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Assessment of iodine and mercury status of Icelandic adolescent girls

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Abstract

Iceland has in the past been known for its high iodine status due to high fish consumption. Fish together with milk and other dairy products are the main sources of iodine in the Icelandic diet. In recent years fish intake has decreased, especially among young people. In addition to being a good source of iodine other health benefits of fish intake have been established. Mercury intake is however one of the risks associated with fish intake.

The aim of this thesis was to assess the iodine status of Icelandic adolescent girls and to gather information on intake of fish and milk consumption. Mercury status was additionally assessed.

A random sample of 350 Icelandic adolescent girls born in the years 1987-1992 was selected by Statistics Iceland. Of the original sample 145 accepted to participate and 112 completed the study. Two Food Frequency Questionnaires (FFQ) were used to evaluate food consumption. A blood sample was taken to measure Thyroid-stimulating hormone (TSH) and mercury concentration, and a urine sample to measure iodine and creatinine concentration.

The mean urinary iodine concentration was found to be 186µg/l (SD 145) and the median 140µg/l. When iodine concentration was adjusted for urinary creatinine the mean urinary iodine concentration was 126µg/g (SD 99) and the median 103µg/g. 10-15% of the subjects had iodine concentration below 50µg/l and 24-31% had iodine concentration below 100µg/l. Milk and dairy products were the main source of iodine, providing 43% of the iodine in the diet followed by fish providing 24%. A positive association was seen between milk consumption and iodine concentration ($r=0.275$ and $p=0.003$). The average intake of fish was less than 15g/day. Fish intake was not directly related to urinary iodine concentration.

The mean mercury concentration in blood was found to be 1.47µg/l (SD 1.05) and the median 1.32µg/l. A positive correlation was found between fish consumption and mercury concentration in blood ($r=0.416$ and $p>0.001$). However the mercury concentration was far below exposure limits.

The study showed that the median urinary iodine status of the subjects was optimal but in the lower end of the optimal range. 10-15% of the subjects were classified as having severe to moderate iodine deficiency and 24-31% as having mild iodine deficiency. Milk and dairy products were the major source and determinant of iodine status. Fish intake was below the recommended intake. Fish intake was not directly related to iodine status in the present study, fish intake should nevertheless be encouraged due to its contribution to total iodine intake and multiple health benefits.

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Abbreviations

BEE	Basal Energy Expenditure
BMI	Body Mass Index
DHA	Docosahexaenoic acid
EPA	Eicosapentaenoic acid
EPA	U.S. Environmental Protection Agency
E%	Energy consumed
FDA	U.S. Food and Drug Administration
FFQ	Food Frequency Questionnaire
IDD	Iodine Deficiency Disorders
NHANES	National Health and Nutrition Examination Survey
NNR	Nordic Nutrition Recommendations
NRC	National Research Council
PCB	Polychlorinated Biphenyls
PTWI	Provisional Tolerable Weekly Intake
SD	Standard deviation
TSH	Thyroid-stimulating hormone
WHO	World Health Organization

1 Introduction

Iodine deficiency is considered to be one of the most common nutrition disorders in the world (NNR 2004) and the world's greatest single cause of preventable brain damage (Delange 2001). Despite a worldwide application of successful iodine supplementation programs over the last four decades, iodine deficiency remains a major public health problem throughout the world. In 2004, it was estimated that out of the 2 billion people around the world that are at risk of iodine deficiency, 20 percent live in Europe, with Eastern and Western Europe being both affected. All European countries except Iceland have experienced this health and socioeconomic threat to a greater or lesser extent (WHO/UNICEF 2007). The fact that mild to severe iodine deficiency persists in many European countries has important public health consequences, including impaired intellectual development of infants and children (Delange 2002). Iceland has in the past been known for its high iodine status due to high fish consumption. In the Icelandic National Nutrition Survey 2002, fish was the main source of iodine from the diet. However, fish intake had decreased significantly from previous study conducted in 1990, especially among young people (Steingrimsdottir et al. 2003). This calls for the importance of evaluating iodine status in the Icelandic population.

Seafood is an important part of a healthy diet but is also a dietary source of heavy metals and organic pollutants (Sioen et al. 2007a). Mercury is the most dangerous of all the heavy metals (Salonen et al. 1995). All mercury compounds are toxic to humans and animals, but the organic forms, methyl mercury and dimethyl mercury, have the highest toxicity (Gochfeld 2003). Seafood is the predominant source of human exposure to methyl mercury (WHO 2007). Children exposed to high levels of methyl mercury in uterus have exhibited a variety of developmental neurological abnormalities. The developing brain is thought to be the most sensitive organ for methyl mercury toxicity (WHO 2008). High mercury levels have not been shown in fish species commonly eaten in Iceland.

In this study, the subjects were questioned about their food consumption and their iodine and mercury status was assessed. This kind of study has not been conducted in Iceland before and might therefore have significant scientific value and add to the existing knowledge on the diet of Icelanders. Adolescent girls are future mothers to be and the nutrients covered in this thesis are important to fetal development and the health of both mother and child.

Aims of the study were to:

- Assess iodine and mercury status of Icelandic adolescent girls.
- Gather information on fish consumption of Icelandic adolescent girls.
- Gather information on the consumption of other food sources such as milk and dairy products among Icelandic adolescent girls.

2 Review of literature

2.1 Iodine

Iodine is a trace element present in the human body in small amounts, almost exclusively in the thyroid gland (WHO/UNICEF 2007). Iodine is an essential component of the thyroid hormones, tetraodothyronine (T_4) and triodothyronine (T_3), which are necessary for normal growth, development and metabolism during pregnancy, infancy and throughout life (Caldwell et al. 2005). Uptake of iodine by the thyroid is dependent on need. This process is largely mediated by TSH which is secreted by the anterior pituitary gland. TSH activity increases in iodine deficiency (Dunn 2006). To meet the demand for adequate hormones, the thyroid has developed an elaborate mechanism for concentrating iodine from the circulation and converting it into thyroid hormones, which it stores and releases into the circulation as needed (Dunn 1998). Most of the remaining iodine is excreted in urine (Dunn 2006). The thyroid hormones act through specific receptors to selectively regulate gene expression in target tissues, particularly liver, pituitary gland, muscle and developing brain. Inadequate iodine supply leads first to inadequate hormone production and then to inadequate tissue response, i.e. hypothyroidism (Dunn 1998). Thyroid hormones promote synthesis of enzymes and other proteins to increase metabolic activity in tissues. Hyperthyroidism is characterized by increased nervous activity, temperature, energy consumption and heart rate, whereas hypothyroidism involves an overall slowing of metabolism, including lower body temperature and mental and physical sluggishness (Dunn 2006).

2.1.1 Recommendations

The iodine required to prevent goiter is estimated to be 50-75µg/day or approximately 1µg/kg body weight per day. Long-term intakes below the lower level of intake are associated with an increased risk of developing deficiency symptoms. The average requirement is estimated to be 100µg/day iodine for both adult women and men. The recommended intake of iodine is 150µg/day for adults and adolescents. An extra 25µg/day is recommended during pregnancy and extra 50µg/day during lactation to provide sufficient iodine in the breast milk (NNR 2004). WHO recommends also 150µg/day for adolescents and adults, but 250µg/day for pregnant and lactating women (WHO/UNICEF 2007).

2.1.2 Sources of iodine

Fish has the highest natural concentration of iodine (Reykdal et al. 2000; Dahl et al. 2004; Haldimann et al. 2005). Analysis on various Icelandic fish species shows that the iodine content in haddock is 191µg/100g, in cod 170µg/100g, in wild salmon 36µg/100g and in farmed salmon 30µg/100g (ISGEM 2008). Marine fish species low in fat, like cod have the highest iodine contents (Dahl et al. 2004). Iodine in fish reflects the content in the water they inhabit. A Swiss study showed that marine fish had about six-fold higher iodine level than freshwater fish (Haldimann et al. 2005). The iodine content in raw food is reduced by cooking (WHO 1996), boiling or grilling/frying fish results in about 50-82% and 20% respectively loss of iodine (Harrison et al. 1965).

Another important source of iodine is milk and other dairy products (Reykdal et al. 2000; Dahl et al. 2004; Haldimann et al. 2005). Icelandic milk, dairy products and eggs are generally high in iodine (Reykdal et al. 2000). Iodine content of milk and dairy products varies considerably depending on feed and use of disinfectants containing iodine in connection with milking (NNR 2004). In Iceland, fish products have traditionally been used for animal feeding causing high iodine content in milk and dairy products. Fish can contain between 30-300µg iodine per 100g. There is therefore a considerable amount of iodine in fishmeal, although some is lost through processing (Reykdal et al. 2000). Iodine content for various food items is summarized in Table 1.

Table 1: Iodine content in Icelandic food items

Food	Iodine (µg/100g)
Liverwurst[1]	13.5
Lamb liver[1]	12.9
Lamb[1]	1.2-2.9
Beef[1]	1.3
Pork[1]	2.7
Chicken[1]	3.2
Cheese, 26%[1]	42
Milk[1]	9.7-12.7
Skyr[1]	23
Eggs[1]	57.2
Chocolate, bitter[1]	2.3
Chocolate, milk[1]	62.3
Haddock, raw[2]	117-238
Haddock, boiled[2]	220
Wild salmon, raw[2]	35.8
Salmon, boiled[2]	35.4
Cod, raw[2]	170

[1] Reykdal et al. 2000.
[2] ISGEM 2008.

Iodine is readily enriched in milk, whereas meat of dairy cows does not have high concentrations (Swanson et al. 1990; Haldimann et al. 2005). In the Icelandic National Nutrition Survey 2002 fish was the main source of iodine followed by milk and dairy products (Steingrimsdottir et al. 2003). In Norway, milk and dairy products are the main source of iodine. This is due to high consumption of milk and dairy products combined with relatively high concentration of iodine in milk because of mandatory iodine fortification of cow feed since 1950 (Dahl et al. 2004; Brantsæter et al. 2007). In the U.S., dairy products are also the most important source of iodine (Dunn 2006). In a study conducted in Switzerland, bread was shown to be the major dietary source of iodine, which is due to the use of iodized salt. Milk contained also relatively high amounts of iodine which can be partially explained by iodized cattle feed supplements used in the dairy industry (Haldimann et al. 2005). As the only source of iodine in humans is through diet, iodine deficiency can be corrected either through iodine supplementation or alternatively by consuming food produced in iodine sufficient areas. In industrialized societies, diet diversity is the main ensurance for adequate iodine intake. That might explain why iodine deficiency today is more likely to occur in poor and more remote areas, even of industrialized Europe, for economical reasons rather than geographical location (Vitti 2001).

2.1.3 Iodine intake

Regular consumption of fish, both fatty and lean varieties is recommended as a part of a balanced diet. The Public Health Institute of Iceland (2006) recommends fish consumption at least twice a week as a main meal, in addition to fish used as bread spread and in salads. Two fish meals a week contribute to the total iodine intake but recommendations are also based on the fact that fish is a good source of nutrients such as selenium, vitamin D and long-chain n-3 fatty acids (NNR 2004). Fish reduces the risk of cardiovascular disease (Whelton et al. 2004). Furthermore, intake of fish and fish oil has been negatively linked to many other diseases such as stroke, hypertension (Mozaffarian 2007) rheumatoid arthritis (Pattison et al. 2004) and non-insulin dependent diabetes mellitus (Ukropec et al. 2003). Fish consumption has also been shown to have positive effects on weight loss (Thorsdottir et al. 2007). Fish can however be a contributor of contaminants in the diet such as mercury and polychlorinated biphenyls (PCB). High fish intake used to be one of the main characteristics of the Icelandic diet and was higher than in any other European country in 1990. The Icelandic National Nutrition Survey conducted in 2002 showed a dramatic drop in fish consumption from 1990, with a 30% decrease in the average fish consumption of adults. Adolescent girls aged 15-19 years old were reported to consume only 15g fish per day and received only 2/3 of the recommended intake of iodine on average (Steingrimsdottir et al. 2003). Another Icelandic study reported fish consumption only 20g/day for 15 years old girls. The median iodine intake was only 115µg/day and 44% did not to meet their daily recommendations (Thorsdottir and Gunnarsdottir 2006). If a single portion of fish is estimated to be approximately 150g, the previously reported average intake of the Icelandic adolescent girls does not meet the twice a week recommendation.

Milk is another important source of iodine. The recommended intake of milk and dairy products is two glasses, bowls or cans a day, preferably products that are low in fat and added sugar. Cheese can count as a dairy product and 25g of cheese is equivalent to one portion. Those who do not consume dairy products can use calcium enriched soy milk or calcium supplements (Public Health Institute of Iceland 2006). The recommendation for milk consumption is based on the notion that milk provides several nutrients e.g. calcium, potassium, riboflavin and selenium (NNR 2004). Adolescents are encouraged to consume three portions to reach the daily recommendation (Thorsdottir and Gunnarsdottir 2006). In the Icelandic National Nutrition Survey 2002, adolescent girls aged 15-19 years old had an average consumption of 3.2 portions of milk and dairy products per day (Steingrimsdottir et al. 2003). Another Icelandic study on children 9-15 years old showed that 15 years old girls consumed on average 3.3 portions of milk and dairy products (Thorsdottir and Gunnarsdottir 2006) which is in accordance with recommendations.

2.1.4 Iodine Deficiency Disorders

When the physiological requirements for iodine are not met, a series of functional and developmental abnormalities occur. This includes thyroid function abnormalities and when iodine deficiency is severe, hypothyroidism, endemic goiter and cretinism, endemic mental retardation, decreased fertility, increased prenatal death and infant mortality. These complications, which constitute a hindrance to the development of the affected populations, are grouped under the general heading of Iodine Deficiency Disorders (IDD) (Hetzel 1983). IDD refers to all ill effects of iodine deficiency that can be prevented by adequate intake of iodine (WHO/UNICEF/ICCIDD 2001). IDD is best described in relation to four different phases of life, the fetus, the neonate, the child and adolescent and finally the adult. Disorders affecting the fetus are e.g. abortions, stillbirths, congenital anomalies, increased prenatal and infant mortality, cretinism, psychomotor defects and fetal hypothyroidism. Disorders affecting neonates are e.g. neonatal hypothyroidism and neonatal goiter. Disorders affecting children and adolescents are e.g. juvenile hypothyroidism, goiter, impaired mental function, retarded development and cretinism. Disorders affecting adults are e.g. goiter with its complications, hypothyroidism and impaired mental function (Hetzel 1986).

All degrees of iodine deficiency (mild: iodine intake of 50-99µg/day, moderate: 20-49µg/day and severe: $<$20µg/day) affect thyroid function of the mother and the neonate, as well as the mental development of the child. The damage increases with the degree of the deficiency, with overt endemic cretinism as the most severe consequence (Delange et al. 2001). Goiter is the first and most visible sign of iodine deficiency. The thyroid enlarges as an adaptation to the threat of inadequate hormone. In mild iodine deficiency, the response may be adequate to preserve euthyroidism, but at the cost of an enlarged thyroid and the attendant risk of neck compression and eventual hyperfunctioning autonomous nodules with hyperthyroidism. An insufficient adaptation in adults produces hypothyroidism with its usual clinical stigmata (Dunn 1998). Cretinism is characterized by gross mental retardation, deaf-mutism, short stature, spasticity and other neuromuscular retardation. Cretinism from iodine deficiency has now become rare because of improved iodine nutrition (Dunn 2003). However, less obvious damage to the brain occurs with milder degrees of iodine deficiency and such damage is much more widespread within the affected population (Holloweell et al. 1998). Thyroid hormone is particularly important for myelination of the central nervous system, which is most active in the prenatal period and during fetal and early postnatal development (Dunn 2006). Individuals, who are hypothyroid at this critical period, frequently have permanent mental retardation, which cannot be corrected by later administration of thyroid hormone or iodine (Dunn 1998). This has an important public health consequence, putting most people in an iodine deficient area at some risk for defective brain development (Dunn 2003). A meta-analysis on the relationship between children's intelligence and factors such as iodine deficiency showed that iodine deficiency played a role of intermediated strength, compared with other causes in delaying brain development, lowering the IQ of children by at least 10 points in IDD areas (Qian et al. 2002). Another study on Mexican schoolchildren showed that moderate iodine deficiency was associated with a 4.26 times higher risk of low IQ (Pineda-Lucatero et al. 2008).

2.1.5 Iodine and adolescence

Inadequacy of iodine may put adolescents at risk for developing delayed IDD, as sensitivity to iodine deficiency of thyroid gland metabolism is enhanced during sexual maturation (Als et al. 2000b). Puberty is a crucial period of hormonal interactions in the human life cycle (Hanna & LaFranchi 2002). Marked changes in thyroid functions occur during puberty as an adaptation to body and sexual development (Flueury et al. 2001). Thyroid hormones are one of the major growth regulators along with growth hormone (GH) and the Insulin Growth Factors (IGFs) (Markou et al. 2008). They influence almost all aspects of normal child development and thus play a crucial role as a regulator of nervous system, myelination, dental and skeletal development, metabolism, organ functions (Larsen et al. 1998), growth and puberty (Larsen et al. 1998; Hanna and LaFranchi 2002). Thyroid disorders in adolescents may present as goiter, or as a general cluster of abnormal symptoms and physical findings (Bettendorf 2002).

Adolescent girls as well as young women are likely to become pregnant in the near future. Before becoming pregnant, women should ideally have an average daily iodine intake of 150µg, to ensure that their intra-thyroidal iodine stores are replenished before pregnancy

(Glinoer 1995). A survey conducted in the U.S. indicated that as many as 14.9% of women in the child-bearing age and 6.7% during pregnancy had iodine excretion levels into the range of iodine deficiency, or below 50µg/l (Dunn 1998). A Swiss study showed that adolescents reached their recommendation in only 13% of cases. Girls and women of childbearing age (age 13-35 years) had the most unsatisfactory results (Als et al. 2000b). Iodine status has never before been assessed in Icelandic adolescents.

2.1.6 Prevention of IDD

Insufficient iodine intake is the major cause of low iodine status. Over a century and a half ago iodine deficiency had already been recognized. At the beginning of the 19^{th} century, it was first suggested that the use of salt fortified with iodine would lead to good health in people living in mountainous regions (WHO/UNICEF 2007). In 1999, WHO's Regional Office for Europe adopted elimination of iodine deficiency as one of the targets in its nutrition action plan (WHO/UNICEF/ICCIDD 1999).

Universal salt iodization is considered to be the most efficient way to improve iodine intake since availability of iodine rich foods, such as marine fish, is decreasing worldwide (Winger et al. 2008). Salt is one of the few commodities that is consumed by virtually everyone and salt consumption is fairly stable throughout the year. Salt iodization technology is easy to implement and available at a reasonable cost. The addition of iodine to salt does not affect its color, taste or odor and the quality of iodized salt can be monitored at production and retail (Delange et al. 2001). Iodized table salt is available in Denmark, Sweden, Finland and Norway and contributes to iodine intake (NNR 2004) but not in Iceland. While there is a general agreement on the benefit of salt iodization for the improvement of iodine status in the population, this appears to be inadequate to further secure sufficient iodine supply for the prevention of IDD. The use of salt for household purpose is decreasing, salt used in food industry is generally not iodized and it is considered undesirable to encourage increased salt intake for other health reasons (Winger et al. 2008). It is argued that the strategy for the prevention and treatment of iodine deficiency in Europe has to start with information and health education, not only of the public but also of the health professionals, who are often insufficiently aware of the problem (Delange 2002).

2.1.7 Upper intake levels for iodine

For some nutrients, high intake may cause adverse or even toxic symptoms. Tolerable upper intake levels refer to the highest average daily nutrient intake level unlikely to pose risk of adverse health effects to almost all (97.5%) healthy individuals in an age- and sex specific population group (WHO/UNICEF 2007). Upper intake levels have been established for some nutrients. Prolonged intakes above these levels can, for certain nutrients, induce an increased risk of toxic effects. The upper levels are derived for the normal healthy population and values are given for adults (NNR 2004).

An iodine intake in excess of 2mg/day can in rare cases cause reactions such as rhinitis, nasal congestion, swollen salivary glands, headache and acne-like skin changes. High iodine intake can also cause disturbances in the thyroid function. Symptoms include inflammation in the thyroid gland, goiter and hypo- or hyperthyroidism (NNR 2004). The Scientific Committee on Food has proposed 600µg/day of iodine as a safe upper level for adults (European Commission 2002). Individuals especially sensitive to the toxic effect of excess iodine include children of all ages, pregnant women, fetuses and newborns of breast feeding women and those under previous or current supervision for thyroid disease (WHO 2003a).

Not many populations in the world have a high iodine intake from natural sources. Iodine excess occurs in Japan due to high seaweed consumption and in Chile due to high environmental iodine, widespread use of iodine for water purification and iodization of salt at excessive levels in recent years (Dunn 2006).

2.1.8 Analysis of iodine status

Three measures are recommended for assessment of iodine in populations, urinary iodine, goiter rate and serum TSH (WHO/UNICEF/ICCIDD 2001). According to WHO recommendations, the median urinary iodine excretion is the most reliable indicator for assessment of a population’s iodine nutrition (WHO/UNICEF 2007). TSH is a sensitive measure of iodine status in the newborn period (Pardede et al. 1998; WHO/UNICEF/ICCIDD 2001). Thyroid size decreases slowly after iodine repletion and goiter rate may remain high for several years after introduction of iodized salt (Pardede et al. 1998; WHO/UNICEF/ICCIDD 2001).

Urinary iodine concentration is influenced by gender, age, sociocultural and dietary factors, drug interferences, geographical location and season (Als et al. 2000a). It is not a direct measure of thyroid function, but reflects recent iodine intake and thyroid hormone catabolism (Parede et al. 1998; van den Briel et al. 2001; Hambidge 2003). Above 90% of iodine is absorbed in the body and eventually appears in the urine. Therefore, urinary iodine excretion is considered a good marker of very recent dietary iodine intake (WHO/UNICEF/ICCIDD 2001). In physiological conditions, losses by perspiration and feces are negligible (Follis et al. 1962; Vought et al. 1963). According to WHO, a population's median urine iodine value is optimal between 100 and 200μg/l with no more than 20% of individual values less than 50μg/l (WHO/UNICEF/ICCIDD 2001). In non-pregnant, non-lactating women, a urinary iodine concentration of 100μg/l corresponds roughly to a daily iodine intake of about 150μg/day under steady state conditions (WHO/UNICEF/ICCIDD 2001).

Urine is a non-regulated body fluid and the concentration of iodine may vary even if the daily internal doses are kept constant. Generally, for this reason, either 24 hour urine samples must be obtained for analysis or spot samples must be corrected for dilutions (Caldwell et al. 2005). The disadvantages of spot urine samples include the variability in the volume of urine and the concentrations of endogenous and exogenous chemicals from sample to sample (Barr et al. 2005). Therefore, it cannot give a precise measure of iodine excretion in a single individual due to the large inter individual variations in 24 hour urinary volume (Knudsen et al. 2000). However, this variation tends to even out among populations (WHO/UNICEF/ICCIDD 2001). It has been demonstrated that there is a highly significant circadian rhythm in urinary iodine concentration with nadirs in early morning and peaks in the evening (Als et al. 2000a). Urinary biomonitoring data are typically adjusted to a constant creatinine concentration to correct for variable dilutions among spot samples. Traditionally, this approach has been used in population groups without much diversity (Barr et al. 2005). Creatinine adjustment involves dividing the analytic concentration (micrograms analytic per liter urine) by the creatinine concentration (grams creatinine per liter urine). Such correction is applied because it is assumed that excretion of creatinine is constant, not only between individuals but also within individuals throughout the day and on a daily basis (Furnee et al. 1994).

Spot samples have been used to assess iodine status in large population studies like the National Health and Nutrition Examination Survey (NHANES) (Hollowell et al. 1998) and were also used in this study. According to Bourdoux et al. (1985) the measurement of urinary iodine concentrations in fifty to one hundred spot samples gives a good index for iodine status in a population.

2.2 Mercury

Mercury is a natural element and as such it can neither be created nor destroyed, unlike many other toxicants, and the same amount has existed on the planet since the earth was formed (Pirrone and Barth 2007). It is relatively uncommon in the Earth's crust, from which it is liberated by natural processes such as erosion and volcanism as well as mining (Gochfeld 2003). Icelandic lava is however not high in mercury (Geirsson 1994). Mercury is the most dangerous of all the heavy metals (Salonen et al. 1995). It exists in various forms, elemental (or metallic), inorganic (e.g. mercuric chloride) and organic (e.g. methyl- and ethylmercury) (WHO 2007). All mercury compounds are toxic to humans and animals, but the organic forms, particularly methyl mercury and dimethyl mercury, have the highest toxicity (Gochfeld 2003).

From the atmosphere, mercury cycles from rainwater into lakes and oceans, where it is converted by microbial activity into organic methyl mercury. Inorganic mercury is poorly absorbed following ingestion and elemental mercury does not readily cross tissue barriers. In contrast, methyl mercury is readily absorbed and actively transported into tissues (EPA 1997). Approximately 95% of the methyl mercury ingested is absorbed and peak blood methyl mercury levels are reached within 6 hours. Methyl mercury readily crosses the blood-brain barrier and the placenta. Methyl mercury accumulates in the brain and is slowly converted to inorganic mercury (NRC 2000; WHO 2004).

2.2.1 Sources of mercury

Most of the mercury in the environment results from human activity, particularly from coal-fired power stations, residential heating systems and waste incinerators (WHO 2007). Significant releases of mercury to the environment result from the use of thermometers and blood pressure monitors, and from the incineration of medical waste (WHO 2005a; WHO 2005b). Mercury is an environmental contaminant that is present in seafood products largely as methyl mercury. Consumption of contaminated fish and shellfish is the main source of methyl mercury exposure, especially in populations that rely heavily on consumption of predatory fish (Gochfeld 2003; WHO 2008). Food sources other than seafood products may contain mercury but mostly in the form of inorganic mercury, which is considerably less toxic than methyl mercury (WHO 2008). In general, white meat fish such as cod and haddock tend to have lower mercury levels but also lower levels of long-chain n-3 fatty acids, whereas dark meat fish, such as swordfish, mackerel and other large long-lived predatory fish, tend to contain both more mercury and more long-chain n-3 fatty acids. Small fatty fish such as sardines and canned light tuna may contain relatively more fatty acids with less mercury (Oken et al. 2005). Methyl mercury has a half-life of approximately 2 years in fish. Larger, older fish such as swordfish and sharks therefore have the highest levels of methyl mercury (Maycock and Benford 2007). Methyl mercury in fish meat is bound to proteins. Skimming, trimming of fish, or cooking does not significantly reduce its concentration (Costa 2007).

In Iceland, as elsewhere, the maximum permissible level of contaminants in food items is subject to regulations. Should the content of a food product exceed the maximum level permitted, the product must not be distributed or sold. Two maximum levels apply to mercury, a lower maximum level for seafood that is consumed often and a higher level for seafood consumed rarely, such as shark and very large halibut. The Icelandic seafood which is eaten in the largest quantities is well below the lower maximum permissible level for mercury, e.g. haddock, shrimp, cod and halibut (Asmundsdottir et al. 2006). Mercury content in various Icelandic food items is summarized in Table 2.

Table 2: Mercury content in Icelandic food items

Food	Mercury (µg/100g)
Liverwurst[1]	3.9
Pork[1]	2.2
Chicken[1]	1.5
Cheese, 26%[1]	<2.3
Eggs[1]	3
Wheat[1]	<3.2
Haddock, raw[2]	2.53-4.31
Haddock, boiled[2]	4.13
Wild salmon, raw[2]	5.72
Prawn, boiled[2]	0.95-5.78
Halibut, raw[2]	10.6
Cod, raw[2]	6.5

[1] Reykdal et al 2000.
[2] ISGEM 2008.

Fish and shellfish are recommended dietary constituents providing high-quality proteins, vitamins and the fatty acids docosahexaenoic acid (DHA) and eicosapentaenoic acid (EPA). In addition, fish and shellfish provide a favourable ratio of n-6 to n-3 fatty acids (FNB 2005). The potential health risks related to fish consumption may be due to the presence of carcinogenic (e.g. dieldrin, heptachlor, PCBs, dioxin) and non-carcinogenic (e.g. methyl mercury) environmental contaminants in fish tissues. These contaminants are present in low levels in lakes, rivers, seas and oceans. However, the fish species can concentrate the environmental contaminants by bioaccumulation and biomagnification (Sidhu 2003). Fish contaminated with methyl mercury appears less protective against atherosclerosis (Landmark and Aursness 2004) than might be predicted based on its long-chain n-3 fatty acids content alone (Salonen et al. 1995). According to Houston (2007) mercury diminishes the protective effect of fish and long-chain n-3 fatty acids. The increasing daily fish consumption in health conscious modern societies may result in chronic low-level dietary intake of methyl mercury and thus can pose a significant toxicological problem, especially to susceptible groups such as developing embryos and fetuses (Castoldi et al. 2001). However the benefits of fish consumption usually outweigh the risks of mercury (Hibbeln et al. 2007).

2.2.2 Exposure limits for methyl mercury

The Provisional Tolerable Weekly Intake (PTWI) for total mercury is 5µg/kg body weight (FAO/WHO 1978) and for methyl mercury from 0.7µg/kg body weight (NRC 2000) to 1.6µg/kg body weight (WHO 2004; EFSA 2004; JECFA 2004). In January 2001 the U.S. Food and Drug Administration (FDA) issued a nutrition advisory for pregnant women to avoid consuming specified long-lived predatory fish, which may contain high levels of organic mercury and to limit ingestion of all other fish products (FDA 2001). A study performed after the dissemination of this national advisory showed that women reported reduced consumption of fish, including tuna, dark meat fish and white meat fish, whereas there was no change in shellfish consumption. The decline in total fish consumption included a decrease in the proportion of women who consumed more than three fish servings per week (Oken et al. 2003). In 2004 the U.S. Food and Drug Administration (FDA) and U.S. Environmental Protection Agency (EPA) issued a new nutrition advisory counselling advising pregnant women, women who may become pregnant, nursing mothers and young children to avoid some types of fish and eat fish and shellfish that are lower in mercury (HHS/EPA 2004).

Methyl mercury toxicity has been demonstrated at low exposure levels (WHO 2008). People who consume large amounts of fish (even species with relatively low mercury content) can accumulate sufficient levels of methyl mercury to cause symptoms and pregnant women can transfer to a fetus amounts of methyl mercury that are sufficient to impair nervous system development (Gochfeld 2003). Methyl mercury is removed from the body naturally, but it may take over a year for the levels to drop significantly. Therefore it may still be present in women when they become pregnant (HHS/EPA 2004). In Iceland, pregnant women and women who may become pregnant are recommended to consume fish at least twice a week. They are recommended to avoid raw fish like smoked and dried fish, pickled whale and sushi. They are also recommended to avoid swordfish, shark and cod liver (Public Health Institute of Iceland 2004).

2.2.3 Mercury toxicity

Concerns about exposure to mercury were first raised as the result of three severe exposure episodes (Myers and Davidson 2000). Two incidents took place in Japan and resulted from consumption over an extended period of time of highly contaminated fish during the 1950s and 1960s (Harada 1995). The third event occurred in Iraq during the early 1970s following consumption of seed grain that had been treated with a fungicide containing methyl mercury (Amin-Zaki et al. 1974). In all of these cases, children exposed before birth suffered the greatest symptoms of central nervous system injury, with the most severely affected exhibiting blindness, deafness, general paralysis, hyperactive reflexes and impaired mental development (Amin-Zaki et al. 1974).

Methyl mercury is a potent neurotoxin (Myers and Davidson 2000; Maycock and Benford 2007) though the mechanism by which this occurs is still unclear (Maycock and Benford 2007). The central nervous system is the principal target for the effects of methyl mercury in humans. Sensory, visual and auditory functions, which are concerned with coordination, are the most common functions to be affected (WHO 1990). The developing brain is thought to be the most sensitive organ for methyl mercury toxicity (WHO 2008). The placental barrier can stop many toxic elements, but methyl mercury is an exception in that it not only crosses the placenta, but it also accumulates at higher concentrations on the fetal side than on the maternal side (Iyengar and Rapp 2001). Even worse for the developing fetus, mercury also crosses the blood-brain barrier and exhibits long-term retention once it gets across (Kerper et al. 1992). The fetal brain may be affected even if the mother shows no signs of poisoning (Castoldi et al. 2001). In 2006, JECFA clarified that life-stages other than the embryo and fetus may be less sensitive to the adverse effects of methyl mercury (JECFA 2006). Unlike focal damage in adults, the developing brain shows a diffuse and widespread damage. Neurological symptoms include mental retardation, seizures, vision and hearing loss, delayed development, language disorders and memory loss. In children, a syndrome characterized by red and painful extremities called acrodynia has been reported to result from chronic mercury exposure (WHO 2007).

A study conducted in the Faroe Islands, where children were followed from birth to 7 years of age, indicated that higher blood methyl mercury concentration in the umbilical cord was associated with lower scores on several developmental and cognitive tests (Grandjean et al. 1997). Another study in the Faroe Islands showed that prenatal exposure to methyl mercury from contaminated seafood was associated with an increased risk of neurodevelopmental deficit (Steuerwald et al. 2000). Results from a prospective U.S. pregnancy and child cohort study, showed that higher mercury exposure in pregnancy was associated with lower offspring cognitive scores. In addition, higher maternal fish intake was associated with higher mercury levels. However, higher maternal fish consumption was associated with better infant cognition. This benefit appeared greatest among infants whose mothers consumed more fish but had lower mercury levels (Oken et al. 2005). The most likely explanation is that the benefit is conferred by consuming fish types with the combination of relatively little mercury and high amounts of beneficial nutrients (Oken et al. 2005).

2.2.4 Mercury and heart disease

The clinical consequences of mercury toxicity include hypertension, Coronary Heart Disease (CHD), Myocardial Infarction (MI), increase in Carotid Intimal Medial Thickness (IMT) and carotid obstruction, generalized atherosclerosis, renal dysfunction and proteinuria and an overall increase in total mortality (Houston 2007).

Two major epidemiological studies (Salonen et al. 1995; Guallar et al. 2002) have identified an adverse effect of methyl mercury exposure on cardiovascular endpoints. Salonen et al. (1995) reported an increased risk of coronary heart disease among residents of the Kuopio area in Finland whose hair samples had increased levels of mercury. The participants in that study had relatively high levels of mercury which were derived largely from locally contaminated freshwater fish. Guallar et al. (2002) showed an independent and graded association between toenail mercury levels and the risk of myocardial infarction. High levels of mercury appeared to be a risk factor for cardiovascular disease, attenuating the protective effects of long-chain n-3 fatty acids on cardiovascular health (Mozaffarian et al. 2005).

Mercury may also promote atherosclerosis and hence increase the risk of myocardial infarction in several ways. Among these, mercury promotes the formation of free radicals, binds to thiol groups and may induce lipid peroxidation. It also binds to selenium and reduces bioavailability of selenium for incorporation into glutathione peroxidase (Virtanen et al. 2007). The overall vascular effects of mercury include oxidative stress, inflammation, thrombosis, vascular smooth muscle proliferation and migration, endothelial dysfunction, dyslipidemia, immune dysfunction and mitochondrial dysfunction. All of these functional abnormalities have the potential to increase the risk for hypertension and vascular disease (Houston 2007).

2.2.5 Diagnosis of mercury poisoning

Exposures of mercury can be estimated by measuring pollutant levels in various body tissues e.g. hair, blood, umbilical cord, urine, human milk and nails (WHO/UNEP 2008). Because mercury is widely distributed in the environment, a small amount of mercury may be detected in urine and blood in those without history of exposure (Ng et al. 2007)

Total blood mercury is an indicator of methyl mercury exposure in people who consume fish and have no significant exposure to inorganic or elemental mercury (WHO 1990). The presence of mercury in blood indicates recent or current exposure. Methyl mercury in the diet is readily absorbed through the gastrointestinal tract and distributed throughout the body by the blood. Usually blood methyl mercury concentration reaches a maximum within 4 to 14 hours and undergoes clearance from the blood to other body tissues after 20 to 30 hours (WHO/UNEP 2008). Blood test cannot exclude the presence of mercury owing to its short half-life and it is more useful in detecting methyl mercury than inorganic mercury (Ng et al. 2007).

The presence of mercury in urine generally represents exposure to inorganic and/or elemental mercury. Urine test is a generally accepted, non-invasive and convenient method for mercury level detection, especially low-level exposure. Blood and urine tests together give a better measurement of mercury exposure (Ng et al. 2007).

Mercury level in hair reflects past exposure to mercury. However, hair analysis as a laboratory test has a serious intrinsic defect, namely irreproducibility of results (Ng et al. 2007). Once incorporated in the hair, mercury does not return to the blood, thus it provides a good long-term marker of exposure to methyl mercury (WHO/UNEP 2008).

3 Materials and methods

3.1 Study protocol

A randomly selected sample of 350 teenage girls (born in the years 1987-1992) living in the capital area was received from Statistics Iceland. From June 2007 to March 2008 the subjects received a letter, providing them with information about the study. Approximately one week after receiving the letter, the subjects were contacted by telephone and were invited to participate. Over the phone they had the opportunity to ask questions regarding the study protocol. On acceptance, an appointment was made. A text message was sent out one day prior to the visit at a clinic, the Centre for Antenatal Health Services, Reykjavik. Subjects were excluded if they had a disease that affected thyroid function or did not understand Icelandic. All subjects participating in the study, or legal guardian for those under 18 years of age, gave their informed consent. This study was approved by The National Bioethics Committee and The Icelandic Data Protection Commission.

3.2 Subjects

Data collection was conducted during the period of June 2007 to May 2008. Of the 350 girls invited to participate, 145 girls or 41% accepted to participate in the study. Of the 145 girls who accepted to participate, 112 completed the study or 32%. See Figure 1 for details. Of the girls invited to participate, 139 or 40% declined participation, the most common reasons being lack of time and lack of interest. 6% of the girls were excluded, main reasons being that they had moved from the capital area, did not speak the language, had thyroid disease or were mentally retarded. Of the 350 girls from the original sample approximately 13% could not be reached. Several could not be reached by phone in spite of repeated attempts and others had no registered phone numbers.

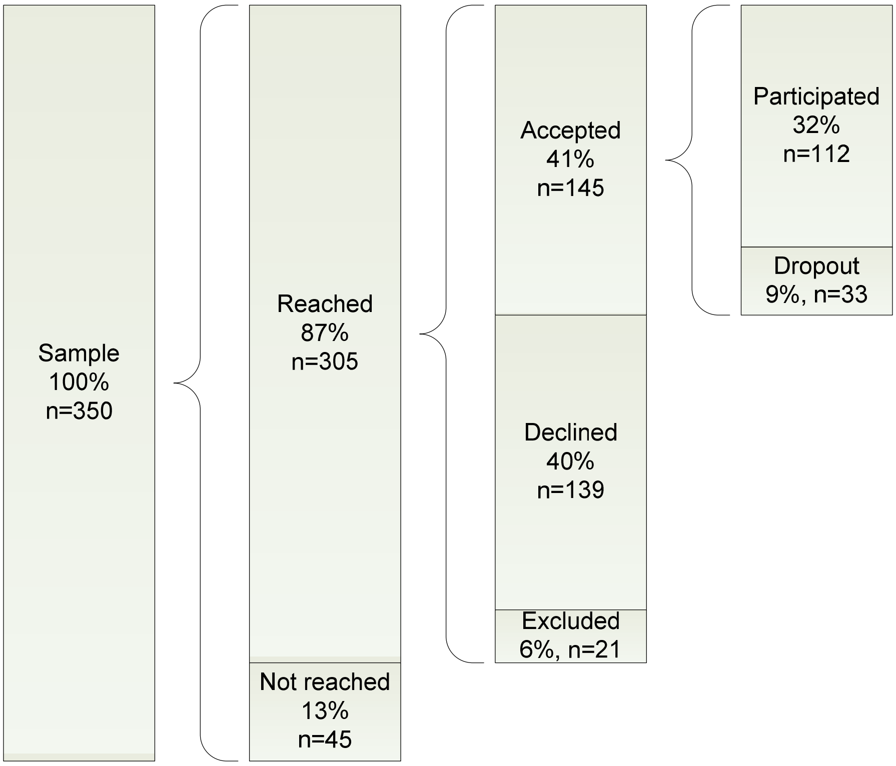

Figure 1: Distribution of subjects, percentage of original sample

3.3 Dietary assessment

Dietary intake was assessed by two Food Frequency Questionnaires (FFQ), one for total food consumption (total food FFQ) and another for seafood consumption (seafood FFQ). Of all methods to assess dietary intake, FFQ are the most frequently used in cohort studies in epidemiology. They are designed to assess usual eating habits over recent months or years and comprise a list of foods most informative about the nutrients or foods of interest. The main advantages of questionnaires are their ease and uniformity of administration, their low cost and their use with samples which are geographically widespread. The primary disadvantages are the amount of work required for their development and validation and the level of imprecision in the estimates of usual food consumption or nutrient intake (Margetts and Nelson 2006).

A total food FFQ was developed at the Icelandic Nutrition Council. It provides information on the consumption of 130 different food items and is designed to reflect intake over the previous three months. Portion sizes are estimated from pictures of three portion sizes of common food items and from general household measures. The National Nutrition Database

(ISGEM 2008) was used to evaluate the information. The total food FFQ was validated by Thorsdottir et al. (2004) and used e.g. in a study on the diet and lifestyle of women of childbearing age by Olafsdottir (2006).

To assess fish consumption and the intake of fish oil specifically, the participants also answered seafood FFQ about fish and fish oil consumption (Birgisdottir et al. 2008). This FFQ focuses solely on seafood consumption and does not provide information that might be critical in some studies to provide information on total diet. Therefore energy adjustments are not possible, nor can the FFQ be used to investigate diet-disease relationships fully. However, it can be useful for ranking individuals according to frequency of fish consumption, fish oil intake and in identifying consumers with low and high consumption of seafood (Birgisdottir et al. 2008).

3.4 Lifestyle questions

Subjects were asked about their smoking habits, if they had ever smoked and if they smoked today. Subjects were also asked if they used an oral contraceptive.

3.5 Anthropometry

The subjects' weight was measured to the nearest 100g, with light clothing and without shoes. Height was measured to the nearest 0.1cm using standard procedures and Body Mass Index (BMI) calculated. BMI is a simple index of weight-for-height that is commonly used to classify underweight, overweight and obesity in adults (WHO 2006).

3.6 Blood samples

TSH was measured with ELCIA (Electrochemiluminescence immunoasssy) using MODULAR ANALYTICS E170 from Roche. Measurements were conducted the same day blood samples were taken, at Landspitali-University Hospital in Reykjavik, Iceland.

Mercury was analysed in whole blood using Perkin Elmer AAnalyst 600 atomic absorption spectrometer using Zeeman background correction and equipped with transversely heated graphite furnace (THGA), a FIAS-400 flow injection station and autosamplers for both flame (AS-91) and furnace (AS-800). With each batch of samples that were taken through sample decomposition procedures at least one blank sample and at least one of each of the two reference samples were included. The measurement results for all such blank samples were used to calculate values for the limit of detection and limit of quantification respectively. Average values for the determinations of mercury in the two reference samples were well within the reference limits stated by their producer. The limit of detection was estimated as a value that is three times the standard deviation of the measurement results for all the digested blank samples. Blood samples with EDTA used as anticoagulant were kept frozen at -80°C at Landspitali-University Hospital in Reykjavik, Iceland until they were transported to Keldnaholt, Agricultural University of Iceland where they were analysed.

3.7 Urine samples

Urine spot samples were collected for iodine and creatinine measurements. Samples were collected in vials between 9:00am and 3:00pm.

Creatinine was measured with VITROS CREA Slide method using the VITROS CREA Slides and the VITROS Chemistry Products Calibrator Kit 1 (National Institute of Standards and Technology) on VITROS Chemistry Systems. The VITROS CREA Slide is a multilayered, analytical element coated on a polyester support. Measurements were conducted the same day urine samples were taken at Landspitali-University Hospital in Reykjavik, Iceland.

Urine samples, used for iodine measurements were kept frozen at -80°C at Landspitali-University Hospital in Reykjavik, Iceland, until all samples had been collected. The samples were sent with a courier in dry ice packages to the National Institute of Nutrition and Seafood Research (NIFES) in Bergen, Norway. An Agilent quadrupole ICP-MS 7500c (Yokogawa Analytical System Inc. Tokyo, Japan) was used as an iodine specific detector for urinary determination. Data was collected using the Agilent Chemstation ICP-MS chromatographic software (Julshamn et al. 2001; Dahl et al. 2003; Dahl et al. 2004). Certified reference material (CRM) (Seronorm™ Trace Elements; Nycomed, Norway) of iodine in human urine

was included in each analytical series of 25 samples in order to control the systematic errors of the analytical method.
Adjusting iodine excretion by creatinine excretion corrects for changes in water clearance and lean body mass. It has been shown to be more likely to represent the 24 hour excretion of iodine in a random spot urine sample than urinary iodine unadjusted for creatinine excretion (Thomson et al. 1996). In healthy populations, creatinine is excreted from the body at a relatively constant rate over time (Caldwell et al. 2005). Creatinine adjustment changes the distribution of individual measurements within the population and might either clarify or confound a possible association between urine iodine and thyroid deficiency (Haddow et al. 2007). The WHO guidelines for valid urine samples for occupational monitoring are often used. WHO recommends that if a sample is too dilute (creatinine concentration <30mg/dl) or too concentrated (creatinine concentration >300mg/dl), another urine sample should be collected (WHO 1996). In developing countries were protein intake and consequently creatinine excretion is low, iodine/creatinine ratio may be misleading and unreliable with regards to iodine deficiency (Furnee et al. 1994; WHO/UNICEF/ICCIDD 2001). In this thesis the results for iodine concentration are presented as µg/l and iodine creatinine ratio as µg/g.

3.8 Definitions

Iodine Deficiency Disorders (IDD) refers to all consequences of iodine deficiency in a population that can be prevented by ensuring that the population has an adequate intake of iodine (WHO/UNICEF 2007). A population's median urine iodine value is optimal between 100 and 200µg/l, with no more than 20% of individual values less than 50µg/l (WHO/UNICEF/ICCIDD 1994). See Table 3 for epidemiological criteria for assessing iodine nutrition based on median urinary iodine concentrations.

Table 3: Iodine intake based on median urinary iodine concentrations

Median urinary iodine (µg/l)	Iodine intake
<20	Insufficient
20-49	Insufficient
50-99	Insufficient
100-199	Adequate
200-299	Above requirements
≥300	Excessive

WHO/UNICEF/ICCIDD (2007).

Body Mass Index (BMI) is defined as the weight in kilograms divided by the square of the height in meters (kg/m^2). Underweight is defined as <18.5, normal weight as 18.5-24.99, overweight ≥25 and obese ≥30 (WHO 2006).

For subjects who had not reached 18 years of age, Cole et al. (2007) was used to define underweight subjects and Cole et al. (2000) was used to define overweight and obese subjects. International cut-off points for BMI by sex between 2 and 18 years were obtained by averaging data from Brazil, Great Britain, Hong Kong, Netherlands, Singapore and United States. See Table 4.

Table 4: BMI for underweight, overweight and obesity for females, age 15 to 18 years

Age years	Underweight kg/m^2	Overweight kg/m^2	Obesity kg/m^2
15.0	17.45	23.94	29.11
15.5	17.69	24.17	29.29
16.0	17.91	24.37	29.43
16.5	18.09	24.54	29.56
17.0	18.25	24.70	29.69
17.5	18.38	24.85	29.84
18.0	18.50	25.00	30.00

Adapted from Cole et al. 2000 and Cole et al. 2007.

3.9 Statistical analysis

The data were analysed using the computer program SPSS version 11 (SPSS Inc. 2003), and Microsoft Excel was used for simple calculations. All variables were checked for normal distribution with one Kolmogorov-Smirnov test. The level of significance was taken as $p<0.05$. Assessing relationship between two different variables was conducted with bivariate correlation, using Spearman’s rho for non-normally distributed variables. Non-parametric Mann-Whitney U-tests were used to assess the difference between two groups and to assess difference in means among three groups, non-parametric Kruskal Wallis H tests were used.

4 Results

4.1 Subjects

The subjects were born in the years 1987-1992 and the mean age was 17.7 years (range 15.1-20.3 years old). There was a similar distribution of residence in the original random sample and the subjects sample. As seen in Table 5 a large part of the subjects have residence in Reykjavik or 58.9% which was the same as the original sample.

Table 5: Distribution of residence

Town	Original sample (%)	Subjects (%)
Reykjavik	206 (58.9)	66 (58.9)
Kopavogur	53 (15.1)	18 (16.1)
Gardabær	25 (7.1)	8 (7.1)
Hafnarfjordur	44 (12.6)	11 (9.8)
Mosfellsbær	22 (6.3)	9 (8.1)
Total	350 (100)	112 (100)

Subjects were asked about their smoking habits and use of oral contraceptives. As shown in Table 6, 14% of the adolescent girls were current smokers and 32% had been smokers. 39% used oral contraceptives.

Table 6: Lifestyle questions concerning smoking habits and birth control use

	Yes n (%)	No n (%)
Have smoked	36 (32.4)	75 (67.6)
Currently smoke	16 (14.4)	95 (86.6)
Oral contraceptives	43 (38.7)	68 (61.3)

Information from one subject was missing, n=111.

The average weight of subjects was 63.6kg (SD 12.2) and the mean BMI 22.6kg/m^2 (SD 4.0) for all subjects. See Table 7. The subjects were divided into two groups based on their age, subjects born in the years 1987-1989 and subjects born in the years 1990-1992. The average weight for subjects born 1987-1989 was 67.1kg (SD 13.6) and for subjects born 1990-1992 the average weight was 60.4kg (SD 9.9). For subjects born 1987-1989 the mean BMI was 23.7kg/m^2 (SD 4.3) and for subjects born 1990-1992 the mean BMI was 21.7kg/m^2 (SD 3.4). As expected there was a significant difference between the two age groups for weight (p=0.007) and BMI (p=0.01) (Mann Whitney U-test)

Table 7: Anthropometrical measurements

	Total	Percentiles			1987-1989	1990-1992	
	Mean (SD)	25	50	75	n=54	n=58	p value[1]
Weight (kg)	63.6 (12.2)	56.2	61.7	67.2	67.1 (13.6)	60.4 (9.9)	0.007
Height (m)	1.68 (0.06)	1.64	1.67	1.73	1.68 (0.06)	1.67 (0.06)	0.08
BMI (kg/m^2)[2]	22.6 (4.0)	20.4	21.8	24.2	23.7 (4.3)	21.7 (3.4)	0.01

[1] Mann-Whitney U-test.
[2] The lowest BMI was 15.6 kg/m^2 and the highest was 39.7 kg/m^2.

As seen in Table 8, close to 71% of the subjects were normal weight, 8% were underweight and 21% were overweight or obese. Underweight was more common in the younger age group and overweight and obesity was more common in the older age group.

Table 8: Weight distribution

	1987-1989	1990-1992	All subjects
	n (%)	n (%)	n (%)
Underweight	2 (3.7)	7 (12.1)	9 (8.1)
Normal weight	37 (68.5)	42 (72.4)	79 (70.5)
Overweight	9 (16.7)	7 (12.1)	16 (14.3)
Obesity	6 (11.1)	2 (3.4)	8 (7.1)
Total	54 (100)	58 (100)	112 (100)

4.2 Urinary iodine

The mean urinary iodine concentration was 236µg/l (SD 169) for all the subjects and the median concentration 200µg/l. WHO recommends that if a sample is too dilute (creatinine concentration <30mg/dl) or too concentrated (creatinine concentration >300mg/dl), another urine sample should be collected (WHO 1996). None of the subjects had too diluted urine sample. However, 40 subjects had too concentrated urine sample, leaving 71 subjects with valid sample. When the subjects with invalid urine sample were excluded the mean urinary iodine concentration was 186µg/l (SD 145) and the median concentration 140µg/l. When iodine concentration was adjusted for urinary creatinine the mean urinary iodine concentration was 126µg/g (SD 99) and the median concentration 103µg/g. In the following sections, discussion on iodine concentration and frequency of iodine deficiency will be based on subjects with valid urinary sample (n=71). As shown in Table 9, 10-15% of the subjects had median iodine concentration below 50µg/l which is according to WHO defined as severe to moderate iodine deficiency (WHO/UNICEF/ICCIDD 1999). No correlation was between TSH and iodine.

Table 9: Distribution for TSH, creatinine, iodine and iodine/creatinine ratio (I/CR)

				Percentiles						
		n	Mean (SD)	5	10	15	50	85	90	95
TSH (mU/l)[1]	valid	100	2.0 (1.2)	0.7	0.8	1.1	1.8	2.8	3.4	4.9
Creatinine (mg/dl)[2]	valid	111	246 (139)	54	72	86	234	378	450	500
Iodine (µg/l)[2]	invalid	111	236 (169)	44	57	77	200	404	512	608
Iodine (µg/l)[3]	valid	71	186 (145)	41	50	55	140	284	390	490
I/Cr (µg/g)[3]	valid	71	126 (99)	36	41	49	103	194	207	299

[1] Blood samples were missing from 12 participants.
[2] Urine sample was missing from 1 participant.
[3] Samples from 40 participants were too concentrated and therefore excluded.

In Table 10 subjects were divided into six groups depending on their urinary iodine concentration according to WHO (WHO/UNICEF/ICCIDD 1999) in order to assess distribution. One subject was classified with severe iodine deficiency using iodine/creatinine ratio, 10% with moderate iodine deficiency, 31% with mild iodine deficiency and 4% had iodine/creatinine ratio above 300µg/g. Using urinary iodine concentration no subject had severe iodine deficiency, 10% of the subjects were classified with moderate iodine deficiency, 24% with mild iodine deficiency and 14% had iodine concentration above 300µg/g which according to WHO poses a risk of adverse health consequences.

Table 10: Urinary iodine status

Iodine nutrition	Iodine reference value (µg/l)	Iodine n=71 n (%)	I/Cr n=71 n (%)
Severe iodine deficiency	<20	0 (0)	1 (1.4)
Moderate iodine deficiency	20-40	7 (9.9)	10 (14.1)
Mild iodine deficiency	50-99	17 (23.9)	22 (31.0)
Optimal	100-199	19 (26.8)	30 (42.3)
Risk of iodine-induced hyperthyroidism	200-299	18 (25.3)	5 (7.0)
Risk of adverse health consequences	>300	10 (14.1)	3 (4.2)

Adapted from WHO/UNICEF/ICCIDD 1999.

4.3 Fish consumption

The subjects consumed fish on average 1.4 times per week (SD 0.9) and their average consumption was 14.8g/day (SD 13.3). Of the 112 subjects 54 (48.2%) consumed cod liver oil. Of the subjects who consumed cod liver oil the average intake was 4.5 times a week. See Table 11. Use of liquid cod liver oil capsules was more common than liquid cod liver oil (63% versus 37%).

Table 11: Distribution of fish consumption and cod liver oil

			Percentiles						
	n	Mean (SD)	5	10	25	50	75	90	95
Fish (times/week)[1]	112	1.36 (0.92)	0.25	0.30	0.63	1.0	2.5	2.5	2.5
Fish (g/day)[1]	112	14.8 (13.3)	1.7	3.0	6.0	11	21	29	44
Cod liver oil (times/week)[2]	54	4.5 (2.6)	0.5	0.5	2.0	5.0	7.0	7.0	7.0

[1] Total food FFQ.
[2] Seafood FFQ.

Subjects were asked to rank the fish species in the order they consumed most often. Haddock was the most commonly consumed fish specie or by 84%, salmon was second and cod was third. See Table 12.

Table 12: Fish species consumed most often

Fish specie	n (%)
Haddock	90 (84.1)
Cod	8 (7.5)
Salmon	7 (6.6)
Halibut	1(0.9)
Plaice	1 (0.9)
Total	107 (100)[1]

[1] 5 subjects did not consume fish.

Subjects were also asked if they consumed fish as a starter or a side dish and what kind of fish they consumed most often as a starter or a side dish. 38% of the subjects never consumed fish as a starter or a side dish. Tuna was consumed most often (47%), second was salmon (33%) and third was prawn (13%). See Table 13.

Table 13: Fish species consumed most often as a starter or a side dish

Fish species	n (%)
Tuna	33 (47.1)
Salmon smoked or cured	23 (32.9)
Prawn	9 (12.9)
Herring	3 (4.4)
Caviar	1 (0.9)
Sardine	1 (0.9)
Total	70 (100)[1]

[1] 42 subjects did not consume fish as a starter or a side dish.

As shown in Table 14 fish consumption was divided into tertiles and mean urinary iodine concentration and iodine/creatinine ratio shown for each tertile. Urinary iodine concentration or iodine/creatinine ratio was not different between tertiles of fish intake.

Table 14: Mean fish consumption, iodine and iodine/creatinine ratio (I/CR) by tertiles

	n	Fish g (SD)	Iodine µg/l (SD)	I/CR µg/l (SD)
Tertile 1	28	4.0 (2.2)	189 (142)	134 (134)
Tertile 2	23	11.6 (2.9)	189 (130)	127 (61)
Tertile 3	20	27.8 (11.9)	179 (171)	116 (77)
	71	13.2 (11.8)	186 (145)	126 (99)

4.4 Mercury

The subjects mean mercury concentration in blood was 1.47µg/l (SD 1.05) and the median 1.32µg/l. The highest mercury concentration was 5.51µg/l. See Table 15.

Table 15: Mercury concentration in blood

		Percentiles						
	Mean (SD)	5	10	25	50	75	90	95
Mercury (µg/l)	1.47 (1.05)	0.29	0.39	0.65	1.32	1.88	3.13	3.85

Mercury concentration, n=98.

As shown in Table 16, fish consumption in g/day was divided into tertiles and mercury concentration in blood shown for each tertile. Subjects in tertile 3, who consumed on average 29.4g of fish per day, had the highest mercury concentration in blood (1.86µg/l). Subjects in tertile 1, who consumed on average 4.2g of fish per day, had the lowest mercury concentration in blood (0.96µg/l). Fish consumption was positively correlated to mercury concentration in blood ($r=0.416$ and $p<0.001$) (Spearman's rho).

Table 16: Mercury concentration in blood and fish consumption by tertiles

	n	Fish g/day (SD)	Mercury µg/l (SD)
Tertile 1	36	4.2 (2.1)	0.96 (0.63)
Tertile 2	29	11.8 (3.0)	1.68 (0.63)
Tertile 3	33	29.4 (13.3)	1.86 (1.15)
	98	14.8 (13.3)	1.47 (1.05)

4.5 Milk consumption

The daily average consumption of milk and dairy products other than cheese was 539g/day. Intake of cheese was on average 20g/day. The total consumption of milk and all dairy products was equal to 3 portions per day[1]. Recommended amount of milk and dairy products for adolescent girls are 3 portions per day, however 60% of the adolescent girls consumed less than that. The recommended amount for adults are 2 milk portions per day and only 40% reached 2 portions per day. See Table 17.

Table 17: Milk and dairy product consumption in portions per day

			Percentiles						
	n	Mean (SD)	5	10	25	50	75	90	95
Milk and dairy products	112	3.0 (2.3)	0.7	1.0	1.4	2.2	4.0	6.2	7.9

In Table 18 the subjects were divided into tertiles depending on their milk and dairy product consumption and mean urinary iodine concentration and iodine/creatinine ratio shown for each tertile. Urinary iodine concentration was significantly higher in the group with the highest milk consumption compared to subjects with the lowest milk consumption (p=0.02) (Kruskal Wallis). Milk and dairy product consumption was positively correlated with urinary iodine concentration (r=0.275 and p=0.003) and iodine/creatinine ratio (r=0.520 and p<0.001) (Spearman's rho).

Table 18: Milk and dairy portions, iodine and iodine creatinine ratio (I/CR) by tertiles

	n	Milk and dairy portions (SD)	Iodine µg/l (SD)	I/CR µg/l (SD)
Tertile 1	23	1.19 (1.21)	156 (92)	89 (49)
Tertile 2	25	2.25 (0.44)	137 (96)	109 (63)
Tertile 3	23	5.87 (2.43)	270 (193)	182 (139)
	71	3.08 (2.44)	186 (145)	126 (99)

When milk and dairy products were divided into separate variables for milk and dairy products other than cheese, and cheese respectively, only milk was a significant predictor of urinary iodine concentration (r=0.305 and p=0.001) (Spearman's rho). There was a trend towards negative correlation between BMI and milk and dairy product consumption however it was only borderline significant (r=-0.180 and p=0.06). There was a positive correlation between fish consumption and milk and dairy product consumption (r=0.294 and p=0.002).

[1] One portion of milk and dairy products was estimated to be equal to 250g and one portion of cheese to be equal to 25g.

4.6 Iodine and mercury from the diet

The mean iodine intake was 170µg/day (SD 100) and the median 148µg/day. Only half of the subjects met the daily recommendations for iodine which is 150µg/day. The average requirement is estimated to be 100µg/day and close to 30% of the subject did not reach 100µg/day of iodine. The lower level of intake is 70µg/day and 10% of the subjects had iodine intake below the lower level of intake. The safe upper level for iodine is 600µg/day, none of the subjects had iodine intake above the upper level. See Table 19.

The subjects mean mercury intake was 2.4µg/day (SD 1.4) and the median 2.1µg/day. The subject with the highest mercury intake had mercury intake of 6.7µg/day. See Table 19.

Table 19: Distribution of iodine and mercury consumption

			Percentiles						
	n	Mean (SD)	5	10	25	50	75	90	95
Iodine (µg)	112	170 (100)	62	66	90	148	218	312	386
Mercury (µg)	112	2.4 (1.4)	0.7	0.9	1.3	2.1	2.9	4.5	5.8

Figure 2 shows from which food sources iodine in the diet originated. Milk and dairy products were the main contributors to iodine consumption providing 43% of the iodine, 24% came from fish and the rest from other food sources.

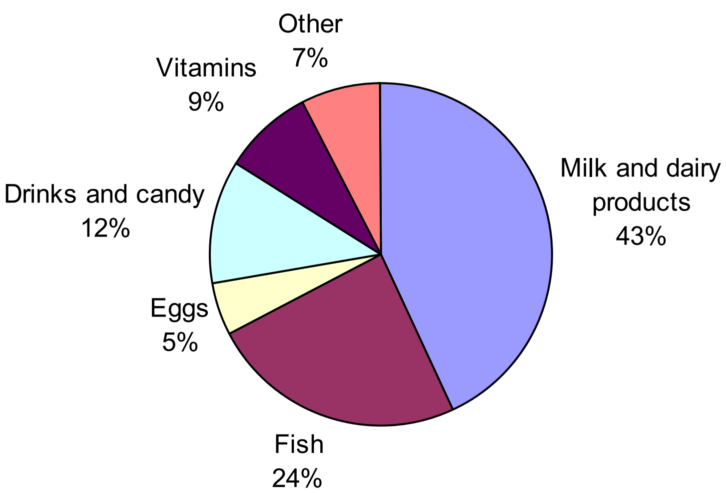

Figure 2: Origin of iodine in the diet

Figure 3 shows from which food sources mercury in the diet originated. Most of the mercury consumed came from fish or 39%, 15% from eggs, 14% from meat, 13% from cereals, 12% from milk and dairy products and the rest from other food sources.

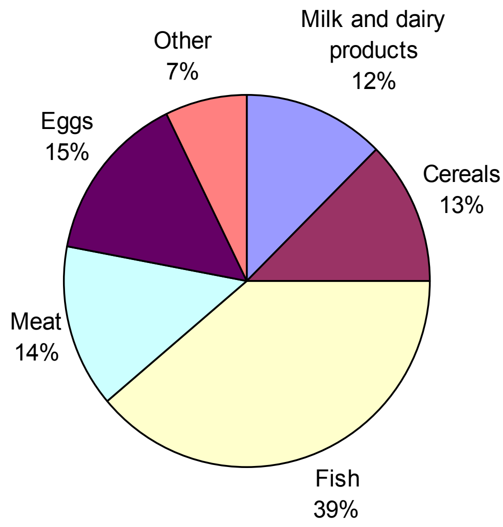

Figure 3: Origin of mercury in the diet

5 Discussion

The median urinary iodine status of the subjects was within the optimal range although in the lower end. Between 34-47% of the subjects were classified as having severe, moderate or mild iodine deficiency. The mean iodine intake was within the daily recommended intake, however 25% of the subjects had iodine intake below the average requirement. Milk and dairy products were the main source and determinant of iodine status. Fish intake was below the recommendations and fish intake was not directly related to iodine status. Mercury intake was well below exposure limits and mercury concentration in blood was below reference range as well.

5.1 Iodine status

Studies have been performed to assess the iodine status of the Icelandic nation. In 1939 the thyroid gland was measured and the results showed that the thyroid gland in the general Icelandic population was very small compared to the accepted normal size in other countries (Sigurjonsson 1940). Iodine status was measured in 1988 in people aged 20-59 years old. The mean iodine excretion for men was found to be 395µg/day (iodine/creatinine ratio 232.5µg/g) and for women 269.9µg/day (iodine/creatinine ratio 243.15µg/g). The results indicated that the iodine intake in Iceland was relatively high (Sigurdsson and Franzson 1988). Ten years later iodine status was measured in 66-70 year old Icelanders and the results showed lower iodine status (Laurberg et al. 1998). It was thought that the change towards lower values could be caused by changes in eating habits (Laurberg et al. 1998). In the present study iodine status of the subjects was considerably lower. The mean urinary iodine concentration was found to be 186µg/l (SD 145) and the median 140µg/l (iodine/creatinine ratio 126µg/g and the median 103µg/g). A population's median urine iodine value is optimal between 100 and 200µg/l, with no more than 20% of the individual values less than 50µg/l (WHO/UNICEF/ICCIDD 1994). According to this definition the iodine status of the subjects was optimal but in the lower end of the optimal range. However 10-15% of the subjects had iodine concentration below 50µg/l and 24-31% below 100µg/l which is noteworthy considering that Iceland has been known for its high iodine status.

A Swedish study found the median urinary iodine concentration to be 194µg/l in 7-9 years old children, 246µg/l in 15-17 year old teenagers and 190µg/l in 60-65 year old adults (Milakovic et al. 2004) which is somewhat higher than in the present study. In Sweden, a salt iodine supplementation programme was initiated in 1936 (Milakovic et al. 2004). However, the results in the present study are similar to results for the U.S. population in NHANES 2001-2002, where the median urinary iodine concentration was found to be 167.8µg/l and the median iodine creatinine/ratio 151.4µg/g. On average 12.3% (SD 2.4) of females aged 12-19 years old had iodine concentration below 50µg/l (Caldwell et al. 2005). This is in spite of available iodized salt in the U.S. (Hollowell et al. 1998).

Adolescent girls are future mothers to be. When pregnancy takes place in conditions with iodine restriction or deficiency, the more severe the iodine deficiency, the more obvious, frequent and profound the potential maternal and fetal impact. The main changes in thyroid function associated with pregnancy are related to increased hormone requirements, which begin in the first trimester of pregnancy. Increased hormone requirements can only be met by proportional increased hormone production, directly depending upon availability of iodine in the diet (Glinoer 2001). The most critical period for the fetus is from the second trimester of pregnancy to the third year after birth (WHO/UNICEF 2007). The fetal thyroid begins functioning around the 12th week of pregnancy. Before this, adequate maternal thyroid hormone is essential for normal human development (Dunn 2003). If iodine insufficiency leads to inadequate production of thyroid hormones and hypothyroidism during pregnancy, irreversible fetal brain damage can result (Public Health Committee of the American Thyroid Association 2006).

Iodine deficiency in pregnancy has been associated with an increased incidence of spontaneous abortion, stillbirth and congenital abnormalities (WHO 1996). In the U.S. overt hypothyroidism has been seen in about 0.3%-0.7% of women of reproductive age and subclinical hypothyroidism in approximately 2.5% (Smallridge et al. 2005). Als et al. (2000b) did a survey in Switzerland on subjects aged 0-90 years and the results showed that women of childbearing age (13-35 years old) had mild iodine deficiency. Adolescents reached their recommended threshold of >200µg/day iodine in only 13% of cases. A recent report from Boston, U.S. showed that approximately half of the pregnant women had urinary iodine concentration below 150µg/l and 9% had below 50µg/l, with the latter corresponding to severe to moderate iodine deficiency (Pearce et al. 2004). A study from the north east of

England, where the population is assumed to be replete in iodine, demonstrated insufficient urinary iodine concentration in about 50% of pregnant women (Kibirige et al. 2004).

5.2 Iodine intake

In the past, fish has been the most important source of iodine in the Icelandic diet, followed by milk and dairy products (Reykdal et al. 2000). In the Icelandic National Nutrition Survey 2002, for subjects aged 15-80 years old, fish was the main source of iodine providing 41% and milk and dairy products were the second main source providing 27% (Steingrimsdottir et al. 2003). In the present study milk and dairy products were the main sources of iodine, contributing 43% of the iodine intake and fish 24%. This is in accordance with studies from other countries. In Denmark milk and dairy products contributed 44% of iodine intake and fish 15% (Rasmussen et al. 2002). In the UK milk and dairy products were also found to be the main source accounting for 35% of the iodine intake whereas fish provided less than 10% (Lightowler and Davies 1998). In Norway milk and dairy products were also the main source of iodine, contributing 58% of the iodine intake and fish 18% (Brantsæter et al. 2007).

In the present study the mean iodine intake was 170µg/day and the median 148µg/day. Half of the subjects did not meet the daily recommendations and had iodine intake below 150µg/day. 25% did not meet the average requirement which is 100µg/day and 10% had daily intake below the lower level of recommended intake of 70µg/day (NNR 2004). In Norway, the mean iodine intake was found to be 136µg/day among women 16-79 years old (Dahl et al. 2004) and in Slovenia, 15 years old adolescents had median iodine intake of 156µg/day (Stimec et al. 2007). In the Icelandic National Nutrition Survey 2002 girls 15-19 years old consumed iodine on average 104µg/day (Steingrimsdottir et al. 2003). Long-term intakes below the lower level of intake are associated with an increased risk of developing deficiency symptoms (NNR 2004). In the present study, 10-15% of the subjects had urinary iodine concentration below 50µg/l which is defined as severe to moderate iodine deficiency. This is in accordance with the iodine intake but 10% of the subjects had iodine intake below 70µg/day though energy intake was underestimated by close to 45% of the subjects. Furthermore, the present study showed that none of the subjects had an intake of iodine above the safe upper limit of intake, 600µg/day (NNR 2004) which leaves room for increased consumption of fish or other iodine rich food like milk and dairy products.

5.3 Fish consumption

The food-based dietary guidelines published by the Public Health Institute of Iceland (2006) recommend that fish should be consumed at least twice a week, in addition to fish used as bread spread and in salads. Fish consumption in Iceland has for a long time been considered high relative to many other countries but in recent decades a dramatic decrease has occurred. In an Icelandic Nutrition Survey on 9-15 years old adolescents, 9 years old girls were reported to consume on average 22g/day and 15 years old 20g/day (Thorsdottir and Gunnarsdottir 2006). In the Icelandic National Nutrition Survey 2002 the average fish consumption was 15g/day for girls aged 15-19 years old (Steingrimsdottir et al. 2003). The present study showed similar results as the Icelandic National Survey 2002 where subjects consumed fish on average 14.8g/day and 1.4 times a week.

In the present study intake of fish was not directly related to urinary iodine. However 24% of the iodine in the diet came from fish. Iodine content is substantial in seafood or up to 190µg/100g in haddock compared to 11.2µg/100g in milk, 31.7µg/100g in eggs and 42µg/100g in cheese (ISGEM 2008). Haddock was the most commonly eaten fish specie among the subjects. A plausible explanation for lack of association is a large variation in urinary iodine excretion (Rasmussen et al. 1999). In the present study urinary iodine concentration was measured in spot samples which are a good marker of very recent dietary iodine intake (WHO/UNICEF/ICCIDD 2001) and the subjects answered total food FFQ which is designed to reflect intake over the previous three months. It is possible that had the subjects recorded their recent dietary intake before the urinary sample was collected, a correlation between fish consumption and urinary iodine concentration could have been found. Milk and dairy products are however consumed with high regularity, and maybe easier to recall than foods consumed less regularly like fish.

Recommendations to eat fish and other seafood are included in most national dietary guidelines (WHO 2003b) due to the positive health effects related to seafood consumption. Low intake of fish is of concern as fish provides a range of nutrients and contributes especially to the intake of vitamin D, iodine and selenium. Fatty fish is also a major source of long-chain n-3 fatty acids (NNR 2004). A large number of studies indicate an inverse association between intake of fish and risk for cardiovascular disease and coronary mortality (Hu et al. 2002; He et al. 2004). Seafood consumption has also been shown to have positive effects on weight loss. In a recent 8 week cross-European intervention study (Iceland, Spain and Ireland) young overweight men, that consumed regularly either fatty or lean fish, or fish oil as a part of energy-restricted diet, lost significantly more weight than men that had isocaloric energy-restricted diet without seafood (Thorsdottir et al. 2007). Additionally, the weight-loss diet including oily fish resulted in greater triglyceride reduction than a diet without fish or fish oil (Gunnarsdottir et al. 2008) and the consumption of fish oil in the diet exerted positive effects on insulin resistance independently from changes in body weight, triglycerides, erythrocyte membrane or adiponectin (Ramel et al. 2008).

5.4 Milk consumption

The recommendation for milk consumption is based on the notion that milk provides several nutrients, e.g. calcium, potassium, riboflavin and selenium. Milk fat is rich in saturated fatty acids, which is the main reason for recommending low-fat varieties (NNR 2004). The recommended intake of milk and dairy products is two glasses, bowls or cans a day, preferably products that are low in fat and added sugar. Cheese counts as a dairy product and 25g of cheese is equivalent to one portion (Public Health Institute of Iceland 2006). In the present study the subjects consumed on average 539g/day milk and dairy products other than cheese and 20g/day cheese compared to 358g/day milk and dairy products other than cheese and 38g/day cheese in the Icelandic National Nutrition Survey 2002 (Steingrimsdottir et al. 2003). In the present study the subjects consumed on average 3 portions of milk and dairy products per day which is in accordance with recommendations for adolescent girls in order to reach the daily recommendations for calcium (Thorsdottir and Gunnarsdottir 2006). However 60% of the subjects consumed less than 3 portions and only 40% consumed 2 portions per day. Half of the subjects did not reach the daily recommendations for calcium which are 1000mg for girls aged 14-17 years old and 800mg for women 18-30 years old (Public Health Institute of Iceland 2006). Maintaining adequate calcium intake during childhood and adolescence is necessary for the development of peak bone mass, which may be important in reducing the risk of fractures and osteoporosis later in life. Optimal calcium intake is especially relevant during adolescence, when most bone mineral accretion occurs (Institute of Medicine 1997). In the Icelandic National Nutrition Survey 2002 milk and dairy products were the most important calcium source in the diet providing 68% (Steingrimsdottir et al. 2003).

In the present study milk and dairy products were the main source of iodine. Milk consumption has decreased in the last decade especially among young people, while consumption of other dairy products like skyr has increased (Public Health Institute of Iceland 2008). This might have positive effects on the iodine status as milk contains 9.7-12.7µg/100g compared to 23.0µg/100g in skyr (ISGEM 2008). Correlations between urinary iodine excretion and milk and dairy products intake have been reported in numerous countries (Rasmussen et al. 2002; Girelli et al. 2004; Brantsæter et al. 2007). The contribution of iodine from milk and dairy products in these studies is primarily attributed to iodine fortification of industrially produced animal feed. Iodine was measured last in Icelandic agricultural products 1997 (Reykdal et al. 2000) and the database used to process the data is built on that information (ISGEM 2008). Today there is an ongoing study to update the database. Since 1997 composition of feed for agricultural animals has changed and iodine content of agricultural products is expected to have decreased considerably (Reykdal et al. 2007).

5.5 Cod liver oil consumption

Although not the main focus of this thesis, information on use of cod liver oil was gathered from the seafood FFQ. Until recently, there was a long time tradition of cod liver oil intake by the Icelandic population. However only 48% of the subjects consumed cod liver oil and only 21% consumed cod liver oil daily. This is in accordance with the Icelandic National Nutrition Survey 2002 where only 19% consumed cod liver oil daily (Steingrimsdottir et al. 2003). On average the subjects in the present study consumed cod liver oil 4.5 times a week. Of the subjects who consumed cod liver oil, 63% used cod liver oil capsules and 37% used liquid cod liver oil. The liquid cod liver oil has some advantages over the cod liver oil capsules. It contains more long-chain n-3 fatty acids, 1 tablespoon (10ml) contains 600mg EPA and 800mg DHA compared to cod liver oil capsules which contain considerably less, 109mg EPA and 151mg DHA in 6 capsules (Lysi 2008). EPA and DHA have been linked to the prevention of non-communicable diseases because they modify the key risk factors for cardiovascular disease in several ways (NNR 2004). Studies indicate that long-chain n-3 fatty acids have anti-arrhythmic, anti-thrombotic, anti-atherosclerotic and anti-inflammatory properties (Calder 2004). Liquid cod liver oil is the main source of long-chain n-3 fatty acids in the Icelandic diet, providing 42% of all long-chain n-3 fatty acids in the diet of the whole population (Steingrimsdottir et al. 2003).

Vitamin D is primarily synthesized in the skin after exposure to ultraviolet radiation (UVR) and less than 10% is derived from dietary sources (Norris 2001). Due to the latitude of the Nordic countries vitamin D deficiency may occur if the diet is devoid of the vitamin. Vitamin D is crucial for the regulation of calcium homeostasis, bone formation and for soft tissue health. Deficiency of vitamin D may result in defective mineralization with development of rickets in children and osteomalacia in adults (NNR 2004). Cod liver oil is an important source of vitamin D in the diet of the Icelandic population. The use of cod liver oil as a source of vitamin D during the winter months is highly recommended (Steingrimsdottir et al. 2003). The liquid cod liver oil has again some advantages over the cod liver oil capsules. The liquid cod liver oil contains more vitamin D, it contains 18.4µg in 10ml whereas the cod liver oil capsules contain 9µg in 6 capsules (Lysi 2008). Recommended daily intake of vitamin D is 10µg/day (Public Health Institute of Iceland 2006). From the total food FFQ the subjects answered, their vitamin D consumption was low. The average consumption was 5µg/day and 80% of the subjects consumed less than 10µg (data not shown).

5.6 Mercury

Fish and seafood are an important part of a healthy diet. Seafood is naturally rich in long-chain n-3 fatty acids, vitamin D and iodine, but also a dietary source of heavy metals and organic pollutants (Sioen et al. 2007a). The primary human exposure to methyl mercury is from fish consumption (Raymond and Ralston 2004). In the Icelandic National Nutrition Survey 2002 fish was the major source of mercury or 56% (Steingrimsdottir et al. 2003) and in the present study 39% of the mercury in the diet came from fish.

Exposure limits for mercury have been established, the PTWI for total mercury is 5µg/kg body weight (FAO/WHO 1978) and for methyl mercury from 0.7µg/kg body weight (NRC 2000) to 1.6µg/kg body weight (WHO 2004; EFSA 2004; JECFA 2004). When PTWI was calculated for the average weight of the subjects in the present study, 63.6kg, the PTWI was found to be 45µg/day. The subjects in the present study all had mercury intake well below the PTWI. The subjects consumed mercury on average 2.4µg/day. There was a positive correlation between fish consumption and mercury concentration in blood, which is not surprising considering that all fish consumers are exposed to some level of methyl mercury.

The presence of mercury in blood indicates recent exposure to mercury. There is a direct relationship between mercury concentrations in human blood and consumption of fish contaminated with methyl mercury. WHO considers the normal mean concentration of total mercury in blood to be between 5 to 10µg/l in individuals with no consumption of contaminated fish (UNIDO 2003). The NRC identifies 2µg/l as the normal mean concentration for populations with little or no fish consumption (NRC 2000). In the present study the subjects mean blood mercury concentration was found to be 1.48µg/l (SD 1.05) and the median concentration 1.32µg/l. In an Icelandic study on pregnant women the mean blood mercury concentration was found to be 2.9µg/l (AMAP 1997). One subject in the present study had blood concentration of 5.51µg/l. Using both the reference range from WHO for individuals and from NRC for populations the blood mercury concentration in the subjects is safe.

It is of great importance that consumption of mercury is low. The toxic effects of methyl mercury can result in a potential health problem and it is listed by the International Program of Chemical Safety as one of the most dangerous chemicals in the environment (Gilbert and Grant-Webster 1995). The risk from mercury in fish and shellfish depends on the amount eaten and the levels of mercury in the fish and shellfish. The subjects in the present study are future mothers to be and this is an important matter considering that the FDA and the EPA are advising women who may become pregnant, pregnant women, nursing mothers and young children to avoid some types of fish and eat fish and shellfish that are lower in mercury (2004).

5.7 Actions against iodine deficiency

Iodine deficiency occurs when iodine intake falls below the recommended level. In nearly all countries where iodine deficiency occurs, it is well recognized that universal salt iodization is the most effective way to achieve virtual elimination of IDD (WHO 2007). Iodized table salt is available in Denmark, Sweden, Finland and Norway and contributes to iodine intake (NNR 2004). In the present study, part of the subjects had low iodine status whereas another part of the subjects had high iodine status. Searching for the best way to optimize iodine status of the Icelandic population is something that requires consideration. The Public Health Institute of Iceland (2006) recommends that salt consumption should not be more than 6g/day for women and 7g/day for men. This recommendation is based on evidence for association between dietary sodium chloride intake and blood pressure (NNR 2004). In the Icelandic National Nutrition Survey 2002 average salt consumption was estimated to be 9g/day (Steingrimsdottir et al. 2003). Considering that salt consumption is high in Iceland it would probably not be the most optimal way to recommend salt in the diet for people to reach the recommendations for iodine. In Norway salt enriched with iodine is not allowed in the food industry and there is very limited use in the private household (Dahl et al. 2004). Iodization of cow feed has been more important for the iodine intake in Norway than iodized table salt (Dahl et al. 2003). In Northern Europe use of fortified feed is more widespread than in Southern Europe, where pasture is available for a greater part of the year, which may partially explain why countries in Northern Europe are more likely to be iodine sufficient (Dahl et al. 2003). In Norway and Iceland milk and dairy products are the main sources of iodine in the diet which highlights the importance to continue iodization of cow feed. Fortification of cow feed seems to be an effective way to provide a population with sufficient iodine (Dahl et al. 2003). In Norway iodine fortification of cow feed is mandatory but this is not the case in Iceland. This underlines the importance of cooperation between the Public Health Sector of Iceland and the Ministry of Agriculture.

Marine fish is the richest natural food source of iodine, however the contribution of iodine is low because fish is consumed to a lesser extent (Haldimann et al. 2005). An intervention study conducted in Ghana showed that goitrous individuals had a significant ability to improve their iodine status following 14 days on a diet containing elevated amounts of iodine due to marine fish inclusion (Maage et al. 2008). Even though the use of iodized salt is the major alleviating factor for iodine deficiency, for parts of the population it is not always efficient. Diets containing fish could be a helpful supplement in alleviating the IDD in some areas (Maage et al. 2008).

Regular intake of milk and dairy products as well as fish products is important to meet the iodine requirements in the Icelandic diet. It is known that important health-related lifestyles choices, like dietary patterns, are already established during childhood and adolescence (Sioen et al. 2007b). Food habits change significantly during adolescence and health promotion during the adolescent years therefore ought to be encouraged (Post-Skagegard et al. 2002). Adolescents are easily influenced by movements and changes in the society (Fogelholm 1998). It is therefore vital that young people are guided towards a healthy eating pattern.

Knowing that the consumption of seafood can play a role in the prevention of chronic diseases and that processes such as atherosclerosis may start during teenage years, it can be stated that creating the habit of regular seafood consumption at a younger age can create health benefits favourable now and later (Sioen et al. 2007b). In the present study the mean iodine intake was found to be in the range of sufficient, even though there were some subjects with very low intake of iodine. Certain groups of the population might be at risk of low iodine intake, e.g. subjects with allergy to milk or fish, vegetarians who do not consume fish, milk and dairy products and others with a low consumption of milk and fish. The study verifies the importance to encourage a regular intake of milk and dairy products as well as seafood, in order to meet the requirements for iodine.

5.8 Limitations

As the study sample is not big, its representativeness could be questioned. It is possible that individuals, who live a healthier lifestyle, might be more willing to participate in a health oriented study, thereby biasing the results. Factors that might affect healthy lifestyle are e.g. social status (Darmon and Drewnowski 2008), body weight (Rashad 2005), smoking (Dallongeville et al. 1998) and health promotion (Sjostrom et al. 1999).

In the present study 21.4% of the subjects were overweight or obese compared to 15% of women aged 15-24 years old in the Icelandic National Nutrition Survey 2002 (Steingrimsdottir et al. 2002). There is however a difference in the assessment of weight between the two surveys. In the Icelandic National Nutrition Survey 2002 the subjects self reported their weight and in the present study the subject were weighed and their height measured. A U.S. study showed that obese women who seek weight loss assistance tended to under-report their weight and over-report height, suggesting that self-reported data is likely to be inaccurate (Nawaz et al. 2001). Another factor that might affect the results is that close to 45% of the subjects under-reported their energy consumption and 76% of the overweight and obese subjects (data not shown). Under-reporting of food intake is one of the fundamental obstacles preventing the collection of accurate habitual dietary intake data. Women are more likely to under-report than men (Macdiarmid and Blundell 1998) and a high BMI has been related to a higher degree of under-reporting for adolescents as well as for adults (Bandini et al. 1990; Bratteby et al. 1998; Johansson et al. 1998). Ballard-Barbash et al. (1996) reported the prevalence of underreporting to be as high as 71% in overweight women (BMI>27.3). Another limitation could be that underreporters have been shown to specifically report less of high sugar and fatty foods (Olafsdottir et al. 2006). Consumption of food considered healthy might also be overestimated. Overestimation by FFQ are found to be especially amplified for food perceived as healthy and socially acceptable, such as fruits and vegetables (Amanatidis et al. 2001). Similar overestimation might be likely for seafood (Birgisdottir et al. 2008).

Based on the common understanding that iodine concentration in most food items is low and that only a few food items are regarded as good sources of iodine, the frequency and the amount of intake of these iodine rich sources are of great importance. In general, no dietary survey methods exist today which allow estimation of the dietary intake of one or more individuals without weaknesses being connected to the data. Estimating the intake of specific nutrients, such as iodine, is further complicated by the fact that one of the two major sources of the element, i.e. fish, is normally not eaten daily. There are also more sources of errors when assessing iodine intake by a FFQ, e.g. choice of food portion sizes, frequency of intake and particularly the values used for iodine content in food, than by measuring iodine excretion (Rasmussen et al. 2002). The advantage of a biological reference is that it does not depend on the respondent's ability to recall or report dietary intake correctly, which is a major source of error in all dietary assessments (Kaaks 1997). Due to weakness factors in nutrition epidemiological methods, biomarkers and other indicators of status or intake are of importance.

Another limitation could be that urinary iodine was measured with a spot sample and not a 24 hour urine collection. When measuring iodine, preferably 24 hour urine collection should be obtained. However as compliance might be low for 24 hour collections, estimates based on spot urine samples are often preferred (Knudsen et al. 2000). The major disadvantages of a spot urine sample include the variability in the volume of urine and the concentration of endogenous and exogenous chemicals from sample to sample (Barr et al. 2005). To compensate for the lack of a 24 hour urine collection, in populations with adequate nutrition, the creatinine concentration has been used to adjust for factors that may affect the concentrations. Adjusting iodine excretion by creatinine has been shown to be more likely to represent 24 hour excretion of iodine in spot samples than urinary iodine unadjusted for creatinine excretion (Thomson et al. 1996). In the present study the urinary iodine was adjusted for creatinine excretion and the results might therefore more likely represent 24 hour urine collection.

Additionally, considering that the database the results are built on has not been updated since 1997 (Reykdal et al. 1997) there might be some errors in the results of iodine intake. Today there is an ongoing study to update the database. It is known that the composition of feed for agricultural animals has changed and iodine content of agricultural products is expected to have decreased (Reykdal et al. 2007).

6 Conclusion

The median urinary iodine status of the subjects was within the lower end of the optimal range. 10-15% of the subjects were defined as having severe to moderate iodine deficiency and additionally 24-31% were defined as having mild iodine deficiency. The average iodine intake was also within recommendation. However 10% of the subjects had iodine intake below the lower level of recommended intake and 25% below the estimated average requirement. Milk and other dairy products as well as fish were the main sources of iodine from the diet. Average milk and dairy product consumption was within the recommendations. 60% of the subjects however consumed less than 3 portions per day and 40% consumed less than 2 portions per day. Milk and dairy products were the major source and determinant of iodine status. Fish intake was below the recommendations, the subjects consumed on average 14.8g fish per day. Fish intake was not found to be directly related to iodine status in the present study. A positive correlation between fish consumption and mercury concentration in blood was seen. However mercury intake was well below exposure limits and mercury concentration in blood was below reference range as well.

The iodine status of the Icelandic adolescent girls was within the optimal range however there were subjects with low iodine status and subjects with high iodine status. Salt iodization is considered to be the most efficient way to improve iodine intake. However, before decisions are made concerning iodization of salt in Iceland all benefits and drawbacks need to be considered. Today the key to increase iodine intake in subjects with low iodine status might lie in added encouragement to increase consumption of iodine rich food items like fish and milk and dairy products.

7 References

Als, C., Helbling, A., Peter, K., Haldimann, M., Zimmerli, B. & Gerber, H. (2000a). Urinary iodine concentration follows a circadian rhythm: A study with 3023 spot urine samples in adults and children. *The Journal of Clinical Endocrinology and Metabolism, 85*(4), 1367-1369.

Als, C., Keller, A., Minder, C., Haldimann, M. & Gerber, H. (2000b). Age- and gender-dependent urinary iodine concentrations in an area-covering population sample from the Bernese region in Switzerland. *European Journal of Endocrinology / European Federation of Endocrine Societies, 143*(5), 629-637.

AMAP (Artic Monitoring and Assessment Programme) (1997). *A State of the Artic Environment Report.* Oslo: Artic Pollution.

Amin-Zaki L., Elhassani S. B., Majeed M. A., Clarkson T.W., Doherty R. A. & Greenwood M. R. (1974). Intrauterine methyl mercury poisoning in Iraq. *Pediatrics, 54*, 587–595.

Asmundsdottir, A. M., Baldursdottir, V., Rabieh, S. & Gunnlaugsdottir, H. (2008). *Undesirable substances in seafood products - Results from monitoring activities in 2006.* Reykjavik: Matis-Food Research, Innovation and Safety.

Ballard-Barbash, R., Graubard, I., Krebs-Smith, S. M., Schatzkin, A. & Thompson, F. E. (1996). Contribution of dieting to the inverse association between energy intake and body mass index. *European Journal of Clinical Nutrition, 50*(2), 98-106.

Bandini, L. G., Schoeller, D. A., Cyr, H. N. & Dietz, W. H. (1990). Validity of reported energy intake in obese and nonobese adolescents. *The American Journal of Clinical Nutrition, 52*(3), 421-425.

Barr, D. B., Wilder, L. C., Caudill, S. P., Gonzalez, A. J., Needham, L. L. & Pirkle J. L. (2005). Urinary creatine concentrations in the U.S. population: Implications for urinary biologic monitoring measurements. *Environmental Health Perspectives, 113*, 192-200.

Bettendorf, M. (2002). Thyroid disorders in children from birth to adolescence. *European Journal of Nuclear Medicine, 29,* 439-446.

Birgisdottir, B. E., Kiely, M., Martinez, J. A. & Thorsdottir, I. (2008). Validity of a food frequency questionnaire to assess intake of seafood in adults in three European countries. *Food control, 19,* 648-653.

Bourdoux, P., Delange, F., Filetti, S., Thilly, C. & Ermans, A. M. (1985). Reliability of the iodine/creatinine ratio: a myth? In Hall R. & Kobberling J. (Eds.). *Thyroid disorders associated with iodine deficiency and excess.* New York: Raven Press.

Brantsæter, A. L., Haugen, M., Julshamn, K., Alexander, J. & Meltzer, H. M. (2007). Evaluation of urinary iodine excretion as a biomarker for intake of milk and dairy products in pregnant women in the Norwegian Mother and Child Chort Study (MoBa). *European Journal of Clinical Nutrition,* 1-8.

Bratteby, L. E., Sandhagen, B., Fan, H., Enghardt, H. & Samuelson, G. (1998). Total energy expenditure and physical activity as assessed by the doubly labelled water method in Swedish adolescents in whom energy intake was underestimated by 7-d diet records. *American Journal Clinical Nutrition 67*, 905-911.

Calder, P. C. (2004). Fatty acids and cardiovascualar disease: Evidence explained and mechanisms explored. *Clinical Science, 107*, 1-11.

Caldwell, K. L., Jones, R. & Hollowell, J. G. (2005). Urinary iodine concentration: United States national health and nutrition examination survey 2001-2002. *Thyroid, 15*, 692-699.

Castoldi, A. F., Coccini, T., Ceccatelli, S. & Manzo, L. (2001). Neurotoxicity and molecular effects of methyl mercury. *Bain Research Bulletin, 55*, 197-203.

Cole, T. J., Bellizzi, M. C., Flegal, K. M., & Dietz, W. H. (2000). Establishing a standard definition for child overweight and obesity worldwide: International survey. *BMJ (Clinical Research Ed.), 320*(7244), 1240-1243.

Cole, T. J., Flegal, K. M., Nicholls, D., & Jackson, A. A. (2007). Body mass index cut offs to define thinness in children and adolescents: International survey. *BMJ (Clinical Research Ed.), 335*(7612), 194.

Costa, L. G. (2007). Contaminants in fish: Risk-benefit considerations. *Arhiv Za Higijenu Rada i Toksikologiju, 58*(3), 367-374.

Dahl, L., Johansson, L., Julshamn, K. & Meltzer, H. M. (2004). The iodine content of Norwegian foods and diets. *Public Health Nutrition, 7*, 569-576.

Dahl, L., Opsahl, J. A., Meltzer, H. M. & Julshamn, K. (2003). Iodine concentration in Norwegian milk and dairy products. *The British Journal of Nutrition, 90*, 679-85.

Dallongeville, J., Marecaux, N., Fruchart, J. C. & Amouyel, P. (1998). Cigarette smoking is associated with unhealthy patterns of nutrient intake: A Meta-analysis. *The Journal of Nutrition, 128,* 1450-1457.

Darmon, N. & Drewnowski, A. (2008). Does social class predict diet quality? *American Journal of Clinical Nutrition, 87,* 1107-1117.

Delange, F. (2001). Iodine deficiency as a cause of brain damage. *Postgraduate Medical Journal, 77*(906), 217-220.

Delange, F. (2002). Iodine deficiency in Europe and its consequences: An update. *European Journal of Nuclear Medicine and Molecular Imaging, 29 Suppl 2*, S404-16.

Delange, F., Benoist, B., Pretell, E. and Dunn, J. T. (2001). Iodine deficiency in the World: Where do we stand at the turn of the century? *Thyroid 11*, 437-447.

Dunn, J. T. (1998). What's happening to our iodine? *The Journal of Clinical Endocrinology and Metabolism, 83*(10), 3398-3400.

Dunn, J. T. (2003). Iodine should be routinely added to complementary foods. *The Journal of Nutrition, 133,* 3008S-3010S.

Dunn, J. T. (2006). Iodine. In: Shils M. E., Shike M., Ross A. C., Caballero B. & Cousins R.J. (Eds.). *Modern Nutrition in Health and Disease 10th ed.* Philadelphia: Lippincott Williams and Wilkins.

EFSA (European Food Safety Agency) (2004). Opinion of the scientific panel on contaminants in the food chain on a request from the commission related to mercury and methylmercury in food. The EFSA Journal, 34, 1-14.

European Commission. Health and Consumer Protection Directorate-general (2002). *Opinion of the Scientific Committee on food on the tolerable upper intake level of iodine.* Retrieved September 13th 2008, from, http://ec.europa.eu/food/fs/sc/scf/out146_en.pdf.

EPA (U.S. Environmental Protection Agency) (1997). *Mercury Study report to Congress*. Retrieved August 4th 2008, from, http://www.epa.gov/Mercury/report.htm.

FAO/WHO (Food and Agriculture Organization and World Health Organization) (1978). *Evaluation of certain food additives and contaminants. Twenty-second report of the Joint FAO/WHO Expert Committee on Food Additives*. Geneva: World Health Organization.

FDA (Food and Drug Administration) (2001). *Consumer advisory: An important message for pregnant women and women of childbearing age who may become pregnant, nursing mothers, and young children.* Retrieved August 15th 2008, from, http://vm.cfsan.fda.gov/~dms/admehg.html.

FDA/EPA (U.S. Food and Drug Administration and US Environmental Protection Agency) (2004). *What you need to know about Mercury in fish and shellfish*. Retrieved June 26th 2008, from, http://www.cfsan.fda.gov/~dms/admemercury3.html.

Flueury, Y., Melle, G. V., Woringer, V., Gaillard, R. C. & Portmann, L. (2001). Sex-dependent variations and timing of thyroid growth during puberty. *The Journal of Clinical Endocrinology and Metabolism, 86,* 750-754.

FNB (Food and Nutrition Board) (2005). *Dietary reference intakes for energy, carbohydrate, fiber, fat, fatty acids, cholesterol, protein, and amino acids (Macronutrients).* Retrieved July 12th 2008 from, http://www.nap.edu/openbook.php?isbn=0309085373

Fogelholm, M. (1998). Diet, physical activity and health in Finnish adolescents in the 1990s. *Scandinavian Journal of Nutrition, 42*, 10-12.

Follis, R. H., Vanprava, K. & Damrongsakdi, D. (1962). Studies on iodine nutrition in Thailand. *The Journal of Nutrition, 76,* 159–173.

Furnee, C. A., van der Haar, F., West, C. E. & Hautvast, J. G. (1994). A critical appraisal of goiter assessment and the ratio of urinary iodine to creatinine for evaluating iodine status. *The American Journal of Clinical Nutrition, 59*(6), 1415-1417.

Geirsson, K. (1994). *Natturuleg viðmiðunargildi á styrk þungmalma i islensku umhverfi.* Kopavogur: Siglingamalastofnun rikisins.

Gilbert, S. G. & Grant-Webster, K. S. (1995). Neurobehavioral effects of developmental methylmercury exposure. *Environmental Health Perspectives, 103 Suppl 6*, 135-142.

Girelli, M. E., Coin, P., Mian, C., Nacamulli, D., Zambonin, L., Piccolo, M., Vianello-Dri, A., Gottardo, F. & Busnardo, B. (2004). Milk represents an important source of iodine in schoolchildren of the Veneto region, Italy. *Journal of Endocrinological Investigation, 27*(8), 709-713.

Glinoer, D. (1995).The thyroid in pregnancy: a European perspective. *Thyroid Today, 58*, 1-11.

Glinoer, D. (2001). Pregnancy and iodine. *Thyroid : Official Journal of the American Thyroid Association, 11*(5), 471-481.

Gochfeld, M. (2003). Cases of mercury exposure, bioavailability, and absorption. *Ecotoxicology and Environmental Safety, 56*(1), 174-179.

Grandjean, P., Weihe, P., White, R. F., Debes, F., Araki, S., Yokoyama, K., Murata, K., Sorensen, N., Dahl, R. & Jorgensen, P. J. (1997). Cognitive deficit in 7-year-old children with prenatal exposure to methylmercury. *Neurotoxicology and Teratology, 19*(6), 417-428.

Guallar, E., Sanz-Gallardo, M. I., van't Veer, P., Bode, P., Aro, A., Gomez-Aracena, J., Kark, J. D., Riemersma, R. A., Martin-Moreno, J. M. & Kok, F. J. (2002). Mercury, fish oils, and the risk of myocardial infarction. *The New England Journal of Medicine, 347*(22), 1747-1754.

Gunnarsdottir, I., Tomasson, H., Kiely, M., Martinez, J. A., Bandarra, N. M., Morais, M. G. & Thorsdottir, I. (2008). Inclusion of fish or fish oil in weight-loss diets for young adults: Effects on blood lipids. *International Journal of Obesity (2005), 32*(7), 1105-1112.

Haddow, J. E., McClain, M. R., Palomaki, G. E. & Hollowell, J. G. (2007). Urine iodine measurements, creatine adjustment, and thyroid deficiency in an adult United States population. *The Journal of Endocrinology and Metabolism, 92*, 1019-1022.

Haldimann, M., Alt, A., Blanc, A. & Blondeau, K. (2005). Iodine content of food groups. *Journal of Food Composition and Analysis, 18,* 461-471.

Hambidge, M. (2003). Biomarkers of trace mineral intake and status. *The Journal of Nutrition, 133*, 948S-955S.

Hanna, C. E. & LaFranchi, S. H. (2002). Adolescent thyroid disorders. *Adolescent Medicine, 13*, 13-35.

Harada, M. (1995). Minamata disease: Methyl mercury. Poisoning in Japan caused by environmental pollution. *Critical Reviews in Toxicology, 25*, 1-24.

Harrison, M. T., Harden, R., Alexander, W. D. & Wayne, E. (1965). Iodine balance studies and the availability of iodine. *Proceedings of the International Thyroid Conference, Rome*, 241-248.

He, K., Song, Y., Daviglus, M. L., Liu, K., Van Horn, L., Dyer, A. R. & Greenland, P. (2004). Accumulated evidence on fish consumption and coronary heart disease mortality: A meta-analysis of cohort studies. *Circulation, 109*(22), 2705-2711.

Hetzel, B. S. (1983). Iodine deficiency disorders (IDD) and their eradication. *Lancet, 2*, 1126-1127.

Hetzel, B. S. (1986). Iodine deficiency disorders (IDD): A maternal and child health issue. In Jelliffe, D. B. & Jelliffe, E.F.P. (Eds). *Advances in international maternal and child health Volume 6*. Oxford: Clarendon Press.

HHS/EPA (U.S. Department of Health and Human Services and US Environmental Protection Agency) (2004). *What you need to know about mercury in fish and shellfish. 2004 EPA and FDA Advice for Women who might become pregnant, women who are pregnant, nursing mothers and young children*. Retrieved August 4th 2008, from, http://www.cfsan.fda.gov/~dms/admehg3.html.

Hibbeln, J. R., Davis, J. M., Steer, C., Emmett, P., Rogers, I., Williams, C. & Golding, J. (2007). Maternal seafood consumption in pregnancy and neurodevelopmental outcomes in childhood (ALSPAC study): An observational cohort study. *Lancet, 369*(9561), 578-585.

Hollowell, J. G., Staehling, N. W., Hannon, W. H., Flanders, D. W., Gunter, E. W., Maberly, G. F., Braverman, L. E., Pino, S., Miller, D. T., Garbe, P. L., DeLozier, D. M. & Jackson, R. J. (1998). Iodine nutrition in the United States. trends and public health implications: Iodine excretion data from national health and nutrition examination surveys I and III (1971-1974 and 1988-1994). *The Journal of Clinical Endocrinology and Metabolism, 83*(10), 3401-3408.

Houston, M. C. (2007). The role of mercury and cadmium heavy metals in vascular disease, hypertension, coronary heart disease, and myocardial infarction. *Alternative Therapies in Health and Medicine, 13*(2), S128-33.

Hu, F. B., Manson, J. E., & Willett, W. C. (2001). Types of dietary fat and risk of coronary heart disease: A critical review. *Journal of the American College of Nutrition, 20*(1), 5-19.

Institute of Medicine, Food and Nutrition Board (1997). *Dietary Reference Intakes for Calcium, Phosphorus, Magnesium, Vitamin D, and Fluoride*. Washington: National Academy Press.

ISGEM (2008). *Icelandic database on food composition*. Reykjavik: MATIS.

Iyengar, G. V. & Rapp, A. (2001). Human placenta as a 'dual' biomarker for monitoring fetal and maternal environment with special reference to potentially toxic trace elements. Part 3: Toxic trace elements in placenta and placenta as a biomarker for these elements. *The Science of the Total Environment, 280*(1-3), 221-238.

JECFA (Joint FAO/WHO Expert Committee on Food Additives) (2004). Methyl mercury. In: *Safety evaluation of certain food additives and contaminants. Report of the 61st Joint FAO/WHO Expert Committee on Food Additives.* Retrieved August 15th 2008, from, http://whqlibdoc.who.int/trs/WHO_TRS_922.pdf.

JECFA (Joint FAO/WHO Expert Committee on Food Additives) (2006). Methyl mercury. Summary and conclusions of the 67th Joint FAO/WHO Expert Committee on Food Additives. Retrieved August 15th 2008, from, http://www.who.int/ipcs/food/jecfa/summaries/summary67.pdf.

Johansson, L., Solvoll, K., Bjorneboe, G. E. & Drevon, C. A. (1998). Under- and overreporting of energy intake related to weight status and lifestyle in a nationwide sample. *The American Journal of Clinical Nutrition, 68*(2), 266-274.

Julshamn, K., Dahl, L. & Eckhoff, K. (2001). Determination of iodine in seafood by inductively coupled plasma/mass spectrometry. *Journal of AOAC International, 84*, 1976-83.

Kaaks, R. J. (1997). Biochemical markers as additional measurements in studies of the accuracy of dietary questionnaire measurements: Conceptual issues. *American Journal of Clinical Nutrition, 65*, 1232S–1239S.

Kerper, L. E., Ballatori, N. & Clarkson, T. W. (1992). Methyl mercury transport across the blood-brain barrier by an amino acid carrier. *The American Journal of Physiology, 267*, R761–R765.

Kibirige, M. D., Hutchison, S., Owen, C. J. & Delves, H. T. (2004). Prevalence of maternal dietary iodine insufficiency in the north east of England: Implications for the fetus. *Archives of Disease in Childhood Fetal and Neonatal Edition, 89*, 436-439.

Knudsen, N., Christiansen, E., Brandt-Christensen, M., Nygaard, B. & Perrild, H. (2000). Age- and sex-adjusted iodine/creatinine ratio. A new standard in epidemiological surveys? Evaluation of three different estimates of iodine excretion based on casual urine samples and comparison to 24h values. *European Journal of Clinical Nutrition, 54*, 361-363.

Landmark, K. & Aursnes, I. (2004). Mercury fish, fish oil and the risk of cardiovascular disease. *Tidskrift for Den Norske Legeforening, 124*, 198–200.

Larsen, P.R., Davis, T.F. & Hay, I.D. (1998). The thyroid gland. In Wilson J.D., Foster D.W., Kronenberg H.M. & Larsen P.R (Eds). *Williams's textbook of endocrinology*. Philadelphia: WB Saunders.

Laurberg, P., Pedersen, K. M., Hreidarsson, A., Sigfusson, N., Iversen, E. & Knudsen, P. R. (1998). Iodine intake and the pattern of thyroid disorders: A comparative epidemiological study of thyroid abnormalities in the elderly in Iceland and in Jutland, Denmark. *Journal of Clinical Endocrinology and Metabolism, 83*, 765-769.

Lightowler, H.J. & Davies, G.J. (1998). Iodine intake and iodine deficiency in vegans as assessed by the duplicate-portion technique and urinary iodine excretion. *British Journal of Nutrition, 80*, 529-535.

Lysi (2008). *Neytendavara*. Retrieved August 8th 2008, from, http://lysi.is/Neytendavara/.

Maage, A., Toppe, J., Steiner-Asiedu, M., Asibey-Berko, E. & Lied, E. (2008). Inclusion of marine fish in traditional meals improved iodine status of children in an iodine deficient area. *African Journal of Food Science, 2*, 45-53.

Macdiarmid, J. & Blundell, J. (1998). Assessing dietary intake: Who, what and why of under-reporting. *Nutrition Research Reviews, 11*, 231-253.

Margetts, B. M. & Nelson, M. (2006). *Design concepts in Nutritional Epidemiology* (2nd ed.). New York: Oxford University Press.

Markou, K. B., Tsekouras, A., Anastasiou, E., Vlassopoulou, B., Koukkou, E., Vagenakis, G. A., Mylonas, P., Vasilopoulos, C., Theodoropoulou, A., Rottstein, L., Lampropoulou, E., Apostolopoulos, D., Jabarov, R., Vagenakis, A. G. & Georgopoulos N. A. (2008). Treating iodine deficiency: Long-term effects of iodine repletion on growth and pubertal development in school-age children. *Thyroid: Official Journal of the American Thyroid Association, 18*(4), 449-454.

Maycock, B.J. & Benford, D.J. (2007). Risk assessment of dietary exposure to methyl mercury in fish in the UK. *Human and Experimental Toxicology, 26*, 185-190.

Milakovic, M., Berg, G., Nystrom, E., Lindstedt, G., Gebre-Medhin, M. & Eggertsen, R. (2004). Urinary iodine and thyroid volume in a Swedish population. *Journal of Internal Medicine, 255,* 610–614.

Mozaffarian, D. (2007). Fish, n-3 fatty acids, and cardiovascular haemodynamics. *Journal of Cardiovascular Medicine, 8,* S23-S26.

Mozaffarian, D., Longstreth, W. T.,Jr, Lemaitre, R. N., Manolio, T. A., Kuller, L. H., Burke, G. L. & Siscovick D. S. (2005). Fish consumption and stroke risk in elderly individuals: The cardiovascular health study. *Archives of Internal Medicine, 165*(2), 200-206.

Myers, G. J. & Davidson, P. W. (2000). Does methyl mercury have a role in causing developmental disabilities in children? *Environmental Health Perspectives, 108*, 413-420.

Nawaz, H., Chan, W., Abdulrahman, M., Larson, D. & Katz, D. L. (2001). Self-reported weight and height implications for obesity research. *American Journal of Preventive Medicine, 20*, 294-298.

Ng, D. K., Chan, C. H., Soo, M. T., & Lee, R. S. (2007). Low-level chronic mercury exposure in children and adolescents: Meta-analysis. *Pediatrics International: Official Journal of the Japan Pediatric Society, 49*(1), 80-87.

NNR (Nordic Nutrition Recommendations) (2004). *Iodine* (4th ed.). Copenhagen: Nordic Council of Ministers.

Norris, J. M. (2001). Can the sunshine vitamin shed light on type 1 diabetes? *Lancet, 358*, 1476-1478.

NRC (National Research Council) (2000). *Toxicological effects of methylmercury*. Retrieved August 8th 2008, from, http://www.nap.edu/openbook.php?isbn=0309071402

Oken, E., Kleinman, K. P., Berland, W. E., Simon, S. R., Rich-Edwards, J. W. & Gillman, M. W. (2003). Decline in fish consumption among pregnant women after a national mercury advisory. *Obstetrics and Gynecology, 102*(2), 346-351.

Oken, E., Wright, R. O., Kleinman, K. P., Bellinger, D., Amarasiriwardena, C. J., Hu, H., Rich-Edwards, J. W. & Gillman, M. W. (2005). Maternal fish consumption, hair mercury, and infant cognition in a U.S. cohort. *Environmental Health Perspectives, 113*(10), 1376-1380.

Olafsdottir, A. S. (2006). Diet and lifestyle of women at childbearing age: Impact of cod liver oil consumption and associations between diet, lifestyle and weight gain in pregnancy. PhD dissertation: University of Iceland, Faculty of Science.

Olafsdottir, A.S., Thorsdottir, I., Gunnarsdottir, I., Thorgeirsdottir, H. and Steingrimsdottir, L. (2006). Comparison of women´s diet assessed by FFQs and 24-hour recalls with and without underreporters: Associations with biomarkers. Annals of Nutrition and Metabolism, 50(5), 450-460.

Pardede, L. V., Hardjowasito, W., Gross, R., Dillon, D. H., Totoprajogo, O. S., Yosoprawoto, M., Waskito, L. & Untoro J. (1998). Urinary iodine excretion is the most appropriate outcome indicator for iodine deficiency at field conditions at district level. *The Journal of Nutrition, 128*(7), 1122-1126.

Pattison, D. J., Symmons, D. P. & Young, A. (2004). Does diet have a role in the aetiology of rheumatoid arthritis? *Proceedings of the Nutrition Society, 63*, 137-143.

Pearce, E. N., Bazrafshan, H. R., He, X., Pino, S. & Braverman, L. E. (2004). Dietary iodine in pregnant women from the Boston, Massachusetts area. *Thyroid: Official Journal of the American Thyroid Association, 14*(4), 327-328.

Pineda-Lucatero, A., Avila-Jimenez, L., Ramos-Hernandez, R. I., Magos, C. & Martinez, H. (2008). Iodine deficiency and its association with intelligence quotient in schoolchildren from Colima, Mexico. *Public Health Nutrition, 11*(7), 690-698.

Pirrone, N. & Barth, H. (2007). Preface. *Marine Chemistry, 107*, 1–3.

Post-Skagegard, M., Samuelson, G., Karlstrom, B., Mohsen, R., Berglund, L. & Bratteby, L. E. (2002). Changes in food habits in healthy Swedish adolescents during the transition from adolescence to adulthood. *European Journal of Clinical Nutrition, 56*, 532-538.

Public Health Committee of the American Thyroid Association, Becker, D. V., Braverman, L. E., Delange, F., Dunn, J. T., Franklyn, J. A., et al. (2006). Iodine supplementation for pregnancy and lactation - United States and Canada: Recommendations of the American thyroid association. *Thyroid: Official Journal of the American Thyroid Association, 16*(10), 949-951.

Public Health Institute of Iceland (2006). *Radleggingar um mataræði og næringarefni fyrir fullordna og born fra tveggja ara aldri*. Retrieved August 05th 2008, from, http://www.lydheilsustod.is/media/manneldi/utgefid//mataraedi-lowres.pdf

Public Health Institute of Iceland (2008). *Fæduframbod a Islandi*. Retrieved September 10th 2008, from, http://www.lydheilsustod.is/greinar/greinasafn/manneldi//nr/334.

Public Health Institute of Iceland, Center of Prentatal Care - Reykjavík Health Care Center, Environment and Food Agency (2004). *Matur og meðganga, froðleikur fyrir konur a barneignaaldri*. Retrieved August 05th 2008, from, http://www.lydheilsustod.is/media/manneldi/utgefid/Matur&medganga_net_utg.pdf

Qian, M., Yan, Y., Chen, Z. & Wang, D. (2002). Meta-analysis on the relationship between children's intelligence and factors as iodine deficiency, supplement iodine and excessive iodine. *Zhonghua Liu Xing Bing Xue Za Zhi = Zhonghua Liuxingbingxue Zazhi, 23*(4), 246-249.

Ramel, A., Martinez, A., Kiely, M., Morais, G., Bandarra, N. M. & Thorsdottir, I. (2008). Beneficial effects of long-chain n-3 fatty acids included in an energy-restricted diet on insulin resistance in overweight and obese european young adults. *Diabetologia, 51*(7), 1261-1268.

Rashad, I. (2005). Structural estimation of caloric intake, exercise, smoking, and obesity. *The Quarterly Review of Economics and Finance, 46*, 268-283.

Rasmussen, L. B., Ovesen, L. & Christiansen, E. (1999). Day-to-day and within-day variation in urinary iodine excretion. *European Journal of Clinical Nutrition, 53*, 401-407.

Rasmussen, L. B., Ovesen, L., Bulow, I., Jorgensen, T., Knudsen, N., Laurberg, P. & Pertild H. (2002). Dietary iodine intake and urinary iodine excretion in a Danish population: Effect of geography, supplements and food choice. *The British Journal of Nutrition, 87*(1), 61-69.

Raymond, L. J. & Ralston, N. V. C. (2004). Mercury: Selenium interactions and health implications. *Seycelles Medical and Dental Journal, 7*, 72-77.

Reykdal, O., Hilmarsson, O.Þ. & Audunsson, G.A. (2007). *Joð í landbunadarafurdum*. Retrieved Juli 10th 2008, from, http://landbunadur.is/landbunadur/wgsamvef.nsf/8bbba2777ac88c4000256a89000a2ddb/f807d67fa91fbc04002572e60055f6a0/$FILE/Jod%20i%20landbunadarafurdum_grein.pdf

Reykdal, O., Thorlacius, A., Audunsson, G.A. & Steingrimsdottir, L. (2000). Selen, jod, fluor, jarn, kopar, sink, mangan, kadmin, kvikasilfur og bly í landbunaðarafurðum. *Fjolrit Rala*, *204*, 7-36.

Salonen, J. T., Seppanen, K., Nyyssonen, K., Korpela, H., Kauhanen, J., Kantola, M., Tuomilehto, J., Esterbauer, H., Tatzber, F. & Salonen R. (1995). Intake of mercury from fish, lipid peroxidation, and the risk of myocardial infarction and coronary, cardiovascular, and any death in eastern Finnish men. *Circulation, 91*(3), 645-655.

Sidhu, K.S. (2003). Health benefits and potential risks related to consumption of fish or fish oil. *Regulatory Toxiology and Pharmacology, 38*, 336-344.

Sigurdsson, G. and Franzson, L. (1998). Urine excretion of iodine in an Icelandic population. *Icelandic Medicine Journal, 74*, 179-181.

Sigurjonsson, J. (1940). *Studies on the human thyroid in Iceland*. Doctorial dissertation, University of Iceland, Faculty of medicine.

Sioen, I., De Henauw, S. & Van Camp, J. (2007a). Evaluation of benefits and risks related to seafood consumption. *Verhandelingen - Koninklijke Academie Voor Geneeskunde Van Belgie, 69*(5-6), 249-289.

Sioen, I., Matthys, C., De Backer, G., Van Camp, J. & Henauw, S. D. (2007b). Importance of seafood as nutrient source in the diet of Belgian adolescents. *Journal of Human Nutrition and Dietetics: The Official Journal of the British Dietetic Association, 20*(6), 580-589.

Sjostrom, M., Yngve, A., Poortvliet, E., Warm, D. & Ekelund U. (1999). Diet and physical activity – interactions for health, public health nutrition in the European perspective. *Public Health Nutrition, 2*, 453-459.

Smallridge, R. C., Glinoer, D. G., Hollowell, J. G. & Brent, G. (2005). Thyroid function inside and outside of pregnancy: What do we know and what don't we know? *Thyroid, 15*, 54-59.

Steingrimsdottir, L., Thorgeirsdottir, H. & Olafsdottir, A. S. (2003). *The Icelandic National Nutrition Survey 2002*. Public Health Institute of Iceland, Reykjavik.

Steuerwald, U., Weihe, P., Jorgensen, P. J., Bjerve, K., Brock, J., Heinzow, B., Budtz-Jorgensen, E. & Grandjean, P. (2000). Maternal seafood diet, methylmercury exposure, and neonatal neurologic function. *The Journal of Pediatrics, 136*(5), 599-605.

Stimec, M., Mis, N. F., Smole, K., Sirca-Campa, A., Kotnik, P., Zupancic, M., Battelino, T. & Krizisnik C. (2007). Iodine intake of Slovenian adolescents. *Annals of Nutrition & Metabolism, 51*(5), 439-447.

Swanson, E. W., Miller, J. K., Mueller, F. J., Patton, C. S., Bacon, J. A. & Ramsey, N. (1990). Iodine in milk and meat of dairy cows fed different amounts of potassium iodide or ethylenediamine dihydroiodide. *Journal of Dairy Science, 73*(2), 398-405.

Thomson, C. D., Smith, T. E., Butler, K. A. & Packer, M. A. (1996). An evaluation of urinary measures of iodine and selenium status. *Journal of Trace Elements in Medicine and Biology: Organ of the Society for Minerals and Trace Elements (GMS), 10*(4), 214-222.

Thorsdottir, I. & Gunnarsdottir, I. (2006). *The diet of Icelandic 9- and 15-year-old children and adolescents.* Reykjavik: *Unit for Nutrition Research 2002-2003*.

Thorsdottir, I., Gunnarsdottir, I. & Steingrimsdottir, L. (2004). Mat a gildi spurningarlista Manneldisrads Islands til konnunar á mataræði fullordinna. *Læknabladid, 90*, 37-41.

Thorsdottir, I., Tomasson, H., Gunnarsdottir, I., Gisladottir, E., Kiely, M., Parra, M. D., Bandarra, N. M., Schaafsma, G. & Martinez J.A. (2007). Randomized trial of weight-loss-diets for young adults varying in fish and fish oil content. *International Journal of Obesity (2005), 31*(10), 1560-1566.

Ukropec, J., Reseland, J. E., Gasperikova, D., Demcakova, E., Madsen, L., Berge, R. K., Rustan, A.C., Klimes, I., Drevon, C.A. and Sebokova E. (2003). The hypotriglyceridemic effect of dietary n-3 FA is associated with increased beta-oxidation and reduced leptin expression. *Lipids, 38*(10), 1023-1029.

UNIDO (United Nations Industrial Development Organization) (2003). *Removal of barriers to introduction of cleaner artisanal gold mining and extraction technologies*. Retrieved September 4th 2008, from, http://www.cetem.gov.br/gmp/GMP_News/ GMP_News_January_2003.pdf.

van den Briel, T., West, C. E., Hautvast, J. G., Vulsma, T., de Vijlder, J. J. & Ategbo, E. A. (2001). Serum thyroglobulin and urinary iodine concentration are the most appropriate indicators of iodine status and thyroid function under conditions of increasing iodine supply in schoolchildren in benin. *The Journal of Nutrition, 131*(10), 2701-2706.

Virtanen, J. K., Rissanen, T. H., Voutilainen, S. & Tuomainen, T. P. (2007). Mercury as a risk factor for cardiovascular diseases. *The Journal of Nutritional Biochemistry, 18*, 75-85.

Vitti, P., Rago, T., Aghini-Lombardi, F. & Pinchera, A. (2001). Iodine deficiency disorders in *Europe. Public Health Nutrition, 4*, 529-535.

Vought, R. L., London, W. T., Lutwak, L. & Dublin, T. D. (1963). Reliability of estimates of serum inorganic iodine and daily fecal and urinary iodine excretion form single casual specimens. *The Journal of Clinical Endocrinology and Metabolism, 23*, 1218–1228.

Whelton, S. P., He, J., Whelton, P. K., & Muntner, P. (2004). Meta-analysis of observational studies on fish intake and coronary heart disease. *The American Journal of Cardiology, 93*(9), 1119-1123.

WHO (World Health Organization) (1990). *Environmental Health Criteria 101: Methylmercury*. Geneva: WHO. Retrieved August 29th 2008 from, http://www.inchem.org/documents/ehc/ehc/ehc101.htm

WHO (World Health Organization) (1994). *World summit for children-mid decade goal: Iodine Deficiency Disorders. UNIEF–WHO Joint Committee on Health Policy*. United Nations Children's Fund, World Health Organization, Geneva,

WHO (World Health Organization) (1996). *Trace elements in human nutrition and health*. Geneva: WHO.

WHO (World Health Organization) (2003a). *Iodine: some products contain more than the RDA*. Retrieved June 15th 2008, from, http://www.who.int/medicines/publications/newsletter/en/ news2003_3.pdf.

WHO (World Health Organization) (2003b). *Food based dietary guideline in the WHO European Region.* Geneva: WHO. Retrieved August 22nd 2008, from, http://www.euro.who.int/Document/E79832.pdf

WHO (World Health Organization) (2004). *WHO Food Additive Series 52. Safety evaluation of certain food additives and contaminants. International Programme on Chemical Safety*. Retrieved September 4th 2008, from, http://www.inchem.org/documents/jecfa/ jecmono/v52je23.htm.

WHO (World Health Organization) (2005a). *Policy paper: Mercury in health care*. Retrieved August 22nd 2008, from, http://who.int/water_sanitation_health/medicalwaste/ Mercury/en/index.html.

WHO (World Health Organization) (2005b). *Affordable technology: Blood pressure measuring devices for low resource settings.* Retrieved August 22nd 2008, from, http://whqlibdoc.who.int/publications/2005/9241592648.pdf.

WHO (World Health Organization) (2006). *Global database on body mass index an interactive surveillance tool for monitoring nutrition status.* Retrieved June 8th 2008, from, http://www.who.int/bmi/.

WHO (World Health Organization) (2007). *Preventing disease through healthy environments. Exposure to mercury: A major public health concern*. Retrieved June 30th 2008, from, http://www.who.int/phe/news/Mercury-flyer.pdf.

WHO (World Health Organization) (2008). *Exposure of children to chemical hazards in European environment and health information system.* Retrieved August 16th 2008, from, http://www.enhis.org/object_document/o4736n27387.html.

WHO/UNEP (World Health Organization and United Nations Environment Programme) (2008). *Guidance for identifying populations at risk from mercury exposure*. Retrieved September 4th 2008, from, http://www.who.int/foodsafety/publications/chem/mercuryexposure.pdf.

WHO/UNICEF (World Health Organization and United Nations Children's Fund) (2007). *Iodine deficiency in Europe: A continuing public health problem.* Retrieved June 15th 2008, from, http://whqlibdoc.who.int/publications/2007/9789241593960_eng.pdf.

WHO/UNICEF/ICCIDD (World Health Organization, United Nations Children's Fund and International Council for the Control of Iodine Deficiency) (1994). *Indicators for assessing iodine deficiency disorders and their control through salt iodization.* Geneva: WHO.

WHO/UNICEF/ICCIDD (World Health Organization, United Nations Children's Fund and International Council for the Control of Iodine Deficiency) (2001). *Assessment of the iodine deficiency disorders and monitoring their elimination. A Guide for Programme Managers.* (2nd ed). Retrieved June 30th 2008, from, http://who.int/reproductive-health/docs/iodine_deficiency.pdf.

WHO/UNICEF/ICCIDD (World Health Organization, United Nations Children's Fund and International Council for the Control of Iodine Deficiency) (2007). *Assessment of iodine deficiency disorders and monitoring their elimination. A guide for programme managers,* 3ed. Retrieved August 2nd 2008, from, http://whqlibdoc.who.int/publications/2007/9789241595827_eng.pdf.

WHO/UNICEF/ICCIDD (World Health Organization, United Nations International Children's Fund and International Council for the Control of Iodine Deficiency) (1999). *Progress towards the elimination of Iodine Deficiency Disorders (IDD).* Geneva: WHO.

Winger, R. J., Konig, J. & House, D. A. (2008). Technological issues associated with iodine fortification of foods. *Trends in Food Science and Technology, 19*, 94-101.

Appendix A – Information letter 16-18 years old and legal guardian

Upplýsingar fyrir þátttakendur og forráðamenn.

Joðhagur unglingsstúlkna og þungaðra kvenna

Tilgangur rannsóknarinnar er að rannsaka joðhag unglingsstúlkna og þungaðra kvenna út frá joðstyrk í þvagi ásamt spurningalista um fæðuval sem verða notuð til að kanna samband milli joðhags og neyslu ákveðinna fæðutegunda. Joðhagur hefur ekki verið kannaður á beinan hátt á Íslandi síðastliðin 20 ár og aldrei meðal unglingsstúlkna eða þungaðra kvenna. Ungar og þungaðar konur er sá hópur sem er hvað viðkvæmastur fyrir joðskorti.

Ábyrgðarmaður rannsóknarinnar:	Ingibjörg Gunnarsdóttir, dósent við HÍ Rannsóknarstofa í næringarfræði Landspítali Háskólasjúkrahús Eiríksgötu 29, ris 101 Reykjavík Símar: 543-8416 / 543-8420 / 891-7017 Tölvupóstur: ingigun@landspitali.is

Aðrir sem að rannsókninni koma eru Guðrún Kristín Sigurgeirsdóttir, næringarfræðingur (M.Sc.) á Rannsóknastofu í næringarfræði, Ari. J. Jóhannesson, sérfræðingur í lyflækningum, innkirtla- og efnaskiptasjúkdómum á Landspítala-háskólasjúkrahúsi, dr. Amund Maage, rannsóknamaður við NIFES í Noregi, dr. Laufey Steingrímsdóttir næringarfræðingur auk Heilsugæslunnar á höfuðborgarsvæðinu.

Úrtak 350 þungaðra kvenna á 2. og 3. þriðjungi meðgöngu verður tekið úr mæðraskrám Heilsugæslunnar á höfuðborgarsvæðinu á tímabilinu júní 2007 – maí 2008. Handahófsúrtak 350 unglingsstúlkna (16-20 ára) verður tekið úr Þjóðskrá Íslands á tímabilinu júní 2007 – maí 2008 á höfuðborgarsvæðinu.

Kjósir þú að taka þátt mun framlag þitt til rannsóknarinnar felast í eftirfarandi:

1. Svörun spurningalista um neyslu matvæla (tekur u.þ.b. 20 mín).
2. Mæting í blóðprufu til mælinga á TSH (mælikvarði á virkni skjaldkirtils), Hg (kvikasilfur) og Selen.
3. Að skila inn þvagprufu á staðnum.
4. Mælingar á hæð og þyngd. Spurt um heilsugæslu, heimilislækni, getnaðarvarnarlyf og reykingar.

Rannsóknin er styrkt af Rannsóknamiðstöð Íslands (RANNÍS), hefur leyfi Vísinda-siðanefndar auk þess að hafa fengið leyfi Persónuverndar.

Öll rannsóknagögn, blóð- og þvagprufur verða kóðaðar til að gæta trúnaðar við þátttakendur. Ábyrgðarmaður rannsóknar er til aðstoðar við framkvæmd viðtals ef þarf. Þátttakendur geta ávallt haft samband við ábyrgðarmann rannsóknarinnar eða aðstoðarmann hans.

Rannsóknagögn verða varðveitt á Rannsósknastofu í næringarfræði. Þeim verður eytt eigi síðar en 5 árum eftir lífsýnatöku. Lífsýni sem send verða til Noregs verða undir umsjón og ábyrgð Dr. Amund Maage sem tekur ábyrgð á að eyða þeim að mælingum loknum.

Ávinningur og áhætta af þátttöku

Ávinningur þátttakenda felst fyrst og fremst í því að ef niðurstöður mælinga á TSH eða joðstyrk er ekki innan eðlilegra marka þá munu rannsakendur grípa til viðeigandi ráðstafana til að stuðla að því að joðhagur þátttakenda verði leiðréttur.

Allir þátttakendur munu að rannsókn lokinni fá leiðbeiningar um fæðuval.

Áhætta þátttakenda í rannsókninni er mjög lítil. Ströngum gæðakröfum verður framfylgt við blóðprufutöku og þær allar framkvæmdar af reyndu heilbrigðisstarfsfólki. Sýkingahætta samfara þvagsöfnun er hverfandi sé gætt að almennum handþvotti.

Ekki er unnt að greiða sérstaklega fyrir þátttöku í rannsókninni.

Þátttakendum er frjálst að hafna þátttöku eða hætta í rannsókninni á hvaða stigi sem er, án útskýringa og mun það ekki hafa neinar afleiðingar.

Með góðum kveðjum,

Dr. Ingibjörg Gunnarsdóttir
Dósent við Háskóla Íslands

Guðrún Kr. Sigurgeirsd.

Guðrún Kristín Sigurgeirsdóttir
Næringarfræðingur (MSc)

Ef þú hefur spurningar um rétt þinn sem þátttakandi í vísindarannsókn eða vilt hætta þátttöku í rannsókninni getur þú snúið þér til Vísindasiðanefndar, Vegmúla 3, 108 Reykjavík. Sími: 551-7100, fax: 551-1444.

Appendix B – Information letter 18-20 years old

Upplýsingar fyrir þátttakendur.

Joðhagur unglingsstúlkna og þungaðra kvenna

Tilgangur rannsóknarinnar er að rannsaka joðhag unglingsstúlkna og þungaðra kvenna út frá joðstyrk í þvagi ásamt spurningalista um fæðuval sem verða notuð til að kanna samband milli joðhags og neyslu ákveðinna fæðutegunda. Joðhagur hefur ekki verið kannaður á beinan hátt á Íslandi síðastliðin 20 ár og aldrei meðal unglingsstúlkna eða þungaðra kvenna. Ungar og þungaðar konur er sá hópur sem er hvað viðkvæmastur fyrir joðskorti.

Ábyrgðarmaður rannsóknarinnar:	Ingibjörg Gunnarsdóttir, dósent við HÍ Rannsóknarstofa í næringarfræði Landspítali Háskólasjúkrahús Eiríksgötu 29, ris 101 Reykjavík Símar: 543-8416 / 543-8420 / 891-7017 Tölvupóstur: ingigun@landspitali.is

Aðrir sem að rannsókninni koma eru Guðrún Kristín Sigurgeirsdóttir, næringarfræðingur (M.Sc.) á Rannsóknastofu í næringarfræði, Ari. J. Jóhannesson, sérfræðingur í lyflækningum, innkirtla- og efnaskiptasjúkdómum á Landspítala-háskólasjúkrahúsi, dr. Amund Maage, rannsóknamaður við NIFES í Noregi, dr. Laufey Steingrímsdóttir næringarfræðingur auk Heilsugæslunnar á höfuðborgarsvæðinu.

Úrtak 350 þungaðra kvenna á 2. og 3. þriðjungi meðgöngu verður tekið úr mæðraskrám Heilsugæslunnar á höfuðborgarsvæðinu á tímabilinu júní 2007 – maí 2008. Handahófsúrtak 350 unglingsstúlkna (16-20 ára) verður tekið úr Þjóðskrá Íslands á tímabilinu júní 2007 – maí 2008 á höfuðborgarsvæðinu.

Kjósir þú að taka þátt mun framlag þitt til rannsóknarinnar felast í eftirfarandi:

5. Svörun spurningalista um neyslu matvæla (tekur u.þ.b. 20 mín).
6. Mæting í blóðprufu til mælinga á TSH (mælikvarði á virkni skjaldkirtils), Hg (kvikasilfur) og Selen.
7. Að skila inn þvagprufu á staðnum.
8. Mælingar á hæð og þyngd. Spurt um heilsugæslu, heimilislækni, getnaðarvarnarlyf og reykingar.

Rannsóknin er styrkt af Rannsóknamiðstöð Íslands (RANNÍS), hefur leyfi Vísinda-siðanefndar auk þess að hafa fengið leyfi Persónuverndar.

Öll rannsóknagögn, blóð- og þvagprufur verða kóðaðar til að gæta trúnaðar við þátttakendur. Ábyrgðarmaður rannsóknar er til aðstoðar við framkvæmd viðtals ef þarf. Þátttakendur geta ávallt haft samband við ábyrgðarmann rannsóknarinnar eða aðstoðarmann hans.

Rannsóknagögn verða varðveitt á Rannsósknastofu í næringarfræði. Þeim verður eytt eigi síðar en 5 árum eftir lífsýnatöku. Lífsýni sem send verða til Noregs verða undir umsjón og ábyrgð Dr. Amund Maage sem tekur ábyrgð á að eyða þeim að mælingum loknum.

Ávinningur og áhætta af þátttöku

Ávinningur þátttakenda felst fyrst og fremst í því að ef niðurstöður mælinga á TSH eða joðstyrk er ekki innan eðlilegra marka þá munu rannsakendur grípa til viðeigandi ráðstafana til að stuðla að því að joðhagur þátttakenda verði leiðréttur.

Allir þátttakendur munu að rannsókn lokinni fá leiðbeiningar um fæðuval.

Áhætta þátttakenda í rannsókninni er mjög lítil. Ströngum gæðakröfum verður framfylgt við blóðprufutöku og þær allar framkvæmdar af reyndu heilbrigðisstarfsfólki. Sýkingahætta samfara þvagsöfnun er hverfandi sé gætt að almennum handþvotti.

Ekki er unnt að greiða sérstaklega fyrir þátttöku í rannsókninni.

Þátttakendum er frjálst að hafna þátttöku eða hætta í rannsókninni á hvaða stigi sem er, án útskýringa og mun það ekki hafa neinar afleiðingar.

Með góðum kveðjum,

Dr. Ingibjörg Gunnarsdóttir
Dósent við Háskóla Íslands

Guðrún Kristín Sigurgeirsdóttir
Næringarfræðingur (MSc)

Ef þú hefur spurningar um rétt þinn sem þátttakandi í vísindarannsókn eða vilt hætta þátttöku í rannsókninni getur þú snúið þér til Vísindasiðanefndar, Vegmúla 3, 108 Reykjavík. Sími: 551-7100, fax: 551-1444.

Appendix C – Consent form for 16-18 years old and legal guardian

UPPLÝST SAMÞYKKI unglingsstúlkna og forráðamanna fyrir þátttöku í vísindarannsókninni *„ **Joðhagur unglingsstúlkna og þungaðra kvenna á Íslandi** “*

Þátttakandi samþykkir hér með þátttöku á rannsókninni “Joðhagur unglingsstúlkna og þungaðra kvenna”. Þátttakandi samþykkir að svara spurningalista um neyslu matvæla og skila inn þvagprufu. Einu sinni verður tekin blóðprufa.

Niðurstöður rannsóknarinnar munu verða sendar til birtingar í virtum erlendum vísindatímaritum.

Þátttakandi hefur lesið kynningarbréf um rannsóknina “Joðhagur unglingsstúlkna og þungaðra kvenna”.

Undirskrift þátttakanda:

Ég, ____

lýsi því hér með yfir að ég gef samþykki mitt af fúsum og frjálsum vilja fyrir því taka þátt í þessari rannsókn “Joðhagur unglingsstúlkna og þungaðra kvenna”sem styrkt er af Rannsóknamiðstöð Íslands (RANNÍS). Ég samþykki að lífsýni (ópersónugreinanleg) megi flytja til Noregs þar sem þau eru unnin undir stjórn og ábyrgð Amund Maage. Sýnum verður eytt að vinnslu lokinni. Ég hef fengið nauðsynlegar upplýsingar og lesið þær yfir.

Mér hefur verið kynnt eðli og umfang þessarar vísindarannsóknar og ég er samþykk(ur) þátttöku og skrifa því undir þessi tvö eintök:

____

Dagsetning og staður:

____

Kennitala þátttakanda

____________________________ ____________________________

Undirskrift þátttakanda Undirskrift foreldris/forráðamanns

____

Undirritun þess sem aflar samþykkis

Appendix D – Consent form for 18-20 years old

UPPLÝST SAMÞYKKI fyrir þátttöku í vísindarannsókninni
*„ **Joðhagur unglingsstúlkna og þungaðra kvenna á Íslandi** “*

Þátttakandi samþykkir hér með þátttöku á rannsókninni “Joðhagur unglingsstúlkna og þungaðra kvenna”. Þátttakandi samþykkir að svara spurningalista um neyslu matvæla og skila inn þvagprufu. Einu sinni verður tekin blóðprufa.

Niðurstöður rannsóknarinnar munu verða sendar til birtingar í virtum erlendum vísindatímaritum.

Þátttakandi hefur lesið kynningarbréf um rannsóknina “Joðhagur unglingsstúlkna og þungaðra kvenna”.

Undirskrift þátttakanda:

Ég, ____
lýsi því hér með yfir að ég gef samþykki mitt af fúsum og frjálsum vilja fyrir því taka þátt í þessari rannsókn “Joðhagur unglingsstúlkna og þungaðra kvenna” sem styrkt er af Rannsóknamiðstöð Íslands (RANNÍS). Ég samþykki að lífsýni (ópersónugreinanleg) megi flytja til Noregs þar sem þau eru unnin undir stjórn og ábyrgð Amund Maage. Sýnum verður eytt að vinnslu lokinni. Ég hef fengið nauðsynlegar upplýsingar og lesið þær yfir.

Mér hefur verið kynnt eðli og umfang þessarar vísindarannsóknar og ég er samþykk þátttöku og skrifa því undir þessi tvö eintök:

____
Dagsetning og staður:

____
Undirskrift þátttakanda

____

Undirritun þess sem aflar samþykkis

Appendix E – Health information form

Upplýsingar um þátttakendur

*Númer:*____________ *Hæð:*____________ cm *Þyngd:*____________ kg

Spurningalisti fyrir rannsakendur

<u>Spurningar um heilsuvernd (spurningar 1-2)</u>

1) Hjá hvaða heilsugæslu ertu skráð?

____

2) Hjá hvaða heimilislækni ertu skráð?

____

<u>Spurningar um reykingar (spurningar 3-4)</u>

3) Hefur þú einhvern tíma reykt?
- ❑ já
- ❑ nei (ef svarað neitandi farðu beint að spurningu 5)

4) Reykir þú núna?
- ❑ já
- ❑ nei

<u>Spurningar um lyf (spurningar 5-6)</u>

5) Hefur þú einhvern tíma notað getnaðarvarnalyf?
- ❑ já (nafn lyfs:______________________________)
- ❑ nei

6) Notar þú getnaðarvarnalyf núna?
- ❑ já (nafn lyfs:______________________________)
- ❑ nei

Appendix F – Validated Food Frequency Questionnaire

Rannsóknastofa í næringarfræði
Landspítali-Háskólasjúkrahús
Eiríksgata 29, 1. hæð
101 Reykjavík
Sími: 543 8416
Fax: 543 4824
ingigun@landspitali.is

Fæðuvenjur: Nokkrir fæðuþættir

Spurningar vegna rannsóknarinnar
„Joðhagur þungaðra kvenna og unglingsstúlkna á Íslandi”

Raðnúmer:

Hvernig á að fylla út spurningalistann

Vinsamlega lestu spurningarnar og valmöguleikana vandlega áður en þú svarar spurningunum. Flestar spurningarnar eru þannig að það á bara að merkja við einn reit. Í nokkrum spurningum má merkja við fleiri möguleika ef við á. Í nokkrum spurningum biðjum við þig um að skrifa svarið.

Þú ert beðinn að svara með tilliti til þess hvernig þú varst að borða að meðaltali síðustu 3-4 vikurnar.

Fiskur – hvað borðar þú vanalega?

Aðalréttur

F1. Hvað borðar þú oft fisk sem aðalrétt?

- ☐ Aldrei
- ☐ 1-3 sinnum í mánuði
- ☐ 4-6 sinnum í mánuði
- ☐ 2 sinnum í viku
- ☐ 3 sinnum í viku
- ☐ 4-6 sinnum í viku
- ☐ Á hverjum degi
- ☐ Annað (skrifaðu):

____

F2. Hvaða gerðir/tegundir fiska borðar þú oftast sem aðalrétt? (merktu við með tölum 1-4 ef við á)

- ☐ Ýsu
- ☐ Þorsk
- ☐ Lúðu
- ☐ Lax
- ☐ Annað (skrifaðu):

____

F3. Þegar þú borðar fisk sem aðalrétt, hve mikið borðar þú í hverjum matmálstíma? (veldu að segja frá eins og þér hentar best að lýsa því)

_______ grömm

_____ flak (t.d. 1 flak, ½ flak, ⅓ flak o.s.frv.)

Fékk mér______ sinnum á diskinn

Annað (skrifaðu):

____

Brauðálegg, forréttur

F4. Hvað borðar þú oft fisk sem brauðálegg eða sem forrétt?

- ☐ Aldrei (ef aldrei farðu beint í spurningu F7)
- ☐ 1-3 sinnum í mánuði
- ☐ 4-6 sinnum í mánuði
- ☐ 2 sinnum í viku
- ☐ 3 sinnum í viku
- ☐ 4-6 sinnum í viku
- ☐ Á hverjum degi
- ☐ Annað (skrifaðu):

____

F5. Hvaða gerðir/tegundir fiska borðar þú oftast sem álegg eða sem forrétt? (merktu við með tölum 1-6 ef við á)

- ☐ Síld
- ☐ Reyktan/grafinn lax
- ☐ Kavíar
- ☐ Sardínur
- ☐ Makríl
- ☐ Rækjur
- ☐ Annað (skrifaðu):

____

F6. Hve mikið borðar þú í hvert sinn? (t.d. hve margar sneiðar, bita eða matskeiðar á eina brauðsneið og hve margar brauðsneiðar)

____

Lýsi

(Merktu bara einu sinni við hverja spurningu)

F7. Tekur þú inn lýsi eða lýsisperlur? **(Ef nei þá máttu sleppa þeim spurningum sem eftir eru)**

- ☐ Nei
- ☐ Já

F8. Hvaða tegund?

- ☐ Þorskalýsi
- ☐ Ufsalýsi
- ☐ Lúðulýsi
- ☐ Hákarlalýsi
- ☐ Annað (skrifaðu):

____

F9. Hve oft?

- ☐ Á hverjum degi
- ☐ 4-6 sinnum í viku
- ☐ 2-3 sinnum í viku
- ☐ Annað (skrifaðu):

____

F10. Hve mikið?

- ☐ Teskeið
- ☐ Barnaskeið
- ☐ Matskeið
- ☐ Fjöldi lýsisperla (skrifaðu):

____

Appendix G – Semi-quantitative Food Frequency Questionnaire

Dæmi um hvernig á að fylla út eyðublaðið

Sjá spurningu 1. Brauð, kökur, kex

Hér er spurt hversu margar brauðsneiðar, kexkökur og kökusneiðar þú borðar yfirleitt í mánuði, viku eða á dag.

Segjum sem svo að Jón Jónsson borði yfirleitt tvær heilhveitibrauðsneiðar á morgnana alla virka daga, hann fái sér ævinlega samloku í hádeginu úr heilhveitibrauði og auk þess eina til tvær grófar brauðsneiðar síðdegis 5 daga vikunnar. Franskbrauð, rúgbrauð eða hrökkbrauð borðar hann hins vegar aldrei. Hann áætlar neysluna á heilhveitibrauði og grófu brauði þannig:

Hann áttar sig á því að 5 daga vikunnar borðar hann 5-6 sneiðar á dag en tvo daga vikunnar aðeins tvær sneiðar. Að jafnaði borðar hann því 4-5 sneiðar á dag. Á eyðublaðinu er hvergi hægt að merkja við 4-5 sneiðar á dag heldur annað hvort 3-4 á dag eða 5 eða fleiri. Jón metur stöðuna þannig að réttara sé að merkja við reitinn 5 eða fleiri, því það sé líklega nær hinu rétta.

Jón fær sér ævinlega vínarbrauð á sunnudagsmorgnum og merkir því í reitinn 1/viku. Pönnukökur fær hann u.þ.b. einu sinni í mánuði en borðar þá 4-5 pönnukökur og merkir því við 1/viku (4 í mánuði). Kleinur eða jólaköku fær hann sér með kaffinu flesta daga vikunnar og borðar þá gjarnan 2 kleinur eða kökusneiðar. Hann merkir því við reitinn 2 á dag. Rjóma setur hann aðeins á tvær pönnukökur í mánuði og merkir í samræmi við það. Kex borðar hann aldrei.

Útfyllt eyðublað Jóns lítur þannig út fyrir spurningu 1:

1. Brauð, kökur, kex

Hversu margar brauðsneiðar, stykki, borðar þú á dag, í viku eða mánuði? Eitt rúnnstykki eða langloka teljast tvær brauðsneiðar. Merktu við einn reit í hverri línu, annað hvort við sneiðar á dag, í viku, í mánuði eða aldrei eftir því sem við á.

Fæðutegund	Aldrei	Í mánuði sneiðar			Í viku sneiðar			Á dag sneiðar			
	Aldrei	Færri en 1	1	2-3	1	2-3	4-6	1	2	3-4	5 eða fleiri
Franskbrauð, hvítt brauð	☒	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Rúg-, malt-, flatbrauð	☒	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Heilhveitibrauð, gróft brauð	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☒
Hrökkbrauð, bruður, kringlur	☒	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Vínarbrauð, smjörkaka	☐	☐	☐	☐	☒	☐	☐	☐	☐	☐	☐
Snúður, pönnukökur, vöfflur	☐	☐	☐	☐	☒	☐	☐	☐	☐	☐	☐
Kleinur, kökur, annað sætabrauð	☐	☐	☐	☐	☐	☐	☐	☐	☒	☐	☐
Kex, sætt, ósætt	☒	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Rjómi á kökur, vöfflur	☐	☐	☐	☒	☐	☐	☐	☐	☐	☐	☐

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1. Brauð, kökur, kex

<u>Hversu margar</u> brauðsneiðar, stykki, borðar þú á dag, í viku eða mánuði? Eitt rúnnstykki eða langloka teljast tvær brauðsneiðar. Merktu við einn reit í hverri línu, <u>annað hvort</u> við sneiðar á dag, í viku, í mánuði eða aldrei eftir því sem við á.

Fæðutegund	Aldrei	Í mánuði sneiðar			Í viku sneiðar			Á dag sneiðar			
	Aldrei	Færri en 1	1	2-3	1	2-3	4-6	1	2	3-4	5 eða fleiri
Franskbrauð, hvítt brauð	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Rúg-, malt-, flatbrauð	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Heilhveitibrauð, gróft brauð	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Hrökkbrauð, bruður, kringlur	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Vínarbrauð, smjörkaka	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Snúður, pönnukökur, vöfflur	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Kleinur, kökur, annað sætabrauð	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Kex, sætt, ósætt	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Rjómi á kökur, vöfflur	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐

2. Með hverju er brauðið (kexið) smurt?

<u>Hversu margar</u> brauðsneiðar eða kexkökur smyrðu á dag eða á viku með hverju feitmeti fyrir sig? Teldu samanlagðan fjölda sneiða eða stykkja af öllu brauði, hrökkbrauði eða kexi sem er smurt.

Fæðutegund	Aldrei	Í mánuði sneiðar			Í viku sneiðar			Á dag sneiðar			
Fjöldi brauðsneiða	Aldrei	Færri en 1	1	2-3	1	2-3	4-6	1	2	3-4	5 eða fleiri
Smjör (fjöldi sneiða)	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Smjörvi	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Létt og laggott	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Klípa	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Sólblóma (65%), Olivio	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Létta, Létt-Sólblóma, Lína, Smyrill	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐

MR98-02

3. Álegg. Fjöldi brauðsneiða með hverju áleggi

Skráðu hér samanlagðan **fjölda** brauðsneiða eða kexa á dag, viku eða mánuði með hverri áleggstegund. Ef tvær eða fleiri áleggstegundir eru hafðar á einni sneið, skal merkja við allar tegundirnar.

Fæðutegund	Aldrei	Í mánuði fjöldi			Í viku fjöldi			Á dag fjöldi			
	Aldrei	Færri en 1	1	2-3	1	2-3	4-6	1	2	3-4	5 eða fleiri
Ostur, 26% eða feitari	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Ostur, 17%, 11%, smurostur	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Camembert, Brie, rjómaostur, gráðostur	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Skinka, hangikjöt, kjötpylsa, steik	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Spægipylsa, pepperóní, rúllupylsa, beikon	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Kæfa, paté	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Salat með majonesi/sýrðum rjóma	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Lax, silungur, síld	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Rækjur, egg	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Grænmeti/ávextir sem álegg	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Sulta, marmelaði, hunang	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Hnetusmjör	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐

4. Mjólk <u>til drykkjar.</u>

Hversu mörg mjólkurglös drekkur þú á dag, viku eða í mánuði (1 glas=2dl)
(Ekki telja með mjólk út á graut o.s.frv. eða út í kaffi),

Fæðutegund	Aldrei	Í mánuði fjöldi glasa			Í viku fjöldi glasa			Á dag fjöldi glasa			
	Aldrei	Færri en 1	1	2-3	1	2-3	4-6	1	2	3-4	5 eða fleiri
Nýmjólk, kakó úr nýmjólk	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Léttmjólk, sælumjólk, kakó úr léttmjólk	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Undanrenna, fjörmjólk	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Mysa	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐

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5. Drykkjarvörur

Hversu mörg glös, bolla o.s.frv. drekkur þú af hverjum drykk?

Fæðutegund	Aldrei	Í mánuði fjöldi			Í viku fjöldi			Á dag fjöldi			
	Aldrei	Færri en 1	1	2-3	1	2-3	4-6	1	2	3-4	5 eða fleiri
Vatn, sódavatn, sykurlaus gosdrykkur (glas)											
Ávaxtasafi, hreinn (lítil ferna)											
Svali, djús (lítil ferna)											
Gosdrykkur (glas)											
Pilsner, maltöl (glas)											
Bjór (glas)											
Léttvín, borðvín (vínglas)											
Millisterkt vín (sherry, púrtvín, líkjör) (glas)											
Sterkt vín (sjúss)											
Kaffi (bolli)											
Te, jurtate (bolli)											

6. Notar þú mjólk, sykur o.s.frv. í kaffi, te?

Skráðu **fjölda** bolla með hverju fyrir sig.

Fæðutegund	Aldrei	Í mánuði fjöldi			Í viku fjöldi			Á dag fjöldi			
	Aldrei	Færri en 1	1	2-3	1	2-3	4-6	1	2	3-4	5 eða fleiri
Mjólk, léttmjólk í kaffi, te											
Rjómi, kaffirjómi í kaffi, te											
Sykur, hunang í kaffi, te											

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Athugið: Hér eftir verður spurt um <u>tíðni</u>, þ.e. hversu oft þú borðar tilgreindar fæðutegundir

7. Morgunkorn, grautar, sýrðar mjólkurvörur

<u>Hversu OFT</u> borðar þú þessar vörur?

Fæðutegund	Aldrei	Í mánuði skipti			Í viku skipti			Á dag skipti			
	Aldrei	Sjaldnar en 1	1	2-3	1	2-3	4-6	1	2	3-4	5 eða oftar
Hafragrautur	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
All-bran, múslí, haframjöl	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Morgunkorn, (Cheerios o.þ.h.)	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Hrísgrjónagrautur, mjólkursúpa	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Súrmjólk, AB-mjólk, ABT-mjólk, jógúrt	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Sýrð léttmjólk, léttjógúrt, fismjólk	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Skyr, berjaskyr	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Rjómaskyr, þykkmjólk, engjaþykkni	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Sykur á morgunkorn, skyr, súrmjólk, graut	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐

8. Mjólk út á graut, morgunkorn eða skyr

Hversu oft hefurðu þessar vörur sem útálát? (Ekki til drykkjar eða í kaffi, te)

Fæðutegund	Aldrei	Í mánuði skipti			Í viku skipti			Á dag skipti			
	Aldrei	Sjaldnar en 1	1	2-3	1	2-3	4-6	1	2	3-4	5 eða oftar
Nýmjólk	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Léttmjólk, sælumjólk	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Undanrenna, fjörmjólk	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Rjómi	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Rjómabland, kaffirjómi	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐

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9. Ávextir og ber. <u>Hversu oft</u> borðar þú ávexti og ber?

Fæðutegund	**Aldrei**	**Í mánuði** skipti			**Í viku** skipti			**Á dag** skipti			
	Aldrei	Sjaldnar en 1	1	2-3	1	2-3	4-6	1	2	3-4	5 eða oftar
Epli, perur	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Appelsínur, mandarínur, greip, kíví	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Bananar	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Rúsínur, sveskjur, döðlur	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Aðrir ávextir eða ber	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐

10. Súpur, eftirréttir

Ef um er að ræða matarmiklar súpur skal skrá kjöt, fisk, grænmeti, kartöflur o.s.frv. úr súpunni á viðeigandi stað í spurningum nr. 12 til 16 en skrá sjálfa súpuna hér.

Fæðutegund	**Aldrei**	**Í mánuði** skipti			**Í viku** skipti			**Á dag** skipti			
	Aldrei	Sjaldnar en 1	1	2-3	1	2-3	4-6	1	2	3-4	5 eða oftar
Súpa, tær	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Súpa, þykkt eða rjómalöguð	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Búðingur	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Ís, frómas	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐

11. Heitur matur. Yfirlit. <u>Hversu oft</u> borðar þú þessar fæðutegundir eða rétti?

(Ekki telja með álegg á brauð).

Fæðutegund	**Aldrei**	**Í mánuði** skipti			**Í viku** skipti			**Á dag** skipti			
	Aldrei	Sjaldnar en 1	1	2-3	1	2-3	4-6	1	2	3-4	5 eða oftar
Fiskur, fiskfarsréttir	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Kjöt, pylsur, hamborgari, hakkréttir	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Pizza	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Pastaréttir (**<u>sem aðalréttur</u>**)	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Grænmetis-, bauna- og hrísgrjónaréttir (**<u>sem aðalréttur</u>**)	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Eggjaréttir (**<u>sem aðalréttur</u>**)	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐

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12. *Fiskréttir.* **Hversu oft** borðar þú þessa fiskrétti?

Fæðutegund	Aldrei	Í mánuði skipti			Í viku skipti			Á dag skipti			
	Aldrei	Sjaldnar en 1	1	2-3	1	2-3	4-6	1	2	3-4	5 eða oftar
Soðinn nýr fiskur, bakaður, glóðaður	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Steiktur fiskur	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Fiskibollur, fiskibúðingur	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Saltfiskur, reyktur fiskur	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐

13. *Kjötréttir.* **Hversu oft** borðar þú þessa kjötrétti?

Fæðutegund	Aldrei	Í mánuði skipti			Í viku skipti			Á dag skipti			
	Aldrei	Sjaldnar en 1	1	2-3	1	2-3	4-6	1	2	3-4	5 eða oftar
Hakkréttur, hamborgari	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Pylsur, kjötfars, kjötbúðingur	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Bjúgu	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Feitt og millifeitt kjöt (t.d. kótilettur, frampartur)	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Magurt kjöt (t.d. læri, gúllas eða fita skorin burt)	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Saltkjöt, hangikjöt, reykt kjöt	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Blóðmör, lifrarpylsa, lifur	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Kjúklingur, annað fuglakjöt	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐

14. *Meðlæti.* **Hversu oft** borðar þú þessar fæðutegundir sem **meðlæti**

Fæðutegund	Aldrei	Í mánuði skipti			Í viku skipti			Á dag skipti			
	Aldrei	Sjaldnar en 1	1	2-3	1	2-3	4-6	1	2	3-4	5 eða oftar
Bakaðar baunir	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Egg (soðin, steikt, hrærð)	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Kartöflur (soðnar, bakaðar)	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Kartöflur (steiktar, franskar)	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Hrísgrjón	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Pasta, spaghetti	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Hamborgara-, pylsu-, pítubrauð	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐

15. Grænmeti. Hrátt eða soðið. <u>Hversu oft</u> borðar þú:

Fæðutegund	Aldrei	Í mánuði skipti			Í viku skipti			Á dag skipti			
	Aldrei	Sjaldnar en 1	1	2-3	1	2-3	4-6	1	2	3-4	5 eða oftar
Hrátt grænmeti, grænmetissalat	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Soðið grænmeti (nýtt, frosið, niðursoðið)	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐

16. Grænmeti. <u>Hversu oft</u> borðar þú þessar <u>**grænmetistegundir**</u>? Teldu bæði með grænmeti sem fer í pottrétti og blandaða rétti eða borðað sér. Ekki skal telja með grænmeti sem álegg á brauð eða pizzur.

Fæðutegund	Aldrei	Í mánuði skipti			Í viku skipti			Á dag skipti			
	Aldrei	Sjaldnar en 1	1	2-3	1	2-3	4-6	1	2	3-4	5 eða oftar
Gulrætur, rófur	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Hvítkál, blóm-, spergil-, rauðkál	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Grænar baunir, maískorn frosin grænmetisblanda	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Laukur, púrra, hvítlaukur	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Gúrka, tómatur, paprika, salat	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐

17. Sósur og feiti. Hér er spurt <u>**hversu oft**</u> þú borðar sósur og feiti, hvort heldur er með fiski, kjöti grænmeti, salati eða pasta. <u>Telja skal með sósur í pottréttum og í bökuðum fiskréttum</u>. Einnig skal telja með sósur með hamborgurum, pítum, frönskum kartöflum og þess háttar.

Fæðutegund	Aldrei	Í mánuði skipti			Í viku skipti			Á dag skipti			
	Aldrei	Sjaldnar en 1	1	2-3	1	2-3	4-6	1	2	3-4	5 eða oftar
Smjör, smjörvi, kalt, brætt	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Létt og laggott, Klípa, Létt smjörlíki	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Annað smjörlíki	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Flot, tólg	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Brún sósa, sósa úr tómötum, grænmeti	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Rjómasósa, ostasósa, sýrður rjómi	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Mjólkurjafningur, sósa úr súrmjólk, jógúrti	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Hollandais-, bernaissósa	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Kokteilsósa, remúlaði, hamborgarasósa	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Salatsósa í flösku, létt sósa, létt majónes	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐

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18. Sælgæti, snakk. <u>**Hversu oft**</u> borðar þú þessar vörur?

Fæðutegund	Aldrei	Í mánuði skipti			Í viku skipti			Á dag skipti			
	Aldrei	Sjaldnar en 1	1	2-3	1	2-3	4-6	1	2	3-4	5 eða oftar
Súkkulaði, súkkulaðikex	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Brjóstsykur	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Lakkrís (1-3 stk)	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Lakkrís (poki)	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Hlaup, Ópal, Tópas	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Poppkorn	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Kartöfluflögur, skrúfur, saltkex	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Salthnetur	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Harðfiskur, hákarl	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Ídýfur	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Ostur, ekki sem álegg	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐

19. Tekurðu vítamín, lýsi eða önnur fæðubótarefni?

Fæðutegund	Aldrei	Í mánuði skipti			Í viku skipti			Á dag skipti			
	Aldrei	Sjaldnar en 1	1	2-3	1	2-3	4-6	1	2	3-4	5 eða oftar
Fjölvítamín **með** A- og D-vítamíni	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Fjölvítamín **án** A- og D-vítamíns	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Lýsi (þorska-, ufsa-)	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Lýsispillur	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
E-vítamíntöflur	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
B-vítamíntöflur	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Fólasíntöflur (fólín, fólínsýra)	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
C-vítamíntöflur	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Járntöflur, járnmixtúra	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Kalktöflur	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Omega-3 hylki, hákarlalýsishylki	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐

MR98-09

20. *Máltíðir.* Merktu við **hversu oft** þú borðar þessar máltíðir eða millibita.
Drykkur án annarrar fæðu telst hvorki máltíð né millibiti.

Fæðutegund	Aldrei	Í mánuði skipti			Í viku skipti			Á dag skipti			
	Aldrei	Sjaldnar en 1	1	2-3	1	2-3	4-6	1	2	3-4	5 eða oftar
Morgunverður	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Millibiti að morgni	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Hádegisverður	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Síðdegiskaffi, síðdegishressing	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Kvöldverður	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Kvöldhressing	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Millibiti að nóttu	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐

21. Hversu oft eru þessar fitutegundir notaðar til steikingar þar sem þú borðar?
(þegar matur er steiktur á annað borð)

Fæðutegund	Veit ekki	Sjaldan eða aldrei	Stundum	Oftast eða alltaf
Steikingasmjörlíki	☐	☐	☐	☐
Soja-, sólblóma-, maís-, matarolía	☐	☐	☐	☐
Ólífuolía	☐	☐	☐	☐
Smjör	☐	☐	☐	☐

MR98-10

22. *Hvað færðu þér yfirleitt mikið af hverju fyrir sig?*

Skoðaðu ljósmyndirnar af mismunandi skammtastærðum á næstu blaðsíðum og merktu við í töflunni hér á eftir hvaða skammtur er líkastur því sem þú færð þér oftast af hverri fæðu eða sambærilegri fæðu. Mundu að taka með í reikninginn ef þú færð þér yfirleitt oftar en einu sinni á diskinn, þá þarftu að áætla samanlagt magn.

Fæðutegund	Minna en A	A	B	C	Meira en C
Fiskur	☐	☐	☐	☐	☐
Kjötréttur	☐	☐	☐	☐	☐
Kartöflur	☐	☐	☐	☐	☐
Hrísgrjón	☐	☐	☐	☐	☐
Soðið grænmeti	☐	☐	☐	☐	☐
Grænmetissalat	☐	☐	☐	☐	☐
Hvernig er brauðið smurt	☐	☐	☐	☐	☐

23. *Hér er spurt um algengan sósuskammt í matskeiðum (msk) eða desilítrum (dl)*

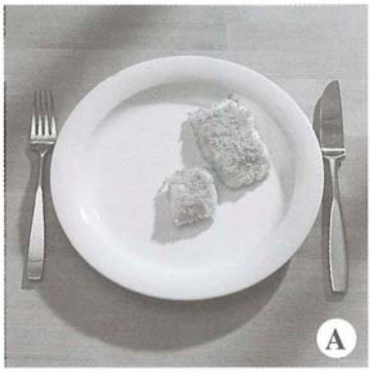
Fæðutegund	Minna en 1 msk	1 msk	2-3 msk	½-1 dl	Meira en 1 dl
Rjómasósa, ostasósa	☐	☐	☐	☐	☐
Kokteilsósa, salatsósa	☐	☐	☐	☐	☐
Feiti	☐	☐	☐	☐	☐

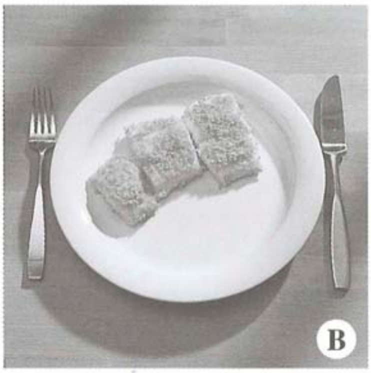
Bestu þakkir fyrir þátttökuna!

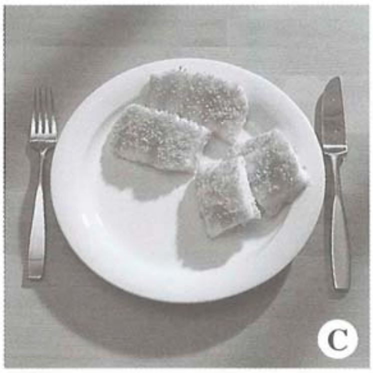
Veldu þá skammtastærð sem mest líkist því sem þú færð þér oftast

Færðu svörin inn í spurningu 22 hér að framan

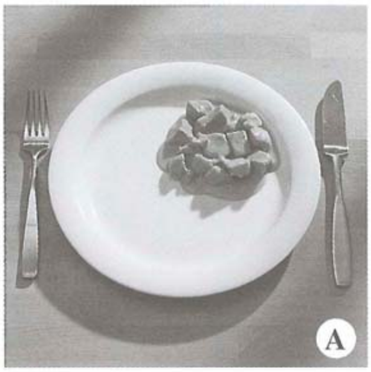
Fiskur ______

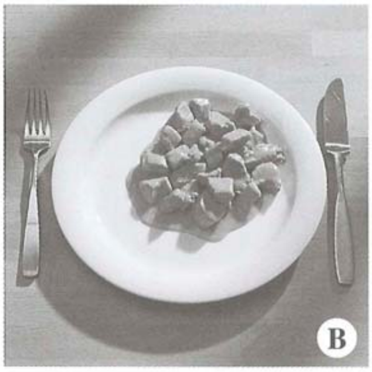


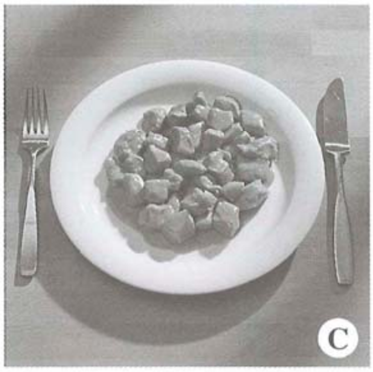




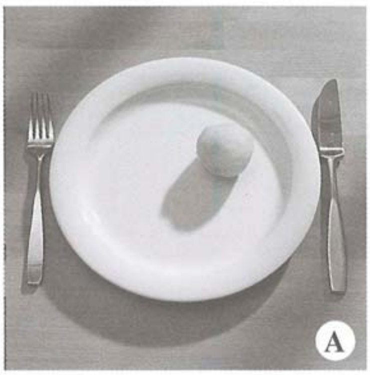
Kjötréttur ______

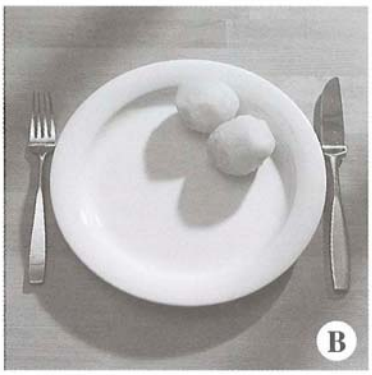


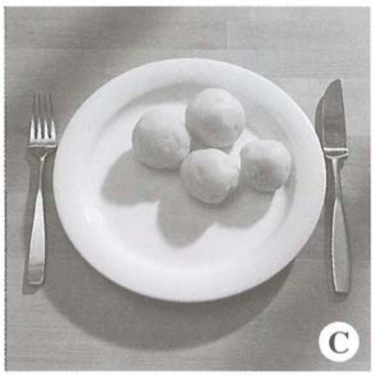




Kartöflur ______



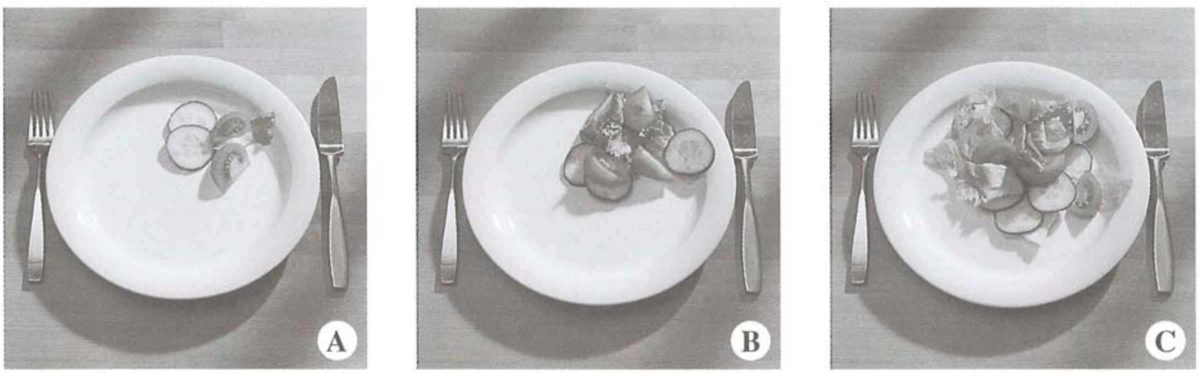




Hrísgrjón

Soðið grænmeti

Grænmetissalat



Hvernig er brauðið smurt