THYROID HORMONE DEFICIENCY IN BOVINE AND ITS INTERACTION WITH OTHER TRACE ELEMENTS

Kafil Hussain, Assistant Professor (Sr.Sc.),
Division of Veterinary Clinical Medicine & J, FVSc & AH, SKUAST-J, R S Pura Jammu-181102
Email: kafil.hussain@gmail.com

Introduction

Iodine plays a very important function being a constituent of the thyroid hormones thyroxin (T4) and triiodothyronine (T3). The primary function of T3 and T4 is to stimulate the oxygen consumption of almost all metabolically active tissues, with the exception of adult brain, testes, uterus, lymph nodes, spleen and anterior pituitary. Thyroid hormones play active role in development of foetus, digestion, muscle function, immune defense and circulation. Thyroid gland nearly consist half of the total amount of iodine present in the body and is essential for the formation of thyroxin which is a secretion of this gland. A lack of this secretion lowers basal metabolism and the thyroid gland then fails to control the rate of body metabolism. The animal body tries to increase secretion of this gland by enlarging it. This enlargement is called goitre. A high incidence of stillbirth and weak newborn animals with harsh coat and hairlessness is the most important manifestation of iodine deficiency.

Thyroid hormone

Iodine is a component of the thyroid hormones. The action of T3 and T4 via oxygen consumption has secondary effects i.e. heat production for maintenance of constant body temperature. It is also involved in catabolism of endogenous protein and fat stores, increases cardiac output due to combined action of thyroid hormones and catecholamines on the heart. Thyroid hormones are necessary for hepatic conversion of carotene to vitamin A. It is also responsible for increased activity of nervous system (Follet and Potts, 1990).

Iodine is actively transported from the circulation into thyroid cells, then it diffuses into the colloid. In the gland Iodine is rapidly oxidised and bound to the 3rd position of tyrosine molecules that are bound to thyroglobulin. Transport mechanism is stimulated by thyroid stimulating hormone (TSH) and depends on Na+, K+-ATPase (Hetzel and Welby, 1997). Thyroid cells ingest colloid by endocytosis. In the cells, globules of colloid merge with lysosomes. Peptide bonds between iodinated residues and thyroglobulin broken by proteases in the lysosomes resulting into liberation of T3, T4, diiodotyrosine (T2) and monoiodotyrosine in the cytoplasm T3 and T4 pass on into the circulation. Thyroid uptake of iodide and secretion of thyroid hormones are regulated by TSH feedback system (Beckett and Arthur, 1994).

Iodine is excreted in both urine and faeces. Urinary excretion is the primary regulating mechanism. Iodide in the form of iodine moves in either direction between the blood and mammary gland. Milk iodine content of cow's milk increases in direct proportion to intake (Hillman and Curtis, 1980).

Thyroid hormone and trace minerals

Copper and thyroid hormone

Interaction of thyroid hormones with trace minerals have been reported by various workers. Thyroid uptake of iodine is reduced by arsenic, iron and cobalt. Copper deficiency enhances the effects of PIU-induced hypothyroidism. Thus, copper-deficient and hypothyroid states were considerably enhanced when the two existed concurrently giving added meaning and necessity to close surveillance of trace mineral concentration and thyroid gland status. Kralik et al. (1996) investigated the effect of copper deficiency on thyroid hormone metabolism in rats. They found that the copper-deficient rats had an increased concentration of T3 in serum whereas the concentrations of total and free T4 as well as the activity of hepatic 5' D were not different compared with copper adequate control rats. The copper is necessary for proper iodine metabolism and consequently of proper thyroid hormone synthesis (Esipenko and Marskuova 1990).

Oliver (1975) demonstrated the interrelationship between copper deficient and hypothyroid states in rats. He observed severe clinical signs in the thiouracil treated rats fed copper-deficient diet. The copper-deficient and hypothyroid states were considerably enhanced.

Zinc and thyroid hormone

Zinc increases thyroid function and is usually high in hyperthyroid and low in hypothyroid animals. Hypers usually have low zinc in the blood and this information seems to be the source of the very common nutritional advice that hypers need zinc.

Oliver et al. (1987) studied the interactive combination of altered zinc and thyroid status in rats to assess pathophysiologic effects. They observed the clinical signs of zinc deficiency or thyroid alteration were limited to effects on growth rate. Changes in organ and glandular weights and serum thyrotropin levels reflected changes in serum thyrotropin levels reflected changes in serum thyroid hormone concentrations.

Iron, cobalt and thyroid hormone

Deshpande and Nadkarni (1992) concluded from the experiment, that ferritin metabolism is influenced by thyroid hormone as well as by iron. They further observed the significant increase in liver iron in both hypo and hyperthyroid animals. However, liver ferritin synthesis rate was reduced by 36% in hypothyroid rats. It appears from the present data that ferritin metabolism is influenced by thyroid hormone as well as by iron. The long term cobalt deprivation along with analysis of thyroid hormone status indicated a slight reduction of type one thyroxine monodeiodinase activity in liver accompanied by a significant reduction of the triiodothyroxine level in serum of cattle (Stangl et al., 1999).

Deficiency

Deficiency is a geographical problem, occurs when feeds and water are low in iodine, goitrogenic substances such as brassicas (e.g. rape, kale, turnips, cabbage) present in feed aggravate a deficiency (Fitzgerald, 1983). Star grass and

clover can cause an indirect iodine deficiency through the production of cyanogens. Thiocynate and perchlorate block thyroid uptake of iodine. Thiouracil blocks organification of iodine.

Deficiency signs are more likely in newborn. Goiter thyroid hypertrophy under continued stimulation by TSH, hairlessnes in newborn pigs and calves are common deficiency signs (Sihombing et aI., 1974). Long term deficiencies may result in decreased milk yield and some signs of hypothyroidism (Hill, 1991). Myxedema puffiness of the skin due to accumulated protein complexes which promote water retention also develops (Ludke and Schone, 1988). Calves born to cows suffering from iodine deficiency will have enlarged thyroid glands. Reduced bone growth and delayed epiphyseal closure have also been reported. In cows, an iodine deficiency results in arrested foetal development at any stage of pregnancy leading to death of the foetus, abortions and stillbirths. Infertility usually occurs at early stages of a deficiency. Decline in sexual vigor, and deterioration in semen quality is reported in bulls suffering from the iodine deficiency.

Diagnosis

Diagnosis is mainly based on morphological, histological or biochemical status of iodine. If dissection of a full-term calf, either born dead or which dies soon after birth, reveals a thyroid with a mass of over 8 g, then an iodine deficiency is indicated.

Thyroxin assay can reflect the thyroid and iodine status of domestic animals whereas triiodothyronine assays reflect the cause of hypothyrodism as impair conversion of T4 to T3 in case of iodine antgonist like thiouracil type goitrogens selenium deficiency where T4 measurements are not reliable (Underwood, 1977).

Prevention and control

Recommended daily allowances for dairy cattle is 0.25 ppm to 0.5 ppm in feed (NRC, 2001). Iodine requirements for growing animals are 0.12 ppm dry matter of the diet and for pregnant and lactating cows the concentration of iodine should be 0.8 ppm dry matter of feed. Reliable published values are scarce because of analytical problem. Most analyses indicate 0.25 ppm will usually be attained in most feeds except in areas where iodine is deficient in soil and water (ARC, 1980). Diets are usually supplemented by addition of iodized salt containing Nal, KI, KIO3, calcium iodiate, pentacalcium orthoperiodate or ethylene-diamine-dihydiodide (Underwood and Suttle, 1999). Physical availability of iodine present in a form not lost by volatilization, leaching, or migration into the centre of a salt block, NaI and KI not always physically available (Miller and Ammerman, 1995). Nutritional availability depends on a form of iodized salt that can be absorbed and utilized efficiently for formation of thyroid hormone.

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