Dairy production research at the University of Alberta is a mix of the applied and the basic. While the applied is aimed at developing and evaluating new feeding and management practices, the objective of basic research is to improve our understanding of the mechanisms involved in the conversion of feed to milk.

One of the mechanisms which is critical to the cow’s ability to sustain high production is the manufacture of milk lactose. The rate of lactose synthesis is a primary determinant of milk yield since lactose is responsible for drawing water into the space where milk is synthesized. This is the reason that lactose is the most constant constituent in milk, at a concentration of about 5%.

Lactose is synthesized in the mammary gland from glucose, the sugar that circulates in the blood. A cow producing 50 kg of milk is required to synthesize a staggering 2.5 kg of lactose daily. In addition, glucose provides much of the energy required for milk synthesis and is essential to the manufacture of milk fat. In total, the mammary gland requires about 72 grams of glucose to produce 1 kg of milk.

Non-ruminant animals absorb a large proportion of their glucose requirements from the digestive tract, derived from the digestion of starch and sugars. But, when ruminants consume starch and other carbohydrates, much of it is fermented by rumen microbes. So where does the cow producing 50 kg of milk get the 3.6 kg of glucose required by her mammary gland?

**Starch degradation in the rumen**

The primary energy source for the high-producing cow is starch, derived mainly from cereal silages and grains. As suggested in figure 1, starch is composed of long chains of glucose molecules. Depending on the source, level of intake and method of processing, from 42 to 94% of the starch consumed can be degraded in the rumen (see article IB3). Although the first step in starch degradation involves the cleavage of chains into individual glucose units, the end products of this degradation are mainly volatile fatty acids (VFAs) including acetic, butyric, propionic and lactic acids.

These VFAs are absorbed into the portal bloodstream which carries the products of digestion to the liver. Here, propionic and lactic acids are largely converted back to glucose. From 43 to 67% of the glucose synthesized in the liver is derived from propionic acid; up to 12% is derived from lactic acid. Amino acids absorbed from protein digested in the small intestine provide much of the remainder.

**Figure 1**: Pathways in the conversion of feed starch to milk lactose. The magnified part of the diagram illustrates the uptake of glucose into cells lining the small intestine and its utilization as a source of energy for these cells. NRG: energy, VFA: volatile fatty acids.
Since glucose breakdown in the rumen followed by resynthesis in the liver is clearly inefficient, we have been interested in determining the extent to which glucose might be directly available from intestinal starch digestion. On high concentrate corn- or barley-based diets, as much as 3.5 kg/day of starch may escape rumen degradation. Complete digestion of this with absorption of the resulting glucose could make a sizeable contribution to total requirements.

**Starch digestion in the small intestine**

A study published in 1991 indicated that the activity of the intestinal starch-digesting enzyme, *amylase*, was lower in cattle fed grain than in those fed forage. Furthermore, early research on intestinal starch digestion suggested that the cow had the capacity to digest only 100-200 grams of starch per day. This conclusion was based on experiments which estimated starch digestion by measuring glucose transfer into portal blood. Neither of these ideas seemed compatible with practical experience.

Recent results from our laboratory help to explain these results. In the forage versus grain trial, the forage diet had a higher crude protein level (17.4%) than the grain diet (14.9%). Our studies suggest that amylase secretion is sensitive to the protein level in intestinal digesta. Higher protein levels effectively stimulate the secretion of cholecystokinin (CCK) which, in turn, stimulates amylase release from the pancreas. And our current understanding of the fate of glucose released from starch digestion explains the low starch digestion capacity observed in the early work, as explained below:

**What happens to glucose in the small intestine?**

Although starch seems to be readily digested in the small intestine of the cow, the amount of glucose that subsequently appears in portal blood is very limited. Our research has shown that 45-88% of the glucose released by digestion may be transported into the cells lining the small intestine. However, we have also demonstrated that only a small proportion of the glucose entering these cells is transported out into the portal bloodstream – most is used to supply cellular energy requirements. The low starch digestion capacity calculated in the earlier work was based on the assumption that all glucose derived from starch digestion would appear in portal blood, an assumption we now know to be incorrect.

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**Figure 2 : Starch and glucose flow in a 520 kg cow consuming 21 kg of dry matter and producing 32 kg of milk. All amounts are grams of starch or glucose.**

Recognition that glucose released from starch digestion may be used to meet the energy requirements of intestinal cells raises the question: How do these cells satisfy their requirements when low-starch diets are fed? The answer has 2 parts:

1. Intestinal energy requirements are lower when low starch diets are fed, and;
2. When the amount of glucose absorbed from the small intestine is limited, glucose is extracted from blood to supply the energy requirements of the intestinal cells.

Therefore, although the glucose derived from starch digestion may not appear in the bloodstream, its direct use by intestinal cells spares blood glucose for use by other tissues (figure 2). In particular, the mammary gland is left with a greater supply of blood glucose to support lactose synthesis.

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