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RESEARCH ARTICLE

INCIDENCE OF GOITRE AND OTHER IODINE DEFICIENCY DISORDERS IN SELECTED VILLAGES OF DISTRICT CHAMOLI IN GARHWAL HIMALAYA

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Abstract

Garhwal Himalaya was known to be iodine deficient. It was reported that 25% of population lived here were mentally retareded. To overcome this Govt of India lauches Salt Iodization program. But due to tough terrain it was not distributed adequately. In the present work we assess the impact of iodize salt in the population of Chamoli Garhwal .

Total 11 villages were surveyed for Goiter and IDDs. Thyroid hormone was measured in blood serum and iodine content was measured in Urine.

We found that Goiter and IDD was very low in the region and hormonal values were on normal side. Despite the populations were not using iodized salt adequately. We concluded that iodine is important for metabolism but nature is sufficient to provide. There is no need to given externally.

Introduction:-

Iodine is an essential micronutrient required for the synthesis of thyroid hormones (Thyroxine, T_4, Triiodothyronine, T_3). Deficiency of iodine in the body is a major cause of insufficient thyroid hormone synthesis. Thyroid hormones influence most vital functions in the human body and play crucial role in growth, development and metabolism. Underproduction of thyroid hormones, referred to as hypothyroidism, leads to a series of abnormalities in physiology. The spectrum of these complications is known as iodine deficiency disorders (IDD i.e. goitre, cretinism, mental retardation, spastic diplegia, deaf/mutism, strabismus).

In the Indian subcontinent an iodine deficient endemic goitre belt has been identified passing through various districts along the southern slopes of the Himalaya extending from J & K in the North West to Arunachal Pradesh, Nagaland and Manipur in the East. Rough distribution maps for goitre in India were prepared for the first time by McCarrison 1915. Later studies on goitre were stated from some parts of Indian sub continent (Karmarkar et. al. 1974, Agarwal and Agarwal, 1983, Udupa et.al. 1983) and now extended to Pondicherry (Kapil et.al 2002), Tamilnadu (Kapil et.al 2004), West Bengal (Biswas et.al 2004 & Sen et. al 2006), Kerala (Kapil et.al 2006). But detailed studies in the Himalayan context are, surprisingly very few e.g sub – Himalayan area in eastern U.P, Panjab, Kangra valley in Himanchal Pradesh and Tarai area of Uttarakhand (Sooch and Ramalingaswami 1965, Kapil 2003, 2007 and Toteja et al 2004) and higher reaches of Uttarakhand (Lakhera & Chandola-Saklani A 1989, Bamola 2005).

Almost no scientific base line data are available on the status of iodine deficiency in this Himalayan region which is a pre-requisite for the development of adequate IDD management plans for the region. National Goitre Control Board (NGCB) launched National Goitre Control Programme (NGCP) in 1962 to carry out surveys which, however; appear to be inadequate for the want of appropriate data.

Prophylactic programs involving iodine fortification in diet (salt) and iodized oil injections have been implemented in large areas of China, Andean South America, Central Africa and even India. Following the examples of some other countries in India in 1983 the Government took a policy decision to iodise the entire edible salt in the country

by the year 1992. The program started in April 1986 in a phased manner. Ever since then, the approach has been to increase production, demand and supply of iodized salt. All States and Union Territories have been advised to ban the sale of non-iodized salt under the Prevention of Food Adulteration (PFA) Act, 1954. Claims are periodically made by Government on eradication of goiter through Universal Salt Iodization program (USI).

Adequate goitre & IDD management plans through community based prophylactic programs first require sufficient prerequisite infrastructural scientific data base. In the Indian context this data base is inadequate and decisions have been taken two decades ago on the basis of conclusions drawn from case studies elsewhere, often not scientifically sound.

Our earlier studies were of an extensive nature drawing data from various districts (Lakhera 1990 and Bamola 2005) that indicate Goiter was invariably found to occur in pockets interspersed with areas not affected. The present intensive study focuses on a few villages (district Chamoli) with systematic in depth investigation on various parameters often from same individuals.

Also, of late since methods for iodine determination have been available, more and more IDD surveys are based on iodine measurement without concomitant data on actual prevalence of goiter and even less on the associated IDD, information on which is conspicuous by its paucity.

In the present work we conduct intensive studies based on 11 villages in Chamoli of Garhwal region on the current status of thyroid hormone disorders, hormonal profiles of T3 & T4, and iodine measurements in urine.

Material and methods:-

Study Site:-

Garhwal Himalaya (29^{O} 26 to 31^{O} 5 N & 78^{O} to 88^{O}E, 30090 sq km) exhibit great altitudinal variations (400m to above 4000m), varied topography and climate. The mountain populations in Garhwal Himalaya are spread out in patches in remote interior. Living in the difficult rough terrain here is a challenge with climatic extremes ranging from sub-tropical heat to freezing temperatures of tundra. Access to habitations is often through hilly tracks, quite often walking distance of several kilometers from the road head.

Garhwal constitutes six districts including five in hills (Chamoli, Rudraprayag, Tehri Garhwal, Pauri Garhwal and Uttrakashi) and one in foothills (Dehradun). The area borders on China in north, Himachal Pradesh in northwest & Uttar Pradesh in southwest. The economy is entirely agriculture based but self cultivation is not sufficient to fulfill the annual food requirements, sufficient in majority of cases for less than six months. The main crops are pulses, millet, grain, vegetables.

Health camps were organized in association with Health Department, Uttrakhand in 11 selected villages in district Chamoli viz., Syun, Bamaru, Ratoli, Salla, Gonna, Bayara, Nijmulla, Nargoli, Ludho, Kamyar, Awani of Garhwal Himalaya to assess the IDD (goitre, strabismus, hearing defects, speech defects, deaf mutism, spastic diplegia, dwarfism, cretinism, mental retardation). Total 1106 individuals were examined physically in district Chamoli. More than 10% of total village's population was surveyed except in 2 cases. Awani and Bayara where only 6% & 5.3% could be surveyed.

Individuals were examined physically for the incidence of IDD if any. Surveyed villages were situated between 1600-2200 m altitudes, most of them in remote interior (up to 14 km away from road head). Few villages are connected by regular roads but access to majority is by walking tracks only.

The survey team composed of four to six members. Prior to any camp Govt. Health Department and Chief Medical Officers of the respective districts were contacted to ensure the participatory help of local Doctor / paramedical staff in the camp. To enlist local help and cooperation during the camp village '*Pradhan'* (chief of the village) were contacted. During the camps organized for the assessment of IDD people were also screened for different physiological abnormalities & infectious diseases using modern diagnostic tools.

Adequate health care and diagnostic facilities in most of the areas of rural Garhwal Himalaya are poor. Existing Govt. Health Centers as well as Private Diagnostic Centers are few, limited to larger towns and poorly equipped in expertise and infrastructure. People have to travel far to get simple manual routine diagnostic tests done. Considering the poor status of diagnostic facilities and hence health care system in this rural area of Uttaranchal, our

lab (with the help of infrastructure available through DoEn, CSIR, UGC projects) has set up an Endocrine & Immunodiagnostic Facility in collaboration with the Base Hospital, Srikot, Srinagar. This Facility as a humble service to the society has provided desperately needed diagnostic facility to the region for the diagnosis of different endocrine disorders, infectious & nutritional diseases through health camps various physiological and biochemical abnormalities. Following tests are available: T4, T3, TSH, LH, FSH, Prolactin (RIA based), FT4, FT3 (ELISA based), ToRCH, Hepatitis, HIV, Staphylococcus, Tuberculosis Screening, Typhoid Screening, ASO, RA, CRP (ELISA based).

Goitre grading:-

Methods of carrying out goitre surveys have shown considerable variation in different parts of the world. Classification based on grading are the method of choice for large population surveys because of rapidity and simplicity. Since Kimball and Marine (1918) differentiated between "slight", "moderate" and "marked" enlargements, many other qualitative classifications using diverse criteria have been proposed (Clements et al 1968, Delange 1974, WHO 2001). The method of Perez et al 1960, which incorporates salient features of these classifications, was proposed for international use in 1960 by the world Health Organization (WHO). The method of Perez et al (1960) was subsequently modified by different observers and organizations from time to time (IDD Newsletter 1987). In the present surveys grading method of WHO 1979, as modified by Pan American Health Organization, 1986 & WHO 2001, has been employed. A brief description follows:

For grading of goitre or detection of thyroid enlargement the individual is asked to sit on a chair and neck of individual is carefully examined for any sign of visible enlargement of thyroid. The individual was then asked to look up and to fully extend the neck. This activity pushes the thyroid gland forward and makes any enlargement more obvious. Finally the individual is palpated from behind by gentle sliding of thumb along the side of the trachea between the cricoid cartilage and the top of the sternum. Both sides of the trachea were checked and the size and consistency of the thyroid gland were noted. Where necessary, the individual was asked to swallow some water during examination. The thyroid moves up on swallowing. The size of each lobe of the thyroid was compared to the tip of the thumb of the individual being examined. Goitre was graded according to the following classification.

Grade 0 - No palpable and visible goitre
Grade Ia- Thyroid distinctly palpable and definitely larger than normal but not visible with normal and extended neck
Grade Ib - Thyroid easily palpable and visible with extended neck
Grade II - Thyroid easily visible with normal neck (without extended)
Grade III - Goitre visible at a considerable distance.

Each individual was examined for physical appearance and thyroid size using PAHO/WHO recommended method. Individuals were graded for goitre according to their thyroid enlargement viz. grade 0, Ia, Ib, II, III. Information on pathophysiological symptoms of the individual, sex, age, diet, source of drinking water & salt samples were also collected.

Iodine deficiency disorders (IDD):-

Intensive information on incidence of iodine deficiency disorders (IDD) were generated through door to door survey with questionnaires in selected villages of district Chamoli in Garhwal Himalaya Observations were also made on age, sex, affected since when. Data were obtained from the family directly or immediate neighbors with more than one affected individuals & with three or more different generations.

Data on prevalence of neurological disorders (neurodevelopment & neuromotor) associated with iodine deficiency were recorded according to following symptoms (Michaelides and Mooore 2004, Hetzel 1989).
Strabismus - Misalignment of eyes
Cretinism - Short stature, some hearing defect & unintelligible speech, mental dullness, Physical appearance is not normal as compared to other people of area
Dwarfism - Stunted growth
Spastic diplegia - Weakness in legs, spasticity, unable to stand or walk without the support of hands on ground hand support, deaf Mute
Deaf mute - Speech & hearing defect
Hearing defect -Hearing capability was assessed during normal conversation in group

Speech defect - Stammering in speech/ unintelligible speech.
Mental retardation - Loss of intellectuality

Hormonal profile:-
Blood samples were collected from individuals of selected villages of district Chamoli of Garhwal Himalaya for the measurement of circulating thyroid hormone levels. Help of the Govt. Health Department Uttranchal, CMOs/ CMS of district Chamoli, local medics & health visitors was enlisted for blood sampling.

Intravenous blood samples were obtained from individuals of different villages of district Chamoli after informed consent. Blood samples were collected irrespective of sex and age from a subset cross-section of population. During sampling individuals were palpated and observed for the assessment of thyroid size and graded for goitre.

Collection of blood samples:-
Intravenous samples were obtained through vaccutainer/ disposable syringe. Blood was allowed to coagulate at room temperature and followed by centrifugation for serum separation. Since most villages are situated at considerable distance from motor road, a portable centrifuge was carried everywhere. Separated serum samples were immediately placed in a cooling kit (0-2^{O}) filled with coolant bags ice and salt. Samples were transported (invariably reached destination within 5-6 hours) to the BARC approved Radioisotope Laboratory, Reproductive and Wild life Biology Unit Department of Zoology, HNB Garhwal university Srinagar Garhwal, where they were aliquoted into numbered eppendorf tubes and stored at -20^{O}. Care was taken to avoid direct contact with blood samples.

Hormonal Estimation:-
Thyroxine (T4) and tri-iodothyronine (T3) were measured in serum by Radioimmunoassay by the in house method (Chandola and Pathak 1980, Kar and Chandola –Saklani 1985) based on techniques developed by Ekins 1960, 1982, 1986 and Brown et al 1970). High specific activity ^{125} I-T4 and ^{125}I-T3 were obtained from BRIT Bhaba Atomic Research Center, Trombay. Quality controls were used from BRIT kits T4 & T3. Precision profile (Ekins 1960) of each assay was developed for validations. All measurements were within 10% range of precision profile (Fig).

Hormone Standards:-
Sodium salt of T4 and T3 (Sigma) were stored at -20^{O}C securely sealed to prevent water absorption. At the time of preparation the salt was allowed to come to room temperature prior to weighing out the substance. This was dissolved in propylene glycol/water 50:50 rendered alkaline (ph 8.0), 0.2 ml was removed and was diluted with 2ml of 0.04M sodium hydroxide and the extinction measured against a solvent blank at 320nM using a mercury vapor lamp and a light path of 19mm. The exact concentration of sodium T3 or T4 in the solution was calculated. Remaining solution was aliquoted in approximately 200µl volume in ampoules, which were sealed and stored at -20^{O}C until required. At the time of assay working standards were prepared following further dilutions in barbitone buffer containing 0.2% bovine serum albumin.

Urinary iodine:-
Total 68 urine samples (43 from male & 25 from females) were collected in studied villages of district Chamoli. Urine samples were obtained from each individual in clean sterilsed bottles. These samples were immediately placed in a cooling kit (0-2^{o}) filled with ice. Samples were transported to the laboratory (Reproductive and Wild life Biology Unit department of Zoology, HNB Garhwal university Srinagar Garhwal,(invariably reached destination within 5-7 hours) samples were stored at -20^{o} until assayed.

The urinary iodine content in urine was measured by Sanger-Kolhoff reaction (Sandell et al 1937). Urine is digested with ammonium persulfate. The released free iodine acts in the presence of the arsenius solution as a catalyst in the reduction of Ceric ammonium sulfate (yellow) to Cerous form (colorless) in urine samples. By the rate of color disappearance the measurement of total iodine content were done.

Ammonium persulfate was added to the urine sample followed by Incubation at 100^{O}C for 1 hr. After that arsenius solution was added and the mixture is shaken. Ceric ammonium sulfate was added at successive intervals of 15-20 second and the tubes left when OD was read at 420. nm (Dunn et al 1993). Quality controls were run simultaneously.

Results:-

Overall Incidence of IDD in 11 villages:-

Results are summarized in table 1 and fig 1

- Incidence of goitre was less than 0.2% of surveyed populations.
- Other IDDs viz cretinism, mental retardation, deaf mute, spastic diplegia ranged between 0.1 to 0.2% of surveyed population. Speech & hearing defect ranged from 0.67 to 0.81%. Strabismus showed highest overall incidence i.e 4.5%.
- Most prevalent disorders were strabismus > hearing defect > speech defect in that order.

Village wise incidence:-

Results are summarized in table 2 and fig 2

- Goitre ranged from grade 0 to Ia, non visible & palpable goitre was 0.2% of surveyed population.
- No visible goitre was recorded from any villages.
- Among IDDs strabismus was highest in % incidence as well as distribution followed by > hearing > speech defect & other form of disorders.
- Among the villages only two, Gonna and Nijmulla exhibited majority of IDDs.
- In three villages i. e Ratoli, Salla and Awani IDD was entirely absent except for strabismus.

Age group wise incidence:-

Results are summarized in table 3 and fig 3

- In general all age groups were affected by strabismus, speech and hearing defect.
- Of all the IDD, strabismus was most prevalent in younger population ($p<.01$ 1-15 years vs older groups).
- There was no relationship of neurological disorders with % incidence of total goiter (Fig 4).

Correlation among other IDDs:-

- Hearing defect and spastic diplegia show significant +ve correlation ($p<0.001$).
- Dwarfism and cretinism also show a significant +ve relationship ($p<0.001$).
- This indicates a co-existence of hearing & spastic diplegia as well as dwarfism and cretinism (Fig 5).

Hormonal Profiles:-

Results are summarized in table 4, 5 and fig 6

Overall hormonal profile

- T3 and T4 levels were within normal range (T3 .7 to 2 µg/ml, T4 5.5 to 13.5µg/ml) in both male & female.
- T4 levels were significantly higher in females as compared to that in males ($p< 0.007$).
- T3 levels were significantly lower in females as compared to that in males ($p< 0.004$).

Hormonal Profiles in Age group wise comparison:

Results are summarized in fig 7

- No significant differences are indicated in serum T3 & T4 concentration as a function of age (p ns).

Urine analysis:-

Results are summarized in fig 8

- Median urinary iodine excretion is less than 100.0 µg/l of iodine, 35.3% of the samples had urinary iodine excretion level less than 50.0µg/l and 47.1% had more than 100µg/l in iodine.

Discussion:-

Goitre:-

Results indicate that overall incidence of total goitre(non visible + visible goitre) was 0.2% in different villages of district Chamoli (Table1). According to WHO criteria (WHO 2001), this is indicative of no iodine deficiency :

Total goitre rate	WHO criteria
0.0 – 4.9%	No iodine deficiency
5.0 – 19.9%	Mild iodine deficiency
20.0 – 29.9%	Moderate iodine deficiency
More than 30%	Severe iodine deficiency

Only three sets of data are available from western Himalaya for comparison. In the first ever report of Himalayan goitre (Stott et al 1931) a high prevelance of goitre was reported in this region (36%). During that time there was no widely accepted grading system of thyroid enlargement. Next report was that from National Goitre Control Board (NGCB), i.e 25.4% in Tehri and 25.4% in Pauri (1983) and 3.5% in Tehri, 20.69% in Pauri and 40% in Chamoli (1989).However surveys do not provide details of endemics surveyed, sample size, methodology, population structure, grading of goitre etc. Subsequent data were reported from our laboratory (Lakhera & Chandola Saklani 1989, Lakhera 1990, Bamola 2005). These data including the present studies indicate a significant decline in overall goitre incidence over the years.

Comparing present data with those obtained 2 – 3 decades earlier or recently from other regions of Himalaya and sub – Himalayan endemics a much lower incidence was indicated e.g goitre prevalence was reported 50% in Simla, Himanchal Pradesh, North Western Himalaya, (Sooch et. al. 1973), 13-47% in school children in Manipur East Himalaya, (Agarwal and Agarwal 1983). 41-85% goitre in Bhutan (Kochupillai et. al. 1986), 78% prevalence in school children in Gilgit and Chitral valley Pakistan. Recent surveys reported 31.02% goitre in Himalayan foothills, Dehradun and 19.8% Kangra in H P (Toteja et. al. 2004, Kapil et. al. 2007).

Incidence of goitre in our area was also low when compared to that in some other endemics in different parts of the Indian sub- continent, viz.,17 -73% prevalence was reported from school children in Champaran (Agarwal and Agarwal 1983), 25.9% in West Bengal (Biswas et. al. 2006) & 22% in Madhubani (Pandav et. al 1986). The prevalence of goitre in North east India was reported 34.96% (Chandra et. al 2006). 46 -67% goitre in Burma and 30-35% in Bangladesh (Stone 1984).

What is most striking is that there has been no iodine prophylaxis in Garhwal region to explain the low incidence of goitre. Despite the ban on the sale of uniodised salt our surveyes showed majority of local inhabitants continue to use crude crystalline noniodised salt which they obtain once a year is barter with local produce (see section II).

Obviously either the normal iodine intake from other food sources has increased or iodine deficiency is not a severe factor causing goitre**.**

Other IDDs:-

Cretinism:-

Results indicate 1.56 % cretinism in village Bayara and 0.54 % in village Syun (Table 2). Cretinism was absent in all other surveyed villages of district Chamoli. Reported figures on the incidence of cretinism are relatively low when compared to that from different parts of Indian subcontinent & other parts of the world. About 7 decades earlier Stoott et. al (1931, 34) reported high incidence of cretinism in the area, Lakhera (1990) found a drastic decline in some of the same villages studied. Bamola (2005) also found overall decline in the last decades. An overall average of 0-13 % population was suggested to be affected by cretinism in India, 6-18 % in Burma and 0-10 % in Indonesia (Pandava and Kochupillai 1986). But these figures were obtained generally through extrapolation of limited population & not based on actual surveys. Almost no direct data are available on the incidence of IDD other than goitre, rendering comparisons difficult. In Bhutan cretinism was found relatively common ranging to the extreme of 35% in parts of Chirang district (Kochupillai 1986 et. al). All these reports have demonstrated iodine deficiency as a major causative factor of endemic cretinism. Severe iodine deficiency, exposure to dietary goitrogens and protein malnutrition have been suggested to be the chief etiological factors for endemic cretinism (Stanbury 1972)

Current results showed that cretinism is confined to pockets and generally to younger population (<35 years). Perhaps cretins have a shorter life span.

Among the developmental actions of thyroid hormones their effect on maturity of neurons is well documented (Wilson, 1974, DeGroot, 1979, Delong, 1987, Haddow, 1999, Bernal et al, 2003). Earlier it was believed that foetoplacental unit acted as a barrier for T_4, as for many other substances and thus goitre considered more of a cosmetic problem. . But with the demonstration that T_4 crosses over placenta to developing foetus (Silva, 1985, Ekins et. al 1986, Zoeller, 2003) there has been a spurt of papers indicating involvement of thyroid hormone in human brain maturation (Fisher et al.1998, Delange 2002). Thyroid hormones have been detected in human coelomic and amniotic fluids as early as 8 weeks of gestation, before the onset of fetal thyroid function at 10-12 weeks (Zoeller et. al. 2002). The functional consequences of deficits in thyroid hormone during fetal development

are suggested to result in two kinds of pathological conditions. The first is cretinism, a condition usually associated with severe iodine insufficiency in the diet and the second is that of subtle, undiagnosed maternal hypothyroxinemia (Zoeller et. al. 2002). Recently Godbole and his group have shown hypothyroidism causes alterations in mitochondrial morphology, gene expression and release of apoptogenic proteins during rat cerebellar development.

There are two forms of cretinism, sporadic and endemic. The 'sporadic' form occurs in population with an incidence of approximately 1 per 4000 births (Pharoah, 1985) and is distributed in all parts of the world & is used to refer to congenital hypothyroidism not associated with endemic cretinism or iodine deficiency. 'Endemic cretinism' is confined to certain population/ areas of the world and associated with endemic goitre.It was Mc Carrision (1908) who first clearly distinguished two types of endemic cretinism from the erstwhile Northwest frontier of India (now the Karakoram Mountains in Northwest Pakistan) – 1) the neurological & 2) the myxoedematous – in a series of 230 patients. Neurological cretinism is characterized by extreme mental retardation, deaf-mutism, squint, impaired voluntary motor activity, spastic diplegia and spastic rigidity usually affecting the legs with a characteristic goitre. Myxoedematous cretinism is characterized by less severe mental retardation and all the major clinical symptoms of persistent hypothyroidism e. g. dry swelled skin & tongue, deep hoarse voice, apathy & mental deficiency. In our population cretinism appeared to be predominantly of neurological not myxoedematous origin.
Incidence of Mental retardation was observed 0.73%in village Nijmulla, 1% in village Kamyar, 0.54% in village Syun. % incidence of mental retardation was high in female as compared to male individuals (Table2).

Incidence of Dwarfism was observed 1.56% in village Bayara, 0.54% in village Ludho (Table 2). % incidence of Dwarfism was high in male as compared to female individuals Dwarfism and cretinism also show a significant ($p<0.001$) +ve relationship. This indicates a co-existence of these traits.

Strabismus, Hearing defect, spastic diplegia, speech defect, deaf mute:-
Results indicate1.47% strabismus in village Nijmulla, 4.93% in village Gonna, 9.3% in village Bayara, 6 % in village Ratoli, 1.6% in village Nargoli, 14% in village Kamyar, 5 % in village Awani, 2.3% in village Bamaru, 3.33% in village Salla, 1.78 % in village Syun (Table 2).

Strabismus was the most prevalent of all IDDs in percent incidence as well as distribution. Strabismus was more prevalent in female as compared to male individuals and also found prevalent in lower age groups (1-15 years).
Incidence of Hearing defect was observed 3.6% in village Nijmulla, 3.08% in village Gonna, 1% in village Kamyar, 0.76% in village Bamaru, 0.54% in village Ludho. % incidence of hearing defect was high in female as compared to male individuals and % incidences were increased in higher age groups.

Percent Incidence of Spastic diplegia was observed 0.73% in village Nijmulla, 0.61% in village Gonna and only found in female. In our study there was no relationship of neurological disorders with % incidence of total goitre.

However among other IDDs, hearing defect and spastic diplegia show significant ($p<0.001$) +ve correlation

Incidence of Speech defect was observed 3.70 % in village Gonna, 3.22 % in village Nargoli, 0.54% in village Ludho. % incidence of speech defect was high in female as compared to male individuals and found in all age groups but % incidence was found high in lower age groups (1-15 years). % Incidence Deaf mute was observed 1.6% in village Gonna.

In the present population same individuals were screened for goitre & IDDs. In the near absence of goitre the prevalence of IDDs known to be associated with iodine deficiency was rather intriguing. This called for a thorough investigation of iodine status in these populations.

Hormonal profile:-
Tri- iodoithyronine (T3) and thyroxine (T4) are two major circulating hormones secreted from thyroid gland. Presence of iodine on the outer ring (3' 5' position) of T4 & T3 imparts biological activity to the molecules. Present results indicated low incidence of goitre and associated IDD in Western Himalaya despite presumed environmental iodine deficiency. To assess physiological implications of these findings thyroid hormone profiles were obtained from a subset of current population.

Overall hormonal profile from two villages in district Chamoli indicates high normal T4 and normal T3 levels. These values are in harmony with the near absence of goitre in the area. The observed normal thyroid status, however, is in contrast with the relatively high incidence of overall IDDs as reflected by neurodevelopmental & neuromotor disorders in the population. Since deficiency of iodine brings about effects through thyroid, which in this case is normal, doubts are raised as to these disorders attributed being to iodine deficiency. Obviously iodine deficiency disorders need not necessarily be caused by iodine deficiency.

What then, are the other possible factors which are responsible for observed high incidence of IDDs in the absence of goitre in the area? Dietary, physiological or genetic? These questions are addressed in section II of this dissertation.

Iodine content in urine:-

Most iodine absorbed in the body eventually appears in the urine. Therefore urinary iodine excretion (UIE) is a good marker of dietary iodine intake. Results indicate that median urinary iodine excretion is less than 100.0 µg/l of iodine. 35.3% of the samples had urinary iodine excretion level less than 50.0µg/l and 47.1% had more than 100µg/l in iodine. According to the WHO (WHO 2001) criteria these levels are indicative of only mild iodine deficiency.

Urinary iodine excretion is increasingly becoming the favored method to asses iodine deficiency & information is now available on several populations e.g West Bengal (Biswas 2004), Kangra valley in Himachal Pradesh (Kapil et. al 2007), tarai region of Uttrakhand (Toteja et al 2004) and Tamil Nadu (Kapil 2004).

The mild iodine deficiency status as observed through UIE is indeed striking since Himalayan goitre belt of which the area is an integral part, has long been considered severely iodine deficient.

Table 1:- Incidence of Overall iodine deficiency disorders (IDD) in district Chamoli (based on overall averages of 11 villages surveyed).

Total village Surveyed	11		
Total population	10300		
Surveyed population	1106		
IDD	**mean ±se (% of surveyed population)**	**Male mean ±se (% of surveyed population)**	**Female mean ±se (% of surveyed population)**
Visible goitre	0	0	0
Non visible (Palpable)	0.2	0.13	0.06
Speech defect	0.67±0.41	0.41±0.25	0.25±0.17
Mental retardation	0.2±0.11	0.06	0.04
Cretinism	0.19±0.14	0	0.19±0.14
Dwarfism (normal with any abnormalities)	0.14	0.14	0
Deaf mute	0.14	0.05	0.05
Spastic diplegia	0.12±0.08	0	0.12±0.08
Hearing defect	0.81±0.39	0.55±0.35	0.27±0.14
Strabismus	4.51±1.23	2.3±0.69	2.4±0.79

Table 2: Incidence of iodine deficiency disorders (IDD) in inhabitants of district Chamoli (11 villages)

Village	Total Population	No of families Surveyed	Surveyed population	%Surveyed population	**IDD Percent of surveyed population**								
					♣Goitre	Cretinism	Spastic diplegia	Mental retardation	Deaf-mute	Hearing defect	Speech defect	Strabismus	Dwarfism
Nijmulla	1200	18	136	11.33	0	0	0.73	0.73	0	3.6	0	1.47	0
Gonna	2000	23	162	8.1	0	0	0.61	0	1.6	3.08	3.70	4.93	0
Bayara	1200	10	64	5.3	0	1.56	0	0	0	0	0	9.3	1.56
Ratoli	800	14	100	12.5	0	0	0	0	0	0	0	6	0
Nargoli	600	10	62	10.33	0	0	0	0	0	0	3.22	1.6	0
Kamyar	1200	20	100	8.33	0	0	0	1	0	1	0	14	0
Awani	1000	11	60	06	0	0	0	0	0	0	0	5	0
Bamaru	1000	24	130	13	2.3*	0	0	0	0	0.76	0	2.3	0
Salla	300	08	30	10	0	0	0	0	0	0	0	3.33	0
Syun	600	33	185	30.83	0	0.54	0	0.54	0	0	0	1.78	0
Ludho	400	13	77	19.25	0	0	0	0	0	0.54	0.54	0	0

Table 3:- Percent Incidence of various iodine deficiency disorders (IDD) in individuals of different age groups in villages of district Chamoli.

Age group	**Strabismus mean ± se**	**Hearing defect mean ± se**	**Mental retardation mean ± se**	**Spasticdiplegia mean ± se**	**Dwarfism mean ± se**	**Speech defect mean ± Se**	**Deaf mute mean ± se**	**Cretinism mean ± se**	Goitre **mean ± se**
1-15	2.67±0.85	0.23±0.12	0	0.06	0.14	0.37	0.05	0	0
16-30	1.53±0.5	0	0.04	0.05	0	0.16	0	0.14	0.2
31-45	0.66±0.37	0.13±0.08	0.06	0	0	0.11	0.05	0.04	0
46-60	0.13±0.09	0.2±0.02	0	0	0	0.14	0	0	0
61-above	0.09	0.3±0.13	0	0	0	0	0	0	0

Table 4:- Serum thyroxine (T4), triiodothyronine (T3) concentration in habitants of two villages of district Chamoli

Villages	**T4 (ng/ml) mean±se**	**T3 (ng/ml) mean±se**
Bamaru (98) + Syun (49)	124.29±3.74	0.82±0.05

Table 5:- Serum thyroxine (T4), triiodothyronine (T3) concentration in males & females in two villages (Syun & Bamaru) of district Chamoli.

Male **(92)**		Female **(55)**	P values male vs female
T4 (ng/ml) mean±se	117.54±4.28	134.65±6.83	< 0.007
T3 (ng/ml) mean±se	0.89±0.08	0.69±0.06	< 0.04

Figures in parenthesis indicate number of individuals.

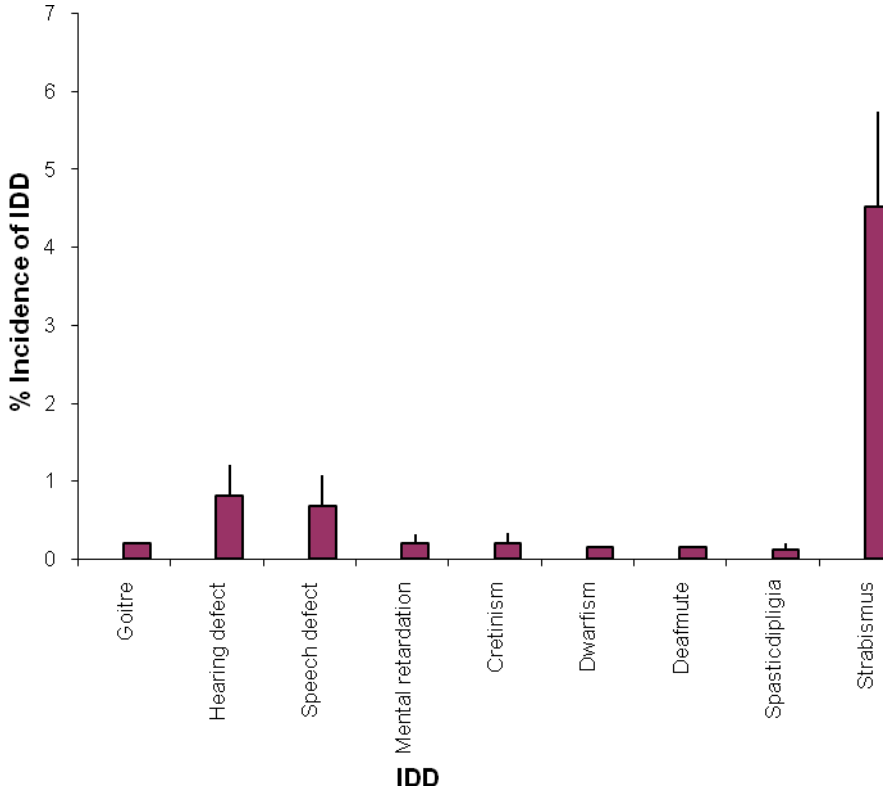

Fig 1 % Incidence of IDDs in district Chamoli in Garhwal Himalaya

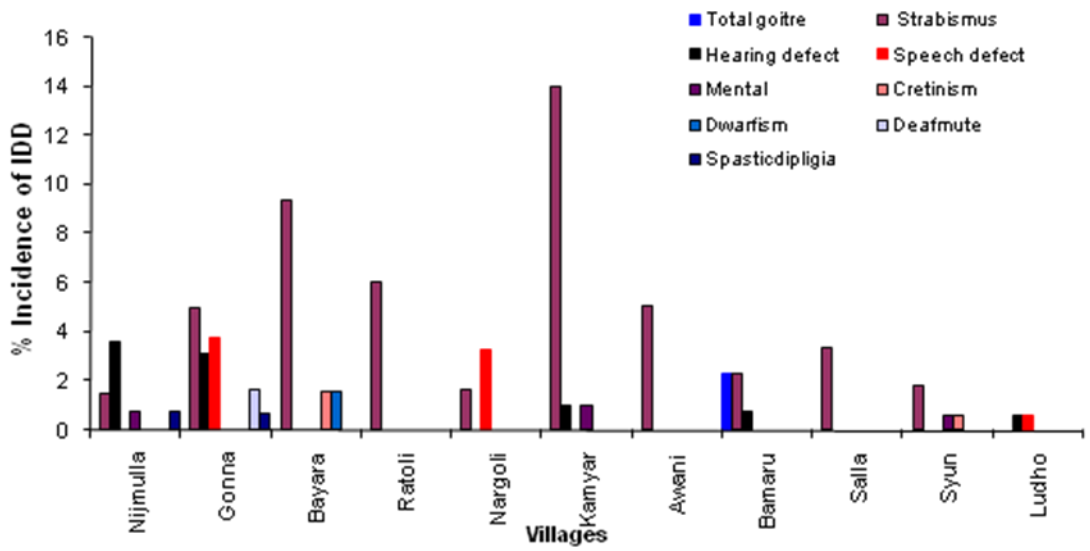

Fig 2 % incidence of iodine deficiency disorders in different villages of district Chamoli of Garhwal Himalaya

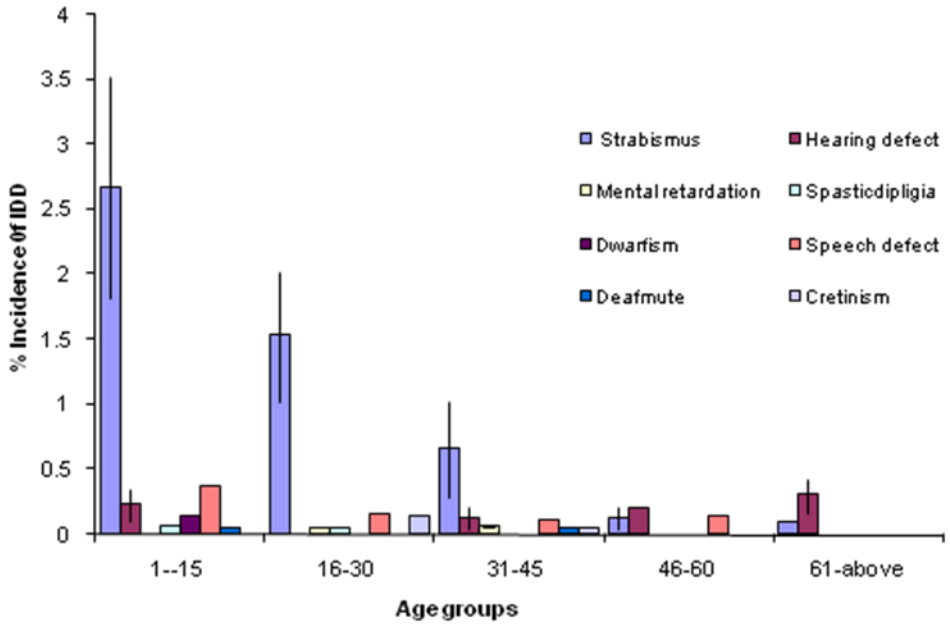

Figure 3 % incidence of overall iodine deficiency disorders in different age groups in district Chamoli of Garhwal Himalaya

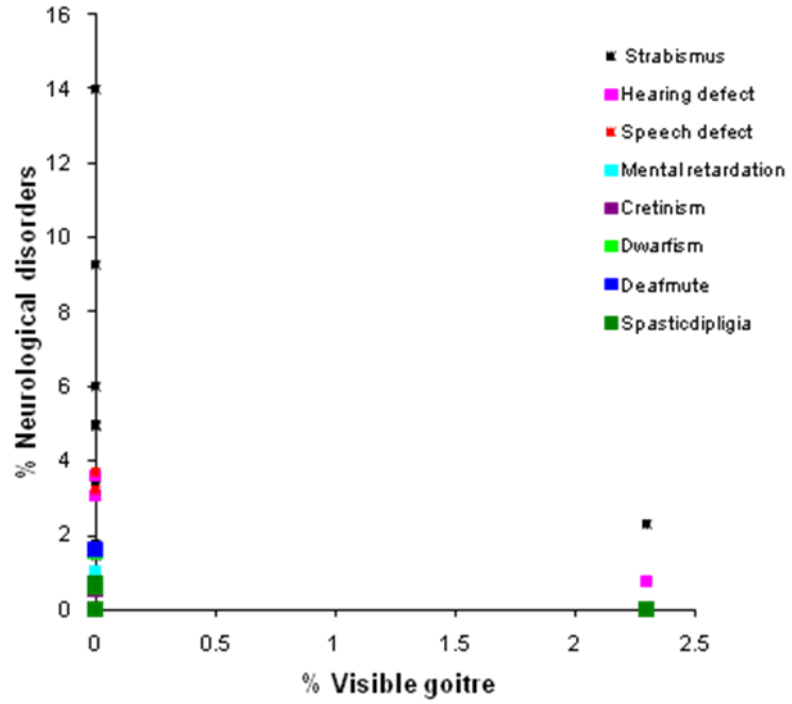

Figure 4 Correlation between visible goitre and neurological disorders in the different villages of district Chamoli

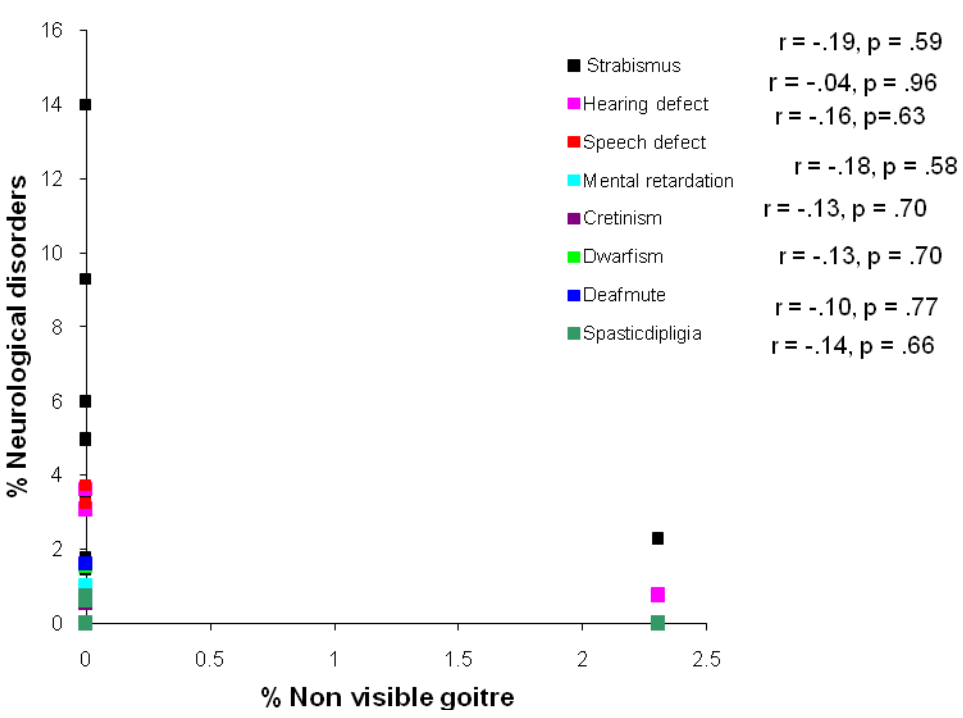

Figure 5 Correlation between nonvisible goitre and neurological disorders in the different villages of district Chamoli

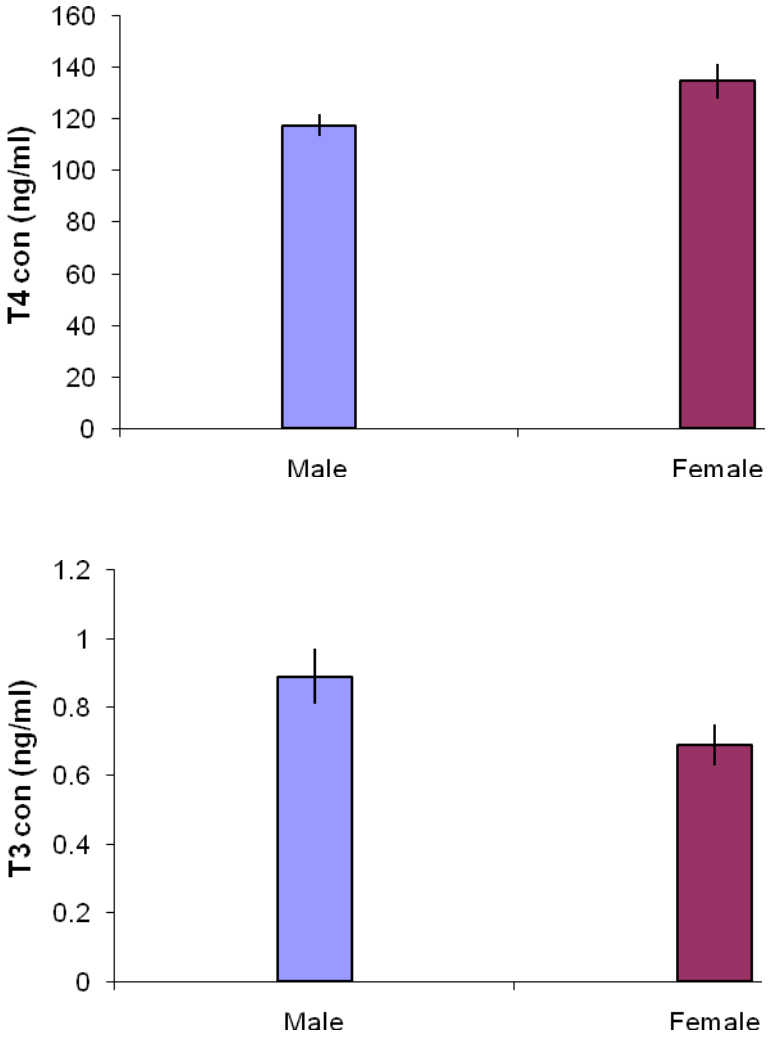

Fig 6:- Serum T3/T4 concentration in villages of Syun & Bamru of district Chamoli.

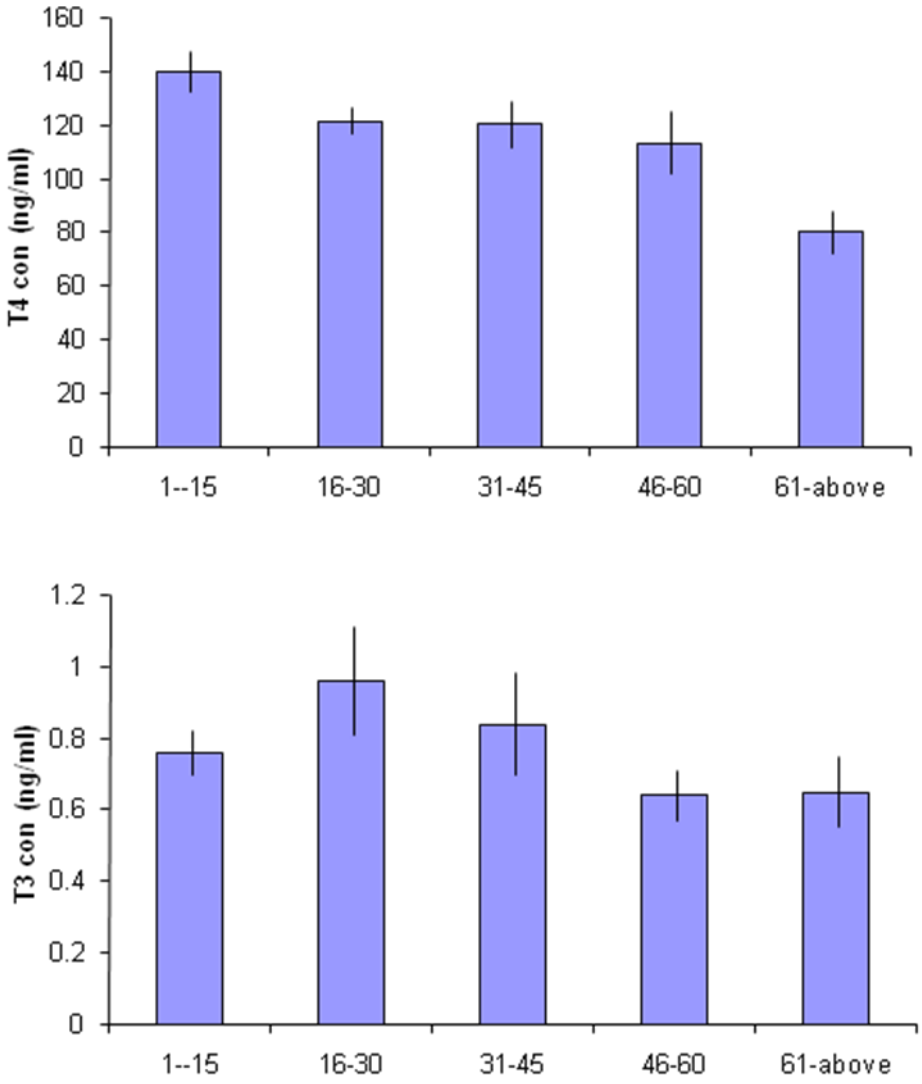

Fig 7: Serum T3, T4 concentration in different age groups in village Syun & Bamru of district Chamoli

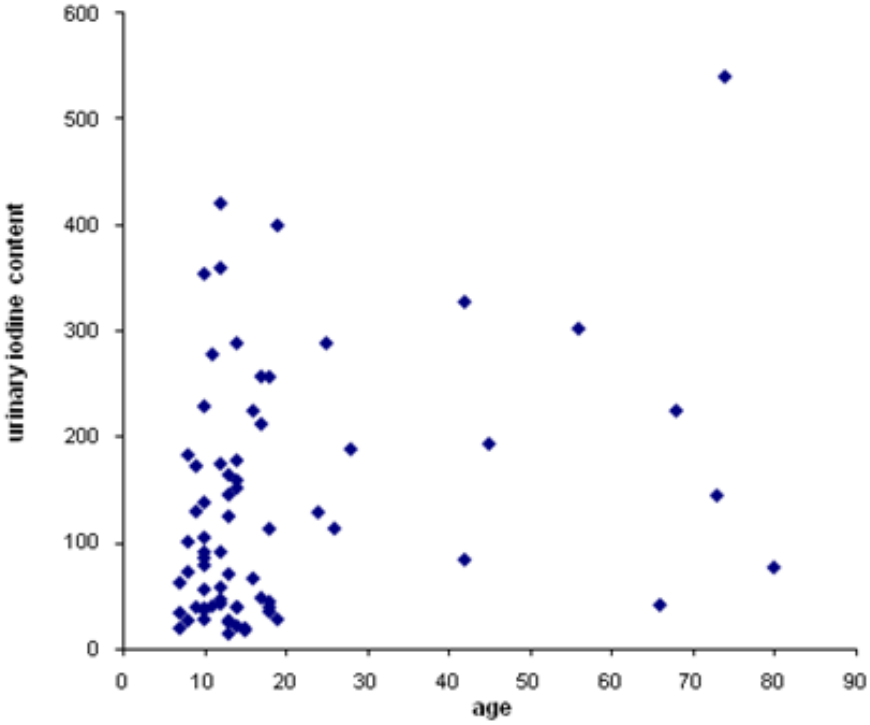

Fig 8:- Urinary iodine Content (µg/L) in different age group of Village Syun & Bamru

References:-

1. Agarwal, D K & Agarwal, K N (1983) Current Status of Endemic Goitre in Some Areas of Sub-Himalayan Belt. Ind. Pediat. 20: 471-477.
2. Bamola V D (2005) Current Status of Iodine deficiency and iodine deficiency disorders in Garhwal Himalaya. Ph D Dissertation. HNB Garhwal University, Uttrakhand.
3. Bernal J, Guadano-Ferraz A, Morte B.(2003) Perspectives in the study of thyroid hormone action on Brain Development and Function.Thyroid 13: 1005-1012.
4. Biswas A B, Chakraborty I, Das D.K, Roy R N, Mukhopadhyay S and Chatterjee S.(2004) Iodine Deficiency Disorders Among School Children of Birbhum, West Bengal.Current Science, Vol. 87, No.1
5. Chandola A & Pathak V K (1980) Migration and annual thyroid activity cycle in Red-headed bunting. In: J R Stockigt & S Nagataki (eds.) Thyroid Research VIII in Australian Acad. Sci. 244-247.
6. Clements F W, Gibson H B & Howelers – Coy J F (1968) Goitre StudiesClements F W & Wischart J W (1956) A Thyroid- Blocking Agent in The Etilogy of Endemic goitre Metabolism 5: 623-639.
7. Degroot L J (1979) Endocrinology. W B Saunders Company, USA.
8. Delange F (1974) Endemic Goitre and Thyroid Function in Central Africa. Monograph in Paediatrics, Basal S Karger Vol. 2.
9. Delong R (1987) Neurological involvement in Iodine Deficiency Disorders. In Hetzel B S, Dunn J T, Stanbury J B, rds. The Prevention and Control of Iodine deficiency Disorders. Elseiver Science Publisher, 49-64.
10. Dunn J. T, Daza C H & Viteri F E (1986) Towards the Eradication of Endemic Goitre, Critinism and Iodine Deficiency, Wasington PAHO/WHO, Scientific Pub. No.502.
11. Dunn J., Crutchfield H., Gutekunst R. & Dunn A. (1993) Methods of measuring iodine in urine 1993 ICCIDD/UNICEF/WHO Wageningen, The Netherlands. .
12. Ekins R P, Sinha A K & Woods R J (1986) Maternal Thyroid Hormone and Development, Tata McGraw Hill New Delhi 222-245.
13. Haddow J E, Palomaki G E, Allan W C, Williams J R, Knight G J, Gagnon J, O Heir C E, Mitchell M L, Hermos RJ, Waisbern S E, Faix J D, Klein R Z (1999). Maternal Thyroid Deficiency during pregnancy and subsequent neuropsychological development of the child. N Engl J Med 341: 549-555.
14. Hetzel B S (1989) Iodine Deficiency Disorders (IDD) and their Eradication. The Lancet 2: 1126-1129.
15. Kapil U, Sharma TD, Singh Preeti (2007) Iodine status and goitre prevalence after 40 years of salt iodization in the Kangra district, India. Vol. 74 Issue 2: 135-137.

16. Kapil U, Singh P, Dwevedi S N, Pathak P (2004) Profile of iodine content of salt and urinary excretion levels in selected districts of Tamil Nadu. Indian J of Pediatrics, Vol. 71, Issue 9: 785-787
17. Kapil U, Singh P, Dwivedi SN, Pathak P. Status of iodine nutriture and universal salt iodisation at beneficiaries levels in Kerala State, India. J Indian Med Assoc. 2006 Apr;104(4):165-7.
18. Kapil, U Singh P and Pathak P (2002) Status of Iodine in Union Territory of Pondeycherry, India. Pakistan Journal of Nutrition 1 (5): 234-235.
19. Kar A & Chandola-Saklani A (1985) Extrathyroidal conversion of thyroxin to tri-iodothyroxine in calotes versicolor. Gen Comp Endocinol 59: 214-218.
20. Karmakar M G Deo M G, Kuchupillai N Ramalingaswami V (1974) Pathophysiology of Himalayan endemic goitre. Am. J Clin Nutri 27:96.
21. Kimball O P & Marine D (1918) The Prevention or Simple Goitre in Man. Arch Int.Med 22 : 41.
22. Kochupillai N & Pandav C S (1987) Neonatal Chemical hypothyroidism in Iodine Deficient Environment. In : B S Hetzel, J T Dunn & J B Stanby (eds) The Prevention and Control of Iodine Deficiency Disorders. Elsvier Sci. Pub. Div. 85-93.
23. Lakhera P & Chandola- Saklani A (1987) Current Status of Goitre and Critinism in Some Endemias of Garhwal Himalaya. Proc Thyroid Assocition (India) AIIMS, New Delhi.
24. Lakhera P (1990) Incidence of Endemic Goitre in Garhwal Himalaya. Ph D Dissertation. HNB Garhwal University, Uttrakhand.
25. Lakhera P and Chandola- Saklani A (1989) Preliminary Reports on Prevalence of Goitre and Cretinism in Area of Garhwal Himalaya Proc. Symp. Env. Gene of Thyroid Disorderes AIIMS, New Delhi.
26. M Michaelides, A T Moore J Med genet (2004) 41:641646. doi:10.11 36/jmg. 021667. Genetics of Strabismus.
27. Mc Carrison R (1915) Observation on endemic Cretinism in the Chithral and Gilgit Valleys. Lancet 2:1275-1280
28. Pandav C S & Kochupillai N (1986) Endemic Goitre and Endemic Cretinism in Southeast Asia: Current Status of Extent, Severity and Control measures. In : J T Dunn, E A Pretall, C H Daza & E E Viteri (eds) Towards the Eradication of the Endemic Goitre, Cretinism and Iodine deficiency. Washingoton PAHO/WHO Sci.Pub. No. 502-366.
29. Perez C, Scrimshaw Ns & Munoz J A (1960) Techniqes of Endemic Goitre survey. In: Endemic Goitre, WHO, Geneva, Monograph Series No. 44 : 369-383.
30. Sandell E B, Kolthoff I M (1937). Micro determination of iodine by catalytic method. Microchem Acta 1: 9 – 25.
31. Sen T K, Biswas A B, Chakrabarty I, Das D K, Ramakrishanan R, Manickam P, Hutin Y (2006) Persistence of iodine deficiency in gangetic flood-prone area, West Bengal, India. Asia Pac J Clin. Nutr 15(4): 538-32.
32. Silva J E (1985) The response of the body to iodine deficiency and hypothyroinaemia: a source of variability in the clinical presentation of endemic goitre of cretinism. In G Medeiros Neto, R M B Maciel & A halpern (eds) Iodine deficiency disorders and congenitial hypothroidinism, Sao Paulo, Ache 80-88.
33. Sooch S S & Ramalingaswami, V (1965) Preliminary Report of an Experiment in the Kangra Valley for the Prevention of Himalayan Endemic Goitre with Iodinated salt. Bull WHO 32:229.
34. Stanbury J B (1972) Cretinism and fetal maternal relationship. Adv Exp Med Biol 30:487
35. Stone T (1984) Endemic Goitre Nutrition and Landholding in Bangladesh Int.J Epidermiology 13: 58-64.
36. Stott H & Gupta S P (1934) The Distribution of Goitre in United Provinces.Ind.J Med Res. XXI: 649-654.
37. Stott H, Bhatia B B, Lal R S & Rai K C (1931) The distribution and cause endemic goitre in the united provinces. Ind J Med Res XVIII 1059-1085.
38. Toteja G S, Padam Singh, B.S.Dhillon and saxena (2004) Iodine Deficiency Disorders in 15 Districts of India. Indian Journal of Pediatrics, Volume 71-January 2004.
39. Udupa K N, Mishr S K & Agarwal J K (1983) Disorders of the Thyroid Gland in tropics. Sds. Vikas Publishing House Pvt. Ltd. New Delhi.
40. W H O /UNICEF/ICCIDD.Assesment of Iodine Deficiency Disorders and monitering their Elimination .Geneva, 2001,WHO/NHD/01.1.
41. Zoellar R.T, A. L.S. Dowling, C. T.A. Herzig, E. A. Lannacone, K. J.Gauger, and Ruby Bansal (2002) Thyroid Hormone, Brain Development, and the Environment. Environmental Health Perspectives Vol. 110 Supplement 31 June 2002
42. Zoeller R.T, (2003) Tran placenta thyroxine and Fetal Brain Development, J Clin. Invest. 111:985-957.
43. Zoeller R.T, Rovett J (2004) Timing of Thyroid Hormone Action in the Developing Brain: Clinical Observations and Experimental Findings. Journal of Neuroendocrinology, 2004, Vol. 16, 809-818.