Volume 7 Issue 3



BIOCHEN An Indian Journal Regular Paper

BCAIJ, 7(3), 2013 [97-101]

Assessment of iodine status of some selected populations in Anambra state, Nigeria

Ifeyinwa Chidiogo Olife^{1*}, Benedette Azuka Anajekwu², Agwu Kama Onuogbu¹ ¹Raw Materials Research and Development Council, Abuja, (NIGERIA) ²Department of Biochemistry Nnamdi Azikiwe University, Awka, (NIGERIA) E-mail: ifeeolife@yahoo.com

ABSTRACT

The study was designed to determine the iodine status of selected population in Oba and Nanka communities of Anambra State and assess their risk to Iodine Deficiency Disorders (IDD). Iodine is excreted mainly in the urine with smaller amounts appearing in the feaces and sweat. Urinary iodine excretion represents 75-90% of total intake and is therefore the accepted method of assessing iodine status. Urinary iodine levels of selected school children and women of child bearing age in both communities were determined. The result showed that the mean urinary iodine concentration of 170.65±27.17µg/L in school children from Oba is significantly higher (p<0.05) compared to the mean concentration of 156.12±16.48µg/L found in school children from Nanka. However, the mean urinary iodine concentrations of the women were not significantly different in the two communities. Extensive dietary supplementation programmes, often based on the addition of iodized oil, iodized salt or iodination of irrigation water have successfully lowered the incidence of IDD in countries such as China, USA, Switzerland, Papua Guinea and India. It is now well recognized that the most effective way to achieve the virtual elimination of IDD is through universal salt iodization. Universal salt iodization involves iodization of all human and livestock salt, including salt used in food industries. Adequate iodization of all salt will deliver iodine in the required quantities to the population on a continuous and self-sustaining basis. Therefore, the levels of iodine in salt samples purchased from local markets in the two communities were determined. The results showed that the levels of iodine in salt samples from Oba market ranged from 7-30ppm while the levels of iodine in salt samples from Nanka market ranged from 0-30ppm. One teaspoon of iodine is all a person requires in a lifetime and yet its deficiency at critical stages of fetal life and early childhood remains the world's single most important and preventable cause of mental retardation. © 2013 Trade Science Inc. - INDIA

KEYWORDS

Iodine; Urinary iodine; School children; Women salt; Iodine deficiency disorders (IDD).

Regular Paper 🛥 INTRODUCTION

Iodine is a trace element, which is essential to human and animal health in small doses. Iodine forms an important constituent of the thyroid hormones, thyroxine (T4) and triiodothyronine (T3). These hormones play a fundamental biological role controlling growth and development^[1]. If the amount of utilizable iodine reaching the thyroid gland is deficient or if the thyroid gland is not functioning properly, the hormone production will be reduced resulting in a group of conditions in man, collectively referred to as Iodine Deficiency Disorders (IDD)^[2].

A turning point in the struggle against iodine deficiency came in 1983 when the term "Iodine Deficiency Disorders" (IDD) was introduced by Hetzel^[3]. This redefined and emphasized that the problem extends far beyond goiter and cretinism and drew particular attention to the devastating effects that iodine deficiency has on the developing brain^[4]. It became recognized that the effect of iodine deficiency on the function of the thyroid gland is critical, rather than its effect on its structure. The most devastating effect is on the developing brain resulting in mental retardation^[5]. The severity of the mental retardation varies from mild intellectual blunting to frank cretinism. Since 1993, further research has demonstrated that in iodine deficient communities, there is a general diminution of mental ability and as many as ten IQ points may be lost compared to similar but noniodine deficient communities^[6].

One teaspoon of iodine is all a person requires in a lifetime and yet its deficiency at critical stages of fetal life and early childhood remains the world's single most important and preventable cause of mental retardation^[7,8]. Knowledge of the global magnitude of IDD and thus its real significance for health and socio-economic development has improved considerably in recent years^[9]. The World Health Organization (WHO), International Council for the Control of Iodine Deficiency Disorders (ICCIDD) and the United Nations Children's Education Fund (UNICEF) have worked closely for decades to combat IDD. In 1999, WHO^[10] estimated that of its 191 member countries, 130 had significant IDD problems. Extensive dietary supplementation programmes, often based on the addition of iodized oil, iodized salt or iodination of irrigation water^[11] have successfully lowered the incidence of IDD in countries such as China, the USA, Switzerland, Papua Guinea and India. Despite the success of these programmes, WHO declared that IDD remain a serious global health issue affecting 1.6 billion people worldwide^[12].

It is now well recognized that the most effective way to achieve the virtual elimination of IDD is through salt iodization. Universal salt iodization involves iodization of all human and livestock salt, including salt used in the food industries. Adequate iodization of all salt will deliver iodine in the required quantities to the population on a continuous and self-sustaining basis. Although most people now use iodized salt, some people add the salt before cooking. Much of the iodine added this way may be lost to the atmosphere during the cooking process. Storing iodized salt for long periods or adding it to food before cooking may reduce its effectiveness^[13]. Iodized salt can also deteriorate due to excessive or long-term exposure to moisture, light, heat and contaminants. Under these circumstances, iodine losses can be up to 50% or more from the moment salt is produced until it is actually consumed^[14]. Also, there is a reduced salt usage in cooking and at the table in response to health guidelines to reduce salt intake^[15].

In addition to inadequate amounts of iodine in the environment, consumption of goitrogenic substances further reduces thyroid hormone production through a variety of mechanisms^[16]. Goitrogens exert their effects by inhibiting the uptake or concentration of iodine into the thyroid gland^[17]. The anti-thyroid effects of flavonoids in millet have beed demonstrated^[18]. Flavonoids contained in millet and other cereals inhibit thyroid peroxidase, the enzyme catalyzing hormone system in the thyroid. Goitrogens also interact with iodothyronine deiodinase enzymes, inhibiting the peripheral metabolism of thyroid hormones^[19]. Some staple foods in developing countries, particularly cassava, contain goitrogens, which worsen the iodine deficiency problems. Other foods, such as legumes, also have goitrogenic effects.

Iodine is excreted mainly in the urine with small amounts appearing in the feaces and sweat. Urinary iodine excretion represents 75-90% of total intake and is therefore the accepted method of assessing iodine status^[20,21]. The study was designed to determine the iodine status of selected populations in Oba and Nanka

BIOCHEMISTRY Au Iudian Journal

Regular Paper

communities of Anambra State and assess their risk to IDD.

METHODOLOGY

Population

The study populations resided in two communities (Oba and Nanka) in Anambra State. 200 school-age children (4-10yrs) were sampled after informed consent and due permission from their head teacher. In each community, 100 school children from four different primary schools were sampled.

A total of 120 women of child bearing age were sampled from different hospitals and health centres in the two communities (60 pregnant and 60 non-pregnant). In all, informed consent was obtained.

Sampling strategy

Casual urine samples were collected from all the subjects in tightly sealed specimen bottles. The samples were then kept in the freezer until analysis.

Salt sampling

In Oba market, salt are sold in 0.5 - 1kg branded sachets. 4 salt samples of different brands were purchased from local market in Oba. However, in Nanka, salts are purchased in 20 - 30kg bags and poured into different sizes of basin for display. So, in the absence of branded salt in Nanka market, 6 unbranded salt samples sold by retailers were purchased and labeled A – F.

Analytical methods

The method for measuring iodine in urine^[22] was used to measure urinary iodine. Urine samples were completely defrosted before analysis and were mixed to suspend sediments.

Iodine contents of salt samples were estimated using field test kit (MBI Kit manufactured by Machine Build Industries, India was used).

RESULTS AND DISCUSSION

Mean Urinary Iodine (UI) levels of school in four different schools (A-D) in both Nanka nad Oba are shown in TABLE 1. There is no significant difference when the values from the different schools in Oba were compared (p>0.05). However, there is significant difference between values from school A and B in Nanka (p<0.05). The mean UI concentration of school children in Oba is 170.65µg/L while the mean UI concentration of school children from Nanka is156.12 µg/L. The difference between the UI levels of school children from both communities is highly significant (p<0.05). The mean iodine intake for school children in the study were 140.5µg/d for Nanka and 153.59µg/d for Oba. The values were comparable to the recommended nutrient intake of 120µg/d for school children between the ages of 6 and $12^{[20]}$.

TABLE 1 : Mean urinary iodine levels of sampled children in Oba and Nanka (values in $\mu g/L$)

Community	School A	School B	School C	School D
Oba	185.0±17.89	167.0±16.73	134.4±11.95	181.0±20.48
Nanka	147.2±9.96 ^a	$143.6{\pm}19.09^{a}$	152.6±10.88	155.0±10.95

^a represents p<0.05 for school A compared to school B in Nanka; Values are ±SD; T-tests were used; N=25 for each school

Mean UI concentrations of pregnant women and non-pregnant women in Nanka are 163.93µg/L and 154.03µg/L respectively while the values for Oba women are 169µg/L and 164.55µg/L respectively (TABLES 2 and 3). When compared, there were no significant differences between all the groups of women. The mean iodine intakes for pregnant women in the study were 147.54µg/d and 144.0µg/d for Nanka and Oba respectively. Also, mean values for non-pregnant women were 138.63µg/d for Nanka and 148.10µg/d for Oba. The values for all groups of women are less that the recommended nutrient intake of 150µg/d for adults^[20]. This is of particular concern for the pregnant women as the requirement for iodine is increased during pregnancy, with an addition of 30µg during pregnancy and 50µg during lactation^[20].

TABLE 2 : Mean urinary iodine levels of sampled women of child bearing age in Nanka (values in $\mu g/L$)

Population	Hospital A	Hospital B	Hospital C
Pregnant women	146.6±15.90 ^a	164.4±11.82	$169.0{\pm}26.08^{a}$
Non-pregnant women	146.0±10.91	150.0±19.21	154.0±19.47

^a represents p<0.05 for hospital A compared to C; Values shown are mean \pm SD; T-tests were used; N=20 for each group of women in each hospital

Levels of salt samples from Oba market range from





Regular Paper

7 to > 30ppm, while salt from Nanka market have iodine levels ranging from 0 to > 30ppm (TABLE 4).

TABLE 3 : Mean urinary iodine levels of sampled women of child bearing age in Oba (values in $\mu g/L$)

Population	Hospital A	Hospital B	Hospital C
Pregnant women	155.4±21.8	151.8±18.65	153.8±20.57
Non-pregnant women	149.8±8.17	174.4±24.59	164.4±11.82

Values shown are mean \pm SD; T-tests were used; N=20 for each group of women in each hospital; No significant differences between the groups.

 TABLE 4 : Levels of iodine in salt samples collected from

 Oba and Nanka markets (values in ppm)

Oba (Brandedsamples)	Nanka (unbranded salt A -F)		
Dangote Salt >30	A 0		
Royal salt 7	B > 30		
Dicon salt >30	C 15		
Uncle palm salt 15	D>30		
	E 15		
	F 7		

CONCLUSION

The results showed that people living in the two communities, especially women, are at risk of IDD. In reviewing the prevalence data on goiter throughout the world, females fro adolescence onwards generally have a higher prevalence of goiter than males, perhaps due to differences in metabolism of iodine during adolescent growth^[12]. In 1993, it was estimated that the prevalence of goiter in Nigeria is above 20% with the highest endemic rate in the mid Southern part of the country stretching from East to the West^[23,24]. Within countries, the levels of IDD often vary significantly from area to area and a single national estimate, many a times, does not reflect sub-national variability in the prevalence of IDD.

It is recommended that an education programme be initiated to increase the effectiveness of the iodized salt programme, educating people that to store iodized salt for long periods or adding salt to food before cooking reduces the effectiveness of the iodine in the salt. Also goitrogen-containing foods such as cassava and cereals must be adequately processed to reduce the anti-nutritional effects. Avoidance of severe iodine deficiency is a must for any official health care. Surveillance and control of iodine is highly cost effective compared to diagnosing, therapy and control of many individual cases of IDD that might have been prevented. The need for continued vigilance is underlined, as is the importance of periodic urinary iodine surveys.

REFERENCES

- [1] H.S.Hetzel, G.F.maberly; Iodine. In: W.Mertz, (Ed); Trace elements in human and animal nutrition. Academic Press Inc. London, 139-197 (**1986**).
- [2] M.A.Fernando, S.Balasuriya, K.B.Herath, S.Katugampola; Endemic goiter in Sri Lanka. In. C.B.Dissanayake, L.Gunatilaka, (Eds); Some aspects of the environment of Sri Lanka. Colombo: Sri Lanka Association for the Advancement of Science, 46-64 (1987).
- [3] B.S.Hetzel; Iodine Deficiency Disorders (IDD) and their eradication. The Lancet, 2, 1126-1129 (1983).
- [4] R.DeLong; Neurological involvement in iodine deficiency disorders. In. Hetzel, et al., (Eds); The prevention and control of iodine deficiency disorders. Elsevier, Amsterdam, 49-63 (**1987**).
- [5] G.R.DeLong, J.B.Stanbury, R.Fierro-Benitez; Neurological signs in congenital iodine deficiency disorder (endemic cretinism). Dev.Med.Child Neurol, 27, 317-324 (1985).
- [6] N.Bleichrodt, M.P.Born; A meta-analysis of research on iodine and its relationship to cognitive development. In: J.B.Stanbury, (Ed); The damaged brain of iodine deficiency. Cognizant Communication, New York, 195 (1994).
- [7] B.S.Hetzel; Iodine deficiency and fetal brain damage. New Eng.J.Med, 331, 1770-1771 (1994).
- [8] G.R.DeLong; Effects of nutrition on brain development in humans. Am.J.Clin Nutr., 57, 2865-2905 (1993).
- [9] K.V.Bailey, G.A.Clugston; Iodine deficiency disorders. In: C.J.Murray, A.D.Lopez, (Eds); The global burden of disease and risk factors. World Health Organization/World Bank, Geneva, (1990).
- [10] World Health Organization. Progress towards the elimination of iodine deficiency disorders. UNICEF/ ICCIDD/WHO, Geneva, (1999).
- [11] G.R.DeLong, P.W.Leslie, S.H.Wang et al.; Effects on infant mortality iodination of irrigation water in a severely iodine in a severely iodine-deficient area



Regular Paper

of China. Lancet, 350(9080), 771-773 (1997).

- [12] World Health Organization. Global prevalence of iodine deficiency disorders. UNICEF/ICCIDD/ WHO, Geneva, (1993).
- [13] F.M.Fordyce, C.C.Johnson, R.B.Udaya, U.R.Navaratna et al.; Selenium and iodine soil, rice, drinking water in relation to endemic goiter in Sri Lanka.Sci.Total Environment, 263(1-3), 127-141 (2000).
- [14] World Health Organization. Recommended iodine levels in salt and guidelines for monitoring their adequacy and effectiveness. WHO, Geneva, (1996).
- [15] C.D.Thomson, A.Colls, J.Conaglen, M.Macormack, M.Stiles, J.Mann; Iodine status of New Zealand residents as assessed by urinary iodine excretion and thyroid hormones. Br.J.Nutr, 78, 902-912 (1997).
- [16] F.M.Delange, A.M.Ermans; Endemic goiter and cretinism. Naturally occurring goitrogens. In: J.M.Hershman, G.A.Bray, (Eds); The thyroid-physiology and treatment of disease. Oxford, Pergamon press, 415-451 (1979).
- [17] A.W.Halverson, M.Zepplin, E.B.Hart; Relation of iodine to the goitrogenic properties of soybeans. Journal of Nutrition, 38, 115 (1949).

- [18] H.Sartelet, S.Serghat, A.Lobstein; Flavonoids extracted from fonio millet reveal potent anti-thyroid properties. Nutrition, 12, 100 (1996).
- [19] E.Gaitan, R.C.Cooksey, J.Legan, R.H.Lindsey; Antithyroid effects *in vivo* and *invitro* of vitenin: ac-glycosylflavone in millet. J.Clin.Endocrinol. Metabol, 3(5), 170-175 (1995).
- [20] A.S.Truswell, I.E.Dreost, R.M.English, I.H.Rutishauser, N.Palmer; Recommended Nutrient intakes. Australian papers, Australian Professional Publications, Sydney, (1990).
- [21] R.S.Gibson; Principles of Nutritional Assessment. Oxford University Press Incorporated, New York, (1990).
- [22] J.T.Dunn, H.E.Crutchfield, R.Gutekunst, A.D.Dunn; Methods for measuring iodine urine. ICCIDD/UNICEF/WHO, The Netherlands, (1993).
- [23] J.Egbuta; UNICEF Consultancy report on IDD situation in Nigeria, (1993).
- [24] R.Gutekunst; Report of Consultancy on the Use of Ultrasonography for IDD survey in Nigeria, (1993).

BIOCHEMISTRY

An Indian Journal