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Vitamin D deficiency in early childhood: prevalent in the sunny South Pacific

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Abstract

Objective: To estimate the prevalence of and risk factors for vitamin D deficiency in young urban children in Auckland, New Zealand, where there is no routine vitamin D supplementation.

Design: A random sample of urban children. Vitamin D deficiency was defined as serum 25-hydroxyvitamin D <27·5 nmol/l (<11 ng/ml). Logistic regression analysis was used to calculate odds ratios and, from these, relative risks (RR) and 95 % confidence intervals were estimated.

Setting: Auckland, New Zealand (36°52′S), where the daily vitamin D production by solar irradiation varies between summer and winter at least 10-fold.

Subjects: Children aged 6 to 23 months enrolled from 1999 to 2002.

Results: Vitamin D deficiency was present in forty-six of 353 (10 %; 95 % CI 7, 13 %). In a multivariate model there was an increased risk of vitamin D deficiency associated with measurement in winter or spring (RR = 7·24, 95 % CI 1·55, 23·58), Pacific ethnicity (RR = 7·60, 95 % CI 1·80, 20·11), not receiving any infant or follow-on formula (RR = 5·69, 95 % CI 2·66, 10·16), not currently receiving vitamin supplements (RR = 5·32, 95 % CI 2·04, 11·85) and living in a more crowded household (RR = 2·36, 95 % CI 1·04, 4·88).

Conclusions: Vitamin D deficiency is prevalent in early childhood in New Zealand. Prevalence varies with season and ethnicity. Dietary factors are important determinants of vitamin D status in this age group. Vitamin D supplementation should be considered as part of New Zealand's child health policy.

Keywords
Child
Diet
Ethnic groups
New Zealand
Sunlight
Vitamin D deficiency

New Zealand is a South Pacific island nation that lies between latitudes 34°S and 47°S[1]. Solar UV radiation is very high in the summer and New Zealand has some of the highest rates of skin cancer in the world[2]. Sun protection to prevent skin damage and skin cancer when the UV index is high is an important and necessary health message[3].

However, there are also large seasonal variations in UV radiation in New Zealand, with the daily winter erythemal dose being between 5 % and 10 % of the summer dose[4]. The daily vitamin D production by solar UV irradiation varies between summer and winter at least 10-fold in Auckland (36°52′S) and at least 20-fold in Invercargill (46°24′S)[5].

In contrast to the USA and the UK, routine vitamin D supplementation during early childhood is not recommended[6,7]. Current recommendations in New Zealand are for intakes of 200 IU vitamin D/d from birth to age 50 years[8]. These same recommendations apply to pregnant and breast-feeding women[9]. For premature or low-birth-weight infants (<32 weeks' gestation or <1800 g) the recommended intake is 400 IU vitamin D/d with oral supplementation if the infant is consuming <180 ml preterm milk formula or fortified breast milk/kg per d[10]. For children it is recommended that hands, face and arms or arms and legs be exposed to the sun two to three times weekly for 5 to 10 min (non-pigmented skin) or 10 to 15 min (pigmented skin). The Cancer Society of New Zealand recommends that this exposure not be between 11.00 and 16.00 hours in the summer months[11].

In Auckland we have observed a significant number of hospitalised children with vitamin D-deficient rickets. Eighteen hospitalised children (median age 12 months) with radiological evidence of rickets and serum 25-hydroxyvitamin D (25(OH)D) <25 nmol/l were identified in an audit of one calendar year (1998) of admissions, giving a prevalence of approximately 6 per 10 000 children less than 3 years old[12,13]. Deficiency of other

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micronutrients (Fe deficiency 14% and vitamin A deficiency 9%) are also prevalent in Auckland children aged 6 to 23 months[14]. We therefore sought to determine the population prevalence of vitamin D deficiency in the first two years of life and the factors associated with vitamin D deficiency in this age group.

Subjects and methods

Study design

An ethnically stratified random sample of children aged 6 to 23 months residing in Auckland was enrolled. In this age group 18% are Maori (New Zealand's indigenous population), 24% are Pacific (from Samoa, Tonga and other Pacific Island nations) and 58% are of European and other ethnic groups[15]. There were no exclusion criteria. Enrolment of children with an acute infection was deferred for one month. The Health Funding Authority Ethics Committee approved the study.

Eligible children were identified from random residential address start points with cluster sampling from the twenty consecutive dwellings starting with the dwelling immediately to the right of each start point address. A list of random start points in the form of street addresses was obtained from the New Zealand Department of Statistics. A researcher identified eligible children by visiting the twenty dwellings at each start point.

In a manner identical to that used in the national census, ethnicity was defined by the child's caregiver. This was the parent's ethnicity or the ethnicity they identified with most if the parents were from more than one ethnic group. An ethnically stratified sampling ratio was applied so the enrolled sample would include approximately equal numbers of three groups: Maori, Pacific and European or other children. A sample of 450 children was sufficient to have 95% confidence limits of ±2% if the observed prevalence of vitamin D deficiency was 5% and ±6% if it was 50%[16].

Socio-economic status was measured using the New Zealand Index of Social Deprivation[17,18]. This measure uses census data that describe household income and ownership, household member employment and education, and household amenities and crowding. Based on these variables households in New Zealand are ranked on a 10-point deprivation scale with 1 being the least deprived and 10 the most deprived households.

Data collection

Data were collected in the child's home. Written informed consent was obtained, the child's caregiver was interviewed and topical anaesthetic cream applied to the child's antecubital fosse (2·5% lignocaine, 2·5% prilocaine, EMLA™; AstraZeneca Ltd, Auckland, New Zealand).

One hour following application of the anaesthetic cream a paediatric phlebotomist visited and obtained a 5 ml venous blood sample. This was facilitated by the research nurse using play therapy to assist the child to cope with the venepuncture[19]. Blood was collected into trace-element-free Vacutainer tubes, protected from light and oxygen, transported on ice and frozen until analysis.

25(OH)D concentration was measured using RIA (DiaSorin, Stillwater, MN, USA). Inter-batch CV was 6·4% (at 22 nmol/l) and intra-batch CV was 6·6% (at 25 nmol/l). The assay measured total 25(OH)D (both vitamin D_2 and D_3). Vitamin D deficiency was defined as 25(OH)D < 27·5 nmol/l (<11 ng/ml)[7,20,21].

A full blood count was measured using an automated Coulter Counter (Beckman Coulter Inc., Fullerton, CA, USA). Serum ferritin concentration was measured by immunoturbidimetry. Serum Fe and total Fe binding capacity were measured calorimetrically using the Ferro-Zine reagent[22]. Fe deficiency was defined as abnormal values for two or more of serum ferritin (<10 μg/l), Fe saturation (<10%) and mean cell volume (<73 fl)[14,23,24]. C-reactive protein (CRP) concentration was measured by immunoturbidimetry. An elevated CRP was defined as >4 mg/l.

Weight and length were measured supine. The mean of three serial measurements from each child, wearing light clothing and no shoes, were taken. Height-for-age and weight-for-age *Z*-scores were computed in the Epi-Info™ 2000 software (Centers for Disease Control and Prevention (CDC), Atlanta, GA, USA) using the 2000 CDC reference data[16].

Statistical analyses

Double-entered data were analysed using Epi-Info™ 2000 and the SAS-PC version 9·1 statistical software package (SAS Institute Inc., Cary, NC, USA). Estimations of variable distribution, proportions and odds ratios were adjusted for clustering and weighted for ethnic stratification using SAS-callable SUDAAN version 9·0·1 (Research Triangle Institute, Research Triangle Park, NC, USA).

Univariate associations with vitamin D deficiency were determined by estimating odds ratios and 95% confidence intervals. Variables examined included those describing the presence of Fe deficiency; season of enrolment; child demographics (age, ethnicity, gestation, birth weight), feeding habits (breast-feeding duration, use of vitamin D-fortified milk formula, weaning foods, use of vitamin and mineral supplements) and UV light exposure; and family and household characteristics. Within each of these areas the potential for covariance was explored. Where covariance was present between pairs of variables only the variable with the strongest association with vitamin D was included in the regression model.

Exposure to UVB radiation was measured indirectly by determining the number of hours the child spent outdoors in the sun during the preceding four weeks. In New Zealand, reported sunlight exposure has been shown to correlate with serum 25(OH)D concentrations[25].

A multivariate model was built by successively adding variables in order of the amount of variance in vitamin D deficiency that they explained in univariate analyses. Variables were included in the model if their inclusion increased the explanatory power of the model and if the variable was significantly associated with vitamin D deficiency ($P < 0{\cdot}05$) after adjustment for the effect of other variables previously added to the model.

Multivariate logistic regression analysis was performed using SAS-callable SUDAAN (PROC MULTILOG) to estimate odds ratios and 95% confidence intervals. As the odds ratios were determined on a cohort in which vitamin D deficiency was prevalent, the relative risk (RR) estimated from the odds ratio was adjusted to more accurately represent the true relative risk[26].

Results

Study sample enrolment

Enrolment occurred from June 1999 to September 2002 and is summarised in Fig. 1. Application of the ethnic specific sampling frames resulted in 575 of the 995 identified eligible children being invited to enrol (100% of the identified Maori children, 75% of the identified Pacific children and 31% of the identified children of New Zealand European and other ethnic groups).

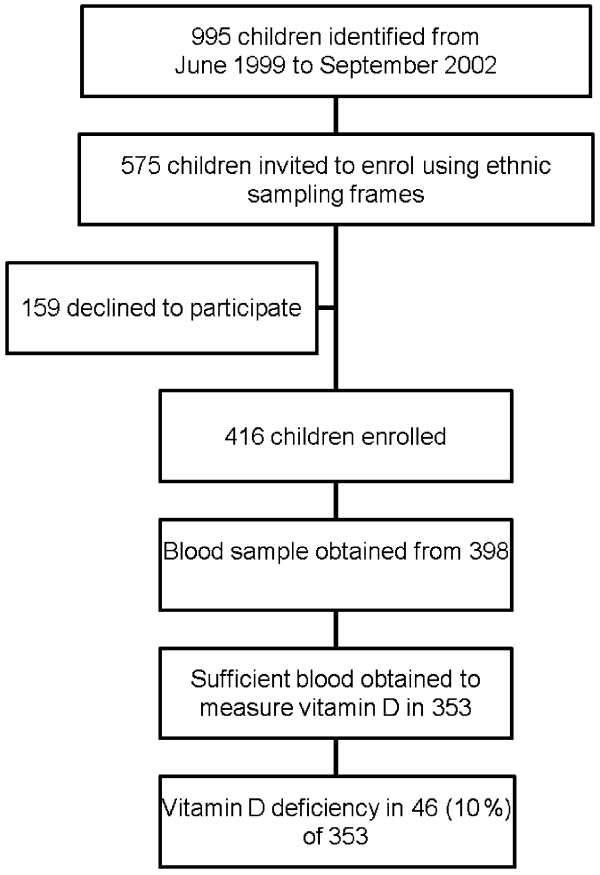

Fig. 1 Summary of enrolment, blood sampling and prevalence of vitamin D deficiency in the studied population: random sample of urban children aged 6 to 23 months, Auckland, New Zealand, 1999–2002

The most frequently stated reason for non-enrolment was that the caregiver did not want the child to have a blood test. Social deprivation, as measured by the median household deprivation score, did not differ between enrolled (*n* 416) *v.* non-enrolled (*n* 159). The 416 enrolled children were identified from 345 start points, with between one and five (median one) enrolled from each start point.

Vitamin D status was measured in 353 (85%) of 416 enrolled children. The median (25th, 75th centile) vitamin D concentration was 55 (42, 70) nmol/l. Vitamin D deficiency was present in forty-six (10%; 95% CI 7, 13%).

Serum vitamin D concentrations varied with age, ethnicity and season (Table 1). The median vitamin D concentration for children aged 18–23 months was lower than that for children aged 6–11 ($P = 0{\cdot}002$) or 12–17 months ($P = 0{\cdot}002$). In comparison with European children, vitamin D concentrations were lower for Maori ($P = 0{\cdot}02$) and Pacific ($P < 0{\cdot}001$) children. In comparison with those measured in summer, vitamin D concentrations were lower when measured during winter ($P < 0{\cdot}001$) or spring ($P < 0{\cdot}001$). The median vitamin D concentration did not differ between children with a normal *v.* elevated (>4 mg/l) CRP concentration (53 *v.* 50 nmol/l, $P = 0{\cdot}55$).

Characteristics of the children

The median age (25th, 75th centile) was 14·7 (11·0, 19·6) months. Associations of children's ethnicity and feeding habits with risk of vitamin D deficiency are shown in Table 2. There was an increased risk of vitamin D deficiency

Table 1 Vitamin D concentration by age, gender, ethnicity and season: random sample of urban children aged 6 to 23 months, Auckland, New Zealand, 1999–2002

	Vitamin D concentration (nmol/l)		
Variable	Median	25th, 75th centile	*P* value
Age in months (*n* 340)*			<0·001
6 to 11 (*n* 107)	62	42, 78	
12 to 17 (*n* 112)	58	44, 76	
18 to 23 (*n* 121)	49	39, 61	
Gender (*n* 353)			
Male (*n* 168)	57	42, 75	0·11
Female (*n* 185)	52	41, 67	
Ethnicity (*n* 353)			<0·001
Maori (*n* 90)	53	36, 67	
Pacific (*n* 120)	43	29, 58	
Other (*n* 25)	50	34, 69	
European (*n* 118)	59	47, 76	
Season (*n* 353)†			<0·001
Summer (*n* 47)	71	51, 81	
Autumn (*n* 96)	61	49, 74	
Winter (*n* 125)	47	34, 63	
Spring (*n* 85)	47	35, 62	

*Seven children were aged 5 months, five children were aged 24 months and one child was aged 27 months when vitamin D status was measured.
†Summer (December to February), autumn (March to May), winter (June to August), spring (September to November).

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Table 2 Associations of child's ethnicity and feeding habits, season of enrolment and sunlight exposure with risk of vitamin D deficiency: random sample of urban children aged 6 to 23 months, Auckland, New Zealand, 1999–2002

	Vitamin D deficiency present					
	Yes (*n* 46)		No (*n* 307)			
Variable	*n*	%*	*n*	%*	RR†	95 % CI†
Child's ethnicity and feeding habits						
Ethnic group (*n* 353)						
Maori	10	11	80	89	4·30	1·01, 13·78
Pacific	29	24	91	76	9·14	2·78, 20·35
Other	4	16	21	84	6·14	1·14, 19·63
European	3	3	115	97	1·00	
Age stopped breast milk (*n* 324)						
7 months or older	11	6	138	94	2·08	0·94, 4·22
0 to 6 months	30	12	145	88	1·00	
Any infant or follow-on formula (n 350)						
No	23	26	45	74	4·84	2·62, 8·03
Yes	21	5	261	96	1·00	
Weaning foods first eaten (*n* 347)						
Only home-made	15	20	52	80	2·82	1·37, 5·21
Commercial and/or home-made	29	7	251	93	1·00	
Child currently taking vitamin supplements (*n* 349)						
No	43	10	285	90	3·91	2·24, 6·57
Yes	1	3	20	97	1·00	
Sunlight exposure and season of enrolment						
Season of enrolment‡						
Winter	27	15	98	85	12·88	7·34, 21·62
Spring	15	14	70	86	11·84	5·90, 22·36
Autumn	3	3	93	97	2·53	0·41, 14·03
Summer	1	1	46	99	1·00	
Hours per day outside over past 4 weeks§ (*n* 347)						
0	7	19	23	81	2·17	0·84, 4·67
1 or more	37	9	280	91	1·00	
Clothing and sun protection used when outside						
Long-sleeved top and pants‖ (*n* 349)						
Yes	41	15	164	85	7·57	1·33, 26·87
No	3	2	141	98	1·00	
Sunhat (*n* 349)						
Yes	23	10	179	90	1·01	0·53, 1·85
No	21	9	126	91	1·00	
Sunscreen (*n* 349)						
Yes	3	3	92	97	0·18	0·03, 1·13
No	41	13	213	87	1·00	

RR, relative risk.
*Row percentages adjusted for clustering and weighted for the ethnic stratification.
†Proportions and relative risks and 95 % confidence intervals weighted for ethnicity and adjusted for clustering.
‡Summer (December to February), autumn (March to May), winter (June to August), spring (September to November).
§Number of hours per day in the last 4 weeks averaged over weekdays and weekend days that the child spent outdoors in the sun.
‖Alternatives include T-shirt and shorts, underpants and singlet, or nothing.

for all non-European ethnic groups with the risk being greatest for children of Pacific ethnicity (RR = 9·14, 95 % CI 2·78, 20·35). Although vitamin D concentration decreased with increasing age the risk of vitamin D deficiency did not vary with age.

Vitamin D status did not vary with birth weight. The mean height-for-age *Z*-score (0·24 *v.* 0·20, $P = 0·81$) and weight-for-age *Z*-score (0·62 *v.* 0·56, $P = 0·78$) did not differ between those with low *v.* normal vitamin D concentrations. Being of premature gestation or low birth weight was not associated with the risk of vitamin D deficiency (RR = 0·47, 95 % CI 0·06, 3·04).

Most (92 %) children were breast-fed. The risk of vitamin D deficiency was not associated with duration of breast-feeding but was increased in children who had not received any milk formula (RR = 4·84, 95 % CI 2·62, 8·03), the only type of milk in New Zealand that is fortified with vitamin D. Milk formula in New Zealand is required to contain between 0·25 and 0·63 μg of vitamin D_2 or vitamin D_3 per 100 kJ[27].

Children whose first weaning foods were home-made rather than commercial were at increased risk of vitamin D deficiency (RR = 2·82, 95 % CI 1·37, 5·21) as were those children not currently taking vitamin or mineral supplements (RR = 3·91, 95 % CI 2·24, 6·57). Children who ate no eggs, meat or fish were not at increased risk of vitamin D deficiency (RR = 0·68, 95 % CI 0·30, 1·51).

Fourteen per cent of the children were Fe-deficient. The risk of vitamin D deficiency was increased in children with Fe deficiency (RR = 2·50, 95 % CI 1·17, 4·71).

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Season of enrolment and sunlight exposure

Table 2 also shows associations of season of enrolment and sunlight exposure with risk of vitamin D deficiency. Children enrolled during either winter (RR = 12·88, 95% CI 7·34, 21·62) or spring (RR = 11·84, 95% CI 5·90, 22·36) were at increased risk of vitamin D deficiency. The median (25th, 75th centiles) number of hours spent outdoors per day over the preceding month was 1·5 (0·9, 2·3). There was no association between spending less than one hour per day outside and the risk of vitamin D deficiency. Nor did the risk of vitamin D deficiency vary with the time of day when the child was usually outside (data not shown). The lack of association between time spent outside and risk of vitamin D deficiency persisted after adjustment for either season or ethnicity (data not shown). In comparison with children who wore less clothing, children who usually wore a long-sleeved top and pants when outside were at increased risk of vitamin D deficiency (RR = 7·57, 95% CI 1·33, 26·87).

Parents and siblings

The median (25th, 75th centile) maternal age was 31 (26, 34) years. Family and household characteristics and associated risk of vitamin D deficiency are shown in Table 3. The risk of vitamin D deficiency did not vary with maternal age or education (data not shown). There was an increased risk of vitamin D deficiency if neither parent was employed (RR = 2·34, 95% CI 1·02, 4·69). Sixty per cent of the children had siblings. Having two or more siblings was associated with an increased risk of vitamin D deficiency (RR = 2·29, 95% CI 1·23, 3·98).

The total household income from wages and benefits, before tax, in the last 12 months was provided for 316 (90%) of the households. There was an increased risk of vitamin D deficiency associated with annual household income <$NZ 15 000 (RR = 2·91, 95% CI 1·13, 6·02) and with a higher household expenditure to income ratio (RR = 9·75, 95% CI 6·01, 15·45). Children living in households where there were six or more residents (RR = 3·64, 95% CI 1·99, 6·08) or in households that were more crowded (RR = 4·11, 95% CI 2·30, 6·68) were at increased risk of vitamin D deficiency.

Household income and family size vary with ethnicity, with a larger proportion of Pacific and Maori children living in lower-income and more crowded households[28,29]. After adjustment for ethnicity, the increased risk of vitamin D deficiency associated with higher household expenditure to income ratio (RR = 7·98, 95% CI 4·59, 13·55), and with households that contained more people (RR = 2·19, 95% CI 1·09, 4·09) and were more crowded (RR = 2·24, 95% CI 1·10, 4·23), persisted.

Multivariate analyses

In the multivariate model (Table 4) there was an increased risk of vitamin D deficiency associated with being enrolled in either winter or spring (RR = 7·24, 95% CI 1·55, 23·58), Pacific ethnicity (RR = 7·60, 95% CI 1·80,

Table 3 Family and household characteristics and associated risk of vitamin D deficiency: random sample of urban children aged 6 to 23 months, Auckland, New Zealand, 1999–2002

	Vitamin D deficiency present							
	Yes (*n* 46)		No (*n* 307)		Univariate risk†		Adjusted for ethnic group†	
Variable	*n*	%*	*n*	%*	RR	95% CI	RR	95% CI
Family characteristics								
Neither parent employed (*n* 316)								
Yes	10	19	40	81	2·34	1·02, 4·69	1·65	0·66, 3·70
No	29	8	237	92	1·00		1·00	
Number of siblings (*n* 350)								
2 or more	20	16	85	84	2·29	1·23, 3·98	1·49	0·75, 2·82
0 or 1	24	7	221	93	1·00		1·00	
Household characteristics								
Household income before tax in past 12 months in $NZ‡ (*n* 316)								
1–15 000	9	22	29	78	2·91	1·13, 6·02	1·15	0·36, 3·17
15 001–40 000	15	8	110	92	1·09	0·48, 2·33	0·53	0·25, 1·13
40 001 or greater	15	8	138	92	1·00		1·00	
Household expenditure to income ratio (*n* 296)								
0·34 or greater	35	11	212	89	9·75	6·01, 15·45	7·98	4·59, 13·55
<0·34	1	1	48	99	1·00		1·00	
Number of people living in house (*n* 348)								
Six or more	21	23	68	77	3·64	1·99, 6·08	2·19	1·09, 4·09
Two to five	23	6	236	94	1·00		1·00	
Person to room ratio (*n* 348)								
1·0 or greater	22	25	62	75	4·11	2·30, 6·68	2·24	1·10, 4·23
<1·0	22	6	242	94	1·00		1·00	

RR, relative risk.
*Row percentages adjusted for clustering and weighted for the ethnic stratification.
†Proportions and relative risks and 95% confidence intervals weighted for ethnicity and adjusted for clustering.
‡From all sources in the past 12 months including wages and benefits, before tax or any other deductions.

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Table 4 Multivariate analysis of risk factors for vitamin D deficiency: random sample of urban children aged 6 to 23 months, Auckland, New Zealand, 1999–2002

	Univariate risk*		Multivariate risk*†	
Variable	RR	95% CI	RR	95% CI
Season				
Winter and spring	6·23	1·55, 19·39	7·24	1·55, 23·58
Summer and autumn	1·00		1·00	
Ethnic group				
Maori	4·30	1·01, 13·78	3·41	0·66, 13·06
Pacific	9·14	2·78, 20·35	7·60	1·80, 20·11
Other	6·14	1·14, 19·63	4·09	0·58, 17·54
New Zealand European	1·00		1·00	
Any infant or follow-on formula				
No	4·84	2·62, 8·03	5·69	2·66, 10·16
Yes	1·00		1·00	
Child currently taking vitamin supplements				
No	3·91	2·24, 6·57	5·32	2·04, 11·85
Yes	1·00		1·00	
Person to room ratio				
1·0 or greater	4·11	2·30, 6·68	2·36	1·04, 4·86
<1·0	1·00		1·00	

RR, relative risk.
*Relative risks and 95% confidence intervals weighted for ethnicity and adjusted for clustering.
†Sample size for multivariate model = 348.

20·11), not receiving any infant or follow-on formula (RR = 5·69, 95% CI 2·66, 10·16), not currently receiving vitamin supplements (RR = 5·32, 95% CI 2·04, 11·85) and living in a more crowded household (RR = 2·36, 95% CI 1·04, 4·86).

Discussion

Vitamin D deficiency is prevalent in the first two years of life in New Zealand, particularly among Pacific children. There was more than a 5-fold variability in prevalence with season, with this magnitude of seasonal effect evident during both winter and spring. The limited dietary sources of additional vitamin D available in New Zealand through supplementation or using fortified milk formula were both independently protective against vitamin D deficiency.

The vitamin D-deficient children identified in the present sample were dissimilar in several respects from those described in recent series from developed countries. Unlike many other studies of young urban children, the vitamin D-deficient children in the current sample were not of low height- or weight-for-age[30–33]. Neither breast-feeding past 6 months of age nor restrictive diets were associated with an increased risk of vitamin D deficiency[34,35]. In the present study inadequate dietary intake of vitamin D was not due to any parental omission or deliberate dietary restriction. The children were placed at dietary risk because there is no current policy that seeks to ensure adequate vitamin D status except for infants born at less than 32 weeks' gestation or 1800 g birth weight[10].

Although most measures of sunlight exposure were not associated with the risk of vitamin D deficiency, a very strong seasonal effect was evident. Auckland has mild but wet winters with on average more rainy days per month than London, Birmingham or Edinburgh for seven of the twelve calendar months, and an average of only 4·3 h of sunshine per day during the winter months (June, July and August)[36]. Relative to similar northern hemisphere latitudes, most of New Zealand's excess UV irradiation occurs during the summer months. Auckland (36°52′S) lies at a similar latitude to European cities where vitamin D deficiency has recently been shown to be prevalent in breast-fed infants; for example, Athens[37] (37°59′N) and Ioannina[38] (39°42′N) in Greece and Pisa[39] in Italy (43°43′N). In these countries, like Auckland, there has been a misperception that because of high summer sunlight intensity vitamin D supplementation in early childhood is unnecessary.

Increased skin pigmentation is one of the factors that explain the increased frequency of vitamin D deficiency in non-European ethnic groups. Clearly though, it is not the sole nor necessarily the most significant factor[40]. Generally speaking, the skin pigmentation of Maori and Pacific peoples is similar; however, the risk of vitamin D deficiency for Pacific children was twice that for Maori (RR = 2·18, 95% CI 1·09, 3·83).

Ethnic-specific dietary habits alter the risk of rickets. In the UK, the higher frequency of rickets in Asian (Pakistani and Indian) children in comparison with children with more pigmented skin was thought to be due to the higher phytate content of their diet[41,42]. However, the mechanism of this effect is primarily by reducing the bioavailability of dietary Ca rather than any specific effect on vitamin D[43]. Ethnic differences in the frequency of the genetic polymorphisms which regulate vitamin D receptor activity explain differing risks of disease associated with vitamin D

deficiency but not vitamin D concentrations themselves[44]. Thus it seems more likely that cultural and lifestyle rather than genetic differences explain the increased risk of vitamin D deficiency in Pacific people.

There was an increased risk of vitamin D deficiency associated with two measures of lower socio-economic status: higher expenditure to income ratios and more crowded households. Living in poor urban households is recognised as a risk factor for rickets both in countries with and without high sunlight exposure[30,45]. It is likely to be the most significant factor contributing to the excessive risk of vitamin D deficiency in Pacific children. Within New Zealand, Pacific people have the highest level of food insecurity and more than 40 % of New Zealand Pacific people live in crowded housing[46,47].

The vitamin D deficiency evident in the present sample is likely to have several adverse effects on the health of these children. Vitamin D deficiency in early childhood may have long-term adverse effects on skeletal bone mineralisation in addition to causing rickets[48]. It may also play a causative role in periodontal disease[49]. Vitamin D deficiency (as manifested by rickets) is associated an increased risk of subsequent development of type 1 diabetes[50–52].

Vitamin D influences the function of both the innate and adaptive immune systems. Vitamin D increases the production of innate antimicrobial peptides and promotes the induction of T-regulatory cells[53,54]. These immune modulator effects provide a potential explanation for the increased risk of pneumonia that is associated with rickets in children of pre-school age and with the increased risk of wheezy respiratory illnesses in children whose mothers had lower intakes of vitamin D during pregnancy[55]. The potential role that vitamin D deficiency plays in the excessive burden of lower respiratory tract disease experienced by New Zealand children requires further examination[56].

The poor vitamin D status of the young children in the present study is consistent with that described for New Zealand adults and school-aged children. A national nutrition survey of adults in 1997 showed that the mean vitamin D concentration (50 nmol/l) of New Zealand adults was lower than the mean value recorded in the adult US population (>70 nmol/l) living at comparable latitudes[57,58]. A national survey of children aged 5 to 14 years in 2002 showed that the mean vitamin D concentration was also 50 nmol/l[59]. This population average is lower than that reported for the UK population aged 4 to 10 years (mean ≈70 nmol/l) and 11 to 14 years (mean 56 nmol/l)[59,60]. Suboptimal vitamin D status therefore appears to be a public health issue in New Zealand from infancy through adulthood.

Thus the public health strategy to address this issue for young children needs to consider the vitamin D status of the child and mother, and is likely to require both vitamin D supplementation and vitamin D fortification of staple foods. In vitamin D-sufficient women, breast milk vitamin D concentration ranges from 15 to 50 IU/l and is even lower in women who are vitamin D-deficient[61]. Therefore policy that recommends vitamin D supplementation for breast-fed infants and those not consuming sufficient infant formula to meet their daily requirements will be necessary if currently recommended intakes in infancy (200 IU/d) are to be met[7]. The poor compliance with daily vitamin supplementation of infants in the UK suggests that alternative strategies, such as less frequent dosing administered at well child visits, requires consideration[62]. The protection against vitamin D deficiency provided by infant formula consumption in our study indicates that supplementation of other milks and solid foods consumed by young children is also likely to be beneficial.

Improving maternal vitamin D status is essential if infant vitamin D status is to be normal from birth. Newborn infant vitamin D status is determined by maternal vitamin D status during pregnancy[63]. Both mandatory fortification and vitamin D supplementation would appear to be necessary to achieve adequate maternal and therefore newborn vitamin D status. In the New Zealand adult nutrition survey more than 50 % of women aged 19 to 44 years had vitamin D concentrations ≤50 nmol/l and more than 80 % had concentrations ≤80 nmol/l[57]. The intake of vitamin D from dietary sources in New Zealand is not known[57]. Based on literature from other countries that do not have mandatory fortification, intake is likely to be well below the currently recommended intake of 200 IU/d[57].

Mandatory fortification of food with vitamin D would be expected to increase vitamin D intake[64]. However, in countries where this is already policy, it is not sufficient to prevent vitamin D deficiency in higher-risk groups[64]. Maternal supplementation during pregnancy would also seem necessary. The dose required is likely to be higher than the currently recommended 200–400 IU/d and compliance issues indicate that alternatives to daily supplementation need to be considered[65–67].

In conclusion, the present study shows that the prevalence of vitamin D deficiency is such that it is liable to be having an adverse effect on child health in New Zealand. It could well be one of the more important factors that determine the poorer health status of Maori and Pacific children in New Zealand. New Zealand must develop health policy which reflects the needs of its ethnically diverse population.

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