SITE OF STARCH DIGESTION: IMPACT ON ENERGETIC EFFICIENCY AND GLUCOSE METABOLISM IN BEEF AND DAIRY CATTLE

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SUMMARY

Stoichiometric relationships and controlled infusion experiments clearly show that the efficiency of converting starch energy to tissue energy is enhanced when starch is digested in the small intestine rather than in the rumen. However, limitations in small intestinal starch digestion can prevent realization of this enhanced efficiency at productive levels of starch intake; this is due to incomplete digestion or energy losses associated with large intestinal fermentation. Nevertheless, delivery of starch or glucose to the small intestine increases net PDV glucose flux and glucose entry rate in cattle. In lactating cows, the fraction of the glucose entry rate that is used for lactose synthesis, versus that which is oxidized or incorporated into tissue, appears to be dependent upon the metabolic "pull" of the mammary gland for lactose synthesis and milk production. Alternatively, in growing beef steers, an increased glucose entry rate is associated with adiposity, particularly in the alimentary fat depots. However it is unclear whether this increase in adiposity is due to increased glucose oxidation that spares acetate carbon for lipogenesis, or a direct effect of glucose on the abundance and activity of rate-limiting lipogenic enzymes.

INTRODUCTION

Starch from cereal grains is the primary dietary energy source for highly productive cattle, representing up to 50 and 70% of the ration dry matter for lactating dairy cows and feedlot finishing cattle, respectively. Provided with the capacity to digest starch in both the rumen and intestines, cattle are equipped with the ability to digest large amounts of starch, often exceeding 90% of starch intake (Owens and Zinn, 2005). Although pregastric fermentation in cattle provides an advantage, particularly in forage systems, in that energy is derived from digestion of cellulosic feedstuffs and protein is synthesized from non-protein nitrogen, this advantage is tempered due to fermentative energy losses when high grain diets are fed. In contrast, heat loss associated with small intestinal starch digestion is minimal relative to ruminal and large intestinal fermentation. Estimates of small intestinal starch digestibility are highly variable and are generally indicative of limitations in the capacity to capture energy by cattle consuming high starch diets. Optimization of starch utilization in lactating dairy and growing and finishing beef cattle is impaired by our current inability to precisely predict site of starch digestion, and thus accurately assess the impact of absorption and metabolism of glucose vs. fermentative end products. The goals of this paper are to discuss 1) how site of digestion affects the net energy value of starch, in terms of energy and heat losses associated with starch digestion and subsequent assimilation of substrate into tissue, 2) the integration of potential differences in energetic efficiency with site-specific capacities to digest starch, and 3) implications of increased glucose supply on metabolism and tissue synthesis by lactating cows and growing beef cattle.

ENERGETIC EFFICIENCY OF RUMINAL AND INTESTINAL STARCH DIGESTION

It generally is maintained that conversion of dietary starch energy to tissue energy is greater if assimilation occurs via intestinal glucose absorption rather than ruminal fermentation and subsequent VFA absorption. However, scientific data that directly supports this contention or quantifies the divergence in energetic efficiency associated with site of starch delivery is sparse. Nevertheless, inferences can be made regarding this divergence based upon estimates of energy or heat losses associated with the individual processes involved in digestion and assimilation of substrate into body tissue. Additionally, in vivo approaches have been used to develop total and partial efficiencies of converting ME supplied from purified carbohydrate sources to body tissue energy.

Methane, an end product of anaerobic fermentation, represents a fraction of carbon that is not available for reconversion into usable substrates by either the microbes or the host animal (Hungate, 1966). Accordingly, methanogenesis, 90 to 95% of

which occurs in the rumen, represents a net energetic loss in the conversion of dietary energy to animal tissues or milk. Based on in vivo measures (Blaxter and Clapperton, 1965; Moe and Tyrrell, 1980; Kreuzer et al., 1986) and the stoichiometric relationships between substrates and products (Hungate, 1966; Baldwin et al., 1970), energy losses associated with methane formation range from 3 to 18% of digestible energy. This wide variation is largely attributable to diet composition. Moe and Tyrrell (1980), summarizing data from 404 energy balance trials with dairy cows, reported that the variation in methane production across diets depended largely upon the carbohydrate fractions being digested; energy loss was greater when digestible energy was derived from structural carbohydrate (11-33.6%) rather than from soluble carbohydrate (6.5 %) fractions. These differences in methane formation due to carbohydrate fraction most likely reflect differences in the rumen microflora and in fermentation patterns. Diets rich in fiber support growth of cellulolytic-methanogenic bacteria, whereas diets rich in readily-available carbohydrates (e.g., starch) alter the fermentation pattern so that excess reducing equivalents are consumed for propionate synthesis rather than for reduction of CO_2 to methane. For a typical high-grain feedlot diet, Beever (1993) calculated that 0.38 mole of methane is produced per mole of starch fermented. Based on the heats of combustion of starch (672 kcal/mol) and methane (212 kcal/mol), this is equal to 12% of the DE from starch that would be lost as methane. This value is greater than the value of 8.5% of DE generated in an experiment in which partially hydrolyzed starch was infused into the rumen at a rate of 20% of dietary ME intake (McLeod et al., 2001). Nevertheless, these values indicate that a typical feedlot steer consuming 6.0 kg of starch (25.2 Mcal of intake energy), with a ruminal starch digestibility of 80%, would lose between 1.7 to 2.4 Mcal DE from starch or 6.8 to 9.6% of starch intake energy as methane from the rumen. This loss contrasts with essentially no loss of energy in the form of methane when starch is digested in the small intestine and absorbed as glucose; only negligible amounts of methane are produced in the small intestine (Hungate, 1966). However, given the similarities in fermentation end products between the rumen and large intestine, one would assume that any starch reaching the large intestine would incur a similar energy loss from methane production as that for ruminal fermentation.

Dietary energy also is lost as heat during fermentation as a result of the inefficiency of converting substrates to end products of fermentation. Heat of fermentation is calculated as the difference between the heats of combustion of the substrates used and products formed. Stoichiometric relationships describing the fermentation of starch to a typical ratio of VFA (62 acetate : 22 propionate : 16 butyrate) indicates that 6.4% of the fermentable starch would be lost as heat (Hungate, 1966). This calculation, however, assumes that the microbial mass is static with no capture of hexose energy by the microbiota. Because up to 30% of the hexose may be incorporated by microbial cells (Baldwin et al., 1970), this simple calculation overestimates heat loss to the degree by which hexose energy is incorporated into bacterial cells. However, for starch digested in the rumen, but not in the large intestine, a portion of hexose energy captured in the form of bacterial polysaccharides and amino acids subsequently would be released as heat as a result of bond breakage via enzymatic hydrolysis in the small intestine. Approaches using both in vitro and in vivo techniques provide estimates of heats of fermentation that range from 3 to 12% of DE from both purified substrates and mixed diets (Blaxter, 1962; Webster, 1980). This large variation and the deviation from stoichiometric estimates likely are due to differences in molar ratios of VFA end products and the precision of the experimental techniques used to quantify heat production. Heat of fermentation of starch in the small intestine is negligible; however, heat is released as a result of glucosidic bond cleavage by host enzymes. Given that the free energy of hydrolysis of glucosidic bonds is 4.3 kcal/mol of starch, it is estimated that heat released from digestion of starch in the small intestine would equal 0.6% of DE (Baldwin, 1968).

Absorption of VFA from the rumen and large intestine of ruminants is primarily a passive process; hence, the energy costs directly associated with absorption are negligible (Rechkemmer et al., 1995). Conversely, small intestinal absorption of glucose occurs via a Na+-dependent cotransporter coupled with Na^+/K^+-ATPase, an energy dependent process (Shirazi-Beechey et al., 1995). Providing that absorption of 1 mole of glucose (686 kcal) requires the use of 1 mole of ATP (18 kcal), 2.6% of glucose energy would be expended during absorption (Baldwin, 1968).

Aside from direct energy costs attributed to digestion and absorption, indirect costs such as synthesis of proteins necessary for digestion and maintenance of gut mass and cell ion balance must be considered. Estimated rates of digestive protein secretions in the gastrointestinal tract vary from 3.5 to 7.5 g/$BW^{0.75}$ (Baldwin, 1995). Considering the average molecular weight of protein equals 110g / mole of amino acid in protein and 5 ATP equivalents are required per peptide bond synthesized, heat loss associated with the synthesis and secretion of digestive proteins of a 250 kg steer would equal 285 kcal/d or approximately 4% of maintenance energy. Although this cost is significant, the relative difference in the rate or quantity of digestive proteins secreted due to site of starch digestion gut is considered to be small. In contrast, the difference in costs of gut tissue maintenance between ruminal and small intestinal digestion may be substantial. McLeod et al. (2007) demonstrated that supplying ruminal starch at a rate of 20% of the dietary ME increased gut mass; however the increase in mass of the stomach complex was 12% greater than when an equal amount of starch was supplied to the small intestine. Although an increase in gut tissue mass increases heat losses associated with cellular processes such as ion balance and protein turnover, estimating such costs are difficult and it is not clear whether these changes in mass are a linear function of starch supply. Additionally, Richards (1999) demonstrated that hepatic glucose production was lower for beef cattle receiving abomasal versus ruminal starch infusion, implying that dietary glucose is insufficient when starch is digested in the rumen. Thus, additional metabolic costs of converting gluconeogenic carbon into glucose for essential function or lactose synthesis could also be considered. These costs range from 4% of the energy derived from propionate to 12% of the energy derived from glucogenic amino acids. Nevertheless, excluding indirect cost, stoichiometric relationships indicate that energetic losses associated with starch digestion and subsequent glucose transport in the small intestine equal 3.2% of starch DE or approximately 15-20% of the energy loss incurred if the starch were fermented in the rumen. These same relationships show that losses associated with ruminal fermentation of starch are only about 50% of those that would be incurred with glucose fermentation in the large intestine due to flow and subsequent digestion of bacteria in the small intestine.

The efficiency of converting energy from starch fermented in the rumen or digested in the small intestine to tissue energy is difficult to assess based on feeding studies with grain. Starch from grain is not digested in only one location within the digestive tract. Therefore, one cannot quantitatively deliver dietary starch to specific organs for digestion. To circumvent this problem, we infused a partial cornstarch hydrolysate into either the rumen or the abomasum of growing beef steers (McLeod et al., 2001). Steers were fed a basal forage diet at 1.5 times maintenance energy requirements; starch hydrolysate was infused at a rate of 20% of total ME intake [12.6 g/(d^{-1} kg $BW^{.75}$)]. The partial efficiency (K_r) of converting ME from starch to tissue energy was calculated as the increase in retained energy above the basal diet divided by the ME supplied by the infused starch. Thus, Kr reflects both direct and indirect heat losses associated with digestion, absorption, and assimilation of substrate into tissue. Our K_r estimates averaged 0.48 and 0.60 for ruminally and abomasally infused starch, respectively. These K_r values are somewhat lower than those determined previously with sheep for ruminally-supplied (0.55) and abomasally-supplied (0.72) glucose (Armstrong et al., 1960). However, the relative increases in K_r observed (25 and 31%) for abomasal versus ruminal starch or glucose supply are reasonably consistent between studies. Branco et al. (1999) determined that 88% of duodenally-infused cornstarch hydrolysate disappeared from the small intestine of steers. Because we used a similar rate of infusion in our energy balance experiments, some of the abomasally-infused starch in our experiment may have escaped small intestinal digestion to be fermented in the large intestine. Adjusting the data set by 0.88 creates a theoretical maximum K_r value for small-intestinally supplied starch of 0.68. Therefore, the actual K_r value for small intestinally-supplied starch probably falls between the observed 0.60 and the calculated maximal value of 0.68. Based on these partial efficiencies, and an average loss of 10% of DE for methane formation, the total energetic efficiency of ruminally-fermented starch is only 65 to 72% of that for starch digested in the small intestine. Although the magnitude of the total energy loss seems greater, the differences in these efficiencies agree reasonably well with differences based on stoichiometric relationships of substrates and products and the cost of absorption. Therefore, both approaches indicate that accurate prediction of the energy values of cereal grains

requires quantitative data describing starch digestion in terms of the extent of digestion in the rumen, small intestine, and large intestine.

INTEGRATION OF ENERGETIC EFFICIENCY AND DIGESTION LIMITS

Cattle are efficient at and have high capacity for digesting starch from cereal grains. Owens and Zinn (2005) summarized results from published and unpublished trials that measured site and extent of starch digestion by lactating dairy and feedlot beef cattle. Across grain types and processing methods, total tract starch digestibility averaged 92% and 98% for lactating dairy and feedlot cattle, respectively. However, based on the calculations of energetic efficiency presented above, the net energy value of starch from grains can vary not only due to the extent, but also due to the site of digestion. Several excellent reviews have been published that discuss the capacity for and factors that affect ruminal and intestinal starch digestion by cattle (Huntington et al., 1997; Harmon and McLeod, 2001; Harmon et al., 2004; Owens and Zinn, 2005; Huntington et al., 2006); the reader is referred to these reviews for detailed discussions. This paper will briefly discuss potential limitations to starch digestion in the rumen and small intestine and how these limitations impact the net energy value of starch from cereal grains.

Using a data set generated from 16 published studies conducted in beef cattle (n=79) consuming 1 to 5 kg of starch per day supplied from varying sources of grain (corn, sorghum, and barley), Harmon et al. (2004) demonstrated a linear relationship between starch intake and ruminal starch digestion with a slope (i.e., digestion coefficient) of 0.77. This approach also revealed variation in ruminal starch digestion due to source of grain, with digestibility being higher for corn-based diets (0.80) than for sorghum-based diets (0.75). However, these authors found no relationship between starch intake and ruminal digestibility, indicating that ruminal starch digestion was not limiting, at least within the starch intake parameters of the data set. Owens and Zinn (2005), summarizing data from 49 trials, showed that ruminal digestibility of starch from corn, in both lactating dairy and feedlot beef cattle, was increased by processing corn with added moisture, mechanical pressure, and(or) heat. When averaged across processing method and weighted by the number of observations, ruminal digestibility of starch from corn in beef cattle was identical between the data sets (80%) used by Harmon et al. (2004) and Owens and Zinn (2005). In contrast to beef cattle, Owens and Zinn (2005) further reported that the ruminal fraction of total tract starch digestion that occurs in the rumen is substantially lower in lactating dairy cows. Again, averaged across processing method by using weighted means from the data set of Owens and Zinn, an average ruminal digestibility of 55% was calculated for starch from corn in lactating cows. It is unlikely that the lower ruminal starch digestibility by lactating dairy cows relative to beef cattle reflects a lower fermentation capacity, but rather a decreased rumen retention time due to higher feed or NDF intake, or to anatomical differences in the reticulo-omasal orifice (Owens and Zinn, 2005).

Postruminal starch digestion includes digestion in both the small and large intestines. As previously described in their summary of studies in beef cattle where intestinal starch digestibility was measured, Harmon et al (2004) reported that the digestibility of starch entering each segment averaged 62% and 47% for the small and large intestines, respectively. Additionally, these authors applied linear regression to the data and found a reasonable relationship between starch entering the large intestine and large intestinal digestion (slope = 0.44). In contrast, the linear fit for small intestinal entry and digestion was comparatively low (r^2= 0.36; slope = 0.40), which reflects a tendency for small intestinal digestibility to decline at a higher starch intake. In an effort to further define the relationship between starch entry and digestion in the small intestine, these same data were subsequently fit to a nonlinear kinetic-based model (Huntington et al., 2006). Output from this model showed that the capacity for the small intestine to digest starch approached an upper asymptote between 600 and 700g/d. As a consequence, small intestinal digestibility was predicted to decrease from approximately 85% to 44% as the amount for starch entering the small intestine increased from 300 to 1,500 g/d. Parallel data describing intestinal starch digestion in lactating cows is extremely limited; however, similar limitations in digestion would be expected. Limitations in small intestinal starch digestibility have been ascribed to particle size or physiochemical properties of intact starch, insufficient pancreatic α- amylase and(or) brush-border carbohydrases (Owens et al., 1986; Harmon et al., 2004). Conversely, low digestibility of starch in the

large intestine probably reflects the fact that starch particles, having resisted digestion in the rumen and small intestine, inherently are resistant to digestion.

Based on these capacity estimates of starch digestion in cattle, maximal net energy value of cereal grain is limited by small intestinal starch digestibility. Case-in-point, using a ruminal starch digestibility coefficient of 0.80 and the kinetic model of Huntington et al. (2006) to predict small intestinal digestibility, a feedlot steer consuming 5 kg of starch would have a postruminal starch flow of approximately 1200g/d. Of this amount only 50% would be digested in the small intestine, leaving 600 g to flow to the large intestine. Because of this large flow of starch to the large intestine and its associated energy losses, any advantage in energetic efficiency achieved by digesting starch in the small intestinal digestion relative to the rumen is lost. Huntington et al. (2006), using a simulation model, demonstrated that shifting starch digestion from the rumen to the small intestine would increase energy yield only when small intestinal digestibility exceeds 75%. Therefore, in order to capitalize on the energetic efficiency of shifting starch digestion from the rumen to the small intestine, starch flow to the large intestine must be minimized.

SMALL INTESTINAL STARCH DIGESTION AND GLUCOSE METABOLISM

Typically ruminants obtain the majority of their glucose supply from hepatic gluconeogenesis, which is derived primarily from propionate (43 to 77%) and amino acid (10 to 30%) carbon. One putative advantage conferred by postruminal digestion of grain starch is an increase in glucose absorption or a decreased need for de novo synthesis of glucose to meet demands of production. Indeed, Amaral et al. (1990) reported that the fractional contributions of propionate to hepatic glucose output decreased when dairy cows were infused intravenously with glucose. Data from experiments using short-term intravenous or intra-duodenal infusions of glucose would support this contention in that endogenous glucose synthesis was decreased (Bartley and Black, 1966; Leng, 1970). Relative to water infusion, long-term abomasal infusion of wheat starch (1200g/day) increases PDV appearance of glucose without decreasing hepatic output (Reynolds et al., 1998). Furthermore, in growing beef steers infused with 800 g of partially hydrolyzed starch ruminally or abomasally, net increases in glucose absorption and total splanchnic output were observed for abomasal vs. ruminal infusion (Richards, 1999). Accompanying the observed increase in glucose supply, Richards (1999) found an increase in both glucose entry rate (i.e., rate of appearance and utilization under steady state conditions) and in peripheral utilization of glucose. Based on these findings, one would expect that an increase in the quantity of starch digested in the small intestine would be accompanied by an increase in glucose utilization.

Although glucose entry rate apparently is increased with small intestinal starch digestion, subsequent production responses have been mixed and dependent on productive state, reflecting a complex interaction between productive tissues, endocrine controls, and nutrient supply. When glucogenic precursors (ruminal propionate or duodenal glucose) each were infused at two different rates (1.72 or 3.45 Mcal NE_L/d), Lemonsquet et al. (2004) detected an increase in glucose appearance that exceeded the increase in lactose synthesis in early lactating dairy cattle. In contrast, Reynolds, (2001) reported that with late lactating dairy cattle, an abomasal infusion of 1200g/d wheat starch did not increase milk energy output when compared to water-infused controls. In that same report, Reynolds (2001) infused incrementally increasing amounts of corn starch (700, 1400 and 2100 g/d) in early lactating dairy cattle and observed that milk energy output increased only at the highest rate of starch infusion. Moreover, across these studies, the increase in milk energy output with increased postruminal supply of starch represented only a small portion of the increase in ME from the infusate. Thus, the balance of energy from glucose must either be oxidized or used for tissue gain. Supporting this observation, abomasal infusion of partially hydrolyzed starch (1.5 kg/d) relative to ruminal infusion (1.5 kg/d) increased glucose entry rate but did not affect lactose synthesis, thus resulting only in a tendency for milk yield of mid-lactation dairy cows to increase (Knowlton et al., 1998). Abomasal infusion of starch did increase the fraction of carbon dioxide that was derived from glucose. Taken together, this indicates that under normal physiologic conditions with adequate energy supply, the quantitative supply of glucose does not appear to limit milk production. However, at high levels of milk production, the fraction of glucose entry that appears in lactose increases, while the fraction

oxidized to carbon dioxide decreases (Baumann et al., 1988).

In growing beef steers, we demonstrated that both ruminal and abomasal infusion of partially hydrolyzed starch at a rate of 20 % of the ME supply increased retained tissue energy above that observed for the basal forage diet alone, with greater retention from abomasal rather than from ruminal starch delivery (McLeod et al., 2001). Partitioning of the increased retained tissue energy, using C-N balance techniques, revealed that retained energy deposited as protein and lipid comprised 30 and 70% for ruminally-infused energy compared to 16 and 84% for abomasally-infused energy. After accounting for protein accretion, the increase in tissue energy retention from abomasal as compared with ruminal infusion of starch was accounted for solely as adipose tissue. In a subsequent terminal experiment using the same infusion model, McLeod et al. (2007) further confirmed the stimulatory effect of abomasal starch delivery on adipose accretion in growing beef steers. Specifically, the absolute and relative amounts of alimentary fat mass were greater following infusion of starch abomasally as compared to ruminal infusion. Because an isoenergetic glucose infusion treatment was included in this experiment, it was apparent that the increase in abdominal adiposity was exacerbated by compared to starch infused abomasally. It is unclear weather this reflects a difference in energy supply to the tissues or in glucose entry rate.

In an effort to further examine the functional response of the mesenteric, omental, and subcutaneous adipose depots to intestinal carbohydrate infusion by growing beef steers, we collected adipose samples following the 35 d infusions from the aforementioned steers for ex-vivo analysis of lipogenic and lipolytic activity (Baldwin et al., 2007) as well as analysis of lipogenic enzyme and adipose regulatory protein gene expression (Baldwin, 2006). Lipolytic rates were largely unaffected by infusion treatment. However, incorporation rates for both acetate and glucose into fatty acids were greater for adipose tissues harvested from steers abomasally-infused with either glucose or starch compared with those receiving ruminal infusion (Baldwin et al., 2007). Similar to the observed mass changes, incorporation of lipogenic substrate was greater with abomasal infusion of glucose than of starch (McLeod et al., 2007). However, given that the rate of glucose incorporation was only a fraction of that observed for acetate, it seems unlikely that direct incorporation of glucose carbon into adipose is responsible for the increased adiposity. Although insulin has been shown to increase the uptake of glucose and acetate by muscle and adipose tissue, these actions are permissive; the major role of insulin in adipose accretion in ruminants is mediated via antilipolytic actions rather than by stimulation of fatty acid synthesis (Brockman, 1986). In our experiment, circulating insulin concentrations were not changed by carbohydrate infusion treatment at the end of the 35 d treatment period (Baldwin et al., 2007). Therefore in the absence of a change in circulating insulin concentrations, it seems likely that an increase in the glucose supply stimulated lipogenesis independent of insulin, by either sparing acetate carbon for de novo lipogenesis and/or directly stimulating lipogenic gene expression. In support of the latter idea, glucose has been shown to stimulate expression of lipogenic enzyme mRNA (Fatty Acid Synthetase and Acyl-CoA Carboxylase) in rat adipose tissue via elevation of intracellular glucose-6-phosphate concentrations (Girard et al., 1997). Moreover, abomasal glucose infusion induced increases in the transcription of genes encoding for lipogenic regulatory nuclear proteins including: carbohydrate response element binding protein, sterol regulatory element-binding protein 1, and Spot 14, as well as their established targets - FAS and ACC (Baldwin et al., 2006). However, more research is necessary to ascertain the exact mechanism(s) responsible for stimulation of adipose accretion observed with abomasal carbohydrate infusion and to discern whether this increase in adiposity depends specifically to carbohydrate or to other energy sources and whether it applies to grain feeding programs.

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QUESTIONS AND ANSWERS

Q: Kyle, you mentioned that starch digested in the lower GI tract may increase the amount of omental fat. Would you elaborate on the mechanism?

A: We don't know the mechanism. Think about the kid drinking soda pop at school; he is likely to have a big waistline. In studies where we have increased the amount of circulating glucose, we have seen a similar response. Glucose may have a direct effect. We may be increasing the expression of nuclear regulatory proteins that have been shown to increase fatty acid synthesis; these are prominent in omental fat. Perhaps omental fat is less insulin dependent than other tissues or its anatomical location allows more direct use of absorbed glucose.

Q: Kyle, as you increase glucose supply to the small intestine, the additional energy appeared in fat. What depots were affected primarily?

A: In our slaughter experiments, we saw some increase in subcutaneous fat, but the fat depth measurement was not very quantitative. In the alimentary fat, where we have separated the adipose depot into omental and mesenteric fat, the increase is in the omental fraction and not the mesenteric. Whether this is based on our method of separating these fractions or if there is truly a difference in metabolism of omental and mesenteric fat is not known.