Determination of iodine content of household salts in the area of Srimangal in Bangladesh.

A Thesis Paper submitted to the Department of Pharmacy, East West University in conformity with the requirements for the degree of Bachelor of Pharmacy.

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December, 2017

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Declaration by the Research Candidate

I, Afsana Haque Mim , hereby declare that the dissertation entitled **"**Determination of iodine content of household salts in the area of Srimangal in Bangladesh**"** submitted by me to the Department of Pharmacy, East West University and in the partial fulfillment of the requirement for the award of the degree Bachelor of Pharmacy, work carried out by me during the period 2017 of my research in the Department of Pharmacy, East West University, under the supervision and guidance of Sufia Islam, Professor Department of Pharmacy, East West University. The thesis paper has not formed the basis for the award of any other degree/diploma/fellowship or other similar title to any candidate of any university.

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This is to certify that the thesis entitled **"**Determination of iodine content of household salts in the area of Srimangal in Bangladesh**"** submitted to the Department of Pharmacy, East West University for the partial fulfillment of the requirement for the award of the degree Bachelor of Pharmacy, was carried out by Afsana Haque Mim, ID: 2014-1-70-063, during the period 2017 of her research in the Department of Pharmacy, East West University, under the supervision and guidance of me. The thesis has not formed the basis for the award of any other degree/diploma/fellowship or other similar title to any candidate of any university.

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

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Dedication

This Research Paper is Dedicated
To
My Beloved Parents

Abstract

IDD (Iodine deficiency disorders) are recognized as a major global public health problem. Insufficient iodine intake was observed in about 2.5 billion people worldwide. Among them 313 million are from South-eastern Asian region including Bangladesh. Many people suffers from IDD in Bangladesh. To ensure adequate iodine intake, salt iodinization is an effective strategy which is safe, sustainable and cost-effective as well. The objective of the study was to determine the iodine concentration of household salts collected from 20 households of tea plantation worker from Srimangal area of Bangladesh. 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. According to WHO guidline the acceptable range of iodine content of salt is 20-50 ppm. Out of 20 samples, 7 did not show the iodine content within the standard range. Evidence is now available from both controlled trials and successful iodization programs that IDD disorders can be successfully prevented by correction of iodine deficiency. Inadequate iodine fortification in correcting & preventing IDD still persists in household in Srimangal area. Appropiate measurement should be taken in salt iodinization in commercially available salts in Bangladesh.

Keyword:- Iodine deficiency disorders, Iodinization, Household salts

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CHAPTER -1

INTRODUCTION

1.1 Iodine

Iodine is a chemical element with symbol I and atomic number 53. The heaviest of the stable halogens, it exists as a lustrous, purple-black metallic solid at standard conditions that sublimes readily to form a violet gas. The first description of iodine was by Bernard Courtois in 1811, when he saw a violet vapor arising from sea weed ash during the manufacture of gun powder for Napoleon's army. The name *iodine*, from the Greek for "violet," was subsequently suggested by Joseph Louis Gay-Lussac. In 1895, Eugen Baumann identified iodine in thyroid glands and by 1917, it was understood that thyroid gland enlargement (goiter) was caused by iodine deficiency and could be prevented by iodine supplementation (Zicker 2012).

Iodine is a micronutrient of crucial importance for the health and well-being of all individuals. It is a trace element, just 5 gm of which are sufficient to meet the life-time needs of an individual with a life-span of 70 years. Iodine is mostly concentrated in thyroid gland. A healthy adult body contains 15-20 mg of iodine, 70-80% of which is stored in the thyroid gland. Daily intake of iodine by an individual amounts to 500 micrograms; daily physiological requirement during adult life is 150 micrograms; during pregnancy and lactation period is 200 micrograms; and during neonatal period is 40 micrograms. The thyroid gland normally takes up about 120 micrograms of iodide for the synthesis of thyroid hormones. The body does not make iodine, so it is an essential part of diet. If there is insufficient iodine in our body, enough thyroid hormone is not making (Ahad and Shafiq 2010).

1.2 Sources of iodine

Iodine is mostly obtained from food sources particularly vegetables grown on iodine-rich soil; the remaining requirement is met from drinking water. Seaweeds such as wakame, nori or mekabu, which are widely used in some Asian cultures for making soups, salads and condiments, are rich sources of iodine. Iodine is found in nature in various forms: inorganic sodium and potassium salts (iodides and iodates); inorganic diatomic iodine(molecular iodine or I), and organic monoatomic iodine (Ahad and Shafiq 2010).

Table 1.1: Common Dietary Sources of Iodine (Mary Ann Liebert, Inc. Publisher 2016)

Food	Iodine content (µg per 100g)
Breads (made with iodised salt)	46
Cheddar Cheese	23
Cow's Milk	13
Eggs	22
Frozen Yogurt	16
Ice-Cream	21
Tap water (varies depending on site)	0.5-20.0
Apples, oranges, grapes, bananas	<0.5

1.3 Geographical distribution of iodine

Iodine occurs widely in trace amounts, mainly as iodide salts, with local concentrations depending on geologic and water conditions. Generally, the highest concentration of iodide (50 to 60 µg/L) is found in seawater; the concentration in fresh water is1to10µg/L. Iodide in seawater oxidizes and sublimes when exposed to air and is transported inland, where it is dissolved into water droplets that fall as rain. Leaching due to glaciation, flooding, and erosion depletes surface soils of iodide, resulting in most mountainous and some interior in land regions being deficient. Crops grown in these areas may be 10-fold lower in iodine content (~10 µg/kg) than those from iodine-sufficient regions (Zicker 2012).

1.4 Metabolism of Iodine

Iodine trapping is the first step in the metabolism of iodine. The process commences with the uptake of iodide from the capillary into the follicular cell of the gland by an active transport system. This occurs against chemical and electrical gradients by sodium / iodine symported protein (NIS) found in the basolateral membrane of the follicular cell; the

energy required by this process is linked to the ATPase dependent Na^{+};- K pump (Ahad and Shafiq2010).

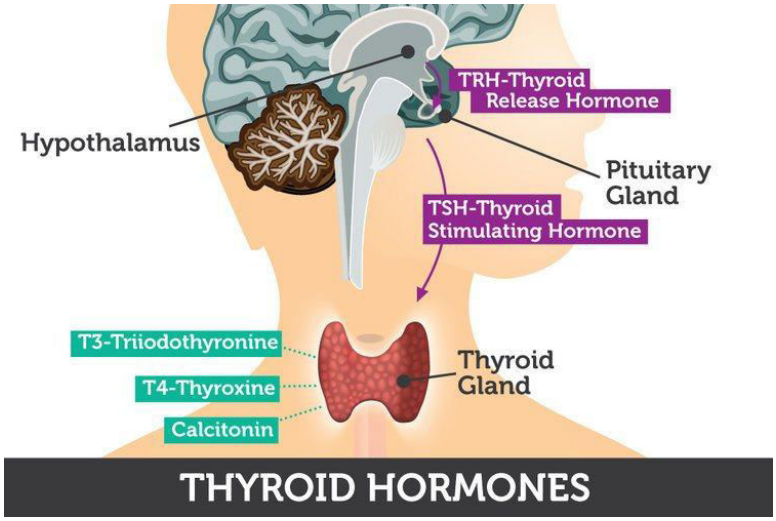


Fig 1.1: Thyroid hormone

1.5 Thyroid gland

1.5.1 Definition of Thyroid Gland

The thyroid is a small, butterfly-shaped gland located at the front of your neck, sort of like an internal bowtie. The thyroid consists of two lobes joined together by a narrow band of thyroid tissue called the isthmus. These lobes are themselves made up of many small lobules joined together with connective tissue.

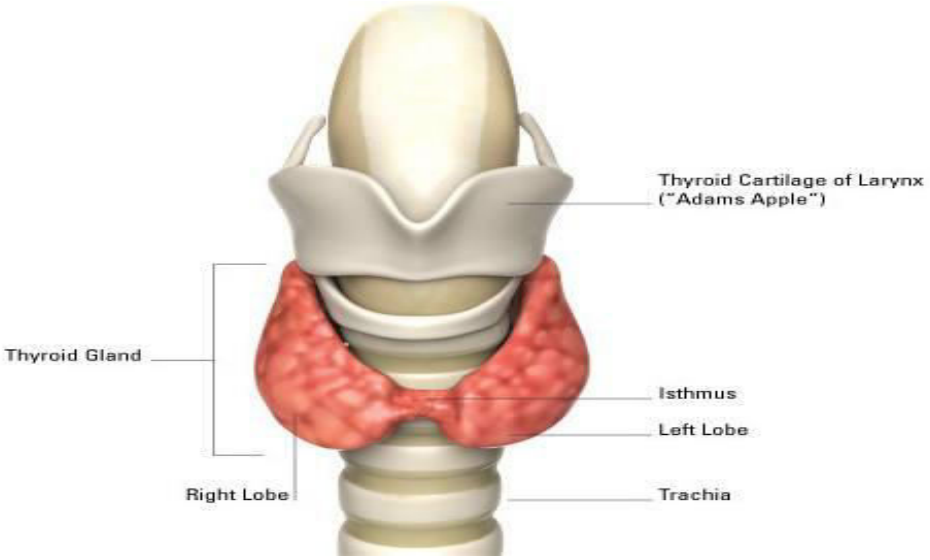


Fig1.2: Thyroid gland

Thyroid gland secretes 80 micrograms of iodine in the form of T3 and T4 hormones per day; 40 micrograms of iodine secreted appear in extracellular fluid (ECF) per day. T3 and T4 are metabolized in liver which releases about 60 micrograms of iodine into ECF and 20 micrograms of iodine into the bile to be excreted in stools. On an average, 480 micrograms of iodine get excreted in urine and 20 micrograms in stools per day (Ahad and Shafiq 2010).

1.5.2 Thyroid hormone synthesis

Iodide is removed from circulating blood primarily by the thyroid gland and kidneys. The body can also concentrate iodide in the salivary glands, breast tissue, gastric mucosa, and choroid plexus, among other sites. Sodium=iodide transporters—protein molecules also known as ‘‘symporters’’—take up iodide from the blood into the thyroid gland across a concentration gradient that may be as high as 50-fold, and concentrate the iodide in the cells of the gland to a level adequate for hormone synthesis. This iodide is incorporated into precursors that are transformed into thyroxine, or T4, a hormone secreted primarily by the thyroid, which is converted in peripheral tissues to the hormone triiodothyronine (T3), which regulates growth and cellular metabolism (Mary Ann Liebert, Inc. Publisher 2016)

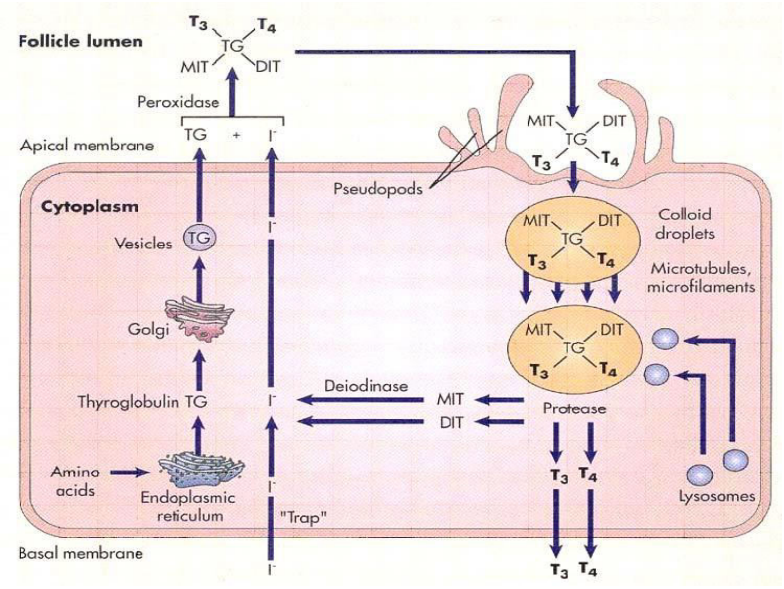


Fig 1.3: Synthesis of thyroid hormone

1.5.3 Distribution and elimination

Absorbed iodide is distributed through the extracellular space with a half-life of approximately 10 h in plasma. The half-life may be shortened in iodine- deficient or hyperthyroid animals due to more rapid thyroid uptake and in- creased GFR. When dietary iodide intake is abundant, approximately 90% of ingested iodide is excreted in urine and there main derinfeces. Renal iodide clearance remains constant as a percent age of filtered iodide in plasma, even in response to variable iodine intake. This results in decreased urinary iodide when iodine intake is low and increased urinary iodide when intake is high. Biliary conjugation of thyroxine may occur, with concomitant fecal elimination almost entirely as conjugated thyroid hormone (Zicker 2012).

1.5.4 Role of iodine in thyroid physiology

Iodine is a trace element in soil and water that is ingested in several chemical forms. Most forms of iodine are reduced to iodide in the gut. Iodide is nearly completely absorbed in the stomach and duodenum. Iodine is cleared from the circulation primarily by the thyroid and kidney. Under normal circumstances, plasma iodine has a half-life of approximately 10 hours, but this is shortened if the thyroid is overactive, as in iodine deficiency or hyperthyroidism. The mean daily turnover of iodine by the thyroid is approximately 60-95 µg in adults in iodine-sufficient areas. The body of a healthy adult contains from 15 to 20 mg of iodine, 70%-80% of which is in the thyroid. In the basolateral membrane of the thyroid cell, the sodium/iodine symporter (NIS) transfers iodide into the thyroid across a concentration gradient 20-50 times that of plasma by

active transport. Degradation of T4 and T3 in the periphery releases iodine that re-enters the plasma iodine pool. Most ingested iodine is eventually excreted in the urine. Only a small amount appears in the feces. The mammary gland concentrates iodine and secretes it into breast milk to provide for the newborn. The salivary glands, gastric mucosa, and choroid plexus also take up small amounts of iodine. The NIS and pendrin have been reported in trophoblasts, and the placental iodine content is approximately 3% that of the thyroid(Chung 2014).

1.5.5 Control of the thyroid by iodine

Iodide is known to control thyroid function. Its main effects are to decrease the response of the thyroid to thyrotropin (TSH); to acutely inhibit its own oxidation; to reduce its trapping after a delay; and, at high concentrations, to inhibit thyroid hormone secretion. Small changes in iodine intake are sufficient to reset the thyroid system at different serum TSH levels. This suggests that modulation of the thyroid response to TSH by iodide plays a major role in the negative feedback loop. In response to increasing doses of iodide, iodine organification increases initially and then decreases.

In vitro, iodide has been reported to inhibit various metabolic steps in the thyroid cell. Iodide inhibits the cyclic adenosine monophosphate cascade and the Ca^{2+} -phosphatidylinositol 4, 5-bisphosphate (PIP_2) cascade. Iodide also activates H_2O_2 generation and thus protein iodination in the thyroid of some species, including humans (Chung 2014).

1.5.6 Role of iodine in Healthy Pregnancy

Women who are pregnant or lactating have increased dietary iodine requirements. Severe iodine deficiency leads to adverse maternal and fetal consequences. Even mild-to-moderate iodine deficiency in pregnancy has adverse effects on obstetric and neonatal outcomes. Recent data on the neonatal neuro cognitive impact of early iodine supplementation suggests that adequate iodine intake should start as soon as the patient is aware she is pregnant, or, even better, should be incorporated as part of preconception planning (Yarrington and Elizabeth2011).

1.5.7 Role of Iodine in the brain effects mechanisms

Iodine is an essential micronutrient needed in human diets. As iodine is an integral component of thyroid hormone, it mediates the effects of thyroid hormone on brain development. Iodine deficiency is the most prevalent and preventable cause of mental

impairment in the world. The exact mechanism through which iodine influences the brain is unclear, but is generally thought to begin with genetic expression. Many brain structures and systems appear to be affected with iodine deficiency, including areas such as the hippocampus, microstructures such as myelin, and neurotransmitters. The clearest evidence comes from the studies examining cognition in the cases of iodine deprivation or interventions involving iodine supplementation (Redman et al. 2016).

1.5.8 Role of iodine in antioxidant defence in thyroid and breast disease

The role of iodine played in thyroid hormonogenesis by iodide oxidation to iodine (organification) is well established. Iodine deficiency may produce conditions of oxidative stress with high TSH producing a level of hydrogen peroxide, which because of lack of iodide is not being used to form thyroid hormones. The cytotoxic actions of excess iodide in thyroid cells may depend on the formation of free radicals and can be attributed to both necrotic and apoptotic mechanisms with necrosis predominating in goiter development and apoptosis during iodide induced involution. These cytotoxic effects appear to depend on the status of antioxidative enzymes and may only be evident in conditions of selenium deficiency where the activity of selenium containing antioxidative enzymes is impaired. Less compelling evidence exists of a role for iodide as an antioxidant in the breast (Smyth 2003).

1.5.9 Radiation-induced thyroid cancer

Nuclear accidents can release radioactive iodine into the environment, increasing the risk of thyroid cancer in people who are exposed to the radioactive iodine, especially children. People with iodine deficiency who are exposed to radioactive iodine are especially at risk of developing thyroid cancer. The U.S. Food and Drug Administration has approved potassium iodide as a thyroid-blocking agent to reduce the risk of thyroid cancer in radiation emergencies. The amount of iodine needed each day depends on age. Average daily recommended amounts are listed below in micrograms (mcg) (Smyth 2003).

Table 1.2: Recommended Iodine intake (Smyth 2003).

Life Stage	Recommended Amount
Birth to 6 months	110 mcg
Infants 7–12 months	130 mcg

Life Stage	Recommended Amount
Children 1–8 years	90 mcg
Children 9–13 years	120 mcg
Teens 14–18 years	150 mcg
Adults	150 mcg
Pregnant teens and women	220 mcg
Breastfeeding teens and women	290 mcg

1.6 Iodine deficiency disorders (IDD)

1.6.1 IDD definition

IDDs are defined as all the consequences of iodine deficiency in a population that can be prevented by ensuring that the population has an adequate intake of iodine.

Insufficient iodine during pregnancy and infancy results in neurological and psychological deficits in children. Iodine deficiency remains the leading cause of preventable mental retardation worldwide. In adults, mild-to-moderate iodine deficiency increases the incidence of hyperthyroidism due to toxic goiter.

The iodine status of most premature infants worldwide is that of iodine deficiency. Persistent decreases in TSH and increases in free T4 were observed in a previously iodine insufficient population, even though the present iodine status was adequate, suggesting that low iodine intake at young age leads to thyroid autonomy that persists despite normal iodine intake later in life.

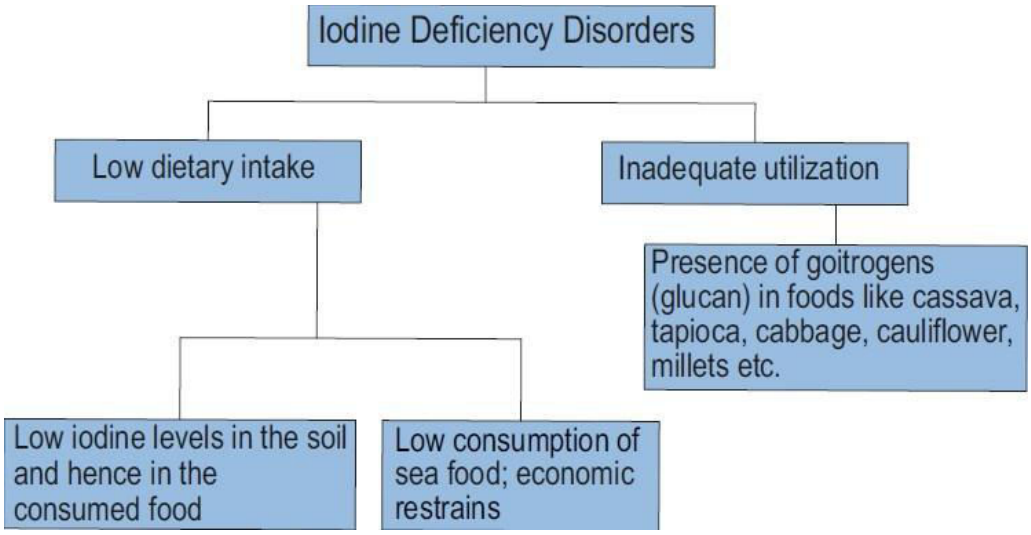


Fig 1.4: Iodine Deficiency Disorder

1.6.2 Causes of iodine deficiency disorders

IDD is a term that collectively reflects the clinical and sub-clinical manifestations of iodine deficiency. Iodine being an indispensable component of T3 and T4 hormones, its deficiency interferes seriously with the synthesis of these hormones. For a time thyroid responds by releasing the hormones stored as components of thyroglobulin molecules. But when the stores are exhausted and blood level of T4 start declining, pituitary intervenes by increasing TSH output which stimulates the thyroid to increase the uptake of iodide and ensure the release of thyroid hormones in adequate strength. However, in the state of deficiency when iodide uptake of thyroid is seriously hampered TSH fails to promote the release of T4 and only ends up with the hyperplasia of follicular cells. In a situation of severe iodine deficiency, while the level of T4 remains low, the level of TSH remains high . Under continuing TSH stimulation in endemic areas, thyroid gland undergoes hypertrophy and hyperplasia of follicular cells and in the process, enlarges in size and appears as a goiter which may in certain cases attain an enormous size (Ahad and Shafiq 2010).

Table 1.3: The damage caused to the human body due to iodine deficiency is in fact the result of deficiency of thyroid hormones. The effects of IDD in humans at different ages (Ahad and Shafiq 2010).

Pregnancy	• **.Spontaneous abortions** • **Still births** • **Mal development of fetal brain** • **Birth of cretins**
Childhood	• Goiter • Low IQ • Impaired learning • Mental retardation • Delayed motor development • Stunted growth • Apathy • Muscular disorders • Paralysis • Speech & hearing defects • High perinatal mortality • High infant mortality
Adolescent	• Mental retardation • Growth retardation
Adult & all ages	• Goiter • Hypothyroidism • Apathy • Impaired mental function • Reduced work output

1.6.3 Effect of Iodine Deficiency Disorder

A growing fetus in the womb of an iodine deficient mother is at high risk. The pregnancy may end in abortion, still birth, congenital anomalies or low birth weight outcome. Infants born to iodine deficient mothers, who survive the critical postnatal phase, may develop endemic cretinism. The neurological form of endemic cretinism is characterized by severe mental retardation and is usually associated with cerebral diplegia and deaf-mutism. Children in endemic areas show retarded physical and mental development, low I.Q. levels and impaired school performance. The grave implications of iodine deficiency on child's learning capacity and the quality of life of child population is thus evident in the erosion of quality of our human resources .

Iodine deficiency has emerged as a socio-medical problem of vast dimensions associated with physical and mental retardation, neurological disorders, feeble mindedness, low educability, poor performance, social handicaps, dependability and disfigurement (Ahad and Shafiq 2010).

1.6.4 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 ldeficiency 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 µg/L in a population(Hernando, Anliza and Herman 2015).

Table 1.4: Recommendations for iodine intake by age or life-stage group and tolerable upper intake levels for iodine (Hernando, Anliza and Herman 2015).

	Iodine intake	**Dietary iodine contribution**
School-age children		

<20 µg/L	Insufficient	Severe deficiency
20-49 µg/L	Insufficient	Moderate deficiency
50-99 µg/L	Insufficient	Mild deficiency
100-199 µg/L	Adequate	Optimal
200-299 µg/L	More than adequate	Risk of iodine-induced hyperthyroidism in susceptible groups
>300 µg/L	Excess	Risk of harmful consequences for health (hyperthyroidism, autoimmune thyroiddisease)
Pregnant women		
<150 µg/L	Insufficient	-
150-249 µg/L	Adequate	-
250-499 µg/L	More than adequate	-
≥500 µg/L	Excess*	-
Breastfeeding women**		
<100 µg/L	Insufficient	-
≥100 µg/L	Adequate	-
Children< 2 years of age		
<100 µg/L	Insufficient	-
≥100 µg/L	Adequate	-

1.6.5 Iodine supplementation strategies

Since iodine is released from the body through urine, the best way to determine iodine deficiency across a large population is to measure the amounts of iodine in urine samples. The WHO defines iodine deficiency as a median urinary iodine concentration less than 50 µg/L in a population .Median population urinary iodine values and iodine nutrition .With increasing

awareness of the wide spectrum of iodine deficiency disorders, a steady increase has been noticed in the estimation of the magnitude of the problem in the world. In 1990, WHO reported that the total population at risk of iodine deficiency in developing countries was 1 billion, of which 200 million suffered from goiter; over 5 million were cretins with gross mental retardation; and 15 million had less degrees of mental defect (Ahad and Shafiq 2010).

1.6.6 IDD prevention and treatment

The most effective way to control IDD is universal salt iodization, which refers to iodization of all salt used for human consumption (industrial and household use). This strategy is recommended because salt is used essentially in all foods and its intake is consistent throughout the year. Iodization is a simple, inexpensive technique, and does not affect salt color or flavor. The amount of iodine in the salt may be monitored in production, retail and in the home. The WHO/UNICEF/ICCIDD recommendation is to add iodine to the salt at a concentration of 20-40 mg of iodine per kilogram of salt. Iodine may be added in the form of potassium iodide or iodate; however, considering that potassium iodate is more stable than iodide when in contact with moisture and impurities, it is the recommended iodization form in tropical countries. Iodine is usually added once the salt has gone through a "drying" process. Bread may be a good vehicle for adjusting salt intake by introducing iodine-enriched salt in the baking process. Iodization of water and irrigation systems may also be useful, but this requires costly methods that limit its application. Countries like Switzerland and the United States have additional iodine sources through milk in the diet, more because of the use of iodophors in the food industry than the deliberate addition of iodine. In countries affected by IDD, it is considered that iodine must be added routinely to complementary foods in order to increase iodine content derived from daily intake.

In remote areas or areas of difficult access, or where small-scale salt producers exist, salt iodization programs may not work or create the expected social impact. In those situations, the recommendation is to replace iodine by means of iodized oils administered orally or intramuscularly. The oral route is easier, but the intramuscular route is more effective and has longer lasting effects. The oral dose ranges between 200 and 400 mg of iodine per year and it is usually administered to the most vulnerable population (pregnant women, children and women in childbearing age). Iodine may also be given in the form of potassium iodide or iodate drops or

tablets; the monthly (30 mg) or biweekly (8 mg) dose of potassium iodide may besufficient to provide the adequate amount of iodine, in particular to the population at risk(Hernando, Anliza and Herman 2015)

1.7 Iodine Deficiency

1.7.1 Iodine Deficiency and Pregnancy

Iodine deficiency is especially important in women who are pregnant or nursing their infants. The World Health Organization (WHO) recently increased their recommended iodine intake during pregnancy from 200 to 250 microg/d and suggested that a median urinary iodine (UI) concentration of 150-249 microg/L indicates adequate iodine intake in pregnant women. Thyrotropin concentrations in blood collected from newborns 3-4 d after birth may be a sensitive indicator of even mild iodine deficiency during late pregnancy; a <3% frequency of thyrotropin values >5 mU/L indicates iodine sufficiency. New reference data and a simple collection system may facilitate use of the median UI concentration as an indicator of iodine status in newborns. In areas of severe iodine deficiency, maternal and fetal hypothyroxinemia can cause cretinism and adversely affect cognitive development in children; to prevent fetal damage, iodine should be given before or early in pregnancy. Whether mild-to-moderate maternal iodine deficiency produces more subtle changes in cognitive function in offspring is unclear; no controlled intervention studies have measured long-term clinical outcomes. Cross-sectional studies have, with few exceptions, reported impaired intellectual function and motor skills in children from iodine-deficient areas, but many of these studies were likely confounded by other factors that affect child development. In countries or regions where <90% of households are using iodized salt and the median UI concentration in school-age children is <100 microg/L, the WHO recommends iodinesupplementation in pregnancy and infancy (Zimmerman 2009).

1.7.2 Iodine Deficiency in the Fetus

The most important biological role played by thyroxin is in the early foetal stage of life. It ensures the growth, differentiation and maturation of different organs of the body, and particularly the brain. Iodine deficiency has been identified as the world's major cause of

preventable mental retardation. Its severity can vary from mild intellectual blunting to frank cretinism, a condition that includes gross mental retardation, deaf-mutism, short stature and various other defects. In areas of severe iodine deficiency, the majority of individuals risk some degree of mental impairment. The damage to the developing brain results in individuals poorly equipped to fight disease, learn, work effectively, or reproduce satisfactorily. The spectrum of disorders caused due to iodine deficiency affects all the stages of life, from foetus to adult age (Kapil 2007).

1.7.3 Iodine Deficiency in the Adults

Iodine deficiency remains the leading cause of preventable mental retardation worldwide. In adults, mild-to-moderate iodine deficiency increases the incidence of hyperthyroidism due to toxic goiter. Persistent decreases in TSH and increases in free T4 were observed in a previously iodine insufficient population, even though the present iodine status was adequate, suggesting that low iodine intake at young age leads to thyroid autonomy that persists despite normal iodine intake later in life (Chung 2014).

1.7.4 Iodine Deficiency in the Children and Adolescents

The majority of children with ID disorders brought to psychiatric outpatient units by their families or referred to these units by their teachers present with complaints of lack of attention and consequent failure. In a substantial portion of these children, attention deficit/hyperactivity disorder (ADHD) needs to be considered in the differential diagnosis. ADHD is a disorder in the etiology of which neurological, genetic, environmental, dietary, biological, and psychosocial factors are possibly involved (Yuksek, Ayceramd and Oner 2016).

1.7.5 Specific Iodine Deficiency Disorders

Endemic Cretinism

Endemic cretinism is the extreme clinical manifestation of severe hypothyroidism during foetal, neonatal and childhood stages of development. It is characterised by severe and irreversible mental retardation, short stature, deaf-mutism, spastic dysplegia and squints.

Cretinism seen in severe endemic areas is predominantly of two types

(a) neurological cretinism, where the neurological manifestations of thyroxin deficiency early in life, i.e. hypothyroidism, were confined to the in-utero or neonatal stages.

Fig1.5: Male with the typical facies of neurological cretinism, who is also deaf-mute and suffering from less severe proximal muscle weakness in lower limbs

(b) Myxedematous cretinism, where besides having mental retardation, sufferers also have myxoedema and dwarfism. This variant of cretinism is presumably because of continuing hypothyroidism through all phases of life.

Fig1.6 : Myxedematous endemic cretinism. Four inhabitants aged 15-20 years : a normal male and three females with severe longstanding hypothyroidism with dwarfism, retarded sexual development, puffy features, dry skin and hair and severe mental retardation.

1.7.6 Cretinoids

Besides the few children who manifest as cretins in an endemic goiter area, a large number of individuals with lesser degrees of mental retardation, speech and hearing defects, psychomotor retardation, as well as gait defects may be seen. Such individuals are known as cretinoids. The prevalence of cretinoids in severely endemic regions may be ten-fold greater or more than fully manifested cretins..

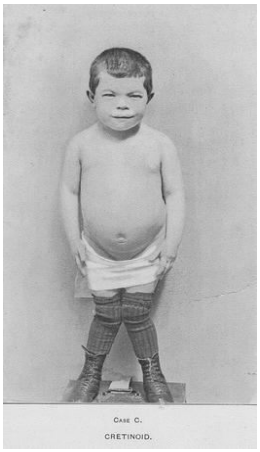


Fig1.7: Cretinoids

1.7.7 Neonatal and Childhood hypothyroidism

Studies have documented that more than 30% of the goitrous subjects in endemic areas are functionally decompensated and hypothyroid despite the 'adaptive' enlargement of the thyroid.

1.7.8 Adult hypothyroidism

A large number of goitrous adults in an endemic region can have varying degrees of hypothyroidism leading to a variety of clinical symptomatologies and complications related to hypo-metabolic states. This symptomatology can seriously hamper human energy and work capacity with resultant erosion of the economic productivity of endemic regions (Kapil 2007).

Fig 1.8. Three women of the Himalayas with stage II goiters.

1.7.9 Endemic goiter

Endemic goiter is characterized by enlargement of the thyroid gland in a significantly large fraction of a population group, and is generally considered to be due to insufficient iodine in the daily diet. Endemic goiter exists in a population when >5% of 6-12 year-old children have enlarged thyroid glands

Fig1.9: A young girl with a soft diffuse goitre and an elderly woman with a huge, longstanding multinodular goiter, both resulting from iodine deficiency.

1.8 Current Global Status of IDD Control Programs

Until 1990, only a few countries Switzerland, some of the Scandinavian countries, Australia, the U.S. and Canada were completely iodine sufficient. Since then, globally, the number of households using iodized salt has risen from <20% to >70%, dramatically reducing iodine deficiency . This effort has been achieved by a coalition of international organizations,

including ICCIDD (now IGN), WHO, MI and UNICEF, working closely with national IDD control committees and the salt industry; this informal partnership was established after the World Summit for Children in 1990.

The two most commonly used approaches to assessing iodine nutrition on the population level are estimation of the household penetration of adequately iodized salt (HHIS) and measurement of urinary iodine concentrations (UICs). UIC surveys are usually done in school aged children (SAC), because they are a convenient population, easy to reach through school based surveys and usually representative of the general population. Therefore, WHO use UICs from 6-12 y-old children in nationally- representative surveys, expressed as the median in µg/L, to classify a population's iodine status . More countries are beginning to carry out studies in high-risk population groups, i.e. women of reproductive age, pregnant women and younger children, however data is limited and the majority of countries still conduct routine iodine monitoring in SAC .

In 2017, representative UIC surveys are available for 139 countries. There are no up-to- date UIC data available for 55 countries. Available UIC data now cover >98% of the world's population of SAC .

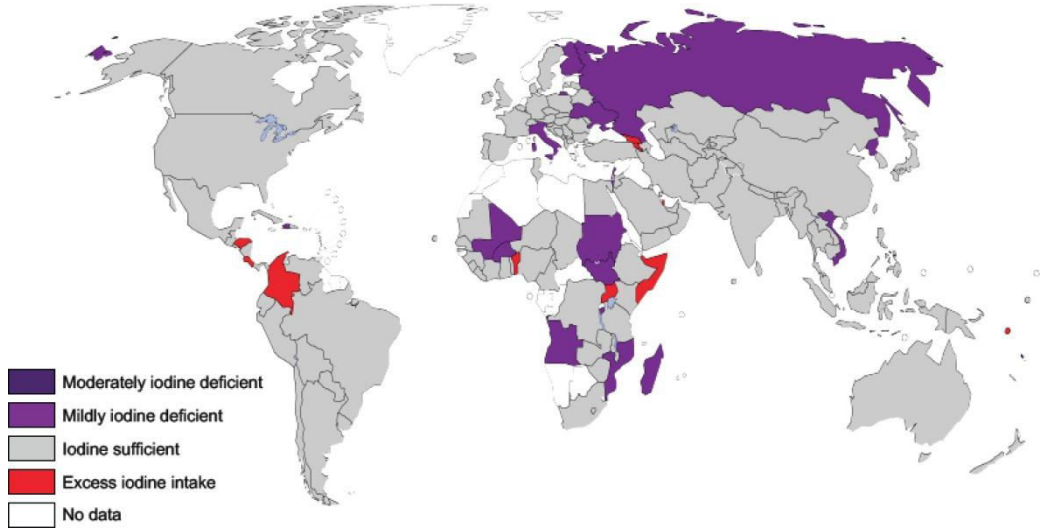


Fig 1.10: Shows countries classified by iodine nutrition in 2017 according to degree of public health importance based on the median UIC. Iodine intake is inadequate in 19 countries, adequate in 110 and excessive in 10. There are no up- to-date UIC data available for 55 countries.

Overall, approximately 75% of households worldwide have access to iodized salt. Those with the greatest access are living in the WHO regions of the Western Pacific and the Americas, and those with the least access are residing in the Eastern Mediterranean region (Zimmermann 2009).

1.8.1 Iodine deficiency disorder in Bangladesh

The widespread prevalence of goitre in Bangladesh was first revealed by the nutrition survey of 1962-64 by the Department of Biochemistry, University of Dhaka. The national goitre prevalence study conducted by the Institute of Public Health and Nutrition (IPHN), Dhaka in 1981-82 reported that the overall goitre prevalence was 10.5%. The survey was done on 214, 608 subjects and the prevalence varied between 2.6% and 29.3%.

A nationwide comprehensive IDD survey was conducted by a Dhaka University team in 1993 to assess the current status in the country. It revealed a severe IDD situation; the current total goitre rate (TGR) in Bangladesh is 47.1% (8.8% visible and 38.3% palpable), and the rate of cretinism is about 0.5%. The survey also showed that about 68.9% are biochemically iodine deficient. Children and women are the worst sufferers. In Bangladesh certain northern districts, particularly Rangpur, Gaibandha, Nilphamari and Dinajpur are considered as goiter-prone areas.

In 1981-82, the Government of Bangladesh and WHO jointly conducted a survey on the status of iodine deficiency disorders of the country. It was reported that more than 30 million people in the country have the benign or primary state of goiter and other form of iodine deficiency maladies. About 11 percent of the population is affected by visible goiter, the prevalence being much higher in females. About 30 percent of the population of greater Rangpur and Dinajpur districts in the north show incidences of goiter. The incidence in Chittagong, Khulna and Mymensingh areas is also fairly high. Endemic goiters may slowly diminish in size with iodine administration. Some sporadic goiters respond to treatment with replacement doses of thyroid extract.

Programmes to mitigate goiter and other iodine deficiency disorders started in Bangladesh in 1977. The Institute of Nutrition and Food Science of Dhaka University in cooperation with UNICEF administered Lepiodol injection to about 95,000 people in nine districts. Similarly, in

1983 the Institute of Public Health with assistance from UNICEF provided injections to about 80,000 people of Jaldhakaupazila in Rangpur district. The project to incorporate iodine in edible salt started from 1985. It is now mandatory to market iodized salts, and nearly two dozen enterprises produce such edible salts according to government specifications. The Institute of Food Sciences and Technology (IFST) of the Bangladesh Council of Scientific and Industrial Research (BCSIR) has developed a simple procedure and technology of producing, preserving, and the marketing this commodity. IFST also monitors the effectiveness of this programme.

In areas where the iodine content of the diet is low, a satisfactory way of increasing the intake of iodine is the use of 'iodized salt'. This is prepared by adding about one part of potassium iodide to about 40,000 parts of salt. Potassium iodide is soluble in water and is rapidly absorbed into the blood, any surplus being quite harmless. The use of iodized salt is a simple and harmless way of supplementing the iodine obtained from food. Bangladesh government has made it mandatory to market iodised salts (Zimmermann 2009).

1.8.2 Current situation of iodine deficiency in Bangladesh:

The current prevalence of I deficiency, based on urinary I concentration <100 µg/l, in school-age children is as high as 40 %, reflecting a figure similar to the 42·5 % reported in 1999 and an increase from the 33·8 % reported in 2004–2005. There are no estimates on the current prevalence of goitres (a severe form of I deficiency) in school-age children. However, previous surveys indicated a substantial reduction in goitre prevalence in school-age children from 50 % in 1993to 17·2 % in 1999to 6·2 % in 2004–2005.

The NMS 2011–2012 reported a 42 % prevalence of I deficiency, based on urinary I level, in NPNL women, which was 45·6 % in 1999and 38·6 % in 2004–2005. The National Iodine Deficiency Disorder Survey in 2004–2005 reported goitre rates of 11·7 and 14·6 % in NPNL women and pregnant women, respectively, with the higher risk of I deficiency among women either living in rural areas and/or belonging to lower socio-economic groups.

1.8.3 Major risk factors of I deficiency in the Bangladeshi population:

Household food insecurity, lack of access to iodised packaged salt, rural residency, low levels of awareness about the health benefits of I and iodised salts, consumption of industrial salt (non-

iodised) and lack of preservation knowledge about iodised salts have been identified as the major risk factors of I deficiency in the Bangladeshi population. (Ahmed, Prendiville and Narayan 2017)

Table1.5: The studies and the prevalence of iodine deficiencies in children and women in Bangladesh(Ahmed, Prendiville and Narayan 2017)

Study period	Study design	Sample size	Prevelance of deficiency
2004-2005	National survey	Goiter survey : school children (n 6400) NPNL women (n2447) Urinary I: school children (n 2400) NPNL women (n 2401)	Goiter : School children,6.2% NPNL women,11.7% I deficiency: School children,33.8% NPNL women,38.6%
2011-2012	National survey	Urinary I: school children (n 1350) NPNL women (n 1500)	I deficiency: School children,40% NPNL women,42.1%

1.9 Indicators of Sustainable IDD Elimination

Adequate iodine intake during pregnancy, breastfeeding and early childhood is extremely important for optimal brain development in utero and in infants 6-24 months of age. While the

primary strategy for sustainable elimination of IDD continues to be universal salt iodization, international agencies have recommended the complementary strategy of providing iodine supplements on a temporal basis in those cases where iodization cannot be implemented. Consequently, salt iodization is the most sustainable approach to eliminating IDD in the long run, and iodine supplementation must be considered as a short-term measure, especially in those areas where salt iodization cannot be implemented promptly.

The following are topics can be considered by the country that are planning to implement an iodine supplementation program for IDD prevention:

- Effective and efficient supplement management programs

Supplements need to be distributed monthly on a regular and timely basis.Maintenance of continuing education and communication programs. Authors involved in healthcare must be trained and provided with the necessary knowledge regarding the programs.

- Strong monitoring systems

Monitoring of nutrition status in relation to iodine is a way to ensure that results and achievements are used for appropriate decision-making; hence the need to share results with the general public.

- Establishment of supplementation goals

Vulnerable groups must be the main targets for the intervention. These include pregnant women, breastfeeding mothers, and children under 2 years of age. In pregnant women, programs must be instituted as early as possible during gestation.

Additionally, there are some programs indicators for sustainable IDD elimination. Of these, at least eight must be met in order to consider that eradication objectives are being achieved.

1. Existence of a multi-stake holder coalition responsible for managing the national IDD elimination program, of the following characteristics:
    - ✓ National scope.
    - ✓ All stakeholders, including the salt industry, represented with their own roles andresponsibilities.
    - ✓ Call to action at least twice early.

2. Adoption of legislation and regulation to support universal salt iodization that provides for a consistent mechanism to assure external quality.
3. Establish evaluation methods to measure progress in IDD elimination (programe reports at a national level designed to assess progress every three years).
4. Access to laboratories with the ability to provide accurate data on iodine levels in soil and urine, and thyroid function tests.
5. Establish an education and social awareness program consisting of information about the importance of iodine and the use of iodized salt, provided as part of an education curriculum.
6. Consistent and routine availability of data on iodine content in salt, in the form of regular data coming from producers at least every month andfromhouseholdsatleastevery5years.
7. Availability of population data regarding ioduria values, at least every five years.
8. Evidence of continuous cooperation from the salt industry, in the form of consistent quality control measurements and lowering of iodide and iodate costs.
9. Existence of a national database for registering the results of regular monitoring procedures, including coverage of household and ioduria levels, together with other iodine indicators in the population, and assessment of thyroid function, when available (Hernando, Anliza and Herman 2015).

CHAPTER – 2

LITERATURE REVIEW

2.1 A study was conducted in Sri Lanka has shown the concentrations of iodine species in commercial edible salt products, the stability of iodine at different conditions and the iodine exposure at the consumer level. The result shows that the six different brands of most commonly sold commercial edible iodized salts in different grocery stores have excess iodine that is above the fortification level of 15–30 mg kg^{-1}.As a total 95.8 % cases can cause Iodine Induced Hyperthyroidism (IIH) and only 4.1 % of them can provide optimal iodine nutrition in a population (Vithanageet al. 2016).

2.2 A study was designed by Nepal et al. to determine the salt types and the household salt iodine content of school aged children in the hilly and the plain districts of eastern Nepal in the year of 2013,has shown that, there were 270 (38.2%) households which consumed crystal salt and 437(61.8%) of the households consumed packet salts. The mean ±SD values of the salt iodine content in the four districts, namely, Sunsari , Dhankuta, Sankhuwasabha and Tehrathum were 34.2±17.9, 33.2±14.5, 27.4±15.1 and 48.4±15.6 parts per million (ppm) respectively. The salt types were categorized and the salt iodine content was estimated by using rapid test kits and iodometric titration methods . Among the 707 salt samples which were collected from the households had <15 ppm iodine were 123 (17.3%) and those which had >15 ppm iodine were 584 (82.6%) (Nepal et al. 2013).

2.3 A quantitative analysis of iodine was performed on table salts collected from different local markets, and local eateries of Nigeria in the year of 2013 has shown that, the majority of the Nigerian manufactured table salts were compliant with the standards of the World Health Organization standards and USI mandate. However, one had low iodine content due to long term storage and exposure to harsh weather conditions. Salts tested from the local eateries had average iodine levels of 71ppm which will be adjusted to permissible limits due when the concentration is reduced by approximately 10% when exposed to high temperatures during cooking. Both commercially available salts and those from local eateries are compliant with the USI mandate and are therefore provide dietary intake of preventing nutritional problems such as Iodine Deficiency Disorders (Tyndall et al.2013).

2.4 A survey was conducted in Bangladesh in the year of 2008 to monitor the iodine deficiency

disorders in children (aged 6-12 years) and women (aged 15-44 years). It was conducted between September 2004 and March 2005, and a total of 7233 children and 6408 women were examined for goiter and 4848 urine samples (2447 from children and 2401 from women) were analyzed for iodine. In addition, 5321 household salt samples were analyzed for iodine. In children, the total goitre rate (TGR) was 6.2%, compared to 49.9% in 1993 and the TGR among women was 11.7%, while in 1993 it was 55.6%. Prevalence of iodine deficiency (Urinary Iodine Excretion <100 microg/L) was 33.8% in children and 38.6% in women (compared to 71.0% and 70.2%, respectively in 1993).In the study it has shown that the iodine nutrition status in urban areas was considerably better than in rural areas and there was a clear inverse relationship between iodine deficiency and the coverage of households using adequately iodized salt (>or =15 ppm).

The result revealed that Bangladesh has achieved a commendable progress in reducing goitre rates and iodine deficiency among children and women ever since the universal salt iodization programme was instituted 10 years ago. However, physiological iodine deficiency still persists among more than one-third of children and women, which points to the need for all stakeholders to redouble their efforts in achieving universal salt iodization (Yusuf et al.2008).

2.5 A study was conducted in the year of 2010 in Bngladesh, has shown that, the sub-clinical iodine deficiency is still prevalent in adolescent girls and pregnant womens. For doing this study a total number of 354 adolescent girls and 256 pregnant women were randomly selected from the six divisions of Bangladesh. The salt samples were collected from the household and spot urine samples were collected from the respondents. The result shows that

in total, 37% adolescent girls and more than fifty percent of pregnant women were subclinically iodine deficient. Analysis of salt samples from the households shows that a proper level of iodine in the salt is not being maintained and it was found that almost half of the households were still consuming table salt containing an inadequate iodine concentration (<15 $\mu g\ g^{-1}$). According to urinary iodine concentration classified by the WHO cut off points (optimal iodine concentration >99 $\mu g\ L^{-1}$; mild deficiency 50-99 $\mu g\ L^{-1}$; moderate deficiency 20-49 $\mu g\ L^{-1}$ and severe deficiency <20 $\mu g\ L^{-1}$) the level of iodine deficiency was severe in 4% of the adolescent girls.

Mild to moderate deficiency existed in 33% of the girls. One fifth (20%) of pregnant women had severe iodine deficiency and 37% women who demonstrated mild to moderate levels of urinary iodine (Ara *et al.,*2010).

CHAPTER – 3

AIM

AND

OBJECTIVE OF THE STUDY

3.1 Aim and Objectives of the Study

The aim of the study is to determine the iodine content of the table salts collected from the area Srlimangal of Bangladesh.

Objectives:

- To determine the concentration of iodine present in the household salts, in the area of Srimangal of Bangladesh.

- To determine the iodine content, compliant with the WHO guideline or not.

CHAPTER – 4

MATERIALS AND METHODS

4.1 Study design

The study was designed to determine the household salts iodine content and collected from the area Srimangal.

4.2 Study period

The frist ten samples were collected in 11thmay of 2017 and the another ten samples were collected in18th October of 2017.

4.3 Samples

The samples for this study were collected from the different peoples household salts in the area of Srimangal.Thefrist ten samples are collected in 11thmay of 2017and these are S 1,S 2,S 3,S 4,S 5,S 6,S 7,S 8,S 9,S 10.
The another ten samples were collected in18th October of 2017 and these are S 11,S 12,S 13,S 14,S 15,S 16,S 17,S 18 S 19,S 20.

4.4 Chemicals and reagents used:

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

- Starch
- Sulfuric Acid

4.5 Glass apparatus/Accessories:

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

4.6 Equipments/Machineries:

- Electronic Balance
- Hot Plate
- Distillation Equipment

4.7 Distillation procedure:

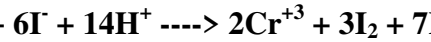
The device used in distillation, consists at a minimum of a reboiler or *pot* in which the source material is heated, a condenser in which the heated vapour is cooled back to the liquid state, and a receiver in which the concentrated or purified liquid, called the distillate, is collected. By this technique distilled water collected.

4.8 Methods:

4.9 Preparation of Standard Solutions and Reagents

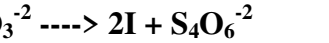
Principle

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.



$$Cr_2O_7^{-2} + 6I^- + 14H^+ \text{----> } 2Cr^{+3} + 3I_2 + 7H_2O$$

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



$$I_2 + 2S_2O_3^{-2} \text{----> } 2I + S_4O_6^{-2}$$

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 0.005N $K_2Cr_2O_7$

Molecular weight of $K_2Cr_2O_7$ = 294 mg

Equivalent weight of $K_2Cr_2O_7$ = 49 mg

Calculation:

n= Normality

e= Equivalent weight

v=Volume in litre

g= n e v

= 0.005×49×50/1000 gm

= 0.01225 gm

0.01225 gm 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 50 ml of 0.005N $Na_2S_2O_3$

Molecular weight of $Na_2S_2O_3$= 248mg
Equivalent weight of $Na_2S_2O_3$=248mg

Calculation:
n= Normality
e= Equivalent weight
v=Volume in litre

g= n e v
= 0.005×248×50/1000 gm

= 0.062 gm

0.062 gm of $Na_2S_2O_3$ was accurately measured on an electronic balance, and placed into a 50 ml 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 50 ml mark.

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 6 M HCL

A small amount of water was added to a 50ml volumetric flask. 31.98ml 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.

Standardization of Sodium Thiosulphate ($Na_2S_2O_3$)

Since $Na_2S_2O_3$ standard compound , so is needed to standardize the compound with primary standard compound $K_2Cr_2O_7$. From 50 ml prepared $K_2Cr_2O_7$ solution ,10 ml $K_2Cr_2O_7$ solution was transferred in each of the three conical flask.1gm of KI and 1gm of $NaHCO_3$ were placed in that conical 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 5 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 $Na_2S_2O_3$ required for titration was then determined to calculate the strength.

Method of Standardization of $Na_2S_2O_3$

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 $Na_2S_2O_3$ is calculated using the following formula:

Volume of $Na_2S_2O_3$, $V_1 = 10.10$ ml

Strength of $K_2Cr_2O_7$, $S_2 = 0.1$ N

Volume of $K_2Cr_2O_7$, $V_2 = 10$ml

$$S_1 V_1 = S_2 V_2$$

$$S1 = (0.1 \times 10)$$

$$= 0.099N$$

4.10 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 (KIO_3).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:

$$IO^{3-} + 5I^- + 6H^+ \rightarrow 3I_2 + 3H_2O$$

Then the resulting iodine is titrated with Thiosulphate as follows:

$$I_2 + 2S_2O_3^{2-} \rightarrow 2I^- + S_4O_6^{2}$$

Method of Titration of salt:

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 $Na_2S_2O_3$ 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 $Na_2S_2O_3$ was determined.

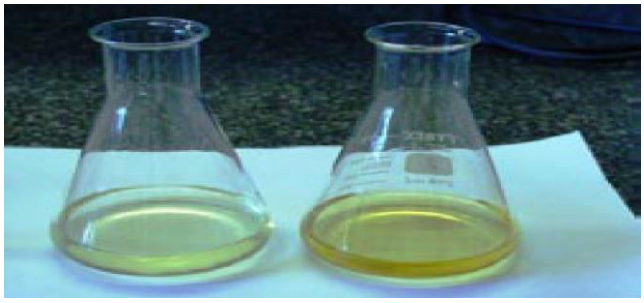


Fig. 4.1.: Right flask: yellow/brown color of iodine formed from reaction of iodated from salt with acidic iodide solution. Left flask: pale yellow color left when nearly all iodine has reacted with added thiosulfate during titration.

After addition of starch

Fig 4.2: Right flask: yellow/brown solution containing last trace of iodine. Left flask: darkblue-black color formed when starch indicator is added to solution containing iodine near endpoint

After addition thiosulfate

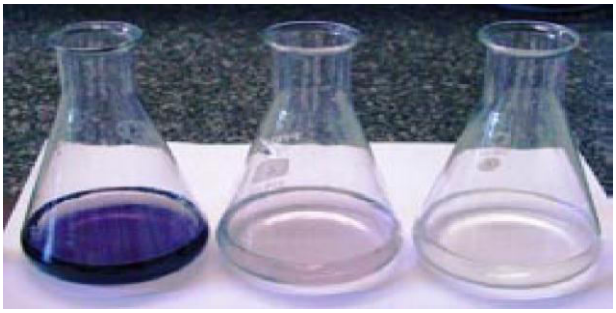


Fig 4.3: A series of flasks showing the color change as the last remaining iodine (with added starch indicator) is titrated with thiosulfate. The dark blue-black color disappears, leaving a colorless solution at the endpoint.

Iodine ppm = (R×100×1000×0.127×N)/6

Where,

R = Average volume of $Na_2S_2O_3$

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 Thiosulphatesolution

N is normality of Thiosulphate solution which was calculated after standardization

6 is to arrive at the value that corresponds to 1 atom of iodine liberate.

CHAPTER – 5

CALCULATION AND RESULTS

5.1 Calculation

Table 5.1: Standardization of $Na_2S_2O_3$

Observation No.	Initial Burette Reading (ml)	Final Burette Reading (ml)	Difference (ml)	Average (ml)
1	3.2	13.1	9.9	
2	13.1	23.2	10.1	9.77
3	23.2	32.5	9.3	

5he strength of the $Na_2S_2O_3$ is calculated using the following formula:

Volume of $Na_2S_2O_3$, $V_1 = 9.77$ml

Strength of $K_2Cr_2O_7$, $S_2 = 0.005$ N

Volume of $K_2Cr_2O_7$, $V_2 = 10$ml

$$S_1 V_1 = S_2 V_2$$

$$S1 = (10 \times 0.005)/9.77$$

$$= 0.00512N$$

Table 5.2: Iodine content of salts(S 1,S 2,S 3) after iodometric titration

Sample Code	Initial Burette Reading (ml)	Final Burette Reading (ml)	Difference (ml)	Iodine (ppm) (R×100×1000×0.127×N)/6
S 1	32.5	35.5	3	32.51
S 2	35.5	36.4	0.9	9.75
S 3	36.4	37.2	0.8	8.67

From the iodine ppm of S 1,S 2,S 3 it is shown that, S1 is in acceptable range (20-50 ppm) and S2 & S3 are below the range.

Table 5.3: Standardization of $Na_2S_2O_3$

Observation No.	Initial Burette Reading (ml)	Final Burette Reading (ml)	Difference (ml)	Average (ml)
1	7.4	18.4	11	
2	18.4	29.9	11.5	10.93
3	29.9	40.2	10.3	

$$S_1 V_1 = S_2 V_2$$

$$S1 = (10 \times 0.005)/10.9$$

$$= 0.004573 \text{ N}$$

Table 5.4: Iodine content of salts(S7, S8) after iodometric titration

Sample Code	Initial Burette Reading (ml)	Final Burette Reading (ml)	Difference (ml)	Iodine (ppm) (R×100×1000 ×0.127×N)/6
S 7	47.3	47.3	3	35.81
S 8	47.3	50	2.7	26.13

From the iodine ppm of S7, S8 it is shown that, both are in acceptable range (20-50 ppm).

Table 5.5: Standardization of $Na_2S_2O_3$

Observation No.	Initial Burette Reading (ml)	Final Burette Reading (ml)	Difference (ml)	Average (ml)
1	3.5	14	10.5	
2	14	24	10	10.20
3	24	34.1	10.1	

$$S_1 V_1 = S_2 V_2$$

$$S1 = (10 \times 0.005)/10.20$$

$$= 0.004902 \text{ N}$$

Table 5.6: Iodine content of salts(S 4,S 5,S 6,S 9,S 10) after iodometric titration

Sample Code	Initial Burette Reading (ml)	Final Burette Reading (ml)	Difference (ml)	Iodine (ppm) (R×100×1000 ×0.127×N)/6
S 4	34.1	37.3	3.2	33.20
S 5	37.3	40.3	3	31.13
S 6	40.3	43.8	3.5	36.32
S 9	43.8	47	3.2	33.20
S10	47	50	3	31.13

From the iodine ppm of S4, S5, S 6, S9 ,S10 it is shown that all are in acceptable range (20-50 ppm).

Table 5.7: Standardization of $Na_2S_2O_3$

Observation No.	Initial Burette Reading (ml)	Final Burette Reading (ml)	Difference (ml)	Average (ml)
1	3	13.3	10.3	
2	13.4	23.2	9.8	10.47
3	23.2	34.5	11.3	

$$S_1 V_1 = S_2 V_2$$

$$S1 = (10 \times 0.005)/10.47$$

$$= 0.00478 \text{ N}$$

Table 5.8: Iodine content of salts(S 11,S 12,S 13,S 14,S 15) after iodometric titration

Sample Code	Initial Burette Reading (ml)	Final Burette Reading (ml)	Difference (ml)	Iodine (ppm) (R×100×1000 ×0.127×N)/6
S 15	34.5	37.5	3	30.35
S 14	37.6	41.6	4	40.46
S 13	41.7	42.9	1.2	12.14
S 12	43	44.7	1.7	17.20
S 11	35.4	38.9	3.5	35.41

From the iodine ppm of S 11, S 12, S13 ,S14 , S15 it is shown that, S11, S14,S15 are in acceptable range (20-50 ppm) and S12 & S13 are below the range.

Table 5.9: Standardization of $Na_2S_2O_3$

Observation No.	Initial Burette Reading (ml)	Final Burette Reading (ml)	Difference (ml)	Average (ml)
1	0.2	12	11.8	
2	12	23	11	10.83
3	23	32.7	9.7	

$$S_1 V_1 = S_2 V_2$$

$$S1 = (10 \times 0.005)/10.83$$

$$= 0.004615 \text{ N}$$

Table 5.10: Iodine content of salts(S 16,S 17,S 18,S 19,S 20) after iodometric titration

Sample Code	Initial Burette Reading (ml)	Final Burette Reading (ml)	Difference (ml)	Iodine (ppm) (R×100×1000 ×0.127×N)/6
S 16	32.7	34.7	2	19.54
S 17	34.7	36.5	1.8	17.58
S 18	36.7	38.6	1.9	18.56
S 19	38.6	42.3	3.7	36.14
S 20	42.3	47	4.7	45.91

From the iodine ppm of S16, S17, S18, S19, S20 it is shown that, S16, S17, S18 are in acceptable range (20-50 ppm) and S19 & S20 are below the range.

5.2 Result:

Table 5.11: Result of the iodine content of ten salt samples

Serial No.	Salt No	Iodine ppm	Serial No.	Salt No.	Iodine ppm
1	S1	32.51	6	S6	36.32
2	S 2	9.75	7	S7	35.81
3	S 3	8.67	8	S8	26.13
4	S 4	33.20	9	S 9	33.20
5	S5	31.13	10	S10	31.13

Table 5.11 shows the iodine content (ppm) in S1, S2, S3, S4, S5, S6, S7, S8, S9, S10 salts.The iodine comtent of S1,S2, S3, S4, S5, S6, S7, S7, S8,S9, S10 were 32.5, 9.75, 8.67, 33.20, 31.13, 36.32, 35.81, 26.13, 33.20, 31.13 respectively.

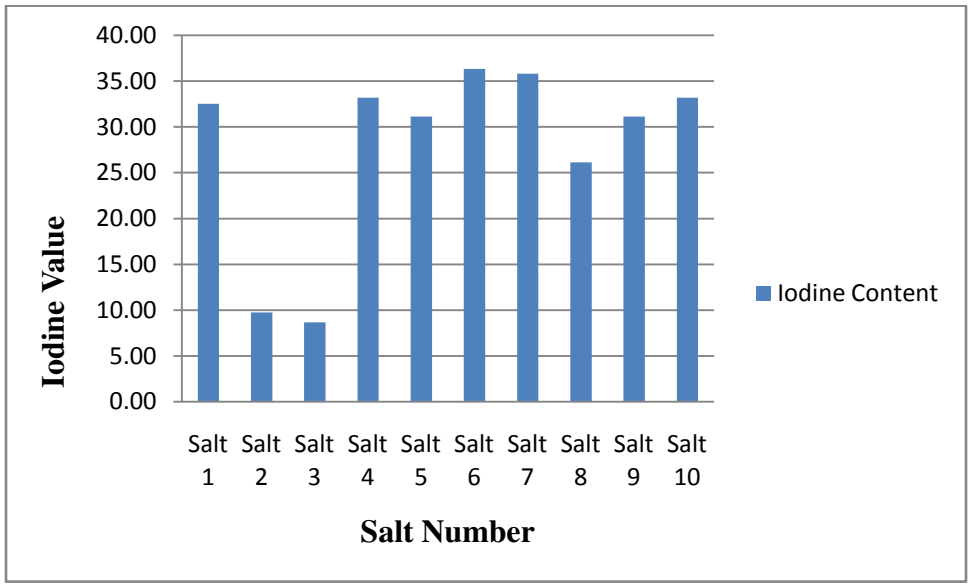


Fig 5.1: Iodine content (ppm) in different household salt samples (S1-S10)

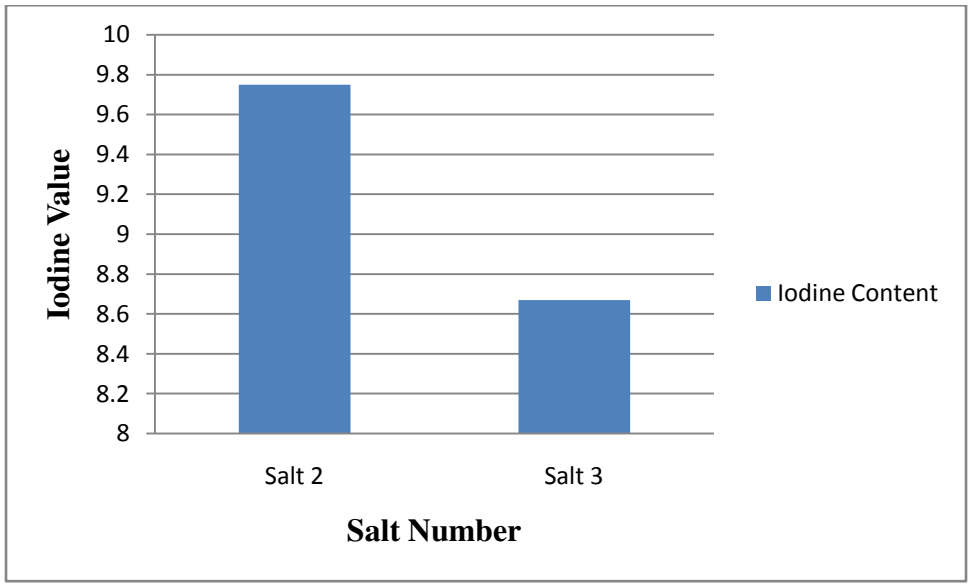


Fig 5.2: Low iodine content in different household salts (S2, S3)

Table 5.12: Result of the iodine content of another ten salt samples

Serial No.	Salt No.	Iodine ppm	Serial No.	Salt No.	Iodine ppm
11	S11	35.41	16	S16	19.54
12	S12	17.20	17	S17	17.58
13	S13	12.14	18	S18	18.56
14	S14	40.46	19	S19	36.14
15	S 15	30.35	20	S20	45.91

Table 5.11 shows the iodine content (ppm) in S11, S12, S13, S14, S15, S16, S17, S18, S19, S20 salts. The iodine content of S11, S12, S13, S14, S15, S16, S17, S18, S19, S20 were 35.41, 17.20, 12.14, 40.46, 30.35, 19.54, 17.58, 18.56, 36.14, 45.91 respectively.

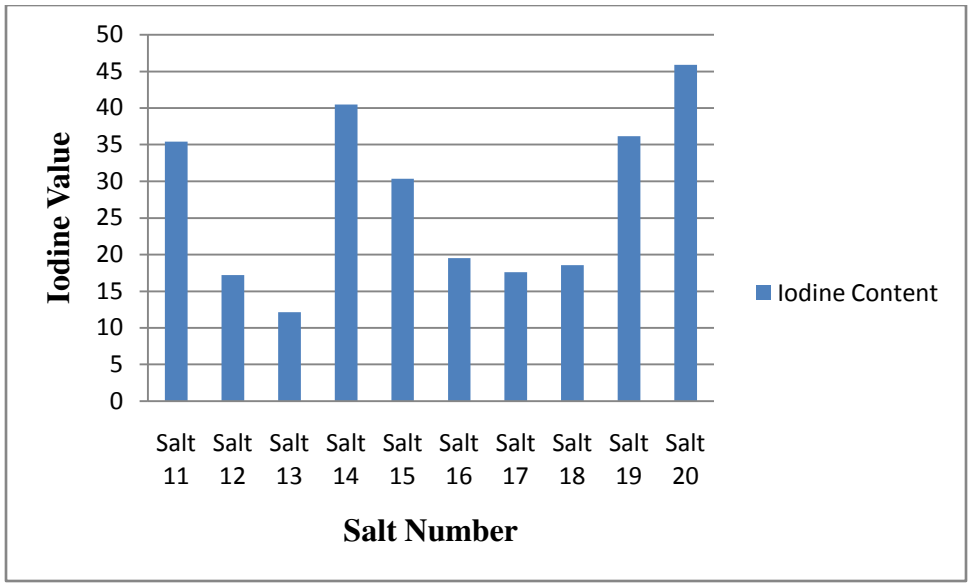


Fig 5.3: Iodine content (ppm) in different household salt samples (S11-S20).

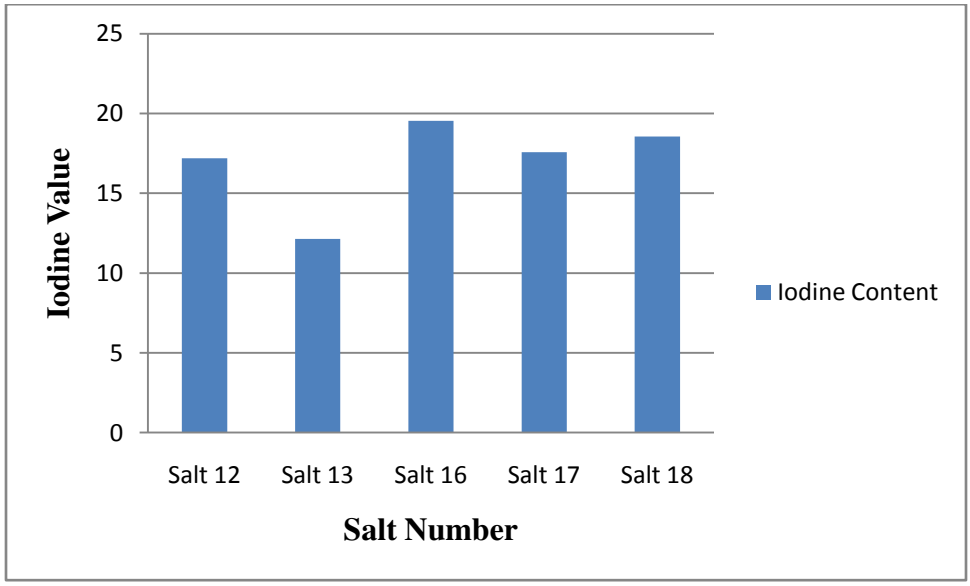


Fig 5.4: Low iodine content in different household salt samples(S 12, S 13, S 16,S 17,S18)

Table 5.13: Compliant and non-compliant iodine content in different household salts.

Salt No.	Consequences	Salt No.	Consequences
1	Compliant with WHO guideline (20-50 ppm)	11	Compliant with WHO guideline (20-50 ppm)
2	Do not Compliant with WHO guideline (20-50 ppm)	12	Do not Compliant with WHO guideline (20-50 ppm)
3	Do not Compliant with WHO guideline (20-50 ppm	13	Do not Compliant with WHO guideline (20-50 ppm)
4	Compliant with WHO guideline (20-50 ppm)	14	Compliant with WHO guideline (20-50 ppm)
5	Compliant with WHO guideline (20-50 ppm)	15	Compliant with WHO guideline (20-50 ppm)
6	Compliant with WHO guideline (20-50 ppm)	16	Do not Compliant with WHO guideline (20-50 ppm)
7	Compliant with WHO guideline (20-50 ppm)	17	Do not Compliant with WHO guideline (20-50 ppm)
8	Compliant with WHO guideline (20-50 ppm)	18	Do not Compliant with WHO guideline (20-50 ppm)
9	Compliant with WHO guideline (20-50 ppm)	19	Compliant with WHO guideline (20-50 ppm)
10	Compliant with WHO guideline (20-50 ppm)	20	Compliant with WHO guideline (20-50 ppm)

CHAPTER -6

DISCUSSION AND CONCLUSION

6.1 Discussion

Iodine is a micronutrient of crucial importance for the health and well-being of all individuals. The body does not make iodine, so it is an essential part of diet. If there is insufficient iodine in our body, enough thyroid hormone is not make. IDDs are defined as all the consequences of iodine deficiency in a population that can be prevented by ensuring that the population has an adequate intake of iodine. The WHO/UNICEF/ICCIDD recommendation is to add iodine to the salt at a concentration of 20-50 ppm(parts per million) (Chung 2014) .

The major risk factors of I deficiency in the Bangladeshi population are household food insecurity, lack of access to iodised packaged salt, rural residency, low levels of awareness about the health benefits of iodine and iodised salts, consumption of industrial salt (non-iodised) and lack of preservation knowledge about iodised salts have been identified .
A study in Sri Lanka has shown that the six different brands of most commonly sold commercial edible iodized salts in different grocery stores have excess iodine that is above the fortification level of 15–30 mg kg^{-1}. As a total 95.8 % cases can cause Iodine Induced Hyperthyroidism (IIH) and only 4.1 % of them can provide optimal iodine nutrition in a population (Vinthanage et al. 2016).

A quantitative analysis on table salts collected from different local markets, and local eateries of Nigeria has shown that the both commercially available salts and those from local eateries are compliant with the USI mandate and are therefore provide dietary intake of preventing nutritional problems such as Iodine Deficiency Disorders (Tyndall et al. 2013)

The household salt iodine content of school aged children in the hilly and the plain districts of eastern was held in Nepal. Among the 707 salt samples which were collected from the households, had <15 ppm iodine , were 123 (17.3%) and those which had >15 ppm iodine , were 584 (82.6%) (Nepal et al. 2013).

A study was conducted between September 2004 and March 2005,and it was found that in children, the total goitre rate (TGR) was 6.2%, compared to 49.9% in 1993 and the TGR among women was 11.7%, while in 1993 it was 55.6%. Prevalence of iodine deficiency (Urinary Iodine

Excretion <100 micro g/L) was 33.8% in children and 38.6% in women (compared to 71.0% and 70.2%, respectively in 1993).The result shows that Bangladesh has achieved a commendable progress in reducing goitre rates and iodine deficiency among children and women ever since the universal salt iodization programme was instituted 10 years ago (Yusuf et al. 2008).

Another study was conducted in the year of 2010 and according to urinary iodine concentration classified by the WHO, the level of iodine deficiency was severe in 4% of the adolescent girls. mild to moderate deficiency existed in 33% of the girls. One fifth (20%) of pregnant women had severe iodine deficiency and 37% women who demonstrated mild to moderate levels of urinary iodine. From that study it can be found that, the sub-clinical iodine deficiency is still prevalent in adolescent girls and pregnant womens (Ara et al. 2010).

In our study among 20 salt samples which are collected from the household of tea plantation worker in the area of Srimangal, 7 samples has shown low content of iodine and rest of the 13 samples shows the iodine content in acceptable range according to WHO guideline. From our study it can be measured that these 7 houses people are in a risk of iodine deficiency.

The most effective way to control IDD is universal salt iodization, which refers to iodization of all salt used for human consumption (industrial and household use). This strategy is recommended because salt is used essentially in all foods and its intake is consistent throughout the year. Iodization is a simple, inexpensive technique, and does not affect salt color or flavor. The amount of iodine in the salt may be monitored in production, retail and in the home.

6.2 Conclusion

The major impact of hypothyroidism due to iodine deficiency is impaired neurodevelopment, particularly early in life. In the fetal brain, inadequate thyroid hormone impairs myelination, cell migration, differentiation and maturation. Thus, with a focus on vulnerable groups such as pregnant women and young children, iodine prophylaxis of deficient populations is essential to avoid the many adverse effects of ID on growth and development (Zimmermann2011).

So that the salt iodine content of the household salts should be regularly monitored to know the fortification status of the iodine in the salt. As salt iodine is used as a universal vehicle for the salt iodization, to know the levels of iodine in ppm in the household salts. The use of the iodometric titration is recommended for the laboratory analysis of the salt iodine content. The use of iodized salt is a simple and harmless way of supplementing the iodine obtained from food .Bangladesh has achieved a commendable progress in reducing goitre rates and iodine deficiency among children and women by the universal salt iodization programme. More awareness campaigns regarding the usage and the practice of iodized salts are required to be conducted by governmental agencies, to ensure the consumption of iodized salt for the elimination of the iodine deficiency and also Bangladesh government has made it mandatory to market iodised salts.

CHAPTER -7

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