

Metabolic Fate of Products of Starch Digestion and Absorption in Beef and Dairy Cattle

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Summary:

- Gluconeogenesis is the principal route of glucose supply for ruminants
- Several products of starch digestion and absorption are key carbon sources for gluconeogenesis
- Propionate
- Lactate
- Amino acids from microbial protein
- Metabolic requirements for glucose are linked to production demands and priorities; they can be estimated or calculated as a function of energy intake across a wide range of body weights, breeds, or productive purposes
- Propionate use for gluconeogenesis in the liver is insulin-independent
- Increased propionate production in the rumen will increase gluconeogenesis, but there has to be a concomitant demand, or “pull” to avoid oxidation of increased glucose production
- Glucose use can be divided into two main categories: constitutive use, which is independent of control by the hormone insulin, and insulin-dependent use
 - ◊ Approximately 10% of total glucose use is constitutive use by the brain and other nervous tissues
 - ◊ The lactating mammary gland has insulin-independent glucose uptake but insulin affects uptake of other milk precursors
- Capacity of insulin to enhance glucose use varies with age, body composition, nutritional status, and productive state
- Shifting the site of starch digestion from the rumen to the intestine changes the profile of absorbed compounds, but it does not necessarily increase overall metabolizable energy supply absorbed to support carcass growth or milk production

Introduction:

The scientific community has come full circle on the function and importance of glucose for the metabolic processes of ruminants. Before the 1940's, ruminants were presumed to use glucose as a major energy substrate, like other mammals (Mephram, 1993; Annison and Bryden, 1999). The discovery of absorption of short-chain fatty acids and the use of acetate as a lipid precursor, linked with information that showed ruminants use gluconeogenesis to meet glucose needs, led experts to conclude that glucose metabolism is not particularly important for ruminants. The advent of high-grain, high-energy diets to improved feed and milk efficiencies has been key to economic success of modern feedlots and dairies and has renewed interest in glucose metabolism, because starch is a major energy component in those diets. We now know that starch digestion and absorption provide fermentation products (lactate, amino acids, nucleic acids, short-chain fatty acids) as well as glucose required for metabolic processes; many of the fermentation products support gluconeogenesis after they are absorbed from the gut (Ortigue-Marty et al., 2003). We also know that gluconeogenesis ensures that high-producing beef and dairy cattle are rarely, if ever, deficient in glucose supply.

In order to make sound nutrition and management decisions, nutritionists need to evaluate and incorporate a wide variety of available information. A good understanding of the metabolic purpose of absorbed products is an important component of “available information”. The premise of this review is that metabolic priorities control fate of these products of digestion. Main metabolic processes and products will be discussed in relation to gluconeogenesis. Quantitative scenarios of glucose supply and use will be presented for a lactating dairy cow and a growing beef steer.

Gluconeogenesis

Relationship to Energy Intake

Ruminants derive 25% or less of their glucose supply directly as the absorbed product of amylolytic starch digestion in the gut, so gluconeogenesis is the principal route of glucose supply for ruminants (Huntington, 1997). Metabolic requirements for glucose are linked to production demands and priorities; they can be estimated or calculated as a function of energy intake (ME) across a wide range of body weights, breeds, or productive purposes, at least in cattle (Herbein et al., 1978; Russell et al., 1986; Wiegart et al., 1986). **Figure 1** shows three linear regression lines that represent glucose and propionate metabolism, expressed as grams of carbon per day, as a function of ME intake of beef and dairy cattle; one line represents glucose irreversible loss from 32 data points (Herbein et al., 1978; Schmidt and Keith, 1983; Lyle et al., 1984; Armentano et al., 1984; Bauman et al., 1988; Veenhuizen et al., 1988; Amaral et al., 1990; Knowlton et al., 1998; Richards, 1999; Rigout et al., 2002; Lemosquet et al., 2003). Glucose irreversible loss represents glucose that is used by the animal and will not be recycled (for example, excretion as milk lactose, or exhalation as CO_2). The second line represents liver gluconeogenesis from 49 data points (Baird et al., 1980; Lomax and Baird, 1983; Harmon et al., 1991, 1993; Krehbiel et al., 1992; Casse et al., 1994; Eisemann et al., 1996; Reynolds et al., 1988, 1992, 1994,

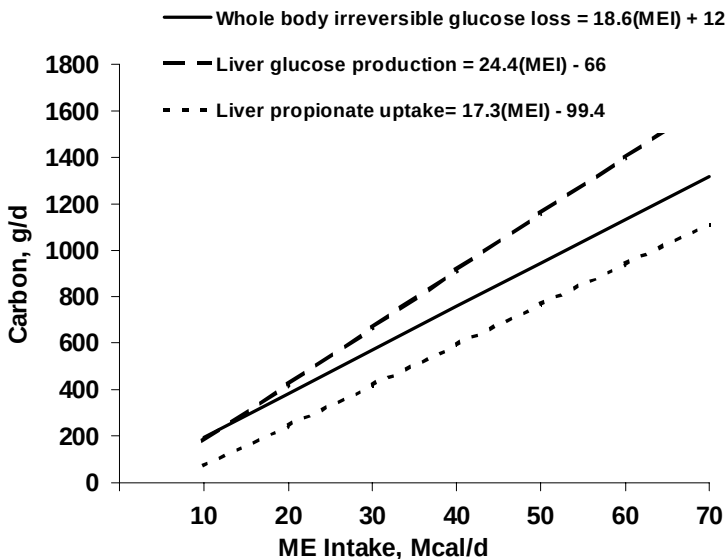


Figure 1. Regression of glucose whole body irreversible loss, liver glucose production and liver propionate uptake (carbon, g/d) against metabolizable energy (ME) intake from previously published literature.

2003; Eisemann and Huntington, 1994; Taniguchi et al., 1995; Huntington et al., 1996; DeVisser et al., 1997; Lozano et al., 2000; Benson et al., 2002). The third line represents propionate's contribution to liver gluconeogenesis from the same database used for liver gluconeogenesis. Coefficients of correlation (R^2) are 0.92 for regression of irreversible loss on ME intake, 0.93 for regression of liver gluconeogenesis on ME intake, and 0.93 for liver propionate uptake. Therefore, these relationships of glucose metabolism with ME intake can explain most of the variation in the data sets across a variety of body weights, diet composition, and production priorities. Of course, many sources of variation are reflected in ME intake; for example, the data points for high ME intake came from large, lactating, Holstein cows. Furthermore, the most likely way to increase ME intake is to increase energy density of the diet by including grain or other starch-containing feedstuffs.

Most published information supports the general concept that gluconeogenesis is a positive function of energy intake. Intensive work with ruminal or duodenal infusion of glucose or propionate into lactating dairy cows (Rigout et al., 2002; Lemosquet et al., 2003) and work with hydrolyzed starch infusion into beef steers (Richards, 1999) suggests direct use of absorbed glucose by visceral tissues is equal to 28% of glucose entering the small intestine. In our view, the work with lactating cows cited above shows similar response in glucose irreversible loss to duodenal infusion of glucose or to ruminal infusion of propionate. Almost none of the published information specifies or discriminates among dietary sources of energy; however, extensive analysis of data from cattle and sheep indicates that the presence of corn grain in the diet will increase gluconeogenesis more than isoenergetic diets that do not contain corn (Ortigue-Marty et al., 2003). This underlines the fact that ruminants will continue to synthesize glucose even when they are provided a diet rich in starch, largely through use of glucogenic ruminal fermentation products. The relationship between liver propionate uptake and liver glucose production exemplifies the link between precursor supply to the liver and glucose production. Figure 1 also indicates that propionate does not supply all the carbon for liver glucose production or whole body irreversible glucose loss.

Glucose Precursors

Several products of starch digestion and absorption are key carbon sources for gluconeogenesis. Propionate is quantitatively most important; the slopes of lines in Figure 1 indicate that propionate uptake by the liver accounts for about 70% (100 times 17.3 divided by 24.4) of liver glucose production across a range of ME intakes. Propionate is followed by L-lactate and glucogenic amino acids (**Table 1**). Propionate and lactate are direct fermentation products, and amino acids are, at least in part, from microbial protein. However, amino acids and lactate may arrive in the liver as immediate products of the Cori cycle (lactate to and from glucose in the gut or peripheral tissues) or deamination or transamination of glucogenic amino acids (again in the gut or peripheral tissues). Because data in Table 1 represent maximal theoretical calculations based on one glucose unit produced from 2 units of the precursor, the sums may be greater than 100%. Transition dairy cows likely use relatively more lactate and glycerol and less propionate to support gluconeogenesis immediately before and after calving (Reynolds et al., 2003), because they mobilize body tissues to support the onset of lactation.

Table 1. Liver glucose production and maximal theoretical contribution of propionate, L-lactate, and amino acids to liver glucose production in cattle^a.

	Beef steers				Lactating dairy cows	
		1.70-	0.69-	1.12-	3.08 -	
Liver glucose production, kg/d	0.78	2.59	1.18	1.67	3.11	3.6
Contribution to glucose, %						
Propionate	73	48-64	60-77	43-54	55-58	66
L-lactate	13	26-36	16-35	16-20	18-21	8
Amino acids	12	16-30	11-28	17-22	15-17	
Alanine						2
Glycerol						0.4

^aData from Reynolds et al., 1988, 2003; Huntington, 1990; Taniguchi et al., 1995; Eisemann et al., 1996; Lozano et al., 2000.

The studies summarized in Table 1 make two points about gluconeogenesis in beef steers. First, as steers get older and fatter, the proportional contribution of propionate to gluconeogenesis became lower in favor of increased contribution of gluconeogenic amino acids (Eisemann et al., 1996). Second, in steers fed high concentrate, flaked sorghum grain diets, decreasing flake density (increased ruminal fermentation rate) increased supply of propionate, which was directly linked to increased gluconeogenesis (Lozano et al., 2000). Studies of the effect of insulin on liver glucose production show that increased insulin supply will reduce liver glucose production to the point where all glucose production could be derived from propionate removal by the liver (Eisemann and Huntington, 1994). This implies that propionate use for gluconeogenesis is independent of insulin's control, and that there is a high metabolic priority for the liver to remove propionate from blood, and use it to satisfy the need for glucogenic precursors. Insulin-independent use of propionate for gluconeogenesis also supports the observations that high starch diets (corn, wheat, or sorghum) that promote production of propionate in the rumen likely will promote higher rates of gluconeogenesis. The relative insensitivity to control by insulin also implies that use of ionophores or other feed additives that promote propionate production should spare other glucose precursors, such as glycerol or glucogenic amino acids.

Glucose Use

Glucose use in beef and dairy cattle is similar in rates and fates of glucose use in nonruminants. Glucose is transported from blood by almost all tissues to be oxidized for production of ATP, to provide carbon chains for production of glycerol needed for production of esterified fatty acids, for production of fatty acids in muscle, and for production of glycogen in the liver or muscle (Ortigue-Marty et al., 2003). Glucose use can be divided into two main categories: constitutive use, which is independent of control by the hormone insulin, and insulin-dependent use. Approximately 10% of total glucose use is constitutive use by the brain and other nervous tissues (Brockman, 1993), which reflects the high priority given to the function of those tissues. The lactating mammary gland also has insulin-independent glucose uptake (Nielsen et al., 2001), but the situation is complicated by the fact that insulin does affect uptake of other milk precursors, which may in turn increase glucose use by the mammary gland. Milk production by the mammary gland is positively related to the rate of glucose supply in blood perfusing the gland with glucose being the sole precursor of lactose synthesis (Mephram, 1993; Rigout et al., 2002). Insulin enhances glucose uptake by muscle and adipose tissue of beef and dairy cattle, which in turn increases use of glucose by those tissues for oxidation or synthetic pathways.

The capacity, or effectiveness, of insulin to enhance glucose use varies with age, body composition, nutritional status, and productive state of the animal; in simple terms, insulin helps ensure that the nervous system has priority when glucose and(or) total energy supply is low, by not promoting uptake of glucose by insulin-sensitive tissues. It also appears that cattle, like humans and other species, develop increased resistance to insulin's effect as they age, and increase in percentage of body tissue as fat (Sano et al., 1991; Eisemann et al., 1997). Extensive analysis of the relationship between glucose irreversible loss and energy intake in sheep and cattle indicates decreased response in adults compared with younger animals (Ortigue-Marty et al., 2003), which is consistent with increased resistance to insulin with increased age. This resistance to insulin's ability to promote glucose absorption by adipose tissue likely has some negative effect on overall feed efficiency of finishing feedlot cattle.

Heat stress, cold stress, and other stressors increase blood concentrations of glucocorticoids and cause changes in glucose use by ruminants. Elevated levels of glucocorticoids promote glycogenolysis and gluconeogenesis, and they also appear to inhibit insulin's effects on glucose uptake, which is consistent with decreased efficiency of energy use in stressed animals.

Recycling or Oxidation of Glucose

In addition to exemplifying the relationship between energy intake and glucose metabolism in cattle, Figure 1 also sheds some light on the recycling of glucose in response to production needs. If liver glucose production (liver gluconeogenesis) represents 75% or more of glucose entry rate (the sum of glucose that enters the bloodstream from all sources; Brockman, 1993) and irreversible loss represents permanent removal of glucose, then the space between the liver glucose production and glucose irreversible loss lines in Figure 1 represents additions to glucose supply from the kidneys or tissues other than the liver, glucose absorption from the gut, and glucose recycling within the animal's body. Possible avenues of recycling include glycogen production and glycogenolysis in the liver or muscle, movement of carbon through synthesis and catabolism of glucogenic amino acids, or movement of carbon through lipid metabolism in the form of glycerol. Therefore, the energy delivered by high starch diets moves through many metabolites to meet needs in carbohydrate, protein, and lipid metabolism. Those metabolites may be the result of ruminal fermentation (propionate, lactate, amino acids) or may emanate from pathways of intermediary metabolism involved in recycling of glucose carbon. Glucose oxidation to CO_2 in growing Holstein steers consuming a 30% concentrate diet (44.4% of irreversible loss, Veenhuizen et al., 1988) and lactating dairy cows that need glucose for milk lactose synthesis (17.2% of irreversible loss, Bauman et al., 1988) shows that ruminants have more than adequate gluconeogenic capacity to meet glucose needs as well as to meet other requirements for metabolic balance, or homeostasis. The growing gap between liver gluconeogenesis and irreversible loss at higher ME intakes (Figure 1) indicates that glucose supply from sources other than gluconeogenesis in the liver are metabolically more significant in lactating dairy cows than in growing animals.

Veenhuizen et al. (1988) fed steers 600 g/d of sodium propionate and increased gluconeogenesis from propionate, increased irreversible loss of glucose by 59%, increased oxidation of glucose to CO_2 , and increased the percentage of CO_2 supplied by oxidation of glucose from 7.8 to 13.1%. Increased oxidation of glucose to CO_2 accounted for essentially all of the increased irreversible loss of glucose by the steers. Amaral et al. (1990) increased

glucose supply for lactating cows by intravenous infusion of glucose (up to 737 g/d) and increased milk production by 6% (not statistically significant), increased irreversible loss of glucose by 53%, decreased gluconeogenesis from propionate, and increased the percentage of CO₂ supplied by oxidation of glucose from 4.1 to 6.8%. Knowlton et al. (1998) increased potential glucose supply in lactating cows by abomasal infusion of 1500 g/d of partially hydrolyzed starch and increased milk production by 5%, increased irreversible loss of glucose by 21%, and increased the percentage of CO₂ supplied by oxidation of glucose from 5.4 to 7%. Taken together, these results indicate that glucose supply was not a major limitation to growth or milk production in these studies, and that it is difficult to 'push' the metabolic system by enhancing glucose supply. Therefore, these intensive, metabolic studies tell us that ruminants are capable of synthesizing sufficient glucose for their needs, and will oxidize, or 'burn off' excess glucose that does not fit into their metabolic balance. However, there may be some benefit by sparing amino acids that otherwise would have been used to support gluconeogenesis. Propionate production in the rumen represents a relatively efficient capture of energy for subsequent production of glucose, but there has to be a concomitant demand, or "pull" to avoid oxidation of extra glucose.

Site of Starch Digestion Affects Glucose and Energy Metabolism

The regressions in **Figure 1** suggest that irreversible loss is based on ME intake across a wide range of production states and ration compositions. Using feed efficiencies and grain starch digestibilities from many trials, Owens et al. (1986) determined that if starch digestion in the small intestine exceeds 70% of its digestibility in the rumen, small intestinal digestion improves efficiency of energy capture from dietary sources. This estimate is similar to previous estimates by Armstrong et al. (1960) and Black (1971) using other methods. A second conclusion from Owens et al. (1986) is that starch digestion in the small intestine provides 42% more energy than starch digested in the rumen.

As would be expected, shifting starch digestion from the rumen to the small intestine increases portal-drained visceral (digestive tract, pancreas, spleen and mesenteric fat) glucose absorption (Taniguchi et al., 1995; Reynolds et al., 1998; Richards, 1999). Richards (1999) determined that shifting the site of digestion to the small intestine increased portal-drained visceral tissue use of glucose from 120 to 281 g/d. However, as indicated by oxygen consumption in beef steers (Richards, 1999) and lactating dairy cows (Reynolds et al. 1998), the increase in glucose use is not associated with an increase in energy use by the portal-drained visceral tissues indicating that other energy substrates are conserved. Taniguchi et al. (1995) only reported increases in portal-drained visceral acetate and valerate fluxes while Richards (1999) reported increases in butyrate and valerate with ruminal starch infusion where ruminal VFA production increases would be expected. When the heat of combustion estimates are applied to each of the energy substrates absorbed across and oxygen consumed by the portal-drained viscera, there was no difference due to site of starch digestion in either of the above experiments.

Despite differences in substrates absorbed and utilized by the portal-drained viscera, the supply of major precursors reaching the liver for gluconeogenesis were not affected by the site of starch digestion. Consequently, as noted previously, liver glucose production was similar for both sites of starch digestion in steers (Taniguchi et al., 1995; Richards, 1999). Taniguchi et al. (1995) reported metabolism in the liver of ruminally infused calves resulted in decreased glucose and lactate, but increased acetate and b-hydroxybutyrate releases to peripheral tissues, which is similar to the findings in Richards

(1999) except, in that experiment, acetate was higher with abomasal starch infusion. Both experiments infused the same quantity of starch, but Richards (1999) supplied a partially hydrolyzed form, which resulted in greater net glucose absorption. In lactating dairy cows, Reynolds et al. (1998) infused starch into the abomasum and increased net glucose absorption, but did not affect liver glucose production when compared to a water infusion. However, starch infusion into the rumen increased liver glucose production.

If we look at the total energy available for use by peripheral tissues (total splanchnic flux), there are the differences in substrates available as described above. Greater quantities of glucose available with postruminal starch digestion result in greater quantities of total glucose irreversible loss and peripheral tissue use by steers (Richards, 1999), and greater total glucose irreversible loss rates with lactating cows (Knowlton, et al., 1998). However, if we look at energy available for use by peripheral tissues, the data differ in quantities of glucose available from postruminal digestion between the experiments of Taniguchi et al. (1995) and Richards (1999). The quantity of acetate available for peripheral metabolism was greater when more glucose was absorbed postruminally, which would agree with the concept that in portal-drained viscera tissues, acetate use is associated primarily with oxidative metabolism rather than incorporation into mesenteric fat. Oxidation of acetate to CO₂ can represent up to 25% of the whole body acetate turnover on forage-based diets (Pethick et al., 1981). Bartley and Black (1966) and Knowlton et al. (1998) both discuss the possibility postruminal starch digestion resulting in preferential use of glucose by the portal-drained viscera tissues, thereby reserving amino acids and VFA for other functions. A numerical increase in acetate flux between a basal diet control and the postruminal starch infusion of Taniguchi et al. (1995) would indicate that the addition of glucose supply does result in some conservation of acetate. These differences resulted in 635 kcal/day greater peripheral energy availability for the ruminal treatment in Taniguchi et al. (1995) and 1759 kcal/d greater energy for the abomasal digestion in Richards (1999).

Table 2. Glucose and starch needs of a lactating dairy cow and finishing beef steer.

	Dairy cow	Beef steer
Body Weight, kg	700	400
Dry Matter Intake, kg/d	22	10
Milk yield, kg/d ^a	44	
Weight gain, kg/d		1.6
Metabolizable energy intake, Mcal/d	64	27
Liver gluconeogenesis, kg/d ^b	3.75	1.48
Glucose irreversible loss, kg/d ^b	2.73	1.20
Glucose oxidized to CO ₂ , kg/d ^c	0.47	0.61
Gluconeogenesis from blood supply of precursors, %		
Propionate ^b	67	62
Lactate	10	17
Amino acids, other precursors	23	21
Starch Intake to support glucose irreversible loss, kg/d ^d		
No ruminal fermentation of dietary starch	3.90	1.71
50% of dietary starch fermented in rumen	5.85	2.56
75% of dietary starch fermented in rumen	6.82	3.00

^aAssumes 80% of irreversible loss is milk lactose, and milk is 5% lactose by weight.

^bCalculated from regressions in Figure 1.

^cCalculated as 0.172 times irreversible loss for dairy cows (Bauman et al., 1988)

and 0.444 times irreversible loss for beef steers (Veenhuizen et al., 1988).

^dCalculated from the irreversible loss in this table and the following assumptions: 35% of carbon from starch digested ruminally appears as blood glucose; and 70% of starch entering the small intestine appears as blood glucose (Richards, 1999).

In an attempt to look at the metabolic consequences of changing site of starch digestion, we created scenarios for a lactating dairy cow and finishing beef steer (**Table 2**). Liver gluconeogenesis and irreversible loss were calculated using the equations shown in **Figure 1**. We assumed that ruminal conversion of carbon from starch digested in the rumen to blood glucose is approximately 50% as efficient as the conversion in the small intestine. This results in an estimate of 35% of carbon from ruminally digested starch appearing as blood glucose. In contrast, approximately 70% of available starch entering the small intestine is appears as blood glucose (Richards, 1999).

In general, the lactating cow doubles the rates and amounts of the beef steer in terms of dry matter intake, metabolizable energy intake, starch intake, and gluconeogenesis. The cow uses a slightly higher proportion of propionate to support gluconeogenesis than the steer, with a concomitant decrease in the proportions of lactate or amino acids used for that purpose. The cow differs dramatically from the steer in daily output of product (milk production versus weight gain) and in the amount and proportion of glucose oxidized to CO₂. We infer from the data in **Table 2** that the cow has a greater potential demand, or “pull”, and therefore is more likely than the steer to increase production in response to increased glucose supply. The amount of dietary starch required to support glucose irreversible loss is less than normal consumption of dairy cows or steers in production situations. However, if potential capacity of intestinal starch digestion and glucose transport is maximally 1.5 kg/g/d for the steer and 3.0 kg/d for the lactating cow, and more likely capacity is less than 1 kg/d for the steer and 2 kg/d for the lactating cow (Huntington, 1997), then the data in **Table 2** indicate that at least 50% of the starch needs to be fermented in the rumen to accommodate the animals’ requirements for glucose irreversible loss. The amount of dietary starch required to support glucose irreversible loss increases as rumen degradation of starch increases, largely in response to inefficiencies associated with fermentation. However, that does not mean that there are not other benefits of ruminal starch fermentation in terms of overall nutrient supply to support metabolism.

Conclusions and Recommendations

For almost fifty years, grain has been fed to beef and dairy cattle in the U.S. as an economically attractive way to market grain in the form of animal products. As we integrate more information on intermediary metabolism of starch from grain sources, we will be able to continue improvement of the economic aspects of production by fine-tuning demand and supply of glucose, glucose precursors, and other energy sources. Differences in site of digestion, amount of starch digested, and inclusion of fermentation modifiers can affect both the quantity and source of energy substrates available for productive use in beef and dairy cattle. Evaluation of these product changes in metabolic products can be helpful in determining responses to different dietary formulations and feeding strategies. For example, while glucose supply to the mammary gland is a primary determinant of milk production (Kronfeld, 1976), our review of data indicates that the capacity for glucose production is not a primary limiter of glucose supply when substrate is available. Furthermore, Rigout et al. (2003) point out that changes in VFA as well as glucose supply affect milk fat production and fatty acid composition. Similar responses in growing beef animals are likely and may differ as primary growth shifts from lean to adipose tissues. These data do not indicate that methods of ration formulation and feeding strategies should be altered on the basis of increasing supplying carbon for glucose because glucose supply does not appear to be a major limitation in beef or dairy cattle diets.

Areas of Needed Information

The relationship between maximal glucose production (supply) and most efficient use of metabolizable energy for carcass growth or milk production (demand) is not clear. We believe that there likely is a minimal level of glucose that must be oxidized, but clear identification of that level is needed. Also needed is more basic information on the relationship between supply of glucose and other oxidizable substrates at production levels of energy intake.

References

- Amaral, D. M., J. J. Veenhuizen, J. K. Drackley, M. H. Cooley, A. D. McGilliard, and J. W. Young. 1990. Metabolism of propionate, glucose, and carbon dioxide as affected by exogenous glucose in dairy cows at energy equilibrium. *J. Dairy Sci.* 73:1244-1254.
- Annisson, E. F. and W. L. Bryden 1999. Perspectives on ruminant nutrition and metabolism. II. Metabolism in ruminant tissues. *Nutr. Res. Rev.* 12:147-177.
- Armentano, L. E., S. E. Mills, G. DeBoer, and J. W. Young. 1984. Effects of feeding frequency on glucose concentration, glucose turnover, and insulin concentration in steers. *J. Dairy Sci.* 67:1445-1451.
- Armstrong, D. G., K. L. Blaxter, and N. McCGram. 1960. Fat synthesis from glucose by sheep. *Brit. J. Nutri.* 19:31A.
- Baird, G. D., M. A. Lomax, H. W. Symonds, and S. R. Shaw. 1980. Net hepatic and splanchnic metabolism of lactate, pyruvate and propionate in dairy cows in vivo in relation to lactation and nutrient supply. *Biochem. J.* 186:47-57.
- Bartley, J. C. and A. L. Black. 1966. Effect of exogenous glucose on glucose metabolism in dairy cows. *J. Nutr.* 89:317-328.
- Bauman, D. E., C. J. Peel, W. D. Steinhour, P. J. Reynolds, H. F. Tyrrell, A. C. G. Brown, and G. L. Haaland. 1988. Effect of bovine somatotropin on metabolism of lactating dairy cows: Influence on rates of irreversible loss and oxidation of glucose and nonesterified fatty acids. *J. Nutr.* 118:1031-1040.
- Benson, J. A., C. K. Reynolds, P. C. Aikman, P. Lupoli, and D. E. Beever. 2002. Effects of abomasal vegetable oil infusions on splanchnic nutrient metabolism in lactating dairy cows. *J. Dairy Sci.* 85:1804-1814.
- Black, J. L. 1971. A theoretical consideration fo the effect of preventing rumen fermentation on the efficiency of utilization of dietary energy and protein in lambs. *Br. J. Nutr.* 25:31-55.
- Brockman, R. P. 1993. Glucose and short-chain fatty acid metabolism. In: Forbes, J.M. (ed.) *Quantitative aspects of ruminant digestion and metabolism*. Cambridge. Pp. 249-265.
- Casse, E. A., H. Rulquin, and G. B. Huntington. 1994. Effect of mesenteric vein infusion of propionate on splanchnic metabolism in primiparous Holstein cows. *J. Dairy Sci.* 77:3296-3303.
- De Visser, H., Valk, H., Klop, A., Van Der Meulen, J., Bakker, J. G. M., and G. B. Huntington. 1997. Nutrient fluxes in splanchnic tissue of dairy cows: Influence of grass quality. *J. Dairy Sci.* 80:1666-1673.
- Eisemann, J. H. and G. B. Huntington. 1994. Metabolite flux across portal-drained viscera, liver, and hindquarters of hyperinsulinemic, euglycemic beef steers. *J. Anim. Sci.* 72:2919-2929.
- Eisemann, J. H., G. B. Huntington, and D. R. Catherman. 1996. Patterns of nutrient interchange and oxygen use among portal-drained viscera, liver, and hindquarters of beef steers from 235 to 525 kg body weight. *J. Anim. Sci.* 74:1812-1831.

- Eisemann, J. H., G. B. Huntington, and D. A. Catherman. 1997. Insulin sensitivity and responsiveness of portal-drained viscera, liver, hindquarters, and whole body of beef steers weighing 275 or 490 kilograms. *J. Anim. Sci.* 75:2084-2091.
- Harmon, D.L., K. L. Gross, K.K. Kreikemeier, K. P. Coffey, T. B. Avery, and J. Klindt. 1991. Effects of feeding endophyte-infected fescue hay on portal and hepatic nutrient flux in steers. *J. Anim. Sci.* 69:1223-1231.
- Harmon, D. L., K. K. Kriekemeier, and K. L. Gross. 1993. Influence of addition of monensin to an alfalfa hay diet on net portal and hepatic nutrient flux in steers. *J. Anim. Sci.* 71:218-225.
- Herbein, J. H., R. W. Van Maanen, A. D. McGilliard, and J. W. Young. 1978. Rumen propionate and blood glucose kinetics in growing cattle fed isoenergetic diets. *J. Nutr.* 108:994-1001.
- Huntington, G. B. 1990. Energy metabolism in the digestive tract and liver of cattle: influence of physiological state and nutrition. *Reprod. Nutr. Dev.* 30:35-47.
- Huntington, G. B. 1997. Starch utilization by ruminants: From basics to the bunk. *J. Animal Sci.* 75:852-867.
- Huntington, G. B., E. J. Zetina, J. M. Whitt, and W. Potts. 1996. Effects of dietary concentrate level on nutrient absorption, liver metabolism, and urea kinetics of beef steers fed isonitrogenous and isoenergetic diets. *J. Anim. Sci.* 74:908-916.
- Knowlton, K. F., T. E. Dawson, B. P. Glenn, G. B. Huntington, and R. A. Erdman. 1998. Glucose metabolism and milk yield of cows infused abomasally or ruminally with starch. *J. Dairy Sci.* 81:3248-3258.
- Krehbiel, C. R., D. L. Harmon, and J. E. Schnieder. 1992. Effect of increasing ruminal butyrate on portal and hepatic nutrient flux in steers. *J. Anim. Sci.* 70:904-914.
- Kronfeld, D. S., 1976. The potential importance of proportions of glucogenic, lipogenic and aminogenic nutrients in regard to the health and productivity of dairy cows. *Adv. Anim. Physiol. Anim. Nutr.* 7:5.
- Lemosquet, S., S. Rigout, A. Bach, H. Rulquin, and J. W. Blum. 2003. Glucose metabolism in lactating cows in response to isoenergetic infusions of propionic acid or duodenal glucose. *J. Dairy Sci.* 87:1767-1777.
- Lomax, M. A. and G. D. Baird. 1983. Blood flow and nutrient exchange across the liver and gut of the dairy cow. *Brit. J. Nutr.* 49:481-496.
- Lozano, O., C. B. Theurer, A. Alio, J. T. Huber, A. Delgado-Eldorduy, P. Cuneo, D. DeYoung, M. Sadik, and R. S. Swingle. 2000. Net absorption and hepatic metabolism of glucose, L-lactate, and volatile fatty acids by steers fed diets containing sorghum grain processed as dry-rolled or steam-flaked at different densities. *J. Anim. Sci.* 78:1364-1371.
- Lyle, R. R., G. DeBoer, R. O. Harrison, and J. W. Young. 1984. Plasma and liver metabolites and glucose kinetics as affected by prolonged ketonemia-glucosuria and fasting in steers. *J. Dairy Sci.* 67:2274-2282.
- Mephram, T. B. 1993. The development of ideas on the role of glucose in regulating milk secretion. *Aust. J. Agric. Res.* 44:509-522.
- Nielsen, M. O., T. B. Madsen, and A. M. Hedeboe. 2001. Regulation of mammary gland uptake in goats: role of mammary gland supply, insulin, IGF-1, and synthetic capacity. *J. Dairy Res.* 68:337-349.
- Ortigue-Marty, I., J. Vernet, and L. Majdoub. 2003. Whole body glucose turnover in growing and non-productive adult ruminants: meta-analysis and review. *Reprod. Nutr. Dev.* 43:371-383.
- Owens, F. N., R. A. Zinn, and Y. K. Kim. 1986. Limits to starch digestion in the ruminant small intestine. *J. Anim. Sci.* 63:1634-1648.

- Pethick, D. W., D. B. Lindsay, P. J. Barker, and R. T. Northrup. 1981. Acetate supply and utilization by tissues of sheep in vivo. *Br. J. Nutr.* 46:97.
- Reynolds, C.K., P. C. Aikman, B. Lupoli, D. J. Humphries, and D. E. Beever. 2003. Splanchnic metabolism of dairy cows during the transition from late gestation through early gestation. *J. Dairy Sci.* 86:1201-1217.
- Reynolds, C. K., D. L. Harmon, R. L. Prior, and H. F. Tyrrell. 1994. Effects of mesenteric alanine infusion on liver metabolism of organic acids by beef heifers fed diets differing in forage:concentrate ratio. *J. Anim. Sci.* 72:3196-3206.
- Reynolds, C. K., D. J. Humphries, S. B. Cammell, J. Benson, J. D. Sutton, and D. E. Beever. 1998. Effects of abomasal wheat starch infusion on splanchnic metabolism and energy balance of lactating cows. In: K. McCracken, E.F. Unsworth, and A..R.G. Wylie (Ed.) *Energy Metabolism of Farm Animals*. pp 39-42. CAB International New York, NY.
- Reynolds, C. K., G. B. Huntington, H. F. Tyrrell, and P. J. Reynolds. 1988. Net portaldrained visceral and hepatic metabolism of glucose, Lactate, and nitrogenous compounds in lactating Holstein cows. *J. Dairy Sci.* 71:1803-1812.
- Reynolds, C. K., H. Lapierre, H. F. Tyrrell, T. H. Elsasser, R. C. Staples, P. Gaudreau, and P. Brazeau. 1992. Effects of growth hormone releasing factor and feed intake on energy metabolism in growing beef steers: Net nutrient metabolism by portal-drained viscera and liver. *J. Anim. Sci.* 70:752-763.
- Richards, C. J. 1999. Influence of small intestinal protein on carbohydrate assimilation and metabolism in beef cattle. Ph.D. Dissert. Univ. of Kentucky.
- Rigout, S., C. Hurtaud, S. Lemosquet, A. Bach, and H. Rulquin. 2003. Lactational effect of propionic acid and duodenal glucose in cows. *J. Dairy Sci.* 86:243-253.
- Rigout, S., S. Lemosquet, J. E. van Eys, J. W. Blum, and H. Rulquin. 2002. Duodenal glucose increases glucose fluxes and lactose synthesis in grass silage-fed dairy cows. *J. Dairy. Sci.* 85:595-606.
- Russell, R. W., L. Moss, S. P. Schmidt, and J. W. Young. 1986. Effects of body size on kinetics of glucose metabolism and on nitrogen balance in growing cattle. *J. Nutr.* 116:2229-2243.
- Sano, H. M., T. Nakai, T. Kondo, and Y. Terashima. 1991. Insulin responsiveness to glucose and tissue responsiveness to insulin in lactating, pregnant, and nonpregnant, nonlactating beef cows. *J. Anim. Sci.* 69:1112-1127.
- Schmidt, S. P., and R. K. Keith. 1983. Effects of diet and energy intake on kinetics of glucose metabolism in steers. *J. Nutr.* 113:2155-2163.
- Taniguchi, K., G. B. Huntington, and B. P. Glenn. 1995. Net nutrient flux by visceral tissues of beef steers given abomasal and ruminal infusion of casein and starch. *J. Anim. Sci.* 73:236-249.
- Veenhuizen, J. J., R. W. Russell, and J. W. Young. 1988. Kinetics of metabolism of glucose, propionate and CO₂ in steers as affected by injecting phlorizin and feeding propionate. *J. Nutr.* 118:1366-1375.
- Wiegart, M., R. Slepatis, J. M. Elliot, and D. R. Smith. 1986. Glucose absorption and hepatic gluconeogenesis in dairy cows fed diets varying in forage content. *J. Nutr.* 116:839-850.