

# Skeletal and hormonal responses to sunlight deprivation in Antarctic expeditioners

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## Abstract

**Summary** Serum 25(OH)D levels decline without sunlight exposure. We studied 120 expeditioners to Antarctica to determine the skeletal and hormonal responses to sunlight deprivation. With emerging vitamin D insufficiency, serum calcium decreased, PTH increased, and bone loss at the proximal femur was observed. Baseline serum 25(OH)D levels >100 nmol/L prevented vitamin D insufficiency.

**Introduction** Vitamin D stores deplete without adequate sunlight exposure unless supplementation is provided. We studied 120 healthy adults who spent a year in Antarctica as a model for sunlight deprivation to define the timing and magnitude of the skeletal and hormonal responses to emerging vitamin D insufficiency.

**Methods** Fasting blood samples were assessed at baseline, 6 and 12 months for serum 25-hydroxyvitamin D (25(OH)D), osteocalcin (OC), bone formation (P1NP) and resorption (CTx), PTH and calcium. Lumbar spine

and proximal femur BMD was measured using DXA. Differences over time were determined using repeated measures ANOVA. Percent changes were expressed as  $(\Delta \text{ value}/(\text{value } A + \text{value } B)/2) \times 100$ . Relationships between outcome measures were determined using Spearman's correlations.

**Results** Vitamin D insufficiency (<50 nmol/L) was observed in 85% of expeditioners by 6 months when serum calcium decreased and PTH increased ( $p < 0.01$ ). By 12 months, OC increased by  $7.4 \pm 3.0\%$  ( $p < 0.05$ ), and BMD decreased by  $1.0 \pm 2.0\%$  at the total proximal femur ( $p < 0.05$ ). For those with vitamin D sufficiency at baseline (>50 nmol/L), sunlight deprivation produced vitamin D insufficiency within 4 months unless baseline values were >100 nmol/L.

**Conclusion** Supplementation may be necessary for expeditioners with limited access to UV light.

**Keywords** Bone loss · Sunlight · Vitamin D

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## Introduction

Vitamin D stores become depleted without adequate sunlight exposure unless supplementation is provided. For this reason, vitamin D insufficiency (serum 25(OH)D < 50 nmol/L) is common during winter in many countries, even in those where sunlight exposure is plentiful in summer [1–4]. Little is known about the rate of reduction in serum 25(OH)D during sunlight deprivation and its effects on serum calcium, the emergence of secondary hyperparathyroidism, increased bone remodeling, and subsequent bone loss.

Although bone loss may be partly reversed following re-exposure to sunlight because the remodeling space deficits produced by accelerated remodeling as deficiency appears

is reversed as remodeling slows with repletion, there may be an irreversible component in adults, particularly older adults because of the negative bone balance produced by the reduced volume of bone formed by each basic multicellular unit (BMU) than is resorbed by each BMU [5, 6]. Repeated periods of prolonged sunlight deprivation during adulthood may thus result in permanent bone loss [7].

Expeditioners to Antarctica provide a model for the skeletal and bone metabolic responses to sunlight deprivation. UV exposure from March to August is negligible, and marginal in the shouldering months, which corresponds to autumn (March–May), winter (June–August), and the commencement of spring (September–November) in the southern hemisphere [8]. Extreme conditions also limit skin exposure. Reductions in 25(OH)D have been reported in Antarctic expeditioners; however, skeletal changes are inconclusive [9–12]. We studied Australian Antarctic expeditioners to determine the temporal pattern and magnitude of emerging vitamin D insufficiency, changes in circulating measures of bone remodeling, and rates of bone loss during long-term sunlight deprivation.

## Materials and methods

This prospective study involved 120 healthy adults (19 females, 101 males) free of disease known to affect bone who spent a year at Australian Antarctic or sub-Antarctic coastal stations for the 2004–2005 ( $n=71$ ) or 2005–2006 ( $n=49$ ) seasons. Departures to Antarctica were scheduled between October and March, depending on station destination. Baseline data was collected before departure at the Australian Antarctic Division (Tasmania, Australia) or during the ship voyage to Antarctica. At baseline, each participant completed a standard medical questionnaire. Height ( $\pm 0.1$  cm) was measured using a stadiometer (Medizintechnik, Germany) and weight ( $\pm 0.1$  kg) measured on a beam scale (Wedderburn, UK). Fasting blood samples were taken at baseline, then as near as possible to quarterly (2004–2005 season) or biannually (2005–2006 season). All samples were stored at  $-80^{\circ}\text{C}$  to be assayed in single batches. Dietary intake of macronutrients and calcium was assessed at 6 months ( $n=107$ ) using a 3-day weighed food diary, and analyzed using FoodWorks (version 2.10, Xyris Software). Dietary vitamin D was negligible, as foods were not fortified and deep-sea fish and other vitamin D rich

**Table 1** Characteristics of Australian Antarctic expeditioners

	All $n=120$	25(OH) D > 50 nmol/L ( $n=68$ )	25(OH)D < 50 nmol/L ( $n=52$ )
Anthropometry			
Age (years) (range)	40.3 (21.1–64.4)	41.7 (21.1–64.4)	38.0 (24.1–59.9)*
Sex (no. male/female)	101/19	57/11	44/8
Height (cm)	176.9 $\pm$ 7.9	176.7 $\pm$ 7.6	176.9 $\pm$ 8.5
Weight (kg)	84.6 $\pm$ 13.2	83.2 $\pm$ 11.4	85.9 $\pm$ 14.4
Serum measures			
25(OH)D (nmol/L)	56.8 $\pm$ 20.6	71.3 $\pm$ 14.5	37.8 $\pm$ 8.0**
PTH (pmol/L) <sup>a</sup>	3.9 $\pm$ 1.8	4.1 $\pm$ 2.0	3.6 $\pm$ 1.4
Calcium (mmol/L) <sup>a</sup>	2.42 $\pm$ 0.10	2.39 $\pm$ 0.10	2.46 $\pm$ 0.08*
Calcium (adjusted) (mmol/L)	2.26 $\pm$ 0.11	2.23 $\pm$ 0.10	2.30 $\pm$ 0.08*
Osteocalcin (ng/ml) <sup>a</sup>	23.2 $\pm$ 8.0	22.3 $\pm$ 7.8	24.5 $\pm$ 8.2
CTx (ng/ml) <sup>a</sup>	0.31 $\pm$ 0.14	0.32 $\pm$ 0.15	0.29 $\pm$ 0.13
PINP (ng/ml) <sup>a</sup>	55.2 $\pm$ 19.4	55.4 $\pm$ 20.7	55.3 $\pm$ 17.8
Bone mineral density (g/cm <sup>2</sup> ) <sup>b</sup>			
Total hip	1.08 $\pm$ 0.11	1.08 $\pm$ 0.13	1.07 $\pm$ 0.12
Lumbar spine	1.07 $\pm$ 0.14	1.08 $\pm$ 0.16	1.03 $\pm$ 0.03
Mean daily dietary intake <sup>c</sup>			
Dietary calcium (mg)	749 $\pm$ 508	759 $\pm$ 630	741 $\pm$ 334
Energy intake (kJ)	8999 $\pm$ 328	9125 $\pm$ 390	8462 $\pm$ 466
Protein (g)	100 $\pm$ 30	102 $\pm$ 31	97 $\pm$ 28
Fat (g)	81 $\pm$ 31	82 $\pm$ 35	81 $\pm$ 31
Carbohydrate (g)	253 $\pm$ 110	256 $\pm$ 90	253 $\pm$ 135
Alcohol (g)	20 $\pm$ 25	17 $\pm$ 22	22 $\pm$ 28

\* $p < 0.05$ ; \*\* $p < 0.001$

<sup>a</sup> *R* reference, *M* measurement ranges: PTH (*R* 1.3–7.2 pmol/L), serum calcium (*R* 1.80–2.65 mmol/L), PINP (*M* 5–1200 mg/ml), CTx (*M*: 0.010–6.00 ng/ml), OC (*M*: 0.500–300 ng/ml). Means $\pm$ SD are shown unless otherwise stated

<sup>b</sup>  $n=38$  (2005–2006)

<sup>c</sup> Data collected at 6 months ( $n=107$ )

**Table 2** Means ( $\pm$ SD) absolute values and percentage change from baseline for serum bone metabolic and endocrine markers from 93 Australian Antarctic expeditioners with serial measurements

	Baseline	6 Months	12 months
25-hydroxy vitamin D (nmol/L)	58.8 $\pm$ 20.5	38.0 $\pm$ 16.7*	37.1 $\pm$ 14.3*
PTH (pmol/L)	3.9 $\pm$ 1.8	4.7 $\pm$ 2.2**	4.6 $\pm$ 2.6***
Calcium (mmol/L)	2.42 $\pm$ 0.10	2.36 $\pm$ 0.11**	2.35 $\pm$ 0.10**
Calcium (adjusted) (mmol/L)	2.26 $\pm$ 0.10	2.25 $\pm$ 0.10	2.25 $\pm$ 0.10
Osteocalcin (ng/ml)	23.0 $\pm$ 7.9	23.3 $\pm$ 8.3	25.0 $\pm$ 9.6*****
CTx (ng/ml)	0.30 $\pm$ 0.14	0.31 $\pm$ 0.18	0.34 $\pm$ 0.17*****
PINP (ng/ml)	55.1 $\pm$ 20.2	57.9 $\pm$ 23.0	60.4 $\pm$ 26.0***

Different to baseline \* $p$ <0.001, \*\* $p$ <0.01, \*\*\* $p$ <0.05

Different to 6 months \*\*\*\* $p$ <0.05

sources not staples. Bone mineral density (BMD) at the total proximal femur (neck, trochanter, and proximal shaft) and lumbar spine (L1–4) was assessed at the Menzies Research Institute (Tasmania, Australia) before and after the 2005–2006 expedition using DXA (Hologic Delphi W Version 11.1, CV=1%) Hologic, Waltham, MA, USA.

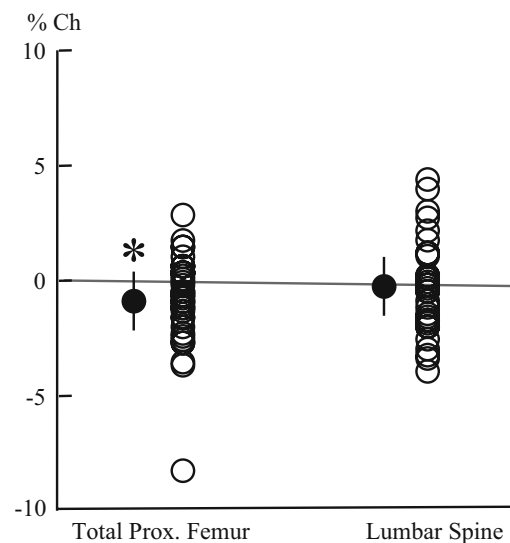
Serum 25(OH)D was assessed using chemiluminescent immunoassay (CLIA; Liason, DiaSorin, Stillwater, USA). Serum 25(OH)D levels of <50 nmol/L are insufficient based on Australian recommendations [13]. Serum N-Mid Osteocalcin (OC), total procollagen type 1 amino-terminal propeptide (PINP), and c-terminal telopeptide (CTx) were measured using the electrochemiluminescence immunoassay (Elecsys 1010 Analytics, Roche Diagnostics, Germany) [14] and intact PTH assessed using CLIA (DPC Immulite 2000, Los Angeles, USA). Serum calcium was assessed by indirect potentiometry (SYNCHRON LX, Beckman Coulter USA). The intra- and interassay CVs for serum measures were 7–13% [15]. Due to late arrivals to, or early departures from Antarctica ( $n=22$ ), dropouts ( $n=3$ ), and field trips ( $n=2$ ), full serum data sets were available from 93 of 120 participants. A total of 38 baseline and final BMD assessments were performed during the 2005–2006 expedition (84% males). One female participant consumed a calcium supplement, four were postmenopausal, two were taking the OCP, and none were on HRT. Mean age for women was 36.7 $\pm$ 9.6 years. Approval for the study was obtained from the Australian Antarctic Division and Austin Hospital Human Research Ethics Committees.

Comparisons between time points (0, 6, 12 months) were determined using repeated measures analysis of variance ( $n=93$ ).  $\Delta$  time=age at time  $B$ –age at time  $A$ . Percent changes were calculated as  $(\Delta \text{ value}/(\text{value } A + \text{value } B)/2) \times 100$ , accounting for regression to the mean and producing a conservative estimate of changes [16]. Correlations between outcome measures were determined using Spearman's correlations to account for skewness of some variables. A curve linear regression was used for changes in serum 25(OH)D [3]. A sample of 35 was needed to detect seasonal

differences in 25(OH)D and 32 to detect changes in BMD with 80% power using a two-tailed test at  $p$ <0.05 [17]. Data were analyzed using StatView (Version 5.0, SAS Institute, Berkeley, USA). Mean $\pm$ SD are presented unless otherwise stated.

## Results

Vitamin D insufficiency (<50 nmol/L) was observed at baseline in 44% of expeditioners and 85% by mid-expedition (6 months), when serum calcium decreased, and PTH increased ( $p$ <0.01), with 10% of participants having PTH levels >7.2 pmol/L (Tables 1 and 2). By 12 months, serum OC increased ( $p$ <0.05), BMD decreased by 1.0 $\pm$ 2.0% at the total proximal femur ( $p$ <0.05), and 25(OH)D remained 37% below baseline (Table 2 and Fig. 1). There was a trend toward increasing PTH levels with time



**Fig. 1** Yearly rate of change (%) in total proximal femur and lumbar spine BMD in 38 healthy adults after prolonged sunlight deprivation during an Antarctic expedition. Open circles represent individual rates of change and the mean $\pm$ SD is represented by the solid circles. \* $p$ <0.05

**Table 3** Correlations between baseline serum markers, changes to 25(OH) vitamin D levels (baseline to 6 months), and annual rate of bone loss at the total proximal femur in expeditioners to Antarctica

	25(OH) D	CTx	P1NP	OC	PTH	Cal	$\Delta$ 25(OH)D	Percent $\Delta$ 25(OH) D
CTx	-0.11	–	–	–	–	–	–	–
P1NP	-0.10	0.49*	–	–	–	–	–	–
OC	0.23**	0.51*	0.70*	–	–	–	–	–
PTH	0.12	-0.03	-0.21***	0.05	–	–	–	–
Cal	-0.40**	0.14	0.17	0.20	-0.37**	–	–	–
$\Delta$ 25(OH) D	-0.65*	-0.17***	0.03	0.12	-0.03	0.17	–	–
% $\Delta$ 25(OH) D	-0.21**	-0.21**	-0.05	-0.01	-0.03	0.02	0.84*	–
Prox. femur	-0.28***	-0.27***	-0.40**	-0.30***	0.04	-0.10	-0.11	-0.05

\* $p < 0.001$ ; \*\* $p < 0.05$ ; \*\*\* $p \leq 0.1$

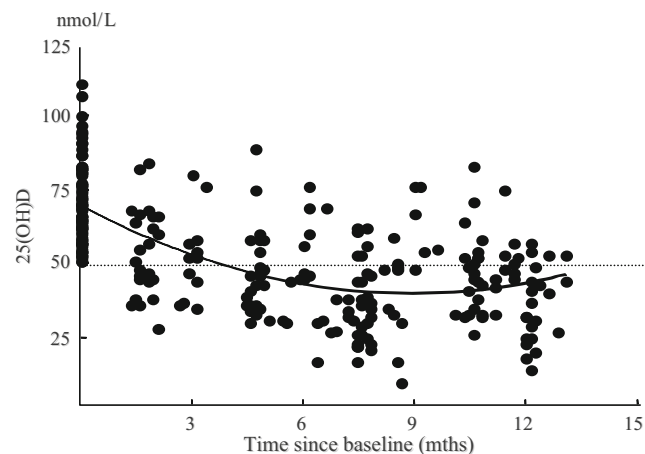
since baseline measure ( $r=0.12$ ,  $p < 0.07$ ), which was significant for those with vitamin D insufficiency at baseline ( $r=0.18$ ,  $p < 0.05$ ). Those with vitamin D insufficiency at baseline were younger and had higher baseline serum calcium levels ( $p < 0.05$ ) (Table 1). Vitamin D insufficiency at baseline was not related to usual residency, occupation, number of prior expeditions, weight, or BMI (data not shown). Those who experienced bone loss at the total proximal femur had higher baseline OC levels ( $23.2 \pm 1.3$  vs  $19.0 \pm 1.3$  ng/ml,  $p < 0.05$ ) and lower calcium intakes ( $678 \pm 49$  vs  $1,189 \pm 345$  mg,  $p < 0.07$ ) compared to those who did not, but they did not differ by age ( $41.5 \pm 9.9$  vs  $44.1 \pm 9.2$  years) or baseline 25(OH)D levels ( $63.4 \pm 23.3$  vs  $53.9 \pm 22.7$  nmol/L). Those with high rates of bone remodeling tended to lose more bone at the total proximal femur (Table 3). For those who were vitamin D replete at baseline (serum 25(OH)D  $> 50$  nmol/L), sunlight deprivation produced vitamin D insufficiency within 4 months unless starting values were greater than 100 nmol/L (Fig. 2).

## Discussion

The conditions experienced by Antarctic expeditioners: limited UV light, protective clothing, unfortified food, and lack of supplementation, provides a model for the study of skeletal and metabolic effects of sunlight deprivation. For all expeditioners, serum 25(OH)D and serum calcium decreased and PTH increased within 6 months of limited UV exposure, but the increase in bone turnover was observed about 6 months later. A reduction in total proximal femur, but not lumbar spine BMD, was observed following the expedition. Those who experienced bone loss had higher initial bone turnover rates and lower dietary calcium intakes. Bone loss was associated with higher bone remodeling. In vitamin D replete adults, sunlight deprivation resulted in insufficiency within 4 months, unless baseline values were greater than 100 nmol/L.

In countries without routine food fortification, periods of immobilization, hospitalization, or institutionalization, especially in the elderly may cause a similar skeletal response to that observed in expeditioners [18]. The effectiveness of sunlight exposure or vitamin D supplementation to correct vitamin D deficiency and reduce fracture risk in the elderly living in aged care is documented, but less is known about the efficacy of supplementation and food fortification in the general population [19, 20]. Controlled sunlight exposure at appropriate times is an effective means of maintaining adequate serum 25(OH)D levels, but is difficult to monitor to prevent overexposure which may, in turn, increase skin cancer risk [21].

Despite the rapid reduction in serum 25(OH)D in the first 6 months and 62% of expeditioners reporting increases in PTH levels, only 10% of expeditioners demonstrated PTH levels above the reference range ( $> 7.2$  pmol/L), and serum calcium remained within normal limits with no detectable change to markers of bone remodeling. An increase in OC was observed at 12 months in this and



**Fig. 2** Changes in serum 25(OH)D with duration of sunlight deprivation during an Antarctic winter in 52 adults who were vitamin D replete ( $> 50$  nmol/L) at baseline

previous Antarctic expeditioners perhaps indicating a new steady state at a higher remodeling rate at this time [11]. Vitamin D supplementation is not routine; therefore, the stabilization of serum 25(OH)D at 12 months may have resulted from UV exposure during the warmer months (November–January) [22].

Total proximal femur BMD, but not lumbar spine bone loss, was observed suggesting that bone loss may proceed by cortical rather than trabecular bone loss due to secondary hyperparathyroidism, as previous studies have reported that cortical bone loss was not always reversed after correction of hyperparathyroidism [5]. Those with bone loss had higher bone turnover at baseline. Baseline OC was associated with higher rates of bone loss over 5 years in older women [23]. Moreover, those who demonstrated bone loss had lower dietary calcium intakes; therefore, supplementation of these individuals may be beneficial in suppressing bone turnover [24]. Despite the high prevalence of vitamin D insufficiency (<50 nmol/L), an association between baseline or change in vitamin D levels and bone loss was not observed, which may be due to the low incidence (<1%) of severe deficiency (<12.5 nmol/L) in this group, or the error associated with serum 25(OH)D measurements [25].

The bone loss observed at the total proximal femur is similar to that reported by others during winter [17]. Wintertime bone loss may be higher in groups living at moderate to high latitudes and in persons with limited sunlight opportunities such as veiled immigrant women, persons with very dark skin, or institutionalized elderly unless they are supplemented. For example in Hobart, Australia (latitude 39°), it is estimated that in winter, to produce sufficient serum 25(OH)D, more than 55 min of UV exposure is needed to produce one sixth–one third MED for people with white skin (Fitzpatrick classification II) with 15% of the body exposed [26]. This is unlikely to be achieved in high-risk groups, and supplementation is an option in such cases [27].

A serum 25(OH)D level >100 nmol/L has been suggested as optimal for numerous health outcomes and was estimated to prevent insufficiency after 4 months of limited UV exposure. However, this level is not often achieved, even during summer [3, 28]. Adequate sunlight exposure during winter is unlikely in regions at latitudes greater than 37°, but wintertime vitamin D insufficiency is also observed in countries with unlimited sunshine and routine food fortification, supporting a global trend toward vitamin D insufficiency [3, 4, 29]. Factors that may contribute to vitamin D insufficiency include the aging of the population, sun protection, and religious practices, migration of people from equatorial regions to more extreme latitudes, and a reduction in sunlight exposure due to pollution [13].

This study is limited by the small sample of women and the wide age range preventing sex comparisons or

investigation of the influence of menopausal status on the observed responses, or if lumbar spine bone loss occurred in women. The bone loss at the total proximal femur was small and of uncertain biological significance. Moreover, follow-up bone density data was unavailable; therefore, the permanency of the bone loss cannot be confirmed. Following up expeditioners in the future may elucidate this.

Vitamin D insufficiency is likely after 4 months of limited sunlight exposure, especially for those with initial serum 25(OH)D levels less than 100 nmol/L. If bone loss is only partly reversed after prolonged sunlight deprivation, then BMD may remain reduced and results in an increased fracture risk. Baseline assessments prior to prolonged sunlight deprivation are advisable, and supplementation may be necessary for those with serum 25(OH)D levels < 100 nmol/L and limited access to UV light.

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**Conflicts of interest** None.

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