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Glucose, starch, and dextrin utilization in the small intestine of steers

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Glucose, Starch, and Dextrin Utilization in the Small Intestine of Steers

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T.B. Avery¹, and R.T. Brandt, Jr.²

Summary

Glucose infused into the abomasum of Holstein steers resulted in higher arterial glucose concentrations and increased net glucose absorption than either starch or dextrin infusions. Increasing infusion rates above 20 g/hr for both starch and dextrin resulted in no further increases in net glucose absorption. Even though the enzymatic starch and dextrin hydrolysis became saturated above 25 g/hr, the amount of starch and dextrin disappearing in the small intestine increased with higher infusion rates. This was accompanied by increased volatile fatty acid concentrations in the ileal fluid with starch and dextrin infusions, but not when glucose was infused. These data support two concepts: (1) microbial fermentation is involved in small-intestinal starch disappearance and (2) starch and dextrin hydrolysis in the small intestine of steers is more rate limiting than glucose absorptive capacity.

Introduction

Because feed grains are about 70% starch, starch is the primary energy source in the diet of finishing beef cattle. Digestion of starch can occur either by microbial fermentation in the rumen and hindgut or by enzymatic hydrolysis in the small intestine. Total tract starch digestion in beef cattle ranges from 80 to 95% and is affected by grain type, as well as processing method. Extensive processing of grain will increase its digestibility, but may increase the potential for acidosis. Underprocessing, however, results in decreased starch digestion and poor feed efficiency. Starch being digested and absorbed in the small intestine as glucose is more energetically efficient than its fermentation to volatile fatty acids. However, as the amount of starch escaping ruminal fermentation increases, so does fecal starch excretion, indicating a limit to the rate of small-intestinal starch digestion. Therefore, a series of experiments was conducted to evaluate small-intestinal starch digestion and to determine factors that may be limiting.

Experimental Procedure

Two groups of four Holstein steers (group 1, 660 lbs; group 2, 890 lbs) were surgically fitted with abomasal and ileal cannulae, portal and mesenteric catheters, and an elevated carotid. A temporary catheter was placed in the carotid

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artery during sampling periods. Glucose, corn starch, and corn dextrin were infused abomasally at 0, 20, 40, and 60 g/hr. Ileal digesta samples were collected, and disappearance of carbohydrate in the small intestine was determined using Cr:EDTA as an indigestible marker. Simultaneous blood samples were collected from the portal vein and carotid artery, and glucose absorption across the small intestine was calculated. Portal blood flow was determined by a primed continuous infusion of para-amino-hippuric acid (PAH) into a small mesenteric vein.

With the first group of steers, glucose (Experiment 1) or corn starch (Experiment 2) were continuously infused into the abomasum at 0, 20, 40, or 60 g/hr for 10 h. Infused volume was 250 ml of solution per hour, consisting of tap water, the appropriate carbohydrate, and Cr:EDTA. Steers were fed chopped alfalfa hay at 1.5% of body weight (dry matter basis). Animals and treatments were randomized to an 8-period crossover design for each experiment. During each infusion period, 7 ileal digesta samples and 5 sets of blood samples were collected from each steer. Ileal digesta was analyzed for VFA concentration, dry matter, starch, glucose, and chromium. Plasma samples were analyzed for glucose and PAH.

The second group of steers was infused with glucose (Experiment 3), corn starch (Experiment 4), or corn dextrin (Experiment 5). Infusions, digesta and blood collections, lab analyses, and animal care were identical to those in Experiments 1 and 2. Animals and treatments were randomized to a 4 x 4 Latin square design for Experiments 3 and 4, whereas Experiment 5 was an 8-period crossover design.

Results and Discussion

Experiment 1. Steers consumed 9 lbs of alfalfa hay daily (dry matter basis) during the glucose infusions (Table 35.1). Increasing levels of abomasal glucose infusion resulted in glucose passing the ileum into the large intestine, yet there was no change in ileal fluid VFA concentration. This indicated that there was little microbial fermentation of glucose in the small intestine. Arterial glucose concentration as well as net glucose absorption continued to increase with higher glucose infusions. The amount of glucose absorbed was approximately equal to the amount of glucose disappearing in the small intestine.

Experiment 2. Steers consumed 7.25 lbs of alfalfa daily (Table 35.2). Even though corn starch was infused, both free glucose and starch passed the ileum. The amount of starch that escaped small-intestinal digestion increased with increasing amounts of starch infusion. In addition, as starch passing the ileum increased, ileal fluid VFA concentration also increased. This indicates that small-intestinal starch digestion included microbial fermentation. Arterial glucose and net glucose absorption increased as the infusion rate was raised from 0 to 20 g/hr, with no additional change as infusion increased further. It appears that the processes for small-intestinal starch hydrolysis became saturated at approximately the 20 g/hr infusion level.

Experiment 3, 4, 5. The results of Experiments 3 and 4 (Table 35.3 and 35.4) conducted with group 2 steers were similar to trends observed in the first two experiments. The final experiment (Table 35.5) was the infusion of corn dextrin. Dextrin is partially hydrolyzed starch, consisting of straight chain glucose polymers with an average chain length of 17 glucose units. Dextrin has none of the granules

or molecular branching points that are found in starch and may limit enzymatic hydrolysis. When corn dextrin was infused, steers consumed 11.5 lbs of alfalfa hay daily. At higher levels of infusion, free glucose as well as dextrin flowed past the ileum to the large intestine. There was an increase in ileal fluid VFA concentration, whereas arterial glucose levels and net glucose absorption both plateaued at the 20 g/hr infusion rate, as in previous experiments with starch.

Regardless of the type of carbohydrate infused, increased infusion rates resulted in increased amounts of small-intestinal carbohydrate disappearance. When glucose was infused, most of the disappearance could be accounted for by glucose absorption. With starch and dextrin infusions, arterial glucose and glucose absorption plateaued near the 20 g/hr infusion rate. This was accompanied by a gradual increase in ileal fluid VFA concentration. Therefore, it is probable that a large amount of starch digestion in the small intestine is by microbial fermentation. It also appears that enzymatic processes responsible for starch and dextrin hydrolysis are more rate limiting than the glucose absorption capacity of the small intestine.

Table 35.1. Effect of Abomasal Glucose Infusions on Small-intestinal Disappearance and Net Portal Glucose Absorption (Experiment 1)

Item	Glucose Infusion rate, g/hr				SE
	0	20	40	60	
Daily feed, lbs	9.2	9.7	9.2	8.6	0.4
Glucose flowing past ileum, g/hr ^{ab}	0	0.8	8.0	20.6	1.5
VFA in ileal fluid, mM	20.8	21.3	22.0	21.3	1.5
Arterial glucose, mM ^a	4.1	4.5	4.6	5.0	0.1
Net portal glucose absorption, g/hr ^a	-2.5	13.4	18.2	34.2	5.5

^aLinear effect, $P < .01$,

^bQuadratic effect, $P < .01$.

Table 35.2. Effect of Abomasal Starch Infusions on Small-intestinal Disappearance and Net Portal Glucose Absorption (Experiment 2)

Item	Starch Infusion rate, g/hr				SE
	0	20	40	60	
Daily feed, lbs	7.5	7.5	6.6	7.5	0.4
Glucose flowing past ileum, g/hr ^a	0	0.5	0.9	1.1	0.1
Starch flowing past ileum, g/hr ^{ab}	0	1.3	13.3	26.2	1.8
VFA in ileal fluid, mM ^a	23.3	26.2	28.9	30.2	1.9
Arterial glucose, mM ^a	4.1	4.3	4.4	4.3	0.1
Net portal glucose absorption, g/hr ^a	-5.7	5.0	2.1	6.3	3.2

^aLinear effect, $P < .05$.

^bQuadratic effect, $P < .05$.

Table 35.3. Effect of Abomasal Glucose Infusions on Small-intestinal Disappearance and Net Portal Glucose Absorption (Experiment 3)

Item	Glucose Infusion rate, g/hr				SE
	0	20	40	60	
Daily feed, lbs	12.5	13.0	10.4	12.3	1.3
Glucose flowing past ileum, g/hr ^a	0	0	4.90	13.8	2.8
VFA in ileal fluid, mM	23.9	25.8	27.9	20.5	2.8
Arterial glucose, mM ^a	4.4	4.9	5.1	5.1	0.1
Net portal glucose absorption, g/hr ^a	-7.3	19.8	20.0	36.6	7.9

^aLinear effect, $P < .01$.

Table 35.4. Effect of Abomasal Starch Infusions on Small-intestinal Disappearance and Net Portal Glucose Absorption (Experiment 4)

Item	Starch Infusion rate, g/hr				SE
	0	20	40	60	
Daily feed, lbs	11.9	13.2	13.2	13.0	0.9
Glucose flowing past ileum, g/hr ^a	0	0.7	2.3	2.7	0.5
Starch flowing past ileum, g/hr ^a	0	5.5	10.9	26.1	4.1
VFA in ileal fluid, mM	26.9	30.9	29.8	29.6	2.3
Arterial glucose, mM ^a	4.1	4.2	4.3	4.4	0.1
Net portal glucose absorption, g/hr ^a	-6.7	8.5	12.0	12.1	1.9

^aLinear effect, P<.01^bQuadratic effect, P<.01.

Table 35.5. Effect of Abomasal Dextrin Infusions on Small-intestinal Disappearance and Net Portal Glucose Absorption (Experiment 5)

Item	Dextrin Infusion rate, g/hr				SE
	0	20	40	60	
Daily feed, lbs	11.0	13.2	9.9	10.6	1.4
Glucose flowing past ileum, g/hr ^{ab}	0	0	0.4	1.0	1.3
Dextrin flowing past ileum, g/hr ^a	0	6.2	5.1	11.1	1.3
VFA in ileal fluid, mM ^a	22.3	24.7	22.4	30.7	3.2
Arterial glucose, mM ^{ab}	4.3	4.6	4.7	4.6	0.1
Net portal glucose absorption, g/hr ^{ab}	-7.3	14.4	14.3	9.4	4.6

^aLinear effect, P<.05.^bQuadratic effect, P<.05.