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Review article

Selenium and iodine intakes and status in New Zealand and Australia

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Most New Zealand soils contain relatively low concentrations of the anionic trace elements F, I and Se. Some areas of Australia also have a history of I deficiency. In view of current interest in establishing nutrient reference intakes for Se and I in New Zealand and Australia, it is timely to review current understanding of the intakes and status of these two elements. In spite of a recent increase in Se status, the status of New Zealanders remains low compared with populations of many other countries and may still be considered marginal, although the clinical consequences of the marginal Se status are unclear. There are no recent reports of blood Se levels in Australia, but earlier reports indicate that they were generally greater than those of New Zealanders. Similarly, the consequences of decreasing I status in Australia and New Zealand are unclear. Mild I deficiency in New Zealand has resulted in enlarged thyroid glands indicating an increased risk of goitre. Currently there is little evidence, however, of any associated clinical disease. Public health recommendations to reduce salt intake, together with the reduction in I content of dairy products, are likely to result in further decreases in the I status of New Zealand and Australian residents. Some action is needed to prevent this decline and it may be necessary to consider other means of fortification than iodized salt. The consequences of possible interactions between Se and I in human nutrition are also unclear and no practical recommendations can be made.

Selenium: Iodine: Selenium status: Iodine status: New Zealand: Australia

Most New Zealand soils contain relatively low concentrations of the anionic trace elements F, I and Se. This, in part, has highlighted the risks of deficiency, and has led to considerable research on nutritional aspects of these trace elements in New Zealand (Robinson, 1989, 1992; Thomson & Robinson, 1996). I-deficiency goitre was very common in the late 1800s and early 1900s before the iodization of salt in 1924. Some parts of Australia also have a history of I deficiency (Stewart *et al.* 1971; Gibson, 1995). There is little evidence, however, that Se nutrition is inadequate in Australia, and the importation of Australian wheat with relatively high Se concentrations may have prevented potential Se deficiency in some parts of New Zealand. In view of current interest in establishing reference nutrient intakes for Se and I in Australasia, it is timely to review current understanding of the intakes and status of these two elements.

Selenium intakes in New Zealand and Australia

Food sources of selenium

Food is the major source of Se, with drinking water and air being minor contributors (Barceloux, 1999). Dietary intake varies with the geographical source of the foods and the eating habits of the local populations. Soil Se concentrations around the world typically range from $<0{\cdot}01$ μg/g in Se-deficient areas to >1000 μg/g elsewhere. Plant-food concentrations roughly reflect the Se concentration of the soils in which plants are grown. Hence, dietary intakes around the world can range from <10 μg/d in the low soil-Se areas of China where Keshan disease is endemic (Yang *et al.* 1983) to 20–60 μg in New Zealand (Thomson & Robinson, 1988; Russell *et al.* 1999) and up to 5000 μg/d in seleniferous areas of China (Yang *et al.* 1983). Some plant foods are particularly high in Se, including Brazil nuts and mushrooms (Thomson & Robinson, 1988; Vannoort *et al.* 2000).

Variability in Se concentrations is less evident in animal foods: fish and organ meats are the richest sources followed by muscle meats, but the bioavailability of Se is greatest from Se-enriched yeast, cereals and grains. Dairy products, fruits and vegetables tend to be the poorest sources of Se (Thomson & Robinson, 1988). In New Zealand (Thomson & Robinson, 1988, 1990; Vannoort *et al.* 2000) and Australia (Fardy *et al.* 1989, 1994; Tinggi *et al.* 1992; Tinggi, 1999) the main sources of Se in the

Abbreviation: GPx, glutathione peroxidase.

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diet are seafood, poultry and eggs, and to a lesser extent other muscle meats. The contribution of cereals to dietary Se intakes varies with the source of the crop.

Selenium intakes in New Zealand

Se intakes in New Zealand have increased during the past 10 years, due largely to an increase in the importation of Australian wheat and other cereal products and to an increased use of supplemental Se in animal feeds in New Zealand (Thomson & Robinson, 1996). Early studies indicated mean Se intakes, determined from duplicate diets, were <30 μg/d (Robinson & Thomson, 1987). Even in 1999, intakes as low as 28 μg/d (determined by duplicate diets and diet records) were found in Dunedin residents with low Se status and who had been asked not to consume high-Se foods such as fish, liver, kidney and Brazil nuts (Duffield & Thomson, 1999). A recent study of Dunedin smokers showed mean intakes from diet records of 56 μg/d for male and 38 μg/d for female subjects (Paterson, 2000). Similar intakes, determined by 24 h recall, were reported from the New Zealand National Nutrition Survey (Russell *et al.* 1999). Median intakes were 56 (10th percentile 36, 90th percentile 91) μg/d and 39 (10th percentile 25, 90th percentile 68) μg/d for male and female subjects respectively. Se intakes in non-breast-fed infants and toddlers, determined using diet records, were 7·9 (SD 6·2) and 13·7 (SD 8·4) μg/d respectively (McLachlan, 2003), while those estimated from 5–15-year-old children from the pilot pre-test of the Children's Nutrition Survey using a computerized 24 h recall, were 34 (SD 24) μg/d (Scragg & Metcalf, 2001) (Table 1).

The New Zealand Food Composition Database for Se, however, may not be sufficiently accurate to reliably estimate Se intakes because of the regional variation of Se concentrations in bread and other wheat products. This is due to the greater use of Australian wheat (100 %) in bread-making in the north of the North Island, while in the South Island all wheat used is normally grown locally. In the south of the North Island about 30–35 % of wheat used is Australian (N Athar, New Zealand Institute of Crop and Food Research, personal communication). A further factor is that some bread manufactured in the North Island is distributed in the South Island. The 1997–98 New Zealand Total Diet Survey found Se concentrations in white bread of about 25 μg/kg in Dunedin and Christchurch (South Island), 80–104 μg/kg in Napier (mid-east of the North Island) and 78–114 μg/kg in Auckland (north of the North island). The mean value for New Zealand, from which intake data for the country are calculated, is 59 μg/kg (Vannoort *et al.* 2000). Intakes were derived from the 1997–98 Total Diet Survey; this involved sampling 114 different foods, of which 105 were considered to be those most commonly consumed by the majority of New Zealanders, and simulation of diets from available data on food consumption patterns. Estimated intakes were somewhat higher than those measured by dietary assessment methods, with mean intakes of 78–82 μg/d in male subjects, 55 μg/d in female subjects and 25–30 μg/d in children (Vannoort *et al.* 2000).

Selenium intakes in Australia

There has been only limited study of dietary Se intakes in Australia (Tinggi, 2003). Estimates of Australian Se intakes from the National Market Basket Survey in which fifty representative foods from each of the State capitals were analysed, and combined according to hypothetical diets, were 87 μg/d for adult male subjects,

Table 1. Selenium and iodine intakes (μg/d) for New Zealand and Australian adults

				Male		Female		
	Year	Methodology	*n*	Mean	SD	Mean	SD	Author(s)
Se								
New Zealand	1973	Duplicate diets	F, 4			24	20–34*	Robinson & Thomson (1987)
	1983		F, 4			11·4	9–14*	
	1997	Duplicate diets	M, 15; F, 28	35	7	26	14	Duffield & Thomson (1999)
		Diet records		30	13	27	16	
	1997	24 h recall		56†	36, 91‡	39†	25, 68‡	Russell *et al.* (1999)
	1997–98	Simulated diets		78–82*§		55*¶		Vannoort *et al.* (2000)
	1998–99	Diet records	F, 302			38	25	McLachlan (2003)
	1999	Diet records	M, F	59	19	38	21	Paterson (2000)
Australia	1989	Simulated diets		87	24	57	12	Fardy *et al.* (1989)
	1993	Diet records		89	35–204*	59	21–141*	Reilly (1993)
I								
New Zealand	1997	Duplicate diets	M, 15; F, 28	100	72	77	51	Duffield & Thomson (1999)
	1997–98	Simulated diets		84–93*§		65–67*¶		Vannoort *et al.* (2000)
		Estimated from urinary excretion (90 % intake)				60–100*‖		

F, female; M, male.
* Range.
† Median value.
‡ 10th, 90th centile.
§ Simulated diets; values for adult male and young male subjects.
¶ Simulated diets; values for adult female and vegetarian female subjects.
‖ Median values from several studies.

57 μg/d for adult female subjects, 43 μg/d for a 2-year-old child and 23 μg/d for an infant (Fardy *et al.* 1989). These estimates are similar to those obtained in a study of Brisbane residents: 89 and 59 μg/d for adult male and female subjects respectively, and 56 μg/d for children (Reilly, 1993). Se intakes were also assessed in the 1994 Australian Market Basket Survey, but are not included because values were expressed per kg body weight (Marro, 1996). In another study the mean Se intake of ninety-seven preschool children aged 4–6 years, measured by food-frequency questionnaire and 24 h recall, was 56 (SD 18) μg/d (Reilly *et al.* 1991). For comparison, daily dietary intakes of Se in the UK were reported to be 29–39 μg/d (Rayman, 2000), and the 1988–94 Third National Health and Nutrition Examination Survey of US subjects reported median intakes of 106 μg/d (Standing Committee on the Evaluation of Dietary Reference Intakes of the Food and Nutrition Board Institute of Medicine, the National Academies and Health Canada, 2000). Further country comparisons may be found in the comprehensive review compiled by Combs (2001), although values for England and New Zealand may now be out of date.

Selenium status in New Zealand and Australia

Assessment of selenium status

Blood Se concentration is generally considered a useful measure of both Se status and intake, but other tissues such as hair and toenails are also often assessed (Burk & Levander, 1999; Sheehan & Halls, 1999). Plasma or serum Se reflects short-term status (several days); erythrocyte Se reflects longer-term status (several weeks to months). There are no internationally accepted 'normal' reference ranges because of variations in Se status from country to country. Tissue concentrations of Se are likely to be unreliable indicators, however, as they do not accurately reflect the functional activity, which varies with the form of Se ingested (Nève, 1991, 1995; Thomson *et al.* 1993). Measurement of individual selenoproteins such as the Se-containing enzyme glutathione peroxidase (GPx), provides more accurate and useful information than does total Se alone (Patching & Gardiner, 1999). The close relationship between plasma GPx (GPx-3) or erythrocyte GPx (GPx-1) activity and Se concentration is useful for assessment in individuals with relatively low status, but not beyond the maximal activity of the enzyme, i.e. blood Se greater than about 1·27 μmol (100 μg)/l (Rea *et al.* 1979). It is often difficult, however, to compare results of GPx activities from different laboratories because of variations in methodology. Other selenoproteins such as selenoprotein P may be used, but simple assays are not yet available. Currently, plasma or serum Se is still the favoured measure of Se status for international comparisons.

Selenium status of New Zealanders

The relative paucity of Se in New Zealand soils has spurred the study of the significance of Se in human nutrition. In the 1950s white muscle disease and ill thrift in cattle and sheep in certain regions was found to be due to Se deficiency in soils. Subsequently, over the last 30 years, extensive investigations of the Se status of New Zealanders, the consequences of low Se status, Se metabolism, and the effects of supplementation of the diet with Se have been conducted in our laboratory. All investigations indicate that the Se intake and status of New Zealand residents is low (Griffiths & Thomson, 1974; Robinson, 1989; Thomson & Robinson, 1996; Duffield *et al.* 1999), and since supplementation results in an increase in GPx and selenoprotein P (Thomson *et al.* 1982, 1985, 1988, 1993; Duffield *et al.* 1999), intakes may be considered inadequate. More recently this low Se status has been confirmed in independently living, ambulatory, elderly Otago residents, particularly among those >80 years old (de Jong *et al.* 2001), with 80 % of subjects at risk of Se deficiency. Moreover, serum Se and Zn values were positively associated ($P<0·05$) with a physical functioning score, suggesting that suboptimal Se and Zn status may contribute to an inferior health status. Se intakes of most New Zealanders are likely to be below recommended intakes in many countries (Standing Committee on the Evaluation of Dietary Reference Intakes of the Food and Nutrition Board Institute of Medicine, the National Academies and Health Canada, 2000; Thomson & Paterson, 2001). In spite of this, no clinical signs of deficiency in New Zealand have been identified, apart from in one patient on total parenteral nutrition (van Rij *et al.* 1979). This may be due partly to the lack of specific clinical markers of Se deficiency. Other studies in New Zealand have confirmed the low Se status and have suggested a link with a number of conditions, such as CVD (Kay & Knight, 1979) and respiratory complications of premature newborn infants (Darlow *et al.* 1995).

Several reports suggest that blood Se concentrations of residents of the South Island of New Zealand have increased during the past 10 years due in part to greater importation of Australian wheat and other cereal products (Winterbourne *et al.* 1992; Thomson & Robinson, 1996). The impact of wheat importation into New Zealand, however, is inconsistent as little Australian wheat is used in the South Island. The increase in Se status is likely to be due also to other factors such as the supplementation of animal feeds leading to higher concentrations in meat and poultry (Vannoort *et al.* 2000), and also to changes in dietary patterns such as greater use of multigrain breads and imported legumes and nuts. Nevertheless, recent studies indicate that the Se status of the New Zealand population remains low compared with the population of many other countries, and may still be considered marginal (Table 2). This view is supported by a response of increased levels of the functional selenoproteins, GPx and plasma selenoprotein P to Se supplementation (Duffield *et al.* 1999; CD Thomson, E Paterson and AM Grant, unpublished results). The current plasma Se levels of residents of the Otago region of the South Island are in the range 0·76–1·65 μmol (60–130 μg)/l, but there is inadequate data from other areas of New Zealand to estimate a 'normal' reference range for the country. Mean blood Se concentrations in residents of two North Island provincial regions, Waikato and Taranaki, were 1·08 μmol

Table 2. Blood selenium status of adult residents of New Zealand and Australia
(Mean values and standard deviations)

Subject	Year	Gender	*n*	Plasma Se (μmol/l) Mean	Plasma Se (μmol/l) SD	Whole blood Se (μmol/l) Mean	Whole blood Se (μmol/l) SD	Authors
New Zealand*								
Otago blood donors	1994	M and F	206	0·90	0·14	1·14	0·18	Colls (1996)
Waikato & Taranaki blood donors	1994		199	1·08	0·18	1·37	0·18	Colls (1996)
Dunedin residents	1995	M and F	122	–		1·24	0·25	Duffield *et al.* (1999)
Dunedin residents†	1996	M and F	52	0·84	0·15	0·97	0·17	Duffield *et al.* (1999)
New Zealand	1997	F	449	1·03	0·24	–		McLachlan (2003)
South Island	1998–99	F	197	0·98	0·18	–		McLachlan (2003)
Dunedin								
Smokers		M and F	60	0·92	0·16	1·13	0·18	Paterson (2000)
Non-smokers		M and F	30	1·12	0·16	1·29	0·17	
Dunedin elderly	2000	F	103	0·90	0·25	–		de Jong *et al.* (2001)
Dunedin residents	2001–2002	M and F	188	1·11	0·18	–		CD Thomson & AM Grant, unpublished results
Australia								
Sydney workers	1989	M and F				1·36		Fardy *et al.* (1989)
Adelaide residents		M and F				1·54		Judson (1987–88)
Adelaide residents	1990	M and F	19	1·11	0·25			Daniels *et al.* (2000)
Tasmanian blood donors	1990	M and F	25	0·97	0·16			Daniels *et al.* (2000)

M, male; F, female.
* Values reported from 1994. Earlier values reported from 1972 are reported by Thomson & Robinson (1996).
† Screened for low Se status.

(85 μg)/l plasma and 1·38 μmol (109 μg)/l whole blood in 1994 compared with those from Otago residents in the same year of 0·90 μmol (71 μg)/l and 1·14 μmol (90 μg)/l respectively (Colls, 1996). Thus, the national mean value is likely to be higher than that for Otago. Table 2 summarizes plasma and whole blood Se concentrations in New Zealand subjects obtained as baseline measures in various studies from 1992. Earlier values for blood Se concentrations in Otago residents from 1972 (plasma Se ranging from 0·56 (SD 0·14) to 0·89 (SD 0·13) μmol/l) and residents of other New Zealand cities have been summarized by Thomson & Robinson (1996).

Selenium status of Australians

There is little information on the Se status of Australian populations. Dietary surveys in Australia have not included Se status. Mean Se concentrations reported in whole blood of Sydney workers in 1989 were 1·37 and 1·35 μmol (105 and 108 μg)/l for male and female subjects respectively (Fardy *et al.* 1989), while in Adelaide a mean a value of 1·54 μmol (122 μg)/l for blood Se was reported by Judson (1987–88). Plasma Se in nineteen adults from Adelaide and twenty-five adults from Tasmania in 1990 was 1·11 (SD 0·25) and 0·97 (SD 0·16) μmol (88 (SD 20) and 77 (SD 13) μg)/l respectively (Daniels *et al.* 2000), and in children aged ≤1–14 years, 0·82 (S 0·15) μmol/l (Reilly *et al.* 1990). These few reports indicate that blood Se levels, with a mean estimate for plasma of 1·12 μmol (94 μg)/l (Lyons *et al.* 2003), were generally greater than those of New Zealanders.

Iodine intakes in New Zealand and Australia

Food sources of iodine

The major sources of I are seafood, iodized salt (NaCl), milk and eggs, while meat and cereals are secondary sources (Kidd *et al.* 1974; Vannoort *et al.* 2000). Foods of marine origin (sea fish and shellfish, sea-meal (custard made of ground seaweed) and seaweeds) are rich in I, reflecting the much higher I concentration in seawater compared with most freshwaters (Kidd *et al.* 1974). The I content of plants and animals depends on the environment in which they grow. Vegetables, fruits and cereals grown in soils with low I content are poor sources of I. Different cooking procedures produce varying levels of I loss, depending on cooking time, temperature and the nature of the food being cooked (Wang *et al.* 1999). The greatest losses occur as a result of leaching into water during boiling (Goindi *et al.* 1995).

In addition, there are adventitious sources of I including iodates in bread, iodophors (used as cleaning agents in the dairy industry) in dairy products and I-containing food colours (Gibson, 1990). Iodophors have been used in the dairy industry in New Zealand and also in Australia, since 1962, for sanitizing milking machines and other equipment (Twomey, 1968; Joerin & Bowering, 1972; Eastman, 1999). Kelp tablets and drugs, beverages (raspberry drinks) and products such as maraschino cherries often contain the food colouring erythrosine, a component of which is I. The bioavailability of I from erythrosine is, however, probably low (Wenlock, *et al.* 1982; Sumar & Ismail, 1997).

Iodized salt probably remains an important source of I in the diets of New Zealanders. There are no data, however,

on its contribution to total I intake, due to the fact that estimates of discretionary salt intakes in the New Zealand population are not available. Estimates from other countries are derived from calculations for household salt purchases; these calculations do not take into account salt used for other purposes, such as in preservation and cleaning, and salt lost in cooking water (Bull & Buss, 1980). In the UK, a Li-marker technique was used to determine the proportion of salt derived from discretionary sources (Sanchez-Castillo *et al.* 1987), which was found to be only 15 % total salt intake, while in the less developed countries Guatemala and Benin, discretionary use of salt contributed 77 and 52 % respectively to the total salt intake of mothers (Melse-Boonstra *et al.* 1998). The impact of the public health recommendation to decrease salt intake on the I status of the New Zealand population is currently unknown. A recent survey in Otago indicated that 93 % of subjects purchased iodized salt; 48 %, however, never added salt at the table and 30 % never used salt in cooking (Thomson *et al.* 1997*a*). Similarly, although 83 % of caregivers of children studied in Dunedin and Wellington reported that iodized salt was used in the home, almost 30 % did not use iodized salt in cooking and 51 % of the children did not use iodized salt at the table (Skeaff *et al.* 2002). It appears likely that recommendations to reduce salt intake, if adopted by an increasing number of New Zealanders, could exacerbate the risk of suboptimal I status, especially since non-iodized salt is used commercially in food manufacturing. In Australia, as little as 10 % of the population may be using iodized salt in the home because of the availability of non-iodized salt in supermarkets (Li *et al.* 2001).

Iodine intakes in New Zealand

Most soils in New Zealand are relatively low in I, resulting in low concentrations in locally produced foods. Goitre was endemic in many parts of New Zealand in the late 1800s and early 1900s. Table salt was iodized at a low level in 1924, but was increased to the current level of 40–80 mg I/kg salt in 1939, and goitre had virtually disappeared by the 1950s. Currently, however, non-iodized table salt is widely available in New Zealand, and almost all salt used in manufactured foods is non-iodized.

As with other trace elements, the nutritional status of I in the New Zealand population may be influenced by changes in food supply, location of production and the amounts and origins of imported foods. In recent years I in dairy products has made a major contribution to the daily intake as a result of iodophor contamination (Joerin & Bowering, 1972; Sutcliffe, 1990), which has raised the I intake of full-fat milk and dairy-product users. In 1972, the level of I in milk from farms in New Zealand using iodophors ranged from 10–750 μg/l, compared with 30–110 μg/l from farms that did not use iodophors (Joerin & Bowering, 1972). Sometimes intakes were raised to undesirable levels close to 1000 μg/d, compared with the recommended intake of 150 μg/d (Robinson, 1992). Some intakes, where kelp tablets or I containing medications were taken regularly, approached the potentially harmful level of 2000 μg/d.

During the 1980s iodophors started to be replaced in the New Zealand and Australian dairy industries by other cleaning compounds, such as quaternary ammonium compounds, resulting in decreased I levels in milk and dairy products (Sutcliffe, 1990; Knowles *et al.* 1997). Overall there has been a reduction in I intakes in New Zealand since monitoring began in 1982. Intakes estimated in the 1997–98 New Zealand Total Diet Survey from simulated diets were 84–93 μg/d for male and 65–67 μg/d for female subjects (Vannoort *et al.* 2000). These intakes did not take into account the addition of discretionary salt used during cooking or added at the table. The contribution of dairy products to total I intake remains relatively high: 42 % of the intake of a young male and 68 % of that of a 1–3-year-old child (Cressey & Vannoort, 1998; Vannoort *et al.* 2000). The reduction in I intakes also reflects other dietary changes, such as the increased use of ready-to-eat and bought prepared foods (which contain non-iodized salt) and a probable decrease in the use of salt in cooking and at the table. I intake was not assessed in the 1997 National Nutrition Survey because of inadequate food composition data. Analysis of 3 d duplicate diets collected in a recent study of fifty Otago residents indicated intakes ranging from 12 to 812 (median 108) μg/d (CD Thomson, unpublished results). These diets included discretionary salt used in cooking, but may not have included all salt used at the table.

Since most I is excreted in urine, daily I intake may be estimated from 24 h urinary iodide excretion on the assumption that 80–90 % of I ingested is excreted. Thus, I intakes of 60–95 μg/d were estimated for North and South Island blood donors (Thomson *et al.* 1997*a*), 60–70 μg/d for pregnant and lactating New Zealand women (Thomson *et al.* 2001) and 100 μg/d for a group of Otago residents in 1997. Such intakes are considerably less than the 200 μg/d recommended by the Nutrition Taskforce (Department of Health, 1991) and the 150 μg/d recommended in most countries (Food & Nutrition Board Institute of Medicine, 2001; Thomson, 2002). The low intakes of pregnant women are of particular concern in view of the role of I in fetal brain development and the possible impact of inadequate I on psychomotor performance.

Iodine intakes in Australia

Little information is available on I intakes in Australia. Intakes cannot be estimated from surveys of daily urinary excretion as random urine samples only have been collected so far (Eastman, 1999; Gunton *et al.* 1999; Hynes, 2001; Li *et al.* 2001). Several recent surveys of schoolchildren, healthy adults, pregnant women and diabetic patients report that urinary I excretion of Australians is decreasing (Gunton *et al.* 1999; Hynes, 2001; Li *et al.* 2001). Thus, I intakes are probably also decreasing. As in New Zealand, the principal source of I in Australia has been from dairy products as a result of contamination by iodophors (Connolly, 1971), and in Tasmania, from bread as a result of the addition of potassium iodate to improve the flour (Eastman, 1993). A decrease in I intakes in Australia, as in New Zealand, is probably due to

reduction in the use of iodophors in the dairy industry and decreasing iodized salt consumption (Eastman, 1999; Gunton *et al.* 1999). Li *et al.* (2001) reported that as little as 10 % of the Australian population currently purchases iodized salt for domestic use. The Tasmanian State Government adopted the addition of iodate to bread as an I supplement in 1966, but this is no longer mandatory due to an increase in I-induced thyrotoxicosis. In October 2001, the Department of Health and Human Services of Tasmania introduced an intervention to increase intakes of I by 50 μg/d (Department of Health & Human Services, 2001). To achieve this the Department has negotiated a memorandum of understanding with the bread manufacturers for them to use iodized salt (40 mg I/kg NaCl) for bread manufacture.

Iodine status in New Zealand and Australia

Assessment of iodine status

Daily urinary excretion of iodide closely reflects I intake, as only a small fraction is excreted in faeces. Thus, urinary iodide excretion in a 24 h urine specimen is used as an index of I nutriture (Hetzel & Dunn, 1989; Gibson, 1990; Dunn *et al.* 1993). Twenty-four h urine collections are preferable, but are not practical for large field surveys where I deficiency is being assessed. Non-fasting casual urine specimens are usually obtained in such cases. Casual urine samples, however, are not suitable for estimating iodide excretion of individuals, even when expressed as the iodide:creatinine ratio (Nicolau *et al.* 1989; Thomson *et al.* 1996, 1997*a*). First-voided fasting morning urine samples or double-voided fasting samples are sometimes collected, as these are less affected by immediately preceding intakes of I. These measures correlate better with 24 h iodide excretion than do non-fasting urine samples (Thomson *et al.* 1996, 1997*b*). In populations with adequate general nutrition, urinary iodide concentration correlates well with the urinary iodide:creatinine ratio, so urinary iodide excretion relative to creatinine may be determined on the assumption that creatinine excretion is constant over time. There is some disagreement, however, about the suitability of the iodide:creatinine ratio for assessing I status (Furnée *et al.* 1994; Remer & Manz, 1994; Thomson *et al.* 1996, 1997*a*; Rasmussen *et al.* 1999). Creatinine excretion increases with age until adulthood due to increases in muscle mass and creatinine production (Remer & Manz, 1994), and is also influenced by malnutrition, strenuous exercise, fever and trauma (Gibson, 1990). In surveys for assessing I deficiency within a population, iodide concentration in casual urine samples appears to be adequate (Frey *et al.* 1978).

Iodine status of New Zealanders

Surveys during the 1960s to 1980s indicated more than adequate I intakes as measured by urinary iodide excretion (North & Fraser, 1965; Cooper *et al.* 1984; Simpson *et al.* 1984). Median urinary iodide excretions in these surveys ranged from 202 μg/d in Wellington in 1965 (North & Fraser, 1965), to 266 and 216 μg/d in Otago male and female subjects (with those on a salt-restricted diet 228 and 190 μg/d respectively; Simpson *et al.* 1984) and 305 μg/d in subjects in Auckland in 1984 (Cooper *et al.* 1984).

More recently a series of studies (see later) carried out in the Department of Human Nutrition, University of Otago, in the 1990s suggested reduced I intakes reflected in lower I status as measured by urinary iodide excretion (Table 3). WHO/UNICEF/Centre for the Control of Iodine Deficiency Disorders criteria for assessing I deficiency disorders are (urinary iodide concentration, μg/l): mild I deficiency 50–99; moderate I deficiency 20–49; severe I deficiency <20) (World Health Organization/United Nations International Children's Emergency Fund/International Council for the Control of Iodine Deficiency Disorders, 1994).

In a combined population of blood donors residing in Otago and Waikato, the median iodide excretions for non-supplementing male and female subjects were 70 and 59 μg/d respectively, with urinary iodide concentrations of 45 and 42 μg/l. Fifty percent of the participants had a risk of mild I deficiency disorder, 35 % had moderate risk and 7 % had a risk of severe I deficiency disorder (Thomson *et al.* 1997*a*,*b*) according to the World Health Organization/United Nations International Children's Emergency Fund/ Centre for the Control of Iodine Deficiency Disorders (1994) criteria. Thyroid hormone concentrations (thyroid stimulating hormone, triiodothyronine, thyroxine) in the blood of all donors, however, were within normal ranges.

Similarly, in a study of pregnant and non-pregnant women, 55 % of the daily urinary excretions were <50 μg/d, indicating moderate I deficiency (Thomson *et al.* 2001). Twenty-four h urinary iodide excretion measured on twelve separate occasions over 18 months, varied considerably from month to month for each subject, but there were no obvious trends throughout the period. Mean values for daily urinary iodide and for iodide concentrations were reasonably consistent from month to month, ranging from 52 to 72 μg iodide/d and from 37 to 52 μg iodide/l respectively, with no significant differences between pregnant and non-pregnant women. Median iodide concentrations were 38 and 33 μg/l in pregnant and non-pregnant women respectively.

In a recent survey of Otago residents, low iodide excretions were confirmed (median urinary iodide concentration, 59 μg/l) and significant correlations were found between measures of urinary iodide excretion and thyroid volume and serum thyroglobulin levels (but not thyroid stimulating hormone or thyroxine). Multiple regression analysis of data for subjects divided into three groups, according to 24 h urinary iodide excretion, showed significant differences in thyroid volume and serum thyroglobulin among the groups, with those with the lowest excretions (median urinary iodide concentration, 41 μg/l) showing elevated thyroglobulin and greater thyroid volume. These results indicate that the fall in I status is being reflected in clinical measures of thyroid status, including enlarged thyroid glands and elevated thyroglobulin, suggesting a possible re-emergence of mild I deficiency in New Zealand.

Table 3. Urinary iodide excretion in New Zealand and Australia adults and children

Subjects	Year	Gender	*n*	Urinary iodide (μg/l) Median	Urinary iodide (μg/l) Range	Author(s)
New Zealand*						
Pregnant women	1991–92	F	27	38	1–170	Thomson *et al.* (2001*a*)
Non-pregnant women			14	33	1–187	
Otago blood donors	1992–93	M and F	63	43	5–111	Thomson *et al.* (1996)
Otago blood donors	1993–94	M and F	183†	42	6–126	Thomson *et al.* (1997*a*)
Waikato & Taranaki blood donors	1993–94	M and F	128†	48	17–152	Thomson *et al.* (1997*a*)
Dunedin residents	1995	M and F	52	49	10–239	Duffield *et al.* (1999)
Otago blood donors	1997–98	M and F	233	54	1–200	Thomson *et al.* (2001*b*)
Dunedin and Wellington children‡	1996–97	M and F	282	66	45–91§	Skeaff *et al.* (2002)
South Island children	1998–99	M and F	230	67	37–115§	Skeaff *et al.* (2004)
New Zealand children	2002	M and F	1796	66	1–260	Parnell *et al.* (2003)
Australia‡						
Sydney residents	1992	M and F		180		Eastman (1993)
Sydney adults	1998–99	M and F	63	88	12–200	Li *et al.* (2001)
			19	64	54–75	Gunton *et al.* (1999)
Pregnant women	1998–99	F	101	88	20–448	Li *et al.* (2001)
			81	104	89–129	Gunton *et al.* (1999)
Postpartum women	1998–99	F	26	79	44–229	Gunton *et al.* (1999)
Sydney children	1998–99	M and F	94	84	28–312	Li *et al.* (2001)
Pregnant women	2000	F	84	109	65–168	McElduff *et al.* (2002)
Tasmanian children	1996	M and F	93	42	25–71	Hynes (2001)
	1998–99	M and F	241	75	60–96	Hynes (2001)
	2002	M and F	125	84	57–110	Guttikonda *et al.* (2002)

F, female; M, male.
* Values for New Zealand adults are concentrations in 24 h urine samples.
† Non-supplementers.
‡ All values for Australian and for New Zealand children are concentrations in casual urine samples.
§ Inter-quartile range.

In a sample of New Zealand children from Dunedin and Wellington, median urinary iodide excretion (66 μg/l) was indicative of mild I deficiency (Skeaff *et al.* 1999, 2002). Thirty-one percent of the children had urinary iodide levels <50 μg/l. Median urinary iodide excretion was 66 μg/l. Comparison of thyroid volume with the 2001 WHO age- and gender-specific, and age- and body surface area (BSA)-specific cut-off values (Zimmerman *et al.* 2001) showed that 11·3 and 12·0 % of the children respectively had thyroid gland enlargement greater than the upper limit of normal (Skeaff *et al.* 2002). In the pilot of the Children's Nutrition Survey of children aged 5–14 years carried out in New Zealand in 2000, urinary iodide excretion in casual urine samples (67 μg/l) was also indicative of mild I deficiency (Scragg & Metcalf, 2001). This low I status (66 μg/l) was confirmed in a representative national sample in the 2002 Children's Nutrition Survey (Parnell *et al.* 2003). Mild I deficiency was also found in a group of New Zealand infants and toddlers, 37 % of whom had urinary I concentrations <50 μg/l (Skeaff *et al.* 2004). When children were classified according to feeding practice, those who were currently formula-fed had significantly higher median urinary I concentration (99 μg/l) than those who were currently breast-fed (44 μg/l; $P=0000$), indicating that infant formulas are better sources of I in New Zealand than breast milk.

Iodine status of Australians

Tasmania is the only state in Australia where there are records of I nutrition as a result of regular surveillance (Eastman, 1999). Other data are available from the Australian Centre for the Control of Iodine Deficiency Disorders from sporadic surveys in the past 20 years of urinary iodide excretion levels in small samples of Australians (Eastman, 1999). In the early 1990s it was reported that there was no evidence of I deficiency in any part of Australia (Eastman, 1993). In 1992 the mean urinary iodide excretion was 180 μg/l in Sydney residents, and >200 μg/l in Tasmanian children (Eastman, 1993).

More recent studies in Australia, however, have shown a decreasing trend in I intakes, as is the case in New Zealand (Eastman, 1999; Gunton *et al.* 1999; Hynes, 2001; Li *et al.* 2001). Sporadic surveys have indicated a gradual sustained decline in urinary iodide excretion in Sydney residents (Eastman, 1999) (Table 3). In 1988–99 primary-school children from Western Sydney had a median urinary iodide concentration of 84 μg/l, indicative of mild I deficiency. Similarly, in 1998–99 and 2000–2001 the median urinary iodide level in school-aged children in Tasmania was 75 μg/l. The I status of four groups at a Sydney hospital was determined in 1998–99 (Gunton *et al.* 1999; Public and Environmental Health Service, unpublished results). Median urinary iodide concentrations were 104 μg/l in pregnant women, 79 μg/l in postpartum women, 65 μg/l in patients with diabetes mellitus and 64 μg/l in volunteers. Moderate I deficiency was found in 19–34 % of subjects across the four groups, and mild I deficiency in an additional 30–47 %. A more recent survey confirms the marginal I status of pregnant women (median urinary iodide excretion, 109 μg/l; McElduff *et al.* 2002). As a result of these observations the Tasmanian

Department of Health and Human Services mounted a survey of the urinary I status and thyroid volume of Tasmanian primary-school children (Hynes, 2001; Public and Environmental Health Service, unpublished results). Median urinary iodide excretion of 225 children aged 4–17 years was 84 μg/l (Guttikonda *et al.* 2002) and the prevalence of elevated thyroid volume was 24·6 % of boys and 20·7 % for girls when compared with 2001 WHO age- and gender-specific and age- and BSA-specific cut-off values (Zimmerman *et al.* 2001).

Aetiology of iodine deficiency disorders in New Zealand and Australia

Before the introduction of iodized salt in New Zealand in 1924, goitre was endemic in many parts of the country due largely to the relatively low levels of I in New Zealand soils. In 1920, the incidence of enlarged thyroid gland was approximately 32 % in schoolchildren, with a further 29 % having thyroid glands exhibiting pathological enlargement. In 1938, the incidence of goitre in schoolchildren was 15·1 %, but by 1953 had fallen to 1·1 % (Hercus *et al.* 1925; Purves, 1974). Surveys of I status in 1965, 1982 and 1984 found that most New Zealanders had a more than adequate I status (North & Fraser, 1965; Cooper *et al.* 1984; Simpson *et al.* 1984). Recent research, however, provides evidence of a re-emergence of mild I deficiency due to a fall in dietary I intakes (Thomson *et al.* 1997*a*; Skeaff *et al.* 2002).

Endemic goitre was recorded in Tasmania during the 19th century, but there are few references in the medical literature before the early 20th century (Clements & Wishart, 1956). In 1949, goitre prevalence in schoolgirls aged 5–17 years was 51 %, with palpable goitre being 39 % and visible goitre 12 % (Eastman, 1993). I deficiency and goitre were also recognized in the Australian Capital Territory and surrounding districts before 1950. It was believed that the ingestion of goitrogens was a significant contributory factor in the development of endemic goitre in this area (Clements & Wishart, 1956). Iodization of bread introduced in Tasmania in 1966 and the Australian Capital Territory in 1963, reduced the incidence of goitre but also increased the incidence of I-induced thyrotoxicosis (Tasmanian Thyroid Advisory Committee, 1981). As in New Zealand, however, there may also be a current re-emergence of I deficiency in parts of Australia (Eastman, 1999; Gunton *et al.* 1999; Hynes, 2001; Li *et al.* 2001; Guttikonda *et al.* 2002) due to decreasing I intakes.

Reduced I intakes in recent years are reflected in lower urinary iodide excretion in both New Zealand and Australia (Thomson *et al.* 1997*a*, 2001*a*; Eastman, 1999; Gunton *et al.* 1999; Li *et al.* 2001; Scragg & Metcalf, 2001; Guttikonda *et al.* 2002; Skeaff *et al.* 2002; Public and Environmental Health Service, unpublished results). Studies in New Zealand and Tasmania indicate that the fall in I status is reflected in clinical measures of thyroid status and enlarged thyroid glands (Guttikonda *et al.* 2002; Skeaff *et al.* 2002). Groups who are likely to be at particular risk are those restricting salt intake, and those whose consumption of dairy products or fish is negligible, especially those following vegan diets. Other 'at risk' groups are pregnant and lactating women, and infants, because of the association of suboptimal intelligence development with inadequate I intake (Pharoah *et al.* 1984). At present there is little evidence of any associated clinical disease. Nonetheless, it is important to continue to monitor the urinary iodide excretion of New Zealanders and Australians. It would appear to be desirable to introduce measures that increase I intakes in both countries.

The selenium–iodine interrelationship

The discovery of the role of Se in thyroid hormone production as a component of hepatic type I 5′-iodothyronine deiodinase, and subsequently type II and III deiodinases, presented an exciting new challenge for Se research, particularly in New Zealand where soils are relatively low in both Se and I. These enzymes catalyse the conversion of thyroxine to its active metabolite triiodothyronine. Se deficiency results in an increase in plasma thyroxine and a corresponding decrease in triidodthyronine (Arthur, 1999). Se status may relate directly to health and well-being through impairment of thyroid hormone metabolism (Arthur *et al.* 1990). Goitre and hypothyroidism in rats, brought about by I deficiency, were exacerbated by concurrent Se deficiency (Arthur *et al.* 1990; Beckett *et al.* 1993), probably as a consequence of adverse effects of both deficiencies on thyroid hormone metabolism and on thyroid gland I content. There is increasing evidence that combined Se and I deficiencies also have significant physiological and metabolic consequences in human subjects (Corvilain *et al.* 1993; Vanderpas *et al.* 1993). An interaction between Se deficiency and I deficiency has been implicated in the pathogenesis of cretinism in Africa (Goyens *et al.* 1987). More recently, Derumeaux *et al.* (2003) have shown an inverse association between Se status and thyroid volume in elderly women, but not in elderly men, participating in the SU.VI.MAX study. This suggests that Se may protect against goitre. Se also appears to be implicated in thyroid echostructure and thus may protect against autoimmune disease (Derumeaux *et al.* 2003). Kvícala *et al.* (1995) have shown associations among measures of Se status (serum Se and urinary Se excretion) and those of thyroid status (thyroid stimulating hormone, thyroxine, triiodothyronine, thyroid volume) in a population with low Se status. Such associations might be used in the biological assessment of the magnitude of Se deficiency.

The coincidence of low-Se and low-I areas of the South Island of New Zealand is intriguing. Has the incidence of goitre in New Zealand been aggravated by low Se status? This would be difficult to investigate directly now that salt iodization has been introduced, but the nature of the relationship between Se and I is of interest, especially if the downward trend in I status continues.

Preliminary investigations in New Zealand (Thomson *et al.* 1996), although not designed specifically to investigate the relationship between Se and I, showed correlation between total daily excretions of Se and I. There was also a correlation between the urinary concentrations of the two elements in fasting and in 24 h urine specimens. Similar

correlations were also found in pregnant and non-pregnant women (Thomson *et al.* 2001). This may merely reflect the fact that excretion of Se, and probably also that of I, is related to body size. It may, however, also reflect a relationship between the relatively low dietary intakes of the two elements in the South Island. In another New Zealand study, Se supplementation resulted in a small decline in plasma thryroxine in subjects with low Se status (Duffield *et al.* 1999), as was also observed in elderly subjects in Italy (Olivieri *et al.* 1995). Further studies are underway to investigate the possibility of an association among measures of Se status and of thyroid status in New Zealand.

Conclusions

The Se status of the New Zealand population remains low compared with the population of many other countries, and may still be considered marginal, although the clinical consequences of the marginal Se status are unclear. There are no recent reports of blood Se levels in Australia, but earlier reports indicate that they were generally higher than those of New Zealanders. Current recommended intakes are based on the requirement for maximal activities of the selenoprotein GPx (Standing Committee on the Evaluation of Dietary Reference Intakes of the Food and Nutrition Board, 2000; Thomson & Paterson, 2001). The results of large cancer intervention studies currently in progress should determine whether intakes, higher than those now recommended in USA and elsewhere, are beneficial to health. Individuals may increase their Se intake by choosing high Se foods such as seafood, Brazil nuts and other good sources such as poultry and eggs (Vannoort *et al.* 2000). Commercially available supplements usually contain organic Se as selenomethionine or high-Se yeast, but caution is advisable in their use to prevent unsafe high intakes.

Similarly, the consequences of decreasing I status in Australia and New Zealand are unclear. Mild I deficiency in New Zealand has resulted in enlarged thyroid glands, indicating an increased risk of goitre. Currently there is little evidence, of any associated clinical disease. Public health recommendations to reduce salt intake, however, together with the reduction in I content of dairy products, are likely to result in further decreases in the I status of New Zealand and Australian residents. Eventually some action may be necessary to prevent this decline. The consumption of rich sources of I such as eggs, fish and shellfish can be encouraged. Foods containing small quantities of seaweed, such as some types of sushi, are also effective dietary sources of I. The excessive consumption of seaweed and of I-containing dietary supplements, such as kelp tablets, is not recommended as it may lead to intakes beyond the safe upper limit. The use of iodized salt in cooking or at the table is beneficial. Advice to reduce salt intake, and the increased consumption of processed foods (in which salt is non-iodized) means that iodized salt may no longer be as effective a vehicle for I fortification as it was previously. It may be prudent now, or in the near future, for public health authorities in Australia and New Zealand to consider other foods, such as bread, as a suitable vehicle for fortification.

The consequences of possible interactions between Se and I in human nutrition are unclear, and no practical recommendations can be made. Caution is required in supplementing intakes as work from areas with severe deficiencies in Se and I indicates that Se supplementation may aggravate, rather than alleviate problems, both for children with borderline hypothyroidism and for the fetuses of pregnant mothers (Contempré *et al.* 1991*b*). These authors have warned that any Se supplementation should not be undertaken without simultaneous I supplementation (Contempré *et al.* 1991*a*,*b*).

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