

Determination of iodine content in different brands table salt of Bangladesh

A Thesis Paper submitted to the Department of Pharmacy, East West
University in partial fulfillment of the requirement for the degree of
Bachelor of Pharmacy

SUBMITTED BY

Mahfuza Fardousi

ID- 2008 3-70-077

JULY 2012



**DEPARTMENT OF PHARMACY
EAST WEST UNIVERSITY**

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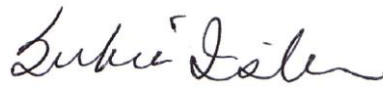


**DEPARTMENT OF PHARMACY
EAST WEST UNIVERSITY**

**In the name of “ALLAH”
The most Gracious The most
Merciful**

CERTIFICATE

This is to certify that the thesis submitted to the Department of Pharmacy, East West University, Aftabnagar, Dhaka in partial fulfillment of the requirement for the degree of Bachelor of Pharmacy was carried out by Mahfuza Fardousi ID-2008-3-70-077.



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This is to certify that the thesis submitted to the Department of Pharmacy, East West University, Aftabnagar Dhaka in partial fulfillment of the requirement for the degree of Bachelor of Pharmacy was carried out by Mahfuza Fardousi ID:2008-3-70-077 under our guidance and supervision and that no part of the thesis has been submitted for any other degree. All the sources of information, laboratory facilities availed of this connection is dully acknowledged.



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Abstract

IDD (Iodine deficiency disorders) are recognized as a major global public health problem; it is possible to root out this problem by universal salt iodization. The study was designed to determine iodine concentration in different branded salt collected from different area of Bangladesh. All samples were collected both Dhaka and out of Dhaka city. Concentration of iodine in salt was determined by iodometric titration method. Reagent was $K_2Cr_2O_7$, standardized $Na_2S_2O_3$, KI, $NaHCO_3$, starch, concentrated HCl, H_2SO_4 . Most of the salts collected show iodine level to be in the acceptable range. Result showing that out of seven brand only one shown to has a poor content of iodine. It can be decided from the result that most of the people of our country are now using iodized salt. Evidence is now available from both controlled trials and successful iodization programs that these disorders can be successfully prevented by correction of iodine deficiency. A major achievement is the spectacular reduction of IDD in our country that has implemented universal salt iodization.

Keyword:- Iodine deficiency disorders, symptoms, treatment, iodine determination

CHAPTER -1

INTRODUCTION

1.1 Overview

Iodine is an element that is needed for the production of thyroid hormone. The body does not make iodine, so it is an essential part of your diet. If we do not have enough iodine in our body, we cannot make enough thyroid hormone. Thus, iodine deficiency can lead to enlargement of the thyroid hypothyroidism and to mental retardation in infants and children whose mothers were iodine deficient during pregnancy (American Thyroid Association, 2007)

Iodine deficiency affects humans at every stage of life and leads to several severe disorders. Iodine deficiency is the leading cause of brain damage and mental retardation in the world. In addition to mental retardation, iodine deficiency causes endemic goiter, cretinism, dwarfism, mental retardation, muscular disorders, spontaneous abortions, sterilization, and stillbirths (Verma and Raghuvanshi, 2001).

In the 1990s, 1.6 billion people, one third of the world's population, was at risk for iodine deficiency disorders (IDD); less than 20 percent of people at risk had access to iodized salt. Universal Salt Iodization (USI), the primary strategy to prevent IDD, was adopted in 1993. Since then, more than 90 million newborns are protected each year from learning disabilities caused by IDD (WHO). More than 90 percent of the populations of 21 developing countries use iodized salt. In 1998, nearly 60% of world's edible salt was iodized and there was a significant decrease in number of children born at risk of IDD. The number of cretin births halved to less than 55,000 per year (Kiwani, 1999) By 2000, 70 percent of households in developing countries used iodized salt (Kiwani, 2000).

UNICEF has declared that 85 million children will be born free of iodine deficiency disorders (IDD) this year. Because of the global effort to wipe out IDD it is now estimated that more than 70 percent of the world has access to iodized salt, the most practical vehicle for providing iodine in the diet (Kiwani, 2011)

Iodized salts stored in atmospheres of relative humidities of 50per cent lose smaller quantities of their iodine than salts stored under similar conditions at other humidities. Iodized salts rendered alkaline by the addition of NaHCO_3 lose practically none of their iodine during storage, while neutral salts or salts rendered acid lose appreciable quantities. Salts iodized with KIO_3 lose none of their iodine when stored for extended periods. Exposure of iodized salts to sunlight effects a loss of iodine from neutral or acid

salts, only a slight loss from salt rendered alkaline, and practically no loss from salts iodized with KIO_3 . Exposure to heat alone effects losses of iodine from acid, neutral, and alkaline iodized salts and from salt iodized with KIO_3 in precisely the same order as did exposure to sunlight. Exposure to light and heat simultaneously effects greater losses of iodine from salts of neutral or acid reaction than exposure to light alone or heat alone. In the case of iodized salts of alkaline reaction the losses of iodine are again insignificant or negligible, even though the salts are exposed to light and heat simultaneously. The quantity of iodine liberated from a neutral salt iodized with KI appears to depend on the quantity of KI present. (Arnold H. Johnson and B. L. Herrington August, 1927)

Universal salt iodization is the recommended intervention for preventing and correcting iodine deficiency. In the past, recommendations for iodine levels in salt were made on the assumption that, from producer to consumer, iodine losses from iodized salt were commonly between 25% and 50%, and that average salt intakes were commonly between 5 and 10 g/person/day. Substantial experience has been gained in the last decade in implementing universal salt iodization and assessing its impact on iodine deficiency disorders (IDD). A major achievement is the spectacular reduction of IDD in countries that have implemented universal salt iodization (Geneva, 8-9 July 1996).

The aim of the study is to determine the iodine content in different brand salts collected from different areas of Bangladesh.

1.2 Iodine

Iodine is a bluish-black, lustrous solid that mainly occurs in nature as stable iodine-127. A small amount of radioactive iodine-129 is produced naturally in the upper atmosphere by the interaction of high-energy particles with xenon. Iodine volatilizes at ambient temperatures into a pretty blue-violet gas with an irritating odor. Iodine exhibits some metal-like properties and is only slightly soluble in water. It occurs in nature as iodide ions, and it is in this form that it is taken into our bodies. (Human Health Fact Sheet, 2005) .

1.3 Sources

Iodine is naturally present in seaweeds, sponges and such other materials. Iodine is assimilated by seaweeds and sponges (from which it may be recovered) and is found in Chilean saltpeter, caliches, and brine associated with salt deposits.

Table 1. Common Dietary Sources of Iodine (American Thyroid Association, 2007)

Breads	Iodized table salt
Cheese	Saltwater fish
Cow's Milk	Seaweed
Eggs	Shellfish
Frozen Yogurt	Soy Milk
Ice-Cream	Soy Sauce
Iodine-containing multivitamin	Yoghurt

1.4 Uses

Iodine is used to treat cuts and scrapes on the skin as a tincture of iodine, which is a dilute mixture of alcohol and iodine. Iodine is also used in photography and lasers (silver iodide), in dyes, and as a nutrient added to table salt. Iodine-131 is used for a number of medical procedures, including monitoring and tracing the flow of thyroxin from the thyroid. With its short half-life of 8 days, it is essentially gone in less than three months. Iodine-129 has no important commercial uses(Human Health Fact Sheet, 2005).

1.5 Role of iodine in human body

Iodine is a chemical element required for good health, as it has several roles in the body. Iodine is needed to make thyroid hormones and promote a healthy pregnancy, as well as to prevent a number of conditions. Because foods don't naturally contain much iodine, salt is fortified with iodine. It's estimated that iodized salt is used regularly by about half the U.S. population, according to Medline (Brownstein, 2008).

1.5.1 Role of iodine in Thyroid Function

The thyroid gland can't function without iodine because it's needed to make thyroid hormones. Insufficient iodine can cause an enlarged thyroid gland. This unsightly swelling in the front of the neck is known as a goiter. Insufficient iodine can also affect thyroid function by causing hypothyroidism, or an under active thyroid. Symptoms include fatigue, weight gain and depression. Thyroid function is important for fertility, as well. Iodine deficiency leading to low levels of thyroid hormones can cause women to stop ovulating, leading to infertility (Brownstein, 2008).

1.5.2 Role of iodine in Normal Metabolism

Iodine is needed for the normal metabolism of cells. Metabolism is the process of converting food into energy. Insufficient iodine can slow down the body's metabolism, which can result in weight gain. "Because thyroid hormones regulate metabolism and growth, when production of these hormones is slowed or stopped due to iodine deficiency, your metabolism can become sluggish," notes Beth Reardon, registered dietitian and Director of Nutrition at Duke University Integrative Medicine. The metabolic rate determines how fast calories are burned. A higher metabolic rate is thought to speed up weight loss. (Brownstein, 2008).

1.5.3 Role of iodine in Healthy Pregnancy

Iodine plays a crucial role in promoting a healthy pregnancy. Iodine deficiency during pregnancy can cause high blood pressure in the mother and mental retardation in the baby. In extreme cases, iodine deficiency during pregnancy can cause the baby to be born with a disorder that involves severely stunted physical and mental growth. The Lines Paling Institute indicates that iodine deficiency during pregnancy may also increase the risk of miscarriage, stillbirth and birth defects (Brownstein, 2008).

1.5.4 Role of iodine in protection of breast diseases

It is hypothesized that dietary iodine deficiency is associated with the development of mammary pathology and cancer. A review of the literature on this correlation and of the author's own work on the antioxidant function of iodide in iodide-concentrating extra thyroidal cells is reported. Mammary gland embryo genetically derived from primitive iodide-concentrating ectoderm, and alveolar and ductal cells of the breast specialize in uptake and secretion of iodine in milk in order to supply offspring with this important trace-element. Breast and thyroid share an important iodide-concentrating ability and an efficient peroxidase activity, which transfers electrons from iodides to the oxygen of hydrogen peroxide, forming iodoproteins and iodolipids, and so protects the cells from peroxidative damage (Brownstein, 2008).

1.5.5. Role of inorganic and organic forms of iodine as an antioxidant

Iodine is one of the most abundant electron-rich essential elements in the diet of marine and terrestrial organisms. It is transported from the diet to the cells via iodide transporters. Iodide, which acts as a primitive electron-donor through peroxides enzymes, seems to have an ancestral antioxidant function in all iodide-concentrating cells from primitive marine algae to more recent terrestrial vertebrates. Thyroxin and iodothyronines have an antioxidant activity too and, through denominate enzymes, are donors of iodides and indirectly of electrons. Thyroid cells phylogenetically derived from primitive gastro enteric cells, which during evolution of vertebrates migrated and specialized in uptake and storage of iodo-compounds in a new follicular “thyroidal” structure, for a better adaptation to iodine-deficient terrestrial environment. Finally, some animal human chronic diseases, such as cancer and cardiovascular diseases, favored by dietary antioxidant deficiency (Brownstein, 2008).

1.5.6 Role of iodine in oral mucosa and in salivary glands physiology

It is hypothesized that dietary deficiency or excess of iodine (I) has an important role in oral mucosa and in salivary glands physiology. Salivary glands derived from primitive I-concentrating oral cells, which during embryogenesis, migrate and specialize in secretion of saliva and iodine. Gastro-salivary clearance and secretions of iodides are a considerable part of "gastro-intestinal cycle of iodides", which constitutes about 23% of iodides pool in the human body. Salivary glands, stomach and thyroid share I-concentrating ability by sodium iodide symporter (NIS) and peroxides activity, which transfers electrons from iodides to the oxygen of hydrogen peroxide and so protects the cells from per oxidation. Iodide seems to have an ancestral antioxidant function in all I-concentrating organisms from primitive marine algae to more recent terrestrial vertebrates. The high I-concentration of thymus supports the important role of iodine in the immune system and in the oral immune defines. In Europe and in the world, I-deficiency is surprisingly present in a large part of the population (Brownstein, 2008).

1.5.7 Iodine and delayed immunity

Iodine was and is sometimes used therapeutically in various pathologies where the immune mechanism is known to play a dominant role. It has in fact been administered to patients with tubercular glaucomatous, lepromatous, syphilitic and mycotic lesions where it facilitates cure. This effect does not depend on iodine's action on the micro-organism responsible. Iodine may also be used in Villanova-Panof Panniculitis, in erythema nodosum, in nodular vasculitis, erythema multiform and Sweet's syndrome. Oral iodine is also very effective in the lymphatic-cutaneous form of sporotrichosis. In order to establish a relationship between dietary iodine and immune response, 607 infants residing in an area of endemic goiter were studied: 215 were given Lugol solution (2 drops a week for about 8 months) and 392 not. The immune response was assessed by the skin test method using tetanic toxoid and a clear correlation was shown between this and lymphocyte stimulation and monocytic chemotaxis tests. The test was considered positive when an infiltration of at least 5 mm in diameter was shown after 48 hours (in the U.S. 80% of pediatric cases aged 2-10 years old were positive). A significant difference was noted in the average diameter of the infiltrations after the tetanic toxoid skin test in the two groups considered (P less than 0.001). The results appear to indicate that an adequate iodine intake is necessary for normal retarded immune response. The molecular mechanism by which iodine increases immune response is still to be decided (Brownstein, 2008)

Table 2. Recommended Iodine intake (State Government of Victoria, 2010)

Classes of People	Recommended Daily intake (RDI) for Iodine
Younger children (1 to 8 years)	90µg
Older children (9 to 13 years, boys and girls)	120µg
Adolescents (14 to 18 years)	150µg
Men	150µg
Women: Pregnancy	220µg

1.6 Thyroid Gland

The human body contains 10 to 20 milligrams of iodine, of which more than 90% is contained in the thyroid gland. The thyroid is a 2-inch-long, butterfly-shaped gland weighing less than an ounce. Located in the front of the neck below the larynx, or voice box, it is composed of two lobes, one on each side of the windpipe. The thyroid is one of the glands that make up the endocrine system. The endocrine glands produce, store, and release hormones into the bloodstream that travel through the body and direct the activity of the body's cells. Thyroid hormones regulate metabolism and affect nearly every organ in the body. The thyroid gland makes two thyroid hormones, triiodothyronine (T_3) and thyroxine (T_4). Thyroid hormones affect metabolism, brain development, breathing, heart and nervous system functions, body temperature, muscle strength, skin dryness, menstrual cycles, weight, and cholesterol levels. A third hormone produced by the thyroid gland, calcitonin, is not considered a thyroid hormone as such, but affects calcium levels in the blood and controls the buildup of calcium in the bones. Thyroid hormone production is regulated by thyroid-stimulating hormone (TSH), which is made by another gland in the endocrine system called the pituitary, located in the brain (NIDDK, 2008)

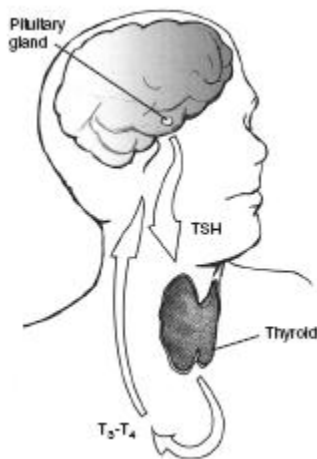


Fig 1. The thyroid gland's production of thyroid hormones (T_3 and T_4) is triggered by thyroid-stimulating hormone (TSH), which is made by the pituitary gland.(NIDDK,2008)

1.7 Iodine Deficiency Disorder

Iodine is an element that is needed for the production of thyroid hormone. The body does not make iodine, so it is an essential part of our diet. If we do not have enough iodine in our body, we cannot make enough thyroid hormone. Thus, iodine deficiency can lead to enlargement of the thyroid hypothyroidism and to mental retardation in infants and children whose mothers were iodine deficient during pregnancy (American Thyroid Association, 2007)

The significance of iodine in fetal development and thyroid health is not prioritized by majority of the population. Very few people realize that iodine deficiency is a serious health problem currently affecting over 740 million people in 130 countries (WHO).

In many areas around the world the soil does not contain an adequate amount of iodine. In mountainous regions, especially the Himalayas, Andes, and Alps, and in areas of frequent flooding, the soil has an inadequate amount of the essential element, leading to the prevalence of iodine deficiency disorders (IDD). Central Africa, Central Asia, and much of Europe also have iodine-depleted soil (ICCIDD).

Iodine deficiency affects humans at every stage of life and leads to several severe disorders. Iodine deficiency is the leading cause of brain damage and mental retardation in the world. In addition to mental retardation, iodine deficiency causes endemic goiter, cretinism, dwarfism, mental retardation, muscular disorders, spontaneous abortions, sterilization, and stillbirths (Verma and Raghuvanshi, 2001).

In addition to these specific effects on the individual, IDD also negatively affects the community as a whole. Since iodine deficiency is prevalent in entire communities where the soil is iodine-depleted, the entire community may suffer from levels of mental retardation, cretinism, etc. According to the ICCIDD, the average IQ of an iodine-deficient community about 13.5 points lower than other communities. Individuals in iodine-deficient communities, therefore, have more difficulty learning and developing a productive economy. By providing iodine to these communities, a dramatic socio-economic improvement is possible for the entire community (ICCIDD).

1.7.1 Causes of Iodine Deficiency

Worldwide, we are experiencing epidemic proportions of iodine deficiency, in part due to deforestation, soil erosion, and poor farming practices that deplete minerals from the soil and yield iodine-deficient crops. There are other contributing factors that exacerbate this disturbing global problem. Exposure to toxic chemicals hinders the uptake of iodine in the body as the toxins compete for iodine receptor sites and inhibit the body's ability to absorb this valuable mineral. These toxins include a group of elements known as halides (and their derivatives), all of which have similar chemical structures. The halides consist of bromide, fluoride, chloride and iodide, the latter being the only one with therapeutic effects in the body. In the 1980s, bromine (a bromide derivative) replaced iodine as a bread dough ingredient. Bromine is a known breast carcinogen. This singular change by the food industry resulted in an epidemic of bromide toxicity and increases in thyroid disorders, thyroid cancer and other illnesses resulting from iodine deficiency.

Exposure to chlorine (the oxidized form of chloride), as well as fluoride found in toothpaste, the water supply and many pharmaceutical drugs, further compound the deficiency dilemma as these toxins compete with iodine for absorption by bodily tissue. Sufficient iodine saturation in bodily tissues prevents the binding of halides and allows for their elimination from the body (Brownstein, 2008)

Since the body does not make iodine, it relies on the diet to have enough iodine. Thus, iodine deficiency is caused but not having enough iodine in the diet. The availability of iodine in foods differs in various regions of the world (American Thyroid Association, 2007)

1.7.2 Diagnosis of Iodine Deficiency Disorder

Iodine deficiency is diagnosed across populations and not specifically in individuals. Since iodine is released from the body through the urine, the best way to determine iodine deficiency across a large population is to measure the amounts of iodine in urine samples. Iodine deficiency is defined as a median urinary iodine concentration less than 50 $\mu\text{g/L}$ in a population.

Table 3. Median Population Urinary Iodine Values and Iodine Nutrition (American Thyroid Association, 2007)

Median Urinary Iodine Concentration ($\mu\text{G/L}$)	Corresponding Intake ($\mu\text{G/DAY}$)	Iodine Nutrition
<20	<30	Severe Deficiency
20-49	30-74	Moderate Deficiency
50-79	149-175	Mild Deficiency
100-199	150-299	Optimal
200-299	300-449	More than adequate
>299	>449	Possible Excess

1.7.3 Treatment

The solution to iodine deficiency disorders (IDD) is relatively simple and inexpensive compared to other prevalent diseases such as AIDS. Iodized salt, the preferred method of intervention, costs only \$0.05 per person per year to prevent IDD, according to the World Health Organization (WHO). A teaspoon of iodine is all a person requires in a lifetime, but it must be provided regularly, in tiny amounts to prevent IDD. Other methods are used, such as iodized vegetable oil, which is administered orally or by intramuscular injection, iodized drinking water, vitamins, sugar, bread, and tea (ICCIDD). Since even mild deficiency during pregnancy can have effects on delivery and the developing baby, all pregnant and breastfeeding women should take a multivitamin containing at least 150 μg iodine per day (American Thyroid Association, 2007)

1.8 Iodine Deficiency and Pregnancy

Iodine deficiency is especially important in women who are pregnant or nursing their infants. Severe iodine deficiency in the mother has been associated with miscarriages, stillbirth, preterm delivery, and congenital abnormalities in their babies. Children of mothers with severe iodine deficiency during pregnancy can have mental retardation and problems with growth, hearing, and speech. In the most severe form, an under active

thyroid can result in cretinism (a syndrome characterized by permanent brain damage, mental retardation, deaf mutism, spasticity, and short stature). Congenital hypothyroidism due to iodine deficiency is the most common preventable cause of mental retardation in the world. Even mild iodine deficiency during pregnancy, may be associated with low intelligence in children (American Thyroid Association, 2007)

1.9 Iodine Deficiency in the Fetus

Iodine deficiency in the fetus is the result of iodine deficiency in the mother. It is associated with a greater incidence of stillbirths, abortions and congenital abnormalities, which can be reduced by iodization. Another major effect of fetal iodine deficiency is endemic cretinism. This condition is still widely prevalent, Its commonest form is referred to as the 'nervous' (neurological) type in contrast with the less common "myxoedematous" type characterized by hypothyroidism with dwarfism. (Hetzl, 1993)

1.10 Iodine Deficiency in the Children and Adolescents

Iodine deficiency in this period is characteristically associated with endemic goiter. Prevalence increases with age, reaching a maximum after the first decade of life. The condition can be effectively prevented by iodization. There is increasing evidence of impaired mental function in apparently normal children living in iodine-deficient areas. (Hetzl, 1993)

1.11 Iodine Deficiency in the Adults

The common result of iodine deficiency in adults is endemic goiter. One of its accompanying effects is a high degree of apathy. It is apparent that reduced mental function is widely prevalent in iodine-deficient communities with effects on their capacity for initiative and decision-making. Characteristically there is an absence of classical clinical hypothyroidism, but laboratory evidence of hypothyroidism with reduced T-4 levels is common. This is often accompanied by normal T-3 and raised TSH levels. Iodine administration in the form of iodized salt, iodized bread or iodized oil have all been demonstrated as effective in preventing goiter in adults. Iodine

supplementation may also reduce existing goiter. This is particularly true of iodized oil injections. The obvious benefit leads to ready acceptance of the measure by people living in iodine-deficient communities .A rise in circulating thyroxin can be easily demonstrated in adult subjects following iodization. This could mean a rise in brain T-3 levels with improvement in brain function (Hetzl 1993)

1.12 Specific Iodine Deficiency Disorder

1.12 Hyperthyroidism

The term hyperthyroidism refers to any condition in which there is too much thyroid hormone in the body. In other words, the thyroid gland is overactive. Hyperthyroidism is a disorder that occurs when the thyroid gland makes more thyroid hormone than the body needs. It is sometimes called thyrotoxicosis, the technical term for too much thyroid hormone in the blood. Women are much more likely to develop hyperthyroidism than men. (American Thyroid Association, 2005)

Symptoms

Hyperthyroidism has many symptoms that can vary from person to person. Some common symptoms of hyperthyroidism are:

- nervousness or irritability
- fatigue or muscle weakness
- trouble sleeping
- heat intolerance
- hand tremors
- rapid and irregular heartbeat
- frequent bowel movements or diarrhea
- weight loss
- mood swings
- goiter, which is an enlarged thyroid that may cause your neck to look swollen (NIDDK, 2008)

Causes

Hyperthyroidism has several causes, including

- Graves' disease
- one or more thyroid nodules
- thyroiditis, or inflammation of the thyroid gland
- overmedicating with synthetic thyroid hormone, which is used to treat under active thyroid

Rarely, hyperthyroidism is caused by a pituitary adenoma, which is a non-cancerous tumor of the pituitary gland. In this case, hyperthyroidism is due to too much TSH.

1.12.3 Endemic Goiter

A goiter is an enlarged thyroid gland. The enlargement can be due either to an increased number of cells or to an increase in the size of individual cells. The enlargement is an adaptive response, as the thyroid attempts to produce more thyroid hormone. It attempts to produce more thyroid hormone due to Thyroid Stimulating Hormone (TSH) from the pituitary, in response to low levels of thyroid hormones in the blood (Zoe, 2006).

Thyroid gland enlargement can be generalized and smooth, a so called diffuse goiter; or it can become larger due to growth of one or more discrete lumps (nodules) within the gland, a nodular goiter. A goitrous gland can continue producing the proper amounts of thyroid hormones, in which case it is called a euthyroid or nontoxic goiter; or a goiter can develop in conditions with either overproduction of thyroid hormone, called toxic goiter, or the inability to make sufficient thyroid hormones, called goitrous hypothyroidism (Ladenson, 2008)

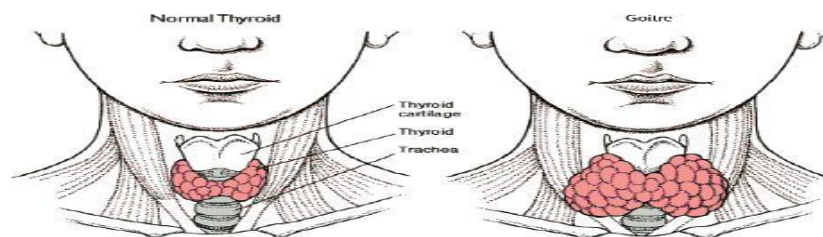


Fig 2. Endemic Goiter (The Daily Sun)

Symptoms

The primary symptom of goiter is a swelling of the thyroid, a butterfly gland at the front of the neck just below the Adam's Apple. Other possible symptoms are the following:

- A tight feeling in the throat
- Coughing
- Hoarseness
- Difficulty in swallowing
- Difficulty in breathing
- Choking awake at night
- Food getting stuck in the upper throat (Zoe, 2006)



Fig3. Boy with Goiter (Science photo Library)

Causes

Three categories of problems are responsible for almost all cases of thyroid gland enlargement: inefficient thyroid hormone production, gland inflammation, and tumors in the thyroid. First, when the gland is inefficient in making sufficient thyroid hormone, it compensates by getting bigger. Worldwide, the most common cause is dietary iodine deficiency, a condition estimated still to affect 100 million people who live in poverty-stricken societies. Iodine is an essential building block for thyroid hormones; in the absence of adequate supply, the gland becomes larger. When more than 10% of a population has goiter due to iodine deficiency, it is called endemic goiter. Other consequences of severe iodine deficiency include hypothyroidism and cretinism, a syndrome of mental retardation, short stature, deafness, and characteristic facial deformities that affects children born to hypothyroid mothers in iodine deficient regions. People with defects in their genetic blueprints for the proteins that permit the thyroid gland to make thyroid hormone (e.g., mutations in the molecular pump that enable the thyroid to concentrate iodine within itself) typically develop a goiter. Certain drugs can also interfere with normal thyroid function and lead to compensatory gland enlargement, such as lithium carbonate, which causes a goiter in 10% of individuals taking this medicine. Second, inflammation of the thyroid gland (thyroiditis) can produce gland swelling. Some forms of thyroid inflammation are quite common, such as autoimmune thyroiditis and painless (postpartum) thyroiditis. Autoimmune thyroiditis (also called Hashimoto thyroiditis) occurs when a person's immune system turns against their own thyroid gland, inflaming it, usually causing the gland to swell, and often making it permanently under active. Autoimmune thyroiditis can first appear in children and young Adults, but its incidence increases sharply in middle aged and elderly people. Other types of thyroiditis causing goiter include:

- painless (postpartum) thyroiditis, a self-limited inflammation of the thyroid that can resolve without treatment and affects at least five per cent of women in the year after
- pregnancy
- subacute thyroiditis, which causes painful thyroid enlargement as the result of viral infection

- other rarer forms of infectious thyroiditis
- drug-induced thyroiditis, such as those caused by amiodarone and interferon alpha
- a rare fibrosing condition called Reidel thyroiditis.

Third, goiter can be the result of thyroid tumors, which are usually benign, but sometimes malignant. Most thyroid tumors present as discrete nodules, but there are several kinds of thyroid cancer that can cause generalized swelling of the gland. These include infiltrating papillary thyroid cancer, lymphoma, and Anaplastic thyroid cancer. Certain facts make it important to consider the possibility that a goiter might be malignant. These include one or more of the following symptoms: rapid enlargement of a goiter over a few weeks, the onset of new thyroid-related pain, difficulty swallowing, shortness of breath, or coughing up blood; or a goiter in someone with risk factors for thyroid cancer, such as a person who had childhood radiation to their neck or who has a close relative with thyroid cancer (Ladenson, 2008)

Treatment

Whether a goiter needs treatment depends on the answers to the three key clinical questions. If the thyroid is so large as to cause symptoms by stretching or compressing adjacent structures, or if it is so big as to be unsightly, surgical removal of the thyroid gland (thyroidectomy) may be required. If the goiter is related to a condition causing hyperthyroidism, as in Graves's disease or toxic nodular goiter, treatment with radioactive iodine may be effective in both controlling gland over activity and decreasing its size. Some normally functioning (nontoxic) nodular goiters can also be shrunk with radioactive iodine therapy. If the thyroid is enlarged as the result of autoimmune (Hashimoto) thyroiditis and the gland is also under active with a high blood TSH level, then starting thyroid hormone medication (L-thyroxine) may both treat the hypothyroidism and partially shrink the gland. (Ladenson, 2008)

1.12.4 Cretinism

Cretinism is a situation induced by an insufficiency of thyroid hormone at birth and during minority, as a result of abnormal development of the thyroid gland. Cretinism reasons fatal retardation of physical and mental progress; if the situation is left untreated, progress is stunted and the physical stature progress is that of a dwarf. In addition, the skin is thick, flabby, and waxy in color, the nose is flat, the abdomen protrudes, and there is a common deliberate of movement and speech (Health-Issues, 2011)

Causes

Hypothyroidism in the newborn may be caused by :A missing or abnormally developed thyroid gland .Pituitary gland's failure to stimulate the thyroid defective or abnormal formation of thyroid hormones (Harris, 2007)

Symptoms of Cretinism

Symptoms include execute growth, twined facial features, increased tongue and mental detainment. The typical:

- floppy infant
- thick, protruding tongue
- poor feeding
- choking episodes
- constipation
- prolonged jaundice
- short stature

Diagnosis and Treatment of Cretinism

Advance diagnosis, and life long cure with thyroid hormone by mouth, gives the child better chances of recovering. Nonattendance of thyroid hormone during beginning life leads to a poor outlook in terms of mental progress. Replacement therapy with thyroxine is the common approach to treatment of hypothyroidism. Once medication begins, the blood levels of T3 and T4 are monitored to observed

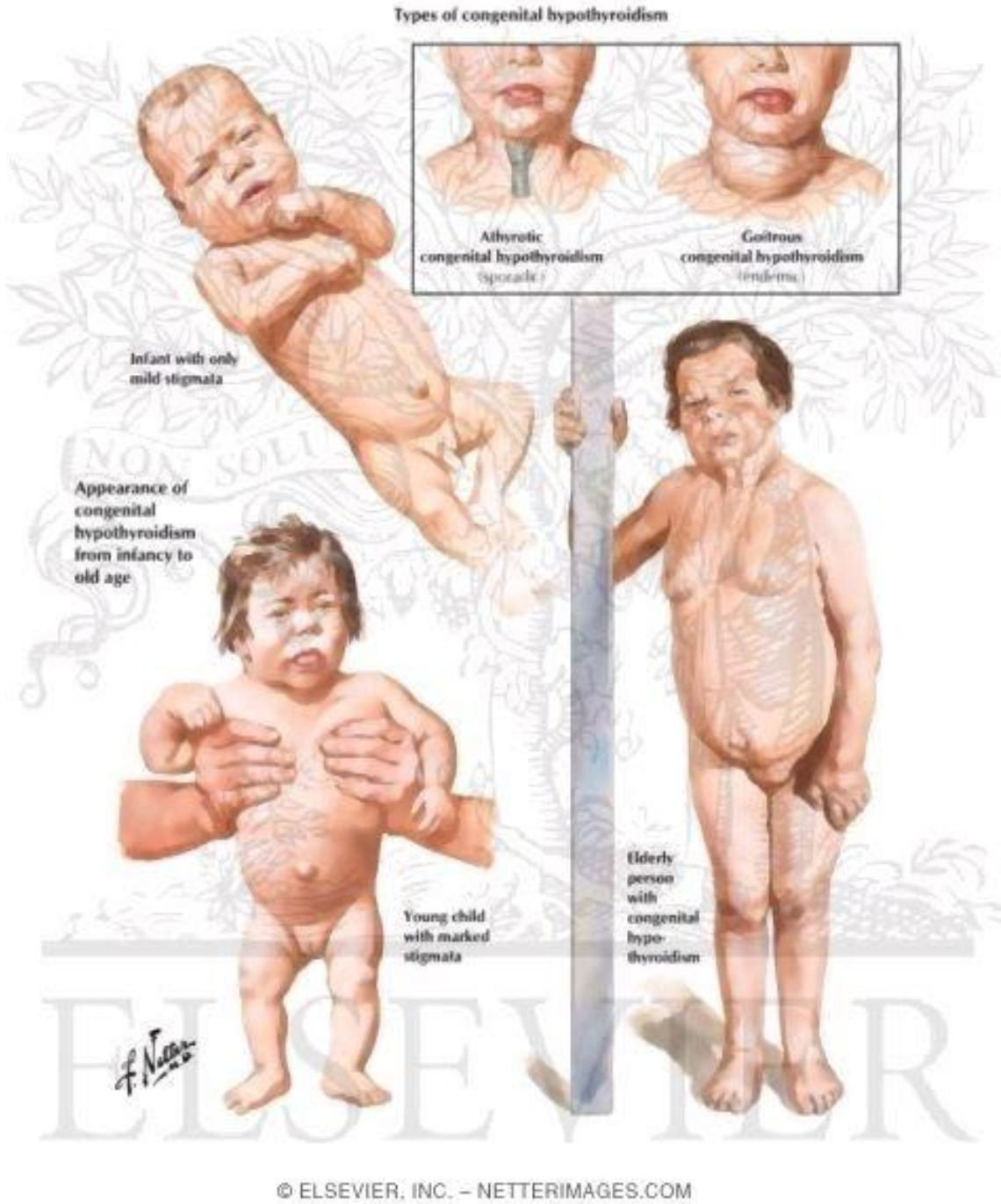
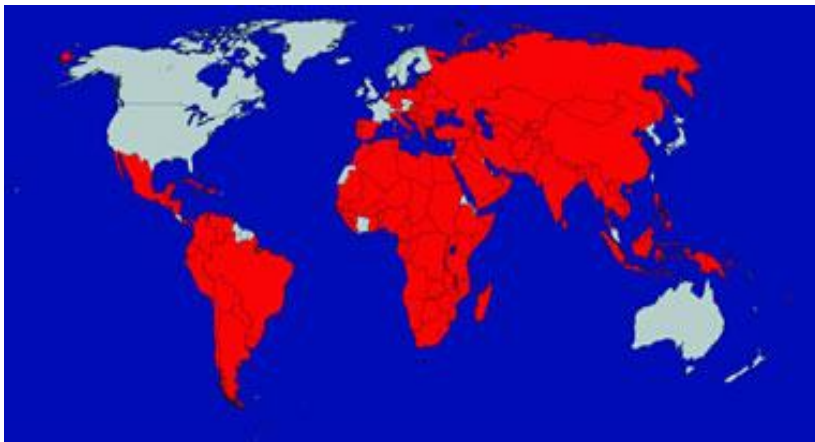


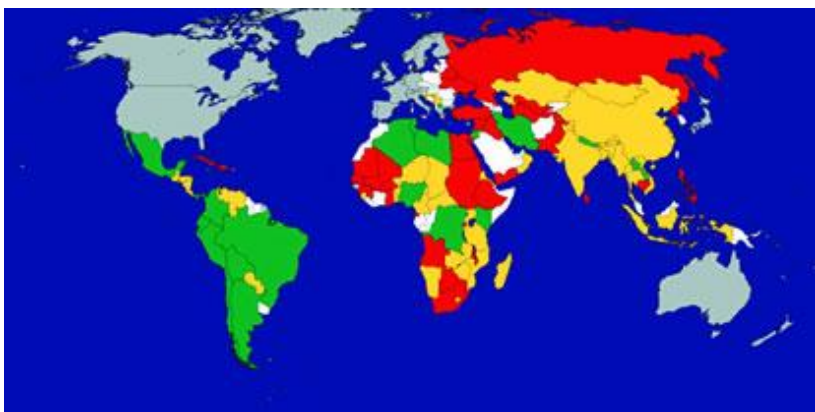
Fig4. Types of Cretinism (Buja & Krueger, 2009)

1.13 Current Status of Iodine Deficiency

The world in 1990



The world in 1998



Fig;5 Prevalence of IDD green: 90% or more yellow: 50-89% red: less than 50% white: no data grey: Countries not assisted by UNICEF *Fig 6. Prevalence of IDD from 1990 to 1998 (Kiwaniis, 2000)*

In the 1990s, 1.6 billion people, one third of the world's population, was at risk for iodine deficiency disorders (IDD); less than 20 percent of people at risk had access to iodized salt. Universal Salt Iodization (USI), the primary strategy to prevent IDD, was adopted in 1993. Since then, more than 90 million newborns are protected each year from learning disabilities caused by IDD (WHO). More than 90 percent of the populations of 21 developing countries use iodized salt. In 1998, nearly 60% of world's edible salt was iodized and there was a significant decrease in number of children born at risk of IDD. The number of cretin births halved to less than 55,000 per year (Kiwani, 1999) By 2000, 70 percent of households in developing countries used iodized salt (Kiwani, 2000). UNICEF has declared that 85 million children will be born free of iodine deficiency disorders (IDD) this year. Because of the global effort to wipe out IDD it is now estimated that more than 70 percent of the world has access to iodized salt, the most practical vehicle for providing iodine in the diet (Kiwani, 2011)

1.14 Iodine Deficiency in Bangladesh

Surveys carried out since 1960s has shown that high levels of iodine deficiency are widespread in Bangladesh. The Nutrition Survey of East Pakistan 1962-1964 reported a goiter rate of 28.9% in former East Pakistan, now Bangladesh. The National Iodine Deficiency Disorder Survey 1993 revealed a goiter rate of 47.1% (Rasheed, et al., 2001)

A study was conducted in eight unions of Chakaria upazilla in the Cox's Bazar district of Bangladesh during 1997-1998 to determine the prevalence of use of iodized salt, explore the reasons behind non-use, and identify the socioeconomic correlates of its use. A quantitative survey was conducted to collect information from 21,190 households on socioeconomic status, demographic characteristics, and the kind of salt used. The results revealed that only 1.9% of the households used iodized salt (Rasheed, et al., 2001)

Another similar study carried out in Rajshahi among 500 mothers, aged 19-49 years showed the prevalence of iodine deficiency disorders in the form of palpable goiter to be 12%. The study revealed that 93% of the mothers were housewife, of which 7% had not heard about iodized salt, 60% had heard about it from television and radio. The majority of the households had open containers to store salt. About 56% of the

households consumed iodized salt. The main cause on not using iodized salt was that it was not bought by their husbands as it was more expensive than non-iodized salt and was not available in nearby shops or markets (Azad, Molla & Bhuyan, 2002)

Another study carried out in Dhaka among college girls aged 16-18 years was done by determining the level of salt iodine, which they were consuming in their households, and also their urinary iodine excretion (UIE), which is an indicator of biochemical iodine deficiency. The results showed that 15.3% of the students had biochemical iodine deficiency. The mean iodine value of salts consumed in the household level was 49 ppm, but as per law, 15 ppm iodine should be present at household level. On the other hand, some salt samples contained no iodine. Only 8.1% of the salt samples contained iodine in the range of 10 to 20 ppm and more than 34% of the salt samples contained iodine greater than standard factory level (Jamal, Salamatullah & Yusuf, 2006)

1.15 Salt Iodization in Bangladesh

In 1989, the Government of Bangladesh passed the Iodine Deficiency Disease Prevention Act. The Act declared universal iodization of edible salt for human and animal consumption and included prevention, enforcement, and education efforts. Under this act, the Bangladesh Council of Scientific and Industrial Research (BCSIR) and other institutions would be responsible for monitoring the quality of iodized salt manufactured and sold from that time onwards. Most salt-crushing units have been provided with iodization equipment, and UNICEF supplies the iodizing agent—potassium iodate—free of charge. Despite this law and this assistance, much of the salt used by the people is not iodized. A survey in 1995 showed that only 30% of iodized salt manufactured in Bangladesh contained an acceptable level of iodine (Khorasani, 1999). Surprisingly, 10% of commercial brands contained no iodine at all. Only 30% of producers were using the recommended level of iodine, 10% were using mixtures of fortifying agents, and 10% were not using any iodine at all. Another survey conducted with UNICEF support in 1997 showed that the situation had not improved (Yusuf, et al., 1996). This survey found that only 57% of salt factories with iodization facilities were in regular production, 7% produced iodized salts only irregularly, and 36% were closed. Of 379 samples collected from 138 factories, only 5% contained adequate amounts of iodine, 46% contained too

little, 1% contained no iodine at all, and 49% contained too much; some contained significantly more than it should, i.e. up to 20 times the recommended amount of iodine. Of 1,104 samples collected from retail outlets, 7% contained no iodine, and only one contained the recommended amount whereas 44% contained too little, and 56% contained a very large excess. Another cross-sectional study conducted in a coastal area in southern Bangladesh, during 1997-1998, comprising 21,190 households revealed that only 1.9% of the households used iodized salt in daily cooking (Rasheed et al., 2001). In the Baseline Survey of the National Nutrition Programme in 2004, 39.5% of households were consuming table salt containing an inadequate concentration of iodine (<15 ppm) (Ahmed *et al.*, 2005).

For two decades, UNICEF has supported salt-iodization as means of preventing iodine deficiency disorders. Although 84 per cent of households now consume iodized salt (up from 70 per cent in 2004), the quality of the salt is often unreliable. UNICEF's current program provides monitoring and technical assistance to salt manufacturers to ensure proper iodization and works to increase public awareness of the importance of consuming adequately iodized salt (UNICEF, 2006)

1.16 Iodine Stability in iodized salt

Elemental iodine readily sublimates and is then rapidly lost to the atmosphere through diffusion. Potassium iodide is less stable than potassium iodate, as it can be oxidized to elemental iodine by oxygen or other oxidizing agents, especially in the presence of impurities, such as metal ions and moisture, which catalyze the reaction. Potassium iodate may be reduced to the elemental iodine by a variety of reducing agents in the salt, such as ferrous ions. Moisture is naturally present in the salt, or is abstracted from the air by hygroscopic impurities such as magnesium chloride. The pH of the condensed moisture on the salt is very much influenced by the type and quantity of impurities present, and this affects the stability of the iodine compounds. Elevated temperatures increase the rates of iodine loss. The levels and types of impurities, moisture content and pH of salt produced for human consumption vary widely. Salt is produced from sea water, rock salt deposits and lake sediments, by solar evaporation of brines and dry or

solution mining of rock salt deposits. Refining processes range from simple washing methods to large-scale mechanized vacuum evaporation systems which require trained operators and rigorous quality assurance. Based on the chemical properties of salt aimed at human consumption, losses of iodine were not unexpected, and there have been numerous published and unpublished studies on iodine stability in salt during the past 75 years. A review of this literature showed that iodate is superior to iodide in terms of stability as a fortificant in salt. Published evidence of the stability of iodine, added in the form of iodate without stabilizers, is relatively meagre, but indicates iodine losses ranging from around 5% to 66% after 12 months. The studies undertaken between 1923 and 1996 are difficult to compare. Variations between rates of iodine loss reflect impurities, moisture content, and processing methods. Conditions of packaging and storage, such as humidity and temperature also affect the final iodine content of the salt, yet these factors were not always clearly defined in earlier studies. Sample sizes and reproducibility of results were not always reported, making it more difficult to assess the statistical significance of results. A comprehensive review of the literature by Kelly (1953) concluded that the stability of iodine in salt is determined by (i) the moisture content of the salt and the humidity of the atmosphere (ii) light, (iii) heat (iv) impurities in the salt (v) alkalinity or acidity (vi) the form in which the iodine is present. He concluded that the iodine content will remain relatively constant if the salt is packed dry with an impervious lining, and kept dry, cool, and away from light. He recommended that iodate be used under adverse conditions such as found in developing countries where the salt being iodized is crude, unprocessed and usually not dried sufficiently. Potassium iodate was stabilized by calcium carbonate in crude sea salt stored in hemp fibre sacks for up to eight months at ambient temperature and relative humidity between 70% and 84%. Only some 3.5% of the added iodine was lost. (Arroyave, et al. 1956) Chauhan et. al., (1992) compared iodine stability over 300 days in common salt iodized with iodate, packed in 5 kg solid high density polyethylene (HDPE) bags or left in open heaps. The relative humidity and temperature varied from 41 - 83% and 30-39 °C respectively. Both the salt packed in HDPE bags and in the open lost 9-10% of the added iodine within the first month, after which values remained practically constant. While the actual magnitude of iodine losses were not well defined, a number of researchers investigated the effects of

stabilizing compounds. Considerable variations in iodine stability were found in control samples without stabilizers. Ranganathan, et al. (1986) found that coarse salt iodized with iodate at "normal" room temperature and humidity showed iodine losses of 20% after 12 months, while samples stabilized with calcium carbonate lost no iodine over an 18-month period. In a later study analyses of five types of Indian salt (including powder and crystal) iodized with iodate showed losses of 28-51% after 3 months, 35-52% after 6 months, and up to 66% after 12 months. (Ranganathan, 1999). Iodine losses from refined solar salt packed in open 1 kg plastic film bags, heated to 130°C for 2.5 hr. to simulate drying, and stored at ambient temperature were reported to be 5.7% after 12 months and 11% after 3 years (Zigong Institute, 1992).

1.17 Factor influencing in loss of iodine from iodized salt

Iodized salts stored in atmospheres of relative humidities of 50 per cent lose smaller quantities of their iodine than salts stored under similar conditions at other humidities. Iodized salts rendered alkaline by the addition of NaHCO_3 lose practically none of their iodine during storage, while neutral salts or salts rendered acid lose appreciable quantities. Salts iodized with KIO_3 lose none of their iodine when stored for extended periods. Exposure of iodized salts to sunlight effects a loss of iodine from neutral or acid salts, only a slight loss from salt rendered alkaline and practically no loss from salts iodized with KIO_3 . Exposure to heat alone effects losses of iodine from acid, neutral and alkaline iodized salts and from salt iodized with KIO_3 in precisely the same order as did exposure to sunlight. Exposure to light and heat simultaneously effects greater losses of iodine from salts of neutral or acid reaction than exposure to light alone or heat alone. In the case of iodized salts of alkaline reaction the losses of iodine are again insignificant or negligible, even though the salts are exposed to light and heat simultaneously. The quantity of iodine liberated from a neutral salt iodized with KI appears to depend on the quantity of KI present. (Arnold H. Johnson and B. L. Herrington August 1927)

1.17.1 The effect of relative humidity on the loss of iodine from iodized salt

Since iodized salts prepared on the range are likely to be stored under conditions differing in the relative humidity of the surrounding atmosphere, an experiment was devised to study this factor. The iodized salts were stored in desiccators in which the relative humidity was controlled by some substance or solution contained in the desiccators. The following relative humidities were maintained: 100, 50, 20, and 0 per cent. The 100 per cent relative humidity was maintained by having water in the desiccators, the 50 and 20 per cent by solutions of KOH, and the 0 per cent by solid CaCl_2 . Potassium hydroxide was used for the intermediate relative humidities; as it would remove any free iodine from the atmosphere and hence would not allow the reaction by which iodine was liberated to stop or slow up because equilibrium had been reached. Data indicate that iodized salt stored over water loses considerable iodine. After May 9, 1926, it was impossible to sample this salt as it had taken up so much water. On February 8, 1927, the whole mass of salt was dissolved in water and the iodine content determined. The salt stored in an atmosphere of 100 per cent relative humidity lost nearly 20 per cent of its iodine during storage from October, 1925, to February, 1927. The salt stored over the solution of KOH of such concentration as to give 50 per cent relative humidity lost no iodine during storage, while the two salts stored in drier atmospheres, over the more concentrated solution of KOH and over CaCl_2 , lost small quantities of iodine. Further observations concerning the loss of iodine from very dry iodized salts will be given later. Fellenberg obtained similar data when he stored dry and moist iodized salts. (Arnold H. Johnson and B. L. Herrington August 1927)

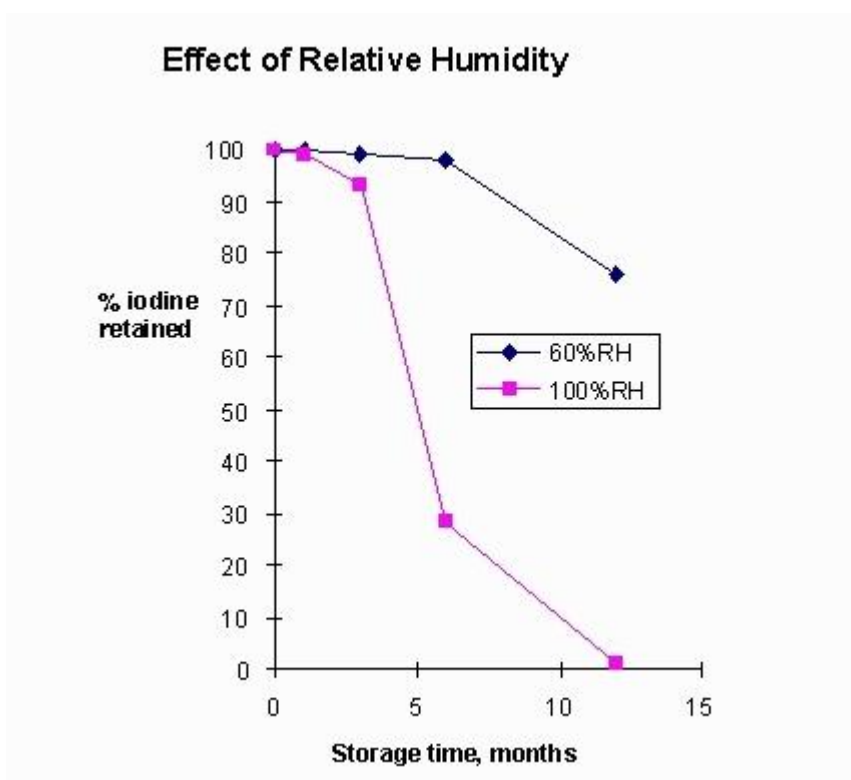


Figure 7. - Effect of relative humidity on iodine retention - Refined salt stored in HDPE

1.17.2 The effect of sunlight on the loss of iodine from iodized salt

Since in the feeding of iodized salts to stock the salts are likely to be exposed to sunlight for various periods of time, and since it is known that sunlight hastens the decomposition of potassium iodide, a study was made of the effect of sunlight on the rate of loss of iodine from iodized salts. The salts were stored in open crystallizing dishes on the sill of a south window and determinations of the iodine content were made periodically* Four salts were used in this study, a salt iodized with finely divided KIO_3 , a salt iodized with KI solution, and two iodized salts which had been recrystallized according to the method used in the preceding section. The most significant conclusion that can be drawn from these data is that salts iodized with KIO_3 retain their iodine when exposed to sunlight, while salts iodized with KI lose considerable portions of it. Thus, the salt which originally contained 0.0620 per cent of KI contained only 0.0165 per cent at the end of 63 weeks. The recrystallized salts show no advantage over ordinary iodized salts in regard to retaining their iodine when exposed to sunlight. Salt was stored in a

crystallizing dish of 4 inches diameter while the other salts were stored in dishes of 7⁴ inches diameter. Hence, a smaller surface of the salt was exposed to the light. This probably accounts for the smaller loss of iodine in this case. Since the surface exposed to the light appeared to be a factor in determining the quantity of iodine lost from an iodized salt, this factor was subjected to further study. Ten gram samples of iodized salt were weighed out on watch glasses and some of the watch glasses containing the salt were placed in dark cupboards, others were placed on the roof in direct sunlight, and still others were placed on the roof in the sunlight but covered with other watch glasses. The losses in iodine which occurred are relatively laid. Thus, exposure of the salt for two days resulted in a loss of 40 per cent of the iodine. When the watch glass containing the salt was covered the loss was less, or only 14.9 per cent. In the experiment in which one day's exposure was allowed the sky was somewhat cloudy. This may account for its lower loss in proportion to the loss for two days. The losses in all cases, however, are relatively large.

1.18 Actions taken to treat Iodine Deficiency Disorders in Bangladesh

Iodine Deficiency Disorders (IDD) can be prevented by consuming tiny amounts of iodine on a regular basis. UNICEF is working with a range of partners in Bangladesh to promote iodization of salt, so that everyone receives enough of this micronutrient in their diet to avert IDD. The main aim is to ensure that the activities are cost-effective and sustainable in the long term. This means

- Maintaining through advocacy the Government's strong political commitment to the Control of Iodine Deficiency Disorders (CIDD) by Universal Salt Iodization (USI).
- Collaboration with and participation of the private sector in sharing the costs of USI and in monitoring the quality of iodized salt.
- Ensuring appropriate salt iodization at factory level and quality maintenance at wholesaler and retailer levels through monitoring, supervising and evaluating salt iodization at all levels, with particular emphasis on the performance of salt factories.
- Strengthening Behavioral Change Communication (BCC) to create more demand for iodized salt among consumers, retailers, wholesalers and producers.
- Intensive monitoring of the enforcement of the salt law (UNICEF, 2005).

1.19 Future plans for sustained IDD elimination

IDD control activities has created a good professional network of various research institutes, government implementing agencies, donor agencies, and private sectors to achieve its goals. This network will be used to strengthen a coordination mechanism for programs for micronutrient deficiency control. USI for CIDD as one of the most extensive fortification efforts in Bangladesh indicates that a large-scale food fortification is possible. This program has provided knowledge and experience that can be utilized in upcoming fortification efforts such as Vitamin A and iron (UNICEF, 2004)

CHAPTER – 2

**SIGNIFICANCE AND OBJECTIVE OF
THE STUDY**

2.1 Significance of the Study

In many situations in developing countries, however, despite improvements in salt production and marketing technology, the quality of available salt is poor, or salt is incorrectly iodized, or salt that has been correctly iodized deteriorates due to excessive or long-term exposure to moisture, light, heat and contaminants. Under these circumstances, iodine losses can be 50% or more from the moment salt is produced until it is actually consumed, and median urinary iodine levels could thus fall below the recommended range (100—200 $\mu\text{g/l}$). In addition, salt consumption is sometimes considerably less than 10 g/person/day. All these factors should be taken carefully into account, particularly when establishing the initial level of iodine in salt (Geneva, 8-9 July 1996).

Recent evidence indicates a wide spectrum of disorders resulting from severe iodine deficiency which puts at risk more than 400 million people in Asia as well as millions in Africa and South America. These iodine deficiency disorders (IDD) include goiter at all ages, with associated impairment of mental function; endemic cretinism characterized most commonly by mental deficiency, deaf-mutism and spastic diplegia and lesser degrees of neurological defect related to fetal iodine deficiency; increased stillbirths, prenatal and infant mortality. Evidence is now available from both controlled trials and successful iodization programs that these disorders can be successfully prevented by correction of iodine deficiency (Hetzel, 1993)

.The social impact of IDD is great. Prevention will result in improved quality of life, productivity, and educability of children and adults. It is now clear that iodine deficiency is a major impediment to human development. Iodized salt and iodized oil (by injection or by mouth) are suitable for correction of the condition on a mass scale. Alternative vehicles for iodine supplementation are required. Prevalence of Iodine Deficiency Disorder in Bangladesh has been reported to be decreasing over the years, as can be seen in this study. As simple as it is, Iodine Deficiency can be prevented by taking a single spoonful of iodine every day. However, as the situation has gone out of hand, additional aid has been required. UNICEF, WHO ICDDR, and various institutions have all been contributing efforts to improve the situation. Universal salt iodization is the recommended intervention for preventing and correcting iodine deficiency. In the past, recommendations for iodine levels in salt were made on the assumption that, from

producer to consumer, iodine losses from iodized salt were commonly between 25% and 50%, and that average salt intakes were commonly between 5 and 10 g/person/day. Substantial experience has been gained in the last decade in implementing universal salt iodization and assessing its impact on iodine deficiency disorders (IDD) (1). A major achievement is the spectacular reduction of IDD in countries that have implemented universal salt iodization (Geneva, 8-9 July 1996).

The use of iodized salt has dramatically increased, from 20 per cent of households in the early 1990s to 84 per cent in 2006. As a result, the prevalence of goiter has reduced significantly, from 49.9 per cent of children in the mid-90s to 6.2 per cent in 2005. The main challenge now is ensuring that salt is adequately iodized. A recent national survey found that only 51 per cent of the salt samples tested contained adequate iodine (UNICEF, 2009).

This study may help to find out branded and open market salt having poor quality and to take step against of the selling of those salts as a result most of the people of our country will get iodized salts.

2.2 Aim and Objectives of the Study:

The aim of the study is to determine the iodine content in different brands of the table salts collected from different areas of Bangladesh.

Objectives:

- To determine the concentration of iodine present in widely used brand salt in different areas of our country.
- To compare the quality salt of different brands.

CHAPTER – 3

MATERIALS AND METHODS

3.1 Study design:

The study was designed to determine the iodine content in branded table salts and collected from different area of Bangladesh.

3.2 Study period: Study period was August to December in 2011

3.3 Samples

The samples for this study are seven branded and three open market salt collected from different shop in Dhaka city and also from outside of Dhaka .

3.4 Chemicals and reagents used

- Potassium Dichromate
- Sodium Thiosulphate
- Potassium Iodide
- Sodium Bicarbonate
- Distilled Water
- Concentrated Hydrochloric Acid
- Starch
- Sulfuric Acid

3.5 Glass apparatus/Accessories

- 50 ml Volumetric Flask
- 500 ml Volumetric Flask
- Conical Flasks
- Watch Glass
- Measuring Cylinder
- Burette and Stand
- Pipette
- Beaker
- Dropper
- Pipette Pump
- Spatula

3.6 Equipments/Machineries

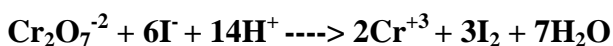
- Electronic Balance
- Hot Plate
- Distillation Equipment

3.7 Methods

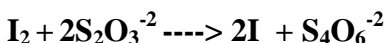
3.8 Preparation of Standard Solutions and Reagents

Principle

The objective of the preparation was to standardize Sodium Thiosulphate using Potassium Dichromate. An 'indirect iodine method' was used to standardize the sodium Thiosulphate. The first step in the reaction involved oxidation of iodide to iodine using potassium dichromate.



This iodine was then reduced to iodide using sodium Thiosulphate which formed a light green solution of tetrathionate.



Starch was used as an indicator in this reaction as unreacted I_2 will form a deep blue complex with the starch.

Preparation of a 50ml solution of 0.1N $\text{K}_2\text{Cr}_2\text{O}_7$

Molecular weight of $\text{K}_2\text{Cr}_2\text{O}_7 = 294\text{mg}$

Equivalent weight of $\text{K}_2\text{Cr}_2\text{O}_7 = 49\text{mg}$

Calculation:

For 1000ml of 1N equivalent weight = 49mg

For 1ml of 1N equivalent weight = 49/1000 gm

Therefore, for 50ml of 0.1N equivalent weight = $(49 \times 50 \times 0.1) / 1000 \text{ gm} = 0.245\text{gm}$

0.245gm of $K_2Cr_2O_7$ was accurately measured on an electronic balance, and placed into a 50ml volumetric flask with the help of a funnel. Distilled water was then added to flask in a small amount and then shaken until the solute dissolved. The flask was then filled with distilled water, up to the 50ml mark.

Preparation of 500ml of 0.1N $Na_2S_2O_3$

Molecular weight of $Na_2S_2O_3 = 248\text{mg}$

Equivalent weight of $Na_2S_2O_3 = 248\text{mg}$

Calculation:

For 1000ml of 1N equivalent weight = 248mg

For 1ml of 1N equivalent weight = $248/1000$ gm

Therefore, for 500ml of 0.1N equivalent weight = $(248 \times 500 \times 0.1)/1000\text{gm} = 12.5\text{gm}$

12.5gm of $Na_2S_2O_3$ was accurately measured on an electronic balance, and placed into a 500ml volumetric flask with the help of a funnel. Distilled water was then added to flask in a small amount and then shaken until the solute dissolved. The flask was then filled with distilled water, up to the 500ml mark.

Standardization of Sodium Thiosulphate ($Na_2S_2O_3$)

1gm of KI and 1gm of $NaHCO_3$ were placed in a conical flask. 25ml of water and 10ml of potassium dichromate were then added to the flask. The flask was swirled to mix the contents. 5ml of HCl (conc.) was then added to the conical flask. The flask was covered with a watch glass and the contents are gently mixed. The flask was kept in the dark for 7 - 8 minutes. After the specified time, the flask was taken out of the dark, gently swirled to mix, and the sides were rinsed with a small amount of distilled water. The next step was to place the Sodium Thiosulphate in a 50ml burette, and set it up on a stand using a clamp. The $K_2Cr_2O_7$ was then titrated against $Na_2S_2O_3$. As the titration proceeds, the color of the dichromate solution turned from a deep brown to a lighter color. Once the color started turning lighter, two to three drops of starch solution (1%) was added to the flask. The starch imparted a blue black color to the solution since there was the presence

of iodine. At the end point of the titration, the solution in the flask turns light green as all the iodine liberated had reacted with the thiosulfate. To accurately determine the end point of the titration, one or two drops of starch solution (1%) can be added to the conical flask again. Any unreacted iodine will color the solution dark blue. The process was repeated two more times. The average volume of $\text{Na}_2\text{S}_2\text{O}_3$ required for titration was then determined to calculate the strength.

Table 4. Standardization of $\text{Na}_2\text{S}_2\text{O}_3$

Observation No.	Initial Burette Reading (ml)	Final Burette Reading (ml)	Difference (ml)	Average (ml)
1	0.00	10.00	10.00	
2	10.00	20.20	10.20	10.10
3	20.20	30.10	10.10	

The strength of the $\text{Na}_2\text{S}_2\text{O}_3$ is calculated using the following formula:

Volume of $\text{Na}_2\text{S}_2\text{O}_3$, $V_1 = 10.10$ ml

Strength of $\text{K}_2\text{Cr}_2\text{O}_7$, $S_2 = 0.1$ N

Volume of $\text{K}_2\text{Cr}_2\text{O}_7$, $V_2 = 10$ ml

$$S_1 V_1 = S_2 V_2$$

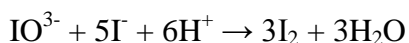
$$S_1 = (0.1 \times 10)$$

$$= 0.099\text{N}$$

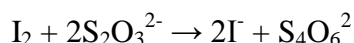
3.9 Determination of Iodine content in salt

Principle

In earlier times salt was “iodized” by the addition of potassium iodide; however, nowadays iodine is more commonly added in the form of potassium iodate (KIO₃). This potassium iodate can be determined by the process of redox titration. In this method the amount of iodate (IO₃⁻) in iodized salt is determined by first reacting the iodate added iodide (I⁻), under acid conditions, to produce iodine:



Then the resulting iodine is titrated with Thiosulphate as follows:



Preparation of Sodium Thiosulphate

From the calculated strength of the standardized Sodium Thiosulfate, the volume required to react with each mole of iodate was determined as follows:

Volume of Na₂S₂O₃ required to titrate iodate, V₁ = ?

Strength of Na₂S₂O₃, S₁ = 0.099N

Volume of Diluted Na₂S₂O₃, V₂ = 100ml

Strength of Diluted Na₂S₂O₃, S₂ = 0.005N

$$S_1V_1 = S_2V_2$$

$$V_1 = (0.005 \times 100) / 0.099$$

$$= 25.25\text{ml}$$

Amount of water required to dilute Na₂S₂O₃ = 100 - 25.25 = 74.75ml

Preparation of 1% starch indicator solution

1g of soluble starch was weighed and placed into a 100ml conical flask or beaker. The starch was then dissolved in 100ml of water by heating and stirring the solution at 79°C, for 5 minutes, being careful not to exceed the specified temperature. The solution was then allowed to cool to room temperature.

Preparation of 10% Potassium Iodide

10g of Potassium Iodide was weighed and then made up to 100ml with distilled water in a volumetric flask.

Preparation of Sulfuric Acid (about 2N)

A small amount of water was added to a 50ml volumetric flask. 2.8ml of Sulfuric Acid (conc.) was measured out by a pipette and slowly added to the water in the flask. The solution was then filled with water up to the 50ml mark.

Titration

10g of salt was weighed using electronic balance and placed into a conical flask. To the flask, 50ml of water, 5ml of 10% KI and 1ml of H_2SO_4 were all added, one by one. The solution turned a yellow/brown color, as iodine was produced. The solution was then titrated against the Standardized and Diluted $\text{Na}_2\text{S}_2\text{O}_3$ until the yellow/brown color became very pale. Then, 2-3 drops of Starch indicator solution was added, which produced a dark blue-black colored complex with iodine. The titration was continued until the color completely disappears. The process was repeated two more times and an average value for the volume of $\text{Na}_2\text{S}_2\text{O}_3$ was determined.

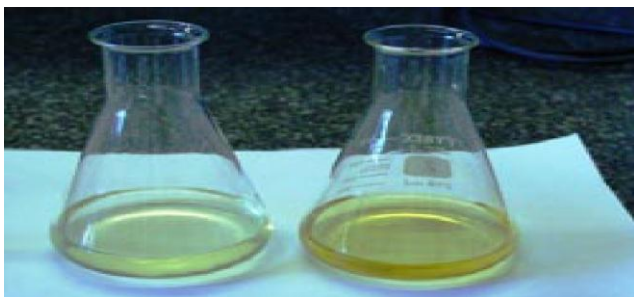


Fig.8. Right flask: yellow/brown color of iodine formed from reaction of iodated from salt with acidic iodide solution. Left flask: pale yellow color left when nearly all iodine has reacted with added thiosulfate during titration.

After addition of starch



Fig9. Right flask: yellow/brown solution containing last trace of iodine. Left flask: dark blue-black color formed when starch indicator is added to solution containing iodine near endpoint

After addition thiosulfate



Figur 10. A series of flasks showing the color change as the last remaining iodine (with added starch indicator) is titrated with thiosulfate. The dark blue-black color disappears, leaving a colorless solution at the endpoint.

CHAPTER – 4

RESULTS

4.1 Calculation

From the average volume of $\text{Na}_2\text{S}_2\text{O}_3$ determined, the number of ppm of iodine in the salt samples was calculated with the following formula:

$$\text{Iodine ppm} = (\mathbf{R} \times \mathbf{100} \times \mathbf{1000} \times \mathbf{0.127} \times \mathbf{N}) / \mathbf{6}$$

Where,

R = Average volume of $\text{Na}_2\text{S}_2\text{O}_3$

100 is to convert the reading for 1000g of salt

1000 is to convert gram of iodine to milligram of iodine

0.127 is the weight of iodine equivalent to 1ml of normal Thiosulphate solution

N is normality of Thiosulphate solution (which is 0.005N)

6 is to arrive at the value that corresponds to 1 atom of iodine liberated (Srivastava, 2006)

4.2 Result

Following the standard procedure, as mentioned before, the following results were obtained for each sample collected:

Table5. Iodine content for Super Salt

Sample Code	Observation No.	Initial Burette Reading (ml)	Final Burette Reading (ml)	Difference (ml)	Average	Iodine (ppm)
	1	0.00	3.5	3.5		
001	2	3.5	8.0	4.5	4.16	44.022
	3	8.00	12.5	4.5		

From this result it is seen that super salt contains iodine above minimum level.

Table6. Iodine content for Confident Salt

Sample Code	Observation No.	Initial Burette Reading (ml)	Final Burette Reading (ml)	Difference (ml)	Average	Iodine (ppm)
	1	0.00	3	3.00		
002	2	3	6	3.0	2.93	31.009
	3	6	8.8	2.8 0		

From this result it is seen that confident salt contains iodine above minimum level.

Table7. Iodine content for Ifad Salt

Sample Code	Observation No.	Initial Burette Reading (ml)	Final Burette Reading (ml)	Difference (ml)	Average	Iodine (ppm)
	1	0	2.4	2.4		
003	2	2.7	5	2.3	2.7	28.575
	3	5	7.4	3.4		

From this result it is seen that Ifad salt contains iodine above minimum level.

Table8. Iodine content for Brac Salt

Sample Code	Observation No.	Initial Burette Reading (ml)	Final Burette Reading (ml)	Difference (ml)	Average	Iodine (ppm)
	1	8	12.3	4.3		
004	2	12.3	17.1	4.8	5.00	52.91
	3	17.1	23.0	5.9		

From this result it is seen that Brac salt contains iodine above minimum level.

Table9. Iodine content for Molla Salt

Sample Code	Observation No.	Initial Burette Reading (ml)	Final Burette Reading (ml)	Difference (ml)	Average	Iodine (ppm)
	1	13	16	3.0		
005	2	16	19.6	3.60	3.30	35.270
	3	20	23.5	3.50		

From this result it is seen that Molla salt contains iodine above minimum level.

Table10. Iodine content for Surza Salt

Sample Code	Observation No.	Initial Burette Reading (ml)	Final Burette Reading (ml)	Difference (ml)	Average	Iodine (ppm)
	1	23	23.9	.9		
006	2	23.9	24.6	.7	0.73	7.76
	3	24.6	25.2	.6		

From this result it is seen that Surza salt contains iodine below minimum level.

Table11. Iodine content for ACI Salt

Sample Code	Observation No.	Initial Burette Reading (ml)	Final Burette Reading (ml)	Difference (ml)	Average	Iodine (ppm)
	1	27	30.5	3.5		
007	2	30.5	34.5	3.5	3.5	37.04
	3	34.5	37.5	3.5		

From this result it is seen that ACI salt contains iodine above minimum level.

Comparison of different branded table salt on iodine content

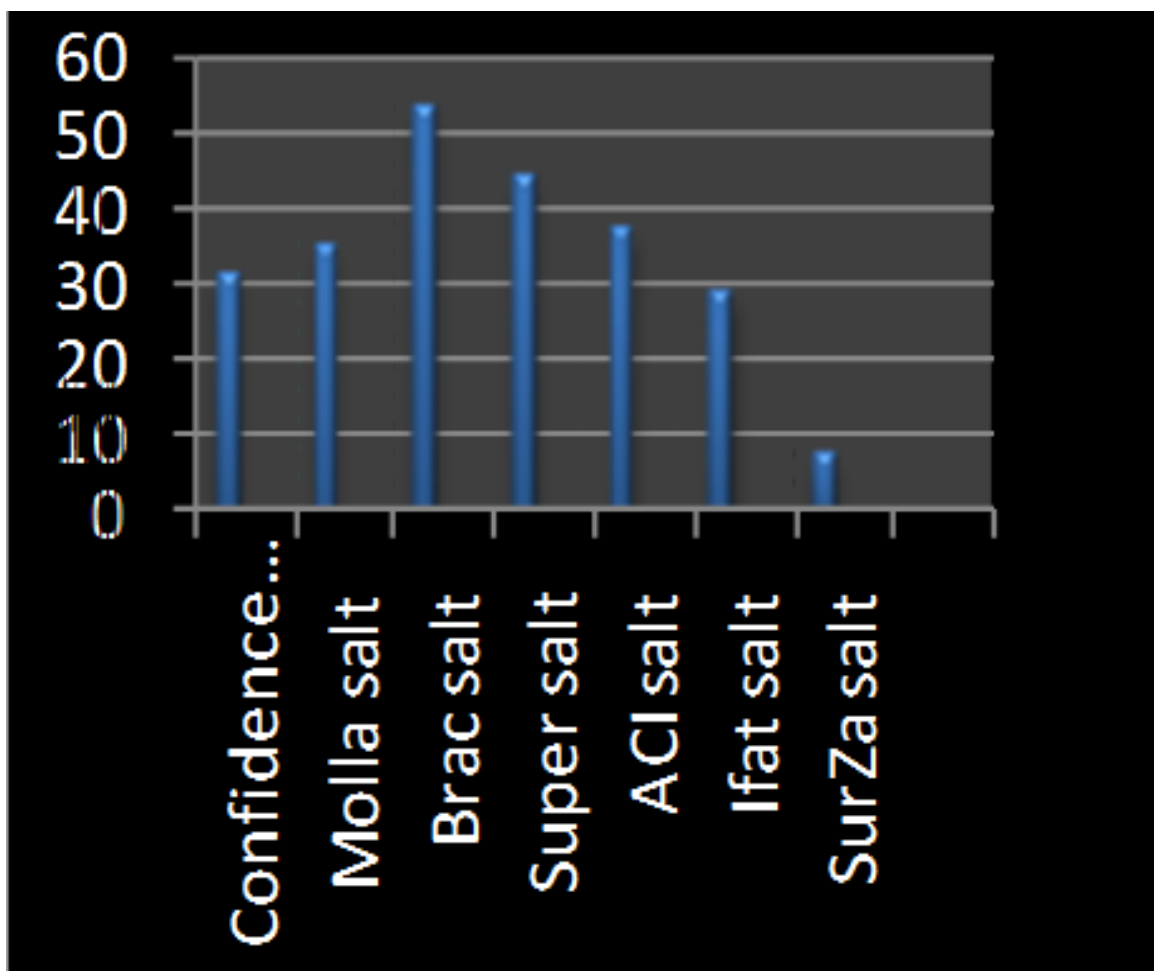


Fig 11: Comparison of different branded table salt on iodine content .From this diagram it is seen that most iodine containing brand is Brac salt and least iodine containing brand is surza salt (less than minimum level).

CHAPTER- 5

DISCUSSION

5.1 Discussion

Iodine deficiency disorders (IDD) are recognized as a major global public health problem. According to the recent estimates, about 2.5 billion people worldwide (38% of the world's population) have insufficient iodine intake, of which 313 million are in the South-eastern Asian region that includes Bangladesh (WHO, 2005). Iodine deficiency is the single most preventable cause of neurological and intellectual impairment (cretinism) in the world. The Government of Bangladesh is officially committed to IDD elimination through national, as well as international, commitments. In 1989, the Government of Bangladesh passed a law making it mandatory that all edible salt be iodized. The law stipulates that all salt for human consumption must contain 45-50 parts per million (ppm) of iodine at the time of production and not less than 20 ppm iodine at the time of retail, to ensure a minimum of 15 ppm iodine at the household level. Accordingly, a plan was undertaken to institute a Universal Salt Iodization (USI) in the country.

Little progress was made in salt iodization until the results of the first National IDD Survey in 1993 were published. The survey revealed a very high prevalence of total goiter (47.1%), visible goiter (8.8%), cretinism (0.5%), and biochemical iodine deficiency (68.9%) as indicated by a low urinary iodine excretion ($<100 \mu\text{g/L}$) (Dhaka University, ICCIDD & UNICEF, 1993). Following the release of the survey findings, the Salt Bye-Law was passed in 1994 and large investments were made in the infrastructure for salt iodization. By 1995, all 267 registered salt factories in the country were equipped with Salt Iodization Plants (SIP) with UNICEF assistance, and iodized salt became available throughout the country by the beginning of 1995. Two National USI Surveys were conducted to assess the progress in achieving USI, the first in 1996 and the second in 1999. The surveys revealed that during the period 1996-9, the percentage of adequately iodized salt at household level ($=15 \text{ ppm}$) was maintained at 54 to 55%. In 1999, the first follow-up National IDD Survey was conducted to measure progress in eliminating IDD (Dhaka University, IPHN, BSCIC, UNICEF & ICCIDD, 2001). This survey showed that the substantial investments in salt iodization and resulting increase in consumption of iodized salt by households resulted in a remarkable reduction in the prevalence of IDD. Between 1993 and 1999, the total goiter rate among the population fell from 47.1% to 17.8% and biochemical iodine deficiency fell from 68.9% to 43.1%. As part of the

roadmap to eliminate iodine deficiency in Bangladesh, a National IDD and USI Survey was again conducted in 2004-05 to monitor the situation. Nationally, 81.4% of household salt was iodized (iodine content =5 ppm), and 51.2% of household salt was adequately iodized (iodine content =15 ppm). Between 1993 and 2004-05, TGR decreased from 49.9% to 6.2% in children, and from 55.6% to 11.7% in women. Median UIE increased between 1993 and 2005 among both children and women, and there was a concomitant decrease in iodine deficiency. In children, UIE increased from 54 µg/L in 1993 to 126 µg/L in 1999 and further to 162µg/L in 2005, and accordingly iodine deficiency decreased from 71.0% in 1993 to 42.5% in 1999 and then to 33.8% in 2005. In women, UIE increased from 47 µg/L in 1993, to 111µg/L in 1999 and to 140 µg/L in 2005, while iodine deficiency decreased from 70.2% in 1993 to 45.6% in 1999 and to 38.6% in 2005 (Yusuf, *et al.*,2008).

If all the substantial research-work mentioned above is brought into comparison with this trivial effort to determine the current situation of IDD in Dhaka City, the progress mentioned is evident. Even most of the salts collected show iodine levels to be in the acceptable range (iodine content = 15 ppm). Out of the 7 samples, only one has shown to have poor content of iodine, but that may also be due to long exposure times to moisture or heat while working in the laboratory. Inaccuracies and oversights due to faulty equipments or while working, should also be taken into consideration. we can predict that most of the people of our country were not deprived of the required amount of iodine in salts. . Overall the study portrays that the effort made by various organizations and the government, for decades, is aiding to wipe out IDD from the nation. Since the situation has been taken seriously by the authorities, it has been possible to supply iodized salt to the all levels of people

CHAPTER- 6

CONCLUSION

6.1 Conclusion

The causes, prevention and cure of goiter and cretinism, now included in the more general term iodine deficiency disorders (IDD), have been known for more than half a century; yet their total eradication remains an elusive goal. We now know that iodine deficiency causes a spectrum of effects on growth and development, particularly brain development in the foetus, neonate and child, justifying a much higher priority now for its prevention and control than in the past. Apart from diminishing the toll in human misery, the prevention of IDD would mean improved educability of children, greater productivity, and better quality of life for many millions living in the iodine-deficient regions of the world. It is now clear that iodine deficiency is a major impediment to human development (Hetzel, 1993).

Bangladesh has achieved a commendable progress in reducing prevalence of goiter and iodine deficiency. Progressing at this rate, may bring the target of eliminating IDD from the country closer in no time. Although the study carried out in some widely used braded salt which is too minute to conclude that the rate of IDD is decreasing in the nation, but the results showing that almost all the salts containing iodine above the minimum level make it quite marked. Despite of most of people of our country live below poverty line, it can be seen from the study, that the people are now using iodized salts.

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