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Understanding Starch Utilization in the Small Intestine of Cattle

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ABSTRACT : Ruminants possess the capacity to digest very large amounts of starch. However, in many cases diets approach 60% starch and even small inefficiencies present opportunities for energetic losses. Ruminal starch digestion is typically 75-80% of starch intake. On average, 35-60% of starch entering the small intestine is degraded. Of the fraction that escapes small-intestinal digestion, 35-50% is degraded in the large intestine. The low digestibility in the large intestine and the inability to reclaim microbial cells imposes a large toll on post-ruminal digestive efficiency. Therefore, digestibility in the small intestine must be optimized. The process of starch assimilation in the ruminant is complex and remains an avenue by which increases in production efficiency can be gained. A more thorough description of these processes is needed before we can accurately predict digestion occurring in the small intestine and formulate diets to optimize site of starch digestion. (**Key Words :** Bovine, Starch, Digestion, Amylase, Enzyme, Intestine)

INTRODUCTION

The ruminant digestive system provides the powerful advantage of pregastric fermentation that enables the use of structural carbohydrates and the production of microbial protein to meet the needs of the host. This complexity of ruminant digestion also offers a challenge towards optimizing nutrient supply for the host. Despite the advantages for use of structural carbohydrates, the system is not optimally designed for use of non-structural carbohydrates. The pregastric fermentation results in fermentation losses of 13-18% of gross energy (Harmon and McLeod, 2001) and is at risk of carbohydrate overload if excessive amounts are consumed (Dunlop, 1972).

Energetically, small intestinal digestion offers efficiency advantages over ruminal fermentation of non-structural carbohydrates thus digestion in the small intestine must be optimized. We have calculated that small intestinal starch digestibility must be at least 75% or the energetic inefficiencies of large intestinal digestion result in a decreased efficiency of postruminal starch digestion (Figure 1). To be able to formulate diets for optimum efficiency of digestion, we must be able to optimize digestion in the different regions of the gastrointestinal tract. We must be able to take advantage of the increased efficiency of having the starch digested and absorbed as glucose in the small intestine, thus avoiding energetic losses of ruminal fermentation. To optimize diet formulation we must understand the limitations of the animal to digest and absorb both structural and non-structural carbohydrates thereby preventing the inefficiency of large intestinal fermentation. The goal of this review is to describe processes occurring during starch assimilation in cattle. Greater emphasis will be given to the animal rather than the diet, and to digestion in the small intestine as this is where the greatest energetic advantages are to be gained. Much of the understanding of these processes comes from other species which will collectively be described as non-ruminants. Where ruminant information is lacking attempts will be made to point out research opportunities.

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INTESTINAL STARCH ASSIMILATION

Pancreatic α-amylase

The process of intestinal starch assimilation begins in the lumen of the small intestine with the secretion and action of pancreatic α-amylase. α-Amylase is synthesized in the pancreatic acinar cells. Once synthesized, α-amylase and other enzymes are packaged into zymogen granules and stored until a stimulus signals the cell to initiate an exocytosis event to release the enzymes into the duodenum. α-Amylase is an endoglucosidase, that is, it does not require free ends of amylose chains for activity, but rather is capable of hydrolyzing internal α-1-4 glucosidic bonds.

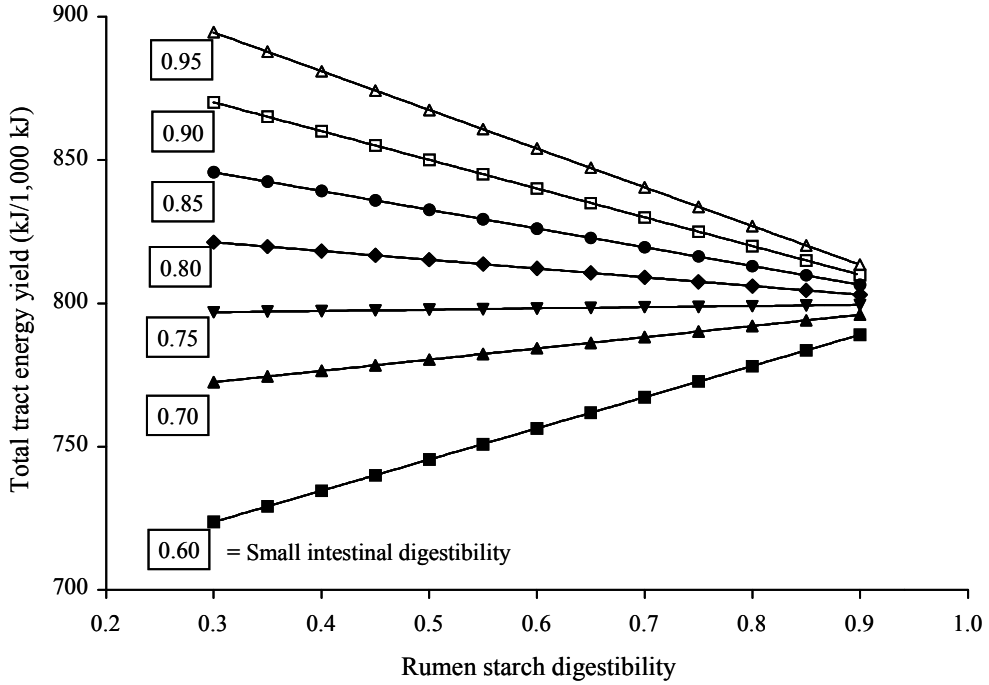

Figure 1. Calculated energy yield from total tract starch digestion that occurs when shifting digestion from the rumen to the small intestine in cattle.

Investigations of bovine α-amylase have reported characteristics similar to those in non-ruminants. Michaelis constants and activation energies are similar (Clary et al., 1969) with a pH optimum of 6.9 (Russell et al., 1981). Products of this initial phase of intestinal starch assimilation are a mixture of maltose, maltotriose and various limit dextrins that occur as a result of α-1,6 branch points (Harmon, 1993).

Because ruminants evolved as cellulose fermenters it has long been thought they have a limited ability for intestinal starch assimilation. At birth calves have low concentrations of pancreatic α-amylase (Siddons, 1968), which increases with age (Morrill et al., 1970) as does total pancreatic secretion (McCormick and Stewart, 1966). It is thought that the near continuous flow of digesta in ruminants minimizes large diurnal fluctuations in intestinal flow and pancreatic juice secretion that occur in the non-ruminant (Merchen and Church, 1988). Because of this near continuous flow nutritional regulation of pancreatic enzyme output must therefore result in changes in pancreatic enzyme synthesis to impact intestinal enzyme supply.

Early studies comparing concentrate to forage-based diets on concentrations of pancreatic α-amylase were confounded by energy intake. Clary et al. (1969) maintained steers on pasture or an all-concentrate diet for 126 days prior to slaughter. Steers consuming the all-concentrate diet had 40% higher activity of α-amylase in pancreatic tissue than those maintained on pasture. A similar tendency was reported for sheep fed either dried grass or ground corn-based diets for 4 weeks (Janes et al., 1985). Pancreatic α-amylase concentration was 34% greater in lambs consuming the ground corn-based diets.

Russell et al. (1981) were the first to evaluate the effects of diet or energy intake on postruminal digestive enzymes by feeding steers either alfalfa hay or a corn-corn silage based diet to meet maintenance energy requirements or they fed a corn-corn silage based diet at two or three-times their maintenance energy requirements. At equal energies, intake of corn and corn silage-based diets resulted in slightly lower pancreatic concentrations of α-amylase compared with the alfalfa diet, whereas increasing intake of the corn-corn silage based diet from one to two times maintenance energy increased pancreatic concentrations of α-amylase approximately two-fold. There were no further increases as energy intake increased to three times maintenance energy. To evaluate both forage and dietary energy effects on pancreatic α-amylase concentration, calves were fed 90% forage (alfalfa) or 90% grain (wheat:sorghum) diets at one or two times maintenance energy for 140 d (Kreikemeier et al., 1990). Both pancreatic concentration (55%) of α-amylase and total content of α-amylase in the pancreas (140%) increased with energy intake, regardless of diet. However, both pancreatic concentration of α-amylase and total content of α-amylase in the pancreas were decreased in calves consuming the 90% grain diet (34 and 44%, respectively) compared with those fed forage. Calves fed forage had greater (34%) concentrations of α-amylase in

the small intestinal digesta and greater total content of α-amylase (50%) in the small intestinal digesta, indicating greater pancreatic secretion of α-amylase. These decreases in pancreatic α-amylase concentrations with increased starch intake are in contrast to other reports indicating greater pancreatic α-amylase concentrations with increased starch intake (Clary et al., 1969; Janes et al., 1985). However, the results agree with those of Russell et al. (1981) which compared forage and grain at maintenance energy intakes. All studies that suggested increased pancreatic α-amylase with increased starch intake also had concurrent increases in total energy intake.

Concentration and secretion of pancreatic α-amylase can be manipulated nutritionally; however, studies to determine the exact regulatory mechanisms in ruminants are lacking. Attempts have been made to investigate the regulation of α-amylase secretion by infusing carbohydrate post-ruminally. Chittenden et al. (1984) duodenally infused wethers with glucose, maltose, or starch (200 g/d) for up to 23 d while monitoring pancreatic α-amylase secretion. Glucose infusion increased pancreatic α-amylase secretion at 16 d but not at 23 d. Maltose infusion did not change pancreatic α-amylase secretion at either 16 or 23 d, whereas starch infusion decreased pancreatic α-amylase secretion at both times. To define relations between intestinal carbohydrate supply and pancreatic enzyme secretion, Walker and Harmon (1995) infused steers fitted with pancreatic cannulas either ruminally or abomasally with a partially hydrolyzed starch solution. Infusing partially hydrolyzed starch into the abomasum decreased secretion of pancreatic α-amylase by 60% compared with a control water infusion. This decrease occurred despite increased (19%) secretion of pancreatic juice with the abomasal carbohydrate. Abomasal carbohydrate infusion also increased portal blood glucose concentrations; however, insulin concentrations were unaffected. It is clear that increased small intestinal carbohydrate can decrease pancreatic α-amylase secretion. However, it is unclear if the negative effects of carbohydrate occur because of increased carbohydrate in the lumen of the small intestine or result from increased absorbed glucose. To test this hypothesis Swanson et al. (2002b) infused glucose and partially hydrolyzed starch abomasally in steers fitted with pancreatic cannulas. As in the previous experiment (Walker and Harmon, 1995) infusion of carbohydrate increased pancreatic juice secretion, but both sources of carbohydrate resulted in linear decreases in pancreatic α-amylase secretion. This indicates that complex carbohydrate in the lumen of the gastrointestinal tract is not solely responsible for the down-regulation of α-amylase. Similar changes are elicited by glucose; whether they occur via luminal or post-absorptive effects remains unclear. However feeding high-starch diets (Kreikemeier et al., 1990) or infusing carbohydrate post-ruminally into cattle (Swanson et al., 2002b; Walker and Harmon, 1995) or increasing intake of a high-concentrate diet (Swanson et al., 2008) has consistently reduced pancreatic α-amylase concentration and/or secretion.

Taniguchi et al. (1995) infused casein and starch post-ruminally in steers and demonstrated that in the presence of casein, the supply of glucose from the portal-drained viscera was increased. This suggested that casein (or protein) may somehow improve intestinal starch disappearance. Richards et al. (2002) measured intestinal disappearance of starch in steers abomasally infused with starch and casein and showed that starch disappearance was increased with casein infusion. Further research showed that pancreatic α-amylase secretion also increased when casein was infused abomasally (Richards et al., 2003). These studies demonstrated that casein (protein) does influence the regulation of pancreatic α-amylase. To study how casein and starch interact, calves were infused abomasally with starch and/or casein and the pancreas was collected at slaughter for analysis (Swanson et al., 2002a). Infusing starch decreased pancreatic α-amylase activity (63%), protein content (71%) and tended to decrease α-amylase mRNA. These changes are consistent with our previous results in cattle (Walker and Harmon, 1995; Swanson et al., 2002b). However, infusing casein increased pancreatic α-amylase activity (28%), protein content (38%) and increased α-amylase mRNA (69%). When starch and casein were infused together the effects closely resembled those of the starch; pancreatic α-amylase activity (53%), protein content (79%) and α-amylase mRNA decreased (21%). Thus, the beneficial effects of casein on pancreatic α-amylase were not maintained when starch was infused. To determine how these changes in pancreatic enzyme content would relate to pancreatic enzyme secretion an additional experiment was performed using steers with pancreatic cannulas (Swanson et al., 2004). Infusion of starch, with or without casein increased secretion of pancreatic juice and decreased the concentration of α-amylase in pancreatic juice. However, total secretion of α-amylase was unchanged because of the increased total juice secretion. Casein infusion increased secretion of α-amylase, but only when starch was not infused. Accompanying the increased secretion of pancreatic juice were increased concentrations of insulin and cholecystokinin (CCK) but not glucagon for steers receiving the starch infusions. Casein infusion actually produced lower plasma CCK concentrations than the control. These differences show that pancreatic enzyme content and secretion can be manipulated nutritionally.

The relationship between pancreatic α-amylase and casein is difficult to explain. Non-ruminants respond to

increased dietary starch much like ruminants do with casein (Brannon, 1990). The failure to maintain the increased pancreatic α-amylase secretion when casein and starch are combined suggests that it may be difficult to increase pancreatic α-amylase secretion through formulation of practical diets. To further investigate the relationship between dietary protein and pancreatic α-amylase content steers were fed diets with differing dietary protein concentrations (Swanson et al., 2008). As dietary protein increased, there were increases in intake, gain, pancreatic trypsin and α-amylase contents. These data support the previous observation that the pancreas is highly responsive to protein but may also support the concept that these responses occur in the absence of starch as dietary starch intake decreased in this study as dietary protein increased.

Mucosal enzymes

There is comparatively little information available describing the regulation and composition of the mucosal dissaccharidases in ruminants. The information has been reviewed (Harmon, 1993) and I will only briefly touch on it here.

There are four proteins possessing carbohydrase activity in the small intestinal mucosa of the non-ruminant. It has been proposed that sucrase-isomaltase contributes approximately 80% of the mucosal maltase activity and maltase-glucoamylase contributes 20% (Galand, 1989). However, recent work in the non-ruminant has more thoroughly characterized the role of maltase-glucoamylase (Quezada-Calvillo et al., 2007a) and found that maltase-glucoamylase is the predominate disaccharidase at low substrate concentrations. As substrate concentrations increase maltase-glucoamylase is inhibited by products of digestion and sucrose-isomaltase predominates. Additional work showed that ablation of maltase-glucoamylase slowed mucosal carbohydrate assimilation providing further evidence for a critical role for maltase-glucoamylase (Quezada-Calvillo et al., 2007b). These authors (Quezada-Calvillo et al., 2008) also demonstrated that the inhibitory or "brake" portion of maltase-glucoamylase activity was associated with the glucoamylase and that removal of maltase-glucoamylase decreased the capacity for intestinal starch digestion and its contribution to blood glucose by 40% (Nichols et al., 2009). These studies have greatly increased our understanding of the role of this important intestinal dissacharidase and similar descriptive information is needed in ruminants.

The ruminant possesses a similar complement of enzyme activities to the non-ruminant, with the exception of sucrase (Kreikemeier et al., 1990). The sucrase-isomaltase gene has been characterized in the bovine (Threadgill and Womack, 1991) but sucrase is apparently not expressed or the protein is not translated. Because sucrose-isomaltase is synthesized as a single protein it may suggest that ruminants differ at some post-translational step or there is a difference in the maltase component as well. Using heat inactivation Coombe and Siddons (1973) suggested that maltase activity involved two proteins which could be solely maltase-glucoamylase. Trehalase also contributes α-glycosidase activity (Kreikemeier et al., 1990), but its nutritional significance has not been established. The other nutritionally important carbohydrase is lactase. Studies characterizing these proteins and their regulation in ruminants are needed before we can understand limitations to intestinal carbohydrate assimilation. Research evaluating nutritional influences on enzyme activities has shown that neither energy nor starch intake influences the concentration of dissacharidases in mucosal tissue from sheep and cattle (Janes et al., 1985; Kreikemeier et al., 1990; Russell et al., 1981). Maltase specific activity is highest in the mid-small intestine and declines abruptly towards the ileum. These studies show a rather limited capacity of the intestinal mucosa to alter disaccharidase activities in response to changes in diet. However, dramatic changes in small intestinal maltase activities have been reported for wethers fed alfalfa hay and infused duodenally with glucose to supply 60, 120, and 180 g/d (McNeill et al., 1974). As glucose increased from 0 to 180 g/d for 2 d, small intestinal maltase increased 28-fold but then decreased to approximately two-fold the initial concentrations by 5 d at 180 g/d of glucose. Other examples of increases in mucosal maltase in animals infused post-ruminally with carbohydrate have been variable. Bauer et al. (2001b) reported increased jejunal maltase in sheep infused abomasally with partially hydrolyzed starch but jejunal maltase decreased in cattle in the same experiment or was unchanged in a companion experiment (Bauer et al., 2001a). An additional experiment infusing various forms of carbohydrate post-ruminally in steers for 40 days reported increased maltase for steers receiving both glucose and partially hydrolyzed starch (Rodriguez et al., 2004). These latter studies (Bauer et al., 2001a; Bauer et al., 2001b; Rodriguez et al., 2004) report maltase measured in isolated enterocytes. While these may provide a more sensitive assay, it also would focus on enterocytes easily dislodged from the villus. However, all approaches indicate that the mucosal enzymes in the ruminant are highly variable and most respond little to dietary manipulation.

Glucose transport

Several processes have been proposed for the entry of luminal sugars into the vasculature draining the small intestine. A mechanism of absorption has been proposed whereby sugars exit the lumen via the intercellular spaces, a process termed solvent drag (Madara and Pappenheimer, 1987; Pappenheimer, 1990, 1987; Pappenheimer and Reiss,

1987). For this process to occur, luminal glucose must be present at high concentrations (>25 mM), and concentrations must exceed approximately 200 mM before paracellular absorption would exceed active transport, (Pappenheimer and Reiss, 1987) which may not occur under physiological conditions (Ferraris et al., 1990). However, these processes may contribute in experiments where glucose is infused post-ruminally (Kreikemeier and Harmon, 1995; Kreikemeier et al., 1991).

The second means whereby sugars may cross the luminal membrane is the facilitated transporter GLUT5. This transporter is responsible for the entry of fructose into the intestinal enterocytes (Burant et al., 1992) but does not transport glucose or galactose. Being a facilitated transporter, GLUT5 will transport fructose down a concentration gradient. Fructose, as a component of sucrose, would represent a significant contribution to the supply of luminal carbohydrate in man. However, its significance in ruminants is unknown since little fructose passes to the small intestine in typical ruminant diets.

The third and usually considered major means whereby glucose crosses the brush-border membrane is via the sodium-dependant glucose transporter, SGLT1 (Hediger and Rhoads, 1994). The SGLT1 transporter is a high affinity glucose transporter (Km~100 μM; Wright, 1993) that couples glucose transport to an inwardly directed Na^+ gradient. This Na^+ gradient is maintained by Na^+-K^+-ATPase in the basolateral membrane.

The final transporter that contributes to sugar entry and exit from enterocytes is GLUT2. The GLUT2 transporter has long been considered the major route of glucose exit from the cells as well as entry of glucose from the blood into enterocytes (Thorens, 1993). Fructose can also cross the basolateral membrane via the activity of GLUT2 (Cheeseman, 1993). However, more recent evidence suggests that apical GLUT2 may be the principle route of glucose absorption from the intestinal lumen as well (Kellett and Helliwell, 2000). Since GLUT2 is a facilitated transporter it may represent what was previously thought to be diffusion or paracellular absorption. The current model of absorption suggests that GLUT2 is rapidly inserted into the apical membrane in response to glucose in the intestinal lumen (Kellett and Brot-Laroche, 2005). The cue is the presence of glucose in the intestinal lumen and its interaction with sweet taste receptors in the brush border membrane (Mace et al., 2007). This then signals for the insertion of GLUT2 into the brush border membrane. It has been shown that artificial sweeteners also stimulate this response (Mace et al., 2007). Estimates in non-ruminants indicate that GLUT-2 facilitated diffusion accounts for up to 3-times the rate of glucose transport mediated by SGLT1 (Kellett and Helliwell, 2000).

Studies on the role of GLUT2 in ruminants are lacking. In studies infusing glucose abomasally (Kreikemeier and Harmon, 1995; Kreikemeier et al., 1991) intestinal glucose disappearance was complete indicating an apparently large capacity for glucose transport. Recent work (Liao et al., unpublished) reported that ileal GLUT2 mRNA expression increased 6-fold whereas SGLT1 expression increased 1.3 fold in steers infused abomasally with starch suggesting that perhaps GLUT2 readily adapts to increased intestinal carbohydrate. However, these results do not demonstrate a localization of GLUT2 to the brush border membrane in ruminants.

Studies using membrane vesicles have reported that ruminants possess a sodium-dependent, saturable system of glucose transport (Crooker and Clark, 1986; Moe et al., 1985). Zhao et al. (1998) prepared brush border membrane vesicles from lactating dairy cows and observed SGLT1 activity throughout the intestine. They also determined SGLT1 expression in several tissues and found high amounts in the stomach tissues, rumen and omasum, as well as in the intestinal tissues, the duodenum, jejunum and ileum.

Nutritional influences on transport

Lambs differing in age and rumen development have been used to measure glucose and galactose disappearance from isolated intestinal loops (Scharrer et al., 1997a) and uptake has been measured *in vitro* using isolated pieces of jejunum (Scharrer et al., 1979b). Both studies demonstrated that sugar uptake was greater in milk-fed lambs. The rate of absorption decreased as age increased, and decreased most in the distal small intestine (Scharrer et al., 1979a). Similar conclusions were drawn by Shirazi-Beechey et al. (1989) using lambs at 1- and 3-wks-old (milk-fed), 5-wks-old (transition period) and 12-wks-old (ruminant). Sodium-dependent glucose transport was present in all regions of the small intestine in pre-ruminant lambs, but was absent in the small intestine of ruminant lambs. In a more detailed report of developmental changes in glucose transport in the lamb, Shirazi-Beechey et al. (1991) found that glucose transporter activity peaked at 2-wks of age and declined to negligible levels by 8-wks of age. This decreased glucose transporter activity was maintained at 2 to 3-yr of age in adult sheep; however, the decline could be prevented by maintaining the lambs on a milk-replacer diet beyond the normal weaning period. Furthermore, when 2 to 3 yr-old sheep were intraduodenally infused with a 30 mM glucose solution for 4 d, glucose transporter activity in brush border membrane vesicles increased 40 to 80-fold. This increase in glucose transporter activity was accompanied by an increase in abundance of SGLT1 protein in the brush-border membrane. This was the first study to demonstrate that the presence of glucose in the intestinal lumen regulates glucose transporter expression in the brush-border

membrane of ruminants.

While an adaptive response to increased luminal glucose is indicative that ruminants can adapt to increase their capacity for carbohydrate assimilation, adaptive responses to starch in the intestine have been less clear, particularly in cattle (Mayes and Orskov, 1974; Bauer et al., 1995). Bauer et al. (2001b) used cattle (8) and sheep (12) in an experiment to study adaptation of glucose transport in the proximal jejunum. Animals were fed fescue hay and infused either ruminally (control) or abomasally (adapted) with a partially hydrolyzed cornstarch solution for 7d. Animals were killed and 1 m of jejunum was harvested and used to prepare brush border membrane vesicles. Animals that were adapted to the hydrolyzed starch (infused abomasally) had higher (2-fold) rates of Na^+ dependant glucose transport. This increased Na^+ dependant glucose transporter activity was greater in sheep than in cattle. This adaptive response was studied in more detail in a second experiment using 13 steers (Bauer et al., 2001a). Steers were again fed fescue hay and infused for 7 d either ruminally (control, n = 6) or abomasally (adapted, n = 7) with a partially hydrolyzed cornstarch solution. On d 7 steers were killed and the entire intestine removed and 5 equally spaced, 1-m segments of small intestine were used for BBMV preparation and analysis of SGLT1 activity. In this experiment, adaptation did not affect SGLT1 activity in the small intestine. Activity of SGLT1 was greatest in the mid jejunum and declined towards the ileum. Similar results were seen in a follow-up study where steers were infused for 35 d either ruminally or abomasally with water, starch hydrolysate or glucose and the intestine was removed at slaughter and BBMV were prepared from 5 sites throughout the small intestine (Rodriguez et al., 2004). There was no effect of treatment on small intestinal glucose transport. Collectively, these studies bring into question the ability of the small intestine of cattle to up-regulate their SGLT1 glucose transporter activity in the presence of complex carbohydrates. Further studies are needed to clearly define the role of SGLT1 and GLUT2 in glucose absorption from the small intestine of cattle.

CONCLUSION

Much progress has been made in understanding the processes of starch assimilation over the past 20 years in many species. Ruminant pancreatic α-amylase can exhibit wide fluctuations in secretion in response to diet. However, the negative adaptive response to dietary starch remains quizzical. The control system seems to have evolved to focus on dietary energy intake. Energy intake in turn increases microbial protein flow which stimulates α-amylase. Thus, the major nutritional controlling factors are energy (protein) which increases α-amylase production and secretion and starch (glucose) which decreases α-amylase production and secretion. Mucosal hydrolysis of the products of α-amylase remains poorly described in ruminants. Crude activities of the carbohydrases have been measured with apparently little change induced nutritionally. While much progress has been made in non-ruminants in describing this phase of digestion little is still known about these processes in ruminants. The understanding of the third and final phase of intestinal carbohydrate assimilation, glucose transport, has also increased greatly for non-ruminants. The addition of GLUT2 as a potential mechanism of mucosal transport may be the key to explaining previous work studying SGLT1 in ruminants. The lack of an adaptive response, yet a perceived high capacity for glucose transport has been troubling, particularly in experiments focusing on cattle. Recent data showing changes in expression of GLUT2 in cattle suggest it may be the primary adaptive transporter, but confirming data is yet lacking. Information building upon these key findings in non-ruminants need to be transferred to ruminants, particularly cattle, to enable a thorough understanding of intestinal digestion.

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