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Glucose transporter in ruminants: Mechanisms and Regulation

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Abstract
The significance of glucose as a ruminant energy source is explored in this article, with particular attention paid to the body's absorption and metabolism of the substance. It emphasizes the function of glucose transporters in the kidney, liver, and small intestine, in addition to the influence of dietary and hormonal variables on glucose metabolism. The hormonal control of glucose metabolism in ruminants is covered, with particular attention paid to the functions of insulin, Growth Hormone (GH), and glucose transporters. It also emphasizes how these hormones affect the uptake and utilization of glucose in different tissues, such as the kidneys, liver, and muscle cells. The effect of GH and GH-Releasing Factor (GHRF) on the expression of glucose transporters and glucose partitioning in ruminants is also examined in this review. It also explores the peripheral tissues' sensitivity to insulin and how it relates to animal health. The importance of dietary components like cellulose and cereal grains in supplying metabolizable energy for rumen microbial fermentation is emphasized in this review. It emphasizes the relevance of the rumen's ability to digest starch, ferment volatile fatty acids, and provide glucose for several physiological processes. It also examines the connection between rumen digestion and starch intake, highlighting the crucial role that dietary factors play in ruminant animals' glucose metabolism.

Keywords: GULT, SGLT, Hormonal regulation, Nutritional regulation

Introduction
While most animals prefer glucose as their primary energy source, ruminants primarily rely on carbohydrates (Aboragah *et al*., 2023) [2]. Because it is more effective than glucose derived from carbohydrates that ferment into volatile fatty acids (VFAs), glucose absorbed through the small intestine ultimately serves as the body's primary energy source. Propionate, the primary glucogenic precursor among the VFAs generated in the rumen, supplies more than 80% of the needed glucose (Wang *et al*., 2024) [41]. A key factor in sustaining ruminants' glucose requirements is hepatic gluconeogenesis. Specifically, the generation of milk by high-yielding dairy cows is dependent on a high rate of glucose synthesis (Rigout *et al*., 2003; Wang *et al*., 2024) [36, 41].
Because VFAs are produced in the rumen through the fermentation of non-structural carbohydrates, a minimal amount of dietary glucose seems to be accessible through direct absorption from the small intestine (Owens *et al*., 1986; 1997) [33, 32]. In any event, via the action of digestive enzymes in the small intestine, some non-structural carbohydrates, like starch, bypass rumen fermentation and transform into glucose and numerous other forms before being absorbed via the epithelium (Harmon, 2009; Noziere *et al*., 2010) [13, 28].
The apical region of the small intestine's villi contains two glucose transporters that are involved in the active glucose absorption process: (1) sodium-dependent glucose transporter 1 (SGLT1) and (2) facilitated diffusive component assisted by the temporary embedding of glucose transporter type 2 (GLUT2), (Kellett *et al*., 2008) [18]. Furthermore, the ability to absorb glucose is influenced by the surface area of the small intestine's villi (Mao *et al*., 2013) [27]. Energy and carbohydrate intakes are the main factors influencing the activity of these transport systems (Liao *et al*., 2010) [25].
The primary endocrine factor regulating glucose homeostasis is insulin. The endocrine pancreas secretes insulin during hyperglycemic episodes, which promotes glucose absorption by skeletal muscles and adipose tissue. Insulin-dependent glucose transport into peripheral tissues is another aspect of glucose metabolism in ruminants (Duehlmeier *et al*., 2007) [8].

The glucose transporters (GULTs) are a family of particular facilitative transport proteins that mediate the absorption and distribution of glucose. Only glucose transporter 4 (GLUT4) is insulin-dependent, primarily in muscles and adipocytes (Duehlmeier *et al*., 2010) [7], whereas GLUT2 is more prevalent in the small intestine (Noziere *et al*., 2010) [28]. Eleven of the fourteen transporters are involved in sugar transport (Scheepers *et al*., 2004) [38].

This review's primary objective is to examine recent studies on the hormonal and dietary factors that control ruminant glucose metabolism by influencing glucose transporters in the small intestine, muscle, and adipose tissues.

Glucose Transporter: Types and Mechanisms

Two types of membrane carrier proteins known as glucose transporters are responsible for the movement of glucose across plasma membranes (Gould and Holman, 1993) [11]. The sodium-dependent glucose transporter transfers glucose across the apical membranes of polarized intestinal and renal epithelial cells. The facilitative glucose transporter family facilitates the net efflux of glucose from the intestinal, liver, and kidney cells to plasma as well as the net intake of blood glucose for cellular metabolism in the majority of cell types.

According to Hediger *et al*. (1987) [14], SGLT-l is the first sodium-dependent glucose transporter to be cloned. The facilitative glucose transporter family includes at least six more glucose transporters (Gould and Holman, 1993) [11]. Every transporter is specialized to a particular tissue or cell type and has a unique kinetics. The net release of endogenous glucose in the liver and reabsorbed glucose in the kidney are influenced by GLUT2, the primary glucose transporter isoform expressed in hepatocytes and the kidney (Olson and Pessin, 1996) [29]. Although the liver and kidney of the nursing cow also exhibit a comparatively high abundance of GLUT5 (Zhao *et al*., 1993) [49], it is unknown what role GLUT5 plays in these tissues. The kidney of nursing cows also expresses the GLUT1 mRNA (Zhao *et al*., 1996; Aboragah *et al*., 2023) [50, 2]. According to Zhao *et al*. (1996) [50], GLUT1 may have a role in the kidney's uptake of glucose from the blood as a source of metabolizable energy for urine formation's acidity or alkalinization.

Na+/Glucose Cotransporters

The active, sodium-linked glucose transport process is mediated by sodium-dependent glucose transporters, also known as Na+/glucose cotransporters (SGLT). The Na+/glucose cotransporters are primarily found in the brush-border membranes of small intestine epithelial cells and the kidney's proximal convoluted tubule, which is consistent with this active transport role (Wright and Turk, 2004) named SGLT1 after isolating the first cDNA clone that encoded a protein with every characteristic of the small intestine brush-border transporter (Hediger *et al*., 1987) [14]. Since the cloning of SGLT1, other isoforms of SGLT genes have been discovered, including SGLT2 (Wells *et al*., 1992) [43], SGLT3 (Kong *et al*., 1993) [21], and at least 6 SGLT-related orphan cDNA (Wright, 2001) [45]. These proteins feature 14 transmembrane domains and several distinctive and conserved sodium solute symporter family characteristics. SGLT1 is presently thought to perform a limited function in glucose reabsorption in the kidney, possibly in the distal tubule. It primarily transports glucose and galactose across the intestinal brush border. Because of its low affinity for sugars and outstanding selectivity for glucose over galactose, SGLT2 is primarily responsible for glucose transport in the kidney's proximal tubule. Other Na+/glucose cotransporters' roles and transport activities are unknown (Abbas *et al*., 2020) [1].

Facilitative Glucose Transporters

The facilitative glucose transporters (GLUT) mediate the energy-independent facilitated diffusion. According to the order of publication, 12 functional facilitative glucose transporter isoforms have been cloned, described, and assigned the designations GLUT1-GLUT12 thus far (Zhao and Keating, 2007) [48]. The 12 transmembrane domains of these structurally related and conserved transporters have an N-glycosylation site on either the first or ninth extracellular loop, and the amino and carboxy termini are both in the cytoplasm. Each transporter isoform has a unique function in glucose absorption in different tissues and glucose homeostasis, as evidenced by their tissue-specific distribution, unique kinetic characteristics, and differential regulation by ambient glucose and hormones, particularly insulin (Wood and Trayhurn, 2003) [44].

The glucose transporters GLUT1 to GLUT5 have been extensively studied. GLUT1 has been ubiquitously detected in cells and tissues, including the mammary gland (Zhao *et al*., 1996) [49]. In many tissues in which it is expressed, GLUT1 is concentrated in the cells of blood-tissue barriers (Olson and Pessin, 1996) [29]. Because of the ubiquitous distribution and cellular localization, GLUT1 is considered to be the primary transporter responsible for basal glucose uptake. GLUT2 is involved in the release of hepatic glucose, in the release of absorbed and reabsorbed glucose in the small intestine and kidney, respectively, and in the regulation of insulin secretion from β-cells. GLUT3 plays a significant role as the brain neuronal glucose transporter. In skeletal muscle and adipose tissues, insulin-stimulated glucose uptake is mediated by GLUT4 (Holman and Sandoval, 2001) [15]. GLUT5 may help the small intestine's lumen absorb dietary fructose. The most recent facilitative glucose transporter isoforms that have been more thoroughly studied are GLUT6 to GLUT12 and HMIT (Zhao and Keating, 2007) [48].

Glucose Transporter Regulation

According to earlier research, GH therapy *in vitro* raised the breastfeeding cow's hepatic rates of gluconeogenesis (Knapp *et al*., 1992) [20]. Additionally, GH prevents insulin's influence on hepatic gluconeogenesis, while insulin prevents hepatic glucose production (Debras *et al*., 1989) [6]. As a result, GH ought to boost hepatic glucose production. The facilitative glucose transporters' mRNA abundance in the liver and kidney of nursing cows was unaffected by the 63-day administration of bovine GH and bovine GHRF. The mRNA abundance of GLUT2 in the liver remained unchanged by both bovine GH and GHRF, indicating a constant high maximal velocity of GLUT2 for glucose (Zhao *et al*., 1996) [50]. Therefore, at healthy glucose concentrations, the glucose flux through this transporter would be almost linear with both internal and external glucose concentrations, and transporter saturation by glucose would probably not be rate-limiting (Zhao *et al*., 1996) [50].

The effects of administering GH and GHRF were examined in a study. GLUT1 mRNA and protein were unaffected by exogenous GH, although milk yield rose by 17%. On the other hand, GHRF raised GLUT1 mRNA levels and milk supply (by 14%) (Zhao *et al*., 1993, 1996) [50]. Bovine GH and GHRF significantly reduced GLUT4 expression in peripheral adipose and muscular tissues. This aligns with the hormones' control of nutritional partitioning, which moves more glucose from these tissues to the mammary gland to boost milk synthesis (Zhao *et al*., 1996) [50]. Furthermore, in a bovine mammary gland explant culture investigation, leptin did not affect GLUT1 mRNA expression or glucose uptake (Pratt *et al*., 2007) [34].

The developmental regulation of the glucose transporters prior to and after the initiation of lactation shows that their expression and function are likely to be regulated by lactogenic hormones around the time of lactation. Intracellular GLUT-1 concentrations in mouse mammary epithelial cells have been reported to increase approximately 15-fold in response to prolactin and hydrocortisone (Zwierzchowski *et al*., 2021) [51]. It is yet unknown how these hormones control the glucose transporter's expression in the cows' mammary gland.

The most significant anabolic hormone in mammalian energy metabolism, insulin controls fat metabolism and glucose balance, among other things. Scientific studies on horses have long focused on insulin-regulated glucose elimination and the sensitivity of peripheral organs to insulin. For instance, it was hypothesized in the 1980s that Shetland ponies' breed-dependent insulin resistance was a factor in equine hyperlipaemia (Jeffcott *et al*., 1986) [16]. Treiber *et al*. (2006) [40] also found that insulin resistance is a significant risk factor for developing horse laminitis. The mechanisms that supply glucose to the skeletal muscles, especially in exercising horses, remain of great interest (de Graaf-Roelfsema *et al*., 2006) [5]. Several recent studies have focused on the role of skeletal muscle glucose transporters in improving intra- and post-exercise skeletal muscle glucose uptake (de Graaf-Roelfsema *et al*., 2006; Pratt *et al*., 2007) [5, 34]. The two carrier proteins glucose transporter 1 (GLUT1) and 4 (GLUT4) are primarily expressed by skeletal muscle cells and adipocytes. The plasma membrane contains the insulin-independent GLUT1, which is known to be in charge of the basal glucose supply (Kahn, 1996). GLUT4 is linked to membrane structures and recycles between the intracellular tubulo-vesicular pool and the plasma membrane. Through two post-insulin receptor signaling pathways, insulin and muscle contraction enhance GLUT4 translocation to and fusion with the plasma membrane (Watson and Pessin, 2006) [42]. In people and lab animals, a single exercise session enhances GLUT4 translocation to the muscle cell plasma membrane (Holman and Sandoval, 2001) [15]. Long-term exercise increases the amount of GLUT4 protein and GLUT4 mRNA in muscle cells and improves insulin-stimulated glucose absorption (Holman and Sandoval, 2001) [15].

Influence of Nutritional Factors on Glucose Metabolism

Cellulose provided the majority of the metabolizable energy for the rumen microbial fermentation during the evolution of ruminants. Particularly in feedlots and dairies, cereal grains which are mostly made of starch are an important feed source for ruminant production systems. Since ruminants lack salivary α-amylase, the rumen is the first place where starch is broken down and converted to volatile fatty acids (Kotarski *et al*., 1992) [23]. There are no restrictions on ruminal starch digestion, according to Harmon *et al*. (2004) [37], who found a linear relationship between starch intake and starch processed in the rumen. However, ruminal and systemic acidosis can occur when readily fermentable carbohydrates are fermented too quickly and excessively (Owens *et al*., 1998) [33]. Therefore, factors regulating the fermentation rate, such as grain source and processing method, must be considered for forage source and inclusion level.

Depending on the grain source and processing, between 40 and 60 percent of the dietary starch intake in cattle given high-concentrate diets goes to the small intestine (Theurer, 1986) [39]. Pancreatic α-amylase hydrolyzes the starch before it enters the small intestine. The small intestine's mucosal disaccharidases then hydrolyze the starch breakdown products. The sodium-dependent glucose transporter 1 (SGLT1; Bauer *et al*., 2001) [3] is the primary mechanism by which the mucosa absorbs free glucose. Only 55% and 53%, respectively, of the starch that enters the small intestine of calves fed high-concentrate diets dissipates in the small intestine, according to Harmon *et al*. (2004) [37], who analyzed multiple studies. In steers injected abomasally with increasing doses of glucose, corn dextrin, or corn starch, assessed the portal appearance of glucose and the elimination of small intestine carbohydrates simultaneously. A potential limit in carbohydrase activity was suggested because only the glucose infusion caused a linear proportional increase in net portal glucose absorption. This is further confirmed by the fact that when starch is fed post-ruminally at 60 g/h, 15 times as much starch as glucose passes through the ileum (Bauer *et al*., 2001) [3].

GLUC availability is one of the primary elements influencing ruminant lipogenesis, marbling, and meat quality. The production of glucose, a universal fuel for cellular, tissue, and whole-animal processes, depends on gluconeogenesis. Another difference between ruminants is that the primary precursor for gluconeogenesis is propionate, resulting from ruminal fermentation. Furthermore, it was shown by Harmon *et al*. (2004) [37] that diets high in grains enhance the absorption of L-lactate, which in turn plays a significant role in gluconeogenesis. As a result, ruminants and non-ruminants have different significance levels for the pathways involved in precursor entrance into gluconeogenesis.

According to Zhang *et al*. (2016) [47], propionate substantially increases the stimulation of cytosolic phosphoenolpyruvate carboxykinase transcription compared to cyclic adenosine monophosphate and dexamethasone, and insulin does not reverse this effect as it does in non-ruminants. The impact of glucose sensing on ruminant liver metabolism is not as well characterized. Glucose sensing in non-ruminant livers is believed to affect blood glucose management and energy metabolism (Oosterveer and Schoonjans, 2014) [30].

Koser *et al*. (2008) [22] claim that propionate positively regulates the expression of the gene encoding PEPCK, bovine phosphoenolpyruvate carboxykinase 1 (PCK1), forming a feed-forward mechanism of substrate control for hepatic gluconeogenesis connected to the end products of rumen fermentation.

It has been demonstrated that in ruminants, the expression of maltase-glucoamylase and sucrase-isomaltase is highly

responsive to dietary changes in mice, regressing when fed resistant starch and increasing in response to increased digestible starch (Goda and Honma, 2018) [9]. Since similar increases in glucose transporter (SGLT1) accompany increases in sucrase-isomaltase with both glucose and fructose feeding, with the response being more significant for fructose, it is believed that this reaction is triggered by accessible hexose (Kishi *et al*., 1999) [19].

According to Gorka *et al*. (2017) [10], ruminant mucosal carbohydrase activities typically do not react to dietary changes. Ruminants do not appear to adjust to higher carbohydrate intake, based on the apparent lack of changes in mucosal carbohydrase activities. However, as calorie intake rose, the colon and jejunum's total hydrolytic capacity increased due to increased intestinal length (Kreikemeier *et al*., 1990) [24] and increased mucosal mass (Gorka *et al*., 2017) [10]. These findings imply that ruminants' capacity to digest starch in the small intestine increases when they consume higher quantities of high-concentrate meals. However, that capacity, compared with the ability of the non-ruminant to adapt, and perhaps with a more efficient complement of enzymes, may explain the inefficiencies of ruminant small intestinal digestion.

They found that SGLT1 increased by 2.1 times in the proximal jejunum of rats receiving the abomasal infusion as opposed to the ruminal infusion. Nevertheless, a later study (Bauer *et al*., 2001) [3] failed to show that abomasal vs. ruminal infusion of partly hydrolyzed starch altered SGLT1 activity throughout the small intestine. The conversion of starch hydrolysate to glucose or the fact that mechanisms other than SGLT1 contribute to the elimination of small intestinal glucose in cattle are undoubtedly limitations of this model.

Glucose was injected into steers and compared to steers that received either ruminal or abomasal partially hydrolyzed starch to see if more glucose in the small intestine upregulates glucose transport (Rodriguez *et al*., 2004) [37]. Treatment did not affect sodium-dependent glucose absorption, whereas uptake declined distally down the gut. Results from dairy cows (Lohrenz *et al*., 2011) [26] fed diets high in starch (24%) and low in starch (12%) lend credence to this work. These researchers found that brush boundary membrane vesicles made from mid-duodenum and mid-jejunum did not vary in SGLT1 or GLUT2 mRNA or protein expression. Therefore, SGLT1 is active in cattle, with the proximal intestine showing the highest activity. However, increased consumption of starch-based diets does not affect activity.

According to a study by Ran *et al*. (2016) [35], dietary and developmental changes significantly changed the mRNA expression of monosaccharide transporters (SGLT1, SGLT3, GLUT5, and GLUT2). Goats' GIT's capacity to absorb monosaccharides changed with developmental stages, peaking at the early stage (suckling). When detecting and absorbing monosaccharides, the duodenum and jejunum are essential. Additionally, throughout all developmental stages, there was a strong correlation between the expression of sugar transporters and sweet taste receptors in the duodenum and jejunum.

Conclusion

This study emphasizes how ruminant glucose metabolism is influenced by dietary and hormonal factors such as insulin, growth hormone (GH), and glucagon-like peptide-1 (GLP-1), as well as the function of glucose transporters in the small intestine, liver, and kidney. It examined the effects of GH on glucose transporters in the liver and kidneys of nursing cows. It focused on the function of insulin in regulating glucose homeostasis and its influence on glucose uptake in peripheral tissues. The hormonal effects of prolactin and hydrocortisone on glucose transporter expression in the mammary gland were also discussed. Focusing on the importance of cellulose and cereal grains in ruminant diets and their effects on glucose metabolism, the impact of nutritional variables, namely dietary composition, on glucose metabolism in ruminants was also addressed. It emphasizes how the rumen breaks down starch, how volatile fatty acids ferment, and how crucial glucose availability is for lipogenesis and other animal processes.

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