Season and Ethnicity Are Determinants of Serum 25-Hydroxyvitamin D Concentrations in New Zealand Children Aged 5–14 y[1,2]

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ABSTRACT New Zealand children, particularly those of Māori and Pacific ethnicity, may be at risk for low vitamin D status because of low vitamin D intakes, the country's latitude (35–46°S), and skin color. The aim of this study was to determine 25-hydroxyvitamin D concentrations and their determinants in a national sample of New Zealand children aged 5–14 y. The 2002 National Children's Nutrition Survey was designed to survey New Zealand children, including oversampling of Māori and Pacific children to allow ethnic-specific analyses. A 2-stage recruitment process occurred using a random selection of schools, and children within each school. Serum 25-hydroxyvitamin D concentration [mean (99% CI) nmol/L] in Māori children (n = 456) was 43 (38,49), in Pacific (n = 646) 36 (31,42), and in New Zealand European and Others (NZEO) (n = 483) 53 (47,59). Among Māori, Pacific, and NZEO, the prevalence (%, 99% CI) of serum 25-hydroxyvitamin D deficiency (<17.5 nmol/L) was 5 (2,12), 8 (5,14), and 3 (1,7), respectively. The prevalence of insufficiency (<37.5 nmol/L) was 41 (29,53), 59 (42,75), and 25 (15,35), respectively. Multiple regression analysis found that 25-hydroxyvitamin D concentrations were lower in winter than summer [adjusted mean difference (99% CI) nmol/L; 15 (8,22)], lower in girls than boys [5 (1,10)], and lower in obese children than in those of "normal" weight [6 (1,11)]. Relative to NZEO, 25-hydroxyvitamin D concentrations were lower in Māori [9 (3,15)] and Pacific children [16 (10,22)]. Ethnicity and season are major determinants of serum 25-hydroxyvitamin D. There is a high prevalence of vitamin D insufficiency in New Zealand children, which may or may not contribute to increased risk of osteoporosis and other chronic disease. There is a pressing need for more convincing evidence concerning the health risks associated with the low vitamin D status in New Zealand children. J. Nutr. 135: 2602–2608, 2005.

KEY WORDS: • *25-hydroxyvitamin D* • *children* • *New Zealand* • *Māori* • *Pacific*

Vitamin D plays an essential role in calcium and phosphorus homeostasis. The most serious clinical consequence of vitamin D deficiency in children is rickets, a condition characterized by soft and weakened bones, resulting from poor mineralization of newly formed bone tissue (1). Although still uncommon in Western countries, it would appear from the increasing number of case reports being published that rickets is reemerging as a public health problem (2–4). Lesser degrees of vitamin D deficiency, often referred to as insufficiency, are associated with lower bone mineral density (BMD)[4] (5) and bone accretion rates in children (6), as well as elevated serum parathyroid (PTH) hormone concentrations (5,7); these effects are consistent with secondary hyperparathyroidism. Recent discoveries indicate that vitamin D has functions unrelated to calcium, specifically in cell differentiation and in the immune system (8,9). These biological effects add plausibility to reports that low vitamin D status is associated with increased risk of childhood-onset Type 1 diabetes (10) and increased risk of some types of cancer in adults (11–13).

This widening spectrum of diseases in which vitamin D may play an etiological role and the reemergence of rickets give greater relevance and priority to monitoring the vitamin D

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[4] Abbreviations used: BMD, bone mineral density; CNS02, 2002 National Children's Nutrition Survey; NHANES, National Health and Nutrition Examination Survey; NZEO, New Zealand European and Others; PTH, parathyroid hormone.

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status of populations, particularly children. The best indicator of vitamin D status is the concentration of circulating 25-hydroxyvitamin D. The determinants of serum 25-hydroxyvitamin D concentrations in children include dietary vitamin D intake, which in the absence of fortified foods or supplements is generally low, and conditions that affect the synthesis of vitamin D in the skin such as skin color and sun exposure; the latter are affected by season, geographic latitude, sunscreen use, and clothing (1). Obesity has been associated with lower vitamin D status in adults (14,15), an effect that may be stronger in young than older adults (16). The effect of obesity on the vitamin D status of children is not clear.

There have been several community or clinic-based studies of the vitamin D status of children (5,17–23), but few studies have reported vitamin D status of a nationally representative group of children (24,25). Results from the 3rd U.S. National Health and Nutrition Examination Survey (NHANES III) indicated that the prevalence of insufficient serum 25-hydroxyvitamin D (<37.5 nmol/L) in the 12- to 19-y-old U.S. population ranged from 2 to 12%, although this is likely to be an underestimate because vitamin D status was measured in the northern United States during summer and in the southern United States during winter. These rates prevail despite the widespread fortification of milk and breakfast cereals in the United States (18,24).

We present the serum 25-hydroxyvitamin D results from the Children's Nutrition Survey (CNS02), a large national survey of New Zealand school-aged children in 2002. The vitamin D analysis was initiated in response to evidence from Tasmania, Australia (42°S) that there was a high prevalence of low vitamin D status among children and adolescents (17). New Zealand lies geographically from ~35°S to ~47°S (equivalent to Duluth, MN, and Los Angeles, CA) and has a food supply with no mandatory and minimal voluntary vitamin D (some margarines and some edible fat spreads) fortification. Furthermore, there are 3 main ethnic groups with varying skin color: Māori, Pacific, and New Zealand European. Moreover, the survey provides a unique opportunity to explore the independent effects of season, age, ethnicity, and obesity on serum 25-hydroxyvitamin D concentrations in children 5–14 y old.

SUBJECTS AND METHODS

Study design and population. The 2002 National Children's Nutrition Survey (CNS02) was a cross-sectional survey of a national sample of New Zealand school children and adolescents aged 5 to 14 y, conducted during the 2002 school year. A school-based sampling frame of children was used with oversampling of Māori and Pacific children to allow for ethnic-specific analysis. The CNS02 aimed to recruit 3000 children with 1000 from each of 3 ethnic groups: Māori, Pacific, and New Zealand Europeans and Others (NZEO). After exclusion of some schools for reasons of cost (schools on the Chatham Islands, correspondence schools, and schools with <50 students), 160 schools were randomly selected from Ministry of Education rolls. Because 16 schools declined to participate, a further sample of 30 schools was selected, with 2 schools declining. The final sample was selected from 172 schools. Recruitment from each school was in proportion to the number of students on the school roll. Children from selected schools were assigned to 1 of 3 ethnic groups with a different probability of selection for each ethnic group. Details of the survey methodology are described more fully elsewhere (26). Although the survey sample covered rural and urban areas across New Zealand, blood samples were not collected from rural areas due to concerns about blood stability and cost. The total number of children invited to participate was 4728; of these, 3275 participated and 1927 provided blood samples. Blood was available for vitamin D analysis for 1659 participants. Of these, 1585 children had all data available that were relevant to this study, yielding an overall response rate of 33.5%. The survey received ethical approval from the Auckland Ethics Committee. Informed written consent was obtained from both the children and their parents or guardians.

Demographic data were obtained from interviews, at children's homes in the presence of their parents or guardians, although some took place at school. Interviews were conducted from the last week of February 2002 through the second week of December 2002. In this paper, the term "Pacific" includes children who identify as Samoan (55%), Tongan (24%), Cook Island Māori (16%), Niuean (10%), Tokelauan (2%), Fijian (3%), and other Pacific ethnic groups (3%). The New Zealand European and Others (NZEO) group included: New Zealand European (80%), Asian (10%), Other European (6%), Indian (5%), and other (4%). In cases in which the child or parent/guardian indicated that they belonged to more than one ethnic group, a single ethnic category was assigned to the child using a priority system as follows: if Māori was one of the groups reported, the participant was assigned to Māori. If Māori was not reported, but any of the Pacific groups were reported, the participant was assigned to Pacific. All remaining participants were assigned to NZEO.

Anthropometric measurements and blood samples were taken at each child's school. Height and weight measurements were taken using portable standardized equipment. BMI was calculated as weight in kilograms divided by height in meters squared. Prevalences of overweight and obesity were determined using the reference cutoff values of Cole et al. (27). Blood samples were collected by trained phlebotomists. The children were not instructed to fast. Blood was drawn from an antecubital vein into vacuum-evacuated tubes. Blood samples were centrifuged (2500 × *g*, 7 min), the serum was removed, and put into cryovials; surplus blood was stored at −80°C for subsequent analyses. The samples underwent no freeze-thaw cycles and were stored for up to 18 mo before being analyzed for 25-hydroxyvitamin D.

Serum 25-hydroxyvitamin D analysis. Serum 25-hydroxyvitamin D was determined on surplus blood using an RIA kit (DiaSorin) that recognizes both cholecalciferol and ergocalciferol equally and measures total 25-hydroxycholecalciferol (25-hydroxyvitamin D). Two levels of control provided by the manufacturer were run in each assay. Inter- and intra-assay CVs based on repeated analysis of a pooled control were 13 and 9%, respectively. The sensitivity of the assay is 7 nmol/L. Certified reference material for serum 25-hydroxyvitamin D is not available; however, to verify the relative accuracy of our results, we sent aliquots of 20 randomly selected samples to the Steroid Laboratory of Canterbury Health Laboratories. This laboratory, using the same method, reported a mean serum 25-hydroxyvitamin D concentration of 51 nmol/L, compared with our measured mean of 53 nmol/L. Using a paired *t* test, the difference was [mean (95% CI)] 1.7 (−4.2, 7.5). The Bland and Altman test showed the 95% limits of agreement were −23.4, 26.7.

Data analysis. Statistical analyses were carried out using STATA 8.0, adjusting for the complex survey design. Tests for independence were used to examine the association between those who did and did not give blood and demographic variables such as gender and ethnicity. The χ^2 statistic was corrected for the survey design and converted into an F-statistic. Sampling weights were used in all analyses to obtain unbiased estimates of population serum vitamin D concentrations. Sampling weights were based on the provision of a blood sample rather than participation in the survey because only children from urban areas were sampled for blood. Sample weights were based on the inverse probability of selection, and adjusted for differential nonresponse and poststratification by age, sex, and ethnicity. Because age, sex, ethnicity, season, geographical location, and obesity were reported to affect serum 25-hydroxyvitamin D concentrations, we used multiple linear regression models to examine the independent relations between each of these variables and serum 25-hydroxyvitamin D. We estimated adjusted means based on these models. Because the prevalence of deficiency was <10%, Poisson regression was used to estimate the prevalence (99% CI) for the groups of interest. Logistic regression was used for estimating prevalence of insufficiency. For the purposes of analysis, the months of April through September were defined as "winter" months, and October to December, and March, defined as "summer" months. We

used Poisson regression to identify characteristics predicting risk of vitamin D deficiency (serum 25-hydroxyvitamin D < 17.5 nmol/L) and logistic regression for predictors of risk insufficiency (<37.5 nmol/L) so that relative risks (for Poisson) and odds ratios were obtained. There were no significant interactions in the multivariate regression models. Estimates were considered significant if $P < 0.01$.

RESULTS

Characteristics of the children for whom a serum 25-hydroxyvitamin D concentration was obtained ($n = 1585$) were compared with those of all participants in the Children's Nutrition Survey ($n = 3275$) (**Table 1**). The distribution of sex, age, season, and overweight or obese did not differ between these groups. The vitamin D subgroup comprised only children from urban schools, and there were greater proportions of Pacific ($\chi^2 = 60.7$, $P = 0.0002$) and North Island ($\chi^2 = 95.5$, $P = 0.0005$) children than in the survey group.

New Zealand children had a mean serum 25-hydroxyvitamin D concentration of 50 nmol/L, with mean concentrations in subgroups ranging from 32 nmol/L in Pacific girls aged 11–14 y, to 62 nmol/L in NZEO boys aged 5–6 y (**Table 2**). Of New Zealand children, 4% were vitamin D deficient (<17.5 nmol/L), and nearly one third were insufficient (<37.5 nmol/L). The prevalence of vitamin D deficiency ranged from 0% in NZEO boys and girls 5–6 y old, to 13% in Pacific girls 11–14 y old. Similarly, the prevalence of vitamin D insufficiency ranged from 13% in NZEO boys 5–6 y old to 71% in Pacific girls 11–14 y old. Percentiles of serum 25-hydroxyvitamin D by ethnicity and sex are presented in **Table 3**. In "winter" months, the unadjusted prevalence of deficiency was 5%, and for insufficiency, 43%; in "summer" months, they were 2 and 16%, respectively.

When adjusted for all other variables [age, ethnicity, latitude (North vs. South Island), season ("summer" vs. "winter" months), and overweight/obesity], boys had higher 25-hydroxyvitamin D concentrations than girls by [adjusted mean difference (99% CI)] 5 (1,9) nmol/L (**Table 4**). The effect of age category on serum 25-hydroxyvitamin D was not significant. Ethnicity was a strong determinant of 25-hydroxyvita-

TABLE 1

Characteristics of all participants in the Children's Nutrition Survey 2002 and participants with a serum 25-hydroxyvitamin D result

Characteristic	All survey participants[1]	Participants with a 25-hydroxyvitamin D result
	n (%)	
All	3275	1585
Sex		
Boys	1697 (52)	801 (51)
Girls	1578 (48)	784 (49)
Age, *y*		
5–6	692 (21)	294 (19)
7–10	1425 (44)	722 (46)
11–14	1158 (35)	569 (36)
Ethnicity		
Māori	1224 (37)	456 (29)
Pacific	1058 (32)	646 (41)*
NZEO	993 (30)	483 (30)
Region		
South Island	538 (16)	174 (11)
North Island	2737 (84)	1411 (89)*
Season[2]		
Winter	1994 (61)	924 (58)
Summer	1281 (39)	661 (42)*
Obesity[3]		
Obesity	517 (17)	288 (18)
Overweight	789 (26)	422 (27)
Normal weight	1743 (57)	875 (55)
School		
Urban	2792 (85)	1585 (100)
Rural	483 (15)	0 (0)

[1] In the Children's Nutrition Survey, a participant was defined as a child or adolescent who completed a 24-h recall.
[2] "Winter" months: April–September; "Summer" months March, October–December.
[3] Normal weight, overweight, or obese classification according to Cole et al. (27).
* Difference between participants with and without a blood result for serum 25-hydroxyvitamin D, χ^2 test, $P < 0.01$.

TABLE 2

Serum 25-hydroxyvitamin D concentrations and prevalence of deficiency and insufficiency, in New Zealand children and adolescents by age, sex and ethnicity[1,2,3]

Sex, age and ethnicity	*n*	Serum 25-hydroxy vitamin D	Deficient	Insufficient
		nmol/L	%	
All children	1585	50 (45, 54)	4 (2, 6)	31 (22, 40)
Boys	801	52 (47, 58)	3 (1, 6)	27 (17, 37)
5–6 y	157	57 (49, 65)	1 (0, 5)	19 (7, 32)
7–10 y	360	53 (48, 58)	3 (1, 8)	24 (15, 33)
11–14 y	284	50 (41, 59)	4 (2, 10)	33 (16, 50)
Girls	784	47 (41, 53)	4 (2, 9)	36 (24, 47)
5–6 y	137	48 (43, 54)	1 (0, 6)	29 (15, 43)
7–10 y	362	51 (44, 58)	2 (1, 7)	31 (19, 44)
11–14 y	285	42 (33, 51)	7 (3, 17)	43 (24, 63)
Māori				
All	456	43 (38, 49)	5 (2, 12)	41 (29, 53)
Boys	232	47 (40, 53)	4 (2, 10)	36 (21, 50)
5–6 y	52	48 (41, 55)	4 (0, 26)	26 (6, 46)
7–10 y	116	47 (40, 54)	4 (1, 13)	38 (20, 56)
11–14 y	64	45 (34, 57)	4 (1, 2)	40 (18, 62)
Girls	224	40 (35, 45)	7 (3, 18)	46 (33, 59)
5–6 y	50	42 (36, 48)	4 (1, 23)	45 (24, 65)
7–10 y	116	46 (40, 52)	2 (0, 13)	33 (17, 48)
11–14	58	35 (28, 42)	12 (3, 43)	58 (36, 79)
Pacific				
All	646	36 (31, 42)	8 (5, 14)	59 (42, 75)
Boys	297	38 (33, 44)	7 (4, 15)	53 (34, 72)
5–6 y	64	42 (34, 49)	6 (1, 25)	49 (28, 70)
7–10 y	129	40 (34, 46)	6 (2, 17)	50 (29, 71)
11–14 y	104	36 (30, 42)	10 (3, 30)	61 (41, 81)
Girls	349	34 (29, 40)	9 (5, 17)	64 (49, 79)
5–6 y	47	39 (31, 47)	3 (1, 19)	47 (27, 68)
7–10 y	145	34 (28, 40)	8 (3, 23)	66 (49, 83)
11–14 y	157	32 (25, 39)	13 (7, 24)	71 (54, 88)
NZEO				
All	483	53 (47, 59)	3 (1, 7)	25 (15, 35)
Boys	272	56 (49, 63)	2 (1, 8)	21 (9, 33)
5–6 y	41	62 (52, 72)	0 (0, 0)	13 (0, 27)
7–10 y	115	56 (50, 63)	2 (0, 14)	17 (8, 26)
11–14 y	116	53 (41, 65)	3 (1, 16)	28 (6, 50)
Girls	211	50 (43, 57)	3 (1, 12)	29 (16, 42)
5–6 y	40	52 (45, 59)	0 (0, 0)	20 (3, 38)
7–10 y	101	54 (45, 64)	1 (1, 17)	27 (12, 42)
11–14 y	70	45 (34, 56)	5 (1, 25)	36 (13, 59)

[1] Values are means (99% CI) or % (99% CI). Deficient: <17.5 nmol/L; insufficient: <37.5 nmol/L.
[2] All data were adjusted for survey weighting.
[3] Data rounded to whole numbers.

TABLE 3

Serum 25-hydroxyvitamin D by centile in New Zealand children and adolescents by sex and ethnicity[1]

Sex, ethnicity	*n*	Percentile of serum 25-hydroxyvitamin D (nmol/L)								
		1st	5th	10th	25th	50th	75th	90th	95th	99th
All children										
All	1585	13	20	23	34	47	62	77	89	111
Boys	801	13	21	25	36	51	65	81	89	116
Girls	784	12	18	22	32	44	56	74	89	111
Māori										
All	456	13	17	20	29	43	55	68	74	85
Boys	232	12	19	22	30	48	60	71	76	84
Girls	224	15	17	19	26	40	51	63	69	87
Pacific										
All	646	13	16	18	24	34	44	57	67	91
Boys	297	13	16	19	26	35	46	61	75	103
Girls	349	12	15	18	23	32	42	52	64	88
NZEO										
All	483	12	22	26	37	50	65	83	95	120
Boys	272	16	24	28	39	53	68	83	96	120
Girls	211	12	21	24	35	48	60	78	92	111

[1] All data were adjusted for survey weighting.

min D concentration; Māori children were 7 (2,11) nmol/L and Pacific children 15 (11,20) nmol/L lower than NZEO children. Children living in the South Island (40–47°S) and the North Island (35–40°S) did not differ. Obese children had a 7 (2,11) nmol/L lower serum 25-hydroxyvitamin D concentration than normal weight children. There was a marked independent effect of season on mean 25-hydroxyvitamin D concentrations, with a difference of 15 (9,21) nmol/L between "winter" and "summer" (Table 4). Mean 25-hydroxyvitamin D concentrations adjusted for age, sex, ethnicity, latitude, and overweight/obesity, fell from a peak in March of 69 (58,80) nmol/L to a nadir in August of 36 (32,40) nmol/L before rising through the end of the year (**Fig. 1**). Unadjusted means differed little from adjusted means when analyzed by month.

The results of logistic regression analysis indicated that ethnicity and season, but not age, sex, latitude, or obesity were significant determinants of having insufficient vitamin D status (Table 4). In the fully adjusted model, the prevalence of insufficiency among Māori children was 15% higher than in NZEO children, which translates into an odds ratio of 1.63 ($P = 0.006$). Pacific children had a 35% higher prevalence of insufficiency than NZEO children (OR = 2.49, $P < 0.001$). The prevalence of insufficiency in "winter" months was 27% higher than in "summer" months (OR = 2.88, $P < 0.001$). Age was the only significant independent predictor for the prevalence of deficiency. Children 11–14 y had a 3% greater prevalence of deficiency than children aged 5–6 y (RR = 4.35, $P = 0.002$).

DISCUSSION

We found that 1 in 25 (4%) New Zealand children aged 5–14 y had a serum 25-hydroxyvitamin D concentration low enough (<17.5 nmol/L) to be classified as vitamin D deficient and 31% as insufficient (<37.5 nmol/L). Vitamin D deficiency in children is generally defined as a concentration of serum 25-hydroxyvitamin D below which there is overt evidence of compromised skeletal health such as the presence of rickets, most common in infancy and at puberty, other radiological evidence of poor bone development, or marked biochemical abnormalities associated with metabolic bone disease (28).

TABLE 4

Adjusted serum 25-hydroxyvitamin D concentrations and prevalence of deficiency and insufficiency in New Zealand children and adolescents[1–4]

	n	Serum 25-hydroxy vitamin D	Deficient	Insufficient
		nmol/L		%
Sex				
Boys	801	52 (48, 56)[a]	2 (1, 4)	24 (18, 33)
Girls	784	47 (43, 51)[b]	3 (1, 6)	33 (24, 43)
Age, *y*				
5–6	294	52 (47, 58)	1 (0, 3)[a]	21 (13, 32)
7–10	722	51 (47, 55)	2 (1, 4)	25 (18, 34)
11–14	569	47 (41, 52)	4 (2, 9)[b]	35 (23, 49)
Ethnicity				
Māori	456	44 (39, 48)[a]	4 (1, 11)	38 (26, 52)[a]
Pacific	646	37 (34, 40)[b]	7 (4, 14)	58 (48, 67)[b]
NZEO	483	53 (48, 57)[c]	2 (1, 4)	23 (16, 32)[c]
Region				
South Island	174	48 (43, 53)	2 (0, 8)	27 (15, 45)
North Island	1411	50 (45, 54)	3 (1, 5)	29 (21, 38)
Season[5]				
Winter	924	43 (39, 47)[a]	4 (2, 7)	42 (32, 53)[a]
Summer	661	58 (52, 64)[b]	1 (0, 4)	14 (9, 24)[b]
Obesity[6]				
Obesity	288	44 (39, 49)[a]	2 (1, 4)	42 (26, 60)
Overweight	422	50 (45, 54)[b]	1 (1, 3)	28 (18, 41)
Normal weight	875	50 (46, 54)[b]	3 (1, 6)	27 (20, 35)

[1] Values are adjusted means (99% CI) or adjusted prevalence [% (99% CI)]. Estimates within a column subgroup not sharing a common superscript letter differ, $P < 0.01$.

[2] Deficient: <17.5 nmol/L; insufficient: <37.5 nmol/L.

[3] All data were adjusted for survey weighting and rounded to whole numbers.

[4] Estimates adjusted for age, sex, ethnicity, season, region, and obesity, using multiple linear, Poisson, and logistic regression where appropriate.

[5] "Winter" months: April–September; "Summer" months March, October–December.

[6] Normal weight, overweight, or obese classification according to Cole et al. (27).

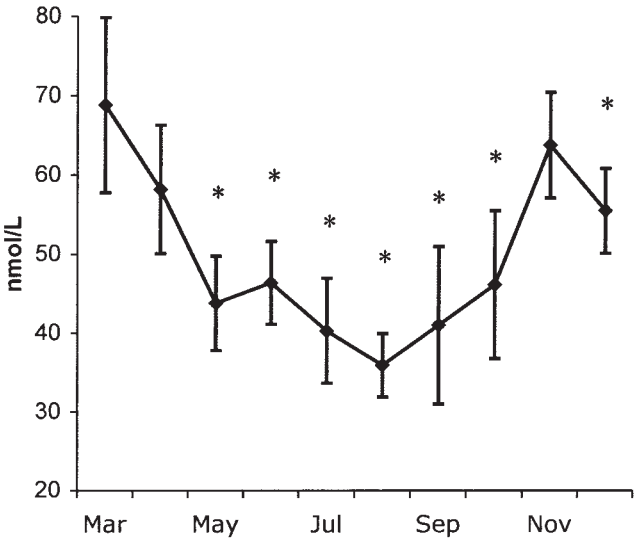

FIGURE 1 Serum 25-hydroxyvitamin D concentrations by month, of New Zealand children and adolescents (n = 1583) who participated in the 2002 National Children's Nutrition Survey. Values are means (99% CI) adjusted for age, sex, ethnicity, latitude, and obesity using multiple linear regression, and weighted to account for the complex survey design. *Different from March, $P < 0.01$.

Cutoff values indicative of vitamin D deficiency in children, based on 25-hydroxyvitamin D, range from 10 to 25 nmol/L (1,21). A cutoff value of 17.5 nmol/L was used to define vitamin D deficiency, in adolescents and adults in the U.S. NHANES III (24).

There is a continuum of increasing risk of compromised growth and development in children as serum 25-hydroxyvitamin D concentration decreases from sufficiency to deficiency. Although there is no current consensus on the optimal definition of vitamin D insufficiency, studies (primarily of older adults) suggest that higher serum 25-hydroxyvitamin D concentrations (50–80 nmol/L) (29) may be necessary to prevent secondary hyperparathyroidism (30–32), increased bone loss (33), or fracture (34) and to maximize fractional calcium absorption (35). Because older people tend to have a reduced capacity for intestinal calcium absorption (36) and may be more resistant to vitamin D than children, applying adult cutoff values to children and adolescents may be inappropriate.

Health indicators used to define vitamin D insufficiency in children include minimization of PTH concentrations and maximization of bone mass. Estimates of the serum 25-hydroxyvitamin D concentration indicative of insufficiency in children based on suppression of PTH range from 30 to 83 nmol/L (5,19,22). Defining vitamin D insufficiency by PTH suppression should be interpreted with caution because at higher calcium intakes, absorption of calcium is sufficient to maintain normal serum calcium and PTH is not elevated. The daily calcium intake of New Zealand children (mean ± SEM) was 767 ± 13 mg; however, in Pacific children, it was 558 ± 28 mg. Achieving peak bone mass during childhood is important for the prevention of osteoporosis later in life. Vitamin D insufficiency during childhood may limit the attainment of peak bone mass (37), but there is scant information on what constitutes an optimal serum 25-hydroxyvitamin D concentration to achieve this. In the study of Cheng et al. (5), girls with 25-hydroxyvitamin D concentrations < 25 nmol/L had lower cortical volumetric BMD of the distal radius and tibial shaft than girls with concentrations > 25 nmol/L. Another Finnish study of girls aged 14–16 y found serum concentrations < 40 nmol/L concomitant with low forearm BMD (7). Defining cutoff values for vitamin D insufficiency is further complicated by differences in values obtained by different 25-hydroxyvitamin D assays (38). Cognizant of the uncertainty of what defines vitamin D insufficiency, we chose a 25-hydroxyvitamin D value of 37.5 nmol/L as a cutoff value for vitamin D insufficiency.

Two large nationally representative surveys also reported serum 25-hydroxyvitamin D concentrations in children and adolescents; the NHANES III included data for adolescents 12–19 y (24) and the UK National Diet and Nutrition Survey (NDNS) included data on children 4–14 y (25). It would appear that the vitamin D status of UK children and adolescents is somewhat better, and of U.S. adolescents substantially better, than that of New Zealand children and adolescents. Data from NHANES III are confounded by latitude and season. Nevertheless, mean serum 25-hydroxyvitamin D in the winter in the American South (25–41°N) was 64.9 nmol/L in females and 78.6 nmol/L in males; <1% of 12–19 y olds had a serum 25-hydroxyvitamin D < 17.5 nmol/L and only 5 and 12% of males and females, respectively, had serum concentrations < 37.5 nmol/L. In NZ in the winter months (April-September), the mean serum 25-hydroxyvitamin D in children and adolescents 5–14 y was 43 nmol/L with 5 and 43% having vitamin D concentrations < 17.5 and < 37.5 nmol/L, respectively. In the UK NDNS, the mean serum 25-hydroxyvitamin D for children aged 4–10 y was ~70 nmol/L and for ages 11–14 y the mean was 56 nmol/L. Almost no British children were defined as vitamin D deficient, based on a cutoff value of <12 nmol/L. The prevalence of insufficiency was lower than in NZ children; 8–15% of UK children aged 4–10 y were vitamin D insufficient (<40 nmol/L). In the 11–14 y age group, UK and NZ boys were similar but 30% of UK girls were vitamin D insufficient (<40 nmol/L) (25), compared with 43% of NZ girls aged 11–14 y having concentrations < 37.5 nmol/L. The better vitamin D status of British and U.S. than New Zealand children probably reflects higher consumption of vitamin D–fortified foods such as fortified milks and breakfast cereals, which are readily available in the U.S. and UK but not in New Zealand.

The 2 strongest determinants of serum 25-hydroxyvitamin D concentrations in New Zealand children were season and ethnicity. The decline of 25-hydroxyvitamin D concentrations by 30 nmol/L (a 50% decline) between March and August is typical of differences reported in regions of similar or higher latitude (39–41). Because the survey had no blood samples collected during January and February, our results may underestimate the true mean serum 25-hydroxyvitamin D status. In the National Nutrition Survey of New Zealand adults, the mean serum 25-hydroxyvitamin D concentration in January and February was 81 and 70 nmol/L, respectively, with a population mean of 50 nmol/L for the whole year (42). Assuming that the concentrations in children during January and February are the same as in adults, we estimate the mean serum 25-hydroxyvitamin D concentration for the whole year to be 54 nmol/L, only slightly higher than the survey result of 50 nmol/L.

Ethnicity had the next greatest effect on vitamin D status, with an adjusted mean difference of 16 nmol/L in serum 25-hydroxyvitamin D between Pacific and NZEO children. Pacific children had twice the risk of vitamin D insufficiency compared with NZEO children. There is a large variability in skin color within each of the 3 ethnic groups, particularly Māori and Pacific children. However, overall differences in

average skin color among the 3 ethnic groups likely account for the ethnic differences in vitamin D status. The health implications of lower vitamin D status in Māori and Pacific children are not known. Paradoxically, Pacific adults in New Zealand have lower serum 25-hydroxyvitamin D concentrations (42,43), yet higher bone mineral content (44), BMD (45), and lower fracture rates (46) than European New Zealanders. To date, there are no data associating vitamin D status with chronic diseases in Māori or Pacific Peoples.

Obesity and sex each had an independent effect on serum vitamin D concentrations, although of lesser magnitude than season or ethnicity. Results from the CNS02 showed that physical inactivity increased considerably across age categories, was higher in girls than boys (26), and was higher in children who were obese rather than "normal" weight (unpublished findings). It is possible that sex and obesity are acting as surrogate markers for sunlight exposure through their association with physical activity. In the case of obesity, there is also evidence that serum 25-hydroxyvitamin D may be sequestered in adipose tissue (15). There was no effect of latitude on serum 25-hydroxyvitamin D. Only 174 participants were recruited from the South Island, and it may be that there was insufficient power to detect a difference in 25-hydroxyvitamin D concentrations across the narrow range of latitude (32–44°S).

Of the children who participated in the CNS02, 16% were from schools classified as rural and were not asked to provide a blood sample. The greater proportion of Pacific and North Island (89 vs. 84%) children likely reflects the exclusion of rural participants, who were more likely to be NZEO and live in the South Island. Differences in proportions of children who gave blood compared with those in the larger survey (Table 1) were accounted for by applying appropriate weights in the statistical analysis.

Using a cutoff value of 537.5 nmol/L for 25-hydroxyvitamin D concentration, we report a high prevalence of insufficient vitamin D status in New Zealand school-age children, from 31% for all children to a high of 59% in Pacific children. Unfortunately, there is little direct evidence to indicate that vitamin D concentrations at this level in children cause poor skeletal health or lead to a lower attainment of peak bone mass and higher rates of osteoporosis in older age. Moreover, the epidemiologic evidence linking low vitamin D status with increased risk of diabetes or some types of cancer is not yet of sufficient quality or strength to be convincing. The results of this survey warrant an urgent evaluation of what health risks may be experienced by New Zealand children with respect to their current vitamin D status.

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