

Factors Influencing Assimilation of Dietary Starch in Beef and Dairy Cattle

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Summary:

- The process of starch assimilation in the ruminant is complex and remains an avenue by which increases in production efficiency can be gained.
- Ruminal starch digestion is typically 75 to 80% of starch intake. Starch that escapes fermentation and flows to the small intestine may be more resistant to enzymatic digestion. On average, 35 to 60% of starch entering the small intestine is degraded there, and of the fraction that escapes small intestinal digestion, 35 to 50% is degraded in the large intestine. This suggests that limitations to small intestinal starch digestion do exist.

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, this system is not designed for use of non-structural carbohydrates. 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 maximized. However, maximum small intestinal digestion does not simply refer to the quantity of starch digested. Increasing starch flow to the small intestine may be accompanied by decreased total tract digestion (Theurer, 1986). Maximum starch flow to the small intestine may also result in greater quantities of starch flowing to the large intestine; digestive efficiency in the large intestine is least and thus large intestinal digestion should generally be avoided (Harmon and McLeod, 2001). We must be able to optimize digestion in the different regions of the gastrointestinal tract if nutritionists are to formulate diets for optimum efficiency of digestion in the total tract. To achieve this we must understand the limitations of the animal to digest and absorb both structural and non-structural carbohydrates. The goal of this review is to describe factors affecting intestinal starch availability and use 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.

GRAIN CHARACTERISTICS

While a detailed analysis of starch structure is beyond the scope of this paper, a rudimentary understanding is necessary to integrate the properties of the starch in feed grains with the digestion processes occurring in the animal. The first impediment to digestion is the seed coat; this is generally overcome with processing but whole corn is still routinely fed. The second obstacle to digestion depends on how the starch is packaged within the kernel.

Starch structure, while simple in concept, is in fact quite complex. Amylose, the linear chain of glucose, is composed of α -1, 4 linked glucose molecules. Amylopectin is comprised of α -1, 4 glucose chains with α -1, 6 linked glucose molecules every 20-30 glucose units, producing a highly branched polysaccharide with relatively short linear chains. While the distribution of these molecules varies with starch source (**Table 1**) most feed grains are typically 75% amylopectin. The packaging of these two molecules into plant starch occurs in granules that are semi-crystalline in nature and are organized as layers with “crystalline” regions composed of primarily amylopectin and “amorphous” regions of mostly amylose. Amylose and amylopectin are held together within the starch granules by extensive hydrogen bonding. Enzyme access to the crystalline regions of raw starch is limited and the altering of this structure is one means whereby steam flaking improves starch availability.

Table 1. Distribution of Amylose and Amylopectin in Various Starch Sources.

	Corn	Waxy Corn	Wheat	Potato	Tapioca
Amylose, %	25	1-5	25	20	17
Amylopectin, %	75	95-99	75	80	83

Considerable effort has been expended in recent years examining the impact of corn genotype on starch availability. Almost all of the kernel area is comprised of endosperm, the structure containing starch granules surrounded by a protein matrix. The protein profile of this protein matrix determines whether the endosperm can be defined as floury or vitreous. Starch granules in vitreous endosperm are imbedded in an insoluble protein matrix whereas granules in floury endosperm are more loosely associated with a soluble protein matrix. Dent corn types tend to have a greater percentage of floury endosperm which is more accessible than the vitreous endosperm in flint corn varieties.

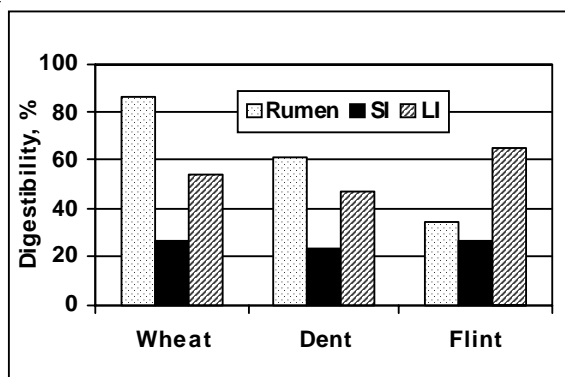


Figure 1. Digestibility of starch in the rumen, small intestine (SI) and large intestine (LI) of steers. (Philippeau et al., 1999b)

(Philippeau et al., 1999a) estimated ruminal starch degradation using in situ disappearance and found that ruminal starch digestion ranged from 40 to 77% for 14 varieties of corn. These same authors (Philippeau et al., 1999b) also compared the digestibility of wheat with dent and flint corn using intestinally cannulated steers. Ruminal starch digestibility (**Figure 1**) decreased from 87% for wheat to 61 and 35 % for dent and flint corns, respectively. Small intestinal starch digestibility ranged from only 23 to 27%; however, starch digestibility in the large intestine ranged from 48% for dent corn to over 65% for flint corn. Because flint corn markedly reduced ruminal starch digestibility the amount of starch digested in the large intestine was approaching 1 kg per day. Large intestinal fermentation of up to a kilogram per day of starch is certainly indicative of a limited capacity for small intestinal starch assimilation. These limitations are exacerbated when the starch is inherently refractory to digestion such as with vitreous endosperm in flint corn.

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, meaning it does not require free ends of amylose chains for activity but is capable of hydrolyzing internal α -1, 4 glucosidic bonds. Bovine α -amylase has reported characteristics similar to those of non-ruminant α -amylase; Michaelis constants and activation energies are similar (Clary et al., 1968) with a pH optimum of 6.9 (Russell et al., 1981). Products of luminal starch digestion by α -amylase are a mixture of maltose, maltotriose and various limit dextrins (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., 1969), concurrent with increased total pancreatic secretion with increasing age (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 nonruminant (Merchen, 1988). Nutritional regulation of pancreatic enzyme output must therefore result in changes in pancreatic enzyme synthesis to impact intestinal enzyme supply.

Early experiments evaluating effects of concentrate or forage-based diets on concentrations of pancreatic α -amylase were confounded by energy intake. Clary et al. (1969) maintained steers (24 each group) on pasture or an all-concentrate diet for 126 days prior to slaughter. Steers consuming the all-concentrate diet had 40% higher α -amylase activity in pancreatic tissue than those maintained on pasture. A similar tendency was reported for sheep (6 per group) 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 postprandial digestive enzymes. Steers were fed either alfalfa hay to meet maintenance energy requirements or a corn/corn silage-based diet fed at one, two, or three times their maintenance energy requirements. At equal energies, the corn/corn silage-based diet slightly reduced pancreatic α -amylase concentrations compared to alfalfa hay. Increasing intake of the corn/corn silage-based diet from one to two times maintenance energy increased pancreatic α -amylase concentrations approximately two-fold, but there were no further increases in pancreatic α -amylase concentrations as energy intake increased to three times maintenance energy. To evaluate the interactions of diet composition and energy content on pancreatic α -amylase concentration, calves were fed either 90% forage (alfalfa) or 90% grain (wheat:sorghum) diets at one or two times maintenance energy intake for 140 d (Kreikemeier et al., 1990). Regardless of diet, increasing energy intake (from one to two times maintenance energy) increased pancreatic α -amylase concentration (55%) and total content of α -amylase in the pancreas (140%). However, both pancreatic α -amylase concentration and total content of α -amylase in the pancreas were lower in calves consuming

the 90% grain diet compared with those fed forage (34 and 44%, respectively). Compared to grain-fed calves, calves fed forage had greater concentrations of α -amylase in the small intestinal digesta (34%) and greater total content of α -amylase in the small intestinal digesta (50%), indicating greater pancreatic secretion of α -amylase. These results agree with those of Russell et al. (1981) which compared forage and grain at maintenance energy intakes. The decreases in pancreatic α -amylase concentrations with greater starch intake observed by Kreikemeier et al. (1990) 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 experiments that suggested greater pancreatic α -amylase as starch intake increased also had concurrent increases in total energy intake.

Concentration and secretion of pancreatic α -amylase can be manipulated nutritionally, but experiments 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) infused either glucose, maltose, or starch (200 g/d) into the duodenum of wethers 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 time points. To my knowledge, this was the first report to suggest that increased small intestinal starch could result in decreased secretion of pancreatic α -amylase.

To further define the relationship between intestinal carbohydrate supply and pancreatic enzyme secretion, Walker and Harmon (1995) infused a partially hydrolyzed starch solution or water into either the rumen or abomasum of steers fitted with pancreatic cannulas. Abomasal infusion of partially hydrolyzed starch decreased secretion of pancreatic α -amylase by 60% compared with control water infusion. This decrease occurred despite greater (19%) pancreatic juice secretion in steers with abomasal carbohydrate infusion. Abomasal carbohydrate infusion also increased portal blood glucose concentrations; however, insulin concentrations were unaffected. It is clear that increasing small intestinal carbohydrate can decrease pancreatic α -amylase secretion, but it is unclear if the negative effects of carbohydrate occur because of increased carbohydrate in the small intestinal lumen or result from increased absorbed glucose. To test this hypothesis Swanson et al. (2002b) abomasally infused water, glucose, or partially hydrolyzed starch in steers fitted with pancreatic cannulas. Similar to the experiment (Walker and Harmon, 1995), infusion of both carbohydrate sources increased pancreatic juice secretion but resulted in linear decreases in pancreatic α -amylase secretion compared to control water infusion. This indicates that complex carbohydrate in the lumen of the gastrointestinal tract is not solely responsible for the down-regulation of α -amylase secretion because similar changes are elicited by glucose. Whether the negative effects of luminal carbohydrate (both simple and complex) on pancreatic α -amylase secretion occur via luminal or post-absorptive effects remains unclear.

Both high-starch diets (Kreikemeier et al., 1990) or and postruminal carbohydrate infusion infusing carbohydrate post-ruminally into cattle (Swanson et al., 2002b; Walker and Harmon, 1995) have consistently reduced pancreatic α -amylase concentration and/or secretion in cattle. Attempts to study the dietary regulation of α -amylase in sheep have produced differing different results. Janes et al. (1985) fed sheep grass or corn-based diets and found only a tendency for an increase in pancreatic α -amylase. Swanson et al. (2000) fed lambs either forage or concentrate based- diets at 1.2 or 1.8 times their

net energy for maintenance requirement with diets that were either forage or concentrate based. Diet had little impact on pancreatic α -amylase with the exception of the high-energy, high-starch diet; . Lambs receiving this diet had higher concentrations of pancreatic α -amylase activity and, greater amounts of α -amylase protein but the lowest amounts of α -amylase mRNA. These differences demonstrate the complexity of the α -amylase regulation of α -amylase and indicate that nutritional influences on enzyme regulation may not be comparable between different species of ruminants.

Other luminal nutrients may interact with small intestinal carbohydrate assimilation. Taniguchi et al. (1995) infused casein and starch post-ruminally in steers and demonstrated that glucose supply from the portal-drained viscera was increased 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. Abomasal infusion of starch with casein increased starch disappearance from the small intestine of steers compared to starch infusion alone (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 with abomasal casein was infused abomasally infusion (Richards et al., 2003). These studies experiments demonstrated that casein (protein) does influences the regulation of pancreatic α -amylase. To study how casein and starch interact to affect pancreatic α -amylase, calves were infused abomasally with starch and/or casein and the pancreas was collected at slaughter for analysis (Swanson et al., 2002a). Starch iInfusing on 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 (Swanson et al., 2002b; Walker and Harmon, 1995). However, casein infusing infusion casein increased pancreatic α -amylase activity (28%), protein content (38%) and increased α -amylase mRNA (69%). When Infusion of both starch and casein were infused together the effects closely resembled those of the starch; decreased 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 (Benson et al., 2002). Infusion of starch, with or without casein, increased secretion of pancreatic juice and decreased the α -amylase concentration of α -amylase in pancreatic juice compared to control water infusion,. Howeverbut, total secretion of α -amylase was unchanged because of the increased increase in total juice secretion. Casein infusion increased α -amylase secretion compared to control of α -amylase, but only when starch was not also infused. Starch infusion, in addition to Accompanying the increaseding secretion of pancreatic juice, were increased plasma concentrations of insulin and cholecystokinin (CCK) but not reduced plasma glucagon concentration compared to control for steers receiving the starch infusions. Casein infusion increased plasma insulin concentration, actually produced lowerreduced plasma CCK concentrations, and did not affect plasma glucagon concentration than the compared to 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 pancreatic α -amylase secretion responds to increased dietary starch much similar to like how pancreatic α -amylase secretion in ruminants responds to withto 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.

Mucosal Enzymes

There is comparatively little information available describing the regulation and composition of the mucosal disaccharidases. 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. Sucrase-isomaltase contributes approximately 80% of the mucosal maltase activity and maltase-glucoamylase contributes 20% (Galand, 1989). Trehalase also contributes α -glycosidase activity (Kreikemeier et al., 1990), but its nutritional significance has not been established. The fourth other nutritionally important carbohydrase is lactase. 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 the sucrase subunit is apparently not expressed. Neither the sucrase-isomaltase nor the maltase-glucoamylase proteins have been characterized in the ruminant. Studies characterizing these proteins and their regulation in ruminants are needed before we can understand limitations to intestinal carbohydrate assimilation.

Researcher has shown that neither energy or starch intake influences the concentration of disaccharidases 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 experiments 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 or 180 g/d (McNeill et al., 1974). As glucose infusion increased from 0 to 180 g/d for 2 d, small intestinal maltase concentration increased 28-fold after 2 d of infusion of 180 g/d glucose, but then decreased to approximately two-fold the initial concentrations by 5 d after infusion of 180 g/d glucose. Other examples of increases in mucosal maltase in animals infused post-ruminally with carbohydrate have been variable. Bauer et al. (2001b) reported increased greater jejunal maltase in sheep infused abomasally with partially hydrolyzed starch but in cattle 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 Hazleton, 2002). These latter studies (Bauer et al., 2001a, 2001b; Rodriguez Hazleton, 2002) 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 do not respond to dietary manipulation.

Glucose Transport

Comparatively few aspects of the process of glucose absorption have been described in detail for ruminants. General aspects for ruminants have been reviewed

(Harmon and McLeod, 2001) and only a brief description will be included here. Excellent detailed reviews describing the structure and function of the Na⁺-dependent glucose transporter (SGLT1) are available (Hediger and Rhoads, 1994; Wright, 1993).

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; 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 indeed contribute in experiments where glucose is infused post- ruminally (Kreikemeier et al., 1991; Kreikemeier and Harmon, 1995).

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 Because it is a facilitated transporter, GLUT5 will transport fructose down a concentration gradient. Fructose, as a component of sucrose, would could represent a significant contribution to the supply of luminal carbohydrate in humans. However, its significance in ruminants is unknown since little fructose passes to the small intestine in typical ruminant diets.

The third transporter that contributes to sugar entry and exit from enterocytes is GLUT2. This transporter is present in the basolateral membrane and serves as the major route of glucose exit from the cells as well as entry of glucose from the blood into enterocytes (Thorens, 1993). Fructose also crosses the basolateral membrane via the activity of GLUT2 (Cheeseman, 1993). Recent evidence in rodents also suggests that GLUT2 insertion into the luminal membrane of enterocytes can occur when glucose uptake (via the Na⁺-dependent glucose transporter, SGLT1) increases and luminal concentrations of glucose are high (Gouyan et al., 2003; Helliwell and Kellet, 2002). This mechanism could significantly contribute to glucose uptake by the intestine with high concentrations of luminal glucose but has not been characterized in ruminants.

The fourth and primary means by which glucose crosses the brush-border membrane is via the Na⁺-dependent 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.

Ruminants possess a Na⁺-sodium-dependent, saturable system of glucose transport (Crooker and Clark, 1986; Moe et al., 1985). Zhao et al. (1998) prepared brush border membrane vesicles (BBMV) 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). Whether this expression is translated into protein activity has not been demonstrated. This transporter could play a role in maintaining Na⁺ and water balance in these tissues (Loo, 1999) if it was active.

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., 1979a) and uptake has been measured in vitro using isolated pieces of jejunum (Scharrer et al., 1979b). Both experiments demonstrated that sugar uptake was greater in milk-fed lambs compared with conveniently fed. The rate of absorption decreased as age increased, and decreased most in the distal small intestine (Scharrer et al., 1979b). 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). Na⁺-dependent glucose transport was present in all regions of the small intestine in preruminant 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 decrease in 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 (BBMV) 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 (Bauer et al., 1995; Mayes and Orskov, 1974). 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 7 d. Animals were killed and 1 m of jejunum was harvested and used to prepare BBMV. Animals that were adapted to the hydrolyzed starch (infused abomasally) had higher (2-fold) rates of Na⁺-dependant dependent glucose transport. This increased Na⁺-dependant dependent glucose transporter activity was greater in sheep than in cattle. and tThis 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-another study experiment where steers were infused for 35 d either ruminally or abomasally with water, starch hydrolysate or glucose (Rodriguez Hazleton, 2002) and the intestine was removed at slaughter and BBMV were prepared from 5 sites throughout the small intestine (Rodriguez Hazleton, 2002). There was no effect of treatment on small intestinal glucose transport. These studies experiments bring into question the ability of the small intestine of cattle to upregulate their glucose transporter activity in the presence of complex carbohydrates.

Optimizing Site of Digestion Advantages of Small Intertine

The dilemma we face as nutritionists is how to take advantage of the increased efficiency of small intestinal digestion. In order to evaluate the impact of site of digestion on energetic efficiency, (McLeod et al., 2001) determined energy retention in steers receiving ruminal versus abomasal starch infusions for 28 days, and . From these animals they estimated partial efficiencies of infused substrates using indirect calorimetry. Abomasal infusion resulted in a partial efficiencies efficiency of 0.60, whereas ruminal infusions resulted in a partial efficiencies efficiency of 0.48. It was known that the amounts of starch infused abomasally in these studies experiments would not exceed the capacity of the small intestine to digest the starch and absorb glucose (Branco et al., 1999). These data demonstrate that an energetic advantage can be gained if starch is digested and absorbed in the small intestine. At the very least we should avoid digestion in the large intestine because of its low efficiency of nutrient utilization which is the least efficient site (Owens et al., 1986). (Zinn et al., 2002) summarized available literature and suggested that post-ruminal starch digestion appears to be limited by accessibility of the amyolytic enzymes and not limitations in the amount of enzymes. It is clear that processing, particularly steam-flaking enhances digestive efficiency. However, starch that escapes digestion in the small intestine is extensively fermented in the large intestine (Philippeau et al., 1999b), thus indicating that additional starch is potentially available in the small intestine. It has not been demonstrated that additional amyolytic activity would improve digestion in the small intestine (Remillard et al., 1990), suggesting that either mucosal enzymes or glucose transport may be limiting to assimilation. To apply these concepts in practice we need an accurate depiction of small intestinal digestion. We need to predict starch flow and disappearance from the small intestine at the time of diet formulation. Based on our current methods, adequate means of reliably assessing small intestinal digestion are not available. Without the ability to accurately describe and predict small intestinal starch digestion we cannot optimize starch digestive efficiency. We can only hope to avoid fecal starch excretion through grain processing and nutritional management.

Another factor making the application of increased complicating changes in our ability to predict small intestinal starch digestion and glucose absorption difficult is the changing visceral metabolism. Just because starch disappears in the small intestine does not mean it contributes to absorbed glucose (Kreikemeier et al., 1991). To determine how site of starch digestion interacts with visceral glucose metabolism, steers (Harmon et al., 2001) were infused either ruminally or post-ruminally with starch (Harmon et al., 2001). Visceral nutrient flux and glucose metabolism were measured while infusing ¹⁴C-glucose. Based on an estimated small intestinal digestibility of 90% for the infused starch (Branco et al., 1999), estimates of net portal-drained visceral glucose absorption indicated that from 0.38 to 0.5638 to 56% of the infused carbohydrate was presented to the liver as glucose. However, when the estimates were corrected for changes in visceral glucose metabolism this estimate increased to 0.7777%. These types of measurements, where we measure glucose net absorption and visceral metabolism, may provide more meaningful and precise estimates of small intestinal starch availability, upon which we can begin to build these much needed dietary predictions and thereby improve digestive efficiency. We need data such as these in beef and dairy cattle with high levels of feed consumption.

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