European Journal of Clinical Nutrition (2002) 56, 1169–1175
© 2002 Nature Publishing Group All rights reserved 0954–3007/02 $25.00
www.nature.com/ejcn

ORIGINAL COMMUNICATION

Mild iodine deficiency in a sample of New Zealand schoolchildren

SA Skeaff[1]*, CD Thomson[1] and RS Gibson[1]

[1]*Department of Human Nutrition, University of Otago, Dunedin, New Zealand*

Objective: To assess the iodine status of New Zealand schoolchildren.
Design: A proportionate to population size school-based cluster survey was used to randomly select children from two cities. The indicators used to assess iodine status were urinary iodine, as determined in a casual urine sample, and thyroid volume, as measured by ultrasonography. A qualitative food frequency questionnaire designed to ascertain frequency of consumption over the previous 3 months of foods or food groups that are good sources of dietary iodine, including iodized salt, was administered to each child.
Setting: Dunedin and Wellington, New Zealand.
Participants: Three-hundred children aged 8 – 10 y from 30 schools.
Results: The median urinary iodine concentration of the children was 6.6 μg/dl (interquartile range, 4.5 – 9.1). The percentage of children who had urinary iodine levels less than 5 μg/dl was 31.4 (95% confidence interval (CI), 24.2 – 38.6). Comparison of thyroid volume with 2001 World Health Organization age/sex-specific and age/BSA-specific cut-off values resulted in a goitre prevalence of 11.3% (95% CI, 7.6 – 15.1) and 12.0% (95% CI, 7.9 – 16.1), respectively. Almost 30% of the children's caregivers did not use iodized salt in cooking and 51% of the children did not use iodized salt at the table.
Conclusions: Mild iodine deficiency was found in this sample of children. Iodized table salt may no longer be making a significant contribution to the iodine intakes of New Zealand children.

Sponsorship: This research was funded by the Health Research Council of New Zealand and the Ministry of Health in New Zealand.
European Journal of Clinical Nutrition (2002) **56,** 1169 – 1175. doi:10.1038/sj.ejcn.1601468

Keywords: iodine; iodine deficiency; thyroid gland; iodized salt; children

Introduction

New Zealand has low soil iodine levels. Studies carried out in the 1920s and 1930s showed that in many parts of New Zealand there was mild to moderate iodine deficiency characterized by low urinary iodine levels and high goitre rates; in some areas up to 30% of schoolchildren had goitre (Purves, 1974). The introduction of iodized salt in 1939 helped to eradicate goitre by the 1950s. Furthermore, in the 1960s, an adventitious source of iodine was introduced into the diet from iodophors used as sanitizers in the dairy industry (Sutcliffe, 1990) and in the 1970s and 1980s adequate intakes of iodine were reported (Cooper *et al*, 1984; North & Fraser, 1965; Simpson *et al*, 1984). More recent studies, however, report urinary iodine levels in adults indicative of mild iodine deficiency (Thomson *et al*, 1997). Dietary iodine intakes may have decreased in New Zealand for two reasons. Firstly, there has been a decline in the use of iodophors in the dairy industry over the last two decades and, secondly, there are public health recommendations to decrease discretionary salt consumption. Decreased iodine intakes have also been reported in the United States and Australia (Gunton *et al*, 1999; Hollowell *et al*, 1998).

Iodine deficiency disorders (IDD) is the term used to describe the wide range of adverse effects low iodine intakes can have on health (Hetzel & Dunn, 1989). These adverse

---

***Correspondence: SA Skeaff, Department of Human Nutrition, University of Otago, PO Box 56, Dunedin, New Zealand.**
E-mail: sheila.skeaff@stonebow.otago.ac.nz
Guarantor: SA Skeaff.
Contributors: **SAS participated in the design of the study, recruited the subjects, collected data, carried out statistical analysis and data interpretation, and prepared first and subsequent drafts of the manuscript. CDT and RSG participated in the design of the study, data interpretation and manuscript preparation.**
Received 16 August 2001; revised 1 March 2002; accepted 11 March 2002

effects can occur at all stages of the life cycle but have the greatest impact during periods of rapid growth and development. Accordingly, the World Health Organization has identified several groups in the population who are particularly vulnerable to sub-optimal iodine intakes, such as pregnant women and children (WHO *et al*, 1993). Iodine deficiency during pregnancy has been associated with a higher incidence of stillbirths, abortions and congenital abnormalities with the most severe consequence being cretinism (Hetzel *et al*, 1990). Cases of cretinism have never been reported in New Zealand. In children, iodine deficiency typically results in goitre. A number of studies have also reported adverse effects on hearing capacity, motor and cognitive function in children (Azizi *et al*, 1995; Bleichrodt *et al*, 1989; Huda *et al*, 1999; Shrestha, 1994; Valeix *et al*, 1994). Most of these studies, however, have been conducted in populations with severe or moderate iodine deficiency. It is not known whether in mild iodine deficiency there are similar but more subtle adverse effects, which the current tests are not sensitive enough to detect.

The aim of this study was to assess the iodine status of New Zealand schoolchildren aged 8–10 y. As subjects, school-aged children are readily accessible, enthusiastic and honest participants, and the indicators used to assess iodine deficiency in this group are simple to obtain and well defined. In children, mild iodine deficiency is typically characterized by a median urinary iodine concentration between 5 and 10 μg/dl and a goitre incidence greater than 5% (WHO *et al*, 1994). Other indices of iodine status, such as thyroxine, tri-iodothyronine and thyroid stimulating hormone, are not recommended for assessing mild iodine deficiency (Benmiloud *et al*, 1994; WHO *et al*, 1993). In this study, we measured urinary iodine levels in a casual urine sample and thyroid volume by ultrasonography.

Methods

Study design

A proportionate to population size school-based cluster survey was used to randomly select children from two New Zealand cities. In such surveys, 10 children should be selected from each of 30 clusters or schools resulting in a minimum sample size of 300 (WHO *et al*, 1994). In Dunedin, a city in the South Island with a population of approximately 100 000 people, children were selected from eight schools from June 1996 until June 1997. In Wellington, a city in the North Island with a population of approximately 300 000 people, children were selected from 22 schools from March 1999 until May 1999. Ethical approval was obtained from the Southern Regional Health Authority Ethics Committee Otago in Dunedin and the Wellington Ethics Committee in Wellington.

Subjects and experimental protocol

In Dunedin, schools were randomly selected. In Wellington, schools were selected using a sampling interval and the cumulative school roll. Because different methods were used to select children in the two cities, the probability of a child being included in the sample was not equal; therefore a weighting factor was used to account for this difference and used in all statistical analyses.

Apparently healthy children aged between 8 and 10 y were randomly selected from the school roll. In most instances 25 children from each school were sent a letter by post outlining the study and inviting them to participate; a few schools indicated that participation in such studies was usually low and at these schools 30 children were invited to take part. Children and their caregivers were then contacted by telephone and those who consented were assigned interview times. Most of the interviews and data collection was carried out on the school premises by trained research assistants with the exception of thyroid volume, which was measured by an ultrasonographer. Participating children attended either a single morning interview on weekends accompanied by their primary caregiver, or during the week, two interviews, one in the morning and a second after school on the same day at which time their primary caregiver also attended. Two interviews on weekdays were necessary for the following reasons: many caregivers were unavailable during school hours, the ultrasonographers were available only during the morning, and the shorter interview on a weekday school morning reduced the time children missed from school.

Dietary, anthropometric, biochemical and clinical data were obtained from each child. Regardless of the day, anthropometric and clinical measurements and urine samples were obtained from all children between 08:00 and 12:00 h. Children who were identified by their primary caregiver as Maori or a Pacific (ie born in a Pacific Island) person or who had a parent who was Maori or a Pacific person, were identified as Maori or Pacific persons, respectively.

During the interviews attended by the child's primary caregiver, dietary data were collected via a qualitative food frequency questionnaire designed to solicit information on the frequency of consumption of foods over the previous 3 months that are good sources of iodine. The list of foods were: milk, dairy products, red meat, chicken, fish, seafoods and eggs; and the frequency-of-use response categories were: daily, weekly, monthly, rarely or unknown. Data on typical serving sizes were not obtained. Information about the use of iodized salt in the home and general health status of the child was also obtained at this time.

Anthropometric assessment included height and weight using calibrated equipment and standardized techniques (Lohman *et al*, 1988) with children wearing light clothing and no shoes. Height was measured to the nearest 0.1 cm using a portable stadiometer designed and built by the University of Otago. Weight was measured to the nearest 100 g using Seca digital platform scales (Model 770, Alpha) calibrated with a 5 kg weight. On school days these measurements were taken during the first session. Children were asked to void urine into a school toilet fitted with a plastic bag and bowl; urine was then transferred to plastic test tubes, stored in a styrofoam chilly bin containing a cold pack and frozen at −20°C within 24 h of collection.

The clinical assessment involved a general health questionnaire administered to the primary caregiver and an assessment of thyroid volume. Thyroid volume was determined by ultrasonography using an Aloka SSD-500 portable ultrasound unit with a 7.5 MHz transducer. Children were in the supine position with the neck hyper-extended during imaging. A transverse and sagittal scan was taken of each lobe and lobe volume was calculated according to the following formula: lobe volume = length × depth × width × 0.479 (Delange *et al*, 1997). The thyroid volume reported is the sum of the volumes of both lobes and does not include the isthmus. Three ultrasonographers were used in the study, one in Dunedin and two in Wellington; each had more than 10 y experience. The Dunedin ultrasonographer carried out training sessions with the two Wellington ultrasonographers on volunteer children to standardize the protocol for assessing thyroid volume among the three ultrasonographers; the same ultrasound machine was used at both study sites. Analysis of variance found no significant differences in the thyroid volume measurements of the three ultrasonographers.

Every attempt was made to standardize the techniques used by the trained research assistants and ultrasonographers in Dunedin and Wellington to decrease inter-observer variability. A site check was carried out half-way through the Wellington phase of the study to ensure that the standardized protocols and methods used were identical to those used in Dunedin.

Urinary iodine was determined using a modification of the method of Pino *et al* (1998) and performed by a single technician. At the time of the study there were no available standard reference materials for urinary iodine. However, previously this laboratory had successfully participated in two international quality control programmes for the determination of urinary iodine (the Program Against Micronutrient Malnutrition (PAMM) International Quality Control Program, Center for Disease Control, Atlanta, GA, USA; and the ICCIDD Urinary Iodine Reference Laboratory, Charlottesville, VA, USA). A pooled urine sample (8.5 ± 0.3 μg/dl) was analysed with each batch of samples giving a coefficient of variation of 3.7% and an average recovery of 101% ($n = 14$).

Statistics and reference data

Data processing and statistics were carried out using Stata 5.0 (Stata Corporation, College Station, Texas, USA). Because individual children within schools could not be considered independently, this was taken into account using the survey commands in Stata. All estimates presented are weighted for probability of selection. Regression analysis was used to test for differences in thyroid volumes and urinary iodine by age and sex. The level of significance for all tests was $P < 0.05$. Urinary iodine concentrations were expressed as a median and compared to ranges reported by the World Health Organization (WHO *et al*, 1994). Thyroid volumes were compared to values published by ICCIDD (Zimmerman *et al*, 2001).

Results

In New Zealand each school is assigned a socio-economic decile rating. Deciles 1–3 are schools drawn from areas of very low socio-economic status, while decile 8–10 schools are from areas of high socio-economic status. Of the participating schools in this study, 27% had a decile rating of 1–3, 27% had a decile rating of 4–7, and 46% had a decile rating of 8–10. On average, just over half of the children (51%) selected agreed to participate in the study. The main reason given for non-participation was an inability to attend the interview on the appointed day because of other commitments. Of the 320 children who took part, 11 children did not fall within the age range and nine did not provide their date of birth, data on general health and diet, resulting in a final sample size of 300 children (75 from Dunedin in the South Island and 225 from Wellington in the North Island). Urinary iodine levels were measured in 282 children and thyroid volume measured in 298 children. The food frequency questionnaire was completed by 299 children.

The ethnic background of the children was as follows: 82% European, 11% Maori, 5% other, and 1.6% Pacific Persons. Almost three-quarters of the children (74.3%) were considered healthy by their primary caregiver. The remainder of the children reported minor health problems such as mild forms of asthma (18%), otitis media (2.3%), and hyperactivity (< 1%). The majority of the children (74.7%) did not regularly consume a multi-vitamin and mineral supplement, with the remainder consuming such supplements occasionally. Almost all of the children (94.4%) did not consume other types of dietary supplements and none of the children took kelp tablets. The mean height, weight and body surface area (BSA) for the children are presented in Table 1. BSA was calculated using the following formula, where W is the weight in kg and H is the height in cm (WHO & ICCIDD, 1997):

$$\text{Body surface area } (\text{m}^2) = W^{0.425} \times H^{0.725} \times 71.84 \times 10^{-4}$$

Most (84.6%) of the children's caregivers reported that they had iodized salt in the home, 9.7% did not use iodized salt, and 5.7% of caregivers did not know what type of salt was used in the home. Access to iodized salt in the home did not mean that iodized salt was used in cooking as only 69% of the caregivers used iodized salt in cooking. Just over a quarter (26.3%) of caregivers did not use iodized salt in cooking. Data on the quantity and frequency of use of iodized salt in cooking were not obtained. Fifty-one percent of children ($n = 152$) did not use iodized salt at the table, while the other 49% ($n = 147$) reported that they did use

Table 1 Mean (s.d.) of weight, height and body surface area (BSA) by age and sex

Sex	*Age (y)*	n	*Weight (kg)*	*Height (cm)*	*BSA* (m^2)
Girls	8	37	29.6 (5.8)	130.7 (6.4)	1.0 (0.13)
	9	62	32.3 (6.9)	135.4 (6.1)	1.1 (0.13)
	10	51	35.0 (7.0)	139.4 (6.5)	1.2 (0.13)
Boys	8	58	29.2 (6.6)	131.1 (6.5)	1.0 (0.13)
	9	60	33.2 (6.2)	136.7 (6.2)	1.1 (0.12)
	10	32	37.1 (8.3)	140.7 (6.7)	1.2 (0.15)

iodized salt at the table. One child and his/her caregiver were unable to categorize the child's consumption of food or salt and were unaware of whether iodized salt was used in the home. Table 2 shows the frequency of consumption of foods that are good sources of dietary iodine.

The median urinary iodine concentration for the whole group was 6.6 μg/dl (interquartile range (IQR), 4.5–9.1; Table 3). There were no significant gender or age differences in urinary iodine levels. The percentage of children who had urinary iodine levels less than 2 μg/dl was 3.6 (95% CI, 1.1–6.2), less than 5 μg/dl, 31.4 (95% CI, 24.2–38.6) and less than 10 μg/dl, 79.7 (95% CI, 74.1–85.3). These urinary iodine concentrations are indicative of severe, moderate and mild IDD, respectively (WHO *et al*, 1994).

Table 2 Frequency of consumption over the previous three months of foods that are good sources of dietary iodine (% of total (*n*))

Food item	*Daily*	*Weekly*	*Monthly*	*Rarely*	*Unknown*
Milk	88 (265)	10 (30)	< 1 (2)	< 1 (2)	< 1 (1)
Dairy products	84 (252)	16 (47)	0 (0)	0 (0)	< 1 (1)
Red meat	15 (46)	77 (232)	3 (8)	4 (13)	< 1 (1)
Chicken	2 (5)	87 (232)	8 (23)	3 (9)	< 1 (1)
Fish	0 (0)	47 (141)	30 (89)	21 (64)	1 (7)
Seafood	< 1 (1)	3 (8)	18 (53)	78 (235)	1 (3)
Eggs	2 (6)	64 (191)	22 (67)	10 (31)	2 (5)
Iodized salt	14 (42)	26 (79)	9 (26)	50 (152)	< 1 (1)

Table 3 Urinary iodine levels (μg/dl) and percentage of children below cutoffs[a] for severe, moderate and mild iodine deficiency

Age (y)	n	*Median (IQR)*[b]	*Percentage < 2 μg/dl*	*Percentage < 5 μg/dl*	*Percentage < 10 μg/dl*
Girls					
8	34	6.7 (4.1, 9.3)	3.1	42.8	76.5
9	57	6.7 (4.6, 8.4)	1.9	26.2	85.2
10	48	6.1 (4.1, 8.2)	6.4	35.8	87.2
Boys					
8	54	5.6 (4.7, 9.3)	5.4	34.0	77.4
9	57	7.1 (4.6, 9.6)	3.7	27.6	78.8
10	32	7.5 (6.0, 10.2)	0.0	24.0	68.1
Total	282	6.6 (4.5, 9.1)	3.6	31.4	79.7

[a] WHO and ICCIDD (1997).
[b] IQR, interquartile range.

Table 4 compares the median thyroid volumes found in the present study with the values reported for children from Malaysia (Foo *et al*, 1999), Switzerland (Hess & Zimmermann, 2000), and Germany (Liesenkötter *et al*, 1997) as well as the 2001 WHO/ICCIDD reference data. Consistent with other studies, the right lobe of the thyroid gland was larger than the left lobe of the gland (data not shown). None of the children had abnormal thyroid scans. There were no significant differences in thyroid volume between males and females. Of the children, those aged 8 y had significantly smaller thyroid glands than those aged 9 and 10 y ($P = 0.004$); there were no differences in thyroid volumes between the 9 and 10-y-olds. The percentage of children with thyroid volumes greater than the age/sex-specific upper limit of normal (> ULN) is compared with the 2001 WHO/ICCIDD reference data (Table 5). Median thyroid volumes based on BSA were also calculated and the percentage of children with thyroid volumes greater than the BSA/sex-specific cut-offs are shown in Table 6.

Discussion

The WHO/ICCIDD/UNICEF criterion for assessing mild IDD in a population is a median urinary iodine concentration between 5.0 and 9.9 μg/dl (WHO *et al*, 1994). The value of 6.6 μg/dl found in this sample of New Zealand schoolchildren clearly indicates mild IDD. Furthermore, WHO/ICCIDD/UNICEF state that no more than 20% of the population should have urinary iodine concentrations

Table 4 Comparison of thyroid volumes (ml) of children in this study with other studies of similarly aged children

	This study		*Other studies*			
Age (y)	n	*Median (IQR)*[a]	*Foo et al (1999)*	*Hess et al (2000)*[b]	*Liesenkötter et al (1997)*	*ICCIDD (2001)*
Girls						
8	36	3.3 (2.8, 4.4)	2.4	2.9	2.5	2.8
9	61	3.6 (2.8, 4.1)	2.9	3.4	2.7	3.1
10	51	3.9 (3.1, 5.2)	3.3	3.6	4.2	3.6
Boys						
8	58	3.3 (2.7, 3.8)	2.5	2.9	2.4	2.6
9	60	3.9 (2.9, 4.7)	2.9	3.4	3.0	2.9
10	32	4.1 (3.2, 4.8)	3.2	3.6	3.8	3.2

[a] IQR, interquartile range.
[b] Median values reported for sexes combined.

Table 5 Percentage of children (95% Confidence interval) with thyroid volumes > upper limit of normal using 2001 WHO/ICCIDD cut-offs by age

Age	*Girls*	*Boys*	*Total*
8	11.3 (0.0–22.8)	10.2 (1.6–18.7)	10.6 (4.3–17.0)
9	5.4 (0.0–10.9)	20.3 (11.8–28.8)	12.9 (8.9–16.9)
10	8.2 (1.3–15.2)	12.6 (0.4–24.7)	9.9 (2.8–17.1)
Total	7.9 (3.3–12.5)	14.6 (9.2–20.1)	11.3 (7.6–15.1)

Table 6 Percentage of children (95% confidence interval) with thyroid volumes >upper limit of normal using 2001 WHO/ICCIDD cut-offs by body surface area (BSA)

BSA (m^2)	n	*Girls*	*Boys*	*Total*
0.8	1	NA	NA	NA
0.9	36	6.6 (0.0–19.9)	24.0 (3.8–44.1)	16.2 (3.2–29.2)
1.0	75	9.1 (0.0–18.4)	24.0 (10.8–37.1)	17.3 (9.9–24.7)
1.1	88	7.6 (0.0–15.5)	17.3 (3.6–31.0)	12.3 (4.8–19.8)
1.2	54	3.4 (0.0–10.3)	8.8 (0.0–21.3)	5.7 (0.0–12.2)
1.3	26	0	15.6 (0.0–35.1)	9.9 (0.0–23.0)
1.4	13	12.5 (0.0–37.6)	0	5.7 (0.0–17.3)
1.5	3	NA	NA	NA
1.6	2	NA	NA	NA
Total	298	6.4 (1.7–11.1)	17.4 (10.4–24.3)	12.0 (7.9–16.1)

NA = not applicable.

less than 5 μg/dl (WHO *et al*, 1994). In the present study, over 30% of the children had levels below this figure. These results support those of Thomson *et al* (1997), who found mild IDD in New Zealand adults. A recent study in Sydney, Australia also found mild IDD as determined by casual urine samples in school-aged children (Li *et al*, 2001).

A prevalence of thyroid volume >ULN between 5 and 20% of children is suggested by WHO/ICCIDD/UNICEF to be indicative of mild IDD (WHO *et al*, 1994). In 1997, the WHO in conjunction with ICCIDD published median and upper normal limits of thyroid volumes for boys and girls, by age and BSA, for the interpretation of ultrasonography data (WHO & ICCIDD, 1997). These data were obtained from the measurements of a large sample of iodine-replete (ie urinary iodine excretion greater than 10 μg/dl) schoolchildren aged 7–15 y from different parts of Europe (Delange *et al*, 1997). When compared to the 1997 WHO reference data, less than 3% of our children had thyroid volumes above this level, a result that is inconsistent with a median urinary iodine excretion of 6.6 μg/dl.

There has been discussion in the literature regarding the appropriateness of the 1997 reference data; the WHO/ICCIDD/UNICEF median and ULN values are similar to those found in a few studies of iodine-replete children (Büurgi *et al*, 1999; Vitti *et al*, 1994), but higher than those found in a number of other studies (Foo *et al*, 1999; Gutekunst & Martin-Teichert, 1993; Hess & Zimmermann, 2000; Liesenkötter *et al*, 1997; Xu *et al*, 1999). Recently a set of provisional reference values has been published that are approximately 30% smaller than the 1997 values, in an effort to correct for the large systematic measurement bias shown to be apparent in the 1997 reference data (Zimmerman *et al*, 2001). When compared to the provisional 2001 WHO/ICCIDD reference values by sex and age, 11.3% of the children studied here had enlarged thyroid glands, indicating mild IDD in this group of children, a result that is consistent with the median urinary iodine excretion of 6.6 μg/dl. When the 2001 reference values were based on sex and BSA, a similar (12.0%) prevalence of goitre was found.

It is interesting to note that, regardless of whether the 2001 thyroid volume reference values are based on age or BSA, in our study more boys than girls had enlarged thyroid glands. There is no reason to believe that girls in New Zealand would have a lower prevalence of goitre than boys in an environment of mild iodine deficiency. Table 4 compares the median thyroid volumes of boys and girls from a number of different studies. There were no significant differences between the median thyroid volumes of boys and girls in this study. Only Delange *et al* (1997) and Foo *et al* (1999) report significant differences in thyroid volumes between boys and girls, while most other studies have found no differences (Hess & Zimmermann, 2000; Langer *et al*, 1994; Liesenkötter *et al*, 1997; Vitti *et al*, 1994; Xu *et al*, 1999). Foo *et al* (1999) state, however, that in their study the differences in thyroid volumes between boys and girls were small and suggest a unisex reference for children aged up to 10 y old. Since the 2001 reference values are based on the data generated by Delange *et al* (1997), almost all the ULN values for girls are higher than for boys by age and BSA. The 95% confidence intervals in our data indicate that a number of girls fall just below the cut-off, suggesting that if the ULN values for girls were lower (ie closer to the values for boys) more girls in our study would have been classified with an enlarged thyroid gland. Xu *et al* (1999) propose that the larger thyroid volumes seen in girls in the Delange *et al* (1997) data might be the result of two factors. Firstly, girls are more affected by marginal iodine deficiency than boys, and secondly, that despite an adequate urinary iodine concentration the thyroid glands of girls take longer to regress than boys after iodine intervention.

It is possible that there is some variation in the thyroid volumes of children, which may be related to factors such as gender, growth, developmental age, iodine status and genetic make-up. However, it is also possible that the measurement of children's thyroid glands using ultrasonography is not as simple as it appears. At present, there is no standardized protocol for assessing the thyroid volume of children using this method and no means of ascertaining the precision of the results obtained. Inter-observer error, type of transducer, type of instrument, and position of the child may all contribute to the variations seen among studies (Anonymous, 2000). Indeed, the recent publication of a set of corrected reference values suggests that researchers need to obtain and interpret ultrasonography data with care. The use of ultrasonography as a tool for assessing the iodine status in children is relatively recent and it is important that it is used in conjunction with other, more established, indices of iodine status such as urinary iodine excretion and, if possible, dietary iodine intakes.

Because most of the iodine consumed in the diet (ie 90%) eventually appears in the urine, our results suggest that iodine intakes of New Zealand children are low. This is not surprising given the limited sources of iodine found in the New Zealand diet. Firstly, New Zealand has low soil iodine levels. Secondly, the use of iodized salt in food manufacturing and processing is not used in New Zealand so that iodized salt can only be consumed if used in the home. Close to 30% of the homes surveyed in this study did not use iodized salt in cooking and 50% of the children in the present study did not use iodized salt at the table. We suggest that public health recommendations to decrease the sodium level of the diet have been interpreted by the New Zealand public as a need to decrease their use of salt in cooking and at the table, rather than to decrease their intake of processed foods. Thirdly, there has been some concern that the level of iodine in dairy products is declining as a result of a decline in the use of iodophors in the New Zealand dairy industry. This decline is anecdotal and has not been documented. Indeed, a government report found that the iodine level of dairy products has not changed in the last 10 y (Cressey & Vannoort, 1998). Finally, alternative sources of iodine commonly found in other countries, such as iodates in bread, are not used in New Zealand and iodine-supplemented feeds for livestock are used less extensively here than elsewhere.

Directly assessing the iodine intake of the diet is complex for a number of reasons. The New Zealand food composition database does not contain the iodine content of foods. Furthermore, most dietary assessment methods do not quantify the contribution of household salt to the diet. The 1997/1998 New Zealand Total Diet Study (NZTDS; Cressey *et al*, 2000) estimated the iodine intake of children aged 4–6 y to be 71 μg of iodine per day, well below the RDA of 90 μg/day; the contribution of discretionary salt to the diets of these children was not included in this estimate. Dairy products contributed to 68% of the total estimated iodine intake of young children (1–3 y) and 42% of the intake of young males (19–24 y). The combined consumption of chicken, eggs, meat and fish was the next largest contributor to iodine intakes after dairy products in the 1997/1998 NZTDS; 15% in young children and 26% in young males.

In the present study, a qualitative FFQ assessed the frequency of consumption, but not the quantity, over the past 3 months, of foods or food groups in children's diets that are good sources of iodine. We found that the majority of the children consumed milk and dairy products on a daily basis, while the remainder consumed such foods weekly. In our study, the majority of children ate meat, chicken, fish and eggs on a weekly basis. Although half of the children in this study used iodized salt at the table, only 14% did so on a daily basis. Of concern were the large number of children who rarely used iodized salt at the table and whose parents did not cook with iodized salt. Although the results of our FFQ are by no means definitive, we believe that they indicate that dairy products probably make the largest contribution to total iodine intakes in New Zealand children. Furthermore, our results highlight the irregular use of iodized salt by New Zealand children of this age group and indicate that iodized salt is unlikely to be making a significant contribution to total iodine intake.

It is important that children obtain sufficient amounts of iodine in the diet to maintain normal growth and development. Although the clinical consequences of mild iodine deficiency found in this sample of New Zealand children are unknown, it seems prudent to increase the iodine intakes of children in New Zealand. Iodized salt has been used for more than 50 y as a cheap, safe and effective means of adding iodine to the New Zealand diet. Reliance on iodized salt used in the home as the sole means of fortifying the New Zealand diet may no longer be appropriate. Other avenues of supplementing the New Zealand diet with iodine, for instance the use of iodized salt in some processed foods, should be explored.

Acknowledgements

We are indebted to Dr Neil Morrison of Otago Radiology for the use of the ultrasound machine and thank ultrasonographers Jill Muirhead, Annemieke Arron and Colin Gerrard. We gratefully acknowledge the help of Natassja Essed and Dianne Brown in Dunedin, Diana Clear in Wellington, technician Jody Joseph, and statistician Jo McKenzie. Finally we would like to thank all the children and their caregivers who participated in the study.

References

Anonymous (2000): Standardization of ultrasound and urinary iodine determination for assessing iodine status: report of a technical consultation. *IDD Newsletter* **16**, 19–23.

Azizi F, Kalani H, Kimiagar M, Ghazi A, Sarshar A, Nafarabadi M, Rahbar N, Noohi S, Mohajer M & Yassai M (1995): Physical, neuromotor and intellectual impairment in non-cretinous schoolchildren with iodine deficiency. *Int. J. Vit. Nutr. Res.* **65**, 199–205.

Benmiloud M, Chaouki ML, Gutekunst R, Teichert H-M, Wood WG & Dunn JT (1994): Oral iodized oil for correcting iodine deficiency: optimal dosing and outcome indicator selection. *J. Clin. Endocrinol. Metab.* **79**, 20–24.

Bleichrodt N, Escobar del Ray F, Morreale de Escobar G, Garcia I & Rubio C (1989): Iodine deficiency, implications for mental and psychomotor development in children. In: *Iodine and the Brain* ed. GR DeLong, J Robbins & PG Condliffe, pp 269–287. New York: Plenum Press.

Bürgi H, Portmann L, Podoba J, Vertongen F & Srbecky M (1999): Thyroid volumes and urinary iodine in Swiss school children. *Eur. J. Endocrinol.* **140**, 104–106.

Cooper GJS, Croxson MS & Ibbertson HK (1984): Iodine intake in an urban environment: a study of urine iodine excretion in Auckland. *NZ Med. J.* **97**, 142–145.

Cressey PJ & Vannoort RW (1998): *Iodine Content of New Zealand Dairy Products*. Wellington: Ministry of Health/Institute of Environmental Science and Research Limited.

Cressey PJ, Vannoort RW, Silvers K & Thomson B (2000): *1997/98 New Zealand Total Diet Survey*. Wellington: Ministry of Health.

Delange F, Benker G, Caron P, Eber O, Ott W, Peter F, Podoba J, Simescu M, Szybinsky Z, Vertongen F, Vitti P, Wiersinga W & Zamrazil V (1997): Thyroid volume and urinary iodine in European schoolchildren: standardization of values for assessment of iodine deficiency. *Eur. J. Endocrinol.* **136**, 180–187.

Foo LC, Zulfiqar A, Nafikudin M, Fadzil MT & Asmah AS (1999): Local versus WHO/International Council for Control of Iodine Deficiency Disorders-recommended thyroid volume reference in the assessment of iodine deficiency disorders. *Eur. J. Endocrinol.* **140**, 491–497.

Gunton JE, Hams G, Fiegert M & McElduff A (1999): Iodine deficiency in ambulatory participants at a Sydney teaching hospital: is Australia truly iodine replete? *Med. J. Aust.* **171**, 467 – 470.

Gutekunst R & Martin-Teichert H (1993): Requirements for goiter surveys and the determination of thyroid size. In: ed. F Delange, Jt Dunn & D Glinoer, *Iodine Deficiency in Europe, a Continuing Concern*, pp 109 – 118. New York: Plenum Press.

Hess SY & Zimmermann MB (2000): Thyroid volumes in a national sample of iodine-sufficient swiss school children: comparison with the World Health Organization/International Council for the Control of Iodine Deficiency Disorders normative thyroid volume criteria. *Eur. J. Endocrinol.* **142**, 599 – 603.

Hetzel BS & Dunn JT (1989): The iodine deficiency disorders: their nature and prevention. *A. Rev. Nut.* **9**, 21 – 38.

Hetzel BS, Potter BJ & Dulberg EM (1990): The iodine deficiency disorders: nature, pathogenesis and epidemiology. *World Rev. Nutr. Diet.* **62**, 59 – 119.

Hollowell JG, Staehling NW, Hannon WH, Flanders DW, Gunter EW, Maberly GF, Braverman LE, Pino S, Miller DT, Garbe PL, DeLozier DM & Jackson RJ (1998): Iodine nutrition in the United States. Trends and public health implications: iodine excretion data from National Health and Nutrition Examination Surveys I and III (1971 – 1974 and 1988 – 1994). *J. Clin. Endocrinol. Metab.* **83**, 3401 – 3408.

Huda SN, Grantham-McGregor SM, Rahman KM & Tomkins A (1999): Biochemical hypothyroidism secondary to iodine deficiency is associated with poor school ahievement and cognition in Bangladeshi children. *J. Nutr.* **129**, 980 – 987.

Langer P, Tajtáková M, Podoba J, Kost'álová L & Gutekunst R (1994): Thyroid volume and urinary iodine in school children and adolescents in Slovakia after 40 years of iodine prophylaxis. *Exp. Clin. Endocrinol.* **102**, 394 – 398.

Li M, Ma G, Guttikonda K, Boyages SC & Eastman CJ (2001): Re-emergence of iodine deficiency in Australia. *Asia Pacific. J Clin. Nutr.* **10**, 200 – 203.

Liesenkötter KP, Kiebler A, Stach B, Willgerodt H & Grüters A (1997): Small thyroid volumes and normal iodine excretion in Berlin schoolchildren indicate full normalization of iodine supply. *Exp. Clin. Endocrinol. Diabetes* **105**, 46 – 50.

Lohman TG, Roche AF & Martorell R (eds) (1988): *Anthropometric Standardization Reference Manual* Champaign, IL: Human Kinetics.

North KAK & Fraser S (1965): Iodine intake as revealed by urinary iodine excretion. *NZ Med. J.* **65**, 512 – 513.

Pino S, Fang SL & Braverman LE (1998): Ammonium persulfate: a new and safe method for measuring urinary iodine by ammonium persulfate oxidation. *Exp. Clin. Endocrinol. Diabetes* **106**, S22 – 27.

Purves HD (1974): The aetiology and prophylaxis of endemic goitre and cretinism: the New Zealand experience. *NZ Med. J.* **80**, 477 – 479.

Shrestha RM (1994): *Effect of Iodine and Iron Supplementation on Physical, Psychomotor and Mental Development in Primary School Children in Malawi*. Wageningen: Grafisch Service Centrum.

Simpson FO, Thaler BI, Paulin EI & Cooper GJS (1984): Iodine excretion in a salt restriction trial. *NZ Med. J.* **97**, 890 – 893.

Sutcliffe E (1990): Iodine in New Zealand milk. *Food Tech. NZ* July, 32 – 38.

Thomson CD, Colls AJ, Conaglen JV, Macormack M, Stiles M & Mann J (1997): Iodine status of New Zealand residents as assessed by urinary iodide excretion and thyroid hormones. *Br. J. Nutr.* **78**, 901 – 912.

Valeix P, Preziosi P, Rossignol C, Farnier M-A & Hercberg S (1994): Relationship between urinary iodine concentration and hearing capacity in children. *Eur. J. Clin. Nutr.* **48**, 54 – 59.

Vitti P, Martino E, Aghini-Lombardi F, Rago T, Antonangeli L, Maccherini D, Paolo N, Loviselli A, Balestrieri A, Araneo G & Pinchera A (1994): Thyroid volume measurement by ultrasound in children as a tool for the assessment of mild iodine deficiency. *J. Clin. Endocrinol. Metab* **79**, 600 – 603.

WHO & ICCIDD (1997): Recommended normative values for thyroid volume in children aged 6 – 15 years. *Bull. WHO* **75**, 95 – 97.

WHO, UNICEF & ICCIDD (1993): *Indicators for assessing iodine deficiency disorders and their control programmes*. WHO/NUT/93.1. Geneva: WHO.

WHO, UNICEF & ICCIDD (1994): *Indicators for assessing iodine deficiency disorders and their control through salt iodization*. WHO/NUT/94.6. Geneva: WHO.

Xu F, Sullivan K, Houston R, Zhao J, May W & Maberly G (1999): Thyroid volumes in US and Bangladeshi schoolchildren: comparison with European schoolchildren. *Eur. J. Endocrinol.* **140**, 498 – 504.

Zimmerman MB, Molinar L, Spehl M, Weidinger-Toth J, Podoba J, Hess S & Delange F (2001): Updated provisional WHO/ICCIDD reference values for sonographic thyroid volume in iodine-replete school-age children. *IDD Newsletter* **17**, 12.