

Udayan Bhattacharya and Amar K. Chandra*

Socioeconomic status of the population – a prime determinant in evaluating iodine nutritional status even in a post salt iodization scenario

<https://doi.org/10.1515/jpem-2018-0344>

Received August 3, 2018; accepted December 16, 2018; previously published online February 2, 2019

Abstract

Background: To compare the state of iodine nutrition among school age children (SAC) in high- (HSGs) and low-socioeconomic groups (LSGs) during a post iodation scenario in Kolkata.

Methods: Clinical examinations of the goiter, median urinary iodine (MUI), mean urinary thiocyanate (MUSCN) in SAC (6–12 years) from both sexes in the different socio-economic groups were carried out and the iodine content of edible salt was measured.

Results: A total of 5315 SAC, of which 2875 SAC were from a HSG and another 2440 SAC from an LSG were clinically examined for goiter. In the HSGs the total goiter prevalence (TGP) was 3.2% and in the LSGs the TGP was 9.1% and the difference was statistically significant ($p < 0.001$). The MUI of the HSGs was 242 $\mu\text{g/L}$ as compared to 155 $\mu\text{g/L}$ in the LSGs ($p < 0.001$). MUSCN of the HSGs was 0.77 ± 0.45 mg/dL while in the LSGs it was 0.94 ± 0.44 mg/dL and the difference was statistically significant ($p < 0.01$). In the HSGs 19.4% salt samples had 15–30 ppm iodine and 80.6% salt samples were above 30 ppm as compared to 26.3% salt samples which were below 15 ppm, 37.1% salt samples which were between 15 and 30 ppm and 36.6% salt samples which were above 30 ppm in the LSGs.

Conclusions: The population of the LSGs was clinically mildly iodine deficient having no biochemical iodine deficiency while in the HSGs it was more than the adequate requirement and the HSG children are possibly at risk of

excess iodine induced thyroid diseases. Existing goiter prevalence in the LSGs was from their relatively high consumption of dietary goitrogens. Therefore, socioeconomic status plays a pivotal role in the management of iodine nutrition even in a post salt iodation scenario.

Keywords: goiter; iodine deficiency disorders; urinary iodine; urinary thiocyanate.

Introduction

Iodine is a trace element belongs to the halogen family in the periodic table and is required for the production of thyroid hormones in thyrocytes. In environmental iodine deficiency thyroid usually produces the required thyroid hormone if iodine deficiency is not so severe but increases its own volume through a feedback mechanism hypothalamo-pituitary-thyroid-axis. It is manifested by goiter (enlargement of thyroid more than normal), increased susceptibility to stillbirths, spontaneous abortion, congenital anomalies including endemic cretinism and irreversible brain damage collectively called iodine deficiency disorders (IDDs) [1]. Children living in areas affected by iodine deficiency may have an intelligence quotient of up to 13.5 points below that of those living in areas where there is no iodine deficiency [2]. This mental paucity has an effect on a child's learning aptitude, women's health, quality of life and socioeconomic productivity [3]. The World Health Organization (WHO) have announced that globally 2 billion people are iodine deficient of which one-third are school age children (SAC) [4]. In India 350 million people are at risk of IDD [5]. Out of 587 districts in India, 282 have been surveyed for IDD and 241 have been found to be experiencing a goiter endemic [6]. IDD are thus not restricted to the classical hilly sub-Himalayan goiter endemic belt of India. It has also been reported from the plains, (especially areas that are subject to annual flooding), riverine areas, deltas and even in coastal regions [7].

The Kolkata district covers an area of 185 km². As per the 2011 Census it had a population of 4,496,694 [8]. As regards the post salt iodization scenario, reports on the current state of iodine nutrition were not available. Thus,

*Corresponding author: Dr. Amar K. Chandra, UGC Emeritus Professor, Endocrinology and Reproductive Physiology Laboratory, Department of Physiology, University College of Science and Technology, University of Calcutta, 92, A.P.C. Road, Kolkata – 700 009, West Bengal, India, Mobile: +91 94331 61840, E-mail: physiology.ac@gmail.com

Udayan Bhattacharya: Senior Research Fellow, Endocrinology and Reproductive Physiology Laboratory, Department of Physiology, University College of Science and Technology, University of Calcutta, Kolkata, West Bengal, India

the present investigation was conducted to evaluate the variation if any, in the state of the iodine nutrition in two different socioeconomic groups of the population with the possible etiological factors that might exist between high socioeconomic groups (HSGs) and low socioeconomic groups (LSGs).

Materials and methods

Sampling

This was a cross-sectional study; SAC in the age group of 6–12 years of both sexes were selected from two different socioeconomic groups, i.e. HSGs and LSGs. For the HSGs, standard missionary schools in wealthy areas of the city were selected while for the LSGs schools from underdeveloped and slum areas were screened. Furthermore, for the correct representation of the socioeconomic strata, each of the SAC groups was socioeconomically categorized by the socioeconomic evaluation scale developed by Aggarwal et al., in a pre-tested proforma [9]. The list of schools and their enrolment number was previously collected from the School Education Department. The list of schools was divided as per socioeconomic groups into two different tables. From each table, schools were selected using the probability proportionate to size (PPS) methodology [1]. A minimum sample size was calculated based on an assumed goiter prevalence of 5%, 95% confidence interval and a 20% permissible error of margin. Using these parameters a sample size of 1900 was obtained [10]. In the present study, a total 5315 SAC of which 2875 SAC (54.09%) were from the HSGs and other 2440 SAC (45.91%) were from the LSGs. All the SAC from both groups were clinically examined for goiter.

Ethical clearance

The necessary permission was obtained from the Institutional Human Ethical Clearance Committee of the Department of Physiology, University of Calcutta.

Clinical examination of goiter

IDD is chiefly manifested by goiter [11]. SAC were examined clinically for goiter using the palpation method. Grading of goiter was done as follows:

- Grade 0 – No palpable or visible goiter;
- Grade 1 – a goiter that was palpable but not visible when the neck was in the normal position;
- Grade 2 – a goiter that was visible when the neck was in the normal position.

Total goiter prevalence (TGP) (Grade 1 + Grade 2) in SAC in the age group of 6–12 years is most acceptable markers of IDDs in a region. A prevalence rate of 5%–19.9% is considered as mild, 20.0%–29.9% is considered as moderate and prevalence rate of above 30.0% is considered as a severe public health problem [1].

Measurement of iodine in urine samples

Median urinary iodine (MUI) in casual urine samples is currently used as the most practical indicator to assess recent dietary intake of iodine because once the need for thyroidal iodine has been met, excess iodine is excreted by kidneys [12]. To evaluate the state of iodine nutrition, casual spot urine samples were collected from 40 SAC irrespective of their thyroid status from each of the socioeconomic groups of clinically examined enrolled students at a definite interval maintaining proportionate representation from the entire population of studied schools. Iodine in urine was assayed by the kinetic method after mineralization of organic iodide by alkaline ashing in the presence of a strong alkali [13]. An internal quality control methodology was adopted during analysis [6]. A total of 400 urine samples were measured for iodine of which 200 were from the HSGs and another 200 were from the LSGs. An MUI below 99 $\mu\text{g/L}$ was considered as insufficient, 100–199 $\mu\text{g/L}$ was considered as adequate, 200–299 $\mu\text{g/L}$ was considered as above the requirements and greater than or equal to 300 $\mu\text{g/L}$ was considered as excessive iodine intake [1].

Measurement of thiocyanate in urine samples

The quantity of thiocyanate (SCN^-) excreted in urine is an good indicator for the consumption of the most common goitrogenic plant food stuffs in a community [14]. Dietary supplies of cyanogenic glycosides and glucosinolates might be estimated from the quantity of SCN^- in the urine [15]. Urinary SCN^- was estimated from urine samples which were already collected for iodine assay using Aldridge's method [16] and modified by Michajlovskij and Langer [17].

Measurement of iodine in edible salt samples

In India iodine is supplemented through edible salt because it is inexpensive and consumed by every class of person in a community in a uniform manner throughout the year [1]. A total of 350 edible salt samples were collected of which 175 edible salts were from the HSGs and another 175 were from the LSGs at a definite interval. Iodine content of edible salt samples was measured by iodometric titration [18].

Statistical analysis

A nonparametric chi-square (χ^2) test was applied for any significant difference in TGP in the two different socioeconomic groups and for a comparison of TGP between boys and girls [10]. The contingency coefficient (C) was calculated for the correlation between socioeconomic status and goiter prevalence. The computed C was then converted to χ^2 to test the null hypothesis [19]. As per recommendations of WHO/UNICEF/ICCIDD, the mean, median, mode, standard deviation (SD), skewness (Sk), kurtosis (κ) and percentile values (P20 and P80) were computed for urinary iodine values [1]. Student's t-test was computed for comparison of mean urinary iodine between the two different socioeconomic groups. For urinary thiocyanate data mean and SD were computed. Salt iodine values were categorized below 15 ppm, 15–30 ppm and more than 30 ppm. All descriptive statistical analysis was done using Microsoft Office Excel 2007.

Results

Goiter prevalence

In the HSGs, the TGP was 3.2% of which palpable (grade 1) goiter was 3.1% and visible (grade 2) goiter was 0.1%. In the LSGs, the TGP was 9.1% of which palpable goiter was 7.9% and visible goiter was 1.2%. The TGP of the LSGs was significantly higher than in the HSGs ($p < 0.001$). A significant positive correlation was found between TGP and socioeconomic status ($C = 0.130$, $p < 0.001$) (Table 1).

Urinary iodine

In the HSGs the median and mean \pm SD value was 242.00 and 253.78 ± 84.15 $\mu\text{g/L}$. The P_{20} and P_{80} value were 202.50 $\mu\text{g/L}$ and 295.50 $\mu\text{g/L}$, respectively. The 95% confidence level was between 241.94 $\mu\text{g/L}$ and 265.62 $\mu\text{g/L}$. As per the WHO/UNICEF/ICCIDD criteria the MUI values were calculated, because MUI values are asymmetric. The mean was much higher than the median, indicating that distribution was heavily skewed to the right ($Sk = +1.227421$) and the histogram was platykurtic ($\kappa = 2.633317$). The frequency distribution of MUI in the HSGs indicated that there was no iodine deficiency, 19.5% of the populations' iodine intake was adequate, 61.5% of the populations' iodine intake was more than adequate and 19% of the populations' iodine intake was excessive. In the HSGs no salt samples were found below 50 $\mu\text{g/L}$ and 100 $\mu\text{g/L}$ (Table 2, Figure 1).

In the LSGs the median and mean \pm SD urinary iodine excretion pattern was 155.00 and 184.00 ± 109.67 $\mu\text{g/L}$.

P_{20} and P_{80} values were 117.50 $\mu\text{g/L}$ and 212.50 $\mu\text{g/L}$, respectively. The 95% confidence level was between 168.81 $\mu\text{g/L}$ and 199.19 $\mu\text{g/L}$. The distribution was heavily skewed to right ($Sk = +1.952313$) and the histogram was platykurtic ($\kappa = 3.860113$). The frequency distribution of MUI indicated that to date 11% of the population were iodine deficient, 64% of the populations' iodine intake was adequate, 15% of the populations' iodine intake was more than adequate and 10% of the populations' iodine intake was excessive. In the LSGs of Kolkata, 11% of the urine samples were below 100 $\mu\text{g/L}$ and 3% of the urine samples were below 50 $\mu\text{g/L}$ of iodine. The median and mean \pm SD urinary iodine excretion of the HSGs was significantly higher than the LSGs in the studied population ($p < 0.001$) (Table 2, Figure 1).

Urinary SCN⁻

In the HSGs, the mean urinary thiocyanate (MUSCN) excretion was 0.77 ± 0.45 mg/dL. In the LSGs, MUSCN excretion was 0.94 ± 0.44 mg/dL. The difference of the MUSCN excretion pattern between the HSGs and the LSGs was statistically significant ($p < 0.01$) (Table 3, Figure 2).

Edible salt samples

In the HSGs no families were found to consume salt below 15 ppm of iodine; 19.4% of families consumed salt containing 15–30 ppm of iodine and 80.6% of the families consumed salt containing iodine with more than 30 ppm.

Table 1: Severity of goiter prevalence (in grades) of the school children in different socioeconomic strata.

Socioeconomic group	Studied population	Goiter prevalence			Severity as a public health problem
		1	2	TGP (1+2)	
HSG	2875	89 (3.1)	2 (0.1)	91 (3.2) ^a	None
LSG	2440	192 (7.9)	30 (1.2)	222 (9.1) ^a	Mild

^a $\chi^2 = 91.37$; $df = 1$; $p < 0.001$; $C = 0.130$; $df = 2$; $p < 0.001$ (values in parenthesis indicates percentage).

Table 2: Urinary iodine excretion pattern of the studied population in different socioeconomic groups.

Socioeconomic status	Median mean \pm SD, $\mu\text{g/L}$	Percentage of urine samples < 50 , $\mu\text{g/L}$	Percentage of urine samples < 100 , $\mu\text{g/L}$
HSG	242.00 253.20 ± 85.47^a	0	0
LSG	155.00 184.00 ± 109.67^a	6 (3.0%)	22 (11.0%)

^aThe mean \pm SD difference is statistically significant ($p < 0.001$).

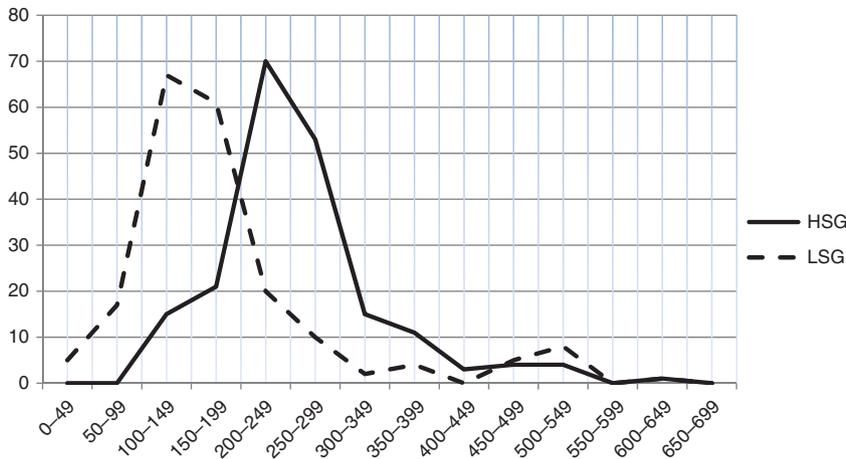


Figure 1: Histogram of MUI excretion pattern of the HSGs and the LSGs of Kolkata district.

Table 3: Urinary thiocyanate excretion pattern of the studied population in different socioeconomic groups.

Socioeconomic status	Thiocyanate excretion pattern mean ± SD, mg/dL
HSG	0.77 ± 0.45 ^a
LSG	0.94 ± 0.44 ^a

^aThe difference was statistically significant (p < 0.01).

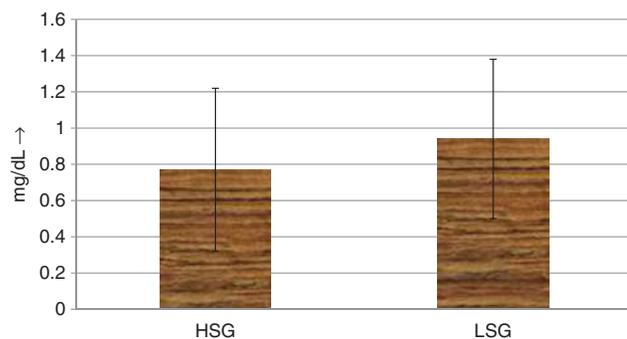


Figure 2: Bar diagrammatic representation of mean ± SD urinary thiocyanate excretion patterns of the HSGs and the LSGs of Kolkata district.

In the LSGs 26.3% of the families consumed salt containing less than 15 ppm of iodine, 37.1% of the families consumed iodine in the range 15–30 ppm and 36.6% of the

Table 4: Distribution of iodine content of edible salt samples collected from different socioeconomic groups.

Socioeconomic status	<15 ppm	15–30 ppm	>30 ppm
HSG	0	34 (19.40%)	141 (80.60%)
LSG	46 (26.30%)	65 (37.10%)	64 (36.60%)

ppm, parts per million.

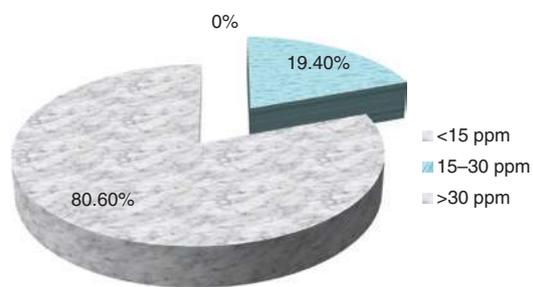


Figure 3: Pie diagram of edible salt iodation status of the HSGs of Kolkata district in a post iodation scenario.

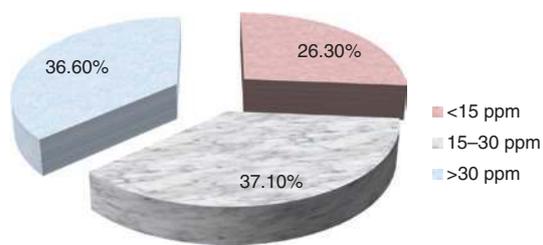


Figure 4: Pie diagram of edible salt iodation status of the LSGs of Kolkata district in a post iodation scenario.

families consumes more than 30 ppm of iodine (Table 4, Figures 3 and 4).

Discussion

West Bengal is located in the eastern part of India. Its northern part is hilly and is in the classical sub-Himalayan goiter endemic belt of India. Kolkata is the capital of the state West Bengal as well as a metropolitan city which is spread roughly north-south along the east bank of the Hooghly River (Bhagirathi) and is situated within

the lower Ganges delta (Sundarban) of eastern India. In Kolkata during a post iodation scenario the TGP of the HSGs was below the recommended margin of severity but in the LSGs, TGP was a mild public health problem. TGP of the LSGs was found to be significantly higher than in the HSGs in Kolkata and a significant positive correlation was found between TGP and socioeconomic status. Iodine nutritional status of SAC in the LSGs was not adequate as was evidenced by their TGP and this group is more susceptible to IDD. It has previously been reported that TGP was higher in public schools than in private schools and in the lower socioeconomic and lower educated strata [20]. The higher TGP in the lower socioeconomic strata was also reported by earlier observers [21, 22].

The MUI of the SAC of the LSGs was adequate and their iodine nutritional status was optimum while the iodine nutritional status of the HSGs was more than adequate and the group is probably at risk of iodine induced hyperthyroidism [1]. MUIs of the HSGs were found to be 242.00 $\mu\text{g/L}$ which is much higher than the recommended level by WHO/UNICEF/ICCIDD. If such median iodine values continued among the HSGs for a longer period, that is, throughout their entire lives it may cause adverse effect of excess iodine in the long run. The primary cause of endemic goiter includes iodine deficiency and iodine excess. Iodine deficiency causes goiter through the hypothalamic-pituitary-thyroid axis by the secretion of less thyroid hormone or by the interference of environmental substances that interfere iodine nutrition [23]. On the other hand, excess iodine in the thyroid inhibits thyroid hormone synthesis and secretion resulting thyroid enlargement or goiter [24].

Cyanogenic glycosides (SCN^- precursors) are found in plant foods such as cassava, maize, cabbage, cauliflower, bamboo shoots, radish, sweet potatoes and many others eaten in the third world. After ingestion these glycosides are readily converted to thiocyanate by glycosidases and sulfur transferases. SCN^- has been demonstrated as a goitrogenic constituent in plants from the Cruciferae family [25]. It has same ionic charge and ionic volume to that of iodide and thus is a potent inhibitor of iodide (I^-) transport through the basolateral membrane of thyroid follicular cell. Consumption of naturally occurring goitrogens/cyanogenic glycosides can significantly increase SCN^- concentrations to levels potentially capable of affecting the thyroid [25]. Despite certain similarities SCN^- has with I^- , it differs in several aspects: (i) It is not concentrated by thyroid tissue. (ii) It has a more marked effect on iodide efflux *in vivo* and *in vitro*. (iii) It is actively metabolized by thyroid tissue. (iv) SCN^- is an inhibitor of organic iodination at slightly higher concentrations than that inhibiting

iodide transport. It has been proposed that SCN^- is a competitive substrate for thyroid peroxidase (TPO) explaining its biotransformation and ability to inhibit iodination [26]. SCN^- overload have also been implicated as a cause of goiter development. Consumption of poorly detoxified cassava and vegetables from the Brassica family aggravates iodine deficiency by releasing SCN^- anions, which, in turn, competes with I^- for thyroidal uptake [27]. Bamboo shoots are rich in cyanogenic constituents and its consumption may affect thyroid function in spite of adequate iodine intake [28]. Large numbers of cyanogenic plant foods (SCN^- precursors) are consumed in India as common vegetables and thus IDD persists in many such pockets in spite of recommended iodine intake [29–32]. Boiled extracts showed maximum inhibition of TPO activity followed by cooked and raw extracts [33]. Regular consumption of this type of cyanogenic plant foods affect the thyroid and may develop endemic goiter in the long run [34]. It has been reported that SCN^- appeared to play an important role in goiter formation especially among poor children in India during a post iodation scenario [22]. The mean \pm SD value of urinary SCN^- obtained from the non-goitrous population was 0.71 ± 0.34 mg/dL. In the present study, MUSCN excretion of the HSGs was not significantly higher than MUSCN excretion of the non-goitrous population ($p > 0.05$) but in the LSGs, excretion of MUSCN was significantly higher than MUSCN excretion of the non-goitrous population ($p < 0.0005$). In addition, MUSCN excretion in the LSGs was significantly higher than in the HSGs. All these observations suggest that school children in the LSGs of the studied region are exposed to excess thiocyanate load through the consumption of cyanogenic plant foods and thiocyanate may have a possible role in the etiopathogenesis of endemic goiter in those belonging to the LSGs.

In India salt is the best and most ideal physiological vehicle for dietary supplementation of iodine because salt is a dietary necessity and is consumed daily and is cost effective, e.g. in India the estimated cost of iodine per person per year is negligible [35]. In 1962, the Government of India (GOI) launched the National Goiter Control Programme (NGCP). In August 1992, the NGCP was renamed as the National Iodine Deficiency Disorders Control Programme (NIDDCP) [36]. In 1997 the GOI banned the sale of non-iodized salt. In September 2000, the GOI lifted the ban on the sale of non-iodized salt stating “On a point of principle, compulsions in the matter of individual choice are undesirable”, resulting in a 12% decrease in the coverage of iodized salt nationwide. In May 2006 the GOI re-imposed the ban [25, 37]. However, the GOI has implemented a “Universal Salt Iodization Programme” (USIP) to meet the

body's iodine status in the community since 1984. The Ministry of Health and Family Welfare fixed standards for edible common salt in 1981 under the Prevention of Food Adulteration Act (PFA), 1954. Under this act the iodine content shall be not less than 30 ppm at the manufacturers' level and shall be not less than 15 ppm in the distribution channel including at the retail level. The Director of Health Services (Food Health Authority) of each state and union territory issues notifications banning the entry and sale of non-iodized salt in each state and union territory under the PFA, 1954 [35]. In NFHS-3, in the third cycle of the survey 51% of households in India were using adequately iodized salt whose iodine content was ≥ 15 ppm. Another 25% were using salt that had iodine less than 15 ppm and 24% of households were found to be using non-iodized salt [38]. In our study during the USIP in Kolkata, in the HSGs no household was found using salt below the recommended level. In the LSGs, 73.7% of households were using properly iodized salt and 26.3% of households were using edible salt below the recommended level. The goal of a successful salt iodization program is that 90% of household should be using iodized salt [1]. The results of the median and mean \pm SD urinary iodine excretion patterns indicate that the population belonging to the LSGs consume less iodine than the HSGs because salt consumed by the LSGs was not adequately iodized. According to the NFHS-3, people living in low socioeconomic strata are at maximum risk to the effects of iodine deficiency because this group is consuming salt with less iodine or inadequately iodized salt. In India about half of the population is covered by properly iodized salt and coverage of iodized salt is poor in low socioeconomic and low educated strata [38]. Allowing for choice and price difference will lead this group to opt to use common salt and therefore, this makes them vulnerable to the consequent deleterious medical effects of iodine deficiency. For the successful implementation of a USIP special attention is needed for people of the LSGs in Kolkata. In sub-Saharan Africa 67% of households are using iodized salt. In Sudan, Mauritania, Guinea-Bissau and Gambia the coverage of iodized salt is less than 10% [24]. In Italy, both Italian and immigrating women with a low income or without access to the public health system have a poor adherence both to the salt iodization policy and iodine supplements in preconception and pregnancy. They also indicated a low-frequency intake of iodine-rich foods [39].

This study reveals that the overall goiter prevalence, urinary iodine excretion pattern, intake of iodine through iodized salt available in the market, consumption pattern of cyanogenic plant food as shown by the urinary SCN^- excretion pattern of the studied population are very

different between LSGs and HSGs though they are from the same geographical region. The population of the LSGs are exposed to an SCN^- load that may interfere with iodine metabolism leading to iodine deficiency while the HSGs are exposed to more than adequate iodine intake and they are at risk of excess iodine induced hyperthyroidism, hypothyroidism and iodine induced thyroiditis. Such iodine intake in the long run might affect thyroid function. The underlying cause for such differences is their socioeconomic status or in other words the socioeconomic status of the community plays a pivotal role in the management of iodine nutritional status.

Acknowledgments: The authors duly acknowledge the students who participated in this study.

Author statements: The article is original in nature and based on the studies of school children in the age group 6–12 years from low- and high-socioeconomic groups in the Kolkata metropolitan city of India following the guidelines of WHO/UNICEF/Global Iodine Network.

Authors contribution: Dr. Udayan Bhattacharya wrote the first draft of the manuscript and conducted the whole research work under direct supervision of Prof. Amar K Chandra. All the authors have accepted responsibility for the entire content of this submitted manuscript and approved submission.

Research funding: This work was supported by University of Calcutta research grants BI (92) for Endocrinology and Reproductive Physiology Laboratory, Department of Physiology.

Employment or leadership: None declared.

Honorarium: None declared.

Conflict of interest: The funding organization(s) played no role in the study design; in the collection, analysis, and interpretation of data; in the writing of the report; or in the decision to submit the report for publication.

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