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IODINE DEFICIENCY IN LIVESTOCK: CAUSES, CLINICAL MANIFESTATIONS, AND MANAGEMENT

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Abstract

Iodine is an essential trace mineral required for thyroid hormone synthesis, which regulates growth, metabolism, reproduction, thermoregulation, and development in livestock. Iodine deficiency is a common nutritional disorder, especially in regions with iodine-poor soils or where goitrogenic feeds are used. It leads to hypothyroidism, resulting in goiter, poor growth, reduced milk production, reproductive failure, infertility, weak offspring, and increased neonatal mortality. Young animals are particularly affected due to their high developmental requirements. Diagnosis is based on clinical signs, herd history, dietary evaluation, and laboratory tests such as thyroid hormone levels and iodine concentration in biological samples. Effective management includes balanced mineral nutrition, iodized salt or mineral mixtures, proper feed formulation, and control of goitrogenic feeds. Regular monitoring and appropriate supplementation are essential to prevent both deficiency and excess. This review highlights the causes, effects, diagnosis, and management of iodine deficiency in livestock, emphasizing preventive nutrition and herd health management to improve productivity and animal welfare.

Keywords: Iodine deficiency, Livestock, Thyroid hormones, Goiter, Trace minerals, Mineral nutrition, Reproductive performance, Growth disorders, Iodine supplementation, Herd health management.

1. INTRODUCTON

Iodine is an essential trace element required for the synthesis of thyroid hormones, thyroxine (T_4) and triiodothyronine (T_3), which play a vital role in regulating normal growth, development, and metabolic processes in both humans and animals. Although the daily requirement of iodine is only a few micrograms, iodine deficiency disorders (IDD) remain a significant public health concern worldwide. Epidemiological studies indicate that approximately 29% of the global population is at risk of IDD, with goitre prevalence estimated at about 12%. In recent decades, sustained efforts by international organizations such as the ICCIDD, WHO, and UNICEF have been directed toward the elimination of iodine deficiency disorders globally. India is among the most severely affected countries. According to the Ministry of Health (Government of India), surveys conducted across 239 districts in 28 states and Union Territories revealed that 197 districts are iodine deficient. This indicates that no state in the country is entirely free from iodine deficiency. A major endemic belt, extending approximately 2400 km along the Himalayan and sub-Himalayan regions from Kashmir in the northwest to the Naga Hills in the east, is particularly affected. It is generally assumed that iodine deficiency in humans is often accompanied by similar deficiencies in livestock, as animals depend on locally grown feed and fodder derived from iodine-deficient soils. Approximately 160 million livestock animals, including cattle, buffalo, goats, and sheep, are estimated to be at risk of IDD in the Himalayan and sub-Himalayan regions of India. Recent studies have also reported a widespread occurrence of subclinical iodine deficiency among cattle and buffalo populations in Punjab (Randhawa, 1999; Singh, 2002).

2. IMPORTANCE OF IODINE IN LIVESTOCK NUTRITION

Iodine is an essential trace mineral required in minute quantities for the normal growth, development, reproduction, and productivity of livestock. Despite its low dietary requirement, iodine plays a critical role in maintaining physiological homeostasis through its involvement in the synthesis of thyroid hormones. Since animals cannot synthesize iodine, it must be obtained from dietary sources such as forages, grains, mineral supplements, and drinking water. The iodine content of these sources largely depends on soil iodine concentration, environmental conditions, and agricultural practices. Consequently, livestock raised in iodine-deficient regions are at an increased risk of developing iodine deficiency disorders unless appropriate supplementation programs are implemented. Iodine deficiency remains one of the most widespread micronutrient deficiencies affecting livestock worldwide, particularly in mountainous regions, areas with high rainfall, and locations where soils are naturally low in iodine. In addition to inadequate dietary intake, the consumption of feeds

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containing goitrogenic compounds can impair iodine utilization, further increasing the risk of deficiency. Although iodine toxicity is relatively uncommon, excessive supplementation can also adversely affect thyroid function, emphasizing the importance of maintaining an optimal dietary iodine balance.

Biological Functions of Iodine

The principal biological function of iodine is its incorporation into the thyroid hormones thyroxine (T4) and triiodothyronine (T3), which regulate numerous metabolic and developmental processes. Approximately 70–80% of the body's iodine is stored in the thyroid gland, where it serves as the substrate for hormone synthesis. These hormones influence nearly every organ system by regulating cellular metabolism, oxygen consumption, protein synthesis, and energy production.

Beyond thyroid hormone production, iodine contributes indirectly to several physiological functions, including:

- Regulation of basal metabolic rate and energy metabolism.
- Maintenance of normal body temperature through thermoregulation.
- Promotion of skeletal growth and muscular development.
- Normal maturation of the nervous system during fetal and neonatal life.
- Enhancement of reproductive efficiency in both males and females.
- Support of mammary gland function and lactation.
- Modulation of immune responses and disease resistance.
- Maintenance of skin, hair, and hoof integrity.

Adequate iodine nutrition is therefore essential for achieving optimal growth performance, reproductive success, and productive efficiency in livestock species.

Thyroid Hormone Synthesis

Thyroid hormone synthesis is a tightly regulated process that depends on an adequate supply of iodine. Dietary iodine is absorbed primarily in the small intestine as iodide and transported via the bloodstream to the thyroid gland. Within thyroid follicular cells, iodide is actively concentrated by the sodium–iodide symporter (NIS), allowing the gland to accumulate iodine even when circulating concentrations are low.

Inside the thyroid gland, iodide is oxidized by the enzyme thyroid peroxidase (TPO) in the presence of hydrogen peroxide. The activated iodine is then incorporated into specific tyrosine residues of the glycoprotein thyroglobulin, forming monoiodotyrosine (MIT) and diiodotyrosine (DIT). Coupling reactions catalyzed by TPO subsequently generate the thyroid hormones:

- Two DIT molecules combine to form thyroxine (T4).
- One MIT and one DIT combine to form triiodothyronine (T3).

Following synthesis, T3 and T4 remain stored within thyroglobulin in the thyroid follicles until stimulated for release by thyroid-stimulating hormone (TSH), which is secreted by the anterior pituitary gland under the control of hypothalamic thyrotropin-releasing hormone (TRH). Circulating T4 is converted into the biologically more active T3 in peripheral tissues by deiodinase enzymes.

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When iodine intake is insufficient, thyroid hormone synthesis declines, resulting in increased secretion of TSH. Chronic TSH stimulation causes thyroid follicular cell hypertrophy and hyperplasia, leading to enlargement of the thyroid gland (goiter). Prolonged deficiency eventually produces hypothyroidism, adversely affecting growth, metabolism, reproduction, and productivity.

Physiological Roles in Growth, Metabolism, Reproduction, and Immunity

Thyroid hormones synthesized from iodine regulate a wide range of physiological functions that are essential for animal health and production.

Growth and Development: Thyroid hormones are indispensable for normal fetal development, postnatal growth, skeletal maturation, and central nervous system development. They stimulate protein synthesis, bone mineralization, and tissue differentiation. Iodine deficiency during gestation may result in fetal death, stillbirths, congenital goiter, weak neonates, reduced birth weight, delayed ossification, and impaired neurological development. Young animals are particularly susceptible because of their high metabolic demands.

Metabolism: T3 and T4 regulate basal metabolic rate by increasing cellular oxygen consumption, mitochondrial activity, and energy production. They influence the metabolism of carbohydrates, proteins, and lipids by promoting glucose utilization, glycogen breakdown, protein turnover, and fatty acid oxidation. These hormones also enhance nutrient digestibility and feed conversion efficiency, thereby contributing to improved growth and productive performance. Deficiency leads to reduced appetite, lethargy; poor feed utilization, weight loss or poor weight gain, and decreased milk production.

Reproduction: Normal thyroid function is closely associated with reproductive performance in both sexes. Thyroid hormones influence ovarian follicular development, estrous cyclicity, ovulation, implantation, placental development, spermatogenesis, and fetal growth. Iodine deficiency has been linked to delayed puberty, irregular estrous cycles, infertility, reduced conception rates, abortions, retained placenta, stillbirths, weak offspring, increased neonatal mortality, and reduced semen quality in breeding males. Adequate iodine nutrition during pregnancy is particularly important to ensure proper fetal thyroid development and neonatal survival.

Immunity: Thyroid hormones contribute to maintaining immune competence by regulating the activity of immune cells, cytokine production, and antioxidant defense mechanisms. Adequate iodine status supports both innate and adaptive immune responses, improving resistance to infectious diseases. Conversely, iodine deficiency may impair leukocyte function, reduce antibody production, increase susceptibility to infections, delay wound healing, and compromise overall health. Improved thyroid function has also been associated with enhanced vaccine responsiveness and greater resilience to environmental stressors.

Overall, iodine is indispensable for maintaining endocrine balance and ensuring optimal growth, reproduction, immune function, and productive efficiency in livestock. Appropriate dietary supplementation and regular monitoring of iodine status are therefore essential components of livestock nutritional management, particularly in regions where natural iodine availability is limited.

3. SOURCES AND METABOLISM OF IODINE

The iodine status of livestock largely depends on the iodine content of feed, water, and mineral supplements, which in turn is influenced by soil characteristics, environmental conditions, and feeding practices.

Natural Dietary Sources: The iodine concentration of animal feeds varies considerably depending on geographic location, soil iodine content, climatic conditions, and agricultural management. Unlike other minerals, iodine is unevenly distributed in nature, making its availability highly variable across different ecosystems.

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Forages and Pastures: Fresh pasture grasses, legumes, and conserved forages constitute the principal source of iodine for grazing livestock. However, iodine concentrations in plants generally reflect the iodine content of the soil in which they are grown. Coastal regions often produce iodine-rich forages because of marine aerosols, whereas mountainous regions, inland areas, and regions with high rainfall commonly have iodine-deficient soils, resulting in low-iodine forage. Excessive fertilization and acidic soils may further reduce iodine uptake by plants.

Cereal Grains and Crop Residues: Cereal grains such as maize, wheat, barley, oats, sorghum, and rice generally contain relatively low concentrations of iodine. Crop residues including straw and stover are similarly poor sources. Animals maintained predominantly on cereal-based diets without mineral supplementation are therefore more susceptible to iodine deficiency.

Oilseed Meals and Protein Supplements: Protein supplements derived from soybean, sunflower, cottonseed, and rapeseed contribute varying amounts of iodine. However, certain oilseed meals, particularly rapeseed and canola products, contain glucosinolates and other goitrogenic compounds that interfere with iodine utilization and thyroid hormone synthesis.

Animal-Origin Feedstuffs: Fish meal, seaweed products, marine algae, and other seafood-derived ingredients are naturally rich sources of iodine and are frequently incorporated into commercial livestock diets. Milk and dairy by-products may also contribute dietary iodine depending on the iodine status of the producing animals.

Mineral Supplements: Commercial mineral mixtures, iodized salt, mineral blocks, and complete feed supplements are the most reliable sources of iodine in modern livestock production systems. These products commonly contain iodine in the form of potassium iodide, sodium iodide, calcium iodate, or potassium iodate, ensuring consistent iodine intake irrespective of natural feed variability.

Drinking Water: Although water usually contributes only a small proportion of daily iodine intake, groundwater in certain coastal or volcanic regions may contain appreciable iodine concentrations. Conversely, water from mountainous and inland regions generally provides negligible amounts.

Table 1: Iodine content of different feeds and fodders.

Feed/Fodder	Iodine content (µg/g DM)
Cereal (maize, wheat, baley)	0.115-0.192
Oil cakes (De-oiled GNC, mustard, mahua)	0.085-0.263
Brans, wheat, and rice (de-oiled)	0.095-0.287
Green fodders (maize, bajra, berseem,oat, subabul)	0.216-0.574
Straw (Wheat, rice)	0.293-0.309

Table 2: Iodine content of berseem fodder

Month	December	January	February	March	April
(cut)	(I)	(II)	(III)	(IV)	(V)
Iodine content	0.196±	0.159±	0.351±	0.368±	0.303±
(µg/g DM)	0.081	0.078	0.158	0.106	0.930

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4. FACTORS AFFECTING IODINE BIOAVAILABILITY

Although dietary iodine concentration is an important determinant of iodine status, several nutritional, environmental, and physiological factors influence its absorption, metabolism, and utilization.

Soil Characteristics: The iodine content of soils largely determines the iodine concentration of plants. Sandy, acidic, and heavily leached soils generally contain low iodine levels. Flooding, erosion, prolonged rainfall, and glaciation further reduce soil iodine availability, predisposing livestock in these regions to deficiency.

Goitrogens: Goitrogens are chemical substances found in certain plants that interfere with normal iodine metabolism and can precipitate IDD despite an otherwise adequate iodine intake (1-4 ppm DM). Presence of these goitrogens in feeds and fodders increase the usual dietary requirements of dairy animals by 4-5 times. Three general categories of goitrogens exist which affect animals: i) cyanogenic glycosides ii) mimosine iii) other iodine antagonists e.g., several inorganic elements.

Cruciferous plants contain variable amounts of potential goitrogens of the Thiouracil type (e.g. progoitrin), whereas most brassicas (kale, turnip, cabbage)/white clover (cyanogenic glycosides), cassava (Linamarin and lotoaustralin) rapeseed meal (glucosynolate, isothiocyanate etc.) contain cyanogenetic glycosides that are goitrogenic because of the release of hydrocyanic acid after structural damage to plant cells and conversion in to thiocyanate in the animals. Subabul, a leguminous fast growing perennial green bushy fodder used for animal feeding contains adequate to high levels of iodine (0.3 to 0.5 µg/g DM). Shastry (1989) reported that feeding of *subabul* to female adult goats adversely affects the fertility, neonatal mortality and congenital goitre in the kids born. These studies also show that subabul fodder has high content of mimocine (3-6%) which not only inhibits the utilization of iodine but also blocks the availability of iodine from other ingredients in the ration fed to animals. Presence of excess calcium in diet also reduces absorption from gastro-intestinal tract. High fluoride intakes have also been contributing factors in the incidence of goitre in some parts of India (Miller, 1975). Selenium is also a goitrogenic element.

Parasitic infection: Hypothyroidism in animals may also be aggravated by parasitic infection. Histopathological evidence has demonstrated sclerotic lesions in the thyroid glands of cows due to Onchocerca infections. The effect of intestinal worms in decreasing iron absorption in humans is perhaps analogous to iodine uptake in parasitized animals.

Environmental Stress: Cold temperatures, infectious diseases, transportation, and other physiological stressors increase metabolic activity and thyroid hormone demand, potentially increasing iodine requirements. Exposure to environmental pollutants such as perchlorates may also inhibit iodine uptake by the thyroid gland.

5. CLINICAL MANIFESTATION OF IODINE DEFICIENCY

Iodine deficiency occurs in many species of animals and manifests differently in different animals. Goitre is a cardinal sign of iodine deficiency in human beings, sheep, goats and calves, however it is not identifiable in adult dairy animals.

Cattle: Birth of calves with goitre is a sign of borderline or definite iodine deficiency although cows may appear normal and yet be afflicted. Retardation of foetal development during pregnancy is the hallmark of this deficiency. Iodine deficient calves may be born blind, hairless, weak, or dead depending on the severity of the deficiency. Neonatal weakness and stillbirths are accompanied by prolonged gestation and retention of placenta in iodine deficient herds. The calf mortality in deficient herds varies from 10-60%. Newly born surviving calves from severely iodine-deficient cows may show baldness. Iodine is also essential for growth and body metabolism in post-natal life and therefore, failure of growth, unthriftiness and general weakness is a common consequence in calves and yearlings. Long-term deficiency may result in decreased milk yield (Hill 1991). Iodine deficiency has also been identified as one of the main causes of dystrocia in farm animals. Ill-thrift and emaciation has been also reported in adult iodine deficient cows. The loss of libido with deterioration of semen quality in bulls, a failure to express estrus in the cow, and a high incidence of aborted, still born or weak calves have been suggested as manifestations of hypothyroidism in cattle (Smyth *et al.,* 1992). Deficient cows are less able to resist environmental

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stresses and may have higher incidence of other diseases like ketosis. Thyroid hormones regulate heat production. Sub-optimal hormonal production in iodine-deficient animals may cause hypothermia (sub-normal temperature) and is well pronounced during winter season.

Sheep and Goats: Iodine deficiency leads to thyroid enlargement in sheep but are clinically normal in other aspects. Newborn lambs show weakness, extensive alopecia, and palpable, if not visible enlargement of thyroid glands. Prolonged gestation is reported in ewes. Groppel *et al*. (1986) found that iodine deficiency affects normal growth in young sheep and goats and causes reproductive disorders in adults. Reduced quantity and quality of wool growth have been widely associated with goitre in sheep since normal development of wool producing secondary follicles requires thyroid activity in excess of that needed for growth (Ferguson *et al*., 1956). Goats present a similar clinical picture except that all abnormalities are more severe than in lambs. Kids are more susceptible and degree of enlargement is also more than their dams (Pachauri, 1981). Administration of the goitrogens, methyl-thiouracil, to cashmere goats reduced the proportion of active secondary hair follicles and delayed the onset of moult (Rhind and McMillen, 1996)

Table 3: Influence of iodine supply on reproduction of goats.

Description	Iodine content of 0.04	DM in feed 0.40
Success of conception (%)	79	83
Success of first insemination (%)	27	73
Abortion rate of gravid goats (%)	47	0
Length of gravidity (days)	158	152
Kids per gravid goat	1.4	1.7
Kids carried to term per gravid goat	0.9	1.7

Venkatesh and Pandav (1997)

In rams, decline in libido and deterioration in semen quality has been observed. Miller (1975) reported reduced alimentary tract motility in hypothyroid ruminants. It was reported in Australia that ewes given 20 mg of KI twice weekly during the last 3 months of pregnancy gave birth to lambs having higher birth weight (Knights *et al.*, 1979).

Pigs: In pigs, the major findings are the birth of hairless, still born or weak piglets often with myxedema of the skin of the neck. Thyroid enlargement may be present but it is never sufficiently large enough to cause visible swelling in the live pigs. Survivors are lethargic, do not grow well, have a waddling gait, and have leg weaknesses due to weakness of ligaments and joints.

Poultry: Iodine deficiency in breeding hens results in reduced egg production, decreased hatchability, prolonged hatching time and thyroid enlargement in the embryos (March *et al.,* 1984)

Identification of deficiency: In clinical deficiency some or all of the clinical signs are present in 10-30% or more individuals of a herd and diagnosis is relatively easier. But still another 10-40% usually have low production but with no

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obvious signs. The subclinical deficiency is of more importance as it goes unnoticed. Such animals show few or no clinical signs but productivity (fertility, growth rate or milk yield) is depressed in 20-50% of the group.

Thyroid hormone (T_3 & T_4) assay commonly employed as diagnostic tests in man and dogs are not diagnostic of failing thyroid function in dairy animals (Singh, 2002).

Estimation of iodine in blood is highly sensitive to gross iodine deficiency. The rate of excretion of iodine in urine and milk can provide useful diagnostic criteria in simple iodine deficiency since iodine intake is positively correlated with urinary iodine excretion. Iodine in milk is also extremely responsive to dietary iodine intake and milk iodine determination is used as a means of establishing the iodine status of an area. Milk iodine content 25 µg/L indicates dietary iodine deficiency in cows.

Thyroid morphology also helps to diagnose iodine deficiency as in frank cases thyroid glands are enlarged. Diagnosis of less severe form requires histopathology and iodine concentration of gland. McCoy *et al.,* 1997 reported that hyperplasia of thyroid gland is more reliable index of iodine deficiency than any other test.

Supplementation: Daily iodine requirement of an adult animal is 8-12 mg/day in the absence of iodine antagonists and animals thriving on goitrogenic feeds require 30-60mg of iodine per day. This could be provided through water, supplemented iodine through mineral mixture and injections of iodine. Supplementation of potassium iodide, sodium iodide or calcium iodide is recommended (Miller 1979). But these salts are subjected to loss of iodine by volatilization and leaching in hot and humid climates. Cuprous iodide, Orthoperiodates are the alternate options since these are stable and absorbed readily. Dosing or drenching is effective in controlling the incidence of endemic goitre however it is time consuming and costly. Extra feeding of iodine compounds to prevent neonatal mortality and associated goitre in lambs over and above the requirements can be done with few doses (e.g., two doses of 280 mg KI or 360 mg KIO_3 one at the beginning of the fourth month and the other at the beginning of the fifth month of pregnancy). Intramuscular injection of iodize poppy seed oil (4ml to each cows) prevents iodine deficiency successfully. Slow release intra ruminal devices have been developed. Randhawa and Randhawa (2001) observed that administration of 2ml of iodized oil containing 780mg of elemental iodine by S/C route at brisket region was sufficient to maintain level in Cattle for 3-4 months.

In Himachal Pradesh, India, supplementation of 10ml Lugol's iodine in water daily for ten days to anestrus cows resulted in 62 percent of the animals coming into estrus (Ryot *et al*. 1990). A relatively higher proportion (73%) of animals came into estrus when they received an iodine spray on the cervix in addition to Lugol's iodine supplementation. 64% of the treated cows and 57 per cent of the treated buffaloes conceived.

The most common and convenient supplement is iodized salt. The NRC (1971) recommended 0.0190 per cent iodine content in salt. The general recommendation for common salt supplement for livestock is 2 per cent of the concentrate or 0.5 per cent of the total dry matter intake. The daily iodized salt requirement for the average adult cattle and buffalo is 40-50 gm, 20-25 g for adult sheep and 4.5 g (2-2.5 g for young) for goats and pigs. Daily feeding of 100g of mineral mixture containing 500ppm of iodine also provides required amount. Alternatively, drinking water could be medicated with 0.5-1.0 ml of 5 % tincture of iodine for each adult dairy animal per day. Painting of tincture iodine (5%) on the thin skin of pocket of flank fold once in each week for 5 weeks before calving or breeding is another option in cows only as buffaloes have thick skin.

Iodine deficiency caused by thiouracil type of goitrogens or selenium deficiency requires special measures of control. Treatment of thiouracil rich feed with copper sulphate solution can eliminate these goiterogens from feed, which could not be achieved through iodine supplementation.

Iodine toxicity: There have been instances where administration of iodine to human and animal systems have proved toxic. Therefore, adequate caution should be exercised while taking up therapy Dairy cattle require a minimum level of 0.60 ppm of I in their diet and a maximum level of 50 ppm which is almost 100 times the minimum requirement (NRC, 1988). The signs of toxicity included anorexia, pityriasis, nasal and lacrymal discharge, conjunctivitis, coughing, bronchopneumonia,

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hair loss and dermatitis (Hillman and Curtis, 1980). Metabolic rate is elevated and indicated by raised body temperature and heart rate. Blood glucose and urea concentration are raised and immunosuppression has been reported (Haggard *et al.,* 1980). High iodine intakes which are tolerable by the dairy cow may increase iodine levels in milk sufficiently to increase the risk of thyrotoxicosis in human (Phillips *et al.,* 1988)

6. DIAGNOSIS

A combination of evidence from affected animals, feed analysis, thyroid hormone measurements, and iodine status assessment provides the most reliable diagnosis. Early identification is particularly important in breeding herds, where iodine deficiency can cause significant reproductive losses and neonatal mortality before obvious clinical signs become apparent.

Clinical Examination*:* Clinical examination is the initial step in diagnosing iodine deficiency and should include both individual animal assessment and herd-level evaluation. Veterinarians should obtain a detailed history regarding feeding practices, mineral supplementation, geographical location, reproductive performance, and the occurrence of congenital abnormalities.

The most characteristic clinical finding is **goiter**, which results from enlargement of the thyroid gland due to prolonged stimulation by thyroid-stimulating hormone (TSH). The enlargement may be unilateral or bilateral and varies from slight swelling to massive cervical enlargement that interferes with swallowing or breathing.

In young animals, congenital hypothyroidism may manifest as prolonged gestation, enlarged thyroid glands, delayed skeletal development, muscle weakness, poor suckling reflex, and impaired neurological function.

Because goiter may not always be present, particularly during early stages of deficiency, its absence does not exclude iodine deficiency.

Feed and Water Analysis*:* Evaluation of dietary iodine intake is an essential component of diagnosis. Feed and water samples should be analyzed to determine whether dietary iodine levels are sufficient to meet species-specific nutritional requirements. Laboratory analysis determines iodine concentration, usually expressed as milligrams (mg) or micrograms (µg) per kilogram of dry matter.

In addition to iodine concentration, dietary evaluation should identify factors that reduce iodine availability, including:

- Rapeseed and canola meal
- Mustard and Brassica forages
- White clover
- Cassava
- Sorghum
- High nitrate-containing feeds

Assessment of ration formulation also helps determine whether mineral supplementation is adequate or whether feed mixing errors have occurred.

Laboratory Investigations*:* Laboratory investigations provide objective evidence of iodine deficiency and thyroid dysfunction.

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Serum or Plasma Iodine: Measurement of circulating iodine concentration may provide information regarding recent iodine intake. However, serum iodine fluctuates with dietary intake and therefore has limited diagnostic value when used alone.

Urinary Iodine Concentration: Urinary iodine excretion reflects recent iodine intake because excess iodine is primarily eliminated through the kidneys. It is widely used for herd-level monitoring, particularly when multiple animals are sampled. Low urinary iodine concentrations generally indicate inadequate iodine intake.

Milk Iodine Concentration*:* Milk iodine concentration serves as a useful indicator of iodine status in dairy herds. Reduced milk iodine often reflects insufficient dietary iodine, whereas excessively high concentrations may indicate over-supplementation.

Histopathology*:* Microscopic examination of thyroid tissue obtained during necropsy or biopsy provides valuable diagnostic information.

Typical lesions include:

- Follicular epithelial hyperplasia
- Hypertrophy of thyroid follicular cells
- Reduced colloid content
- Increased vascularity
- Enlarged thyroid follicles
- Diffuse thyroid enlargement

These pathological changes are characteristic of chronic iodine deficiency.

Thyroid Hormone Assays: Measurement of thyroid hormones represents one of the most informative laboratory methods for confirming iodine deficiency.

Thyroxine (T4): Serum thyroxine concentration is commonly used to evaluate thyroid function.

Animals with iodine deficiency generally exhibit:

- Reduced total T4
- Reduced free T4

Because T4 is the major hormone secreted by the thyroid gland, decreased concentrations strongly suggest impaired thyroid hormone synthesis.

Triiodothyronine (T3): Serum T3 concentrations may also decrease during iodine deficiency. However, T3 levels are often maintained longer than T4 because peripheral tissues convert available T4 into T3 to preserve metabolic function. Consequently, T3 is considered less sensitive than T4 for detecting early iodine deficiency.

Thyroid-Stimulating Hormone (TSH): Reduced thyroid hormone production results in increased secretion of TSH by the anterior pituitary gland through negative feedback mechanisms.

Typical hormonal profile includes:

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- Low T4
- Low or normal T3
- Elevated TSH

Persistently elevated TSH stimulates thyroid hyperplasia and ultimately produces goiter.

Thyroglobulin*:* Serum thyroglobulin concentration may increase in animals with chronic iodine deficiency because of thyroid follicular damage and increased glandular activity. It is considered a useful biomarker in some research and herd-monitoring programs.

7. IMPACT ON LIVESTOCK PRODUCTION AND ECONOMICS

Iodine deficiency is one of the most important micronutrient disorders affecting livestock productivity worldwide. Although the clinical manifestations are often subtle during the early stages, prolonged iodine deficiency significantly impairs growth, reproduction, lactation, and overall animal performance. The resulting production losses adversely affect farm profitability through decreased productivity, increased veterinary expenses, reproductive inefficiency, and higher mortality rates. The economic impact is particularly severe in intensive dairy and breeding operations where optimal thyroid function is essential for maintaining production efficiency.

Milk Production: Adequate iodine nutrition is essential for maintaining normal thyroid hormone synthesis, which regulates energy metabolism, mammary gland development, and lactogenesis. Thyroid hormones stimulate nutrient utilization and metabolic processes necessary for efficient milk synthesis. Consequently, iodine deficiency adversely affects both the quantity and quality of milk produced.

Growth Performance: Thyroid hormones regulate protein synthesis, skeletal development, muscle growth, cellular metabolism, and nutrient utilization. Consequently, iodine deficiency has profound effects on growth performance, particularly in young and rapidly growing animals. Young calves, lambs, kids, piglets, and poultry are especially susceptible because thyroid hormones play a critical role in fetal and postnatal development. Congenital iodine deficiency often results in weak offspring with poor survival and reduced lifetime productivity.

Fertility: Reproductive performance is one of the most sensitive indicators of iodine deficiency in livestock. Thyroid hormones influence nearly every aspect of reproduction, including puberty, estrous cyclicity, ovulation, implantation, placental development, fetal growth, and neonatal viability.

Mortality: Severe iodine deficiency significantly increases mortality, particularly among fetuses and neonatal animals. Neonates born with congenital goiter often experience respiratory distress because enlarged thyroid glands compress the trachea and esophagus. These animals frequently die shortly after birth unless immediate supportive care is provided. In adult animals, mortality is generally uncommon but may occur indirectly due to chronic debilitation, secondary infections, reproductive complications, or severe metabolic disturbances associated with prolonged hypothyroidism.

Economic Losses: The economic consequences of iodine deficiency extend beyond individual animal health and affect the overall profitability and sustainability of livestock enterprises. Losses result from decreased production efficiency, increased veterinary interventions, reproductive failures, and premature culling.

8. MANAGEMENT AND PREVENTION

Effective management and prevention of iodine deficiency in livestock rely primarily on ensuring an adequate and balanced dietary iodine supply. Since iodine deficiency is largely a nutritional disorder, preventive strategies are considerably more economical and effective than treating clinically affected animals. Comprehensive prevention programs should include

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appropriate iodine supplementation, balanced feed formulation, regular monitoring of iodine status, control of dietary goitrogens, and periodic evaluation of herd health. Such integrated management practices not only prevent thyroid dysfunction but also improve reproductive performance, productivity, and overall animal welfare.

Dietary Iodine Supplementation: Dietary supplementation is the most practical and effective method for preventing iodine deficiency in livestock. Supplementation should be based on species, age, physiological status, production level, and regional iodine availability. Animals rose in areas with iodine-deficient soils or those consuming feeds containing goitrogenic compounds generally require additional iodine supplementation.

Common iodine supplements include:

- Potassium iodide (KI)
- Potassium iodate (KIO_3)
- Calcium iodate [$Ca(IO_3)_2$]
- Sodium iodide (NaI)
- Ethylenediamine dihydroiodide (EDDI)

Among these, **calcium iodate and potassium iodate** are widely preferred because they are more stable during feed processing and storage than iodide salts.

In areas with severe iodine deficiency, veterinarians may recommend injectable iodine preparations for pregnant animals or valuable breeding stock. However, injectable supplementation should be used judiciously under professional supervision to avoid iodine toxicity. Excessive iodine intake should also be avoided because chronic over-supplementation can impair thyroid function and reduce animal performance.

Mineral Mixtures and Iodized Salt: The routine use of mineral mixtures fortified with iodine is the most reliable method of ensuring adequate iodine intake under field conditions.

Iodized salt remains one of the simplest and most cost-effective methods of iodine supplementation.

Management of Goitrogenic Feeds

Certain plants and feed ingredients contain compounds that interfere with iodine metabolism and thyroid hormone synthesis. Proper management of these feeds is essential for preventing iodine deficiency. These feeds contain glucosinolates, thiocyanates, cyanogenic glycosides, or related compounds that inhibit iodine uptake by the thyroid gland or interfere with thyroid hormone synthesis.

Herd Health Monitoring: Routine herd monitoring enables early detection of iodine deficiency before significant production losses occur.

9. CONCLUSION

Iodine is an essential trace mineral required for thyroid hormone synthesis and plays a vital role in regulating growth, metabolism, reproduction, and immune function in livestock. Iodine deficiency is a significant and preventable nutritional disorder, most commonly occurring in regions with iodine-deficient soils and water, leading to low iodine content in feeds and fodders. The condition is further aggravated by the inclusion of goitrogenic feeds, which interfere with iodine uptake and thyroid hormone synthesis.

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Iodine deficiency may occur in both clinical and subclinical forms, with the latter being more widespread and often overlooked. It results in reduced milk production, poor growth, reproductive inefficiency, weak offspring, increased neonatal mortality, and economic losses, including reduced wool and hide quality in certain species. Different livestock species exhibit varying degrees of susceptibility and clinical expression of the disorder.

Accurate diagnosis requires a combination of clinical assessment, dietary evaluation, and laboratory testing of thyroid hormones and iodine status, as clinical signs alone are often nonspecific. Effective prevention and control depend on regular iodine supplementation through iodized salt, mineral mixtures, and balanced ration formulation, along with proper management of goitrogenic feeds and continuous herd health monitoring.

In conclusion, iodine deficiency remains an economically important but entirely preventable disorder in livestock production systems. Sustained improvement in productivity and animal health can be achieved through routine dietary supplementation, improved feed management, and regular monitoring of iodine status across herds.

10. CONFLICT OF INTREST

There is no conflict of interest regarding the publication of this manuscript. The research and preparation of this review were conducted without any commercial, financial, or personal relationships that could be interpreted as influencing the content or interpretation of the work.

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