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Full Length Research Paper

Assessment of current iodine status of pregnant women in a suburban area of Imo State Nigeria, twelve years after universal salt iodization

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Three hundred and two pregnant women participated in this study. Our results showed that the range, mean and median urinary iodine excretion (UIE) were 28.1 to 218.1, 152.09 ± 41.65 and 163.1 µg/l, respectively. The range, mean and median TSH concentration were 0.7 - 5.9, 1.4 ± 0.7 and 1.3 µIU/ml, respectively. Our results showed that none of the women have severe iodine deficiency, 2% had moderate iodine deficiency, 12% had mild iodine deficiency, while 80% had optimal iodine nutrition and 6% have more than adequate. We observed a progressive and significant (P = 0.0009) decrease in the mean UIE from the 1st to the 3rd trimester. We observed also that 95% of the pregnant women had TSH concentration within the normal range. The TSH values between the three trimesters showed no significant difference (P = 1.20). The Urinary Iodine Excretion and Thyroid Stimulating Hormone concentration values suggest that iodine deficiency has been eliminated as a public health problem in Orlu. The progressive decrease in the median UIE from the 1st to the 3rd trimester should be addressed to meet the increased demand of iodine as a result of the pregnancy.

Key words: Urinary iodine, thyroid stimulating hormone, iodine deficiency, hyperthyroidism, pregnant women, Orlu, Nigeria.

INTRODUCTION

Iodine is an essential trace element necessary for the synthesis of thyroid hormones (Delange, 1994; Hetzel and Maberly, 1989). These hormones promote growth and development of bone, muscle, height and weight and maintain the stabilization of energy and material metabolism (Fuge and Johnson, 1986). The thyroid hormones are also vital for growth and development of all organs especially the brain, reproductive organs, nerves, skins, nails and teeth (Fisher and Delange, 1998). Deficiency of iodine resulting from inadequate dietary intake is related to a spectrum of diseases collectively known as iodine deficiency disorders (IDD) (Hetzel, 1983). IDD can be corrected by re-supplying iodine in the diet (Delange, 2000). The impact of IDD is enormous and it affects all the stages of life (Hetzel, 1983; ICCIDD/UNICEF/WHO, 2001). Iodine deficiency disorders are primarily the result of inadequate amounts of iodine in soil, water and food as well as consumption of foods rich in goitrogenic substances (Aston and Brazier, 1979; Sharma et al., 1999; Ene-Obong, 2001). Iodine deficiency in the fetus is the result of iodine deficiency in the mother. The consequence of iodine deficiency during pregnancy is impaired synthesis of thyroid hormones by the mother and the fetus. An insufficient supply of thyroid hormones to the developing brain may result in mental retardation (Morreale et al., 2004; Auso et al., 2004; Koibuchi and Chin, 2000; Delange, 2001). It has been established through some experimental evidence that the varying manifestation of IDD in fetus could be as a result of low thyroxine level in the blood of the iodine deficient mother and the lower the level of thyroid hormone of the pregnant women, the greater the threat to the fetus development (WHO, 1996). These children also have a greater occurrence of congenital abnormalities, lower birth weight and lower mortality rate as indicated by higher perinatal and infant mortality (US Foods and Nutrition Board, 2001). The commonest manifestation of iodine deficiency is goitre, which occurs when the iodine level of the blood is low; the cells of the thyroid gland enlarge in an attempt to trap as many particles of iodine

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as possible. Sometimes the gland enlarges until it is visible (Hamilton et al., 1998; Chatterjea and Rana, 2004). This gland enlargement is caused by an increased production of thyroid stimulating hormone (TSH) (Ubom, 1991).

Inadequate dietary iodine leads to reduced synthesis of thyroid hormones (T3 and T4). Lower level of T4 in the blood stimulates the pituitary gland to secrete TSH to fulfill the production of thyroid gland hormones. It is important not to over consume iodine as it has a relatively narrow range of intakes that reliably support good thyroid function. Consumption of an excessive amount of iodized salt or seaweeds could readily result to complex disrupt-tive effect on the thyroid and may cause either hyperthy-roidism or hypothyroidism in susceptible individuals, as well increasing the risk of thyroid cancer. A large percentage of the world population is at a risk of IDD (Delange and Hetzel, 2003). Several parts of Nigeria have been before now identified with goiter endemicity and hence labeled the "goitre belt" (Nwokolo and Ekpechi, 1966; Olurin, 1975; Isichie et al., 1987; Ubom, 1991). In 1993 a national goitre rate of 20% was reported and 20 million Nigerians were estimated to be affected by IDD (UNICEF, 1993). The Participatory Information Collection Study (1993), using thyroid hormone concen-trations as indicators of iodine status reported an iodine deficiency prevalence of 65.6% in South-East, 41% in the South-West, 43% in the North- West of Nigeria. As part of the strategies to reduce the prevalence of IDD in Nigeria, the Universal Salt Iodization (USI) Programme was introduced in 1995. The update from the report of the Nigeria Demographic and Health Survey (NDHS, 2003) showed that almost all Nigerian households (97.3%) consumed adequately iodized salt, while about 1.7% consumed uniodized salt. This study was used to eva-luate the iodine nutrition in Orlu suburban area of Nigeria using pregnant women as a case study after several years of availability and consumption of iodized salt.

MATERIALS AND METHODS

Experimental design

This study is a hospital based study conducted amongst pregnant women attending the antenatal clinic of Imo State University Teaching Hospital, a government hospital, in Orlu senatorial district of Imo State South-East, Nigeria. Three hundred and two pregnant women (mean age 33 years) participated in the study. All subjects were volunteers and were selected from among pregnant women visiting the antenatal clinic. All subjects provided written informed consent in accordance with the ethical standards of the local ethical committee. Selection criteria were the absence of chronic disease such as thyroid disease, diabetes mellitus, anemia, hypertension and coronary artery. Casual urine samples were obtained from the three hundred and two pregnant women, labelled and immediately preserved in a cooler with ice chips. Blood samples were collected from two hundred and nine of the women in standardized conditions to reduce sources of pre-analytical variation. Venipuncture was performed after an overnight fast. All blood samples were collected by experienced medical laboratory scientists using conventional venipuncture. The blood samples were allowed to clot before separation by centrifugation at 3000 g for 15 min. All serum samples were stored frozen at refrigerated until testing at the end of the collection period. The urine and blood samples obtained from the women were used for the determination of urinary iodine excretion (UIE) and thyroid stimulating hormone (TSH).

Determination of urinary iodine excretion

Measurement of urinary iodine is the most common method to monitor dietary iodine intake (Fray et al., 1973). This makes urinary iodine a good biochemical marker for control of iodine deficiency disorders. The iodine in the urine is measured by a modification of the traditional colorimetric method of Sandell and Kolthoff (1937). This was done using the Ammonium Persulfate Method as described by Pino et al. (1996). Urine was digested with ammonium persulphate. The iodine in the urine samples catalyses the reduction of ceric ammonium sulphate (yellow colour) to the cerous form (colourless) in the presence of arsenious acid. The degree of reduction in colour intensity of the yellow ceric ammonium sulphate is proportional to the iodine content in the urine sample. This method was applied to all urine samples.

Measurement of the TSH serum concentration

Serum TSH concentration was measured by enzyme-linked immunosorbent assay using commercial kits (Syntron Bioresearch, Inc. Carlsbad, CA - USA). The normal range of TSH concentration determined with this kit was 0.5 - 4.10.

Statistical analysis

The data obtained were subjected to statistical analysis using the statistical software package, Statistical Analysis Software (SAS). The mean, Median and range of the data was determined. Results will be considered significant when $P<0.05$ at 95% confidence.

RESULTS

Urinary iodine excretion (UIE)

Urinary iodine excretion (UIE) of 302 pregnant women was used to monitor the effect of Universal Salt Iodization in Nigeria. The results obtained from the 1st to the 3rd trimester showed that the mean and median UIE were 152.09 ± 41.65 and 163.1 µg/l, respectively. The UIE ranged from 28.1 to 218.1 µg/l. The results showed that none of the women have severe (<20 µg/l) iodine deficiency, 5 (2%) have moderate (20 - 49 µg/l) iodine deficiency, 37 (12%) have mild (50 - 99 µg/l) iodine deficiency, while 242 (80%) have optimal (100 - 199 µg/l) iodine nutrition range and 18 (6%) have more than adequate (200 - 299). The UIE for the three trimesters varied significantly ($P = 0.0009$). The UIE between the 1st trimester and the 2nd trimester varied slightly ($P = 0.046$), while UIE between the 1st and 3rd trimester varied significantly ($P = 0.0001$). There was no significant difference between UIE in the 2nd and 3rd trimester ($P = 0.06$). From the result, a progressive decrease in the

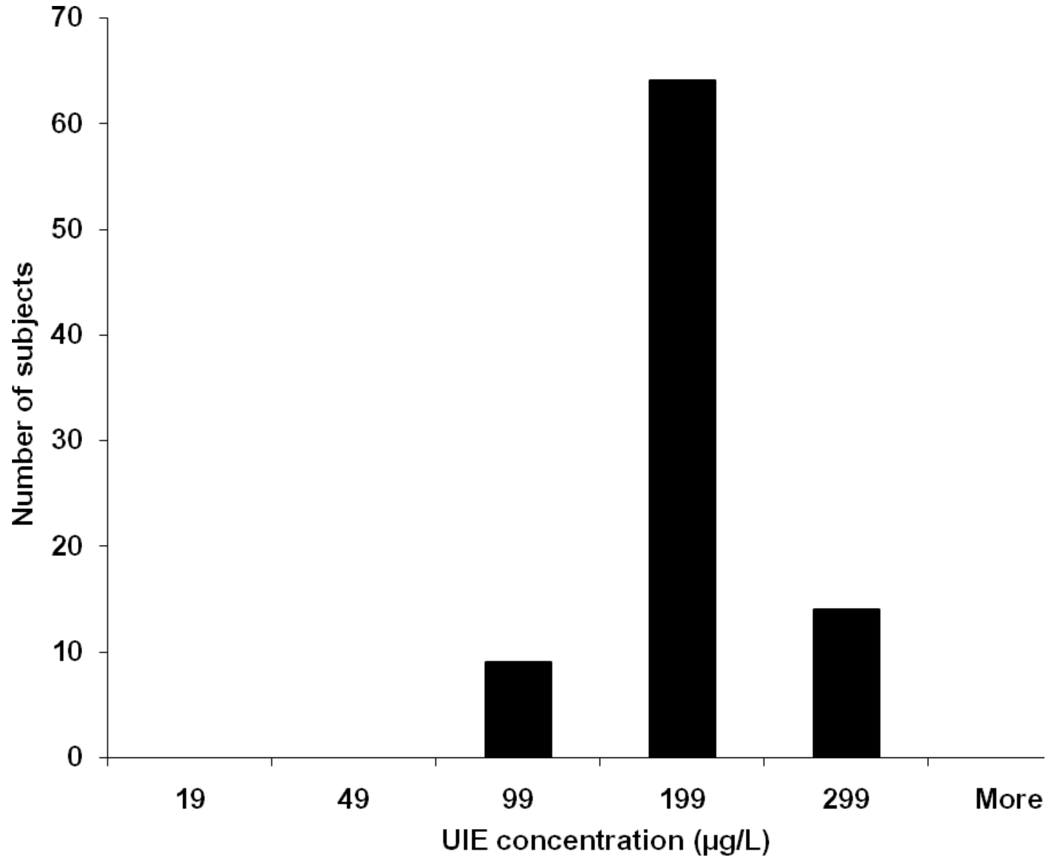

Figure 1. Distribution of UIE concentration values of 87 pregnant women in 1st trimester of pregnancy.

mean UIE from the 1st to the 3rd trimester was observed and the decrease was significant (P = 0.0009). The results obtained from each trimester are presented in Figures 1, 2 and 3.

The result of UIE of 87 pregnant women in their first trimester is shown in Figure 1. The result showed that none of the women have severe iodine deficiency but 9 (10%) have mild iodine deficiency, 64 (74%) have optimal iodine nutrition and 14 (16%) have more than adequate. The results from the pregnant women in the first trimester gave a mean UIE of 164.22 ± 40.56 µg/l, median UIE of 173.1 µg/l and the UIE ranged from 53.1 to 218.1 µg/l.

The result UIE of 112 pregnant women in their 2nd trimester is shown in Figure 2. The results showed that none of the women have severe iodine deficiency, 2 (2%) have moderate iodine deficiency, 17 (15%) have mild iodine deficiency. The result also showed that, 90 (80%) have optimal iodine nutrition and 3 (3%) have more than adequate. From the result, the mean UIE was 152.22 ± 42.80 µg/l, median UIE was 170.6 µg/l. The UIE ranged from 48.1 to 205.6 µg/l.

The result of UIE of 103 pregnant women in their 3rd trimester is shown in Figure 3. The results showed that none of the women have severe iodine deficiency, 3 (3%) have moderate iodine deficiency. Also, 11 (11%) have mild iodine deficiency, 88 (85%) have optimal iodine nutrition and 1 (1%) have more than adequate. From the result, the mean UIE was 141.69 ± 38.78 µg/l, median UIE was 150.6 µg/l. The UIE ranged from 28.1 to 200.6 µg/l.

Thyroid Stimulating Hormone

The result of TSH concentration measurements, of 58 pregnant women in their 1st trimester of pregnancy are showed in Figure 4. The result showed that 7 (12 %) of the pregnant women had TSH values between 0.0 to 0.5 µIU/ml (Hyperthyroid status). A total of 50 (86%) of the women had TSH level between 0.6 to 4.10 µIU/ml (normal thyroid status). Also the result showed that 1(2%) of the pregnant women had TSH value > 4.10 µIU/ml (Hypothyroid). From the result, the mean and median TSH concentration values were 1.11 ± 0.76 and 1.07 µIU/ml, respectively and the TSH values ranged from 0.39 to 6.00 µIU/ml.

The result of TSH concentration measurements, of 82 pregnant women in their 2nd trimester of pregnancy are showed in Figure 5. The result showed that 2(2%) of the pregnant women had TSH values between 0.0 to 0.5 µIU/ml (Hyperthyroid status). A total of 80 (98%) of the pregnant women had TSH level between 0.6 to 4.10 µIU/ml

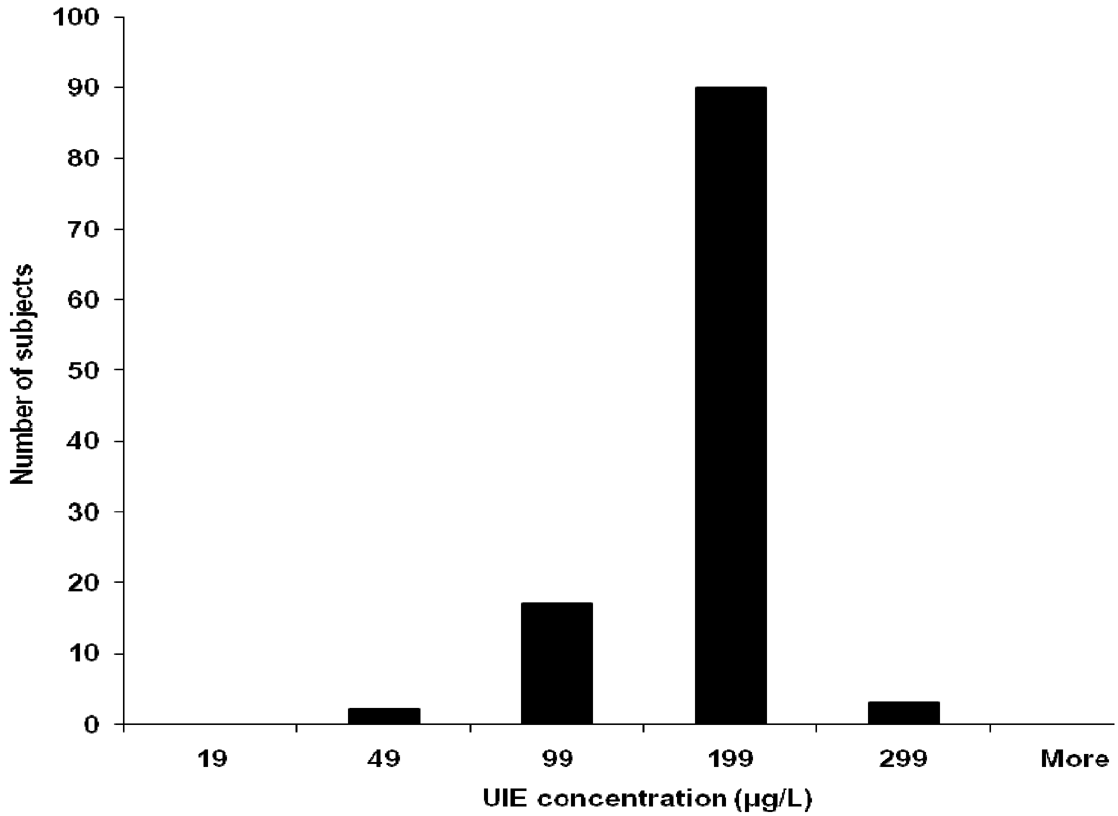

Figure 2. Distribution of UIE concentration values of 112 pregnant women in 2nd trimester of pregnancy.

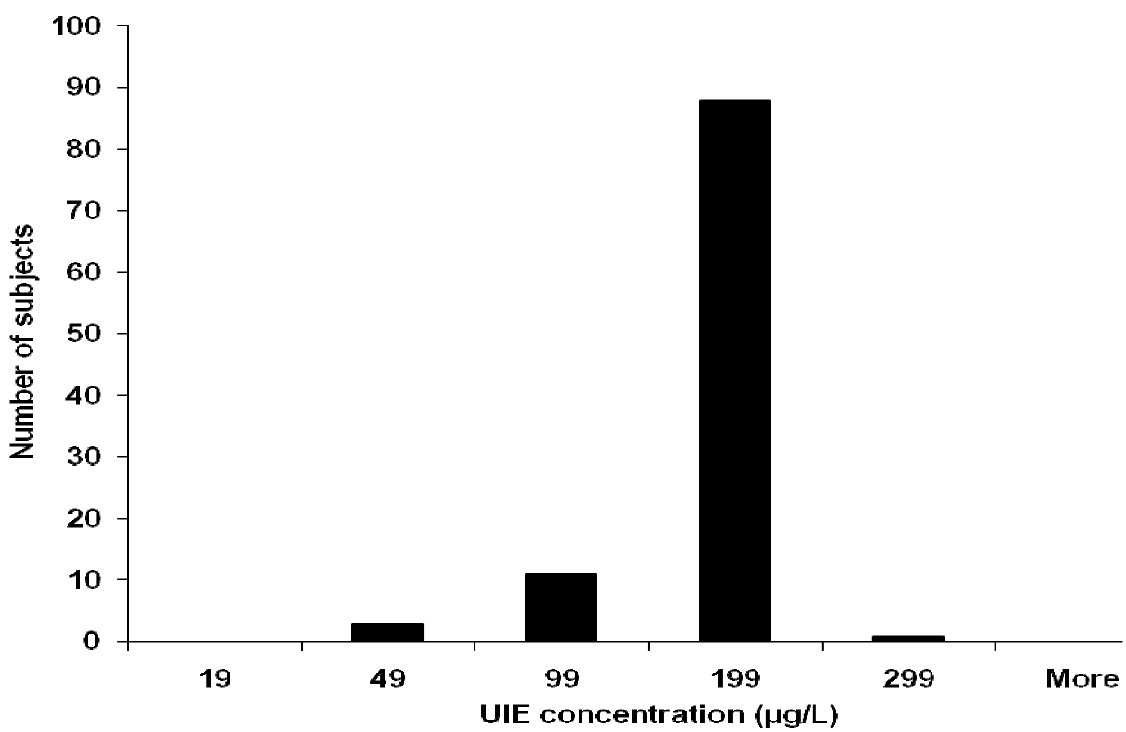

Figure 3. Distribution of UIE concentration values of 103 pregnant women in 3rd trimester of pregnancy.

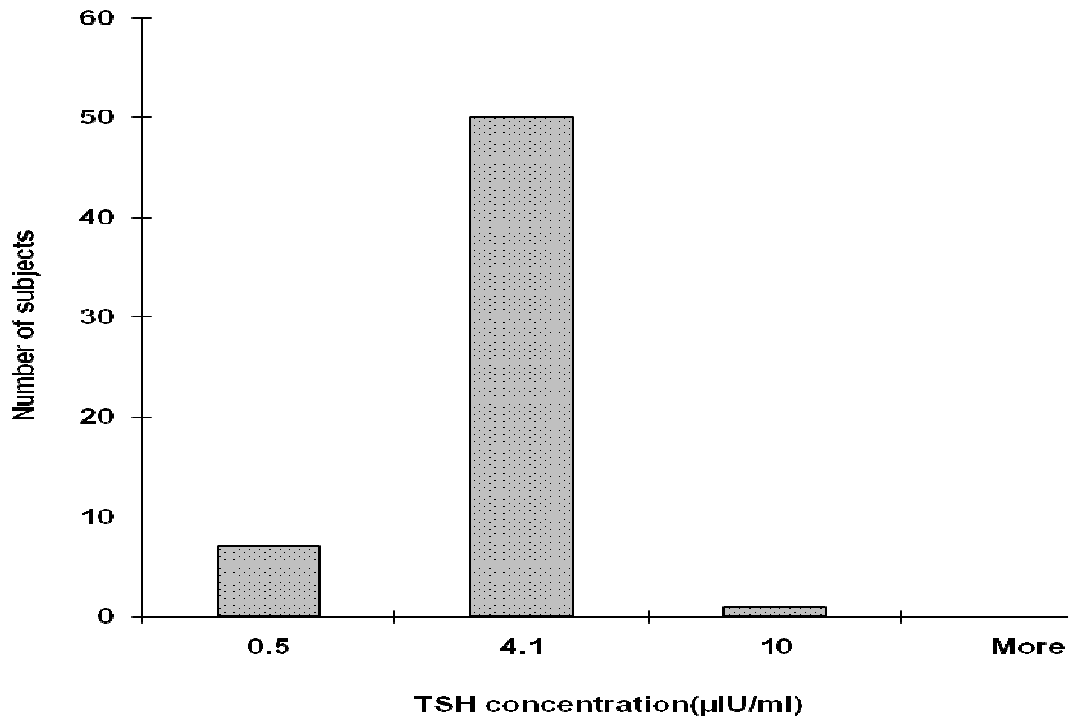

Figure 4. TSH concentration distribution of 58 pregnant women in 1st trimester of pregnancy.

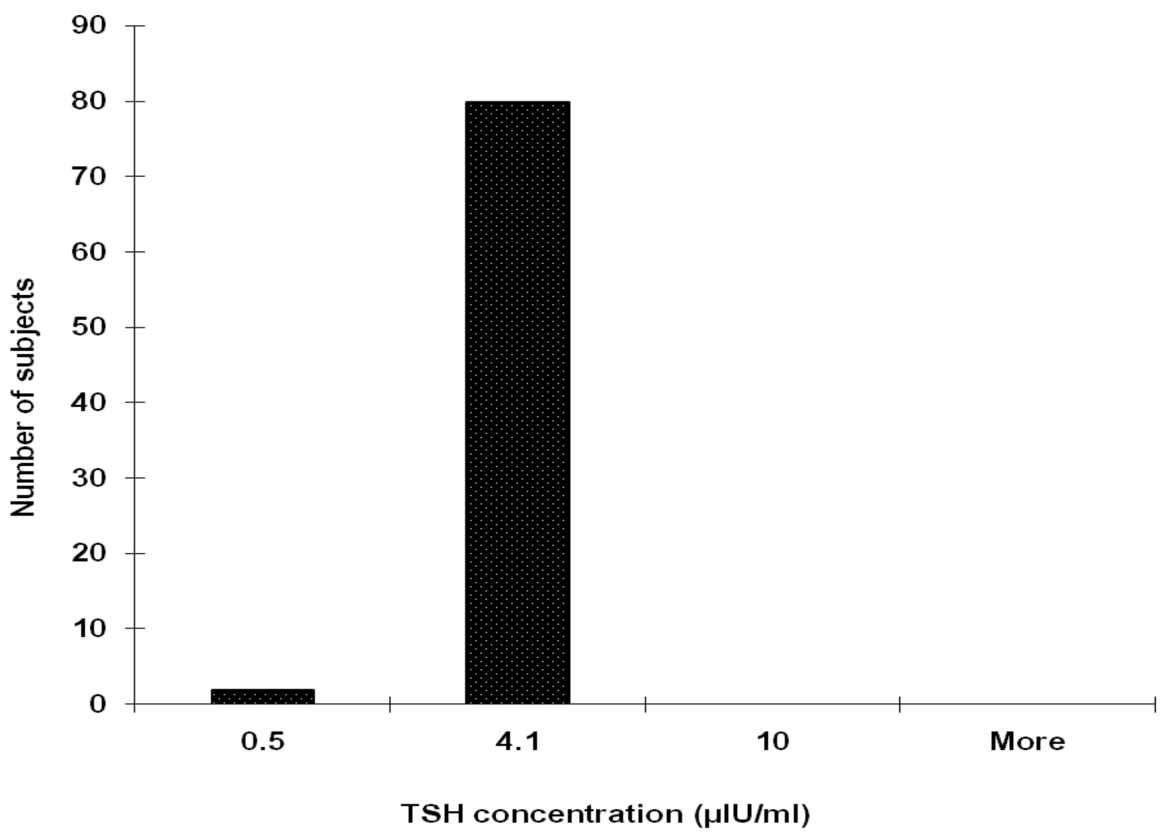

Figure 5. TSH concentration distribution of 82 pregnant women in 2nd trimester of pregnancy.

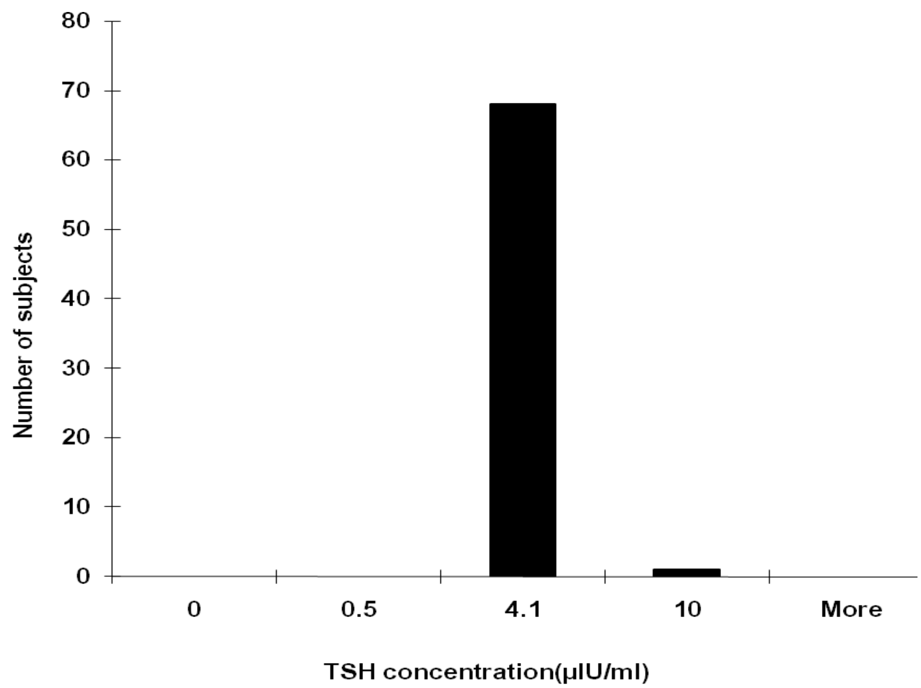

Figure 6. TSH concentration distribution of 69 pregnant women in 3rd trimester of pregnancy.

(normal thyroid status). The result also showed that none of the pregnant women had TSH value > 4.10 µIU/ml (Hypothyroid condition). From the result, the mean and median TSH concentration values were 1.39 ± 0.53 and 1.39 µIU/ml, respectively. The TSH values ranged from 0.47 to 3.09 µIU/ml.

The result of TSH concentration measurements, of 69 pregnant women in their 3rd trimester of pregnancy are showed in Figure 6. The result showed that none of the pregnant women had TSH values between 0.0 to 0.5 µIU/ml (Hyperthyroid status), while 68 (99%) of the pregnant women had TSH level between 0.6 to 4.10 µIU/ml (normal thyroid status). The result also showed that 1(1%) of the pregnant women have TSH value > 4.10 µIU/ml (Hypothyroid condition). From the result, the mean and median TSH concentration values were 1.7 ± 0.8 and 1.62 µIU/ml, respectively. The TSH values ranged from 0.78 to 5.92 µIU/ml

DISCUSSION

The results obtained showed a progressive decrease in the median UIE from the first to the third trimester. This decrease can be attributed to the increased demand of iodine as a result of the pregnancy (Aboul-Khair et al., 1964). Increased demand for micronutrients predisposes women of reproductive age, pregnant and lactating women to micronutrient deficiencies (McGuire, 1993; ICCCIDD/UNICEF/WHO, 2001). This result also suggests that the quantity of iodized salt consumed is not meeting the increased demand for iodine. Increased iodine requirement during pregnancy is to provide for the needs of the fetus and to compensate for increased loss of iodine. It has being reported that intake between 200 - 250 µg/day, for pregnant women is very important to prevent goiter development and to keep serum levels of T4 and T3 stable (ICCIDD/WHO/UNICEF, 2001; Glinoer et al., 1995). The UIE value results showed that 80% of the pregnant women had optimal iodine nutrition. Also, our results showed that 5 (2%) of 302 pregnant women had moderate (20 - 49 µg/l) iodine deficiency and 37 (12%) had mild (50 - 99 µg/l) iodine deficiency. These results suggested that 42 pregnant women (14%) did not consume the necessary amount of iodine.

The results of TSH measurements showed that over 90% was observed to have serum TSH within normal suggesting the optimal iodine nutrition, in which most of them were was able to keep serum levels of T4 and T3 stable.

The number of the women (80%) with UIE values corresponding to the optimal nutrition is in line with the result of the TSH in which over 90% was observed to have serum TSH within normal, the normal range which suggests good thyroid function status and an adequate dietary intake of iodide. The results of the TSH for women consuming adequate iodine is in line with the requirement by WHO/UNICEF/ICCIDD (1994) and the report of Delange (1999). The result of the TSH showed that the mean concentration of the TSH in the three trimesters increased gradually as pregnancy progressed, however variation of the TSH concentration between the three trimesters was not significant (P = 1.20). The value of the mean serum TSH concentration of the pregnant women suggests that, they had normal thyroid function status. The result also showed that only 4% of the women had serum TSH concentration below normal (hyper-thyroidism), this justifies the 6% of women observed to have UIE values above optimal. These findings are in line with that obtained in Australia where lowest TSH was associated with UIE values of between 200 - 300 µg/l (Buchinger et al., 1996).

The results obtained in this study, in measuring USI effect on UIE and TSH concentration, of pregnant women indicate that iodine deficiency was eliminated as a public health problem in Orlu. Also, with these results and the results obtained in previous studies in measuring the effect of USI on iodide nutrition, Nigeria can be said to be succeeding in, mitigating the large-scale losses of brain-power and productivity caused by Iodine Deficiency Disorders. The challenge faced today by the Nigerian government, salt industries, the communities and other stakeholders in the IDD elimination drive will be to sustain these achievements and consolidate the gains of the universal salt iodization through certification and monitoring at all levels as a routine by all concerned.

REFERENCES

Aboul-Khair SA, Turnbull AC, Hytten FE (1964). The physiological changes in thyroid function during pregnancy. Clin. Sci. 27: 195-207.

Aston SR, Brazier PH (1979). Endemic goiter, the factors controlling iodine deficiency in soils. Sci. Total Environ. 11: 99-104.

Auso E, Lavado-Autric R, Cuevas E, Escbar del Rey F, Morreale de Escobar G, Berbel PA (2004). moderate and transient deficiency of maternal thyroid function at the beginning of fetal neocoticogenesis alters neuronal migration. Endocrinol., 145: 4037-4047.

Buchinger W, Lorenz-Wawschinek O, Binter G, Langsteger W, Bonelli R, Eber O (1996). Relation between serum thyrotropin and thyroglobulin with urinary iodine excretion. In: The Thyroid and Iodine. Nauman J, Glinoer D, Braverman LE, Hostalek U, eds. pp. 189-190. Stuttgart, Germany, Schattauer.

Chartterjea MM, Rana S (2004). Medical Biochemistry, 6th edition Oxford Press London pp. 700-750.

Delange F (1994). The disorders induced by iodine deficiency. Thyroids 4: 107.

Delange F (1999). Neonatal thyroid screening as a monitoring tool for the control of iodine deficiency. Acta. Padiat. Scand. Suppl. 432: 21-24.

Delange F (2000). Iodine deficiency. In The thyroid. A fundamental and clinical text. Braverman LE, Utiger RD, editors. Philadelphia: Lippincott publ. pp. 295-316.

Delange F (2001). Iodine deficiency as a cause of brain damage. Postgrad. Med. J. 77: 217-220.

Delange F, Hetzel BS (2003). The iodine deficiency disorders. In: The Thyroid and its Diseases. Basle karger pp. 324-44.

Ene-Obong HN (2001). Substances occurring in foods. In: Eating Right (Nutrition Guide). University of Calaber Press, Nigeria p. 48.

Fisher DA, Delange F (1998). Thyroid hormone and iodine requirements in man during brain development. In: Stanbury J.B., Delange F., Dunn J.T. and Pandav C.S. eds. Iodine in pregnancy. New Delhi, Oxford University Press pp. 1–33.

Fray HM, Rosenlund B, Torgersen JB (1973). Value of single urine specimens in estimation of 24 h urine iodine excretion. Acta. Endocrinol. 72: 287-92.

Fuge R, Johnson CC (1986). The geochemistry of iodine. Environ Geochem. Health, 8: 3l–54.

Glinoer D, de Nayer P, Delange F, Lemone M, Toppet V, Spehl M, Grün JP, Kinthaert J, Lejeune B (1995). A randomized trial for the treatment of mild iodine deficiency during pregnancy: Maternal and neonatal effects. J. Clin. Endocrin. Metab., 80: 258-269.

Hamilton EM, Whitney EN, Sizer FS (1998). Nutrition concepts and controversies. 5th edition west publishing company, Washington pp. 255 – 493.

Hetzel BS (1983). Iodine Deficiency Disorders (IDD) and their eradication. Lancet, 11: 1126-1129.

Hetzel BS (1989). The Story of Iodine Deficiency, 1st edition. Oxford Medical Publications, Great Britain p. 3.

Hetzel BS, Maberly GF (1986). Iodine. In: Mertz W, ed. Trace Elements in Human and Animal Nutrition, Vol. 2, 5th ed. New York: Academic Press pp.139–208.

ICCIDD/UCICEF/WHO (2001). Assessment of iodine deficiency disorders and monitoring their elimination. A guide for programme managers; WHO document WHO/NHD/01.1.

Isichie UP, Das SC, Egbuta JO, Banwo AI, Marimot I, Nagataki S (1987). Endemic goitre in plateau state, Nigeria; the possible aetiological factor and the establishment of endemic goitre map for the region. Proceedings of Nigeria/Japan Conferences, Jos pp. 78-81.

Koibuchi N, Chin WW (2000). Thyroid hormone action and brain development. Trends Endocrinol. Metab., 4: 123-128.

McGuire J (1993). Best practice in addressing micronutrient malnutrition. In: SCN News Geneva. No. 9.ACC/SCN pp. 1-10.

Morreale de Escobar G, Obregon M, Escobar del Rey F (2004). Role of thyroid hormone during early brain development. Eur. J. Endocrinol., 15: 25- 37.

Nigeria Demographic and Health Survey (2003). Infant feeding and children and women nutritional status. National Population Commission, Federal Republic of Nigeria pp. 160.

Nwokolo C, Ekpechi OL (1966). New foci of on the handling and storage of table salt. endemic goitre in Eastern Nigeria. Trans. Roy. Soc. Trop. Med. Hyg., 6: 97-108.

Olurin EO (1975). The fire of life (The Thyroid Gland). Inaugural lecture. University of Ibadan.

Pino S, Fang SL, Braverman LE (1996). Ammonium persulfate: A safe alternative oxidizing reagent for measuring urinary iodine .Clin. Chem. 42(2): 239-243

Sandell EB, Kolthoff I (1937). Micro determination of iodine by catalytic method. Mikrochim Acta, 1: 9-25.

Sharma SK, Chelleng PK, Gogoi S (1999). Iodine status of food and drinking water of a sub-Himalayan zone of India. Int. J. Food Sci. Nutr., 50: 95-98.

The Participatory Information Collection Study, 1993.

Ubom GA (1991). The Goitre-Soil-Water-Diet Relationship: Case Study in Plateau State, Nigeria. The Sci. the Total Environ. 107: 1-11.

UNICEF (1993). Regional goitre survey in school children, Nutrition Section, Lagos Nigeria.

US Food and Nutrition Board (2001). Dietary Reference Intakes. A report of the Institute of Medicine. pp 8-1 to 8-27, National Academy Press, Washington, DC.

WHO, UNICEF, ICCIDD (1994). Indicators for assessing Iodine Deficiency Disorders and their control through salt iodization. Geneva: WHO publ. WHO/NUT/94.6 pp. 1-55.

WHO (World Health Organisation) (1996). Trace elements in human nutrition and health. WHO, Geneva.