Are breast-fed infants and toddlers in New Zealand at risk of iodine deficiency?

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Abstract

Objective: This study assessed the iodine status of New Zealand infants and toddlers and explored factors that might influence their iodine status.

Methods: A community-based, cross-sectional survey of 6- to 24-mo-old children was conducted in three cities in the South Island of New Zealand. Iodine status was determined by a casual urine sample. Breast-feeding mothers were asked to provide a breast milk sample for iodine determination. Caregivers collected a 3-d weighed diet record from their children to investigate associations between dietary patterns and urinary iodine excretion.

Results: The median urinary iodine concentration for the group (n = 230) was 67 μg/L (interquartile range 37–115) with 37% (95% confidence interval 30.5–43.4) of children having a urinary iodine concentration lower than 50 μg/L. When children were classified by current feeding method, those children who were currently formula-fed had a significantly higher median urinary iodine concentration (99 μg/L) than did children who were currently breast-fed (44 μg/L; P < 0.000). The mean iodine concentration in breast milk was 22 μg/L (n = 39). After multivariate analysis using estimates from 3-d diet records, only percentage of energy from infant formula was significantly associated with urinary iodine concentration (P = 0.005).

Conclusions: This study found mild iodine deficiency in a group of New Zealand infants and toddlers. Children who consumed infant formula, which is fortified with iodine, had better iodine status than did children who were currently breast-fed because breast milk contained low levels of iodine. © 2005 Elsevier Inc. All rights reserved.

Keywords: Iodine; Human milk; Breast feeding; Bottle feeding; Children

Introduction

Iodine deficiency is one of the most common nutrient deficiencies in the world, estimated to affect more than 1 billion people. Iodine deficiency disorders (IDDs) is the term used to describe the wide range of adverse health effects caused by low iodine intakes [1]. These adverse effects can occur throughout life but have the greatest effect during periods of rapid growth and development. In the first 2 y of life, the brain grows rapidly. An inadequate supply of iodine can limit the production of iodine-containing hormones, thyroxine and triiodothyronine, leading to abnormal brain development that can manifest itself in impaired cognitive and psychomotor function [2–6]. It is not surprising, therefore, that many studies have assessed the iodine status of children, particularly those aged 4 to 15 y [7–13]. However, there is clearly a need for more studies investigating the iodine status of children younger than 2 y.

Before the introduction of weaning foods, the iodine intake of the infant depends solely on the iodine content of breast milk or infant formula. Infant formula produced in Australia and New Zealand is permitted to have an iodine content of 30 to 315 μg/L [14]. Levels of iodine in breast milk reflect the iodine intake of the mother. In countries with IDD, breast milk iodine concentrations typically are...
lower than 50 μg/L; when iodine intakes are adequate, breast milk iodine concentrations typically range from 60 to 150 μg/L [15,16]. High concentrations of iodine (e.g., >400 μg/L) are found in the breast milk of women who consume iodine supplements or large amounts of seaweed and those who have been exposed to iodine-containing disinfectants [15,16]. Recent studies in New Zealand, a country with low levels of iodine in soil, have found mild iodine deficiency in some groups of the population [13,17–19]. It is likely, therefore, that breast-feeding women in New Zealand have low dietary iodine intakes, which in turn may result in low concentrations of iodine in their breast milk and, hence, suboptimal iodine status in their breast-fed infants.

To date, no one has examined the iodine status of New Zealand infants who are formula-fed and compared them with breast-fed infants. It is also possible that the iodine intakes of weaned infants and toddlers may not be adequate because foods produced in New Zealand, other than iodized salt and seafoods, are not good dietary sources of iodine [20]. Food manufacturers in New Zealand do not use iodized salt [21]. For this reason, this study investigated the iodine status of New Zealand infants and toddlers and explored factors that might influence their iodine status. This study is part of a comprehensive survey designed to investigate dietary and biochemical trace element status, including iron, zinc and iodine, of a randomly selected sample of healthy 6- to 24-month-old urban South Island New Zealand children. This report presents the iodine results of the survey.

**Materials and methods**

**Study design**

A community-based, cross-sectional survey of 6- to 24-month-old children was conducted in three cities (Christchurch, Dunedin, and Invercargill) in the South Island of New Zealand between May 1998 and March 1999. A casual urine sample, a non-consecutive, 3-d, weighed diet record and general questionnaire were collected from each child during two home visits. A breast milk sample was obtained from those mothers who were still breast-feeding their children. Ethical approval was obtained from the ethics committee of the University of Otago in Dunedin. Informed written consent was given by all participants after the nature of the study had been fully explained to them.

**Recruitment of participants**

Children were randomly selected by using multistage cluster sampling. A detailed description of the recruitment procedure can be found in Soh et al. [22]. In brief, the number of children recruited in each city was in proportion to the population in that city (i.e., 217 children in Christchurch, 66 in Dunedin, and 40 in Invercargill). In each city, address start points were randomly selected across census area units (CAUs) after weighting each CAU for the number of households. Thus, the probability of having an address start point selected per CAU was proportional to the number of households in the CAU. At each start point, 80 households were visited on three separate occasions to minimize the selection bias created by missing an eligible child because household members were not found at home. If there was more than one eligible child in a household, then one was randomly selected to participate in the study. Children were eligible to participate if they were 6 to 24 months of age and apparently healthy. Of the eligible 532 children identified, 323 agreed to participate in the study (response rate 61%). However, a urine sample was not obtained from 93 children, resulting in a final sample size of 230 children for the iodine component of the study (final response rate 43%).

**Biochemical assessment**

A casual urine sample was obtained from children during the first home visit. At the beginning of the visit, the genital region of the child was thoroughly cleaned and the child was fitted with a pediatric urine collection bag (pediatric size U-Bag, Hollister Inc, Libertyville, IL, USA). A disposable diaper was then loosely placed on the child. If urine was passed during the home visit, the collection bag was removed and an aliquot of the urine placed in a test tube. If urine was not passed during the home visit, the primary caregiver was asked to keep the collection bag on the child for another hour. An aliquot of urine was then placed in a specimen bottle and kept refrigerated until collection within the next 24 h. All urine samples were stored at −20°C until analysis. The main reasons that urine samples were not obtained from children were the collection bag becoming displaced, the child not producing urine during the allotted time, or the child refusing to have the collection bag fitted. The iodine content of the samples was determined by method A recommended by the International Council for the Control of Iodine Deficiency Disorders (ICCIDD) [23]. A certified reference sample (Seronorm, Sero AS, Asker, Norway) was analyzed, with each batch of samples producing an analyzed value of 93 μg/L (expected value 97 μg/L) and a coefficient of variation of 2.1% (n = 42).

**Dietary assessment**

A 3-d weighed diet record was collected by the primary caregiver of each child by using dietary scales accurate to within 1 g (Salter Electronic, Salter Housewares Ltd, Tonbridge, UK). The randomly selected, non-consecutive 3 d included 2 weekdays and 1 weekend day over a 3-wk period. Efforts were made to ensure that all days of the week were represented and that different days of the week were equally chosen for the first, second, or third recording day. The caregiver of the child was given detailed written and oral instructions on how to record the child’s diet and
received a schedule of the days to be recorded. A telephone call was made to the caregiver the day before and another on the first day of dietary recording to provide encouragement and to answer any questions. Caregivers were also asked to record the child’s health status on each recording day and the effect of any illness on the child’s food intake. A research assistant checked the diet record with the caregiver within a few days of completing the third and final day of recording.

Diet records were analyzed with the software program Diet Cruncher 1997 [24] using the New Zealand Food Composition Database [25] to estimate the energy and nutrient intakes of each child and the food sources of energy and nutrients. The database does not contain the iodine content of foods so iodine intakes could not be calculated.

**Breast milk iodine**

All breast-feeding mothers (n = 79) were asked to provide a breast milk sample. Consenting breast-feeding mothers (n = 39) were supplied with a detailed set of instructions and an electric breast pump (Mini Electric, Medela AG, Baar, Switzerland) on the second home visit. Women were asked to collect approximately 120 mL of breast milk into an acid-washed opaque polystyrene bottle between 1:00 and 3:00 pm on a single day. The sample included fore and hind milk, although to date there is no evidence that iodine concentrations in breast milk vary with regard to fore or hind milk, time of day, or right or left breast. Further, studies investigating whether iodine concentrations change with time of lactation are inconsistent [15]. After collection, mothers were requested to place the breast milk sample in their home freezer until collected by a research assistant. Breast milk samples were then stored at −20°C until analysis. Breast milk iodine was measured with the Sandell-Kolthoff [26] reaction with a Technicon Auto-Analyzer II (Technicon Instruments Corporation, Tarrytown, NY, USA) [27]. This method has an average recovery of 99% and a coefficient of variation of 3.1% (n = 110).

**Statistical analysis**

All statistical analyses were performed with STATA 7.0 (STATA Corp., College Station, TX, USA) by using the set of survey commands to adjust for the complex sampling design. Spearman’s correlation and chi-square analysis were used to determine associations between continuous and categorical variables, respectively.

Multiple linear regression analysis was used to determine dietary (formula, dairy, meat, meat products, fish, and eggs), socioeconomic (income), and demographic (age and sex) variables associated with urinary iodine. Urinary iodine concentration was log-transformed to normalize the distribution. The exponential of the β-coefficients from regression analysis with log-transformed outcome data provided comparisons between predictor variables on a ratio scale.

The model building process consisted of initially including variables that in univariate analyses with urinary iodine had a P less than 0.25. Variables that had been excluded were then re-entered to assess their effect on the parameter estimates of variables already included in the model. Variables that were not significantly associated with urinary iodine and did not affect parameter estimates of other dietary variables in the model by more than 10% were removed. Age and sex were retained in the model on biological grounds. Energy was retained in the model to adjust for differences in intakes [28]. Model assumptions were tested using standard techniques.

**Results**

Of the 323 children who agreed to participate in the study, 93 children did not provide a urine sample. There were no statistically significant differences between those children who provided a urine sample and those children who did not with regard to the following factors: sex (P = 0.869), age (P = 0.514), ethnicity (P = 0.276), household income (P = 0.599), caregiver’s use of iodized salt (P = 0.377), and current feeding method (i.e., breast feeding and/or formula feeding; P = 0.467). Table 1 outlines the demographic and socioeconomic characteristics of the 230 children who provided a urine sample and their caregivers.

<table>
<thead>
<tr>
<th>Table 1</th>
<th>Subject characteristics (n = 230)*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Boys</td>
<td>57 (131)</td>
</tr>
<tr>
<td>Age</td>
<td></td>
</tr>
<tr>
<td>Infant</td>
<td>33 (75)</td>
</tr>
<tr>
<td>Toddler</td>
<td>67 (155)</td>
</tr>
<tr>
<td>White</td>
<td>86 (197)</td>
</tr>
<tr>
<td>Household income†</td>
<td></td>
</tr>
<tr>
<td>Low (&lt;20 000 NZS)</td>
<td>14 (29)</td>
</tr>
<tr>
<td>Medium (20 000-50 000 NZS)</td>
<td>53 (108)</td>
</tr>
<tr>
<td>High (&gt;50 000 NZS)</td>
<td>33 (67)</td>
</tr>
<tr>
<td>Caregiver uses iodized salt</td>
<td>75 (173)</td>
</tr>
</tbody>
</table>

* Values are percentage (number of subjects).
† Not all caregivers answered this question.

The mean concentration of iodine in milk samples obtained from 39 breast-feeding women was 22 μg/L (95% confidence interval [CI] 18–26). The correlation between iodine levels in breast milk in mothers and urinary iodine concentrations of their children was 0.073 (n = 29).

The median urinary iodine concentration of all children was 67 μg/L (interquartile range 37–115), which was between 50 and 100 μg/L and thus indicative of mild IDD (Table 2). We found that 11.7% of children (95% CI 7.9—15.6) had a urinary iodine concentration lower than 20 μg/L, 37.0% (95% CI 30.5–43.4) had a concentration lower than 50 μg/L, and 67.4% (95% CI 60.5–74.3) had a concentration lower than 100 μg/L. There were no significant differences in mean urinary iodine concentrations between...
infants and toddlers \((P = 0.133)\), between boys and girls \((P = 0.776)\), white and non-white children \((P = 0.340)\), children of caregivers with different household incomes \((P = 0.416)\), and between children whose caregivers used iodized salt and those whose caregivers did not \((P = 0.113)\).

Table 2 also presents the median urinary iodine concentration for children by current feeding method (as identified in the 3-d diet record and confirmed by the general questionnaire). The median urinary iodine concentration of 99 \(\mu g/L\) (interquartile range 6.8–16.7) in children currently fed with formula was very close to the 100-\(\mu g/L\) cutoff indicating adequate iodine status [23]. In contrast, the median urinary iodine concentration of children who were currently breast fed was 44 \(\mu g/L\) (interquartile range 23–82), which is in the 25- to 50-\(\mu g/L\) range indicative of moderate IDD. The World Health Organization, ICCIDD, and United Nations Children’s Fund have stated that no more than 20% of the population should have a urinary iodine concentration lower than 50 mu;g/L [23]. In our study, 13.7% (95% CI 4.7–22.7) of children currently fed with formula had a urinary iodine concentration below this level compared with 51.2% (95% CI 36.1–66.2) of breast-fed children.

Children who were currently fed with formula also had a significantly higher (geometric) mean urinary iodine excretion than did children who were currently breast fed \((P = 0.000)\), those currently fed with formula and breast milk \((P = 0.002)\), and those currently fed with neither formula nor breast milk \((P = 0.000;\) Table 3). Compared with children who were currently fed with breast milk, there were no significant differences in the (geometric) mean urinary iodine concentrations of children who were currently fed with formula and breast milk \((P = 0.165)\) and currently fed with neither breast milk nor formula \((P = 0.091)\).

Of the 230 children in the study, the diet records of children who were currently fed with breast milk were excluded because total energy intakes could not be accurately determined \((n = 60)\). In addition, diet records were not obtained from some non-breast-fed children \((n = 8)\), and the diet record of one child was excluded from analysis because the calculated energy intake was suggestive of an error in the recording process \((n = 1)\). As a result, complete diet records were obtained from 161 children. The 3-d diet record was used to examine associations between dietary patterns and urinary iodine excretion. Table 4 presents the results of univariate and multivariate analyses. In the univariate analysis, only age and percentage of energy from formula had a significant effect on urinary iodine excretion. In the multivariate analysis, only percentage of energy from formula was significantly associated with urinary iodine excretion after controlling for total energy, percentage of energy from dairy foods (includes milk and other dairy products), age, and sex. It is estimated from this model that a 5% increase in energy from formula would be associated with a 9% (95% CI 3–16) increase in urinary iodine while maintaining the same total energy and dairy food intake.

**Discussion**

It is surprising that the iodine status of 6- to 24-mo-old children has been the subject of so few studies despite the fact that thyroid hormones play a critical role in brain development in the first 2 y of life. Measurement of iodine concentration in urine is a widely accepted method to determine the iodine status of a population [23]. Collecting a casual urine sample, even from a very young child, is relatively simple and non-invasive. Nevertheless, this is the first study to measure the iodine status of infants and tod-

### Table 2

<table>
<thead>
<tr>
<th>Feeding Status</th>
<th>Median (IQR)</th>
<th>Percentage &lt;50 (\mu g/L)</th>
<th>Percentage &lt;100 (\mu g/L)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Currently formula fed</td>
<td>99 (68–167)</td>
<td>13.7</td>
<td>51.0</td>
</tr>
<tr>
<td>Currently breast fed</td>
<td>44 (23–82)</td>
<td>51.2</td>
<td>83.7</td>
</tr>
<tr>
<td>Currently breast fed and formula fed</td>
<td>59 (39–103)</td>
<td>47.1</td>
<td>70.6</td>
</tr>
<tr>
<td>Currently neither breast fed nor formula fed</td>
<td>59 (36–112)</td>
<td>39.5</td>
<td>66.4</td>
</tr>
<tr>
<td>Total</td>
<td>67 (37–115)</td>
<td>37.0</td>
<td>67.4</td>
</tr>
</tbody>
</table>

IQR, interquartile range.

### Table 3

<table>
<thead>
<tr>
<th>Feeding Status</th>
<th>Mean*</th>
<th>Ratio†</th>
</tr>
</thead>
<tbody>
<tr>
<td>Currently formula fed</td>
<td>105</td>
<td>1.0</td>
</tr>
<tr>
<td>Currently breast fed</td>
<td>40</td>
<td>0.38 (0.26–0.56)</td>
</tr>
<tr>
<td>Currently breast fed and formula fed</td>
<td>56</td>
<td>0.54 (0.39–0.79)</td>
</tr>
<tr>
<td>Currently neither breast fed nor formula fed</td>
<td>56</td>
<td>0.54 (0.39–0.74)</td>
</tr>
</tbody>
</table>

* Geometric mean.
† Ratio of geometric means (95% confidence interval).
dlers in New Zealand. In our study, the median urinary iodine status for this group of 6- to 24-mo-old New Zealand children was 67 μg/L, a value indicative of mild IDD in school children and adults according to the ICCIDD [23]. Further, 37% of the children in the study had a urinary iodine excretion lower than 50 μg/L, which is above the 20% ICCIDD cutoff for mild IDD [23]. Clearly the iodine status of 6- to 24-mo-old urban South Island New Zealand children is suboptimal. The results from our study are consistent with those from studies of other age groups in New Zealand, including schoolchildren, pregnant women, and adult blood donors [13,17–19,29] and provides further evidence that the iodine level of the current New Zealand diet is low. The median urinary iodine concentration of these young New Zealand children is also lower than that of French children aged 10 mo (181 μg/L) and 2 y (134 μg/L) [30], Belgian children aged 6 to 36 mo (101 μg/L) [31] but similar to German toddlers (~50 μg/L) [32].

Until age 4 to 6 mo, the iodine intake of an infant is dependent on the iodine content of breast milk and infant formula. Iodine in breast milk is present primarily (>80%) as inorganic iodide [15]. Organic forms of iodine, mainly as thyroxine and triiodothyronine, are also present in breast milk but do not contribute significantly to total thyroid hormone levels in the infant [15]. By age 3 or 4 mo, these hormones are likely to be destroyed by digestive enzymes. Therefore, adequate iodine needs to be present in breast milk to enable the infant to synthesize sufficient amounts of thyroid hormones. Results of iodine balance studies of full-term and preterm infants have indicated that an iodine intake of 15 μg/kg of body weight per day is needed to maintain a positive iodine balance [33]. This level of intake equates to a breast milk iodine concentration between 100 and 200 μg/L. This is considerably higher than the mean iodine concentration of 22 μg/L found in the mature breast milk collected from 39 women in this study.

Iodine concentrations in breast milk reflect maternal intake, and concentrations in the range of 100 to 200 μg/L have been reported in studies of women living in the Netherlands, Spain, Italy, and the United States [15]. In contrast, low concentrations of iodine (<50 μg/L) have been reported in breast milk samples from women living in Finland, Sicily, Germany, and Zaire, primarily in goitrous regions in those countries [15,16]. To our knowledge, three studies have investigated iodine concentration of breast milk in New Zealand. Two small studies (n < 20) published in 1927 and 1931 and before salt iodization reported breast milk iodine concentrations ranging from 9 to 43 μg/L [34,35]. In 1990 the third study found breast milk iodine concentrations of 50 μg/L in women whose infants were older than 3 mo [36]. These values compare favorably to the mean concentration of 22 μg/L found in our study. Because iodine concentrations in breast milk reflect maternal intake, the low iodine concentration found in breast milk in our study suggests that urban South Island New Zealand mothers who choose to breast feed longer than 6 mo are likely to have low levels of iodine in their diets. A limitation of this aspect of our study is its small sample size, which makes it difficult to extrapolate the results to the wider population. A larger survey of breast-feeding women living throughout New Zealand with infants 0 to 24 mo old should be conducted to confirm our finding.

The reported mean iodine concentration of a variety of infant formulas available in New Zealand is 95 μg/L (range 30–270) [20]. This value is more than four times the average iodine concentration in breast milk observed in this study. There is some evidence to suggest that iodine in breast milk is better absorbed than iodine in infant formulas [15], thereby making a straight numerical comparison simplistic. Nonetheless, in our study, the urinary iodine concentration of infants currently fed with breast milk was significantly lower than that of infants currently fed with
were currently fed with formula (99 indicative of moderate IDD, whereas that of infants who Australia and New Zealand of 90 /H9262 average 18 this study, iodine intakes for a breast-fed child would be on average 18 μg/d of iodine compared with 81 μg/d for a 6-mo-old child fed infant formula. Published iodine requirements for infants 6 mo of age vary from 60 μg/d to 110 μg/d [23,38,39], with a recently proposed requirement for Australia and New Zealand of 90 μg/d for children [21].

Using our data, the iodine intake of an exclusively breast-fed child would not meet any of these recommended requirements. Thus it is not surprising that the median urinary iodine excretion (44 μg/L) of infants who were currently fed with breast milk was lower than the 50 μg/L cutoff indicative of moderate IDD, whereas that of infants who were currently fed with formula (99 μg/L) was very close to the 100-μg/L cutoff suggesting adequate iodine status.

From 6 to 8 mo of age, the recommended diet for infants in New Zealand consists of the progressive introduction of cereals, vegetables, and fruits, foods that are not particularly good sources of iodine. At around 8 mo, meat, fish, eggs, and some dairy foods such as yoghurt may be consumed [40]. Milk and dairy products, although estimated to contribute up to 70% of the total iodine in the diet of a child aged 1 to 3 y [41], are no longer such a good a source of iodine as they were in the 1960s and 1970s, when iodophors were widely used in the dairy industry [42]. Fish and seafood remain excellent sources of iodine but they are eaten only by a small number of children younger than 24 mo, so these foods make at most a minor contribution to the iodine content of the diets of most infants and toddlers [43]. Further, parents are advised not to add salt to foods prepared for infants [40] and, in New Zealand, iodized salt is not used in processed foods including ready-to-eat baby foods. Thus, it is not surprising that children in this study who were currently fed with neither breast milk nor formula had a median urinary iodine concentration indicating inadequate levels of iodine in their diets. Indeed, the New Zealand Total Diet Study of 1997 to 1998 calculated that the iodine intake of children ages 1 to 3 y was only 58 μg/d [41].

A novel aspect of our study was the use of univariate and multivariate analyses to explore associations between dietary patterns of these children and urinary iodine excretion. The variables percentage of energy from formula and age were significantly associated with urinary iodine in univariate analysis. However, only percentage energy from formula remained significant after adjusting for energy, percentage of energy from dairy foods, age, and sex in the multivariate analysis. Each 1% increase in energy from infant formula was associated with a concomitant 1.8% estimated increase in urinary iodine levels. Several reasons may account for the lack of association noted in this study for foods other than infant formula. For example, the amount of iodine provided by the food group may have been too small to affect urinary iodine concentrations, or the measurement error associated with the estimation of intake may have been too large. Alternatively, the number of children who ate the food may have been too small, or the range of percentage of energy intakes provided by these food groups may have been too narrow. Limitations also may be associated with the use of a single casual urine sample as an estimate of iodine intake at the individual level. Differences in fluid consumption could not be accounted for and could affect urinary iodine concentrations. There was also a lag time between the collection of the urine sample on the first home visit and collection of the 3-d diet record.

Because its nutrient composition is regulated and monitored, infant formula may be a better source of iodine than breast milk of New Zealand mothers whose diets are low in iodine. Fortunately, iodine concentrations in breast milk are easily modified and steps should be taken in New Zealand to increase the iodine intakes of lactating mothers. In the short term, this is best achieved by recommending that women who use salt ensure that it is iodized salt and that they regularly consume foods that are rich sources of iodine such as fish and other seafoods. For women who do not use iodized salt and consume little or no seafood, a multiminerol tablet containing 50 to 100 μg of iodine is suggested. In the long term, mandatory iodine fortification of staple foods such as bread may be a more appropriate strategy because it would also benefit infants and toddlers, an age group found to have a low iodine status in this study. Our findings demonstrate the need for ongoing surveillance of the iodine status of populations who live in low-iodine environments such as New Zealand. Given the dynamic nature of modern food consumption patterns, such surveillance is vital to avoid the risks of impaired psychomotor and cognitive development that can occur in children with iodine deficiency.

References

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