

Assessment of Urinary Iodine Status of Primary School Children in Saki, in South Western Nigeria

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ABSTRACT

Urinary iodine excretion is a good marker of the dietary intake of iodine and is the index for evaluating the degree of iodine deficiency, correction and toxicity. Previous studies on urinary iodine excretion measured in school children in Saki showed that iodine deficiency was still endemic. The current iodine status in the area has not been evaluated, thus this study.

Random urinary iodine was determined in 280 primary school children in Saki. The ages of the subjects ranged from 8-12 years with a mean of 9.23±1.28 years. Urinary iodine level was measured using the standard method of ammonium persulphate reaction.

Classifying the urinary iodine level obtained based on World Health Organization (WHO), United Nation's International Children Emergency Fund (UNICEF) and International Council for the Control of Iodine Deficiency Disorders (ICCIDD) recommendation, results showed that of the 280 primary schoolchildren studied, 0.7% (2) had sever iodine deficiency, 3.2% (9) had moderate iodine deficiency, 5% (14) had mild iodine deficiency, 41.1%(115) fell into the sufficient group and 50%(140) had excess, with urinary iodine level greater than 300µg/L

These findings suggest that iodine nutrition has greatly improved in Saki, nevertheless, current efforts should be targeted towards sustaining the achievements thereby preventing over iodization of the population.

Key words: Iodine, Iodine deficiency, Iodine Induced Hyperthyroidism.

INTRODUCTION

Iodine is an essential micronutrient for all animal species, including humans. It is necessary for thyroid hormone synthesis. Thyroid hormone influences general body metabolism. Based on this important physiological function of thyroid hormone, all other functions of iodine are mediated through this mechanism.

The association between iodine deficiency and endemic goiter has been known for centuries [1]. These disorders are collectively described as iodine deficiency disorders (IDD) and it includes: endemic goiter, hypothyroidism, cretinism and congenital abnormalities [1,2]. While endemic goiter is the most visible consequence of iodine deficiency, the most significant and profound are on the developing brain. The potential impact of iodine deficiency on the intellectual development of large segments of the populations in underdeveloped countries is of particular concern, especially when all of the adverse effects of iodine deficiency can be prevented by long-term, sustainable iodine prophylaxis [1, 2].

Universal salt iodization has been extremely effective at reducing the burden of IDD and represents a major global public health success [3]. In Nigeria, great progress has been made towards the elimination of iodine deficiency. This has been greatly achieved through the concerted efforts of various agencies including the National Agency for Food, Drug and Administration Control (NAFDAC) and the Standards Organization of Nigeria (SON) [4]

Iodine Deficiency Disorder (IDD) is a major global cause of morbidity, mortality and impaired development [1, 2, 3, 4]. As far back as 1967, Ekpechi alerted the Federal government of Nigeria of the existence of IDD in the country [5]. The studies of Isichie et al [6] led to the production of goiter map for Nigeria of which Saki, a city in South-Western Nigeria was inclusive. In 1998, Ojule and Osotimehin [7] reported that iodine deficiency was still present in Saki. Egbuta et al [8] conducted a study to evaluate the urinary iodine level in some of the local governments within the goiter belt region of Nigeria. Their report showed that of all the local governments studied, only data from Saki

showed lower median urinary iodine concentration. This implies that iodine deficiency was still present in that population.

Urinary iodine estimation has been widely applied as an indicator to measure iodine status of a given population. This is because, with normal renal function, the amount of iodine excreted in urine is an index for measuring iodine nutrition.

The aim of the study was to ascertain the current iodine status of children and population in Saki. The study hypothesis was that current government efforts at improving iodine nutrition in Nigerian citizens have yielded improved results in Saki population.

MATERIALS AND METHOD

Selection of patients and sample collection: This study was a public health study. In sample selection, the systematic sampling method as recommended by World Health Organization (WHO), (2007) was adopted. Subjects comprised of 280 apparently health primary school children from Saki. Their ages ranged between 8-12 years and comprised both males and females. The subjects had been resident in Saki for at least five years. Primary school children are appropriate population group for the assessment of iodine status because of their physiological vulnerability. Furthermore, measurement of urinary iodine level in school children is important for public health considerations, as this group effectively reflects the current status of IDD in the general population, as well as the extent to which IDD control measures have had an impact on the population. All the pupils were in their early primary school years.

Ethical clearance: In spite of obtaining the appropriate ethical clearance for the study, prior to sampling, consent was received from the school authorities, the parents and the pupils.

Sample collection:

10ml random urine sample was collected into clean and sterile universal bottle from all the pupils who were selected for the study using the systematic sampling method. Their anthropometric measurements were taken using standard methods. Since the samples were not analyzed immediately, it was stored frozen at -20°C until ready for analysis.

Analytical method: The standard method, the ammonium persulphate technique was used for estimating the level of iodine in the urine. Urine is digested with ammonium persulphate. Iodine present in the urine acts like a catalyst in the reduction of ceric ammonium sulphate (yellow) to cerous ammonium sulphate (colourless). The degree of disappearance of the yellow colour is a measure of iodine content in the urine. A standard curve plotted during the analysis was used to extrapolate the concentration of iodine in the urine samples.

RESULTS

TABLE 1: Measured parameters in the study population

Variables	Mean ± SD
AGE (yrs)	9.23 ±1.28
Height (m)	1.24±0.09
Weight (kg)	21.82± 4.46
Waist Circumference (m)	0.58±0.05
*MUI (µg/l)	284.75±114.94

*Medium Urinary Iodine.

Table 2: Classification of Iodine Nutrition of the Studied Population based on the Epidemiological Criteria for Assessing Iodine Nutrition Using Joint Criteria of WHO, UNICEF and ICCIDD (2001)

Range (µg/l)	% distribution
Severe (< 20)	0.7% (2)
Moderate (20-49)	3.2% (9)
Mild (50-99)	5 % (14)
Sufficient (100-199)	41.1 (115)
Excess (> 300)	50 (140)

DISCUSSION

Urinary iodine excretion is a good marker of the dietary intake of iodine, and is the index for evaluating the degree of iodine deficiency, correction and toxicity [1,2]. Many countries including Nigeria have adopted massive salt iodization as a means of correcting IDD in countries where they were prevalent [4].

Saki, a south-western Nigeria has been classified from previous studies as a goiter region in Nigeria with known environmental iodine deficiency. Over the recent times, studies have not been conducted to evaluate the present iodine status of its inhabitants.

Egbuta *et al* [8] conducted a study to evaluate the urinary iodine level in some of the local governments within the goiter belt region of Nigeria. Their report showed that of all the local governments studied; only data from Saki showed lower urinary iodine concentration. This lower urine iodine concentration was attributed to the numerous supply of non-iodized salts to the area by unscrupulous traders. From our study, it was evident that the median urinary iodine excretion in the population studied was 284.75µg/l, with greater percentage falling into the sufficient group as recommended by WHO. This shows that iodine nutrition has greatly improved in the area. Prior to the report of Egbuta *et al* [8], several government agencies saddled with the responsibility of improving iodine nutrition in the country were not active. It is not impossible that the inactivity of the agencies, created porous ports and weak environments, which made room available for the importation of non-iodized salt by non-patriotic business men. No doubt, result from this study has shown that concerted efforts by respective government agencies have improved the supply of iodized salt to the area contrarily to previous reports.

Of interest in this study is the presence of mild to moderate iodine deficiency in Saki. Previous reports have shown that the prevalence of IDD in Saki was 36% in 1993 and 16.2 % in 1998 [8]. It is very evident that with the current efforts of government towards the supply of iodized salt, the prevalence could be lower and this could be attested to by a very low percentage of iodine deficiency found in our study.

Furthermore, it may also not be impossible that only a hand full of all households' purchase iodized salt for domestic use. This however needs to be evaluated through further study.

The contribution of iodized table salt makes to iodine nutrition is probably insignificant [9,10]. Most of the salt in our diet comes from salt added in the preparation and processing of food. The assessment, monitoring and evaluation of the iodine content of salts imported and those produced within the country is currently poor. It is not unlikely that household salt may not contain the recommended level of iodine.

Reports have shown that iodine deficiency is not the sole cause of endemic goiter [10]. Indeed, the disease has been found in regions where there is no iodine deficiency. Conversely, in other regions with an extremely severe iodine deficiency, endemic goitre is not observed [11,12,13]. That iodine deficiency is still present in Saki several years after the introduction of iodized salt could show that several other environmental factors could contribute to iodine deficiency. Previous finding have also shown that some environmental goitrogenic factors in the diet or environment, other than iodine, could play a critical role in the aetiology of the disease [14]. Natural goitrogens were first found in vegetables of the genus *Brassica* (the *Cruciferae* family), which possesses goitrogenic properties in animals [15]. Their antithyroid action is related to the presence of thioglucosides, which, after digestion, release thiocyanate and isothiocyanate. Another important group of naturally occurring goitrogens is the cyanoglucosides, which have been found in several staples (cassava, maize, bamboo shoots, sweet potatoes, lima beans) [16,17,18]. After ingestion, these glucosides release cyanide, which is detoxified by conversion to thiocyanate, a powerful goitrogenic agent that inhibits thyroid iodide transport and, at higher doses, competes with iodide in organification processes. Saki, a popular city in South Western Nigeria is well known for the growth and production of sweet potato, maize and cassava products. These produce contribute to the greater percentage of the meal consumed in the region. It is therefore not unlikely that their ingestion could produce by products which could interfere with the body's need for iodine, thus reducing urinary iodine.

From this study, a greater percentage of the population, 50% (150) had urinary iodine concentration within the excess level. This is consistent with the findings of Delange and Lantun [19,20] who reported high concentrations of urinary iodine in some African countries few years after the introduction of massive iodization programme. It is therefore not impossible that the massive

iodization programme embarked by the government has exposed residents to higher concentrations of iodine.

Furthermore, the daily recommended allowance of iodine for children is about 150µg. Large quantities of iodide are present in drugs, antiseptics, bread, food preservatives and some fast food products. Considering the flair that school children have for bread and fast foods, it is likely that these food products could contribute to the increased level of iodine observed. Further reasons that could be responsible for the high urinary iodine level observed could be due to poor monitoring of the production, quantity and quality of iodine used for food production and preservation. In most instances, the monitoring of iodized salt within this environment only focuses mainly on whether iodine is present or not but the quantity is not always considered.

The World Health Organization has reported that population exposed to excess concentrations of urinary iodine could be prone to developing Iodine Induced Hyperthyroidism (IIH) and Iodine Induced Thyroiditis (IIT) [20,21]. The thyroid gland has intrinsic mechanisms that maintain normal thyroid function even in the presence of iodine excess; however this mechanism can be depleted at increased iodine levels [22]. It has widely been reported that administration of iodine in any chemical form could induce the development of IIH and IIT in populations previously deficient in iodine [23,24]. The presence of IIH and IIT needs to be investigated in Saki.

CONCLUSION

This study showed that high urinary iodine level is present in the studied population and it has public health implication. Despite remarkable progress in the control of the iodine deficiency disorders (IDD), it remains a significant global public health problem. Assessing the control of IDD and preventing the development of IIH and IIT, monitoring the progress of salt iodization programmes are cornerstones of a control strategy. It is recommended that government and government agencies regularly monitor the quality and quantity of iodized salt present in the country. Further and elaborate studies should also investigate the presence of goitrogens, IIH and IIT in Saki population.

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