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 and calcitonin, a regulator of calcium homeostasis. 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 ($T_4$) in the blood stimulates the pituitary-hypothalamic axis to secrete excess thyrotropin (TSH) form 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. IDD refers to a broad spectrum of conditions that affect the foetus, neonate, child and adult (Hetzel 1987). The major consequences 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 role of iodine deficiency as an environmental determinant in the development of IDD is well established (Stanbury and Hetzel 1980). The multifactorial 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). However, three observations indicate the existence of other factors in the etiopathogenesis of IDD. First, iodine deficiency does not always result in endemic goitre. Second, there are epidemiological and experimental evidences that concomitant exposure to other naturally occurring anti-thyroid agents viz. cyanogenic glucosides and flavonoids present in staple 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 agents in the environment, both naturally occurring and man made, have the role to interfere with thyroid gland morphology and function (Gaitan 1986a).

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 (Bourdoux 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 1969; Montgomery 1969). Moreover, goitrogenic / anti-thyroid potential also depend on the processing of foods, in the areas where these plant foods are used commonly (Oke 1982). Recently much attention has been given to identify the specific edible portion of the plant food responsible for their goitrogenic / anti-thyroid activity with an aim to neutralize their effects.

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-thyroidal 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 thyroxine associated with enlargement of thyroid gland (Langer and Greer 1977).

In India, consumption of cyanogenic plants viz. cabbage, cauliflower, radish, mustard seeds etc. is a common practice and the thyroid hypofunction as evidenced by clinical and subclinical manifestations persists in spite of iodised salt intake (Chandra and Ray 2002; Marwaha et al. 2003; Chandra et al. 2003). The effect of feeding goitrogens in different conditions (viz fresh, cooked or boiled) present in different cyanogenic plant foods of Indian origin, on thyroid morphological and functional status and the goitrogenic / anti-thyroid potentiality of those plant foods consumed by the people of the country have not been evaluated adequately. The available literature shows that in most of the studies powerful anti-thyroid drugs are used to investigate the alteration in thyroid physiology (Yamazaki et al. 1960; Fragu et al. 1979). A number of Indian cyanogenic plants have potent thyroid peroxidase (TPO) inhibiting activity in \textit{in vitro} experiments (Chandra et al. 2004a). Endemic goitre and associated IDD has also been reported in the country during post salt iodization phase and the consumption of cyanogenic foods has been considered as etiological factor (Chandra and Ray 2002; Marwaha et al. 2003; Chandra et al. 2004b).

Therefore in the present study attempt has been made to gather scientific knowledge to evaluate the morphological and functional status of thyroid gland in experimental animals under the influence of selective cyanogenic plants mentioned above under uncooked and cooked conditions in addition to their goitrogen content and \textit{in vitro} thyroid peroxidase inhibiting activity, which may be utilized for the management of thyroid disorders despite adequate iodine prophylaxis in the country.