

ORIGINAL ARTICLE**PREVALENCE AND SEVERITY OF IODINE DEFICIENCY DISORDER AMONG CHILDREN 6-12 YEARS OF AGE IN SHEBE SENBO DISTRICT, JIMMA ZONE, SOUTHWEST ETHIOPIA**Yinebeb Mezgebu¹, Andualem Mossie¹, P.N. Rajesh², Getenet Beyene³**ABSTRACT**

BACKGROUND: Iodine deficiency disorder is a major problem worldwide, especially during pregnancy and childhood. The magnitude of the problem is quite big in Ethiopia. The main aim of the present study was to determine the prevalence and severity of iodine deficiency disorders.

METHODS: A cross-sectional survey was conducted in Shebe Senbo District on January 2011. Three elementary schools were selected by lottery method from 20 schools. From each school, students were selected by simple random sampling. Spot urine sample (5 ml) was taken to measure urine iodine level; physical exam was made to palpate goiter and salt samples were collected to estimate iodine content.

RESULTS: Out of 389 participants, 179 (46%) were males. The total goiter rate was 59.1% (Grade 1: 35.2%; Grade 2: 23.9%). The median urinary iodine level was 56 µg/L that indicates iodine deficiency. Out of 389 households in the study area, 277 (71.2%) were using non-iodinated salt, 102 (26.2%) of the households were using iodinated salt. Cabbage usage was significantly associated with goiter.

CONCLUSION: Endemic goiter is quite prevalent in the study area. Median urinary iodine value of the study samples was found to be far lower than standards. Quality of the salt used by the study population was found to be poor in its iodine content. The use of cabbage (goitrogen) has shown remarkable influence on the development of goiter. Therefore, awareness creation and distribution of iodized salt are highly recommended.

KEYWORDS: Iodine deficiency disorders, goiter, median urinary iodine, severity, prevalence

INTRODUCTION

Iodine deficiency disorder (IDD) is a major problem worldwide, especially during pregnancy and childhood (1). The most devastating outcomes of iodine deficiency are high prenatal mortality and mental retardation. Iodine deficiency is the major preventable cause of brain damage in children and needs a universal public health concern (2).

According to a situational analysis carried out by Ministry of Health (MOH) and the United Nations Children's Fund (UNICEF) in 1993, 42

million people (78%) of the total population of Ethiopia are exposed to iodine deficiency, 35 million (62%) are iodine deficient, 14 million (26%) have goiter and at least one in 1000 people is cretin; with about 50,000 prenatal deaths (3). A recent report in 2009 on IDD newsletter revealed that severe iodine deficiency in Ethiopian women leads to 50,000 stillbirths annually and the country's goitre rate has increased from 26% in 1980 40% in 2009 (4).

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Endemic goiter is conveniently referred to iodine deficiency that occurs in more than 10% of the population in a defined area. Iodine deficiency results from either low iodine intake in the diet or ingestion of goitrogens (7). A total goiter rate (TGR) of 5% or more is recommended as the cut-off point to indicate a public health problem as per the decision made by the key international concerned bodies, WHO/UNICEF/ICCIDD (8).

Iodine deficiency causes reduced production of thyroid hormones (T_3/T_4). A decrease in the serum level of T_3/T_4 triggers the secretion of high amount of pituitary thyroid stimulating hormone, which stimulates thyroid glandular activities that results in enlargement of the gland (9). Thyroid hormones play an important role in cellular metabolism, early growth and development of most organs, especially synaptic development and myelination of the brain tissue (10). As a consequence of iodine deficiency at the early age, there will be hypothyroidism that causes low metabolic activities, stunted growth and mental retardation leading to poor school performance, reduced intellectual ability and impaired work capacity (5, 6, 11).

The recommended intake of iodine is 90 μ g/day for preschool children (0 to 59 months); 120 μ g/day for school children; 150 μ g/day for adolescents and adults, and 250 μ g/day for pregnant and lactating women. The thyroid follicles have to collect iodine 60 μ g/day to maintain an adequate pool (12). The excretion of ingested dietary iodine in the feces has been reported to be 30% (13) and is dependent on the type of food taken. About 150 μ g /day is sufficient to prevent clinical manifestation of iodine deficiency disorders for at least several months even if iodine would become absent from the diet (14, 15). Even though inadequate intake of iodine is the principal cause of IDD, goitrogenic food items like cabbage, cassava, millet, soya bean, bamboo shoot, turnip, kale, which interfere with the metabolism of iodine and hormone secretion (16,17).

Supplying properly iodinated salt is considered the most effective long-term public health intervention for achieving optimal iodine. Effective salt iodination is a pre-requisite for the sustainable elimination of iodine deficiency disorders. It has been recommended by WHO/UNICEF/ICCIDD that 90% of the

household should get iodized salt at least 15 ppm for the successful management of IDD in a population (8).

Most iodine absorbed in the body eventually appears in the urine; therefore, urinary iodine concentration is a good marker for very recent dietary iodine intake (18). Median urinary iodine (MUI) value is usually considered as an indicator of iodine nutritional status of a population (20).

The magnitude of iodine deficiency is quite high in pregnant mothers and in school children as evidenced by research articles. Especially in Ethiopia, the problem is so severe perhaps for three possible reasons. First, the iodine content of the soil is decreasing from time to time because of soil bleaching by flood due to high rainfall in certain areas such as Shebe Senbo District. Second, goitrogenic food is cheaply available in the community that may contribute to the development of goiter. Third, the quality of salt distributed to the community is not well inspected if it contains adequate amount of iodine. Therefore, the main aim of the present study is to determine the prevalence and severity of iodine deficiency in school children.

MATERIALS AND METHODS

The study was conducted on school children age 6 to 12 years at Shebe Senbo District from January 1 to 30, 2011. Three out of 20 elementary schools in Shebe Senbo District, Jimma Zone, South West Ethiopia, were selected randomly. Totally there were 2,988 students in the three schools, out of which 389 students of both sexes were interviewed and examined (19) using a single population proportion formula. Each child was subjected to both goiter palpation and urine test for iodine content. These include Shebe, Bekekobo and Chekorsa Elementary Schools. All schools in the district were listed. Primary Sampling Units (3 schools) were selected by lottery method from the list of all schools in the Shebe Senbo District. Proportion of samples was allocated to each of the three schools. From the list of students in each school, individual students were selected by simple random sampling technique.

Structured questionnaire for the interview, formats for physical examination, urinary iodine test and household salt iodine content was developed by the principal investigator.

Structure questionnaire was used to collect information regarding socio demographic variables (like age, sex ethnicity, religion); frequency of cabbage intake; monthly income of the families and type of salt used

IDD manifested as goiter was palpated and graded by physicians using standard procedures as per the criteria of WHO/UNICEF/ICCIDD. According to these criteria, goiter was graded as follows: Grade zero: no palpable or visible goiter; Grade 1: a goiter that is palpable but not visible when the neck is in the normal position i.e., the thyroid is not visibly enlarged and Grade 2: A swelling in the neck, that is visible when the neck is in a normal position (21). Based on severity, goiter is categorized as mild if TGR = 5.0-19.9%, moderate if TGR = 20-29.9% severe if TGR \geq 30% (32).

About 5 ml spot urine samples were collected from every student in a properly labeled and sterile screw-capped plastic vials. These vials were immediately transferred to the thermo cool box containing ice bags and were transported to the biochemistry laboratory, Jimma University. The samples were kept at 4°C in a refrigerator with all precautionary measures until analysis.

Analysis of urinary iodine was done using spectrophotometric procedure. It was based on ammonium persulfate method which was suggested and approved by WHO/UNICEF/ICCIDD (21). This involves the spectrophotometric analysis (measuring the absorbance) of a reaction medium, which utilizes iodine as a catalyst. During this reaction, ceric ammonium sulfate (yellow in color), one of the reactants is reduced to cerrous (colorless) form, which uses iodine as a catalyst. The absorbance value/ optical density (OD) at 405 nm gives a clue about the iodine content of urine sample. It indicates that the more the absorbance value is the lesser the iodine content. There is an inverse relationship between absorbance values and iodine concentration. The absorbance value can be used for the determination of the actual iodine concentration using a standard graph prepared by using a range of standard KIO₃ solutions (21).

Urinary iodine status is a clear cut indicator of iodine nutritional status. WHO/ UNICEF/ ICCIDD have made some guidelines (18) regarding categorizing individuals to different iodine nutritional groups based on urinary iodine level.

Accordingly, < 20 µg iodine/L of urine indicates severe iodine deficiency, 20-49.9 µg iodine/L indicates moderate iodine deficiency, 50-99.9 µg iodine/L indicates mild iodine deficiency, 100-199.9 µg iodine/L indicates adequate or sufficient level, 200-300 µg iodine/L indicates above the requirement and >300 µg iodine/L indicates a excessive level of iodine in the body (20).

Salt samples were collected from each house of the sampled children of the study area and from local market (shops), packed in air tight plastic containers and transported to the laboratory for analysis. The samples were kept in desiccators at room temperature until iodine content was analyzed. Salt samples were grouped as following based on criteria such as processed or unprocessed, powdered or crystal, iodinated or non-iodinated and based on the method of packing. Type I: Ordinary, non- iodinated, unpacked. Type II: Ordinary, non-iodinated, packed in plastic/ polythene sheets. Type III: Processed, iodinated and packed in plastic/ polythene sheets. Type IV: Processed, iodinated and packed in plastic bottle.

Twenty salt samples from each category (I, II, III & IV) were analyzed for iodine content using standard iodometric titration method suggested by WHO/UNICEF/ICCIDD (33). This method involves the titration of a solution of salt against standard Sodium thiosulphate (Na₂S₂O₃ 5H₂O) solution to find out the end point. The volume of Na₂S₂O₃ 5H₂O used up is a direct measure of the content of iodine present in the salt. As the study children were not mature enough to know their family income, they were told to ask their parents about monthly income of the family one day before data collection is completed. Income was categorized base on the assumption of faire and convenient distribution of family income. Parents (Fathers and mothers) were key informants of family income.

Ethical clearance for the study was obtained from Ethical Review Board of Jimma University. Permission was obtained from the parents or guardians of the children and from the directors of schools to undertake the study. Confidentiality was maintained.

Data analysis was done using SPSS version 16 Software. Association between dependent & independent variables were examined. P-value \leq 0.05 was considered significant.

RESULTS

Prevalence and severity of goiter: Among 389 children 179 (46%) were males and 210 (54%) were females. The total goiter rate (TGR) was found to be 59.1% (Grade 1: 35.2%; Grade 2:23.9%) (Table 1).

The total goiter prevalence was more severe 147 (70.0%) among females compared to males 83 (46.4%). Significant difference of goiter prevalence rate between males and females, ($p = 0.00$) was observed.

Association between goiter and income: High goiter prevalence, 66.7% was observed in children whose parent's monthly income was between 500-1000 birr, and the lowest 5 (23.8%) was observed

in children whose parent's monthly income was > 1500 Birr. Statistically significant association was observed ($p = 0.027$) between goiter prevalence rate and monthly income of the parents. Since cabbage is a locally cultivated and cheaply available food item, the daily intake of cabbage was observed to be very high in the study population. Highest prevalence of goiter, 76.7% was observed among children who consumed cabbage every day. Children who had never used cabbage in their diet had the lowest prevalence, 41.7% of goiter. It is shown that significant association existed between the frequency of cabbage intake and development of goiter ($p = 0.05$) (Table 1).

Table 1: Association between severity of goiter and socioeconomic variables of school children in Shebe Senbo District, Jimma Zone, Southwestern Ethiopia, 2011.

Variables	Grade 0 N (%)	Grade 1 N (%)	Grade 2 N (%)	TGR (Grade 1+2) N (%)	X^2	p-value
Sex						
Male (n=179)	96(53.6)	54(30.2)	29(16.2)	83(46.4)		
Female(n=210)	63(30)	83(39.5)	64(30.5)	147(70)	23.4	0.00
Monthly income (Birr)						
< 500 (n=10)	4(40)	4(40)	2(20)	6(60)		
500-1000 (n=90)	30(33.3)	39(43.3)	21(23.3)	60(66.7)		
1001-1500 (n=268)	109(40.7)	91(34)	68(25.4)	159(59.3)	14.281	0.027
>1500 (n=21)	16(76.2)	3(14.3)	2(9.5)	5(23.8)		
Freq. of cabbage intake						
Every day (n=43)	10(23.3)	21(48.8)	12(27.9)	33(76.7)		
3x/ week (n=151)	57(37.7)	58(38.4)	36(23.8)	94(62.3)		
2x/ week (n=137)	58(42.3)	46(33.6)	33(24.1)	79(57.7)	15.17	0.05
1x/ week (n=46)	27(58.7)	9(19.6)	10(21.7)	19(41.7)		
Never (n=12)	7(58.3)	32(5.0)	2(16.7)	5(41.3)		
Total (n= 389)	159(40.9)	137(35.2)	93(23.9)	230(59.1)		

Association between urinary iodine level and sex: Among a total of 389 urine samples tested, 148 (82.7%) male students and 177 (84.3%) female students were found to have iodine level below 100 µg/L. The median urinary iodine (MUI) value was 56 µg/L for both genders while 74 µg/L and 54 µg/L in males and females, respectively (Table 2).

Logistic regression was used to determine which background variables were independently related to the development of goiter. This model showed that females were more likely to be vulnerable to develop goiter than males [$OR=2.384$, 95.0% CI (1.372, 4.144)]. Children with median urinary iodine (MUI) value < 56 µg/L were more likely to develop goiter than those who had MUI \geq 56 µg/L. [$OR=0.044$, 95% CI (0.025,0.081)] (Table 3).

Table 2: Association between urinary iodine level and sex among school children in Shebe Senbo District, Jimma Zone, South West Ethiopia, 2011.

	Urinary Iodine ($\mu\text{g/L}$)*							Poor Iodine Status N (%)
	MUI ($\mu\text{g/L}$)	< 20 N (%)	20-49.9 N (%)	50-99.9 N (%)	100-199.9 N (%)	200-299.9 N (%)	>300 [‡] N (%)	
Sex								
Male	74	5(2.8)	59(33)	84(54.9)	25(19.7)	1(0.6)	5(2.8)	148 (82.7)
Female	54	7(3.3)	91(43.3)	79(37.6)	26(12.4)	5(2.4)	2 (1)	177 (84.3)
Total	56	12(3.1)	150(38.6)	163(41.9)	51(13.1)	6(1.5)	7(1.8)	325 (83.5)

* Urinary iodine < 20 $\mu\text{g/L}$ = severe, 20-49.99 $\mu\text{g/L}$ = moderate, 50-99.99 $\mu\text{g/L}$ = mild, 100-199.99 $\mu\text{g/L}$ = adequate, 200-299.99 $\mu\text{g/L}$ = above requirement, >300 $\mu\text{g/L}$ = excessive

[‡]Risk of adverse health consequences (iodine-induced hyperthyroidism, autoimmune thyroid diseases)

Table 3: Multiple logistic regression analysis predicting the likelihood of having goiter by socio demographic variables and MUI among school children in Shebe Senbo District, Jimma Zone, South West Ethiopia, 2011.

Variables	Presence of Goiter		AOR	95% CI
	Normal No (%)	Goitrous No (%)		
School				
Shebe (n=92)	41 (44.6)	51 (55.4)	1.00	
Chekorsa (n=194)	56 (28.9)	138 (71.1)	0.291	(0.125, 6.770)
Beke kobo (n=103)	62 (60.2)	41 (39.8)	0.982	(0.436, 2.211)
Sex				
Male (n=179)	96 (53.6)	83(46.4)	1.00	
Female (n=210)	63 (30.0)	147 (70.0)	2.384*	(1.372, 4.144)
Age (10 years is mean age)				
< 10 (n = 101)	49 (48.5)	52 (51.5)	1.00	
\geq 10 (n = 288)	110 (38.2)	178 (61.8)	0.142	(0.853, 3.028)
Freq. of cabbage intake				
Every day (n= 43)	10 (23.3)	33 (76.7)	4.620*	(1.200, 17.789)
3x/ week (n = 151)	57 (37.7)	94 (62.3)	2.309	(0.700, 7.619)
2x/ week (n = 137)	58 (42.3)	79 (57.7)	1.907	(0.576, 6.310)
1x/ week (n= 46)	27 (58.7)	19 (41.3)	0.985	(0.271, 3.575)
Never (n = 12)	7 (58.3)	5 (41.7)	1.00	
Monthly income (Birr)				
< 500(n=10)	4 (40.0)	6 (60.0)	3.436	(0.401, 29.474)
500-1000 (n=90)	30 (33.3)	60 (66.7)	2.799	(0.650, 129.053)
1001-1500 (n=268)	109(40.7)	159 (59.3)	1.848	(0.457, 7.475)
>1500(n=21)	16 (76.2)	5 (23.8)	1.00	
MUI (56 $\mu\text{g/L}$)				
< 56 (n=196)	20 (10.2)	176 (89.8)	1.00	
\geq 56 (n=193)	139 (72.0)	54 (28.0)	0.044*	(0.025, 0.081)

Iodine content of salt samples: In the study area large majority of the households were using only non-iodinated salt 277 (71.2%), a less number 102 (26.2%) of the households were using iodinated

salt and a very less percent 10 (2.6%) of the households were using both iodinated and non-iodinated salt, which is a mixed type (Table 4).

Table 4: Grouping of salt samples collected from children households and from local markets, Shebe Senbo District, Jimma Zone, South West Ethiopia, 2011.

General type of salt*	Households Count N (%)	Detail type of salt [‡]	Households Count N (%)
Non-iodinated salt	277(71.2)	Type I	186(67.15)
		Type II	91(32.85)
Iodinated salt	102(26.2)	Type III	71(69.61)
		Type IV	31(30.39)
Mixed	10(2.6)	Iodinated + non-iodinated	10(2.6)
Total	389(100)	-	389(100)

* General typing of salt before any analysis, [‡]Classification of salt type after iodine analysis (33).

The result revealed that ordinary salt, which are type I and type II, both non-iodinated, were of poor quality, i.e. iodine level < 15ppm. From iodinated salt samples, 15 (75.0%) were of good

quality (iodine level >15 ppm) and 5 (25.0%) were found to be inadequate in their iodine content (Table 5).

Table 5: Iodine content of salt samples collected from household and local market in Shebe Senbo District, Jimma Zone, South West Ethiopia, 2011.

Type of salt sample	MSI content (ppm)	Salt iodine content (ppm)			Salt samples with Inadequate iodine N (%)
		<15ppm N (%)	15-29ppm N (%)	>30ppm N (%)	
Type I (n=20)	10.46	20(100)	Nil	Nil	20(100)
Type II (n=20)	9.78	20(100)	Nil	Nil	20(100)
Type III (n=20)	45	5(25)	Nil	15(75)	5(25)
Type IV (n=20)	58	Nil	Nil	20(100)	Nil
Total	80	45 (56.6)		35 (43.4)	45(56.6)

MSI = Median salt iodine

DISCUSSION

In the studied area, the total goiter prevalence was found to be high (59.1%), indicating that IDD is a severe public health problem. The present finding is in line with the study reported by Abuye et al., in 2007 (22) in SNNPR, as a prevalence rate of 56.2%. A total goiter rate of 64.1% was also reported by Asseffa et al, 1996 (23) from another

location of Shebe Senbo District. The prevalence of goiter in this study is far higher than the one that has been reported earlier by Abuye et al in 2007 (22) from the entire Oromia region, as TGR is 42%. The possible reason for higher prevalence of iodine deficiency goiter in the present study could be due to heavy rainfall in Shebe Senbo District nine months a year that erodes the soil iodine content.

A cross-sectional community based goiter prevalence survey in Neksege Sub District in Tigray revealed very high goiter prevalence of 71.4% (25) compared to the magnitude of the problem reported in the present study in Shebe Senbo District. The high prevalence in Tigray region is due to high rainfall in the mountainous areas as a result soil iodine is bleached by running flood (25).

According to WHO/UNICEF/ICCIDD a TGR of > 5% can be taken as a public health problem in a population. Therefore, a TGR of 59.1% in the study population has to be considered as a severe public health problem. In the present study, iodine deficiency was found to be more prevalent in females compared to males. This is in agreement with studies conducted by Kidane et al. 2006 in Tigray region (25). Abuye et al in 2007 (22), reported that goiter is commoner in females than males as it is conducted in Oromia region. Negalign et al in 2004 reported that the TGR was higher in females than males in Kefa Zone, South West Ethiopia (24). Amar K Chandra in 2008 (26) reported that in Tarai region of India females showed a higher TGR (34.4%) as compared to males (26.9%). The possible explanation is that females are the more vulnerable category because of physiological reasons such as early puberty, which starts about 2 years earlier than males. As a result their iodine demand is higher than males. In addition, estrogen, which is a female hormone, has a well-known inhibitory effect on iodine uptake by thyroid follicular cells. Beside, estrogen also increases thyroid follicular proliferation, leading to thyroid gland enlargement (27).

A correlation was found to be existed between the income of the household and goiter prevalence. Children who belong to the parents earning monthly income > 1500 birr have the least TGR (23%) as compared to those who earn <1000 birr. The higher TGR in low income category could be because of poor food security, including less meat and vegetable consumption, use of non-iodized salt, lack of medical attention. Related study results reported that the prevalence of goiter is common in economically low families. This suggests that living standard has direct relationship with iodine nutritional status and thereby with prevalence of IDD in a population (22).

A direct correlation was also seen between the frequency of cabbage intake and goiter prevalence. Children who take cabbage on regular bases were exposed to goiter more than those who have never taken cabbage. The present finding is supported by the experimental work of Chesney et al 1928. (28) reporting that those rabbits that were fed cabbage regularly developed goiter. Additional evidence was reported by Abuye et al 2008 (29). As per this study in Oromia region, which includes Shebe Senbo District, the mothers who take cassava regularly showed higher prevalence of goiter compared to non-users. Cabbage contains thiocyanate and isothiocyanate that inhibit iodine uptake by the thyroid follicular cells and also blocks the thyroid peroxidase enzyme (29, 34). In the presence of goitrogens, iodination of thyroglobulin protein will be impaired, resulting in poor thyroxine production and enlargement of the thyroid gland.

The urinary iodine level is used as a valuable indicator for measuring the iodine nutritional status and for the assessment of IDD in an individual, because 90% of body iodine is excreted through urine (30). Among the participants, majority (83.5%) had urinary iodine level below 100 $\mu\text{g/L}$ and this suggests the presence of iodine deficiency. Median urinary iodine (MUI) level is also important indicator of iodine deficiency of the entire population. According to the current finding, the MUI was found to be 56 $\mu\text{g/L}$, which is far below 100 $\mu\text{g/L}$. This is in agreement with earlier nationwide reports (22), which have shown that MUI 24.5 $\mu\text{g/L}$. This indicates that the study area is highly affected by iodine deficiency. Abuye et al 2007 reported a median urinary iodine level of 25 $\mu\text{g/L}$ in Oromia region, suggesting a very severe case of iodine deficiency in the population (22).

Generally salt is one of the easily available sources of iodine for a population. But the iodine content of salt samples may vary widely depending on the source, the storage place, the method of packaging and the methods used for processing (in the case of commercial /branded items).

As per the result of salt samples collected from children's household and local market, 71.0% of the households were using non-iodized salt containing iodine level less than 15 ppm. This could be one of the main reasons for the high

prevalence of goiter in the population. It has been recommended by WHO that 90% of the households in a population should be able to get iodized salt containing iodine level >15 ppm for the effective elimination of IDD. The result of present study is comparable with earlier study reports (31) indicating that 81% of household salt samples of the study area had iodine level below the minimum standard set by the Quality and Standard Authority of Ethiopia (QSAE). Among branded iodized salts, 25% of them were having iodine level less than ordinary non-iodized salt. This may be because of the loss of iodine during the time of processing, washing, and storage. According to WHO/UNICEF/ICCIDD the iodine level must be above 15 ppm for non-iodinated salt and above 30 ppm for iodinated salt at the consumer level for the effective management of IDDs in a population (8).

Thus, as a serious concern, quality checking mechanism should be developed before approving a brand as iodized salt. Using regression analysis, among the expected predictors; sex, frequency of cabbage intake and median urinary iodine level were found to be risk factors for the development of goiter.

To conclude, high prevalence of goiter among school children in the study area has been identified, which is a severe public health problem. The problem was found to be more prevalent among females, low family income and high frequency of cabbage intake.

Therefore, awareness creation via health education and inspection of the distribution of iodized salt are highly recommended.

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REFERENCES

1. Dunn JT. Endemic goiter and cretinism: an update on iodine status. *Journal of Pediatric Endocrinology*, 2001; 14 S6:1469-1473.
2. De BB, Andersson M, Takkouche B, Egli I. Prevalence of iodine deficiency worldwide. *Lancet*, 2003; 362: 1859-1860.
3. Ministry of health/United Nations Children's Fund. Nutrition policy: The miracle of iodated salt, Ethiopia's commitment to salt iodation. Joint Report on Situation Analysis, Addis Ababa, Family Health Department, MOH. 1995.
4. IDD Newsletter. International Council for Control of Iodine Deficiency Disorders. 2009; 33(3): 13.
5. Andersson M, Takkouche B, Egli I, Allen HE, de BB. Current global iodine status and progress over the last decade towards the elimination of iodine deficiency. *Bulletin of WHO*, 2005; 83(7): 518-525.
6. Cavature A, Delarosa J, Rally AA, Disarray JN, Bellies G, Roux F, et al. Endemic goiter: a research protocol elaboration for eradication. *Collegium Antropologicum*, 1998; 22(1): 1-8.
7. Delange F. Iodine deficiency in Europe and its consequences: an update. *European Journal of Nuclear Medicine & Molecular Imaging*, 2002; 29(S2):S404-S416.
8. Azizi F, Duffiel A, Bürgi H, Dunn J, Hetzel B, Delange F. et al. Assessment of iodine deficiency disorders and monitoring their elimination. A guide for programme managers, 2nd ed. Geneva, World Health Organization, 2001 (WHO/NHD/01.1).
9. Hetzel BS, Maberly GF. Iodine. In: Mertz W. Trace Elements in Human and Animal Nutrition. 5th Ed. Academic Press, New York. 1986; 2: 139-208.
10. Taurog A. Hormone synthesis and thyroid iodine metabolism. in: Braverman LE, Utiger RD, editors. The Thyroid: A Fundamental and Clinical Text. Philadelphia, PA: JB Lippincott Publishing, 1991; 51-97.
11. Davis PJ. Cellular action of thyroid hormones. in: Braverman LE, Utiger RD, editors. The Thyroid: A Fundamental and Clinical Text. Philadelphia, PA: JB Lippincott Publishing, 1991; 190-203.
12. Fisher DA. Thyroid hormone effects on growth and development, in: Delange, Fisher, Malvaux, Pediatrics Thyroidology, Basel S. Karger Publishing, 1985; 85-89.
13. Follis RH. Patterns of urinary iodine excretion in goitrous and non-goitrous area, *Anal. J. Clin.Nutr.* 1964; 14: 253-268.
14. Dunn JT, Crutchfield H.E, Gutekunst R, Dunn AD. Two simple methods for measuring iodine in urine, *Thyroid*, 1993; 3: 119-123.
15. Stanbury JB. Iodine, in: Shils ME, Young VR, Modern Nutrition in Health and Disease, Lea and Febiger Publishing, Philadelphia, PA, 1998; 252-262.

16. Wolde-Gebriel Z, Demeke T, West CE, Haar FVD. Goiter in Ethiopia. In: Wolde-Gebriel Z. Micronutrient deficiencies in Ethiopia and their inter relationships. Wageningen, Grafisch Service Centrum Wageningen, Netherlands. LUW 1992; 41-56.
17. Bekele A, Wolde-Gebriel Z, Kloos H. Food, diet and nutrition. In: Zein AZ, Kloos H. The ecology of health and disease in Ethiopia. West view Press, Boulder. Sanfrancisco. Oxford, 1993; 85-102.
18. Hetzel BS. Iodine deficiency disorders (IDD) and their eradication. *Lancet*, 1983; 2:1126-1129.
19. CSA. Population and housing census of Ethiopia. Result for Oromia Region, July 2010 (Projected from May & November 2007 Ethiopian Census).
20. De Maeyer EM, Lowenstein FW, Thilly CH. The control of endemic goiter. Geneva, World Health Organization, 1979.
21. ICCIDD/WHO/UNICEF. A Practical guide to the correction of IDD. In: Dunn JT, Vander Harr F. Netherlands: *International Council for Control for Iodine Deficiency Disorders*; 1990.
22. Abuye C, Berhane Y, Akalu G, Getahun Z, Ersumo T. Prevalence of goiter in children 6 to 12 years of age in Ethiopia. *Food and Nutrition Bulletin* 2007; 28(4): 391.
23. Assefa T, Argaw H. Prevalence and prominent factors for iodine deficiency disorders in Shebe area Seka Chekorsa district, south Western Ethiopia. *Bull.inst.health sci.* 1997; 7(1):63
24. Berhanu N, Wolde Michael K, Bezabih M. Endemic goiter in School Children in Southwestern Ethiopia. *Ethiop .J. Health Dev.* 2004; 18(3):175-178.
25. Kidane T, Woldegebriel A. Prevalence of Iodine deficiency disorder in a highland district in Tigray. *Ethiop. J. Health Dev.* 2006; 20(1):58-59.
26. Chandra AK, Bhattacharjee A, Malik T, Ghosh S. Goiter Prevalence and Iodine Nutritional status of Shool Children in a Sub Himalayan Tarai Region of Estern Uttra Pradesh. *Indian Pediatrics*, 2008; 45(28): 469-474.
27. Furlanetto TW, Nunes Jr RB, Sopelsa AMI, and Maciel RMB. Estradiol decreases iodide uptake by rat thyroid follicular FRTL-5 cells. *Braz J Med Biol Res*, 2001; 34(2): 259-263.
28. WHO/UNICEF/ICCIDD. Indicators for Assessing Iodine Deficiency Disorders and their Control through salt Iodization WHO/NUT94.6 WHO: Geneva; 1994.
29. Abuye C, Berhane Y, and Ersumo T. The role of changing diet and altitude on goitre prevalence in five regional states in Ethiopia. *East African Journal of Public Health*, 2008; 5(3):164.
30. World Health Organization/International Council for the Control of the Iodine Deficiency Disorders/United Nations Children Fund (WHO/ICCIDD/UNICEF). Assessment of the iodine deficiency disorders and monitoring their elimination. Geneva: World Health Organization, 2007.
31. Takele L, Belachew T, Bekele T. Iodine concentration in salt at household and retail shop levels in Shebe town, south west Ethiopia. *East African medical journal*, 2000; 80(10): 532.
32. Delange F. Determining median urinary iodine concentration that indicates adequate iodine intake at population level. *Bulletin of the World Health Organization* 2002; 80(8): 633–636.
33. Titration methods for salt iodine analysis. In: Sullivan KM, Houston E, Gorestein J, Cervinskias J, eds. Monitoring Universal Salt Iodization program. Atlanta, Georgia, USA: UNICEF/ ICCIDD/PAMM/WHO 1995; 11.
34. Amar KC, Sanjukta M, Dishari L & Smritiratan T. Goitrogenic content of Indian cyanogenic plant foods & their in vitro anti-thyroidal activity. *Indian J, Med Res.* 2004; 119: 180-185.