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Goiter in cattle and its relationship with iodine: Literature review

Bócio em bovinos e sua relação com o iodo: revisão de literatura

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A B S T R A C T

Goiter is a non-inflammatory, non-neoplastic condition characterized by an increase in the volume of the thyroid gland, accompanied by a basal reduction in serum levels of tetraiodothyronine (T4) and triiodothyronine (T3). These hormones are produced by the thyroid under the condition of the presence and availability of iodine in the bloodstream. In this sense, the objective of this study was to highlight the relationship between iodine and the occurrence of goiter in cattle. Searches were carried out in the following databases: SciELO, Google Scholar and Repository of Theses and Dissertations. The survey of the studies occurred through the use of the respective keywords: Goiter in cattle, Iodine, Goiter substances and Thyroid gland. Studies that associated the occurrence of goiter in cattle with iodine deficiency and ingestion of goiter substances were selected, prioritizing those in Portuguese and English. It was found that iodine deficiency in cattle, whether by feeding only on pasture, the ingestion of goiter compounds or the absence of iodized salt supplementation, induced the continuous release of thyroid-stimulating hormone (TSH), responsible for causing gland swelling. Therefore, it was concluded that the nutritional deficiency of iodine in cattle diets is the main cause of the occurrence of goiter in adult animals and also of congenital goiter

R E S U M O

O bócio é uma condição não inflamatória e não neoplásica caracterizada pelo aumento do volume da glândula tireoide, acompanhada da redução basal dos níveis séricos de tetraiodotironina (T4) e tri-iodotironina (T3). Estes hormônios são produzidos pela tireoide sob a condição da presença e da disponibilidade de iodo na corrente sanguínea. Neste sentido, objetivou-se evidenciar a relação do iodo com a ocorrência de bócio em bovinos. Foram realizadas pesquisas nas bases de dados: SciELO, Google Acadêmico e Repositório de Teses e Dissertações. O levantamento dos estudos ocorreu por meio da utilização das respectivas palavras-chave: Bócio em bovinos, Iodo, Substâncias bociogênicas e Glândula tireoide. Foram selecionados os trabalhos que associavam a ocorrência de bócio em bovinos à deficiência de iodo e à ingestão de substâncias bociogênicas, priorizando-se aqueles em língua portuguesa e inglesa. Constatou-se que a deficiência de iodo em bovinos, seja pela alimentação unicamente a pasto, a ingestão de compostos bociogênicos ou a ausência de suplementação com sal iodado, induziu a liberação contínua do hormônio estimulador da tireoide (TSH), responsável por provocar o inchaço da glândula. Portanto, concluiu-se que a deficiência nutricional de iodo em dietas para bovinos é a principal causa da ocorrência de bócio em animais adultos e, também, de bócio congênito.

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Introduction

The thyroid is one of the most important endocrine glands in the body. Its main function is to produce tetraiodothyronine (T4) and triiodothyronine (T3). These hormones are fundamental for the regulation of energy metabolism and body temperature in homeothermic animals. They potentiate the effect of insulin, resulting in the optimization of glucose absorption by body tissues and higher growth rates. The biosynthesis of T4 and T3 is conditioned by the presence and availability of iodine in the bloodstream. This microelement is captured by the thyroid through stimuli from the hypothalamic-pituitary-thyroid gland system. The hypothalamus is responsible for releasing thyrotropin-releasing hormone (TRH), which stimulates the pituitary gland to release thyrostimulating hormone (TSH). Thus, the thyroid is induced to capture iodine in order to produce, mainly, triiodothyronine, whose action is faster due to greater affinity with the blood fluid (CHEEKE, 1987; TEIXEIRA, 2001; BOTTINI & WILDBERGER, 2022).

Before iodine is incorporated into the tyrosine residues of thyroglobulin, giving rise to thyroid hormones, it undergoes metabolic processes, which begin in the gastrointestinal tract (TGI). About 90% of its absorption occurs in the rumen and omasum in the form of potassium iodide (KI) or sodium iodide (NaI). In the TGI, inorganic iodine (I^-) is converted to iodide and then transported by blood plasma, which is rich in carrier proteins, to the region where the thyrocytes are located. The iodide is taken up by the NIS (Sodium Iodine Symporter - Na^+/I^-) system, going to the apical region of the thyroid. The enzyme thyroperoxidase (TPO) is activated through the energy transfer carried out by hydrogen peroxide (H_2O_2), which promotes the oxidation of iodide to atomic iodine (AZEREDO & GONZÁLEZ, 2004; COLODEL, 2005; BOTTINI & WILDBERGER, 2022).

If an iodine atom binds to the tyrosine residue of thyroglobulin, which has a benzene ring structure with carbon atoms, it will give rise to monoiodothyronine (MIT). However, if one more iodine binds, it will form diiodothyronine (DIT), since each tyrosyl structure of tyrosine can receive up to a maximum of two iodine atoms. In this sense, the formation of T4 will occur through the union of two DIT molecules, while T3 will be formed from the junction of an MIT molecule with a DIT molecule (AZEREDO & GONZÁLEZ, 2004; COLODEL, 2005; NICODEMO et al., 2008).

The production, release, and action of T3 and T4 can be inhibited by goiter compounds such as progoitrins, thiocyanates, isothiocyanates, nitriles, cyanogenic glycosides, and perchlorates. These substances are present in several forage plant species such as those of the *Brassicaceae* family, *Poaceae*, *Leguminosae*, *Euphorbiaceae* and *Leucaena leucocephala*, which also offer only 0.09 mg/kg of iodine in dry matter. Goiter compounds are able to reduce the bioavailability of iodine readily usable by the thyroid by inhibiting the action of thyroperoxidase (TPO), causing a basal reduction in the concentration of T3 and T4. The

presence of goiter agents combined with the low supply of iodine by forages contributes to the occurrence of goiter in animals fed exclusively on pasture (GONZÁLEZ, 2000; AZEREDO & GONZÁLEZ, 2004; NICODEMO et al., 2008).

Goiter is characterized by an increase in the volume of the thyroid gland, without involvement of an inflammatory or neoplastic process, due to the basal reduction of T3 and T4 hormones. The nutritional status of iodine deficiency, whether by feeding exclusively on pasture, not supplementing with iodized salt or ingestion of goiteric agents, induces the release of thyroid-stimulating hormone (TSH) by the pituitary gland. TSH is responsible for stimulating the uptake of iodine from other tissues by the thyroid in an attempt to establish hormonal homeostasis (CAPEN, 1993; CUNNINGHAN & KLEIN, 2008; EILER, 2012).

As the hormonal regulation system is characterized by negative feedback, severe iodine deficiency stimulates the continuous release of TSH, as there is no significant concentration of tetraiodothyronine and triiodothyronine to stop the release of thyrotropin. Thus, the thyroid gland swells occur due to the excessive and continuous stimulation caused by TSH. Despite this, the condition can be reversed through iodized supplementation, which is usually done from the supply of potassium iodate or sodium iodate (CAPEN, 1993; GONZÁLEZ, 2000; AZEREDO & GONZÁLEZ, 2004; CUNNINGHAN & KLEIN; EILER, 2012).

Therefore, the objective of this research is to evidence the relationship between iodine and the occurrence of goiter in cattle, taking into account that most of the Brazilian herd is raised exclusively on pasture, which by itself does not meet the nutritional need for iodine in animals.

Methodology

For the construction of this literature review, searches were carried out in the following search databases: SciELO, Google Scholar and Repository of Theses and Dissertations. The survey of studies on the subject occurred through the use of the respective keywords: Goiter in cattle, Iodine, Goiter substances and Thyroid gland. Studies that associated the occurrence of goiter in cattle with iodine deficiency and the ingestion of goitogenic substances were selected, prioritizing research in Portuguese and English. The timeliness of the studies was not a selection criterion for their inclusion, since there are no current studies in the literature on the topic addressed in this review.

Theoretical framework

The thyroid gland

The thyroid is the largest endocrine gland in the body and the first to emerge during embryonic development. It is present in all vertebrates, and among the glands of internal secretion, it is the most important, because according to Nelson (2010), the hormones produced by the thyroid affect many metabolic processes, influencing the concentration and activity of several enzymes, the metabolism of substrates, vitamins and minerals, the ratio between secretion and degradation of several other hormones and the response of target tissues to them (TEIXEIRA, 2008; OLIVEIRA, 2011)

It consists of two lobes located below the larynx, on each side of the trachea. Each wolf is 5 cm long and 1.5 cm wide in an adult animal. Its size varies according to the animal species, diet and iodine content (THOINET, 1996).

The lobes, when they are in normal conditions, cannot be palpated, because they are located in a ventrolateral position to the trachea and dorsal position to the medial borders of the sternothyroid and sternohyoid muscles. However, in a condition of thyroid hypofunction, characterized by decreased production of T3 triiodothyronine and T4 tetraiodothyronine, the lobes have their volume increased, becoming palpable (FERGUSON & FREEDMAN, 2006).

The main function of the thyroid gland is to produce T3 triiodothyronine and T4 tetraiodothyronine. For the biosynthesis of these hormones, four elements are required: iodine, thyroglobulin (TG), TPO thyroperoxidase, and a hydrogen peroxide generator system (CHASTAIN, 1993). Elemental iodine (I_2) is transported to the gastrointestinal tract where it is oxidized to iodide (I^-) by thyroperoxidase, in the presence of hydrogen peroxide (H_2O_2), which acts as an oxygen donor (Oliveira, 2011). It is then picked up by the thyroid gland from the NIS protein (Sodium Iodide Symporter) (CONTRERAS, 2020).

Iodide is transported from the extracellular medium into the glandular cells and thyroid follicles. Thyroid follicles are the basic functional unit of the thyroid and secrete a substance called colloid, which is rich in thyroglobulin, iodinated amino acids, or iodotyrosine, such as monoiodotyrosine (MIT) and diiodotyrosine (DIT) (BASTOS, 2014).

Subsequently, iodide is incorporated into the tyrosine residue of thyroglobulin and, consequently, the binding between it and the tyrosine molecule occurs, giving rise to the thyroid hormones tetraiodothyronine T4 and triiodothyronine T3 (Trepainer, 2007). The T4 hormone is formed by the union of two molecules of di-iodotyrosine (DIT) and the hormone T3 by the junction of a molecule of di-iodotyrosine (DIT) with a molecule of monoiodotyrosine (MIT) (ROESLER, 2012).

Thyroid hormones participate in the most important biochemical processes in the body. Some of its actions include the regulation of basal metabolism, the maintenance of body temperature and homeostasis, the potentiation of insulin action and the increase of glucose

absorption, normal growth and development of the skeletal and neurological system, bone synthesis and resorption, in addition to being fundamental for fetal development (GUYTON & HALL, 1999; YEN, 2001).

Iodine

Iodine is quantitatively classified as a microelement, however, based on the biological function it performs, it is considered an essential element. It is widely distributed throughout nature, so that 0,7 μg/m^3 is present in the air, 300 μg/Kg on the soil, 5 μg/L fresh water and in lower concentration in the animal body, with only 0.4 mg/kg (BERNADÁ & GONZALEZ, 2004).

The oceans concentrate most of the iodine in the environment. (WARKENTIN & GONZALEZ, 2003; BERNADÁ & GONZALEZ, 2004). However, these food sources are not consumed by ruminants. In addition, tropical forages offer only 0.09 to 0.05 mg/kg^{-1} depending on soil type, forage species, climatic conditions, maturity stage, growth speed, genotype and management (NICODEMO et al., 2008). These quantities are insufficient to meet the demand of 0.50 mg/kg/day of cattle.

Most of the elemental iodine (I_2) present in the body comes from food (VECCHIATTI, 2009). This trace element is essential for the synthesis of thyroid hormones, as well as being essential for normal growth and development, particularly of the brain and central nervous system (MEZZOMO & NADAL, 2016). Its importance for animal health has been recognized since the beginning (BERNADÁ & GONZALEZ, 2004), since it is one of the few minerals whose deficiency causes known clinical signs, which can be used as a parameter for the identification of thyroid dysfunctions.

Based on the importance of iodine for animal physiology and the negative impacts that its deficiency can cause, the World Health Organization (WHO) created a term entitled “iodine deficiency diseases” with the aim of describing the deleterious effects on individuals whose intake is insufficient to meet the body's needs and warning that such effects are indicators of the reduction of the productive capacity and quality of life of all patients affected individuals (VECCHIATTI, 2009).

Iodine deficiency causes a decrease in the production of tetraiodothyronine T4 and stimulates the pituitary gland to produce and release greater amounts of thyroid-stimulating hormone (TSH). This hormone, due to the lack of iodine, excessively encourages the thyroid gland to establish hormonal balance. However, it causes an increase in the volume of the thyroid, better known as thyroid hyperplasia or goiter (KANECO, 2008; OLIVEIRA, 2011; MARTINS et al., 2018).

The effects of iodine deficiency on the reproductive performance of different animal species are reported by researchers. According to Contreras (2020), cattle with insufficient iodine intake mainly have reduced libido, lower sperm quality in males, and decreased

pregnancy rate in females. In addition, this fact is pointed out as one of the main nutritional causes of miscarriages, stillbirth, retained placentas and congenital goiter, which is characterized by the birth of calves with increased volume of the thyroid gland (CABELL, 2007; MARTINS et al., 2018).

Development

The influence of iodine on thyroid function

Iodine is essential for animals, as it acts as an indispensable component for the formation of the hormones tetraiodothyronine T4 and triiodothyronine T3, formed by the conjugation of two molecules of diiodothyronine and a molecule of monoiodothyronine with diiodothyronine, respectively. Its deficiency is pointed out by several studies as the main inducer of goiter. This pathology is easy to identify, since the clinical and external signs expressed by cattle are easily interpreted and perceived (OLIVEIRA, 2011; ESTIEBE ET AL., 2020), due to the evident enlargement of the thyroid gland.

Thyroid activity is mediated through communication between the hypothalamic-pituitary-thyroid axis. This communication starts from the low blood concentration of T3 and T4. In this condition, the hypothalamus synthesizes thyrotropin-releasing hormone (TRH), which is responsible for signaling to the pituitary gland about the need to stimulate the thyroid via activity performed by thyroid-stimulating hormone (TSH). However, the formation of T3 and T4 also depends on the presence of two molecules: Tyrosine, which is part of thyroglobulin, and the availability of iodine. This mineral has a dietary origin and, when it reaches the gastrointestinal tract, it is converted into iodide, which is transported through plasma proteins to the thyroid. Its entry into this gland is made possible by a specific transporter, sodium-iodide co-transporter (Na^+/I simport - NIS), ATP dependent (CUNNINGHAN & KLEIN, 2008; KANECO, 2008; OLIVEIRA, 2011; GUYTON & HALL, 2017; ESTIEBE et al., 2020).

In the thyroid, iodide is incorporated into tyrosine and then converted to thyroglobulin by the action of the enzyme thyroperoxidase (TPO), being stored in the thyroid follicular colloid. Once formed, the hormones will only be released as needed by the body. But in general, for this to occur, thyroglobulin will be hydrolyzed by lysosomal enzymes, thus releasing T3 and T4 into the bloodstream (OLIVEIRA, 2011; PORTO, 2016). T3 is the biologically active form of hormone. It circulates freely through the plasma and with lower energy expenditure, as it has a greater affinity with the medium. T4, on the other hand, needs to be converted to T3 in order to perform its function. This reaction is enabled and executed by the enzyme deiodinase, which is responsible for the deiodination of T4 (KANECO, 2008; OLIVEIRA, 2011; GUYTON & HALL, 2017; ESTIEBE et al., 2020).

Neuroendocrine activity is mediated and influenced by the amount of iodine available

(OLIVEIRA, 2011). According to Kimura (2008), the availability of iodine allows the thyroid to produce T3 and T4 without the need for neural stimulation, a process known as autoregulatory hormonegenesis, thus there is no detectable change in the levels of thyrostimulating hormone TSH. However, when this mineral is lacking, the thyroid loses its ability to self-regulate. In this context, the pituitary gland releases higher levels of TSH, which stimulates increased iodine transport from other tissues, with the aim of encouraging the thyroid to produce its hormones. But this alternative mechanism, over time, becomes taxing on the gland, due to continuous hyperstimulation.

The statement made by Mezzomo & Nadal (2016) reinforces the above explanation, because according to them, insufficient iodine consumption forces the thyroid to adapt to this nutritional state, so that the gland becomes dependent on the pituitary neural stimulus promoted by the action of TSH, since in this condition, this hormone is in high concentration. This increase triggers plasma uptake of inorganic iodine through the activation of the immobilization system, known as the Sodium Iodine Symporter (NIS). In this scenario, the preferential secretion of T3 occurs due to the greater ease of formation and speed of action. But as a consequence of the high synthesis and release of TSH, an increase in the volume of the thyroid lobes, popularly known as goiter, is inevitable.

According to Hetzel & Maberly (1986), iodine deficiency in cattle causes subnormal synthesis of T3 and T4, reducing the reproductive performance of these animals, which is expressed by the high incidence of spontaneous abortions, stillbirth and the birth of calves with congenital goiter. Other reproductive failures were reported, such as irregular cyclicity, low conception rates and decreased libido of the bulls (MCDOWELL, 1992; WHO, 1996).

Correction of iodine deficiency is necessary for the regression of thyroid dysfunction and the reestablishment of T3 and T4 levels. Thus, iodine deficiency in cattle is suppressed by dietary supplementation with iodized salt, in which the intake of 0.45 mg/kg is recommended for lactating cows, 0.33 mg/kg for non-lactating cows and 0.50 mg/kg for beef cattle, regardless of physiological status (NRC, 2000; NRC, 2001; NRC, 2006).

The following table shows the need for iodine for cattle, according to physiological status.

Table 1.

Iodine requirements for cattle, according to the National Research Council (NRC).

Species	Physiological state	Requirements	Reference
Beef cattle	All Classes	0.50 mg/kg	NRC (2000)
Dairy cattle	Lactation	0.50 mg/kg	NRC (2000)

Source: NRC (2000).

Congenital goiter in calves

One of the main consequences of iodine deficiency in pregnant cows is the birth of calves with goiter. During the initial third of pregnancy, the embryo depends on an adequate supply of transplacental T3 and T4, as its thyroid is developing and is not able to meet the physiological need. Thyroid hormones are essential for the development of the central nervous system of the bovine embryo, the formation and development of tissues, including thyroid cells, and the optimization of glucose utilization (AZEREDO & GONZÁLEZ, 2004; MARTINS et al., 2018; REZENDE et al., 2019; NINA et al., 2020).

In the final third of gestation, the growth rate of the fetus becomes accelerated. Thus, the calf's requirement for iodine and energy in the form of glucose is increased. The use of glucose by the fetus is optimized when the cow's T3 and T4 levels are in homeostasis. These hormones increase the uptake of glucose by cells and potentiate the effect of insulin. Thus, iodine deficiency in pregnant cows, in addition to causing congenital goiter, slows the growth of the embryo, contributing to the birth of weak, underweight calves with brittle hair, alopecia, and cold intolerance (MARTINS et al., 2018; NINA et al., 2020), since iodine directly influences energy metabolism.

The absence of iodinated supplementation or administration in an unbalanced way during the gestation period, associated with the consumption of forages containing goiter agents, are the main nutritional factors responsible for iodine deficiency in pregnant cows and, consequently, for the appearance of congenital goiter. In three farms in Mato Grosso, iodized salt was mixed with common salt in the ratio of one bag of iodized salt to two bags of common salt, which resulted in lower iodized salt consumption and iodine deficiency in pregnant cows. After the calves were born, the clinical-pathological findings from the thyroid necropsy revealed an increase in the volume of the gland and the presence of follicles devoid of colloid (MARTINS et al., 2018).

Congenital goiter in calves can be prevented and reversed, respectively, by means of adequate iodinated mineral supplementation of the cow during gestation and treatment of calves with confirmed diagnosis. When diagnosing congenital goiter in three calves whose thyroid gland volume was enlarged, Martins et al. (2018) evaluated the effects of parenteral iodine supplementation and observed regression of thyroid volume. After iodine is supplied, the thyroid's ability to produce T3 and T4 is re-established. These hormones, after being released into the bloodstream, give negative feedback to the hypothalamus and pituitary gland. Subsequently, the release of thyroid-stimulating hormone (TSH) is ceased. Thus, regression of the volume of the thyroid gland occurs.

Administration of 15 mL of parenteral iodine to heifers with congenital goiter increased the concentration of tetraiodothyronine (T4) produced by the thyroid from 0.42 nanograms

per deciliter ng/dl (pre-treatment) to 0.60 ng/dl (post-treatment) (NINA et al., 2020).

Final considerations

Nutritional deficiency of iodine in cattle diets is the main cause of the occurrence of goiter. However, the clinical signs in adult cattle are, in most cases, discrete, making it difficult to detect the pathology and correct the iodine deficit in diets. The pathology is easily identified during the gestational period of iodine-deficient cows, due to the reproductive failures presented, including, mainly, the birth of calves with congenital goiter.

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