

## Original article

# Urinary iodine excretion in relation to goiter prevalence in households of goiter endemic and nonendemic regions of Ethiopia

**Cherinet Abuye<sup>1</sup>, Bantirgu Haile Mariarn, Hanna Neka Tibeb, Kelbessa Urga and Zewdie Wolde-Gebriel**

**Abstract:** A base line survey of goitre prevalence, among population of five endemic and four nonendemic regions of Ethiopia was carried out prior to the distribution of iodated salt. Urine samples were collected from 327 subjects selected by systematic random sampling from endemic and 276 subjects in sites taken as nonendemic. The lowest mean urinary iodine excretion (UIE) value was recorded in Bure (22 µg/day) and the highest in Alemmaya (148 µg/day). The highest total goitre rate (% TGR) was recorded in Sawla (55.6%) and the lowest (0.6%) in Yabello. Iodine content of drinking water was in the range 0.04- 48.5 µg/l. Iodine content of water source was correlated positively ( $r = 0.8399$ ) with the mean UIE in all study sites. The relationship between UIE and TGR, however, indicates that sites considered as nonendemic seem to be affected by iodine deficiency. The present study results urge the need for intervention in controlling Iodine deficiency disorders (IDD). [Ethiop. J. Health Dev.1995;9(1):111-116]

## Introduction

Iodine is an essential trace element required for human growth and development. It is mainly obtained by way of food and water consumed. Determination of its level in ready-to-eat food provides a reliable estimate of the amount of iodine ingested (I). Iodine intake can also be evaluated by indirect indices such as estimation of the thyroid uptake of the radioiodine, measuring thyroid hormones using radioimmunoassay and determination of the urinary iodine (2,3,4). Of all these methods urinary iodine measurement has the advantage of applicability in field conditions. Estimation of daily urinary iodine concentration in a sufficient number of casual samples as urinary-iodine to creatinine ratio or microgram iodine per day (µg/day) had been a good approach and remains the most convenient index of iodine intake.

Investigators in a large number of studies of iodine deficiency disorders (IDD) have used urinary iodine to evaluate dietary intake of iodine in communities (5,6). Studies in Thailand (7) have found that urinary iodine

distribution patterns correlate well with the regional prevalence of goiter. In order to ensure safe plasma inorganic iodine level of 0.1 µg/dL, an average intake of 70 µg is required. This would allow for 50 µg of urinary iodine excretion which is considered as the minimum quantity of adequate thyroid hormone synthesis (8).

Several reports (7,9,10) indicate that moderate IDD is generally associated with urinary iodine ranging from 25 to 50 µg per day while, severe IDD occurs when daily excretion falls below 25 µg iodine per day.

Therefore, urinary iodine assessment, helps in the evaluation of available nutritional iodine in a given community .Iodine content in water is also an indication of the level of iodine consumed with locally produced food. However, drinking water provides ten percent of the average daily body requirement (11). Observation of its level in water could give supportive evidence to the consumption level of iodine and may reflect the urinary iodine excretion in a given locality. This study was part of a baseline survey of IDD, carried out before implementing iodine intervention programme to identify the existing situation of goitre. It was substantiated with determination of VIE which could be used as a reference in evaluating the impact after distribution of iodated salt.

---

<sup>1</sup>From Ethiopian Nutrition Institute P.O.Box 5664  
Addis Ababa

## **Methods**

**Study Sites;** The country-wide goitre prevalence survey of 1981 (12) was used to select five goitre endemic regions (prevalence greater than 20% ) and four nonendemic regions (prevalence less than 5% ). The endemic regions and their corresponding sampling sites were Shoa (Majetie), GamuGofa (Sawla), Shoa (Gohatsion), Bale (Adaba) and Gojam (Bure) while sites taken as nonendemic regions were Harerghie (Alemmaya), Shoa (Mojo), Sidamo (Yabello), and Arsi (Huruta). The study was conducted from 1988 to 1991. **Sampling procedure;** In all the survey sites, data on household members were obtained from Kebele (lowest administrative units) and then properly labeled call cards were distributed to households selected systematically for goitre examination and obtaining information like age, sex, and drinking water source. Sub-samples were selected systematically matching to age and sex, from sample populations with predetermined sample size of 50 to 100 for collection of urine samples. A total number of 5399 subjects from areas taken as goiters endemic and 60 10 from sites taken as non- endemic were included in goitre examination. Thyroid size estimation was accomplished according to the criterion established by WHO /PAHO (13). Urine samples were collected in duplicate from subjects in all study sites. Creatinine was determined in the urine samples prior to determining the urinary iodine (14). Creatinine value was used as a basis for computing urinary iodine as iodine / creatinine ratio in casual urine samples and expressed in microgram iodine per day (p.gl/day) (15). Analysis of urinary iodine was carried out by the method of Sandell and Kelthoff, as described by Barker (15). Water samples were collected from drinking water sources in polyethylene bottles washed with distilled water and then rinsed with deionized water except from Sidamo (Yabello).

Iodine content in water samples was determined according to the method of Rogina and Dubravcic (16). Classification of urinary iodine excretion ranges were based on the method of Follis (6) which indicates iodine consumption level and IDD endemicity of a group or community.

*Statistical analysis:* The data were tested for skewedness and transformed to square root. The level of correlation was performed on a SPSS/PC soft-ware package.

## **Results**

Table I shows total goitre rate, urinary iodine excretion and the level of iodine in water. As indicated, Alemmaya (Harerghie) and Yabello (Sidamo) have UIE rates of greater than 100 p.gl/day whereas Mojo (Shoa) and Huruta (Arsi) have UIE between 50 and 00 p.gl/day. Other study sites have UIE values of less than 50 p.gl/day. TGR higher



Table 1: Mean urinary iodine in relation to total goitre rate and water iodine by study sites

No. Site	% TGR Community	Mean + Se(Range) UIE (mcgI/day)	Water iodine (mcgI/L)
1. Harerghie/Alemmaya	15.70 (1465)	148.00±16.71 (59)	48.50
2. Shoa/Mojo	9.60 (1219)	83.50±7.90 (55)	48.00
3. Sidamo/Yabello	0.60 (1667)	104.00±7.66 (90)	-
4. Arsi/Huruta	24.60 (16559)	54.50±3.27 (72)	5.80
5. Shoa/Majetie	32.40 (840)	30.40±2.62 (67)	3.00
6. Gamogofa/Sawla	55.60 (978)	25.30±2.11 (97)	3.40
7. Shoa/Gohatsion	9.20 (870)	24.90±2.65 (57)	15.30
8. Bale/Adaba	27.60 (1138)	34.20±3.57 (50)	0.80
9. Bojam/Bure	21.30 (1573)	22.00±2.37 (56)	0.40

SE = Standard error Number in parentheses are number of samples taken (r = 0.839 for UIE and water iodine), (4 = -0.5148 for UIE and TGR)

than 20% was noted in 80% of the study subjects with VIE less than 50 JLgI/day. Consequently, a mean VIE rate of 58.50 ±14.75 JLgI/day was observed against total mean goitre rate of 21.80 ±.5.40 was noted for both endemic and nonendemic sites.

A positive and highly significant (p<0.05) correlation (r=0.8399) was also observed between UIE and iodine concentration of water sources. Mean urinary iodine excretion values in Bure (Oojam) and Oohatsion (Shoa) were below 25 JLgI/day whereas concentrations of iodine in water in Bure (Oojam) and Oohatsion (Shoa) were 0.4 JLg/1 and 15 JLg/1, respectively. Table 1, shows UIE range, total goitre rate (% TOR) and absolute number of people having goitre out of the biochemical groups, by sex.

In females, the highest value, 50.9% TOR, was recorded within the range of 0.01-24.99  $\mu\text{gI/day}$ . This value was about 33% for male subjects with similar range of UIE rate. For all ranges of UIE, % TOR was greater in females than in males. For all UIE ranges, %TOR was higher in sites taken as goitre endemic than those sites taken as nonendemic (Table 3). However, % TOR in sites taken as nonendemic was found to be greater than 20.0%.

### Discussion

This is most probably the first study on VIE to be done in relation to endemicity of goitre in Ethiopia. It was reported by different investigators that no region in Ethiopia under investigation was free from iodine deficiency despite an indication of wide variation in the magnitude of endemicity (12,17). Our study which covers a total of nine sites ascertained the existence of goitre supported by biochemical findings. Both endemic and nonendemic sites of the present study selected from the national survey report of 1981 (16) were found to have high prevalence of goitre than previously reported (16).

The nonendemic sites were also found to be endemic. This indicates that most parts of Ethiopia, including sites regarded as nonendemic (16) were exposed to iodine deficiency problems through time. Of the total of nine study sites, mean VIE in Bure (Gojam), Shoa (Gohatsion) and Sawla (Gamogofa) appears to be quite low (Table 1). The low rate of VIE combined with high TGR is a good indication that these areas are highly affected by iodine deficiency problems. Similarly, Majetie (Shoa) and Adaba (Bale) have also low VIE and high TGR that they

Table 2: Urinary iodine excretion by range and Total Goitre Rate by Sex

Urinary iodine range ( $\mu\text{gI/day}$ )	Male		Female	
	N	% TGR	N	% TGR
0.01-24.99	95	32.60	173	50.90
25.0-49.99	67	32.80	64	37.50
50.0-99.99	60	13.30	55	36.40
100+	59	25.40	30	40.00
Total	281		322	

N = Biochemical groups

too are categorized under the area of high IDD endemicity .UIE thus have a negative correlation ( $r=-0.5148$ ) with total goitre rate in tile study sites. The TGR in Mojo (Shoa) is relatively high although with a high UIE value.

The water iodine value for Mojo was also found to be among the highest and this gives rise to other speculations which again requires further investigation. Mojo lies within the Rift Valley area where high fluoride intake is observed (18). The mechanism of interference of high intake of fluoride has a possible influence upon the amino acid precursors of thyroxin, tyrosine and its metabolites rather than upon iodine (19) .The study results also indicate that UIE did not linearly increase with the decrease in TGR, which might be due to the presence of some goitrogenic factors and / or water contaminats (3,20) which are known to contribute to the lack of absolute correlation between iodine deficiency and goitre size.

Microorganisms like Escherichia coli and other dietary goitrogenic factors like thiocyanate impose their adverse effects on bioformation of thyroid hormones in the thyroid gland by inhibiting iodine uptake and organification and increasing iodine excretion through the urine. Our study results also indicated that with increasing ranges of UIE, TGR decreased in both male and female study subjects. %TGR dramatically decreased in male than in female for UIE ranges of 25.99-49.99 to 50.00-99.99 JLgI/day which could perhaps be explained by ,exceptional exposure of females for high :physiological demand of iodine that could laccelerate its shortage in the body (21,22).

There is a concomitant decrease of TGR for both sexes until UIE rate reached a level of 99.9 p.mg/day urinary iodine (Table 2).

Beyond this range, the TGR shifted forward with increasing tendency. There is also unexpected increment of TGR with increasing UIE (Above 49.99 (p.mg/day) in regions considered as nonendemic. This might be due to seasonal migration of people for food search and trade from goitre endemic areas, to less endemic regions which may have improved UIE while without significant change in goitre size. Once goitre has developed as a result of poor iodine intake the thyroid enlargement may not disappear when the individual is taken to iodine sufficient environment (23). Our study results also indicate that TGR in females is higher than in males for the same range of UIE. This can be attributed to the higher physiological requirement of iodine and sex- based low thyroxine binding. Pre-albumin (PA) biosynthesis in females than in males would aggravate TOR when dietary intake is insufficient or marginal (22,24). The relationship of UIE to total goitre rate shown both in endemic and in sites taken as nonendemic is also a good indicator of the severe iodine dificiency situation in study sites. There is also an indication of IDD agravating factors besides iodine deficiency. It was reported (6) that the effect of goitrogenic factors can be overcome by iodine intervention.

Hence, the findings presented here urge for implementing, control and eradication programme of IDD in the affected areas.

**Table 3 urinary Iodine excretion by range and Total Goitre Rate by Endemicity**

	endemic	Non endemic
--	---------	-------------

Urinary Iodine range (µg/l/day)	N	% TGR	N	% TGR
0.01-24.99	197	47.20	84	26.2
25.00-49.99	83	51.80	40	20.0
50.00-99.99	40	45.00	55	21.8
100+	7	28.60	97	22.7
Total	327		276	

N = biochemical groups

### Acknowledgement

The financial support of UNICEF for goiter survey and IDD control programme in Ethiopia is gratefully acknowledged.

### Reference

1. Moxon RED and Dixon PJ .Semi-automatic method for determination of total iodine in food. Analyst 1980; 105:344.
2. Jalin T and Escobar FD. Evaluation of Iodine Creatinine ratios of casual Samples as indices of daily urinary iodine output during field studies. J:Clin.Endocrinol.metab. 1965;25:540.
3. Jose R. Varea Teran Nutritional and public health considerations relating to endemic goitre and cretinism. In:F.Delange and R. Ahluwalia,(eds). Cassava toxicity and thyroid research and public health issues proceedings of a workshop held in Ottawa, 31 May- 2 June 1982; p. 55.
4. Dourdoux P, Thilly C, Denlange F, and Ermans AM. A new look at old concepts in laboratory evaluation of endemic goitre. In: John T. Dunn,Eduardo A.pretell,Carlos Hernan Daza,Fernando E. Viteri.(eds). Towards eradication of endemic goitre,cretinism and iodine deficiency PAHO. WHO Scientific publication 1986;502: 115.
5. Jerome M. Hershman,Glenn A. Melnick,and Rosemary Kastner .Economic Consequences of endemic goitre In: John T. Dunn, Eduardo A. Pretell, Carlos Hernan Daza,Fernando E. Viteri,(eds). Towards eradication of endemic goitre, cretinism and iodine deficiency. PAHO. WHO Scientific Publication 1986;502:36.
6. Hennart Dourdoux P, Vis HF, Yunga Y, Seghers P and Delange. Epidemiology of goitre and malnutrition and dietary supply of iodine, thiocyanate and protein in Daszaire, Kivu and Yubang In:Delange FB, Itezkand A.M. Ermans,(eds). Nutritional factors involved in goitrogenic action of Cassava. International development research center publ. Ottawa Canada,IDRC -184e 1982 p 25.
7. Richard H, Follis JR. Patterns of Urinary Iodine Excretion in goitrous and non-goitrous areas. The American I. Clin.Nut. 1964;14:253.

8. Wayne PJ, Kentros DA and Alexander WD. Clinical Aspect of Iodine Metabolism, Blackwell, Oxford, UK. 1964; p 303.
9. De Visscher MC, Deckers HG, Von Den Schrieck, M. De Smet, Ermans AM, Galperin H, and Dastenie PA. Endemic goitre in Uele Region (Republic of Congo) *J. Clin. Endocrinol Metab.* 1961;21:175.
10. Gill MC, Taylor Pe and Suppoery A. new focus of endemic goitre in Mwezi, Tanzania East Africa *Medical Journal.* 1970;47:66.
11. Koutras DA. Iodine: Distribution, availability, and effect of deficiency on thyroid. In: John T. Dunn, Eduardo A. Pretell, Carlos Hernan Daza, Fernando E. Viteri, (eds). Towards eradication of endemic goitre, cretinism and iodine deficiency P AHO Scientific publication 1986;502: 15.
12. Wolde-Gebriel Z, Demeke T, Clive E. West and Frits Van der Haar. Goitre in Ethiopia. *British Journal of Nutrition* (in press).
13. Hetzel BS. The prevention and control of iodine deficiency disorders. Nutrition policy discussion paper 1988;3:89.
14. Roscoe MH. Creatinine in serum or urine. *J. Clin. Pathol.* 1958;11:173. ,
15. Barker SB et al. The clinical determination of protein bound iodine *J. Clin Invest* 1951; 50:30  
Lynch  
MU. et al. Estimation of protein bound Iodine. *Medical laboratory technology* W.B, Sounds Co. Philadelphia 1969; p 542.
16. Rogina B and Dubravcic: Microdetermination of iodine by arresting the catalytic reduction of ceric ions *Analyst* 1953;78:594.
17. Inter-departmental Committee on Nutrition for National Defence (ICNND). Ethiopian Nutrition Survey Washington DC: US Government printing Office 1959.
18. Teklehaimanot R, Fekadu A, Bushra B. Endemic Fluorosis in the Rift Valley Ethiopia. *Trop. Geogr. Med.* 1987;39:209.
19. Day TK and Powell-Jackson PR: Fluoride Water Hardness and Endemic Goitre. *the Lancet* 1972;1:1135.
20. Bourdoux p, Mafuta M, Hanson A and Ermans AM. Cassava toxicity the role of linamarin in: A.M. Ermans, N.W Mbulamoko, F. Delange, endemic goiter and cretinism. IDRC- 136e 1980; p 15.
21. Delange F, Thilly CH and Ermans AM. Endemic Goitre in kivu area Africa: FOCUS on cassava In: A.M. Ermans, N.M. Mbulamako, F. Delange, and Ahluwalia, (eds). Role of cassava in the etiology of endemic goitre and cretinism. IDRC-136e 1980; I' 29.
22. Ingenbleek Y and Visscher DM. Hormonal and nutritional status: Critical conditions for endemic goitre epidemiology? *Metabolism* 1979;28:9.
23. Thilly CH, Delange F, Ermans AM: Five years follow-up in the treatment of endemic goitre with iodized oil. *Acta Endocrinol (Suppl) (kbh)* 1973;74:11.
24. Braveman LE, Foster AE, Ingbar SH: Sex related differences in the binding in serum of thyroid hormones. *J. Clin. Endocrinol Metab.* 1967;27:227.