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REPORT

Goitre: An Iodine Deficiency Disorder

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Goitre is one of the well known manifestations of iodine deficiency disorder. It can be prevented by consuming iodized salt to remove iodine deficiency.

Goitre is one of the better-known manifestations of iodine deficiency. The normal or subnormal activity, the condition formally attributed to an insufficient supply of iodine with marked enlargement of the thyroid gland is known as goitre. It is well known that several types of familiar goitre with hypothyroidism are associated defects in intra-thyroidal metabolism of iodine. Deficient thyroid activity produces myxedema, mental and physical sluggishness, edema and stunted growth. Iodine is a micronutrient. Approximately 8 mg iodine is found in the normal secretion of thyroid gland which represents about 20–40 per cent of total iodine content of the adult body. The effect of iodine deficiency can occur during every stage of life.

Iodine deficiency is no longer a problem related to goitre only; another condition known as cretinism is also associated with iodine deficiency, commonly characterized by mental retardation, deaf-mutism, squint disorder, stunted growth and hypothyroidism—a condition of insufficiency of thyroxine which is a hormone synthesised from iodine by the thyroid gland, an endrocine gland which releases a specialised biological secretion directly into the blood stream and exerts specific effects on the target organs. Thyroxine is a hormone which contains iodine, synthesised and secreted by the thyroid gland. The word 'goitre' has come from the Latin word 'gutter' which means throat. This condition was known to the ancient Romans who called it 'bronchocele'. 'Hypo' in Greek means deficient or low or below. In case of a pregnant lady the deficiency of iodine causes retardation in foetal growth. Hypothyroid children are intellectually sub-normal and lack the aptitudes of normal children of same age.

Soil lacking iodine exists not only in mountanous (Himalayan and Sub-Himalayan) regions but also in densely populated plains and river basins which are blighted by periodic rains and floods. Floods and unseasonal heavy rains sometimes cause deficiency of iodine in soil and iodine deficiency disorder might appear in that particular regions. According to recent survey, more than 600–700 millions of people in Asia live in these regions and at the risk of iodine deficiency disorder. Flood and unseasonal rains sometimes cause deficiency of iodine in soil

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and iodine deficiency disorder appears in that particular area. Due to these causes, more and more areas are becoming iodine deficient. Himalayan sub-Himalayan and Tarai regions show about 15–20 per cent of newly born babies affected by hypothyroidism.

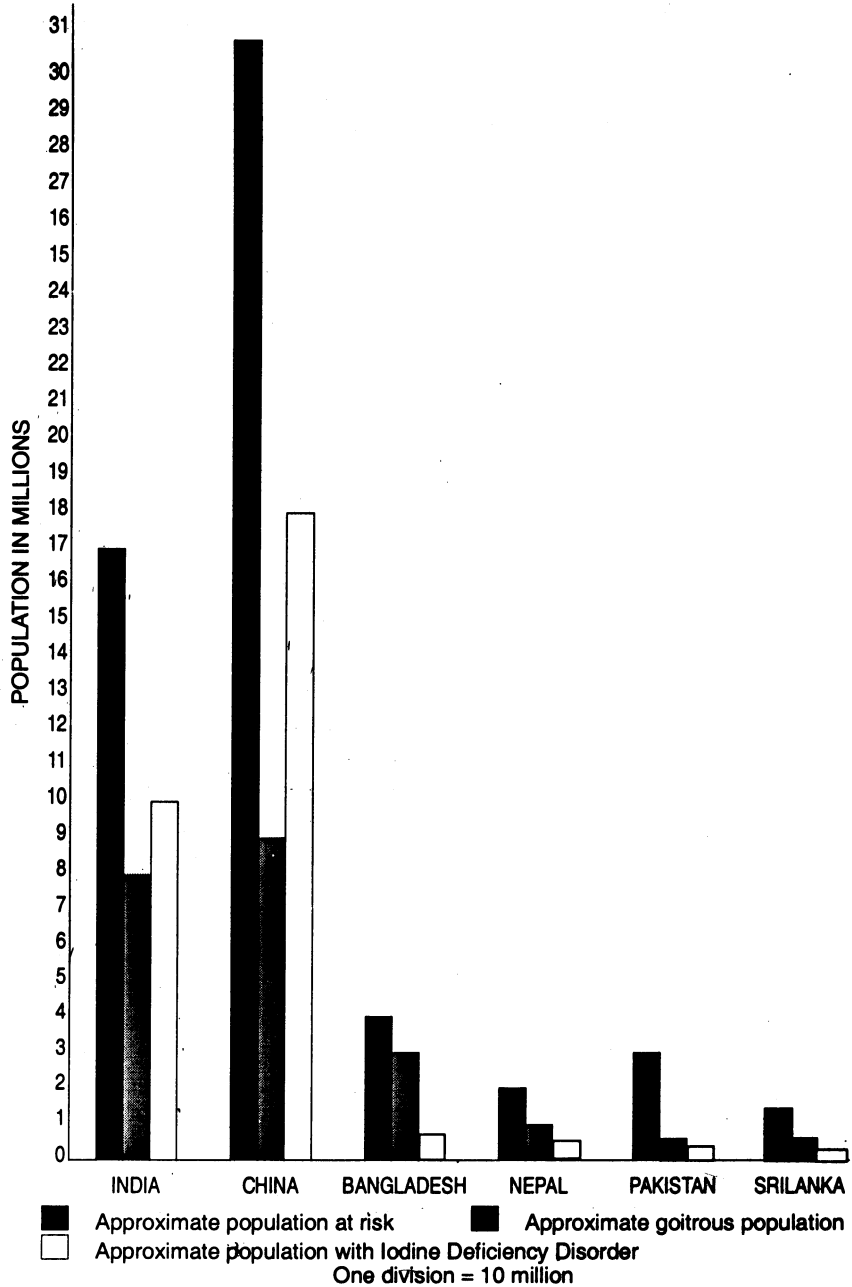

Fig. 1. Extent of Iodine Deficiency Disorder in some Asian countries

The relaton between iodine intake and iodine deficiency disorder is shown in Table-1.

TABLE-1
RELATIONSHIP BETWEEN IODINE INTAKE AND IDD

Nutritional status	Daily iodine intake (micrograms)
Associated with cretinism	20 or less
Associated with goitre	20–50
Marginal	50–100
Normal	100–300
High	300–500
Possible excessive	500–1000

In ancient times, the people of China used seaweeds to treat goitre. Afterwards scientists discovered that seaweeds are rich in iodine. The mechanism of thyroid gland and the hormone thyroxine, essential for growth and development of body and brain cells, and its dependence on iodine intake became known only at the end of the nineteenth century. Fig. 1 and Fig. 2 show the extent of iodine deficiency disorder in some Asian countries and in goitre-recorded areas respectively.

Fig. 2. Goitre-recorded areas

In 1812, the French chemist Bernard Curtois studied the use of kelp, a seaweed, and discovered a new substance iodine which in Greek means 'violet'. Later, Humphrey Davy, an English scientist, in 1829 studied the element and named it iodine similar to other two elements chlorine and fluorine of the halogen group both of which he had already discovered. In 1889, the French pathologist Gley

established that iodine is present in a gland and then in 1912, E.C. Kendall (USA) isolated an iodine-bearing substance when studying thyroxine. Ultimately the chemical nature of thyroxine was established by Harington, an English chemist, in 1926. The production of iodised salt as an aid in preventing goitre was described by Holman in 1956.

Thyroxine is not destroyed during digestion, so that inexpensive preparations of desiccated thyroxine gland can be given orally and are fully active. Thyroxine may also be determined chemically by its iodine content but it must be remembered that thyro-globulin also contains the more soluble amino acid diodotyrosine (Fig. 3).

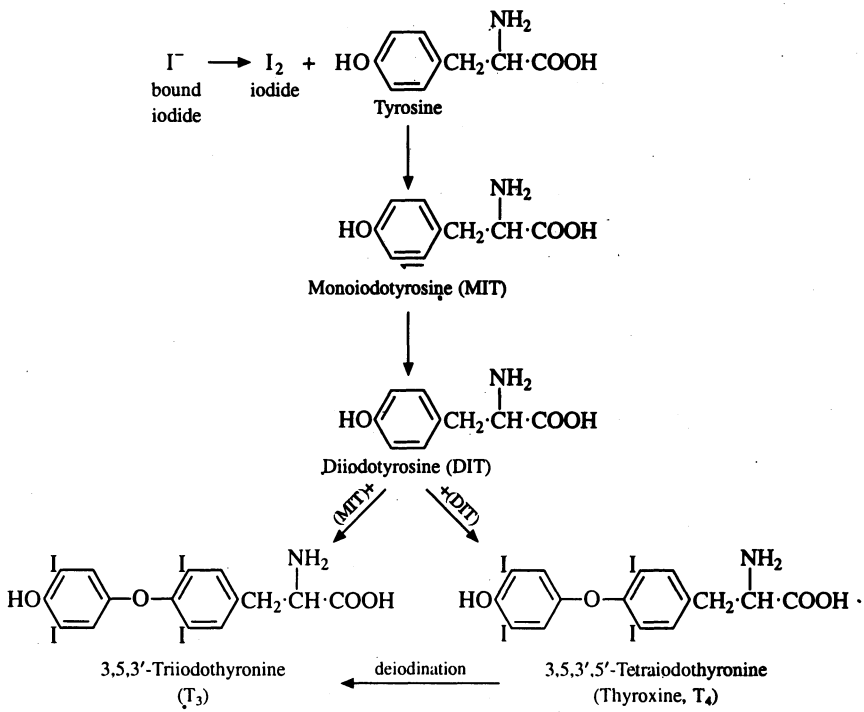

Fig. 3. Biosynthesis of thyroid hormone

The first step in the biosynthesis is the concentration of inorganic iodine within the thyroid gland (iodine trapping). Thyroid iodine is oxidised to iodine *via* a peroxidase enzyme. The amino acid tyrosin is progressively iodinated to monoiodothyrosine (MIT) and then diiodothyrosine (DIT). Two molecules of diiodotyrosine are coupled to form one molecule of thyroxine. Tetraiodothyroxine (T4) and triiodotyrosine (T3) could be obtained from the deiodination of thyroxine or coupling of monoiodotyrosine with diiodotyrosine.

Iodine deficiency disorder and goitre can be prevented by regular use of salt fortified with iodine (iodised salt) if not already affected. It can also be prevented by administration of iodine-rich oil under the advice of a medical practitioner.

Iodised oil can be swallowed or injected. It is a compound of iodised ethyl esters of fatty acids or unsaturated fatty acids (36% iodine by weight) like fatty acids of poppy seeds, soyabean, etc. The fortification of common salt (NaCl) is done with potassium iodate (KIO_3) because it is a relatively stable compound containing 59.3% iodine. The result is generally expressed as iodine in parts per million (mg of iodine present in 1000 g of salt). The Ministry of Health and Family Welfare, Government of India has made it statutory to iodise edible common salt under the provisions of PFA Act (1954). It has laid down the minimum prescribed limits of iodine content at manufacturing level and retailer level along with other essential parameters for salt under item No. A-15.01 of PFA rules. There is no upper limit of iodine prescribed in the Rules (Table-2).

TABLE-2

A. 15.01: IODISED SALT means a crystalline solid, white or pale, pink or light grey in colour, free from visible contamination with clay, grit and other extraneous adulterants and impurities. It shall conform to the following standards, namely:

Moisture	Not more than 6.0 per cent by weight of the undried sample.
Sodium chloride (NaCl)	Not less than 96.0 per cent by weight on dry basis.
Matter insoluble in water	Not more than 1.0 percent by weight on dry basis.
Matter soluble in water other than sodium chloride	Not more than 3.0 per cent by weight on dry basis.
Iodine content at:	
(a) Manufacture level	Not less than 30 parts per million on dry weight basis.
(b) Distribution channel including retail level	Not less than 15 parts per million on dry weight basis.

Provided that table iodised salt may contain aluminium silicate as an anticaking agent to a maximum extent of 20 percent by weight:

Provided further that the total matter insoluble in water in such cases shall not exceed 22 per cent and sodium chloride content on dry basis shall not be less than 97.0 per cent by weight.

Iodised salt prevents hypothyroidism of the coming generation when consumed in child-bearing age. Iodised salt is highly effective as a preventive measure of iodine deficiency disorder. The normal human requires 150 micrograms iodine per day (1,000,000 m/g). About 90% of this comes from food and the rest from water which derives it from soil. Iodised salt is the most inexpensive and effective method to prevent iodine deficiency disorder.

The iodisation of salt is a very simple method which can be adopted by big

and small manufacturing enterprises of iodised salt. The continuous spray mixing of KIO_3 with very fine free flowing crystals of common salt is most common and economical. In this process a more stable compound of iodine, potassium iodate (KIO_3), is usually used, mixed in screw conveyor and bagged under controlled system to adjust iodine content. Increasing the concentration of iodine has no adverse effect, but too low iodine content may not prevent iodine deficiency disorder.

Due to volatile unstable nature, the iodine content of iodised salt constantly goes down. The loss becomes more due to high moisture content, sunlight and bad storing conditions. If moisture content goes high, water is absorbed by salt and iodine migrates to the bottom of the bag. This problem is less acute in small packets of one kg or half kg generally used for domestic consumption. Iodised salt should be packed in moisture-proof and food grade polythene bags to maintain the nutritional value as well as iodine content in accordance with the minimum prescribed limits laid down in PFA Rules (1955). There should be constant careful monitoring of iodine content at the production as well as the retailer stages. Periodical lifting of iodised salt samples by PFA enforcement officials is very essential to curb adulteration.

The iodine content can be determined in the chemical laboratory by the simple method of iodometric titration.

Iodine deficiency disorder is very much alarming in India. General mass dueling in rural and urban areas are not fully aware regarding the problem of iodine deficiency disorder. It is absolutely essential to educate the common people about the ill-effect of iodine deficiency disorder through Government enforcement machineries, Medical Centres, wide Publication through Newspapers, Door Darshan and All India Radio so that the iodine deficiency disorder can be minimise. A stingent action for violating the statutory Food Act (PFA Act 1954) is also desirable apart from high level seminars.

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