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# Assessment of Iodine Deficiency Disorders Among School Children in Amuma District, Beneshangul Gumuz, Ethiopia

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# 1. INTRODUCTION

Iodine is an essential element required for normal human growth and development, even before birth. Although iodine is an important micronutrient, it is needed only in very small quantities. In order to prevent deficiency, a person needs only 250 μg of iodine per day and over a life time, the total quantity of iodine needed is only one teaspoonful. [1]

Iodine is an essential component of the thyroid hormones. Failure to have adequate iodine leads to insufficient production of these hormones, which affect many different parts of the body resulting in a number of pathologic conditions known as the iodine deficiency disorders (IDD). Iodine deficiency disorders refer to all of the consequences of iodine deficiency in a population that can be prevented by ensuring that the population has an adequate intake of iodine. Iodine deficiency continues to be a significant public health problem in many countries, its deficiency not only causes goiter, it may also result in abortion, still birth, mental retardation, growth retardation, irreversible brain damage and retarded psychomotor development in the fetus, infant and in the child. It also affects reproductive function and impedes children learning ability.[2]

The thyroid gland is dependent on dietary iodine for the production of thyroid hormones, normal iodine requirement being about 150-200 μg/day. Long – term deficiency in iodine intake is associated with the development of goiter. When the prevalence of goiter in a population rises above 5-10%, the problem is considered as endemic. So iodine is an essential component of the thyroid hormones, and adequate iodine intake is necessary for normal thyroid function. Thyroid hormones, thyroxine contains four atoms of iodine and $T_3$ contains three atoms of iodine. Iodine enters the thyroid in the inorganic form as iodide that is derived either from food, water and drugs or from deiodination of thyroid hormones. IDD are now considered as a major public health problem all over the world.[2, 3]

Its major manifestations are goiter (enlargement of thyroid gland than normal), mental defects, deaf mutism, still birth and miscarriages, weakness and paralysis of muscles as well as lesser degree of physical and mental dysfunction. Iodine deficiency also affects the socio-economic development of a community. Several indicators exist for the assessment of iodine status, both on the individual and population level by the criteria that WHO developed. Among them goiter rate and iodine content of edible salts are the common one. [3]

In Ethiopia IDD has been recognized as a public health problem for many decades. A recent study[4] that did both clinical and biochemical assessment has conformed that the situation of IDD deteriorated. In high goiter endemic regions the prevalence of goiter in children was greater than 30% while in less endemic regions it was less than 15%. In order to ensure adequate availability and use of iodized salt, the government of Ethiopia designs a five year strategic plan to eliminate IDD through out the country through universal availability of iodized salt to the entire population.

Amuma district is a known iodine deficiency disorders endemic area in Wombera wereda in which two – third of the populations have visible goiter. A study conducted in 2007 in the region reported a goiter prevalence of 37.3% in the region. Despite the recognition of the problem in the region, an iodine deficiency controlling program was never officially implemented and no survey has been conducted on the status of iodine deficiency in Amuma district. Hence the present study was conducted inorder to assess the prevalence of IDD and to estimate the iodine content of salt consumed in the households in Amuma district, wombera.

# 2. LITERATURE REVIEW

## 2.1. Iodine

Iodine (atomic wt 126.9 g/atom) is an essential component of the hormones produced by thyroid gland. Thyroid hormones and therefore iodine are essential for mammalian life, including humans. Iodine is a trace element found naturally and unevenly distributed in soil.
It is very volatile and sublimates easily or passes from a solid state directly to gas form ($I_2$). It is also water soluble. Access to iodine on a daily basis is through drinking water and consuming food originating from crops and plants grown on the earth. For instance, people living in the islands or coastal areas have access to marine foods, including fish and seaweeds that are known to be rich in iodine.[3, 5] The foods grown on iodine-deficient soil have lower iodine content than those produced in iodine-rich soil and man and animals consuming water and crops from the area of iodine deficient soils themselves become deficient in iodine. Iodine deficiency is the main cause of endemic goiter, but other dietary substances (termed goiterogens) that interfere with thyroid metabolism can aggravate the effect (Table 1).[5]

**Table 2.1.** Goitrogens and their mechanism[5].

| **Foods** | **Mechanism** |
|---|---|
| Cassava, lima beans, linseed, sourghum,sweet potato | Contain cytogenic glycosides; they are metabolized to thiocyanate that compete with iodine for thyroidal uptake. |
| Cabbage, kale, cauliflower, broccoli, turnips, rapeseed | Contain glucosinolates; metabolites compete for thyroidal uptake. |
| **Industrial pollutants** | |
| perchlorate | Competitive inhibitor of the sodium/iodine symportor, decreasing iodine transport into the thyroid. |
| smoking | An important goiterogen; smoking during breast feeding is associated with reduced iodine concentrations in breast milk. |
| **Nutrients** | |
| Iron deficiency | Reduces heame-dependent thyroperoxidase activity in the thyroid and might blunt the efficacy of iodine. |

IDD is a globalizing name for a spectrum of disorders caused by iodine deficiency, manifested by enlargement of thyroid gland, also referred as goiter.[6] Brain damage and irreversible mental retardation are the most important disorders induced by iodine deficiency. Cretinism - a condition resulting from extreme form of iodine deficiency in and endemic goiter have been recognized as public health problems for centuries. The primary etiologic factor, iodine deficiency, was hypothesized in 1851 as the cause of goiter[7], although goiter had been recognized in the earliest of ancient history and was treated by giving seaweeds or burnt sponges to eat. [8]

The modern history of global efforts to eliminate goiter and cretinism started after the long debate in 1932, when scientists meeting in Bern, Switzerland, correctly identified the cause of the problem as iodine deficiency and consensually advocated iodine prophylaxis on a national scale. Today, control of iodine deficiency is an integral part of the most national nutrition strategies.[7, 9]

## 2.2. Pathophysiology of Iodine Deficiency

Thyroid enlargement (goiter) is the classic sign of iodine deficiency, and can take place at any age, even in newborn babies. It is a physiological adaptation to chronic iodine deficiency. As the iodine intake falls, secretion of thyroid stimulating hormone increases in an effort to maximize uptake of available iodine, and TSH stimulates thyroid gland to produce the thyroid hormones.[10] Initially goiters are characterized by diffuse, homogeneous enlargement, but over time nodules often develop. Iodine is rapidly and nearly wholly absorbed (>90%) in the stomach and duodenum. Iodate, widely used in iodization of salt, is reduced in the gut and absorbed as iodide. Originally bound iodine is typically digested and the released iodide absorbed, but about 75% of an oral dose of the thyroid hormone $T_4$ is absorbed (as shown in fig. 2.1).

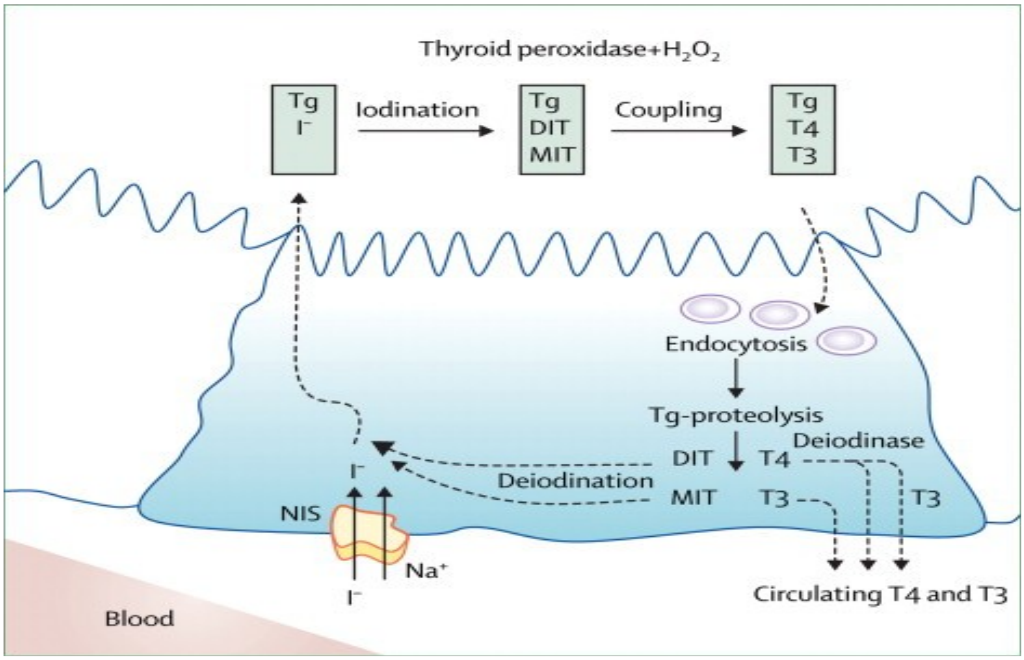
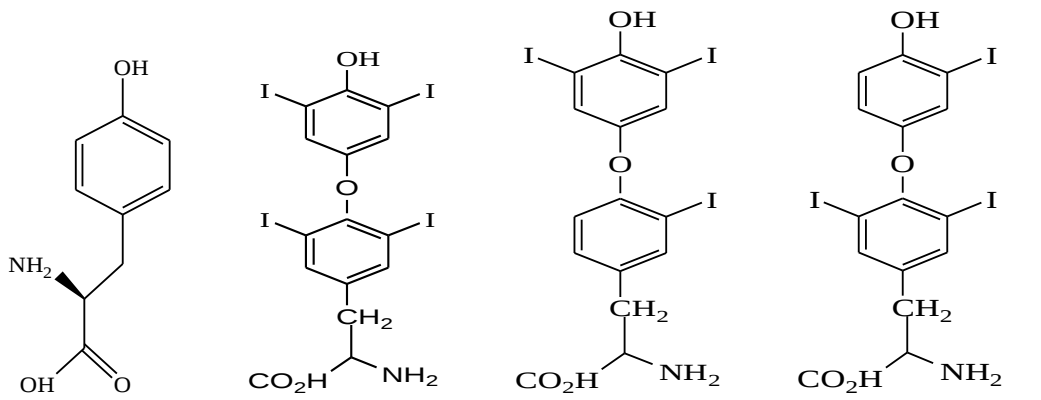



**Figure 2.1.** Iodine pathway in the thyroid cell

**Bioinorganic chemistry of iodine**

Iodide is an essential substrate for the production of thyroid hormone (Fig.2.2). The source of iodide (iodine) is dietary. Since iodine is a scarce nutrient, the thyroid gland has a transport protein that concentrates iodide from the serum. This protein, located on the basolateral membrane of the thyroid follicular cell, actively transports iodide against a concentration gradient, because sodium is co-transported with iodide, the transport protein is called the sodium/iodide symporter (NIS).[11]




Tyrosine Thyroxine ($T_4$) Triiodothyronine ($T_3$) `` Reverse $T_3$ ``

(inactive)

**Figure 2.2.** The structure of thyroid hormones.

**Dietary Iodine Requirements: -** The dietary iodine requirement for adequate synthesis of thyroid hormone is at least 100 µg/day. This is reflected in urine iodine of about 150 µg/l creatinine.

**Thyroid peroxidase: -** Thyroid peroxidase (TPO) is a glycoprotein located on the apical membrane of the thyroid follicular cell. Its function is to oxidize iodine to a higher valence state for incorporation into tyrosine (organification) to produce monoiodotyrosine and diiodotyrosine with in the thymoglobulin molecule. It is also responsible for the coupling of iodotyrosine and diiodotyrosine with in thymoglobulin molecule.

**Hydrogen peroxide generation ($H_2O_2$):-** Hydrogen peroxide is essential for oxidation of iodations of iodide, organification of iodide and coupling of iodotyrosines. The $H_2O_2$ generation system is located on the apical membrane where TPO also resides. The precise biochemical mechanism resulting in generation of $H_2O_2$ remains controversial. TSH stimulates the process and high concentration of iodide inhibits it.[1,12] It is generally believed that an enzyme of the peroxidase group, thyroid peroxidase, is responsible for the iodination of the amino acid and iodide in the thyroid gland as shown in Fig.2.3.

The thyroid gland makes thyroid hormones from iodine absorbed from the food we eat, the larger the amount of thyroid hormone produced, the faster the cells work. When less thyroid hormone is produced, the cells work slower. The pituitary glands-stimulating hormone controls levels of hormones secreted by the thyroid.[12]

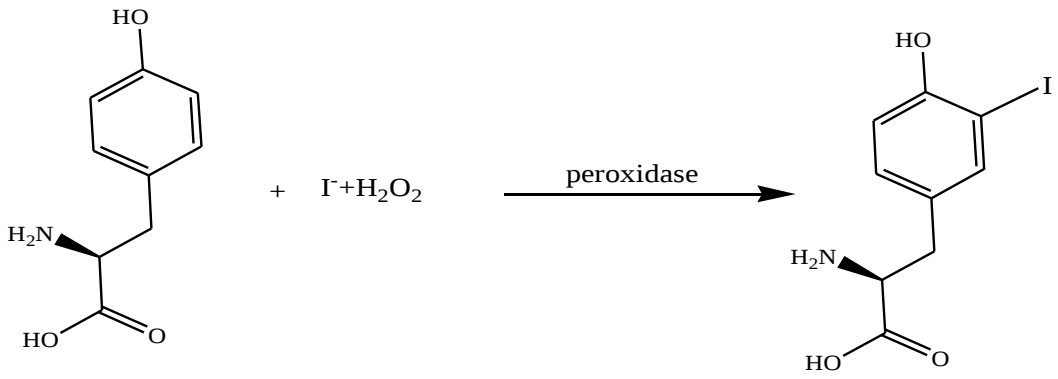



**Figure 2.3**. Iodination of tyrosine by using TPO and $H_2O_2$

The thyroid gland produces thyroid hormone in a mechanism by which it combines the element iodine with the amino acid tyrosine. In this process, one or two iodine molecules attach to a tyrosine molecule to form iodinated tyrosine (iodotyrosines). Compounds containing one iodine or two iodine molecules then combine with one another in a process known as coupling to form the primary thyroid hormones $T_4$ (tetra iodothyronine or thyroxin with four iodine molecules) and $T_3$ (triiodothyronine with three iodine molecules ).

The synthesis of $T_4$ by TPO involves two independent steps:-

1. Iodination of tyrosine and phenol coupling of the resulting iodotyrosin residues.
2. Thyroxine ($T_4$) is then converted to its biologically active form $T_3$ by an outer ring deiodination Pathway.

Although both $T_3$ and $T_4$ are important for normal growth, development and energy metabolism, $T_3$ is more active than $T_4$.

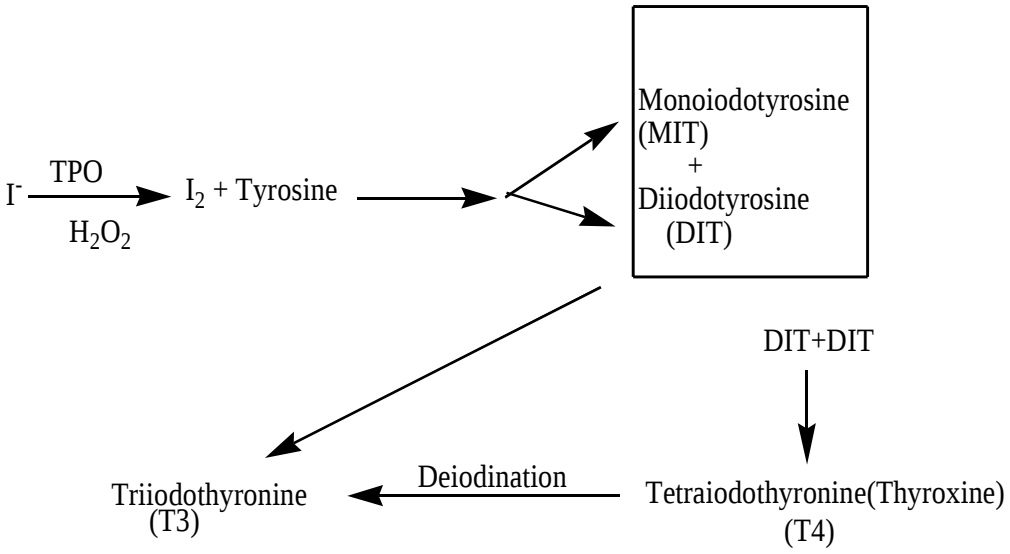



**Figure 2.4.** Iodination of tyrosine and formation of thyroid hormones.

As shown in Fig. 2.3, organification (oxidation) of iodide ($I^-$) to iodine ($I_2$) is accomplished by $H_2O_2$ catalyzed by the enzyme thyroid peroxidase (TPO) and leads to the iodination of iodotyrosines and eventual formation of thyroid hormones triiodothyronine ($T_3$) and thyroxine ($T_4$). Since $H_2O_2$ is major oxidant in the body, the more that is used up in the oxidation of $I^-$, the less is available for other potentially damaging oxidative processes and hence the role of $I^-$ as an antioxidant. Normally, the thyroid gland secretes an abundance of $T_4$ along with smaller amount of $T_3$. When $T_4$ reacts with the cell, liver brain and skeletal muscle, it loses an iodine atom and become $T_3$. $T_3$ is approximately 10 times more potent than $T_4$, although both $T_4$ and $T_3$ serve vital function.[13, 14]

### 2.2.1 Iodine deficiency disorders (IDD)

Iodine Deficiency Disorders refer to all of the ill-effects of iodine deficiency in a population that can be prevented by ensuring that the population has an adequate intake of iodine. Iodine deficiency occurs when iodine intake falls below recommended levels.

For more than 100 years, iodine has been known as the element that is necessary for thyroid hormone production. Iodine is found in each of the trillions of cells in the body- with out adequate iodine level, life itself is not possible.[15, 16]

Iodine is not only necessary for the production of thyroid hormone; it is also responsible for the production of all of the other hormones of the body. Adequate iodine levels are necessary for proper immune system function. Iodine contains potent antibacterial, anti parasitic, antiviral and anticancer properties. Iodine deficiency disorder can result in mental retardation, goiter, increased child and infant mortality, and socioeconomic decline. Every cell in the body contains and utilizes iodine, but iodine is concentrated in the glandular system of the body. The thyroid gland contains higher concentration of iodine than any other organ. Iodine is essential for the normal growth and development of children. Sever iodine deficiency can result in sever mental deficiency and deafness (i.e. cretinism). In addition, spontaneous abortion as well as delayed physical and intellectual development is associated with iodine deficiency. Attention deficit /hyperactivity disorder is also related to iodine deficiency.[17]

### 2.2.2. Impact of iodine deficiency disorders (IDD)

A large proportion of the world's population is at risk of iodine deficiency, which is responsible for a variety of iodine deficiency disorders. The problems associated with these disorders include goiter, hyperthyroidism, impaired mental function, spontaneous abortions, congenital abnormalities, increased infant mortality and cretinism.

**Hypothyroidism**

Hypothyroidism is the result of thyroid hormone insufficiency, which occurs during severe and prolonged iodine deficiency. Hypothyroidism is characterized by reduced metabolic rate, diminished vigor, decreased basal body temperature and cold intolerance.

In spite of decreased appetite, there is weight gain. The skin becomes dry, rough and thickened, the hair become coarse. Loss of hair is also a characteristic symptom.[18]

Metabolic changes resulting from hypothyroidism include positive nitrogen balance due to slowing of protein degradation; slowed glucose absorption due to delayed insulin secretion; a flat glucose tolerance curve; decreased lipid metabolism; accumulation of triglycerides and cholesterol in the blood. The other result is to decrease conversion of carotene to vitamin A, which makes the skin yellowish color. Additional symptoms that may appear in juvenile hypothyroidism include delayed eruption and deformation of teeth, increased dental caries, excessive body and facial hair, brittle scalp hair, increases muscle size, growth retardation and decreased intellectual performance. These complications of adult and juvenile hypothyroidism due to iodine deficiency are reversible with iodine therapy and supplementation. [18,19]

**Goiter**

Goiter, an enlargement of the thyroid gland, usually represents thyroid hyperplasia in response to insufficient iodine intake. With iodine deficiency, $T_4$ concentrations in the blood fall, and the feedback of low $T_4$ on the pituitary leads to increased production of TSH. TSH stimulates hyperplasia of the thyroid with increased uptake of iodide, and the size of the thyroid increases, resulting in a goiter. By palpation on physical examination, goiter is defined as enlargement of the thyroid such that the lateral lobes are larger than the terminal phalanx of the thumb of the person who is being examined. Iodine deficiency is responsible for simple goiter, a condition characterized by swelling in the neck caused by enlargement of the thyroid gland. The enlargement is apparently a compensatory adaptation to the lacked of iodine required for the synthesis of thyroid hormones. The direct stimulus of the enlargement is an abnormally high level of TSH, itself brought about by low plasma levels of the thyroid hormones.[19] Iodine intakes consistently lower than 50 μg per day usually result in goiter, however, not all people with iodine deficiency develop goiters. Women and particularly adolescent girls seem to be especially at risk. In female, endemic goiters generally persist throughout life, while in males they tend to decrease following adolescence.[20]

**Table 2.2.** Recommendations for iodine intake by age or population group

**US Institute Medicine recommendation**

| **Age/state** | **Iodine intake (µg per day)** |
|---|---|
| Infants 0-12 month | 110-130 |
| 1-8 years | 90 |
| Children 9-13 years | 120 |
| Children ≥ 14 year+adults | 150 |
| Pregnancy | 220 |
| Lactation | 290 |

**WHO recommendation**

| | |
|---|---|
| Children 0-5 years | 90 |
| Children 6-12 years | 120 |
| Children ≥ 12 years + adults | 150 |
| Pregnancy | 250 |
| Lactation | 250 |

(Source: WHO, 2008 trace elements in human nutrition)

Persons with large goiter are more likely than others to have manifestations of poor thyroid function, especially hypothyroidism. A large goiter, and especially one that enlarges behind the upper part of the sternum, may cause pressure on the trachea. And esophagus, which may interfere with breathing, cause and irritable cough or voice changes and occasionally affect swallowing. Also, goiters are associated with an increased risk of other thyroid diseases and malignant growth.

Administration of appropriate doses of iodine results in a slow reduction in the thyroid gland size, although surgical removal of part of the gland may be required in the most severe cases. When surgery is necessary, it is complicated by the presence of parathyroid glands, which are entrenched in the thyroid gland.[19, 20]

**Cretinism**

Cretinism is the most severe consequences of hypothyroidism occurring during fetal and neonatal life. The effects of cretinism, which is congenital defect of mental and physical development, are irreversible. Two types of cretinism are found: neurological cretinism and myxedematous cretinism. The key feature of neurological cretinism is mental retardation but short stature, spasticity, rigidity of muscles and deaf mutism are also often found. The main organs affected are the inner ear, cochlea and the brain. The most significant stages in the development of these organs occur during the second trimester of pregnancy, and iodine deficiency during this critical time is believed to be responsible for the major manifestations of neurological cretinism. Myxedematous cretinism is also associated with mental retardation, but its victim are shorter in stature than neurological cretins and have clear hypothyroidism, but do not usually suffer from spasticity, goiter or deaf mutism.[21]

Concerns about potential increases in iodine induced thyroid disease continue to delay or limit the implementation of iodine in iodine- deficient populations. Looking at the benefits vs the risks of iodine prophylaxis, it is clear that severe iodine deficiency in pregnancy can cause hyperthyroidism, poor pregnancy outcome, cretinism and irreversible mental retardation. Mild to moderate iodine deficiency in children results in severe learning disability, poor growth and diffuse goiter. In adults, it causes higher rates of more aggressive subtype of thyroid cancer and increases risk for toxic nodular goiter and associated hyperthyroidism.

## 2.3. Epidemiology of Iodine Deficiency Disorder

### 2.3.1. Global prospective of IDD

In 2007, WHO estimated that nearly 2 billion individuals have an insufficient intake of iodine including all school- aged children (Table 2.3). The lowest prevalence of iodine deficiency is in the Americas (10.6%), where the proportion of the households consuming iodized salt is the highest in the world (about 90%).

The highest prevalence of iodine deficiency is in Europe (52%), where the household coverage with iodized salt is the lowest (25%) and many of the countries have weak or non-existent control program for iodine – deficiency disorders. 64% of households in sub-Saharan Africa use iodized salt, but coverage varies widely from county to county. It is currently estimated that 70% of households throughout the world have access to (and use) iodized salt. [20, 22]

**Table 2.3.** Prevalence of iodine deficiency in 2007 and percentage of households with access to iodized salt by WHO regions.

| | Population with urinary iodine < 100 µg/L | | Proportion of households with access to iodized salt |
|---|---|---|---|
| | As proportion of general population (all age groups) | As proportion of school age children (6-12 years) | |
| Africa | 41.5% (312.9) | 40.8% (57.7) | 66.6% |
| Americas | 11.0%(98.6) | 10.6% (11.6) | 86.8% |
| South-East Asia | 30.0% (503.6) | 30.3% (73.3) | 61.0% |
| Europe | 52.0% (459.7) | 52.4% (38.7) | 49.2% |
| Eastern Mediterranean | 47.2% (259.3) | 48.8% (43.3) | 47.3% |
| Pacific Western | 21.2% (374.7) | 22.7% (41.6) | 89.5% |
| Total | 30.6% (2000.0) | 31.5% (263.7) | 70.0% |

**Source**: WHO, 2007 global prospective of prevalence of iodine deficiency

### 2.3.2. Iodine Deficiency Disorders in Ethiopia

In Ethiopia IDD has been recognized as public health problem for more than four decades since 1950. Many Studies have shown that goiter is prevalent in the various parts of the country. According to the 1978 nation- wide survey (ENI report 1980) the total goiter rate (TGR) and visible goiter rate (VGR) were 54% and 28% respectively; where in one area the TGR was as high as 71%.[23]

In 1980 and 1981 a stratified goiter survey was conducted on school children and household members in all regions of Ethiopia except Eritrea and Tigray.[20] The gross goiter prevalence among school children and household members was 30.6% and 18.7% respectively, while that of visible goiter was 1.6% and 3.2% respectively. Prevalence was higher in females than males and increased with age more in females than in males. The prevalence rates are at higher altitudes were higher than those at lower altitudes in both schoolchildren and household members. According to this survey and a situational analysis carried out by Ministry of health (MOH) and the United Nations Children's Fund (UNICEF) in 1993, 42 million people (78%) of the total population of Ethiopia are exposed to iodine deficiency, 35 million (62%) are iodine deficient, 14 million (26%) have goiter; with about 50,000 prenatal deaths and at least one in 1000 people is cretin, and that three times as many people (3.5/1000) may show some degree of developmental and neurological impairment attributed to IDD.[21] This report claims that many health problems in the country are attributable to iodine deficiency.

In 1995 and 1996, following the above knowledge and report; the Ethiopian Government has embarked on the universal salt iodization initiative. According the ENHRI 80% of the population in Ethiopia had been utilizing iodized salt prior to 1998; however, this figure has dropped to 4.2% of households due to the conflict with neighboring Eritrea- a major producer iodized salt. Since the 1998 border conflict between Eritrea and Ethiopia, Ethiopia's salt supply from Eritrea has been cut off. In the late 1990s, the Ethiopian Health and Nutrition Research Institute, Addis Ababa, undertook a cross-sectional study selecting 2,485 elementary school children belonging to ten villages from four administrative regions of Ethiopia.[22,23] The prevalence rate of goiter (average of male and female values) among school children was 53.3%. The prevalence was higher in girls (56.1%) than in boys (50.8%). The highest prevalence was observed in the villages of Lotte (82%) and Kodowono (91%) and the lowest in the village of Abossara (31%). The Government of Ethiopia in coordination with UNICEF (2005) has planned several strategies for achievement of virtual elimination of iodine deficiency disorders by the year 2015 through Universal Salt iodization in Ethiopia.

Although the National Guideline for Control and Prevention of Micronutrient Deficiencies formulated by the Federal Ministry of Health, Family Health Department (June 2004) states that "in Ethiopia an iodine content of 80-100 ppm is required as $KIO_3$ at the Port of entry or at the packaging factory. Iodine content for a level of 80 ppm or 80 mg/kg $KIO_3$ in one ton of salt = 80 grams $KIO_3$ in one ton of salt." The legislation requiring that salt for sale for human consumption should be iodized is currently not being enforced due to the non-availability of enough nationally produced iodized salt. In a recent study[24] that did both clinical and biochemical assessment has confirmed that the situation of IDD has deteriorated. In high goiter endemic regions the prevalence of goiter in children was greater than 30% while in less endemic regions it was less than 15%. In a more recent cross-section community based study conducted in women in child bearing age of 15 to 49 years.[24] The total goiter prevalence (weighted) was 35.8% (95% CI 34.5-37.1), 24.3% was palpable and 11.5% was visible goiter. This report demonstrates that more than 6 million women were affected by goiter. Goiter prevalence in four regional states namely southern Nation Nationalities and people (SNNP), Oromia, Benshangul-Gumuz and Tigray was greater than 30%, an indication of severe iodine deficiency.

**Table 2.4**. Goiter rate of women by regional states

| Region | Number examined | Palpable %(95% Cl) | Visible %(95%Cl) | TGR% (95% Cl) |
|---|---|---|---|---|
| Amhara | 1637 | 20.2% (17.4,23.0) | 8.6% (6.7,10.5) | 28.8% (25.7,31.9) |
| Oromia | 1816 | 20.0% (17.4,22.6) | 11.3% (9.2,13.4) | 31.3% (28.3,34.3) |
| Tigray | 1796 | 22.2% (19.5,24.9) | 13.4% (11.2,15.5) | 35.6% (32.5,38.7) |
| SNNPR | 1702 | 43.2% (39.9,46.5) | 17.7% (15.1,20.3) | 59.9% (57.6,64.2) |
| Adis Ababa | 804 | 15.0% (11.5,18.5) | 7.3% (4.8.9.8) | 22.3% (18.2,26.4) |
| Afar | 728 | 14.1% (10.5,17.7) | 1.5% (0.2.2.8) | 15.6% (11.9,19.3) |
| Benshangul | 888 | 21.8% (18.0,25.6) | 15.5% (11.8,19.2) | 37.3% (32.8,41.8) |
| Dire Dawa | 749 | 11.5% (8.3,14.7) | 0.9% (0,1.8) | 12.4% (9.1,15.7) |
| Harari | 730 | 4.1% (2.1,6.1) | 2.6% (1.0,4.2) | 6.7% (4.0,9.3) |
| Gambela | 148 | 1.4% (0,4.1) | - | 1.4% (0,4.1) |
| Total goiter rate | 10998 | 24.3% (23.2,25.4) | 11.5% (10.7,12.3) | 35.8% (34.5,37.1) |

**Source** : MOH, micronutrient deficiencies in human nutrition.

Following the research carried out by a UNICEF consultant, it was evident that consumers in Ethiopia were aware of the link between goiter and lack of iodine. However, they were not aware of the wider consequences of IDD in mental capacity. In Ethiopia, awareness of IDD problems and the benefits of iodized salt and iodized oil supplements by the public are low. Only 4.2% percent of Ethiopian 77 million people consume iodized salt- among the lowest percentage in the world. In Ethiopia if the problem continues a generation will suffer (ICIDD News, February 2008).

Currently, much of Ethiopia's salt is imported from Djibouti and is not iodized, although producers in Djibouti have the capacity to iodize it. There are only two main iodization plans in Ethiopia, one at Mekelle and another near Addis Ababa but hundreds of small-scale units are functioning along the shores of Lake Afdera with limited knowledge and skill in salt iodization, each with an individual production varying between 3,000 to 5,000 tons. In 2009 less than 1% of salt produced in the country is iodized[25].

In 2009, the Federal Ministry of health and the afar regional state in collaboration with UNICEF and Micronutrient Initiative (MI) have initiated salt iodization launching and scale up program at the national level by 2010 through the establishment of a central iodization facility in Afdera equipped with iodization machines, which repack salt for distribution across the country.[25, 26] It is therefore crucial to embrace Universal Salt iodization initiative as the most feasible and cost effective strategy for combating IDD and enhance the health and the prosperity of the nation.

## 2.4. Assessment and diagnosis of IDD

Several indicators exist for the assessment of iodine status, both on the individual and population level. The WHO has developed criteria for iodine deficiency as a public health problem in populations.[27, 28, 29]

### 2.4.1. Urinary iodine excretion (UIE)

Most iodine absorbed in the body eventually appears in the urine. Therefore, urinary iodine excretion is a good marker of very recent dietary iodine intake. Median UIC is recommended for defining the population iodine status through a specific group of people, such as pregnant women, lactating women, infants and school-age children.[30] UIC assesses iodine nutrition status only at the time of measurement. In individuals, urinary iodine excretion can vary some what from day to day and even within a given day. However, this variation tends to even out among populations.

Studies have convincingly demonstrated that a profile of iodine concentrations in morning or other casual urine specimens (child or adult) provides an adequate assessment of a population's iodine nutrition, provided a sufficient number of specimens are collected. Round the clock urine samples are difficult to obtain and are not necessary. Relating urinary iodine to creatinine, as has been done in the past, is cumbersome, expensive, and unnecessary. Indeed, urinary iodine/creatinine ratios are unreliable, particularly when protein intake and consequently creatinine excretion – is low.

**Table 2.5.** Criteria for assessing iodine nutrition based on median urinary iodine concentrations of school-age children (≥ 6 years).

| Median urinary iodine | Iodine intake | Iodine nutrition status |
|---|---|---|
| < 20 | Insufficient | sever iodine deficiency |
| 20-49 | Insufficient | moderate iodine deficiency |
| 50-99 | Insufficient | mild iodine deficiency |
| 100-1999 | Adequate | optimal |
| 200-299 | More than adequate | risk of induced - hyperthyroidism |
| >300 | Excessive | risk of adverse health consequence |

**Source** : WHO/UNICEF/ICCIDD. Assessment of IDD and monitoring their elimination[33].

### 2.4.2. Thyroid size or goiter grading

The size of the thyroid gland changes inversely in response to alterations in iodine intake, with a lag interval that varies from a few months to several years, depending on many factors. These include the severity and duration of iodine deficiency, the type and effectiveness of iodine supplementation, age and sex. The term “goiter” refers to a thyroid gland that is enlarged. The statement that “a thyroid gland each of whose lobes have a volume greater than the terminal phalanges of the thumb of the person examined will be considered goitrous”. It has been used in most epidemiological studies of endemic goiter and is still recommended. [31]

Palpation of the thyroid is particularly useful in assessing goiter prevalence before the introduction of any intervention to control IDD. Costs are associated with mounting a survey, which is relatively easy to conduct, and training of personnel. The goiter rate is often used for the assessment of iodine status in a population, and the goiter rate includes both visible and palpable goiter. The WHO has adopted a grading classification for goiter.

Determination of thyroid size by palpation method is more important method, especially in areas where iodine deficiency is high endemic. Health workers can easily conduct up to 100 examinations per day. The WHO and the ICCIDD has recently recommended reference values for thyroid volume in school-aged children based on variation in thyroid size.[32]

Classification for goiter according to WHO:-

**Grade 0**:- No palpable or visible goiter.

**Grade 1**:- A mass in the neck that is consistent with an enlarged thyroid that is palpable, but not visible when the neck is in the normal position: it moves upward in the neck as the subject swallows: nodular alteration(s) can occur even when the thyroid is not enlarged.

**Grade 2** :- A swelling in the neck that is not visible when the neck is in a normal position and is consistent with an enlarged thyroid when the neck is palpated.

It is recommended that a total goiter rate or TGR (number with goiters of grades 1 and 2 divided by total examined) of 5% or more in school children 6-18 years of age be used to signal the presence of a public health problem. This recommendation is based on the observation that in normal, iodine-replete populations, the prevalence of goiter should be quite low. The cut-off point of 5% allows both for some margin of error of goiter assessment.

**Total Goiter Rate** = Sum of goiter grades 1 and 2

**Table 2.6.** Epidemiological criteria for assessing the severity of IDD based on the prevalence of goiter in school-aged children.

| Prevalence of goiter | Severity of IDD | Traffic light color |
|---|---|---|
| 0.0-4.9% | None | Green |
| 5.0-19.9% | Mild | Yellow |
| 20.0-29.9% | Moderate | Orange |
| ≥ 30% | Severe | Red |

**Source** : WHO/UNICEF/ICCIDD. Assessment of IDD and monitoring their elimination[33].

### 2.4.3. Blood constitutes

Two blood constituents, TSH (thyroid stimulating hormone or thyrotropin) and thyroglobulin (Tg) can serve as surveillance indicators. In a population survey, blood spots on filter paper or serum samples can be used to measure TSH and/or Tg. Determining serum concentrations of the thyroid hormones, thyroxine ($T_4$) and triiodothyronine ($T_3$), is usually not recommended for monitoring iodine nutrition, because these tests are more cumbersome, more expensive, and less sensitive indicators.[32, 33]

**A/ Thyroid stimulating hormone (TSH)**

The pituitary secretes TSH in response to circulating levels of $T_4$. The serum TSH rises when serum $T_4$ concentrations are low, and falls when they are high. Iodine deficiency lowers circulating $T_4$ and raises the serum TSH, so that iodine-deficient populations generally have higher serum TSH concentrations than do iodine-sufficient groups. However, the difference is not great and much overlap occurs between individual TSH values.

Therefore, the blood TSH concentration in school-age children and adults is not a practical marker for iodine deficiency, and its routine use in school-based surveys is not recommended. In iodine sufficient populations, about 1 in 4000 neonates has congenital hypothyroidism, usually because of thyroid dysplasia. Prompt correction with thyroid hormone is essential to avoid permanent mental retardation. TSH screening is inappropriate for developing countries where health budgets are low. In such countries, mortality among children under five is high due to nutritional deficiencies and infectious diseases, and screening programmes for congenital hypothyroidism are not cost effective.

**B/ Thyroglobulin (Tg)**

Thyroglobulin is the most abundant protein of the thyroid, providing the matrix for thyroid hormone synthesis. Normally, small amounts are secreted or leak from the thyroid into the circulation. When the thyroid is injured, much larger amounts are released. The thyroid hyperplasia of iodine deficiency is regularly associated with increased serum Tg levels. In this setting, it reflects iodine nutrition over a period of months or years. This contrasts to urinary iodine concentration, which assesses more immediate iodine intake. Several studies have shown a good correlation with other markers of iodine deficiency, particularly goiter. The laboratory technique is similar to that for TSH and other immunoassay. It has been successfully applied to blood spots, but this particular application has not yet been developed commercially or studied further.

**C/ Thyroid hormone concentrations**

In contrast, thyroid hormone concentrations are poor indicators of iodine status. In iodine deficient populations, serum $T_3$ increases or remains unchanged, and serum $T_4$ usually decreases. However, these changes are often within the normal range, and the overlap with iodine sufficient populations is large enough to make thyroid hormone levels an intensive measure of iodine nutrition.[33]

## 2.5. Prevention and Treatment of IDD

### 2.5.1. Universal salt iodination

WHO and UNICEF Joint Committee on Health Policy recommended USI as a safe, cost-effective, and sustainable strategy to ensure sufficient intake of iodine by all individuals.
In nearly all countries where iodine deficiency occurs, it is now well recognized that the most effective way to achieve the virtual elimination of IDD is through USI.[34, 35] They recommend that iodine is added at a concentration of 20-40 mg iodine per kg salt, dependent on local salt intake. Iodine can be added to salt in the form of potassium iodate or potassium iodide. Because potassium iodate has higher stability than does potassium iodide in the presence of salt impurities, humidity and porous packaging, it is the recommended from tropical countries and those with low grade salt.[36]

USI involves the iodization of all human and livestock salt, including salt used in the food industry. Adequate iodization of all salt will deliver iodine in the required quantities to the population on a continuous and self-sustaining basis. The additional cost of iodine fortification in the process of salt production should eventually be borne by the consumer, but is negligible.

National salt iodization programs are now implemented worldwide and have followed a common pattern of evolution, which includes the following phases: Decision, implementation and consolidation. Successful salt iodization programme depends upon the implementation of a set of activities at the national level by various sectors includes: Government ministries, Salt producers, Concerned civic groups, nutrition, food and medical scientists and other key opinion markers. Opening the channels of communication and maintaining commitment and cooperation across these various groups is perhaps the greatest challenge to reaching the IDD elimination goal and sustaining it for the long term. Salt producers and distributors are critical in ensuring that IDD is eliminated.

Protecting consumers requires that a framework be established to ensure quality control of the production of iodized salt, as well as the distribution of adequately packaged and labeled iodized salt. The establishment of this framework is the main responsibility of the government. Ensuring a demand for the product and understanding the reason for insisting upon only iodized salt is a shared responsibility of the private sector and government. Establishment of iodization as the norm and ensuring customer demand will determine the success and sustainability of the programme. By adding a fixed does of iodine to salt at centralized location, the majority of the population would get adequate amounts of iodine. The mixing of salt is simple operation with no adverse chemical reactions. It will not impart any color, test or odor to the salt as shown in Fig 2.5. bellow. Cost of iodization is low too.[36]

**Figure 2.5.** Salt iodination using knapsack sprayers with manual mixing.

Salt iodations experimentation using knapsack sprayers and manual mixing led to improved methods that could reliably achieve the recommended level of 60-ppm iodine, using technologies and materials available on site.

USI, which ensures that all salt for human and animal consumption is adequately iodized, has been remarkably successful in many countries. Over 30 countries have achieved the goal of USI (>90% of household using iodized salt), and many others are on track. Most countries that have failed to achieve coverage over 20% have conflict situations that hinder all health efforts.

The quality control of salt iodization through testing is critical to the over all success of any iodine deficiency disorders (IDD) elimination program. Rapid spot tests are highly sensitive tests that can be performed rapidly to detect levels of iodine in salt and play an important role in salt monitoring programs.[37] Spot tests are technically simple, rapid check methods for detecting salt iodine, and can be readily performed even outside the laboratory. The tests can be classified in to two main categories:-

**Qualitative tests:** These indicate only the presence or absence of iodine over a broad range.
E.g. A positive test result may simply indicate a salt sample with an iodine of content some where between 5 to 100 parts per million [ppm].

**Semi-quantitative tests:** These give an appropriate concentration of the iodine content in salt. These tests generally use some form of color chart by which the iodine levels in a salt sample are estimated. E.g. <10ppm, 10-24ppm, 25-40ppm.

Various spot test methods use the same general reaction mechanism: a starch –based reagent solution, which produces a blue color when iodine is present in the salt sample.

Because spot tests are simple, rapid, and are easily applied in field setting, individuals with specific chemistry train can easily verify whether a salt sample has been iodized. Spot tests can be used at the production; distribution retail and house hold levels. They are particularly appropriate for small-scale salt producers. In many countries, salt producers, health workers, child development organizations, urban services organizations, community leaders, schoolchildren, teachers, and retailers use spot testing kits. They provide valuable information for the monitoring of salt quality as well as creating awareness and demand with in the community to consume only iodized salt.[38]

There is an internationally used test kit supplied by UNICEF. Some test kits will provide an additional neutralizing solution for use alkaline salt samples. In some cases the alkalinity of the salt will lead to a negative result even if iodine is actually present in the sample. Such kits recommend retesting a negative test result applying one drop of the neutralizing solution to salt, and then two drops of test solution. [39, 40]

Most of the countries are using potassium iodate as iodizing agent and some of the South American countries are using potassium iodide as iodizing agent. However, international agreement is made to use $KIO_3$ as iodizing agent to use UNICEF salt test kit as a common for testing the quality of salt or iodine content of salt. [41] In iodized salt some times alkalinity is brought in by the free flow agent that is added to salt. Some time even during normal occurrence, the salt contains sodium carbonate. The test solution does not readily answer the presence of iodine in this type of salts. In such cases, a recheck solution is to be added before trying the test solution.

Titration, or an equivalent method, is preferred for accurate testing of salt batches produced in factories or upon their arrival in a country, and in Household salt samples for analysis. This method is recommended for determining the concentration of iodine in salt at various levels of the distribution system where such accurate testing is required, and for testing when there are legal enforcement issues. Once the method is established, it is necessary to adhere to proper internal and external quality control measures.

### 2.5.2. Methods for supplementation of iodine to deficient population

**A. Iodine tablet**: tablet containing 100-500 mg of $KIO_3$ are available in the market for daily consumption. Single oral doses of potassium iodate monthly (30 mg) can provide adequate iodine for school aged children. The series of studies carried out from 1891 to 1915 proved that after taking sodium iodide tablets for one year, goiter among school children regressed to about 75%.

Supervision of daily intake is difficult and there are no ways of ensuring that the affected persons consume the tablets regularly. Oral administration needs direct target contact. But the bolus can not be stored in the body.[42]

**B. Iodized water:** water is a dietary necessity and must be consumed daily. Potassium iodate is added to the water vessel in which water is stored for drinking. The adverse effect is that this water may not be acceptable for drinking due to its bad smell and test [Fish, et al , 1993].

**C. Iodized capsules (tablet oil):** Iodized oil supplement can be also used to prevent IDD**.** Iodized oil is prepared by esterification of the unsaturated fatty acids in seed or vegetable oils and addition of iodine to the double bonds. It can be given orally or by injection. In rare instances, it may happen that salt iodization efforts are unable to meet the requirement of women during pregnancy, exposing the progeny to potential developmental risks. In such situations, while efforts to improve the salt iodization program continue, iodine supplementation may be considered for both pregnant women and children less than two years of age as a daily oral dose of iodine or a single oral dose of iodized oil every six to 12 months. [43]

**D. Iodized salt:** In 1922, iodized salt was introduced. Salt has been the most commonly accepted way of supplying iodine to the society. The reasons are that salt is universally consumed by all sections of the community irrespective of the economic level and that they consumed some level through out the year. WHO/UNICEF/ICCIDD recommends that iodine is added at a level of 20-40 mg iodine /Kg salt, depending on local salt intake. Iodine can be added to salt in the form of KI in or potassium idate ($kIO_3$). By adding a fixed does of iodine to salt at centralized location, the majority of the population would get adequate amounts of iodine. The mixing of salt is simple operation with no adverse chemical reactions. It will not impart any color, test or odor to the salt.[44]

# 3. STATEMENT OF THE PROBLEM

## 3.1. Project Aims and Goals

Iodine deficiency disorder is a common public health problem in Ethiopia especially in Beneshangul Gumuz. In the 2005 EDHS it has been shown that only 13.6% households in the region consumed salt that is adequately iodized. In another recent national survey (Cherinet Abuye and Yemane Berhane, 2007), goiter prevalence in Beneshangul was 37.3 %, an indication of severe iodine deficiency in the region. Despite some efforts by the government to eliminate IDD through universal salt iodization and public health education, little current data exists in iodine nutrition status in Beneshangul Gumuz, specially at wombera woreda. The main aim of this study is to provide current and reliable data on iodine nutrition status of schoolchildren in Amuma district, Beneshaangul Gumuz, Ethiopia. The study aims at establishing base / line data for planning, monitoring and evaluating of intervention programs by the policy makers.

## 3.2. General objective

- Assessing and quantify, the prevalence and frequency of iodine deficiency and endemic goiter in Metekel zone of wombera wereda at Amuma district.

## 3.3. Specific objectives

The specific objectives of this study include:

- To quantify the state of iodine deficiency in school children in Amuma district aged between 6 and 18 by using WHO/UNICEF/ICCIDD recommended method of goiter classification by palpation.
- To determine the use of iodized salt by school children in Amuma district aged between 6 - 18 by determining the level of iodine in salt used in their households using a UNICEF measuring kit for iodine in a salt and iodometric titration.
- To study the state of iodine deficiency in school children in Amuma district aged between 6 and 18 by studying the availability, price, use and storage of iodated salts in the district.
- To create a base line data for planning, implementing monitoring and evaluation of intervention health programs with regard to IDD.

# 4. EXPERIMENTAL METHODS

## 4.1. Study Area and Design

Across sectional study, which focuses on goiter and related variables, are conducted among schoolchildren in Ammuma Keble, Wombera, Benishangul Gumuz, Ethiopia. Amuma Kebele is one of the most thickly populated districts in northern part of the wombera woreda, found approximately 20 km to north of the wereda`s town. Most of the population in the kebele is farmers living in rural areas. Based on the figures from the (WSE) in 1999 this woreda has an estimated total population of 50,225 whom 24,470 are men and 25,755 are women , 4179 or 7.43% of population are urban dwellers. Among all the kebeles in this woreda, Amuma kebele is one of the thickly populated area in which its population size is estimated to be 6,785. These populations are mostly farmers. Farming and cattle breeding are predominant occupation in the Kebele. Sorghum, teff, coffee, wheat and mango are among the majors' agricultural products. The woreda has three types of agro climatic zones Dega(10%), winadega(15%) and kolla (75%). from these climatic conditions, Amuma keble is found in winadega part. A summary of the study area where shown in Figure 4.1.

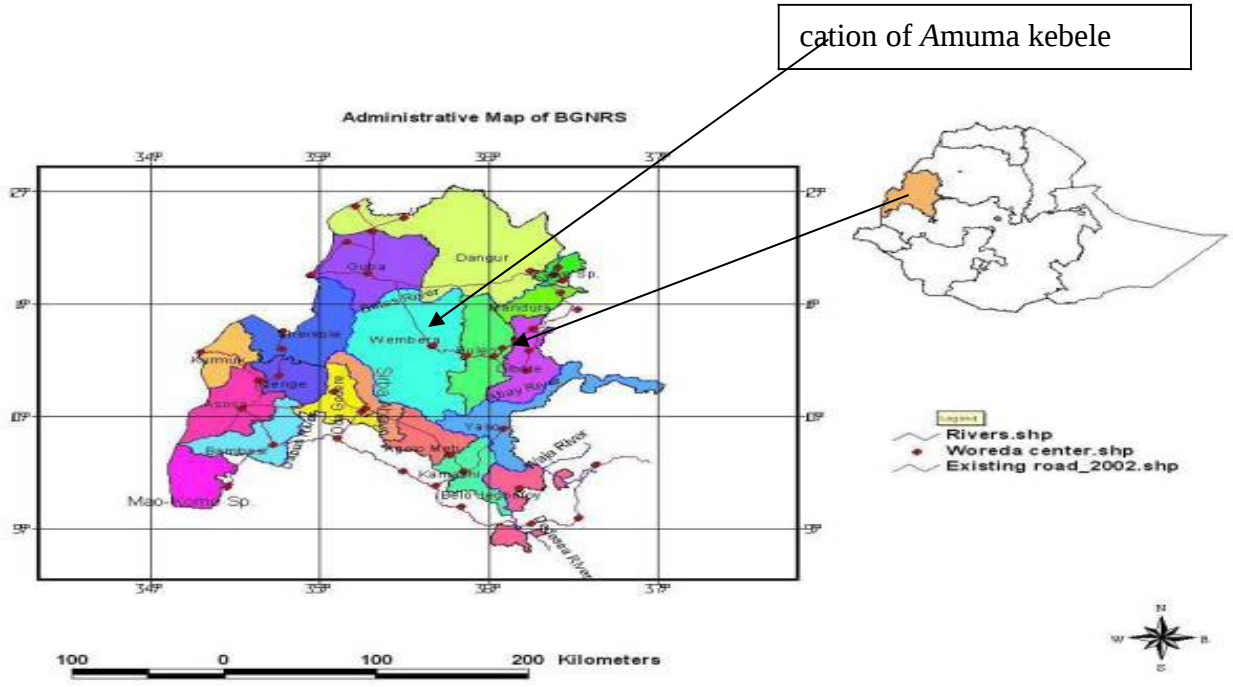



**Figure 4.1.** Map of study area

## 4.2. Sample preparation and shipment for chemistry tests

As per recommendation of WHO, the school children from both sexes were selected for the study.[44, 45] School children in the Kebele are selected as a target for the population in Amuma. The iodine nutritional status of school children represents the state of iodine deficiency in the general population. The clinical goiter surveys and evaluation of iodine content in edible salt samples are used as indicators to evaluate the iodine nutritional status in the area.

A cross-sectional community based descriptive study among two hundred School aged children of age 6 to 18 years in Amuma District in Beneshangul Gumuz, Ethiopia are studied during February to May 2011. Two hundred school aged children of ages 6 to 18 undergo clinical assessment for goiter grading and salt samples brought from their households are studied for iodine nutrition status.

A multistage “Proportional to population Size” (PPS) sampling method was used to determine the number of school aged children to be included in the study. The school-based PPS cluster sampling method is recommended as the most efficient and practical approach for performing an iodine status or an IDD prevalence survey. [46, 47] This method has been in use for many years for the evaluation of immunization coverage, and can be applied to many other health indicators. The principal requirement for applying the PPS method is that a listing (sampling frame) is available populations. For this survey, the sampling units are the schoolchildren.

**Sample size**

To determine the sample size of this study 95% confidence interval was used. A systematic random sampling technique using roster as a sampling frame was used to select school children. Sample size was determined by the following formula developed by Bland (1989):

**Steps:**-

1. Guessing / anticipate the proportion you are about to measure.
2. Deciding an acceptable margin of error for your estimate. Usually 5% = 0.05 for large population and 10% = 0.1 for small population.
3. Choosing approximate design effect. Usually 2 for small population and 10 for large population.

4. Calculate the sample size using the formula:-

$$Sample\ size = \frac{4xpx(1-p)xD}{M^2}$$

Where: - P= proportion of population to be measured

D= confidence level (95%).

M= margin error (standard value of 0.01)

Then using this formula to determine sample size out of 750 total school children:-

Let the proportion to measure is 360 children, then:

p= 48% = 0.48 M = 0.01

D= 2 for small population

Sample size = $\frac{4x0.48x\ (1\text{-}0.48)\ x2}{(0.1)^2}$ = 200

So two hundred schoolchildren aged between 6 and 18 in Amuma district were selected from a total of 750 school children from the school's database by PPS method.

Then, the following method was used to select the students from the school data base:

**Step one:** There are 750 students, then N = 750.

**Step two:** The number of students to sample is 200; then n = 200.

**Step three**: The sampling interval (k) is 750 / 200 = 3.75; then, rounding to the nearest integer, k = 4.

**Step four**: Using a random number table, a number from 1 to (and including) 4 were selected. In this case, suppose the number selected had been 3. Accordingly, the first student to be selected would be the third on the list.

**Step five:** Select every four thereafter; the selected students would be the 3rd,7th,11th, 15th,19th,etc

Using the same technique for salt sample out of 200 children 50 students are required.

**Steps:-**

1. N= 200
2. n = 50
3. K= 200/50 = 4
4. Suppose the number selected between 1 and 4 would be 2

5**.** Select every four thereafter; the selected students would be the 2nd, 6th, 10th,14th, etc.

Then a total of 50 students were selected. These selected students collect salt samples for determination of iodine content using salt iodine test kit and iodometric titration. Advance notice was given so that the children selected would bring salt to school on the day in question. Household salt samples from the students' house were collected and carefully packed in a screw-cap plastic for laboratory analysis.

## 4.3. Measurements and Data collecting schemes

Determining Goiter rate and iodine content in salt samples are the main variables to be accounted in this study. Goiter prevalence is tested using palpation method and iodine in salt is measured using salt testing kit-UNICEF and iodometric titration.

### 4.3.1. Measurement of thyroid size by palpation method

#### Clinical goiter survey for the determination of goiter prevalence

Goiter prevalence can be determined by using WHO/UNICEF/ICCIDD classification scheme using the physical examination of the thyroid gland. The person performing the palpation is health personnel who has 13 years of working experience and had an experience of doing palpation in another area in the region. A doctor how to do the clinical evaluations of the participants also supervises him.

The WHO/UNICEF/ICCIDD classification scheme is as follows (ICCIDD 2001):

**Grade 0**: None or no goiter (palpable or visible)

**Grade 1 or Palpable**: A goiter that is palpable but not visible when the neck is in the normal Position.

**Grade 2 or Palpable and Visible**

Goiter is palpable and visible in the normal position of the neck Grade 2 or perceptible from distance. A swelling in the neck that is clearly visible when the neck is in normal position and is Consistent with an enlarged thyroid when the neck is palpated.

**Total Goiter Rate**: Sum of goiter grades 1 and 2

**Technique**

The examiner faces the subject and looks for visible thyroid enlargement. The subject then looks up, extending the neck and making any thyroid enlargement more visible. The examiner palpates the thyroid by sliding his thumb along each side of trachea between the thyroid cartilage and the top of sternum and the size and consistency of the thyroid is carefully noted. The thyroid moves upwards when the subject swallows (Report from WHO November, 1992).

### 4.3.2. Measurement of iodine content in salt intake

There are a no/ of methods for testing the iodine content of salt, ranging from qualitative spot test by rapid salt testing kit to the more quantitative methods such as iodometric titration performed in laboratories for validation purpose.

**A. Determining iodine content of salt samples using international rapid salt testing kits supplied by UNICEF**

Iodine content of at least 50 salt samples were collected at random from a locality provides a valid estimate of the iodine content of the salt samples of the locality.[48] To monitor the iodine content of salt samples available in the area 50 tight plastic containers were distributed at random to the students and they were asked to bring samples of edible salt from their homes in the next day. The salt samples were kept at room temperature in the laboratory and iodine content was measured using internationally used rapid salt testing kits supplied by UNICEF.

**Rapid salt testing kits**

Improved iodized salt field test kit for salt fortified with potassium iodate.
Ranges: - 0 ppm, less than 15 ppm, more than 15 ppm.
The test kit contains the following reagents:-

- Two test solution ampoules of 10 ml.
- One recheck solution ampoule of 10 ml.
- One chart and one white cup.
- A detailed instruction sheet in the local language
- All the contents are packed in a small kit box that can fit into a shirt pocket.

**Chemicals (reagents) used**

Solution A: 0.5% weight/volume (w/v) starch solution, made by boiling 0.5 g soluble starch (or rice starch) in 100 ml deionized water.

Alternate

Solution A: Mix 10 g wheat starch with 15 g $H_2O$ and 90 g glycerol, warm to 90 $^oC$ in a Water bath until mixture becomes uniformly translucent.

Solution B: 1 %( w/v) sodium nitrite (0.25 g in 25 ml $H_2O$).

Solution C: 20% volume/volume (v/v) $H_2SO_4$ solution (2 ml + 8 ml $H_2O$).

Solution D: 12 %( w/v) potassium iodide (3 g in 25 ml $H_2O$).

Solution E: 5 N hydrochloric acid solution, made by mixing 10 ml concentrated HCl (12 N).

**Procedures**

1. Fill small cup with salt, then spread the salt surface flat.
2. Two drops of the test solution were added on the surface of the salt by piercing the white ampoule with a pin and gently squeezing the ampoule.
3. The solution will turn from light to dark violate depending on the iodine content.
4. Comparing the color on the salt with the color chart, the iodine content can be determine with in one minute.
5. If no color appears on the salt (after one minute), on a fresh sample 5 drops of the recheck solution in red ampoule were added and then 2 drops of test solution on the same spot add were added.
6. Now compare the color with the color chart and determine the iodine content.

**Note:**

1. The solutions should be shaked well before use.
2. The kit can be used for coarse salt also.
3. For precise results an analytical check is recommended
4. Shelf life is 18 months.
5. For reliable results do not use an open ampoule beyond 180 days.
6. Note for oral consumption.
7. Store in a dry cool place.
8. Addition of more than a few drops of test solution will not alter the test result, simply wastes the solution.

**B. Titration methods for salt iodine analysis**

**Sampling of salt**

The iodine content of salt sample is measured using the iodometric titration method.[49] The method consists of preparing of the reagents. For community or population surveys, 10 g salt samples are sufficient. Prior to taking a 10 g salt sample for analysis, salt should be thoroughly mixed to ensure that the iodine is homogeneously distributed in the salt.

**Reagent preparation**

Water required for the titration must be boiled, distilled water, which requires provision of a distillation unit. As a simpler alternative, regular tap water treated with a mixed bed deionizing resin can be used, thus avoiding the need for an expensive distillation unit.

• **0.005 M Sodium thiosulfate ($Na_2S_2O_3$):-** is prepared by dissolving 1.24 g $Na_2S_2O_3.5H_2O$ in 1000 mL water. Stored in a cool, dark place. This volume is sufficient for 100-200 samples, depending on the iodine content of samples.

• **2 N Sulfuric acid ($H_2SO_4$):-** Slowly 6 mL concentrated $H_2SO_4$ was added to 90 mL water. Make to l00 mL with water. This volume is sufficient for 100 samples. The solution is stable indefinitely.

• **10% Potassium iodide (KI):** prepared by dissolving l00 g KI in 1000 mL water and Stored in a cool, dark place. This volume is sufficient for 200 samples.

• **Starch indicator solution:** Make l00 mL of a saturated NaCl solution, by adding NaCl to approximately 80 mL water in a beaker, with stirring and/or heating, until no further solid will dissolve. Weighing 1g soluble starch into l00 mL beaker, and l0 mL water was added and, heated to dissolve. Saturated NaCl solution was added to the hot starch solution to make up to l00 mL Stored in a cool, dark place. This volume is sufficient for 50 samples.

**Procedural Steps**

**Step 1.** l0 g of the salt sample was weight and placed into a 250 mL Erlenmeyer flask with a stopper.

**Step 2.** Approximately 30 mL water was added to dissolve salt sample through stirring.

**Step 3.** Water was added to make volume up to 50 mL.

**Step 4.** 1 mL 2 N $H_2SO_4$ was added.

**Step 5.** 5 mL 10% KI was added. The solution turns to yellow when iodine is present, but do not when iodine is absent.

**Step 6.** The flask with stopper was put in the dark (cupboard or drawer) for 10 minutes.

**Step 7.** The burette was rinsed and filled with 0.005 M $Na_2S_2O_3$, and adjust level to zero.

**Step 8.** The flask was removed from drawer, and some $Na_2S_2O_3$ was added from the titration burette until the solution turns pale yellow.

**Step 9.** Approximately 2 mL of starch indicator solution was added (the solution turn dark purple) and continuing titrating until the solution becomes pink, and finally colorless.

**Step 10. T**he level of thiosulfate in the burette is recorded and converted to parts per million (ppm) using the conversion table (Appendix B) .

**Description of Reaction**

**Reaction 1**: Liberation of free Iodine from salt

- Addition of $H_2SO_4$ liberates free iodine from the iodate in the salt sample.
- The Excess KI is added to help solubilise the free iodine, which is quite insoluble in pure water under normal conditions.

**Reaction 2:** Titration of free Iodine with thiosulfate.

- Free iodine is consumed by sodium thiosulfate in the titration step. The amount of thiosulfate used is proportional to the amount of free iodine liberated from the salt.
- Starch is added as an external (indirect) indicator of this reaction, and reacts with free iodine to produce a blue color.

When added towards the end of the titration (that is, when only a trace amount of free iodine is left) the loss of blue color, or endpoint, which occurs with further filtration, indicates that all remaining free iodine has been consumed by thiosulfate.

**Reaction steps:-**

1. $IO_3^-$ + $5I^-$ + $6H^+$ ⟶ $3I_2$ + $3H_2O$

   (From salt) (From KI) (From $H_2SO_4$)

2. $2Na_2S_2O_3$ + $I_2$ ⟶ $2NaI$ + $Na_2S_4O_6$

   (Sodium thiosulfate) (Sodium tetrathionate)

### 4.4 Statistical analysis

Excel programs and statistical software SPSS were used to carry out data analysis. Origin software was also used for data entry and validation.

## 4.5. Ethical approval

The protocols for considering surveys were presented and approved by the research ethical committee of the university. The ethical risks in this study might otherwise have been the breaching of confidentiality, such as disclosure of sample findings of the individual and disclosure of households iodine status. School children were informed through school teachers on the nature of the intended studies. So that the school children can inform their family to allow them to participate and bring salt samples. The consents of these children and their parents were obtained for the participation to the study.

# 5. RESULTS AND DISCUSSION

## 5.1 Evaluation of Goiter Grading

### Summery for clinical goiter survey results

A total of 200 school children in age 6-18 years were enrolled in the study, which were clinically examined for goiter. The total goiter prevalence rate was found to be 39.5% as shown in table 5.1 bellow.

**Table 5.1** Age specific goiter prevalence in the school children of Ammuma district and total number of students with their respective age group.

| **Age (year)** | **Total no/ of students exam.** | **Number of children with goiter** | | | | | |
|---|---|---|---|---|---|---|---|
| | | **Grade 1** | | **Grade 2** | | **Total (1+2)** | |
| | | No/ | % | No/ | % | No/ | % |
| 6 | 16 | 5 | 31.5 | 1 | 6.3 | 6 | 37.5 |
| 7 | 13 | 3 | 23 | 1 | 7.7 | 4 | 30.5 |
| 8 | 24 | 7 | 29.2 | 2 | 8.3 | 9 | 37.5 |
| 9 | 20 | 7 | 35 | 2 | 10 | 9 | 45 |
| 10 | 19 | 6 | 31.5 | 1 | 5.3 | 7 | 36.8 |
| 11 | 11 | 4 | 36.4 | -- | -- | 4 | 36.4 |
| 12 | 14 | 5 | 35.7 | 1 | 7.2 | 6 | 42.9 |
| 13 | 20 | 7 | 35 | 2 | 10 | 9 | 45 |
| 14 | 14 | 4 | 28.6 | 2 | 7.1 | 6 | 42.9 |
| 15 | 18 | 6 | 33.5 | 2 | 11 | 8 | 44.5 |
| 16 | 18 | 5 | 27.8 | -- | -- | 5 | 27.8 |
| 17 | 9 | 3 | 33.3 | 1 | 5.5 | 4 | 44.4 |
| 18 | 5 | 1 | 20 | 1 | 11.1 | 2 | 40 |
| Total | 200 | 63 | 31.5 | 16 | 7.9 | 79 | 39.5 |

In all 200 students were screened out of which 16 were found to have goiter of grade 2, that means visible goiter and 63 were found to have goiter grade 1 which makes 79 of them have total goiter rate of 39.5% . The sex wise prevalence is as shown in table 5.2:-

**Table 5.2.** Age and sex-wise prevalence of goiter among school children

| Sex | samples | Grade 1 | | Grade 2 | | Total (1+2) | |
|---|---|---|---|---|---|---|---|
| | | No/ | % | No/ | % | No/ | % |
| Boys | 112 | 28 | 14 | 7 | 3.4 | 36 | 18 |
| Girls | 88 | 34 | 17 | 9 | 4.5 | 43 | 21.5 |
| Total | 200 | 63 | 31.5 | 16 | 7.9 | 79 | 39.5 |

In girls the prevalence was 21.5% percent which is more than that among boys 18 % percent as shown in Fig. 5.1. The higher rate among girls may be due to physiological characteristic, during puberty and quantitative dietary deficiency.

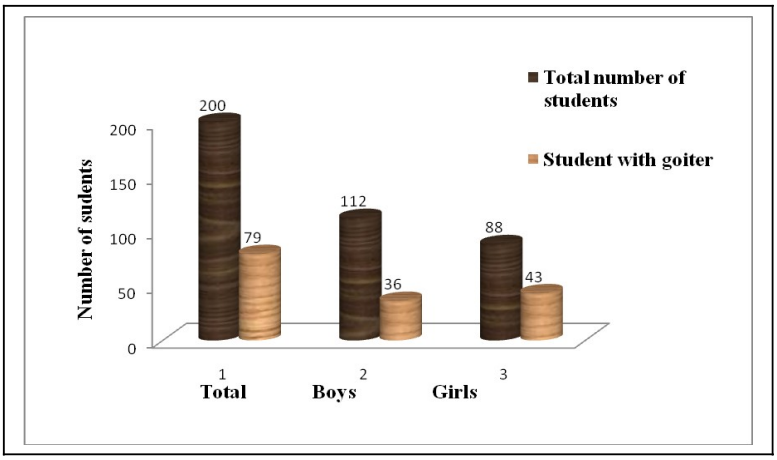



**Figure 5.1**. Sex wise prevalence of goiter

## 5.2. Evaluation of iodine content in salt intake

According to the recommendation of ICCIDD,(1999) for salt analysis for iodine quality monitoring, 50 samples were collected by the children from their house hold and the iodine content were checked on the spot using the internationally used rapid salt test kit and iodometric titration method. Most of the samples of crystal salt contain no any quantity of detectable iodine. Though it may be the quantity of iodine is negligible and can not be detected. The iodine content of the edible salts were measured and it was found that most of the salt samples tested had iodine level of 0-ppm (60%), and 30% of salt samples had iodine level less than 15ppm and only 10% had adequate iodine level which is $\geq$ 15 ppm. The iodine content of salt samples collected and analyzed is depicted in table 5.3.

**A. Determining iodine content of salt samples using international rapid salt testing kits Supplied by UNICEF**

Two spoons of salt samples consumed in their households from 50 students were collected and analyzed by using internationally recommended salt test kit supplied by UNICEF. The iodine content from each sample is indicated in appendix, Table 2A,3A.

**Table 5.3.** Results of salt sample analysis for iodine content using UNICEF test kit for salt consumed in the households of school children in Amuma district.

| Type | No/ of samples | 0-ppm | Less than 15ppm | ≥15ppm |
|---|---|---|---|---|
| crystal | 36 (72%) | 23 (46%) | 10 (20%) | 3 (6%) |
| Powdered | 14 (28%) | 7 (14%) | 5 (10%) | 2 (4%) |
| Total | 50 | 30 (60%) | 15 (30%) | 5 (10%) |

WHO/UNICEF/ICCIDD/ further recommends that 90% of the household salts should get iodized at the recommended level of 15ppm.[50] However, the study shows that about 90% of the households in Amuma are consuming salt which is at inadequate level. i.e. 60 % of the salt samples had iodine level of 0-ppm, 30% had less than 15ppm and only 10% had adequate iodine level ≥ 15ppm as shown in table 5.4.

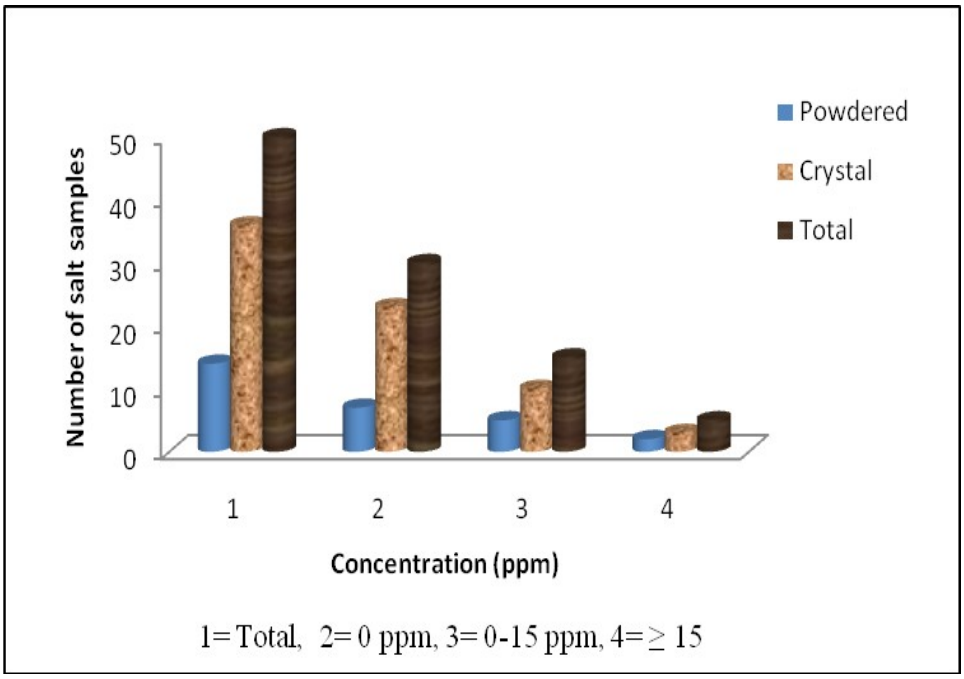



**Figure 5.2.** Number of salt samples verses concentration of Iodine in salt in ppm

**B. Titration methods for salt iodine analysis results**

The results of iodine content of the salt samples using the titration method varied from 0-ppm to 31.73- ppm of iodine (Table 5.4). The 15 salt samples showed iodine content which varied between 5.29 to 10.57 ppm and only 5 salt samples had iodine content of more than 15-ppm. While the rest of the samples did not show any iodine content (Table 5.4).

**Calculation**

***mg/kg(ppm) iodine = titration volume in ml x 21.15 x Normality of sodium thiosulfate x 1000 / salt sample weight in g.***

**Table 5.4.** Results of iodine content of salt samples by iodometric titration

| S.no/ | Burret reading | Iodine content in ppm | | S.no/ | Burret reading | Iodine content in ppm | |
|---|---|---|---|---|---|---|---|
| 1. | 0.0 | Nil | | 26. | 0.0 | Nil | |
| 2. | 0.0 | Nil | | 27. | 0.0 | Nil | |
| 3. | 2.0 | 21.15 | | 28. | 0.0 | Nil | |
| 4. | 0.0 | Nil | | 29. | 0.0 | Nil | |
| 5. | 0.0 | Nil | | 30. | 1.0 | 10.57 | |
| 6. | 1.0 | 10.57 | | 31. | 0.0 | Nil | |
| 7. | 0.0 | Nil | | 32. | 0.0 | Nil | |
| 8. | 0.0 | Nil | | 33. | 0.0 | Nil | |
| 9. | 1.0 | 10.57 | | 34. | 0.5 | 5.29 | |
| 10. | 1.0 | 10.57 | | 35. | 1.5 | 31.73 | |
| 11. | 0.0 | Nil | | 36. | 0.0 | Nil | |
| 12. | 0.0 | Nil | | 37. | 0.5 | 5.29 | |
| 13. | 1.0 | 10.57 | | 38. | 0.0 | Nil | |
| 14. | 0.0 | Nil | | 39. | 0.0 | Nil | |
| 15. | 3.0 | 31.73 | | 40. | 1.0 | 10.57 | |
| 16. | 0.0 | Nil | | 41. | 0.0 | Nil | |
| 17. | 0.0 | Nil | | 42. | 0.5 | 5.29 | |
| 18. | 1.8 | 19.04 | | 43. | 1.8 | 19.04 | |
| 19. | 0.8 | 8.46 | | 44. | 0.0 | Nil | |
| 20. | 0.0 | Nil | | 45. | 0.0 | Nil | |
| 21. | 0.5 | 5.29 | | 46. | 0.0 | Nil | |
| 22. | 1.0 | 10. 57 | | 47. | 0.0 | Nil | |
| 23. | 0.8 | 8.46 | | 48. | 0.0 | Nil | |
| 24 | 0.6 | 6.35 | | 49. | 0.0 | Nil | |
| 25 | 0.5 | 5.29 | | 50 | 0.0 | Nil | |

A total of 50 salt samples were analyzed using the standard idometric titration method (Table 5.4). The proportion of households consuming adequate iodized salt (salt with iodine level of at least 15 parts per million- 15 milligrams of iodine kilogram of common salt) was only 10%. Overall high proportion of School children (90 %) consumed salt having inadequate iodine content. The school children were distributed in to three categories, Normal (≥ 15 ppm), Moderate (less than15 ppm) and Severe (0- ppm). Overall 60 percent school children were in the severe category i.e. almost consuming no iodine through edible salt.

Similar to that of results obtained from salt test kit, the iodometric titration result shows that most of the salt samples had no iodine content. As shown in Table 12, 60 % of the salt samples had no iodine content (nil) , 30 % had iodine content between 5.29 ppm to 10.57 ppm and only 10% had adequate iodine level ( ≥ 15ppm).

## 5.3. Discussion

The most widely accepted marker to evaluate the severity of IDD in a region is the prevalence of endemic goiter in school children. It has been recommended that if more than 5 percent of school aged children (6-18 years) are suffering from goiter, the area should be classified as endemic for iodine deficiency. On the basis of its prevalence, WHO/UNICEF/ICCIDD recommended the criteria to understand the severity of IDD as a public health problem in a region.

The majority of the children with goiter belong to the age group 12—15 and 70% percent of the goiter cases are seen among them. The goiter prevalence is increasing as the age increases except in 16 year age group. This finding is in conformity with the statement that goiter increases with age and reaches the maximum with adolescence [ Hetzel,B. 1997].

Goiter is prevalent at endemic level in the study area among school children and this was evidenced by total goiter rate of 39.5% (grade 1 = 31.5%, grade 2 = 7.9%), indicating that IDD is a sever public health problem in the district so that they are found at the red traffic light. In the present study 60 percent of the beneficiaries consumed salt with an iodine content of 0-ppm (nil) and 30 percent were consume salt with an iodine content of less than 15ppm . So a total of 90 percent consumed salt with an iodine content which was bellow the stipulated level.

From the iodometric titration result (Table 5.4) shows that in the crystalline salt samples, 63.8% had iodine level of 0ppm, 27.8 % of the samples had iodine level between less than 15ppm and only 8.3% had adequate iodine level, which is ≥ 15ppm. While in the powdered salt samples 50% had iodine level of 0ppm, 35.7% had less than 15ppm and only 14.3% had that recommended value. All these results suggest that most of the salt samples that the society uses were not adequately iodized.

So there is need to strengthen the awareness on the community to use only the packed iodized salt for monitoring iodine deficiency disorder. This finding also revealed that salt was treated as either inadequate quantity of iodine was added to it at the production level or there were losses of iodine at the different points of distribution. Loss of iodine might be lost by the following ways [50] :-

**Storage of salt:** - Depending upon packing, transportation and storage, 20 to 40 percent iodine may be lost from the salt.[50] Salt should not be stored in open space or in damp places. It must be shielded from moisture, sunlight and high temperature. It should be stored in airtight containers made of plastic, wood, glass or clay with well-fitting lid. The moisture content in the salt, humidity in the air, acidity of the salt and chemical form of iodine are important factors limiting the stability of iodine (bulletin, June 1996).In the study area the societies do not understand these limitations. Even in wet seasons, people put their salt with its open containers on ceiling just above the fire place to protect it from losing their salt by moisture or dissolution. This process could cause iodated salt to loose its iodine due to exposure to high temperature.

**Adding salt while cooking:** - Iodized salt should be added to the food substances after cooking to reduce the lose of iodine. Addition of salt before cooking results in loss of iodine and hastens the loss of the other nutrients. Losses in cooking and extent of absorption are other factors which determine the ultimate availability of iodine to the body. Washing salt before use in order to remove impurities would remove all the iodine. It is seen that the majority of households in the study area follow the practice of adding salt before cooking.

In the study areas crystal salt was available in unpacked form with some impurities and attempts to remove the impurities by washing salt before use could remove the iodine content of the salt. Powdered iodized salt are sold currently in very little in packets.

## 6. CONCLUSION AND RECOMMENDATIONS

The present study showed sever goiter prevalence in primary school children in Amuma district of Wombera woreda due to inadequate iodine intake or content from salt at household level. It was evident from the findings of this study that biochemical indicators as measurement by goiter grading and determination of salt iodine content are more reliable in measuring the actual situation of iodine intake. The study demonstrate that Amuma population are in the endemic goiter prevalent range and the society do not use adequately iodized salt, so there is need to improve awareness among society to use iodized salt. This will have prevented thousands of children from mental retardation.

This study show current and up-to-date information on the IDD prevalence in the study area and recommended strategies in controlling and eliminating the deficiency of iodine in the study area in particular and in Ethiopia in general. Universal salt iodization, with time supplements and fortification of food and water using iodine are some strategies recommended by WHO/UNICEF/ICCIDD joint consultation. The government of Ethiopia, however still need to strengthen its efforts to control and eliminate IDD in populations. The existing provision, controlling and monitoring systems for quality iodized salt distribution is not enough to guarantee adequate iodine to healthy population. This study showed that a good proportion of the study population are consuming salt with out iodine fortification (90 percent), due to the fact that the communities have no awareness about the importance of iodized salt and the edible salts available in the area are still not iodized.

The suppliers are not complying with the standards and specifications which make the addition of iodine to the salt as mandatory. The successful application of universal salt iodization in combating IDD in a country requires adequate supplies and regular monitoring at the production and consumption level. Universal iodization of Salt has to go on hand in hand with laboratory development and research, capacity building, advocacy and public education on IDD.

Commitment by health professionals and scientists to the assessment and reassessment of the progress in iodine nutrition status by measuring iodine in salt and urine in school aged children, women and infants, and periodic surveys and analysis of their data are very important. Surveillance of the iodine nutrition status including urine iodine determination and monitoring the quality of salt is needed to provide the basis for continuing iodine deficiency disorder elimination work. Results from such efforts are vital for policy and program management decision and continuing and renewing commitment[49,50]. With the problem clear, the impact understood, and the solution affordable and sustainable we should not allow even a single child entering our world and growing up without the iodine protection against brain damage. If we should fail to attain the goal of sustained IDD elimination, what prospects does we have in tackling the more development tasks?

Iodized salt utilization need to be universal through out the country. Efforts to educate the society to use iodized salt and monitoring the sales of iodated salt needs to be stepped up so that every citizen can benefit from this cost-effective and smarter health intervention.

Based on the above facts the following recommendations can be drown:-

1. More attention should be given in the future to use iodized salt in monitoring IDD.
2. A comprehensive monitoring plan should be implemented to aware all communities to use only the packed iodized salt.
3. Development of program to improve knowledge, attitude and practice in using iodated salt.
4. The fortification of iodine to the other food products which used salt as main ingredient in processing or for preservation.
5. To ensure availability of a minimum of 150 μg of iodine (15ppm) at consumption level, a network of Iodine Monitoring Laboratories should be established, which would carry analysis of salt samples at regular intervals.

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# Appendices A

## Terminology

**Iodine deficiency disorders (IDD):- r**efer to all of the consequences of iodine deficiency in a population that can be prevented by ensuring that the population has an adequate intake of iodine.

**Endemic goiter**:-When the prevalence of goiter in a population rises above 5-10%, the problem is considered as.

**Table 1A :** Epidemiological criteria for assessing the severity of IDD based on the prevalence of goiter in school-aged children.

| Prevalence of goiter | Severity of IDD | Traffic light color |
|---|---|---|
| 0.0-4.9% | None | Green |
| 5.0-19.9% | Mild | Yellow |
| 20.0-29.9% | Moderate | Orange |
| ≥ 30% | Severe | Red |

**USI**: - involves the iodization of all human and livestock salt, including salt used in the food industry.

**Rapid spot tests**: - are highly sensitive tests that can be performed rapidly to detect levels of iodine in salt and play an important role in salt monitoring programs.

## ppm:- Calculations

***Mg/kg(ppm) iodine = titration volume in ml x 21.15 x Normality of sodium thiosulfate x 1000 / salt sample weight in g.***

**Determining iodine content of salt samples using international rapid salt testing kits**

**Table 2A**. Iodine content of salt samples consumed by school children in north of Amuma district.

| S. No/ | Type | Iodine content in ppm | | |
|---|---|---|---|---|
| | (powdered or crystal) | 0-ppm | Less than 15ppm | ≥15ppm |
| 1 | crystal | x | | |
| 2 | // | x | | |
| 3 | // | | | x |
| 4 | // | x | | |
| 5 | // | x | | |
| 6 | powdered | x | | |
| 7 | // | | x | |
| 8 | crystal | x | | |
| 9 | // | | x | |
| 10 | // | | x | |
| 11 | // | x | | |
| 12 | // | x | | |
| 13 | powdered | | x | |
| 14 | crystal | x | | |
| 15 | crystal | | | x |
| 16 | powdered | x | | |
| 17 | // | x | | |
| 18 | // | | | x |
| 19 | crystal | | x | |
| 20 | // | x | | |
| 21 | // | | x | |
| 22 | // | | x | |
| 23 | // | | x | |
| 24 | // | | x | |
| 25 | // | | x | |

**Table 3A**. Iodine content of salt samples consumed by school children in Minjo area of Amuma district.

| S. No/ | Type | Iodine in ppm | | |
|---|---|---|---|---|
| | (powdered or crystal) | 0-ppm | Less than 15 ppm | ≥15ppm |
| 26 | crystal | x | | |
| 27 | // | x | | |
| 28 | // | x | | |
| 29 | // | x | | |
| 30 | powdered | | *x* | |
| 31 | powdered | x | | |
| 32 | crystal | x | | |
| 33 | // | x | | |
| 34 | // | | x | |
| 35 | // | | | x |
| 36 | // | x | | |
| 37 | // | | x | |
| 38 | // | x | | |
| 39 | // | x | | |
| 40 | // | | x | |
| 41 | powdered | x | | |
| 42 | // | | x | |
| 43 | // | | | x |
| 44 | // | x | | |
| 45 | // | x | | |
| 46 | // | x | | |
| 47 | crystal | x | | |
| 48 | crystal | x | | |
| 49 | crystal | x | | |
| 50 | crystal | x | | |

**Appendices B**

**Conversion table: Iodine content in parts per million**

| **Burette reading** | **Parts per million (ppm)** |
|---|---|
| 0.0 | 0.0 |
| 0.1 | 1.1 |
| 0.2 | 2.1 |
| 0.3 | 3.2 |
| 0.4 | 4.2 |
| 0.5 | 5.3 |
| 0.6 | 6.3 |
| 0.7 | 7.4 |
| 0.8 | 8.5 |
| 0.9 | 9.5 |
| 1.0 | 10.6 |
| 1.1 | 11.6 |
| 1.2 | 12.7 |
| 1.3 | 13.8 |
| 1.4 | 14.8 |
| 1.5 | 15.9 |
| 1.6 | 16.9 |
| 1.7 | 18.0 |
| 1.8 | 19.0 |
| 1.9 | 20.1 |
| 2.0 | 21.2 |
| 2.1 | 22.2 |
| 2.2 | 23.3 |
| 2.3 | 24.3 |
| 2.4 | 25.4 |
| 2.5 | 26.5 |
| 2.6 | 27.5 |
| 2.7 | 28.6 |
| 2.8 | 29.6 |
| 2.9 | 30.7 |
| 3.0 | 31.7 |
| 3.1 | 32.8 |
| 3.2 | 33.9 |
| 3.3 | 34.9 |
| 3.4 | 36.0 |
| 3.5 | 37.0 |
| 3.6 | 38.1 |
| 3.7 | 39.1 |
| 3.8 | 40.2 |
| 3.9 | 41.3 |
| 4.0 | 42.3 |