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A survey of the iodine content of school-children diets fortified with marine fish

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The present study was carried out in an iodine deficient area in Northern Ghana, and the purpose of the study was: (1) to undertake a food dietary survey on school children in an iodine deficient area, (2) measure the iodine content in the staple food of this area, (3) carry out an intervention study with a traditional food supplemented with marine fish as a natural iodine source. Sixty school children, in the village of Sekoti in the Upper-East region of Ghana, participated in the study. The dietary survey was performed as repeated 24 h recall, and staple foods were analysed for iodine by ICP-MS. A traditional diet fortified with 10% codfish-powder was given to the children every day for two weeks, and iodine status was measured before and after the intervention. The foodstuffs normally eaten by the children contained little iodine. However, beans prepared at a market nearby had surprisingly high values of iodine, explained by the addition of saltpetre, which contained very high amounts of iodine. About 60% of the children in the study area had goitre and the prevalence was highest among girls. Following the intervention period, the urinary iodine concentration was significantly increased and the TSH level in serum was significantly decreased, indicating an elevated iodine status. The children having goitre had the strongest tendency to elevate their iodine status.

Key words: Iodine, Fish, Goitre, Urinary iodine, TSH.

INTRODUCTION

Iodine deficiency disorders (IDD) continue to be a major health problem in the world, particularly in Third World countries. Globally, the prevalence of goitre in 1999 was estimated to be 13% (WHO/UNICEF/ICCIDD, 2001). However, the prevalence of IDD is likely to be greater than this. Results based on prevalence of urinary iodine (UI) below 100 µg/l, estimates that 36.5% of school children worldwide have an inadequate iodine nutrition. This number is 42.3% in Africa and as high as 59.9% in Europe (WHO, 2004). In 1998, Asibey-Berko and co-workers reported that goitre rate of children (8 - 14years) in Sekoti (Northern Ghana) was 59.1% and median urinary iodine for both Sekoti and Buila (also in Northern Ghana) was 16 µg/l. A national survey in 2001 found that

the median urinary iodine was 196 µg/l, while the national goitre rate was 20% with a range of 7% to 30% (NSEID 2007). The most severe consequence of IDD is irreversible brain damage, which can affect up to 5 - 15% of a population in severely affected areas (WHO, 2004). Iodine deficiency is the single most important cause of preventable mental handicap in the world by the turn of the century (WHO/UNICEF/ICCIDD, 2001). The physiological function of iodine is as part of the thyroid hormones thyroxin (T4) and the more active form triiodotyronine (T3), which influences metabolism in all cells, including stimulation of growth and development of the central nerve system (Delange and Fisher, 1995).

The recommendations of dietary iodine given by NRC (1989) were critically reviewed by Delange (1993) and the recommendations were set at 90 µg/day for children 0 to 6 years and 120 µg/day for children 6 to 12 years. For all persons older than 12 years the recommendation is 150

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µg/day. However 200 µg/day is recommended for pregnant and lactating women (WHO/UNICEF/ICCIDD, 2001).

Several indicators are used to assess IDD in individuals and communities, including the size of the thyroid gland and urinary iodine. The size of the thyroid gland is inversely correlated with the average iodine intake, and indicates a person's iodine status (WHO/UNICEF/ICCIDD, 2001). Urinary iodine excretion is a commonly used parameter to evaluate iodine status, showing a strong correlation with the iodine intake (Vought et al., 1963). Blood constituents are also used to assess the iodine status. The level of thyroid-stimulating hormone (TSH) and thyroglobulin (Tg) increases when the iodine intake is low (Delange, 1995).

In 1992 United Nations put forward a plan to eradicate iodine deficiency as a major health problem within the year of 2001 (FAO/WHO, 1992) through increasing: 1) iodine fortification of foods and food ingredients such as salt, water and flour; 2) distribution of iodine capsules in areas where IDD is severe, 3) use of iodine rich nutrients, like seafood, and decreasing the consumption of goitrogens. Although the last decade has seen significant progress in combating IDD, particularly in Africa and Asia, the total number of people affected by IDD is still high. Accoriding to WHO/UNICEF/ICCIDD (2001) the IDD problem was underestimated in earlier studies and eradication policies have not been as effective as expected.

Iodine is normally obtained from food but most foods are poor sources of the element with the exeption of sea foods which are good sources of iodine (Lee et al., 1994; Coultate, 1996; Eckhoff and Maage, 1997; Hou et al., 1997; Karl et al., 2001; Rose et al., 2001). The iodine content in fish is typically in the range of 300 to 1000 g/kg while most vegetable product had less than 30 g/kg. Marine fish species low in fat, like cod and saithe have the highest iodine contents (Dahl et al., 2004).

In some Africa countries iodine deficiency has been a common problem, especially in areas remote from the sea, and Northern Ghana has been no exception with reports on high goiter prevalence (Asibey-Berko, 1995). The recognition of severe IDD problems in Ghana prompted the campaign to use iodized salt. However, household iodized salt use has been low and estimated at about 28 % as of 1998 (NSEID, 2007) . It is therefore also important to study further measures to improve the iodine status in communities. The present study was undertaken to study iodine nutrition and the possible use of marine foods in an intervention to improve this situation.

The aim of this study was:

1) To perform a small dietary survey on school-children.
2) To collect staple foods in the study area, and measure the iodine content of these foodstuffs.
3) To perform an intervention study to evaluate changes in iodine status in school children, following a period of

consuming of traditional foods fortified with marine fish.

METHOD

Study area and subjects

Sekoti, a village 35 km east of Bolgatanga in the Upper -East Region, Ghana, was chosen for the study since it is noted for rates of IDD (Asibey-Berko, 1995). Sixty school children ages 10 to 12 years (23 girls and 37 boys) were randomly selected and participated in the study. The study was approved by an institutional review board and all parents provided informed consent for their children after the study has been explained to them in the local language. The village chief together with his elders permitted the study to be carried out in the village. The study was carried out from February 26th to March 12th 1996.

Dietary assessment

To obtain information on the iodine intake of the children, it was necessary to conduct dietary evaluation. A repeated 24 h recall was conducted each morning with each child to obtained information on the food eaten the previous day. Two trained graduate nutrition students interviewed the children using open ended questionnaire. Two teachers in the village were given a short orientation and served as interpreters for the period. The interviews went fast since food choices in the village are limited. Samples of food portions were shown to the children to help them quantify the amount eaten. Children were asked the type of foods eaten as a composite or single food item. The questionnaire covered all foods eaten through out the day, that is, breakfast, lunch dinner and all snacks. The dietary assessment was necessary since it was the key to knowing what the participants are eating to be able to determine the iodine content of the foods.

Both raw food materials and prepared meals eaten by the participants were collected in Sekoti, or at the market in Bolgatanga for iodine analysis. This was necessary since using literature values for iodine contents of the foods might not give true estimates. The materials sampled were stored in polyethylene bags, frozen transportation to the University of Ghana, Legon. The samples were later air freighted to NIFES, Bergen, Norway, where they were freeze-dried, homogenised and stored in 50 ml Nunc-cups prior to analysis

Intervention diet

The intervention diet was fermented maize/fish blend and this provided each child 460 µg/day of iodine. Table 1 profiles the iodine content in the raw food materials as well as the prepared meals. White maize meal (*Zea mays*) was bought from Indus, Birmingham, UK. For each 5 kilos of maize meal, 4 L of tap water and 1 L of ABC milk (Tine Norske Meierier, Bergen, Norway), containing the bacteria *Lactobacillus acidophilus* (A), *Bifidobacterium bifidum* (B) and *Lactobacillus casei* (C), were mixed to a dough in a food processor (Varimixer Bjørn 93/AR60, Brøndby, Denmark). The dough was then transferred to aluminium trays (20 l), covered with polyethylene foil and fermented at 30ºC for 48 h, until a stable pH of 3.7 was reached. The fermented product was frozen at -20ºC, then vacuum- dried and fortified with 10% (w/w) codfish powder if saithe, *Gadus virens* (Toro A/S, Rieber and Søn, Bergen, Norway): The maize/fish blend was pulverised, bagged into polyethylene bags and packaged into boxes and air freighted to Accra, Ghana. The product was stored at the Nutrition and Food Science Department, University of Ghana prior to the start of the intervention program. The fish maize blend was used to prepare breakfast (8 a.m.) and lunch (1 p.m.) for the children. Breakfast was a popular porridge

Table 1. Dry matter and content of nutrients analysed of raw materials and prepared meals of the intervention diet (dry matter base).

Foodstuff	Dry matter (%)	Protein [1](%)	Fat[1] (%)	Energy[1] MJ/kg	Iodine (n)(mg/kg)	
Codfish powder	95	-	-	-	20.5	(3)
Fermented maize meal	96	-	-	-	0.039[2]	(4)
Fermented maize meal + 10 % codfish	4	15.5	3.4	17.6	2.13	(4)
Koko, prepared	18	12.3	0.7	17.3	1.34	(2)
Banku, prepared	34	15.5	1.0	-	1.82	(2)
Banku and gravy, prepared	32	15.5	6.4	18.0	1.73	(2)

[1]Analysed by Skov (1997).
[2]ABC milk (Tine Norske Meierier, Bergen, Norway) added contained some iodine.

called koko and each child received about 500 ml (2 cups) . Koko was made of 4.5 kg of the raw material mixed with 30 litres of water. The mixture was boiled for about 30 min added 1.5 kg of sugar, and then distributed equally into 60 cups. Lunch was also a popular dumpling called banku and each child received about 600 g. Banku was made by mixing 12.0 kg of the maize/fish blend with 25 L of water, added salt (uniodized) to taste and cooked for about 1 h with constant stirring and kneading. Banku was served with gravy made of 6 kg of tomatoes, 0.6 litres cooking oil (Frytol), red pepper and salt (not iodised).

The preparation and the serving of the food were under the supervision of a home economist/nutritionist. Children ate all the food served at any given time.

Data collection

Baseline and follow-up data collection were done in the mornings before breakfast. Demographic data collected were age and sex. Anthropometric measures included mid-upper arm circumference as well as height and weight for body mass index determination. Physical appraisal by palpation was done by an experienced person to determine goitre grades in the children (Delange, 1995). Physiological samples collected were urine and blood. The field conditions allowed for spot collection of urine. The urine samples were collected in 20 ml polyethylene tubes and stored at -20ºC. Blood samples were collected as drops of whole blood on filter paper (Schleicher and Shuell, grade 903), air dried, put into a polyethylene bag, covered with aluminium foil and then stored at -20ºC.

Analyses

The food, water and urine samples where analysed for iodine by Inductively Coupled Plasma-Mass Spectrometry (ICP-MS) type Perkin Elmer ELAN 5000 A ICP-MS (Ontario, Canada) using tellurium as internal standard. The method is decribed in Eckhoff and Maage (1997) and further in Julshamn et al. (2001). Two replicate portions (0.2 g each) of the dried and pulverised food samples were added nitric acid and perhydrol before digestion in a microwave oven (Milestone- 1200 MEGA, Sorisole, Italy), following addition of internal standard and then diluted to 25 ml. Immediately before the ICP-MS analysis the digested samples were diluted 1:2 with a 3% NH_3 solution. The water samples brought from Ghana

were not digested but some droplets of concentrated NH_3 were added before analyses. The urine sam-ples were thawed overnight at 18ºC. The internal standard was then added and the sample diluted 1:10 with 1% HNO_3 prior to the ICP-MS analysis. The detection and quantification limit of the method was determined to be 0.2 and 0.6 g/l respectively. For analytical quality control of the

iodine analyses, the standard reference materials (SRM) 1566a Oyster Tissue from NIST, USA and BCR 129 Hay Powder from BCR, Belgium were used. Those represented a wide range of iodine concentrations from 167 ± 24 g/kg in the hay to 4460 ± 420 in the oyster tissue. Our analyses of the reference material were well within the uncertainty of the SRM. Since there at the time of analyses were no certified reference materials for iodine in urine, a recovery test was also performed with good results.

The blood samples, collected on filter paper, where analysed by using the enzyme-linked immunosorbant assay (ELISA) methodology. A TSH assay kit (SpectraScreen Dried Blood TSH EIA Kit, EIM Diagnostics, Santee, CA, USA), based on a standardised method (Tseng et al., 1985; Miyai et al., 1981) was used for the analysis.

Statistical procedures

To find significant differences prior to and following the intervention period different tests for dependent variables were used. The level of significance is $P < 0.01$ if nothing else is mentioned. The distribution of the variables was visually checked for normality by using categorised normal probability plot. The results are given as medians and quartiles when the variables were not normally distributed. The result from normally distributed variables is given as means and standard deviations.

The two variables iodine in urine and TSH in serum did not show normality, the nonparametric Wilcoxon matched pairs test was therefore used to test if the changes were significant after the consumption of the intervention diet. For the normally distributed variables weight and upper arm circumference, Student's t-test for dependent variables was used to check if the changes were significant. To test differences between the independent variables boys and girls Mann-Whitney U-test was used for the variables showing no normal distribution, and Student's t -test for independent variables for the normally distributed variables. When looking at the iodine excretion in urine, and TSH level in blood, the differences between groups graded according to the grade of goitre, were tested by the non-parametric Kruskal-Wallis ANOVA median test.

RESULT

Dietary habit

During the 14 days intervention period, the food mostly consumed by the children other than the intervention diet was millet/rice balls ("Tou Zaafi"-TZ) and this was 34% of the food-volume consumed. This was followed by rice which contributed 19%, and then tubani (cowpea paste) which contributed 15% of the food eaten during the

Table 2. Dry matter, energy and iodine analysed in raw materials and prepared meals of the staple foods (dry matter base). Data are presented as means of to parallel determinations.

	Dry matter (%)	Energy (kJ/g)	Iodine (µg/kg)	Place obtained
Yam *Dioscorea sp.*, roasted	58.7	15.9	63	Bolgatanga, market
Gari (cassava *Masihot utilissima*), raw	93.6	16.1	<16	Bolgatanga, market
Cowpea beans *Vigna ungiculata*, raw	95.6	-	25	Bolgatanga, market
Bambara beans *Phaseolus sp.*, raw	95.4	-	19	Bolgatanga, market
Rice *Oryza sativa*, raw	92.1	16.0 [4]	<16	Sekoti, market
Beans *Vigna ungiculata*, prepared	27.2	18.1	234	Bolgatanga, market
Rice/beans, prepared	35.4	-	100	Bolgatanga, market
Sorghum *Sorghum vulgare*, prepared	31.5	17.1	48	Sekoti, school
Fufu (cassava + plantain *Musa paradisica*), prepared	26.6	16.9	46	Bolgatanga, bus-station
Kenkey (maize *Zea mayz*), prepared	32.0	17.7	102	Bolgatanga, market
Tubani (beans), prepared	41.2	19.0	74	Sekoti, school
Tubani (beans), prepared	45.4	19.7	538	Bolgatanga, market
TZ (millet *Pennisetum americanum*), raw	88.2	-	45	Bolgatanga, market
TZ (millet), prepared	19.9	17.0	93	Bolgatanga, market
TZ (millet), prepared	23.3	18.8	110	Bolgatanga, restaurant
Okra *Hibiscus esculentus*, dried	96.6	17.4	518	Bolgatanga, market
Tombrown (maize + cowpea), roasted	95.1	18.0	400	Bolgatanga, market
Tomatoes *Lycopercsicon esculentum*, prepared	27.2	-	949	Sekoti, school
Groundnutpaste *Arachis hypogaea*, raw	93.2	20.9	135	Bolgatanga, market
Salt	99.0	-	119	Bolgatanga, market
Salpetre	98.5	-	50 400	Ghana

[1] Salpetre added under preparation of beans.
[2]Ashed sorghum stalk added instead of salpetre.
[3]TZ made of millet meal. Rice meal is sometimes used instead.
[4]Value obtained from Matvaretabellen (Statens ernæringsråd, 1995).

period. The remaining 7% is from snacks mostly wild fruits that the children picked in and around the village. Food choices and household food availability is limited, hence children received one meal at school which is normally sorghum. However, during the intervention the 60 participants did not eat the school food (sorghum) but instead received the intervention diet (banku and koko) and this formed 25% of the bulk of food eaten.

Iodine content of foods and other food additives

The iodine content of foods mostly eaten and available in the region is shown in Table 2. The iodine content in the foods ranges from less than 16 µg/kg (raw cassava and rice) to 538 µg/kg (tubani). Salpetre which is usually a food additive in Ghana contains the highest amount of iodine (50,400 µg/ kg) and it boosted the iodine levels in foods where it had been added. The two main water sources in the area are well and borehole and both showed very low iodine concentrations; 0.2 and 0.8 µg/l respectively.

Antropometric indicators

The mean values of weight, height, Body Mass Index (BMI) and mid-upper arm circumference are presented in Table 3. A fortnight is too short a time to see any significant change in height for children, however, weight did increase significantly on average by 0.9 kg, (P < 0.01). BMI which is an index including weight also increased significantly by 0.4 kg/m^2 (P < 0.01). Mid- upper arm circumference also increased significantly by 0.4 cm (p < 0.01). Nonetheless, when stratified by gender, no significant difference was found between boys and girls, for weight, BMI and mid- upper arm circumference.

Iodine status

The prevalence of goitre was more than 60% among the children. However, 10% of them showed visible goitre (Table 4). Urinary iodine increased significantly, from 34 to 79 µg/l (p < 0.01) by the end of the intervention while reatinine decreased (Table 5). Baseline correlation bet-

Table 3. Weight, height, upper arm circumference and Body Mass Index (BMI) of the school children in Sekoti, Ghana, prior to and following the consumption of the fish containing experimental diet. Significant differences (P < 0.01) within a column are shown by different superscripts. Data are presented as means (SD), n = 54.

	Weight (kg)	Height (cm)	Upper arm circumference (cm)	BMI (kg/m^2)
Before intervention	30.3[a] (4.1)	137 (7)	18.3[a] (1.3)	16.0[a] (1.2)
After intervention	31.2[b] (3.9)	-	18.7[b] (1.3)	16.4[b] (1.1)

Table 4. Distribution of goitre among 10 to 12 years old schoolchildren in Sekoti, Ghana.

Sex (n)	No goitre (%)	Total goitre rate (%)	Goitre, invisible (%)	Goitre, visible (%)
Girls (23)	30	70	52	18
Boys (37)	43	57	52	5
Both (60)	38	62	52	10

Table 5. Iodine concentration, creatinine concentration and the ratio iodine/creatinine in urine samples from schoolchildren in Sekoti, Ghana, prior to and following the consumption of the fish containing diet. Significant differences (P < 0.01) within a column are shown by different superscripts. Data are presented as medians and precentiles (25th - 75th), n = 54.

	Iodine(µg/l)	Creatinine(g/l)	Iodine/creatinine(µg/g)
Before intervention	34[a] (20 - 53)	1.37[a] (0.76 - 1.66)	28[a] (20 - 47)
After intervention	79[b] (37-122)	0.70[b] (0.34-1.32)	115[b] (84 - 156)

between creatinine concentration and iodine/creatinine ratio was significant at start of the intervention (r = 0.39, p < 0.01). Figure 1 depicts the relationship between iodine concentration in urine and the level of goitre. As the level of iodine increased the occurrence of goitre decreased. The concentration of iodine in urine increased after consumption of the fish diet but the increase was significant for the groups without and with invisible goitre (p < 0.01). No significant change was found in the group having visible goitre. There was no significant difference in the concentration of iodine in urine for boys and girls. Figure 2 shows the TSH level in serum before and after the intervention period. The two groups having goitre, invisible and visible, showed a significant decrease in serum TSH level (p < 0.05) after eating the fish con-taining diet.

The group without goitre did not show any significant change in serum TSH level. When stratified by sex, the decrease in serum TSH was only significant among the girls (P < 0.05).

DISCUSSION

Food habits and iodine assessment of foods

Millet/rice balls ("Tuo Zaafi"-TZ), sorghum, tubani (cow-pea paste) and rice contributed more than 90% of the food normally consumed by the schoolchildren in Sekoti.

These carbohydrate rich foods contained low levels of iodine, and did only cover a minor part of the children's recommended daily intake of 120 µg (WHO/UNICEF/ICCIDD, 2001). The low levels of iodine in the raw materials of cereals and beans reflects the low iodine content in the soil in the area were they are grown (Hetzel and Maberly, 1986). The iodine content in the water samples was insignificant, also reflecting the low iodine content in the soil. Tomatoes and okra had higher levels of iodine than the cereals and the beans, probably because they are grown on farms closer to coastal areas, where iodine levels in the soil are higher (Hetzel and Maberly, 1986). Meals were usually served with a stew, often made of tomatoes, okra or groundnuts, which resulted in a slight increase in the iodine content in most of the dishes.

Dishes of beans and tubani bought at the market in Bolgatanga contained surprisingly high amounts of iodine. These high levels of iodine can be explained by the addition of saltpetre during the food preparation. Saltpetre was added to get the desired texture and taste, and had an iodine content of more than 50,000 µg/kg. At Sekoti, however, where the children involved in the study lived, ashed sorghum stalk was used instead of saltpetre as a cheaper alternative. This explains the low levels of iodine found in the prepared beans and tubani bought there. The salt used for food preparation was not iodised, and contained therefore insignificant levels of iodine.
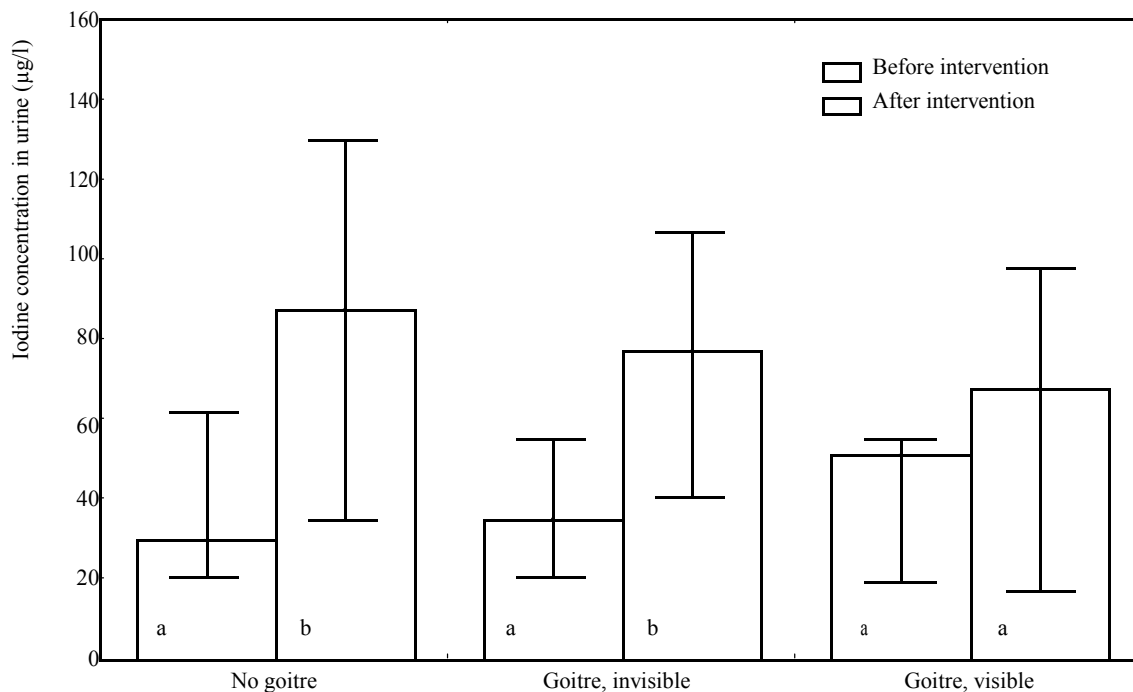
Figure 1. Urinary iodine concentrations of the schoolchildren in Sekoti, Ghana, according to the grade of goitre. Significant difference (P < 0.01) prior to and following the consumption of the fish containing diet within a group is shown by different letters. Data are presented as medians with 25th and 75th percentiles. For the group having no goitre n = 20, n = 28 for the group having invisible goitre and n = 6 for the last group with visible goitre.

The low levels of iodine in the staple foods could be the major explanation of the high incidence of IDD in the Sekoti area. However, other factors than low nutritional iodine may contribute to goitre and diseases related to iodine deficiency. Goitrogenes and selenium deficiency are such factors. Some foods contain anions like tiocya-nate, nitrate and nitrite, which all are goitrogenes (Brown-Grant, 1961; Delange and Fisher, 1995; Ubom, 1991; Fordyce et al., 2000). Saltpetre, which had a high iodine concentration, contains nitrate which can inhibit the utili-sation of iodine (Tajtakova et al., 2006). In Sekoti, millet was one of the most important staple foods, which con-tains a goitrogen called vitexin (Gaitan et al., 1995; Elnour et al., 1997). A probably high intake of vitexin may also contribute to the high incidence of goitre recorded in Sekoti. Selenium deficiency could be a factor contributing to the IDD symptoms as selenium is a part of type I 5`-deiodinase which converts T4 to the more active T3 hormone (Meinhold et al., 1993; Roti et al., 1993). How-ever, selenium status of participants was not measured.

Changes in nutritional status

According to Túron and Chew (1994) protein energy mal-nutrition (PEM), at ages 11 to 13 years, was diagnosed based on a BMI below 15.0 kg/m^2. In the present study, the schoolchildren had at the beginning an average BMI

slightly higher than this cut-off point, and the BMI increased significantly by 0.4 kg/m^2 during the study. The improved nutritional status was also confirmed by the increase in upper arm circumference and the significant weight increase. The consumption of the experimental diets thereby showed a positive effect on the nutritional status, which might be a result of increased dietary pro-tein quality, a higher food consume than normal and an increased intake of specific nutrients like iodine, which had an obvious positive effect on the children's iodine status.

The prevalence of goitre in the Sekoti area was esti-mated to be 59% in 1991, and the area was then defined as an area with severe iodine deficiency (WHO/UNICEF/-ICCIDD, 1993). The same prevalence of goitre was found in this study when started in 1996 and showed that iodine deficiency was still a large problem in this area. The researchers were told that iodine capsules were distri-buted to the population two years prior to the present stu-dy. This seemingly did not correct the low iodine status of the schoolchildren two years after the distribution of the capsules.

Iodine status

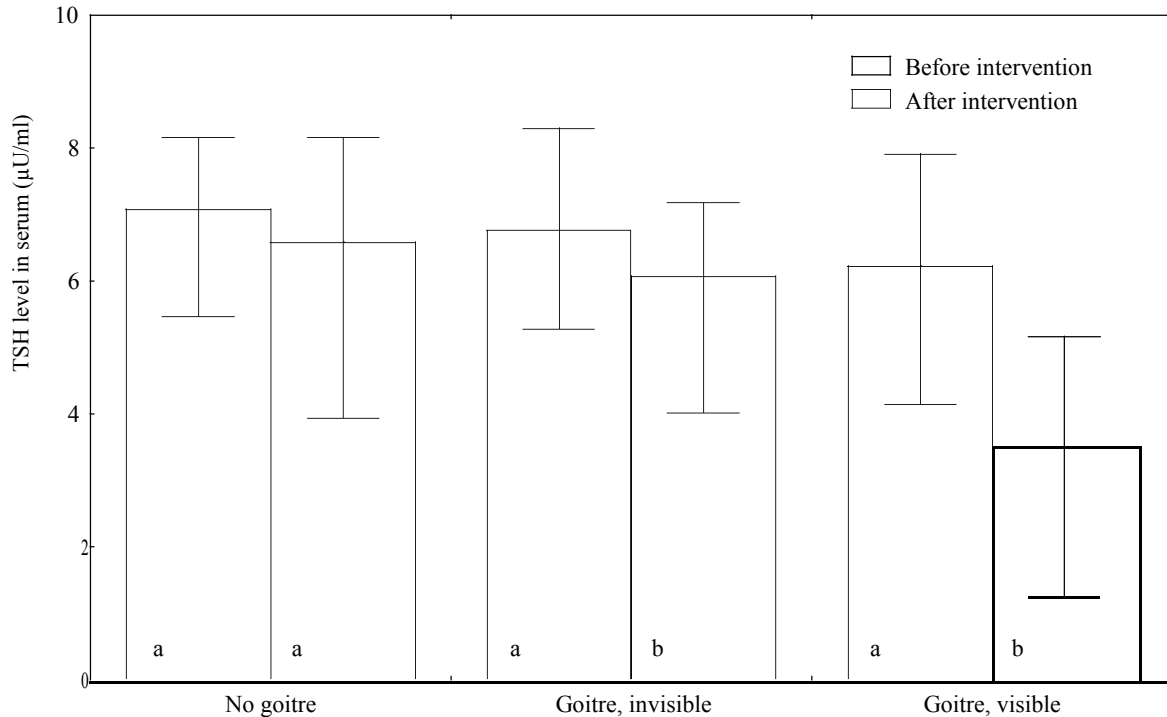Urinary iodine concentration is the major parameter for

Figure 2. TSH levels in serum of the schoolchildren in Sekoti, Ghana, according to the grade of goitre. Significant difference (P < 0.05) prior to and following the consumption of the fish containing diet within a group is shown by different letters. Data are presented as medians with 25th and 75th percentiles. For the group having no goitre n = 20, n = 28 for the group having invisible goitre and n = 6 for the last group with visible goitre.

evaluating iodine deficiency. In casual urine samples the concentration of iodine can be related to urinary createnine (Vought et al., 1963) . However, the urinary iodine-iodine- creatinine ratio is not a good indicator in cases where this ratio is significantly correlated with the createnine concentration itself (Furnée et al., 1994). In this study we also measured this ratio but the iodine-creatinine ratio was not used further as the correlation with the creatinine concentration was found to be significant. The urinary iodine concentrations were therefore preferred as iodine status indicator. According to Bourdoux et al. (1985) the measurement of urinary iodine concentrations in fifty to one hundred casual samples gives a good index for iodine status in a population. Median urinary iodine levels of 20 - 49 µg/l indicate moderate iodine deficiency; median values of less than 20 µg/l indicate severe iodine deficiency (WHO/UNICEF/ICCIDD, 2001). The 25th and 75th percentiles of iodine levels in the urine collected before the intervention was 20 and 53 µg/l respectively, which confirms that IDD was a serious problem in the area. During the study period the average concentration of iodine in urine increased significantly by about two times. The increase was less than expected, because the contribution of iodine from the intervention diet was several times the contribution of iodine from the staple foods usually eaten by the children. It is, however, possi-

ble that the low urinary iodine after eating the intervention diets is an indication that iodine deficient children retained a considerable amount of the ingested iodine.

The TSH concentration in serum decreased signify-cantly as a result of the consumption of the iodine rich diet. For children aged 1 to 15 years the level of TSH in serum is expected to be between 0.6 and 6.3 µU/ml (Delange and Fisher, 1995). In our study, the TSH median value of the school children in the study came below the upper limit of this interval after the intervention period.

The findings from the present study show a relationship between the degree of goitre, urinary iodine concentration and the level of TSH in serum. When grouping the schoolchildren according to the degree of goitre, it was seen that the urinary iodine excretion was not signify-cantly increased in the group having visible goitre, while the TSH level in serum was significantly decreased in this group. In the group having invisible goitre the serum-TSH concentration also significantly decreased and further a significant increase in urinary iodine concentration was seen. The group having no goitre had no significant change in serum TSH, but had the highest increase in urinary iodine concentration. Individuals having goitre seem to have a more efficient iodine trapping mechanism in the thyroid gland, resulting in less iodine excreted by

the urine. More iodine is then available for producing T4 and T3, which in turn reduces the level of TSH in serum. Our data showed higher goitre prevalence among females than among males when the experiment started, the same tendency is reported in other studies from Mali (Fish et al., 1993), Malawi (Furnée et al., 1994) and Sudan (Elnagar et al., 1995). The higher goitre prevalence among the females can explain why it was observed a significant decreased in the TSH level in serum in females while this was not observed in males.

Conclusion/Implication

The high prevalence of goitre in this area showed that iodine deficiency was a major problem. The results from the present study show 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. Even though the use of iodised salt is the major alleviating factor for iodine deficiency, for parts of the population it is not always efficient. Diets containing marine fish could be a helpful supplement in alleviating the IDD in some areas.

ACKNOWLEDGEMENTS

REFERENCE

Asibey-Berko E (1995). Prevalence and severity of Iodine Deficiency Disorders in Ghana. In: Asibey-Berko E, Orraca-Tetteh, R (eds) Proceedings of the national workshop on iodine deficiency disorders in Ghana held in Accra, Ghana. University of Ghana, Ghana, pp. 15-23.

Aisbey-Berko E, Amoah AG, Ado F, Agyepong E (1998). Endemic goitre and unrinary iodine levels in rural communities in the Bolgatanga and Builsa districts of the upper east region of Ghana. East Afr. Med J. 75:5001-5003.

Bourdoux P, Delange F, Filetti S, Thilly C, Ermans AM (1985). Reliability of the iodine/creatinine ratio: a myth? In: Hall R, Köbberling J (eds) Thyroid disorders assosiated with iodine deficiency and excess, Raven Press, New York, pp 145-152.

Brown-Grant K (1961). Extrathyroidal iodide concentrating mechanism. Physiol. Rev. 41:189-213.

Coultate TP (1996). Food - the chemistry of its components, RSC Paperbacks, The Royal Society of Chemistry. 3rd ed. Cambridge.

Dahl L, Johansson L, Julshamn, K, Meltzer, HM (2004). The iodine content of Norwegian foods and diets. Publ. Health Nutr. 7:569-576.

Delange F (1993). Requirements of iodine in humans. In: Delange F, Dunn JT, Glinoer D (eds) Iodine Deficiency in Europe, Plenum Press, New York, pp 5-16.

Delange F (1995). Iodine deficincy in Europe. J. Czech Phys. (Casopis Lékaru Ceských) 134:35-43.

Delange F, Fisher DA (1995). The thyroid gland. In: Brook C (ed) Clinical paediatric endocrinology, 3rd ed., Blackwell publ. Oxford, pp. 397-433.

Eckhoff KM, Maage A (1997). Iodine content of fish and other food products from East Africa analysed by ICP-MS. J. Food Comp. Anal. 10:270-282.

Elnagar R, Eltom M, Karlson FA, Ermans AM, Gebre-Medhin M, Bourdoux PP (1995). The effects of different doses of oral oil on goiter size, urinary iodine, and thyroid-related hormones. J. Clin. Endocrinol. Metab. 80:891-897.

Elnour A, Lieden SÅ, Bourdoux P, Eltom M, Khalid SA & Hombreus L (1997) The goiterogenic effect of two Sudanese pearl millet cultivars in rats. Nutr. Res. 17:533-546.

FAO/WHO (1992). Preventing specific micronutrient deficiencies. International conference in nutrition, theme paper 6, FAO Rome.

Fish A, Pichard E, Prazuck T, Sebbag R, Torres G, Gernez G, Gentilini M (1993). A new approach to combatting iodine deficiency in developing countries: The controlled release of iodine in water by silicone elastomer. Am. J. Public Health 83:540-545.

Fordyce FM, Johnson CC, Navaratna URB, Appleton JD, Dissanayake CB (2000). Selenium and iodine in soil, rice and drinking water in relation to endemic goitre in Sri Lanka. Sci. Tot.Environ. 263:127-141.

Furnée CA, Van der Haar F, West CE, Haustvast JGAJ (1994). A critical appraisal of goiter assessment and the ratio of urinary iodine to creatinin for evaluating iodine status. Am. J. Clin. Nutr. 59:1415-1417.

Gaitan E, Cooksey RC, Legan J, Linsay RH (1995). Antithyroid effects in vivo and in vitro of vitexin: A c-glucosylflavone in millet. J. Clin. Endocrinol. Metab. 80:1144-1147.

Hetzel BS, Maberly GF (1986) Iodine. In: Mertz, W (ed) Trace elements in human and animal nutrition, 5th ed, Academic Press Inc., Orlando, pp 139-197.

Hou X, Chai C, Qian Q, Liu G, Zhang Y, Wang K (1997). The study of iodine in Chinese total diets. Sci. Tot. Environ. 193:161-167.

Jägerstad M, Skog K (2005). Genotoxicity of heat-processed foods. Mutat. Res. 574:156-172.

Julshamn K, Dahl L, Eckhoff K (2001). Determination of iodine in seafood by inductively coupled plasma/mass spectrometry. J. AOAC Int. 84:1976-1983.

Karl H, Münkner W, Krause S, Bagge I (2001). Determination, spatial variation and distribution of iodine in fish. Deutsche Lebensmittel-Rundschau, 97:89-96.

Lee SM, Lewis J, Buss DH, Holcombe GD, Lawrance PR (1994). Iodine in British foods and diets. Br. J. Nutr. 72: 435-446.

Lie , Lied E, Maage A, Njaa LR, Sandnes K (1994). Nutrient content in fish and shellfish. Fisk. Dir. skr., Ser. ernæring 6:83-105.

Meinhold H, Campos-Barros A, Walzog B, Köhler R, Müller F, Behne D (1993). Effects of selenium and iodine deficiency on type I, type II and type III iodothyronine deiodinases and circulating thyroid hormones in the rat. Exp. Clin. Endocrinol. 101:87-93.

Miyai K, Ishibashi K, Kawashima M (1981). Two-site immunoenzymetric assay for thyrotropin in dried blood samples on filter paper. Clin. Chem. 8:1421-1423.

National Research Council (1989). Iodine. In: Recommended Dietary Allowances, 10th ed, National Academy Press. Washington DC, pp. 213-216

NSEID (2007). Country profile, Ghana. Network for Sustained Elimination of Iodine Deficency (NSEID), http://206.191.51.240/countries/ghana.html.

Rose M, Miller P, Baxter M, Appleton G, Crews H, Croasdale M (2001). Bromine and iodine in 1997 UK total diet study samples. J Environ. Monit. 3: 361-365.

Roti E, Minelli R, Gardini E, Bianconi L, Ronchi A, Gatti A, Minoia C (1993). Selenium administration does not cause thyroid insufficiency in subjects with mild iodine deficiency and sufficient selenium intake. J. Endocrinol. Invest. 16:.481-484.

Skov M (1997). Nutrition and humoral immunity in Ghanaian Children; Effects of fish protein supplementation to traditional fermented maize diets Master Thesis, Universitetet i Bergen, Norway. Statens ernæringsråd, Statens næringsmiddeltilsyn (1995). Matvaretabellen (Food composition table), Universitetsforlaget, Oslo.

Tajtakova M, Semanova Z, Tomkova Z, Szökeova E, Majoros J, Radikova Z, Sebökova E, Klimes I, Langer P (2006). Increased thyroid volume and frequency of thyroid disorders signs in school children from nitrate polluted area. Chemosphere. 62(4):.559-564.

Tseng YC, Burmann KD, Baker JR, Wartofsky L (1985). A rapid, sensitive enzymed-linked immunoassay for human thyrotropin. Clin. Chem. 7:1131-1134.

Túron B, Chew F (1994). Protein energy malnutrition. In: Shils ME, Olson JA, Shike M, (eds), Modern nutrition in health and disease, 8th ed, Lea and Febiger, Philadelphia, 1: 950-976.

Ubom GA (1991). The goitre-soil-water-diet relationship: case study in Plateau State, Nigeria. Sci. Tot. Environ. 107:1-11.

Vought RL, London WT, Lutwak L, Dublin TD (1963). Reliability of estimates of serum inorganic iodine and daily fecal and urinary iodine excretion from single casual specimens. J. Clin. Endocrinol. 23:1218-1223.

WHO/UNICEF/ICCIDD (1993). Global prevalence of Iodine Inefficiency disorders. MDIS Working Paper #1), WHO, Geneva.

WHO/UNICEF/ICCIDD (2001). Assessment of Iodine Deficiency Disorders and Monitoring their Elimination. A guide for programme manager (WHO/N/01.1),. 2nd ed, WHO, Geneva.

WHO (2004). Iodine status worldwide. WHO global database on iodine deficiency. WHO, Geneva.