Iodine and selenium status and intake, and iodine nutritional knowledge of women of childbearing-age in Palmerston North, New Zealand.

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ABSTRACT

Objective: To explore the iodine and selenium status and intakes, and iodine nutritional knowledge of women of childbearing-age in Palmerston North, New Zealand (NZ), after the implementation of mandatory fortification of bread with iodine.

Method: Fifty women of childbearing-age (non-pregnant and not breastfeeding) were interviewed (recruited) for the assessment of dietary intake and nutritional knowledge using a researcher-led questionnaire, including a semi-quantitative food frequency questionnaire and 24-hour dietary recall. Fifty 24-hour urine samples were obtained and total volumes were measured. Iodine and selenium were analysed using the inductive-coupled plasma mass spectrometry.

Result: The median urinary iodine concentration was 64.7 mcg/l, which represents a mild iodine deficiency (ID), according to the WHO. Based on the individual iodine status, 70% of the participants were iodine deficient, categorised as marginal (30%), mild (30%) and moderate (10%). Iodine intake estimated from urinary iodine excretion (UIE) showed that 34% did not achieve the Estimated Average Requirement (EAR)(<100 mcg/day) and 46% met the Recommended Dietary Intake (RDI)(150 mcg/day). The median iodine intake was 129.8 mcg/day, indicating suboptimal intake. The major contributors to iodine intake were milk (35.6%), bread (24.6%), fish and seafood (15%) and egg (13.8%). The majority of respondents were unaware of the mandatory fortification of bread with iodine (70%) and also unaware of the ID problem in NZ (52%). The median excretion of selenium was 31.6 mcg/day and the intake estimated from urinary excretion was 57.5 mcg/day, with both values above the safe range for women (30 mcg/day), according to the WHO. Based on the 24-hour recall, the majority (70%) had inadequate selenium intake (<50 mcg/day), whilst only 20% met the RDI intake of 60 mcg/day. There was a moderate correlation between the urinary selenium excretion and UIE (Spearman's rank order; $\underline{r}$ (50)=0.547,$p<0.05$).

Conclusion: ID is still a problem in this population, although mandatory fortification has been implemented. However, this study shows improved iodine status and intake compared to previous studies and it thus signifies the benefits of iodine-fortified bread. In order to help eliminate ID in NZ, an additional strategy, such as the implementation of iodine fortification in another food vehicle should be considered.

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“So, verily, with every difficulty, there is relief. Verily, with every difficulty there is relief” (Holy Quran 94:5-6)

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LIST OF ABBREVIATIONS

EAR	Estimate average requirement
FFQ	Food frequency questionnaire
FNB	Food Nutrition Board
GPx	Glutathione peroxidase
ICCDD	International Council for Control of Iodine Deficiency Disorder
ID	Iodine deficiency
IDD	Iodine deficiency disorder
IDIs	Iodothyronine 5' deiodinases
IOM	Institute of Medicine
MOH	Ministry of Health
NHANES	National Health and Nutrition Examination Survey
NIS	Na/I active transporter
NRV	Nutrient references value
NTD	Neural tube defects
NZ	New Zealand
NZTDS	New Zealand Total Diet Survey
PII	Plasma inorganic iodide
RDA	Recommended dietary allowance
RDI	Recommended dietary intake
TBG	Thyroid-binding globulin
TGR	Total goitre rate
Thg-DIT	Thyroglobulin-3,5-diiodotyrosine
Thg-MIT	Thyroglobulin-3-monoiodotyrosine
TR	Thyroid hormone receptor
TRH	Thyrotropin-releasing hormone
TSH	Thyroid-stimulating hormone
UIC	Urinary iodine concentration
UIE	Urinary iodine excretion
UNICEF	United Nations International Children Emergency Fund
USI	Universal salt iodisation
WHO	World Health Organization

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ETHICS

Ethic approval for this study (Application 10/03) was obtained in March 2010 from the Massey University Ethics Committee (MUHEC) (Appendix 13).

1 INTRODUCTION

1.1 Background

Iodine is an essential element for thyroid hormone synthesis, which is crucial for metabolism and for maintaining growth and development. Iodine deficiency (ID) during pregnancy and lactation is especially serious, since it can cause adverse consequences of iodine deficiency disorder (IDD), for both mothers and their infants. ID is known as the greatest preventable cause of brain damage and mental retardation in the world. Chronic and severe deficiency of ID can also have a detrimental effect across all ages. From 2003 until recent years, approximately two billion people across the global had insufficient iodine intake, which makes ID one of the major worldwide public health problems (Benoist, McLean, Andersson, & Rogers, 2008), and this includes New Zealand (NZ).

ID was prevalent in NZ in the late 1800s and the early 1900s, characterised by endemic goitre and very low urinary iodine concentration. The main cause of ID in NZ is due to low iodine levels in the soil, which provides very limited iodine in food sources in NZ diet. Therefore the NZ government introduced iodisation of salt in the 1920s and this significantly reduced ID and eliminated goitre in NZ (Aitken, 2001; Mann & Aitken, 2003). The previous usage of iodophors in the dairy industry has increased the iodine content in dairy products. Ultimately, ID was eradicated from NZ in the 1970s. However, due to changing eating patterns and other factors, people in NZ have been found to have insufficient iodine intake, since the 1990s (Thomson and Skeaff, 2009). ID has been found to have re-emerged in NZ and many recent studies have reported mild to moderate ID amongst NZ children and adults, including pregnant and lactating women and their infants.

In order to address the re-emergence of ID in NZ, the Ministry of Health (MOH) implemented the mandatory replacement of non-iodised salt with iodised salt in almost all breads in NZ, which commenced in September 2009. This mandatory addition of iodine into bread is targeted at increasing the overall iodine intake of the majority of the NZ population, in order that they are able to achieve the NZ Recommended Dietary Intake (RDI) of iodine. However, this will be insufficient to meet the needs of pregnant and lactating women, since their requirements are much higher and therefore iodine supplement has been introduced for all pregnant and lactating women.

1.2 Significance of the study

Currently, there have been no studies evaluating the effectiveness of iodine-fortified bread on the improvement of iodine status or intake amongst the NZ population. Thus, the present study determined both the iodine status and dietary intake of the participants, following the mandatory fortification of bread with iodine in NZ. The results of the present study were then compared with previous studies in NZ. This study also evaluated the dietary intake of bread and other good food sources of iodine and thus, the contribution of iodine-fortified bread to overall dietary iodine intake can be estimated. It is still questionable whether women of childbearing-age in NZ would be able to achieve the RDI of 150 mcg daily for iodine, even after the mandatory fortification of almost all bread with iodine.

The provision of a supplement to prevent ID in pregnant and breastfeeding women is questionable. For example, a study on pregnant women (n=161) in Christchurch NZ, found that many women did not take the recommended periconceptual folic acid supplements in early pregnancy, which was mainly due to their pregnancy being unplanned (Schader & Corwin, 1999). Thus, ensuring adequate iodine intake amongst women of childbearing-age is important, in order to ensure optimal iodine intake during early pregnancy. Furthermore, iodine deficiency during the early stage of pregnancy may cause disruption of fetal brain growth and body development. These detrimental effects can extend to childhood (or later life), if the deficiency is severe. Therefore, it is important to monitor the iodine status and intake of all women of childbearing-age, including those who are not planning for conception.

The majority of women predominantly take care of their household's food and diet (Behrman & Wolfe, 1987; Lindelow, 2008; Moen, Robison, & Fields, 1994). Therefore, women in the study were investigated regarding the usage of salt at home, particularly use of iodised salt during cooking, or as table salt. The use of processed food in cooking and/or the frequency of eating outside the home (at restaurants or fast food outlets) were also investigated. This investigation is essential when estimating the participants' exposure to food containing very low levels of iodine. Furthermore, the study also determined the usual intake of main food sources of iodine including bread, since this could determine if bread intake provides a significant contribution to the participants' dietary iodine intake. Therefore, this study investigated whether iodine-fortified bread has improved the participants' overall iodine intake.

Since mandatory iodine fortification has only recently been implemented (at the end of 2009), it is of interest to identify whether people are aware of this implementation. Furthermore, the participants' perception towards bread fortification is also important because (as previously discussed) many women determine their household's diet. A positive feedback from women in the study towards iodine-fortified bread intake may help to increase their household intake (including themselves) in the future. A survey undertaken amongst women in Dunedin NZ, has found that the majority of respondents agree with the implementation of iodine-fortified bread and they indicated that they would buy foods fortified with vitamins or minerals, if there was not a great deal of difference in the price, and if they were well-informed about the benefits and risks of fortification (Thurlow, 2006). Therefore, women's knowledge on iodine-fortified bread and other iodine food sources is very important, in addition to their awareness of ID (Charlton, Yeatman, & Houweling, 2010). Studies have found that maternal health and nutritional knowledge could influence a family's health, by providing a more sanitary and safer environment at home, in addition to women being more likely to plan and prepare healthy meals for their family (Behrman & Wolfe, 1987; Chen & Li, 2009; Van Esterik, 1997). Therefore, it is important to determine women's nutritional knowledge, particularly that concerning iodine nutrition knowledge, and also their awareness of ID, as a major public health problem in NZ.

Selenium also plays important roles in maintaining the optimal function of the thyroid gland, particularly in the deiodination of thyroxine (Moreno-Reyes, Victor, Gerard, & Ronald, 2009). Deficiency in selenium may also aggravate the function of the thyroid gland, which may worsen the effect of iodine deficiency disorders (Kvicala, Zamrazil, Soutorova, & Tomiska, 1995; Triggiani et al., 2009). Selenium levels are also low in NZ soil, which results in low selenium levels in the food chain, thus putting the NZ population at risk of selenium deficiency (Thompson, 2004a). Therefore, it is also important to assess the participants' selenium status and dietary intake, in order to see if there is any association (or consequence) to their iodine status.

Research to date, concerning iodine, has been predominantly on people living in the South Island of NZ. Therefore, in order to assure the effectiveness of proposed interventions, it is also necessary to carry out further research on people living in the North Island. This research is important, in order to quantify the degree of ID problem in the country as a whole. This present study, in Palmerston North in the North Island, will add to the knowledge concerning iodine and selenium intake throughout NZ, particularly after the introduction of the mandatory fortification of bread with iodine.

1.3 Aim and Objectives

1.3.1 Aim:

To explore the iodine and selenium status, dietary iodine and selenium intakes and iodine nutritional knowledge, of women of childbearing-age in Palmerston North, NZ, after the implementation of the mandatory fortification of bread with iodine.

1.3.2 Objectives:

i) To assess the iodine status of women of childbearing-age in Palmerston North, via the concentration in 24-hour urine samples;

ii) To evaluate the dietary iodine intake of women of childbearing-age, including the determination of major food contributors to iodine intake;

iii) To investigate the knowledge of women of childbearing-age concerning iodine deficiency and sources of iodine in NZ;

iv) To investigate the knowledge and attitudes of women of childbearing-age concerning the mandatory fortification of bread with iodine in NZ;

v) To determine the selenium status and intake of women of childbearing-age in NZ and its association with iodine status.

1.4 Overview of the study

Chapter 1 (the introduction) provides a brief background of the study, together with the aim and objectives. Chapter 2 of this thesis examines the literature concerning iodine nutrition and it provides the epidemiological data on the global ID problem, including NZ. Methods of iodine status assessment are discussed, in order to clarify the suitability of the method used in the present study. It also discusses the importance of selenium nutrition in reducing the risk of ID. Finally, the chapter identifies the existing approaches or interventions used to correct ID, or improve the iodine status of a population, which include the current situation in NZ. Chapter 3 describes details of the materials and methods used in the study, in addition to the procedures involved. Chapter 4 states the results of the study, which are then discussed further in the following Chapter 5. The discussion is generally relevant to the aims and objectives of the study. The limitations of the research are also addressed in this chapter. Finally, Chapter 6 presents the conclusion of the study and it provides recommendations for future research, in addition to suggested actions required in order to improve the overall iodine status and intake of the NZ population.

2 LITERATURE REVIEW

2.1 Introduction

Iodine deficiency (ID) is a worldwide public health problem, which has long been observed in many parts of the world, including New Zealand (NZ) (Benoist, Andersson, Egli, Takkouche, & Allen, 2004). In NZ, iodine deficiency disorder (IDD) has been prevalent since the end of the 1800s and the beginning of the 1900s (Aitken, 2001). In the 1920s and 1930s, many studies demonstrated mild to moderate ID amongst populations in NZ, which were characterised by high rates of goitre and low urinary iodine concentration (UIC) levels (Purves, 1974; Shore, 1929; Shore & Andrew, 1929, 1934). In 1924, iodised table salt was introduced into NZ in order to overcome this problem; however, the iodine concentration was too low (below 40 mg/kg) and it was therefore insufficient. Thus, in 1938, the iodine concentration in salt was increased to 40-80 mg/kg and it remains at this level today. As a result, goitre appeared to be eradicated by the 1950s and the iodine deficiency problem had dramatically declined. (Aitken, 2001; Mann & Aitken, 2003). In addition, the dairy industry in NZ started to use iodophors as sanitisers, during the 1960s, which ultimately increased the iodine content of dairy products (Sutcliffe, 1990). This helped to increase the iodine supply in the NZ diet, and studies carried out in NZ during the 1970s and 1980s, reported sufficient iodine intake amongst participants (Cooper, Croxson, & Ibberston, 1984; North & Fraser, 1965; Simpson, Thaler, Paulin, Phelan, & Cooper, 1984).

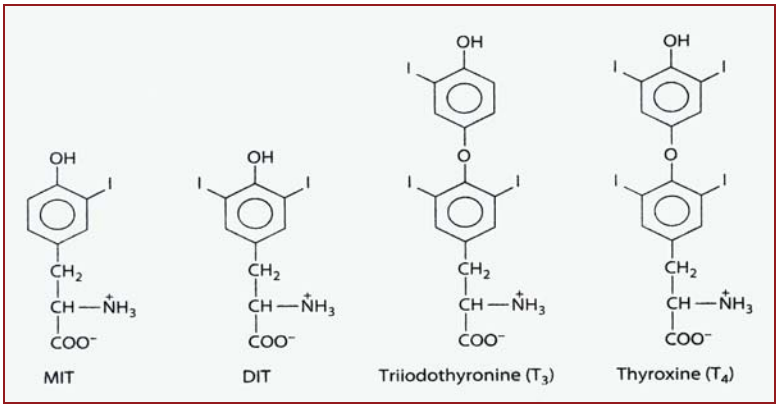
However, in the 1980s, the dairy industry replaced iodophors with different sanitisers (which did not contain iodine) and this lowered the iodine content in dairy products (Aitken, 2001). During the 1990s, dietary iodine intake declined, as reported in the NZ Total Diet Survey (NZTDS) 1990/91, 1997/98 and 2003/04 (Aitken, 2001; Vannoort, Cressey, & Silvers, 2000; Vannoort & Thomson, 2005). Mild to moderate iodine deficiency amongst children and adults in NZ has been reported in recent studies, thus demonstrating that there is a possibility that ID is re-emerging in NZ (Brough & Jin, 2010; Rose, Gordon, & Skeaff, 2009; Skeaff et al., 2005; Skeaff, Menon, & Pettigrew, 2010; Skeaff, Thomson, & Gibson, 2002; C. D. Thomson, 2004b; C. D. Thomson et al., 1997; C. D. Thomson, Packer, et al., 2001; C. D. Thomson, Woodruffe, Colls, Joseph, & Doyle, 2001). For this reason, the NZ Ministry of Health (MOH) implemented the mandatory addition of iodised salt to all bread in NZ (except organic,

par-baked and unleavened bread) from September 2009 (NZFSA, 2009). The content of iodine contant in salt must be within the range of 25 mg/kg to 65 mg/kg (NZFSA, 2009). The food industry will most probably produce iodised salt containing 45 mg/kg; as they will aim for the mid point value of the range, to ensure the iodine amount is always within the required range (Rose, Gordon, & Skeaff, 2009).

2.2 Iodine

Iodine is an important component for all mammals, since it is crucial for the production of thyroid hormones in the body. It is a non-metal mineral that usually occurs in ionic form; iodide (I^-) and iodate (IO_3^-), and molecular form (I_2) (Gibson, 2005; Stipanuk, 2006). The human body needs small amounts of iodine over a lifetime, but requires it on a regular basis: the mass amount of iodine (approximately 70% to 80%) is concentrated in the thyroid gland (Gibson, 2005; Stipanuk, 2006; WHO & FAO, 2004b). Iodine is required to synthesise thyroid hormones, which are crucial for the regulation of biochemical reactions such as enzymatic activities and protein synthesis. It is also needed for normal metabolic processes in the body, especially in maintaining growth and development (WHO & FAO, 2004b). The thyroid hormones in the body are found in the form of thyroxine (T_4) and triiodothyronine (T_3) (Figure 2.1).

Figure 2.1 Structures of thyroid hormones T_3 and T_4, and thyroglobulin MIT and DIT. Figure taken from Gropper et al. (2005).



2.3 Absorption and metabolism of iodine

Most iodine, which is ingested into the body will be absorbed in the stomach and the small intestine, where the process occurs rapidly and the iodine is well absorbed (Higdon, 2003; Stipanuk, 2006). Before absorption, iodine mostly appears freely as iodide and iodate, or it attaches to amino acids. The iodate form is usually reduced to iodide by glutathione in the gut, before it enters the blood circulation (Boron &

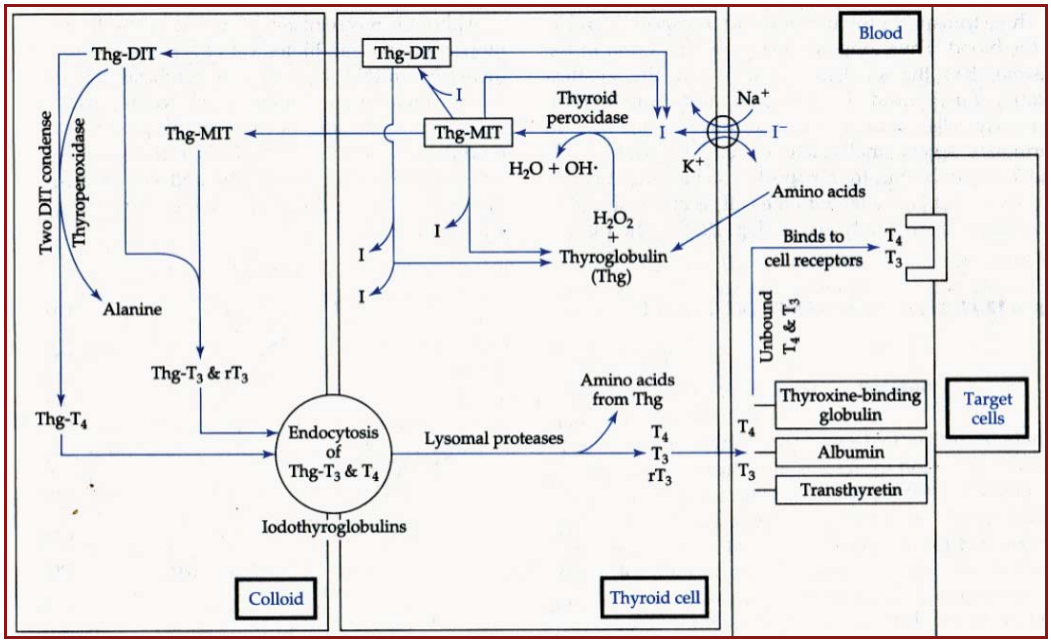
Boulpaep, 2009; Gropper et al., 2005; WHO & FAO, 2004b). The iodide ion is more efficiently absorbed into the circulation compared to iodate or other organic forms of iodide, which can still be absorbed, but only in small amounts (Gropper et al., 2005). Approximately 90% of iodide is absorbed from the gastrointestinal tract into the circulation (Higdon, 2003; Zimmermann, Jooste, & Pandav, 2008). The remaining iodine (less than 10%) will be excreted in the faeces (Gropper et al., 2005). Thyroid hormones in the gut (and both T_3 and T_4) are well absorbed into the circulation with a bioavailability of approximately 75% (Hays, 1991). Since T_4 and T_3 are unchanged and remain stable when they enter into the blood circulation from the GI tract, thyroxine has been prescribed as an oral medication for patients with hypothyroidism (Bolk, Visser, Kalsbeek, Van Domburg, & Berghout, 2007; Hays, 1991).

In the blood circulation, iodine has a tendency to permeate into almost all tissues in the body, primarily into the thyroid gland, followed by other tissues such as kidney and brain, since it is circulated around the entire extracellular fluid. The thyroid gland has the highest concentration of iodine, which is 40 to 50 times higher than plasma concentrations; iodide from the blood is taken up into the thyroid gland by the sodium/iodide (Na/I) active transporter onto the membrane of the thyroid follicular cell (Boron & Boulpaep, 2009; Eskandari et al., 1997; Vanderpas, 2006). Uptake of iodide into the thyroid gland also depends on the thyroid-stimulating hormone (TSH). TSH is released by the anterior pituitary gland, which is stimulated by the thyrotropin-releasing hormone (TRH) that is produced in the hypothalamus. Release of TSH stimulates the uptake of iodide from the blood circulation, by increasing the activity of the Na/I active transporter (NIS) on the membrane of the thyroid follicular cell (Boron & Boulpaep, 2009; Vanderpas, 2006).

Once iodide enters into the thyroid follicle, it is then oxidised to iodine by thyroid peroxidase and hydrogen peroxide. TSH stimulates the oxidised iodine to undergo iodination in the lumen. During this process, iodine is bound to a tyrosyl residue of thyroglobulin at the number three position, catalysed by thyroid peroxidase: and it forms thyroglobulin-3-monoiodotyrosine (Thg-MIT) (Boron & Boulpaep, 2009; Setian, 2007). The Thg-MIT then undergoes further iodination at the number five position, in order to generate thyroglobulin-3,5-diiodotyrosine (Thg-DIT) (Figure 2.2) (Boron & Boulpaep, 2009; Gropper et al., 2005; Vanderpas, 2006). In the colloid, the TSH stimulates the conjugation of Thg-DIT and Thg-MITor two Thg-DITs to form Thg-T_3 and Thg-T_4, respectively, catalysed by thyroid peroxidase. When these components are exposed to proteolytic enzymes, T_3 and T_4 are released from the thyroglobulin.

TSH stimulates T_3 and T_4 to be secreted into the blood circulation and then to be used in target tissues (Boron & Boulpaep, 2009; Setian, 2007; Vanderpas, 2006). The synthesis of thyroid hormones is illustrated in Figure 2.2 below.

Figure 2.2 The synthesis of thyroid hormone. Taken from Gropper et al. (2005).



2.4 Actions of thyroid hormones

Iodine generally functions as a component of thyroid hormones in the body in the form of T_3 and T_4 (FNB & IOM, 2001; Higdon, 2003). T_4 is more abundant in plasma, since its concentration is 50 times higher than T_3, but T_4 is less potent than T_3. This is because 99.98% of T_4 is bound tightly to the thyroid-binding globulin (TBG), but less T_3 binds to TBG, which is 99.50%. This results in the amount of free T_4 in the circulation being only ~2-fold more than free T_3 (Boron & Boulpaep, 2009). Once T_4 enters the target tissues, such as the brain, brown adipose cells, pituitary gland, kidney and liver, it will be converted to T_3 through deiodination. The conversion of T_4 to T_3 requires a selenocysteine-containing enzyme and a deficiency of selenium can impair this process (Berry, Banu, & Larsen, 1991; C. D. Thomson, 2004a; WHO & FAO, 2004a). In the target cells, thyroid hormone receptor (TR) has a higher affinity (~10-fold) for T_3 than for T_4. Therefore, 90% of TR is bound tightly by T_3. The thyroid hormones bind to TR in the nucleus of cells, for normal regulation of gene expression (Boron & Boulpaep, 2009; WHO & FAO, 2004b).

Thyroid hormones are involved in many physiological processes such as the regulation of cell activities and control of basal metabolic rate and metabolism. For

example, thyroid hormones are requiredto increase energy production and lipolysis, and also to regulate gluconeogenesis and glycolysis (WHO & FAO, 2004b). Thus, thyroid hormones are crucial for human body growth and development, including the maturation of the whole body (FNB & IOM, 2001; WHO & FAO, 2004b). This is especially critical for fetal and postnatal growth and development, especially brain and mental development. This is mainly because rapid growth (especially brain growth) occurs from conception until two or three years of age (de Escobar, Obregon, & del Rey, 2007; Delange, 2000; WHO & FAO, 2004b). Inadequate iodine intake during fetal and infant growth can interrupt the development of brain and central nervous system and eventually cause brain damage or mental retardation, depending on the severity of ID (de Escobar et al., 2007; Delange, 2000; WHO & FAO, 2004b). Therefore, the requirement for dietary iodine is higher during pregnancy and lactation in order to ensure a sufficient iodine supply for both mother and fetus/infant (Delange, 2007; Zimmermann, 2009b). Moreover, it is very important to diagnose and treat ID in pregnant and lactating women and women of childbearing-age, in addition to children aged three years and below. Brain damage and mental retardation are avoidable and therefore, the prevention of IDD is a major concern to these vulnerable groups of people, since they have a high risk of iodine deficiency (de Escobar et al., 2007; WHO/UNICEF/ICCIDD, 2007).

2.5 Interaction with goitrogens

The metabolism of iodine is inhibited in the presence of goitrogens, which can worsen iodine deficiency. Goitrogens are naturally occurring substances that inhibit thyroid hormonogenesis: a high uptake of goitrogens in people with ID (and/or a long duration intake of them) can affect the thyroidal iodide uptake and iodide transport (Eastman & Zimmermann, 2009; Gropper et al., 2005; Laurberg et al., 2009). This occurs when the metabolites of goitrogenic compounds such as glucosinolates, thiocyanate and astatide, compete with iodide for the NIS into the follicular cells in the thyroid gland (Ermans, Delange, Van der Velden, & Kinthaert, 1972; Laurberg et al., 2009). Some foods are goitrogenic, mostly the cruciferous vegetables such as cauliflower, cabbage, turnips, kale and broccoli. Thiocyanate is also the metabolite of cyanogenic glucoside that can be produced in the body by the high consumption of some other goitrogenic vegetables, such as cassava, sweet potato, linseed and lima beans (Ermans et al., 1972; Zimmermann, 2009a). The majority of these vegetables, however, would rarely be consumed at the very high quantity that can cause ID or worsen the ID of a population. The exception is cassava, which is mostly consumed at a high quantity as it is the staple food in many developing countries (Eastman & Zimmermann, 2009;

Gropper et al., 2005). Other than natural sources, industrial pollution or tobacco smoking can also result in a high ingestion of cyanide, which will eventually be metabolised to thiocyanate (Laurberg et al., 2009). The amount of cyanide in a cigarette is variable, but it is generally very high (range from 150 to 500 mcg/cigarette) (Scherer, 2006). Moreover, the cyanide in tobacco smoke is rapidly metabolised to thiocyanate by the liver through a sulphation process (Laurberg et al., 2009). Studies report that maternal smoking contributes to a low level of iodine in breast milk and an increase in the risk of IDD in the breast-fed infant (Laurberg, Nohr, Pedersen, & Fuglsang, 2004; Oliveira et al., 2009).

2.6 Dietary requirement of iodine

It is important to ensure sufficient iodine intakes in all age groups of people, but especially in pregnant and lactating women, and also children below three years of age. In general, the requirement of iodine has been estimated based on several indicators which include: daily uptake and turnover of iodine in the body; urinary iodine concentration/excretion; thyroid size and goitre rate; radioactive iodine uptake; thyroid hormones in plasma or serum; and iodine balance in the body (FNB & IOM, 2001; WHO/UNICEF/ICCIDD, 2007). The healthy adult requirement for dietary iodine is 150 mcg/day daily, and the upper limit is extremely high (1000-1100 mcg/day) (FNB & IOM, 2001; WHO & FAO, 2004b; WHO/UNICEF/ICCIDD, 2007). The iodine requirement increases during pregnancy and lactation, when it is needed to ensure a sufficient supply to the fetus and infant, especially in the first trimester of pregnancy, when the fetal thyroid gland is not functioning. During lactation, a high iodine intake is needed to replace the loss of iodide in breast milk through lactation. Whereas, during pregnancy, the demand of maternal T_4 production is increased in order to maintain maternal euthyroidism and also normal metabolism, which are normally higher in pregnant women (Azizi & Smyth, 2009; Delange, 2004, 2007; Kung, 2007; Zimmermann, 2009a). In addition, the mainstream view is that renal clearance increases significantly during pregnancy (Delange, 2007). Therefore, it contributes to a greater loss of iodide through the kidney in pregnant women, and hence, it increases the requirement of dietary iodine during this period (Dafnis & Sabatini, 1992; Delange, 2007; Fantz, Dagogo-Jack, Ladenson, & Gronowski, 1999; Glinoer, 1997, 2007). This loss of iodide would eventually lower the concentration of plasma inorganic iodide (PII) in blood serum. Many studies have shown an increase in iodine excretion during pregnancy (Hess, Zimmermann, Torresani, Bürgi, & Hurrell, 2001; Kung, Lao, Chau, Tam, & Low, 2000; Smyth, 1999; Smyth, Hetherton, Smith, Radcliff, & O'Herlihy, 1997). However, some studies have shown contradictory results, where the loss of

iodide through the kidney is not significant, or it is not more in pregnant mothers. A study in Santiago has shown that the PII concentration in pregnant mothers shows no significant decrease throughout the gestation period, in an iodine-sufficient region (Liberman, Pino, Fang, Braverman, & Emerson, 1998). Moreover, an investigation conducted by Dworkin et al. (1966) found that there is no significant difference of iodine excretion between pregnant mothers and non-pregnant women and adult men, in the same environment. Some studies have also shown that iodine excretion even decreases during pregnancy (Caron et al., 1997; Glinoer et al., 1990; Vermiglio et al., 1992). These inconsistent results show that an increased loss of iodide through increased renal clearance during pregnancy is not confirmed. In general, the high requirement of iodine in pregnant and lactating women is basically due to requirements for their own use, in addition to supplying their fetus and infants.

The recommended daily iodine intake for pregnant and lactating women as set by the World Health Organization (WHO), the United Nations International Children Emergency Fund (UNICEF) and the International Council for Control of Iodine Deficiency Disorder (ICCDD), is 250 mcg/day (WHO/UNICEF/ICCIDD, 2007). Whereas, the Food Nutrition Board (FNB) and the United States (US) Institute of Medicine (IOM) recommends 220 mcg/day for pregnant women and 290 mcg/day for lactating women (FNB & IOM, 2001). In NZ and Australia, the recommended iodine intake for pregnant women is similar to IOM (220 mcg/day), whilst the recommendation for lactating women is 270 mcg/day, which is slightly higher than the WHO recommendation. The recommendation for iodine intake varies and it is slightly different between countries since requirements being based on epidemiologic or population studies in their own countries (FNB & IOM, 2001).

The requirement of dietary iodine intake in adult women recommended by WHO,UNICEF and ICCIDD (2007), and FNB and IOM (2001) and also NZ MOH is 150 mcg/day, which is substantially less than the requirement for pregnant and lactating women. Table 2.1 shows the recommended daily iodine intake for people in all age groups, according to WHO, UNICEF and ICCDD (2007), FNB and IOM (2001)and the Nutrient References Value (NRV) Australia and NZ (Australian National Health and Medical Research Council & New Zealand MOH, 2006b). The latest national survey (the 2003/04 NZ Total Diet Survey) estimated that women and preschool children (agedone to three years) in NZ consumed approximately 60 mcg/day and 47 mcg/day dietary iodine respectively, which is far below the recommended dietary intake (RDI) of 150 mcg/day and 110 mcg/day (Vannoort & Thomson, 2005).

Table 2.1 The recommendation of daily iodine intake by population sub-group.

Population sub group	Amount daily intake according to:		
	WHO/UNICEF /ICCDD	IOM	NRV Australia / NZ
Infants and preschool children	90 mcg/day (0 to 59 months)	90 mcg/day (1 to 8 years)	90 mcg/day (0 to 6 months) 110 mcg/day (7-12 months)
Schoolchildren	120 mcg/day (6 to 12 years)	120 mcg/day (9 to 13years)	120 mcg/day (9-13 year)
Adolescents (boys and girls) / Adults (men and women)	150 mcg/day	150 mcg/day	150 mcg/day
Pregnant women	250 mcg/day	220 mcg/day	220 mcg/day
Lactating women	250 mcg/day	290 mcg/day	270 mcg/day

2.7 Food sources of iodine

Iodine present in food is usually found in the form of inorganic iodide (I^-). The content of iodine in food is extremely variabledue to the variability of iodine levels in the soil in which it is grown, in addition to the nature or amount of iodine in the animal feed or fertiliser that is used in the agricultural industry or plant cultivation. The variability of iodine concentration in the soil is highly variable, depending on geographical location: it is especially low in mountainous, inland regions. Low iodine in the soil is usually caused by the frequency of glaciation and snow and/or heavy rainfall that leach out minerals, which are then carried to the ocean (WHO & FAO, 2004b). Thus, plant and marine life from seawater is always rich in iodine. Seaweed has the capability to concentrate iodine from the ocean and therefore, it has a very high iodine concentration: and marine animals that consume seaweed are also rich in iodine. Therefore, seaweed, sea fish, shellfish and other seafoods, are excellent food sources of iodine (Gibson, 2005; Higdon, 2003; WHO & FAO, 2004b). In certain countries (including NZ and Australia) eggs and dairy products (mainly milk) usually have a moderate iodine content and therefore, they are considered as sources of iodine. On the other hand, the iodine content in meat varies markedly depending on the season and how the animal has been fed. Animals fed with food containing added iodine show a high iodine in their meat and other products (Als, Haldimann, et al., 2003). In certain European countries (e.g. Switzerland, United Kingdom), farm animals freely graze on

grass in the field during summer, with a low iodine content in the soil. Thus, there is a lower iodine content in the meat or products from these animals in the summer months, in comparison to when they are kept in stables during the winter, when they are usually fed with food containing iodine (Als, Haldimann, et al., 2003; FNB & IOM, 2001).

Iodised salt (or food fortified with iodised salt) has been used widely in many parts of the world, especially in areas with ID, including developed countries, such as NZ (FSANZ, 2009) and many European countries, for example, Switzerland, Poland, and Austria (Heinisch et al., 2002; Szybinski et al., 2001; Zimmermann, 2007). Food products such as bread, biscuits or cereal containing iodised salt are good sources of iodine. In addition, food products containing seaweed such as sushi and seameal custard, can provide good iodine sources. Iodised salt, which is used regularly at home as table salt or in cooking, can also provide iodine in the diet on a regular basis. There are also adventitious sources that provide iodine in food, such as dough conditioners containing iodates; disinfectant (containing iodoform) used in the water; iodine containing food colour, such as erythrosine and rose bengal; and sanitiser containing iodophor, which is used in the dairy industry (Gibson, 2005; Kanno, Goda, Sato, Yoshihira, & Hayashi, 1995; Vought, Brown, & Wolff, 1972). All these sources can contribute to an increase in the dietary iodine intake in a population.

2.7.1 Good iodine sources to reduce ID in New Zealand

NZ MOH recommends the inclusion of good food sources of iodine such as fish, seafood, seaweed, sushi, milk, eggs and seameal custard in the NZ diet (MOH, 2010d). Since a large amount of food in NZ contains low iodine, the MOH also encourages people to use iodised salt instead of non-iodised salt. In order to increase the iodine supply in the NZ diet, the mandatory replacement of non-iodised salt with iodised salt in breads was implemented in NZ in September 2009 (MOH, 2010d). Nevertheless, the amount of iodine needed for pregnant and lactating women in NZ might still be inadequate in order to achieve the RDI of 250 mcg/day and 270 mcg/day respectively (MOH, 2010e). Table 2.2 lists the iodine content in good food sources of iodine in NZ according to the NZ Total Diet Survey 2003/04 (Vannoort & Thomson, 2005) and NZ MOH (MOH, 2010d).

Table 2.2 Iodine content in good food sources for iodine in New Zealand.

Food	Iodide content (mcg/100g)
Sushi	92
Eggs	41.5
Sea fish, fresh	23.7
Shell fish – fresh	21.6
Fish, canned	18
Milk	9.6
Seameal custard mixed with milk	11.3
Milk flavoured	7.6
Seafood (processed)	6.5
Cheese	6.1
Yoghurt	5.6

Moreover, soils in NZ have a low concentration of selenium, which is an element that is necessary for thyroid metabolism: in particular the selenocysteine-containing enzyme is needed to convert T_4 to T_3 in the body (C. D. Thomson, 2004b; C. D. Thomson, Campbell, Miller, Skeaff, & Livingstone, 2009; C. D. Thomson & Robinson, 1980). Therefore, good food sources of selenium such as fish, seafoods, kidney, liver and Brazil nuts should be in included in the NZ diet (C. D. Thomson, 2004b; C. D. Thomson, Chisholm, McLachlan, & Campbell, 2008). Table 2.2 lists the iodine content in good food sources of iodine in NZ according to the NZ Total Diet Survey 2003/04 (Vannoort & Thomson, 2005) and NZ MOH (MOH, 2010d).

2.8 Assessment of iodine nutrition

Assessment of iodine nutrition is important in order to monitor and control ID in a population, especially in areas that have prevalent cases of mild to severe IDD. There are three main indicators widely used to assess iodine status in a population and (in particular) they are useful for assessing ID in adult women (pregnancy, lactating and child-bearing age women): (i) the urinary iodine concentration/excretion; (ii) serum levels of thyroglobulin (Tg); and (iii) goitre rate. All these methods have their own sensitivity: from acute detection of recent dietary iodine intake (within 24 hours) shown by the iodine excretion; to the reflection of an intermediate duration of iodine nutrition

(weeks to months) demonstrated by Tg serum; to the response to a long-term effect of iodine nutrition, especially used for iodine control programmes and shown by changes in the goitre rate (within months to years) (Gibson, 2005; WHO/UNICEF/ICCIDD, 2007; Zimmermann, 2008b).

Serum levels of TSH, thyroxine (T_4) and triiodothyronine (T_3) have also been used to assess iodine status in a population. However, these are less sensitive and not preferable compared to the three indicators mentioned above. TSH serum reflects serum T_4 concentration in an inverse relationship: serum TSH rises when serum T_4 concentration falls and vice versa. However, the differences between the concentrations of these two (in serum) are usually not significant in ID, but they remain within the normal range. This has made TSH serum unsuitable for measuring iodine deficiency (WHO/UNICEF/ICCIDD, 2007). However, serum TSH is very responsive in the neonate, when iodine content is low in the thyroid gland. This causes high iodine turnover, which eventually exhibits elevated TSH concentration in serum in the newborn (Delange, 1997; WHO/UNICEF/ICCIDD, 2007). Therefore, this can be used as a screening test for congenital hypothyroidism and it is recommended in iodine-deficient populations (Gibson, 2005; WHO/UNICEF/ICCIDD, 2007). On the other hand, the concentration of thyroid hormones (T_3 and T_4) in adult or school-age children is usually maintained at a normal range, even in cases of mild to severe ID. In addition, serum T_3 is sometimes even higher than the serum T_4 in normal populations (Gibson, 2005; WHO/UNICEF/ICCIDD, 2007). A significant decline of these hormones can only be shown in cases of very severe ID (UIC less than 20 mcg/l) (Lagasse et al., 1982 cited in Gibson, 2005). This shows the very low sensitivity of both serum T_3 and T_4 (Gibson, 2005; WHO/UNICEF/ICCIDD, 2007; Zimmermann, 2008b). Therefore, serum TSH, T_3 and T_4 are not good indicators for assessing the iodine status of a population, according to the WHO, UNICEF and ICCIDD (2007).

2.8.1 Urinary iodine excretion

Approximately 90% of the total dietary iodine intake is excreted in the urine, within 24 hours (C. D. Thomson, 2004b; Zimmermann, 2008b). Therefore, urinary iodine excretion (UIE) over 24 hours is the best indicator to assess the most recent iodine intake. However, the UIE is not useful for individual assessment, since dietary intake is highly variable even within an individual, but the variation evens out at the population level (WHO/UNICEF/ICCIDD, 2007). Nevertheless, a multiple collection of a 24-hour urinary iodine sample from each participant can also help to increase the accuracy of

the result (Rasmussen, Ovesen, & Christiansen, 1999). The UIE can be measured either by 24-hour urine collections, or a casual urine sample, or a fasting morning specimen (one spot specimen). In order to indicate the ID level, the iodine excretion is best expressed as a 24-hour excretion (mcg/day). However, it can also be expressed as UIC (mcg/l), or in relationship to creatinine excretion (mcg iodine/g creatinine) (Als, Minder, et al., 2003; Vejbjerg et al., 2009). The median UIC value of a study sample is generally used as an indicator for the iodine status of a population (WHO/UNICEF/ICCIDD, 2007). This is because the frequency of the distribution of UIC value in study samples is usually not normally distributed. Therefore, the median value is the best one to describe the measure of central tendency for the UIC value of a population study (Gibson, 2005; WHO/UNICEF/ICCIDD, 2007). The WHO has set the cut-off point to indicate the iodine status of a population according to median UIC value (mcg/l) (Table 2.3) (WHO/UNICEF/ICCIDD, 2007).

The measurement of a 24-hour UIE is more representative than casual urine specimens, but it is sometimes impractical to measure a 24-hour urinary excretion in the field. This is especially true if the study samples are children and/or the sample size is large (Vejbjerg et al., 2009; WHO/UNICEF/ICCIDD, 2007). As an alternative, a spot urine specimen can be used to measure the iodine status of a population, which is expressed in the median value of a representative study sample (Pardede et al., 1998; WHO/UNICEF/ICCIDD, 2007).

Studies have demonstrated that the UIC median value, from casual urine specimens, correlate significantly with the concentration of 24-hour urine excretion samples (Knudsen, Christiansen, Brandt-Christensen, Nygaard, & Perrild, 2000; C. D. Thomson et al., 1997), or serum thyroglobulin (van den Briel et al., 2001; Zimmermann, de Benoist, et al., 2006; Zimmermann, Moretti, Chaouki, & Torresani, 2003). In contrast, some studies reported that the casual urine sample is not significantly correlated with the 24-hour UIE (S Andersen, Pedersen, Pedersen, & Laurberg, 2001; Busnardo et al., 2005; Rasmussen et al., 1999). Due to these inconsistent results, casual urine samples may not give an accurate value in measuring ID, but on the other hand, the 24-hour UIE are more preferable and valid in assessing the iodine status of a population (Als, Minder, et al., 2003; Remer, Fonteyn, Alexy, & Berkemeyer, 2006; Ristic-Medic et al., 2009; C. D. Thomson, Woodruffe, et al., 2001).

Table 2.3 Epidemiological criteria for assessing iodine nutrition in a population based on median or range in UIC (WHO/UNICEF/ICCIDD, 2007).

Median UIC (mcg/l)	Iodine intake	Iodine status
*** School-aged children:**		
< 20	Insufficient	Severe iodine deficiency
20 - 49	Insufficient	Moderate iodine deficiency
50 - 99	Insufficient	Mild iodine deficiency
100 - 199	Adequate	Optimal
200 - 299	Above requirements	Likely to provide adequate intake for pregnant/lactating women, but may pose a slight risk of more than adequate intake in the overall population
≥ 300	Excessive**	Risk of adverse health consequences (iodine-induced hyperthyroidism, autoimmune thyroid disease)
Median UIC (mcg/l)		Iodine intake
Pregnant women:		
< 150		Insufficient
150 - 249		Adequate
250 – 499		More than adequate
≥ 500		Excessive**
‡ Lactating women:		
< 100		Insufficient
≥ 100		Adequate
Children less than 2 years old:		
< 100		Insufficient
≥ 100		Adequate

* Applies to adults, but not to pregnant and lactating women
‡ Although lactating women have similar requirement as pregnant women, the median UIC is lower because iodine is excreted in breast milk
** The term 'excessive' means *in excess of the amount required to prevent and control ID*

Overall, the estimation of dietary iodine intake based on the 24-hour UIE is preferable if this is being undertaken in the field, with a large sample size (FNB & IOM, 2001; C. D. Thomson, 2004b; Zimmermann, 2008b). According to FNB and IOM (2001), there

is also a formula than can be used to measure iodine intake based on a 24-hour iodine excretion result, which is as follows: urinary iodine (mcg/l) × 0.0235 × wt (kg) = daily iodine intake. For example, the calculations of this formula would approximately give an average of 150mcg total daily iodine intake if based on the UIC median value of 100 mcg/l in adult women.

2.8.2 Thyroid size

Iodine deficiency causes the thyroid gland (located at the anterior and lateral parts of the larynx and trachea) to enlarge. This thyroid enlargement is known as goitre and the enlargement size depends on the severity of the ID (Dumont et al., 2008). The assessment of thyroid size can be used as an indicator of iodine status: as can the rate of goitre in a population. However, the enlarged thyroid gland will take a long period (months to years) to return to its normal size, after a correction of the iodine deficiency. Therefore, the severity of IDD in a population is better evaluated by the size of the thyroid gland, whilst the degree of ID is better evaluated by the prevalence of the goitre rate (Delange et al., 1997; Peterson et al., 2000). There are two methods which are widely used to measure goitre: the conventional method (neck inspection and palpation) and the modern method (thyroid ultrasonography) which is more precise and objective (Gibson, 2005; Peterson et al., 2000).

According to the WHO, UNICEF and ICCIDD (2007), studies that use the palpation method usually refer to a goitrous thyroid gland as “a thyroid gland each of whose lobes have a volume greater than the terminal phalanges of the thumb of the person examined” (p. 35). The WHO, UNICEF and ICCIDD (2007) have classified goitre into grade 0 to 2, as presented in Table 2.4.

Table 2.4 Simplified classification of goitre by palpation.

Grade 0	No palpation or visible goitre
Grade 1	A goitre that is palpable but not visible when the neck is in the normal position (i.e. the thyroid is not visibly enlarged). Thyroid nodules in a thyroid, which is otherwise not enlarged, fall into this category
Grade 2	A swelling in the neck that is clearly visible when the neck is in a normal position and is consistent with an enlarged thyroid when the neck is palpated

The WHO, UNICEF and ICCIDD (2007) have also categorised the degree of IDD, based on the prevalence of goitre, in school-age children. The total goitre rate (TGR) is the total amount of grade 1 and 2 goitre rates of a population (Table 2.5).

Table 2.5 Epidemiological criteria for assessing the severity of IDD based on the prevalence of goitre in school-age children.

Severity of IDD	Prevalence of goitre (TGR) grades 1 and 2 (expressed as percentage of the total number of children in the study)
None	0.0 – 4.9 %
Mild	5.0 – 19.9 %
Moderate	20.0 – 29.9 %
Severe	≥ 30 %

Epidemiological studies on goitre rate are usually undertaken on school-age children, ranging from 6 to 12 years old, since they are easily accessible (WHO/UNICEF/ICCIDD, 2007; Zimmermann, 2008b). However, goitre is more prevalent in adolescents or women of child-bearing age, according to the WHO, UNICEF and ICCIDD (2007). In addition, performing palpation on young children is relatively difficult, especially with small children since their thyroid size is small, which makes it hard to classify the goitre grade (Gibson, 2005; WHO/UNICEF/ICCIDD, 2007; Zimmermann, 2008b). Moreover, there is a high tendency to misclassify the grade of goitre, particularly between grade 0 and 1, since the thyroid gland is not visibly enlarged with grade 1. Thus, the severity of IDD and the prevalence of goitre in a population may be inaccurate; this is especially so with mild ID, since the sensitivity and specificity of the palpation method is poor, due to the low percentage of visible goitre (Vitti et al., 1994; Zimmermann, Saad, Hess, Torresani, & Chaouki, 2000). Therefore, the imaging method (thyroid ultrasonography) is an improved technique, which can determine the severity of IDD in a population and it is especially useful for mild to moderate ID severity (Gibson, 2005; WHO/UNICEF/ICCIDD, 2007). The ultrasonography imaging technique uses ultrasound technology to measure thyroid volume (Tvol): it is well-known as a safe, quick (two to three minutes per inspection) and non-invasive method (WHO/UNICEF/ICCIDD, 2007; Zimmermann et al., 2000). A portable version of ultrasonography is also available. It can be used with a car battery being the electricity source, thus making it very suitable to be used in remote areas. However this equipment is expensive and it needs to be handled only by a well-trained and experienced operator (Gibson, 2005; WHO/UNICEF/ICCIDD, 2007). Zimmermann et al. (2004) managed to set an international reference for the interpretation of Tvol data measured by ultrasonography. These reference values were developed based on

the outcome of a study in which ultrasonography was used to measure thyroid volume in large samples of children aged 6 to 12 years old (n=3529), living in iodine-sufficient areas around the world (Zimmermann, Hess, et al., 2004). These international reference values were set up as a function of body surface area and age, thus allowing comparison of thyroid volume between countries or regions (Table 2.6).

Table 2.6 Gender-specific 97th percentile (P97) of thyroid volume (ml), by age and body surface area (BSA), measured by ultrasonography.

Age (year)	Boys (P97)	Girls (P97)	**BSA (m^2)**	Boys (P97)	Girls (P97)
6	2.91	2.84	0.7	2.62	2.56
7	3.29	3.26	0.8	2.95	2.91
8	3.71	3.76	0.9	3.32	3.32
9	4.19	4.32	1.0	3.73	3.79
10	4.73	4.98	1.1	4.20	4.32
11	5.34	5.73	1.2	4.73	4.92
12	6.03	6.59	1.3	5.32	5.61
			1.4	5.98	6.40
			1.5	6.73	7.29
			1.6	7.57	8.32

2.8.3 Serum thyroglobulin (Tg) level

Thyroglobulin is only produced in the thyroid gland. It plays an important role in the synthesis of thyroid hormones in the thyroid gland. Approximately, less than 10 mcg/l of Tg in serum is normally seen in adults with iodine sufficiency (Spencer et al., 1998; WHO/UNICEF/ICCIDD, 2007; Zimmermann, 2008b). When dietary iodine is inadequate over a period of time (weeks to months), the concentration of serum Tg increases, which may lead to hyperplasia (proliferation of thyroid cells) and can also cause goitre. Therefore, Tg is usually used as a marker forassessing the chronic effects of ID (Missler, Gutekunst, & Wood, 1994). Thus serum Tg corresponds to iodine nutriture over a period of time, in contrast to UIC that only reflects the immediate iodine intake (Gibson, 2005; WHO/UNICEF/ICCIDD, 2007; Zimmermann, 2008b).

Studies have reported that Tg is also a more sensitive indicator compared to TSH and serum thyroid hormones, which has been demonstrated by the dramatic decline of Tg

corresponding to the correction of iodine deficiency in the study populations (Benmiloud et al., 1994; Knudsen et al., 2001; Missler et al., 1994). A new assay (a dried blood spot) has recently been developed and (as a result) Tg is now easily measured in field studies, especially in remote areas. Since Tg demonstrates an intermediate response (weeks to months), it is suitable to be used for monitoring the treatment of iodine deficiency after iodine repletion (Missler et al., 1994; WHO/UNICEF/ICCIDD, 2007). The standard international reference interval of the dried blood spot assay has been developed in iodine-sufficient school-age children: 4 to 40 mcg/l (WHO/UNICEF/ICCIDD, 2007).

2.9 Iodine deficiency disorders

Iodine deficiency is a common public health problem that affects a large number of people throughout the whole human lifecycle, including the fetus. ID impacts detrimentally on human growth and development, depending on the severity of ID in a population: severe ID during pregnancy or infancy can lead to serious problems, such as irreversible brain damage or mental retardation in infants and children (Fuge, 2007). ID is primarily due to inadequate iodine intake that lessens the synthesis of thyroid hormones and over a period of time, this can contribute to low thyroid hormone concentration, resulting in hypothyroidism (Setian, 2007). This also contributes to the diverse effects of illness in different age groups of people, depending on the severity of ID (WHO/UNICEF/ICCIDD, 2007; Zimmermann, 2006). According to the WHO, all illness caused by mild to severe ID, which can be prevented by a sufficient intake of iodine, are termed as iodine deficiency disorders (IDD), as listed in Table 2.7 (WHO/UNICEF/ICCIDD, 2007).

Hypothyroidism and goitre mostly occur due to ID and severe ID during pregnancy, which causes a pernicious form of hypothyroidism in neonates: cretinism (physical impairment and mental retardation) (D. S. Cooper, 1987; Raiti & Newns, 1971; Zimmermann et al., 2008). Goitre usually occurs amongst people with insufficient daily iodine intake of approximately less than 50 mcg/day (Gibson, 2005). This disorder has long been recognised (since the ancient world) and it is the most common IDD, since it affects all ages including newborn babies (Clements, 1958; Hetzel et al., 1988). On the other hand, the effect of ID on human brain development has only been widely explored during the past 30 years (Hetzel, 2005; Hetzel et al., 1988). Neurological cretinism, congenital abnormalities and other related IDD, which affect fetuses and newborn babies, are the most serious health problem, since these critically harm

fetuses and also increase infant morbidity and mortality rates within a population (Delange, 2000; Fuge, 2007; Zimmermann et al., 2008). This irreversible brain damage and mental retardation occurs mostly in severe iodine-deficient areas: but it can be prevented by iodine correction, in particular by ensuring sufficient iodine intake amongst pregnant and lactating mothers and women of child-bearing age, in addition to young children below three years of age (Delange, 2002; Hetzel, 2000, 2005). Maternal, fetal and neonatal ID demonstrates the spectrum of IDD at this critical stage of human growth and development.

Table 2.7 Iodine deficiency disorders (IDD) across the life-span.*

Population groups	**Iodine deficiency disorders**
Fetus	Abortions Stillbirths Congenital anomalies Neurological cretinism Hypothyroid cretinism Psychomotor defects Increased perinatal mortality
Neonate	Neonatal hypothyroidism Neonatal goitre Retarded mental and physical development and increase in infant mortality
Child and adolescent	Goitre (Subclinical) hypothyroidism Impaired mental function Delayed physical development
Adult	Goitre, with its complications Hypothyroidism Iodine-induced hyperthyroidism
All ages	Goitre Hypothyroidism Increase susceptibility to nuclear radiation

*Adapted from Hetzel (2005) and WHO/UNICEF/ICCIDD(2007).

2.9.1 Maternal and fetal iodine deficiency during gestation period

ID during pregnancy is the greatest cause of preventable intellectual impairment and it may also increase the risk of miscarriage or stillbirth. In the first and second trimesters of pregnancy, thyroid hormones are circulated into the blood stream and transferred across the placenta to the fetus, which is required to maintain normal fetal growth (Delange, 2004, 2007; Sack, Krassas, Darandeliler, Kraiem, & Laron, 2003). Therefore, it is important to ensure that pregnant mothers have sufficient iodine intake

throughout the whole gestation period (Delange, 2004; Glinoer, 2007). This is especially crucial during the early pregnancy period, since as the fetus depends solely on maternal T_4 before the onset of fetal thyroid function (Becker et al., 2006; Delange, 2004). Moreover, the daily requirement of T_4 in a pregnant mother increases gradually throughout the gestation period from 10% to 150% (Delange, 2004). Thus, inadequate iodine intake in a pregnant mother can eventually decrease thyroid hormone concentration in the circulation, thusresulting an inadequate supply of T_4 to the fetus (Pérez-lópez, 2007). Low T_4 in the circulation can cause hypothyroidism only in the mother or in both the mother and fetus, depending on the severity of the ID (Glinoer, 2007; Glinoer et al., 1995). Animal studies have also demonstrated that ID in the fetus is not only caused by the low production of thyroid hormones by the fetus itself, but it is also due to inadequate supplies of maternal thyroid hormones to the fetus, especially during the early gestation period (Meerts et al., 2002; Obregon, Mallol, Pastor, Morreale, & Escobar, 1984; Pathak, Sinha, Mohan, Mitra, & Godbole, 2010; Piosik, van Groenigen, van Doorn, Baas, & de Vijlder, 1997).

Adequate thyroid hormone levels in the fetus are critical during the rapid brain growth period, which occurs from mid-gestation and continues for the first two years of infancy (Gordon, 1997; Meerts et al., 2002). Low thyroid hormone levels in the fetus can impair brain development during gestation and the postnatal period, which eventually delays and slows the intellectual and cognitive development in infancy and later life (Glinoer, 2007; Pérez-lópez, 2007; Sack et al., 2003; Zimmermann, 2008a). Studies, including a meta-analysis (Qian et al., 2005), have indicated that chronic ID in pregnant mothers causes IDD in the fetus, which has been demonstrated by delayed cognitive and psychomotor development in childhood, especially in children from populations with severe ID (Bleichrodt, Drenth, & Querido, 1980; Morreale de Escobar, Obregon, & Escobar del Rey, 2004; Pharoah, Buttfield, & Hetzel, 1971).

2.9.2 Maternal and neonatal iodine deficiency during postnatal period

Following pregnancy, thyroid hormone levels return to normal, since there is no longer a need to transfer thyroxine (T_4) to fetus (Andersson, de Benoist, Delange, & Zupan, 2007; Azizi & Smyth, 2009). However, the iodine intake requirement still continues to be high during lactation: 250 to 290 mcg/day (see section 2.6). This is mainly because iodine is not only being excreted in the urine, but also additional loss occurs in breast milk: mothers with sufficient iodine intake are estimated to lose approximately 75 to 200 mcg/day of iodide in their breast milk (Azizi & Smyth, 2009; Delange, 2004;

Semba & Delange, 2001). Mothers still need to supply iodine to newborn babies through breastfeeding, since infants need to build reserves in their thyroid gland during the postnatal period (Andersson et al., 2007; Remer et al., 2010). Optimal iodine concentrations in breast milk should be in the range of 100 to 180 mcg/l; hence it is vital to ensure adequate intake in lactating mothers, so that sufficient iodine can also be provided to neonates (Azizi & Smyth, 2009; Delange, 2004; Semba & Delange, 2001). This can prevent ID in offspring, especially in areas with a high prevalence of IDD, which in turn, reduces the risk of neonatal hypothyroidism and mental defects (Azizi & Smyth, 2009; Delange, 2007; Remer et al., 2010; Semba & Delange, 2001). Studies have also reported that, in iodine-deficient regions, infants who consume infant formula containing iodine have a better iodine status compared to breast-fed infants, due to the low iodine concentration in breast milk (Bohles et al., 1993; Manz, Fuchs, Terwolbeck, Wiese, & Lombeck, 1993; Skeaff et al., 2005).

Adequate iodine is important for normal neonatal thyroid function, which is essential for the maintenance of normal body growth and development. This is particularly critical during the rapid brain growth in infancy: the human brain achieves only a third of its full size at birth and it gradually expands and rapidly grows from postnatal to the age of two or three years (Gordon, 1997; Hetzel, 2005). Thus, sufficient thyroid hormone production is needed, in order to ensure normal neurological development during the brain growth spurt phase, including the processes of myelination and synaptic (Morreale de Escobar et al., 2004). Adequate thyroid hormones are not only vital in preventing mental retardation or brain damage, they are also needed to achieve maximum brain potential at this stage of life (Dussault & Ruel, 1987; Gordon, 1997; Pérez-lópez, 2007).

Studies have demonstrated that mild to severe ID in pregnant and lactating women affects psychological development and cognitive and psychomotor functions in infancy and later life. This is demonstrated by better psychomotor, cognitive and intellectual performances in children born to mothers supplemented with iodine or those with optimal iodine status, when compared to children born to mothers with mild to severe ID (Berbel et al., 2009; Bleichrodt et al., 1980; Cao et al., 1994; Connolly et al., 1979; Haddow et al., 1999; Pop et al., 2003; Pop et al., 1999; Zimmermann, Connolly et al., 2006). Studies also report that correction of ID should occur at the beginning of pregnancy (during the first to second trimesters), since treatment of ID during the late gestation period does not improve or correct fetal or neonatal neurological development (Cao et al., 1994; Morreale de Escobar et al., 2004; O'Donnell et al.,

2002). Since studies have clearly shown that adequate iodine intake is vital during pregnancy and lactation, ensuring sufficient iodine intake (in women of childbearing age) is very necessary in order to ensure that their iodine intake is always sufficient, from conception through to the postpartum period, especially in iodine-deficient regions. This may reduce the risk of ID in both pregnant/breastfeeding mothers and their fetuses/infants, and it can also prevent any IDD that could adversely affect the fetal and neonatal periods in addition to a person's later life.

2.10 Epidemiology of iodine deficiency (ID)

2.10.1 Iodine deficiency worldwide

One of the most common micronutrient deficiencies is iodine deficiency, which is also the cause of the most common avoidable brain damage in the world (Hetzel, 2002). In 2007, the WHO estimated approximately two billion of people worldwide as having inadequate iodine intake: of whom 266 million were children (about a third of school-age children worldwide) (Benoist, McLean, Andersson, & Rogers, 2008). Europe has the highest prevalence of ID, since their proportion of school-aged children with median UIC below 100 mcg/l is the highest (52.4%), followed by the Eastern Mediterranean (48.8%). Whereas, the smallest proportion of children with insufficient iodine intake are found in America (10.6%), followed by the Western Pacific (22.7%) (Table 2.8) (Benoist et al., 2008). These findings may be strongly associated with iodised salt intake, since the smallest proportion of households that consume iodised salt is found in Europe (about 26%) and the greatest proportion is found in the Americas (approximately 90%) (Bellamy, 2002). ID is a public health problem in many countries around the world, since 10 countries have been classified with moderate ID and 37 countries with mild ID. Iodine intake is optimal in 49 countries, where the population median UIC is between 100 and 199 mcg/l. There are 27 countries, whose populations have more than sufficient iodine intake, with the median UIC being between 200 and 299 mcg/l, and an even greater iodine intake was also found in seven of these countries (median UIC above 300 mcg/l) (Benoist et al., 2008).

Although ID is still a public health problem in several regions around the world, the overall prevalence of ID has decreased since 2003, thus improving the global iodine status. The proportion of school-age children with median UIC below 100 mcg/l has decreased: from 36.5% (in 2003) to 31.5% (in 2007) (Benoist et al., 2008; Benoist et al., 2009). In these statistics, the major improvement has been seen in South-East Asia and Europe, since the prevalence of iodine intake below 100 mcg/l in school-age children has decreased by 9.6% (from 39.9% to 30.3%) and 7.5% (from 59.9% to

52.4%), respectively (Benoist et al., 2008; Benoist et al., 2009). This has also been seen in the decreasing number of countries with ID as a public health problem: a decrease has occurred from 110 to 54 countries, between 1993 and 2003 (characterised by the TGR) and from 54 to 47 countries, between 2003 and 2007 (characterised by the median UIC value) (Benoist et al., 2008). However, there has also been an increase in the number of countries with a population median UIC above 200 mcg/l from 29 (in 2003) to 34 countries (in 2007) (Benoist et al., 2004; Benoist et al., 2008). A continuous excess of iodine intake can lead to iodine toxicity or iodine-induced hyperthyroidism in a population, especially amongst vulnerable groups of people such as people with previous thyroid diseases (Roti & Uberti, 2001). Therefore, salt iodisation programmes need to be monitored and the population's iodine status should always be assessed, especially in a population undergoing iodine repletion (Zimmermann, 2004).

Table 2.8 Prevalence of ID in 2007 and proportion of households with access to iodised salt.

Countries	Insufficient iodine intake (UIC < 100 mcg/l)*			
	School-age children		General population	
	Proportion (%)	Total no. (millions)	Proportion (%)	Total no. (millions)
Africa	40.8	57.7	41.5	312.9
Americas	10.6	11.6	11.0	98.6
Southeast Asia	30.3	73.1	30.0	503.6
Europe	52.4	38.7	52.0	459.7
Eastern Mediterranean	48.8	43.3	47.2	259.3
Western Pacific	22.7	41.6	21.2	374.7
Total worldwide	31.5	266.0	30.6	2,008.8

* Based on population estimates in the year 2006 (Benoist et al., 2008).

In several countries monitored by the WHO, six national surveys have been conducted on both pregnant mothers and school-age children. Five surveys have shown that the median UIC of pregnant women is lower than children and (within these five surveys) three surveys reported pregnant women are also deficient in iodine, with their median UIC being below the cut-off level of 150 mcg/l for pregnant women (Table 2.9) (Benoist et al., 2008). When comparing the two groups within the same country (in these five

surveys) it is shown that, whilst pregnant mothers have low median UIC values, school-age children have adequate (or even more than adequate) dietary iodine intake (based on the median UIC above 100 mcg/l) (Benoist et al., 2008). Hence, although the median UIC of school-age children showed optimal iodine status, the median UIC level in pregnant mothers may indicate a public health problem.

Furthermore, there are also some countries that had no previous problem with ID, but they have recently shown a decreased level of iodine intake amongst the population. This has been demonstrated by a large decrease in the US population median UIC value from 321 mcg/l, during the 1970s, to 160 mcg/l, between 2003 and 2004 (Caldwell, Miller, Wang, Jain, & Jones, 2008). Moreover, Australia and NZ, which used to be iodine sufficient in previous years, have been mildly to moderately iodine deficient since 1990 (Li et al., 2006; Parnell, Scragg, Wilson, Schaaf, & Fitzgerald, 2003; Vannoort & Thomson, 2005). These findings once more emphasise the importance of regularly monitoring the population's iodine status including, the iodine status of pregnant women.

Table 2.9 National survey data for the same year in both pregnant and children.

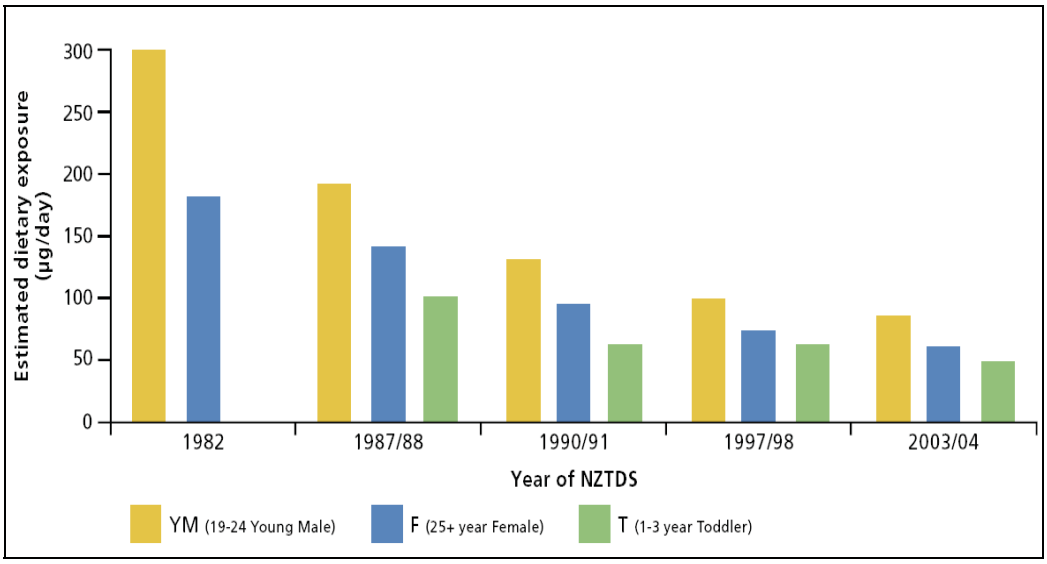
WHO region	Country	Year of survey	Pregnant women		School-age children	
			Median UIC (mcg/l)	Public health problem?	Median UIC (mcg/l)	Public health problem?
Europe	Bulgaria	2003	165	No	198	No
Americas	USA	2001	173	No	249	No
Europe	Switzerland	2004	249	No	141	No
Europe	Romania	2004-05	73	Yes	102	No
Western Pacific	Philippines	2003	142	Yes	201	No
Southeast Asia	Nepal	1997-98	134	Yes	144	No

Source: 'Alfred Rusescu' Institute for Mother and Child Care (2005), Caldwell, Jones and Hollowell (2005), Bulgaria Ministry of Health (2003), Nepal Ministry of Health Ministry of Health (Nepal), UNICEF (Nepal), WHO and The Micronutrient Initiative Ministry of Health (Nepal), UNICEF (Nepal), WHO and The Micronutrient Initiative (1998), Pedro et al., (2003) and Zimmermann, Aeberli, Torresani, and Burgi, (2005).

2.10.2 Re-emergence of iodine deficiency in New Zealand

Iodine intake amongst the NZ population has gradually decreased since the late 1980s. This trend was reported in all age-sex groups of New Zealanders estimated by the five NZ Total Diet Survey (NZTDS) carried out between 1982 and 2003/04. In fact, the NZTDS carried out between 1990/91 and 2003/04 show that iodine intake is predicted to be below the NZ recommendation (Figure 2.3) (Vannoort & Thomson, 2005). This is consistent with the declining of iodine status reported in many studies in NZ, which were generally characterised with a median UIC below 100 mcg/l, as showed in the Table 2.10.

Figure 2.3 Trend of estimated dietary intake (mcg/day) for New Zealanders over five NZTDS from 1982 until 2003/04 (Vannoort & Thomson, 2005).



A pilot study carried out by Thomson et al. (1996) was the first to identify that UIC levels in NZ people (in their study sample) were lower than previous studies in (G. J. Cooper et al., 1984; North & Fraser, 1965; Simpson et al., 1984). This was confirmed by a further study by Thomson et al. (1997), which indicated that the participants (n=333) living in both the North and South Islands (Waikato and Otago areas) of NZ had insufficient iodine intake, with median UIC values of 76 and 60 mcg/day, respectively. A later study, which assessed iodine status in adults living in Otago (n=233), also showed low median 24-hour UIC values in both men (81 mcg/day) and women (69 mcg/day) (C. D. Thomson, Woodruffe et al., 2001). This study also found an inverse association between UIC and thyroid volume and serum Tg levels, when the 24-hour UIC values were categorised into three groups (<60; 60-90; >90 mcg/day), of which the lowest UIC group correlated significantly with greater thyroid volume and

higher Tg levels, and vice versa (C. D. Thomson, Woodruffe et al., 2001). These studies showed that the fall in iodine status had a clinical significance, resulting in a significant increase in thyroid volume and serum Tg levels.

Table 2.10 Median values of UICs in NZ groups.

Population groups / study area	Year	n	Median UIC (ug/l)
Adults			
Otago*	1993-1994	183	42
Waikato/Taranaki*	1993-1994	128	48
Otago*	1997-1998	233	54
Palmerston North[§]	2010	50	65
Pregnant/Lactating women			
Palmerston North**			
Lactating women	2009-2010	24	45
Pregnant women		28	36
Dunedin*	1991-1992	27	38
Dunedin*	2004-2005	109	42
New Zealand*	2005		38
Children			
Dunedin and Wellington*	1996-1997	282	66
South Island*	1998-1999	230	67
New Zealand*	2002	1796	67

Sources : * Thomson & Skeaff, (2009) ** Brough & Jin (2010)[§] Current study

Iodine status was also measured in pregnant and non-pregnant women living in Otago (n=52), where their median UIC values were 38 and 33 mcg/l, respectively, which indicated a moderate ID amongst the study population (C. D. Thomson, Packer et al., 2001). This is consistent with the resultsfrom the latest study, which assessed iodine

status in pregnant (n=24) and lactating women (n=28) living in Palmerston North, NZ (Brough & Jin, 2010). This study reported a low UIC with the median values of 45 mcg/l during pregnancy and 36 mcg/l during lactation, far below the NZ recommendation of 220 mcg/l and 270 mcg/l, respectively. Iodine concentration from breast milk collected from lactating mothers in the study (n=27), was also low (41 mcg/l) compared to the 75 mcg/l minimum concentration that is considered to be adequate (Brough & Jin, 2010).

Assessment of iodine status was also conducted in NZ children in the 2002 National Children's Nutrition Survey. Results indicated that mild ID was found in children aged 5 to 14 years demonstrated by a UIC median of 66 mcg/l (Parnell et al., 2003). Similar results were also found in local studies, indicating mild ID in school-age children with a median UIC of 66 mcg/l (Skeaff et al., 2002), and also in infants and toddlers with a median UIC of 67 mcg/l (Skeaff et al., 2005). Skeaff et al. (2005) also reported that a better iodine status were found in children who consumed iodine fortified infant formula (99 mcg/l), than those who were breast-fed (44 mcg/l). This indicates iodine concentration is low in breast milk and thus, infant formulas are better sources of iodine for children in NZ (Skeaff et al., 2005). A recent study also demonstrated similar results, indicating mild ID in school-aged children with median UIC of 63 mcg/l and serum Tg of 14 mcg/l (Rose et al., 2009). The estimated iodine intake (54 mcg/day) was well below the RDI for children, thus indicating insufficient iodine intake amongst the study samples. However, based on the participants' iodine intake reported in the study, Rose, et al. (2009) estimated that the implementation of iodine-fortified bread could increase the study population's UIC level, from a range of 44-78 to 95-151 mcg/l and therefore, it would be able to achieve optimal iodine status. Overall, studies carried out since the 1990s report insufficient iodine intake and low iodine status amongst all age population groups studied in NZ, thus indicating the re-emergence of mild-to-moderate ID. The mandatory fortification of bread with iodised salt in NZ is one of the main strategies being used to increase iodine intake amongst the population (MOH, 2010).

2.11 Factors contributing to the reduction of iodine intake in NZ

Iodine content in NZ soil is low due to frequent rainfall and snow and glaciation, which leaches out iodine from the soil and carries it to the ocean. This contributes to low levels of iodine in NZ food sources, since crops grown in this the soil have low iodine levels, and animals consuming these plants show low iodine content in their meat and animal products (Aitken, 2001; Mann & Aitken, 2003). Therefore, since people in NZ

consume mostly local plants and animals, they are at risk of not meeting the iodine intake recommendation. Hercus et al. (1925) reported low iodine concentrations in NZ soil, with the lowest amount being approximately 0.3 ppm: this level being the major cause of endemic goitre during the early 1900s. Estimation of the dietary iodine intake of selected foods showed a low intake amongst people in NZ, with the average being approximately 20 mcg/day in people living in goitrous areas and 35 mcg/day in non-goitrous areas (40 mcg/day and 26 mcg/day if fish is included) (Hercus & Roberts, 1927).

Iodised salt was introduced in the 1930s, in order to overcome this problem and the people at that time were also made aware of the importance of using iodised salt. However, this awareness has diminished over the years, due to several possible reasons (Man & Aitken, 2003). Firstly, public health advice about reducing salt intake may have made people more conscious of the problem and therefore, they would have attempted to limit their salt intake and thus reduce their iodine intake (Mann & Aitken, 2003; Seal, Burgess, Taylor, & Doyle, 2007). Studies have also reported that, although iodised salt may be available at home, it is not always used in cooking, or as table salt (Skeaff et al., 2002; C. D. Thomson et al., 1997). Secondly, people are exposed more to food containing non-iodised salt, especially in processed foods, such as pre-prepared or ready-made foods. Frequent eating at fast food outlets or restaurants, or buying take-away food may also contribute to the reduction of iodine intake, since non-iodised salt is mostly used in these places (Aitken, 2001; Mann & Aitken, 2003). The third reason could be the trend of using specialty or mineral salt, such as rock salt, sea salt or garlic and herb salt, which all contain negligible iodine levels (Aitken, 2001; Walrond, 2009). All these factors may have contributed to a decline in the usage of iodised salt at home.

Iodophor, a sanitiser containing iodine, was previously used to clean equipment and containers in the dairy industry in NZ and Australia: an occurrence which accidentally increased iodine intakes. The iodine compounds contained in iodophors were easily dissolved into milk and other dairy products, which increased the iodine content in products and therefore produced adventitious iodine sources in the diet (Li et al., 2001). However, the usage of iodophors has declined (in both countries) and during the 1980s almost all iodophors were replaced with other non-iodised sanitisers in NZ. This resulted in a reduction of the iodine content in dairy products (Cressey, 2003). This change in the type of sanitisers was mainly due to the high variability content of iodine in milk, which predicted that some dairy products would supply excessive iodine

to the population (above 300 mcg/L) (Li et al., 2001). The replacement of non-iodised sanitisers, however, contributed to the reduction of iodine intake amongst the population (Eastman & Li, 2009; Li et al., 2001; Mann & Aitken, 2003; Skeaff et al., 2002). Nevertheless, milk is still considered as being one of the main sources of iodine in NZ and Australia, since it contains moderate levels of iodine (B. M. Thomson, Vannoort, & Haslemore, 2008).

2.12 Selenium and its importance to reduce the risk of ID

2.12.1 Functions of selenium

The thyroid functions at its best when the intake of both iodine and selenium status are sufficient. Selenium is involved in thyroid metabolism: in particular, the selenocysteine-containing enzymes, or selenoproteins, are iodothyronine deiodinases (IDI), which are involved in the deiodination of thyroxine (Germain & Galton, 1997; C. D. Thomson, 2004b; C. D. Thomson et al., 2009; C. D. Thomson & Robinson, 1980). Specifically, Type I and II iodothyronine 5' deiodinases (IDI) are responsible for the conversion of T_4 to T_3, by the removal of iodine at the 5 or 5' position of thyroid hormones (Germain & Galton, 1997; Gropper et al., 2005). Type I deiodinases are mainly found in liver, kidney, pituitary and thyroid gland. The main function of Type I deiodinase is to catalyse the conversion of T_4 in the thyroid gland to T_3, which is then released into circulation (Berry et al., 1991; Gropper et al., 2005). Type II deiodinases are important for activation of T_3 in specific tissues. It is known that T_3 is the active form of thyroid hormone, which is important for metabolic regulation, development and normal growth (Gropper et al., 2005; Triggiani et al., 2009). Regulation of thyroid hormone metabolism is achieved by the deiodination process, whereby T_3 is further converted to T_2, and T_4 can be converted to reverse T_3; both these two forms (T_2 and reverse T_3) are inactivate metabolites (Gropper et al., 2005). In general, if selenium is insufficient, it can affect thyroid hormones levels in the body by significantly increasing serum T_4 levels, but it may not affect serum T_3 and TSH levels (Germain & Galton, 1997; Gibson, 2005; Moreno-Reyes, Victor, Gerard, & Ronald, 2009; Triggiani et al., 2009).

The main role of selenium is in the selenoproteins and glutathione peroxidase (GPx), which plays an important role as an antioxidant enzyme that performs oxidation-reduction reactions in the body (C. D. Thomson & Robinson, 1980; Triggiani et al., 2009; WHO & FAO, 2004a). Selenium acts as an essential cofactor for the enzyme glutathione peroxidase; all GPxs (GPx-1 to -4) are selenium dependent, containing four selenocysteine residues. These GPxs has been characterised in four forms in the body: classical or cellular GPx (GPx-1); gastrointestinal GPx (GPx-2); extracellular or

plasma GPx (GPx-3); and phospholipid hydroperoxide GPx (GPx-4) (Gibson, 2005; Higdon, 2003). All these forms of GPx have the same basic function as an antioxidant, but they react in different tissues in the body. The mechanism of GPx, as an antioxidant enzyme is achieved by catalysing the coupling of the reduced glutathione (GSH) with reactive oxygen species (ROS) such as hydrogen peroxide. This process helps to reduce the oxidative damage of ROS, by converting it to harmless products such as alcohol and water (Gibson, 2005; Higdon, 2003). This helps to protect the oxidative damage in many parts of the body including the thyroid gland (Eastman & Zimmermann, 2009; C. D. Thomson et al., 2009). Therefore, selenium deficiency may diminish the GSH peroxidase in the body, which also contributes to the accumulation of hydrogen peroxide in the thyroid gland: that may eventually lead to thyroid cell death (Contempre, de Escobar, Denef, Dumont, & Many, 2004).

2.12.2 The influence of diet on selenium status

Similar to iodine, the selenium content in food is highly variable due to the variability of selenium concentrations in the soil, depending on geographical locations throughout the entire world (Contempre et al., 1992; Gropper et al., 2005). Thus, low levels of selenium in the soil may affect the entire food system in particular, since both plant and animal feed will also be low in selenium. This situation will affect the selenium intake of people living in the affected areas (Contempre et al., 1992). However, it is difficult to determine selenium deficiency unless it is severe, since mild to moderate selenium deficiency is not significantly associated with any clinical conditions (Moreno-Reyes et al., 2009). However, severe deficiency of selenium has been associated with Kashin-Beck disease in China and Tibet, and myxedematous cretinism in Central Africa, which also have endemic ID (Contempre et al., 2004; Moreno-Reyes et al., 1998). In addition, the effect of selenium deficiency has been found in animal farms, such as sheep and cattle farms, in countries such as NZ and Finland. For example, myopathies shown in farm animals are characterised by white muscle, muscular dystrophy and stiff lamb disease (Andrews, Hartley, & Grant, 1968; Aro, Alfthan, & Varo, 1995; C. D. Thomson & Robinson, 1980). Therefore, the supplementation of fertilisers and use of animal feed containing selenium is carried out in these countries in order to improve the diet of the population (Aro et al., 1995; C. D. Thomson et al., 2009; C. D. Thomson & Robinson, 1980). Good food sources of selenium such as organ meats (mainly kidney and liver) and sea fish (including other seafood) and Brazil nuts, are recommended to be included in the diet, especially for populations living in selenium deficiency areas (C. D. Thomson, 2004b; C. D. Thomson et al., 2008).

2.12.3 Selenium intake requirement

As mentioned previously, selenium content is highly variable in the soil (depending on geographical location) and therefore its content also varies in the normal adult, ranging from about 3 mg in people living in NZ, to 14 mg in many people in the United States (US) (WHO & FAO, 2004a). Populations in certain areas in China and Tibet that were severely deficient in selenium, were found to have a higher incidence of Keshan disease, the selenium-responsive cardiomyopathy (Moreno-Reyes et al., 1998; WHO/FAO/IAEA, 1996). Studies in China found that Keshan disease mostly occurred in areas where a population's mean intake of selenium was below 20 mcg/day (Yang, Ge, Chen, & Chen, 1988; Yang & Xia, 1995). Therefore, it has generally been accepted that 20 mcg/day of selenium is the minimal intake requirement to prevent Keshan disease (Levander, 1997; C. D. Thomson, 2004a).

Nevertheless, the recommendation for selenium intake is higher in order to optimise the requirements for selenoprotein IDI and GPx: at least 30 mcg/day and 45 mcg/day, respectively (C. D. Thomson, 2004a; WHO/FAO/IAEA, 1996). Thus, the WHO recommends a selenium intake of 40 mcg/day for men and 30 mcg/day for women (C. D. Thomson, 2004a; WHO/FAO/IAEA, 1996). The selenium intake recommendation for adult varies between countries (as seen in Table 2.11) but all recommendations are higher than 30 mcg/day, which is the essential level needed to satisfy requirements for selenoproteins.

The recommended selenium intake in many countries, was generally based on studies which investigated selenium intakes needed to achieve elevated levels of plasma selenoproteins concentrations, which are required for maximal GPx activities (Duffield, Thomson, Hill, & Williams, 1999; Gropper et al., 2005; C. D. Thomson, 2004a). In 2000, the Food and Nutrition Board (FNB) set the recommended dietary allowance (RDA) for selenium intake, to 55 mcg/day for men and women, whilst the upper limit is 400 mcg/day (FNB & IOM, 2000). The RDI for selenium in NZ is higher, at 60 mcg/day for women and 70 mcg/day for men (Australian National Health and Medical Research Council & New Zealand Ministry of Health, 2006). Australia and the UK's recommendation for selenium intake is slightly higher than the NZ recommendation (Table 2.11).

Table 2.11 Estimates of requirements for selenium and recommended selenium intake in various countries.

Estimates requirements for selenium intake and recommended intakes of selenium	Selenium intake (mcg/day)
Minimum requirement for prevention of Keshan disease	20
Physiological requirement (EAR) for maximal GPx and selenoprotein P (the most common selenoprotein)	45-50
Requirement for IDIs	30
Protection against some cancers	120
Recommended intakes of selenium according to the WHO (normative requirement)	Men: 40 Women: 30
RDA for selenium in the US and Canada (RDA: recommended dietary allowance)	Men: 55 Women: 55
RDI for selenium in NZ (RDI: recommended dietary intake)	Men: 70 Women: 60
RNI for selenium in the UK (RNI: reference nutrient intake)	Men: 75 Women: 60
PRI for selenium in Europe (PRI: population reference intake)	Men: 55 Women: 55
RNI for selenium in Australia	Men: 85 Women: 70

Source: adapted from Thompson (2004a) and WHO and FAO (2004a).

2.12.4 Assessment of selenium status

Blood selenium concentration has been accepted as a useful biomarker of selenium status and intake (Arthur, 1999; Ashton et al., 2009). However, other tissues, such as nails or hair and urine excretion are also used to access selenium status (Gibson, 2005; C. D. Thomson, 2004a). Daily urinary excretion is the preferred biochemical test, when determining selenium status based on recent dietary intake, especially in populations that live in selenium-deficient areas (Sanz Alaejos & Diaz Romero, 1993; C. D. Thomson, 2004a). Studies have reported that 50% to 60% of dietary intake is closely related to daily urine excretion: therefore, the selenium intake can be estimated as twice the daily urinary excretion concentration value (Gibson, 2005; Sanz Alaejos & Diaz Romero, 1993; C. D. Thomson, 2004a).

The 24-hour urine sample is preferable than a single urine collection, because selenium excretion is affected by time of day, dilution and selenium content of the previous food intake (Gibson, 2005; C. D. Thomson & Robinson, 1980). The urinary selenium level is generally expressed as a concentration (mcg/l) or excretion (mcg/day), especially if the sample is collected over 24-hours (Gibson, 2005; Sanz Alaejos & Diaz Romero, 1993). The daily urinary excretion of selenium is varied and based on certain parameters, such as age and sex. Elderly people have shown a low selenium daily excretion, compared to young adults (Robberecht & Deelstra, 1984; Sanz Alaejos & Diaz Romero, 1993), whilst females have shown lower selenium excretion than males, which these could be due to differences in dietary intake (Sanz Alaejos & Diaz Romero, 1993; C. D. Thomson et al., 1996). Amongst women, pregnant mothers are more likely to have a lower selenium urinary excretion, than non-pregnant women (Sanz Alaejos & Diaz Romero, 1993; Swanson, Reamer, Veillon, King, & Levander, 1983). Studies have suggested that the relationship between different ages and gender, in relation to selenium excretion, may be associated with dissimilarity in muscle mass (Oster & Prellwitz, 1990).

2.13 Action of correction for ID

The WHO/UNICEF/ICCIDD (2007) recommend that any affected countries should have their own national body to implement a specific IDD control programme; in order to correct IDD and reduce the risk of ID in their populations. They also proposed a process model as a guide to operate a nationwide IDD control programme, which comprised of six components: 1) assessment; 2) dissemination of findings; 3) planning; 4) achieving political will; 5) implementation; and 6) monitoring and evaluation.

The first component, ‘assessment’, involves surveys on prevalence of ID and iodised salt usage or intake, and also the measurement of IDD. The second component suggests that the findings should be revealed to the public, showing the status of iodine or the risk of ID in a population. As a result, this may increase the awareness of using iodised salt and increasing iodine intake among population. The third component is the development of intersectoral task in developing strategies for the elimination of ID. The fourth component suggests that the government should create an awareness of its IDD control programme, through education and other political methods. The fifth component involves cooperation from many parties including the government, industries, health professional bodies, non-government organisations and also public

sector in running health interventions of IDD control programs. The intervention programmes such as salt iodisation, iodine supplementation and food fortification is targeted to increase iodine intake, reduce the risk of IDD and improve overall iodine status of the population. These intervention programs require monitoring and evaluation plan (sixth component) in order to ensure that the programs are well-established and run smoothly at all levels. Most importantly, it is necessary to ensure this implementation programme is able to achieve the target of elimination of ID, and can be sustained for future needs (Darnton-Hill, 1998; WHO/UNICEF/ICCIDD, 2007)

2.13.1 Iodisation of salt

The most popular way to reduce ID, eliminate IDD and improve the iodine status of a population, is universal salt iodisation (USI), which has been introduced in both developing and developed countries around the world (Lotfi, Venkatesh Mannar, Merx, & Naber-van den Heuvel, 1996). The low cost method of USI is sustainable in supplying iodine to the population, since it can reach more than 90% of households and it can reach most locations, including remote areas (Benoist et al., 2009). The WHO and UNICEF consider this method to be safe, economical and effectual and it ensures that every individual in a population is able to achieve an adequate intake of iodine (WHO/UNICEF/ICCIDD, 2007). The USI is not only for human consumption but also for farm animals, and therefore almost all food products will contain a certain level of iodine, depending on the iodine requirements of a population (WHO/UNICEF/ICCIDD, 2007).

Iodine is usually added to salt in the form of potassium iodate (KIO_3) or potassium iodide (KI), within the recommended range of 20 to 40 mg of iodine/per 1 kg salt (WHO/UNICEF/ICCIDD (2007). In tropical countries, the KIO_3 form is preferred since it is more stable at high humidity (Zimmermann, 2009a). The iodine level for USI should supply sufficient iodine depending on the requirement of the population, after consideration of potential losses due to food processing and/or storage conditions, in addition to the estimated shelf life of salt (Eastman & Zimmermann, 2009; Hetzel, 2005). In general, salt consumption in developed countries is considered high, at approximately 10-15 g/day, which may increase the risk of hypertension. Hence, public health advice has recommended that people restrict their salt intake to 3-6 g/day or less. However, this has reduced the iodine intake of populations in areas where ID is a public health problem (Eastman & Zimmermann, 2009; Seal, Burgess et al., 2007). Furthermore, the introduction of mineral or flavoured salt, such as sea salt, rock salt, garlic salt and herbs salt, in addition to an increase in the intake of processed foods,

has reduced the usage of iodised salt in countries such as NZ and Australia, and hence there has been a reduction in iodine intake, since these foods contain negligible levels of iodine.

2.13.2 Iodised oil

Iodised oil supplementation, administered either by intramuscular injections or orally, is accepted to be the best method to correct severe IDD when other alternative methods are not effective or appropriate (Hetzel, 2005; Tonglet, Bourdoux, Minga, & Ermans, 1992). The efficacy of iodised oil treatment (by injection) in reducing the goitre rate in a population can usually be shown (at the earliest) at one month or up to three-months, post injection (Buttfield & Hetzel, 1967; Eastman & Zimmermann, 2009). Injection of iodised oil was first used in Papua New Guinea, when the population was severely affected by ID and the treatment showed great effectiveness in treating goitre and correcting ID within three to four years (Caulfield, Richard, Rivera, Musgrove, & Black, 2006; Horton, 2006). Studies and reviews have also reported that injecting iodised oil has been used extensively to correct ID in many populations and to prevent endemic cretinism, with very few side effects caused by the injection (Buttfield & Hetzel, 1967; Delange, 1996; Eltom, Karlsson, Kamal, Bostrom, & Dahlberg, 1985; Furnee, 1997; Wachter et al., 1985). However, this method is much more expensive than others, due to the cost of needles and syringes and also the need for well-trained personnel to administer the injection (Eastman & Zimmermann, 2009; Hetzel, 2005). Alternatively, iodised oil can also be given orally, instead of by injection. However, the downside of this route is that the efficacy rate is much lower and the duration of its effect is reduced (Buttfield & Hetzel, 1967; Leverge, Bergmann, Simoneau, Tillet, & Bonnemain, 2003; Wolff, 2001). This is due to the lack absorption in the gut, since iodised oil is a lipophilic agent (Jeong et al., 2001). This is because, solubility of lipophilic agent is not sufficiently capable of crossing the water layer adjacent to the epithelial cell, which may result in low bioavailability (Holford, 2009).

2.13.3 Iodine fortification

Processed food, particularly staple food, can be an effective vehicle in supplying iodine to a population. Thus, it is important to consider the acceptability of the fortified food to the population, in addition to the ability of ensuring the food reaches remote areas, before the implementation of fortification. Moreover, the stability of iodine in food during processing, storage and packaging needs to be considered, to ensure that the end-users will be able to get the required amount of iodine based on the amount that has been added to the food (Lotfi et al., 1996; Mehra, Srinivasan, Victor, Gerard, & Ronald, 2009). However, it is also important to ensure that no unfavourable effects

occur to the food, such as the iodine altering the physical properties or sensory quality of the food: the originality of the food must be retained. The food vehicle used worldwide is either in liquid form, such as water, milk or sauce, or a solid, such rice, seasoning powders, flour, beverage powders, sugar, cereals, biscuits or bread (Lotfi et al., 1996; Mehra et al., 2009). Potassium iodide (KI) is the most common form of iodine usually used for fortification in commercial food and beverages products, in addition to it being generally available for use in the food industry due to its good solubility (Mehra et al., 2009).

Many iodine-fortified food products have been commercialized and some products have been targeted at certain groups of people with different ages and/or physiological conditions (Mehra et al., 2009). Thailand has implemented its iodine fortification into food products that are primarily being consumed by Thai people, such as seasoning powder for instant noodles, fish sauce and parboiled milled brown rice (Chavasit, Nopburabutr, & Kongkachuichai, 2003; Chavasit & Tontisirin, 1998; Tulyathan, Laokuldilok, & Jongkaewwattana, 2007). Iodine in seasoning powder and fish sauce has been reported to be stable during food processing and storage. Furthermore, it does not have any unfavourable effects on the physical properties and sensory quality of these foods after they have been cooked (Chavasit et al., 2003). However, in the case of par-boiled milled rice, there is significant reduction in iodine content as the time of storage increase (Tulyathan et al., 2007). Foods have also been fortified with iodine together with other micronutrients, mainly iron (Mehra et al., 2009). The National Institute of Nutrition in India reported that there was no adverse effect on the iodine content or its food vehicle, when it is co-fortified with iron in sugar over a twelve month period; and there were no significant differences in the physical properties of the sugar compared to the non-fortified sugar (National Nutrition of Institute, 2003-2004). Studies also have found that the fortification of multiple micronutrients (including iodine) into foods that were targeted for children, such as biscuits, cereals and/or beverages (either in liquid or powdered form), have shown an increase in UIC levels, in addition to positive effects in improving nutritional status, anthropometric measures and cognitive performances were also improved, especially in school children (Abrams et al., 2003; Latham et al., 2003; Solon et al., 2003; Stuijvenberg et al., 1999; Zimmermann, Wegmueller et al., 2004; Zimmermann, Zeder et al., 2003).

Mandatory fortification of iodine in processed food has been undertaken in several countries (including NZ) and it is targeted to overcome the problem of dietary inadequacy within population. As mentioned before, in NZ, the addition of iodised salt

is compulsory in all types of bread, (except organic, par-baked and unleavened bread), although some types of bread are additionally voluntarily fortified with folic acid (MOH, 2010e; NZFSA, 2009). This implementation of iodised salt in NZ is to prevent the IDD, as ID is re-emerging in the country. The amount of iodised salt required in bread is in the range of 25 to 65 mg/kg, and it is targeted to increase the overall intake of iodine within the population (NZFSA, 2009). Nevertheless, it is predicted that it will not to be sufficient for pregnant and breastfeeding women, since they have high requirements for iodine. However, the iodine content in bread could not be increased further, since it was suggested that it might increase the risk of toxicity in children (MOH, 2010e). Mandatory or voluntary iodine fortification in manufactured foods (for example bakery goods, including bread) was implemented earlier in several areas or countries, such as Tasmania in Australia, Denmark, the Netherlands and certain areas in Russia. The populations in these areas/countries have shown a significant increase in their median UIC level, post-iodine fortification (Table 2.12) (Gerasimov et al., 1997; Seal, Doyle, Burgess, Taylor, & Cameron, 2007).

Table 2.12 Iodine fortification of commercial foods in various countries and the effect on the population of the iodine excretion or iodine intake.

Country/ Place	Food vehicles	Urinary iodine concentration (mcg/l or mcgmol/g cr) / iodine intake (mcg/day)		Sources
		Prior-fortification	Post-fortification	
Denmark	Salt and bakery goods	61 mcg/l	101 mcg/l	(Rasmussen et al., 2008)
Tasmania, Australia	Bread	72-75 mcg/l	105-109 mcg/l	(Seal, Doyle et al., 2007)
Russia	Bread	48 mcg/l	126 mcg/l	(Gerasimov et al., 1997)
Netherlands	Bread	<100 mcg/day	>100 mcg/day	(Brussaard et al., 1995)
Northeast Thailand	Fish sauce	3.3 mcgmol/g cr	1339 mcgmol/g cr	(Pongpaew et al., 2002)

cr = creatinine

The success of a mass fortification programme, especially mandatory fortification, is largely dependent on the acceptability of the public or consumers, in addition to the food industry (Darnton-Hill, 1998; Mehra et al., 2009). Therefore, a suitable food vehicle must be chosen to ensure the food is commonly consumed by the majority of

the people or target population, in addition to there being no large increases in the price of the fortified food. Correct information regarding the benefits of fortification should also be provided to the population in order to ensure that everyone understands the importance of an adequate dietary intake, and to avoid any misunderstanding regarding the fortification (Darnton-Hill, 1998; Lotfi et al., 1996). Mandatory iodine fortification however, has been associated with an increase in the incidence of thyroid disease and hyperthyroidism (especially in young people) and this occurrence is presumably of autoimmune origin (Bulow Pedersen et al., 2006; Laurberg et al., 2006). This is because a high iodine intake (if above recommendation) over a period of time, may increase the precipitation of Graves's disease: and the risk increases threefold in women during the postpartum period (Jameson & Weetman, 2008). Therefore, it is recommended that iodine is consumed based on the recommendation, where the intake amount is confirmed to be sufficient in preventing ID (Laurberg, Pedersen, Vestergaard, & Sigurdsson, 1991).

2.13.4 Iodine supplementation

Access to iodine through food fortification or salt iodisation may still be insufficient for certain susceptible groups of people, mainly pregnant and breastfeeding mothers and non-consumers of the iodised food (WHO/UNICEF/ICCIDD, 2007). Thus, iodine supplementation may be an efficient and effective alternative, which provides adequate iodine to pregnant and breastfeeding mothers and their fetus or infant (Berbel, Obregun, Bernal, Rey, & Escobar, 2007; Zimmermann, 2009b; Zimmermann & Delange, 2004). This is characterised by the increased level of urinary iodine in iodine-supplemented women, which have been demonstrated in randomised, controlled trials, involving pregnant mothers with mild to moderate ID, as shown in Table 2.13. Iodine supplementation in these studies (Table 2.13) has show that supplementation in pregnancy is considered safe, since no side effects were reported, and furthermore, there was no increment of maternal thyroid autoimmunity reported (Zimmermann & Delange, 2004).

In adults, an overload of iodine circulation can easily be mediated in the body, by the presence of Na+/I- symporters located at the basolateral membrane of thyroid follicular cells. As iodine increases in the circulation, it will suppress the expression of the symporter and hence, it will reduce the iodine intake (Jameson & Weetman, 2008). This auto-regulatory mechanism is automatically activated once iodine levels are exceeded (Berbel et al., 2007). Hence, the recommended dose of 200 to 250 mcg/day

of iodine supplement is considered safe for pregnant and breastfeeding mothers, as well as for women who are considering conception (Berbel et al., 2007; WHO/UNICEF/ICCIDD, 2007). Moreover, the safe upper level of iodine (1100 mcg/day) is very high and this is difficult to achieve even if women of childbearing-age and pregnant or breastfeeding mothers consume iodine-rich food, together with supplementation, for a long duration throughout their pregnancy and lactation (Berbel et al., 2007). In addition, there is also no clinical data (to date) that reports any adverse effects of iodine supplementation in women and/or their infants (Zimmermann & Delange, 2004). Therefore, pregnant or breastfeeding women living in insufficient areas of iodine are recommended to take iodine supplements throughout gestation and whilst they are breastfeeding.

Table 2.13 Iodine supplementation for pregnant women characterised by median urinary iodine levels.

n	Iodine dose (mcg/day)	Length of administration	Median urinary iodine		Source
			Pre-treatment	Post-treatment	
67	50	18 to 26 weeks	65 mcg/g cr	128 mcg/g cr	(Antonangeli et al., 2002)
	200	29 to 33 weeks	91 mcg/g cr	230 mcg/g cr	
120	100	14 weeks to term	36 mcg/l	80-90 mcg/l	(Glinoer et al., 1995)
35	120-180	1st trimester to term	37 mcg/day	100 mcg/day	(Romano et al., 1991)
66	150	11 weeks to term	50 mcg/l	105 mcg/l	(Nohr & Laurberg, 2000)
54	200	17 weeks to term	55 mcg/l	90-110 mcg/l	(Pedersen et al., 1993)
108	230	11 weeks to term	53 mcg/g cr	104 mcg/g cr	(Liesenkotter et al., 1996)

n = sample size, cr= creatinine

There are also several disadvantages to supplementation. The appropriate level of iodine (in the supplement tablet/capsule or the liquid form) should be monitored, since levels can vary in commercial preparations (Untoro, Timmer, & Schultink, 2010). The quality of the iodine content within the end product of the supplement, could be

reduced by instability during processing; the time which had elapsed since manufacturing; and the storage conditions of the supplements (Untoro et al., 2010; Zimmermann & Delange, 2004). Thus, processing and storage must be carried out properly and monitored vigorously, in order to sustain the optimal iodine bioavailibility of the products (Zimmermann & Delange, 2004). On the other hand, sea kelp and other seaweed based products are the supplemental form of iodine that is not highly recommended, especially for pregnant mothers, since the iodine content is extremely variable (it may contain more than 1000 mcg of iodine per tablet/capsule) (Zimmermann & Delange, 2004). Therefore, pregnant or lactating mothers are recommended to seek professional advice on iodine supplementation or individual dietary iodine requirements during pregnancy and lactation, especially those women with a known thyroid disease. This is because, during the perinatal period, excessive exposure to iodine may lead to transient hypothyroidism in the newborn (Nishiyama et al., 2004). Moreover, an excessive iodine intake of more than 300 mg/day (over a period of time), especially in people with a known thyroid disease, may lead to subclinical hypothyroidism and/or autoimmune thyroiditis (Teng et al., 2006).

3 MATERIALS AND METHODS

3.1 Study design

This study is a cross-sectional designand it includes quantitative and qualitative data that has been collected through a questionnaire and 24-hour recall. The questionnaire and 24-hour recall were administered in the form of an interview. Following the interview, each participant was asked to collect a sample of her urine over a 24-hour period, for analysis of the urinary iodine and selenium concentration levels.

3.2 Study sample

Fifty healthy women, aged 18 to 40 years living in Palmerston North, NZ were recruited. Pregnant, breastfeeding or women who had thyroid disease (or those taking medication containing iodine) were excluded. The mandatory fortification of bread with iodised salt in NZ should increase the UIC level by 20mcg/day in adult women (Rose, Gordon, & Skeaff, 2009). A sample size calculation showed that 34 women would be required, in order to show this difference. However, 50 participants were selected, in order to accommodate any attrition.

3.3 Recruitment of subjects

Advertisements (Appendix 1) were placed at health care, sports and community centres and three tertiary institutions: Massey University, University College of Learning and International Pacific College. Flyers (Appendix 2) were distributed by hand in Palmerston North, primarily at Massey University. An advertisement was published in the local media, through the Massey University advertisement website and CHAFF (the campus newspaper). An advertisement was also circulated in an email to students and staff of Massey University. In order to avoid bias, the information in the advertisement and flyer did not specifically mention that the major focus of the study concerned iodine. This was to prevent participants being influenced by the information and possibly changing their dietary habits, which could ultimately alter the study results. People interested in participating were given an information sheet (Appendix 3), prior to the study. They were then contacted for screening, by phone or email (Appendix 4). During this process, the researcher also checked the participants'

understanding of the information sheet and ensured that they understood their individual details and responses would remain confidential.

3.4 Ethics

The project (Application 10/03) has been approved by the Massey University Human Ethics Committee (MUHEC), 2010. Following ethical procedures, all participants were informed that all data would be kept in a locked filling cabinet for five years: and after this time, the data would be transferred to an official secure archive (Crown Records Management) and then destroyed after 10 years, with permission from the research supervisor. Electronic data would be stored on the researcher's personal computer and it would be password protected. Prior to the interviews, the participants signed a consent form (Appendix 5). After the interview (and once the urine sample had been collected) all participants were also given an information sheet (Appendix 9) relating to good food sources of iodine in NZ.

3.5 Development of questionnaire

A questionnaire (Appendix 6) and a 24-hour recall (Appendix 7) were used to collect data and information from participants. The questionnaire consisted of a mixture of closed and open ended questions and it was divided into six parts: A) salt usage and meal preparation; B) knowledge about and awareness of iodine deficiency; C) dietary intake and consumption of iodine-rich food; D) fish and seafood intake; E) bread intake; and F) demographic background. The questionnaire was mainly developed from the validated questionnaire used in a previous study carried out in Palmerston North, but with a different group of respondents: pregnant and lactating women (Brough & Jin, 2010). Therefore, some of the questions were modified to suit the sample of this study. Some questions were also taken from previous studies, namely, a study on knowledge of iodine nutrition (Jooste, Upson, & Charlton, 2005) for Part B; and the NZ National Nutrition survey 1996 (MOH, 1999) and Canadian First Nations survey (Sharma et al., 2008) for Part E.

3.5.1 Part A : Dietary pattern, salt usage and exposure to non-iodised salts

The first question in this part (Q1) investigated the dietary pattern of the participants, mainly to find out if there were certain foods that the participants avoided, especially the main food sources of iodine: fish, shellfish/seafood, eggs and dairy products. Moreover, this question was also aimed at identifying if there were any vegan or

vegetarian participants. The remainder of the questions in Part A (Q2 to Q12) was used to identify salt usage and the exposure of the participants to non-iodised salt. The questions on regular salt usage (Q2, Q3) asked about the general use of salt, by determining the frequency of consumption. Questions about the purchase and usage of salts (Q8 to Q10) were aimed at identifying the types of salt being used in the home and how they were used. Meal preparation questions (Q4-Q5) determined whether the women themselves regularly prepared their family's meals, in order to clarify if they knew how salt was being used at home in cooking. Questions asking about the frequency of eating out, eating take-away meals and the use of pre-prepared or ready-made food, as part of meal preparation (Q5-Q7), were to gain information on the level of the participants' exposure to non-iodised salt. The final two questions (Q11-Q12), in this part of the questionnaire, were to find out whether participants attempted to limit their salt intake and their reasons for doing so, in addition to what they thought about their overall salt usage level.

3.5.2 Part B: Knowledge and perception of iodine nutrition

This part of the questionnaire explored the participants' knowledge of iodine nutrition and the issue of iodine deficiency in NZ. The first question (Q13) assessed the participants' awareness of iodine deficiency in NZ and how to limit the problem. Q14 and Q15 tested the participants' knowledge about good food sources of iodine in NZ, and which part of the body needs iodine to produce hormones. Q16 investigated the awareness of participants regarding the fortification of iodised salt and folic acid being added to bread in NZ. After answering Q16, the participants were informed about the current status of iodine and folic acid fortification of bread in NZ. They were then asked (Q17-Q19) to give their opinion regarding mandatory iodine fortification, and their reactions to this fortification. The final question (Q20), in this part, asked about the participants' awareness of which groups of people need a high iodine intake, which could result in a high risk of ID.

3.5.3 Part C: Dietary iodine intakes

Part C asked about the frequency of the participants' overall intake of main food sources of iodine, other than fish and seafood and bread. A brief semi-quantitative food frequency questionnaire (FFQ) was used to collect data on the respondents' dietary iodine intake (Q21). Participants needed to describe the portion size and frequency of their intake for each food item. Frequency categories ranged from *never, less than one per month, 1-3 times per month, once a week* and *every day*, to *more than once per day*. There were only 14 food items on the list, since iodine is the nutrient of interest for estimation. Questions in this part of the questionnaire were also

intentionally placed after part B, in order to avoid bias from previous questions, particularly from question 14 in part B, which asked about good food sources of iodine. This is because some earlier questions could have an influence on later responses and the participants might 'pick up' answers from the questionnaire. Therefore, strategically placing the order of questions can help to obtain respondents' unbiased thoughts, instead of hinting or directing them towards a particular response (Clark & Schober, 1992). The final question in this part asked about the participants' general use of supplements (Q22). This question aimed to identify whether the participants were taking iodine supplements or sea kelp. It also identified if other nutrient(s) were being taken, and the reason/s for taking them.

3.5.4 Part D: Fish and seafood intake

Seafish and seafood are the most potent natural sources of iodine. Therefore, part D (Q23 – Q24) was used separately, in order to estimate the iodine intake from these particular sources, in more detail. A similar semi-quantitative FFQ was used in this part, where the participants were required to identify the type/s of fish or seafood that they usually consumed. Food items comprising fish and seafood were divided into categories: fresh, canned, frozen, processed and take-away meals. Next, the participants were asked questions relating to their awareness regarding guidelines for fish and seafood intake during pregnancy (Q25). Then, the participants were asked for their opinion on whether they would change their fish and/or seafood intake in their diet, if they were pregnant; and how they would do this (Q26 - Q27).

3.5.5 Part E: Bread intake

Almost all bread in NZ has been fortified with iodine (as described in the literature review, part 2.1). Thus, questions in part E were used to identify the frequency and types of bread intake amongst the participants. The first table (Q28) in this part determined the frequency of bread intake that is excluded from the mandatory iodine fortification. Another table (Q29) (a similar type of semi-quantitative FFQ) was used to assess the intake of other breads, which are available on the market; and which must contain iodised salt.

3.5.6 Part F: Demographic background

Part D concentrated on the participants' demographic backgrounds (Q30 - Q38) and questions were asked about age, living situation, ethnicity, education level and current or most recent occupation, in addition to smoking status and exposure to smoke.

3.5.7 Part G: 24-hour recall form

A 24-hour diet recall was used in Part G, in order to obtain a clear snapshot of each respondent's diet during the previous 24 hours. The main purpose was to analyse the total selenium intake from each respondent's diet during the previous day. In this part, a detailed description of food, such as type, brand, cooking method, portion size and other measures were used to help respondents recall what they had consumed during the previous day.

3.6 Pre-testing questionnaire

The pre-test questionnaire was completed by 10 volunteers through an interview that allowed feedback. The time taken was recorded and comments from respondents led to changes, mainly in terms of the language and structure of the questionnaire. Although the questionnaire had been validated in previous studies, it had again been tested, since some changes had been made, to ensure its suitability for this study. Moreover, pre-testing the questionnaire could increase the validity and the reliability of the questionnaire itself (Marshall, 2005; Williams, 2003).

3.7 Data collection

3.7.1 Administration of questionnaire

The interviews were performed at the Human Nutrition Research Unit (HNRU) at the Riddet Building, Massey University and each interview took approximately 40 to 60 minutes to complete. For Part C to E, and also the 24-hour recall interview (Part G), participants needed to describe their dietary intake and estimate their portion size. This was done by asking participants to select photos from a food atlas book titled, *Food portion sizes: a user's guide to the photographic atlas* (Nelson, Atkinson, & Meyer, 1997), which contains coloured photographs of various foods with varying portion sizes. Household food measurement utensils, such as cups, spoons and plates were used to demonstrate the actual size of servings. Different sizes of fish cans were also displayed during the interview, to help participants estimate their intake of fish (particularly for the semi-quantitative FFQ questions in part D). All these factors were important in helping the respondents to give correct measurements for their food serving sizes and correct information regarding all food items they had eaten.

3.7.2 24-hour recall interview

In order to obtain an accurate description of the participants' food intake, each interview was conducted by following the three-sweep method, as follows:

i) Firstly, the interviewer asked the participants what they had eaten and drank during the previous 24 hours. At this stage, a quick list of all food and drinks was recorded.

ii) The interviewer then returned to the food list and asked for detailed information about the food and drinks, such as type, brand, cooking method, meal preparation and whether they has been eaten in combination with other foods, or whether anything else had been added to the food or drinks. Information about when and where the food and drink was consumed was also recorded.

iii) The interviewer then ensured that the food record was complete, by reading it back to the respondent, in order to check for omissions. Any missing food or drink (or further detailed information) was added at this stage, if necessary.

3.7.3 24-hour urinary iodine concentration

At the end of the interview session, the participants were asked to collect a 24-hour urine sample. They were provided with a insulated box containing two urine bottles and ice or frozen silica pads, together with instructions (Appendix 8) on how to collect the urine at home. Participants followed these steps for urine collection:

i) On the first collection day, the participants wrote down the time they passed their first urine in the morning. However, this first urine specimen was not collected.

ii) The urine was collected starting from the second time they passed the urine on the same day, for the remainder of that day and night. Urine also needed to be collected when the participants emptied their bowels.

iii) The last specimen was collected on the following day, at the first time they passed urine in the morning. The time was recorded as the finish time of their urine collection.

Following the urine collection procedure, all the participants stored their urine samples in the supplied chilly bin, which contained ice or frozen silica pads. When the participants had completed their urine collection over 24 hours, the researcher collected the urine samples from the participants' homes and brought them immediately to the Human Nutrition Unit laboratory for processing.

3.7.4 Processing the urine samples

When processing the urine samples, the entire amount of samples, from the two urine bottles returned by each participant, were mixed together into a five litre measuring jug. The total volume was then measured and recorded in milliliters (ml). Next, approximately 15 to 20ml of urine from the measuring jug was transferred into 10 sample tubes, each of which was labeled with the participants' identifier code. Urine samples were then stored in batches, at -20 degrees C in a freezer, until analysis. All urine samples were sent in a batch to the laboratory (Hill Laboratories) in Hamilton, NZ, for iodine and selenium analysis using inductive-coupled plasma mass spectrometry (ICP-MS). The ICP-MS is the most accurate method for urinary iodine analysis and it has been recognised as the 'gold standard' technique to measure iodide concentration (Jooste & Strydom, 2010; Vanderpas, 2006).

3.8 Data Handling

3.8.1 Data from questionnaire

Quantitative data from the questionnaires were simplified into understandable components and these were entered (as codes) into SPSS data files (Statistics Package for the Social Sciences). The qualitative data were recorded into an Excel datasheet and were categorised according to the point of information. If possible, these data were then converted into numerical data and were entered (as codes) into SPSS data files.

3.8.2 24-hour recall

Data from the 24-hour recalls were entered into the food record database, FoodWorks Professional Package Version 5 for Windows (FoodWorks 2007, Australia). The nutrient content of the food consumed by each participant was analysed by FoodWorks according to the New Zealand – Standard database. Daily selenium intakes were determined by FoodWorks and then entered into SPSS for statistical analysis.

3.8.3 Semi-quantitative FFQ

The data obtained from the semi-quantitative FFQ in the questionnaire were compiled into an Excel datasheet, in order to calculate the iodine contribution from each food and the total iodine intake from each participant, before entering it into SPSS for statistical analysis. These calculations were based on the frequency of consumption, the participants' usual portion size and the iodine content in each food item (Mina, Fritschi, & Knuiman, 2007; Rasmussen et al., 2001; Woods et al., 2002). Firstly, the

frequencies reported from the semi-quantitative FFQ (based on frequency per month and week) were all converted into frequency per day (Table 3.1).

Table 3.1 Frequencies score of semi-quantitative FFQ.

Frequency (per month)	**Score from FFQ**	**Frequency (per day) (score divided by 30)**
<1 per month	0.5	0.0167
1-3x / month	2	0.0667
Frequency (per week)	**Score from FFQ**	**Frequency (per day) (score divided by 7)**
1	1	0.1429
2	2	0.2857
3	3	0.4286
4	4	0.5714
5	5	0.7143
6	6	0.8571
7	7	1.0000
>1 per day	8	1.1429

Secondly, the participants' usual portion size (in grams) corresponding to the frequencies per day, were multiplied by the iodine factor (iodine content in mcg/100g) of each food item. The weight of each portion size was based on the food atlas book (as described previously) and household food measurement utensils. In addition, the iodine content for food items (Appendix 10) was based on the results from the Total Diet Survey 2009 and the Food Files Database 2006 (The New Zealand Institute for Crop & Food Research, 2006; Vannoort, 2009). However, the latest iodine content for iodine fortified bread was not yet available in NZ, therefore a calculation adapted from Rose et al., (2009) was used to determine the current iodine content in fortified bread (Appendix 11). The study used a formula to calculate the new iodine content in bread:

Previous iodine concentration in bread + (salt content of bread × new concentration of iodine in salt) = New content iodine after addition of iodised salt

A calculation was made for each food item (for each individual) and then summed up for all the participants (n=50). This score produced the total amount of iodine contribution for each food item. In addition, the total scores of iodine contribution for all food items for each participant, could then determine the total individual iodine intake per day.

3.9 Statistical analysis

All data from the questionnaires were analysed using the SPSS Package Version 17 for Windows (SPSS, Inc. Chicago, USA). Frequencies and percentages were used to describe all categorical data variables. Cross tabulation and chi-square tests were used to determine the differences between categorical variables. Frequencies distribution, in addition to the mean, median, standard deviation, percentiles and ranges, were determined from the continuous variables. Normality was tested for the continuous variables by using the Shapiro-Wilks statistical analysis, and they were not all normally distributed. Therefore, Spearman's rank order correlation was used to describe the relationship between two continuous variables for non-parametric data, particularly the data of urinary iodide and selenium excretions and also dietary iodine and selenium intakes. The Wilcoxon Signed Rank Test was used to test the differences of iodine intake from bread, based on prior- and post-fortification of iodine in bread. The significant level for all statistical tests was set at $P \leq 0.05$.

4 RESULTS

The results in this chapter are presented in the same sequence as found in the questionnaire (Appendix 6), with the exception of the participants' descriptive characteristics, which are presented first and then followed by their salt usage; knowledge and awareness about iodine nutrition; dietary iodine intakes; urinary iodine concentration and excretion levels; and selenium status and intakes. Frequency and percentage are reported first (for almost all variables) and this is followed by the cross tabulation and/or statistical analysis results between variables, where necessary.

4.1 Descriptive characteristics

The participants (n=50) were all women of childbearing-age, who were not pregnant or lactating. Table 4.1 shows age groups, ethnicity, education levels, living situation and the smoking status of the study population. Almost half of the participants (46%) were aged between 25 to 34 years old and only six participants (12%) were aged between 35 to 40 years. There were more New Zealanders (60%) than non-New Zealanders (40%) participating in the study. Amongst the New Zealanders: 48% of the respondents described themselves as New Zealand (NZ) European; 12% as Maori; and only two respondents were 'other' New Zealanders. Amongst non-New Zealanders: 24% were identified as Asian; 8% as European; and 10% as others (Australian, American and South African). The majority of participants were highly educated, with their highest educational levels being at tertiary level (90%), including Bachelor degree, Masters and PhD. More than half of the respondents (64%) lived with others (family or flat mates), whilst only 36% lived on their own (alone). There was only one smoker amongst all the participants, whilst 20% of participants had been previous smokers. The majority of respondents (86%) reported having no exposure to second-hand smoke.

Table 4.1 Descriptive characteristics of participants.

Category	n	Percentage (%)
Age groups		
18-24	21	42
25-34	23	46
35-40	6	12
Ethnicity		
New Zealand European	26	47
Maori	6	11
Other New Zealanders	2	4
Asian	12	22
European	4	7
Others	5	9
Highest educational qualification		
School / bursary	2	4
Certificate	3	6
Tertiary levels (university qualifications)	45	90
Living situation		
Live with husband/partner only	14	28
Live with husband/partner and children	8	16
Live with parents	6	12
Live with flat mates	4	8
Live alone	18	36
Smoking status		
Smoker	1	2
Non-smokers	49	98
Exposure to second-hand smoke at home		
Yes	7	14
No	43	86

4.2 Dietary pattern, salt usage and exposure to non-iodised salt

The first question in the questionnaire lists the major food groups consumed by the participants. In Question 1, Part A, cereal and grains were not included, in order to identify vegetarian or vegans amongst the participants and discover whether any participants avoided good food sources of iodine, specifically fish and other seafood. This is because fruits, vegetables and cereals (except bread) in NZ contain low iodine; therefore, the vegetarian/vegan subjects may have been at increased risk of a low dietary iodine intake. The majority of participants (70%) ate all the major food groups: meat, fish, seafood, eggs, dairy and fruits/vegetables (Table 4.2). From all good food sources of iodine, seafood was the most common food avoided by the participants (26%). One participant identified herself as vegetarian, since she avoided all food groups in the list, except fruits, vegetables and dairy products. Thus, she would still manage to obtain iodine from dairy products and also commercially prepared bread and iodised salt, if she ate these foods regularly. However, another participant might have a very low access to iodine intake, since she identified herself as being a vegan and she avoided all food groups in the list, including dairy products (with the exception of fruits and vegetables). She even mentioned that she also avoided bread, since almost all bread contains milk. Therefore, she would have to depend mostly on iodised salt for her daily iodine intake. Seaweed or seaweed products, such as kelp tablets may be also be good sources of iodine, but it is not highly recommended to take these tablets regularly, since the iodine content is extremely variable (MOH, 2010c).

Table 4.2 Dietary patterns of participants (n = 50).

Food	Eat		Avoid	
	n	%	n	%
Meat/poultry	47	94	3	6
Fish	46	92	4	8
Seafood/shellfish	37	74	13	26
Eggs	45	90	5	10
Dairy products	47	94	3	6
Fruits/vegetables	50	100	0	0

4.2.1 Salt usage and exposure to iodised and non-iodised salts

This section describes the usage of iodised salt and non-iodised salt (plain salt and mineral salt, e.g. rock salt, sea salt and herb salt). The highest usage amongst the participants was iodised salt (74%), followed by mineral salt (42%) and table/plain salt (26%) (Table 4.3). From all the participants who had iodised salt at home (n=37), the majority of them (65%) used iodised salt only and approximately one-third (35%) used iodised salt, together with other non-iodised salt. Thirteen participants (26%) do not use iodised salt at all.

Table 4.3 Frequencies of salt usage according to type.

Total (n)	Type of salts	Frequency (n)	Percent (%)
50	Table/plain salt	13	26
50	Mineral salt	21	42
50	Iodised salt	37	74
50	Iodised salt only	24	48
	Iodised salt & other salt	13	26
	Non-iodised salt only	13	26

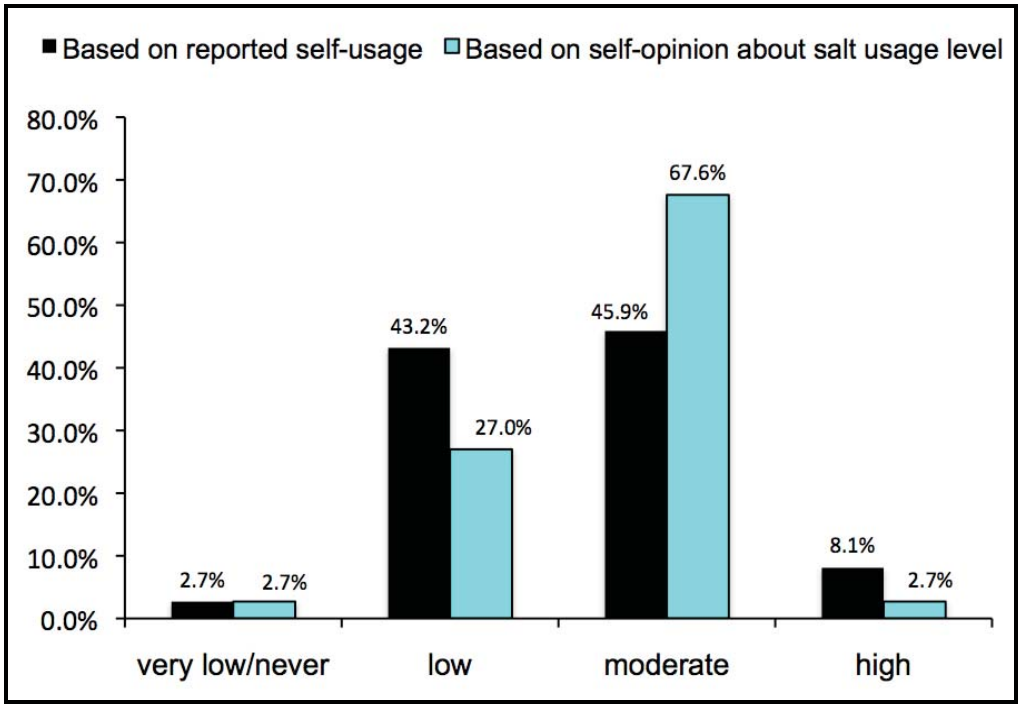
Participants were categorised into groups according to their overall salt usage, ranging from the lowest to the highest access to iodised salt, as shown in Table 4.4. Approximately half of the participants were categorised as having the highest access to iodised salt, since they only used iodised salt at home, whereas 26% were categorised as the lowest access to iodised salt, due to iodised salt not being used at all.

Table 4.4 Usage levels of iodised salts at home (n = 50).

Usage levels	Salt usage	Frequency	Percent (%)
Lowest	non-iodised salts only - no access to iodised salt	13	26
Low	non-iodised salt : regular use in cooking and as table salt, iodised salt: certain particular dish only	4	8
Moderate	non-iodised salt : regular use in cooking, iodised salt : as table salt only, or non-iodised salt : as table salt only, iodised salt : regular use in cooking	6	12
High	iodised salt: regular use in cooking and as table salt, non-iodised salt: certain particular dish only	3	6
Highest	iodised salt only	24	48

The general use of salt amongst the participants was mainly focused on the iodised salt users, since iodine is the main focus of this study. Amongst those participants who had iodised salt at home (n=37), 38% reported that they often added salt to food, whereas 11% reported that they never added salt to food. When asked about their salt usage in detail (salt use at breakfast, lunch and dinner meals and in cooking and baking), the participants were then identified as high users (8.1%), moderate users (45.9%), low users (43.2%) and very low users (3%) (Figure 4.1). More than half (54%) of the iodised salt users (n=37) reported that they prepared their own meals, whereas 46% reported that a partner or other family member usually prepared their meals. From those participants who indicated that someone else usually prepared meals for the participants (n = 17), 41% reported that their partner or other family members used a similar amount of salt to their own salt usage and 29% used less, another 29% used more salt than the participant.

Figure 4.1 Category of iodised salt usage levels amongst participants (n=37).



Approximately a third (32%) of the iodised salt users (n=37) claimed that they often limited their salt intake, whereas others indicated sometimes (38%), seldom (8%) and never (22%). Approximately half (52%) of all the participants stated that being 'health conscious' was the main reason they limited their salt intake, whilst others indicated that they did not like salt (14%), or they have a family background of high blood pressure (4%). Those who seldom or never limited their salt intake indicated that they

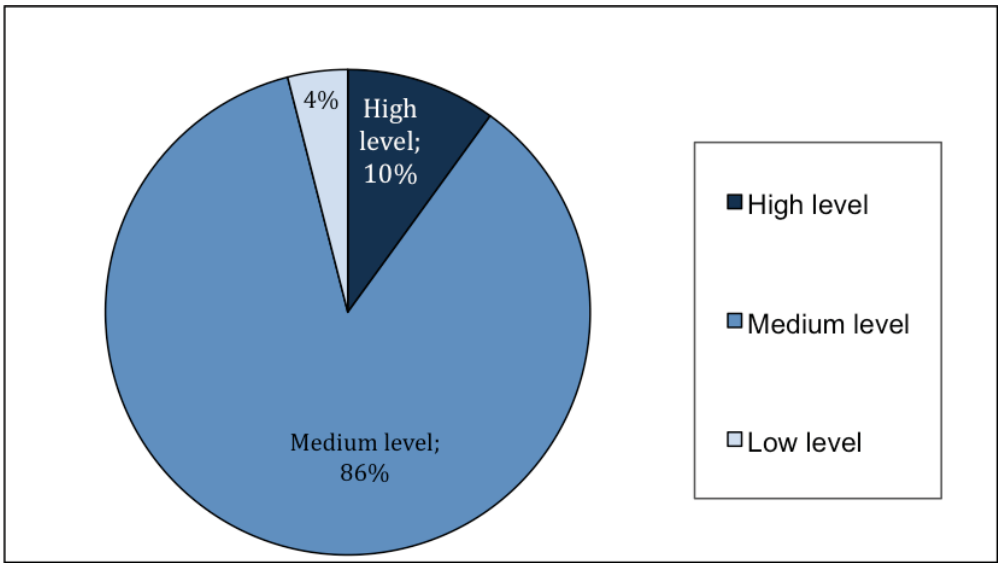
already had a low salt intake in their diet (16%) and some of them were not concerned about their salt usage (14%). When the iodised salt users (n=37) gave their opinion about their overall salt intake levels, the majority (67.6%) described themselves as moderate salt users, follow by low users (27%), and 2.7% each for high and very low salt users (Figure 4.1). There is a significant positive correlation between reported salt usage and the participants' opinions regarding their salt usage levels (Spearman's rank order; $\underline{r}(50)=0.621$, $p<0.05$). This suggests that there is a moderate relationship between these two variables (Durrheim & Tredoux, 2004), which indicates that the participants were likely to have estimated their salt usage levels correctly.

Regarding meal preparation, more participants were likely to prepare them 'from scratch', rather than use pre-prepared food or ready-made food (Table 4.5). In terms of eating out at restaurants or fast food outlets, very few participants (4%) ate out frequently (more than three times per week). Exposure levels to non-iodised salts were determined by summed up total frequency scores of eating pre-prepared and ready-made food, in addition to food at restaurants or fast food outlets (since all these foods generally contain very low iodine or they are considered to be non-iodised foods) (Appendix 12). These scores were then categorised into groups: high, medium and low level of exposure to non-iodised salt. Figure 4.2 shows that the majority of participants appear to be exposed to non-iodised salt at a medium level (86%), whilst 10% are exposed at a high level and only 4% at a low level.

Table 4.5 Frequencies of using pre-prepared and ready-made food and eating at restaurants or fast food outlets (n=50).

Frequency / Meal preparation	Often		Sometimes		Seldom		Never	
	n	%	n	%	n	%	n	%
From scratch	35	70	12	24	2	4	1	2
Use pre-prepared food	10	20	31	62	9	18	0	0
Ready made food	3	6	8	16	20	40	19	38
Frequency / Eating out :	>3 times / week		1-2 times / week		1-2 times / month		<once a month	
At restaurants/fast food outlets	2	4	12	24	27	54	9	18

Figure 4.2 The categories of exposure levels to non-iodised salt (n=50).



4.3 Knowledge and perception about iodine nutrition and action towards it

4.3.1 Knowledge and perception

All the participants were asked about their awareness of ID in NZ and the recommendations or advice, as to how this problem can be reduced. Slightly more than half of the respondents (52%) did not know (or were not sure) about ID (Table 4.6). There was a significant difference in the awareness of ID, according to ethnicity (New Zealanders *vs.* non-New Zealanders; $X^2(1, \underline{N}=50) = 7.065$, $p<0.05$). Table 4.6 demonstrates that the New Zealander participants were more aware than the non-New Zealanders, regarding this problem.

Table 4.6 Cross tabulation: awareness of ID in NZ according to ethnicity.

Awareness of iodine deficiency in NZ		Ethnicity		Total
		Non-new Zealander	New Zealander	
Do not know or not sure	n	15	11	26
	% within Ethnicity	75 %	36.7%	52 %
Yes	n	5	19	24
	% within Ethnicity	25 %	63.3%	48 %
Total	n	20	30	50
	% within Ethnicity	100 %	100 %	100 %

When the participants were asked about the best recommendation to reduce this problem, nearly half of the participants did not know any recommendations. However, iodised salt was the most common suggestion, as shown in Table 4.7.

Table 4.7 Suggested recommendations to reduce iodine deficiency in NZ, indicated by participants (n=50).

Recommendations	Frequency (n)	Percent (%)
No idea / Do not know	21	42
Use iodised salt instead of non-iodised salt	19	38
Use iodised salt and consume food rich in iodine	4	8
Use iodised salt and take supplement / iodine fortified food	4	8
Consume natural source foods (food rich in iodine)	2	4

Participants demonstrated a limited knowledge of good dietary sources of iodine (Table 4.8). Seaweed was mostly identified as a good source of iodine (58%). For other good sources, less than half of the participants gave the correct answers: fish (44%), bread (22%), eggs (20%), dairy products (12%) and seameal custard (16%). Almost half of the participants incorrectly indicated table salt (48%) and sea salt (40%) to be iodine-rich food. The majority of participants gave correct answers for food low in iodine: potato (90%), chicken/beef (92%) and lettuce (92%). Many participants also answered correctly that the thyroid gland needs iodine to produce hormones (74%).

Table 4.8 Good food sources of iodine indicated by participants (n=50).

FOOD	**Good**		**Not a good source**	
	n	**%**	**n**	**%**
Potato	5	10	45	90
Bread	11	22*	39	78
Dairy	6	12√	44	88
Chicken/beef	4	8	46	92
Fish	22	44√	28	56
Eggs	10	20√	40	80
Lettuce	4	8	46	92
Seaweed	29	58√	21	42
Table salt	24	48	26	52
Sea salt	20	40	30	60
Seameal custard	8	16‡	42	48

√ = Correct answer: considered good source, based on NZ Total Diet Survey, 2003/04 (MOH, 2010d).
* = Correct answer: mandatory fortification of iodised salt into bread has been implemented in NZ.
‡ = Correct answer: considered good source, according to Ministry of Health (MOH) and NZFSA.

Participants showed a low knowledge relating to the mandatory fortification of bread with iodised salt, as only two participants (4%) knew that it is now mandatory in NZ. More than half (56%) reported not to have heard anything about this issue and 14% indicated they were not sure (Table 49). In contrast, the participants were more aware of folic acid fortification in bread, since approximately half of the participants (46%) knew that it is voluntarily in NZ. About one third of the participants reported that they were not sure or they have not heard anything about folic acid fortification in bread.

Table 4.9 Knowledge about the status of iodine and folic acid fortification in bread.

	Iodised salt in bread		Folic acid in bread	
Perception	Frequency (n)	Percent (%)	Frequency (n)	Percent (%)
Have not heard	28	56	10	20
Mandatory	2[√]	4[√]	10	20
Voluntary	13	26	23[√]	46[√]
Not sure	7	14	7	14

[√] Correct answers regarding current iodine and folic acid status in bread in NZ.

After the participants were informed about the mandatory addition of iodised salt to bread in NZ, they were asked to give their opinion regarding this issue. Slightly more than half (54%) agreed and 14% strongly agreed with this mandatory addition, whilst 10% disagreed and 22% neither agreed nor disagreed. None of the participants indicated that they strongly disagreed with this issue. Half of the participants (50%) indicated that mandatory fortification of bread with iodised salt can help to reduce the iodine deficiency problem in NZ. However, 20% said that they were not well informed about the benefits or importance of mandatory fortification of bread with iodine. Therefore, they required more information regarding the benefits and risks of fortification. Table 4.10 shows that those who agreed or strongly agreed with mandatory iodine fortification tended to believe that it is good for helping to reduce the iodine deficiency problem. For example, participants agreed that fortification can easily supply more iodine to the NZ diet. In contrast, those who neither agreed nor disagreed mostly reported that they were not well informed on the issue, and those who disagreed were concerned about the toxicity effects, or they were more likely to think that people should get iodine from natural food sources, rather than depend on iodised salt in bread (Table 4.10).

Table 4.10 Cross tabulation: opinions regarding mandatory addition of iodised salt in bread and the reasons for these opinions (n=50).

Opinions regarding mandatory fortification of iodised salt into bread?						
Why do you agree/disagree about the iodine fortification in bread?	disagree	neither agree nor disagree	agree	strongly agree	Total	
					n	%
Not sure/not well informed	0	10	0	0	10	20
Easy and economical vehicle to supply iodine to population	0	0	2	3	5	10
Helps to reduce ID in NZ	0	0	22	3	25	50
Worried about toxicity effect	1	0	0	0	1	2
Cannot depend on bread: supposed to get it from diet/natural food sources	4	1	0	0	5	10
People in NZ need more iodine: those who get hardly any iodine from their diet	0	0	3	1	4	8
n	5	11	27	7	50	100
% of total	10 %	22 %	54 %	14 %	100 %	

4.3.2 Practices and beliefs

The majority of participants (88%) stated they would not change their bread intake after they were informed about the addition of iodised salt to bread, during the interview. A few participants stated that they intended to increase (4%) their intake, since they thought it would be a good source of iodine in their diet (Table 4.11). One person had already changed her bread intake by increasing it, but the main reason for this increase was because she purposely changed her overall diet at that time. One person intended to decrease her bread intake and she said she would prefer to make her own bread, rather than buy it, since she disagreed with mandatory addition of iodised salt in bread.

Very few participants (6%) believed that mandatory iodine fortification in bread could provide sufficient iodine for the whole population, whereas many participants (66%) thought it would not be sufficient to depend only on bread and people would still need to consume other good food sources of iodine. Others (28%) were not sure about the effectiveness of iodine-fortified bread for increasing a daily iodine intake, since they had not been informed about the benefits of fortification. The majority of participants

indicated that pregnant women (82%) were at the highest risk of ID, followed by infants (70%), adult women (66%), breastfeeding women (58%), children (58%) and adult men (16%).

Table 4.11 Cross tabulation: change of bread intake after mandatory addition of iodised salt has been implemented and reason for doing so.

Why would you change, or not change, your bread intake?	Change of bread intake since mandatory fortification has been implemented				Total	
	No change	already increased	intend to decrease	intend to increase	n	%
Do not eat plenty of bread	8	0	0	0	8	16
Just want to remain same intake	23	0	0	0	23	46
Do not really bother	10	0	0	0	10	20
Good source of iodine in my diet	1	0	0	3	4	8
Want to find out more information	2	0	0	1	3	6
Other	0	1	1	0	2	4
Total (n)	44	1	1	4	50	100
% of total	88%	2%	2%	8%	100%	

About half of the participants (48%) were not sure (or did not know) about guidelines or recommendations for fish and seafood intake during pregnancy. Those participants who were already informed about the guidelines or recommendations, indicated that they obtained the information regarding fish and seafood intake during pregnancy, from friends or family (16%); from general reading (2%); or as part of their general knowledge (36%), whilst others (46%) were not aware, or they had not had access to any guidelines or recommendations about fish or seafood intake during pregnancy. Table 4.12 shows the participants' understanding regarding guidelines or recommendations of fish and seafood intake as reported by the participants. Two participants (4%) planned to change their type of fish or seafood intake when they were pregnant, 26% planned to increase their fish/seafood intake during pregnancy, whereas 20% planned to decrease their intake, others (24%) planned to maintain the same intake and another 26% stated that they would be guided by the recommendations of professionals (doctor/midwife/dietitian).

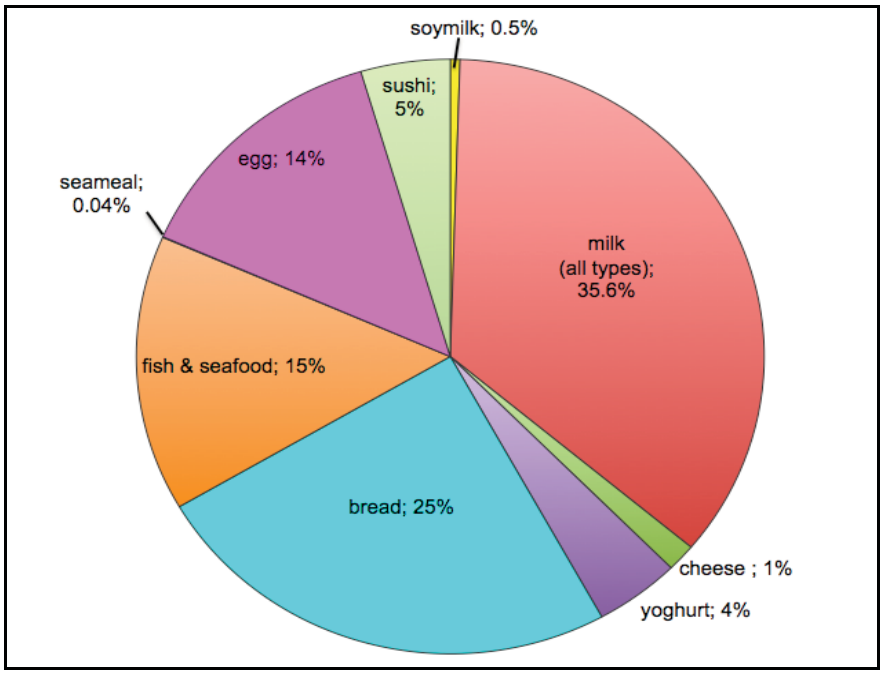
Table 4.12 Perceived guidelines or recommendations on fish or seafood intake during pregnancy, indicated by participants (n=50).

Perceived information	Frequency (n)	Percent (%)
Not sure/don't know	24	48
Eat less fish/seafood	1	2
Eat more fish/seafood	3	6
Avoid eating raw fish/seafood and always make sure it is well cooked	6	12
Avoid certain types (high mercury levels of fish)	8	16
Avoid raw fish/seafood that is categorized as high mercury levels and it must well cooked	3	6
Avoid shellfish/seafood but increase fish intake	5	10

4.4 Dietary iodine intakes

Dietary iodine intakes were determined from a semi-quantitative FFQ in the questionnaire. Milk was the main food source of iodine consumed by the participants, since it contributed approximately 35.6% of their overall dietary iodine intake, followed by bread (24.6%), fish and seafood (15%) and eggs (13.8%). Other food contributed less than 5% (Figure 4.3). Data from the FFQ were only based on the main food sources of iodine and they did not include the use of iodised salt or foods containing iodine at low levels. However, the total iodine content in all other foods was expected to contribute about 5 to 10 mcg of iodine per day (average 7.5 mcg/day) (Rasmussen et al., 2001). Therefore, in order to correct the value, 7.5 mcg/day was added to each individual's total iodine intake per day. As a result, median intakes for the participants based on FFQ data were 65.38 mcg/day. Estimated total iodine intakes were also categorised into groups of low (below the recommendation of EAR value: <100 mcg/day), medium (intake within the EAR range of 100 to 149 mcg/day) and optimal (achieved the RDI value of ≥150 mcg/day) intake levels. The majority of the participants (88%) were defined as having a low iodine intake level (8% medium intake level) and only 4% achieved the RDI. There is no correlation between UIC levels (mcg/day) and reported dietary intake of any food, including milk, fish and seafood, eggs and bread. Regarding supplementation, two participants consumed multivitamins containing iodine, but none of the participants took iodine tablets or sea kelp.

Figure 4.3 Percentage contribution of dietary iodine intakes.



Regarding bread, the top three highest contributors of iodine intake were brown bread such as wholemeal/wholegrain/multigrain bread (59%), white bread (14%) and muffin (13%) (Figure 4.4). Others contributed less than 10% of overall bread intakes. In order to estimate the change in contribution of iodine due to mandatory fortification, the iodine content in bread, prior- and post-fortification (Appendix 11), was calculated based on the calculation adapted from Rose et al. (2009). From this calculation, bread prior to fortification was estimated to contribute only 7.6% of total dietary iodine intakes, but this percentage increased over three-fold (24.6%) post-fortification. The contribution of iodine in iodine-fortified bread is significantly higher than that in bread prior to iodine fortification (Wilcoxon Signed Ranks test; $\underline{z} = -6.154$, $p<0.05$).

Some participants consumed breads that were excluded from mandatory iodine fortification; homemade, par-baked and organic breads. From this group, homemade bread was the most likely to be consumed, whilst the majority of participants indicated that they have never previously eaten par-baked or organic breads (Figure 4.5). From those participants who did consume these unfortified breads, only a very small percentage ate these breads often.

Figure 4.4 The change in contribution of iodine in bread prior to fortification and post-fortification of iodised salt in bread.

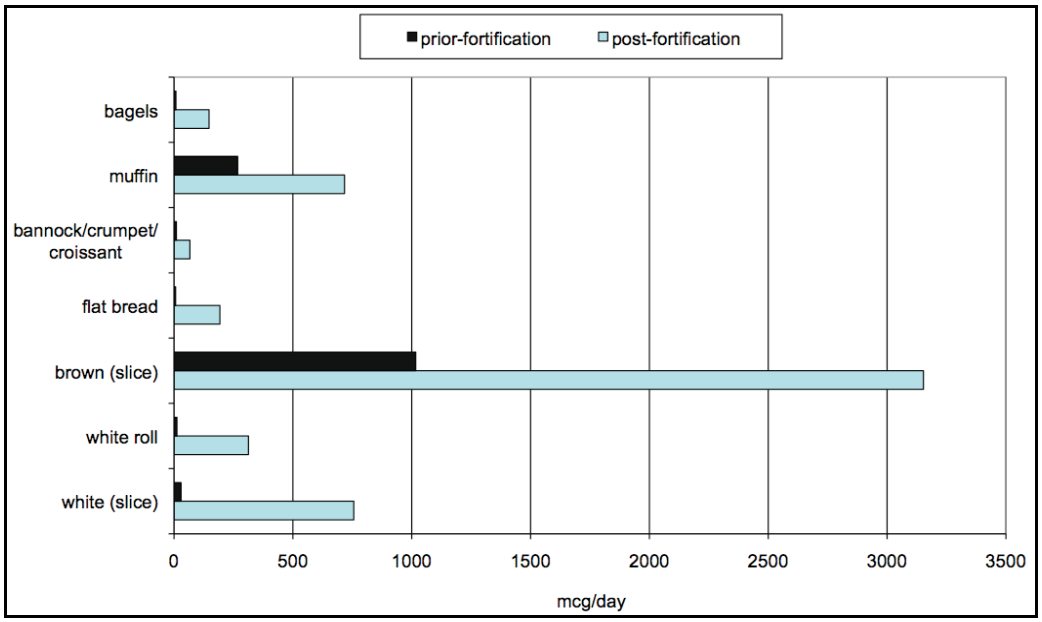
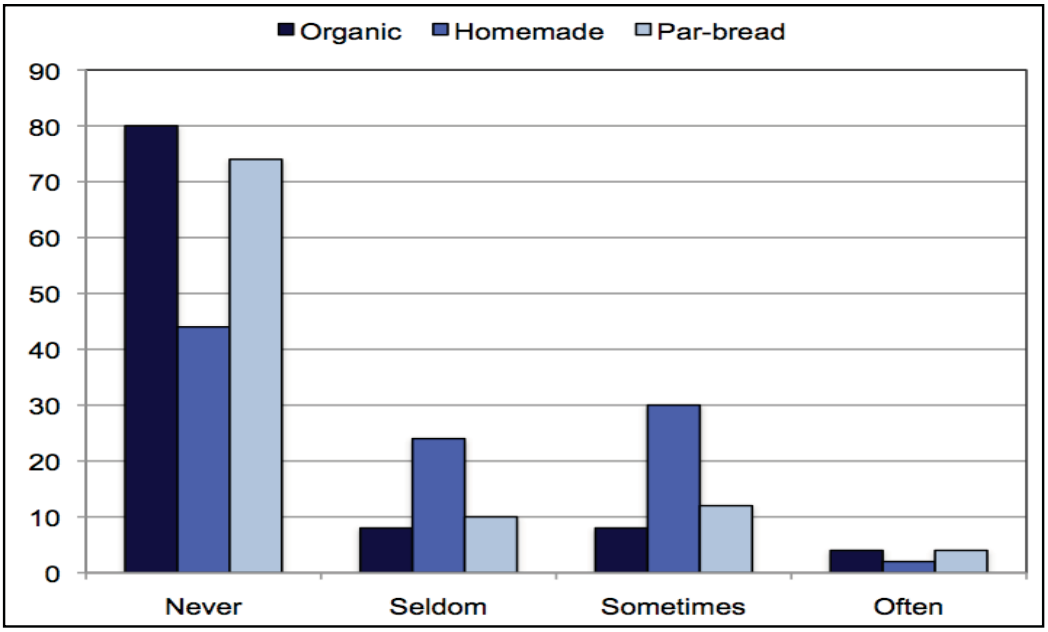


Figure 4.5 Frequency of eating breads that are not fortified with iodine.



4.5 Iodine status based on 24-hour urinary iodide excretion

Table 4.13 shows both the 24-hour urinary iodine concentration (UIC; mcg/L)and the 24-hour urinary iodine excretion (UIE; mcg/day), together with the daily iodine intake estimated from a 24-hour urinary iodine excretion and the semi-quantitative FFQ. The population median value of UIC is 64.71 mcg/l, with 30% of participants having UIC below 50 mcg/l. This classified the study population as being mildly iodine deficient

according to the World Health Organization (WHO); it can be noted that the number of people with UIC below 50 mcg/l should not exceed more than 20% of the population (WHO/UNICEF/ICCIDD, 2007). The 24-hour UIE is significantly correlated with the 24-hour UIC (Spearman's rank order; $r(50)=0.547$, $p<0.05$), which indicates a positive and moderate relationship between these two variables (Durrheim & Tredoux, 2004). Therefore, the participants with higher urinary iodine excretion levels tended to have higher selenium excretion levels.

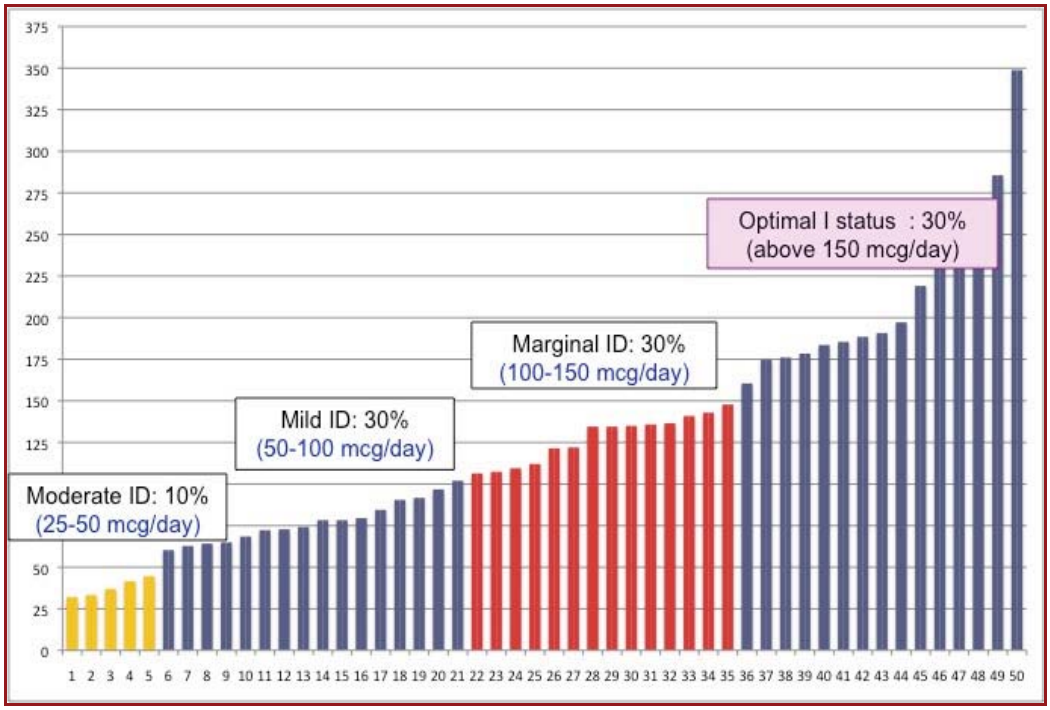
Table 4.13 Urinary iodine excretion and estimated daily iodine intake (n=50).

Descriptive statistics: central tendency		Mean ± standard deviation	Median and percentiles (25^{th}, 75^{th} centile)
24-hour iodine urinary samples	UIC (mcg/L)	82.85 ± 64.97	64.7 (43.8, 97.1)
	UIE (mcg/day)	129.1 ± 69.95	116.8 (73.9, 176.7)
Iodine intake / day (mcg/day)	Estimated from 24-hour UIC	143.4 ± 77.73	129.8 (82.1, 196.3)
	Estimated from semi-quantitative FFQ	69.5 ± 31.9	65.4 (47.6, 83.9)

Als et al. (2003) classified iodine status into categories, which indicates that five of the participants (10%) are moderately iodine deficient, since their UIE were between 25 to 50 mcg/day; 30% (n=15) of participants were classified as mildly iodine deficient (UIE levels of 50-99 mcg/day); and another 30% (n=15) were marginally iodine deficiency (UIE levels of 100-140 mcg/day). None of the participants were classified as severely iodine deficient. Fifteen participants were classified as not being risk of ID, since their UIE levels were above 150 mcg/day (Figure 4.6).

Excretion of iodine in urine over 24-hours is estimated to represent about 90% of total dietary iodine intake over the previous 24 hours (Thomson, 2004b; Zimmermann, 2008). Using this estimation, 54% of the participants were estimated to have an iodine intake below the RDI of 150 mcg/day for adults, and 90% would not meet the RDI of 250 mcg/day for pregnancy, if they become pregnant. Figure 4.7 below shows the estimated iodine intake levels of the participants and the recommended intake if they should become pregnant. There is no correlation between the estimated iodine intake from the semi-quantitative FFQ and that determined from the 24-hour urinary iodine excretion.

Figure 4.6 Urinary excretion of iodine over 24-hours (n=50).



Level of ID status according to Als et al. (2003)

4.6 Selenium status and intake

Table 4.14 shows the 24-hour urinary selenium excretion (mcg/day) and daily selenium intake estimated from the 24-hour diet recall. The median value of selenium urine excretion for the study population was 31.6 mcg/day and the mean was 34.02±15.37 mcg/day. There is no standard reference range for selenium status, since it varies between countries due to the variability of selenium content in different soils (Thomson, 2004a; Young, Nahapetian, & Janghorbani, 1982). The median dietary selenium intake in the current study using 24-hour recall was 31.7 mcg/day, which is below the EAR (50mcg/day) or RDI (60 mcg/day) for women in NZ. After being categorised according to selenium intake levels, the majority of the participants (68%) can be categorised as having a low intake level (below EAR, <50 mcg/day), 12% had a medium intake level (within EAR level, 50-59 mcg/day) and 20% had an optimal intake level (achieved or above RDI, ≥60 mcg/day). No correlation has emerged between reported selenium intake and urinary selenium excretion (mcg/day).

Figure 4.7 Estimated daily iodine intake based on the 24-hour UIE, according to the recommendation for non-pregnant and pregnant women (n=50).

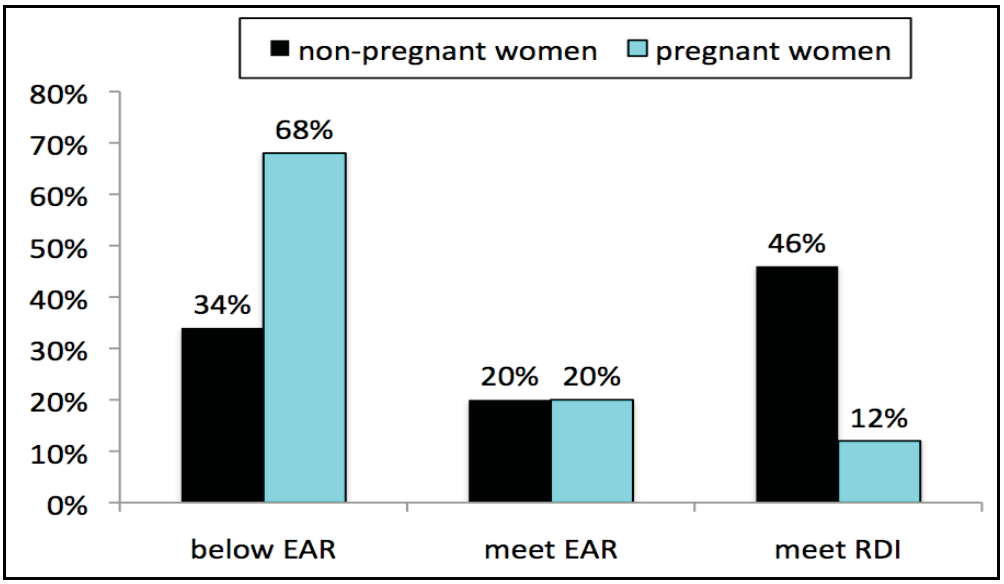
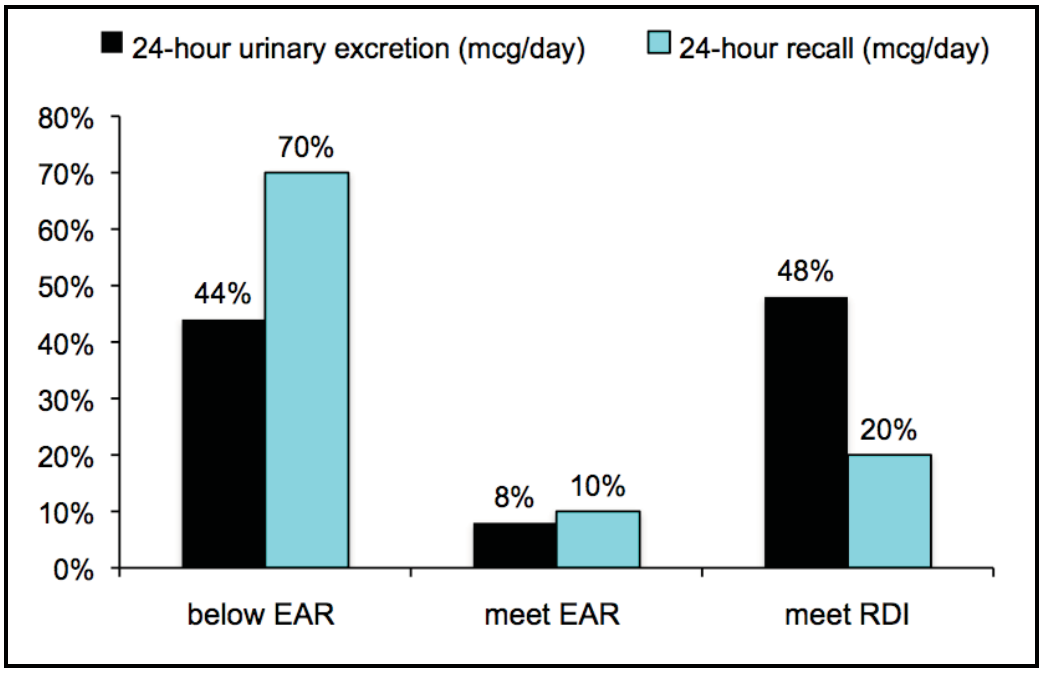


Table 4.14 Urinary selenium excretion and estimated selenium intake (n=50).

Descriptive statistics: central tendency		Mean ± standard deviation	Median and percentiles (25^{th}, 75^{th} centile)
24-hour selenium excretion (mcg/day)		34 ± 15.4	31.6 (22.4, 42.3)
Selenium intake (mcg/day)	Estimated from 24-hour urinary selenium excretion	143.4 ± 77.73	129.8 (82.1, 196.3)
	Based on the 24-hour dietary recall	42.1 ± 34.3	31.7 (21, 54.5)

Many studies have indicated that 24-hour urinary selenium excretion is assumed to represent approximately half (50% to 60%) of total daily dietary selenium intake (Thomson, 2004a, 2004b). Using this calculation, 48% of the participants met the selenium intake RDI of 60 mcg/day for women. However, the estimated selenium intake, based on the 24-hour diet recall, showed that 80% had inadequate selenium intake (Figure 4.8). There was also no correlation between the estimated daily selenium intake based on 24-hour urinary excretion and the 24-hour diet recall.

Figure 4.8 Percentage of selenium intake levels according to intake requirements (n=50), the EAR and RDI.



5 DISCUSSION

5.1 Introduction

The main aim of this study was to assess the iodine status of women of childbearing-age after the mandatory fortification of bread with iodised salt in September 2009, and to investigate the dietary iodine intake of the participants. This study also aimed to identify the participants' knowledge regarding iodine nutrition and their awareness of bread fortification in NZ. The participants' selenium status and intake were also determined, since selenium plays a role in iodine metabolism.

5.2 Participants' demographic background

The study population represented both local and international people residing in Palmerston North, NZ. The majority of the participants are women aged between 18 to 34 years old, which indicates high possibility of participants becoming pregnant in the future. Thus the participants are important groups of people who need to maintain sufficient intake of iodine and have normal iodine status. The majority of the participants were highly educated since many of them were university staff members and students. Thus as education is a proxy for social class, they are mostly of higher social status. Most of participants were also non-smokers and they had no (or very low exposure) to second-hand smoke. Therefore, the participants' iodine status was most probably least affected by goitrogen substances, particularly the thiocyanide, unless they regularly consumed very high amounts of cruciferous vegetables and/or cassava (Ermans, Delange, Van der Velden, & Kinthaert, 1972), which is unlikely to happen, especially among people in NZ. There are several different enthinicities among participants in the study (Table 4.1), however, results shows that there is no significant difference in almost all variables according to ethnicity. Furthermore, because of limited data comparing iodine status or intake between ethnicity in NZ, and small sample size of participants; resulting the finding of the study cannot be generalized to the entire NZ population.

5.3 Iodine status and intake of participants

5.3.1 Iodine status characterised by urinary iodine excretion

The present study population had a median urinary iodine concentration (UIC) value of 64.7 mcg/l, which is classified as mild iodine deficiency according to the WHO (WHO/UNICEF/ICCIDD, 2007). In addition, 30% of the participants had a UIC level below 50 mcg/l, and according to the WHO/ICCIDD/UNICEF, a population is classified as ID if more than 20% of the total population has a UIC below 50 mcg/l. Furthermore, only 30% of the participants were considered to have optimal individual iodine status (Figure 4.6) based on the 24-hour urine iodine excretion (UIE) levels stated by Als et al (2003). These results support the findings from previous studies (Table 2.10), that mild to moderate ID has been identified amongst people in NZ. These previous studies (from the 1990s until recent years) were carried out before the mandatory fortification of bread with iodised salt in NZ that began in September 2010. The present post-fortification study indicates a higher median UIC than these previous studies, which may indicate an improvement of iodine status due to an increase in iodine intake from the fortified bread.

Although the median UIC of the present study is higher than previous studies in NZ adults (Table 2.10), the iodine status of the present study is lower, compared to women in other recent studies worldwide, which were assessed either by UIC or UIE. In the United States (US), women of reproductive age were iodine sufficient, with a population median UIC of 139 mcg/l, as reported in the National Health and Nutrition Examination Survey (NHANES) 2003-2004 (Caldwell, Miller, Wang, Jain, & Jones, 2008). Similarly, Perrine et al.(2010), who analysed the NHANES 2001-2006 data also indicated that women of childbearing-age (aged 15-44 years) in the US have adequate iodine nutrition with a population median UIC of 130 mcg/l (n=1473). A study done in the UK found that the median UIE of women of childbearing-age was 140.5 mcg/day (n=31) (Rayman, Sleeth, Walter, & Taylor, 2008), which was higher than the present study value of 117 mcg/day. Whereas, another study in the UK demonstrated a similar iodine status to the present study, since the median UIC of women of childbearing-age (n=26) in that study was 66 mcg/l (Bath, Walter, Taylor, & Rayman, 2008). However, when considering individual iodine status based on criteria set by Als et al. (2003), the percentage of participants who were at risk of ID (urinary iodine excretion below 150 mcg/day) is found to be higher in the present study (70%), compared to both studies in the UK: 46% and 61% in studies reported by Bath et al., (2008) and Rayman et al., (2008), respectively. These indicate that women in NZ have low iodine intake compare to other countries, which mainly due to low good food sources of iodine in NZ.

5.3.2 Iodine intake estimated from the urinary iodine excretion and semi-quantitative FFQ

UIE over 24 hours can be used to estimate daily iodine intake, since it represents approximately 90% of total dietary intake (C. D. Thomson, 2004b; Zimmermann, 2008b). Median dietary iodine intake in this study (using UIE) was estimated to be 129.8 mcg/day, compared to 65.4 mcg/day estimated from dietary assessment using the semi-quantitative food frequency questionnaire (FFQ). The FFQ results indicate that the participants had insufficient iodine, which low than the EAR of 100 mcg/day for women of childbearing-age. Based on the estimation using UIE, approximately half of participants (46%) achieved the RDI of 150 mcg/day, but only 4% met this recommendation if based on the FFQ results (Figure 4.6). These results are significantly different from each other and no correlation was found. This is probably due to only selective foods (foods that contain high to moderate iodine levels only) being reported in the FFQ (Appendix 6), since iodised salt and other foods containing low iodine were not included when quantifying daily iodine intake. Thus, the results from the FFQ may underestimate the participants' daily iodine intake and this may have caused a large disparity with the results from the UIE. In addition, there is a large variability in the daily intake of iodine. Conversely, Rasmussen et al. (2001) found that the iodine intake from an FFQ tended to be overestimated when compared to UIE, eventhough the FFQ used in the study did not include the iodised salt intake. The authors reported the overestimation was due to the burden that participants have gone through previously; when they completed their dietary records for four consecutive days, which then influenced them to over-report their food intakes in the FFQ, since they found that the FFQ was easier to answer than the dietary records. Nevertheless, the uncertainty of the iodine content in food, especially milk, also contributed to the over-estimation of iodine intake by the FFQ. Thus, this demonstrates that it is difficult to design an FFQ to adequately assess iodine intake. Moreover, the FFQ in the present study, in addition to that of Rasmussen et al. (2001), does not cover the entire intake of food over the entire day, but instead it was just based on selected foods that were listed in the FFQ. Therefore, the semi-quantitative FFQ in this study is specifically focused on determining the percentage contribution of selected foods to overall participants' iodine intake, and this is discussed further in section 5.4.

Based on the urinary iodine excretion, the population median intake of 129.8 mcg/day did not meet the RDI of 150 mcg/day, and a third of the individual's dietary intake did not achieve the EAR (<100 mcg/day) (Figure 4.7). However, this median value is twice as high as the iodine intake of women of childbearing-age in NZ (60 mcg/day), which

was estimated in the NZTDS 2003/04 before iodine fortification was implemented (Vannoort & Thomson, 2005). A recent study of the iodine status in children aged 10-13 years in Dunedin NZ (n=93), estimated that the implementation of mandatory iodine-fortified bread in NZ would increase the median UIC of the study population from the range of 44–78 mcg/l to 95-151 mcg/l (Rose, Gordon, & Skeaff, 2009). In other countries, iodine fortification has also shown a significant increment in iodine intake amongst their populations (Table 2.12). Similar to NZ, the replacement of non-iodised salt with iodised salt is also implemented in Tasmania, Australia, but it is voluntarily. Following this voluntary iodine fortification, a study in Tasmania, Australia showed a significant increase in the median UIC in school-age children after voluntary fortification was implemented; from UIC levels of 75 and 72 mcg/L in 1998 (n=124) and 2000 (n=91), respectively (prior to fortification), to values above 100 mcg/L, following the fortification (years 2003-2005) (Seal, Doyle, Burgess, Taylor, & Cameron, 2007). In Denmark, the required iodine amount for fortification is 13 mg/kg (Rasmussen et al., 2008), which is lower than in NZ (range of 25 to 65 mg/kg) (NZFSA, 2009). However, a study in Denmark showed a significant increase in iodine intake after the mandatory fortification of bread with iodised salt was implemented, in 2000, where the median urinary iodine excretion for women (aged 18-30 years) in Copenhagen (n=24) increased from 140 mcg/day to 201 mcg/day and in Aalborg (n=45) it increased from 96 mcg/day to 171 mcg/day, following iodine fortification (Rasmussen et al., 2008).

Although the iodine intake of the current study's participants following fortification was found to be higher than previous studies in NZ, their iodine status still identified them as iodine-deficient. The iodine fortifcation in outher countries also showed better improvement of iodine status. Therefore, implementation of mandatory iodine fortification can help to reduce the ID problem in NZ, but it may not fully eliminate it. This can be shown by the finding that the majority (68%) of the present study population would not meet the EAR of 160 mcg/day for pregnant women, if the participants became pregnant. Therefore, it is important to ensure that women of childbearing-age have an adequate iodine intake, since some of them may not realise they are pregnant during in the early stages of pregnancy. Sufficient iodine intake is critical during the first trimester of pregnancy, since brain development is rapid during that period. Further investigation is necessary in order to evaluate the effectiveness of mandatory fortification of bread with iodised salt, in increasing the overall dietary iodine intake amongst NZ population, particularly the women on childbearing-age.

5.4 Factors contributing to low urinary iodine excretion

Despite the low iodine content in NZ soils, the reduction of iodine intake in the NZ diet that began in the 1990s, as reported in the NZ Total Diet Survey (NZTDS) 1990/91 and other studies (Aitken, 2001; Thomson et al., 1997; Thomson, Woodruffe, Colls, Joseph, & Doyle, 2001; Vannoort, Cressey, & Silvers, 2000), was also caused by the replacement of iodophors with non-iodised sanitisers in the dairy industry in the late 1980s (Thomson & Skeaff, 2009). The removal of iodised sanitisers has contributed to the reduction of iodine content in milk in NZ, from mean levels of 0.44 mg/kg, to a range of 0.06-0.02 mg/kg (Cressey, 2003; Vannoort & Thomson, 2005), which is much lower than the iodine content of milk in the UK (a mean concentration of 0.30 mg/kg) (UK FSA, 2008). In addition, changes in eating habits have contributed to the reduction of iodine intake, since people tend to eat outside of their home more often, buying takeaway meals, or eating processed foods, such as pre-prepared or ready-made food, which generally contain non-iodised salt (Thomson & Skeaff, 2009). In the current study, the majority of participants (86%) were categorised as having a moderate exposure to non-iodised salt and only 4% were exposed at a low level. This exposure to non-iodised salt was measured based on the frequency of the participants' use of processed food and eating out at restaurants or fast food outlets.

Furthermore, public health recommendations to reduce sodium intake have reduced the usage of iodised salt in NZ and thus reduced the iodine intake (Thomson & Skeaff, 2009). This is demonstrated in the present study, where approximately half of participants (52%) stated that they try to control their salt usage due to health concerns, whilst two participants (4%) specifically mentioned a family history of high blood pressure. In addition, the popularity of non-iodised salts, such as sea salt, rock salt or herb salt has increased, which also reduces the use of iodised salt in NZ. This can also be seen in the salt usage of the present study population, where approximately half of the participants (48%) used only iodised salt and 26% used both iodised and non-iodised salt and thus, 74% of the participants had iodised salt at home. These results are lower than previous studies, as Thomson et al. (1997) and Skeaff et al. (2002) reported 93% and 83% of their participants, respectively, used iodised salt at home. However, the percentage usage in a study on school children in Dunedin was slightly lower than the present study, since 70% of the participants had iodised salt at home and 60% used it at the table, but only 30% used it everyday (Rose et al., 2009). Approximately half of the participants in the present study (54%) used iodised salt regularly, and 12% reported using it moderately (Table 4.4). Similar to other studies, although many of their participants had iodised salt at home, both

Thomson et al. (1997) and Skeaff et al. (2002) reported that 30% of participants never used iodised salt in cooking and approximately half of the participants used iodised salt as a table salt. Overall, it appears that a change in eating patterns, together with public health advice in relation to reducing salt usage and the use of non-iodised salt by industry, are probably the main factors that have contributed to the reduction of iodine intake amongst the NZ population.

In September 2009, the NZ MOH implemented the mandatory fortification of bread with iodised salt in order to improve the overall iodine status of the NZ population and prevent IDD (FSANZ, 2008, 2009). Rose et al. (2009) estimated that this iodine fortification would significantly increase the iodine intake and thus improve the iodine status of schoolchildren in NZ. However, according to the MOH (MOH, 2010e), pregnant and lactating women would remain at risk of inadequate iodine intake, despite mandatory fortification. It was proposed that the iodine level in bread could not be increased since this could result in toxic levels for children, who have a high consumption of bread, relative to their size. Adequate iodine intake during pregnancy and lactation is critical in order to ensure normal thyroid function for both mothers and infants, in addition to preventing neurological dysfunctional, since thyroid hormones are important for the growth and development of the fetus/infant and especially for brain growth. In order to address this issue, an iodine supplement of 150 mcg/day was recently made available at pharmacies in NZ beginning in July 2010, exclusively only for all pregnant and breastfeeding women (MOH, 2010c). However, this supplement should also be available for non-pregnant women in NZ, as iodine supplementation could help women who are considering conception to ensure adequate iodine intake. This is especially important for who are likely to avoid (or eat less of) iodine-rich foods such as dairy products, bread, or fish and seafood. Other studies have also suggested iodine supplementation can help to increase overall iodine intake for women, despite the iodine fortification of food (Charlton, Gemming, Yeatman, & Ma, 2009; Charlton, Yeatman, & Houweling, 2010; Gallego, Goodall, & Eastman, 2010; Yeatman, Player, & Charlton, 2010). Table 2.13 lists studies that have demonstrated the effectiveness of iodine supplementation in pregnancy on an increase in maternal urinary iodine concentration/excretion.

However, there is a possibility that not all women will take the supplement or consume it consistently due to psychological, cultural or socio-demographic factors (Jasti, Siega-Riz, & Bentley, 2003; Ladipo, 2000). For example, a study in the UK found that women from a low social class (and some ethnic minorities) were less likely to take folic acid supplements during pregnancy compared to women from higher social

groups, although the supplement is free (Brough, Rees, Crawford, & Dorman, 2009). In NZ, iodine-only tablet of 150 mcg (known as NeuroKare) is availabale for pregnant and lactating women, and the cost is NZD $3 for a three months' supply (MOH, 2010e). However, it is still important to investigate the accessibility of iodine supplement among women with low socioeconomic background, especially those living in deprived areas in NZ. Good educational strategies, plus extensive health promotion regarding iodine nutrition is also necessary, in order to increase public understanding of the importance of iodine and to develop an awareness of ID amongst the NZ population. This is especially important for pregnant and breastfeeding women, in order to prevent low compliance with the iodine supplement (Winichagoon, 2002). Therefore, health professionals should actively approach pregnant and lactating mothers, in addition to women planning a pregnancy, by advising them on the importance of sufficient iodine intake and regular consumption of an iodine containing supplement. Regular consultation on iodine nutrition, together with the monitoring of dietary iodine intake and usage of iodine supplements by pregnant and lactating mothers is recommended, especially during their visits for maternity services. Despite iodine supplementation, all women of childbearing-age are always needed to have enough dietary iodine intake to prevent insufficient of iodine in the early stage of pregnancy, considering that the possibility of unplanned pregnancies is high. This signifies the importance of iodine food fortification in order to help to increase iodine intake among women, especially pregnant and lactating mothers.

5.5 Dietary intake of study participants

Most participants did not avoid iodine-rich food since only 30% were vegan or vegetarian, or did not eat fish and/or seafood and/or eggs, and almost all participants could obtain iodine from milk (Table 4.2). The NZTDS 1997/98 reported that dairy food (34%) makes the highest contribution to iodine intake for vegetarian females aged 19 to 40 years old (Vannoort et al., 2000). Based on the results from the semi-quantitative FFQ, milk was the highest contributor of iodine, followed by bread, fish/seafood and eggs (Figure 4.3). This dietary intake pattern is also similar to the NZTDS 2003/04, where the major contributors to iodine intake are dairy foods, fish, seafood, eggs and grains (including bread) (Vannoort & Thomson, 2005). Table 5.1 shows that milk, fish/seafood and eggs had a higher iodine content compared to other foods, and all these foods are considered as good sources of iodine by the NZ MOH (MOH, 2010d). In the current study, soymilk and seameal custard contributed a very low percentage (less than 1%) to the overall participants' iodine intake. This is most likely because the participants in the present study are not regularly eating these foods.

Table 5.1 Iodine content according to portion size for selected foods, estimated by NZTDS 2009 (Vannoort, 2009).

Food items	Standard serving size *	Iodine content per portion
Milk	1 glass (300 ml)	29 mcg
Bread (white)**	1 medium slice (28 g)	6.4 mcg
Bread (brown)**	1 medium slice (28 g)	8.1 mcg
Fish / seafood	1 portion (150 g)	35 mcg
Egg	1 medium size (49 g)	20.3 mcg
Yoghurt	1 pottle (130 g)	7.3 mcg
Sushi	1 roll (50 g)	46 mcg
Seameal custard	1 serve (154 g)	17.4 mcg
Cheese	1 slice (28 g)	1.7 mcg
Soymilk	1 glass (300 ml)	4.2 mcg

* Portion size based on Food Atlas (Nelson, Atkinson, & Meyer, 1997)
** Iodine content in bread (post-fortification) is calculated using Rose et al. (2009).

In NZ, although the usage of iodophor sanitisers in the dairy industry has been replaced with non-iodine sanitisers, milk or dairy products are still one of the main sources of dietary iodine. In the current study, 94% of participants consumed milk, with an average of approximately one glass per day. Based on the latest iodine content in food, according to NZTDS 2009 (Vannoort, 2009), it is estimated that a 300ml glass of milk can contribute an average of 29 mcg of iodine, whilst a portion of fish/seafood (150 g) and a roll of sushi can contribute about 35 mcg and 46 mcg, respectively (Table 5.1). Although fish and seafood have higher iodine content (per portion size) than a glass of milk, the higher frequency of the intake of milk has resulted in milk being the largest contributor to iodine intake. A Danish study also found that milk made the most significant contribution to iodine intake, either before or after the mandatory implementation of iodine fortified bread in Denmark (Rasmussen et al., 2008). Other studies in Europe, the UK, Norway and France, also reported that milk is the major contributor to overall iodine intake among populations (Brantsater, Haugen, Julshamn, Alexander, & Meltzer, 2007; Dahl, Johansson, Julshamn, & Meltzer, 2004; Lamand & Tressol, 1992; Lee, Lewis, Buss, Holcombe, & Lawrance, 1994). Conversely, in the US, the major contribution to iodine intake is iodised salt and seafood (U.S. CDC, 2008).

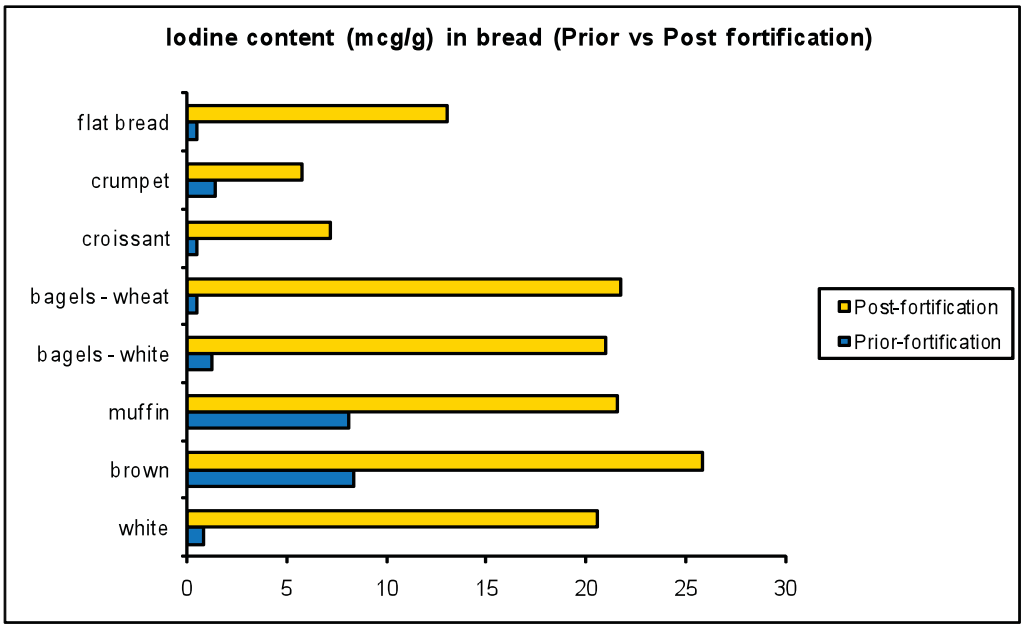
Based on the semi-quantitative FFQ, the participants' average intake of bread post-fortification was only about 2.2 medium slices per day, which contributed approximately 14.1 to 20.3 mcg of iodine per day (based on iodine content per slice of bread as shown in Table 5.1). This result is far below that of the NZ guideline of healthy eating for adults, where the recommendation is for the consumption of at least six servings of breads and cereals per day (MOH, 2010a; Russell et al., 1999). Similarly, the 1997 NZ National Nutrition Survey reported that not many people in NZ followed this guideline, which is only about one in five of the population and (between genders) females were likely to eat less bread than males, since only 9% met the guideline (Russell et al., 1999). However, the result in the present study (only bread) does not take into account the participants other consumption of cereals, since cereal is not listed in the semi-quantitative FFQ that used in the present study.

5.5.1 Fortification of iodine to increase iodine intake

It is estimated that six slices of medium size bread post-fortification can contribute approximately 38.4 to 48.6 mcg of iodine. This iodine amount is significantly higher than the iodine content in bread prior to fortification, since six slices of bread would previously contain only about 1.3 to 14 mcg of iodine. When iodine intakes were estimated based on the iodine content of bread prior to fortification, this reduced the estimated population median iodine intake from 65.4 mcg/day (post-fortification) to 46.81 mcg/day (prior to fortification). This dramatic increase of iodine level in almost all types of breads (post-fortification) can be seen in Figure 5.1. A low bread intake amongst the majority of the participants may be one of the factors why their iodine intake is still low. Moreover, iodine-fortified bread may not have reached all population groups since some people might avoid or prefer not to eat bread, due to several reasons, such as medical (coeliac disease), religious beliefs or cultural reasons (strict vegetarian or vegan), and/or a personal reason (prefer not to eat bread) (Hynes et al., 2009). In addition, some people prefer to make their own bread and they may not use iodised salt when baking, or they may consume organic and/or par-baked bread, which is exempt from the mandatory iodine fortification (NZFSA, 2009). Thus, exchanging bread for another food vehicle (for iodine fortification) may be considered, on the condition that the food is confirmed to be more commonly eating by the majority of people in NZ. However, there are many factors that need to be considered first, in order to ensure the addition of iodine will not have an unfavourable effect on the organoleptic properties of the food vehicle, in addition to maintaining the stability of the iodine in the food during processing and storage (Lotfi, Venkatesh Mannar, Merx, & Naber-van den Heuvel, 1996; Mehra, Srinivasan, Victor, Gerard, & Ronald, 2009).

Alternatively, the fortification of other food or beverages (other than bread) could also be considered, for instance, milk. Milk is the highest contributor to overall iodine intake in NZ, as reported in NZTDS 2003/4 (Vannoort & Thomson, 2005), and this can also be seen in the present study result (Figure 4.3). Hence, milk could be considered as a suitable food vehicle for another fortification in addition to bread, if one is being considered in the future. However, firstly, more study needs to be undertaken to evaluate the effectiveness of iodine-fortified bread, in addition to the suitability of another food vehicle for iodine fortification, as well as the possibility of toxicity effect on children. Acceptance by consumers and the private sector, especially the food industry, should also be considered, in order to ensure the success of such a fortification programme.

Figure 5.1 The differences between iodine content before and after fortification.



The iodine content is estimated, based on the calculation from Rose et al., (2009).

5.6 Iodine nutrition knowledge, awareness and perception

5.6.1 Iodine knowledge

Approximately, half of participants were unaware of the ID problem in NZ, and if according to ethnicity, many of the participants who were not aware of ID were non-New Zealanders (75%) (Table 4.6). However, overall, the participants demonstrated a poor knowledge relating to iodine nutrition since the majority could not identify the good food sources of iodine in NZ (Table 4.8). Other studies in Australia and South Africa also reported that the majority of participants demonstrated limited knowledge

regarding iodine nutrition and they were unaware of the importance of iodine, especially for pregnant and lactating women (Charlton et al., 2009; Charlton et al., 2010; Jooste, Upson, & Charlton, 2005; Yeatman et al., 2010). The present study demonstrates the risk of mild ID amongst the participants which, accompanied with low iodised salt usage and poor iodine knowledge, indicates that it is difficult for women of reproductive-age to achieve optimal iodine status. This signifies the importance of iodine fortification and availibility of iodine supplement for prepregnancy planning.

5.6.2 Awareness of fortification

Regardless of iodine knowledge, the majority of participants were not aware of the mandatory fortification of bread with iodine, which had been recently implemented in NZ and some participants thought it was only voluntary in NZ. Conversely, only a third of the respondents were unaware of the situation regarding the fortification of bread with folic acid. Although a few participants mistakenly thought that this fortification was mandatory, more respondents (46%) were aware that voluntary fortification of bread with folic acid is currently permitted in NZ (Table 4.9). This shows that people were more aware of folic acid than iodine fortification, although the mandatory addition of both nutrients was due to be carried out simultaneously in NZ. Prior to mandatory fortification, there was an intensive discussion in the media regarding folic acid fortification, amongst industry and consumers in NZ. The consumers were arguing about the health risks of fortification, particularly on nutrient imbalances, in addition to the cancer risk and toxicity in children or the elderly (Chauvel et al., 2010; Consumer NZ, 2009; Marks, 2010). Whereas, for bread manufacturers in NZ, the mills needed to have specific equipment that could add vitamins such as folic acid, to the flour and buying this type of equipment would eventually increase the cost of bread. This would also increase the time required to implement the mandatory fortification in NZ (Oakley, Weber, Bell, & Colditz, 2004). Compared to Australia, where bread is already fortified with thiamin at the mills, other vitamins such as folic acid, can also be easily added (Oakley, Weber, Bell, & Colditz, 2004). Iodine fortification is also easy to carry out by replacing non-iodised salt with iodised salt during bread production. These issues discussed in the public arena resulted in the NZ government delaying the mandatory fortification of bread with folic acid until May 2012. However, manufacturers are being encouraged to voluntarily add folic acid into bread at this stage (Chauvel et al., 2010). It is crucial for the government to ensure public health messages (and correct information regarding fortification) is delivered to the population at all levels of socio-economic backgrounds.

In the present study, iodine fortification was deemed acceptable by the majority of the participants. Many participants indicated that they believed the government must have undertaken rigorous research on iodine and therefore, fortification would be the best strategy to increase the iodine intake of the entire population. However, some participants indicated that they needed more information regarding the benefits and risks of fortification, mainly about the risk of excessive iodine intake, since many of them did not know that iodine fortification had already been implemented in NZ. These findings are consistent with other studies in NZ and Australia, where the majority of participants supported iodine fortification, but they also required more information regarding the advantages and risks of fortification (Thurlow, 2006; Yeatman et al., 2010). Thurlow (2006) also found that the majority of respondents showed an interest in buying bread fortified with vitamins rather than non-fortified bread, if the price differences were small. As in the present study, only a few participants indicated they would consume more bread in order to increase their iodine intake, after they had been informed about iodine-fortified bread during the interview. However, more participants would not change their bread intake, or they were not concerned about their iodine intake. Hence, the results have demonstrated the importance of keeping the population well informed, before fortification is implemented, regarding the main purpose of fortification (either iodine or folic acid) and the importance of having an adequate intake of these nutrients. This factor has been previously shown in a qualitative study undertaken in the UK, where people, who had a good understanding of the reason for an intervention, were more likely to accept the fortification (Tedstone et al., 2008). At the beginning of this study, only a minority group of women, who already knew about the importance of folic acid for the prevention of spina bifida, tended to agree with the mandatory folic acid fortification. However, when all the women (n=68) were informed about the causes and impacts of neural tube defects (NTD) and the importance of folic acid to prevent NTD, the majority of the respondents chose mandatory fortification as the best option to increase folic acid intake in the UK population (Tedstone et al., 2008).

5.6.3 Perception towards the intake of selected foods

Some participants indicated that they preferred not to eat lots of bread, and this is consistent with the low intake of bread in the study population (average intake of 2.2 medium slices per day). Moreover, some women have a tendency to eat less, since they are concerned about their diet and they were influenced by the perception of getting fat. This makes it difficult to encourage many women to eat at least six portions of bread and other food cereals daily (if they also eat less cereal), as recommended by

the NZ healthy eating guideline (Bock & Kanarek, 1995; Drichoutis, Lazaridis, & Nayga, 2005; Russell et al., 1999). Furthermore, women still need to be encouraged to eat other good food sources of iodine, particularly fish and other seafood, in order to increase their iodine intake, without being dependent on a high intake of iodine-fortified food. These foods (fish and seafood) can be considered as the best iodine sources, since they can provide high levels iodine from relatively small portions. For example, one can of salmon, or two cans of tuna, or one to two oysters, can provide about 50 mcg of iodine, which equals larger amounts of other foods, such as two to four eggs, two to three cups of milk, eight to ten steaks, six to eight slices of bread and over 2kg of boiled pasta (Australian Academy of Sciences, 2007; Vannoort, 2009). However, seafood and fish are not frequently eaten in NZ, and in the current study, as some participants avoided or preferred not to eat fish and/or seafood. Apart from being vegetarian, the high price of fish in NZ also contributed to less consumption of these foods (Rose et al., 2009; Russell et al., 1999). Thus, it is difficult to encourage people to change their eating patterns regarding fish and other seafood.

There was also misunderstanding regarding the guidelines for fish and seafood intakes during pregnancy amongst the current study participants. In the present study, some participants indicated that they would decrease or totally avoid all or certain types of fish and/or seafood if they were pregnant (Table 4.12), which is contradictory to the NZ Food and Nutrition Guidelines for Healthy Pregnant and Breastfeeding women (MOH, 2006). These guidelines recommend fish and seafood to be included in a pregnant woman's diet and they should be consumed weekly, since these foods are good sources of iodine and long-chain polyunsaturated fatty acids (MOH, 2006, 2010b). Other studies, which have supported the benefits of fish and seafood intakes during pregnancy, outweigh the possible health risk, providing the food is well-cooked and it being consumed moderately (1-2 servings per week) (Dovydaitis, 2008; Mozaffarian & Rimm, 2006). Food safety guidelines during pregnancy for Australia and NZ state that raw and ready-to-eat seafood are labelled as *red = do not eat*, whilst cooked fish and seafood are labelled as *green = safe to eat* (NSW Food Authority, 2008). There are also many other guidelines in NZ and Australia, which explain about food safety and mercury levels in fish and seafood (FSANZ, 2004, 2011; MOH, 2006, 2010b). These complex information may lead to misunderstanding or confusion among consumers. Thus, if all fish and/or seafood intake is being excluded from the diet, or intake has been decreased significantly without sufficient intake of iodine from other foods, this may increase the risk of ID amongst pregnant and lactating women in NZ.

Thus, clearer guidelines regarding which fish are safe to eat and how frequently can be eaten, are needed in order to improve this situation. Apart from fortification and supplementation, a diet plan could be suggested that may recover the loss of iodine from fish and/or seafood, by the consumption of good food sources of iodine, such as breads, eggs, milk and other dairy foods, in addition to emphasising the use of only iodised salt for cooking, or as table salt. On the other hand, as mentioned in section 5.5.1, the addition of another mandatory iodine-fortified food in NZ may be considered in order to eliminate ID in NZ. Therefore, a frequent monitoring programme concerning women's iodine status, or dietary iodine intake and the effectiveness of iodine fortification, is necessary in NZ.

5.7 Selenium status and dietary selenium intake

The mean and median values of present study participants' urinary selenium excretion are 34 and 31.6 mcg/day, respectively, which are higher than that found in studies of women living in the South Island (mean range from 9.4 to 18.7 mcg/day) (Robinson, Robinson, Levander, & Thomson, 1985; Robinson, Thomson, Jenkinson, Luzhen, & Whanger, 1997; Stewart, Griffiths, Thomson, & Robinson, 1978). This is consistent with other findings, which found that people living in the North Island are more likely to have a higher selenium status and intake, than those living in the South Island (Sheck, Davies, & Wilson, 2010; Thomson, 2004b; Thomson et al., 2007; Thomson & Robinson, 1980; Watkinson, 1981). As discussed previously, the low content of selenium in the NZ food chain is affected by the low level of selenium NZ soils. For instance, the average selenium content in grain produced in NZ is 0.1 mg/kg, which is much lower than USA products that contain an average of 2 mg/kg (Combs, 2001). Thus, the low selenium intake amongst people in the South Island is due to the consumption of locally grown food, mainly cereal, whereas people in the North Island consume cereal imported from other countries, mainly Australia, where the selenium content is higher (Sheck et al., 2010; Thomson & Robinson, 1980; Watkinson, 1981).

The selenium dietary intake is estimated to be double the urinary excretion value. Thus, the population median for selenium intake based on the urinary excretion is estimated to be 57.5 mcg/day. The median value for the intake based on the 24-hour dietary recalls is lower, at 31.7mcg/day. These values are considered acceptable, since both fulfill the requirement for iodothyronine 5' deiodinases (IDIs) and maximal GPxs, in addition to being above the minimum requirement to prevent Keshan disease (Table 2.11) (Thomson, 2004a). According to Thomson (2005), the selenium intake of

the NZ population is considered sufficient for the normal function of IDIs. In the present study, the mean selenium intake is 42.1 mcg/day and this can be considered satisfactory, since the WHO proposes 30 mcg/day, as being the lower limit of a safe range for women (Levander, 1997; WHO/FAO/IAEA, 1996). Nevertheless, if based on the individual urinary selenium excretion, only half of the participants met the NZ RDI of selenium (60 mcg/day), whereas the findings from the 24-hour dietary recalls, showed an even lower intake, since 70% of the participants did not meet the NZ EAR of 50 mcg/day. The selenium intake based on urinary excretion is not correlated to the reported 24-hour dietary recall. This is mainly due to the single report of a 24-hour dietary recall being undertaken a day earlier than the single collection of the 24-hour urine sample, thus more likely to show that the selenium consumption is variable from day to day. Furthermore, the participants might have under-reported their food intake since this is more likely to occur amongst women (Macdiarmid & Blundell, 1998).

There is a positive moderate correlation between urinary selenium excretion and iodine excretion, found in the present study. This suggests that the participants who have a low selenium intake, also tend to have a low iodine intake. However, a previous study in NZ found no association between selenium plasma and thyroid status, and that study suggested that selenium did not significantly affect people with marginal iodine status (Thomson et al., 2005). On the other hand, epidemiological data in China, have shown that populations with severe selenium deficiency are also iodine-deficient, but the inverse is not always true since IDD can be endemic in populations with sufficient selenium intake (Ma, Guo, & Wang, 1993). Since the present population study has been categorised as moderate to mild ID, the existence of a correlation between selenium and iodine status in the study, emphasises the importance to monitor selenium intake, especially in people with ID. This is essential in order to ensure that the population might able to have maximal function of IDI and GPx activities, given that selenium plays an important role as selenoproteins in the body (Triggiani et al., 2009; WHO & FAO, 2004).

5.8 Limitations of the study

The findings of this study could not be generalised for the whole population in NZ, since the sample is relatively homogenous, due to the recruitment of only women living in Palmerston North. The recruitment was also more active in the university area compared to other locations in Palmerston North, which resulted in a predominance of highly educated women amongst the participants. This situation arose due to restricted

resources and time constraints. In addition, since the recruitment posters advertised 'nutritional status for women' (Appendix 1 and 2), the nature of the women volunteering may have tended towards those who were more health conscious and therefore concerned about their diet. Thus, future research is recommended to have a larger sample size, with a more representative sample of women over an expansive geographical area in NZ, together with more diverse socio-economic and ethnic backgrounds.

Urinary iodine excretion is an effective biomarker for iodine status and it also reflects the recent iodine intake, particularly amongst non-pregnant or lactating women (Gibson, 2005; Ristic-Medic et al., 2009). As aforementioned, the WHO/ICCIDD/UNICEF also accepts the single 24-hour and casual spot urine collection method for measuring the iodine status and intake of a population (WHO/ICCIDD/UNICEF, 1999). However, studies have reported that a single urinary excretion shows large variations between (and within) individuals from day-to-day (Als et al., 2003; Busnardo et al., 2006; Rasmussen, Ovesen, & Christiansen, 1999). The present study chose the single 24-hour urinary iodine excretion collection in order to encourage participation and to reduce the burden on participants. In order to increase the accuracy of results from UIE, Rasmussen et al. (1999) suggested the collection of samples should be every alternate day/week. Another option is to increase the number of samples, since this would increase the precision of the results. For instance, previous studies have suggested a sample size of more than 150 subjects (Andersen, Karmisholt, Pedersen, & Laurberg, 2008; Vejbjerg et al., 2009).

There is possibility of misreporting in the dietary assessment of the present study (24-hour recall and semi-quantitative FFQ), since these methods were based on the recall and memory of the participants. Moreover, due to time constrains and to lower the responsive burden, this present study only collected a single 24-hour recall, although there is a high possibility of variation in the dietary intake from day-to-day. In future studies, it is recommended that multiple 24-hour recalls are obtained, in order to gain a more accurate result, for instance, two week days and one weekend day (Johnson, Driscoll, & Goran, 1996). In addition, the 24-hour recall (in this study) could only be used to assess the selenium intake, not the iodine intake. This is because the FoodWorks software used for nutrient analysis does not provide the content of iodine in foods.

The semi-quantitative FFQ could not calculate the total daily iodine intake accurately, since it did not cover the entire diet. This is because the FFQ used in the present study only consisted of selected foods: that is, foods with moderate to high iodine content only. This can also prevent a low response rate since atypical FFQ may consist of fifty to one hundred or more food items, which might burden the participants and thereby reduce their compliance (Johansson, Solvoll, Opdahl, Bjorneboe, & Drevon, 1997; Kuskowska-Wolk et al., 1992). Despite these limitations, the results however, do show similar pattern to the findings from NZTDS 2003/04 in terms of the major contribution of foods to the overall iodine intake of the NZ population.

6 CONCLUSION AND RECOMMENDATIONS

6.1 Conclusion

In the present study, the women of child-bearing age had a population median urinary iodine concentration (UIC) of 64.7 mcg/l which represents mild ID according to the WHO (WHO/UNICEF/ICCIDD, 2007); and median urinary iodine excretion (UIE) of 116.8 mcg/day, which is classified as marginal iodine deficiency (Als et al., 2003). These results are consistent with other recent findings indicating moderate to mild ID amongst the NZ population, which also indicates the re-emergence of ID. Although the iodine intake of the present study is suboptimal, it is twice the intake of NZ women estimated by the 2003/04 NZTDS. The overall findings in the study show an improved iodine status and intake compared to previous studies, prior to fortification (Table 2.10). This improvement is most likely to have been influenced by the implementation of mandatory fortification of bread with iodised salt. However, the population is still considered as iodine insufficient, and there would still be potential adverse consequences if the participants were to become pregnant.

This suboptimal iodine intake amongst participants has been caused by several factors: inadequate intake of good food sources of iodine; consumption of processed foods; frequency of eating at food outlets; and usage of non-iodised salt at home. The low intake of iodine-fortified bread amongst the participants also contributed to their overall low iodine intake. The significant contribution of this iodine-fortified bread (to iodine intake) is clearly seen, when the consumption of bread is estimated based on the content of iodine prior- to and post-fortification (Figure 4.4). However, taking into account all major contributors of food to the participants' overall iodine intake, milk is the greatest contributor, followed by bread, fish/seafood and egg. Participants are recommended to increase their iodine intake since the study population median intake of iodine did not achieve the RDI of 150 mcg/day. This can be done by increasing their bread consumption to more than their present average intake of 2.2 slices per day, in addition to using only iodised salt at home in order to improve their overall iodine intake. This is consistent with the NZ healthy eating guideline that recommends people eat bread and/or cereals of at least six servings per day (MOH, 2010a). On the other hand, fortifying other food with iodine could also be considered, in order to increase the iodine supply in the NZ diet.

Poor knowledge about iodine nutrition and the status of iodine deficiency on the population was found amongst the participants. There is a notable lack of awareness amongst the participants regarding the mandatory fortification of bread with iodine. However, the majority of the participants agreed with mandatory fortification of bread with iodised salt, but they required more information regarding fortification. Therefore, implementation of iodine fortification concurrently with good educational strategies that are aimed at increasing people's awareness and iodine knowledge is necessary. This fortification and resulting awareness could encourage people to increase their iodine intake and it might eventually reduce the risk of ID amongst the NZ population.

The selenium status (31.6 mcg/day) of the study population is just above the lower limits of the safe range for women (30 mcg/day) proposed by the WHO (WHO & FAO, 2004). Both the participants' selenium status and intake (Table 4.14) are identified as acceptable, since the population median values are above the requirement for the optimal function of IDIs and maximal GPxs. Nevertheless, if based on individual selenium status, the majority of the participants still did not meet the NZ EAR of 50 mcg/day (Figure 4.8). This suggests further investigation regarding selenium intake and status amongst the NZ population. This is important since selenium plays an important role as selenoproteins, iodothyronine deiodinases and GPx (Triggiani et al., 2009). Thus, an adequate selenium intake is important to help reduce the risk of ID and other health problems in NZ (Sheck, Davies, & Wilson, 2010). Moreover, previous studies have also reported the concerning risk of selenium deficiency amongst NZ people, due to few food sources of selenium in NZ (Thomson, 2004; Thomson et al., 2007).

Overall, the findings in this study show that the implementation of mandatory iodine fortification in bread has increased the iodine supply in the diet and it has improved the iodine status of the study population. Nevertheless, this mandatory fortification is still not sufficient to ensure that most women of childbearing-age achieve the RDI of 150 mcg/day for iodine intake, and this would be more difficult if they became pregnant, since the requirement is higher during pregnancy. This shows that iodine fortification in bread may only help to reduce the risk of ID, but it will not eliminate ID in NZ. Therefore, additional strategies such as implementing iodine fortification in other food vehicles should be considered in the future, since this might help to fully eliminate ID in NZ. Moreover, the prevention of IDD is critical, since it would reduce the risk of potential adverse consequences associated with moderate to mild ID, which have been identified in the NZ population especially amongst pregnant and breastfeeding women. Therefore, the recent introduction of subsidised iodine tablets together with

the introduction of iodine-fortified bread, can help women to increase their overall iodine intake during pregnancy and lactation. In general, the government's strategies of introducing iodine supplementation and implementing iodine fortification, if combined with strong public education and extensive promotion on iodine nutrition, could help to improve the iodine status of the NZ population. Hence, this highlights the importance of continuous assessment of iodine status and the monitoring of iodine and selenium intakes in the NZ population, especially amongst women of childbearing-age, including pregnant and lactating women.

6.2 Recommendations

Further research involving a larger sample size is needed, in order to investigate the effectiveness of iodine-fortified bread in raising the levels of New Zealanders' iodine intake. Other groups that should be included in future studies include pregnant and breastfeeding mothers and their infants, and various ethnic groups, in addition to people from low socioeconomic backgrounds. Further investigation is needed on the usage of iodine supplements and the level of iodine nutrition knowledge amongst these people, especially those living in deprived areas in NZ. More appropriate educational, promotional and marketing strategies on iodine nutrition should be developed for all age groups of people in NZ in order to increase their awareness of iodine deficiency.

Quality control in bread production is necessary to ensure the industry is always following the guideline of iodine fortification. Thus, monitoring the addition of iodised salt into bread is required to ensure correct levels of iodised salt is used for bread production in NZ. Moreover, the latest iodine content in all types of bread in NZ should be updated in the NZ food composition database. On the other hand, given that the average bread intake of the study population is considered low (2.2 slices of medium size bread), future study is required to evaluate the suitability of bread, as the most appropriate food vehicle for fortification. The other alternative is to consider implementing iodine fortification in another food vehicle, such as milk. Thus, more study is needed to investigate the appropriate food vehicle for iodine fortification, in addition to the public's acceptance of fortification.

The NZ MOH recommends a daily iodine supplement for women during pregnancy and lactation (MOH, 2010c). However, the running program of subsidised iodine tablet by the NZ government is not available for non-pregnant women. Therefore, it is recommended that these subsidised iodine tablets are not limited only to pregnant or

breastfeeding women, but should also include women who are considering conception to encouraged their daily intake of iodine. In addition, information about iodine nutrition and availibility of iodine supplement in NZ could be included in the pregnancy testing kits to reach a wider audience including those of unplanned pregnancies. Hence, women of childbearing-age will always be informed about the importance of iodine through these measure. All these suggestion are important to be taken into consideration in order to maintain adequate iodine intake amongst women of reproductive age throughout the stage of their life to prevent any potential adverse consequences in the early stage of pregnancy.

Overall, continued evaluation on the effectiveness of the iodine fortification programme in improving the iodine intake of the NZ population is necessary. Therefore, regular assessment of the iodine and selenium status and continuous monitoring of the intake of these micronutrients is needed, especially amongst women of childbearing-age, particularly pregnant and breastfeeding women in NZ.

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APPENDICES 1 – 13

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Appendix 1

Massey University

INSTITUTE OF FOOD, NUTRITION & HUMAN HEALTH

NUTRIC Study:
Nutritional Status of Women Study

Would you like to take part in a study investigating your nutritional status?

- We are trying to recruit women aged ***18 to 40 years old*** to participate in this study.
- We will invite you to come into the Human Nutrition Unit at Massey for assessment of dietary intake and nutritional status.
- We will ask you a series of questions about your dietary intake, lifestyle and nutritional knowledge.
- We will also analyse your nutritional status by collecting urine samples.
- We will pay all reasonable travel expenses.

If you would like to take part or find out more about the study please contact:

Nurul Husna, Dr Louise Brough, Dr Jane Coad,
Institute of Food Nutrition and Human Health
Massey University, Palmerston North

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Phone: (06) 356 9099 ext 81386
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This project has been reviewed and approved by the Massey University Human Ethics Committee: Southern A, Application 10/03. If you have any concerns about the conduct of this research, please contact Professor Julie Boddy, Chair, Massey University Human Ethics Committee: Southern A telephone 06 350 5799 x 2541, email humanethicsoutha@massey.ac.nz.

Appendix 2

Massey University

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Appendix 3

INSTITUTE OF FOOD, NUTRITION & HUMAN HEALTH

NUTRIC Study :

NUTRITIONAL STATUS OF WOMEN STUDY

Information Sheet

Introduction

My name is Nurul Husna Mohd Shukri. I am a postgraduate student studying human nutrition at Massey University. I am currently engaged in a research project for my master degree in human nutrition, which is "the nutritional status of women study". My supervisors are Dr Louise Brough and Dr Jane Coad, who are human nutritionists working in the Institute of Food, Nutrition and Human Health, at Massey University. I invite you to participate in this study.

The purpose of this project is to look at the dietary intake and nutritional status *of women of childbearing-age.* In the study, we want to *investigate diet and nutrient status* via an interview and a urine sample. This assessment will take place in the Human Nutrition Research Unit at Massey (except for the 24 hour urine collection which you will do at home).

Participant recruitment and selection

We are looking for about **50 to 60 women aged 18 to 40 years old.** If you are interested and contact us, we will telephone you to check you understand the details of the study and can come into the Human Nutrition Research Unit. We appreciate that taking part in this study will take time. We will pay all reasonable travel costs.

We will record details of your diet and nutrient status (via a urine sample) but we will maintain your **confidentiality** by using a code number rather than your name. No identifying details will be recorded on the interview sheets or the measurement records so all information will be **confidential.** The researchers will keep confidential all identifying details known to them. When the study results are presented, you will not be named or recognised from the information.

Appendix 3

Please note that pregnant and breastfeeding women or those with a thyroid disease or currently taking medication containing iodine are excluded from the study.

What the study will involve

We will ask you some easy questions to make sure that you are eligible to take part in the study and feel comfortable doing so.

Initially we will make an appointment for you to come into the Human Nutrition Unit at Massey University.

- **During the interview, we will ask you about your usual diet, lifestyle and knowledge of nutrition based on the questions in the questionnaire.**
- **After that we will do the 24-hour recall, which will ask you about your dietary intake in the past 24 hours.**
- **After the interview session, we will ask you to collect all of your urine over 24 hours. You will be given a container and instructions to do this at home.**

We will come to your home to collect the urine sample. We will analyse this for nutrients such as iodine.

The visit to the Human Nutrition Research Unit should take no more than 1 hour.

Data Management

Data from this study is required to be archived for 10 years. For the first 5 years it will be stored in a locked filing cabinet in the researcher's locked office (Louise Brough). It will be transferred to an official secure archive (Crown Records Management) after 5 years and destroyed by them after the 10 year period following permission from the researcher. The data stored at this archive is identified by barcode and is accessible by nominated people who have pin numbers.

Participant 's Rights

You are under no obligation to accept this invitation. If you decide to participate, you have the right to:

- decline to answer any particular question;
- withdraw from the study at any time;
- ask any questions about the study at any time during participation;
- provide information on the understanding that your name will not be used unless you give permission to the researcher;
- be given access to a summary of the project findings when it is concluded.

It is important for you to understand that if, at any point during the study, you decide that you no longer wish to continue, you may withdraw and you do not need to explain to us the reasons.

Appendix 3

What to do if you would like to participate in this study

If after reading this you would like to take part in our study, please contact Nurul Husna or Dr Brough (details below). Someone will contact you to arrange a time for you to come to the Human Nutrition Research Unit at Massey for your interview session.

If you have any questions about the purpose of the study, or would like to know more about what the interview or measurements involve, please contact us using the details shown below and we will be happy to talk to you.

Contact details

Nurul Husna
RC1.33 Postgraduate room
Institute of Food, Nutrition & Human Health
Massey University, Palmerston North
Phone: (06)3569099 ext 81386
Mobile: 022 642 0889
Email: N.H.MohdShukri@massey.ac.nz

Dr Louise Brough
Institute of Food, Nutrition & Human Health
Massey University, Palmerston North
Phone: (06)356 9099 ext 7732
Mobile: 027 737 5656
Email: Mother-babystudy@massey.ac.nz

This project has been reviewed and approved by the Massey University Human Ethics Committee: Southern A, Application 10/03. If you have any concerns about the conduct of this research, please contact Professor Julie Boddy, Chair, Massey University Human Ethics Committee: Southern A telephone 06 350 5799 x 2541, email humanethicsoutha@massey.ac.nz

Appendix 4

INSTITUTE OF FOOD, NUTRITION & HUMAN HEALTH

Nutritional Status of Women Study (NUTRIC study)

Name

Contact Address

Contact Phone Number(s)

Subject identifier

This page to be detached from remainder of questionnaire at the end of interview.

Confidential information - to be stored separately.

Appendix 4

Screening Questionnaire

Subject identifier: _____

Interview

Thank you volunteering to take part in this study.

I just want to ask you a few questions to check that you are a suitable subject and give you an opportunity to ask any questions that you may have about the study.

We want to recruit childbearing-age woman to ask them about their diet, life style and nutrition knowledge. We would also like to collect some urine.

What is your age?

Are you pregnant or breastfeeding?

Do you have any contagious blood borne disease?

Have you ever been diagnosed with thyroid disease such as thyroid enlargement or goiter/ hyperthyroidism/ hypothyroidism?

If yes, are you currently receiving any treatment or consuming medication containing iodine? Or, are you fully recovered?

Are you taking any other medication? If yes, can you please indicate the type or name of the medication(s) that you are taking?

Would you be willing to provide your urine sample?

Best method of contact:

Appendix 5

INSTITUTE OF FOOD, NUTRITION & HUMAN HEALTH

NUTRIC study
NUTRITIONAL STATUS OF WOMEN STUDY

Consent Form

This consent form will be held for a period of ten (10) years

- I have read the Information Sheet and have had the details of the study explained to me.
- My questions have been answered to my satisfaction, and I understand that I may ask further questions at any time.
- I agree to participate in this study under the conditions set out in the Information Sheet.
- I would/would not like to receive a summary of the findings of the study when it is completed.

Signature: ..... **Date:** .....

Full Name - printed .....

This project has been reviewed and approved by the Massey University Human Ethics Committee: Southern A, Application 10/03. If you have any concerns about the conduct of this research, please contact Professor Julie Boddy, Chair, Massey University Human Ethics Committee: Southern A telephone 06 350 5799 x 2541, email humanethicsoutha@massey.ac.nz.

Appendix 6

Institute of Food Nutrition and Human Health
Te Kura Hangarau o Kai-orangā-ā-tāngata
Human Nutrition & Health

INTERVIEW FOR:

NUTRIC Study

Nutritional Status of Women Study

Date:

Time:

Subject ID:

Urine sample pick up:

Appendix 6

A) I would like to ask you about your dietary intake and meal preparation.

1. Do you eat the following food groups below? (*Tick as many as apply to you*)

Food items	Eat	Avoid
Meat or poultry		
Fish		
Shellfish / Seafood		
Eggs		
Dairy products		
Fruits/ Vegetables		

2. Do you add salt to your food?

- ☐ Never
- ☐ Seldom
- ☐ Sometimes
- ☐ Often

3. How often do you add salt in your **particular** meals/dish?

Categories → **Salt use in:**	**Often** (most meals; e.g. 5 days out of 7)	**Sometimes** (some meals; e.g: lunch only or 2-3 days/week)	**Seldom** (few meals: e.g 1-3 times a month)	**Never** (No salt added at all)
Breakfast meal				
Lunch meal				
Dinner meal				
In cooking				
In baking				
Other _____				

Appendix 6

4. Who regularly prepare most of your meals?

- ☐ Self
- ☐ Partner
- ☐ Other family member (specify:_____)
- ☐ Other (specify : _____)

If other person/people prepares meals for you regularly, do you think that salt use is the same as your own use as reported above (Q3)? If no, how is the different?

_____

_____

5. In general, how would you say most of your meals are prepared?

How often do you prepare it from:	**Often**	**Sometimes**	**Seldom**	**Never**
From scratch				
Use pre-prepared foods (e.g powders soups, stocks, tomato purees, pasta sauce)				
Ready made food (just heat up or open and eat)				

6. How often do you eat out at a restaurant or fast food outlet?

- ☐ <once a month
- ☐ 1-2 times / month
- ☐ 1-2 times / week
- ☐ >3 times / week

7. If you have take away, how often do you add salt to these meals?

- ☐ Never
- ☐ Seldom
- ☐ Sometimes
- ☐ Often

Why or why not?

_____

_____

8. When did you last buy salt? _____

- Size: Bag / Package / Container or other (specify: _____)
- Quantity per purchase : _____
- Frequent of purchase : _____

9. What types of salt do you use at home? (*can tick more than one answer)*

- ☐ Table salt (plain)
- ☐ Iodised salt
- ☐ Other mineral salts: (rock salt/crystal salt/sea salt) or others:

_____

_____

10. <u>If more than one type of salts are chosen</u>, how do you use these salts at home? *(Do you use different ones for cooking/ baking and at the table).*

_____

_____

_____

11. Do you try to limit the usage of salt in your diet?

- ☐ Never
- ☐ Seldom
- ☐ Sometimes
- ☐ Often

Why,or why not? How do you do that? (*probe cooking, baking or/and at the table*)

_____

_____

_____

12. In your opinion, how would you describe your salt usage on the scale from 0-10:

Lowest **Moderate** **Highest**

---0------1------2------3------4------5------6------7------8------9------10--

Appendix 6

B) Now I would like to ask you about the knowledge of iodine nutrition in NZ

13. Do you know if there is any problem with iodine deficiency in New Zealand?

- ☐ No
- ☐ Yes
- ☐ Not sure

Do you know if there is any recommendation/ advice of how to limit/reduce this problem?

Would this recommendation/ advice influence or change how you eat?

_____

_____

_____

_____

14. Which of the following foods do you think are good sources of iodine for NZ population? (*Can tick more than one answer*)

- ☐ Potatoes
- ☐ Bread
- ☐ Dairy products e.g milk, yogurt, cheese
- ☐ Chicken/Beef
- ☐ Fish
- ☐ Eggs
- ☐ Lettuce
- ☐ Seaweed
- ☐ Table salt
- ☐ Sea salt
- ☐ Seameal custard

15. Which part of the body needs iodine to produce hormones?

Liver	Thyroid gland
Kidney	Not sure

16. Now, I would like to ask about the iodine and folic acid fortification into bread. Have you heard about the current status of those fortifications in NZ?

Status	Iodised salt	Folic acid
Have not heard about it		
Mandatory in bread		
Voluntary in bread		
Not sure		

Appendix 6

For your information, Food Standards Australia New Zealand (FSANZ) has introduced a **mandatory iodine fortification** regulation that requires the replacement of **non-iodised salt with iodised salt in all bread**, except organic bread, starting from **September 2009**. For folic acid fortification, it is voluntarily in NZ.

17. What do you think about this mandatory fortification of iodised salt into bread?

- ☐ Strongly disagree
- ☐ Disagree
- ☐ Neither agree or disagree
- ☐ Agree
- ☐ Strongly agree

Why or why not?

_____

_____

_____

18. Do you purposely change your bread intake since the mandatory fortification has been implemented?

- ☐ No
- ☐ Yes, I **increase** my bread intake
- ☐ Yes, I **decrease** my break intake
- ☐ I intend to **change** my bread intake after this interview (increase / decrease?)

Why? Why not?

_____

_____

19. Do you think consuming iodine fortified bread will provide enough iodine for the whole population in NZ?

- ☐ **Yes,** definitely. No need to worry about other iodine food sources
- ☐ **Not really**, will still need to consume more iodine-rich foods
- ☐ Not sure

20. Which groups of people do you think need more iodine? (*tick all which apply*)

- ☐ Adult Men
- ☐ Women of childbearing age
- ☐ Pregnant mothers
- ☐ Lactating women
- ☐ Children
- ☐ Infant

Appendix 6

C) I would like to know about your dietary intake for the following foods:

21. How often do you eat these foods?

FOOD EATEN	Usual portion size or photo code	Never	<1 per month	1-3x / month	Number of days per week							>1 per day
					1	2	3	4	5	6	7	
Yogurt	(Tub/Cup)											
Seaweed	(sheet/piece)											
Egg (boil/simmer/fry etc)												
Plain milk (in glass)	(glass/box)											
In Coffee:	……	……	……	……	……	……	……	……	……	……	……	……
In Tea:	……	……	……	……	……	……	……	……	……	……	……	……
Cereal:	…….....	……	……	……	……	……	……	……	……	……...	……	……
Others:												
Milky drink (flavoured)	(glass/box/cup)											
Soy milk	(glass/box/cup)											
Cheese												
-Cheddar	……	……	……	……	……	……	……	……	……	……	……	……
-Brie	……	……	……	……	……	……	……	……	……	……	……	……
-Cream	…….....	……	……	……	……	……	……	……	……	……...	……	……
others ……..												
Seameal custard	(spoon/cup)											

Appendix 6

22. Do you take any supplements?

(i) Brand & Type : _____

Frequency of consumption: _____

Looking for particular nutrient(s)/ reason: _____

(ii) Brand & Type : _____

Frequency of consumption: _____

Looking for particular nutrient(s)/ reason: _____

(iii) Brand & Type : _____

Frequency of consumption: _____

Looking for particular nutrient(s)/ reason: _____

(iv) Brand & Type : _____

Frequency of consumption: _____

Looking for particular nutrient(s)/ reason: _____

Appendix 6

D) I would like to know about your fish and seafood intakes in your meal/diet.

23. Please tell me about the types of fish you usually eat, and the amount and how often you had each

FISH	Usual portion size /photo code	Never	<1 per month	1-3x / month	Number of days per week							>1 per day
					1	2	3	4	5	6	7	
Fresh fish i._____ ii._____ iii._____	…… …… ……	…… …… ……	…… …… ……	…… …… ……	…… …… ……	…… …… ……	…… …… ……	…… …… ……	…… …… ……	…… …… ……	…… …… ……	…… …… ……
Canned fish i._____ ii._____ iii._____	…… …… ……	…… …… ……	…… …… ……	…… …… ……	…… …… ……	…… …… ……	…… …… ……	…… …… ……	…… …… ……	…… …… ….....	…… …… ……	…… …… ……
Frozen fish i._____ ii._____ iii._____	…… …… ……	…… …… ……	…… …… ……	…… …… ……	…… …… ……	…… …… ……	…… …… ……	…… …… ……	…… …… ……	…… …… ….....	…… …… ……	…… …… ……
Processed fish (fish cake/ fingers) i._____ ii._____ iii._____	…… …… ……	…… …… ……	…… …… ……	…… …… ……	…… …… ……	…… …… ……	…… …… ……	…… …… ……	…… …… ……	…… …… ….....	…… …… ……	…… …… ……
Take out fish (fast food/restaurant) _____ _____	…… ……	…… ……	…… ……	…… ……	…… ……	…… ……	…… ……	…… ……	…… ……	…… ……	…… ……	…… ……
Others:												

Appendix 6

24. Please tell me about the types of seafood you usually eat, and the amount and how often you had each.

SEAFOOD	Usual portion size or photo code	Never	<1 per month	1-3x / month	Number of days per week							>1 per day
					1	2	3	4	5	6	7	
Fresh seafood i. _____ ii. _____ iii. _____	…… …… ……	……	……	……	……	……	……	……	……	……	……	……
Canned seafood i. _____ ii. _____ iii. _____	…… …… ……	……	……	……	……	……	……	……	……	……	……	……
Frozen seafood i._____ ii._____ iii._____	…… …… ……	……	……	……	……	……	……	……	……	……	……	……
Processed seafood (e.g nugget) i. _____ ii. _____ iii. _____	…… …… ……	……	……	……	……	……	……	……	……	……	……	……
Take out fish (fastfood/restaurant) _____ _____	…… ……	……	……	……	……	……	……	……	……	……	……	……
Others:												

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25. Are you aware of any guidelines regarding fish intake during pregnancy?

☐ No ☐ Yes ☐ Not sure

If yes, what is your understanding of the guidelines? *(probe for source of information, avoidance of certain types of fish/seafood)*

_____

_____

_____

26. Would you change the amount or/and type of fish/seafood if you were pregnant?

- ☐ No (*Go direct to **Q28***)
- ☐ Yes, I will **decrease** the fish/seafood intake
- ☐ Yes, I will **increase** the fish/seafood intake
- ☐ Yes, I will change the type of fish/seafood intake
- ☐ Other? _____

27. **How** would you change your fish/seafood intake? (*probe for portion size, frequency, preparation, types of fish avoided*). Why did you make that change? (*Ask each change – probe for reason and source of advice*)

_____

_____

_____

E) Can you tell me more about your bread intakes?

28. Do you usually eat the following bread?

Bread	Often	Sometimes	Seldom	Never
Organic bread				
Homemade bread				
Par-baked bread				

Appendix 6

29. I'm going to ask you how often do you eat different kinds of breads:

BREAD	Usual portion size or photo code	Never	<1 per month	1-3x / month	Number of days per week							More than one per day
					1	2	3	4	5	6	7	
a) white bread (toast/ sliced sourdough bread/garlic bread)	Slices:											
b) white rolls, bun (including sweet bun), topped breads or sub rolls	Rolls:											
c) brown bread (including toast) whole/wheat/multi grain bread	Slices:											
d) flat breads (pita /naan / foccacia /pide @Turkish bread) (*pizza not included*)	Slices /pieces:											
e) bannock (including bannock burger)/ crumpet/ croissant	Pieces:											
f) muffins/ fruits bun or rolls	Pieces:											
g) bagels; choose: (white/wholemeal/sweet)	Pieces:											

Appendix 6

F) Now I would like to ask a few questions about you and your living situation

30. Which of category do you fall into?

- ☐ 18-24 yr
- ☐ 25-34 yr
- ☐ 35-40 yr

31. Who live in your home? (*tick which applies*)

- ☐ Husband / partner
- ☐ Children
- ☐ With parents
- ☐ On own
- ☐ Others (specify) : _____

32. Which ethnic group(s) do you identify with? (*tick that apply*)

- ☐ NZ European
- ☐ NZ Maori
- ☐ NZ other ethnicity (Asian/Arabian/others)
- ☐ Pacific Islander
- ☐ Australian
- ☐ Chinese
- ☐ Other European
- ☐ Other Asian
- ☐ American/Canadian
- ☐ Other (specify) _____

33. What is your highest level of education? Or Are you currently studying?

_____

_____

34. What is your present/most recent occupation or your usual job?

_____

_____

35. If you have a partner/ husband, what is his occupation / usual job?

_____

_____

36. Do you currently smoke?

☐ No ☐ Yes

If yes, how many cigarettes per day?

_____

37. Have you ever smoked?

☐ No ☐ Yes

If yes, how long ago and how many cigarettes per day?

_____

38. Are you regularly exposed to 2nd hand smoke? For example, does someone smoke in your house or a house you visit often?

☐ No

☐ Yes

If yes, how often per day/week are you exposed or when are you usually exposed to 2nd hand smoke?

_____

Thank you very much for your time. Is there anything else you would like to tell us about what you have been eating recently and what influences your food intake?

_____

_____

THANK YOU VERY MUCH ☺

Appendix 7

24-hour Recall

ID: Date:

Time food was eaten	Place eaten	Description of food (preparation/variety/brand)	Amount consume/ portion size

Appendix 8

Massey University
COLLEGE OF SCIENCES
Institute of Food Nutrition and Human Health
Te Kura Hangarau o Kai-orangā-ā-tāngata
Human Nutrition & Health

HUMAN NUTRITION RESEARCH UNIT LABORATORY PROCEDURE

Collection of Urine

Subject identifier :……

You have been provided with:

Two urine collection bottles.

Chilly bins containing ice or frozen slika pads

Measuring jug for urine collection.

Collection:
Start collection first thing in the morning & store bottle in the refrigerator or chilly bin between collecting.

1. Pass urine into the toilet. This urine is **not wanted,** but this is the "Time Commenced". Write the time in "Time and Date Commenced" on bottle label.

2. From now on collect all urine you pass during the rest of the day and night. Use measuring jug for collection and pour carefully into sample bottle. When you empty your bowels, please collect urine so that you do not lose the urine.

3. Collect the last specimen the next morning (24 hours after starting). Even if you do not feel the need to pass this urine, you must empty your bladder completely.

4. Write the time in "Time and Date Finished" on bottle label.

Store urine bottles in chilly bin containing ice or silica pads.

To arrange collection of your sample, please contact:
Nurul Husna 0226420889 or (06) 3569099 extension 81386
Louise Brough (06) 350 9099 extension 7732.

This project has been reviewed and approved by the Massey University Human Ethics Committee: Southern A, Application 10/03. If you have any concerns about the conduct of this research, please contact Professor Julie Boddy, Chair, Massey University Human Ethics Committee: Southern A telephone 06 350 5799 x 2541, email humanethicsoutha@massey.ac.nz."

Appendix 9

Massey University

INSTITUTE OF FOOD, NUTRITION & HUMAN HEALTH

IODINE AND ITS DIETARY SOURCES IN NEW ZEALAND

WHAT is Iodine?

- It is an essential trace mineral needed regularly in a small amount in the body.
- It allows the production of thyroid hormone that is needed for normal metabolism and to support body growth and development.

WHY is it important in New Zealand?

- New Zealand has low soil iodine levels resulting in low iodine in both animal and plant food sources. So, we are limited to good sources from the ocean only
- Early last century goitre was common in New Zealand due to iodine deficiency disorder.
- More people are now eating out or/and more likely to eat pre-prepared food or processed food which contains no iodised salt
- Mineral salts are used widely nowadays but contain a very low level of iodine.
- Studies also have shown that iodine deficiency is re-emerging in New Zealand.

Dietary sources of Iodine:

- **Sea fish & Fish:**
  Fish and sea fish are good sources of iodine. These include sardines, mackerel, tarakihi, blue cod, hoki, warehou and mussels.

- **Iodised salt:**
  Iodised salt is another good source of iodine. Manufacturers do not use iodised salt in their production. Therefore, although processed foods contain a high amount of salt, it is not iodised. It is recommended that you limit total salt intake, when you do use salt, use iodised salts.

- **Bread:**
  Since September 2009, breads in New Zealand have been fortified with iodine. For all manufactures it is mandatory to replace non-iodised salt with iodised salt in bread sold in New Zealand. This excludes organic bread.

- **Seaweed (e.g Sushi, seameal custard) :**
  Seaweed is also a good source of iodine. Although some supplements contain seaweed, the level of iodine is highly variable.

- **Eggs & dairy products:**
  Both eggs and dairy products such as milk and yoghurt are sources of iodine. This is mostly due to the feed the animals are given. In the past, milk contained higher levels of iodine due to contamination from cleaning agents. These cleaners are no longer used in milking sheds, but milk and its derivatives are still important sources of iodine in the diet.

THANK YOU ☺

Appendix 10

Iodine content in good food sources of iodine in NZ:

Food	**Iodide content (mcg/100g)**
Yoghurt	5.6
Sushi	92
Eggs	41.5
Milk (overall)	9.6
Milk trim Milk (whole)	9.7 9.6
Milk flavoured	7.6
Soymilk	1.4
Cheese	6.1
Fish, fresh	23.7
Fish, can	18.0
Fish - process	2.8
Fish, battered (take away)	9.3
Seafood - fresh	21.6
Seafood (process)	6.5
Seameal custard	70.8
Seameal mix with milk	17.43 mcg/616.8g

Sources:

The NZ Total Diet Survey 2009 (Vannoort, 2009).
The Food files (The New Zealand Institute for Crop & Food Research, 2006).

Appendix 11

Iodine content in bread in NZ:

Type of bread	**I content (prior fortification)*** (mcg/100g)	**salt content of bread*** (mg/100 g)	**[I] in salt (ppm)*** (25-65 ppm)√	**new content of iodised salt ‡** (mcg/100g)	**new I in bread** (mcg/100g)	**I available after baking**** (mcg/100g)
white	0.8	489.5	45	22.0275	22.8275	20.54475
brown	8.35	453.7	45	20.4165	28.7665	25.88985
muffin	8.065	354.5	45	15.9525	24.0175	21.61575
bagels - white	1.2	492	45	22.14	23.34	21.006
bagels - wheat	0.48	527	45	23.715	24.195	21.7755
croissant	0.5	167	45	7.515	8.015	7.2135
crumpet	1.41	110	45	4.95	6.36	5.724
flat bread	0.4524	312	45	14.04	14.4924	13.04316

* Based on the previous iodine and salt content, sources from FoodFiles (The New Zealand Institute for Crop & Food Research, 2006).
√ Current content of iodine in salt for bread industry in NZ (iodised salt in bread) (New Zealand Food Safety Authority, 2009).
** Loss about 10% of iodine after baking according to Rose et al., (2009).
‡ Result based on the calculation adapted from Rose et al., (2009). Using the formula:

iodine concentration in bread(prior fortification) + (salt content of bread × concentration of iodine in salt) = new iodine content in bread

Appendix 12

Score for estimation of exposure to iodised salt:

How often do you prepare it from:	**Mark**	**Scores for:**			
		Often	**Sometimes**	**Seldom**	**Never**
From scratch	A	1	2	3	4
Use pre-prepared foods (e.g powders soups, stocks, tomato purees, pasta sauce)	B	2	1.5	1	0.5
Ready made food (just heat up or open and eat)	C	4	3	2	1
How often do you eat out at a restaurant or fast food outlet?	**Mark**	**<once a month**	**1-2 times / month**	**1-2 times / week**	**>3 times / week**
Scores :	D	2	4	6	8

Calculation to estimate the overall exposure to non-iodised salt:

Score from mark A+B+C+D = total exposure to non-iodised salt

Appendix 13

MASSEY UNIVERSITY

24 March 2010

Nurul Husna Mohd Shukri
71 Pacific Drive
PALMERSTON NORTH 4410

Dear Nurul Husna

Re: HEC: Southern A Application – 10/03
Iodine status and dietary iodine intake of women in Palmerston North

Thank you for your letter dated 23 March 2010.

On behalf of the Massey University Human Ethics Committee: Southern A, I am pleased to advise you that the ethics of your application are now approved. Approval is for three years. If this project has not been completed within three years from the date of this letter, reapproval must be requested.

If the nature, content, location, procedures or personnel of your approved application change please advise the Secretary of the Committee.

Yours sincerely

JMBoddy

Professor Julie Boddy, Chair
Massey University Human Ethics Committee: Southern A

cc Dr Louise Brough
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