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ORIGINAL ARTICLE

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Effect of seasonal ultraviolet radiation fluctuations on vitamin D homeostasis during an Antarctic expedition

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Abstract Antarctica is a unique and challenging environment where members of expeditions face a range of conditions not normally experienced. Ultraviolet (uv) radiation levels show marked variation during the year. The 25-hydroxy metabolite of vitamin D [25(OH)D] is largely produced by sunlight and shows a yearly variation in concentration that corresponds to uv radiation levels. The active metabolite 1,25-dihydroxyvitamin D [1,25(OH)2D] does not generally show any such variation provided 25(OH)D concentrations are sufficient. Previous studies have shown a seasonal variation in 25(OH)D with a significant winter drop. No other study of 1,25(OH)2D has been reported on members of Antarctic expeditions. A group of 19 men wintering at Davis Station (68° 34′ S) had four blood samples taken at 3-monthly intervals beginning in the Antarctic summer. Analysis for 25(OH)D showed a drop in concentration for each of the latter three sampling periods ($P < 0.005$). This correlated with uv radiation levels and would suggest that endogenous production of 25(OH)D ceases for at least the duration of the Antarctic winter. There were no significant alterations in 1,25(OH)2D or calcium concentrations over the same period. Providing that individuals with pre-existing vitamin D deficiencies are detected before departure for Antarctica and missions are limited in duration, clinical deficiency is unlikely to occur.

Keywords Antarctica · Vitamin D · Ultraviolet

Introduction

Vitamin D and its metabolites are important in bone and calcium metabolism. Vitamin D is derived from both the skin (vitamin D_3) and food (vitamin D_2 and some vitamin D_3). The major source of vitamin D in a healthy population has been shown to be from sunlight (Stamp and Round 1974; Chesney et al. 1981).

Vitamin D_3 is produced in the skin when ultraviolet (uv) radiation of wavelength 290–320 nm strikes 7-dehydrocholesterol. Subsequent hepatic hydroxylation produces 25-hydroxyvitamin D [25(OH)D] which undergoes a further renal hydroxylation to produce the active metabolite, 1,25-dihydroxyvitamin D [1,25(OH)2D]. Another major metabolite of vitamin D_3 is 24,25-dihydroxyvitamin D [24,25(OH)2D], also produced largely by renal hydroxylation. Skin production of vitamin D_3 has been shown to be influenced by many factors including latitude, skin melanin content, sunscreen useage and skin exposure (Haddad 1992).

Previous studies have shown a seasonal variation in plasma concentrations of 25(OH)D with low levels in winter and a peak during the summer months when uv radiation levels are maximal (McLaughlin et al. 1974); Stamp and Round 1974; Juttmann et al. 1981). The variation is predominately due to variation in vitamin D_3 concentration, with vitamin D_2 forming a smaller, constant proportion of total vitamin D levels in healthy populations (Chesney et al. 1981). There is a tight regulation of the level of the active metabolite 1,25(OH)2D and, although fewer longitudinal studies have been reported for 1,25(OH)2D, studies in temperate climates of young fit adults with normal 25(OH)D concentrations have shown no seasonal variation in 1,25(OH)2D (Chesney et al. 1981; Tjellesen and Christiansen 1983).

Antarctica is a unique environment imposing many environmental extremes on humans living and working there. One of these is extreme variation in terrestrial uv radiation levels, with a winter period of very low levels of radiation and a summer period of continuous exposure. Because of this it might be expected that seasonal variation in 25(OH)D concentrations would be quite marked. Three previous studies on 25(OH)D

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concentrations in Antarctic populations have shown seasonal variation (Fairney et al. 1979; Griffiths and Fairney 1988, 1989). A fourth study of a Japanese expedition showed markedly low concentrations of 25(OH)D with no seasonal alteration (Shigeno et al 1982). It appears that 1,25(OH)2D concentrations have not previously been studied in this environment.

The aim of this study was firstly to determine the extent to which the extreme variation in uv-radiation levels would be reflected in 25(OH)D concentrations of a larger Antarctic population than had been previously studied and secondly to determine if 1,25(OH)2D concentrations would remain static under such conditions.

Methods

The study was conducted during the 1991 Australian National Antarctic Research Expedition (ANARE). A group of 19 Caucasian men wintering at Davis Station were enrolled and signed informed consent forms after the protocol has been explained to them. The study has been approved by the Ethics Committee of the Australian Antarctic Division. All the members of the expedition underwent a medical assessment prior to selection to exclude significant illness. No subject suffered any known condition that could predispose to abnormal vitamin D metabolism. The average age of the subjects was 33.4 years (range 25–49) with an average body mass of 81 kg (range 66–97). Body mass was measured with a Wedderburn beam balance (Wedderburn Scales, Sydney, NSW) to the nearest 0.1 kg while the subjects were clad in underwear. The station diet was similar to a typical Australian diet and there was no vitamin D supplementation. Previous studies at Davis Station has shown that there was no significant dietary change winter and that expedition members spent sufficient time outdoors with face and occasional hand exposure to sunlight to allow endogenous vitamin D production during the summer months (Lugg 1977; Taylor 1992).

The station is located at latitude 68° 35′ South and longitude 77°58′ East on the edge of the Antarctic continent and has a mean annual minimum temperature of −13.0°C with a mean annual maximum temperature of −7.5°C.

During the study period blood samples were collected from each volunteer at 3-monthly intervals. Four samples were collected in total. The blood was collected in the early morning after an overnight fast, allowed to clot at room temperature and the serum separated before being frozen at −70°C. All the subjects successfully completed the study. At the end of the year the serum was transported at −20°C to Australia for analysis. Calcium concentrations were determined on site prior to freezing the serum. These analyses were performed using a Kodak Ektachem DT60 Analyser DTSC module (Eastman Kodak, Rochester, New York).

All 25(OH)D assays were performed with a commercial kit (Buhlmann Laboratories AG, Allschwil, Switzerland) using a modified method involving initial chromatographic purification of serum followed by a competitive protein binding assay (Dean et al. 1988). The 1,25(OH)2D concentrations were determined using a commercial kit produced by Immuno Nuclear Corporation (now Inc Star, Stillwater, Minnesota).

Solar radiation data were collected using an International Light uv-B (280–315 nm) detector (International Light, Newburyport, Mass) incorporating a solar blind vacuum photo-diode, uv-B filter and quartz diffuser. The detector was connected to a datalogger which stored data as 10-min averages. Statistical analyses were performed using a repeated measures design (Hicks 1982).

Table 1 Serum 25-hydroxyvitamin D [25(OH)D], 1,25-dihydroxyvitamin D [1,25(OH)2D] and calcium concentrations of expedition members during a year in Antarctica. Normal ranges of 25(OH)D, 1,25(OH)2D and calcium are given in brackets

Month	25(OH)D (28–165 nmol·l^{-1})		1.25(OH)2D (34–134 pmol·l^{-1})		Calcium (2.27–2.64 mmol·l^{-1})	
	mean	SD	mean	SD	mean	SD
December	108	35	47	10	2.37	0.07
March	91	25	48	13	2.37	0.07
June	87	20	50	13	2.41	0.08
September	75	22*	49	11[a]	2.38	0.07[a]

*$P < 0.005$ for trend test of quarterly averages
[a]not significant

Results

Serum 25(OH)D concentrations declined throughout the study, with trend analysis showing a significant fall ($P < 0.005$) over the duration of the study (Table 1). All results remained within the normal range throughout. All expedition members had left Australia in either October or November during the Southern spring and had therefore been out of Australia for 1 to 2 months prior to the first sample being taken. The largest 3-monthly 25(OH)D concentration decrease was during the first 3 months of the study when the study population has only been in Antarctica for 3 to 4 months. Each of the subsequent two sampling periods showed smaller but consistent falls.

The uv radiation data (Fig. 1) showed a decrease after summer to very low levels during the winter. In the middle of the antarctic summer a 6-week period of continuous sunlight accounts for high uv levels which then fall to a nadir in winter when the sun does not rise (in June the average daily uv B radiance was zero). The early drop in 25(OH)D occurred during a period of rapidly falling uv radiation levels. The uv levels in the month before the March, June and September 25(OH)D samples were less than 40%, 2% and 18% of the summer uv peak, respectively.

There were no significant alteration in 1,25(OH) 2D concentrations during the study. Over this time the 25(OH)D concentrations dropped on average by 30% from the summer peak. Calcium concentrations likewise did not show any significant alteration. Both 1,25(OH)2D and calcium concentrations remained within the normal range throughout.

Discussion

The dramatic change in uv radiation levels at extreme latitudes was well demonstrated in this study. As the diet was not supplemented with vitamin D and dietary intake did not change during the year (Taylor

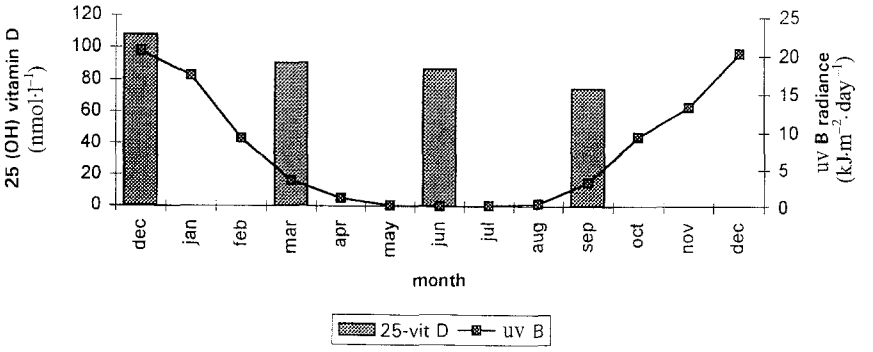

Fig. 1 The 25-hydroxyvitamin D [25(OH)D] concentrations in blood and average total daily ultraviolet (uv) B radiation at different months of the year.

1992), changes in the serum concentration of 25(OH)D should have reflected changes in uv B exposure. The half life of 25(OH)D is 3 weeks and blood samples have been shown to reflect the level of uv exposure in the previous month (Wilske 1993). The falling concentrations of 25(OH)D in March, June and September suggest that uv B exposure in the month prior to each sample was insufficient to maintain vitamin D concentration. The fall in March probably reflected both a falling uv level and the fact that the local climate discouraged skin exposure to whatever uv may have been available. The uv levels prior to the June and September samples were extremely low. Human exposure to the outdoor climate is essentially zero at this time of year. It is therefore reasonable to assume that at least in the period from March to September endogenous production of 25(OH)D was minimal or nonexistant. It has previously been suggested that endogenous vitamin D_3 production ceases for a 6-month period over winter at extreme latitudes (McKenna 1992).

Three of the four previous studies in the Antarctic that have been able to continue into the following summer have shown an increase in 25(OH)D concentrations with the onset of summer, with a peak during or after the summer uv radiation maxima (Fairney et al. 1979; Griffiths and Fairney 1988, 1989). It is likely that this would also have occurred in this study had it been possible to prolong the period during which samples were taken. Because the local conditions tended to restrict the degree of skin exposure, it is unclear whether 25(OH)D concentrations would have risen to the peak of the previous summer. Nonetheless, as it is uncommon for expedition members to spend more than 18 months in Antarctica, clinical deficiency is unlikely to become a problem.

Despite both a 30% drop in concentration during the study and a winter cessation of endogenous production of 25(OH)D, 1,25(OH)2D levels did not alter significantly. Although never before studied in an ANARE population the result is consistent with previous findings in more temperate climates of tight regulation of 1,25(OH)2D concentrations (Chesnay et al. 1981; Tjellesen and Christiansen 1983). It has been found that conversion of 25(OH)D to 1,25(OH)2D is controlled directly and indirectly by calcium, phosphate and 1,25(OH)2D concentrations and 25(OH)D would only be expected to limit 1,25(OH)2D production when it is causing a substrate deficiency (DeLuca and Schnoes 1976). While the exact levels at which this becomes a factor are uncertain, it has been reported that 25(OH)D concentrations, must fall below 5 $nmol \cdot l^{-1}$ before calcium and phosphate concentrations are affected, but that 25(OH)D may limit 1,25(OH)2D production via substrate deficiency at higher levels-possibly as high as 75 $nmol \cdot l^{-1}$ (McKenna 1992). Although average 25(OH)D concentrations dropped by 30%, the minimum levels were still well within the normal range and calcium concentrations were unchanged throughout the study. It is known that in vitamin D deficient states the ratio of 24,25(OH)2D to 1,25(OH)2D falls as relatively more vitamin D undergoes 1-hydroxylation. It may be that 1,25(OH)2D concentrations were maintained in this population despite falling 25(OH)D concentration by a relative increase in 1-hydroxylation to 24-hydroxylation.

Antarctica is a unique environment in which to work and can be difficult and at times dangerous. The dramatic change in uv radiation levels during each year is but one component of this. Despite dramatic changes in uv B levels and subsequent changes in 25(OH)D concentrations during the Antarctic year, 1,25(OH)2D homeostasis would seen to be maintained.

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