Research Article

The Role of Endocrine Glands and Hormones in Regulating Metabolism in Ruminants: A Comprehensive Review

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Abstract | Ruminants possess a unique metabolic architecture, driven by the interplay between ruminal microbial fermentation and host endocrine regulation. This review synthesizes current knowledge on how endocrine glands and their hormones modulate carbohydrate, lipid, and protein metabolism in ruminant species, detailing the roles of pituitary, thyroid, adrenal, pancreatic, gonadal, and adipose-derived (e.g. leptin) hormonal axes. It further explores common endocrine disorders and their metabolic impacts, and highlights recent advances and applications aimed at optimizing metabolic health and production efficiency. Understanding the endocrine control of metabolism is essential not only for basic physiology but also for improving feed efficiency, animal welfare, and sustainable productivity in ruminant systems.

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Introduction

The metabolic network in ruminants is inherently more complex than in monogastric species, owing to their reliance on ruminal fermentation, which converts ingested plant polysaccharides into volatile fatty acids (VFAs) rather than glucose (Lynd *et al.*, 2002; Puniya *et al.*, 2015). Because only a limited amount of glucose is directly absorbed from the small intestine, ruminants depend heavily on hepatic gluconeogenesis, primarily from propionate, lactate, and amino acids, to maintain systemic glucose homeostasis (Ortega Cerrilla and Mendoza Martínez, 2003; Clauss and Hummel, 2017). In this context, endocrine glands and their hormones act as central regulatory controllers, coordinating metabolic fluxes across tissues in response to changing nutritional, physiological, and environmental demands (Baumgard and Rhoads, 2012). This endocrine regulation is especially critical during transitions such as pregnancy, parturition, and early lactation, when nutrient demands shift rapidly (Boisclair and Giesy, 2024; Fazio *et al.*, 2022). Disruptions in hormonal balance can lead to metabolic disorders (e.g. ketosis, fatty liver, hypocalcemia), impairing animal health and performance. This review provides a comprehensive synthesis of the role of endocrine glands and hormones in ruminant metabolism, including (i)

major endocrine organs and their secretions; (ii) hormonal regulation of macronutrient metabolism and energy balance; (iii) endocrine pathologies affecting metabolism; and (iv) recent advances and practical implications for production systems.

Endocrine regulation of metabolism in ruminants

Major endocrine glands and their secretions

Pituitary (Hypophysis)

The anterior pituitary (adenohypophysis) secretes key trophic and metabolic hormones including Growth Hormone (GH), Prolactin (PRL), Adrenocorticotropic Hormone (ACTH), Thyroid-Stimulating Hormone (TSH), Follicle-Stimulating Hormone (FSH), and Luteinizing Hormone (LH) (Melmed and Kleinberg, 2003). The posterior pituitary (neurohypophysis) releases antidiuretic hormone (ADH) and oxytocin, synthesized in the hypothalamus and transported distally (Binder *et al.*, 2009).

GH (somatotropin) is a primary regulator of growth but also exerts profound metabolic effects: it promotes lipolysis, reduces peripheral glucose uptake, and increases gluconeogenesis (Trenkle, 1981; Brockman, 1986). Prolactin, beyond its lactogenic role, may influence nutrient partitioning during lactation, favoring mammary uptake (Childs *et al.*, 2020). TSH stimulates thyroid hormone synthesis; ACTH regulates adrenal cortisol secretion; and the gonadotropins (FSH, LH) influence gonadal sex steroid output, which in turn impacts metabolism (Olds and Mahmoud, 1980; Childs *et al.*, 2020).

Thyroid gland

Thyroid hormones, primarily thyroxine (T4) and triiodothyronine (T3), control basal metabolic rate, thermogenesis, and modulate carbohydrate, lipid, and protein metabolism (Mullur *et al.*, 2014; Kalhan, 2009). In ruminants, variations in diet, energy balance, and climatic stress may influence thyroid hormone levels (Zarrin *et al.*, 2024). A minor thyroid secretion, calcitonin, participates in calcium homeostasis, though its metabolic role is limited relative to PTH (Xie *et al.*, 2020).

Adrenal glands

The adrenal cortex produces glucocorticoids (principally cortisol in many ruminants), mineralocorticoids (aldosterone), and weak androgens (Lloyd, 2023; Katsu and Baker, 2021). Cortisol is a central mediator of metabolic adaptation under stress: it stimulates gluconeogenesis, mobilizes amino acids from muscle, and induces lipolysis (Dutt *et al.*, 2023). Aldosterone regulates sodium and water balance, indirectly influencing circulatory volume and metabolic processes. The adrenal medulla secretes catecholamines (epinephrine, norepinephrine), which acutely drive glycogenolysis and lipolysis during stress (Paravati *et al.*, 2024).

Pancreas (Endocrine portion)

The endocrine pancreas (islets of Langerhans) secretes insulin, glucagon, and somatostatin. Insulin lowers plasma glucose by stimulating uptake in muscle and adipose, promoting glycogenesis and lipogenesis, and suppressing hepatic glucose output. Glucagon antagonizes insulin, promoting hepatic glycogenolysis and gluconeogenesis (Brockman, 1978; Brockman, 1986; Trenkle, 1981). Somatostatin (δ-cells) inhibits both insulin and glucagon secretion, and suppresses GH release (Brockman, 1978).

Gonads (Ovaries/Testes)

Gonadal sex steroids (estrogen, progesterone, testosterone) regulate reproductive physiology but also modulate metabolism. Estrogens may enhance insulin sensitivity, affect lipid metabolism, and influence appetite. Progesterone modulates glucose and fatty acid balance during pregnancy, while testosterone promotes muscle protein accretion and influences fat partitioning (Melmed and Kleinberg, 2003).

Adipose tissue and other hormones (e.g. Leptin)

Adipocytes produce leptin, which signals energy reserve levels to the hypothalamus, modulating feed intake and metabolic rate (Roche *et al.*, 2008). Though less studied in ruminants than in monogastrics, leptin likely plays a role in coordinating energy balance with reproductive and metabolic functions (Boisclair and Giesy, 2024).

Hormonal regulation of macronutrient metabolism

Carbohydrate metabolism

In ruminants, where glucose absorption from the gut is minimal, the hormonal regulation of gluconeogenesis becomes central. After feeding, insulin rises modestly, promoting glucose uptake and storage; yet insulin's effect is less pronounced in ruminants compared to monogastrics (Brockman, 1986; Van der Walt and Linington, 1990). Glucagon and cortisol act as counterregulatory hormones, stimulating hepatic gluconeogenesis and glycogenolysis (Trenkle, 1981;

Brockman, 1978). Thyroid hormones also enhance hepatic gluconeogenic enzyme expression and intestinal glucose transport (Kalhan, 2009).

During negative energy balance, elevated GH supports glucose supply to critical tissues (e.g. muscle, mammary gland) while restraining uptake by adipose tissue (Trenkle, 1981; Brockman, 1986).

Lipid metabolism

Insulin favors lipogenesis and suppresses lipolysis. In contrast, glucagon, cortisol, GH, and catecholamines stimulate mobilization of free fatty acids (FFAs) from adipose tissue (Brockman, 1986; Trenkle, 1981). Thyroid hormones also elevate plasma fatty acid concentrations and enhance β-oxidation rates (Mullur *et al*., 2014).

Rumen microbial biohydrogenation modifies the profile of lipids reaching the intestine, effectively converting unsaturated dietary fatty acids to more saturated forms, thereby influencing substrate availability and hormonal sensitivity (Puniya *et al*., 2015).

Protein metabolism

Insulin and GH promote amino acid uptake and protein synthesis in muscle. Cortisol induces proteolysis, supplying amino acids for gluconeogenesis (Trenkle, 1981; Brockman, 1986). Thyroid hormones at physiological levels stimulate protein synthesis; at excessive concentrations may enhance protein catabolism (Mullur *et al*., 2014).

Ruminants derive much of their amino acids via microbial protein digestion in the small intestine; thus, regulation of rumen nitrogen metabolism is integrally linked to endocrine control of whole-body protein balance (Bach *et al*., 2005).

Integration of hormonal signals & energy balance

Metabolic homeostasis in ruminants arises from cross-talk among endocrine axes. The hypothalamus integrates signals from peripheral hormones (insulin, glucagon, leptin) and nutrient sensors, regulating pituitary output accordingly (Roche *et al*., 2008). During physiological transitions (e.g. early lactation), GH and leptin act in complementary fashion to optimize nutrient partitioning without compromising health (Boisclair and Giesy, 2024).

Endocrine adaptation is crucial under heat stress, feed restriction, or disease: thyroid downregulation, altered GH–IGF axis, and modulated cortisol dynamics are commonly observed (Baumgard and Rhoads, 2012).

Endocrine disorders and metabolic dysregulation in ruminants

Ketosis and negative energy balance

Ketosis is a prevalent metabolic disorder in high-producing dairy ruminants during early lactation, resulting from severe negative energy balance (NEB). In this state, low insulin, high cortisol, GH, and glucagon levels promote excessive lipolysis and ketogenesis in the liver, leading to accumulation of ketone bodies (β-hydroxybutyrate, acetoacetate) (Van der Walt and Linington, 1990). Clinical signs include anorexia, decreased milk yield, and neurologic symptoms in severe cases. Insulin resistance during the periparturient period exacerbates progression (Brockman, 1986; Trenkle, 1981).

Hypocalcemia (Milk fever)

A sudden onset of hypocalcemia immediately postpartum (milk fever) represents a failure of endocrine and homeostatic mechanisms regulating calcium metabolism. Parathyroid hormone (PTH), 1,25-dihydroxyvitamin D_3, and tissue responsiveness fail to meet the rapid calcium demand for colostrum and milk synthesis. This dysfunction, primarily of PTH and vitamin D pathways, leads to severe hypocalcemia, muscle weakness, and recumbency (Goff, 2008; Horst *et al*., 2020).

Thyroid disorders

Hypothyroidism in small ruminants (e.g. goats) leads to reduced metabolic rate, poor growth, lethargy, and decreased milk or wool production. In ruminants, hyperthyroidism is rare. Energy deficits and low adiposity (reduced leptin) may suppress the hypothalamic–pituitary–thyroid (HPT) axis (Roche *et al*., 2008).

Adrenal disorders

Hyperadrenocorticism (Cushing's syndrome) is rare in ruminants but can manifest muscle wasting, insulin resistance, immunosuppression, and abnormal fat distribution. Hypoadrenocorticism (Addison's disease) can lead to severe weakness and electrolyte imbalance (Dutt *et al*., 2023).

Pituitary abnormalities

Pituitary adenomas affecting somatotrophs may

disrupt GH secretion, altering metabolic partitioning. GH excess (acromegaly) or deficiency can impair production traits and energy utilization. Moreover, dysregulation of the hypothalamic–pituitary–adrenal (HPA) or hypothalamic–pituitary–thyroid axes can cascade into metabolic disorders (Trenkle, 1981).

Insulin resistance and rare diabetes

Although frank diabetes mellitus is rare in ruminants, insulin resistance is relatively common around parturition and contributes to metabolic disorders like ketosis or fatty liver. Altered insulin signaling reduces glucose uptake, exacerbating hyperglycemia and lipid mobilization (Brockman, 1986).

Advances, Applications, and Future perspectives

Omics, Molecular signaling, and Endocrine biomarkers

Recent work leverages transcriptomics, proteomics, and metabolomics to decode hormone–tissue interactions in ruminants (Boisclair and Giesy, 2024). Discoveries in endocrine adaptations in high-demand states (e.g. early lactation) offer targets for biomarker discovery and precision management (Boisclair *et al*., 2024).

Nutritional-Endocrine manipulation

Feeding strategies (e.g. propionate precursors, protected fats) can modulate hormonal response—propionate infusion enhances gluconeogenic drive, influencing insulin–glucagon balance. Nutrient restriction or overfeeding can modulate GH pulsatility and thyroid function (Puniya *et al*., 2015; Van der Walt and Linington, 1990).

Hormonal therapies and growth promotants

Exogenous hormone use (e.g. recombinant bovine somatotropin) has been explored to increase milk yield and alter metabolism—with mixed acceptance due to welfare, regulatory, and public concerns. Efforts continue to identify safer, physiologically aligned endocrine modulators (Boisclair and Giesy, 2024).

Genetic selection and endocrine traits

Breeding for metabolic robustness may include selecting for favorable endocrine hormone levels (e.g. efficient GH–IGF axis, insulin sensitivity). Integration of endocrinology into genomic selection is a promising frontier.

Knowledge gaps and research needs

The role of adipokines (beyond leptin) in ruminant energy balance is underexplored.

Mechanistic insights into hypothalamic integration of peripheral endocrine signals remain limited.

More species-specific studies (sheep, goats, buffalo) are needed—much of the literature focuses on dairy cattle.

Longitudinal studies across life stages are scarce.

Conclusions and Recommendations

Endocrine glands and hormones are the master regulators orchestrating the metabolic complexity unique to ruminants. From adjusting fuel partitioning during lactation to safeguarding glucose homeostasis under nutritional stress, hormonal control is indispensable. Metabolic disorders arise when this regulatory balance is disrupted, compromising animal health and productivity. Recent advances in molecular tools and precision nutrition promise to refine our capacity to modulate endocrine–metabolic integration. For sustainable ruminant production, future research must bridge endocrinology, genomics, and management strategies, particularly across diverse species and production environments.

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Novelty Statement

The novelty of this comprehensive review lies in its integrated synthesis of recent advances in ruminant endocrinology, bridging molecular omics technologies with practical nutritional-endocrine manipulations. Furthermore, it uniquely highlights critical knowledge gaps in species-specific metabolic adaptations and adipokine signaling beyond dairy cattle, providing a consolidated framework for future research in sustainable ruminant production.

Author's Contribution

A. J. Mohamed conceptualized the study, performed the literature search, and drafted the initial manuscript. Ali Ahmed Khalaf supervised the study, crit-

ically revised the manuscript for intellectual content, and finalized the overall structure and design of the review. Both authors read and approved the final version of the manuscript.

Generative AI and AI-assisted technology statement

The authors declare that no generative AI and AI assisted technology was used in the creation of this manuscript.

Conflict of interest

The authors have declared no conflict of interest.

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