

Glucose Metabolism and Milk Yield of Cows Infused Abomasally or Ruminally with Starch^{1,2}

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ABSTRACT

The effect of ruminal or abomasal starch infusion on milk yield and glucose metabolism of early lactation cows was measured. Four cows were continuously infused in the rumen or abomasum with partially hydrolyzed starch (1500 g/d) or were not infused (control) for three 14-d periods during wk 4 to 12 postpartum. Milk yield averaged over 40 kg/d throughout the experiment. Milk and milk lactose yields tended to increase when starch was infused and DMI was decreased, regardless of the site of infusion. Starch infusion increased mean insulin concentration and tended to decrease the concentration of serum nonesterified fatty acids. Ruminal starch infusion did not affect glucose irreversible loss rate but tended to increase glucagon concentration and decrease glucose oxidation. The increased milk yield that occurred when starch was infused ruminally relative to the milk yield of control cows could be a result of increased microbial protein supply or increased energy availability. Compared with ruminal starch infusion, abomasal starch infusion tended to increase the irreversible loss rate of glucose and to increase glucose oxidation. Abomasal infusion tended to increase plasma insulin concentration and to decrease the nonesterified fatty acid concentration

relative to ruminal infusion. Infusion of starch abomasally resulted in increases of most uses of glucose, including milk lactose production, glucose oxidation, and the possible storage of glucose as body fat, which indicates that the early lactation dairy cow has a greater capacity for glucose metabolism than is provided by voluntary feed intake of average diets, but that not all available glucose is partitioned to the mammary gland. These data should be useful in testing current concepts and equations in nutritional and metabolic models of dairy cattle.

(**Key words:** dairy cows, starch infusion, glucose metabolism)

Abbreviation key: ILR = irreversible loss rate.

INTRODUCTION

In typical diets of high yielding dairy cattle in the US, large quantities of dietary starch might escape ruminal fermentation and become available for degradation and absorption from the lower gastrointestinal tract. Recent studies with lactating dairy cows (1, 25) indicate that 1 to 5 kg/d of starch might disappear from the lower gut of cows fed high starch diets. Starch that escapes the rumen must be digested and absorbed in the lower tract to be of benefit to the cow. Limits to starch digestion and glucose absorption in the small intestine of the ruminant have been identified (20, 22, 31). Additionally, hepatic gluconeogenesis decreases in response to increased glucose absorption (6, 17), apparently because of insulin secretion (13). Depending on the extent of these limitations, digestion of starch in the small intestine and absorption of glucose is theoretically a more efficient source of glucose than is ruminal fermentation of starch and subsequent hepatic gluconeogenesis (29).

High yielding dairy cows have a specific requirement for glucose for lactose synthesis. The mammary gland utilizes 60 to 85% of the total glucose used in lactating ruminants, and lactose synthesis accounts

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for 50 to 85% of mammary glucose utilization (3). Elliot (14) calculated that, in lactating cows that yield >30 kg/d of milk, all available glucose would be used by the mammary gland, making these cows particularly sensitive to glucose supply. Despite this specific glucose requirement for lactose synthesis, an increase in the supply of glucose to the mammary gland has not consistently increased milk lactose synthesis or milk yield (2, 11, 12).

Further exploration of the effects of site of starch digestion on total glucose supply and subsequent milk yield of lactating cows is needed to estimate the potential contribution of postruminally digested starch and to improve prediction of nutrient availability. The objective of this experiment was to measure the long-term response of milk yield and glucose metabolism of early lactation cows infused either abomasally or ruminally with starch.

MATERIALS AND METHODS

Cows and Management

Four multiparous cows were ruminally cannulated during the dry period preceding the onset of the experiment. Under local anesthetic, each cow was fitted with a 7.6-cm i.d. flexible ruminal cannula, which was replaced with a 10-cm i.d. ruminal cannula 10 d post-surgery. Cows averaged 24 DIM (SD = 2.1) at the start of the experiment and were assigned randomly to one of two incomplete 3 × 3 Latin squares. Two 3 × 3 squares were set up, and one row was randomly selected and removed from each square, leaving two cows in each of two squares and three periods (12 observations). This design balanced our need for statistical power (one 3 × 3 Latin square was insufficient) with our desire to minimize the number of isotope infusions.

Cows were fed diets containing 50% forage (first-cutting, wilted alfalfa silage), 28% NDF, 37% starch, and 18% CP on a DM basis. Complete ingredient and nutrient composition of the diet is presented in Table 1. Cows were housed in a climate-controlled barn (16°C and 65% relative humidity) in tie stalls with rubber mats bedded with wood shavings. Orts were weighed daily, and the amount of feed offered was adjusted to about 10% in excess of intake on the previous day. Water was available *ad libitum* intake. Cows were milked at 0700 and 1900 h and were fed at 0800 and 2000 h. Cows were allowed access to an exercise lot for 1.5 h daily. This experiment was conducted under approval from the Beltsville Area Institutional Animal Care and Use Committee, the USDA Radiation Safety Committee, and

TABLE 1. Ingredient and nutrient composition of the basal diet.

Ingredient	(% of dietary DM)
Alfalfa silage	49.63
Ground high moisture corn grain	37.72
SoyPass ^{TM,1}	10.9
Trace mineral and vitamin mix ²	1.75
Nutrient ³	
NDF	28.0
ADF	21.5
Lignin	5.69
Starch	36.6
Ash	6.10
CP	18.0
Forage NDF	23.4
Ca	0.74
P	0.48
Mg	0.24
Na	1.61
Cl	0.46
S	0.22

¹Soybean meal treated with lignosulfonate (Ligno-Tech USA, Fort Wayne, IN).

²Composition: 14.7 ppm of Co, 97 ppm of Cu, 1174 ppm of Fe, 21 ppm of I, 592 ppm of Mn, 16 ppm of Se, 2275 ppm of Zn, 277 KIU/kg of vitamin A, 140 KIU/kg of vitamin D, and 25 IU/kg of vitamin E.

³Calculated from nutrient composition of individual ingredients. Mineral composition calculated from NRC (28) tabular values.

the University of Maryland Animal Care and Use Committee.

Experimental Procedures

Cows were randomly assigned to one of three treatments. Treatments were continual infusion of a 16% partial starch hydrolysate solution (7) to the rumen or abomasum or no infusion (control). The starch solution was infused at a rate of 7.1 ml/min for 22 h/d throughout the treatment period. The total dosage was 9.4 L/d (1500 g/d starch) of starch solution. The abomasal infusion was through Tygon[®] tubing (6.35 mm i.d.; Norton Performance Plastics, Akron, OH) inserted into the abomasum through the rumen and held in place with a flexible disk (15 cm i.d.). Each experimental period lasted 14 d; the first 10 d were for adjustment to the treatment. Sample periods were kept short to complete the experiment while cows were in early lactation.

Bilateral jugular catheters were inserted on d 10 and kept patent until the end of each experimental period. On d 11 or 14 of each period, cows received a primed continuous jugular infusion of [U-¹⁴C]glucose. Two cows were infused each day, and the day of isotope infusion was randomized for each cow for each

period. At 1100 h, each cow was given a priming dose of 52 μCi , followed immediately by continuous infusion of 1.3 $\mu\text{Ci}/\text{min}$ for 300 min. Jugular blood samples were taken before isotope infusion began, hourly for the first 2 h of the infusion, and then every 20 min for the remaining 3 h ($n = 10$ per cow). Blood was sampled in 10-ml tubes treated with K-EDTA, immediately placed on ice, and then frozen. Separate blood samples used to determine blood CO_2 content were taken every 20 min for the last 3 h of infusion ($n = 7$ per cow) in airtight 3-cc syringes flushed with heparin. Blood samples used to determine insulin, growth hormone, glucagon, glucose, NEFA, and BHBA were drawn from jugular catheters every 30 min over a 4-h period, beginning 2 h before the morning feeding ($n = 9$ per cow) on the day of intravenous [^{14}C]glucose infusion. Plasma was collected and frozen.

Milk weights were recorded on d 11 through 14. Milk samples ($n = 4$ per cow) were collected on d 9 and 10 of each period to avoid contamination of milk with ^{14}C -labeled compounds, and samples were analyzed for milk fat, protein, lactose, and SNF with infrared analysis (Environmental Systems Services, College Park, MD). The feed offered, grain, silage, and orts were sampled on d 11 through 14. Fecal grab samples were taken every 8 h on d 13 and 14. Sample times were shifted forward by 4 h on the 2nd d so that samples represented every 4 h of a 24-h period ($n = 6$ per cow). Starch hydrolysate was sampled on d 10, 12, and 14 of each period.

Chemical Analyses

Carbon dioxide content and specific activity were analyzed immediately on fresh blood samples (32). Carbon dioxide from blood was trapped in $\text{Ba}(\text{OH})_2$, titrated with HCl, then re-trapped with Tris-EDTA, and counted using a liquid scintillation detector. Blood samples were analyzed for the specific activity of glucose (27). Concentrations of plasma insulin (15), glucagon (pancreatic glucagon kit GL-32K; Linco Research, Inc., St. Charles, MO), and growth hormone (15) were measured by radioimmunoassay. Plasma glucose concentration was measured with immobilized glucose oxidase-peroxidase (model 2700 select biochemistry analyzer; Yellow Springs Instruments Inc., Yellow Springs, OH). Plasma NEFA were analyzed enzymatically using acyl-coenzyme A synthetase, acyl-coenzyme A oxidase, and peroxidase (NEFA C kit; Wako Chemicals USA, Inc., Dallas, TX) with modifications to reduce sample size and

reagent volume (26). Plasma BHBA was analyzed enzymatically using BHBA dehydrogenase (B-HBA kit 310-UV; Sigma Chemical Co., St. Louis, MO).

Samples of feces, diet, orts, grain, and silage were dried in a forced-air oven at 60°C for 72 h. All samples were ground through a 1-mm screen in a Wiley mill (Arthur H. Thomas, Philadelphia, PA). Samples were analyzed in duplicate for DM, ash, N, starch, NDF, ADF, and lignin. Ash was determined following sample ignition at 500°C for 6 h (4). Samples were analyzed for total N by micro-Kjeldahl digestion with automated procedures (Technicon Instruments Corp., Tarrytown, NY). Total starch analysis was completed using a two-stage enzymatic hydrolysis method (30), and glucose release was quantified with immobilized glucose oxidase-peroxidase as described for blood glucose. Fecal flows and digestibilities were calculated using lignin as a marker.

Data for the specific activity of plasma glucose were plotted versus time, and the plateau specific activity was identified and calculated for each infusion by averaging the data between 3 and 5 h of infusion (8). The irreversible loss rate (ILR) of glucose was calculated according to the method of Steele et al. (35) using the formula $\text{ILR (moles per day)} = [\text{infusion rate (microcurie per hour)}/\text{plateau specific activity of blood glucose (microcurie per mole)}] \times 24$.

Transfer of labeled glucose to CO_2 was calculated with the formula CO_2 derived from plasma glucose (percentage) = $[\text{plateau specific activity of blood CO}_2 \text{ (microcurie per mole)}/\text{plateau specific activity of blood glucose (microcurie per mole)}] \times 100$.

Statistical Analysis

All data were statistically analyzed using the MIXED procedure of SAS (34). The data were analyzed as replicated ($n = 2$) incomplete 3×3 Latin squares. Plasma hormone and metabolite concentrations were averaged by cow for each period prior to statistical analysis. Data were analyzed with the model

$$Y_{ijkl} = \mu + S_i + C_{j(i)} + P_k + T_l + e_{ijkl}$$

where

- μ = overall mean,
- S_i = random effect of square ($i = 1$ to 2),
- $C_{j(i)}$ = random effect of cow within square ($j = 1$ to 2),
- P_k = fixed effect of period ($k = 1$ to 3),

T_1 = fixed effect of treatment (1 = 1 to 3), and e_{ijkl} = residual error, assumed to be normally distributed.

Preplanned orthogonal contrasts were used to determine the significance of differences among treatments (ruminal vs. abomasal infusion; infusion vs. no infusion). Model effects were declared significant at $P < 0.05$, and trends were declared at $P < 0.15$, unless otherwise noted.

RESULTS AND DISCUSSION

Feed Intake and Nutrient Digestibility

Infusion of starch decreased voluntary feed intake (not including infused starch) by 0.75 kg/d (Table 2), and site of infusion had no effect. Because no control treatment involving the infusion of water was tested, inhibition of feed intake because of liquid infusion per se cannot be ruled out. Inhibition of feed intake by the flexible disk in the abomasum (i.e., impaired outflow from the rumino-reticulum) was also possible. However, the similarity in feed intake between the abomasally infused cows (with the flexible disk) and the ruminally infused cows (no disk) suggests that this inhibition did not occur.

Calculated intake (voluntary feed intake + infused starch) of digestible energy for cows infused with starch increased by 5.5% compared with the intake of

cows on the control treatment. Calculation of net energy balance (28) indicated, that despite their high milk yield, cows on the control treatment were in positive calculated energy balance (+1.9 Mcal/d, or 5% above requirements). When starch was infused, the NE_L intake was 9% greater than requirements. Although positive energy balance was unexpected in early lactation cows, the basal diet was a high starch, energy dense diet. The accuracy of any calculation of energy balance is a function of the accuracy of estimates of NE_L content of dietary ingredients and NE_L requirements.

Digestibility of DM (Table 2) appears low relative to our data from cows fed similar diets (21), possibly because of the use of lignin as a marker. The digestibilities of NDF and N in the current study were within the range of published data from cows fed similar diets (21). In the current study, no effects of treatment were noted for total tract digestibility of NDF, N, or DM (Table 2). Digestibility of starch in the total tract tended to increase ($P < 0.14$) when starch was infused because of the replacement of a portion of the dietary DM with highly digestible, infused starch.

Concentrations of Hormones and Metabolites

The mean insulin concentration increased when starch was infused (Table 3). Similarly, intravenous (2) and duodenal (23) infusion of glucose has been

TABLE 2. Feed and energy intake and nutrient digestibility of four early lactation cows infused with 1500 g/d of starch hydrolysate for 2 wk in the rumen, or the abomasum or not infused.¹

	Infusion site			SE	Contrast		
	None	Abomasum	Rumen		Infusion	Site	
						— $P <^2$ —	
DMI, ³ kg/d	22.5	21.8	21.7	0.170	0.03	0.77	
Calculated energy intake, including infused starch ⁴							
Digestible energy intake, mcal/d	76.0	80.3	80.1	0.573	0.004	0.77	
NE_L Intake, mcal/d	39.6	42.2	42.1	0.298	0.002	0.77	
Total tract NDF digestibility, %	29.3	23.2	25.9	3.82	0.35	0.63	
Total tract N digestibility, %	56.6	58.8	61.9	2.42	0.27	0.41	
Total tract starch digestibility, ⁵ %	92.2	93.1	95.1	0.873	0.14	0.19	
Total tract DM digestibility, %	53.2	54.5	58.9	2.58	0.32	0.28	

¹Data presented are least squares means (n = 4).

²Probability that effect was not different.

³The DMI does not include infused starch.

⁴Calculated dietary digestible energy = 3.38 Mcal/kg, starch digestible energy = 4.41 Mcal/kg. Calculated dietary NE_L = 1.76 Mcal/kg, starch NE_L = 2.54 Mcal/kg.

⁵Total tract starch and DM digestibility calculations include infused starch.

TABLE 3. Serum hormone and metabolite concentrations of four early lactation cows infused with 1500 g/d of starch hydrolysate for 2 wk in the rumen, or the abomasum or not infused.¹

	Infusion site			SE	Contrast		
	None	Abomasum	Rumen		Infusion	Site	
						— <i>P</i> < ² —	
Insulin, ng/ml	0.315	0.531	0.427	0.047	0.05	0.18	
Glucagon, pg/ml	89.6	80.7	92.2	3.98	0.55	0.11	
Growth hormone, ng/ml	8.87	8.91	10.0	0.683	0.51	0.31	
Plasma glucose, mM	3.54	3.77	3.68	0.205	0.49	0.75	
BHBA, mg/dl	8.21	6.55	7.37	0.628	0.17	0.40	
NEFA, μ eq/L	315	198	269	29.1	0.08	0.15	

¹Data presented are least squares means (n = 4).

²Probability that effect was not different.

shown to increase plasma insulin concentrations in lactating cows. Glucagon concentration tended to increase with ruminal infusion versus abomasal infusion. The increase in glucagon with starch that was infused ruminally might have been in response to increased propionate production and absorption (33), which would stimulate glycogen hydrolysis and gluconeogenesis. Growth hormone concentrations were not affected by treatment.

Infusion of starch tended to decrease NEFA concentrations, and abomasal infusion tended to decrease NEFA more than ruminal infusion of starch (Table 3). Decreased NEFA concentrations indicate decreased mobilization of adipose reserves, which is in agreement with the increased energy balance noted when starch was infused. The difference between abomasal and ruminal infusion suggests a difference in peripheral energy metabolism with these two treatments. In support of this hypothesis, lactating cows infused duodenally with glucose had decreased NEFA concentrations (23) relative to control cows with similar NE_L intake and NE_L balance. Lemosquet et al. (23) suggested that lipolysis was inhibited with postruminal glucose infusion, regardless of energy balance.

Neither plasma glucose concentration nor BHBA concentration was affected by treatment. Plasma glucose was similarly unaffected by short-term intravenous glucose infusion in cows that were 60 to 90 d postpartum, although BHBA concentration decreased (2).

Glucose Kinetics

Glucose metabolism may be evaluated through changes in glucose ILR, a measure of glucose leaving the body through oxidation, or lactose synthesis per unit of time. The ILR is measured through isotope

dilution techniques, and, assuming steady-state conditions, equals the glucose absorbed from the digestive tract plus hepatic synthesis. In ruminants, hepatic production of glucose accounts for most of glucose irreversible loss (85% in sheep). Exceptions include a small quantity produced by the kidney (4 to 15% in dogs and humans and 12% in sheep), and reconversion of labeled lactate to glucose in the tissues (9, 10). Primary utilization of glucose in lactating cows is by the mammary gland, with significant utilization also by the portal-drained viscera, the brain and nervous system, and the fetus in pregnant cows. The ILR increases during late pregnancy and lactation and is reduced by lack of feed.

In the current study, glucose ILR tended to increase when starch was infused abomasally compared with ILR when starch was infused ruminally ($P < 0.11$; Table 4). When starch was infused ruminally, the ILR was similar to the ILR of cows on the control treatment. These glucose ILR are somewhat higher than those in other studies with lactating cows (2, 8), but DMI and milk yield of the cows used in the current experiment are higher also.

Carbon dioxide derived from glucose was significantly lower when starch was infused ruminally than when cows were infused abomasally (Table 4). Abomasal infusion of starch appeared to increase the percentage of CO₂ derived from glucose relative to the control treatment, and ruminal infusion of starch appeared to decrease it. Blood CO₂ derived from blood glucose is calculated based on the specific activity of blood glucose and blood CO₂. Oxidation of luminal (unlabeled) glucose by the intestinal mucosa would not contribute to this calculation. In this study, therefore, CO₂ derived from blood glucose after cows were infused with starch in the abomasum is underestimated by the amount of CO₂ derived from oxidation of luminal glucose.

TABLE 4. Glucose kinetics of four early lactation cows infused with 1500 g/d of starch hydrolysate for 2 wk in the rumen or the abomasum or not infused.¹

	Infusion site			SE	Contrast	
	None	Abomasum	Rumen		Infusion	Site
Irreversible loss rate						
mol/d	13.81	16.77	13.78	1.07	0.31	0.11
g/d	2485	3019	2481	192	0.31	0.11
mg/min per kg of MBS ³	15.60	18.93	15.65	1.14	0.31	0.11
Blood CO ₂ from glucose, %	5.40	7.71	3.99	0.318	0.54	0.001

¹Data presented are least squares means (n = 4).

²Probability that effect was not different.

³Metabolic body size = BW^{0.75}.

Starch infusion in the abomasum. The magnitude of the increase in glucose ILR when starch was infused abomasally (+2.96 mol/d of glucose) was 32% of the quantity of exogenous glucose supplied through starch infusion. The infused carbon that did not contribute to glucose ILR might have been lost through limited digestion in the small intestine, limited glucose absorption from the small intestine, increased utilization of luminal glucose by the gut tissue, decreased hepatic gluconeogenesis, or a combination of these factors.

The digestibility of starch in the small intestine has been observed to decrease as duodenal starch flow increases. Infusion of raw corn starch into the abomasum of ileally cannulated steers at 0, 20, 40, and 60 g/h increased intestinal starch disappearance quadratically; disappearance approached a maximal value at the highest level of infusion (22). Starch disappearance was 89% of infused starch at 20 g/h and 58% of infused starch at 60 g/h. There is evidence that digestibility in the small intestine is also affected by the structure of starch that reaches the duodenum. Kreikemeier et al. (22) also infused starch that was partially hydrolyzed to destroy the granular structure (corn dextrin). Disappearance of dextrin from the small intestine was greater at all levels of infusion than was disappearance of raw corn starch, suggesting that the crystalline structure of raw starch inhibits digestion in the small intestine (22). Similarly, starch disappearance from the small intestine, expressed both in grams per day and as a fraction of duodenal flow, increased in lactating cows fed high moisture corn (starch partially gelatinized during ensiling) compared with that in lactating cows fed dry corn despite lower duodenal flow with this treatment (21).

In the current study, the infused starch solution was treated with amylase and heated prior to infu-

sion to gelatinize and partially hydrolyze the starch (7), increasing its potential digestibility within the small intestine. Hind gut fermentation is another possible (but not measured) fate of starch infused to the small intestine in this experiment. With the high total tract starch digestibility observed with all three treatments and the pretreatment of the infused starch, postruminal digestion of the infused starch probably did not limit glucose ILR in this study.

Infused carbon might have been lost through limited glucose absorption from the small intestine. The net flux of glucose across the portal-drained viscera (gastric and intestinal tissues) of ruminants is generally negative when high forage diets are fed (20, 29), indicating utilization of arterial glucose greater than absorption of glucose from the intestine. Postruminal infusion of starch generally increases glucose absorption (20, 22), but, in studies that measured both glucose absorption and starch disappearance from the small intestine (20, 22, 31), only 30 to 40% of the starch that disappeared from the small intestine could be accounted for as glucose appearing in the portal vein.

Increased utilization of glucose in gut tissue as the supply of glucose increased has been observed in sheep with glucose infusion in the mesenteric vein (17). Intestinal tissues might oxidize both arterial glucose and luminal glucose. Luminal glucose oxidized by the small intestine epithelium never enters circulation and, therefore, does not contribute to glucose ILR. Increased utilization of luminal glucose could help to explain the limited contribution of infused starch to glucose ILR when starch was infused into the abomasum in this study. The percentage of CO₂ derived from glucose was increased when starch was infused into the abomasum (Table 4). This increased oxidation of glucose to CO₂ pertains to glucose that was measured as ILR (i.e., blood glucose, not luminal glucose) and is discussed subsequently.

TABLE 5. Milk yield and composition of four early lactation cows infused with 1500 g/d of starch hydrolysate for 2 wk in the rumen or the abomasum or not infused.¹

	Infusion site				Contrast	
	None	Abomasum	Rumen	SE	Infusion	Site
	$P <^2$					
Milk yield, kg/d	40.3	42.1	42.4	0.659	0.07	0.82
Milk fat						
%	3.78	3.73	3.63	0.119	0.52	0.55
kg/d	1.52	1.57	1.53	0.044	0.66	0.51
Milk protein						
%	2.64	2.73	2.60	0.049	0.70	0.14
kg/d	1.06	1.14	1.10	0.030	0.16	0.41
Milk lactose						
%	4.74	4.82	4.70	0.042	0.75	0.13
kg/d	1.91	2.02	1.99	0.044	0.14	0.66
SNF						
%	8.10	8.26	8.02	0.063	0.66	0.06
kg/d	3.26	3.46	3.40	0.075	0.13	0.58

¹Data presented are least squares means (n = 4).

²Probability that effect was not different.

Decreased synthesis of endogenous glucose by the liver is a final explanation for the limited contribution of starch that was infused into the abomasum to glucose ILR. Decreased hepatic gluconeogenesis has been observed for animals with increased exogenous glucose from abomasal or duodenal infusion (6, 11) or intravenous infusion (6, 17). A study (13) using the euglycemic, hyperinsulinemic clamp procedure in steers indicated that insulin release stimulated by glucose rather than glucose per se inhibited gluconeogenesis in response to increased exogenous glucose. Insulin concentration did increase in our study when starch was infused, and so decreased hepatic gluconeogenesis is plausible.

Limitations to starch digestion and glucose absorption in the small intestine, preferential utilization of glucose by the gut tissues, and decreased endogenous glucose synthesis may all limit the contribution of postruminally digested starch to peripheral glucose supply. Despite these potential limits, increased post-ruminal starch digestion did cause some increase in glucose ILR, and increased ruminal starch digestion did not.

Starch infusion in the rumen. Ruminal starch infusion likely increased production of ruminal propionate as well as the production of other VFA, CO₂, and CH₄, but this increase in ruminal propionate was not enough to contribute measurably to the whole body glucose supply. An increase in ruminal propionate production can be predicted using estimates of fermentation stoichiometry. In a mixed diet, Baldwin et al. (5) predicted that fermentation of 1 mol of

soluble carbohydrate would yield 1.14 mol of acetate, 0.43 mol of propionate, and 0.215 mol of butyrate. With the infusion of 1500 g/d of starch, the equivalent of 9.25 mol/d of glucose, the predicted increase in propionate production would be 3.98 mol/d. Utilization of ruminal propionate for hepatic glucose synthesis averaged 36.6% in lactating cows infused intravenously with glucose (2), which is similar to the fraction observed in steers (37). Assuming this utilization rate in our study and allowing 2 mol of propionate for the synthesis of 1 mol of glucose, an increase in glucose ILR of 0.73 mol/d would be predicted from the ruminal infusion of 1.5 kg/d of starch. An increase of this magnitude would be undetectable with our experimental design and the number of cows used.

The effects of increased propionate production on its utilization for glucose synthesis or on its oxidation would also limit the contribution of ruminally digested starch to glucose supply. Propionate utilization for glucose synthesis is linearly related to its concentration in plasma. The absolute amount of propionate utilized for glucose synthesis increases as propionate absorption or intravenous infusion increased, but the fraction of propionate utilized for glucose synthesis is constant under various nutritional conditions (2, 37). Therefore, although propionate production almost certainly increased, the fraction of propionate utilized for glucose synthesis was not a likely source of variation in this study.

The decrease in glucose oxidation as starch was infused into the rumen (Table 4) implied that oxidation of other metabolites increased, sparing glucose.

An increase in oxidation of propionate by peripheral tissues as propionate supply increased has been observed (37). In growing steers fed propionate, 74% of ruminal propionate was oxidized compared with 65% for control steers (37). Carbon dioxide derived from propionate oxidation nearly tripled in steers fed propionate compared with levels in control steers.

Milk Yield and Composition

The infusion of starch tended to increase milk yield by 2 kg/d and to increase milk lactose yield by 95 g/d (Table 5). The site of starch infusion did not affect these measurements. The infusion of starch in the abomasum, but not in the rumen, tended to increase milk lactose concentration. Milk protein yield was not affected by treatment, but milk protein concentration tended to increase when starch was infused abomasally.

Starch infusion in the abomasum. Increases in milk and milk lactose yields when glucose was infused abomasally or intravenously have been reported but not universally observed. Frobish and Davis (18) reported an increase in milk yield of 1.9 kg/d in midlactation cows that were infused abomasally with 2150 g/d of glucose for 5 d. Milk lactose concentrations were not reported, but, if they averaged 4.8%, milk lactose output increased by 91 g/d when glucose was infused abomasally. In another study (16), jugular infusion of 500 or 700 g/d of glucose for 4 d increased milk yield by 0.9 and 1.5 kg/d, respectively, and milk lactose yield increased by 40 and 90 g/d. Intravenous infusion of glucose in goats deprived of feed increased milk yield by 62% and milk lactose by 87% within 3 h and increased milk yield in fed goats by 30% (24). In those studies (24), the increase in milk lactose yield averaged 5 to 15% of the glucose infused.

Studies of lactating cows involving longer term infusions indicate that milk yield responses to exogenous glucose are not sustained. Jugular infusion of 342 or 737 g/d of glucose for 11 d had no effect on milk yield or composition of cows at peak lactation (2). Abomasal infusion of 1000 g/d of glucose for 4 wk in very early lactation cows that were fed an all alfalfa silage diet decreased DMI and had no effect on milk yield (12). Infusion of glucose plus 1200 g/d of soy protein increased milk yield to a greater extent than did infusion of soy protein alone, indicating an interaction between protein flow to the small intestine and glucose absorption or utilization by the mammary gland (12). Abomasal infusion of 450 g/d of glucose to

midlactation cows for 12 d had no effect on milk yield, although a similar infusion of casein increased milk yield (11). Milk yield was also unaffected in lactating cows infused duodenally with 1500 g/d of glucose for 12 d when energy and protein intakes were equalized in both infused and control cows (23).

Duration of infusion appears to be correlated with milk yield response in those studies. In studies in which glucose supply was altered by short-term (<1 wk) infusion of glucose (6, 16, 18, 24), milk yield increased. Longer-term infusion studies (2, 11, 12), however, indicate no prolonged response. As the supply of exogenous glucose increased over a longer period, cows in those studies appeared to adapt metabolically to increased storage of glucose, glucose oxidation, or utilization of glucose by other tissues; yields of milk and lactose were not increased.

Our study with early lactation, high yielding cows is among the first to indicate prolonged increases in milk yield as the exogenous glucose supply increased. The amount of exogenous glucose supplied (9.25 mol/d) was higher in our study than in many others, and the cows in our study yielded greater quantities of milk than did cows in other studies. When starch was infused into the abomasum, the likely mechanism of increased milk yield was increased availability of glucose for lactose synthesis. However, the increase in milk lactose yield (+110 g/d) was just 6.6% of the glucose equivalents infused, which accounts for just 21% of the increased glucose ILR from abomasal infusion. One recent study showed somewhat similar results. Abomasal infusion of wheat starch (1200 g/d) to late lactation cows did increase milk yield, although ruminal infusion had no effect (31). Milk lactose yield from that study was not reported.

Elliot (14) summarized several studies and observed that mammary glucose uptake as a percentage of total glucose entry increased as milk yield increased. Observing that this relationship must become asymptotic as 100% of glucose entry is approached, Elliot predicted that, for cows yielding >30 kg/d of milk, all available glucose would be used by the mammary gland. In this study with early lactation cows yielding >40 kg/d of milk, glucose ILR increased by 534 g/d when starch was infused abomasally, and the observed increase in milk lactose secretion was 110 g/d. Milk lactose secretion generally accounts for about 70% of glucose taken up by the mammary gland (3). The remaining glucose taken up is either oxidized or is used for the synthesis of citrate, milk proteins, and glycerol in milk fat (3). Because of this relationship between glucose uptake

and lactose secretion, the increase in mammary glucose uptake when starch was infused abomasally in this study can be estimated at 160 g/d (110 g/d of lactose/0.7 = 160 g/d). Mammary glucose uptake of 160 g/d accounts for 30% of the increase in glucose ILR. Increased oxidation by peripheral tissues (primarily brain and viscera) was the apparent fate of the remaining 70% of this glucose.

In this study, the percentage of CO₂ derived from glucose increased when glucose ILR increased with abomasal infusion of starch. Other studies have indicated that, when glucose supply increases, glucose is preferentially used for oxidation by many tissues in the body. Jugular or duodenal infusion of glucose in lactating cows increased oxidation of glucose (6), and the fraction of respiratory CO₂ derived from plasma glucose increased from 10 to 13% to nearly 23%. Intravenous infusion of glucose increased the fraction of blood CO₂ derived from plasma glucose in lactating cows from 4.1 to 6.8% in another study (2). Glucose ILR increased by 58% in steers fed 6.1 mol/d of propionate (37), but nearly all of the additional glucose was oxidized. Conversely, with an increased demand for glucose for milk lactose synthesis with bST treatment, CO₂ derived from oxidation of glucose declined from 4.7 to 3.8% (8). In the current study, it is likely that increased oxidation was the fate of much of the increase in glucose ILR when starch was infused abomasally.

Milk protein concentration tended to increase more in cows infused with starch abomasally than in cows infused with starch ruminally (Table 5). Milk protein concentration also increased in lactating cows infused duodenally with 1500 g/d of glucose for 12 d despite equalized energy and protein intakes by both infused and control cows (23). This increase in milk protein concentration might have been due to the sparing of amino acids through preferential utilization of glucose by the gut tissue and decreased hepatic gluconeogenesis. An increase in glucose utilization by gut tissues when starch was infused into the abomasum did limit the contribution of intestinally absorbed glucose to glucose ILR as discussed previously, but also spared amino acids for protein synthesis and VFA for oxidation by peripheral tissues. Similarly, decreased hepatic gluconeogenesis, although a factor that limits the increase in glucose ILR when starch is infused abomasally, would also have the effect of sparing gluconeogenic precursors, including amino acids. The sparing of gluconeogenic amino acids and a subsequent increase in protein supply to the mammary gland might also have been partly responsible for the increase in milk yield observed with abomasal infusion.

Alternatively, the increase in milk protein concentration when starch was infused abomasally might have been due to the increased insulin concentration observed. Recent work (19) demonstrated that chronic elevation of insulin with the hyperinsulinemic, euglycemic clamp protocol combined with abomasal infusion of casein increased milk protein concentration dramatically in lactating cows. Similarly, the increase in milk protein concentration reported (23) for lactating cows duodenally infused with glucose might have been due to the increased basal insulin concentration observed in that study.

Starch infusion in the rumen. The increased milk yield when starch was infused ruminally compared with that from the control treatment might have been due to increased substrate supply or increased availability of energy substrates for oxidation. Because glucose ILR was not measurably increased when starch was infused ruminally, the supply of glucose for lactose synthesis was not a mechanism for increased milk yield. An increase in the supply of absorbed protein is one alternative mechanism, because ruminal infusion of starch likely increased microbial protein synthesis. However, if the increased microbial protein yield from the ruminal infusion of starch caused the increased milk yield, we would expect an increase in milk protein yield with ruminal infusion compared to abomasal infusion, which did not occur (Table 5).

The increase in milk yield when starch was infused ruminally was most likely due to increased energy supply. Starch infusion increased calculated net energy intake by 6.4% over that of the control treatment (Table 2). This increase in energy supply decreased the amount of adipose tissue mobilized in these early lactation cows (indicated by the observed decrease in NEFA concentration when starch was infused ruminally) and is the most logical explanation for the increase in milk yield with this treatment.

CONCLUSIONS

Starch infusion supported sustained increases in milk yield in early lactation cows yielding >40 kg/d of milk. Similar increases in milk yield were observed with both abomasal and ruminal infusion of starch, but the causes of the increase might have differed. When starch was ruminally infused, increased energy supply was the most likely explanation for the increased milk yield. Ruminally infused starch increased neither glucose ILR nor milk protein yield but did decrease glucose oxidation. When starch was infused in the abomasum, increased glucose supply was

a likely cause of the increase in milk yield. Sparing of gluconeogenic amino acids and increased energy availability might have contributed to this increase as well. Starch infused to the abomasum of these early lactation cows made a greater contribution to peripheral glucose supply than did starch infused to the rumen, supporting the contention of increased efficiency of intestinally absorbed glucose (29). Even so, only 32% of infused glucose could be accounted for as increased ILR, implying limited intestinal glucose absorption, increased gut tissue glucose oxidation, or decreased hepatic gluconeogenesis when starch was infused abomasally. Once absorbed, increased oxidation by peripheral tissues was the apparent fate of much of the increased glucose supply in cows infused abomasally, because increases in milk lactose yield were small relative to the increase in glucose ILR. Results indicate that the dairy cow has a greater capacity for glucose metabolism in early lactation than is provided by feed intake of typical diets but that not all available glucose is partitioned to the mammary gland.

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