
Chapter

11

DISCUSSION

Iodine is an important micronutrient and is a raw material for hormones produced by thyroid factory. The consequences of elemental deficiency are collectively known as iodine deficiency disorders (IDD). These are mental and physical retardation, impaired reproductive outcome and goitre. IDD still belong to the preeminent health problems of the world. Iodine deficiency, the principal cause of goitre, can be avoided by using salt fortified with iodine. Alternative options are administration of oral or intramuscular iodised oil, iodization of drinking and irrigation water, iodide tablets and iodized bread.

Apart from primary cause of iodine deficiency, a series of other secondary factors like environmental goitrogens and malnutrition processes contribute to goitrogenesis thus both deteriorating or mitigating the cascade of iodine deficiency that leads to induced growth and proliferation of the thyroid gland.

The basic process in the goitre formation is the generation of new thyrocytes and follicles on one side by replication and mitosis, and hyperplasia of existing follicles on the other hand. These are triggered by

- (a) TSH,
- (b) Growth stimulating immunoglobulins
- (c) Other growth factors already at low concentration

Iodine deficiency as well as dietary goitrogens interact with thyroid hormone synthesis and increase TSH that stimulates thyroid growth directly or indirectly by increasing the sensitivity of thyrocytes to insulin-like growth factor I. The exact mechanism through which thyroid growth is induced in iodine deficiency is somewhat controversial (Bidley SP et al, Burgi U et al, Durgrillon A et al, Durmont JE, Gartner R, Peter HJ, Studer H and Thomas GA). There are mainly two views: Intrathyroidal Autoregulation through TSH (Studer) or direct growth inducing effect of iodine depletion through intrathyroidal mechanisms (Gärtner). Both mechanisms play an important role in goitrogenesis and depending upon the degree of iodine deficiency one of them may prevail. In severe iodine deficiency TSH increases whereas in moderate iodine deficiency Autoregulation predominates.

Thyroid gland can be considered as a factory (importance of raw material, efficient machinery and the response to changing conditions). The output of this factory, are the hormones (Dunn J T, 1996). The raw materials for thyroid factory are iodine, and proteins that form thyroglobulin and enzymes needed for organification, coupling and release. TSH is modulator and provides the major external control of thyroid function. When there is shortage of raw material iodine, the thyroid gland production efficiency is increased by

1. Increasing iodine delivery to thyroid
2. Maximizing the amount of hormone produced per atom of iodine
3. Turning over iodine rapidly and not wasting it in storage
4. Recycling all the iodine that is not secreted as hormone
5. Increasing thyroid mass

A key factor in the thyroid gland's ability to respond to iodine deficiency is its flexibility. It has adaptive responses throughout its metabolic pathways.

The concentration gradient for iodine can be adjusted over a 50-fold range depending on its supply.

A large number of natural agents in the environment are known to affect thyroid gland morphology and function. Goitre is the most prominent effect of these agents. They may cause the goitrous condition by acting directly on the thyroid gland (figure chapter 1) but also indirectly by altering the regulatory mechanisms of the thyroid gland and the peripheral metabolism and excretion of thyroid hormones (Gaitan E, 1989, 1990). The mechanism of goitrogenesis is not well understood because besides TSH other humoral, paracrine, and autocrine growth factors appear to be involved in the process. These agents may enter the human body through food or water and become goitrogenic agents. Their effects may be either additive to iodine deficiency in making the IDD more severe or develop "sporadic" goitres in areas of iodine sufficiency or the persistence of goitre endemia.

The main goitrogens consumed by Gujarat population are thiocyanate and goitrin (cruciferae, capparidaceae and resedaceae family vegetables), isothiocyanates (mustard seeds fried in oil are used to decorate all vegetables), aliphatic disulfides (onion and garlic) and a variety of different flavonoids. These were arachidoside (peanuts and milk), catechin and kaempferol (tea and all vegetables), quercetin (onion), flavenol glycosides (red kidney beans and tomato), anthocyanidin and biflavan (vegetables, cereals, tubers, bulbs, and natural pigments), genistin, daidzin, glycitein, formononetin, biochanin-A and isoflavanone (soy beans, green beans, black beans and in leguminosae family), vitexin and apigenin (pearl millet).

A tremendous gap from a public health point of view persists between the well-documented biochemical knowledge of these goitrogenic compounds and their action in human beings.

Thiocyanate or thiocyanate-like compounds primarily inhibit the iodine concentration mechanism of the thyroid (first step in the formation of

thyroid hormones is active uptake of inorganic iodide and there is competitive inhibition by thiocyanate (SCN) or aliphatic disulfides in onion and garlic) and their goitrogenic activity can be overcome by iodine administration as iodized salt. Thiocyanate at low concentrations inhibits iodide transport by increasing the velocity constant of iodide efflux from the thyroid gland. At higher concentrations the iodide efflux is greatly accelerated while unidirectional iodide clearance into the gland is inhibited. Thiocyanate at higher concentrations also inhibits the incorporation of iodide into thyroglobulin by competing with iodide at the thyroid peroxidase (TPO) level (Gaitan E, 1989). Thiocyanate is rapidly converted to sulphate in the thyroid gland. TSH increases this intrathyroidal catabolism of SCN and reverses the block of iodine uptake produced by this ion.

Isothiocyanates act on the thyroid mainly by rapid conversion to thiocyanate. However, they can react spontaneously with amino acids forming di-substituted thiourea derivative, which produces a thiourea like antithyroid effect.

Goitrin (L-5 vinyl -2 thioxazolidone) is acting like thionamide type of goitrogen such as thiourea. Its action is to interfere with organification of iodide and formation of active thyroid hormones and this action can not usually be antagonised by iodine. Long-term administration of goitrin to rats resulted in increased thyroid weight and decreased radioactive iodine uptake and hormone synthesis (Gaitan E, 1989). Actually goitrin possesses 133% of the potency of propylthiouracil in humans. Goitrin is unique in not being degraded like thioglycosides. Progoitrin in Brassicae vegetables (turnip, cabbage etc) may be converted by hydrolysis to goitrin by normal GIT (large intestine and caecum) bacteria such as *Escherichia coli* and *Proteus vulgaris*. Goitrin inhibits TPO.

The second step for thyroid hormone synthesis entails the incorporation of oxidised iodine into the amino acid tyrosine –within the peptide sequence of thyroglobulin- to form mono-iodo-tyrosine (MIT) and di-iodo-tyrosine

(DIT). This is organification and the process is mediated by the action of thyroidal peroxidase enzyme (TPO). Flavonoids, aliphatic Disulfides and "Goitrin" inhibit organification.

Flavonoids are important stable organic constituents of a wide variety of plants. They are universally present in vascular plants and in a large number of food plants. Because of their widespread occurrence in edible plants such as fruits, vegetables, and grains, flavonoids are an integral part of the human diet. They are present in high concentrations in staple foods of Third World such as millet, beans, sorghum, and groundnuts.

Flavonoids are polyhydroxyphenolic compounds with a C₆-C₃-C₆ structure. Mammals cannot synthesize the flavone nucleus. Flavonoids are strictly exogenous food components of exclusively vegetable origin. They are quickly metabolised in higher organisms hence not found in normal tissue constituents. Most flavonoids are present as B-glucosides which can not be absorbed in the tissues. No mammalian enzymes deglycosylate these compounds to their bioactive aglycone species. Following ingestion by mammals, flavonoid glycosides are hydrolysed by intestinal microbial glycosidases to flavonoid aglycones. These may be absorbed and undergo metabolism by mammalian tissues or be further metabolised by intestinal micro-organisms to undergo B-ring hydroxylation and middle ring fission, with production of various metabolic monomeric compounds (Gaitan E, 1989). Each metabolic step is characterized by a marked increase in antithyroid effects (Gaitan E, 1989). Apigenin (flavonoid aglycone, a metabolite) is 3 to 5 times more potent than the parent compounds, vitexin, glycosylvitexin and glycosylorientin, as inhibitors of TPO. Flavonoids interfere with the synthesis of thyroid hormones by inhibiting the thyroid peroxidase enzyme (catalyzing iodide oxidation); thereby the organification processes and the peripheral metabolism of thyroid hormones by acting on the iodothyronine deiodinase enzyme. This greater inhibitory effect is further enhanced by the additive effects exerted by flavonoid metabolites, which are formed after ingestion of mixtures of flavonoid glycoside present in many plant foodstuffs.

Flavonoids exert multiple actions in biological systems depending on their chemical structure. Flavonoids containing at least one free aromatic phenolic group act as antioxidants (Ng TB et al, 2000). Both monocyclic and polycyclic phenolic aromates are known to interfere with the thyroid axis and to contribute to goitrogenesis. Folk medicines have taken advantage of the antithyroid action of flavonoids (Köhrle J, 2000).

Actions of a synthetic flavonoid F21388 show that it is a potent T₄-antagonist. In vivo administration of F 21388 to intact euthyroid rats immediately (after 3 minutes) but transiently displaced T₄ and T₃ from binding to serum transthyretin thereby increasing free T₄ and T₃ and decreasing TSH subsequently. These changes were reversible. The studies gave evidence that exogenous compounds rapidly disturbed thyroid homeostasis. Whether TTR interaction with naturally occurring flavonoids or their metabolites is of relevance in thyroid hormone physiology remains to be elucidated.

Other well documented multifaceted effects of flavonoids are:

1. Inhibition of TSH secretion
2. Direct inactivation of TSH
3. Inhibition of TPO
4. Competition for thyroid hormone binding to transthyretin
5. Potent inhibition of I 5' – deiodinase activity.

Flavonoid consumption may be very high in Vegas. The food habits of population in Gujarat State are very different from other States in India. There is a trend to consume fresh food three times a day everyday. Bread or cornflakes consumption is less or nil in poor population. The whole grain cereals such as wheat, jowar, pearl millet, maize and nagli are ground to respective flours that is kneaded and used to make pancakes. The rural population consumes at least one main meal (dinner) made from pearl millet pancakes. The snacks consumption is predominantly of fried (in groundnut oil) items made from chickpea flour and potatoes. Frozen food

except ice cream is not consumed at all. Every family consumes at least one kilogram each of tomatoes, potatoes, onions and two green vegetables with herbs and spices. The cooking oils used are groundnut (80%), cottonseed (10%) and sunflower (10%).

Food preparation does not damage flavonoids because they are very stable and resistant to heat, oxygen, dryness and acid but destroyed by illumination. Flavonoids present in foods were considered once non-absorbable but recent evidence indicates that may be absorbed in the large intestine after action by intestinal bacteria. It was also recently reported that the human small intestine possesses an ability to liberate the aglycone from quercetin glycosides. Nevertheless, quercetin glucosides are found to be present in human plasma without metabolic conversion.

Iodine deficiency disorders was assessed (GSI) in the tribal and rural population of Gujarat with the main IDD status indicators that is biochemical prevalence indicators. The urinary iodine determinations showed interferences in the colour reaction by substances in the urine that were detected as goitrogens in the urine. When these interfering substances were removed, the urinary iodine estimations showed mild iodine deficiency in Baroda District and moderate to severe iodine deficiency in Dang district.

Iodine deficiency disorders was assessed (GSII) in the tribal and rural population of Gujarat with all the prevalence indicators to measure the IDD status (biochemical (TSH and urinary iodine) and clinical (thyroid palpation and ultrasound). The biochemical indicators pointed to mild iodine deficiency in Baroda District and moderate to severe iodine deficiency in Dang district. The clinical indicators of measuring the thyroid size by ultrasonography pointed to a severe IDD in Baroda and Dang district. The thyroid enlargement was seen in almost 100% of tribal (Dang district) and rural (Baroda district) children. Thus there was a dissonance of biochemical and clinical indicators. This was attributed to the dietary goitrogens that played an important role in goitrogenesis in this population.

Pearl millet, a staple diet of rural population from the Baroda, contained flavonoids in the form of apigenin, vitexin and glycosyl vitexin. Flavonoid glycosides intake from pearl millet made major contribution to antithyroid effects and were primarily responsible for causation of large goitres in the affected population.

Another important factor contributing to goitrogenesis was malnutrition as evident from BMI. Dang tribal children were more malnourished than Baroda rural children. Still the thyroid enlargement was seen in almost 100% of tribal (severe iodine deficiency and gross malnutrition but less goitrogens) and rural population (mild iodine deficiency and less malnutrition but more goitrogens). This pointed to the multiplication of the confounding factors effects in the presence of iodine deficiency either severe or mild.

In children there was a significant correlation between thyroid volume and the indicator for thinness that is weight for height index and triceps skin fold thickness (chapter 8). In adults there was a significant correlation between thyroid volume and arm circumference that was smaller in them pointing to a long-term malnutrition. These facts point to a role of malnutrition in goitrogenesis.

An urban environment of Braoda district, Gujarat (GSIII) was studied selecting *iodine-replete* and *well-nourished* 6-15 year-old schoolchildren so that two main aetiological factors causing goitrogenesis were removed. Eighty five per cent of these children had enlarged thyroid in comparison to European 1997 reference adopted by WHO. This study showed that endemic goitre was highly prevalent in children and adults who were iodine replete and well nourished and pointed to a very strong role played by dietary goitrogens alone as iodine deficiency and malnutrition were not the confounding factors. This finding challenges the recent definition that IDD refers to all of the ill-effects of primary iodine deficiency in a population, that can be prevented by ensuring that the population has an adequate intake of iodine. Secondary iodine deficiency due to goitrogens

played a very important role in the development of goitre in these otherwise iodine replete children. It is usually stated and understood that presence of goitre in greater than 5% of schoolchildren can only be due to primary iodine deficiency (Dunn JT, 1999). Whereas, it is quite clear from our results that dietary goitrogens causing secondary iodine deficiency cause compensatory adaptation in the form of goitre in a much higher percentage than 5%.

Thyroid enlargement was however less than found in other areas of Gujarat studied in the past i.e. the tribal and rural children under the influence of all confounding etiological factors for goitre acting united (iodine deficiency, malnutrition and goitrogens) (see Table 8.20) but above European WHO reference ranges (1997). The most likely cause of goitre in this urban community was, therefore the high ingestion of dietary goitrogens.

Antithyroid effects of very small amount of purified flavonoid, quercetin and rutin; were investigated (Jeney et al). These were ingested orally dissolved in water for 5 months. Thyroid weights increased whereas the iodide content of the thyroids was reduced. Histological examination demonstrated typical thyroid hyperplasia. The investigators suggested the role of flavonoids in the aetiology of endemic goitre. If such small amounts of flavonoids can cause goitre then large quantities of almost all different types of flavonoids consumed daily for many years could result in the goitre seen in these iodine sufficient and well nourished children.

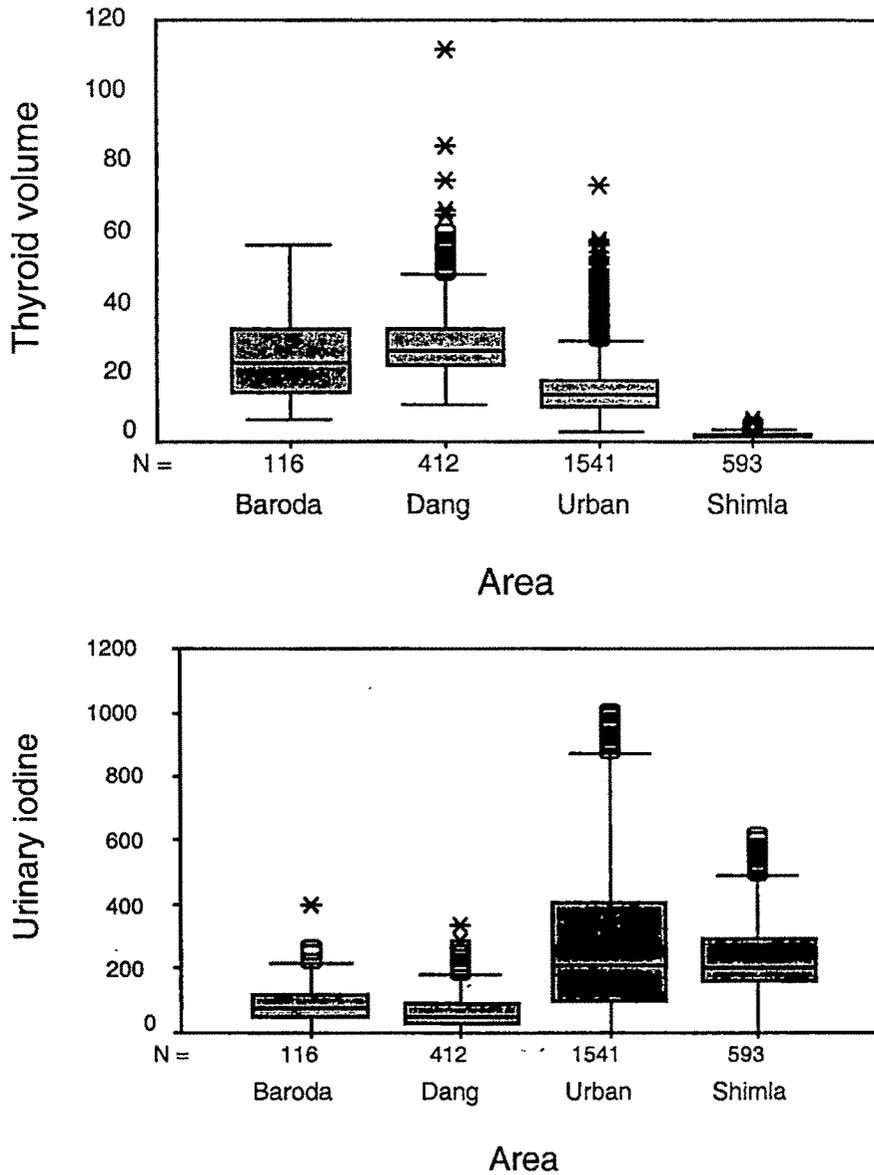
The vegetables produced and consumed in Gujarat (Western India) are exclusive to those consumed in all other parts of India (north, south and East India). We planned other study in the sub-Himalayan belt where these vegetables were not available. As Indian children weigh substantially lower and are shorter than same-age European children, it is possible that thyroid size in children if they are iodine replete, with low goitrogen intake, and well nourished, should be less than and much below European/WHO reference ranges.

This study signals the importance of identifying all known causes of endemic goitre in population surveys for IDD and for establishing appropriate controls and normative reference data. These principles are further explored in chapters 9 and 10 in other parts of India where goitrogen consumption was low. The US data highlight the potential pitfalls of using non-validated reference ranges for thyroid size developed in other environments.

Urinary iodine measurement is cost-effective for a developing country like India. There is too much of dietary diversification amongst different sections of a population. To get a better idea about the whole study group rather than generalisation of data from a small sample, a larger group (comprising of all the study subjects rather than in 25% of the sample) was studied for assessment of the severity of iodine deficiency disorders with biochemical parameters especially urinary iodine. It is necessary to present the overall distribution when the range is too big.

The 65% of the population usually belong to the adult age group. They take the meals at home whereas children may get at least one meal at school. If it is a government run school then the mid day meal is provided by government. If it is a private school for fee-paying students then the parents pay for good quality meal. The adult population survey conducted by household visits, would give the real community iodine consumption and non biased results.

FIGURE 11.1. COMPARISONS OF THYROID VOLUME AND URINARY IODINE IN 6-15 YEAR SCHOOLCHILDREN



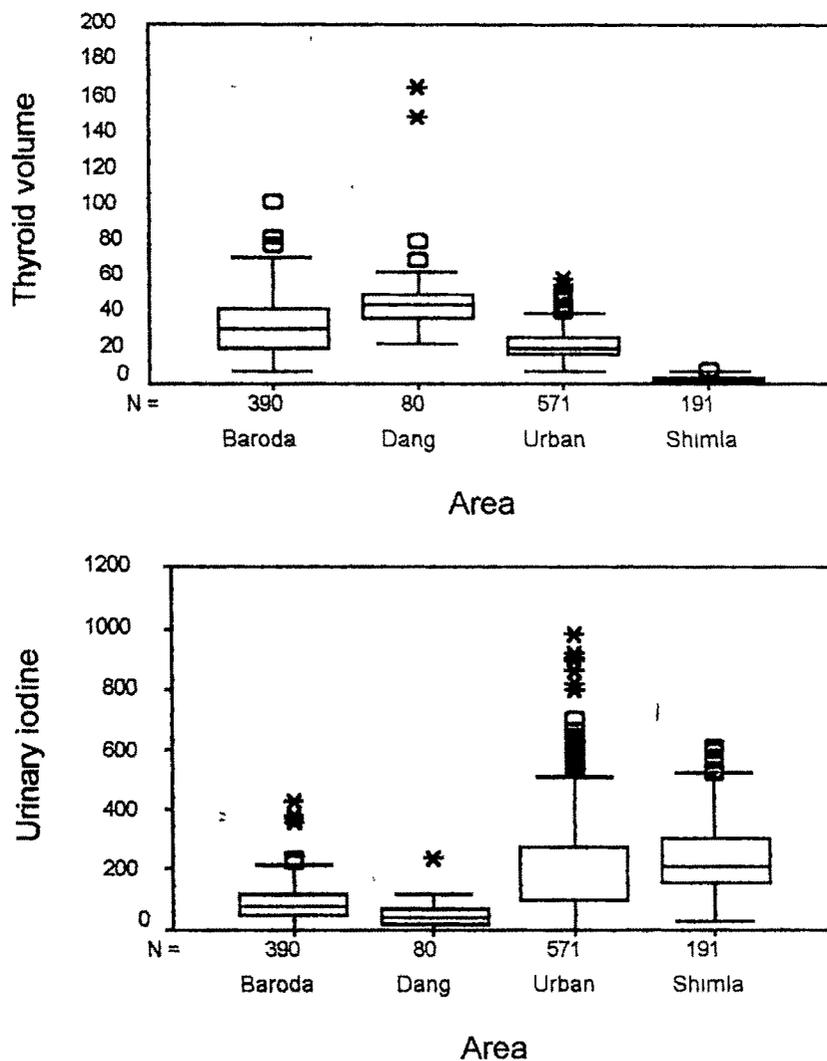
Area	Iodine status	Nutritional status and effect of goitrogens
Baroda	Mild iodine deficiency	Malnutrition and goitrogens positive effect
Dang	Severe iodine deficiency	Malnutrition and goitrogens positive effect
Urban	Iodine replete	Well nourished and goitrogens positive effect
Shimla	Iodine replete	Well nourished and no effect of goitrogens

We studied the IDD status of Gujarat, Tamil Nadu (TN) and Himachal Pradesh (HP). The Gujarat State tribal and rural and TN populations were mildly iodine deficient. There was a dissonance in assessment of IDD with the biochemical and clinical parameters. Clinical parameters of determining the thyroid size showed severe IDD and the biochemical parameters of urinary iodine measurement showed mild IDD. The tribal population was grossly malnourished. The dietary goitrogens played an important role in goitrogenesis. To determine the role of dietary goitrogens alone in goitrogenesis, iodine replete and well nourished urban population (both adults and schoolchildren) was assessed by clinical and biochemical parameters. The other states selected for study were HP on Himalayas as the population in this state had restricted use of vegetables because on mountains and TN on plain lands with no dietary restrictions. Thus there was limited intake of flavonoids and other goitrogens in HP. At the same time the pearl millet cereal and groundnut oil were not consumed.

WHO, UNICEF and ICCIDD published a document to set out principles governing the use of surveillance indicators in monitoring the epidemiology of IDD and implementing the recommended intervention – salt iodization – to prevent and control them in "Indicators for assessing IDD and their control through salt iodization." We do not agree with this document for few points after our study

1. The selection of target group for IDD surveys recommends that "adults in household" have less representativeness, low accessibility and low usefulness for other nutritional surveys but this recommendation seems inappropriate after present study conducted in this target group "adults in household". In the developing country like India 90% of total population lives in villages and farming is the main business. Due to hot climate most of this work is finished before noon. The surveys conducted in the day time (start after 10 AM) had unlimited representativeness because both men and women were at home. Thus accessibility was also unlimited as per the expenses and logistical constraints. Screening adult men women through household survey did provide an

FIGURE 11.2.COMPARISONS OF THYROID VOLUME AND URINARY IODINE IN ADULTS (> 15 YEARS)



Area	Iodine status	Nutritional status and effect of goitrogens
Baroda	Mild iodine deficiency	Malnutrition and goitrogens positive effect
Dang	Severe iodine deficiency	Malnutrition and goitrogens positive effect
Urban	Iodine replete	Well nourished and goitrogens positive effect
Shimla	Iodine replete	Well nourished and no effect of goitrogens

opportunity to establish a sample of a population vulnerable to IDD as mentioned in this document. Goitre rate does not provide **current** iodine intake hence after the age 30, it is a reliable indicator in these adults (especially women because this is the child bearing age), especially if ultrasonography determines the goitre rate. The tribal and rural adults showed presence of prolonged severe malnutrition that had significant negative correlation with thyroid volume.

2. "IDD tends to occur in geographic foci" This statement was not true for our study. IDD occurred in flat terrain zone of Baroda district as well as in hilly Dang district in Gujarat State whereas it was no more seen in Himachal Pradesh State on Himalayas.

3. "The size of the thyroid gland changes inversely in response to alterations in iodine intake, with a lag of 6 to 12 months in children and young adults (i.e. < 30 years of age". In our study the thyroid size did change inversely in all age adults. All the adults > 60 years had enlarged thyroid.

4. "It is recommended that a total goitre rate (TGR, goitre grades 1 and 2) of 5% or more in primary school children (age range approximately 6-12 years) be used to signal the presence of a public health problem. This recommendation is based on the observation that in a normal, iodine-replete population the prevalence of goitre should be quite low. The cut-off of 5% allows some margin of inaccuracy of goitre assessment and for goitre that may occur in iodine replete population due to other causes such as goitrogens and autoimmune thyroid disease. The previously recommended 10% cut-off level has been revised downwards since it has been shown that goitre prevalence rates between 5% and 10% may be associated with a range of abnormalities, including inadequate urinary iodine excretion and/or sub-normal levels of TSH among adults, children and neonates." Our study points to the inaccuracy of thyroid palpation to measure the thyroid size and we recommend measurement of thyroid size by ultrasonography and recommend thyroid ultrasound as the best clinical parameter to

determine IDD status in a population. We observed a goitre prevalence of 85% (with ultrasound in comparison to 1997 reference) in iodine-replete and well-nourished school aged children (6-15 years).

5. "Preferably children 6-12 years of age should be studied. There is a practical reason for not measuring very young age groups: the smaller the child, smaller the thyroid and the more difficult it is to perform palpation." We measured the thyroid volume by ultrasonography in school children of 4 to 5 years age group from nursery and kindergartens sections but due to the lack of a International normative thyroid volume reference, could not report the goitre prevalence rate. The median and 97th percentiles for both these age groups were higher than a 6-year old boy or girl in the International normative thyroid volume reference. However, this is the best target group for studying the thyroid volume relations with other parameters. Linear regression analysis between thyroid volume and some nutritional parameters (triceps skin fold thickness and weight for height index z-score) showed significant negative correlation. Linear regression analysis also showed significant negative correlation between thyroid volume and urinary iodine. Thyroid ultrasonography was not difficult at all in preschool aged children.

6. IDD may be present with TSH levels which are only mildly elevated. While further study of iodine replete population is needed, a cut-off of 5mU/l whole blood may be appropriate for epidemiologic studies of IDD. Our study indicated that the given upper limit of 5mU/l for TSH if decreased to TSH values of 3mU/l as upper limit, would possibly lead to a good correlation between both biochemical parameters i.e. urinary iodine and blood TSH values. 6% of the population had whole blood TSH values > 5mU/l. Significant correlation between UI and blood TSH was not seen with linear regression analysis. However, when the population was divided into urinary iodine bands then median blood TSH levels were higher in

those groups with lower urinary iodine level thereby suggesting that iodine depletion may increase TSH.

TSH was determined in first two surveys to assess IDD in Gujarat as one of the biochemical parameters. It was discontinued after first two surveys because in IDD newsletter (1999) the assessment of IDD was recommended by two parameters: urinary iodine and total goitre rate. Urinary iodine, should be used as the impact indicator and TGR as clinical indicator.

Does thyroid size always depend upon the body size?

There was a good correlation between thyroid size and various anthropometric parameters but only when none of the goitrogenesis aetiological factors like iodine deficiency, malnutrition and goitrogens existed in the environment. In Indian children having body size on par with WHO standards, the thyroid size was much small (almost half of WHO reported thyroid size as shown in chapter 10). The children having smaller body size in comparison to WHO standard had very large thyroid size as shown in chapter 6 that was due to iodine deficiency and malnutrition.

Why thyroid ultrasound is best parameter?

The urinary iodine determinations methods have many drawbacks. The auto analyser method does not separate out the interfering substances taking part in the colour reaction and determines them along with. The recent digestion method with ammonium per-sulfate also has many modifications. The results have shown variations of few micrograms when same sample is repeated. This means that if a sample gives a result of 15 $\mu\text{g/l}$ and upon repeat 40 $\mu\text{g/l}$, (which is always possible because the results depend upon standards calibrations and colour reaction with a mean of 27.5 $\mu\text{g/l}$). this changes the interpretation from severe to moderate iodine deficiency. The ICPMS being the gold method is not feasible for population survey.

The thyroid ultrasound method including intraobserver and interobserver variation of 15 to 30%, if shows the goitre in > 60% of subjects in comparison to WHO international reference, becomes a much reliable method for reporting the severity of IDD.

Intentional supplementation

1. Iodized salt
 - 1.1. All salt for human consumption (including food industry and bakeries).
 - 2.1. House hold salt only
2. Iodised oil
 - 2.1. Intramuscularly
 - 2.2. Oral
3. Iodination of drinking water or of agricultural irrigation water
4. Iodination of bread
5. Iodide tablets

Iodized salt

It is generally agreed that iodized salt should be the long-range objective of any programme. Indian food consists of pancakes, rice, pulses and vegetables and salt is added to all these parts of the meal. The poorest of the poor also consumes salt and pancake. But the question is how many people consume iodised salt? All the salt consumed is at the household level. The common man of India does not eat processed food like sausages, bakery product and cheese. The ban on the consumption of noniodized salt in Dang district has not helped in elimination of IDD. The mandatory salt iodization by the Himachal Pradesh Government has helped elimination of IDD from this severely iodine deficient belt.

Iodized oil

It is a valuable alternative wherever iodized salt consumption has failed. Iodised oil should be given Intramuscularly to the tribal population of Dang district. There is no shortage of trained personnel to give these injections in Gujarat. The pricing might be a concern to the government but the effect is lasting for three years. At the same time there is surety of iodine intake. The disposable syringes are available at very cheap rate which is 1 rupee per syringe equivalent to 1 US cent. Oral iodized oil has shorter duration of action and dependence on compliance. 48% of iodine given orally is lost in the urine within the first days in comparison to 6% by Intramuscular route.

Iodination of drinking water

Water is consumed by everybody due to hot climate hence drinking water is an obvious choice as an iodine carrier. There is always water shortage in India due to high population growth and the municipality-supplied water is used mainly for drinking by poor people. More than 70% of the Indians belong to poor category and these people get washing and cleaning water from wells or rivers. Rich or educated people have their own water bores and afford iodized salt hence government that mainly comes to power on poor votes should iodinate drinking water.

Whenever iodine deficiency exists efforts must be undertaken to eliminate it by iodine supplementation. At a first glance iodine supplementation seems a simple matter, but experience worldwide has shown that the goal of iodine sufficiency is not easily achieved. The main methods used world wide are iodization of salt and administration of iodized oil.

A big step is indicated to eliminate IDD from India. The universal salt iodization did not help and the ban to iodize all salt for human consumption has been lifted up since September 2000. We can't cross this IDD chasm in too small steps and we suggest a big step of the mass iodization by

Lipidol (oral for urban educated population and injectable for rural and tribal uneducated population).

The change always takes place. The direction and degree of change is rapidly altering the courses which were thought be perennial and beneficial. The tempo of technological advances has quickened. The need for more and more information has accelerated. Discoveries not yet imagined will be made. Health related sciences are changing with leaps and bounds. We can't be Rip Van Winkles in this world where the power of ideas and the power of thought are being recognized.

Awake, arise and sleep not till the goal of eliminating IDD from India is achieved be our slogan and our motto.