PREFACE

The thyroid is unique among the endocrine glands for its dependence on an essential micronutrient, iodine, for normal hormone production. The thyroid gland is responsible for producing thyroid hormone, a regulator of growth, development and basal metabolic rate. This gland under hypothalamic-pituitary control concentrates iodide and synthesizes thyroid hormones. Inadequate iodine intake or the interference of thyroid gland functions under the influence of environmental goitrogens lead to reduce the synthesis of thyroid hormones. The lower level of thyroxine (T₄) in the blood stimulates the hypothalamo-pituitary axis to secrete excess thyrotropin (TSH) from the pituitary. As a result hypertrophy and hyperplasia with an enlargement of thyroid gland takes place resulting in goitre (Gaitan, 1990; Wilber and Yamada, 1990).

Endemic goitre and associated iodine deficiency disorders (IDD) constitute a major public health problem. The World Health Organization (WHO) identified nutrition as the major modifiable factor for the occurrence of chronic diseases that increased dramatically over the last decades (WHO, 2003). IDD refers to a broad spectrum of conditions that affects the foetus, neonate, child and adult (Hetzel, 1990). The consequences of iodine deficiency are goitre (enlargement of thyroid gland than normal), still birth and miscarriages, mental defect, deaf mutism, weakness and paralysis of muscles. Iodine deficiency also affects the socio-economic development of a community (Levin, 1987). The multi-factorial nature and complex interactions of host factors (age, sex) with region specific environmental condition in the pathogenesis of endemic goitre constitute a major challenge in understanding and control of the problem of goitrogenic substances in endemic areas (Gaitan and Dunn, 1992). Human goiter remains a significant problem in certain parts of the world. However, three observations indicate the existence of other factors in the etiopathogenesis of IDD. First, iodine deficiency does not always results in endemic goitre. Second, there are epidemiological and experimental evidences that concomitant exposure to other naturally occurring anti-thyroid agent’s viz. cyanogenic glucosides, glucosinolates, thiocyanate and flavonoids (polyphenols) in plant foods; poly hydroxyl phenols and phenol derivatives obtained from coal that are present in drinking water; pyridines occur in aqueous effluents from coal conversion processes as well as in cigarette smoke; phathalate esters and its metabolites, poly chlorinated and poly brominated biphenyl with two or more substituent chlorine and bromine atoms having wide variety of industrial applications; poly cyclic aromatic hydrocarbons found in food and domestic water supplies; even inorganic like excess
iodine, lithium - a pharmacological agent having wide spread use in the treatment of acute mania, bipolar depression etc. are known to interfere with thyroid gland morphology and functions, posing the danger of thyroid disease. Third, iodine supplementation does not always result in complete eradication and prevention of goitre and IDD. Besides a large number of the environmental factors, both naturally occurring and human made, having the role to interfere with thyroid gland morphology and function (Gaitan, 1986).

Pollutants that results in goitre are known as environmental goitrogens that may cause the condition by acting directly on the thyroid gland but also indirectly by altering its regulatory mechanisms and the peripheral metabolism and excretion of thyroid hormones. Anti-thyroid compounds may enter through water, air or food exposure pathways, becoming an important environmental goitrogenic factor in man and other animals. Naturally occurring and anthropogenic agents may act as goitrogens, as well as some drugs, which in the presence of dietary iodine deficiency may exaggerate the goitre and associated disorders (Gaitan, 1988).

Involvements of certain cyanogenic plants present in foodstuff on the alteration of thyroid gland morphology and functions have been studied in certain goitre endemic regions (Delange et al., 1982). But the content of the goitrogenic / anti-thyroid substances even in the same foodstuff show extreme differences due to genetic and ecological factors (Delange et al., 1982). Besides these, the actual concentration of goitrogens in foodstuff do not always represent their true goitrogenic / anti-thyroid potential and even in their absence do not negate the possible anti thyroid effect because the inactive precursors are converted to active goitrogens in the animal body after ingestion (Van Etten & Wolf, 1973; Montgomari, 1969). Recently much attention has been given to identify the specific edible portion of the plant foods responsible for their goitrogenic / antithyroid activity with an aim to neutralize their effects.

Goitrogenic as well as anti-thyroid potential of plants containing thiocyanate or thiocyanate precursors is enhanced in iodine deficient conditions and this anti-thyroid effect may have been reversed by iodine supplementation (Knudsen et al., 2002) while the effects of flavonoids that also have goitrogenic activity may not be reversed by excess iodine supplementation. Consumption of excess cyanogenic plants in relation to iodine intake is considered as an etiological factor for the persistence of iodine deficiency disorders in certain regions of India (Chandra et al., 2001).

Goitrogens present in foods are capable of altering thyroid function by interfering iodide uptake of thyroid tissue or interfering the organification of iodine or inhibiting the
activity of thyroid peroxidase that catalyses the reactions for thyroid hormone synthesis (Gaitan, 1990). Goitrogenic / anti-thyroid effects of feeding certain cyanogenic foods are studied. Most of these investigations have demonstrated a decrease in uptake of labelled iodine by thyroid (Langer and Greer, 1977; Bourdoux et al., 1978) and found to decrease the level in circulating thyroxin associated with enlargement of thyroid gland (Langer and Greer, 1977).

Certain plant foods contain both thyroid disrupting factors like flavonoids and thiocyanate or its precursors. The effect of feeding goitrogens in natural form present in different plant foods on thyroid morphological and functional status has not been investigated adequately. The available literature shows that in earlier experiments powerful antithyroid drugs are used to study the thyroid physiology. Studies on the influence of feeding Indian plant foods containing goitrogens of cyanogenic and polyphenolic origin on thyroid physiology has not been evaluated adequately.

Therefore, in this investigation certain plant foods have been selected viz. spinach (Spinacia oleracea), peanut seed coat (Anachis hypogaed), moringa leaves (Moringa oleifera) and sugarcane juice (Saccharum sp.) that contain both the cyanogenic constituents (cyanogenic glucosides, glucosinolates, thiocyanate) and polyphenols. All the goitrogenic constituents of those plant foods have been measured and the goitrogenic/ anti-thyroid potentiality have been studied respectively after feeding those plant foods as evidenced by thyroid gland morphology, histology, thyroid functional status (T₃, T₄ and TSH), thyroid peroxidase (TPO) activity, 5'-deiodinase- I activity(D1), and (Na⁺-K⁺)-ATPase activity. In addition urinary excretion of iodine and thiocyanate has also been measured to evaluate the iodine nutritional status and goitrogen exposure.

The available literature shows that the goitrogenic / antithyroid potentiality of those selected plant foods consumed by the people of this region have not been evaluated. Therefore, in the present investigation attempt has been made to gather scientific knowledge on those aspects, which may be utilized for the management of thyroid disorders that is prevalent despite adequate iodine prophylaxis is many regions of the country.