Addis Ababa University

School of Graduate Studies

College of Natural Sciences

Center for Food Science and Nutrition

Iodine deficiency in School Aged Children 7-12 years and Associated Factors in Akaki-kality Subcity of Addis Ababa, Ethiopia

By:

Solomon Emiru

Advisors: Mr. Kelbessa Urga (Associate Professor)

Dr. Aweke Kebede

A thesis submitted to the School of Graduate Studies of Addis Ababa University in partial fulfillment of the requirements for the Degree of Master of Science in Food Science and Nutrition

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Approval by examining board:	**Signature**	**Date**
Dr. Cherinet Abuye (External examiner)	_____	_____
Dr. Dawd Gashu (Internal examiner)	_____	_____
Mr. Kelbessa Urga (Advisor)	_____	_____
Dr. Aweke Kebede (Advisor)	_____	_____
Mr. Aynadis Tamene (Chair man)	_____	_____

Declaration

I the undersigned, declare that this thesis is my original work and that all sources of materials used for the thesis have been duly acknowledged.

Name: Solomon Emiru

Signature: _____ Date _____

The thesis has been approved for submission by:

Name of Supervisors:	**Signature**	**Date**
Mr. Kelbessa Urga	_____	_____
Dr. Aweke Kebede	_____	_____

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List of abbreviations

AOAC	Association of Official Analytical Chemistry
AOR	Adjusted Odds Ratio
COR	Crude Odds Ratio
EDHS	Ethiopian Demographic Health Survey
EPHI	Ethiopian Public Health Institute
EFSA	European Food Safety Authority
ICCIDD	International Council for the Control of Iodine Deficiency Disorder
ICP-MS	Inductively Coupled Plasma Mass Spectroscopy
IDD	Iodine deficiency Disorder
IQ	Intellectual Quotient
MUI	Median Urinary Iodine
ppm	parts per million
SAC	School Age Children
SPSS	Statistical Package for Social Science
T_3	Triiodothyronine
T_4	Thyroxine
TGR	Total Goiter Rate
TPO	Thyroid Peroxidase
TRH	Thyrotropin Releasing Hormone
TSH	Thyroid Stimulating Hormone
UIC	Urinary Iodine Concentration
UIE	Urinary Iodine Excretion
UNICEF	United Nations Children's Fund
USI	Universal Salt Iodization
WHO	World Health Organization

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Abstract

Iodine is an essential micronutrient required for normal human growth and development as it is needed for the synthesis of thyroid hormones produced by thyroid glands. Iodine deficiency is a severe public health problem in Ethiopia. The aim of this study was to assess prevalence of goiter, urinary iodine status and to determine iodine content of salt consumed by school age children 7-12 years. A school based cross-sectional study was conducted in five randomly selected primary schools in Akaki-kality subcity of Addis Ababa during April to July 2015. A total of 270 children were included in the study. Questionnaire was used to collect information of socio-demographic, knowledge on iodine deficiency and practices of iodized salt consumption. Clinical examination of thyroid gland was assessed by standard palpation method. Spot urine samples were collected and analyzed by Sandell-Kolthoff reaction. Iodine content of salt samples was determined by iodometric titration method. Descriptive statistics, bivariate and multivariate logistic regression analysis were carried out. The overall prevalence of goiter was 23.3% with (Grade 1=22.2% and Grade 2=1.1%). Prevalence of goiter in females and males was 27.4% and 19.3% respectively. As the age increased the goiter prevalence also increased in age group 10-12 years (AOR=2.6; 95% CI=1.4, 4.8; p=0.003) and school where children learned (AOR=3.8; 95%CI=1.4, 10.1; p=0.009) were factors highly associated with goiter. The median urinary iodine level of school age children was 85.7 µg/L, Interquartile Range was 56.3 – 127.4 µg/L. The UIC was below 100µg/L in 62% of the children and 20% had UIC below 50µg/L. Inadequate iodine status of children was highly associated with age 7-9 years (AOR=2.2; 95%CI=1.1, 4.3; p=0.028), educational status of family (AOR=4.1; 95%CI=1.4, 11.8; p=0.009) and children's consumed coarse salt (AOR=308.4; 95%CI=39.2, 2429.2; p=0.000). Only 20% of the total salt samples were adequately iodized. The finding of this study revealed that iodine deficiency is a moderate public health problem and iodized salt consumption is very low in the study area. Therefore, further strengthen the existing monitoring system for the quality of iodized salt and awareness creation activities on the benefits of iodine nutrition have to be intensified.

Keywords*:* Iodine deficiency, School age children, Urinary iodine excretion, Goiter, Iodized salt

Chapter one

1. Introduction

1.1. Background of the study

Iodine is a trace element essential for the synthesis of thyroid hormones. These hormones regulate the metabolic pattern of most cells and play a vital role in the process of early growth and development of most organs, especially the brain (Kapil, 2007). It is very volatile and sublimates easily, it passes from a solid state directly to gas form (I_2). Source of iodine on a daily basis is obtaining from drinking water and foods originating from plants grown on iodine rich soil (Kunle and Olanrewaju, 2014).

A lack of iodine in the diet leads to an inadequate secretion of thyroid hormone (hypothyroidism), which in turn causes a number of symptoms collectively known as Iodine Deficiency Disorder (IDD). Iodine deficiency can cause goiter, cretinism, mental retardation, brain damage, reduced intelligence, deaf-mutism, in pregnant woman miscarriage and stillbirth (Kunle and Olanrewaju, 2014).

The global burdens of iodine deficiency is estimated to be more than 1.9 billion populations are estimated to have inadequate iodine nutrition. Of these, 285 million are school age children (SAC) (Boonstra and Jaiswal, 2010). In Ethiopia IDD affects million of peoples, nationwide study conducted in 2005 indicated that the prevalence of goiter in SAC (6-12 years) was 39.9% and Median Urinary Iodine (MUI) level was 24.5 μg /L in Ethiopia (Abuye *et al*., 2007). According to Ethiopian Demographic and Health Survey (EDHS, 2011) the household consumption of adequately iodized salt in Ethiopia was 15.6%.

To maintain a sufficient iodine status, the World Health Organization (WHO), the United Nations Children's Fund (UNICEF) and the International Council for Control of Iodine Deficiency Disorders (ICCIDD) recommend that the daily iodine intake should be 90 μg for pre-school children (0 to 59 months), 120 μg for children of 6 to 12 years and 150 μg for adults (above 12 years). The recommendation for pregnant and lactating women has recently been increased to 250 μg per day (Jooste and Zimmermann, 2008).

Methods recommended for assessment of iodine nutrition are the Urinary Iodine Concentration (UIC), thyroid size and thyroid function tests including thyroglobulin. These indicators are complementary, in that urinary iodine is a sensitive indicator of recent iodine intake (days), thyroglobulin and other thyroid function tests shows an intermediate response (weeks to month), while changes in the goiter rate reflect long term iodine nutrition (months to years) (Zimmermann and Crill, 2010).

Since 2001, the indicator for measuring the prevalence of iodine deficiency has changed from goiter prevalence to MUI concentration. Because of dietary iodine is reduced to iodide and is rapidly and almost completely absorbed greater than 90% in the gut. Ultimately more than 90% of iodine is excreted in the urine, making UIC a good indicator of iodine status and consumption (Jooste and Zimmermann, 2008).

Common sources of dietary iodine include seafood, dairy products and food additives such as calcium iodate and potassium iodate (Haldimann *et al*., 2005). Introduction of universal salt iodization (USI) as a global strategy to control iodine deficiency is approaching its 20th anniversary. Low iodine intake is the most common cause of preventable mental impairment worldwide, which is why there is a global drive to eliminate iodine deficiency through the highly effective strategies of salt iodization and iodine supplementation (Andersson *et al*., 2010).

1.2. Statement of the problem

Iodine deficiency is the single most common cause of preventable mental retardation and brain damage in the world. It also decreases child survival, impairs growth and development. Despite remarkable progress of iodine deficiency in previous years, the successes have been regionally variable with some regions showing little progress. Against the general global improvement in iodine status and marked improvement in four of the six WHO regions since 2003, the trend in Africa indicated a small decrease in the proportion of SAC with insufficient iodine intake by UIC below 100 µg/L (Jooste *et al*., 2013).

The regional burden of iodine deficiency in Africa compared to other regions is further emphasized by the finding that seven of the top 10 iodine-deficient countries with the greatest numbers of SAC with insufficient iodine intake in 2011 are from Africa. Among these countries, Ethiopia, Sudan, Algeria, Angola, Mozambique, Ghana, and Morocco were ranked by iodine deficiency in SAC respectively (Jooste *et al*., 2013).

Ethiopia is one of highly affected country by IDD. According to Abuye *et al.* (2007) the Total Goiter Rate (TGR) of SAC in Addis Ababa was 19.3%. Furthermore, the household consumption of adequately iodized salt in Addis Ababa was 29.6% (EDHS, 2011). Both of these finding clearly showed that IDD cause public health problem in Addis Ababa. There was no previous study in the recent years to assess the iodine status of children in Addis Ababa. Therefore, to identify the status of iodine deficiency in Addis Ababa from the ten subcities, Akaki-kality subcity was randomly selected as study area targeted on SAC to determination of urinary iodine status, assessing goiter status and household iodized salt consumption.

1.3. Significance of the study

The importance of this study is to show the current status of iodated salt utilization and iodine deficiency in SAC in the selected subcity. Recently there is no study done specifically in Addis Ababa on iodine status of school children and iodized salt consumption trend after the proclamation and implementation of national iodated salt distribution. This study will help us to identify the problem, evaluate the activities preformed and also gives baseline information for the next intervention plan for the Addis Ababa city Administration Health Bureau. Furthermore, for stakeholder working in this area it helps in order to evaluate the program and to identify opportunities and challenges on the implementation of this program. The study will provide clue for researchers on the consequent areas to be focused.

1.4. Objectives of the study

1.4.1. General objective:

- To determine urinary iodine status, goiter rate and iodine content of salt consumed by school children in selected schools of Akaki-kality Subcity of Addis Ababa.

1.4.2. Specific objectives:

- To determine urinary iodine status of school age children 7-12 years in Akaki-kality subcity of Addis Ababa.
- To assess goiter rate of school age children 7-12 years in Akaki-kality subcity of Addis Ababa.
- To determine iodine content of salt consumed in household of Akaki-kality subcity of Addis Ababa.
- To assess parents knowledge on iodine and iodine deficiency and their practices of iodized salt consumption in Akaki-kality subcity of Addis Ababa.

Chapter two

2. Literature review

2.1. Iodine

Iodine is a micronutrient of crucial importance for health and well-being of all individuals (Farhana and Shaiq, 2010). Iodine as (iodide) is widely but unevenly distributed in the Earth's environment. In many regions, leaching from glaciations, flooding, and erosion have depleted surface soils of iodide and most iodide is found in the oceans. Iodide ions in seawater are oxidized to elemental iodine, which volatilizes into the atmosphere and is returned to the soil by rain, completing the cycle. However, iodine cycles in many regions are slow and incomplete, leaving soils and drinking water iodine depleted (Zimmermann, 2013). Iodine is found in nature in various forms: inorganic sodium and potassium salts (iodide and iodate), inorganic diatomic iodine (molecular iodine or I_2), and organic monoatomic iodine (Patric, 2008).

Iodine is an essential dietary element which is required for the synthesis of the thyroid hormones T_4 and T_3. These hormones are iodinated molecules of the essential amino acid tyrosine regulate cellular oxidation and hence affect calorigenesis, thermoregulation, and intermediary metabolism. These hormones are necessary for protein synthesis. They also promote nitrogen retention, glycogenolysis, intestinal absorption of glucose and galactose, as well as lipolysis and the uptake of glucose by adipocytes (Kapil, 2007).

Most ingested iodine is reduced to iodide in the gut and absorbed almost completely in the duodenum. Iodide is cleared from the circulation mainly by the thyroid and kidney. The thyroid adjusts the amount of iodide uptake to amounts required for adequate thyroid hormone synthesis. In conditions of adequate iodine supply, no more than 10% of the absorbed iodine is taken up by the thyroid. In chronic iodine deficiency, this fraction can exceed 80%. Similarly, during lactation, the mammary gland regulates iodine uptake for secretion in breast milk. Several other tissues can also concentrate iodine, including the salivary glands, choroid plexus, and gastric mucosa, but these are minor pathways of uncertain significance. Once the thyroidal iodine requirement has been met, excess iodine is excreted by the kidney (Hess, 2013).

2.2. Iodine and thyroid metabolism

The healthy human body contains 15–20 mg of iodine, of which about 70–80% is present in the thyroid gland. In a day, 60 μg of circulating iodine needs to be trapped by the thyroid for the adequate supply of T_3 and T_4. To extract this amount of iodine from the circulation, the thyroid daily clears several hundred liters of plasma of its iodine. The thyroid gland traps and concentrates iodide and uses it in the synthesis and storage of thyroid hormones. Iodide is rapidly absorbed from the gastrointestinal tract and distributed to extracellular fluids. But the concentration of iodide in the extracellular fluid is usually low because of the rapid uptake by the thyroid gland and renal clearance (Kapil, 2007).

The synthesis of thyroid hormones requires two principal raw materials. First is the amino acid, tyrosine, provided by a large glycoprotein scaffold called thyroglobulin. A molecule of thyroglobulin contains 134 tyrosines, but only some of these are used to synthesize T_4 and T_3. The second raw material is iodine, or more accurately iodide (I^-). Iodine is absorbed rapidly as either Iodate (IO_3^-) or (I^-) in the stomach and upper small intestine (Bowen, 2003).

Thyroid gland plays a central role in the metabolism of iodine. The gland comprises multiple follicles lined by follicular cells resting on a basement membrane. The follicles are filled by a clear viscous material called colloid. The colloid is a glycoprotein called thyroglobulin. Iodine trapping is the first step in the metabolism of iodine. The process commence 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 symporter protein found in the basolateral membrane of the follicular cell: the energy required by this process is linked to ATPase dependent Na-K pump (Farhana and Shaiq, 2010).

Synthesis and secretion of thyroglobulin is the second step of iodine metabolism. It occurs by another independent process within the follicular cell; the synthesis starts on the rough endoplasmic reticulum as peptide units. Later these units combine into a dimer, followed by addition of carbohydrate moieties, after which the molecule moves to Golgi apparatus. The completed thyroglobulin molecule contains about 140 tyrosine residues, which serve as substrate for synthesis of thyroid hormone (Farhana and Shaiq, 2010).

The third step is the oxidation of iodide. The iodide within the follicular cell moves towards the apical surface of the plasma membrane, to enter into the follicular lumen; this transport by a sodium independent iodide/chloride transporter called pendrin. The iodide is then immediately oxidized to iodine. This followed by organification of thyroglobulin, wherein iodination of the tyrosine residue present within the thyroglobulin molecules occurs. Iodination first occurs at position 3 to form monoiodothyrosine and then at position 5 to form diiodithyrosine. Iodination of tyrosine is followed by coupling reaction, whereby, two molecules of diiodithyrosine couple to form T_4 hormone; and one molecule of monoiodothyrosine couples with one molecule of diiodithyrosine to form T_3 hormone. The reaction catalyzed by Thyroid Peroxidase (TPO). The thyroid hormones are stored inside the thyroid follicles as colloid for several months. The stored hormones can meet the body requirements for up to three months (Ahad and Ganie, 2010).

The processes of hormone synthesis and secretion are stimulated by thyroid stimulating hormone (TSH) is also called thyrotropin from the anterior pituitary gland. The secretion of TSH is modulated by Thyrotropin Releasing Hormone (TRH) from the hypothalamus, being the regulator of iodine metabolism in a feedback mechanism (Bowen, 2003). TSH which operates on feed-back mechanism turned to T_4 level in blood. A fall in T_4 level stimulates the pituitary to increase its TSH secretion which in turn stimulates the thyroid gland to release T_4 in circulation to maintain normal level of the hormone in the blood (Ahad and Ganie, 2010). The thyroid gland produces T_4 and T_3 hormones. These hormones regulate the rate of metabolism and affect physical and mental growth and the rate of function of many other systems in the body (Kapil, 2007).

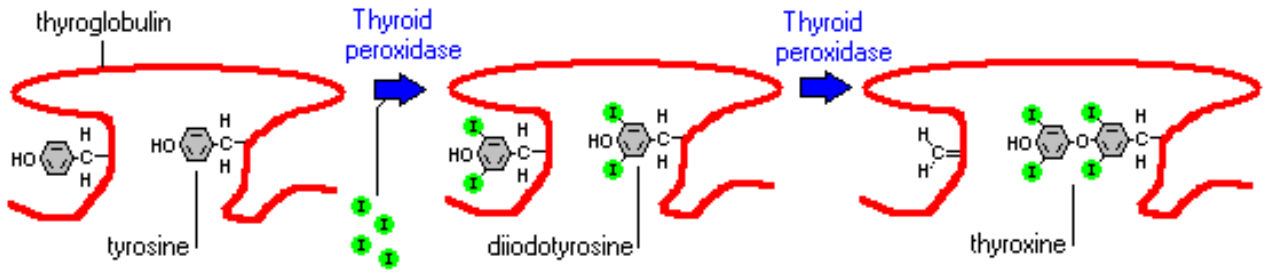


Figure 2.1: Synthesis of thyroxine (Bowen, 2003).

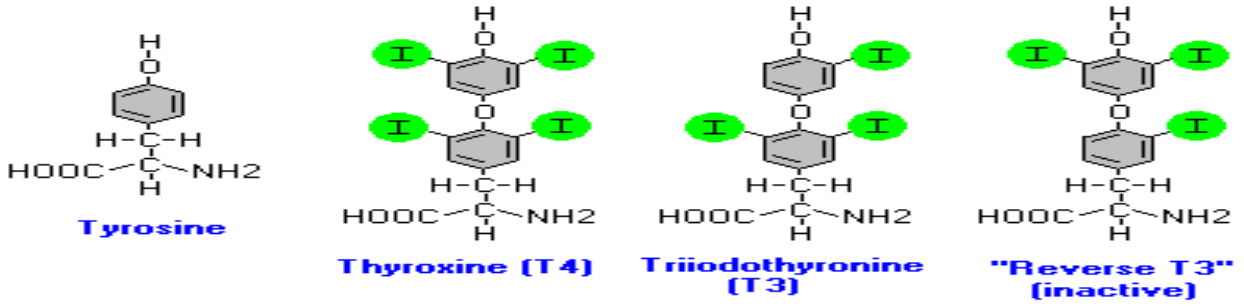


Figure 2.2: The chemical structure of tyrosine and the thyroid hormones (Bowen, 2003).

2.3. Iodine Deficiency

Iodine is a trace element essential for the synthesis of thyroid hormones. These hormones regulate the metabolic pattern of most cells and play a vital role in the process of early growth and development of most organs, especially the brain. In humans, the early development of the brain occurs during feotal and early postnatal life. Inadequate intake of iodine leads to insufficient production of these hormones, which adversely affect the muscle, heart, liver, kidney and the developing brain. This results in the disease states collectively known as Iodine Deficiency Disorders (Kapil, 2007).

Consequence of iodine deficiency includes thyroid function abnormalities, endemic goiter, mental retardation, cretinism, reproductive failure, and prenatal and infant mortality. The most damaging disorder resulting from iodine deficiency is irreversible mental retardation occurring during the critical period from the fetal stage until the third month after birth (Jooste and Zimmermann, 2008). Dietary iodine deficiency stimulates TSH secretion, which results in thyroid hypertrophy. The enlargement of the thyroid gland due to dietary iodine deficiency is called endemic goiter. Iodine intakes consistently less than 50 µg/day usually result in goiter. Severe and prolonged iodine deficiency may lead to a deficient supply of thyroid hormones. This condition is referred to as hypothyroidism (Kapil, 2007).

Due to salt iodization programs worldwide the extreme manifestations of iodine deficiency are becoming increasingly uncommon. However, of major public health concern is the continued iodine deficiency in many countries leading to subtle degrees of mental impairment, poor school performance, reduced intellectual ability and impaired work capacity. Two meta-analyses

reported a reduction of 12.5 to 13.5 Intellectual Quotient (IQ) points in iodine-deficient children. In South Africa, lower language exam marks were reported in goitrous children compared to non-goitrous children before the introduction of mandatory salt iodization. Cognitive impairment appears to be at least partially reversible as shown in a recent clinical trial in 10 to 12 year old moderately iodine deficient Albanian children who received 400 mg of iodine as oral iodized oil or placebo. Iodine treatment significantly improved information processing, fine motor skills, and visual problem solving compared to placebo in these children (Jooste and Zimmermann, 2008).

Ethiopian Health and Nutrition Institute undertook a systematic survey of 70 % of the country at randomly selected sites after stratification by region and altitude. Incidence of goiter ranged from 0.4% to 66.3%, with a mean of 25%. The prevalence of goiter was greatest in areas at altitudes greater than 2000 meter. Another study on IDD in Ethiopia revealed that 3487 women with goiter 16.7% had history of one or more reproductive failure in the form of miscarriage and or stillbirth while among 7515 women without goiter 13.8% reported to have reproductive failure. Weighted TGR in 15 to 49 years-old women were 35.8% goiter (24.3% palpable and 11.5% visible goiter). This indicates that 6 million women in this age category were affected by goiter in Ethiopia (Abuye and Berhane, 2007). The national total goiter weighted prevalence rate among children aged 6 to 12 years was 39.9%, representing more than 4 million children (Abuye *et al*., 2007).

Table 2.1: Iodine deficiency disorder by age group (Zimmermann and Boelaert, 2015)

Age group	Health consequences of iodine deficiency
All ages	Goiter Increase susceptibility of the thyroid gland to nuclear radiation In severe iodine deficiency, hypothyroidism
Fetus	Abortion, stillbirth, congenital anomalies and perinatal mortality
Neonate	Infant mortality, Endemic cretinism
Child and adolescent	Impaired mental function, Delayed physical development
Adults	Impaired mental function, reduced work productivity, Toxic nodular goiter, hyperthyroidism

2.4. Etiology of iodine deficiency

Iodine is widely but unevenly distributed in the earth's environment. In many regions, leaching from glaciations, flooding, and erosion have depleted the superficial layer of soil (in which iodine is present) is washed away of iodide and most iodide is found in the oceans. The concentration of iodide in sea water is approximately 50 g/L. Iodide ions in seawater are oxidized to elemental iodine, which volatilizes into the atmosphere and is returned to the soil by rain, completing the cycle. However, iodine cycling in many regions is slow and incomplete, leaving soils and drinking water iodine depleted. Iodine-deficient soils are common in mountainous areas and areas of frequent flooding, especially in South and Southeast Asia. Although many inland areas, including central Asia and Africa and central and eastern Europe are iodine deficient, iodine deficiency may also affect coastal and island populations (Zimmermann, 2009).

Food crops and water derive iodine from the soil. In areas, where soil has adequate iodine, 90% of the iodine requirements are met by the iodine present in the diet and the rest 10% from water. Consumption of crops and plants grown on iodine deficient soils leads to production of foods deficient in iodine leads to IDD in the population (Kapil, 2008). Food grown in iodine deficient regions can never provide enough iodine to the population and live-stock living there. Unlike nutrients such as iron, calcium or vitamins, iodine does not occur naturally in specific foods; rather, it is present in the soil and is ingested through foods grown on that soil (Kapil, 2007).

Besides nutritional iodine deficiency, a variety of other environmental, socio-cultural and economic factors operate to aggravate iodine deficiency and related thyroid dysfunctions. These include poverty related protein-energy malnutrition, ingestion of goitrogens through unusual diets which interfere with the intestinal absorption of iodine. Moreover, several environmental and genetic factors interfere with the processes of thyroxin synthesis leading to goiter formation. The genetic factors, which are rare, mainly affect the enzymes involved in thyroxin synthesis. Environmental factors are amongst the most common factors that interfere in thyroxin synthesis and lead to goiter formation. The most important environmental factors are environmental iodine deficiency and goitrogens (Kapil, 2007).

2.4.1. Effect of other micronutrients on iodine metabolism

Multiple micronutrients in the bodily metabolism operate synergistically so that supplying several micronutrients may assist in reducing the prevalence of iodine, iron and vitamin A deficiencies in school-age children (Zimmermann *et al*., 2004). Despite ongoing efforts to control micronutrient deficiencies in low-income countries, deficiencies in iodine, iron, zinc, and vitamin A remain major public health problems. The effects of iron and iodine deficiencies on child deaths were estimated to be smaller, though their impacts on cognitive development, educability and future economic productivity potential are considerable (Black *et al*., 2008).

Deficiencies of Iron, Zinc and vitamin A often coexist, possibly because of similar causal factors, such as inadequate dietary intake or absorption from predominantly plant-based diets, sub-optimal breast feeding practices, diseases that either induce excessive losses or impair use of the micronutrients and physiological states that increase requirements during rapid growth during childhood and pregnancy (Winichagoon, 2008). Besides iodine deficiency, other micronutrient deficiencies adversely affect the thyroid hormone synthesis (Hess and Zimmermann, 2004).

Iron is essential for human health because of its capacity to participate in redox reactions and its role in oxygen transport in the body. Iron deficiency can adversely affect cognitive development in childhood, immune function and pregnancy outcomes. Iron deficiency is more likely to occur in populations that rely on plant-based diets, which have low iron bioavailability. The prevalence of iron deficiency is estimated to be high, and it is likely that iron and iodine deficiency often co-exist. In surveys in SAC in West and North Africa, 23–25% suffers from both goiter and iron deficiency anemia (Hess, 2010).

Iron deficiency impairs thyroid hormone metabolism because of the two first steps in the thyroid hormone synthesis catalyzed by TPO, which are iron requiring enzymes. Concurrent iron deficiency anemia impairs the therapeutic response to iodine supplementation, possibly mediated by lowers plasma T_3 and T_4 concentrations, and reduces conversion of T_4 to T_3 or through decreased TPO activity impairing iodide organification (Zimmermann *et al*., 2000a). Because of these impairments in iodine metabolism, goiter in anemic individuals may become less

responsive to iodine treatment. Combining iodine and iron supplements can reduce goiter more rapidly than iodine alone (Zimmermann *et al*., 2000b).

Various mechanisms have been suggested for the interaction between iron and iodine deficiencies. Results from animal studies suggest that iron deficiency anemia may influence thyroid metabolism by altering the central nervous system control, decreasing T_3 binding to hepatic nuclear receptors and reducing TPO activity, an enzyme essential for thyroid hormone synthesis. Iron deficiency anemia could also impair thyroid metabolism through lowered oxygen transport. It is likely that these mechanisms jointly contribute to the impairment of thyroid function in iron deficiency (Hess, 2010).

A series of randomized controlled trials consistently found a significant reduction in thyroid volume in iron-deficient school-age children when iron was provided along with iodized salt either as iron supplement or included into double fortified salt. These findings suggest that a high prevalence of iron deficiency among children in areas of endemic goiter may reduce the effectiveness of iodized salt program. Thus, the prevention of iron deficiency is not only beneficial for iron-related outcomes, but also to improve the response to iodized salt consumption (Hess, 2010).

The coexistence of selenium deficiency with thyroid hormone disturbances may occur during long-term parenteral nutrition, cystic fibrosis, or may be the result of poor nutrition of children, adults and the elderly. A properly functioning thyroid gland has the ability to maintain high levels of selenium in serum even in conditions of inadequate dietary supply of this component. Considerable selenium deficiencies disturb the metabolism of thyroid hormones by inhibiting the synthesis and activity of deiodinase iodothyronine, which is responsible for the conversion of thyroxin into more metabolically active forms (Kawicka and Regulskallow, 2015).

Selenium has been found to be an important co-factor for both physiological function and in autoimmune disease of the thyroid. H_2O_2 is an essential co-substrate for TPO enzyme during the oxidation of inorganic iodine for thyroid hormone synthesis and the number of H_2O_2 molecules produced is proportionate to the intensity of TSH receptor stimulation. However, even in physiological conditions much higher amounts of H_2O_2 are produced than consumed by the

iodination process, potentially exposing the thyroid gland to excessive amount of free radicals in addition to the normal share of a cell. Selenoproteins such as glutathione peroxidase and thioredoxin reductase neutralize these excess H_2O_2 and they are therefore considered as essential Selenoproteins in the thyroid hormone synthesis. In pathological hyperactivity, a large volume of H_2O_2 and reactive oxygen species are produced and proportionately large quantities of Selenium are required to protect the thyroid gland from superoxide damage (Dharmasena, 2014).

Selenium functions largely through an association with proteins, known as selenoproteins. As selenocysteine, it is an integral component of two important enzymes glutathione peroxidase and iodothyronine deiodinase that are present in many tissues, including the thyroid gland. Briefly, there are three types of deiodinases. Two 50-deiodinases (50DI and 50DII) catalyze the activation of the prohormone T_4 to the thyromimetically active thyroid hormone T_3 and 50DI is also involved in the degradation of reserve T_3. The third selenocysteine-containing deiodinase inactivates thyroid hormones, both the prohormone T_4 and its active metabolite such as T_3 and 3, 5-T_2 (Kohrle *et al*., 2005).

Glutathione peroxidase and thioredoxin reductase are expressed in thyroid tissue and protect the thyroid gland from hydrogen peroxide produced during the synthesis of thyroid hormone, thereby protecting against oxidative damage. In conditions of inadequate supply of both iodide and selenium, complex rearrangements of thyroid hormone metabolism enable adaptation by increasing retention of selenium in the brain, endocrine tissues, and especially in the thyroid gland and iodide in the thyroid (Kohrle, 2005).

Zinc is an essential element for the proper synthesis and metabolism of thyroid hormones. Adequate zinc nutrition is essential for human health because of zinc's critical structural and functional roles in multiple enzymes that are involved in gene expression, cell division and growth, immunological and reproductive functions. As a consequence, zinc deficiency affects children's physical growth, and the risk and severity of a variety of infections (Brown *et al*., 2004).

Occurrence of zinc deficiency can reduce thyroid activity and resting metabolic rate. Zinc deficiency reduced the activity of T_3 hormone in blood of animals more than the reduction in the energy supply. There are implications that zinc is also important for normal thyroid homeostasis. Zinc's role is complex and may include effects on both the synthesis and mode of action of the thyroid hormones (Hess, 2010).

Vitamin A supplements can be effective in treating Vitamin A deficiency in areas of mild iodine deficiency children and have an additional benefit through suppression of the pituitary TSH-beta gene, decreasing excess TSH stimulation of the thyroid, and ultimately reducing the risk of goiter and its sequelae (Zimmermann *et al*., 2007). Occurrence of vitamin A deficiencies in autoimmune thyroid diseases may be associated with a reduced uptake of iodine by the thyroid gland and the restricted synthesis and secretion of thyroid hormones (Kawicka and Regulskallow, 2015).

A diet low in vitamin A affects the functioning of the pituitary-thyroid axis. It was proven that a diet low in vitamin A and iodine can be the cause of higher incidence of hypothyroidism compared with a diet low in iodine only. Studies showed that increased secretion of TSH and increased size of the thyroid gland in children with severe deficiency of iodine and vitamin A. Vitamin A supplementation can reduce the impact on the activity of TSH and thus reduce the risk of goiter appearance and its consequences (Kawicka and Regulskallow, 2015).

2.4.2. Effect of goitrogens

Cyanogenic glucosides, thioglucosides and thiocyanate are the goiterogenic (antithyroid) constituents of cyanogenic plants that are often used as food by human and animals. Large differences in glucosides content of plants belonging to the same family and the same taxonomy and grown within the same geographical area owing to their genetic backgrounds and ecological factors have been reported. Cyanogenic constituents affect hormone synthesis in thyroid gland either by inhibiting iodide uptake or interfering the activity of TPO, by inhibiting the organification of iodide (I^- leads to I_2), or iodination of tyrosine in thyroglobulin and coupling reaction (Chandra *et al*., 2004).

Goitrogenic substances interfere with the thyroid hormone production in the thyroid gland, which is where they get their name goiter. Goiter is the enlargement of the thyroid gland, which may occur as a way of trying to compensate for inadequacy of hormone. Goitrogens can induce hypothyroidism and goiter. In hypos, goitrogens can further depress thyroidal function and stimulate the growth of thyroid (goiter). Traditionally, goitrogens are considered as chemicals that interfere with iodine level or thyroid hormone in some way. These goitrogens include foods, drugs and chemicals **(**Enechi *et al*., 2013).

Goitrogens may act directly on the gland or indirectly by altering the regulatory mechanisms of the gland and peripheral metabolism and excretion of T_3/T_4. Thyroid stimulating hormone is the most potent stimulus in the growth and enlargement of the thyrocyte. Food and water were regarded as the sources of these goitrogens and many possible agents were identified (Fernando *et al*., 2012). Even though inadequate intake of iodine is the principal cause of IDD, goiterogenic food items like cabbage, cassava, millet, soya bean, bamboo shoot, turnip, kale, which interfere with the metabolism of iodine and hormone secretion (Mezgebu *et al*., 2012). In addition**,** contamination of drinking water by microbes is also factor that contributes to the occurrence of IDD (Wolka *et al.,* 2014).

Iodide absorption is reduced in the presence of humic acids in drinking water, and of thiocyanates, isothiocyanates, nitrates, fluorides, calcium, magnesium and iron in food and water (EFSA, 2014). The goiterogenic potential of a plant not only depends on the relative concentrations of cyanogenic constituents found in fresh plant but also on its processing as food, so in the areas where these plant foods are consumed, the common measures to reduce the goiterogenic potency include soaking, washing, boiling and cooking (Chandra *et al*., 2004).

Foods containing such chemicals include cassava, cabbage, and root vegetables such as swedes and turnips. Cassava is a staple in Africa and tropical areas in the Pasific basin. It contains cyanogenic glycosides, which are a source of cyanide. If cassava is not treated by boiling in water before human consumption, the cyanide is converted in the body to thiocyanate, which inhibits the activity of TPO, the enzyme responsible for the uptake of iodine by the thyroid gland. Goitrogens in cabbage and related plants also inhibit TPO (Thurnham, 2014).

Cassava plant which plays dominant role in feeding many people in tropical developing countries contain cyanogenic glycoside that inhibit iodine uptake by the thyroid gland thereby aggravate iodine deficiency when unprocessed cassava is consumed. Today this crop is spreading too many parts of Africa including Ethiopia. Where cassava is consumed as staple food, endemic goiter and cretinism are common**.** Cassava is consumed as staple food in many parts of Southern Nations Nationalities and Peoples, Benishngul-Gumuz and some part of Western Oromia region (Abuye *et al*., 2008).

Table 2.2: Goitrogens and micronutrient deficiencies that affect iodine metabolism and thyroid function (Zimmermann *et al*., 2014).

Goitrogens and micronutrient	**Mechanism**
Cassava, lima beans, linseed, sorghum, sweet potato	Contain cyanogenic glucosides that are metabolized to thiocyanates and act by competing with iodine for uptake by thyroid gland
Cruciferous vegetables: cabbage, kale, cauliflower, broccoli, turnips, rapeseed	Contain glucosinolates; these metabolites act by competing with iodine for thyroidal uptake
Soy, millet	Flavonoids impair thyroid peroxidase activity
Industrial pollutants (that enter food and water), Perchlorate, nitrate (disulfides from coal processes)	Competitive inhibitors of the sodium/iodide symporter; they act by decreasing transportation of iodine to the thyroid
Smoking	One of the important goitrogens; during breastfeeding, smoking is known to be linked to reduced iodine content in the breast milk; the mechanism behind this is that in smoking women, the higher serum concentrations of thiocyanate compete with iodine for active transport into the secretory epithelium of the lactating breast
Selenium deficiency	Accumulated peroxides due to selenium deficiency may damage the thyroid, and deiodinase deficiency may impair the synthesis of thyroid hormone
Iron deficiency	The activity of heme dependent TPO is reduced and the efficacy of iodine prophylaxis may be compromised
Vitamin A deficiency	Due to the deficiency there is a decrease in vitamin A mediated suppression of the pituitary TSH beta gene and an associated increase in TSH stimulation and goiter

TPO- Thyroid Peroxidase, TSH- Thyroid Stimulating Hormone

2.5. Assessment of iodine status

Concentration of iodine in salt and iodine in urine is two of the key indicators used in the assessment of both the process side as well as the impact side of iodine nutrition in populations. In Process indicator the iodine concentration in salt needs to be monitored at the production, retail and household levels. For example, the iodine content of iodized salt or the percentage of households using adequately iodized salt. This is necessary to answer different public health questions and to ensure the delivery of adequately iodized salt to the consumer (Zimmermann *et al*., 2008).

Human variables responding to the consumption of iodine are considered impact indicators, for example, the urinary iodine concentration and the volume of the thyroid gland. Impact indicators, also called outcome indicators, indicate whether the iodine consumption by the consumer was insufficient, sufficient or excessive in women and children. Factors influencing the process of supplying iodized salt to the consumer, include the assessment of the iodine content of salt at the point of production, retail and household levels, estimating the coverage of the percentage households using iodized salt, determining the iodine content of processed foods and assessing the level of knowledge regarding iodine nutrition amongst the adult population (Jooste and Zimmermann, 2008).

Urinary iodine is the most useful impact indicator because the rapid turnover of iodine in the body reflects recent changes in iodine intake (Jooste and Zimmermann, 2008). The shift from clinical to biochemical assessment of iodine status has improved the availability and quality of nationally representative iodine deficiency prevalence data. The magnitude of iodine deficiency, based on UIC, was more recently estimated by WHO in 2003 and 2007 (Andersson *et al*., 2010). Methods recommended for assessment of iodine nutrition are the UIC, thyroid size and thyroid function tests, including thyroglobulin (Zimmermann and Crill, 2010).

2.5.1. Determination of iodine in salt

Iodine is normally added to the salt in the form of potassium iodide (KI) or potassium iodate (KIO_3). Iodide can be oxidized to iodine due to exposure to humidity, atmospheric oxygen, heat and sunlight. The iodine produced readily sublimates and is lost from iodized salt (Shabani *et al*.,

2011). Therefore, the use of KIO_3 is usually preferred as an iodine supplement in iodized salt because of it is extremely stable compound that is unlikely to be affected by impurities, and its lower solubility in water gives it another important advantage over KI, because it migrates much less readily and so cannot easily be removed from iodinated salt. Since KIO_3 rapidly breaks down in the human body, large doses of the compound can be tolerated, providing a source of iodine to the thyroid gland for the synthesis of thyroid hormone (Demaeyer *et al*., 1979).

According to the WHO/UNICEF/ICCIDD (2001) 20% of the iodine in salt is lost from production to a household, and another 20% is lost during cooking before consumption. The average salt intake is 10g per person per day. Hence to provide 150μg daily requirement of iodine for each person, the salt iodine concentration at the point of production should be 20–40 mg of iodine/kg of salt (Khazan *et al*., 2013).

For monitoring and evaluating salt iodization programme and for efficient planning and decision making regarding these programmes, the iodine content of salt is, in addition to the testing performed at the production sites, also required at the retail and household levels. Quantitative determination of iodine in salt at each of these three levels answers distinctively different public health questions contributing to the identification of the successful elements of an iodization programme as well as identifying factors weakening the supply of adequately iodized salt to the consumer. Another advantage of quantitative measurement of iodine in salt is the ability to calculate the coverage of percentage of households using inadequately iodized, adequately iodized salt as well as calculating the percentage households using excessively iodized salt **(**Jooste and Strydom, 2010).

Qualitative rapid test kits are often used at the household level for estimating the percentage of households using iodized salt. While these rapid test kits quite accurately distinguish between iodized and non-iodized salt, its ability to measure the iodine concentration in quantitative terms and to distinguish between iodine concentrations in salt below and above the critical level of 15 ppm is questionable. Therefore, the value of the rapid test kits is its ability to demonstrate whether salt is iodized; a characteristic sometimes used as an advocacy tool **(**Jooste and Strydom, 2010).

Quantitative and qualitative evaluations of iodine in salt can be determined by titration and rapid test kits, respectively. Effective quality control in the production of iodate salt requires a simple, reliable and cost-effective analysis method. Depending on the form of iodine (iodate or iodide), different salt iodine testing methods are needed in the fortification process (Khazan *et al*., 2013).

2.5.1.1. Titration method

Titration is the most frequently used method to determine quantities of iodine in salt because of its accuracy, relatively easy to use and incurs low cost. This method is recommended at various levels of a distribution system. Internal and external quality control measures are necessary once a method has been established. The titration method for KIO_3 was introduced in 1979. However, the titration method is not recommended for routine national monitoring purposes because it's a time-consuming process (Khazan *et al*., 2013).

In titration method the iodine content of salt can be determined by liberating iodine from a salt sample then by the titration of iodine with sodium thiosulphate using starch as an external indicator. First, the liberation of free iodine from the salt is carried out by adding concentrated sulfuric acid (H_2SO_4) which liberates the free iodine from the iodate in the salt sample. Excess KI is added to solubilize the free iodine, which is quite insoluble in pure water under normal conditions. Free iodine is consumed by sodium thiosulfate in the titration step (Nepal *et al.,* 2013).

The amount of thiosulfate which is used is proportional to the amount of free iodine which is liberated from the salt. Starch is added as an external (indirect) indicator of this reaction, and it reacts with the free iodine to produce a blue color. When it is added towards the end of the titration (when only a trace amount of free iodine is left) the loss of the blue color, or the endpoint, which occurs with a further filtration, indicates that all the remaining free iodine has been consumed by the thiosulfate. The salt iodine content is estimated in ppm by the iodometric titration method (Nepal *et al.,* 2013).

The titration method is undoubtedly the most popular method used for determining the iodine concentration quantitatively in salt because of its accuracy, relative ease of operation and low cost. This method could be used at any level of monitoring or evaluation of intervention

programmes. The titration method is globally recognized as the reference method for measuring iodine in iodized salt and must always be considered as a back-up method to any alternative method. Furthermore, it should be obligatory for all those producing and selling salt for human consumption to routinely operate and maintain a titration laboratory at the site where salt iodization is undertaken **(**Jooste and Strydom, 2010).

2.5.1.2. Rapid salt testing kits

Rapid test kits are applicable to both qualitative and semi quantitative estimations of iodine content. These kits are rapid, simple and easily applied in a field setting, and need no training of chemistry laboratory personnel. Spot tests can be used at sites of production, distribution, retail, and household. They are especially suitable for small-scale salt producers who may not be able to achieve the level of sophistication needed to establish more quantitative laboratory titration methods. There are various spot tests available, all of which use the same common reaction mechanism, and a starch based reagent solution that produces a blue color when iodine is present in a salt sample (Pandav *et al.,* 2000).

The semi-quantitative estimation of the salt iodine is based on the reaction between starch and iodine to form starch-iodine complex. This test solution contains an acidic buffer and a reducing agent, which convert KIO_3 to elemental iodine (I_2). This elemental iodine reacts with the iodide ion (I^-) to form a tri-iodide anion (I^{3-}) and this further reacts with it to give a penta-iodide anion (I^{5-}). This penta-iodide anion (I^{5-}) forms a visible blue black complex with the amylase of the starch. The color of the test sample is compared with the standard color chart (<15 ppm or >15 ppm) for calculating the salt iodine content (Nepal *et al.,* 2013).

In household surveys where the rapid test kit is used to estimate the percentage of households using iodized salt, it is important that a sub-sample of at least 10%, preferably 20%, of salt samples be sent to a laboratory for back-up titration determinations. Thus the accuracy of coverage estimates can be established and the usefulness of rapid test kits verified. However, titration remains the method of choice for establishing the coverage of percentage households using salt containing >15 parts per million (ppm) **(**Jooste and Strydom, 2010).

2.5.2. Iodine determination in urine

The implementation of IDD control programs uses the principal indicator of urinary iodine rather than thyroid size, thyroid stimulating hormone and thyroglobulin. However, thyroid size is more useful as a baseline assessment of the severity of IDD and it has a role in assessing the long-term impact of control programs (Azizi *et al*., 2003). Because the bioavailability of iodine is high and about 90% of iodine consumed is excreted in urine, the urinary iodine serves as a good reflection of recent dietary iodine intake and therefore of iodine status. Traditionally, studies of iodine nutrition are done mainly on school-age children because of the vulnerability and accessibility of this age group **(**Jooste and Strydom, 2010). Urinary Iodine Excretion (UIE) can be a good indicator of current dietary iodine intake for IDD (Khazan *et al*., 2013).

It can vary in individuals from day-to-day and even within a day. However, more accurate estimates of iodine levels among populations can be determined by a median evaluation of UIE from at least 30 individuals (Khazan *et al*., 2013).The median value, rather than the mean, for a sampled population is often considered as an indicator, as UIC from populations are usually not normally distributed. Iodine deficiency in groups is diagnosed according to specific cut-off values of median UIC **(**Jooste and Strydom, 2010). Random morning spot UIC is the most commonly used method for evaluating urinary iodine assessment. Twenty-four hour urine samples are unsuitable, as they are difficult to obtain where accurate sample collection is critical (Shelor and Dasgupta, 2011).

Table 2.3: Epidemiological criteria for assessing iodine nutrition in a population based on MUI concentration for SAC (Zimmermann, 2008).

MUI (µg /L)	Iodine intake	Iodine nutrition
<20	Insufficient	Severe iodine deficiency
20–49	Insufficient	Moderate iodine deficiency
50–99	Insufficient	Mild iodine deficiency
100–199	Adequate	Optimal
200–299	Above requirements	Likely to provide adequate intake for pregnant/ lactating women, but may pose a slight risk in the overall population.
>300	Excessive	Risk of adverse health consequences (iodine-induced hyperthyroidism, autoimmune thyroid disease)

MUI- Median Urinary Iodine

A suitable method for urinary measurement should consider high accuracy, reliability, high speed, technical demand, low complexity of instrumentation, independence from sole source suppliers, availability of high-quality reagents, safety and low cost **(**Jooste and Strydom, 2010). Methods used to quantified iodide and iodine by a variety of methods, including gas chromatography-mass spectrometry, Inductively coupled plasma atomic emission spectrometry, Ultra violet-Visible spectrophotometry, catalytic spectrophotometric methods, atomic absorption spectrometry, Inductively coupled plasma mass spectrometry **(**ICP-MS), capillary electrophoresis and chromatography (Shelor and Dasgupta, 2011).

Most of these techniques are either very expensive or require several stages of manipulation for routine analysis. Methods applied for assessing levels of nutritional iodine in populations, particularly in developing countries, should be rapid, simple, reliable, affordable, and flexible. Presently, urinary iodine determination is performed almost entirely by one of the two methods; an age-old kinetic spectrophotometric method called the Sandell–Kolthoff reaction and an unsophisticated method, the so called ICP-MS method which permits superb sensitivity, and as such, sometimes allows direct sample analysis following dilution such as in urine samples (Shelor and Dasgupta, 2011).

2.5.2.1. Sandell–Kolthoff reaction

By far the most commonly used laboratory methods for analyzing UIC are based on the Sandell–Kolthoff reaction described in 1937. These analytical methods consist of an initial digestion step to get rid of interfering substances, followed by the Sandell–Kolthoff reaction; of which the reading is obtained spectrophotometrically or colorimetrically **(**Jooste and Strydom, 2010). The catalytic role of iodine in converting yellow Ce^{+4} to the colorless Ce^{+3} is the basis of these measurements (Hedayati *et al*., 2011). The color change that occurs during the reaction can therefore be used to determine the iodine concentration in an unknown urine specimen, when compared to a set of known iodine standards. The reduction of absorbance due to Ce^{+4} is typically measured at 405–420 nm (Shelor and Dasgupta, 2011).

The first step of reaction is elimination of interfering substances and releasing the iodine bound to urine excretory compounds, by chloric acid, ammonium persulfate digestion and ashing

digestion. The iodine-releasing step is an essential procedure during urinary iodine digestion. Ashing is not a suitable method for this purpose due to the potential false negative errors (Hedayati *et al*., 2011). The next step reaction was measured during the reduction of the yellow-coloured ceric ions by arsenic in the presence of iodide to form colorless cerous ions and elemental iodine. The decrease in yellow color over a fixed period is measured by a colorimeter with an appropriate filter or with a spectrophotometer and plotted against a standard curve constructed with known amounts of iodine (Jooste and Strydom, 2010).

Ammonium per-sulfate digestion has recently been reported as a nonhazardous, non-explosive and easy-to use procedure but it remains inadequate for testing because it is time consuming and produces a level of toxic waste that exceeds the allowed limitation (Khazan *et al*., 2013). Despite that this method provides an accurate measurement, it has several disadvantages including toxic waste production from arsenic trioxide in the Sandell–Kolthoff reaction, acidic gas leakage during sample digestion that requires a special fume hood and difficulty in obtaining chloric acid from chemical vendors because of its instability. Sample preparation time to determine urinary iodine levels, can be substantially reduced with the use of rapid microwave digestion and the microplate reading format method (Hedayati *et al*., 2011).

2.5.2.2. Inductively Coupled Plasma Mass Spectrometry

ICP-MS has become the method of choice for reliable determination of iodine in biological samples. The ICP-MS method is fast, accurate, robust and specific to the evaluation of urinary iodine. Since 2000, in the United States National Health and Nutrition Examination Survey, UIC was determined using ICP-MS (Shelor and Dasgupta, 2011). Over years, modifications have been made to ICP-MS procedures, mostly at the dilution step or by varying the internal standard. In general, inter and intra batch coefficients of variation were between 2-5% (Jooste and Strydom, 2010).

As a result of specificity, ICP-MS is not only a resource for quality assurance, but it is also particularly adaptable for long-term monitoring of a population's iodine status. Despite the high cost for instrumentation, the application of ICP-MS may soon become a routine procedure in clinical chemistry, mainly because of its ability to measure several trace elements

simultaneously. However, this expensive, sophisticated procedure makes the process unrealistic for widespread use (Khazan *et al*., 2013). ICP-MS is considered the most accurate method of analysis for urinary iodine and is often used as the 'gold standard'. It is a technically advanced method and is costly to implement and operate **(**Jooste and Strydom, 2010).

A simple manual method for estimating the prevalence of IDD has recently been proposed. The modified methods of the Sandell–Kolthoff reaction, such as spectrophotometric measurement or microplate reading, are frequently applied in most laboratories. Ammonium persulfate digestion on microplate method was compared with the conventional chloric digestion method and the ICP-MS method, and was identified as a sensitive method for urinary iodine detection (Macours *et al*., 2008).

2.5.3. Thyroid size

Thyroid size is a sensitive marker for iodine deficiency because goiter, although not the most severe consequence of iodine deficiency, is the most clinically evident. Assessment by palpation is too crude to be anything more than qualitative except in severe deficiency, but ultrasonography is precise, quantifiable, and easily performed. Two methods are available for measuring goiter: neck inspection and palpation, and thyroid ultrasonography. By palpation, a thyroid is considered goitrous when each lateral lobe has a volume greater than the terminal phalanx of the thumbs of the subject being examined. However, palpation of goiter in mild iodine deficiency has poor sensitivity and specificity and measurement of thyroid volume by ultrasound is preferable (Zimmermann and Crill, 2010).

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 standing behind the subject, sliding his/her fingers along each side of the trachea (windpipe) between the thyroid cartilage (Adam's apple) and the top of the sternum. The size and consistency of the thyroid is carefully noted. The thyroid moves upward when the subject swallows, which can sometimes define its size. The thyroid gland whose lateral lobes have a volume greater than the terminal phalanges of the thumbs of the person examined is considered goitrous (WHO/UNICEF/ICCIDD, 2008).

The thyroid size of SAC 6 to 12 years has been used as biological marker of iodine deficiency for many years, but its usefulness is limited because the thyroid size regresses slowly after increasing the iodine intake in salt iodization programmes and results need to be interpreted cautiously. Thyroid size determined by palpation is graded as Grade 0 when the thyroid is not palpable, Grade 1 when it is palpable but not visible and the size of the lobe is bigger than the size of the end digit of the thumb of the individual being examined, and Grade 2 when the thyroid is palpable and visible when the neck is in the normal position. When more than 5% of school age children have enlarged thyroids, goiter is endemic. Prevalence of goiter from 5% to 19.9%, 20% to 29.9% and more than 30% indicate mild, moderate or severe degrees of IDDs respectively (WHO/UNICEF/ICCIDD, 2007).

Table 2.4: Classification of goiter by palpation method (Zimmermann *et al*., 2003).

Grade	Characteristics
0	No palpable or visible goiter.
1	A goiter that is palpable but not visible when the neck is in the normal position (i.e. the thyroid gland is not visibly enlarged). Nodules in a thyroid that is otherwise not enlarged fall into this category.
2	A swelling in the neck that is clearly visible when the neck is in a normal position and is consistent with an enlarged thyroid gland when the neck is palpated.

Table 2.5: The relationship between the severity of IDD and goiter prevalence

Total goiter rate (%)	Severity of iodine deficiency
0–4.9	None
5–19.9	Mild
20–29.9	Moderate
≥30	Severe

(WHO/UNICEF/ICCIDD, 2008).

2.6. Strategies to prevent and control iodine deficiency

2.6.1. Universal salt iodization

In 1994, a special session of the 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. 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. This will greatly assist sustainability (Sullivan, 2010).

In nearly all regions affected by iodine deficiency, the most effective way to control iodine deficiency is through salt iodization. USI is a term used to describe the iodization of all salts for human (food industry and household) and livestock consumption. Although the ideal, even in countries with successful salt iodization programs, USI is rarely achieved, as food industries are often reluctant to use iodized salt and many countries do not iodize salt for livestock. Recommend that iodine is added at a level of 20–40 mg iodine/kg salt, depending on local salt intake (WHO/UNICEF/ICCIDD, 1993).

Iodine can be added to salt in the form of KI or KIO_3. Because KIO_3 has higher stability than KI in the presence of salt impurities, humidity, and porous packaging, it is the recommended form in tropical countries and those with low-grade salt. Iodine is usually added after the salt has been dried. Two techniques are used such as the wet method (where a solution of KIO_3 is dripped or sprayed at a regular rate on to salt passing by on a conveyor belt) and the dry method (where KI or KIO_3 powder is sprinkled over the dry salt). Optimally, packaging should be in low-density polyethylene bags. In a multi-country study, high humidity combined with porous packing resulted in up to 90% losses of iodine in 1 year of storage in high-density polyethylene bags, compared to 10–15% from low density polyethylene bags (Zimmermann, 2008).

Salt iodization remains the most cost-effective way of delivering iodine and of improving cognition in iodine deficient populations (Engle *et al.,* 2007)**.** Worldwide, the annual costs of salt iodization are estimated at 0.02–0.05 United State (US) $ per child covered, and the costs per child death averted are US$1000 and per disability-adjusted life year gained are US$34–36 US**.** Looked at in another way, prior to widespread salt iodization, the annual potential losses attributable to iodine deficiency in the developing world have been estimated to be US$35.7 billion as compared with an estimated US$0.5 billion annual cost for salt iodization, that is a 70:1 benefit: cost ratio**.** The World Bank strongly recommends that governments invest in micronutrient programs, including salt iodization, to promote development by concluding ‘‘probably no other technology offers as large an opportunity to improve lives at such low cost and in such a short time’’ (Zimmermann, 2008).

Salt iodization is the recommended strategy for control of IDD because salt is consumed by virtually everyone and its intake is fairly consistent throughout the year. Its production or importation is restricted to a few sources in many countries, In addition the iodization technology is simple and inexpensive to implement and also addition of iodine to salt does not affect its colour or taste. The quantity of iodine in salt can be monitored simply during production, retail and in the household (Zimmermann *et al*., 2008).

2.6.2. Iodine supplementation

In some countries and areas with insufficient access to iodized salt for vulnerable groups of the population, additional temporary strategies need to be considered to ensure optimal iodine nutrition for these groups while strengthening the salt iodization programmes to reach universal coverage. In particular, each country should assess the current situation of its salt iodization programme to identify national or sub-national problems and to update its strategies and action plans. For children 7 to 24 months of age, either supplementation or use of iodine-fortified complementary foods may be a possible temporary public health measure (Zimmermann *et al*., 2008).

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 intramuscular injection.

The intramuscular route has a longer duration of action, but oral administration is more common because it is simpler. Usual doses are 200–400 mg iodine per year and it is often targeted to women of child-bearing age, pregnant women, and children. Iodized oil given in the first and second trimesters of pregnancy decreased the prevalence of neurological abnormalities and improved developmental test scores through 7 year, compared with supplementation later in pregnancy or treatment after birth**.** Its disadvantages are an uneven level of iodine in the body over time and the need for direct contact with individuals with the accompanying increased costs (Zimmermann, 2008).

Iodine can also be given as KI or KIO_3 as drops or tablets. Single oral doses of KI monthly (30 mg) or biweekly (8 mg) can provide adequate iodine for SAC**.** Lugol's iodine, containing 6mg iodine per drop and similar preparations are often available as antiseptics in rural dispensaries in developing countries and offer another simple way to deliver iodine locally. Whether providing preterm infants with supplemental iodine prevents morbidity and mortality is uncertain (Ibrahim *et al*., 2006)**.** In countries or regions where a salt iodization program covers 90% of households and has been sustained for years, and the MUI indicates iodine sufficiency, pregnant and lactating women do not need iodine supplementation **(**Andersson *et al*., 2007).

2.7. Dietary source of iodine

The main natural sources of dietary iodide are seafood (200-1000µg/kg) and seaweed (0.1-0.2% iodide by weight). Iodide is also found in cow's milk (20-70µg/liter) and may be added to table salt (100µg/g of sodium chloride) to ensure an adequate intake of iodine (Duressa *et al*., 2014). Foods of marine origin have higher iodine content because iodine in seawater is concentrated in marine plants and animals. In many countries, use of iodized salt in households during cooking and consumption provides additional iodine (Zimmermann and Trumbo, 2013).

In countries in which salt is iodized, it is generally the main dietary source of iodine is salt used in food production. WHO recommends levels of 20–40 mg/kg iodine in salt. Commercially available iodized salt contains iodine in the range of 15–80 mg/kg. In settings in which foods are mainly prepared at home, household salt is the major iodine source. In contrast, in industrialized countries, salt used in processed foods contributes approximately 60–80% of the total salt intake.

The main salt sources from processed foods in typical western diets are bread, dairy products, and processed meat (Zimmermann and Andersson, 2012).

Iodine can be obtained by consumption of foods that naturally contain it (fish, seafood, kelp, some drinking water, and vegetables grown in iodine sufficient soil) or to which it is added (table salt). Cow's milk is a source of iodine owing to iodine in cattle feed and the use of iodophor udder cleansers in the dairy industry. Sea salt naturally contains only a small amount of iodine. Dietary iodine is absorbed as iodide and rapidly distributed in the extracellular fluid, which also contains iodide released from the thyroid and by extrathyroidal deiodination of the iodothyronines. Iodide leaves this pool by transport into the thyroid and excretion into the urine (Zimmermann and Andersson, 2012).

The highest iodine levels were found in cereals and legumes. However, iodine concentration in tubers is low and varied between 12 and 30 μg/kg. The iodine supply of animals is mainly determined by their habitat and their food. The amount of iodine entering the human food chain depends on levels of consumption of key foodstuffs. In some study shows that all food items consumed by the population contained significantly low iodine which is reflected in the iodine content of human breast milk. Therefore breast milk composition indicates maternal deficiency of iodine which affects the nutritional status of the nursing infant (Cherinet and Kelbessa, 2000).

The farming population of Ethiopia relies predominantly on cereals such as maize, sorghum, barley, wheat and teff which provide 70% of daily energy intake. About 5% of the dietary energy comes from pulses, roots and tubers. Nevertheless, it is common to consume *injera* or bread prepared from the cereals with various vegetable sauces. A study shows that they can estimate the daily intake of iodine when *shiro* is included in the meal. For example, 150 g of *shirowot* (spiced legume sauce) in addition to 200g of teff prepared as *injera* will provide 24 μg of iodine. Supplemented with one liter of drinking water from the surface of a river in Kodowono will increase the intake to 40 μg/day. The intake is much lower when *injera* is consumed with sauce prepared from tubers. Estimated iodine intake by the survey population is thus far below the recommended daily allowance 150 μg/day (Cherinet and Kelbessa, 2000).

Chapter three

3. Materials and methods

3.1. Study area and period

Addis Ababa lies 9°1 ′48″N latitude and 38° 44 ′24″E longitudes. The city is located at the heart of the country, at an altitude ranging from 2,100 meters at Akaki in the south to 3,000(9,800 ft) meters at Entoto plateau in the North. Average annual rainfall is 1,184mm, of which about 80% falls between June and September, the months of July and August being the wettest. The hottest and driest months are usually April and May. The short rains fall during March to mid April, characterized by relatively cool nights and warm days.

Akaki-kality subcity is one of the ten sub-cities found in Addis Ababa. It is located in the south part of Addis Ababa. The subcity was randomly selected as study area for this research. The subcity had 10 districts. According to Central Statistic Agency July 2012 report, the projected population of Akaki-kality subcity was 201721 populations. The study was conducted from April to July 2015. According to 2015 data from Akaki-kality subcity Education office, in the subcity there were 12 secondary, 63 primary and 107 kindergarten schools. From the primary schools found in the subcity 18 were government primary schools. In this study 5 government primary schools in five districts were randomly selected.

Figure 3.1: Map of the study area.

3.2. Study design

School based cross-sectional study design was used. Questionnaire was used to collect socio-demographic information and knowledge and practice on iodized salt consumption. Moreover, analytical, biochemical and clinical assessment were used to assess salt iodine content, urinary iodine and goiter rate, respectively.

3.3. Study Population

Sampled populations were school age children from five governmental primary schools of Akaki-kality subcity. As per recommendation of WHO/UNICEF/ICCIDD (2001) the school-aged children 6-12 years from both sexes were selected, because of their high vulnerability to goiter, easy access, high representativeness of their age group in the community and applicability to a variety of surveillance activities were considered.

3.4. Eligibility Criteria

Inclusion criteria: Children who were in the age of 7-12 years and whose parents gave their consents to participate in the study were included.

Exclusion criteria: Children whose age less than 7 years and greater than 12 years and whose parents were not able to provide written consent were excluded from the study.

3.5. Sample size estimation and sampling procedure

Considering p= 0.193 from previous report by Abuye *et al.* (2007). According to the study goiter prevalence in SAC of Addis Ababa was 19.3% the sample size was calculated by considering the aforementioned goiter rate of Addis Ababa, at 95% confidence interval (CI) (z-value =1.96), design effect of 1 (DEFF=1) and margin of error (ME) of 5%. Using the following formula:

$$n = \frac{[(Z)^2\ pq]\ (DEFF)}{(ME)^2}$$

$$n = \frac{[(1.96)^2\ x\ 0.2x0.8]\ (1)}{(0.05)^2}$$

n = 246, by adding (10% non-response rate) = 246 + 25

N= 271

Where: p = 0.19

q = 1- p

Precision = 95%

Margin of error = 5%

Non-response rate and homogenous population (DEFF = 1)

Multi-stage sampling technique was used to select the study participants. In Akaki-kality subcity of Addis Ababa there are 10 districts. In order to get more representative samples the research was targeted in half of districts in the subcity. From each district individually one government primary school and a total of five schools were selected by simple random sampling method. Two schools (Akaki-mengist and Atse-tewodros primary school) where found in semi-urban area which is far away from the main road. Whereas, three schools (Kality-primary, Kality-bulbula and Eweket-wogagen primary school) where found in urban area. The next stage of sampling was systematic random sampling method used to select children's from grade 1 to grade 6 of each selected schools with equal sex and age proportion based on the number and list of students data was obtained from the schools.

3.6. Ethical considerations

Ethical clearance was obtained from ethical review committee of College of Natural Science, Addis Ababa University. In addition, permission was obtained from Akaki-kality subcity education office and the subcity was written support letter for the research to the selected districts education office and government primary schools. Informed verbal consent was obtained from school community after explained the objectives and major activities of the study. Based on the awareness and willingness of children's parent on the study a written assent form was signed and agreed to participate on the study. Data obtained from all study participants was recorded by code to keep their confidentiality and all of the study participants were acknowledged.

3.7. Variables of the study

3.7.1. Independent variables

Socio-demographic characteristics of study participants such as demography of children, occupation, ethnicity, religion, economic status, education status and iodized salt consumption of their family. Besides, families' awareness on iodine deficiency and their practice of iodized salt consumption in household were included.

3.7.2. Dependent variables

Urinary iodine status of children, goiter status of children and Iodine content of salt consumed.

3.8. Data and sample collection tools and techniques

The data was collected using a pre-tested structured and semi-structured questionnaire prepared by reviewing the previous similar studies. The questionnaire was translated in to Amharic and back to English to ensure its consistency. Both questionnaire about socio-demographic and knowledge and practice on iodine deficiency and iodized salt consumption was adopted from the following studies (Takele *et al*., 2003; Berhanu *et al.,* 2004; Nicsic *et al*., 2006; UNICEF, 2005; Sebotsa *et al*., 2009; Girma *et al*., 2012; Buxton and Baguune, 2012; Gebremariam *et al*., 2013; Daba *et al*., 2013; Wolka *et al*., 2013; Edith *et al*., 2013; Zokai *et al*., 2013; Mesele *et al*., 2014; Gebriel *et al*., 2014; Enyew *et al*., 2015; Kebede and Adinew, 2015; Gidey *et al*., 2015; Zoysa *et al*., 2015).

Both consent form and questionnaire were given to the sampled children's in order to signed and filled by their parents and then to return back to the schools. Two trained health professionals and principal investigator were reviewed the collected assent form were signed and information on questionnaire for its accurately and completely filled.

3.8.1. Salt samples collection

Subsamples of 50 children were randomly selected from the total of 270 students. Ten children were selected from each school asked to bring 20g of salt sample using tightly sealed plastic bag from their home. Household salt samples were collected and carefully stored until analysis in desiccators at room temperature. Iodine content in the salt samples was measured according to

Association of Official Analytical Chemistry (AOAC, 2000) by using standard iodometric titration method in Ethiopian Public Health Institute (EPHI) Food analysis laboratory. Each sample was analyzed in duplicate. After analysis, the salt samples were classified according to their iodine level in ppm. The Iodine content of salt expressed in terms of iodine nutrition as [adequately iodized > 15ppm; inadequately iodized <15ppm and no iodine 0 ppm] used for calculating the household consumption of iodized salt.

3.8.2. Urine samples collection

To collect urine sample children were provided coded screw plastic urine cups and 5 ml casual urine sample was collected from each child at school from all sampled children under direct supervision of the principal investigator and other health professionals. Then, the urine sample was immediately transferred into sterile screw-caped cryogenic vial labeled with each children code.

Meanwhile, at each day of sample collection, the samples were put in a conditioned cool box and transported to EPHI within 3 hours after the sample collection and stored in -40^{o}C deep freeze until analysis was performed. Each sample was analyzed in duplicate. According to WHO/UNICEF/ ICCIDD (2001) urinary iodine level < 20 μg iodine/L of urine indicates (severe), 20-49.9 μg iodine/L (moderate), 50-99.9 μg iodine/L (mild), 100-199.9 μg iodine/L (adequate), 200-300 μg iodine/L (above the requirement) and >300 μg iodine/L (excessive) level of iodine in the body.

3.8.3. Goiter assessment

All of the sampled children's who present on the day of assessment were clinically examined for enlargement of thyroid gland (goiter) by two experienced and trained health officers and principal investigator to minimize inter-individual variability by using palpation method as recommendation (WHO/UNICEF/ICCIDD, 2007). Assessment was done by both of three trained professionals in each children clinical examination of goiter. Based on the recommendation goiter assessment was conducted as [Grade 0 for no goiter; Grade1 for thyroid palpable but not visible and Grade 2 for thyroid visible with neck in normal position].The prevalence of goiter was expressed by TGR, which is the sum of (Grade 1 and Grade 2 goiter).

3.9. Data quality management

Questionnaire was objective based, logically sequenced, free of scientific terms, non-leading and pretested. The data collectors were provided one day training on the major activities of the study and for health professional how to assess goiter status of the children to minimize inter-individual variability. The collected data were checked by the principal investigator on daily basis for any incompleteness and consistency and possible correction was made. During data entry great care was given to avoid double entry and data cleaning was also done.

3.10. Analytical Method

3.10.1. Determination of salt iodate content

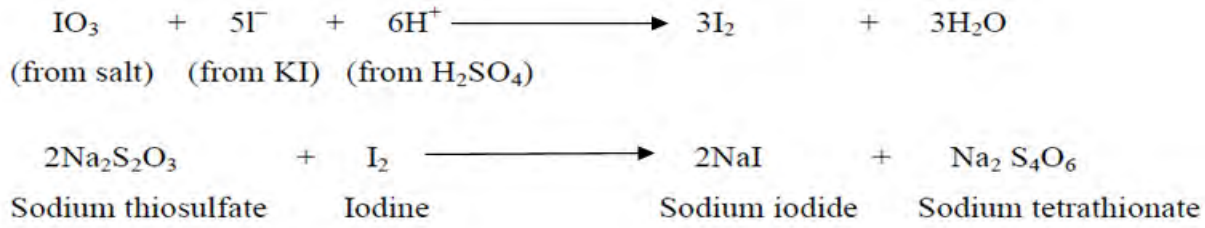
Iodine content of salt was determined according to AOAC (2000) using official method 925.56 by iodometric titration. The principle is that iodine is liberated by adding H_2SO_4 to a solution of iodized salt. KI solution is added to keep the iodine in the dissolved state. Iodine liberated was titrated with sodium thiosulphate ($Na_2S_2O_3$) solution to form sodium iodide and sodium tetrathionate.

The reaction mechanism includes two steps:

***Liberation of free iodine from salt*:** The addition of H_2SO_4 liberates free iodine from the iodate in the salt sample. Excess KI was added to help solubilize the free iodine, which is quite insoluble in pure water under normal conditions.

***Titration of free iodine with thiosulfate*:** free iodine is consumed by $Na_2S_2O_3$ 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 titration (when only a trace amount of free iodine is left) the loss of blue color, or end-point, which occurs with further titration, indicates that all remaining free iodine has been consumed by thiosulfate.

Reaction steps for iodometric titration of iodate:



Procedure:

Prior to taking a 10 g salt sample for analysis, salt should be thoroughly mixed, preferably in tightly sealed plastic bags to ensure that the iodine is homogeneously distributed in the salt. Usually 10 g iodated salt is dissolved in 50 ml distilled water, from which an aliquot of 50 ml could be analyzed as mentioned in the titration step below, without adjusting the concentrations of the reagents or calculation.

Titration: once the salt is dissolved in the measured amount of water, sulfuric acid (1–2 ml) and potassium iodide (5 ml) is added to the salt solution, which in the presence of iodine will turn yellow. The reaction mixture is then kept in a dark place (with no exposure to light) for 5 to 10 minutes to reach the optimal reaction time, before titrated with sodium thiosulfate using starch (2 ml) as the indirect indicator. The solution turned deep purple. Titration was continued until the purple coloration disappears and the solution became colorless. The concentration of iodine in salt is calculated based on the titrated volume (burette reading) of sodium thiosulfate according to the formula mentioned below.

Calculation:

$$\text{Iodine (ppm)} = \frac{\text{Titration volume in ml x 21.15 x normality of sodium thiosulfate x 1000}}{\text{Salt sample weight in gram}}$$

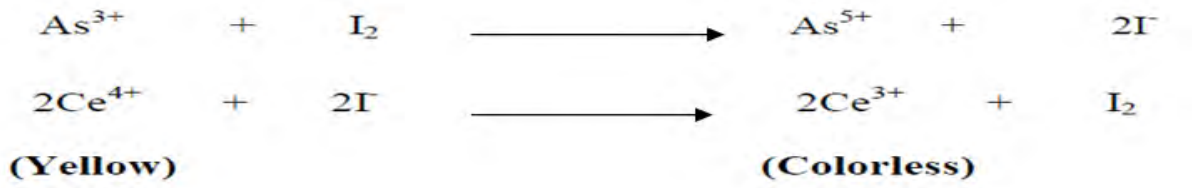
3.10.2. Urinary iodine determination

Urinary iodine was determined according to Dunn *et al.* (1993) by Sandell–Kolthoff reaction method using ammonium persulfate (Method A). This method used to analyze urinary iodine depend on the role of iodide as catalyst in the reduction of ceric ion (Ce^{4+}) to cerous ion (Ce^{3+}) coupled to the oxidation of arsenite (As^{3+}) to arsenate (As^{5+}). This reaction is known as the

Sandell–Kolthoff reaction. The color of the ceric ion is yellow, while the cerous ion is colorless. Thus, the course of the reaction can be followed by the disappearance of the yellow color as the ceric ion is reduced. The color disappearance is directly proportional to the amount of iodide catalyzing it if other reactants are held stable and iodine levels down to several nanograms can be detected. This specificity and high sensitivity has made the Sandell–Kolthoff reaction the basic method for the detection of iodine in urine (Dunn *et al*., 1993).

However, a digestion or ashing step is necessary to prepare urine samples by this method for the removal of interfering substances, such as nitrate and thiocyanate. Ammonium persulfate is considered to be one of the safe alternatives as the oxidizing agent to eliminate the interfering substances in the urine before the colorimetric measurement by the Sandell–Kolthoff reaction (Pino *et al.*, 1996).

Reaction for Sandell-Kolthoff reactions:



Procedure:

According to Sullivan *et al.* (2000) prior to analysis, the urine samples were put out of refrigerator a day before analysis and defrosted in order to reach room temperature. Then the urine samples were mixed until homogenization of the suspended sediment. Standards were prepared by pipetting 0, 10, 20, 40, 60, 100 and 250 µl of standard solution B in duplicate into 12 test tubes contained 250, 240, 230, 210, 190 and 150 µl of H_2O respectively, to give a volume of 250 µl in each tube. The standard curve was obtained with iodine concentrations- 0, 20, 40, 80, 120, 200 and 500 µg/L.

Urine sample of 250 µl was pipetted into 13x100mm test tube and 1 ml of solution of ammonium persulphate was added to each test-tube and mixed slowly. All the test-tubes was placed in a thermostatic block and heated for 1 hour at a temperature 100^oC. Then after digested the sample all test tubes was cooled to room temperature. 2.5 ml of arsenic acid solution was added to each

test-tube, mixed with a "Vortex" mixer and stayed for about 15 minutes. 300 µl of ceric ammonium sulphate were added to each test-tube at 30 second intervals between successive tubes, (which is observed with a stopwatch). Upon added to the solution, it was followed by mixing with the "Vortex" mixer. The sample was allowed to sit room temperature and after 30 minutes ceric ammonium sulphate solution was added to the first test-tube.

Absorbance (at 420 nm) for each sample was read using single-beam spectrophotometer in 30 sec interval. All successive tubes were read at the same intervals as added the ceric ammonium sulfate. Iodine concentration in urine was determined in the base of the value of absorbance plotted from the standard solution. Calibration curve was linear (Sullivan *et al*., 2000). Standard curve was constructed by plotted the log of the absorbance at 420 nm on the X-axis versus the standard iodine concentration in µg/l on the Y-axis with a scattered plot by using Excel on a desktop computer. Finally, iodine concentration in µg/l of each specimen was calculated by using the equation of the linear trend line of this chart.

3.11. Statistical analysis

Data processing and statistical analysis was performed by Statistical Package for the Social Sciences (SPSS) version 20 and Microsoft Excel 2010. The mean iodine content of salt and urine samples was calculated using Microsoft Excel. Furthermore, all data were manually entered in to SPSS for data analysis. Descriptive statistics such as means, median, range, standard deviations and percentage were calculated. In addition, chi-Square test was used to compare proportions of categorical variables and to test the statistically significance ($P<0.05$) between dependent and independent variables.

Moreover, the association between dependent variable and independent categorical variables was assessed by using logistic regression. Bivariant and multivariant analysis was used to describe the data according to some important characteristics of the study population. Multivariant logistic regression analysis was used to control confounding factor and show the effect of independent variables on dependent variables expressed by odds ratio.

Chapter four

4. Results

4.1. Socio-demographic characteristics of the study population

A total of 270 school children with equal proportion of boys 135 (50%) and girls 135 (50%) were participated in the study. Only 33.3% of families attended primary school and 32.2% attended secondary school. However, the remaining 24.9% of families didn't have formal education.

Table 4.1: Socio-demographic characteristics of children and their families in Akaki-kality subcity of Addis Ababa, 2015. (N= 270)

Socio-demographic variables	Frequency	Percent (%)
Sex of children		
Male	135	50.0
Female	135	50.0
Age of children (mean ± standard deviation)	9.5 ± 1.71 years	
Educational Status of Family Head		
Unable to read and write	8	3.0
Able to read and write	59	21.9
Primary school level (1-8)	90	33.3
Secondary school level (9-12)	87	32.2
Diploma and above level	26	9.6
Family head Employment Status		
Government/private employee	109	40.4
Self owned business	103	38.1
Daily laborer	44	16.3
Other	14	5.2
Monthly income of the family		
0-999	131	48.5
1000-1999	70	25.9
≥ 2000	69	25.6
Family size		
<5	152	56.3
≥ 5	118	43.7
Religion of family		
Orthodox	223	82.6
Muslim	29	10.8
Protestant	16	5.9
Catholic	2	0.7
Ethnicity of family		
Oromo	103	38.2
Amhara	110	40.7
Tigray	22	8.1
Others*	35	13.0

Others*- Gurage, Wolayita, Hadiya and Silete

4.2. Knowledge and practices of parents regarding iodine deficiency and iodized salt

Majority (72.6%) of respondents know the importance of iodine. On the subject of the consequence of iodine deficiency on health, majority (90.4%) of the respondents mentioned as it causes goiter. While, only 9.6% of the participants said iodine deficiency can reduce intelligence, cause cretinism, still birth and spontaneous abortion. In addition, majority (70.7%) of the respondents used iodized salt, while (26.7%) of the respondents didn't know type of salt used.

Table 4.2: Knowledge of families on iodine and iodine deficiency disorder in Akaki-kality subcity of Addis Ababa, 2015. (N=270)

Variables	Frequency	Percent (%)
Do you know the importance of iodine		
Yes I know	196	72.6
I don't know	74	27.4
Consequence of iodine deficiency		
Yes I know	219	81.1
I don't know	51	18.9
Health problem of iodine deficiency		
Goiter	244	90.4
Cretinism	14	5.2
Reduced intelligence	6	2.2
Stillbirth	2	0.7
Sudden abortion	4	1.5
Vulnerable group of population for iodine deficiency		
Children's	158	58.5
Pregnant women's	56	20.7
Men's	5	1.9
Female's	44	16.3
Others	5	1.9
I don't know	2	0.7
Iodine enriched food source		
Iodized salt	230	85.2
Meat and meat products	12	4.4
Sea foods	16	5.9
Milk and milk products	12	4.4
Others	0	0
Do you use iodized salt		
Yes I use	191	70.7
No am not use	7	2.6
I don't know	72	26.7
Why you use iodized salt		
Important for health	207	76.7
Food flavor	25	9.3
Other reason	10	3.7
I don't know	28	10.3

Table 4.3: Household consumption practices of iodized salt in Akaki-kality subcity of Addis Ababa, 2015. (N=270)

Variables	Frequency	Percent (%)
Type of salt used		
Fine salt (packed)	56	20.7
Coarse salt (non packed)	214	79.3
How do you check iodized salt when purchase		
Read the label on the pack	225	83.3
Asking the seller	29	10.7
Never checked	16	5.9
Where do you store salt in your home		
Cool and dry place	104	38.5
Moist area and near to fire	3	1.1
I don't know (anywhere)	163	60.4
Kind of container used for salt storage		
Container with cover	255	94.4
Container without cover	12	4.4
I don't know	3	1.1
Time of salt was added during food cooking		
At early of food cooking	21	7.8
Middle of cooking	78	28.8
Late at the end of cooking	171	63.3

4.3. Determination of iodine content of salt

A total of 50 salt samples were analyzed by using standard iodometric titration method. The high proportion of households 40 (80%) had inadequate amount of iodine (<15ppm) and only 10 (20%) of study population consumed adequately iodized salt (less than ≥15 ppm). Consumption of iodized salt in the study populations are shown in Figure 4.1.

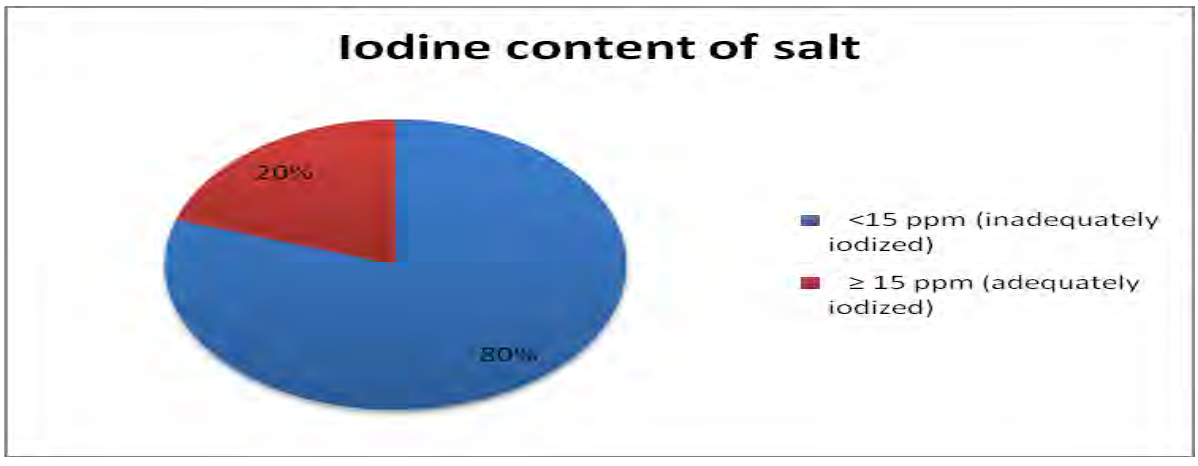


Figure 4.1: Iodine content of salt consumed in Akaki-kality subcity of Addis Ababa, 2015.

4.4. Urinary iodine excretion level

Totally 270 urine samples were analyzed by using Sandell–Kolthoff reaction method to determine urinary iodine level. Median UIC is the main indicator used to assess iodine status of a population. The median UIE level of children was found to be 85.7 µg/L. The proportion of children with UIE less than 100 µg/L was 62 % and less than 50 µg/L was of 20%. In addition, 14(5.2%) and 40(14.8%) of children's had in severe and moderate iodine deficiency respectively. While, 101(37.4%) of children in the study had 100 µg/L or above urinary iodine status. The distribution of urinary iodine values according to the epidemiological criteria for assessing iodine nutrition are shown in Figure 4.2.

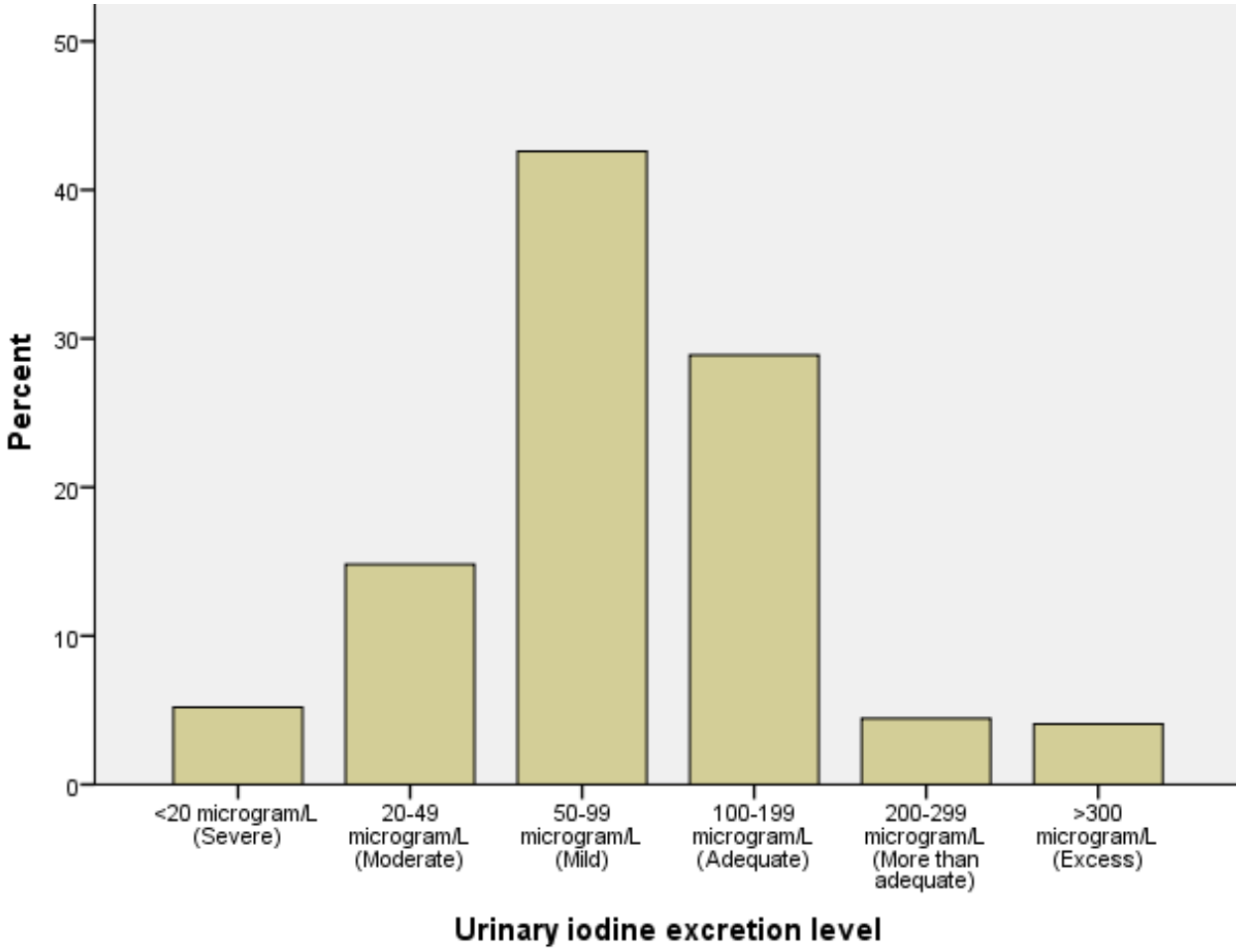


Figure 4.2: Distribution of urinary iodine status of SAC in Akaki-kality subcity of Addis Ababa, 2015.

In Table 4.4 provided that 90(66.9%) children in age 7 to 9 years had urinary iodine status less than 100 µg/L, whereas 79(58.5 %) of children in the age group 10-12 years had less than 100 µg/L.

Table 4.4: Age group based urinary iodine status of SAC in Akaki-kality subcity of Addis Ababa, 2015.

UIE	7-9 year (n=135)	10-12 year (n=135)	Total (%)
<20 µg/L	4 (3%)	10 (7.4%)	14 (5.2%)
20-49 µg/L	22 (16.3%)	18 (13.3%)	40 (14.8%)
50-99 µg/L	64 (47.4%)	51 (37.8%)	115 (42.6%)
≥ 100 µg/L	45 (33.3%)	56 (41.5%)	101 (37.4%)

UIE-Urinary Iodine Excretion

The median UIC of children based on their school such as in Atse-tewodros and Akaki-mengist primary schools had lower median UIC level than other schools in the study area. Even though, median UIC statuses of children in Eweket-wogagen, Kality-primary and Kality-bulbula primary schools were better than the two schools, all schools in the study area were below the recommended level (100 µg/L) and not statistically significant (p=0.074) indicated in Table 4.5.

Table 4.5: School based distribution of urinary iodine status of SAC in Akaki-kality subcity of Addis Ababa, 2015.

UIE	Akaki-mengist (n=51)	Atse-tewodros (n=57)	Eweket-wogagen (n=53)	Kality-bulbula (n=55)	Kality-primary (n=54)	Total (%)
<20 µg/L	2 (3.9%)	8 (14%)	2 (3.8%)	1 (1.8%)	1(1.9%)	14 (5.2%)
20-49 µg/L	6 (11.8%)	10 (17.5%)	6 (11.3%)	13 (23.6%)	5 (9.3%)	40 (14.8%)
50-99 µg/L	27 (52.9%)	25 (43.9%)	20 (37.7%)	16 (29.1%)	27 (50%)	115 (42.6%)
≥ 100 µg/L	16(31.4%)	14 (24.6%)	25 (47.2%)	25 (45.5%)	21 (38.9%)	101 (37.4%)
MUI (µg/L)	75.2	75.1	95.6	88.3	93.5	85.7

MUI-Median Urinary Iodine, UIE-Urinary Iodine Excretion

4.5. Goiter status of the study population

The assessment revealed that the TGR was 23.3% in 7-12 years children. In addition, the prevalence of goiter in this study was more prevalent in female (27.4%) than male (19.3%). Goiter statuses of children in the study area are shown in Figure 4.3.

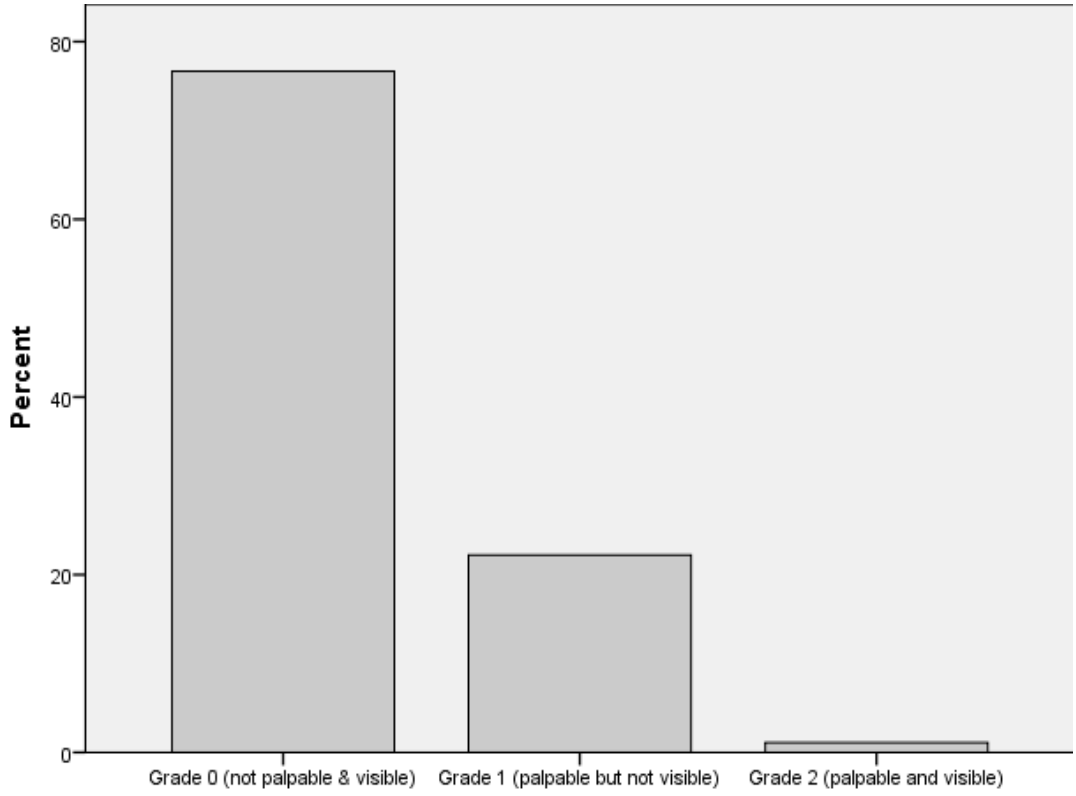


Figure 4.3: Goiter grade of SAC in Akaki-kality subcity of Addis Ababa, 2015.

Table 4.6: Sex wise distribution of goiter prevalence in Akaki-kality subcity of Addis Ababa, 2015.

Goiter grade	Male (n=135)	Female (n=135)	Total (%)
Grade 0	109(80.7%)	98(72.6%)	207 (76.7%)
Grade 1	26 (19.3%)	34 (25.2%)	20 (22.2%)
Grade 2	0(0%)	3 (2.2%)	3(1.1%)
TGR (%)	26 (19.3%)	37 (27.4%)	62 (23.3%)

TGR-Total Goiter Rate

Age group based prevalence of goiter in children whose age 10 to 12 years had TGR of 41(30.4%) which is by far higher than children in the age group 7 to 9 years had 22(16.3%) were presented in Table 4.7.

Table 4.7: Prevalence of goiter by age group in Akaki-kality subcity of Addis Ababa, 2015.

Goiter grade	7-9 year (n=135)	10-12 year (n=135)	Total (%)
Grade 0	113 (87.3%)	94 (69.6%)	207 (76.7 %)
Grade 1	21 (15.6%)	39 (28.9%)	60 (22.2%)
Grade 2	1 (0.7%)	2 (1.5%)	3 (1.1%)
TGR (%)	22(16.3%)	41(30.4%)	63(23.3%)

TGR-Total Goiter Rate

Prevalence of goiter in schools is presented in Table 4.8. The result showed that severe to moderate prevalence of iodine deficiency in Akaki-mengist, Kality-bulbula and Atse-tewodros primary schools. Whereas, goiter prevalence in Kality-primary and Eweket-wogagen primary schools were revealed that mild iodine deficiency problem in the study area. Prevalence of goiter is statistically significant (p=0.007) with schools in the subcity.

Table 4.8: School based prevalence of goiter in Akaki-kality subcity of Addis Ababa, 2015.

Goiter grade	Akaki-mengist (n=51)	Atse-tewodros (n=57)	Eweket-wogagen (n=53)	Kality-bulbula (n=55)	Kality-primary (n=54)	Total (%)
Grade 0	32 (62.7%)	43 (75.4%)	45 (84.9%)	39 (70.9%)	48 (88.9%)	207 (76.7%)
Grade 1	16 (31.4%)	14 (24.6%)	8 (15.1%)	16 (29.1%)	6 (11.1%)	60 (22.2%)
Grade 2	3 (5.9%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	3 (1.1%)
TGR (%)	37.3%	24.6%	15.1%	29.1%	11.1%	63 (23.3%)

TGR-Total Goiter Rate

4.6. Factors associated with urinary iodine level

Urinary iodine status of the children with inadequate (<100 μg/L) and adequate (≥100 μg/L) level were tested for their association with independent variables in binary logistic analysis. Children whose age group 7 to 9 years had Crude Odds Ratio (COR=2.1; 95%CI=1.1, 4.3), family did not follow formal education had (COR=2; 95%CI=1.2, 3.3) and children's which consumed coarse salt had (COR=303.2; 95%CI=38.7, 2377) were highly associated with inadequate iodine level of the children.

All variables that have association at significance level of ($p < 0.05$) with outcome variable in the bivariate analysis were included in the multivariate regression model. Children's in age group 7 to 9 years had Adjusted Odds Ratio (AOR=2.2; 95%CI=1.1, 4.3) and (p=0.028), educational level of family (AOR=4.1; 95%CI=1.4, 11.8) and (p=0.09), and coarse salt consumed children's (AOR=308.4; 95%CI=39.2, 2429.2) and (p=0.000) had independent association with children's iodine level. Children whose family head did not attend formal education were 4.1 times more likely to have inadequate iodine level than those family head attended formal school. Children whose age group had 7 to 9 years were 2.2 times more likely to be lower iodine level than 10 to 12 years age group. Children's who consumed coarse salt had 308.4 times more likely to have iodine deficiency than children's consumed fine salt. The likelihood of lower iodine statuses of children with some independent variables are shown in Table 4.9.

Table 4.9: Association of independent variables with urinary iodine status of SAC by logistic regression in Akaki-kality subcity of Addis Ababa, 2015.

Variables	Urinary iodine status		Odds ratio		p-value
	< 100 µg/L	≥ 100 µg/L	COR (95% CI)	AOR (95% CI)	
Sex					
Male	81 (60%)	54 (40%)	1	1	
Female	88 (65.2%)	47 (34.8%)	0.894(0.456, 1.750)	0.891(0.454, 1.748)	0.737
Age (year)					
10-12	79 (58.5%)	56 (41.5%)	1	1	
7-9	90 (66.7%)	45 (33.3%)	2.148(1.081, 4.271)	**2.169(1.086, 4.328)***	**0.028**
Educational status of family					
Secondary school & above	69 (61.1%)	44 (38.9%)	1	1	
Primary school level	56 (62.2%)	34 (37.8%)	0.526(0.225, 1.229)	1.913(0.818, 4.478)	0.135
Illiterate	44 (65.7%)	23 (34.3%)	1.993(1.187, 3.346)	**4.075(1.409, 11.78)****	**0.009**
Monthly family income					
≥ 1000 birr	92 (66.2%)	47 (33.8%)	1	1	
< 1000 birr	77 (58.8%)	54 (41.2%)	0.556(0.266, 1.162)	0.560(0.265, 1.185)	0.130
Type of salt used					
Fine salt (packed)	1 (1.8%)	55 (98.2%)	1	1	
Coarse salt (non packed)	168 (78.5%)	46 (21.5%)	303.3(38.69, 2377)	**308.4(39.2, 2429.2)****	**0.000**
Salt container used					
Container with cover	160 (62.7%)	95 (37.3%)	1	1	
Container without cover	9 (60%)	6 (40%)	1.152(0.243, 5.459)	1.120(0.231, 5.427)	0.888
Time of salt add					
Lately at the end of cooking	103 (60.2%)	68 (39.8%)	1	1	
Middle of cooking	50 (64.1%)	28 (35.9%)	0.854(0.394, 1.851)	1.171(0.540, 2.537)	0.966
Early at the beginning	16 (76.2%)	5 (23.8%)	0.975(0.294, 3.228)	1.026(0.310, 3.396)	0.690

*statistically significance at p<0.05, ** significance at p<0.01, COR-Crude Odds Ratio, AOR-Adjusted odds ratio

4.7. Underlying factors for goiter

Table 4.10 presents children with goiter and without goiter were compared with different variables. Results of bivariate logistic regression analysis showed that children's group of age 10 to 12 years were significantly associated with presence of goiter (COR=2.5; 95%CI=1.3, 4.6) as compared to age group 7 to 9 years. Children's study in Akaki-mengist primary school had higher likelihood of developing goiter (COR=3.6; 95%CI=1.3, 9.8) than other schools in the study area. Other variables such as sex, educational status of parent's, monthly income of family, family size, use of iodized salt and type of salt used were found to had no significant association with goiter in the bivariate analysis. Variables which were identified to have significant association (at significance level of 0.05) with goiter in the bivariate regression model were entered in to stepwise forward multivariate regression.

In multivariate logistic regression model, children's in the age group 10 to 12 years had (AOR=2.6; 95%CI=1.4, 4.8) and (p=0.003), and children's who study in Akaki-mengist primary school (AOR=3.8; 95%CI=1.4, 10.1) and (p=0.009) had independent association with goiter. Children's in the age group 10 to 12 years had 2.6 times more likely to have goiter than children in the age group 7 to 9 years.

Table 4.10: Association of independent variables with goiter status of children by logistic regression in Akaki-kality subcity of Addis Ababa, 2015.

Variables	Goiter status		Odds ratio		p-value
	Yes	No	COR (95% CI)	AOR (95% CI)	
Sex					
Male	26 (19.3%)	109 (80.7%)	1	1	
Female	37 (27.4%)	98 (72.6%)	0.572(0.310, 1.055)	1.773(0.959, 3.276)	0.068
Age (year)					
7-9	22 (16.3%)	113 (83.7%)	1	1	
10-12	41 (30.4%)	94 (69.6%)	2.472(1.333, 4.585)	**2.556(1.369, 4.773)****	**0.003**
Primary school					
Eweket-wogagen	8 (15.1%)	45 (84.9%)	1	1	
Kality-primary	6 (11.1%)	48 (88.9%)	0.683(0.212, 2.198)	0.706(0.218, 2.285)	0.561
Kality-bulbula	16 (29.1%)	39 (70.9%)	2.418(0.876, 6.674)	2.528(0.907, 7.047)	0.076
Atse-tewodros	14 (24.6%)	43 (75.4%)	2.032(0.735, 5.613)	2.158(0.770, 6.049)	0.144
Akaki-mengist	19 (37.3%)	32 (62.7%)	3.643 (1.358, 9.772)	**3.751 (1.390, 10.122)****	**0.009**
Educational status of family					
Secondary school & above	24 (21.2%)	89 (78.8%)	1	1	
Primary school level	22 (24.4%)	68 (75.6%)	1.374(0.656, 2.881)	1.374(0.656, 2.881)	0.400
Illiterate	17 (25.4%)	50 (74.6%)	1.1(0.481, 2.517)	1.1(0.481, 2.517)	0.822
Monthly family income					
≥ 2000 birr	16 (23.2%)	53 (76.8%)	1	1	
< 2000 birr	47 (23.4%)	154 (76.6%)	1.070(0.500, 2.289)	0.916(0.427, 1.965)	0.822
Family size					
<5	35 (23%)	117 (77%)	1	1	
≥ 5	28 (23.7)	90 (76.3%)	1.042 (0.562, 1.932)	0.950 (0.512, 1.762)	0.870
Use iodized salt					
Yes	42 (22%)	149 (78%)	1	1	
No	21 (26.6%)	58 (73.4%)	0.732(0.379, 1.417)	1.373(0.709, 2.658)	0.348
Type of salt used					
Iodized salt (packed)	11 (19.6%)	45 (80.4%)	1	1	
Coarse salt (non packed)	52 (24.3)	162 (75.7%)	0.951(0.436, 2.072)	1.059(0.486, 2.308)	0.886

** Statistical significance at p<0.01, COR-Crude Odds Ratio, AOR-Adjusted odds ratio

Chapter five

5. Discussion

5.1. Iodine content of salt

Salt iodization is by far the most important population based intervention for IDD control and has been shown to be efficacious in alleviating IDD assuming iodine concentrations in the salt are at appropriate levels at the time of consumption (Emel *et al.,* 1999). According to the present study only 20% of households had used adequately iodized salt. As compared to similar studies in our country, this finding was somewhat higher than study conducted by Kibatu *et al*. (2014) in Benishngul-Gumuz Metekel zone was 10%, due to the implementation of salt iodization program. Besides, this finding also better than similar studies in Sudan and in India districts were 14.4%, 12.6% and 18.2% respectively (Mahfouz *et al*., 2012; Chandra *et al*., 2008; Pandav *et al*., 2012).

However, still the household consumption of adequately iodized salt in this study was lower than similar studies conducted in different region of our country such as in Gondar town, Bale Goba town, Assosa town and Shebe-senbo district of Jimma was 33%, 29.7%, 26.1%, and 26.2% respectively (Gidey *et al*., 2015; Enyew *et al*., 2015; Gebriel *et al*., 2014; Mezgebu *et al*., 2012). In addition, the consumption of iodized salt was lower than other countries studies in Kenya, Nigeria, and Lesetho were 83.3%, 91% and 94.4% respectively (Kishoyian *et al*., 2014; Kunle and Olanrewaju, 2014; Sebotsa *et al*., 2003).

Similarly, in different district of India the iodized salt consumption status was greater than 90% (Singh *et al*., 2015; Sridhar and Kamala, 2014; Biswas *et al*., 2014; Chandwani and Pandor, 2011; Rawal and Kedia, 2011). In Albania and Nepal were also 71.2% and 82.2% respectively (Kaidu *et al*., 2015; Nepal *et al*., 2013). This might be due to availability and accessibility of iodized salt in the market, legislation and policies to fortify salt with iodine, and regular follow up and monitoring regarding utilization of iodized salt in these countries were well established.

Majority (79.3%) of the study population used coarse salt rather than fine salt (packed). A study conducted in Canada showed that iodine content of the salt remained constant and its distribution remained uniform for many months when the salt was packed and kept dry, preferably in a cool

place and away from strong light (Davidson *et al*., 2005). This may be due to coarse salt are crystalline in nature; they are not homogenously iodized with iodine, so that they can't get uniform amount of iodine in the salt.

Furthermore, 61.5% of families participated in this study stored salt anywhere in their home because they don't know iodine is sensitive to heat and moisture. Salt not exposing to sunlight was one of the factors significantly associated with availability of adequately iodized salt. A study conducted in Delhi revealed that there was about 31% iodine loss from iodized salt when exposed to sunlight (Kapil *et al*., 1998). During transportation and storage the salt are exposed to heat and moisture, it can easily loss its iodine content.

The successful application of USI in combating IDD in a country requires adequate supplies and regular monitoring at the production and consumption levels. Universal salt iodization has to go with laboratory development, research, capacity building, advocacy and public education on IDD. Commitment of health professionals and researchers to the assessment and reassessment of the progress in iodine nutrition status is critical for tracking progress towards sustainable elimination of iodine deficiency disorders (Kapil, 2012). Moreover, in order to implement effective USI program in one country they should practice the activities in the community of five As: Awareness, Availability, Accessibility, Acceptability, and Affordability (Sabeeb and Ali, 2014).

5.2. Urinary iodine status of children

For populations, because it is impractical to collect 24 hour urine samples in field studies, UIC can be measured in spot urine specimens from a representative sample of the target group and expressed as the median UIC. Variations in hydration among individuals generally even out in a large number of samples, so that the median UIC in spot samples correlates well with that from 24 hour samples (Zimmermann, 2009). The median UIC in SAC has been used to approximate the iodine status of the general population in countries where salt is the primary vehicle for iodine, because SAC are convenient population group that is easily to reach through school based surveys. Therefore, UIC from 6 to 12 years children in nationally representative survey is used to classify a population's iodine status (Andersson *et al*., 2012).

The finding of this study revealed that MUI concentration of children was 85.7 μg/L, this finding indicated that the study area was mildly affected by iodine deficiency. While, this value is better than previous studies in different region of our country such as nationwide study in 2005, Shebe-senbo district of Jimma, Hawassa town, Burie and Womberma district of west Gojjam, and Metekel zone was 24.5 μg/L, 56 μg/L, 34.2 μg/L, 50 μg/L and 39.9 μg/L respectively (Abuye *et al*., 2007; Mezgebu *et al*., 2012; Girma *et al*., 2012; Aweke *et al*., 2014; Kibatu *et al*., 2014).

Besides, this finding was better than other countries studies in Malaysia, Gouno-Lufa district of Papua New Guinea, New Zealand and India were 47.8 μg/L, 50 µg/L, 68 μg/L and 80 μg/L respectively (Kuay *et al*., 2012; Temple *et al*., 2013; Skeff *et al*., 2012; Makwana *et al*., 2012). So that, the progress in MUI level may be due to the implementation of national salt iodization program in the last three years which is the most effective strategies to improve the iodine deficiency disorder at the household level.

Even though there was an improvement in MUI level than the previous studies, yet this finding was lower than similar studies in other countries such as in Kenya, Nigeria, Malaysia, Romania and in different district of India were 242.2 µg/L, 284.75 µg/L, 109 µg/L , 175.2 µg/L and 125 µg/L, 115 µg/L, 175 µg/L respectively (Kishoyian *et al*., 2014; Augustine *et al.,* 2012*;* Selamat *et al*., 2010; Ursu *et al*., 2014; Kapil *et al*., 2014; Damor *et al*., 2013; Chander *et al*., 2013).

Nevertheless, there was significant association between iodine status of children and their age group 7 to 9 years. The likelihood ratio of children with inadequate iodine status in age group 7 to 9 years was increased by 2.1 times than children 10 to 12 years (AOR=2.1; 95%CI=1.1, 4.2). Moreover, the educational status of family head and those families consumed coarse salt were highly associated with inadequate iodine status of the children. The likelihood of inadequate iodine status in children with illiterate family than following formal education and families who consumed coarse salt than fine salt were 4.1 and 308 times more likely to be iodine deficient children (AOR=4.1; 95%CI=1.4, 11.8) and (AOR=308.4; 95%CI=39.2, 2429.2) respectively.

5.3. Goiter prevalence in children

The overall prevalence of goiter in this study was 23.3%, which revealed that IDD is still a moderate public health problem in the study area. Others similar studies conducted in different

region of our country showed that higher prevalence of goiter in SAC than this study finding. Studies in Assosa town was 26.3%, in Lay Armachiho district of Gondar was 37.6%, in Metekel zone was 39.5%, in nationwide study 39.9%, in Goba town was 50.6%, in Burie and Womberma district of west Gojjam 54 % and in Shebe-Senbo district of Jimma zone was 59.1% (Gebriel *et al*., 2014; Mesele *et al*., 2014; Kibatu *et al*., 2014; Abuye *et al*., 2007; Enyew *et al*., 2015; Aweke *et al*., 2014; Mezgebu *et al*., 2012).

In addition, similar studies in different districts of India revealed that high TGR in SAC was 23.6% in urban slums of Bhubaneswar, 28.4% in Churachanpur of Manipur, and 30.2% in eastern Uttar Pardesh of India (Sethy *et al*., 2007; Singh *et al*., 2015; Chandra *et al*., 2008). The high TGR found in different region of the countries corroborate with the finding of the present study. All this finding signifies that IDD is still not only the problem of Akaki-kality subcity of Addis Ababa but also throughout the country severe public health problem.

According to national wide study conducted by Abuye *et al.* (2007) the prevalence of goiter in SAC of Addis Ababa was 19.3% and the national prevalence was 39.9%. The prevalence of goiter in this study was found to be 23.3% which is higher than the previous study; still the prevalence of goiter in the last decades has increased. This may be due to the study area is high land and poor soil conservation contributed to leaching of the iodine rich top layer soil and the exposition of poor layer beneath. The crops grown in iodine deficient soils are poor in iodine content. Hence the leaching away of iodine in the top layer of the soil may have contributed for high prevalence of goiter in high altitudes than in low land areas (Abuye *et al.,* 2008).

Furthermore, there was no national salt iodization program before three years so the communities were used bare salt. In 2012 Ethiopia launched the national salt iodization program with proclamation, but still not stringent control system was implemented by the regulatory authority to improve the availability of adequately iodized salt at production and retail level. Moreover, there was no collaboration effort in the dissemination of information to sensitize and create awareness about the importance of iodized salt for health by different stakeholder.

Contrary, the prevalence in the present study was higher than the recommendation and other similar studies conducted in Bellur-hobil district of India was (0.1%), Sarawak of eastern

Malaysia was (2.9%) and in Nairobi city of Kenya was (3.5%) (Sirdhar and Kamala, 2014; kuay *et al*., 2014; Kishoyian *et al*., 2014). In addition, similar findings were observed in other countries such as in South Africa was (5.9%), in Romania was (5.8%) and in Saudi Arabia was (7.4%) where IDD elimination programs have entered in the phase of sustainability (Mabapa *et al*., 2014; Omar and Desouky, 2015; Ursu *et al*., 2014). In these countries it may be due to better control of IDD through universal salt iodization.

According to this study the prevalence of goiter was found to be more prevalent in females (27.4%) than in males (19.3%), but there was no statistically significant difference between genders on goiter prevalence in the studied SAC. This finding is in agreement with studies conducted in different regions of Ethiopia (Cherinet and Kelbessa, 2000; Berhanu *et al*., 2004; Girma *et al*., 2012; Wolka *et al*., 2014; Enyew *et al*., 2015). And also other similar studies for both male and female in different district of India also were 7.2% and 21.8% in Belgaum, 7.4% and 15.7% in Jodhpur and 6.8% and 19.9% in Lucknow respectively (Kamath *et al*., 2009; Singh *et al*., 2010; Gupita *et al*., 2015).

The possible explanations for females are the more vulnerable group of population is because of physiological reasons such as early puberty, which starts about 2 years earlier than males. As a result their iodine demand is higher than males. In addition, estrogen, which is a female hormone, has a well-known inhibitory effect on iodine uptake by thyroid follicular cells. Beside, estrogen also increases thyroid follicular proliferation, leading to thyroid gland enlargement (Furlanetto *et al*., 2001).

On analyzing the association of goiter prevalence with children in age group 10-12 was found to be higher goiter rate 30.4% than age group 7-9 years had 16.3%. Children in the age group 10-12 years were highly associated goiter with the likelihood of occurring goiter increased by 2.6 times (AOR=2.6; 95%CI=1.4, 4.8). This result was consistent with another study conducted in Bale-Goba town (53.7%) that for 9-12 years age group and (39 %) for 6-8 years age group (Enyew *et al*., 2015).

Likewise, similar pattern was found in a study by Bhat *et al*. (2008) 12.8% in 9 to 12 years old children as against 10.6% in 6 to 8 years (Bhat *et al*., 2008). In a study in Kottayam also revealed

that a higher prevalence was noted in older children being 12.3% in 10 to12 years as compared to younger children 7-9 years (Ramesh *et al*., 2013). A similar finding was noted in a study conducted in Bhubaneswar which showed significantly high prevalence of goiter in children aged from 10 to 12 years compared with those aged between 6 and 8 years (Sethy *et al*., 2007).

This relationship between age and the prevalence of goiter may be attributed to the increased demand for thyroid hormones during puberty. Another reason could be the long standing iodine deficiency manifesting ultimately as enlargement of thyroid gland in older age groups. Moreover, children learned in the Akaki-mengist primary school were highly associated with goiter, with the likelihood of occurring goiter was increased by 3.8 times (AOR=3.8; 95%CI=1.4, 10.1). This is may be due to the school where found in away from the main road and which is semi-urban area, the community may have less access to get iodized salt and the awareness on the importance of iodized salt also very minimal.

Finally, the above findings of the present study conducted in Akaki-kality subcity of Addis Ababa needs to be looked into in totality with goal of IDD elimination. WHO/UNICEF/ICCIDD (2007) has recommended indicators to determine whether IDD exists as a public health problem in the surveyed region or area.

Table 5.1: Criteria for tracking progress towards eliminating IDD

Indicator	Goal	Akaki-kality subcity
Thyroid size (age 6-12 years)		
Proportion with enlarged thyroid	< 5 %	23.3%
Urinary Iodine		
Median urinary iodine µg/L	> 100 µg/L	85.7 µg/L
Proportion below 100 µg/L	< 50 %	62%
Proportion below 50 µg/L	< 20 %	20%
Salt iodization:		
Proportion of households consuming adequately iodized salt	> 90%	20%

As per these criteria, the impact indicators on SAC in Akaki-kality subcity of Addis Ababa were revealed that TGR of 23.3%, which indicates the prevalence of few years with moderate IDD in children. Similarly, the status of iodine nutrition was shown by the MUI excretion 85.7 µg/L. It indicates that currently there was mild iodine deficiency in the study area. Moreover, the impact indicator showed the consumption status of adequately iodized salt at the household level was 20%. Finally, all these findings signify that in Akaki-kality subcity iodine deficiency is still severe public health problem. The subcity should work in collaboration with other stakeholder in order to significantly improve the availability of adequately iodized salt and to enhance the awareness of the community about the importance of iodized salt for health.

Chapter six

6. Conclusion and recommendations

6.1. Conclusion

According to the WHO/ICCIDD/UNICEF criteria, the finding of this study revealed that the study area was moderately affected by goiter. In addition, the relative proportion of goiter was higher among female children than male children and the prevalence of goiter also increased as age increases. There was significant association of the goiter status of children with increasing age and their area of school found.

MUI concentration of SAC was found to be 85.7 μg/L and also the proportion of children which urinary iodine less than 100 μg/L was 62.6% and less than 50 μg/L was 20%. Thus, all these findings confirmed that presence of mild iodine deficiency in the study area. The age of children, educational status of family and those children who consumed coarse type of salt was highly associated with inadequate urinary iodine status of the children.

The evidence of the current survey also shows that high proportions of household in the study area were consuming inadequately iodized salt. The availability of adequately iodized salt at household level is substantially lower than WHO recommendation to prevent IDD. The results also illustrate the availability of adequately iodized salt in subcity was very low. Besides, the majority of families don't know that iodine is sensitive to heat and moisture.

6.2. Recommendations

Iodine deficiency is a severe public health problem in Akaki-kality subcity of Addis Ababa. The regulatory authority should appropriately implement legislation of national salt iodization program and work with different stakeholders to reduce IDD problems in the population. They should also ensure sufficient supply of adequately iodized salt throughout the country by giving support to salt producers. Distribution of adequately iodized salt through public distribution system can ensure the regular availability of iodized salt with affordable price to access the community easily. So that, it is better to integrate with others basic need commodities supplied by the public facilities to the communities with affordable price.

Households consuming adequately iodized salt are far below the recommended level, indicating weakness in the implementation of national salt iodization program. There should be continuous health education activities by using mass media and health extension workers by house-to-house visits in order to improve the awareness of the community about the impact of IDD and advocate the utilization of iodized salt for the prevention of IDD problems. It is imperative to have a vigilant regulatory mechanism to ensure that the availability of adequately iodized salt at retail level and household level. Finally, perform periodic survey to estimate the prevalence of iodine deficiency disorders in the study area for evaluating the impact of the ongoing salt iodization program.

Further studies on larger population sizes may be needed to confirm the goiter status of children and difference on academic performance. The common belief that goiter is the only consequence of iodine deficiency needs to be corrected and the effects of iodine deficiency on brain development such as cretinism and reduced intelligence, and its subsequent effects on productivity and economic development need to be understood by the general public.

Further studies on the effect of goiterogenic factors on the absorption of iodine and other coexisting micronutrient deficiencies such as iron, zinc, selenium and vitamin A on thyroid hormones synthesis in iodine deficient SAC should be study.

Strengths and limitations of the study

In this study both process and impact indicators for evaluating iodine nutrition were done. The process indicator indicated that the current iodine content of salt consumed by the study population. Beside, the impact indicators such as MUI level revealed the recent iodine status of children and goiter prevalence also showed the long term effect of iodine deficiency on SAC. Therefore, the finding of this study clearly showed the status of iodine deficiency in the study area.

However, the effect of goiterogenic factors on iodine deficiency in the study population was not studied in this research. Subsample of salt was analyzed due to logistic problem, so that it is not sufficient sample size to show the consumption status of iodized salt in the study area.

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Appendixes

Appendix I: Assent form

Title: Iodized salt consumption and goiter status of school children in schools of Akaki-kality Subcity of Addis Ababa.

Purpose: The purpose of this study is to determine iodine status, goiter status and iodized salt consumption of school children 6-12 years in Akaki-kality subcity of Addis Ababa. The study will help you in giving knowledge and awareness of iodine status of your children and also create awareness on consumption of iodized salt.

Procedures

If your children's agree to participate in the study, we will ask children about awareness consumption of iodized salt; will also ask to examine children goiter status by health professional. In addition, the children also ask to give 5ml of urine sample and bringing teaspoon of salt sample from their houses.

Risks: from the assessment of iodine status of the school children, there is no risk other than students will contribute their break time.

Benefits: this study will provide baseline information for next plan and other stakeholder in the sector. Based on this finding it will provide both technical and in-kind support school children. Moreover, the study is important to evaluate the iodized salt consumption and iodine status of school children, so as to show the progress and problems in iodine nutrition.

Cost: there is no cost to you for participating in this research.

Compensation: there will be no financial compensation for participation in the study.

Participant Rights: if I have said any things that are not clear to you, you may ask without hesitation and I will answer. Your participation in the study is entirely voluntary.

Confidentiality: the study results and any information about your children will be kept confidential. Only the research team will have access to your children's information. When I write a report, everyone's information will be put together so that information about your children's cannot be seen because your children's will be coded.

Persons to contact:

If you have any questions, you can ask any time. If you have additional questions or any other concern about the study, you may contact:

Solomon Emiru**;** Phone Number 09 41945410 and email: (emirusolomon@gmail.com)

If you agree your children's to participate in the study, please sign at the space provided below. And I thank you for your cooperation. The study has been explained to me and my questions have been answered to my satisfaction. I agree to participate my children's in this study.

_____	_____	_____
Parent's Name	Signature	Date
_____	_____	_____
Name of researcher	Signature	Date

Student's identification code _____

የስምምነት መግለጫ ቅጽ

ርዕስ፡- በተመረጡ የአዲስ አበባ አቃቂ ቃሊቲ ክፍለ ከተማ አንደኛ ደረጃ ት/ቤቶች አዮዱን የተዋሀደበት ጨው አመጋገብና እንቅርት ያለበት ደረጃ ለማወቅ የሚደረግ ጥናት፡፡

ጥቅም፡ የዚህ ጥናት ጥቅም እድሜያቸው ከ6 እስከ 12 አመት የሆናቸው ልጆች በሰውነታቸው ውስጥ የሚገኘውን የአዮዲን መጠን ለማወቅና የእንቅርት ደረጃ ለመለየት እንዱሁም የሚመገቡት ጨው ምን ያህል አዮዲን የተዋሀደበት ጨው በሚፈለገው መጠን መኖሩን ለማወቅያስችላል፡፡በተጨማሪም ጥናቱ ለሌጆቹ ስለአዮዱን ንጥረ ነገር ለጤና ያለውን አስፈላጊነት እንዲረዱ ያደርጋል፡፡

የሰራ ቅደም ተከተል፤

በዚህ የምርምር ስራ ላይ ልጅዎ እንዲሳተፍ ፈቃደኛ ከሆኑ፤

- ሰለአዮዱን የተዋሀበደት ጨው ያለዎትን እውቀትና የአጠቃቀም ሁኔታ ለማወቅ
- ልጅዎ ያለበትን የዕንቅርት ደረጃ ለማወቅ በጤና ባለሙያ ምርመራ እንዲያደርጉ እንጠይቃለን
- እንዱሁም 5 ሚሊ ሊትር ሽንት ናሙና እንዲሰጡን እና
- አንድ የሻይ ማንኪያ ጨው ከቤታቸው እንዲያመጡ እንጠይቃለን፡፡

የአደጋ መጠን

ጥናቱ የተማሪዎቹን የእረፍት ጊዜ ከመጠቀም ውጭ በተማሪዎቹ ላይ ምንም አይነት ችግር አያስከትልም፡፡

ከምርምሩ የሚገኙ ጥቅሞች

ከዚህ ጥናት ሊገኝ የሚችለው ጥቅም በጥናቱ የሚገኘው መረጃ በቀጣይ ለሚታቀዱ እቅዶች እንደመነሻ የሚያገለግሉ ሲሆን በዚህ ዙሪያ ለሚሰሩ ባለድርሻ አካላትም መረጃ ይሰጣል፡፡በተጨማሪም አዮዲን የተዋሀደበት ጨው አመጋገብ እና የልጆቹ በሰውነታቸው ውስጥ ያለው የአዮዲን ንጥረ ነገር ያለበትን ደረጃ ለማሳየት ለቀጣይ ትኩረት ተደርጎ ሊሰራ የሚገባውን ችግር ያሳያል፡፡

ክፍያ

በዚህ ምርምር ላይ መሳተፍ ምንም አይነት የገንዘብ ክፍያ አይጠይቅም፡፡

ማካካሻ: በዚህ የምርምር ስራ ላይ መሳተፍ ምንም አይነት ገንዘብ አያስገኝም፡፡

የተሳታፊዎች መብት

በምርምሩ ስራ ላይ መሳተፍ ሙሉ በሙሉ በእናነተ ፈቃደኛነት ላይ የተመሰረተ ሲሆን በዚህ ምርምር ስራ ወቅት ግልፅ ያልሆነ ነገር ካለ ያለምንም ማመንታት እና በነፃነት መጠየቅ ይቻላል፡፡

ሚስጥራዊነት

ማንኛውም የልጅዎ መረጃ እና የምርምሩ ውጤቶች በሚስጥር የሚያዝ ሲሆን ተመራማሪው ጋር ብቻ ይገኛል፡፡ በተጨማሪም ሪፖርቶች ሲፃፉ መረጃዎቹ ሌላ ሰው እንዳያገኛቸው በተለየ ስም (ኮድ) ይቀመጣሉ፡፡

ማንኛውም አይነት ጥያቄ ካለዎት የሚከተለውን አድራሻ ይጠቀሙ፡፡

ሰለሞን እምሩ ስልክ ቁጥር 0941945410(emirusolomon@gmail.com)

በዚህ የምርምር ስራ ላይ ልጅዎ እንዲሳተፍ ፈቃደኛ ከሆኑ እባክዎ በክፍት ቦታው ላይ ይፈርሙ፡፡ ስለትብብርዎ እናመሰግናለን፡፡

ስለምርምሩ በበቂ ሁኔታ ተገልፆልኛል እንዲሁም ጥያቄዎቼ በሚገባ ተመልሰዋል፡፡ ስለዚህ ልጄ በዚህ ምርምር ላይ እንዲሳተፍ ተስማምቻለሁ፡፡

_____	_____	_____
የወላጅ ስም	ፊርማ	ቀን
_____	_____	_____
የተመራማሪው ስም	ፊርማ	ቀን

የተማሪው መለያ ቁጥር _____

Appendix II: Questionnaires

Instruction: For parents or caregiver form the listed question answer by circle in code column

Part 1 :- Sociodemographic characteristics of participants			
Sr No	**Variables**	**Multiple choice**	**Answer code**
101	Sex of children	1. Male	0
		2. Female	1
102	Age of children in year		
103	Children school grade		
104	What is the educational status of family head?	1. Unable to read and write	0
		2. Able to read and write	1
		3. Primary education (grade 1-8)	2
		4. Secondary education (grade 9-12)	3
		5. Diploma and above level	4
105	What is the occupation of family head?	1. Government/private employee	0
		2. Self-owned business	1
		3. Daily laborer	2
		4. If other (Specify) _____	3
106	How much is the monthly income of family?	1. 0 - 999 Birr	0
		2. 1000 - 1999 Birr	1
		3. $\geq$ 2000 Birr	2
107	Household family size	1. < 5	0
		2. $\geq$ 5	1
108	Ethnicity of Children?	1. Oromo	0
		2. Amhara	1
		3. Tigray	2
		4. If Other (specify) _____	3
109	Religion of the family?	1. Orthodox Tewahedo	0
		2. Muslim	1
		3. Protestant	2
		4. Catholic	3

Part 2: Knowledge on iodine deficiency and practices of iodized salt consumption of parents			
Sr No	**Variables**	**Multiple choice**	**Answer code**
201	Do you know the importance of iodine?	1. Yes I know	0
		2. I don't know	1
202	Do you know the consequence of iodine deficiency on human health?	1. Yes I know	0
		2. I don't know	1
203	(If your answer on question 202 yes) which health problem do you know?	1. Goiter	0
		2. Cretinism	1
		3. Reduced intelligence	2
		4. Stillbirth	3
		5. Sudden abortion	4
204	Which group of population more vulnerable for iodine deficiency ?	1. Children's	0
		2. Pregnant women's	1
		3. Men's	2
		4. Female's	3
		5. If other (Specify) _____	4
205	Which iodine enriched foods do you know?	1. Iodized salt or oil	0
		2. Meat and meat products	1
		3. Sea foods	2
		4. Milk and milk Products	3
		5. If other (specify) _____	4
206	Do you use iodized salt?	1. Yes I use	0
		2. No am not use	1
		3. I don't know	2
207	(If your answer on question 206 Yes I use it) why you use iodized salt?	1. improtant for health	0
		2. For food flavor	1
		3. If other (specify) _____	2
		4. I don't know	3

208	Which type of salt do you use?	1. Packed	0
		2. Non packed (coarse)	1
		3. I don't know	2
209	How do you check salt is it iodized salt when you purchase?	1. Read the label on pack	0
		2. Asking the seller	1
		3. I don't know	2
210	Where do you store salt in your home?	1. Cool & dry place	0
		2. Moist area & near to fire	1
		3. I don't know (any where)	2
211	What kind of container do you use to store salt?	1. Container with cover	0
		2. container without cover	1
		4. I don't know	2
212	At what time salt was added during food cooking?	1. At early of food cooking	0
		2. At the middle of food cooking	1
		3. Late at the end of cooking	2
		4. I don't know	3

"we have finished our interview thank you for your participation "

ለወላጆች የቀረበ መጠየቅ

መመሪያ፡ ለወላጆች ወይም አሳዳጊዎች ከዚህ በታች ለተዘረዘሩት ጥያቄዎች መልስዎን ከአማራጭ መልሶች በመምረጥ ያከብቡ

ክፍል 1 ፡- ስለ ቤተሰቡ ማህበራዊ ና ስነ ህዝባዊ መረጃ			
ተራ ቁጥር	**ጥያቄ**	**አማራጭ መልሶች**	**የመልስ ኮድ**
101	የልጅዎ ፆታ	1. ወንድ	0
		2. ሴት	1
102	የልጅዎ ዕድሜ		
103	የልጅዎ የትምህርት ደረጃ (ክፍል)		
104	የቤተሰቡ አስተዳዳሪ የትምህርት ደረጃ ?	1.ማንበብና መጻፍ የማይችል	0
		2.ማንበብና መጻፍ የሚችል	1
		3. የመጀመሪያ ደረጃ ትምህርት (1-8 ክፍል)	2
		4. ሁለተኛ ደረጃ ትምህርት (9-12 ክፍል)	3
		5. ዲፕሎማና ከዚያ በላይ	4
105	የቤተሰቡ አስተዳዳሪ የስራ ሁኔታ ?	1. የመንግስት ወይም የግል ተቀጣሪ	0
		2.የግል ንግድ ስራ	1
		3. የቀን ሰራተኛ	2
		4. ሌላ ካለ (ይገለጽ) _____	3
106	የቤተሰቡ ወርሃዊ የገቢ መጠን ?	1. 0 - 999 ብር	0
		2. ከ1000 - 1999 ብር	1
		3. ከ 2000 ብር በላይ	2
107	የቤተሰቡ ብዛት ?	1. ከአምስት በታች	0
		2. አምስትና ከአምስት በላይ	1
108	ብሔር	1. ኦሮሞ	0
		2. አማራ	1
		3. ትግራይ	2
		4. ሌላ ከሆነ (ይገለጽ) _____	3
109	ኃይማኖት	1. ኦርቶዶክስ ተዋህዶ	0
		2. ሙስሊም	1
		3. ፕሮቴስታንት	2
		4. ካቶሊክ	3
		5. ሌላ ካለ (ይገለጽ) _____	4

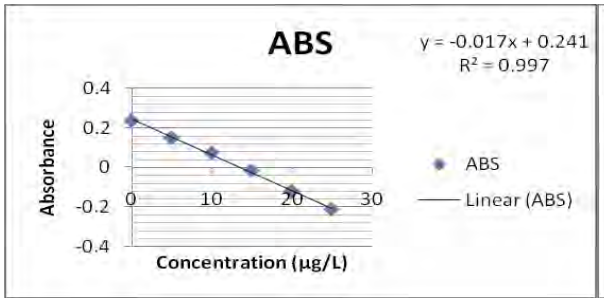
ክፍል 2፡ ስለ አዮዲንና በአዮዲን የበለፀገ ጨው ያለዎትን እዉቀትና አጠቃቀምን በተመለከተ

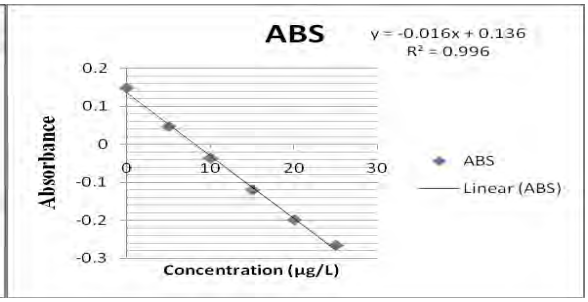
ተራ ቁጥር	ጥያቄ	አማራጭ መልሶች	የመልስ ኮድ
201	ስለ አዮዲን ንጥረ ነገር ጥቅም ያውቃሉ ?	1. አዎ አውቃለሁ	0
		2. አላውቅም	1
202	የአዮዲን እጥረት በጤና ላይ የሚያስከትልውን ችግር ያዉቃሉ ?	1. አዎ አውቃለሁ	0
		2. አላውቅም	1
203	(በጥያቄ 202 ላይ ምላሽዎ አዎ ከሆነ) በጤና ላይ ከሚያስከትለው ችግር የትኛውን ያዉቃሉ ?	1. የእንቅርት በሽታ	0
		2. የአእምሮ እድገት ዝግመት	1
		3. ያለእምሮ የመፍጠር ችሎታ መቀነስ	2
		4. ሞቶ መወለድ	3
		5. ድንገተኛ ውርጃ	4
204	ለአዮዲን እጥረት በይበልጥ ተጋላጭ የሆኑት የህብረተሰብ ክፍሎች የትኞቹ ናቸው ?	1. ሕፃናት	0
		2. ነፍሰጡር እናቶች	1
		3. ወንዶች	2
		4. ሴቶች	3
		5. ሌላ ካለ (ይገለጽ) _____	4
205	በአዮዲን ከበለፀጉ ምግቦችን የትኛውን ያውቃሉ ?	1. በአዮዲን የበለፀገ ጨ ው ወይም ዘይት	0
		2. ስጋና የስጋ ውጤቶች	1
		3. ከሀይቅ ውስጥ የሚገኙ ምግቦች	2
		4. ወተትና የወተት ተዋፅኦዎች	3
		5. ሌላ ካለ (ይገለጽ) _____	4
206	በአዮዲን የበለፀገ ጨው ይጠቀማሉ ?	1. አዎ እጠቀማለሁ	0
		2. አልጠቀምም	1
		3. አላውቅም	2
207	(በጥያቄ 206 ላይ ምላሽዎ አዎ ከሆነ) ለምን በአየዲን የበለፀገ ጨው ይጠቀማሉ ?	1. ለጤና በጣም አስፈላጊ ስለሆነ	0
		2. ለምግብ ጣዕም	1
		3. ሌላ ካለ (ይገለጽ) _____	2
		4. አላውቅም	3

208	የትኛውን አይነት ጨው ይጠቀማሉ?	1. ተፈጨቶ የታሸገውን	0
		2. አንኩዋር ጨውንና ያልታሸገውን	1
		3. አላውቅም	2
209	ጨውን ሲገዙ በአዮዲን የበለፀገ መሆኑን በምን ያውቃሉ ?	1. በእሽጉ ላይ የተጻፈውን በማንበብ	0
		2. ጨውን የሚሸጡትን በመጠየቅ	1
		3. አላውቅም	2
210	በቤትዎ ጨውን በምን አይነት ቦታ ያስቀምጣሉ ?	1. በቀዝቃዛና ደረቅ ቦታ	0
		2. በእርጥበታማና እሳት ባለበት ቦታ	1
		3. አላውቅም	2
211	ጨውን በምን አይነት እቃ ያስቀምጣሉ ?	1. ክዳን ባለው እቃ ውስጥ	0
		2. ክፍት በሆነ እቃ ላይ	1
		4. አላውቅም	2
212	ምግብ ሲያዘጋጁ ጨውን በየትኛው ጊዜ ይጨምራሉ ?	1. ምግቡ መስራት ሲጀመር	0
		2. ምግቡ እየበሰለ እያለ	1
		3. ምግቡ ከበሰለ በኃላና ከምድጃው ሲወጣ	2
		4. አላውቅም	3

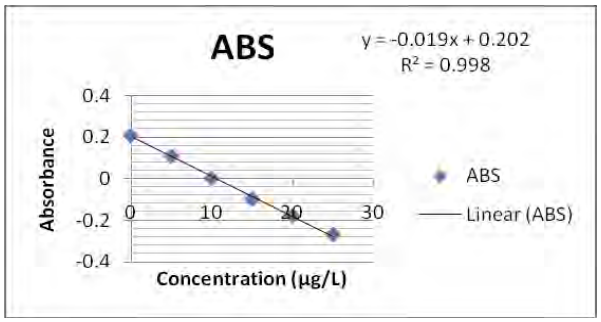
“ ጨርሰናል ስለተሳትፍዎ እናመሰግናለን ”

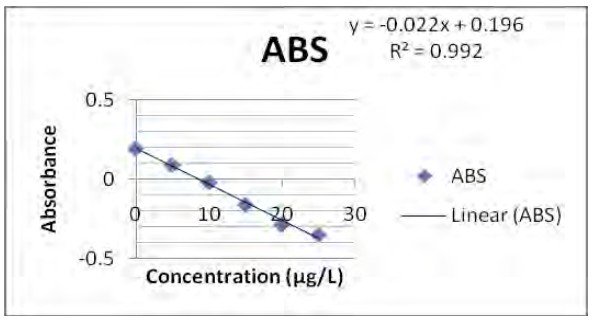
Appendix III: Standard urinary iodine calibration curves



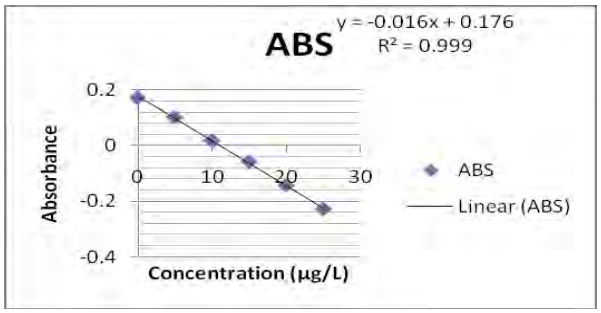


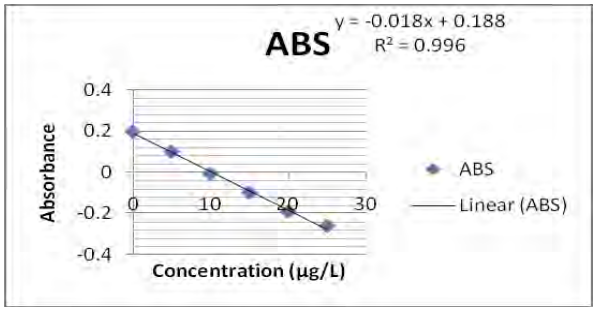
Date of analysis (02/9/2015) Standard A Date of analysis (02/9/2015) Standard B



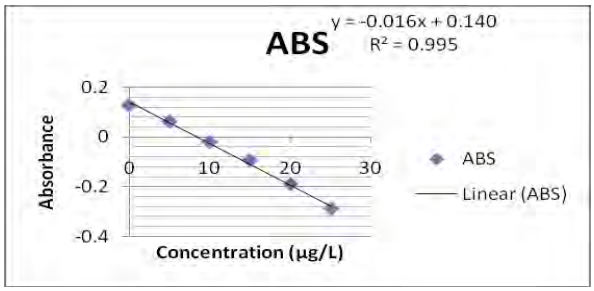


Date of analysis (03/9/2015) Standard A Date of analysis (03/9/2015) Standard B





Date of analysis (08/9/2015) Standard A Date of analysis (08/9/2015) Standard B



Date of analysis (09/9/2015) Standard A

Appendix IV: Ethical approval letter from Ethical Committee of the College of Natural Science, Addis Ababa University.

COLLEGE OF NATURAL SCIENCES
Addis Ababa University
OFFICE OF THE DEAN
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አዲስ አበባ ዩኒቨርሲቲ

Ref: ቁጥር: CNSDO/392/07/15
Date: ቀን: April 2, 2015

To Whom It May Concern

The Ethical Committee of the College of Natural Sciences in its meeting held on 26/03/2015 (Minutes No: IRB/015/2015) has examined the MSc. dissertation project of **Solomon Emiru**, entitled **"Iodized salt consumption and goiter status of school children in schools of Akaki Kality Subcity of Addis Ababa, Ethiopia"** for ethical approval.

The Proposal is approved for implementation.

With regards,

Negussie Retta, (Professor)
Dean, College of Natural Sciences

Encl:

- RERC Minutes

ስ/ቁ/Tel: +251-11-123-9472
ፋክስ/Fax: +251-11-123-9469
ፖ.ሣ.ቁ/POBox: 1176, Addis Ababa, Ethiopia
ኢ.ሜ.አ/Email: dean_cns@aau.edu.et
Please quote our reference number in your correspondence.
"Examine all things; hold fast that which is good"
"ሁሉን መርምሩ፣ መልካሙን ያዙ"