

# EFFECTS AND PREVENTION OF IODINE DEFICIENCY

*Okoroigwe, F. C.*

## **Abstract**

Iodine, an essential trace element, is an essential constituent of the thyroid hormones thyroxine (tetraiodothyronine (T<sub>4</sub>) and triiodothyronine (T<sub>3</sub>). The thyroid hormones play an important part in growth and general development of man and animals. These hormones are involved in regulation of metabolic activities of all cells throughout life cycle. Iodine deficiency is a public health problem included among the "hidden hunger". Globally, about 740 million people are affected by goitre and more than 2 billion are estimated to be at risk of iodine deficiency disorders (IDD). The causes include deficiency of iodine, local environment, and excessive intake of goitrogens. Iodine deficiency leads to a spectrum of disorders IDD, ranging from abortions, still birth, increased infant and perinatal mortality cretinism to goiter and impaired mental functioning. IDD is preventable. The prevention and control approaches include supplementations, dietary diversification and economic improvement. These have been proved to be safe, inexpensive, and readily replicable.

## **Introduction**

Iodine is an essential constituent of the thyroid hormones, thyroxine (3, 5, 3,5- tetraiodothyronine (T<sub>4</sub>) and 3, 5, 3- triiodothyronine (T<sub>3</sub>). The major role of iodine in nutrition arises from the important part played by the thyroid hormones in the growth and development of humans and animals. The thyroid hormones are essential for the regulation of metabolic activities of all cells throughout the life cycle.

Iodine plays a key role in cell replication. This is particularly relevant for the brain since neural cells multiply mainly in utero and during the first two years of life. In other words, iodine is required to ensure normal growth, especially of the brain, which occurs from foetal life to the end of the third postnatal year.

Consumption of goitrogens as well as foods grown in iodine deficient soil, are some of the causes of iodine deficiency. The term "goitre" has been used for many years to describe the effect of iodine deficiency. Although there is no doubt that goitre is indeed the visually obvious and familiar feature of iodine deficiency, understanding of the other consequences of iodine deficiency has expanded to the discovery of wider and more appropriate spectrum of disorder denoted by the term iodine deficiency disorder designated 'IDD'. IDD comprises all the effects of iodine deficiency. Iodine-deficient populations suffer a variety of consequences that include goitre, reduced mental function, widespread lethargy, and increased rates of still births and infant mortality.

Iodine deficiency, though preventable, is a public health problem included among the "hidden hunger". Every country in Africa has IDD. Globally, about 740 million people are affected by goitre, and more than 2 billion (or over 38% of the population living in 130 countries are estimated to be at risk of 11)1). (WIIO/UNICEF/ICCIDD, 199').

## **Causes**

The principal cause of iodine deficiency is a low content of iodine in the local environment. Therefore, iodine-deficient soil, water and food serve as determining factors (ACC/SCN, 1991). Iodine deficiency disorders were found mainly in areas where people consumed foods grown locally in soil depleted of its natural iodine content. Hence IDD are most common in mountainous areas: the entire Andean chain is a major endemic region (ACC/SCN, 1988).

The best-known iodine deficient areas are mountainous-regions, especially the Andes and Himalayas (ACC/SCN, 1987). However coastal areas and plains and areas where repeated flooding occurs, may also be deficient in iodine. Proportion of the population in these areas may be affected by inadequacy of dietary iodine, because the foods grown in these areas are deficient in iodine.

Goitrogens are found in members of cabbage family like cauliflower, cabbage, Brussels, sprouts, turnips, rutabaga, (cooking may inactivate the goitrogenic factor in these), milk from cows that have eaten plants containing goitrogens. Goitrogen is also found in raw soybean but it is rendered less toxic by heating.

Excessive intakes of goitrogens are another important cause of Iodine deficiency.

### **Effects**

The disorders induced by dietary iodine deficiency (IDD) constitute a major global nutrition concern. A broad spectrum of iodine deficiency disorders is made up of mental and physiological effects, which include stillbirths, congenital abnormalities and increased infant deaths (ACC/SCN, 1991). In fact, the spectrum of disorders range from abortions, stillbirth, increased infant and prenatal mortality, neurological cretinism to goiter and impaired mental functioning in adolescents and adults (ACC/SCN, 1988). The greatest risk from iodine deficiency has been found to be during brain development. Iodine deficiency is the world's leading cause of preventable brain damage.

### **Fetal Iodine Deficiency**

Iodine deficiency in fetus is as result of iodine deficiency in mother. This is associated with an increased incidence of stillbirths, abortions and congenital abnormalities, all of which can be avoided by appropriate intervention. Endemic cretinism is a major effect of fetal iodine deficiency. It is characterized by mental deficiency, deaf mutism, and spastic diplegia. This is called "nervous" or neurological type in contrast to "myxedematous" type characterized by hypothyroidism and dwarfism. Individuals affected are called "cretinoid" in China (WHO, 1996.) Low thyroid at crucial development stages (including vulnerability of the foetus to maternal hypothyroidism starting soon after conception) causes irreversible brain damage at its extreme expressed as cretinism and deaf-mutism (Hetzl, 1993).

### **Neonatal Iodine Deficiency**

Iodine deficiency leads to increase in neonatal mortality. Thyroid hormone is essential for normal brain development. Severe iodine deficiency affects neonatal thyroid function and as a result constitutes a threat to early brain development. In neonates, iodine deficiency leads to hypothyroidism, cretinism with severe retarded physical and mental development.

### **Iodine Deficiency in Children and Adolescents**

Iodine deficiency in these periods is characteristically associated with goiter, the goiter rate increases with age, reaching a maximum at adolescence. Goiter rates in school children over the age range 6 - 12 years provide a convenient indicator of the presence of iodine deficiency in a community. Studies using school children have indicated impaired school performance and I.Qs in those living in iodine deficient areas even when the effect of other factors like social deprivation, and other nutritional factors have been taken into account (WHO,1996). IDD causes reduction of up to 10 to 15 I.Q points.

### **Iodine Deficiency in Adults**

The common result of iodine deficiency in adults is goiter. High degree of apathy has been noted in populations living in iodine deficient areas which may even affect domestic animals such as dogs. It is apparent that reduced mental function is widely prevalent in iodine deficient communities, with consequent effects on their capacity for taking the initiative and for decision-making. This shows that iodine deficiency is a major obstacle to the human and social development of communities living in an iodine deficient environment. A typical example to this is a Chinese village (before iodization) where in 1978 there were many cretins which caused the village to be known as "the village of idiots" (WHO, 1996). Severe hypothyroidism during pregnancy results in cretinism in the new born cretinism is a congenital condition (present at birth). The acquired form of this disease, which occurs in older children and adults, is called myxedema.

### **Iodine Deficiency in Animals**

Iodine deficiency in animals results in reproductive and neurological defects. In area of iodine deficiency, the development of the fetus has been retarded or arrested at some stage in gestation resulting in an early death or re-absorption, abortion and still birth or the birth of weak hairier offspring, associated with prolonged gestation and parturition and retention of placental membranes Subnormal thyroid hormone levels in herds of cattle have been accompanied by a high incidence of aborted stillborn and weak calves (Helzel and Maberly, 1986). Iodine deficiency has early effect on neuroblast multiplications that could be important in the pathogenesis of the neurologic-form of endemic cretinism (Hetzl et al, 1988).

### **Goiter**

Iodine deficiency is generally associated with its most visible manifestation, goiter, an enlargement of the thyroid once regarded as a problem with few implications for general health. Iodine deficiency depletes thyroid iodine stores and reduces daily production of thyroxine (T<sub>4</sub>). A fall in the blood level of T<sub>4</sub> triggers the secretion of increased amount of thyroid stimulating hormone (TSH) which increases thyroid activity with consequent hyperplasia of the thyroid. The efficiency of the thyroid iodine pump is increased accompanied by faster turnover of thyroid iodine and enlargement of cells of thyroid follicles as they multiply. The enlargement of the thyroid gland is due to its ability to trap as many particles of iodine as possible and it is this enlargement that gives rise to goiter.

During the initial stages of the adaptation, the goiter is only detectable by palpation. But as the condition progresses, the gland is easily visible. In some cases, goiter may compress the windpipe and produce choking and difficulty in swallowing while in others, it may be asymptomatic. Goiter may be accompanied by more serious manifestations and complications like cancer. Goiter is endemic when its prevalence rate exceeds 10% in a given region. Endemic cretinism is associated with endemic goiter and severe iodine deficiency.

### **Socio-Economic Effect**

Iodine deficiency, causing retardation of intellectual development and lethargy, may have crucial effects on productivity and human welfare in some of the poorest regions of the world. The knowledge of the impact of iodine deficiency on intellectual development and the resulting costs to societies, including delayed socio-economic development, has played a significant role in mobilizing scientists, public health administrations and political leaders the world over to deal effectively with IDD (Pandav, 1996). Finally, goiter and other iodine deficiency disorders lower human aesthetic value.

### **Prevention**

Undisputed evidence shows that IDD could be successfully and inexpensively prevented and controlled. According to Hetzel, IDD "is so easy to prevent" "that it is a crime to let a single child be born mentally handicapped for that reason" (ACC/SCN, 1990). Because the greatest risk from iodine deficiency is during brain development, the highest priority targets for preventive action are women of reproductive age, infants and school-age children. Iodine supplementation has been shown to prevent goiter. The main approaches are the use of iodized salt and oil, fortification of foods, water and condiments, and distribution of iodine tablets.

### **Iodization of Table Salt**

In the long run, iodine deficiency can be prevented (and has been for many years in most industrialized countries) by fortification of salt with iodine. This normally requires legislation, a centralized salt supply, and the necessary equipment, funding and distribution systems. This method is being adopted in a number of developing countries, and requires sustained support and troubleshooting technical problems. (Gillespie and Mason, 1991). By 1991, universal salt iodization, that is all salt for human and animal consumption, was identified as the preferred means of reaching populations, including those consuming salt produced by small-scale artisanal salt works.

Salt is one of the most suitable vehicles because its consumption is universal, consistent, and its sources of production are easy to regulate. Salt for fortification must be dry and refined in order to avoid hydrolysis and ensure stability. Literature has shown that from production site to cooking before consumption, iodine loss from salt is about 40% therefore iodine concentration in salt from the point of production should be 20 - 40mg of iodine per kilogram of salt. If all salt used in food processing is iodized, 20mg is recommended. Also, to reduce the risk of iodine induced hyperthyroidism in a previously severe iodine-deficient population; iodine levels in salts should be set at the lowest level.

Salt iodization is less costly and carries less danger of toxicity than mass dose programmes, but also depends on efficient local level administration and often strong public education campaigns. In India, prevalence of goiter among those consuming fortified salt declined from 38% to 15% within five years, and fell to 3% after a decade (ACC/SCN, 1998). Another striking example in success of eliminating IDD and the benefits is quoted from China. The Jixian village of Heilong-jiang province in northeast China was known as village of idiots. Of its population of about 1313 individuals, goiter rate was 65% and 31.4% were cretins. Since the village began to use iodized salt in 1978, no cretin has been born and by 1982, goiter rate no longer posed a health problem. This wonderful improvement also manifested itself in the economy because the average annual income per head rose from 43 Yuan per year in 1981 to 414 Yuan per person in 1984 (WHO, 1990).

The process of iodization of salt is inexpensive and simple consisting of spray-mixing potassium

iodate with the salt. It does not change the colour, appearance and taste of the salt. Once iodization of salt is established in the country, it becomes a continuous and long-term solution to the problem. The main thrust towards elimination of IDD, has been establishing and sustaining national salt iodization schemes. effective partnerships have been forged between relevant UN agencies, national and international NGOs, and the salt industry. Globally, 68% of households in countries with-IDD now consume iodized salt. Iodization rates are highest in the Americas at 90%. Africa has. achieved a level of 63% (WIIO/UNICEF/ICC/DD, 1999).

### **Iodization of Oil**

Vulnerable individuals can be protected using iodized oil, administered by injection or orally. Single intramuscular injections of iodinated oil prevent deficiency for up to 5 years. Administration by mouth probably gives around 12 months protection (ACC/SCN, 1991). Researchers in Bolivia found that an iodized oil programme markedly reduced goiter after only two years and improved intellectual performance. The efficiency and safety of iodized oil used during pregnancy in the prevention of endemic goiter and cretinism have been established (Delange, 1996).

In the late 1970's, concern emerged about disease transmission through the use of syringes for intramuscular injection of iodized oil. This was mostly in connection with AIDS epidemic. Oral iodized oil was tried and its efficiency established, but the period of protection was observed to be much shorter. At the same time, it was felt that the use of iodized oil (by either route) was not ideal for the long term because it required use of medical personnel and depended on access to individuals and communities (ACC/SCN, 1997).

Intramuscular iodized oil injection, oral iodized oil, drops and tablets can be used in rural areas of the world with endemic goiter and cretinism where salt iodization is not possible due to lack of amenities like good roads and large markets. Oral administration of iodized oil is simpler, cheaper, and safer than iodized oil injection, requiring less technical training, fewer instruments and less time (Hetzel, 1993).

IDD, have been virtually eliminated in twenty (20) developed countries, mainly through iodization assisted by the increased dietary intake of iodine associated with development. Positive examples exist for a number of developing countries. For example, Nepal eliminated cretinism from many mountain districts through mass injections of iodized oil, followed by salt iodization. Similar progress was made in Indonesia and Zaire (ACC/SCN, 1990).

### **Other Possible Approaches to IDD Prophylaxis Include**

Diversification and modification of the habitual diet consumed in the endemic areas, with for example imported foods from outside; Iodine supplementation foods and water

Active prophylaxis of domestic animals; use of iodine materials for plants or iodine deficient soils.

Iodine conservation in the process of meal preparation, elimination of goitrogens, for example adequate soaking in the case of cassava and knowing the benefits of iodine for health are all important if dietary consumption is to be improved without iodine supplementation.

The long-term solution for the sustainable elimination of iodine deficiency would more likely take place through increasing the iodine content of the general food supply. There has been iodization trials using drinking water, bread and sweets but the most promising vehicle was clearly common table salt. In certain situations, however, iodization of salt, use of iodized oil have been found to have limitations, so an alternative method of providing iodine to deficient populations has been developed .by researchers (ACC/SCN, 1993). The system consists of a silicone elastomer platinum matrix containing a dispersion of sodium iodide which, when immersed in water, under regulation; release-iodine at a constant rate over a period of one year. The system was tested in Mali, West Africa, *ova* one year from November 1988 to November 1989, The conclusion given was that for a year the controlled release system supplied a physiological supplement of iodine that was beneficial for all subjects, whatever their age, sex, nutritional status, or previous medical status, while having n: ecological effects (Fisch et al, 1993). Iodine supplements for animals

The enriching of animal feeds with iodized salt allows for better milk output and improve animal production. In Germany, the use of iodine supplements for cattle and pigs has been very successful and deserves to be more extensive. Significant levels are attained in milk and meat, which then act as a vehicle for human dietary intake, resulting in the return of urinary iodine levels to normal values (Bauch et al, 1990).

### **Indication for Different Methods of Iodine Supplementation**

The most appropriate method of iodine supplementation depends on the severity of IDD :~ the

population concerned:

**Mild IDD:** goitre prevalence in the range of 5-19.9. % (school children) and median urinary iodide levels in the range 50-99ug/l. Mild IDD can be eliminated with iodized salt and economic development.

**Moderate IDD:** goitre prevalence of 20-29.9% and some hypothyroidism, and median urinary iodine levels in the range 20-49ug/l. This can be controlled using iodized salt (usually 20 - 40mg/kg at the household level) if this can be effectively produced and distributed. Otherwise, Iodized oil, given orally or by injection should be administered via the primary health care system. **Severe IDD:** a high prevalence of goitre, 30% or more, endemic cretinism, and median urinary iodine below 20ug/l. Severe IDD requires iodized oil, given orally or by injection as an interim measure until effective iodized salt system is operational, if central nervous system defects are to be completely prevented.

### **Problem Associated with Iodization**

The main hazard of iodization is transient thyrotoxicosis or iodine-induced hyperthyroidism seen mainly in adults over the age of 40. It is caused by autonomous thyroid function resulting from long standing iodine deficiency. To minimize this, iodation should be lessened for those over the age of 40. Antithyroid drugs or radio iodine can be used to control this thyrotoxicosis. Associated with this hyperthyroidism is iodide goitre or the so called Wolff-Chaikoff effect

### **Conclusion**

Preventable iodine deficiency and its associated disorders constitute outstanding health problem. It is in fact intolerable, devastating and destructive and affects almost all aspects of human life-aesthetically, socially, economically, politically, nutritionally and otherwise. All round examination of the effects of IDD has shown that IDD tampers with human right: right to live, right to exploitation of environment, right to mental, social and economic development.

But, IDD is preventable. This is why Hetzel made the statement that it is a crime to let a single child be born mentally handicapped. The key to the elimination is mainly through iodization of salt and use of iodized oil. Therefore, let all hands be on deck because IDD needs nothing less than urgent and perfect eradication and elimination.

### **References**

- ACC/SCN (1991). *Some Options for Improving Nutrition in the 1990s*. Supplement to SCN News No. 7 (Mid-1991).
- ACC/SCN (1988). SCN News, ACC/SCN No2, (30<sup>th</sup> March 1988), pg. 8.
- ACC/SCN (1991). SCN News, ACC/SCN No7 (mid 1991) pg.58. ACC/SCN (1993). SCN News, ACC/SCN No 10 (late 1993) pg 28-29. ACC/SCN (1990). SCN News, ACC/SCN NO. 5 early 1990) pg.27.
- ACC/SCN (1997). *Nutrition and Poverty Papers* from the ACC/SCN 24<sup>th</sup> Session Symposium Kahlman Du, (March 1997), pg. 66.
- ACC/SCN (1997). *Third Report on the Work! Nutrition Situation*. Pg. 20.
- Bauch K. et al, (1990). *A Five-Year Interdisciplinary Control of Iodine Deficiency* in the GDR Acta Medica Austriaca, 17 (Suppl. (j) pg. 36-38.)
- Delange F. (1996). Administration of Iodized Oil During Pregnancy: A Summary of the Published Evidence. *Bulletin of the WHO*, 74 (I) pg.101-108.
- Fisch, A et al (1993). A New Approach to Combating Iodine Deficiency in Developing Countries: The Controlled Release of Iodine in Water by a Silicone Elastomer. *American Journal of Public Health* 83 (4) 540-545.
- Gillespie Stuart and Mason John (1991). *Nutrition Relevant Actions*. ACC/SCN State of the Art Series,

Nutrition Policy Discussion Paper No. 10(Oct 1991). Pg. 113.

Hetzel Basil S. (1993). *The Prevention and Control of Iodine Deficiency Disorders*. ACC/SCN State of the Art Series. Nutrition Policy Discussion Paper. No. 3 pg 4, 6.

Hetzel B.S. Chavadej J, potter B.J. (1988). *The Brain in Iodine Deficiency. Neuropathology and Applied Neurobiology*, 14:93-104.

Hetzel B.S. Maberly G.F. (1986). Iodine in Mertz W. ed. *Trace Elements in Human and Animals Nutrition*, 5<sup>th</sup> Ed, Vol. 2, Orland Academic press, pg. 139-208.

Pandau C.s. (1996). *The Economic Benefits of the Elimination of IDD*. In: S.O.S. for a Billion. (Eds) B.S. Hetzel and C.S. Pandau (eds).

WHO (1998). *Consultation on Indications for Assessing Iodine Deficiency Disorders and their Control Through Salt Iodization* (4<sup>th</sup> to 6<sup>th</sup> May, 1998). Geneva, pg. 52.

WHO/UNICEF/ICC IDD (1999). *Progress Towards the Elimination of Iodine Deficiency Disorders (IDD)*. WHO/NHD/99.4 Geneva: WHO.