Using bread as a vehicle to improve the iodine status of New Zealand children

Meredith Rose, Rosie Gordon, Sheila Skeaff

Abstract

**Aim** To determine the iodine status of a sample of Dunedin school children, and to estimate how the addition of iodised salt to bread will improve their iodine status.

**Method** Between October and November 2007, iodine status in a sample of 93 Dunedin school children was assessed by urinary iodine concentration (UIC), serum thyroglobulin (Tg), and dietary iodine intake, estimated using an iodine-specific food frequency questionnaire. Data from the 2002 National Children’s Nutrition Survey and the New Zealand Food Composition Database were used to calculate the increase in total dietary iodine intake if bread is made with iodised salt, and subsequently, the predicted increase in UIC.

**Results** Both the median UIC of 63 mcg/L (interquartile range (IQR) 44–78 mcg/L) and the median serum Tg concentration of 14 mcg/L (IQR 10–23 mcg/L) classify this sample of children as mildly iodine deficient. The estimated dietary iodine intake was 54 mcg/day (IQR 41–65 mcg/L), which is well below the estimated average requirement (EAR) of 75 mcg/day; 83% of children in this study were found to have iodine intakes below the EAR. The addition of iodised salt to bread would increase the average iodine intake of these children to 75–104 mcg/day, thus decreasing the number of children who have an iodine intake less than the EAR to 4–46%. Consequently, the median UIC of these children would increase to 95–151 mcg/L.

**Conclusion** The introduction of iodised salt to bread, which is currently scheduled to become mandatory in September 2009, should improve the iodine status of New Zealand children. The use of iodised salt in other bakery products is encouraged to maximise this improvement.

Iodine is an essential nutrient because it is needed for the formation of thyroid hormones.\(^1\) Due to glaciation, which causes low soil iodine levels, New Zealand has a history of iodine deficiency.\(^2\)

A study carried out by Hercus in the 1920s reported endemic goitre throughout New Zealand,\(^3\) but with the introduction of iodised salt in 1938 at the level of 40–80ppm, the goitre rate fell to 0.1% by 1958.\(^2,3\)

Recently, however, there has been a re-emergence of iodine deficiency in New Zealand, with several studies reporting mild iodine deficiency in New Zealand children.\(^4,6\) The decline in iodine status has been attributed to a decreased concentration of iodine in milk resulting from the reduced use of iodophors as sanitisers in the dairy industry, an increase in processed foods in the diet which are made with non-iodised salt, an increase in the consumption of more fashionable salt products (such as non-iodised rock or sea salt), and decreased consumption of...
discretionary (i.e. table and cooking) iodised salt.\(^7\) A recent study in the USA reported that the iodine content of discretionary salt may not be consistent, both between brands and within an individual container of salt.\(^8\)

The effects of severe to moderate iodine deficiency on health are unequivocal and well documented, and include cretinism, hypothyroidism, and complications during pregnancy. There is a growing body of evidence that mild iodine deficiency also has adverse health consequences, with studies suggesting a link between mild iodine deficiency and decreased cognitive functioning,\(^9-11\) growth,\(^12\) and an increase in the prevalence of attention deficit hyperactivity disorder (ADHD).\(^13-15\)

A study carried out in Spain (n=1221) in a mildly iodine-deficient population reported that children with urinary iodine concentrations (UIC) below 100 mcg/L, the World Health Organization (WHO) cut-off for adequate iodine status, had significantly lower intelligent quotients (IQs) than children with UICs above this level.\(^9\)

Aghini Lombardi et al (n=270) found that children living in an area of mild iodine deficiency, and children born before iodine prophylaxis was introduced to an area that was now considered iodine sufficient, had slower reaction times than children who had always lived in an area of iodine sufficiency.\(^10\) Riaño Galán et al’s study (n=61) observed that in an area of mild iodine deficiency, children born to mothers with UIC below 200 mcg/L had poorer verbal and cognitive development than children born to mothers with UICs above 200 mcg/L.\(^11\)

A study by Zimmermann et al (n=310) reported that when a mildly iodine-deficient, slightly growth-retarded group of children in South Africa were given iodine supplementation, insulin-like growth factor-1 concentrations subsequently increased, thus suggesting a relationship between mild iodine deficiency and growth.\(^12\) One small study (n=16) carried out in Italy reported higher ADHD prevalence in children born to mothers living in a mildly iodine-deficient area compared to children born to mothers living in an iodine-sufficient area.\(^13\)

Other studies also suggest links between ADHD and resistance to thyroid hormone,\(^14\) and ADHD symptoms and thyroid stimulating hormone (TSH) levels that are elevated but still within the normal range.\(^15\) Together, these studies suggest that mild iodine deficiency may present risks to the cognitive abilities, growth, and behaviour of mildly iodine-deficient New Zealand children.

The decrease in iodine status in New Zealand, and its potential risks, has led to the recent amendment of Standard 2.1.1 – Cereals and Cereal Products in the Australia New Zealand Food Standards Code,\(^16\) which mandates the use of iodised salt in the production of bread (i.e. all yeast containing breads and flat breads that are baked, including sliced bread, bread rolls, pita breads, focaccia, bagels, English muffins, sweet buns, and fruit bread) in New Zealand from September 2009.

The aim of this study was to determine the iodine status of a sample of Dunedin school children, and to estimate how the addition of iodised salt to bread will improve their iodine status.

**Method**

**Participants and recruitment**—A convenience sample of children aged 10–12 years from around Dunedin was obtained by approaching four intermediate schools and two high schools; two
intermediate schools agreed to participate. Children were recruited through presentations in their classroom. Advertisements for participants were also placed in the local community newspaper allowing children from other schools the opportunity to participate. Interested parents and children provided a home address and were sent an information sheet, brochure and consent form in the mail. A total of 93 children aged 10 to 13 years participated in this study. Seventy children were recruited through the two intermediate schools, and 23 were recruited through newspaper advertising.

Data collection—For children recruited through their school, data collection was carried out at school, during the school day. Children who were recruited by newspaper advertising visited the Department of Human Nutrition after school or on a Saturday. Prior to sample collection the parent/guardian of each child completed a questionnaire that included contact details, child’s age, and ethnicity (European/Pakeha, Māori, Pacific [Islander], and Other), family doctor, household income, child’s use of supplements, medical conditions, allergies, medication use, and the perceived health status of the child.

The questionnaire also included an iodine-specific food frequency questionnaire (FFQ), which asked parents to describe the frequency of consumption by the child of foods that we considered the main source of iodine for New Zealand children (i.e. dairy products, milk, red meat, poultry, fish, shellfish, pulses and legumes, fruit and eggs).

Frequency categories included ‘never’, ‘less than once a week’, ‘1–3 times per month’, ‘once a week’, ‘2–4 times a week’, ‘5–6 times per week’, ‘once per day’, or ‘2 or more times a day’. All data was collected between October and November 2007.

Upon arrival at their appointment children had their height and weight measured using standard anthropometric techniques. For measurements children removed their shoes, emptied their pockets and, in most cases, wore their school uniform. A causal urine sample was obtained from each child. Children were asked to urinate in a bowl and then one of the investigators transferred approximately 5 mL of urine into a clean plastic specimen container (Labserv Technologies, Canada). Urine was frozen at -20°C until analysis.

Approximately 1 mL of non-fasting, whole capillary blood was collected by a finger prick blood sample from each child using a Tenderlett blood collection device (International Technidyne Corporation, New Jersey, USA). Blood was left to clot at room temperature for 60 minutes before being spun by centrifuge for 10 minutes. Serum was stored at -20°C until analysis.

Data analysis—Raw anthropometric data was entered into EpiInfo (CDC, Atlanta, GA, USA) and z-scores for height-for-age (HAZ) and weight-for-age (WAZ) were calculated using the National Centre for Health Statistics (NCHS)/WHO 1979 growth reference data. (This set of growth reference data was chosen because it better represents the growth patterns of New Zealand children in this age group and was formulated using standardised methodologies.) Calculations of descriptive statistics was carried out using Microsoft Excel 2008 software.

UIC was measured using method A as recommended by the International Council for the Control of Iodine Deficiency Disorders (ICCIDD). A certified reference material (Seronorm Trace Elements Urine, Sero AS, Asker, Norway) was used with each batch of samples and gave a mean iodine concentration of 132 mcg/L, within the expected range of 132–150 mcg/L, and a coefficient of variation (CV) of 4.4% (n=40). An internal standard (i.e. pooled urine sample) was also analysed with each batch of samples and gave a CV of 2.9% (n=20).

Tg concentration was measured from serum samples by Endolab, Christchurch Hospital, Christchurch. Samples were tested for the presence of auto-antibodies to Tg (TgAb) as these antibodies can interfere with Tg determination. Five fasting samples tested positive for TgAb and the results from these subjects were not included in Tg data analysis. Both Tg and TgAb were measured using immunoenzymatic assays with cheliluminscence detection. The Tg assay had an analytical detection limit of 0.1 µg/L and accuracy checked using the CRM 457 Tg standard (European Community Bureau of Reference). The inter-assay CV was 25% at 0.2 mcgTg/L, 8% at 40.4 mcgTg/L, and 5% at 333 mcgTg/L. Intra-assay CV’s were 5% at 0.2 mcgTg/L, 2% at 40.4 mcgTg/L, and 2% at 333 mcgTg/L.

UIC and serum Tg were skewed and are presented as medians and interquartile ranges.

Dietary iodine intake—Using the data obtained from the FFQ, the average total iodine intake was calculated using data (i.e. iodine content and serving sizes) from the New Zealand Food Composition Database (FOODfiles, 2006). This information was then combined with data from the 2002 National
Children’s Nutrition Survey (CNS02) on children’s bread consumption to estimate the effect the introduction of iodised salt to bread will have on the average dietary iodine intake of these children. The formula of the US Institute of Medicine\textsuperscript{20} to estimate average dietary iodine intake from UIC (Daily intake = UIC/0.92 × [0.0009L/hr/kg × 24hr/day × weight]) was used to calculate the average change in UIC from the predicted change in dietary iodine levels.

**Ethical approval**—Ethical approval for this study was obtained from the University of Otago Ethics Committee. All children and their parents provided written informed consent before participating in the study.

**Results**

The study population (n=93) included 43 females and 50 males aged 10–13 years. The sociodemographic characteristics of the study population are summarised in Table 1. The median height of the study population was 152.4 cm, and the HAZ was 0.26 standard deviations greater than the reference population. The median weight was 42 kg, and the WAZ was 0.27 standard deviations greater than the reference population.

The iodine status of the population is summarised in Table 2. Both the median UIC and the median Tg concentration are indicative of mild iodine deficiency according to WHO/United Nations Children’s Fund (UNICEF)/ICCIDD.\textsuperscript{18} The estimated dietary iodine intake of 54 mcg/day is well below the EAR of 75 mcg/day for children of this age group.\textsuperscript{21}

One participant had a much higher daily iodine intake (179 mcg/day) compared to the other participants based on the FFQ results; this participant reportedly ate fish daily and other seafood 2–3 times a week. Sixty percent of parents reported that their child used iodised salt at the table, and 70% of parents reported using iodised salt during cooking. However, only 30% of children reported they used iodised salt regularly (i.e. more than once a day).

From the New Zealand Food Composition Database (FOODfiles, 2006)\textsuperscript{19} bread contains anywhere from trace amounts to 8 mcg of iodine per 100 gm; the median iodine content across all breads listed is 1 mcg iodine per 100mg. Bread also contains from 330 to 750 mg sodium per 100 gm. If we assume that 90% of sodium in bread comes from salt, then the sodium from salt content of bread can be calculated to be 300 to 675 mg per 100 gm.

After September 2009, this salt could contain from 25 to 65 ppm iodine; however, in reality, salt manufactures will aim for the mid point of 45 ppm to ensure they are within this range. Using the formula current iodine concentration in bread + (salt content of bread × concentration of iodine in salt), the new iodine content of bread was calculated to range from 15 to 31 mcg per 100 mg of bread (see Table 3).

Approximately 10% of iodine is lost during the baking process, decreasing the amount of available iodine in 100 gm of bread to from 14 to 28 mcg (see Table 3).
Table 1. Sociodemographic characteristics of study population

<table>
<thead>
<tr>
<th>Variable</th>
<th>Number of participants</th>
<th>Percentage</th>
<th>National data*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gender</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Female</td>
<td>43</td>
<td>46%</td>
<td>49%</td>
</tr>
<tr>
<td>Male</td>
<td>50</td>
<td>54%</td>
<td>51%</td>
</tr>
<tr>
<td>Age</td>
<td></td>
<td></td>
<td>na†</td>
</tr>
<tr>
<td>10–10.99 years</td>
<td>7</td>
<td>8%</td>
<td></td>
</tr>
<tr>
<td>11–11.99 years</td>
<td>41</td>
<td>44%</td>
<td></td>
</tr>
<tr>
<td>12–12.99 years</td>
<td>44</td>
<td>47%</td>
<td></td>
</tr>
<tr>
<td>13–13.99 years</td>
<td>1</td>
<td>1%</td>
<td></td>
</tr>
<tr>
<td>Ethnicity</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>European/Pakeha</td>
<td>73</td>
<td>79%</td>
<td>67%</td>
</tr>
<tr>
<td>Māori</td>
<td>13</td>
<td>14%</td>
<td>14%</td>
</tr>
<tr>
<td>Pacific‡</td>
<td>4</td>
<td>4%</td>
<td>7%</td>
</tr>
<tr>
<td>Other</td>
<td>3</td>
<td>3%</td>
<td>12%</td>
</tr>
<tr>
<td>Parental income</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt;$20,000</td>
<td>6</td>
<td>6%</td>
<td>9%</td>
</tr>
<tr>
<td>$20,000–$50,000</td>
<td>22</td>
<td>24%</td>
<td>32%</td>
</tr>
<tr>
<td>&gt;$50,000</td>
<td>44</td>
<td>47%</td>
<td>59%</td>
</tr>
<tr>
<td>Declined to answer</td>
<td>21</td>
<td>23%</td>
<td></td>
</tr>
<tr>
<td>Health status</td>
<td></td>
<td></td>
<td>na</td>
</tr>
<tr>
<td>Excellent</td>
<td>77</td>
<td>84%</td>
<td></td>
</tr>
<tr>
<td>Fair</td>
<td>14</td>
<td>16%</td>
<td></td>
</tr>
<tr>
<td>Poor</td>
<td>0</td>
<td>0%</td>
<td></td>
</tr>
</tbody>
</table>

*from NZ Census 2006 data‡; †na = not applicable; ‡mostly of Samoan, Tongan, Niuean, or Cook Islands origin.

Table 2. Iodine status of the study population

<table>
<thead>
<tr>
<th>Variable</th>
<th>Median (IQR)*</th>
<th>Recommended value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dietary iodine (mcg/day)</td>
<td>54 (41–65)</td>
<td>75†</td>
</tr>
<tr>
<td>Percentage less than EAR</td>
<td>83%</td>
<td>&lt;50%</td>
</tr>
<tr>
<td>UIC (mcg/L)</td>
<td>63 (44–78)</td>
<td>100–199‡</td>
</tr>
<tr>
<td>Percentage &lt; 50 mcg/L</td>
<td>33%</td>
<td>&lt;20%</td>
</tr>
<tr>
<td>Percentage &lt;100 mcg/L</td>
<td>88%</td>
<td>&lt;50%</td>
</tr>
<tr>
<td>Tg (mcg/L)</td>
<td>14 (10–23)</td>
<td>&lt;10 &lt;8</td>
</tr>
</tbody>
</table>

*IQR = Interquartile range
†EAR for NZ Children aged 9–13 years
‡WHO/UNICEF/ICCIDD cut-offs for adequate iodine status

Results from CNS02 indicate that, on average, children consume from 147 to 180 gm of bread products per day, thereby the iodine consumed from bread products after the addition of iodised salt will range from 21 to 50 mcg/day. When this was applied to the study population’s current iodine intake, it raised the median intake from 54 mcg I/day to 75–104 mcg I/day. This would result in 4–46% of the children in this study having iodine intakes less than the EAR.
Table 3. Proposed Range of Iodine Concentration of Bread due to varying salt content, and varying levels of salt iodisation

<table>
<thead>
<tr>
<th>Variable</th>
<th>Current iodine content of bread mcg/100 gm</th>
<th>Salt content of bread mg/100 gm</th>
<th>Concentration of iodine in salt ppm</th>
<th>New iodine content of bread mcg/100g</th>
<th>Iodine available after baking mcg/100g</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lowest level of salt in bread</td>
<td>1 (0.00–8.00)</td>
<td>300</td>
<td>45</td>
<td>15 (14–22)</td>
<td>14 (13–20)</td>
</tr>
<tr>
<td>Highest level of salt in bread</td>
<td>1 (0.00–8.00)</td>
<td>675</td>
<td>45</td>
<td>31 (30–38)</td>
<td>28 (27–34)</td>
</tr>
</tbody>
</table>

The predicted improvement in iodine status of the study population due to the introduction of iodised salt to bread at the expected level is presented in Table 4 and shows that the calculated amount of iodine that will be added to the diet would result in a median UIC of 95–151 mcg/L. This would lead to 2–6% of the children from this study having UICs less than 50 mcg/L.

Table 4. Predicted iodine status of the study population after iodised salt is added to bread

<table>
<thead>
<tr>
<th>Variable</th>
<th>Median (IQR)</th>
<th>Recommended value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dietary iodine (mcg/day)</td>
<td>75–104</td>
<td>75 *</td>
</tr>
<tr>
<td>Percentage less than EAR</td>
<td>4–46%</td>
<td>&lt;50%</td>
</tr>
<tr>
<td>UIC (mcg/L)</td>
<td>95–151</td>
<td>100–199</td>
</tr>
<tr>
<td>Percentage &lt; 50 mcg/L</td>
<td>2–6%</td>
<td>&lt;20%</td>
</tr>
<tr>
<td>Percentage &lt;100 mcg/L</td>
<td>6–60%</td>
<td>&lt;50%</td>
</tr>
</tbody>
</table>

* EAR for NZ Children aged 9–13 years.

Discussion

Both the median UIC and the median serum Tg concentration indicate the presence of mild iodine deficiency in this population of Dunedin schoolchildren. The WHO/UNICEF/ICCIDD criterion for identifying mild iodine deficiency in a population is a median UIC from 50–99 mcg/L.

The value of 63 mcg/L found in the current study clearly indicates the presence of mild iodine deficiency. Additionally, WHO/UNICEF/ICCIDD state that no more than 20% of the population should have UICs less than 50 mcg/L. In this sample of children, 33% of the children had a UIC below this level.

These results support those of Skeaff et al who reported a median UIC of 66 mcg/L in Dunedin and Wellington schoolchildren (n=300), and the results of the CNS02 (n=3275), which found a median UIC of 66 mcg/L in a nationally representative sample of children between the ages of 5 and 14 years.

The WHO/UNICEF/ICCIDD criterion for identifying mild iodine deficiency in a population using serum Tg is a median Tg concentration from 10–19.9 mcg/L. The value of 14.4 mcg/L found in the current study indicates the presence of mild iodine deficiency in these children, and supports the UIC results.
As stated previously, the results of a large cross-sectional study carried out in Spain indicate that mild iodine deficiency puts children at risk of impaired cognition, and a randomised controlled trial has found that when mildly iodine deficient children are given supplementation, concentrations of growth hormones increased, suggesting that iodine deficiency can impair growth.

The results of the current study indicate that New Zealand children are unlikely to be affected by any iodine deficiency induced growth retardation as both HAZ and WAZ of the study population are greater than those of the reference population. However, the potential risk to cognition is a cause for concern.

In the current study, iodised salt was used at the table by 60% of the children. This figure is slightly higher than that reported in other studies, although use of salt in cooking by 70% of children is similar to other studies carried out in New Zealand. Skeaff et al’s study of 8 to 10 year olds in Dunedin and Wellington found that 50% of children used salt at the table, and 69% of caregivers reported using salt in cooking. Data from the CNS02 reported that 13% of children usually added salt at the table, 36% added salt sometimes, and 52% never added salt to meals at the table.

While iodised salt was used by 70% of the children in the current study, only 30% used salt regularly (i.e. once a day or more), indicating that discretionary use of iodised salt is likely to be making only a minor contribution to the total dietary iodine intake in these children.

The mean daily iodine intake estimated with the FFQ in this sample of children was 54 mcg/day; 83% of children in this study had dietary iodine intakes less than the EAR of 75 mcg/day. This value is comparable to the estimated mean dietary iodine intake reported in the 2003/04 NZTDS, which for 11–14 year old males was 60 mcg/day, and for 11–14 year old females was 50 mcg/day.

In our study iodised salt use was not included when quantifying the daily intake of iodine from the FFQ, meaning that the iodine intake could have been underestimated and may account for the discrepancy between the estimated dietary iodine intake of 54 mcg/day and the median UIC of 63 mcg/L. However, the less than regular use of iodised salt by more than two thirds of the children makes it unlikely that including iodised salt use would have markedly increased the dietary iodine intake measured here. Nonetheless, the FFQ results need to be interpreted with an element of caution.

While FFQs are frequently used in studies due to their low respondent burden, they are not the most accurate method of dietary assessment. Additionally, the FFQ used in this study has not been validated for use in children. Despite these limitations, the fact that the pattern of foods contributing to dietary iodine intake is similar to that of the 2003/04 New Zealand Total Diet Survey, and that unusually high iodine intakes can be easily attributed to specific food groups suggests that that the FFQ used in this study gave a reasonable representation of dietary iodine intake.

From the FFQ an average serving of milk was calculated to contain 20 mcg of iodine, while an average serving of seafood was calculated to contain 152 mcg iodine. However, on average, servings of milk were consumed 1.24 times a day, meaning that milk contributed 43% of the dietary iodine intake, while servings of seafood were only consumed 0.03 times a day, making the contribution of seafood only 9% of the average dietary iodine intake.
The results of one participant who regularly consumed fish and other seafood indicate that frequent consumption of these foods can increase daily iodine intake to above the recommended dietary intake (RDI) of 120 mcg/day. Regardless, recommending an increase in seafood consumption would not be practical way of increasing iodine intake in this age group, as many children find seafood unpalatable, and fish and other seafood is expensive in New Zealand. It seems unlikely that individual dietary modification would be a practical way of increasing iodine intake to the recommended level in this age group.

The recent amendment of Standard 2.1.1 – Cereals and Cereal Products in the Australia New Zealand Food Standards Code\(^\text{16}\) will require the mandatory use of iodised salt in the production of most breads after September 2009. While we cannot ascertain exactly how much bread individual children of this age group will need to consume to improve their individual iodine status, we have estimated that the addition of iodised salt to bread will, on average, increase children’s iodine intakes from 21 to 50 mcg/day and that this increase in iodine intake would decrease the number of children who have an iodine intake less than the EAR to 4-46%, compared to the 83% observed in the current study.

Using the US Institute of Medicine formula\(^\text{20}\) we predicted that the median UIC would increase to 95–151 mcg/L, and that consumption of bread that contains the highest proportion of salt, would result in only 2% of the children having UICs less than 50 mcg/L and only 6% of children having UICs <100mcg/L. According to WHO/UNICEF/ICCIDD, this would mean that the children would have adequate iodine status.

However, with the current emphasis from the National Heart Foundation for the reduction of the sodium content of commercially made bread, the possibility that bread will be able to continue to deliver sufficient iodine to significantly improve iodine status is a concern. It seems prudent, therefore, to encourage bread manufacturers to use iodised salt in all the bakery products they produce to maximise the increase in dietary iodine.

Additionally, a comprehensive monitoring programme should be established to monitor the impact of adding iodised salt to bread, and to allow for the early identification of a reduction in the salt content in bread that might impact on the iodine status of the population. It may be that at some time in the future a vehicle other than salt will need to be identified to ensure adequate intake of iodine in the New Zealand population.

Concerns have been raised over the potential for toxic intakes of iodine with the implementation of mandatory fortification of bread with iodised salt, especially in younger children. However, calculations using the dietary information from the one child in this study with a current iodine intake of 179 mcg/L suggest that it is highly unlikely that a child aged 10–14 years with an unusually high iodine intake would reach the current upper level of intake (UL) of 600 mcg/L.

If this child consumed the higher amount of bread recorded in the CNS (180 g), which contained the highest amount of salt (660 mg/100 gm), with the highest level of salt iodisation (65 ppm), this child would only be consuming 256 mcg iodine/day which is less than half of the UL. Even if this child ate 350 g of bread a day, which equates to...
between 6 and 8 slices of bread, their daily iodine intake would be 329 mcg/day, which is still well below the UL.

The findings of this study indicate that using bread as a vehicle for introducing more iodine to the diet can improve the iodine status of New Zealand children, and could reduce the risk of complications associated with mild iodine deficiency. However, decreasing the salt content of bread has the potential to limit the effectiveness of this intervention, and bread producers should be encouraged to use iodised salt in as many bread products as possible to maximise improvement to the iodine status of New Zealand children.

Competing interests: None known.

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References:


