Effect of feeding fermentable fiber on synthesis of total and mucosal protein in the intestine of the growing pig

A.J. Libao-Mercado, C.L. Zhu, M.F. Fuller, M. Rademacher, B. Sève, C.F.M. de Lange

Abstract

In a previous study, a reduced efficiency of ileal digestible threonine (THR) use for body protein deposition was observed in growing pigs when pectin was included in the diet. This response was not due to increased physical endogenous ileal THR loss. Our aim was to explore the contribution of diet-induced increases in protein synthesis in the colon, especially mucins, to dietary THR requirements. Twelve barrows (21 kg mean BW) were fed either a cornstarch–soybean meal-based diet (Control) or Control with 12% pectin (Pectin). Pigs were given intravenously 1.5 mmol/kg BW of L-1-13C valine (40 mol%) to measure fractional and absolute synthesis rates (FSR, ASR, respectively) of mucosal and whole intestinal protein in the jejunum and colon. Dietary pectin inclusion increased plasma levels of glucose, isoleucine and glutamine ($P_{b}0.05$) but had no effect on insulin or urea nitrogen ($P_{N}0.10$). There were no differences in FSR and ASR of whole intestinal protein in jejunum and colon ($P_{N}0.10$). The FSR of mucosal proteins in colon, not in jejunum, was increased with dietary pectin supplementation ($P_{b}0.05$). Assuming mucosal protein mass is constant, these results imply that the higher protein synthesis in colon mucosa contributes to the reduced THR efficiency observed in pectin-supplemented diet.

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1. Introduction

A reduced efficiency of utilizing ileal digestible threonine (THR) but not lysine intake for whole body protein deposition (PD) was recently observed in growing pigs when pectin, a highly fermentable non-starch polysaccharide (NSP), was included in the diet (Zhu et al., 2005). This response was not due to increased physical endogenous ileal THR loss with increasing dietary pectin level (Zhu et al., 2005), but most likely related to increased metabolic cost associated with higher endogenous protein recycling in the upper gut, or increased endogenous protein synthesis in the lower gut, and possibly the upper gut, induced by enhanced microbial fermentation. Endogenous protein secreted into the intestinal lumen, if not digested and...
reabsorbed, constitutes a physical endogenous protein loss or becomes substrate for microbial fermentation.

In a follow-up study where pectin was continuously infused into the caecum (10% pectin solution; 2.4 L/d), whole body PD, THR utilization for PD, urea pool size and urea flux were reduced (Zhu et al., 2003). These results suggest that the diet-induced effect on THR utilization reflects largely physiological changes in the lower gut. The objective of the current study was to explore the underlying mechanism behind the fermentable fiber-induced effect on THR utilization for PD. It was hypothesized that the synthesis of colonic mucosal proteins is increased with dietary inclusion of fermentable substrates, such as pectin. We suggest that amino acid for synthesis of mucosal proteins in the lower gut contributes to increased dietary amino acid requirements when feeding high fiber feedstuff.

2. Materials and methods

A total of 12 Yorkshire barrows, with initial body weight (BW) of 15.7 kg (SD = 0.90), were given either a cornstarch-soybean meal-based control diet (Control) or the control diet with 12% pectin added at the expense of cornstarch (Pectin). Pigs were allowed to adapt to their respective diets for 10 days prior to infusion of isotopes, and fitted surgically with jugular catheters 7 days prior to infusion and according to Nyachoti et al. (2000). Animals were infused, via jugular vein catheter, with 1.50 mmol/kg BW of L-1-13C valine (40 mol%) for 12 min and were then euthanized 30 min later.

Whole intestinal and mucosal samples for tissue free and protein-bound valine enrichment assay were prepared, essentially as described by Nyachoti et al. (2000). Isotopic enrichment of valine in plasma and tissue free pool was determined by GCMS; protein-bound valine in the intestine and intestinal mucosa was determined by GC-C-IRMS. Plasma insulin, glucose and urea N were determined using commercially available kits. Fractional synthesis rates (FSR; %/d) of mucosal and whole intestinal proteins is increased with dietary inclusion of fermentable substrates, such as pectin. We suggest that amino acid for synthesis of mucosal proteins in the lower gut contributes to increased dietary amino acid requirements when feeding high fiber feedstuff.

3. Results

All pigs recovered quickly from surgery, appeared healthy, and were eating full meals within 6 h post-surgery. Their average BW at the end of infusion was 21.0 kg (SD = 0.90).

Of all amino acids, there were only significant pectin-induced increases in plasma concentration of isoleucine...
Table 1
Effect of dietary inclusion of fermentable fiber (12% pectin) on tissue weights, protein concentration and absolute protein synthesis rates in whole jejunum and colon of growing pigs

<table>
<thead>
<tr>
<th>Tissue Parameters</th>
<th>Control</th>
<th>Mean</th>
<th>SE</th>
<th>Control +pectin</th>
<th>Mean</th>
<th>SE</th>
</tr>
</thead>
<tbody>
<tr>
<td>Body weight (kg)</td>
<td>21.3</td>
<td>0.4</td>
<td>20.5</td>
<td>0.2</td>
<td>21.3</td>
<td>0.4</td>
</tr>
<tr>
<td>Weight (g)</td>
<td>876</td>
<td>37</td>
<td>859</td>
<td>37</td>
<td>876</td>
<td>37</td>
</tr>
<tr>
<td>Protein concentration (mg/g)</td>
<td>63</td>
<td>0.5</td>
<td>61</td>
<td>2</td>
<td>63</td>
<td>0.5</td>
</tr>
<tr>
<td>ASR (g/d)</td>
<td>38.1</td>
<td>3.7</td>
<td>36.3</td>
<td>5.6</td>
<td>38.1</td>
<td>3.7</td>
</tr>
<tr>
<td>Protein concentration (mg/g)</td>
<td>57</td>
<td>0.9</td>
<td>54</td>
<td>1.0</td>
<td>57</td>
<td>0.9</td>
</tr>
<tr>
<td>ASR (g/d)</td>
<td>5.2</td>
<td>0.6</td>
<td>5.4</td>
<td>0.4</td>
<td>5.2</td>
<td>0.6</td>
</tr>
</tbody>
</table>

* a No significant dietary treatment effects were observed (*P* >0.10).

* b Absolute protein synthesis rate (g/d); calculated as fractional synthesis rate (%/d) multiplied by tissue protein mass (g).

and glutamine (294 vs. 350, 846 vs. 1085 μmol/L, respectively). Dietary pectin inclusion also increased plasma levels of glucose (5.8 vs. 6 mmol/L) but not insulin or urea nitrogen (*P* >0.10). There were no differences between treatments in the isotopic enrichment of valine in plasma (33.0 vs. 33.4 MPE; *P* >0.10) or the tissue free pools (jejunum 27.2 vs. 26.8 MPE; colon 28.3 vs. 28.6 MPE; *P* >0.10). The ratio of tissue free to plasma valine enrichment was not different between treatments (0.81 and 0.85 in jejunum and colon, respectively; *P* >0.10).

Inclusion of fermentable fiber (12% pectin) in the diet had no effect on FSR of proteins in whole jejunum and colon (*P* >0.50). It also had no effect on FSR of proteins in jejunal mucosa (*P* >0.50). Fermentable fiber, however, increased the FSR of colonic mucosal proteins by 22–23 percentage units from the control diet (*P* <0.05; Fig. 1). Tissue mass and protein concentration in the jejunum and colon were unaffected by dietary treatments (Table 1; *P* >0.10). It had no impact on ASR of protein in whole jejunum and colon (Table 1; *P* ≥0.75).

### 4. Discussion

The impact of dietary treatment on plasma amino acid and glucose levels may be related to the lower nutrient digestibility, and thus absorption, when feeding pectin-rich diets (Zhu, 2003), but it can also reflect post-absorptive changes in amino acid flux.

The ratio of tissue free to plasma valine enrichment achieved in the current experiment is very close to those achieved in other pig studies (Sève et al., 1993; Nyachoti et al., 2000). More importantly, the ratios were the same for both diets. There is therefore no difference in terms of treatment effects on FSR whether enrichment of plasma free or tissue free amino acid is used to estimate that at the site of protein synthesis.

The lack of diet effects on FSR and ASR of proteins in whole jejunum and colon were similar to the findings obtained in another study where cornstarch–casein vs. barley–canola meal-based diets were compared (Nyachoti et al., 2000). There was however a diet-induced increase in FSR of mucosal protein in colon but not in jejunum. These combined results imply that pectin, and probably other fermentable fiber, promotes synthesis of proteins which are highly expressed in the mucosa relative to the whole colon. If colonic mucosal protein mass is the same for both diets, then the reduced THR use for PD, observed when pectin is included in THR-limiting diets (Zhu et al., 2005), is largely due to increased synthesis of colonic mucosal protein, particularly the threonine and serine-rich mucin proteins (Faure et al., 2002). Colonic mucosal protein synthesized, if secreted, is lost either as physical endogenous protein loss excreted in feces or is fermented by microbes.

The effect of fermentable fiber on colonic mucosal protein synthesis can be direct (physiological concentrations of fibers, viscosity) or indirect, i.e. through release of fiber components (ex. galacturonic for pectin) or through products of bacterial fermentation of fiber (SCFA, ammonia). These effects can be mediated through hormones (PYY, GLP-2), neuropeptide (VIP) and other agents (Plaisancie et al., 1996; Deplancke and Gaskins, 2001; Fukunaga et al., 2003). Understanding both the controls involved in colonic mucosal proliferation and the products of bacterial fermentation for different substrates is essential in order to better predict the response of growing pigs to different types and inclusions of high fiber feedstuffs.

### 5. Conclusions

Feeding purified pectin had no influence on the fractional protein synthesis rates in the jejunum or colon of growing pigs. It also had no effect on the overall absolute protein synthesis in the jejunum and colon. It does, however, increase the fractional synthesis rate of colonic mucosal proteins. It therefore seems likely that the reduced threonine utilization for protein deposition observed in growing pigs fed pectin-supplemented threonine-limiting diet is due to an increase in colonic mucosal protein synthesis, particularly the threonine-rich mucin proteins.
References


