Assessment of Iodine Deficiency Disorders in North 24 Parganas District, West Bengal, India

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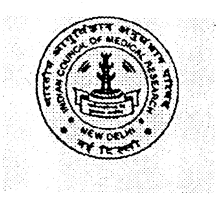

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DECEMBER 2004

CERTIFICATION

This is to certify that this dissertation, entitled **'Assessment of Iodine Deficiency Disorders in North 24 Parganas District, West Bengal, India'**, submitted by Dr. Tapas Kumar Sen, in partial fulfillment of the requirements for the degree of Master of Applied Epidemiology, is the original work done by him and has not been submitted earlier, in part or whole, for any other (Publication or degree) purpose.

Date: 27.12.04

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Abbreviations

CI	Confidence interval
CC	Confidence coefficient
DGHS	Directorate General of Health Services
df	Degrees of freedom
FAO	Food and Agricultural Organization
IDD	Iodine Deficiency Disorders
ICCIDD	International Council for the Control of Iodine deficiency Disorders
IIH	Iodine-induced hyperthyroidism
ICMR	Indian Council of Medical Research
NGCP	National goiter Control Program
NIDDCP	National Iodine Deficiency Disorders Control Program
MIS	Monitoring Information System
NFHS - 2	National Family Health Survey - 2
PDS	Public Distribution System
PPS	Probability Proportional to Size
RoH	Rate of homogeneity
SAARC	South Asian Association for Regional Cooperation
SCN	Standing Committee on Nutrition
SRS	Simple Random Sampling
SD	Standard deviation
TGR	Total Goiter Rate
TRH	Thyrotropin-releasing hormone
TSH	Thyroid-stimulating hormone
USI	Universal Salt Iodization
UN	United Nations
UNDP	United Nations Development Program
UNICEF	United Nations Children's Fund
UIE	Urinary Iodine Excretion
WFP	World Food Program
WHO	World Health Organization

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Date: **Dr. Tapas Kumar Sen**

Abstract

Introduction

In 2000, India revoked the ban on production and sale of non-iodized salt. We conducted a study in the North 24 Parganas district in the state of West Bengal to estimate the magnitude of iodine deficiency disorders and describe the status of the universal salt iodization program.

Methods

We surveyed school children aged eight to ten years selected using a multistage cluster sampling technique. We estimated goiter prevalence and urinary iodine excretion (UIE) using methods and criteria recommended by the World Health Organization. We estimated the iodine content of salt samples collected from the households of the study subjects using spot iodine testing kit.

Results

We included 363 subjects [Mean age (Standard Deviation): 8.9(0.8), proportion of female 177 / 363 (48.8%)], 73 (20.1%) of whom presented with goiter. The median UIE was 160 μg/L (normal range: 100-199 μg/L). Compared with female, a higher proportion of male had UIE level below 100 μg/L (33% and 22% among boys and girls respectively; p=0.02). Of the 363 salt samples, a total of 110 (30.3%) contained insufficient iodine (below 15 ppm).

Conclusions

High goiter prevalence and normal median urinary excretion indicate that the North 24 Parganas District is in transition from iodine deficient to iodine sufficient state. However, the persistence of non-iodized salt consumption indicates that an intensification of universal salt iodization program is needed.

Key words

Goiter, Iodine deficiency, Total goiter rate (TGR), Urinary iodine excretion (UIE), Iodine content of the salt, Cross-sectional study, India.

1. Introduction

The 1990 World Summit for children singled out deficiencies of three micronutrients – iron, vitamin A and iodine – as being particularly common and of special concern for children and women in developing countries, including India [1].

Iodine deficiency is causally related to a spectrum of diseases collectively referred to as iodine deficiency disorders (IDD) and it is one of the most common preventable causes of mental deficiency in the world [2]. An estimated 167 million people in India are at risk of IDD, of whom 54 million suffer from goiter, two million are cretins and 6.6 million children have neurological deficits characterized by mental deficiency, hearing defects, squint and stunted growth [3]. Out of 457 districts (in year 1989) in the country, 275 have been surveyed for IDD and 235 have been found to be endemic of goiter. These districts cover all the states and union territories of India [4].These studies suggested that IDD is not restricted to the classical Himalayan and sub-Himalayan endemic goiter belt involving Jammu and Kashmir, Himachal Pradesh, Punjab, Haryana, Uttar Pradesh, Bihar, West Bengal, Sikkim, Assam, Mizoram, Meghalaya, Tripura, Manipur, Nagaland and Arunachal Pradesh [5]. It has also been reported from the sub-Himalayan flat lands (terai), plains (specially those subjected to annual flooding), riverine areas, deltas and even in coastal regions. We now know that the soil of flooded river valleys is also deprived of iodine as in the Ganges valley in India, which indicates that the problem of iodine deficiency is more widespread than previously suspected [5-7]. IDD are among the easiest and cheapest of all preventable disorders. The addition of a small, constant amount of iodine to the salt that people consume is all that is needed. Considering the impact of IDD on the population, it may be concluded that the elimination of IDD is not only a health issue, rather a critical development issue, and thus should be given the highest priority by governments and international agencies. Both at the national and international level, the problem of IDD has received that priority attention.

To deal with the problem of IDD in India, National Goiter Control Program (NGCP) was launched in 1962, primarily aiming at covering endemic population with iodized salt. Compulsory salt iodization was initiated in 1998 though it was revoked in 2000 considering 'compulsion in such matters of individual choice undesirable' [8]. The

organized efforts were intensified in 1992 with the change of NGCP to National Iodine Deficiency Disorders Control Program (NIDDCP) to reflect an involvement in broader aspects of iodine deficiency beyond goiter in the population.

Since 1992, IDD control program has been in operation in all the states of India, including West Bengal. But successful implementation of such a program needs continuous monitoring and evaluation based on scientifically sound methodology. Globally, International Council for the Control of Iodine deficiency Disorders (ICCIDD), World Health Organization (WHO) and United Nations Children's Fund (UNICEF) have recommended some quantifiable indicators for monitoring the process of the program [9]. The progress of the IDD control program in India needs to be assessed using these recommended indicators.

2. Rationale for the study

The northern part of the State of West Bengal is hilly and located in the classical iodine deficient goiter endemic belt of India; while its major southern part is in the Gangetic basin. North 24 Parganas district is situated at the southern part of the state and extensively riverine and flood prone. Geographical characteristics of the district suggest the IDD proneness of the district.

Moreover, the objectives of the National IDD Control Program were to assess the problem of iodine deficiency in the country, produce and supply iodized salt and then re-assess after five years to estimate the impact of the iodized salt program [5]. Some districts of West Bengal were surveyed in phases to create an information base and to assess the progress of the ongoing intervention program. However, except in two districts those goiter surveys had not been done following methodologies prescribed by WHO, UNICEF and ICCIDD [10, 11]. No such study had been done in the districts of southern part of the state, particularly North 24 Parganas district. Present study will help to understand the present status of salt iodization program of the district at the consumer level and will thus of immense help for the program implementers to take necessary remedial measures.

In this perspective, we conducted the present study with the following objectives.

3. Objectives

- To estimate the prevalence of goiter among school children aged 8-10 years in North 24 Parganas district, West Bengal, India.

- To estimate the Urinary iodine excretion levels of the school children aged 8-10 years in North 24 Parganas district, West Bengal, India.

- To estimate the proportion of the households consuming iodized salt and to estimate the iodine content of the salt consumed.

4. Review of literature

4.1 Introduction

Iodine is a non-metallic trace element. It is an essential component of the thyroid hormones - triiodothyronine (T3) and thyroxine (T4) [7].

Sea fish, other seafoods, and seaweeds are rich sources of iodine suitable for human consumption. Iodine is also found in vegetables grown in soils containing this trace element and in milk products, egg, poultry and meat of animals whose diets contain sufficient iodine. Access to these food products containing iodine is limited in a large portion of the world [12].

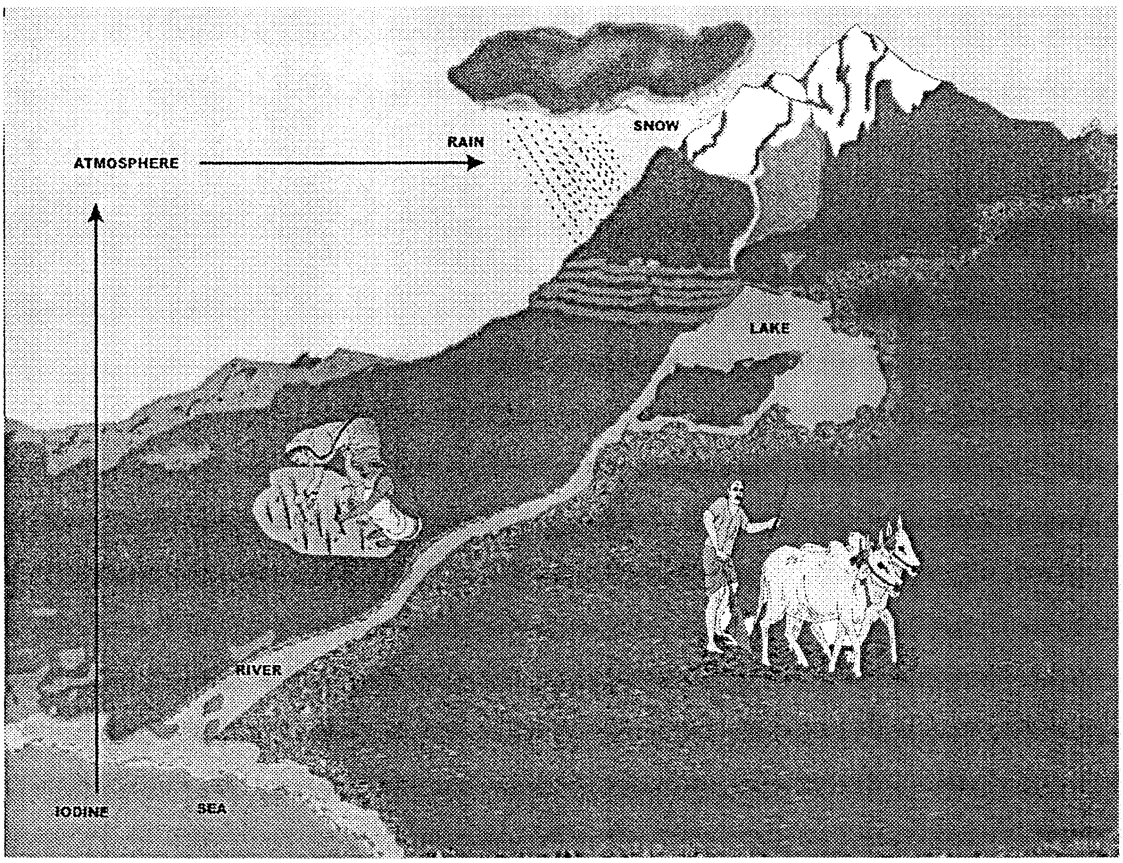
4.1.1. Iodine Cycle in Nature: Ecology of Iodine Deficiency

Most of the Earth's iodine is found in its oceans. It was present during the primordial development of the earth. But large amounts were leached from the surface by glaciations, snow or rain and were carried by the wind, rivers and floods into the sea. In general, the older an exposed soil surface, the more likely the iodine has been leached away by erosion. Mountainous regions, such as the Himalayas, the Andes, and the Alps, and flooded river valleys, such as the Ganges, are among the most severely iodine deficient areas in the world. Iodine occurs in sea and soil as iodide. Iodide ions are oxidized by sunlight to elemental iodine, which is volatile. Every year some 400,000 tonnes of iodine evaporate from the surface of the sea. The concentration of iodine in the seawater is about 50 μg/L; in the air it is approximately 0.7 μg/cubic meter. The iodine in the atmosphere returned to the soil by the rain (concentration 1.8-8.5 μg/L). In this way the cycle is completed [6, 7, 13].

However, the return of the iodine is slow and small in amount compared to the original loss. Subsequent repeated flooding ensures that iodine deficiency in the soil continues. There is no natural correction. So, iodine deficiency persists in the soil indefinitely. Crops grown in that soil will be iodine deficient. As a result, human and animal population dependent on those food crops become iodine deficient. These accounts for the occurrence of severe iodine deficiency among vast population in Asia

that live within systems of subsistence agriculture in flooded river valleys as in India, Bangladesh and Myanmar. Iodine deficiency in affected population will continue unless there is a supplement provided, or alternatively diversification of diet occurs with an increase in iodine intake derived from the food sources outside the iodine deficient areas [6, 7, 13, 14].

Figure 1: Iodine cycle in nature



However, the greater availability of urinary iodine estimation and other methods for assessing iodine deficiency demonstrated that IDD can and do occur in many areas where none of these conditions are met. Indeed, significant iodine deficiency has been found:

- where the prevalence of goiter, based on palpation, is normal;
- in coastal areas;
- in large cities;
- in highly developed countries; and
- where IDD have been considered to have been eliminated, either by prophylactic programs or general dietary changes.

4.1.2. Function of Iodine

Humans require iodine for the synthesis of thyroid hormones. Thyroid gland traps iodine from the blood and converts it into thyroid hormones that are stored and released into the blood circulation when needed. In target tissues such as the liver and the brain, T3, the physiologically active thyroid hormone can bind to thyroid receptors in the nuclei of cells and regulate gene expression. T4, the most abundant circulating thyroid hormone, can be converted to T3 by enzymes known as deiodinases in target tissues. In this manner, thyroid hormones regulate a number of physiologic processes, including growth, development, metabolism and reproductive function [15].

The regulation of thyroid function is a complex process that involves the brain (hypothalamus) and pituitary gland. In response to thyrotropin-releasing hormone (TRH) secretion by the hypothalamus, the pituitary gland secretes thyroid-stimulating hormone (TSH), which stimulates iodine trapping, thyroid hormone synthesis, and release of T3 and T4 by the thyroid gland. The presence of adequate circulating T4 decreases the sensitivity of the pituitary gland to TRH, limiting its secretion of TSH. When circulating T4 levels decrease, the pituitary increases its secretion of TSH, resulting in increased iodine trapping, as well as increased production and release of T3 and T4. Iodine deficiency results in inadequate production of T4. In response to decreased blood levels of T4, the pituitary gland increases its output of TSH. Persistently elevated TSH levels may lead to hypertrophy (enlargement) of the thyroid gland, also known as goiter [16].

4.1.3. Human requirement

An adult human body contains 15-20 mg of iodine (70-80% in thyroid gland). Iodine is rapidly absorbed through gut. The normal intake requirement is 100-150 μg/day. Excess iodine is readily excreted by kidney. WHO, UNICEF, and ICCIDD recommended that the daily intake of iodine should be as follows [4, 17].

Table 1. Recommended daily intake of iodine

Age	Recommended daily intake
0-59 months	90 μg
60 months-12 years	120 μg
Above 12 years	150 μg
Pregnant and lactating women	200 μg

4.2. Definition and Spectrum of Iodine Deficiency Disorders

The Iodine Deficiency Disorders (IDD) refers to all the ill effects of iodine deficiency in a population that can be prevented by ensuring adequate intake of iodine [17].

When iodine intake falls below the recommended levels, the thyroid may no longer be able to synthesize sufficient amount of thyroid hormone. The resulting low level of thyroid hormone in the blood (hypothyroidism) is the principal factor responsible for the damage done to the developing brain and the other harmful effects known collectively as the Iodine Deficiency Disorders [13]. The adoption of this term emphasized that the problem extended far beyond simply goiter and cretinism.

Table 2. The spectrum of Iodine Deficiency Disorders [17]

Fetus	Congenital anomalies; Increased perinatal mortality; Increased infant mortality;Neurological cretinism: mental deficiency, deaf mutism, spastic diplagia, squint Myxoedematous cretinism: mental deficiency, dwarfism, hypothyroidism, psychomotor defects
Neonate	Neonatal hypothyroidism
Child and Adolescent	Retarded mental and physical development
Pregnant and lactating women	Miscarriage, stillbirth; birth defects and congenital hypothyroidism in the offspring; Lactating mother unable to provide sufficient iodine to newborn vulnerable to iodine deficiency
Adult	Goiter and its complications Iodine-induced hyperthyroidism (IIH)
All Ages	Goiter; Hypothyroidism; Impaired mental function; Increased susceptibility to nuclear radiation
Animals	Reduced body weight and wool growth; deformity of skull; reduced bone and brain development; increased incidence of abortion and stillbirth

4.3. Effects of iodine deficiency on growth and development

A deficiency of the thyroid hormone, either produced by the absence of the thyroid, a severe iodine deficiency or a congenitally defective thyroid, is associated with severe retardation of growth and maturation in almost all organ systems. Body weight does not increase and there is retardation of bone growth. The sensitivity of different organs to iodine and hormone deficiency varies. The brain is particularly susceptible to damage during the fetal and early postnatal period. The most critical period is from

the second trimester of pregnancy to the third year after birth. At birth, the human brain is still at a stage of early maturation, as it would have reached less than a third of its mature weight [17-19].

Thyroid enlargement or goiter is one of the earliest and most visible signs of iodine deficiency. The thyroid enlarges in response to persistent stimulation by TSH. In mild iodine deficiency, this adaptation response may be enough to provide the body with sufficient thyroid hormone. However, more severe cases of iodine deficiency result in hypothyroidism. Adequate iodine intake will generally reduce the size of goiters, but the reversibility of the effects of hypothyroidism depends on an individual's stage of development. Iodine deficiency has adverse effects in all stages of development, but is most damaging to the developing brain. In addition to regulating many aspects of growth and development, thyroid hormone is important for the myelination of the central nervous system, which is most active before and shortly after birth [15, 20]. Iodine deficiency, through its effects on the developing brain, has condemned millions of people to a life of few prospects and continued underdevelopment. On a worldwide basis, iodine deficiency is the single most important preventable cause of brain damage [17].

4.3.1. Effects on fetus

Fetal iodine deficiency is caused by iodine deficiency of the mother. One of the most devastating effects of maternal iodine deficiency is congenital hypothyroidism, a condition that is sometimes referred to as cretinism and results in irreversible mental retardation. Congenital hypothyroidism occurs in two forms, although there is considerable overlap between them. The neurological form is characterized by mental and physical retardation and deafness. It is the result of maternal iodine deficiency that affects the fetus before its own thyroid is functional. The myxedematous or hypothyroid form is characterized by short stature and mental retardation. In addition to iodine deficiency, the hypothyroid form has been associated with selenium deficiency and the presence of goitrogens in the diet that interfere with thyroid hormone production. Deficiencies of vitamin A or iron may also exacerbate the effects of iodine deficiency [20, 21]. An important recent issue on thyroid function and regulation in the fetus is the concept that thyroid hormones are transferred from

mother to fetus both before and probably after the onset of fetal thyroid function, contrasting with the previous dogma that this transfer is minimal or does not exist [21].

4.3.2. Effects on infants

Infant mortality is high in areas of iodine deficiency, and several studies have demonstrated an increase in childhood survival when iodine deficiency is corrected [22]. An increased perinatal mortality due to iodine deficiency has been shown in Zaire from the results of a controlled trial of iodized oil injections during the latter half of pregnancy, alternatively with a control injection [23]. In the most severely iodine deficient environments in Northern India, the incidence of neonatal hypothyroidism was 75 to 115 per thousand births. By contrast in Delhi, where only mild iodine deficiency is present with a low prevalence of goiter and no cretinism, the incidence drops to 6 per thousand. In control areas without goiter, the level was only one per thousand [24]. Infancy is a period of rapid brain growth and development. Sufficient thyroid hormone, which depends on adequate iodine intake, is essential for normal brain development. Even in the absence of congenital hypothyroidism, iodine deficiency during infancy may result in abnormal brain development and consequently, impaired intellectual development [25].

4.3.3. Effects on children and adolescents

Iodine deficiency in children and adolescents is often associated with goiter. The incidence of goiter peaks in adolescence and is more common in girls. School children in iodine deficient areas show poorer academic performance, lower Intelligence Quotients (IQ), and a higher incidence of learning disabilities than matched groups from iodine-sufficient areas. A recent meta-analysis of 18 studies concluded that iodine deficiency alone lowered mean IQ scores in children by 13.5 points [26, 27].

In studies in China, lower IQ scores resulted due to nerve deafness and the presence of abnormal neurological signs similar to the pattern of neurological cretinism. It was concluded that iodine deficiency results in a shift of the entire population distribution of cognitive skills to a lower level [28]. In a pioneering study in Ecuador in 1966, Fierro Benitez and his colleagues have reported the long term effects of iodized oil injections

by comparing two highland villages, one being treated and other acting as control [29]. A controlled trial carried out with oral iodized oil in a village in Bolivia indicated that the correction of iodine deficiency could improve the mental performance of the school going children [30].

4.3.4. Effects on pregnant and lactating women

Iodine requirements are increased in pregnant and breastfeeding women [20]. Iodine deficiency during pregnancy has been associated with increased incidence of miscarriage, stillbirth, and birth defects. Moreover, severe iodine deficiency during pregnancy may result in congenital hypothyroidism in the offspring [21]. Iodine deficient women who are breastfeeding may not be able to provide sufficient iodine to their infants, particularly vulnerable to the effects of iodine deficiency [7]. A daily prenatal supplement providing 150 μg of iodine will help to ensure that pregnant and breastfeeding women consume sufficient iodine during these critical periods.

4.3.5. Effects on adults

Inadequate iodine intake may also result in goiter and hypothyroidism in adults. Although the effects of hypothyroidism are subtler in the brains of adults than children, recent research suggests that hypothyroidism results in slower response times and impaired mental function [7].

In Northern India, a high degree of apathy has been noted in populations living in iodine deficient areas. This may even affect domestic animals such as dogs. It is apparent that reduced mental function due to brain hypothyroidism is widely prevalent in iodine deficient communities. This has an effect on their capacity for initiative and decision-making [17].

An instructive example of the possibilities is provided by observations of the effect of iodized salt program dating from 1978 in the village of Jixian in northern China. Between 1978 and 1983, productivity, as measured by per capita income, increased by a factor of 10, school performance improved along with sharp decline in goiter and cretinism prevalence [14].

Iodine deficient individuals of all ages are more susceptible to radiation-induced thyroid cancer as well as to iodine-induced hyperthyroidism as iodine deficiency results in increased iodine trapping by the thyroid [7].

4.3.6. Effects on animals

Epidemiological and experimental studies indicate that reproductive, neurological and other defects are important effects of iodine deficiency in animal population.

In areas of iodine deficiency, retarded or arrested development of the fetus occurs at some stage during gestation; resulting in early death or resorption, abortion and stillbirth, or the birth of a weak, hairless offspring associated with prolonged gestation and parturition and the retention of placental membranes. Subnormal thyroid hormone levels in herds of cattle have been accompanied by a high incidence of aborted, stillborn and weak calves [5].

Experimental work with sheep revealed that apart from the effects mentioned above, the fetus shows reduced body weight, complete absence of wool growth, deformation of the skull and retardation of bone development. There is retardation of brain development as indicated by reduced brain weight and a reduced number of cells (as measured by DNA). Similar effects have been observed in the marmoset monkey [5].

4.4 Role of Goitrogens

Some foods contain substances that interfere with iodine utilization or thyroid hormone production, known as goitrogen. These compounds belong to the following chemical groups: Sulfurated organics (like thiocyanate, isothiocyanate, goitrin and disulphides), flavonoids (polyphenols), polyhydroxyphenols and phenol derivatives, pyridines, phtalate esters and metabolites, polychlorinated (PCB) and polybrominated (PBB) biphenyls, other organochlorines (like DDT), polycyclic aromatic hydrocarbons (PAH), inorganic iodine (in excess), and lithium.

The occurrence of goiter in the Democratic Republic of Congo has been related to the consumption of casava, which contains a compound that is metabolized to thiocyanate and blocks thyroidal uptake of iodine. Some species of millet and cruciferous

vegetables (for example, cabbage, broccoli, cauliflower, and Brussel sprouts) also contain goitrogens. The soybean isoflavones, genistein and daidzein, have also been found to inhibit thyroid hormone synthesis [31].

Most of these goitrogens are not of clinical importance unless they are consumed in large amounts or there is coexisting iodine deficiency. Recent findings also indicate that tobacco smoking may be associated with an increased risk of goiter in iodine deficient areas [32].

4.5. Individuals at risk of iodine deficiency

While the risk of iodine deficiency for populations living in iodine-deficient areas without adequate iodine fortification programs is well recognized, concerns have been raised that certain subpopulation may not consume adequate iodine in countries considered iodine-sufficient. Vegetarian and non-vegetarian diets that exclude iodized salt, fish, and seaweed have been found to contain very little iodine [7, 20, 33, 34]. Urinary iodine excretion studies suggest that iodine intakes are declining in Switzerland, New Zealand and the United States of America (USA), possibly due to increased adherence to dietary recommendations to reduce salt intake. Although iodine intake in the U.S.A. remains sufficient, further monitoring of iodine intake was recommended [35, 36].

4.6. Economic impact of Iodine Deficiency Disorders

The various effects of iodine deficiency impose human, social, and economic costs on individuals and communities. The costs arise from the disabilities of mental deficiency and deaf-mutism. These effects have economic implications in reduced work output in the household and in the labor market, and the costs of medical and institutional care. The more subtle effects on mental status cause poor levels of school performance by children and hence produce long-term effects over the whole life span.

In human population, effects of iodine interventions lead to reduction in mental deficiency, deaf mutism and hypothyroidism. The economic benefits resulted from such effects are: value of higher work output in the household and labor market, reduced costs of medical and custodial care and lower educational costs from reduced

absenteeism and grade repetition. In animal population, iodine interventions lead to increase in live births, weight, and strength. Calves have lesser proportion of birth deformity. It also increases the wool coat in sheep. The economic benefits resulted from such effects are: value of higher output of meat and other animal products and value of higher animal work output [37].

For calculation of IDD load in India, using an epidemiological model and assuming life expectancy of 60 years and the crude birth rate at 33 per 1000 population, WHO estimated that 42,000 new cases of cretins and 126,000 still births and abortions would occur every year if no control program is launched [38].

Loss of productivity was based on the assumptions: cretins have 55% less productivity compared to normal unaffected adult; a person suffering from mild iodine deficiency will have 5% less productivity; severely impaired cretins require full time care; the minimum wage per person is Rs.16 per day; the number of working day in a year is 183 and 90% of the population in the 15-59 years age group is engaged in productive work. Using the above assumptions, the productivity loss is estimated to be Rs.3906 million [39].

Calculation of costs of management of IDD were based on assumptions: only 10% of the affected people seek health care; only people with visible goiters seek treatment; costs include physician costs and cost of treatment; endemic cretins visit a health facility once in a lifetime and no treatment is available for them. Using the above assumptions, total cost of management of patients with IDD was Rs.342 million [39].
The cost of the salt iodization program is Rs.1296 million. So, money saved on the management of IDD is Rs.4248 million (Rs.3906 million + Rs.342 million). The net savings is Rs.2952 million. The cost benefit ratio is therefore 1:3. It justifies the necessity of salt iodization program in India [39].

Similar study conducted in Germany revealed that by spending 122 million DM (German currency) for the IDD control measures, 1.4 billion DM could be saved or a cost benefit balance of 1.3 billion DM would result per year [39].

An outcome description study conducted in Ecuador showed: a 20% reduction of cretinism would increase the annual per capita income by US$ 47 [39].

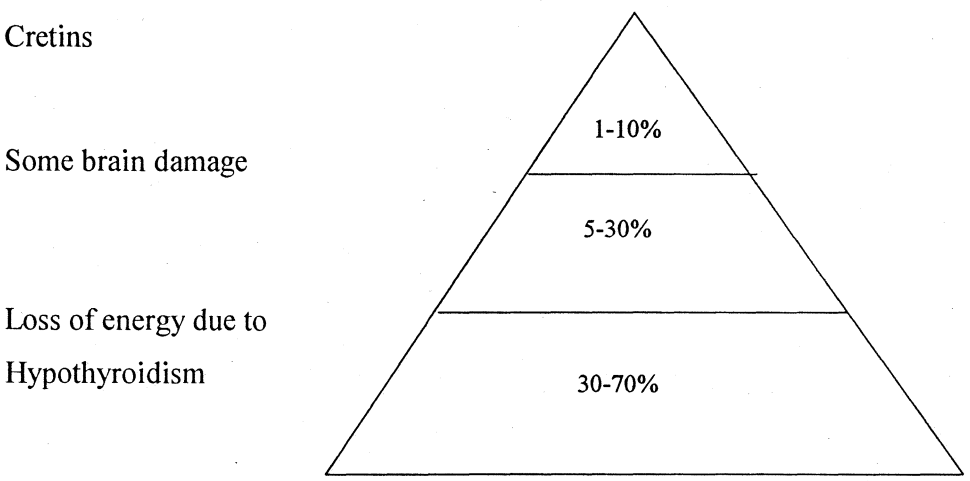
It is evident on the basis of economic evaluation that elimination of IDD confers an overwhelming benefit for human development and social well being.

4.7. Magnitude of the problem

4.7.1. Global scenario

The iodine deficiency disorders (IDD) can be seen as an 'iceberg' of effects [14] in a community or population (Figure 2). In a severely iodine deficient area, cretinism with a prevalence of 1-10% is only the visible portion of the iceberg which includes an invisible but very substantial volume of effects due to lesser degree of brain damage and cerebral hypothyroidism.

Figure 2. The Iodine Deficiency Disorders iceberg



In 1999, World Health Organization (WHO) estimated that 130 of its 191 member countries had a significant IDD problem. A total of 741 million (13%) of the world's total population is affected by goiter [17] (Table 3). Keeping in mind the iceberg phenomenon, it is expected that a much greater proportion of the population suffer from IDD and in particular, from some degree of mental retardation.

Table 3: Magnitude of iodine deficiency disorders by goiter by WHO region (1999)

WHO Region	Population* (in millions)	Population affected by goiter (in millions)	% of the Region
Africa	612	124	20%
America	788	39	5%
South East Asia	1,477	172	12%
Eastern Mediterranean	473	152	32%
Europe	869	130	15%
Western Pacific	1,639	124	8%
Total	5,858	741	13%

* Based on UN Population Division (UN estimates 1997)

The struggle to conquer the IDD started in the early years of the twentieth century. But, it is only the last decade that has seen the most remarkable progress, which has been particularly laudable in Asia and Africa.

In spite of this progress, the estimated total affected population at the global level has not changed substantially compared to the figure previously published in 1993 [40]. The reason is that in 1993 the magnitude of the problem was underestimated because part of the information was not available then.

In 1999, WHO in collaboration with UNICEF and ICCIDD reviewed the IDD global situation [18]. Of the 130 countries with IDD, 98 (75%) now have legislation on salt iodization and a further 12 have it in draft form. Following the promulgation of legislation on salt, and the sensitization of the salt industry, there has been an enormous increase in the consumption of iodized salt [17].

Table 4: Status of salt iodization coverage by WHO region (1999) [17]

WHO Region	Households with access to iodized salt
Africa	63%
Americas	90%
South East Asia	70%
Eastern Mediterranean	66%
Europe	27%
Western Pacific	76%
Overall	68%

Note: Total population of each country multiplied by the % of households with access to iodized salt. Numbers then totaled for each region and divided by the total regional population.

This report emphasizes the importance of monitoring for ensuring the sustainability of IDD control programs. The latest data from the same report, concerning the status of monitoring the programs [17] in the various WHO regions are summarized in Table 5.

Table 5: Status of monitoring activities and laboratory facilities in iodine deficiency disorders affected countries (1999)

WHO Regions	Number of IDD affected countries	Number of IDD affected countries		
		Monitoring salt quality	Monitoring iodine status	Laboratory facilities
Africa	44	29	24	28
Americas	19	19	19	19
South East Asia	9	8	7	6
Eastern Mediterranean	17	14	10	11
Europe	32	17	13	13
Western Pacific	9	8	6	7
Overall	130	95 (73%)	79 (61%)	84(65%)

* These figures reflect countries with the capacity for both urinary iodine and/or salt iodine analyses. Standard of laboratories and expertise for each of these, however, is very different.

4.7.2. Indian scenario

India is the second most populous country in the world with a population of 1027 million (2001 census). There is a high prevalence of goiter and cretinism in the Himalayan and sub-Himalayan belt, from Jammu and Kashmir in the west, to Arunchal Pradesh in the east and, along this entire length, extending at least 500 km. south of the Himalayas into the flat sub-Himalayan terai (plains).

In addition to the well-known Himalayan endemic belt, iodine deficiency has been reported from many other states in the country. In 1984-86, the Indian Council of Medical Research (ICMR) carried out a multicentric IDD prevalence study. Nine states outside the traditional Himalayan goiter belt were studied for the prevalence of goiter and cretinism. A total of 409,923 individuals were examined. Overall goiter prevalence observed was 21.1 percent and the overall cretinism prevalence was 0.7 percent [41].

Results of sample surveys conducted by different agencies in 283 districts of 29 states and 4 Union Territories of India have shown a high prevalence of IDD in 247 districts. This shows that no state or union territory is free of iodine deficiency and free from IDD as a public health problem [42].

A conservative estimate using the epidemiological (Dulberg) model suggests that 150 million are at risk of IDD, 54 million have goiter, 2.2 million suffer from cretinism and an estimated 6.6 million are affected by milder neurological defects attributable to environmental iodine deficiency [3, 5]. In the sub-Himalayan goiter belt of India alone, nearly 55 million are estimated to be suffering from endemic goiter, with an average goiter prevalence rate of about 36% [38].

This places India among the major endemic iodine deficiency countries of the world. Immediate steps are therefore required to ensure that iodine supplementation, in the form of iodized salt, reaches the entire afflicted population, at the earliest.

In 1998, the Government of India introduced a promulgation banning the sale and storage of non-iodized common salt in the country. All states and union territories,

with the exception of Kerala (which did not implement the ban at the state level), Andhra Pradesh and Maharashtra (which implemented the ban partially, i.e. in some districts only), implemented the ban order in their respective states. The Government of India lifted the ban order in 2000 and in its wake only Gujarat has revoked the ban order. Other states have maintained a status quo. Under the NIDDCP, there were provisions made for the setting up of an IDD Cell. Accordingly, the Central Nutrition and Iodine Deficiency Disorders Cell at Directorate General of Health Services (DGHS) is responsible for implementation of NIDDCP in the country.

4.7.3. West Bengal scenario

With a population of 82 million as per 2001 Census, West Bengal is the fourth most populous state of the country. It is situated in the eastern region of India. Accounting for about 2.7% India's area, but about 7.8% of the country's population, this state ranks first in terms of density of 904 persons per sq. km. The boundaries of the state are Nepal, Bhutan and state of Sikkim on the north, Assam and Bangladesh on the east, Bihar on the west and Orissa and Bay of Bengal on the south [43].

Iodine deficiency disorder is a public health problem in the state. Five districts of the state were included in the nationwide survey. All the five districts were declared endemic [42]. National Family Health Survey-2 of India (1998-1999) report stated that 62 percent of households use cooking salt that is iodized at the recommended level of 15 parts per million. It is well below the expected target of 90% coverage of population by iodized salt. In this context, the report suggested that iodine deficiency disorders are likely to be a problem in West Bengal. The report further stated that rural households and households with a low standard of living are much less likely than other households consuming adequately iodized cooking salt [44].

There has been no state level study to assess the status of IDD in West Bengal. Some districts were surveyed in phases by the district authorities. Unfortunately, those studies did not follow the methodologies recommended by WHO, UNICEF and ICCIDD and were conducted by paramedical staff without sufficient training. Two districts were surveyed in the later period following recommended guideline. In both the districts, goiter prevalence was more than 10% (Malda: 11.3%, Birbhum: 12.6%);

however, median urinary iodine excretion levels were more than 100 μg/L [10, 11]. A recent study in Howrah district showed a goiter prevalence of 37.5% among school children of 6-12 years of age [45].

4.8. Elimination of Iodine deficiency Disorders

4.8.1. Correction of iodine deficiency: iodine supplementation

Iodine deficient environment requires the continued addition of iodine. It is most conveniently and cheaply achieved by the addition of iodine to the salt supply. The advantage of supplementing with iodized salt is that it is used by all sections of community irrespective of social and economic status.

In the last 50 years, many countries in North America, Asia, Europe and Oceania have successfully eliminated IDD, or made substantial progress in their control, largely as a result of salt iodization with potassium iodide or iodate and through dietary diversification. For example, in Switzerland, where salt iodization began in 1922, cretinism has been eliminated and goiter has disappeared, while there is negligible evidence of any adverse effects from iodine intake [46-49]. At this stage, however, sustainability of this successful correction of iodine deficiency becomes the challenge, as iodine deficiency may recur at any time [15].

In some regions, iodization of salt may not be a practical option for the sustainable elimination of IDD, at least in the short term. This is, in particular, likely to be the case in remote areas where communications are poor or where there are numerous small-scale producers. In such areas, other options for correction of IDD may have to be considered, such as [17]:

- Administration of iodized oil capsules every 6-18 months [50];
- Direct administration of iodine solution, such as Lugol's iodine, at regular intervals (once a month is sufficient); or
- Iodization of water supplies by direct addition of iodine solution or via a special delivery mechanism.

Universal salt iodization (USI) involves the iodization of all human and livestock salt, including salt used in the food industry. Adequate iodization of all salt will deliver

iodine in the required quantities to the population on a continuous and self-sustaining basis.

4.8.2. Adverse reactions to iodized salt

Since iodine, when ingested in large amounts, is easily excreted in the urine, iodine intake at very high levels can be safe. It is documented scientifically that through adaptive mechanisms, normal people exposed to excess iodine remain euthyroid and free of goiter [51]. A comparative account of various levels of iodine intake to the functional status of the body as a dose response curve has documented that a daily intake in the range of 150-300 µg is absolutely safe [52].

a) Iodide goiter and iodine-induced hypothyroidism

When iodine intake is chronically high, as for example in coastal areas of Japan and China due to the chronic intake of seaweeds rich in iodine such as laminaria or in Eastern China because of the high iodine content of the drinking water from shallow wells, the prevalence of thyroid enlargement and goiter is high as compared to population of other areas of the country who are not exposed to such high iodine level in water or food. As a result, the prevalence of sub clinical hypothyroidism is elevated. The mechanisms behind this impairment of thyroid function are probably both iodine enhancement of thyroid autoimmunity and reversible inhibition of thyroid function by excess iodine (Wolff-Chaikoff effect) in susceptible subjects. However, this type of thyroid failure has not been observed after correction of iodine deficiency, including in neonates after the administration of huge doses of iodized oil to their mothers during pregnancy [6]. High intakes of dietary iodine may induce hypothyroidism in autoimmune thyroid diseases and may inhibit the effects of the thionamide drugs [48].

b) Iodine-induced hyperthyroidism

Iodine induced hyperthyroidism (IIH) is an adverse effect, which may occur primarily in older people when severely iodine deficient populations increase their iodine intake, even when the total amount is within the usual accepted range of 100-200 µg/day. Epidemiologically, iodine induced hyperthyroidism represents a transient

increase in the incidence of hyperthyroidism, which disappears in due course with the correction of iodine deficiency [49].

c) Iodine-induced thyroiditis

Iodine induced hyperthyroidism occurs in people who have pre-existing autonomous nodular goiter. It appears likely that some patients with latent Grave's disease are also at risk. The number of people at risk of iodine-induced hyperthyroidism is directly proportional with number of subjects with nodular goiter [51].

The occurrence of iodine-induced hyperthyroidism is probably related to the relative increase and rapidity of increase of iodine intake, which occurs when iodized salt is introduced in population that have been severely iodine deficient. There is no level of iodine in salt that offers complete protection against some increase in the incidence of hyperthyroidism in a previously iodine-deficient population. From a public health point of view, the benefits of correcting iodine deficiency through Universal Salt Iodization (USI) greatly outweigh the risk of iodine-induced hyperthyroidism [53].

4.8.3. Indicators of the sustainable elimination of Iodine Deficiency Disorders

In considering whether the sustainable elimination of iodine deficiency as a public health problem has been achieved, the following criteria should be met: [9, 17]

1) With regard to salt iodization, availability and consumption of adequately iodized salt (15 ppm or above iodine) must be guaranteed. This is demonstrated by its use by more than 90% of the households. Pre-conditions for this are:

- Local production and/or importation of iodized salt in a quantity sufficient to satisfy potential human demand (about 4-5 kg/person/year);
- 95% of salt for human consumption must be iodized according to government standards for iodine content, at the production or importation levels;
- The percentage of food grade salt with iodine content of at least 15 ppm, in a representative sample of households, must be equal to or greater than 90%; and

- Iodine estimation at the point of production or importation, and at the wholesale and retail levels must be determined by titration; at the household level, it may be determined by either titration or certified kits.

2) With regard to the iodine status in the population:

- The median urinary iodine excretion level should be at least 100 μg/L, with less than 20% of values below 50 μg/L; and
- The most recent monitoring data (national or regional) should have been collected in the last two years.

3) At least eight of the following ten programmatic indicators should occur:

(i) An effective, functional national body (council or committee) responsible to the government for the national program for the elimination of IDD (this control should be multidisciplinary, involving the relevant fields of nutrition, medicine, education, the salt industry, the media, and consumers, with a chairman appointed by the Ministry of Health);
(ii) Evidence of political commitment to universal salt iodization and elimination program;
(iii) Appointment of a responsible executive officer for the IDD elimination program;
(iv) Legislation or regulations on universal salt iodization (while ideally regulations should cover both human and agricultural salt, if the latter is not covered this does not necessarily preclude a country from being certified as IDD-free);
(v) Commitment to assessment and reassessment of progress in the elimination of IDD, with access to laboratories able to provide accurate data on salt and urinary iodine;
(vi) A program of public education and social mobilization on the importance of IDD and the consumption of iodized salt;
(vii) Regular data on salt iodine at the factory, retail and household levels;
(vii) Regular laboratory data on urinary iodine in school-aged children, with appropriate sampling for higher risk areas;
(ix) Cooperation from the salt industry in maintenance of quality control; and

(x) A database for recording of results or regular monitoring procedures, particularly for salt iodine, urinary iodine and, if available, neonatal TSH, with mandatory public reporting.

The criteria [9, 17] for monitoring progress towards sustainable elimination of IDD are summarized in Table 6.

Table 6. Summary of criteria for monitoring progress towards sustainable elimination of IDD as a public health problem

Indicators	Goals
1. Salt iodization:	
* Proportion of households consuming effectively iodized salt	>90%
2. Urinary iodine:	
* Proportion below 100 µg/L	<50%
* Proportion below 50 µg/Lt	<20%
3. Thyroid size:	
In school children 6-12 years age:	
* Proportion with enlarged thyroid, by palpation or ultrasound	<5%
Programmatic indicators	Attainment of at least 8 of the 10

4.8.4. Phases of IDD control and elimination program

In any region, iodine deficiency disorders elimination programs can be seen going through three phases [5, 17, 54]. The phases describe a gradual evolution from the virgin state of iodine deficiency to the iodine-replete state. The status of the indicators of assessment of iodine deficiency varies from phase to phase. The three phases are:

Phase I - Community diagnosis
(Iodine deficiency)

Phase II - Community intervention
(Iodine deficiency to sufficiency)

Phase III - Sustainability
(Ensuring optimum iodine intake)

Phase I – Community Diagnosis

In this phase, the problem is first detected as an existing public health problem. However, efforts to recognize and estimate it have not been done; control measures have not been initiated. During this period the thyroid glands of persons living in iodine-deplete area, being iodine-starved, will take up as much of the iodine as it possibly can and rest is excreted through urine. So, we may get a good association between Total Goiter Rate (TGR) and Urinary Iodine Excretion (UIE) levels. Iodine deficiency manifests with high goiter rate and low urinary iodine excretion values.

Phase II – Community intervention

The intervention has already begun in this phase. So, a mixed picture is seen. The impact indicators are determined by environmental and community factors. Goiter becomes a historic marker of iodine deficiency. Though the median urinary iodine values are on the rise, there is a time lag before the goiter prevalence takes a down hill course. So, the inverse relation between TGR and UIE is not seen. A non-linear relation between total goiter rate and urinary iodine values appear. We may get increased rates of goiter along with adequacy of urinary iodine excretion values.

Phase III – Sustainability

The initial success of an effective program will be the achievement of control of iodine deficiency. Soon after it, the need for sustainability of the control program comes into play. The mainstay of sustainability is to ensure optimum iodine intake on a regular and continuous basis for all time to come. In this phase, once again there is a good association between TGR and UIE. Here again we see a linear relation between low goiter rates and the high urinary iodine values. Iodine deficiency is a disease of the soil. So, the impact indicators are determined by environmental and community factors. It cannot really be eradicated; only eliminated and controlled.

The three indicators mentioned in Table 6 assess different aspects of the IDD status in the community and thus help to track progress towards elimination of IDD. For example, iodine content of salt samples collected in a cross-sectional survey indicate the present level of iodine in salt, but gives no information about the variation occurring in the past. Total goiter rates show long term effects of bioavailability of

iodine and the urinary iodine excretion pattern reflects the existing levels of iodine intake and body iodine stores. To understand the status of IDD elimination programs, the result of these indicators should be viewed in totality. In addition, various environmental and community factors play a role in Phase II of the IDD elimination program. They are:

Severity of iodine deficiency

More severe the iodine deficiency, more intense efforts will be needed to bring the iodine deficiency under control.

Duration of iodine deficiency

How long the area/region has been under the effects of iodine deficiency is very important in the context of the program. Longer the duration, difficult it will be to bring the iodine deficiency under control.

Control measure

Monitoring of the control program must be in place. The persistence of goiter during phase II can be due to the consumption of non-iodized or inadequately iodized salt by part of the population. Moreover, it should be consumed on a regular basis. If it is not so, which is the case in many situations, a scenario emerges where there is no association between total goiter rate and urinary iodine excretion.
The population factors that play a part in phase II are: age, gender and physiological status such as pregnancy and lactation. It is seen that the effects of iodine deficiency tends to show up more vividly in the younger age groups; females during pregnancy and lactation become more vulnerable.

4.8.5. Sustainability of the IDD control program

The remarkable progress of Universal Salt Iodization in the current decade raises the issue of sustainability [5, 17, 54]. Indeed, sustainability is absolutely critical. IDD cannot be eradicated in one great global effort like smallpox and hopefully, poliomyelitis. Smallpox and poliomyelitis are infectious diseases with only one host – man. Once eliminated, they cannot come back. In contrast, IDD is a nutritional deficiency that is

primarily the result of deficiency of iodine in soil and water. It can therefore re-emerge at any time after elimination if iodine deficiency disorders control programs fail. Indeed, there is evidence that iodine deficiency has re-occurred in India (Kangra Valley, Himachal Pradesh) and some countries like Guatemala, Ecuador, Bolivia where it had been eliminated in the past [55].

Communications is an integral part of all actions: to ensure understanding of the problem; to understand the role of each agency; to understand the need for constant quality control and assurance procedures, processes and products; and to sustain the need for financial and other support once begun. There are four major components to consolidate the elimination of IDD and to sustain it forever:

- Sustained Political support
- Effective Administrative infrastructure
- On-going Assessment and monitoring
- Regular Communication

Sustained Political Support

This refers primarily to support at governmental level through the Ministry of Health and the executive group of the Government (cabinet or equivalent). Political support for the elimination of IDD depends on community awareness and understanding of the problem. Political support is essential for the passage of laws or regulations on salt iodization through the legislature. Since governments change, the mechanism to ensure continuity must be in place.

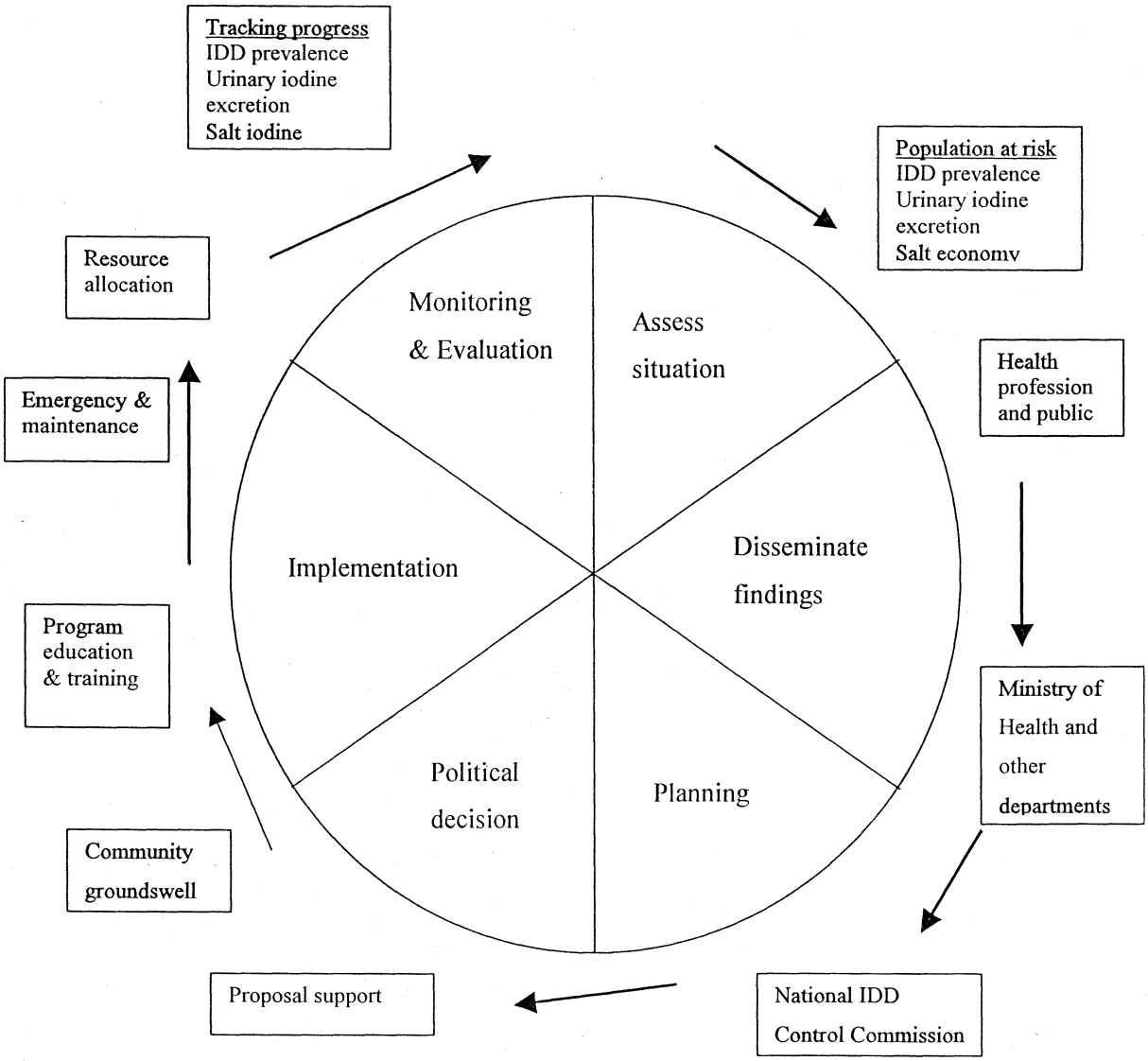
Effective Administrative Infrastructure

The National Body responsible for the management of IDD control program should operate with a process model. A useful example of such a process model is known as the social process model [5, 17, 54] or the 'wheel'. (Figure 3)

The Social Process model ('The Wheel') describes the iterative cycle of IDD elimination. The key lies in the sustainability of IDD elimination, not just its elimination. In order to adequately implement a health care program, a variety of components are necessary. Some of these components may appear to be peripheral to

the actual implementation process. For this reason, it is useful to take a holistic perspective.

Figure 3. Social process model of a national IDD control program



On-going Assessment, Monitoring

It is necessary to have adequate iodine content in the diet to prevent brain damage in the fetus and in the young infant when the brain is growing rapidly. The effectiveness of a national program is providing an adequate amount of iodine to the target population. This is reflected in measurements of salt iodine (at factory, retail and household level) and urinary iodine excretion (measured in casual samples from school children or households). Additional measures that would help in the assessment are estimation of thyroid size and blood tests. All these procedures require internal and external quality control in order to ensure reliability of the data collected. In order to be effective, the assessment and monitoring system needs:

- Laboratories for measurement of salt iodine and urine iodine, which must be available at country and regional level with some support from international laboratories for quality control.
- Production of quality assurance charts and databases at country level for recording the results of the regular monitoring procedures

The above must be backed up by the provision of adequate resources, which includes money, trained manpower, equipment and materials to support the implementation of salt iodization and the establishment of monitoring systems.

Regular Communication

An important aspect of sustaining the success of the elimination of iodine deficiency disorders is to open channels of communication with the various stakeholders, to reaffirm their commitment and dedication to the cause of sustainable IDD elimination.

4.9. Programs to control/eliminate IDD: Genesis and current situation

4.9.1. Global initiative

Successful national programs in Switzerland and USA preceded the birth of international program for elimination of IDD. Switzerland introduced the use of iodized salt in 1922. USA introduced the same in 1924. The success of elimination of IDD in those countries set the international efforts in motion.

There were a number of calls for the elimination of goiter by international organizations beginning with the World Food Council in 1974, followed by the General Assembly of the United Nations (1978) and the International Nutrition Congress (1978). After 1980, the Regional WHO Committees and Pan American Health Organization followed these with similar recommendations [56].

However, comparatively little happened over the decade of 1974-1983 in response to these resolutions. At that time, it was difficult to go beyond the term 'goiter' in discussing the problem of iodine deficiency. The concept of IDD at its present dimension was put forward at the Fourth Asian Congress of Nutrition (Bangkok 1983) when serious attention was given to the problem as worthy of priority in international nutrition. In 1984, this led to the discussion at Standing Committee on Nutrition (SCN) of the UN agencies. In response to that discussion a state-of-the-art review of IDD and the possibility of successful prevention and control on global basis was prepared and submitted by Dr. B.S.Hetzel in 1985 [57, 58].

The report noted the great delay in the application of existing knowledge on IDD and its prevention, to the detriment of the many millions in the developing countries. To bridge this gap, a group of concerned professionals was formed in 1986. This group comprises the International Council for Control of Iodine Deficiency Disorders (ICCIDD), an international non-government organization (NGO), which worked closely with WHO and UNICEF and thus led the crusade against IDD throughout the world [57, 58].

There has been a remarkable increase in the momentum of IDD control programs over the past decades. Great progress has been made through a global partnership between people, governments, non-governmental agencies and the expert multi-disciplinary network of the ICCIDD. In 1993, Kiwanis International along with UNICEF adopted the virtual elimination of IDD as their World Service Project. Salt industry has joined the partnership through a link established with ICCIDD. Global partnership with reference to the opportunity for greater coordination and more effective action to facilitate the achievement of the goal of elimination includes the following [57]

1. People in countries where IDD is a problem.
2. Governments of countries where IDD is a problem.
3. The international agencies, particularly WHO and UNICEF; Other UN agencies – the World Bank, UNDP, WFP and FAO are now playing a significant role.
4. The ICCIDD in association with WHO and UNICEF.
5. The bilateral agencies of developed countries, which have included national programs for IDD control in their development programs.
6. Kiwanis International – an international world service club, which has adopted the elimination of IDD as its international project.
7. The salt industry, which has formed a working relationship with ICCIDD.
8. Micronutrient Initiative – its mission is to facilitate the achievement of virtual elimination of IDD by supporting effective and sustainable program actions.

The ICCIDD has proposed to the WHO and UNICEF the establishment of a Global Verification Commission to assess the progress at the country level towards the goal of elimination. This will ensure longer-term sustainability of the elimination of IDD in a country. It is important particularly with reference to a number of countries in South America, which apparently achieved elimination at a particular point in time, but then in the absence of monitoring, control was lost and the problem recurred [59].

4.9.2. National initiative

National effort to understand the problem of IDD began in the decade of 1950. A survey conducted in district Kangra, Himachal Pradesh in 1956 reported a goiter prevalence of 55%. Similar reports came from other districts of the state. It led to a trial of iodized salt in the district coupled with ban on the sale of non-iodized salt [60]. Following this successful trial Government of India launched National Goiter Control Program in 1962. The objective of the program was to survey the problem of iodine deficiency in the country, produce and supply iodized salt and then resurvey after five years to assess the impact of the iodized salt program [5].

Initially, 12 salt iodization plants were installed. Production was far below the estimated 1 million tones needed annually for the endemic area alone. It was also felt

that the production and distribution through only public sector was not adequate to meet the growing demand. Moreover, distribution only in certain pocket of a district or state was administratively difficult. The technical goiter control review committee considered the entire situation in the backdrop of widespread endemicity of the disease [46]. Based on their recommendations, Government of India took an informed and historic decision in 1984 to universally iodinate all edible salt by 1990 giving the highest priority to hyper-endemic areas. The control of endemic goiter had been included in Prime Minister's 20-point programs. India became signatory to resolutions on elimination of iodine deficiency adopted by World Health Assembly in 1990, World Summit for Children in 1990, International Convention on the Rights of the Child and SAARC conference for children in 1992 [5, 59, 61].

Government also permitted commercial production of iodized salt in private sector. Production situation improved significantly. Salt industry had been mobilized resulting in increased production of iodized salt from a mere 0.2 million tones in 1983 to 4 million tones in 1998. All the states with the exception of Kerala had issued notification prohibiting the sale of salt other than iodized salt for edible purposes. Free trade prevails for iodized salt; however, with a view to make iodized salt available to the most vulnerable groups, 20 states and union territories have included iodized salt in the public distribution system (PDS) list fully or partially [59, 61].

The level of iodization was revised and fixed at 'not less than 30 ppm at the production level and not less than 15 ppm at the consumer level' [62, 63]. National program was re-designated as National Iodine Deficiency Disorders Control Program in 1992. As a part of the drive to prevent iodine deficiency disorders amongst the general public, the Government of India issued a notification with effect from May 1998 making a mandatory for all manufacturers of edible salt to iodize their product [8].

India had been making advances in the elimination of iodine deficiency. Over 70% of the population was consuming adequately iodized salt. India along with several other developing countries was on the brink of eliminating iodine deficiency [5, 63, 64]. Concurrently, the total goiter prevalence and the incidence of neonatal hypothyroidism have also reduced [65]. Further, urinary iodine excretion (UIE) levels among population in different states indicate adequate iodine intake [61, 64-67]. All these

findings have been further substantiated by reports on iodine content of salt samples analyzed and received from different states through monitoring information system (MIS) of Salt department and increase in production and supply of iodized salt. These findings indicate successful implementation of USI program in India [59].

However, over a period of time, a strong view has been expressed that a public health measure like mandatory iodization of edible salt should not be enforced through statutory provision. Any initiative in this regard to beneficial components of diet should be propagated through widespread publicity and dissemination of information. In this backdrop, Government of India issued a press note on May 11, 2000 stating 'It has been argued that, on a point of principle, compulsion in such matters of individual choice, is undesirable'. Government issued a preliminary notification on the same day proposing future withdrawal of the compulsory statutory iodization of edible salt and sought views and suggestions in this regard within 45 days of issuance of the notification [8]. Subsequently, Government of India revoked the statutory provision of mandatory iodization of edible salt in later part of 2000.

The decision of Government of India has the potential of sending wrong signals about the lack of interest of the government in the elimination of iodine deficiency, thus leading to disarray, disaster and dismantling of what had been achieved so patiently. Now, it would appear that policy had forsaken scientific advance, humanism and social development [61].

5. Methods

5.1. Study design

The study was a cross-sectional study.

5.2. Study period

The period of study was August to October 2004.

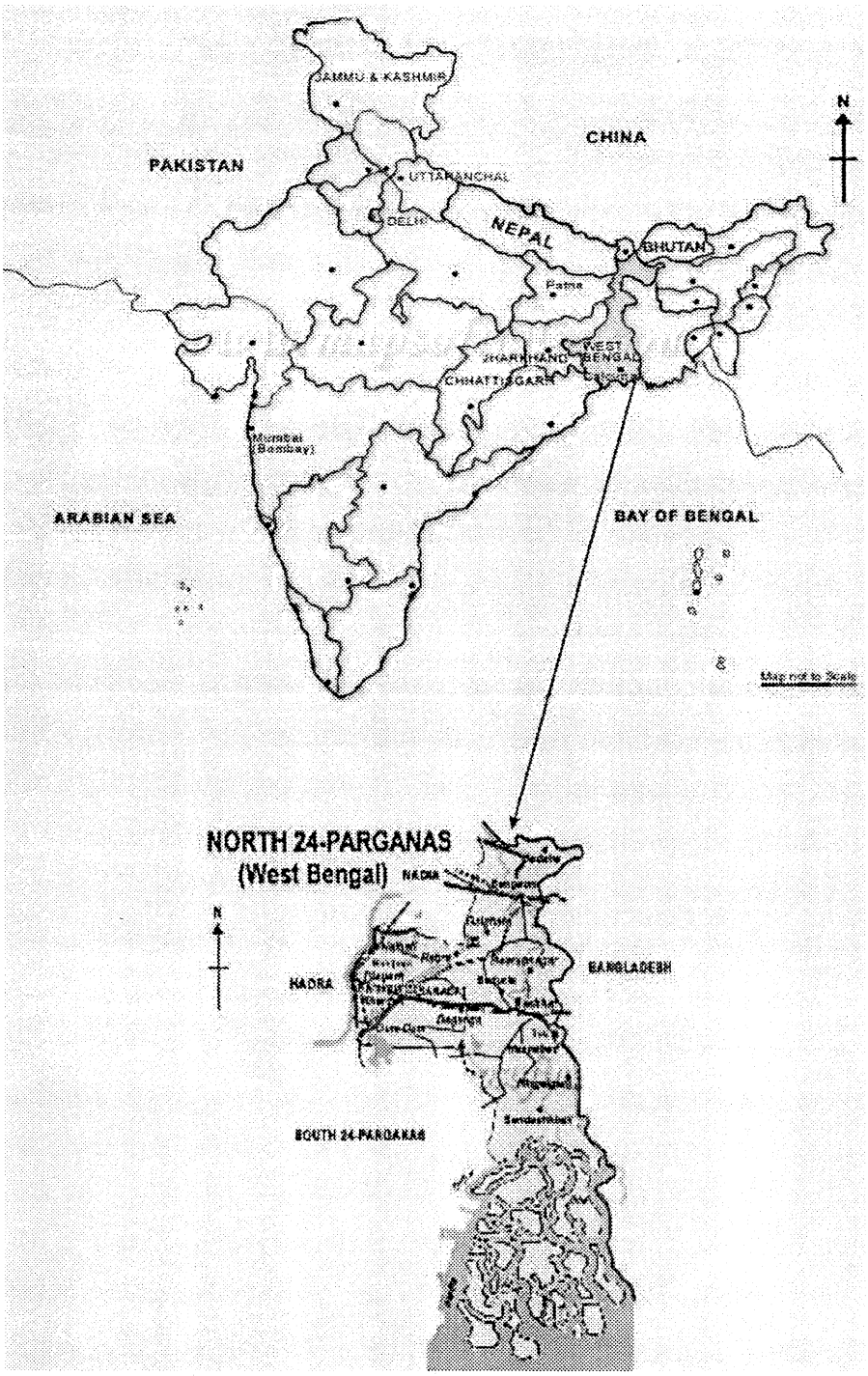
5.3. Study area

The study area was North 24 Parganas district, West Bengal, India. West Bengal is situated in the eastern part of the country. Of the 19 districts of the state, North 24 Parganas is the largest with 8.9 million populations (population density: 2181/sq. km.) as per Census 2001. There are 27 municipalities and 22 community development blocks in the district, of which 10 are riverine [68].

About 24% of the population belongs to backward section i.e. scheduled castes and scheduled tribes. Hindus constitute 75.5% of the population and Muslims 24.2%. Over all literacy rate in 2001 was 78.5%; female literacy rate being 72.1%. The district has a total of 4853 educational institutions; 3799 being primary schools [68].

The district is situated at the southern part of the state bordered by Bay of Bengal. Interspersed with a good number of rivers and rivulets in their way to Bay of Bengal and made up of alluvial soil of Gangetic delta, it is one of the most agriculturally fertile districts of the state. Flood and inundation of land by tidal water are chronic problem of two sub-divisions encompassing 13 community development blocks.

Figure 4. Map of India showing the State of West Bengal and North 24 Parganas district



5.4. Study population

The study was conducted among school children aged 8-10 years. School aged children are a useful target group for IDD surveillance because of their combined high vulnerability, easy access, high representativeness of their age group in the community and applicability to a variety of surveillance activities. They reflect current rather than remote iodine nutrition in the general population [69]. Affected children develop an enlarged thyroid in response to iodine deficiency and can be readily examined in large numbers in school settings. For that, the school based Probability Proportional to Size (PPS) cluster-sampling method is recommended as the most efficient and practical approach for assessing an iodine status or an IDD prevalence survey [9, 17].

A major concern arising in school-based surveys is that children not attending (less than 50%) schools are not represented, which possibly leads to biased prevalence estimates. Same concern is applicable to schools with low enrolment and/or high dropout rates [9, 17]. However, high school enrolment and low drop out rate of the schools of North 24 Parganas district justified the school approach of the present study [70].

Schoolchildren aged 6-12 years are the preferred group. There is a practical reason for not measuring very young age groups. The smaller the child, the smaller the thyroid and the more difficult it is to perform palpation. For that reason some studies have focused on 8-10 years [17].

5.5. Sample size

Recent studies in two districts of West Bengal following standard methodologies reported goiter prevalence of around 12% [10, 11]. The study district, North 24 Parganas, is riverine and flood-prone. So, iodine deficiency may be higher in this district. Thus, assuming goiter prevalence of 15% in the study district, confidence interval (CI) of ±5%, confidence coefficient (CC) of 95%, rate of homogeneity (RoH) as 0.02 [71] and cluster size 30, we calculated the sample size using *Right Size* [72] software. Thus the required sample size was calculated as follows:

Firstly, we calculated the design effect using the formula:

$$RoH = \frac{\text{Design effect} - 1}{\text{Cluster size} - 1}$$

$$0.02 = \frac{\text{Design effect} - 1}{30 - 1}$$ Therefore, Design effect = 1.58

Secondly, we calculated the total sample size using the following formula:

$$n = \frac{1.96^2 \text{ X P X (100-P) X Design effect}}{\text{Precision}^2}$$

$$n = \frac{1.96^2 \text{ X } 15 \text{ X } 85 \text{ X } 1.58}{5^2} = 310$$

We assumed a cluster size of 30; hence, we need to study 330 children from 11 clusters. Considering 10% non-response, final sample size of 363 school children aged 8-10 years was decided i.e. 33 study population per cluster (33 children X 11 clusters)

5.6. Sampling Method

We selected the final sample size of 363 school children by multistage cluster sampling technique as follows:

5.6.1. First stage: selection of clusters

We used the probability proportional to size (PPS) technique [9, 17] to select 11 clusters. We listed all clusters [villages (rural) / wards (urban) areas] in the study district along with their respective population as per Census 2001. Then, we selected 11 clusters in the district using standard PPS technique.

5.6.2. Second stage: selection of schools within the clusters

We met the District inspector of Primary schools and Chairman, District Primary School Council and briefed them the purpose of the study and its possible outcome. We also informed them about IDD and its impact on school going children and sought cooperation and permission for the conduct of the study. We organized two

workshops for sub-inspectors of schools to make them understand the purpose of the study and their role to conduct it successfully.

We collected the list of schools from the district authority of primary education and enlisted all the schools in each of the selected 11 clusters. From the sampling frame of primary schools in each cluster one school was chosen using Simple Random Sampling (SRS) technique for detailed survey. The selected 11 clusters and schools from where the study population was obtained are presented below:

Table 7. Selected clusters and schools in North 24 Parganas district

Cluster No.	Block/Municipality	Village/Ward	Primary School
1	Bhatpara Municipality	Ward 16	Lalita Devi Balika Prathamik Vidyalaya
2	Barrackpur Municipality	Ward10	Anandapuri Prathamik Sikshyatan
3	Panihati Municipality	Ward 6	Agarpara Balika Vidyalaya
4	Baranagar Municipality	Ward 18	Baranagar Sishubhavan Prathamik Vidyalaya
5	South Dumdum Municipality	Ward 33	Dakshindari Urdu Free Primary School
6	Bagda Block	Parkrishnachandrapur	Parkrishnapur FP School
7	Swarupnagar Block	Baro Bankra	Baro Bankra FP School
8	Barrackpur-2 Block	Bilkanda	SN Basu Smriti Free Primary school
9	Baduria Block	Raipur	Raipur FP School
10	Rajarhat Block	Baligori	Baligari FP School
11	Hingalganj Block	Jogeshganj	South Jogeshganj Free Primary School

5.6.3. Third stage: selection of study population within the clusters

We visited the randomly selected school in each cluster before the field survey. We informed the school authorities about the purpose and activities of the study and also informed them about IDD and its impact on school going children. We sought cooperation and permission for the conduct of the study at that school and requested them to ensure the attendance of the students on the day of survey.

On the day of survey, we prepared a sampling frame of all the children aged 8-10 years (based on date of birth in school record) in each selected school of 11 clusters. From that sampling frame, we included 33 children in the study using simple random sampling technique.

5.7. Data collection and assessment

A pre-designed and pre-tested schedule (Annexure) was used to record relevant data.

5.7.1. Assessment of thyroid size

The investigators (faculty members of department of community medicine, R.G.Kar Medical College, Kolkata) assessed thyroid size of 363 study children in 11 clusters. Thyroid size was assessed by standard palpation method. We trained the investigators prior to survey to minimize inter-observer variation. One of the researchers supervised and monitored the clinical assessment of goiter and principal researcher crosschecked 10% of the clinically assessed study subjects for goiter grades. We graded the goiter according to the classification laid down by WHO/UNICEF/ICCIDD: [9, 16, 17, 73]

Grade 0 No palpable or visible goiter.

Grade I A goiter that is palpable but not visible when the neck is in the normal position, (i.e., the thyroid is not visibly enlarged). Thyroid nodules in a thyroid, which is otherwise not enlarged, fall into this category.

Grade II A swelling in the neck that is clearly visible when the neck is in normal position and is consistent with an enlarged thyroid when the neck is palpated.

The Total Goiter rate (TGR) was calculated as the number of students with grade II and I goiter divided by total examined. It is expressed in percentage of the total examined. TGR was interpreted based on the criteria on goiter prevalence in school aged children designed by WHO, UNICEF and ICCIDD[9, 17]. It is given in table 8.

Table 8. Epidemiological criteria for assessing the severity of IDD based on goiter prevalence in school-aged children

Total Goiter Rate (TGR)	Degrees of IDD
0.0-4.9%	None
5.0-19.9%	Mild
20.0-29.9%	Moderate
30% and above	Severe

It is recommended that a TGR of 5% or more in school children 6-12 years of age be used to signal the presence of a public health problem. This recommendation is based on the observation that in normal, iodine-replete populations, the prevalence of goiter should be quite low. The cut off point of 5% allows both for some margin of error of goiter assessment, and for goiter that may occur in iodine-replete populations due to other causes such as goitrogens and auto-immune thyroid diseases. Also, the thyroid size in the community may not return to normal for months or years after correction of iodine deficiency[17].

5.7.2. Estimation of Urinary Iodine Excretion (UIE) level

Urinary iodine excretion levels of all the subjects surveyed in 11 clusters were estimated at the laboratory of department of biochemistry, Burdwan Medical College, Burdwan, West Bengal. Trained laboratory personnel estimated the UIE levels of urine samples. The researcher concerned crosschecked 10% of the samples.

We collected casual on the spot urine sample (5-10 ml) in wide mouthed screw capped plastic bottles containing one/two drops of toluene to minimize any unpleasant smell. We stored the samples in a refrigerator at 4^0C until analysis. We estimated the urinary iodine by 'Wet Digestion Method (Sandell-Kolthoff)' [74, 75] and expressed the result in µg/L of urine.

Urinary iodine values from population are usually not normally distributed. Therefore, the median rather than mean should be used as the measure of central tendency. Likewise, percentiles rather than standard deviation should be used as measure of dispersion. This methodology was followed in the present study. The distribution of median urinary excretion levels is characterized according to certain cut-off points and intervals by WHO/UNICEF/ICCIDD. The cut-off points proposed for classifying iodine nutrition [17, 73] into different degrees of public health significance are described in Table 9.

Table 9. Epidemiological criteria for assessing iodine nutrition based on median urinary iodine concentrations in school-aged children

Median urinary iodine (µg/L)	Iodine intake	Iodine nutrition
< 20	Insufficient	Severe iodine deficiency
20-49	Insufficient	Moderate iodine deficiency
50-99	Insufficient	Mild iodine deficiency
100-199	Adequate	Optimal
200-299	More than adequate	Risk of iodine induced hyperthyroidism within 5-10 years following introduction of iodized salt in susceptible groups
300 and above	Excessive	Risk of adverse health consequences (iodine induced hyperthyroidism, autoimmune thyroid diseases

Median urinary iodine concentrations of 100 µg/L and above define a population with no iodine deficiency i.e., at least 50% of the samples should be above 100 µg/L. In addition, not more than 20% samples should be below 50 µg/L. in population characterized by longstanding iodine deficiency and rapid increment in iodine intake, median value(s) of urinary iodine above 200 µg/L are not recommended because of the risk of iodine-induced hyperthyroidism. This adverse reaction can occur during the 5 to 10 years following the introduction of iodized salt. Beyond this period of time, median values up to 300 µg/L have not demonstrated any side effects, at least not in population with adequately iodized salt [76, 77].

Urinary Iodine Estimation by the Wet Digestion Method (Sandell-Kolthoff) [75]

There are many methods available for the destruction of the organic content of the sample. We used this method in our laboratory for its simplicity and convenience.

Reagents:

1) Percholoric acid 70%, Analytical Reagent (AR) Grade

2) Vanadium pentoxide (V_2O_5) CDH Grade. 250 mg of V_2O_5 dissolved in 25 ml of H_2SO_4 (concentrated sulphuric acid diluted 10 times)

3) Arsenite reagent: A.R. Grade. 1.135 gm of sodium metarsenite and 1 gm of sodium chloride dissolved in 200 ml of water; 46 ml of concentrated sulphuric acid added; the volume to 500 ml was made up with double distilled water.

4) Cerate Reagent: A.R. Grade. 5 gm of ceric ammonium sulphate dissolved in about 200 ml of double distilled water and 135 ml of concentrated sulphuric acid added; the volume to 500 ml was made up with double distilled water.

Stock standard:

100 mg% of iodine i.e. 168.5 mg of KIO_3 / 100 ml of H_2O

Working standard:

100 µg/L, 200 µg/L, 300 µg/L and 400 µg/L

Procedure:

100 µl of sample (urine) and standards were taken. 2 ml of perchloric acid (70%) was added to all the tubes. 150 µl of vanadium pentoxide was added to all the tubes and mixed well. All tubes were then kept in sand bath at 160^0C (in a hot air oven) for 45 minutes. All tubes were removed from the oven, cooled and 2 ml of Arsenite reagent was added to all tubes. Test tube stand with test tubes was placed in a water bath at 48^0C and it was allowed to stabilize. 1 ml of cerate was added to all the test tubes at 30-second interval and read at a wavelength of 420 nm in a colorimeter at 30-second interval.

The assay time was 25 minutes; so a maximum of only 50 tubes was run in one assay.

Factors influencing the rate of reaction:

1. Temperature: The sensitivity of the reaction increases in temperature and is best achieved at a temperature of 48-50^{0}C. But temperature above 56^{0}C should be avoided, as there is cerate reduction even without iodine as catalyst.
2. Reaction time: A time period of 25 minutes is generally employed.

As the method adopted was very tricky, following precautions was followed:

1. All water and glassware used were iodine free.
2. Glassware to be used were kept in chromic acid for 24 hours; then, thoroughly rinsed with tap water and double distilled water (while distilling $KmnO_4$ is added in the distillation flask). Final rinsing was done with methanol.
3. Reagents used were iodine free and also mercury free as they may interfere with Sandel kolthoff reaction.
4. Vanadium pentoxide used was of a same lot.
5. Only AR graded sulphuric acid was used.

5.7.3. Assessment of the iodine content of the salt consumed

There are a large number of test kits available in the market. UNICEF also supplies countries with test kits. Rapid test kits used in this study (Batch No. 076; Mfg. Aug. 2004; Exp. Jan. 2006) were collected from UNICEF sponsored establishment.

These are small bottles of 10-50 ml (in this study, 10 ml bottle), containing a stabilized starch-based solution. One drop of the solution placed on salt produces characteristic coloration. These kits should therefore be regarded as qualitative rather than quantitative. We interpreted the test results based on following criteria:

Coloration	Iodine content (in ppm)	Inference
Nil	0	No iodine
Light blue	7	Insufficient iodine
Blue	15	Sufficient iodine
Deep violet	30	Sufficient iodine

An advantage of the rapid test kit is that it can be used in the field to give an immediate result. However, they do not give a reliable estimate of iodine content, results must be backed by titration [78, 79].

We asked all the study subjects to bring samples (around 20 gm) of cooking salt from their respective households on the day of survey. We supplied auto-seal polythene pouches to the children through the school authorities prior to the day of survey. We collected the salt samples from the study children on the day of survey and estimated the iodine content of the salt samples by rapid iodine testing kit [74].

We assessed the iodine content of the salt samples brought by all the 363 study children on the spot by rapid iodine testing kit. One of the researchers monitored and supervised the collection and testing of the salt samples and 10% of the salt samples were crosschecked.

5.8. Data entry and analysis

The data entry and analysis was done at R.G.Kar Medical College, Kolkata, West Bengal and the office of the principal researcher. We entered the data into a dBase® file and double-checked the data entry for errors. We analyzed the data using *Epi Info 6.04d* [80] epidemiological software program.

We performed univariate analysis to examine any statistical significance between the outcome and response variables. The chi square ($?^2$) test (Yates corrected) was used and 'p' values less than 0.05 were considered statistically significant.

We did two separate multiple linear regression analysis using *Epi Info 2002* [81] to assess the independent association of selected factors with UIE and with salt iodine level. Logistic regression model was carried out to assess the independent association of factors with the categorical outcome variables, namely, presence of goiter and adequate level of iodine in salt. The covariates considered for assessing the association were age, sex, religion and place of residence.

6. Results

6.1. Description of the study population

We studied 363 children aged 8 to 10 years [Mean (S.D): 8.95 (0.8)]. Proportions of males and females were 51.2% and 48.8%, respectively. Mean (S.D) age of males and females were 8.9 (0.8) years and 9.1(0.8) years respectively. 65.3% of the study subjects belonged to Hindu religion and rest Muslims; 54.5% live in rural area. All the sampled school students participated in the survey.

Table 10. Distribution of the study population by age and sex, North 24 Parganas, West Bengal, 2004

Age (Years)	Males	Females	Total
8	75 (40.3%)	50 (28.2%)	125 (34.4%)
9	62 (33.3%)	66 (37.3%)	128 (35.3%)
10	49 (26.4%)	61 (34.5%)	110 (30.3%)
Total	186 (51.2%)	177 (48.8%)	363 (100%)

6.2. Assessment of goiter size of the study population

Table 11 provides the total goiter rate and age and sex-wise prevalence. The Total Goiter Rate (TGR) was 20.1% (95% CI: 16.78-23.44). Age specific TGR of 8, 9 and 10 year groups were not significantly different ($\chi^2 = 0.8$, df = 2, p = 0.7). Difference in sex specific TGR was also not significant ($\chi^2 = 1.33$; df = 1; p = 0.2). TGR among females showed increase with age ($\chi^2 = 0.6$, df = 1, p = 0.4). TGR was comparatively low in 8-year age group among the females and almost same in all age group in boys. Prevalence of Grade I and grade II goiter were 15.4% and 4.7% among the study population.

Table 11. Age and sex wise distribution of goiter prevalence, North 24 Parganas, West Bengal, 2004

Age	Male (n=186)				Female (n=177)				Combined (n=363)
	Goiter grade				Goiter grade				
	0	I	II	TGR (%)	0	I	II	TGR (%)	TGR (%)
8 yrs (n=125)	62	13	0	17.3	41	4	5	18	17.6
9 yrs (n=128)	50	11	1	19.3	50	13	3	24.2	21.9
10 yrs (n=110)	41	6	2	16.3	46	9	6	24.6	20.9
All	153	30	3	17.7	137	26	14	22.6	20.1

Table 12 provides TGR by religion with no significant difference between Hindus and Muslims ($\chi^2 = 0.8$, df = 1, p = 0.4).

Table 12. Goiter grades in children by religion, North 24 Parganas, West Bengal, 2004

Goiter grade	Hindu (n=237)	Muslim (n=126)	Total (n=363)
0	186 (78.5%)	104 (82.5%)	290 (79.9%)
I	38 (16.0%)	18 (14.3%)	56 (15.4%)
II	13 (5.50%)	4 (3.20%)	17 (4.70%)
TGR	21.5%	17.5%	20.1%

Difference in TGR among children residing in rural area and in the urban area (Table 13) was not significant (χ^2 =1.2, df = 1, p = 0.3). However, goiter prevalence was higher among children residing in rural area.

Table 13. Goiter grades in children by residence, North 24 Parganas, West Bengal, 2004

Goiter grade	Rural (n=198)	Urban (n=165)	Total (n=363)
0	154 (77.8%)	136 (82.4%)	290 (79.9%)
I	37 (18.7%)	19 (11.5%)	56 (15.4%)
II	7 (3.50%)	10 (6.10%)	17 (4.70%)
TGR	22.2%	17.6%	20.1%

6.3. Estimation of Urinary Iodine Excretion (UIE) levels

We collected 363 urine samples for estimating urinary iodine excretion levels and analyzed 358 samples. Five urine samples were discarded due to contamination. The median urinary iodine excretion level was found to be 160 µg/L. The range of urinary iodine excretion values was from 5 to 330 µg/L. 72% of the values was 100µg/L or above. The distribution is shown in Figure 5. The median UIE values were 150 µg/L and 160 µg/L among males and females respectively.

Figure 5. Distribution of urinary iodine excretion values in the study population, North 24 Parganas, West Bengal, 2004

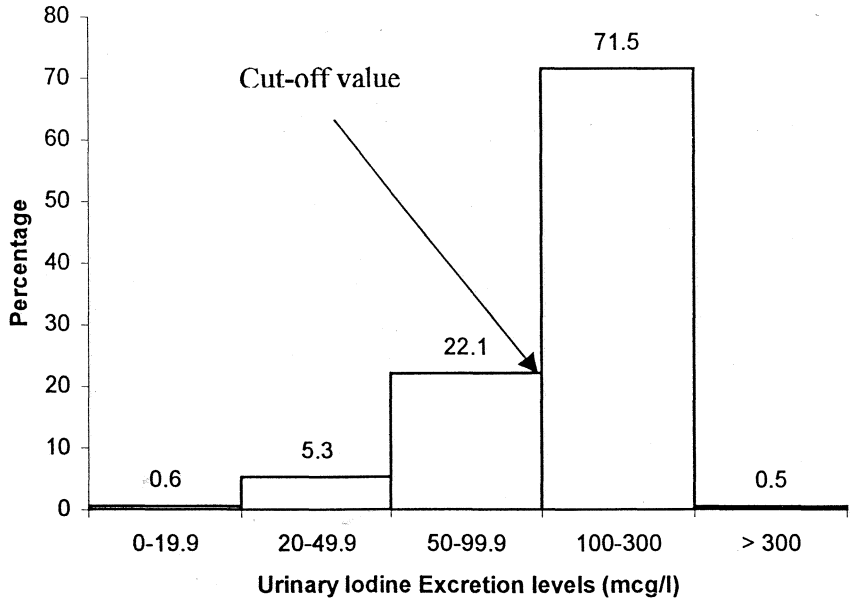


Table 14 provided the age specific UIE levels. There was no significant difference among age groups ($\chi^2 = 2.49$; df = 2; p = 0.3).

Table 14. Distribution of urinary iodine excretion values by age, North 24 Parganas, West Bengal, 2004

UIE (in µg/L)	8 years (n=124)	9 years (n=127)	10 years (n=107)	Total (n=358)
0-19.9	0 (0.0%)	1 (0.80%)	1 (0.90%)	2 (0.60%)
20-49.9	6 (4.80%)	7 (5.50%)	6 (5.60%)	19 (5.30%)
50-99.9	30 (24.2%)	32 (25.2%)	17 (15.9%)	79 (22.1%)
100 or above	88 (71.0%)	87 (68.5%)	83 (77.6%)	258 (72.0%)

The sex specific values of UIE less than 100 µg/L showed significant difference ($\chi^2 = 5.42$; df = 1; p = 0.02). (Table 15)

Table 15. Distribution of urinary iodine excretion values by sex, North 24 Parganas, West Bengal, 2004

UIE (in µg/L)	Male (n=183)	Female (n=175)	Total (n=358)
0-19.9	0 (0.00%)	2 (1.10%)	2 (0.60%)
20-49.9	9 (4.90%)	10 (5.70%)	19 (5.30%)
50-99.9	52 (28.4%)	27 (15.4%)	79 (22.1%)
100 or above	122 (66.7%)	136 (77.8%)	258 (72.0%)

The values of UIE less than 100 µg/L were significantly different among rural and urban residents ($\chi^2 = 4.07$; df = 1; p = 0.04). Higher proportion of children residing in rural area had UIE values less than 100 µg/L (Table 16).

Table 16. Distribution of urinary iodine excretion values by residence, North 24 Parganas, West Bengal, 2004

UIE (in µg/L)	Rural (n=195)	Urban (n=163)	Total (n=358)
0-19.9	0 (0.00%)	2 (1.20%)	2 (0.60%)
20-49.9	13 (6.70%)	6 (3.70%)	19 (5.30%)
50-99.9	50 (25.6%)	29 (17.8%)	79 (22.1%)
100 or above	132 (67.7%)	126 (77.3%)	258 (72.0%)

Table 17 shows that the proportion of UIE values less than 100 µg/L was higher among Muslims than Hindus ($\chi^2 = 0.4$; df = 1; p = 0.5).

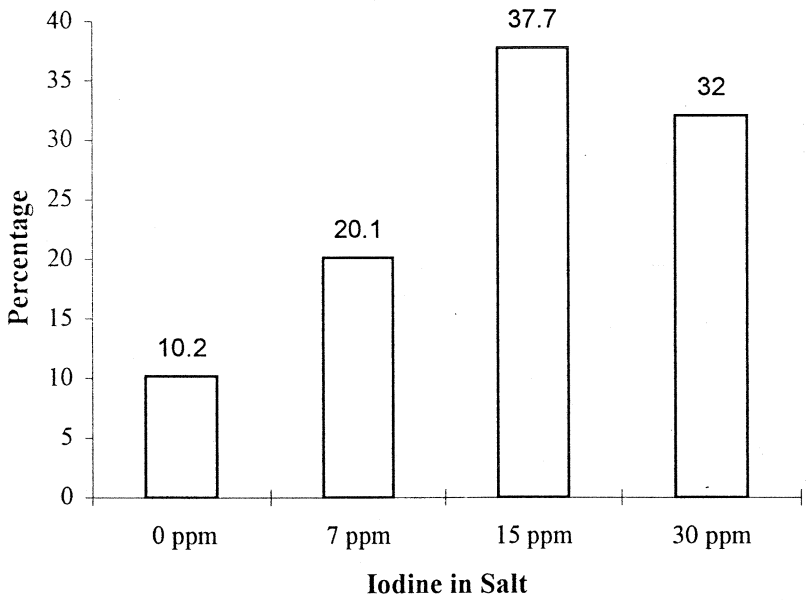
Table 17. Distribution of urinary iodine excretion values by religion, North 24 Parganas, West Bengal, 2004

UIE (in µg/L)	Hindu (n=235)	Muslim (n=123)	Total (n=358)
0-19.9	0 (0.00%)	2 (1.60%)	2 (0.60%)
20-49.9	15 (6.40%)	4 (3.30%)	19 (5.30%)
50-99.9	48 (20.4%)	31 (25.2%)	79 (22.1%)
100 or above	172 (73.2%)	86 (69.9%)	258 (72.0%)

6.4. Assessment of iodine content of the salt samples

We analyzed a total of 363 salt samples. The proportion of households consuming adequately (15 ppm or more) iodized salt was 69.7%. The total number of salt samples with some iodine was 326 (89.8%). The distribution is given in figure 6.

Figure 6. Distribution of salt iodine levels at the households of the study population (n= 363), North 24 Parganas, West Bengal, 2004



31.8% of the study population residing in rural area consumed less iodized salt (< 15 ppm), whereas the proportion was 28.5% in urban area. The difference was not significantly ($\chi^2 = 0.5$; df = 1; p = 0.5). (Table 18)

Table 18. Distribution of salt iodine level by residence, North 24 Parganas, West Bengal, 2004

Salt iodine (in ppm)	Rural (n=198)	Urban (165)	Total (n=363)
0	17 (8.60%)	20 (12.1%)	37 (10.2%)
7	46 (23.2%)	27 (16.4%)	73 (20.1%)
15	80 (40.4%)	57 (34.5%)	137 (37.7%)
30	55 (27.8%)	61 (37.0%)	116 (32.0%)

Consumption of less iodized salt (less than 15 ppm) was significantly higher ($\chi^2 = 24.94$; df = 1; p = 0.00001) among Muslims (46.9%) than the Hindus (21.6%).

Table 19. Distribution of salt iodine level by religion, North 24 Parganas, West Bengal, 2004

Salt iodine (in ppm)	Hindu (n=237)	Muslim (n=126)	Total (n=363)
0	13 (5.50%)	24 (19.1%)	37 (10.2%)
7	38 (16.1%)	35 (27.8%)	73 (20.1%)
15	93 (39.2%)	44 (34.9%)	137 (37.7%)
30	93 (39.2%)	23 (18.2%)	116 (32.0%)

6.5. Multivariate analysis

We considered UIE level as a categorical outcome variable (above and below 100 µg/L) and did a multiple logistic regression analysis to examine whether any of the variables, particularly sex and place of residence (urban or rural), was associated with UIE level, after being adjusted for other variables. None showed any association. However, the variables, place of residence and sex had odds ratios of 1.6 each, and were close to statistical significance level (Table 20).

Table 20. Logistic regression model of associations of UIE with place of residence and sex (adjusting for age and religion), North 24 Parganas, West Bengal, India, 2004

Term	Odds Ratio	95% CI	Coefficient	S. E.	Z-Statistic	P-Value
Residence	1.59	0.97-2.59	0.46	0.25	1.84	0.07
Sex	1.60	0.99-2.60	0.47	0.25	1.90	0.06

Considering actual values of UIE as outcome variable, we did a multiple linear regression analysis to examine whether any of the variables (age, sex, religion and place of residence) was associated with the UIE level after being adjusted for the others. The assessment revealed that place of residence was significantly associated (Coefficient: 1.95; S.E.: 0.77; F-test: 6.45; p-value: 0.01). Considering salt iodine level as the outcome variable, religion was observed to be significantly associated (Coefficient: -6.13; S.E.: 1.10; F-test: 30.97; p-value: <0.0001).

7. Discussion

The present study provided the opportunity to assess the magnitude of iodine deficiency disorders in the North 24 Parganas district of the state of West Bengal by applying standard internationally accepted methods. It highlighted that the district is now passing through the phase of iodine deficiency to iodine sufficiency manifested by median urinary iodine excretion level within normal limit despite high goiter prevalence. The high prevalence of goiter and around 30% less than the expected level of universal salt iodization coverage in the district, as has been found in the present study, underline the importance of strengthening the IDD elimination program.

Demographic characteristics

Both the sexes were almost equally represented among the study population; female-male ratio being 1: 1.05. These suggest almost uniform representation of sex and age in the study population. Lower proportion of female students in 8 years age group in comparison to 9 and 10 years might be due to late entry of females in schools. Further study with larger sample size is needed to infer this.

Hindus by religion were majority among the study population, which corroborates with the population characteristic by religion of the district. The urban-rural divide of the population of the district is almost same. These suggest that the study population characteristics by religion and residence closely resemble with those of the district.

Prevalence of goiter

The prevalence of goiter estimated in this study was considerably higher than the allowable limit of 5%. In similar school-based studies conducted in other districts of the state, the prevalence of goiter were 11.3% in Malda district [10], 12.6% in Birbhum district [11] and 37.6% in Howrah district [45]. The high total goiter rates found in other districts corroborate with the finding of the present study. Considering the TGR found in the present study with the findings of other districts of the state, it may be said that iodine deficiency is still a public health problem in West Bengal.

Similar high TGR was found in other states of India also. Studies in Delhi (2002) [82] showed a TGR of 6.2%, in Bikaner, Rajasthan (1995) [83] 20%, in Bargarh, Orissa (1996) [84] 10.8%, in Meerut, Uttar Pradesh (1999) 11.6% [85], in Kerala (2001) [54] 16.6% and in Bharatpur, Rajasthan (2002) 7.2% [86]. These observations are similar to the finding of the present study and also signify that iodine deficiency is a problem not of the state of West Bengal in particular, but also of the whole country.

Total goiter rate was found higher in other countries too. A study conducted in Bhutan in 1996 showed a TGR of 14% among school aged children [87]; in Nepal (1998) [88] it was found to be 40% among school aged children and 50% among women of child bearing age. School based studies in some central Asian countries also showed high prevalence of goiter. Iran (1996-97) showed a TGR as high as 25% [89]. In Egypt, several studies reported high goiter prevalence among school aged children, e.g. in Kafr Elsheikh, (1995) 27.1% [90], in Cairo (1991) 13.5% [91], in Aswan (1995) 17.5% [92]. A study in Uganda, Africa (1999) TGR was found to be 60.2% [93]. The findings of the studies indicate that IDD is a major problem in the third world countries. However, a study conducted in central coast of Australia [94] in 2000 revealed TGR among girls as 4% and among boys 3% and another study in Lesotho [95], it was 4.9%. Possibly, it was due to better control of IDD in Australia and Lesotho through universal salt iodization. Similar findings were observed in other first world countries where IDD elimination programs have entered in the third phase i.e. the phase of sustainability.

The present study was conducted among school children of 8 to 10 years of age. We observed that total goiter rates by sex and overall age groups were not significantly different. TGR was found ascending with the increase in age among females, though not differing significantly. Grade II goiters were also higher among females than males. This might be due to the effect of puberty. Among males the rates were almost same.

Total Goiter rate was found higher among Hindus than Muslims with no significant difference. We may suggest iodine deficiency affected the population irrespective of their religious belief in the past and it was manifested by TGR, the historical marker of iodine deficiency in the community. The picture was similar when we consider TGR among the study population by their place of residence. Here, also TGR was

higher, though not different significantly, among rural residents in comparison to urban study subjects. It may be due to limited access of the rural population to iodized salt. However, both the issues i.e. religion and residence as variables to influence goiter prevalence have not been addressed in the present study. Further studies with proper sample size and following appropriate methodologies are needed to find out any possible association.

Estimation of urinary iodine excretion level

In the present study, we estimated overall median urinary iodine excretion (UIE) value fairly higher than the cut off value of 100 µg/L; the sex specific median UIE values were also above the cut off limit. The proportions of values less than 100 µg/L and less than 50 µg/L were well below the program targets of <50% and <20%, respectively. These findings along with median UIE values indicate that there is no current iodine deficiency in the district, though high prevalence of goiter signifies that iodine deficiency was present in the same community in the recent past and still it is a public health problem.

Similar observations i.e. high TGR with median UIE level higher than the cut off value of 100 µg/L, were found in a number of studies, particularly where IDD control programs have been going on for last few years. In the state, the study conducted in Malda [10] revealed median UIE level 150 µg/L, in Birbhum [11] 124 µg/L. These findings corroborate with that of the present study. The study conducted in Howrah [45] district revealed median UIE level as high as 350 µg/L.

Studies conducted in the other states of the country also showed similar picture. Median UIE level found in two studies conducted in Delhi were 198 µg/L [96] and 200 µg/L [82]. Studies in Bargah district, Orissa [84], in Meerut, Uttar pradesh [85], in Kerala [54], in Bharatpur, Rajasthan [86] revealed similar findings as in the present study. Studies conducted in other third world countries also showed similar results [87-90, 93, 95]. Study in Bangladesh [54] revealed that it was low (63 µg/L) in the hilly area, whereas it was within normal limit in the plains.

Proportions of study population below 100 µg/L and 50 µg/L by age groups revealed no significant differences suggesting iodine deficiency affected the community irrespective of their age.

We found the urinary iodine excretion (UIE) level below 100 µg/L was significantly higher in males compared with that of females. However, the median UIE level among males was within normal range. These findings should be interpreted cautiously, as the present study was not designed to elicit such associations. Further study with appropriate sample size is necessary to draw any inference.

The proportion of study population having urinary iodine excretion (UIE) level below 100 µg/L was significantly higher among rural residents compared with that of urban study population. TGR was also higher among the rural subjects than the urban ones, though not significantly different. It suggests the possibility of higher degree of iodine deficiency among the rural population. Further study with appropriate sample size and methodology may test this hypothesis.

The proportions of urinary iodine excretion (UIE) levels below 100 µg/L were not significantly higher among Muslims compared with Hindus. It suggests iodine deficiency is currently affecting the community irrespective of their religious belief.

Multiple linear regression analysis suggested that UIE is associated with the place of residence i.e. the rural children is associated with lower UIE values. However, the model was rather weak, with the r^2 (coefficient of determination) value of 0.02. In the logistic regression model, none of the variables were statistically associated. Inadequacy of sample size to detect such association could be a possibility as two variables, namely, place of residence and sex was very close to statistical significance.

Assessment of iodine content of salt samples

The proportion of the households of the study population consuming adequately iodized salt is much less than the expected level (Program target: At least 90% coverage). However, only 10% were consuming salt with no iodine. It suggests that

the health authority is implementing universal salt iodization (USI) program successfully. What is needed may be to monitor and supervise the program more intensively and increase the awareness of the community regarding usefulness of the iodized salt in the context of possible outcome of IDD. Inadequate presence of iodine in salt among substantial proportion of population (salt samples with iodine content 7 ppm) might be due to inappropriate preservation, under or uneven iodination at the production point or loss during transport due to poor packaging. Taking care of these aspects may increase the effectiveness of USI and so improve the status of IDD in the district [5, 6, 14, 97].

The National Family Health Survey - 2 (NFHS - 2), a nationwide survey conducted in the year 1998- 1999, assessed, among other indicators, the proportion of households consuming adequately iodized salt, i.e. salt with an iodine content of at least 15 parts per million (15 mg of iodine per 1 kg of salt). This was done using salt testing kits. As per the survey data, 62% of the households in West Bengal consume adequately iodized salt [44]. It corroborates with the findings of the present study.

Urinary iodine concentration is currently the most practical biochemical marker for iodine nutrition, when carried out with appropriate technology and sampling. It assesses iodine nutrition only at the time of measurement, whereas thyroid size reflects iodine nutrition over months or years. Therefore, populations may have attained iodine sufficiency as expressed by median urinary iodine concentration. Yet goiter may persist even in children as there is a lead time required for the goiter prevalence rate in population to decline as a result of regularly consuming adequately iodized salt. From the above data, it may be reasonable to infer that iodized salt contributes considerably to the iodine intake of the population.

The proportion of study population consuming non-iodized/inadequately iodized salt in urban and rural areas did not show any significant difference. It suggests that the USI program is being implemented in both rural and urban areas and residents of both the areas have almost equal access to iodized salt.

However, consumption of non-iodized/inadequately iodized salt was significantly higher among Muslims than the Hindus. The multiple linear regression model

adjusting for age, sex and place of residence, confirmed the findings of univariate analysis that the religion was associated. That is, Muslims consume less of iodized salt. However, the impact of this was not seen either in the prevalence of goiter or UIE level. This might be due to adequate consumption of iodine through some other sources on which we have no information. However, present study has not addressed this issue, as it has not been designed to do so. Further study with appropriate sample size and methodology is needed to test this hypothesis.

All the above observations suggest that females and rural population are more prone to develop IDD. Salt iodization program had less access among the minority population, the Muslims. However, these findings need cautious interpretation before drawing an inference.

Other possible factors

Some other possibilities may be considered to understand the presence of high goiter rate and adequacy of urinary iodine excretion in North 24 Parganas district. They are: iodine intake from the sea fish, increased intake of goitrogen by food and other sources of iodine [98, 99]. These variables have not been addressed in the present study. However, further studies on these aspects can ascertain their contribution.

Under normal conditions, 99% of the iodine that is ingested gets excreted in the urine provided the body iodine stores are adequate. If one were to get all the iodine from sources other than iodized salt then the urine iodine would have been significantly lower. Also, if other food factors were to have contributed to the iodine intake, the median UIE would have been much higher. However, the results show that nearly 70% of the population is consuming adequately iodized salt and also that 72% of the population have a UIE value above 100 μg/L. This indicates that iodine in the iodized salt contributes considerably to the iodine nutrition in North 24 Parganas district.

In the context of the determinants of IDD elimination, the above findings of the present study conducted in North 24 Parganas district needs to be looked into in totality. WHO/UNICEF/ICCIDD has recommended indicators to determine whether IDD exists as a public health problem in the surveyed region or area. The indicators

and criteria for classifying IDD as a public health problem are outlined in Table 6. As per these criteria, North 24 Parganas district is 'moderately' endemic for iodine deficiency disorders in view of its 20.1% total goiter rate. However, adequacy of iodine nutrition is shown by the median urine iodine excretion (160 µg/L). It indicates that there is no current iodine deficiency. It is evident that a non-linear relationship is manifested in the study district with high goiter rate and high median urinary iodine excretion value. Moreover, in the attainment of adequacy of salt iodization at the household level, it still has to achieve the same success. The present status of IDD elimination in the district is summarized below:

Table 21. Criteria for tracking progress towards eliminating IDD

Indicator	**Goal**	**North 24 Parganas District**
Thyroid size (Age group 6-12)		
Proportion with enlarged thyroid	< 5%	20.1%
Urinary Iodine		
Median Urinary Iodine	> 100 µg/L	160 µg/L
Proportion below 100 µg/L	< 50%	27%
Proportion below 50 µg/L	< 20%	5.9%
Salt Iodization		
Proportion of households consuming adequately iodized salt (15 ppm or above)	> 90%	69.7%

All these signify that the district is passing through the phase II of IDD elimination (phase of community intervention) i.e. from iodine deficient to iodine sufficient state. North 24 Parganas district will likely be able to reduce TGR below 5 percent as soon as the proportion of consumption of iodized salt at the household level reaches > 90%, and is maintained at that level, thereafter. So, the main challenge before the district is to intensify the elimination program so that it can enter into phase III (phase of sustainability) and sustain that to be an IDD free district.

Internal validity

Selection of clusters was done following probability proportional to size (PPS) sampling technique. Schools within the clusters and study subjects within the schools were selected by simple random sampling to avoid any bias in selection.

In the present study, we estimated the goiter prevalence by palpation of the thyroid. The specificity and sensitivity of palpation may be low in grades 0 and I due to high inter-observer variation. Misclassification can be high. To minimize the inter-observer variation, the four field investigators were trained during preparatory phase regarding standard method of assessing goiter. During survey, one of the researchers supervised and monitored the clinical assessment of the investigators and principal researcher crosschecked 10% of the clinically assessed study subjects for goiter grades.

External validity

We conducted the present study among school children of 8-10 years of age. The characteristics of the school children in other districts of the state may not be very different from those of the study population in North 24 Parganas district. Hence, the magnitude of the iodine deficiency disorders may be similar among the school children of 8-10 years age of other districts.

The present study was not designed to find out associations between some demographic variables (e.g. age, sex, residence, religion) and goiter prevalence, UIE values and salt iodine levels. The findings of the present study in relation to these variables cannot be generalized unless tested by further study with appropriate design and sample size.

Limitations of the study

The present study is cross-sectional in design. So, it is limited in finding any causal association.

Dates of birth of the students were collected from the school registers. It was not checked with the parents or crosschecked with the birth certificates.

Measurement bias through inter-observer variation might influence the study estimates. However, training was imparted to investigators and crosschecking of clinical assessments was done to minimize this shortcoming.

Iodine content of the salt was tested by spot iodine testing kits. This method is regarded as qualitative rather than quantitative. Titration, which is a better method of quantitative estimation, was not done.

Future studies

Two factors may explain this insufficient iodization as has been found in the present study. These are: (a) the lack of awareness in the community regarding the impact of iodine deficiency disorders and the role of iodized salt to prevent them and (b) the absence of ban on the production and sale of non-iodized salt. These two factors may affect the poor more. Additional data regarding the vulnerability of the poor to iodine deficiency disorders would allow formulating better prevention policy.

In this context, a study to estimate the prevalence of iodized salt consumption in households in the North 24 Parganas district, West Bengal, India according to the (a) socio-economic characteristics and (b) level of awareness about iodine deficiency disorders may be done. Along with this objective, we may describe the availability and cost of adequately iodized salt at the retail shops in the district.

The proposal for a study with the above-mentioned objectives has already been placed for consideration.

8. Conclusions

1) The present study suggests endemic goiter as a public health problem in North 24 Parganas district.

2) The median urinary iodine excretion value suggests current iodine nutritive status is adequate in North 24 Parganas district.

3) The district is passing through phase II (phase of community intervention) i.e. from iodine deficient to iodine sufficient state.

4) Consumption of adequately iodized salt at the household level is substantially below the program target of greater than 90% of salt iodization coverage of the community. It indicates weakness in the implementation of the universal salt iodization program in the district.

9. Recommendations

1) Endemic goiter is a public health problem in the district. We recommend the district health authority to accelerate the program implementation to reduce goiter prevalence below the internationally accepted level. The authority is to ensure sufficient supply of iodized salt to the community and strengthen monitoring of the salt iodization at the household and retailer level. A system of annual cyclic monitoring could be developed, so that in a five-year monitoring cycle the district is covered for ensuring availability of adequately iodized salt to the people. The monitoring system could be linked to the decision making process.

2) Median urinary levels are above the recommended level, indicating that the current iodine nutritive status of the district is satisfactory. We recommend continuing the program efforts. Distributions of iodized salt through public distribution system can sustain this achievement. Ensure focused attention for the regular availability and proper storage of iodized salt in the public distribution system.

3) Proportion of households consuming adequately iodized salt is below the recommended level, indicating weakness in the implementation of universal salt iodization program. We need to improve the coverage of iodized salt. We recommend measures to improve the awareness of the community about the impact of iodine deficiency disorders and the usefulness of consuming iodized salt in its prevention. In the absence of legislation banning the sale of non-iodized salt, it is imperative to have a vigilant regulatory mechanism to ensure that the labeling is appropriate and the iodized salt logo is appropriately used.

4) Ensure periodic survey, at least once in every five years, to estimate the prevalence of iodine deficiency disorders in the district for evaluating the impact of the ongoing universal salt iodization program.

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Annexure

Assessment of Iodine Deficiency Disorders In North 24 Parganas District, West Bengal, India

Cluster: **Name of the school:** **Date:**

ID No.	Age (yrs)	Sex (M/F)	Residence (urban/rural)	Religion	Goiter grade on clinical examination			Iodine level in salt (ppm)	Urinary iodine		Remarks
					Grade 0	Grade I	Grade II		Collected (Y/N)	Level (mcg/dL)	

Countersigned by: **Signature of the investigator**