INTRODUCTION

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Pregnancy and increased micronutrients requirement: a concern

Pregnancy is associated with increased nutritional needs due to the physiologic changes and the metabolic demands of the embryo/fetus. Hence, the nutritional status of mother prior to conception establishes the quality of the environment in which the fetus will develop and is a key determinant in the life of the newborn. Proper maternal nutrition during pregnancy is thus imperative for the health of both the woman and the offspring. Daily requirements for micronutrients particularly iodine and iron during pregnancy are higher to meet the physiologic changes and increased nutritional needs of pregnancy. Both iodine and iron requirements are increased by 40% during pregnancy.

Micronutrient	RDA non pregnant women	RDA pregnant women	Increase in requirement
Iodine	150 µg/d	250 µg/d	40%
Iron	21 mg/d	35 mg/d	40%

Source: NIN, 2010; WHO/UNICEF/ICCIDD, 2007

Iodine is an essential micronutrient for normal growth and development. The human body contains 15–20 mg of iodine, of which 70–80% is concentrated in the thyroid gland (FAO, 2005).

At present, the only physiological role known for iodine in the human body is for the synthesis of thyroid hormones by the thyroid gland. Therefore, the dietary requirement of iodine is determined by normal thyroxine (T4) production by the thyroid gland without stressing the thyroid iodide trapping mechanism or rising thyroid stimulating hormone (TSH) levels. Iodine from the diet is absorbed throughout the gastrointestinal tract. Dietary iodine is converted into iodide ion before it is absorbed. The iodide ion is 100% bio-available and absorbed

totally from food and water. This is however not true for iodine within thyroid hormones ingested for therapeutic purposes.

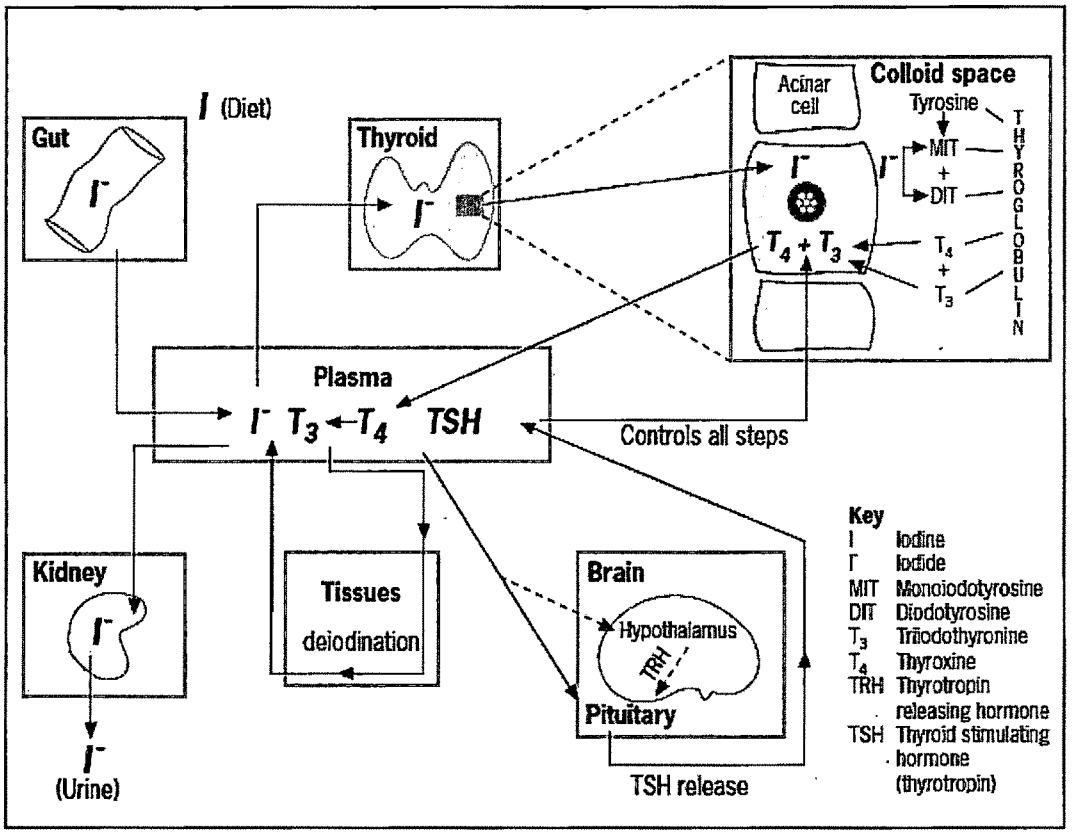
Iodine enters the circulation as plasma inorganic iodide, which is cleared from the circulation by the thyroid and kidney. The iodide is used by the thyroid gland for synthesis of thyroid hormones, and the kidney excretes excess iodine with urine. The excretion of iodine in the urine is a good measure of iodine intake. In a normal population with no evidence of clinical iodine deficiency either in the form of endemic goitre or endemic cretinism, urinary iodine excretion reflects the average daily iodine requirement. Therefore, for determining the iodine requirements and iodine intake, the important indices are serum T4 and TSH levels (exploring thyroid status) and urinary iodine excretion (exploring iodine intake). A simplified diagram of the metabolic circuit of iodine is given in Figure 1.1. All biological actions of iodide are attributed to the thyroid hormones. The major thyroid hormone secreted by the thyroid gland is T4. T4 in circulation is taken up by the cells and is de-iodinated by the enzyme 5'-monodeiodinase in the cytoplasm to convert it into triiodothyronine (T3), the active form of thyroid hormone. T3 traverses to the nucleus and binds to the nuclear receptor.

All the biological actions of T3 are mediated through the binding to the nuclear receptor, which controls the transcription of a particular gene to bring about the synthesis of a specific protein.

The physiological actions of thyroid hormones can be categorized as 1) growth and development and 2) control of metabolic processes in the body. Thyroid hormones play a major role in the growth and development of the brain and central nervous system in humans from the 12th week of gestation to 3 years of age. If iodine deficiency exists during this period and results in thyroid hormone deficiency, the consequence is derangement in the development of the brain and

central nervous system. These derangements are irreversible; the most serious form being that of cretinism.

Figure 1.1: Summary of thyroid hormone production and regulation



Source: Stanbury, 1960

The effect of iodine deficiency at different stages of life is given in Table 1.1. The other physiological role of thyroid hormones is to control several metabolic processes in the body. These include carbohydrate, fat, protein, vitamin, and mineral metabolism. For example, thyroid hormone increases energy production, increases lipolysis, and regulates neoglucogenesis, and glycolysis.

Table 1.1: Effect of iodine deficiency by life stage

Life stage	Effects
All ages	Goitre Hypothyroidism Increased susceptibility to nuclear radiation

Life stage	Effects
Fetus	Abortions
	Stillbirths
	Congenital anomalies
	Increased perinatal mortality
	Increased infant mortality
	Neurological cretinism: mental deficiency, deaf mutism, spastic diplegia, and squint
	Myxedematous cretinism: mental deficiency, hypothyroidism and dwarfism
	Psychomotor defects
	Impairment in development of brain, lung, muscle, nerves, adipose tissue, heart and cardiovascular function
Neonate	Neonatal goitre
	Neonatal hypothyroidism
Child and adolescent	Goitre
	Juvenile hypothyroidism
	Impaired mental function
	Retarded physical development
Adult	Goitre with its complications
	Hypothyroidism
	Impaired mental function
	Iodine-induced hyperthyroidism

Source: Zimmermann et al, 2008

IODINE DEFICIENCY, THYROID DISORDERS AND PREGNANCY

The high global prevalence of iodine deficiency and autoimmune thyroid disorders, the mental and physical consequences of these disorders create a huge human and economic burden that can be prevented, in large part, by early detection and therapeutic measures.

Over the past several years it has been proved that maternal thyroid disorders influence the outcome of mother and fetus, during and also after pregnancy. The most frequent thyroid disorder in pregnancy is maternal hypothyroidism, which is associated with fetal loss, placental abruptions, pre-eclampsia, preterm delivery and reduced intellectual function in the offspring (Abalovich et al, 2002).

Thyroid disorders during pregnancy

Prevalence of thyroid disorders during pregnancy is, hypothyroidism-2% (Vanderpump et al, 1995), congenital hypothyroidism-1/4,000 (Vanderpump et al, 1995), overt hypothyroidism-0.2-0.5% (Allan et al, 2000; Casey et al, 2005), subclinical hypothyroidism-2.2-2.5% (Allan et al, 2000; Casey et al, 2005), hypothyroxinemia-1.3-2.1% (Casey et al, 2007; Cleary-Goldman et al, 2008) and TPO-Ab or TG-Ab positive test results-5% (Cleary-Goldman et al, 2008).

Worldwide more than 20 million people develop neurological disorders due to intra uterine iodine deprivation (Girling, 2008). Another problem related to thyroid disorders during pregnancy is postpartum thyroiditis.

Physiology of thyroid in pregnancy

Thyroid hormones consist of thyroxine (T4) and triiodothyronine (T3) of which active forms are the free portions (fT3, fT4) consisting of 1% of total hormones. The fT3 fraction is biologically more significant and derived from conversion of fT4 at liver, kidney and muscle. The fT3 hormone acts through specific nuclear receptors of fT3, situated in most of the tissues. TSH secreted from anterior pituitary act as negative feedback from fT3 levels. Dietary iodine is essential for this thyroid hormone synthesis.

Fetus: In pregnancy, fetus receives iodine from maternal source in all the trimesters. Fetus receives thyroxine from mother up to 12 weeks through placental circulation but not TSH or fT3. Thyroxine is partially converted to fT3 and combines with receptors in fetal brain and is responsible for fetal brain development. From 12th week, placental changes resist T4 passage to fetus and fetal pituitary thyroid axis start functioning like adult (Girling, 2008).

Pregnant women: Pregnancy has an appreciable effect on thyroid economy. There is an increase in thyroid binding globulins, increase

in total T4 and T3, thyroid stimulation by hCG, increase in renal iodine clearance and increase in serum thyroglobulin during normal pregnancy (Fantz et al, 1999). Hence as a result of these changes, iodine requirements increase during pregnancy.

Maternal aspects of hypothyroidism

Women with hypothyroidism have decreased fertility; even if they conceive, risk of abortion is increased, and risk of gestational hypertension, anemia, placental abruption and postpartum hemorrhage is increased (Abalovich et al, 2002). The risk of these complications is greater in women with overt, rather than subclinical hypothyroidism.

Fetal and neonatal aspects of maternal hypothyroidism

Untreated maternal hypothyroidism can lead to preterm birth, low birth weight, and respiratory distress in the neonate. Enough evidence has accumulated over the years about the role of thyroxine in normal development of the fetal brain. The presence of specific nuclear receptors and thyroid hormone found in fetal brain at 8 week of gestation, free T4 found in the coelomic and amniotic fluids and demonstration of the transfer of maternal thyroid hormones across the placenta, underline the role of thyroid hormones in fetal brain development. Complex interactions between the D2 and D3 iodothyronine deiodinases during gestation help to fine tune the supply of adequate amounts of T3 required for normal brain development.

A number of pioneering studies by Man et al (1971), Haddow et al (1999), Pop et al (1999) and newer studies by Rovet et al (2004) and Vermiglio et al (2004) have conclusively proved that children born to mothers with hypothyroidism had a significantly increased risk of impairment in IQ scores, neuropsychological developmental indices and learning abilities. Children born to untreated hypothyroid women

had an IQ score that was 7 points below the mean IQ of children born to healthy women and women given thyroxine supplements. This risk applies to children born not only of untreated women, but also women with suboptimal supplementation. A study by Rovet et al (2004) found that such children had mild defects in global intelligence, but visual-spatial ability, language, fine motor performance, and preschool ability were unaffected. This study emphasizes the need to follow-up women adequately after initiating treatment. Children born to mothers with iodine deficiency fared even worse, with a greater than 10-point average deficit in global IQ and quite a few also had attention deficit hyperactivity disorder (Vermiglio et al, 2004).

Need for trimester specific reference intervals

Because of physiological changes, values of thyroid hormones during pregnancy differ from non-pregnant values. Values in pregnancy also vary from trimester to trimester and from method to method. Since more and more researchers are aware of the importance of evaluating maternal thyroid function during pregnancy by gestation-specific reference intervals, manufacturer's reference range should not be used for pregnant women. If a non-pregnant reference interval is used, a number of pregnant women with thyroid dysfunction could be potentially misclassified.

IODINE DEFICIENCY AND PREGNANCY

Iodine plays a critical role in the neuropsychological development of the fetus throughout gestation and in the first two years of life. Iodine uptake by the thyroid is higher in pregnancy and iodine reserve in the thyroid can decrease to approximately 40% of preconception levels (Glinoer, 1997*). World Health Organization (WHO) has recently increased their recommended iodine intake during pregnancy from 150 to 250 µg/day.

Thyroid gland stores iodine from the diet and as such maternal iodine status is not entirely dependent on the current dietary intake during gestation. If preconception iodine nutrition is adequate there will be sufficient stores of thyroid hormone to support the mother and foetus, at least in the first trimester. However if preconception dietary intake is deficient the increasing demands of later pregnancy may produce a deficit which untreated can result in a hypothyroxinemic state (Smyth, 2006). There is some evidence suggesting that in areas of mild to moderate iodine deficiency, the maternal thyroid is able to adapt to meet the increased thyroid hormone requirements of pregnancy (Zimmerman, 2009*).

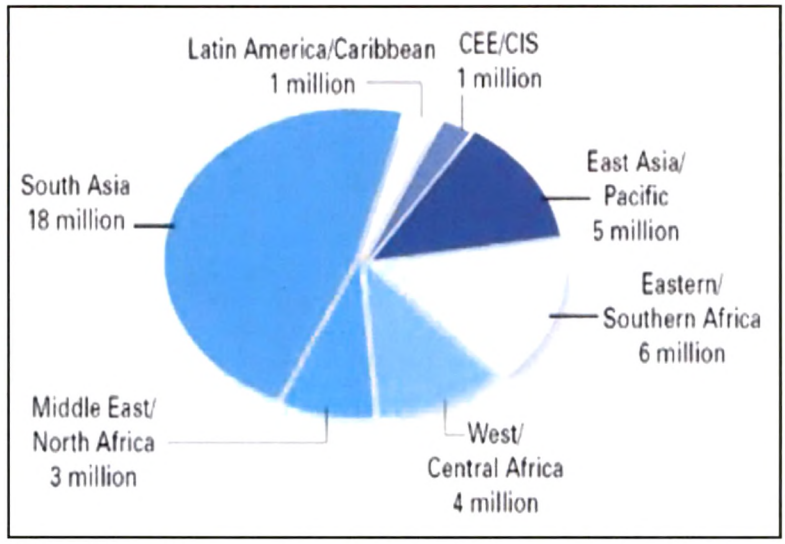
Over the past decade, there has been increasing focus on iodine deficiency during pregnancy as iodine is critical for optimal fetal development, yet 38 million newborns in developing countries every year remain unprotected from the lifelong consequences of brain damage associated with iodine deficiency (Figure 1.2).

GLOBAL iodine nutrition

Only a few countries, Switzerland, some of the Scandinavian countries, Australia, the United States and Canada were completely iodine sufficient before 1990. Since then, there has been a major global effort to introduce salt iodization to ensure sufficient intake in deficient areas. Over two-thirds of the world's population is now covered by iodized salt (UNICEF, 2012).

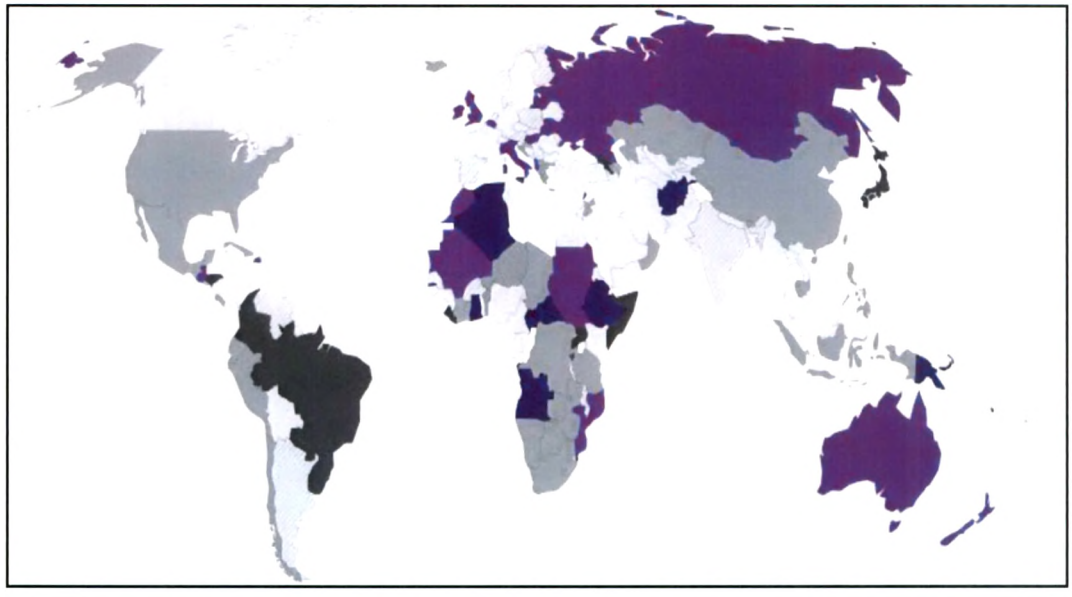
Global iodine nutrition has markedly improved over the past decade (but with strong regional differences) and the number of iodine deficient countries has decreased from 54 in 2003 to 32 in 2011 (Figure 1.4). Yet despite remarkable progress, 1.88 billion of the global population, including 241 million school children, still has insufficient dietary iodine intakes.

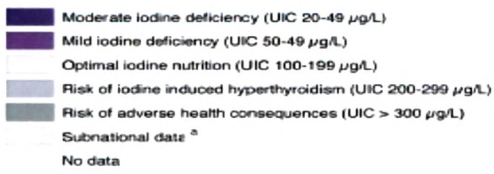
Figure 1.2: Distribution of infants born in developing countries annually who are unprotected against IDD, by region 2000-2006



Source: UNICEF, 2012

Figure 1.3: National iodine status based on urinary iodine concentrations in school aged children

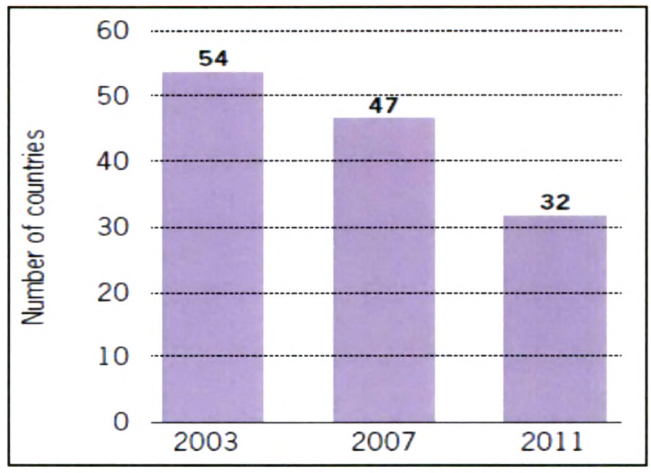




*The country estimates in the cross-hatched countries are based on sub-national data. The national coverage of iodized salt in these countries is incomplete, there are large variations in the iodine intake and some regions likely remain deficient.

Source: IDD Newsletter, Feb 2010

Figure 1.4: Number of iodine deficient countries in 2003, 2007 and 2011



Source: IDD Newsletter, Feb 2012

Table 1.2: Countries (number) by iodine status over the period 2003-2011

Iodine intake	2003	2007	2011
Insufficient: severe iodine deficiency	1	0	0
Insufficient: severe iodine deficiency	13	10	9
Insufficient: severe iodine deficiency	40	37	23
Adequate	43	49	69
More than adequate	24	27	36
Excessive	5	7	11
Countries with data	126	130	148
Total countries	**192**	**193**	**193**

Source: IDD Newsletter, Feb 2012

Iodine nutrition of pregnant women

Only a limited number of countries have completed UIC surveys in pregnant women and women of reproductive age on the national or large sub-national level. Thus, there are insufficient data to directly estimate the regional or global prevalence of low iodine intake in these important target groups. This is a major limitation of the current

estimate because although the median UIC in children may be used to represent iodine status of most of the population, it should not be used as a proxy for iodine status in pregnant women (Wong et al, 2011).

Iodine nutrition in INDIA

There is insufficiency of national data on urinary iodine concentration of population (Figure 1.3). According to global iodine nutrition scorecard of 2010 and 2012, percentage of households consuming iodized salt in India has improved with an improvement in median UIE. However, proportion of population having UIE <100 has not improved (Table 1.3).

Table 1.3: Comparison of country data on iodine nutrition

Global scorecard values for INDIA		**2010***	**2012****
Annual no. of births in 2008* and 2009** (000)		26'913	26'787
Household consuming iodized salt (%)		51.1	71
Median UIE (µg/L)		133	154
Proportion of population with UIE <100 (%)		31.3	34.4
General Population	Iodine deficiency protected population (000)	602'520	625'778
	Iodine deficiency unprotected population (000)	576'892	598'836
Infants	Iodine deficiency unprotected infants (000)	13'726	13'688

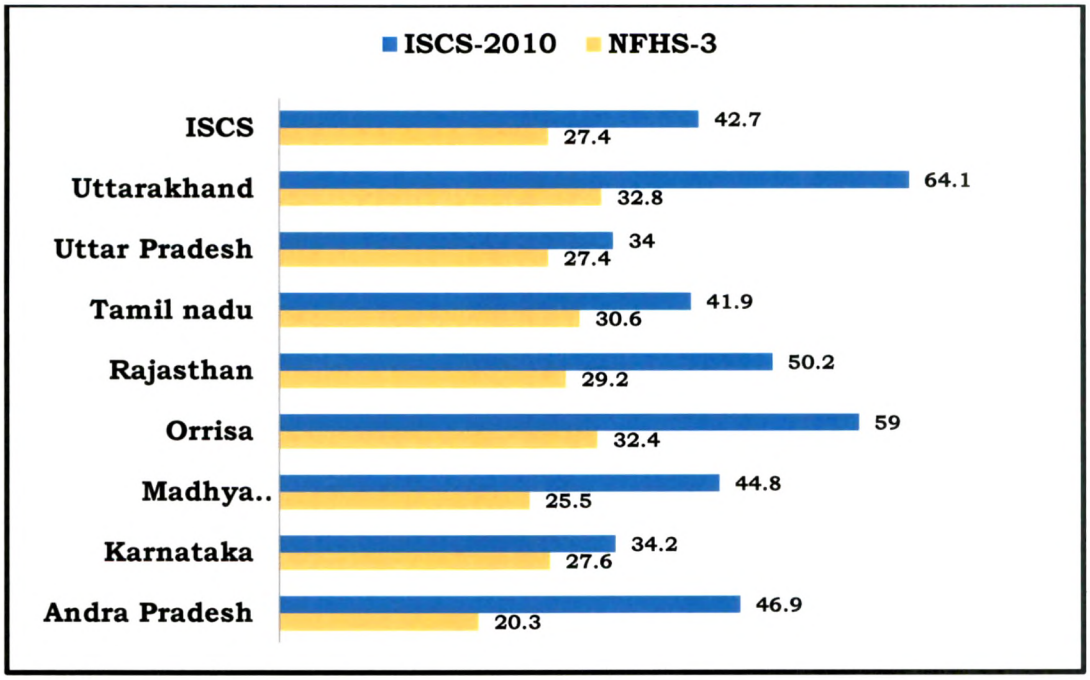
Source: *IDD Newsletter, Feb 2010; ** IDD Newsletter, Feb 2012

Adequately iodized salt availability at household level in 8 states

Recently in 2010, Micronutrient Initiative (MI) has conducted a study on iodine content of edible salt at household level in rural areas of eight states. A comparison between NFHS-3 and Iodized Salt Coverage Study (MI-ISCS, 2010) is shown in Figure 1.5. The use of adequately

iodized salt in rural households has increased across all states. The 8 state averages have gone up from 27% during NFHS-3 to 47.2% during the 2010 study. The highest increase was evidenced in Uttarakhand followed by Orissa, Rajasthan, Andhra Pradesh and Madhya Pradesh. Tamil Nadu and Uttar Pradesh have also reported modest increases. Use of iodized salt has gone up significantly in rural areas in states which were previously considered to be problem states. The amount of adequately iodized salt at the household level has increased and the amount of non-iodized salt has dropped dramatically (MI-ISCS, 2010).

Figure 1.5: A comparison between NFHS-3 and Iodized Salt Coverage Study



Source: MI-ISCS, 2010

Recommendations of thyroid societies (worldwide):

Recently in 2007 (Abalovich et al), all thyroid societies have worked towards development of guidelines for treatment of thyroid disorders. These societies are-Latin American Thyroid Society, the Asia and Oceania Thyroid Society, the American Thyroid Association, the European Thyroid Association and the American Association of Clinical Endocrinologists.

Hypothyroidism and pregnancy: Maternal and fetal aspects

- Both maternal and fetal hypothyroidism is known to have serious adverse effects on the fetus. Therefore maternal hypothyroidism should be avoided.
- If hypothyroidism has been diagnosed before pregnancy, we recommend adjustment of the preconception thyroxine dose to reach a TSH level not higher than 2.5 μU/mL prior to pregnancy.
- The T4 dose usually needs to be incremented by 4-6 wk gestation and may require a 30-50% increase in dosage.
- If overt hypothyroidism is diagnosed during pregnancy, thyroid function tests (TFTs) should be normalized as rapidly as possible. Thyroxine dosage should be titrated to rapidly reach and thereafter maintain serum TSH concentrations of less than 2.5 μU/mL in the first trimester (or 3 μU/mL in the second and third trimester) or to trimester-specific normal TSH ranges. Thyroid function tests should be re-measured within 30-40 days.
- Women with thyroid autoimmunity who are euthyroid in the early stages of pregnancy are at risk of developing hypothyroidism and should be monitored for elevation of TSH above the normal range.
- Subclinical hypothyroidism (serum TSH concentration above the upper limit of the reference range with a normal free T4) has been shown to be associated with an adverse outcome for both the mother and offspring. T4 treatment has been shown to improve obstetrical outcome but has not been proved to modify long-term neurological development in the offspring. However, given that the potential benefits outweigh the potential risks, the panel recommends T4 replacement in women with subclinical hypothyroidism.
- After delivery, most hypothyroid women need a decrease in the thyroxine dosage they received during pregnancy.

Iodine nutrition during pregnancy

- Women in the childbearing age should have an average iodine intake of 150 µg per day. During pregnancy and breast-feeding, women should increase their daily iodine intake to 250 µg on an average.
- Iodine intake during pregnancy and breastfeeding should not exceed twice the daily recommended nutritional intake for iodine, i.e. 500 µg iodine per day.
- To assess the adequacy of the iodine intake during pregnancy in a population, urinary iodine concentration (UIC) should be measured in a cohort of the population. UIC should ideally range between 150 and 250 µg/L.
- To reach the daily recommended nutrient intake for iodine, multiple means must be considered, tailored to the iodine intake level in a given population. Different situations must therefore be distinguished: a) countries with iodine sufficiency and/or with a well-established universal salt iodization (USI) program; b) countries without a USI program or an established USI program where the coverage is known to be only partial; and finally c) remote areas with no accessible USI program and difficult socioeconomic conditions.

Postpartum thyroidtis

- There are insufficient data to recommend screening of all women for postpartum thyroiditis (PPT).
- Women known to be thyroid peroxidase antibody positive should have a TSH performed at 3 and 6 months postpartum.
- The prevalence of PPT in women with type 1 diabetes is threefold greater than in the general population. Postpartum screening (TSH determination) is recommended for women with type 1 diabetes mellitus at 3 and 6 months postpartum.

- Women with a history of PPT have a markedly increased risk of developing permanent primary hypothyroidism in the 5 to 10 year period following the episode of PPT. An annual TSH level should be performed in these women.
- Asymptomatic women with PPT who have a TSH above the reference range but below 10 μU/mL and who are not planning a subsequent pregnancy do not necessarily require intervention, but should, if untreated, be re-monitored in 4–8 weeks. Symptomatic women and women with a TSH above normal and who are attempting pregnancy should be treated with levothyroxine.
- There is insufficient evidence to conclude whether an association exists between postpartum depression (PPD) and either PPT or thyroid antibody positivity (in women who did not develop PPT).
- However, as hypothyroidism is a potentially reversible cause of depression, women with postpartum depression should be screened for hypothyroidism and appropriately treated.

Screening for thyroid dysfunction during pregnancy

Although the benefits of universal screening for thyroid dysfunction (primarily hypothyroidism) may not be justified by the current evidence (presented above), we recommend case finding among the following groups of women at high risk for thyroid disease by measurement of TSH:

- Women with a history of hyperthyroid or hypothyroid disease, PPT, or thyroid lobectomy.
- Women with a family history of thyroid disease.
- Women with a goiter.
- Women with thyroid antibodies (when known).

- Women with symptoms or clinical signs suggestive of thyroid underfunction or overfunction, including anemia, elevated cholesterol, and hyponatremia.
- Women with type I diabetes.
- Women with other autoimmune disorders.
- Women with infertility who should have screening with TSH as part of their infertility work-up.
- Women with previous therapeutic head or neck irradiation.
- Women with a history of miscarriage or preterm delivery.

IRON DEFICIENY ANEMIA DURING PREGNANCY

Iron deficiency anemia is the most common nutritional deficiency in the World. Anemia is a condition of low levels of hemoglobin in the blood. It is a widespread public health problem associated with increased risk of morbidity and mortality. Young children, pregnant and postpartum women are the most severely affected by iron deficiency because their demand for iron is high.

National and state prevalence of anemia during pregnancy

According to DLHS survey percentage of pregnant women having anemia in India is 55.3% with 38.6% mildly anemic, 15% moderately anemic and 1.8% severely anemia (Kothari and Noureddine, 2010). According to NFHS 3 data, 57.9% pregnant women (15-49 years of age) are anemic in Gujarat state.

In developing countries, the majority of women are anemic in the second half of pregnancy. Pregnant women are often iron deficient and iron deficiency has adverse effects on thyroid function. During the second and third trimester, pregnant women are highly vulnerable to iron deficiency because their increased iron needs are rarely met by dietary sources. Iron deficiency has multiple adverse effects on thyroid metabolism. It decreases circulating thyroid hormone concentrations, likely through impairment of the heme-dependent thyroid peroxidase

(TPO) enzyme. Iron deficiency blunts the efficacy of iodine prophylaxis, and iron repletion improves the efficacy of iodized salt in goitrous children with iron deficiency (Zimmermann, 2007).

MICRONUTRIENT DEFICIENCIES AND MDGs

Recent evidence suggests that, micronutrient deficiencies (especially iodine and iron) may play a role in children's development. Micronutrient deficiencies are a critical concern among children throughout the world. Approximately 30% of the world's population lives in iodine-deficient areas and 25% of the world's children <3 years of age have iron-deficiency anemia, with higher rates in developing countries. The relationship between micronutrient deficiency and early cognitive development has captured recent attention because micronutrients are related to specific physiological processes. Therefore, programs designed to prevent or treat micronutrient deficiencies can be targeted toward specific recommendations. The fortification of salt with iodine has been hailed as one of the world's great public health advancements. Now breakthrough technology that allows salt to be double fortified with iron as well as iodine has created an exciting new opportunity to reach the world with supplemental iron easily and inexpensively, without having to change people's habits.

Nutrition actions are critical to achieve the Millennium Development Goals (MDGs). Micronutrient interventions are suggested as cost-effective and programmatically feasible to scale-up worldwide.

Elimination of Iodine Deficiency will contribute to at least six of the Millennium Development goals:

MDG 1, Eradicate extreme poverty and hunger: Eliminating iodine deficiency will increase learning ability and intellectual potential, leading to higher earnings. In addition, the burden of diseases and pathologies related to ID will be eliminated.

MDG 2, Achieve Universal Primary Education: Children will have improved cognitive development and learning capacity,

leading to improved school performance and reduced drop-out rates.

MDG 3, Promote gender equality and empower women: Eliminating ID in children reduces child care burden for women, frees up household resources and allows women more time for income generating work.

MDG 4, Reduce child mortality: Reduced ID contributes to decreased rates of miscarriages, stillbirths, and other pregnancy complications, as well as early neonatal deaths.

MDG 5, Improve maternal health: Eliminating ID in women will reduce rates of miscarriages, thyroid diseases and other clinical outcomes of ID, thus improving the health status of women of reproductive age.

MDG 8, Develop a global partnership for development: The programs for sustainable elimination of iodine deficiency ensure a strong partnership of public, private and civil society at the global, regional, and country level.

DFS can play a major role:

MDG 2, Achieve Universal Primary Education: DFS will improve children's cognitive development and educational outcomes through increased and sustained intake of iron and iodine.

MDG 5, Improve Maternal Health: DFS will improve the survival and health of women by increasing and sustaining their iron and iodine intake and, in turn reducing the consequences of iron deficiency anemia and of poor pregnancy outcomes.

From the above discussion it was worthwhile to work towards-

(1) Preventing fetal brain damage by screening pregnant women during early gestation for thyroid dysfunction, iodine deficiency and iron deficiency anemia.

(2) Improving maternal iodine and iron status with supplementation through double fortified salt.

Apart from screening, pregnant women should also be provided with knowledge regarding antenatal care, delivery care and postnatal care. Correction of iodine deficiency and iron deficiency alone may not result in delivering a healthy baby. In order to ensure healthy baby, it also becomes necessary to check that pregnant women are availing good antenatal care, delivery care and postnatal care. Welfare check-ups for babies once in three months till one year will further reduce morbidity/mortality in children.

In view of the previous section discussion, present study was planned with major objective-

"Maternal thyroid dysfunction and iodine deficiency, its implications on infant development and impact of double fortified salt supplementation"

Specific objectives were:

- To screen pregnant women during first trimester for thyroid dysfunction, iodine deficiency and iron deficiency anemia.
- To provide Nutrition Health Education (NHE) to pregnant women regarding importance of iodine and iron nutrition during early pregnancy.
- To provide knowledge to pregnant women regarding maternal health components (antenatal care, delivery care and postnatal care).
- To record thyroid hormone levels, urinary iodine concentration and hemoglobin status during each trimester.
- To assess the nutritional status of pregnant women (anthropometry, biochemical indicators and 24 hr dietary recall).
- To screen the neonates.
- To test infant development.
- To study postpartum maternal thyroid status.
- To study the impact of double fortified salt supplementation (DFS) on iodine and iron status during lactation.
- To study infant iodine status.