

Intake and excretion of iodine and thiocyanate among school children in goiter endemic sub-Himalayan tarai region.

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Abstract

The present study was undertaken to assess the state of iodine nutrition of the representative population, consumption pattern of cyanogenic plant food that generally interfere with iodine nutrition, and to evaluate whether there is any interrelation between iodine and thiocyanate intake and their excretion pattern among school children in goiter endemic sub-Himalayan tarai region. A total of 560 urine samples from school children, aged 6-12 years of both sexes were collected and analyzed for iodine (I) and thiocyanate (SCN) concentration. Median urinary iodine level was 76.2 µg/l and 32.3% had concentration <50µg/l indicating biochemical iodine deficiency. Mean urinary thiocyanate level was 0.762±0.45 mg/dl indicating relatively higher consumption of cyanogenic plant foods by the population. A significant positive correlation ($r=0.13$, $P<0.01$) between urinary iodine and urinary thiocyanate concentration suggests that there is an interrelationship between mean urinary iodine and thiocyanate excretion pattern of the population in the study region. Body's overall thiocyanate concentration perhaps plays an important role to maintain the overall iodine concentration in the body. Therefore the urinary iodine may not always truly reflect the iodine nutrition status of the body.

Keywords: Urinary iodine, Urinary thiocyanate, Sub-Himalayan tarai region, School children.

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Introduction

The relation of iodine deficiency to endemic goiter is well established and the relation between elevated urinary thiocyanate and goiter prevalence has also been evidenced [1]. In most of our earlier studies in India, findings of high total goiter rate (TGR) in spite of optimal urinary iodine excretion have been reported [2]. Available literature suggests that the development of goiter not only depends on intake of thiocyanate or intake of iodine but critically related to the balance between dietary supplies of iodine and thiocyanate [3]. Therefore a close interrelation may exist between iodine and thiocyanate intake and their excretion pattern in some geographical region where consumption of foods containing thiocyanate precursors is relatively high. Our recent study in six selected areas of Siddharthnagar district (a sub-Himalayan tarai district) showed moderate goiter prevalence in the area and people consume cyanogenic plant foods containing goitrogenic

/anti-thyroidal substances, which are capable of liberating thiocyanate in body after ingestion[4]. Therefore we conducted a cross sectional study covering all the fourteen community development(CD) block of Siddharthnagar to evaluate the iodine nutritional status, consumption pattern of cyanogenic plant food and also to identify whether there is any interrelation between iodine and thiocyanate intake and their excretion pattern among school children in the study region.

Subjects and Methods

The Siddharthnagar district lies between Maharajganj district on the east and Gonda on the west, on the south district Basti separates it while on the north the boundary marches with Nepal. It has 14 Community Development Blocks (CD Blocks) of which 10 are rural and 4 are urban. The total area of the district is about 2895 Sq.km and population is about 20 Lakhs. The present study was conducted in 14 representative localities taking one from each

of the CD Blocks covering the entire Siddharthnagar district by purposive sampling method[5]. The study has been conducted from January to August' 2010. The school children in the age group 6-12 yrs from both sexes were selected as target population, because of their high vulnerability to goiter; easy accessibility and they are representative of their age group in the community[6]. This age group truly reflects the correct status of iodine nutrition in general population [7].

To get the adequate representative target population, in each selected study area, one primary school annexed to a nearby junior high school was randomly chosen. A total of 560 spot urine samples were collected taking 40 urine samples from each study area. Samples were collected from children of both sexes irrespective of their thyroid status at a definite interval maintaining proportionate representation from the entire population of the studied schools following WHO/UNICEF/ICCIDD criteria[8]. Urine samples were kept in a wide mouth screw capped plastic bottles adding a drop of toluene to inhibit bacterial growth and minimize bad odor. The samples were brought to the laboratory and kept at 4°C till analyzed. Iodine in the urine was determined by the arsenite method[9] following dry ashing in presence of potassium carbonate maintaining internal quality control having a known concentration range of iodine content with each batch of test samples. Thiocyanate content in urine was measured from the collected urine samples used for the analysis of iodine by the method of Aldridge[10] and modified by Michajlovskij and Langer[11].

Results

The median value for urinary iodine (MUI) concentration in the studied region was 76.2µg/l, and 32.3% of the urine samples had iodine level below 50 µg/l suggesting biochemical iodine deficiency in the study region. Table 1. shows that in overall 10.7% of the studied population had the iodine excretion level below 20 µg/l showing severe iodine deficiency; 21.6% had iodine level in the range of 20-49 µg/l indicating moderate iodine deficiency; 31.6% had the iodine level in the range of 50-99µg/l indicating mild iodine deficiency, while 36.1% population had no iodine deficiency because their iodine excretion level was equal to or more than 100 µg/l as per WHO/UNICEF/ICCIDD criteria.

The mean urinary thiocyanate level was 0.762±0.45 mg/dl, indicates that the entire studied population is further exposed to thiocyanate load (Table 2). In all the study areas, the mean urinary thiocyanate value was more than the cut off value of 0.504±0.197 mg/dl for non endemic population[12] except in Domriaganj and Bansi. It was found that mean of individual I/SCN ratios obtained from all study areas were 17.518±21.14 µg/mg which is well above the critical level of 7, however, in all 19.4% of the studied individuals had I/SCN ratios below or equal to 7 but more than 3 and 12.1 % had I/SCN ratios below or equal to 3. Therefore a total of 31.5% studied individuals had the I/SCN ratios below or equal to the critical level of 7 (Table 2).

Table 1. Urinary iodine excretion pattern of the studied population of sub-Himalayan tarai region

Study areas (CD Blocks)	Number of Urine sample analyzed	Urinary iodine excretion levels(µg/l)				Iodine Nutrition	
		Median	%Urine Samples <20	%Urine Samples 20-49	%Urine Samples 50-99		%Urine Samples ≥100
Mithwal	40	37.5	25	57.5	12.5	5	Moderate deficiency
Khesraha	40	75.0	7.5	25	35	32.5	Mild deficiency
Uska	40	85.0	2.5	10	42.5	45	Moderate deficiency
Jogia	40	121.2	0	7.5	30	62.5	Optimal
Loton	40	55.0	10	35	35	20	Mild deficiency
Khuniyaon	40	72.5	25	10	35	30	Mild deficiency
Itwa	40	43.7	10	45	35	10	Moderate deficiency
Birdpur	40	142.5	7.5	5	22.5	65	Optimal
Bhanwapur	40	58.7	32.5	15	22.5	30	Mild deficiency
Domriaganj	40	85.0	15	12.5	27.5	45	Mild deficiency
Barhni	40	106.2	2.5	20	25	52.5	Optimal
Sourathgarh	40	100.0	0	12.5	35	52.5	Optimal
Naugarh	40	96.2	2.5	10	42.5	45	Mild deficiency
Bansi	40	52.5	10	37.5	42.5	10	Mild deficiency
All	560	76.2	10.7	21.6	31.6	36.1	Mild deficiency

Table 2. Urinary thiocyanate excretion pattern of studied population of sub-Himalayan tarai region

Study areas (CD Blocks)	Number of urine Sample analyzed	Urinary thiocyanate(USCN) excretion level (mg/dl)	I/SCN ratios ($\mu\text{g}/\text{mg}$)	I/SCN* ($\mu\text{g}/\text{mg}$)	
		Mean \pm SD	Mean \pm SD	<3	3-7
Mithwal	40	0.878 \pm 0.308	5.384 \pm 7.051	16(40)	14(35)
Khesraha	40	0.622 \pm 0.376	27.170 \pm 35.107	2(5)	7(17.5)
Uska	40	0.615 \pm 0.290	24.761 \pm 22.620	1(2.5)	4(10)
Loton	40	0.634 \pm 0.393	13.394 \pm 13.674	2(5)	14(35)
Khuniyaon	40	0.828 \pm 0.443	12.800 \pm 12.396	11(27.5)	5(12.5)
Itwa	40	0.570 \pm 0.156	10.652 \pm 8.761	3(7.5)	13(32.5)
Birdpur	40	1.187 \pm 0.566	21.394 \pm 35.303	0	4(10)
Bhanwapur	40	0.872 \pm 0.540	9.725 \pm 10.183	14(35)	8(20)
Domriaganj	40	0.393 \pm 0.218	34.623 \pm 31.247	6(15)	0
Barhni	40	1.149 \pm 0.550	11.987 \pm 8.706	4(10)	9(22.5)
Sourathgarh	40	0.825 \pm 0.384	18.847 \pm 18.736	1(2.5)	8(20)
Naugarh	40	0.578 \pm 0.293	27.124 \pm 21.646	4(10)	9(22.5)
Bansi	40	0.474 \pm 0.171	14.931 \pm 11.549	2(5)	10(25)
	560	0.762\pm0.456	17.518\pm21.144	68(12.1)	109(19.4)

*Number of individual having I/SCN ratio below or equal to 3 and 7; figures in parentheses indicate percentage.

A significant positive correlation ($r=0.13$, $P<0.01$) found between urinary iodine and urinary thiocyanate concentration of the studied population. The interrelationship between mean urinary iodine and thiocyanate excretion pattern of the population in different study areas are shown

in FigI. Both the lines are running almost parallel to each other i.e. in an area where thiocyanate excretion of the population is high, iodine excretion is proportionally high. Therefore, body's thiocyanate level may be considered as one of the regulator to maintain iodine level in the body.

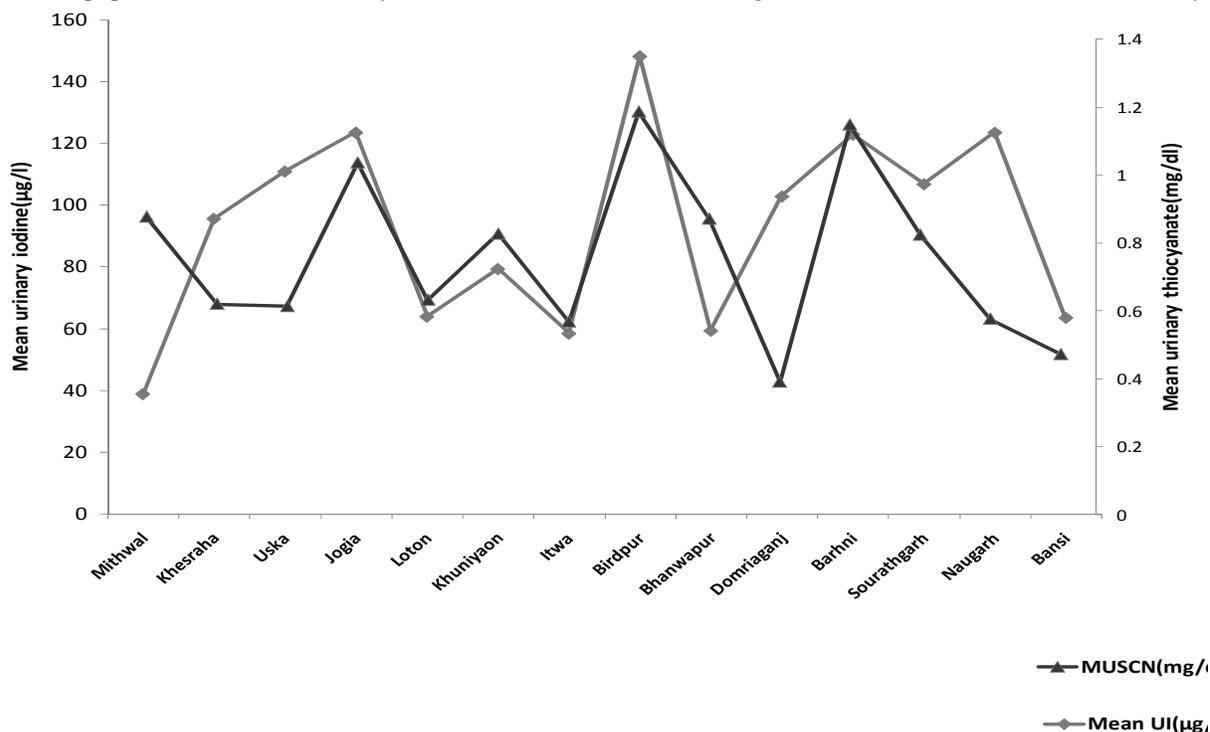


Figure I: Area wise interrelation between urinary iodine(UI) and urinary thiocyanate(USCN) excretion

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Discussion

The cause of endemic goiter has not been completely determined, and is complicated by the existence of the factors varying from region to region. Although the relation of iodine deficiency to endemic goiter is well established, other factors may be involved. Urinary iodine excretion is the most important biochemical marker for the assessment of IDD as 90% of body's iodine is excreted through urine[9]. Urinary excretion pattern of iodine indicates current iodine nutritional status i.e. very recent dietary iodine intake of the population and, therefore, is the index of choice for evaluating the degree of iodine deficiency and of its correction. WHO/UNICEF/ICCIDD have also recommended that no iodine deficiency be indicated in a population when median urinary iodine (MUI) level is 100µg/l or more i.e. more than 50% of the urine samples should have urinary iodine excretion (UIE) level $\geq 100\mu\text{g/l}$ and not more than 20% of the samples have UIE level less than 50 µg/l [6]. But this well known recommendation for the assessment of IDD is not always applicable since the urinary iodine does not always truly reflect the iodine intake/ current iodine nutritional status in an environment where consumption of foods containing thiocyanate precursors is relatively high. In the present study the median urinary iodine concentration in the studied region was 76.2µg/l, which is much lower than the recommended level of 100µg/l and in 32.3% (i.e. more than 20%) of the samples has UIE level less than 50 µg/l, indicating biochemical iodine deficiency of the population in the study region.

In India, a large numbers of cyanogenic plants (SCN precursors) are used as common vegetable and IDD thus persists in many such regions in spite of recommended iodine intake[13]. It was demonstrated that ingestion of the Brassica vegetables by humans causes a rise of thiocyanate ion in the blood followed by its appearance in the urine. The thiocyanate level was found to drop as soon as the eating of Brassica plants was discontinued[14]. Therefore, thiocyanate (SCN^-) in urine reflects the consumption pattern of cyanogenic foods containing goitrogen/anti-thyroid substances such as cyanogenic glucosides and glucosinolates which are precursors of thiocyanate. It has been mentioned that mean \pm SD urinary thiocyanate value obtained from non-endemic population was 0.504 \pm 0.197 mg/dl[12]. In the present study, the mean urinary thiocyanate value was 0.762 \pm 0.45 mg/dl and in all studied areas except in Domriaganj and Bansi, the mean urinary thiocyanate values were much higher than the values found in non-endemic area indicat-

ing that the studied population consumed foods containing cyanogenic glucosides and glucosinolates.

Urinary iodine-thiocyanate ratio (I/SCN) has been recommended as an indicator of the combined effect on the thyroid of low iodine intake and high thiocyanate exposure. The mean ratio is higher than 7 under normal condition and endemic goiter has been found to occur in cassava-eating population in Zaire when the mean ratio is less than critical threshold of 3, and cretinism when it is below 2 [15]. The mean of individual I/SCN ratios obtained from all study areas were well above the critical level of 7. However, a total of 31.5% of the studied population of the sub-Himalayan tarai region had I/SCN ratio below or equal to 7 indicating their susceptibility for the development of goiter.

In the present study, a significant positive correlation ($P < 0.01$) found in between urinary iodine and urinary thiocyanate concentrations which suggests that there is a relationship between urinary iodine and thiocyanate concentration; or in other words when the consumption of thiocyanate is increased as evidenced by increased urinary thiocyanate level, the excretion of iodine is also increased almost proportionally. Thiocyanate and thiocyanate like compounds interfere with iodine metabolism by competitive inhibition of the Γ transport into the thyrocyte, stimulating iodide efflux and replacing iodide by thiocyanate in thyroid gland[16]. Thiocyanate or thiocyanate like compound also inhibit the iodine concentrating mechanism by inhibiting unidirectional clearance of iodide from the thyroid gland[17] or in other words iodine retaining capacity of thyroid/body appears to be dependent on consumption pattern of cyanogenic plant food[18]. Therefore most of the iodine is removed from the thyroid gland and ultimately from the body due to thiocyanate overload leaving less iodine for synthesis of thyroid hormone. In this way thiocyanate interferes with thyroid morphology and thereby iodine nutrition resulting in IDD in certain environmental conditions.

Conclusion

Therefore body's overall thiocyanate concentration perhaps plays an important role to maintain the overall iodine concentration in the body; if the consumption of cyanogenic food is more, the production of thiocyanate becomes high that removes most of the unutilized iodine from the body resulting the excretion of more iodine in comparison to that of the population whose consumption of cyanogenic food is relatively low. Therefore the uri-

nary iodine may not always truly reflect the iodine nutrition status of the body.

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References

1. Chandra AK, Singh H, Tripathi S, Debnath A: Iodine nutritional status of children in North East India. *Indian J of Pediatrics* 2006; 73: 795-798.
2. Chandra AK, Tripathy S, Ghosh D : Goiter Prevalence and the state of iodine nutrition in Sundarban delta of North 24-Parganas in West Bengal. *Asia Pac J Clin Nutr* 2006; 3:357-361.
3. Delange F, Vigneri R, Trimarchi F : Etiological factors of endemic goiter in north-eastern Sicily. *Journal of Endocrinological Investigations* 1978; 1: 137-142.
4. Chandra AK, Bhattacharjee A, Malik T, Ghosh S : Etiological factors for the persistence of endemic goiter in selected areas of Siddharthnagar district in Eastern Uttar Pradesh. *India.J Pediatr Endocrinol Metab.* 2009; 22(4):317-25.
5. Cochran WG. *Sampling technique*, 3rd ed. Calcutta: Wiley Eastern Ltd, 1977.
6. WHO/UNICEF/ICCIDD: Indicators for assessing iodine deficiency disorder and their control through salt iodization. WHO/NUT/94.6, 1994.
7. WHO/UNICEF/ICCIDD. *Global Prevalence of Iodine Deficiency Disorders, Micronutrient Deficiency Information System (MDIS). Working Paper No.1*, 1993; Pp: 5.
8. Dunn JT, Vander Haar F : Detection of iodine deficiency. In: *A practical guide to the correction of iodine deficiency. Technical Manual No.3*, The Netherlands: ICCIDD/UNICEF/WHO Publication,1990; Pp: 13-20.
9. Karmarkar MG, Pandav CS, Krishnamachari KAVR : *Principle and Procedure for Iodine Estimation; A Laboratory Manual*. New Delhi : Indian Council of Medical Research, 1986; Pp:14.
10. Aldridge WN : The Estimation of Micro Quantities of Cyanide and Thiocyanate. *Analyst London* 70, 1945; Pp: 474-5.
11. Michajlovskij N, Langer P: Studien uber benzie hungen zwischen rhodanbildung und kropfbildender eigenschaft von nahrungsmitteln. In: *Gehalt Einiger Nahrungs Mittel an Praformierten Rhodanid*. Hoppe Seyless. *Z Physiol Chem*: 1958; Pp:26-30.
12. Marwaha RK, Tandon N, Gupta N: Residual goiter in the post-iodization phase:iodine status, Thiocyanate exposure and autoimmunity. *Clin Endocrinol* 2003; 59(6): 672-681.
13. Chandra AK, Ray I: Dietary supplies of iodine and thiocyanate in the etiology of endemic goiter in Tripura. *Indian J Pediatr* 2009; 68: 399-404.
14. Langer P. & Michajlovskiji N: The relation between thiocyanate formation and the goitrogenic effects of foods. II. The thiocyanate content of foods, the chief cause of thiocyanate excretion in urine of man and animals. *Zeitschrift fur Physiologische Chemie* 1958; 312: 31-36.
15. Delange F, Bourdoux P, Colinet E, *et al.* Nutritional factors involved in the goitrogenic action of Cassava. In: Delange F and Ahluwalia R, eds. *Cassava Toxicity and Thyroid Research and Public Health Issues*. Ottawa: IDRC, 1982; Pp: 17-50.
16. Gaitan E : Goitrogen in food and water. *Annu Rev Nutr*1990; 10: 21-39.
17. Mitchell M.L. & O'Rourke M.E: Response of thyroid gland to thiocyanate and thyrotropin. *The Journal of Clinical Endocrinology and Metabolism*1960; 20: 47.
18. Chandra AK, Lahari D, Mukhopadhyay S, Tripathy S: Effect of radish (*Raphanus sativus*) on thyroid status under conditions of varying iodine intake in rats. *Indian J Exp Biol* 2006; 44: 653- 661.

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