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A comprehensive review of iodine deficiency in goats

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Abstract
Iodine deficiency indeed poses a significant concern for both humans and livestock worldwide, and goats are particularly susceptible due to their browsing habits and potential lack of access to iodine-rich sources. Additionally, certain plants (Cabbage, Kale, Broccoli, Cauliflower and Turnips) consumed by goats may contain goitrogens, compounds that interfere with iodine utilization, exacerbating the problem. Clinical iodine deficiency in goats typically manifests as goitre, characterized by an enlargement of the thyroid gland. However, subclinical deficiency, where clinical signs are not readily apparent, can be more challenging to diagnose and have adverse effects on goat health and productivity. Clinical signs of iodine deficiency, such as goitre formation, are often more prevalent in kids (young goats) compared to adults. This heightened vulnerability in kids can be attributed to their rapid growth and development, which increases their demand for iodine. Diagnosis relies on clinical observations of goitre and the evaluation of thyroid hormone levels, along with assessing plasma organic iodine levels. Milk and urine iodine levels serve as reliable indicators of iodine deficiency. Deficiency can be prevented by daily supplementation of iodine. Additionally, it's crucial to avoid feeding goats diets high in goitrogens, substances that can interfere with iodine utilization and exacerbate deficiency symptoms.

Keywords: Iodine deficiency, goitre, thyroid gland, goat

Introduction
Iodine, a crucial trace mineral, plays a fundamental role in the synthesis of thyroid hormones such as triiodothyronine and thyroxine. These hormones play a pivotal role in multiple physiological processes including thermoregulation, boosting cellular respiration and energy generation and exerting wide spread effects on intermediary metabolism, growth, development, reproduction, muscle function, immune response and circulation (Bhardwaj *et al.*, 2018) [6]. Iodine availability is reduced by substances such as methylthiouracil, nitrates, perchlorates, soybean meal, and thiocyanates. Additionally, certain minerals like rubidium, arsenic, fluorine, calcium, and potassium interfere with iodine absorption. Iodine tends to be more readily utilized by the body during the winter months and lactation periods. The chemical form of iodine in feed also affects its absorption and iodates are more easily absorbed than iodides (Rankins and Pugh, 2012) [20]. Iodine deficiency is a spectrum of disorders that can occur from the foetal stage to puberty and is a widespread cause of diseases from goitre to neurological and metabolic disorders (Davoodi *et al.*, 2022) [10]. Goitre is an Iodine deficiency disorder and common in newborns and younger animals characterized by non-inflammatory and non-neoplastic enlargement of the thyroid gland (Lafta *et al.*, 2023) [15]. It has a major economic importance because the affected foetus seldom survives and the doe often perishes (Constable *et al.*, 2017; Tufani *et al.*, 2017) [9, 27].

Classification of iodine deficiency
Iodine deficiency primarily classified as primary and secondary iodine deficiency.

a. Primary iodine deficiency
Primary goitre refers to the enlargement of the thyroid gland due to dietary iodine deficiency. Deficiency of iodine in animals is primarily attributed to environmental factors, stemming from low levels of iodine in soil, water, feed, and fodder. The insufficient presence of iodine in

soil, particularly in fodder crops, serves as a primary driver of iodine deficiency among animals. This deficiency in soil can occur due to including the leaching of iodine from surface soil and inadequate replenishment from airborne sources. Additionally, drinking groundwater with iodine levels below 2 µg/L contributes to iodine deficiency in animals (Hetzel and Wellby, 1997) [13].

Sandy soils generally have low iodine levels. The presence of high clay content and elevated soil pH can hinder the uptake of iodine by plants growing in such soils. The iodine content of plants varies depending on factors such as species, strains, climatic conditions, seasonal variations, and the use of chemical fertilizers. Cereals, wheat bran, and oil cakes typically have low iodine levels, whereas straws and green fodders contain iodine levels that are marginally adequate for livestock requirements. The stage of maturity and timing of harvesting significantly influence the iodine content of fodder, with iodine levels decreasing as environmental temperatures drop and vice versa. Excessive use of chemical fertilizers like diammonium phosphate and potash can decrease iodine uptake from the soil. Conversely, supplementing soil with seaweeds can increase soil iodine content (Bharadwaj *et al.*, 2018) [6].

b. Secondary iodine deficiency

Secondary iodine deficiency occurs due to the ingestion of goitrogenic compounds such as brassica plants, soybean by-products, and water with high calcium and nitrate content, which interfere with thyroxinogenesis. In rare instances, excessive dietary iodine intake or genetically determined inherited enzyme defects responsible for thyroid hormone biosynthesis can also contribute to goitre formation (Radostits *et al.*, 2010) [19]. An excessive intake of calcium reduces the absorption of iodine from the gastrointestinal tract. Additionally, high fluoride consumption is considered a significant factor in the development of goitre in animals. A deficiency of cobalt, and consequently vitamin B12, has been associated with elevated thyroxine levels, leading to pronounced hypertrophy and hyperplasia of the thyroid gland (Abdel *et al*., 1999) [1].

Pathogenesis

Ruminants efficiently store iodine in their thyroid glands, maintaining thyroid hormone levels even when iodine intake is low over extended periods. However, unlike ruminants, developing foetuses lack this thyroid hormone reserve, impacting their growth and development when iodine intake is insufficient (Lafta *et al*., 2023) [15]. In circumstances of iodine deficiency, there is a consequential rise in the secretion of thyroid-stimulating hormone (TSH) as a compensatory mechanism due to the diminished levels of circulating thyroid hormones, prompting a notable enlargement of the thyroid glands known as goitre (Hassan *et al*., 2013) [12]. The increased size of the thyroid gland is a physiological response aimed at capturing more iodine from the bloodstream to support thyroid hormone synthesis.

Clinical signs

The most important clinical manifestations of goitre in young kids involve noticeable enlargement of the thyroid lobes, which are generally at least twice their normal size. These enlarged lobes feel soft to the touch and may exhibit a dark red coloration. Goitre arises from the hypertrophy and hyperplasia of the thyroid gland, primarily due to iodine deficiency in the body. This deficiency triggers an overproduction of thyroid-stimulating hormone (TSH) by the pituitary gland, as the thyroid attempts to compensate for the shortfall in thyroid hormones (Singh *et al.*, 2002) [23, 24]. However, in more severe cases, signs of hypotrichosis or alopecia may manifest, alongside pronounced enlargement of the neck. This enlargement is characterized by thickened, flaccid skin that appears swollen and edematous (Constable *et al.*, 2017) [9]. Kids which survive the initial critical period after birth may generally recover, although they might still exhibit partial persistence of goitre. The thyroid gland may exhibit pulsations synchronized with the normal arterial pulse and may extend further down a significant portion of the neck, potentially causing localized swelling. When auscultated and palpated along the jugular furrow, a murmur and thrill (referred to as the thyroid thrill) may be detected due to increased arterial blood supply to the glands (Radostits *et al*., 2010) [19]. Most cases of congenital hypothyroidism are associated with multiple late-term abortions, stillbirths or early postnatal deaths (Jones *et al.*, 1997) [14]. Of the animals born alive, some are weak and partly hairless with subcutaneous oedema of head and neck.

Diagnosis

The diagnosis of iodine deficiency relies on assessing the history of mineral mixture and iodine supplementation in the animal's diet, as well as observing clinical signs such as stillbirths, abortions, and the birth of weak or dead kids with congenital goitre. Clinical iodine deficiency is relatively straightforward to diagnose. However, subclinical deficiency is more concerning as it is challenging to identify and often overlooked. Animals with subclinical iodine deficiency may exhibit minimal or no obvious clinical symptoms, yet their production, growth rate, and fertility can be negatively impacted.

Several methods have been employed to diagnose iodine deficiency, including thyroid weight, thyroid to body weight ratio in newborns, histological evaluation of the gland and T4 concentration in the serum of the animals (Clark *et al.*, 1998) [8]. Assaying thyroid hormone levels, including triiodothyronine and thyroxine, is utilized to confirm iodine deficiency or hypothyroidism in goats. In goats, normal serum thyroxine concentrations have been reported in various studies. According to Capen (2002) [7], the range is 38.6 to 54.4 nmol/L, while Bath *et al.* (1979) [5] and Reap *et al.* (1978) [21] reported a wider range of 64 to 107 nmol/L. According to Smith and Sherman (2023) [26], serum thyroxine levels in goats typically fall within the range of 5.9 to 10.2 µg/dL, while normal levels of triiodothyronine (T3) typically range from 124 to 151 ng/dL. Furthermore, according to Davoodi *et al.* (2022) [10], normal values for thyroxine (T4) in does range from 4.96 to 6.16 µg/dL, and for triiodothyronine (T3) from 144.00 to 162.00 ng/dL. In kids, these values are reported as an average of 8.65 ± 1.86 µg/dL for T4 and 282 ± 1.01 ng/dL for T3. These findings offer additional insights into thyroid hormone levels in both does and kids, aiding in the understanding of thyroid function in goats across different age groups. In small ruminants, the normal mean values for plasma T3 level are reported to range from 0.74 to 1.12 nmol/L, while for T4 level, the range is between 56.8 and 73.35 nmol/L, as documented by Almeida *et al.* (2002) [2] and Atessahin *et al*. (2001) [4]. Additionally, Atessahin *et al.* (2001) [4] documented normal levels for free T3 (FT3) ranging from 3.67 to 5.11 pmol/L and for free T4 (FT4) ranging from 13.38 to 18.53 pmol/L. According to, a thyroxine

concentration of less than 50 nmol/L is considered a significant indicator of goitre (Andrewartha *et al*., 1980) [3].

Triiodothyronine, the active form of thyroid hormone at the cellular level, requires conversion from thyroxine through the action of the selenium-containing enzyme deiodinase. Insufficient selenium levels can hinder this conversion process, leading to elevated thyroxine levels, reduced triiodothyronine levels, and increased plasma cholesterol. The assessment of protein-bound (organic) iodine levels in the blood proves to be highly sensitive in diagnosing iodine deficiency.

A protein-bound iodine level below 8.1 µg/dL indicates a potential deficiency in iodine. Assessing the rate of iodine excretion in milk and urine serves as valuable diagnostic criteria for simple iodine deficiency, as iodine intake typically correlates positively with urinary iodine excretion. In sheep, a milk iodine level of 80 µg/L is considered normal, while levels below 8 µg/L suggest deficiency. Similarly, the lower normal limit of iodine in urine is reported to be 50 µg/L according to Bharadwaj *et al.* (2018) [6]. These measurements offer important insights into iodine status and aid in diagnosing iodine deficiency. Earlier investigations into soil iodine concentration have demonstrated a broad spectrum, ranging from less than 0.1 to 150 mg/kg across different geographic regions. Notably, the iodine content in soil surpasses that found in rocks. Atmospheric iodine, sourced from marine environments, primarily contributes to soil iodine levels. Consequently, regions near seas exhibit higher concentrations of iodine in their soil (Fuge, 2013) [11]. In a normal thyroid gland, acini are lined by low cuboidal epithelium and filled with colloid. However, in cases of goitre, this normal architecture is replaced by tall columnar epithelium, with papillary infolding and reduced colloid content. A hyperplastic goitre presents as an enlarged gland with abundant colloid, also termed colloid goitre, particularly when dietary iodine supplementation is provided to goats (Roy *et al.*, 1964) [22]. In such cases, the iodine content of the thyroid gland is diminished. Histopathologically, subclinical iodine deficiency can be identified by hyperplasia of the thyroid epithelium despite the gland maintaining a grossly normal size (Smith and Sherman, 2023) [26].

Treatment and prevention

The actual dietary iodine requirement is 0.8 mg/kg dry matter for lactating goats and 0.5 mg/kg dry matter for the rest of the flock (Lamand, 1978; NRC, 2007) [16, 18]. When goats are fed cruciferous plants, iodine requirement is approximately 2 mg/kg dry matter for lactating animals and 1.3 mg/kg dry matter for other animals (Smith and Sherman, 2023) [26].

To address or prevent iodine deficiency goitre in goats, especially in pregnant does, iodine supplementation in the form of iodized salts is recommended. The ideal iodine content of salt is typically around 0.0190%. It should be supplemented to livestock at a concentration of 2% in concentrates or at 0.5% of the total dry matter intake. For goats, the daily requirement of iodized salt is approximately 4.5 grams for adults and 2–2.5 grams for kids. Ensuring proper supplementation helps maintain adequate iodine levels, promoting thyroid health and preventing iodine deficiency-related conditions (NRC, 2005; Bharadwaj *et al*., 2018) [17, 6].

Synthetic sodium thyroxine administered orally at a dosage of 0.2 mg/day to goats weighing between 16–20 kg has been shown to effectively alleviate many clinical signs of deficiency, as reported by Singh *et al*. (2006) [25]. However, it's important to note that this supplementation should ideally not be required once the diet is corrected to provide adequate levels of essential nutrients. It serves as a temporary measure to address immediate deficiencies, with the ultimate goal being the correction of dietary imbalances to maintain optimal health without the need for supplementation.

Goat kids suffering from goitre can potentially benefit from the topical application of iodine, similar to the method used for navel dips in newborns. Additionally, in certain regions, injectable iodized poppy seed oil is available as a supplement. This injectable form of iodine provides supplementation that can last for up to two years (Wichtel *et al*., 1996) [28]. These interventions offer alternative methods of iodine supplementation for effectively managing goitre and iodine deficiency-related conditions in goat kids.

Oral daily supplementation of potassium iodide (130mg) or weekly application of 1ml of tincture of iodine to the back throughout gestation was successful in preventing goitre (Smith and Sherman, 2023) [26]. The prophylactic injection of 375 mg of iodized oil administered before servicing of goats has been shown to prevent the relapse of stillbirth and congenital goitre in goats.

Indeed, preventing goitre in goats typically involves several measures. One key approach is to avoid goitrogenic diets and forages, particularly during the gestation period. Goitrogens are substances that interfere with thyroid function and can contribute to the development of goitre. Additionally, regular supplementation of iodine in the diet of goats is essential. Ensuring adequate iodine intake helps maintain thyroid health and reduces the risk of goitre formation. By implementing these strategies, goat owners can effectively minimize the occurrence of goitre in their herds.

Conflict of Interest

Not available

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Not available

References

1. Gadir WSA, Adam SEI. Development of goitre and enterohepatonephropathy in Nubian goats fed with pearl millet (*Pennisetum typhoides*). Vet J. 1999;157(2):178-185.
2. Almeida AM, Schwalbach LM, de Wall HO, Greyling JP, Cardosa LA. Plasma insulin concentrations and thyroid hormone in fed and underfed Boer goat bucks. Israel Vet Med Assoc. 2002;57:1-4.
3. Andrewartha KA, Caple IW, Davies WD, McDonald JW. Observations on serum thyroxine concentrations in lambs and ewes to assess iodine nutrition. Aust Vet J. 1980;56(1):18-21.
4. Atessahin A, Pirincci I, Gursu F, Cikim G. Effects of selenium on thyroid hormone levels in sheep. Turk J Vet Anim Sci. 2002;26(6):1401-1404.
5. Bath GF, Wentzel D, van Tonder E. Cretinism in Angora goats. J S Afr Vet Assoc. 1979;50:237-239.
6. Bhardwaj RK. Iodine deficiency in goats. In: Goat Science; c2018. p. 75-84.
7. Capen CC. Tumors of the endocrine glands. In: Tumors in Domestic Animals. Iowa State Press; c2002. p. 650-654.
8. Clark R, Sargison N, West D, Littlejohn R. Recent information on iodine deficiency in New Zealand sheep flocks. N Z Vet J. 1998;46(6):216-222.

9. Constable PD, Hinchcliff KW, Done SH, Grünberg W. Perinatal diseases. In: Veterinary Medicine: A Textbook of the Diseases of Cattle, Horses, Sheep, Pigs, and Goats. 11th ed. St. Louis, MO: Elsevier; c2017. p. 1830-903.
10. Davoodi F, Zakian A, Rocky A, Raisi A. Incidence of iodine deficiency and congenital goitre in goats and kids of Darreh Garm region, Khorramabad, Iran. Vet Med Sci. 2022;8(1):336-342.
11. Fuge R. Soils and iodine deficiency. In: Essentials of Medical Geology. Revised edition. Dordrecht: Springer Netherlands; c2013. p. 417-432.
12. Hassan N, Randhawa C, Hussian S. Treatment of congenital bilateral goitre in a kid: a case report. Indian J Sci Technol. 2013;1(3):19-20.
13. Hetzel BS, Welby MC. Iodine. In: O'Dell BL, Sunde RA, editors. Handbook of Nutritionally Essential Mineral Elements. New York: Marcel Dekker; c1997. p. 557-582.
14. Jones TC, Hunt RD, King NW. Endocrine glands. In: Veterinary Pathology. Williams & Wilkins; c1997. p. 1236-1237.
15. Lafta MH, Jarad A, Al Saad KM. Goiter in crossbreed goat kids at Basrah Province, Iraq. Arch Razi Inst. 2023;78(2):531-538.
16. Lamand M. Oligoéléments. In: Alimentation des Ruminants. Paris: Institut National de la Recherche Agronomique; c1978. p. 143-149.
17. NRC. Mineral Tolerance of Animals. 2nd Rev. ed. Washington, DC: National Academies Press; c2005.
18. NRC. Nutrient Requirements of Small Ruminants: Sheep, Goats, Cervids, and New World Camelids. Washington, DC: National Academies Press; c2007.
19. Radostitis OM, Gay CC, Hinchcliff KW, Constable PD. Veterinary Medicine: A Textbook of the Diseases of Cattle, Sheep, Goats, Pigs, and Horses. 10th ed. New York: Saunders, Elsevier; c2010.
20. Rankins DL Jr, Pugh DG. Feeding and nutrition. In: Sheep and Goat Medicine. 2nd ed. Maryland Heights, MO: Elsevier; c2012. p. 8-49.
21. Reap M, Cass C, Hightower D. Thyroxine and triiodothyronine levels in 10 species of animals. Southwest Vet. 1978;31:31-34.
22. Roy S, Deo MG, Ramalingaswami V. Pathologic features of Himalayan endemic goiter. Am J Pathol. 1964;44:839-851.
23. Singh JL, Sharma MC, Shiv Prasad SP, Mahesh Kumar MK, Gupta GC, Patnaik AK. Prevalence of endemic goitre in goats in relation to iodine status of the soil, water and fodder. Indian Vet J. 2002;79:657-60.
24. Singh J, Sharma M, Kumar M, Rastogi S, Gupta GC, Singh SP, et al. Assessment of therapy in goitrous goats through some cardiac function tests. Small Rumin Res. 2002;44(2):119-122.
25. Singh JL, Sharma MC, Kumar M, Gupta GC, Kumar S. Immune status of goats in endemic goitre and its therapeutic management. Small Rumin Res. 2006;63(3):249-255.
26. Smith MC, Sherman DM. Goat Medicine. 3rd ed. Ames, Iowa: Wiley-Blackwell; c2023.
27. Tufani NA, Athar H, Gugjoo MB, Ahmad RA, Fazili MR. Successful therapeutic management of congenital goitre in goat kids. Rumin Sci. 2017;6(2):391-392.
28. Wichtel JJ, Thompson KG, Craigie AL, Williamson NB. Effects of selenium and iodine supplementation on the growth rate, mohair production, and thyroid status of Angora goat kids. N Z J Agric Res. 1996;39(1):111-115.

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