Research Note

Dietary Inulin Affects the Morphology but not the Sodium-Dependent Glucose and Glutamine Transport in the Jejunum of Broilers

H. Rehman,* C. Rosenkranz,†[1] J. Böhm,* and J. Zentek*[2]

*Institute of Nutrition, Department of Veterinary Public Health and Food Science, and †Institute of Histology and Embryology, Department of Pathology, University of Veterinary Medicine, Veterinärplatz 1, A-1210 Vienna, Austria

ABSTRACT Inulin, a prebiotic, is a fermentable oligosaccharide that may affect the intestinal mucosal architecture and the electrophysiological parameters. The effects of a diet with added inulin were tested on the jejunal morphology and electrogenic transport of Glc and Gln from the jejunal mucosa in broilers. Short-circuit current and transmucosal tissue resistance of jejunal flaps were measured in Ussing chambers. The feeding experiment was carried out in broilers (n = 40) using 1% inulin with an application period of 5 wk. The inulin-containing diet resulted in longer jejunal villi ($P < 0.05$) and deeper crypts ($P < 0.01$) than in control birds without affecting villus:crypt depth. Basal short-circuit current value remained unaffected by dietary treatment. Inulin supplementation did not modify the electrogenic transport of Glc and Gln in the jejunal mucosa. The basal value of transmucosal tissue resistance was significantly lower ($P < 0.001$) in the inulin-fed group compared with the control group. In conclusion, inulin supplementation affected the jejunal mucosal architecture but did not modify the electrogenic transport of Glc and amino acid under present experimental condition.

Key words: inulin, electrophysiology, jejunum, short-circuit current, transmucosal tissue resistance

2007 Poultry Science 86:118–122

INTRODUCTION

Oligosaccharides that are not hydrolyzed by the digestive enzymes in the upper digestive tract of a host enter in the hindgut, where they are fermented by the intestinal microbiota. Inulin, a prebiotic polyfructan, extracted from chicory (*Cichorium intybus*), contains molecules with a degree of polymerization (**DP**) of 3 to 60, average DP being 10 (Crittenden, 1999). Therefore, it contains both, oligosaccharide components and polysaccharides. Because of the β (2→1) glycosidic bond, it is resistant to host-derived digestive enzymes. Inulin has been reported to decrease the cecal concentrations of *Escherichia coli*, *Salmonella* spp., and *Campylobacter* spp. in broilers (Yusrizal and Chen, 2003) and to increase *Bifidobacterium* spp. (Rada et al., 2001). Inulin supplementation of broiler diets has been found to increase the concentrations of jejunal lactate and cecal butyrate (Rehman et al., 2006b). No information exists regarding the effects of inulin on the histomorphology of the small intestine, though a few reports are available showing that villus height of the small intestine is increased in broilers supplemented with fermentable carbohydrates like fructooligosaccharides (**FOS**; Sonmez and Eren, 1999; Xu et al., 2003) and mannan oligosaccharides (**MOS**; Iji et al., 2001). Kleessen et al. (2003) demonstrated that supplementation of dietary fructans (mixture of FOS and inulin at 1:1) in different rat models (germ-free rats, rats associated with human fecal flora, and rats harboring *Bifidobacterium longum* and *Bacteroides vulgatus*) resulted in higher villi and deeper crypts in the jejunum of human fecal flora-associated rats but not in germ-free rats. The jejunal villus height was also higher ($P < 0.05$) in the rats harboring *B. longum* and *B. vulgatus* without affecting crypt depths of jejunum or distal colon.

It is assumed that an increased villus height is paralleled by an increased digestive and absorptive function of the intestine due to increased absorptive surface area, expression of brush border enzymes, and nutrient transport systems (Pluske et al. 1996). No information is available regarding the effect of inulin on the intestinal morphological changes in broilers and in vitro absorptive capacity of Glc as well as amino acids from the jejunal mucosa.

Therefore, the objective of this study was to investigate the potential effects of inulin on the jejunal mucosal architecture and electrophysiological tissue parameters, Na-dependent Glc, and Na-dependent Gln transport.

MATERIALS AND METHODS

Forty 1-d-old broilers of a commercial strain (Ross 308), procured from a commercial hatchery (Geflügelhof Schulz,

Received July 19, 2006.
Accepted August 28, 2006.
[1]Current address: Institute of Nutrition, Faculty of Veterinary Medicine, Free University, D 14195 Berlin, Germany.
[2]Corresponding author: zentek.juergen@vetmed.fu-berlin.de

Table 1. Ingredient percentages and analysis of basal diet

Ingredients	Composition
Corn	50.50
Soybean meal	30.00
Wheat	10.00
Potato protein	3.00
Soybean oil	1.40
NaCl	0.10
Vitamin-mineral mixture[1]	5.00
Analyzed composition (DM)	
DM (%)	87.9
Ash (%)	6.6
Crude fiber (%)	1.5
CP (%)	24.9
Crude fat (%)	3.5
Calculated ME (kcal/kg)	2,820
Minerals (%)	
Ca	1.08
P	0.87
Mg	0.26
Na	0.33
K	1.27

[1]BR 5 Universal Vetmed, Biomin GmbH, Herzogenburg, Austria. Each kilogram contains Ca, 196 g; P, 64 g; Na, 30 g; Mg, 6 g; Cu, 400 mg; Zn, 1,200 mg; Fe, 2,000 mg; Mn, 1,200 mg; Co, 20 mg; I, 40 mg; Se, 8 mg; vitamin A, 200,000 IU; vitamin D_3, 80,000 IU; vitamin E, 1,600 mg; vitamin K_3, 34 mg; vitamin C, 1,300 mg; vitamin B_1, 35 mg; vitamin B_2, 135 mg; vitamin B_6, 100 mg; vitamin B_{12}, 670 μg; nicotinic acid, 1,340 mg; calcium pantothenic acid, 235 mg; choline chloride, 8,400 mg; folic acid, 34 mg; biotin, 3,350 μg; and Met, 30 g.

Schulweg, Austria), were randomly divided into 2 groups (control and inulin), each comprising 20 birds. The birds were housed in wire-bottomed pens fitted with electrical heaters during the 35-d experimental period. The temperature started at 33°C (from d 0 to 3) and was gradually reduced according to normal management practice (2 to 3°C/wk). Chicks were maintained on a 24-h constant light schedule until the end of the experiment. The experiment was conducted under the permission of the Federal Ministry for Education, Science and Culture, Vienna, Austria, vide letter no. GZ 68.205/69-BrGT/2004.

Diets

Chicks in the control group were fed a basal diet, which was mainly comprised of corn and soybeans (Table 1). The inulin group was fed the basal diet with 1.0% inulin (Raftiline-GR with DP 2 to 60, average being 10 to 12, Orafti Active Food Ingredients, Tienen, Belgium). According to the manufacturer, the inulin mixture contained pure inulin (90 to 94%), Glc, Fru (0 to 4%), and sucrose (4–8%). The inulin was supplemented at the expense of corn. The feed was devoid of any coccidiostats or antibiotics. Feed and water were provided ad libitum.

Histomorphology of the Jejunum

On d 35, 10 birds from each group were randomly selected and killed by cervical dislocation. The tissue samples for histology were taken from the jejunum at the junction of the Meckel's diverticulum and fixed in 4% buffered formalin for 2 d. The processing consisted of serial dehydration, clearing, and impregnation with wax. Tissue sections, 5-μm thick (3 cross-sections from each sample), were cut by a microtome and were fixed on slides. A routine staining procedure was carried out using hematoxylin and eosin. The slides were examined on an Olympus AX70 microscope (Olympus Corp., Tokyo, Japan) fitted with a digital video camera (Sony DXC-930P, Sony, Tokyo, Japan). The images were analyzed using stereological image software, CAST Image System (Version 2.3.1.3, Visiopharm, Hørsholm, Denmark). A total of 15 intact well-oriented, crypt-villus units were selected randomly for each sample. The mean values attributed to individual birds were used in the statistical analysis. Villus height was measured from the tip of the villus to the villus-crypt junction, whereas crypt depth was defined as the depth of the invagination between 2 villi.

Nutrient Transport Activity

On d 35, 8 birds from each group, were killed at the Institute of Nutrition, University of Veterinary Medicine, Vienna, Austria. The jejunum was then removed within 3 min after exsanguination, rinsed several times with ice-cold Ringer buffer (4°C), and transported in ice-cold oxygenated buffer to the laboratory.

Preparation of Tissue. The segments were taken from jejunum at the junction of the Meckel's diverticulum, and tissue flaps were prepared by the method as described earlier (Awad et al., 2005; Rehman et al., 2006a). The intestinal segment was opened longitudinally along the mesenteric border and washed free of intestinal contents several times with Ringer solution at 4°C. Tissues were placed in the cold Ringer buffer and gassed with carbogen (O_2 and CO_2, ratio 95:5) until they were mounted in the Ussing chamber.

Measurements of Electrophysiological Traits. Short-circuit current (**Isc**) and transmucosal tissue resistance (**Rt**) were measured in Ussing chambers with a microprocessor system based on a voltage-current clamp device (Mussler, Microclamp, Aachen, Germany). The serosa and muscularis were stripped manually to obtain a mucosa-submucosa preparation of the jejunum. Thereafter, the epithelial sheets were mounted in the modified Ussing chambers with an exposed tissue area of 1 cm^2. The serosal and mucosal surfaces of the tissues were bathed in 5mL of Ringer solution with the following composition (mmol/L): $CaCl_2$, 1.2; $MgCl_2$, 1.2; Na_2HPO_4, 2.4; NaH_2PO_4, 0.4; $NaHCO_3$, 25; KCl, 5; NaCl, 115; and mannitol, 10. The pH was adjusted to 7.4. The bathing medium in the chambers was aerated with 95% O_2 and 5% CO_2 and maintained at 38°C in a water bath (Julabo USA Inc., Allentown, PA). The solution was continually stirred and oxygenated by bubbling into the chamber by means of a gas lift. The electrode potential and the solution resistance were determined at the beginning of experiment and were automatically corrected before tissues were placed in the chamber. The tissues were first incubated under open circuit conditions for 30 min. for equilibration and then were short-circuited by clamping the voltage at 0 mV to measure Isc

Table 2. Effect of inulin supplementation on histomorphological parameters of the jejunum in broilers (n = 10)

Parameters	Groups		Probability
	Control	Inulin	
Villus height (μm)	781.6 ± 50.96	941.2 ± 37.71	0.022
Crypt depth (μm)	199.3 ± 8.20	260.5 ± 4.78	0.001
Villus height:crypt depth	3.94 ± 0.22	3.63 ± 0.18	0.298

and Rt. After the stabilization, the buffer solution in the mucosal side was replaced with 10 mmol/L of D-Glc instead of mannitol. The electrical response was measured as the peak response obtained 1 min after the addition of Glc. For Gln transport, the amino acid-containing buffer in the mucosal side was substituted for mannitol buffer (final concentration: 10 mmol/L) after the stabilization of tissue. Electrical response was observed as the peak response after 1 min of replacement of mucosal buffer with amino acids. Six jejunal strips (3 each for Glc and Gln, respectively) were prepared from individual birds in each set of experiment. Unless otherwise stated, all the chemicals used in the study were from Sigma-Aldrich, Munich, Germany.

Statistics

Statistical analyses were conducted with the Statistical Package for Social Science (SPSS for Windows Version 12, SPSS GmbH, Munich, Germany) to determine if variables differed between groups. The Kolmogorov-Smirnov test was used to test the normal distribution of the data before statistical analysis was performed. Results are expressed as means ± SE. For histomorphological parameters, an unpaired *t*-test was used between 2 groups. For electrical parameters (nutrient transport study), an unpaired *t*-test was used to find the difference between 2 groups before and after the addition of Glc and Gln, whereas a paired *t*-test was used to evaluate the effects of both substrates after their addition on Isc and Rt. Probability values of less than 0.05 ($P < 0.05$) were considered significant.

RESULTS

The BW of the control group and the inulin-fed group were comparable on d 0 (40.0 ± 0.75 g and 40.2 ± 0.65 g) and d 35 (1,798.5 ± 51.4 g and 1,809.1 ± 25.5 g). The villus height of the jejunal mucosa was higher ($P < 0.05$) for birds fed the diet supplemented with inulin compared with the control group (Table 2). Inulin supplementation also increased ($P < 0.01$) the crypt depth, however, no dietary effect was apparent for villus height:crypt depth for jejunal mucosa.

Inulin did not modify the basal Isc value, because no significant difference was noted between both groups. Addition of Glc in the mucosal compartment resulted in an increased ($P < 0.01$) Isc in both groups compared with respective basal Isc values. The tissues obtained from birds fed the inulin supplementation had a comparable increase of Isc after the addition of Glc as the jejunum from the control group (Table 3). Similarly, application of Gln in the mucosal side of the chamber also increased Isc ($P < 0.001$) compared with the basal value in both groups. The electrogenic current (Isc) induced by the mucosal application of Gln was not statistically different in both groups (Table 3). The basal value of Rt was lower ($P < 0.01$) in the inulin-fed group compared with control group (Table 4) before addition of either Glc or Gln (Table 4). The cumulative basal value of Rt was lower ($P < 0.001$) in the group supplemented with dietary inulin (69.0 ± 4.3Ω.cm^2) compared with the control group (119.6 ± 9.7Ω.cm^2) when data of each group (8 birds each) were pooled.

DISCUSSION

The present study shows changes in the mucosal architecture in terms of increased villus height and deeper crypts in birds fed with an inulin-supplemented diet. The intestinal mucosal architecture can reveal useful information on the intestinal function. Shorter villi have been associated with the presence of toxins such as deoxynivalenol (Awad et al., 2006). The nutrient absorption can be decreased due to shortening of the villi, whereas crypts can be regarded as the production site where stem cells divide to permit renewal of the villus. Deeper crypts indicate fast cellular turnover to permit renewal of the villus as needed in response to normal sloughing or inflammation from pathogens or their toxins and high demands for tissue (Yason et al., 1987; Anonymous, 1999). A shortening of the villi and deeper crypts may lead to poor nutrient absorption, increased secretion in the gastrointestinal tract, and lower performance (Xu et al., 2003). On the other hand, increased villus length means that more energy and nutrients would be required for faster turnover of the gut mucosa. Providing the extra nutrients for higher mucosal growth may be at the cost of energy required for performance. There is a dearth of reports regarding the influence of inulin on the histomorphology of the intestine in broilers. Results of the present study demonstrated that villus height and crypt depth were higher in the inulin-supplemented group (Table 2). Feeding of FOS (0.4%) has been reported to increase the ileal height, crypt depth, and villus height:crypt depth in broilers (Xu et al., 2003). Similarly, MOS has been found to increase the villus length of the small intestine in broilers (Sonmez and Eren, 1999; Iji et

Table 3. Effect of inulin supplementation on short-circuit current (μA/cm^2) of the jenunal mucosa in broilers before and after Glc or Gln addition (n = 8)

Parameters	Groups		Probability
	Control	Inulin	
Glc addition			
Basal	–1.3 ± 11.6	–2.6 ± 9.1	0.931
After Glc addition	20.0 ± 11.1	14.8 ± 9.2	0.726
Gln addition			
Basal	–1.3 ± 5.2	–1.4 ± 10.0	0.991
After Gln addition	19.0 ± 5.0	20.6 ± 10.4	0.887

Table 4. Effect of inulin supplementation on tissue resistance ($\Omega \cdot cm^2$) of the jenunal mucosa in broilers before and after Glc or Gln addition (n = 8)

Parameters	Groups		Probability
	Control	Inulin	
Glc addition			
Basal	139.2 ± 17.7	74.4 ± 6.1	0.004
After Glc addition	137.7 ± 19.6	73.2 ± 6.6	0.009
Gln addition			
Basal	108.3 ± 10.9	65.3 ± 5.9	0.003
After Gln addition	112.5 ± 12.2	70.4 ± 5.4	0.004

al., 2001). Kleessen et al. (2003) demonstrated that supplementation of dietary fructans increased the jejunal villus length and crypt depth in human-flora-associated rats without affecting germ-free rats.

The small and large intestines of domestic birds have high absorptive rates for water and electrolytes. The net movements of ions are responsible for the electrical current across the epithelium. In the intestine, there are 2 mechanisms for the absorption of D-Glc and amino acids, paracellular (passive diffusion) and transcellular transport (Garcia-Amado et al., 2005). In the former, no input of energy is required, whereas for the transcellular transport, energy is required for the absorption of Glc mediated by the Na-Glc cotransporter-1 and amino acids via carrier proteins in the apical and basolateral membranes of the epithelial cells (Pappenheimer, 1993). The chicken has capacity for transcellular transport of D-Glc and amino acids in the small intestine and colon (Dyer et al., 1997; Soriano and Planas, 1998; Garriga et al., 1999; Soriano-Garcia et al., 1999). The jejunum is the most efficient segment for Na-mediated uptake of Glc and amino acids through carrier proteins (Amat et al., 1996). Glucose and amino acids are absorbed from the intestinal lumen by active transport, coupled with Na^+. Addition of theses substrates to the mucosal side of the intestinal tissue stimulates the carrier-mediated transport proteins along with increased uptake of luminal Na^+. The brush border membrane depolarization and rise in cytosolic Na concentration stimulate the Na^+-K^+ adenosine triphosphatase, which, in turn, increases the net flux of Na from the luminal to the serosal side. These events modify the electrical variables of the intestinal tissues and increase Isc (Shimada and Hoshi, 1986; Wright et al., 1994, Amat et al., 1999; Awad et al., 2005; Garcia-Amado et al., 2005). The result of the present study reveals nonsignificant difference in basal Isc in both groups (Table 3). This is in accordance with the findings of Breves et al. (2001), who observed no effect on basal and forskolin-stimulated Isc in the jejunal mucosa of pigs supplemented with 2 different types of oligosaccharides. Nancy et al. (2003) also could not find differences in basal Isc for the jejunum and colon of pigs supplemented with different oligosaccharides compared with the control group. Our results showed that the jejunal transport of Glc by the brush border Na-Glc cotransporter was not significantly altered by the experimental diet (Table 3). As far as we know, no information exists regarding the influence of inulin on the electrical variables of the intestine in broilers. The results of this study are in agreement with Nancy et al. (2003), who demonstrated that mucosal application of Glc in the jejunal, ileal, and colon tissue of FOS-fed pigs did not induce any significant increase in Isc vs. the control group. In contrast, Breves et al. (2001) observed that Glc stimulation of Isc was higher ($P < 0.05$) in the oligosaccharide-fed groups in the proximal jejunum but not in the distal jejunum of pigs. It is an established fact that Gln absorbs Na^+ from the intestinal mucosa by 2 mechanisms, an electrogenic and a neutral absorption. However, the proportion of these 2 mechanisms is still unsettled (Abely et al., 2000). In our result, electrogenic Gln transport was not altered by inulin supplementation (Table 4). Nancy et al. (2003) demonstrated that electrogenic ileal Gln transport was not statistically different from control in pigs fed diets supplemented with FOS, methylcellulose, and soy polysaccharide. However, electrogenic Gln transport was higher ($P < 0.05$) in FOS and soy polysaccharide than in methylcellulose-fed pigs. Iji et al. (2001) observed that absorption of tryptophan by brush border membarne vesicles was not affected by dietary supplementation of MOS in the ileum, but it increased in the jejunum of broilers fed with higher dose of MOS.

The present study reveals that transmural resistance of jejunal tissue was lower in the inulin-supplemented group (Table 4). Nancy et al. (2003) noted lower transmucosal resistance in the ileum of pigs supplemented with FOS. However, resistance of colon and jejunal tissues remained unaffected by the dietary supplementation. Similary, Breves et al. (2001) also demonstrated that tissue conductance tended to be higher in the oligosaccharide-supplemented pigs than in control pigs. It has been found that in vitro incubation of different indigestible disaccharides increases the paracellular permeability of rat intestinal mucosa by opening the tight junction (Mineo et al., 2004). Fructooligosaccharides have been reported to impair the intestinal barrier in rats by increasing the intestinal permeability (Bruggencate et al., 2005). The decrease in the transmucosal Rt as induced by inulin might be due to the effect of prebiotic on transcellular pathways, paracellular pathways, or both. The reason behind this decreased transmucosal Rt as induced by the inulin is not clear. We are not sure whether inulin affected the transmucosal resistance in the jejunal mucosa directly or indirectly in association with the indigenous microflora and a higher production of short chain fatty acids. It could be due to the accumulation of organic acids or other bacterial fermentation metabolites, because those can cause irritation in the mucosal barrier that could lead to impaired intestinal barrier (Rémésy et al., 1993; Lin et al., 2002). In a previous study (Rehman et al., 2006b), we found that inulin elevated the jejunal lactate concentration that could be a cause for this decreased intestinal tissue resistance.

In conclusion, supplementation of inulin resulted in an increase in the villus height and crypt depth of the jejunal mucosa of broilers. Inulin failed to alter the Na-dependent absorption of Glc and Gln from jejunal mucosal flaps. The transmucosal resistance of inulin-fed birds was lowered.

Based on our findings, inulin affected the mucosal architecture but has no positive potential effects on electrogenic nutrient absorptions. Further study is needed to know how inulin affects the transmucosal tissue resistance.

REFERENCES

Abely, M., P. Dallet, M. Boisset, and J. F. Desjeux. 2000. Effect of cholera toxin on glutamine metabolism and transport in rabbit ileum. Am. J. Physiol. Gastrointest. Liver Physiol. 278:789–796.

Amat, C., J. A. Piqueras, J. M. Planas, and M. Moreto. 1999. Electrical properties of the intestinal mucosa of the chicken and the effects of luminal glucose. Poult. Sci. 78:1126–1131.

Amat, C., J. M. Planas, and M. Moreto. 1996. Kinetics of hexose uptake by the small and large intestine of the chicken. Am. J. Physiol. 271:R1085–R1089.

Anonymous. 1999. How do mannanoligosaccharides work? Feed Times 1:7–9.

Awad, W. A., J. Böhm, E. Razzazi-Fazeli, K. Ghareeb, and J. Zentek. 2006. Effect of addition of a probiotic microorganism to broiler diets contaminated with deoxynivalenol on performance and histological alterations of intestinal villi of broiler chickens. Poult. Sci. 85:974–979.

Awad, W. A., H. Rehman, J. Böhm, E. Razzazi-Fazeli, and J. Zentek. 2005. Effects of luminal deoxynivalenol and L-proline on electrophysiological parameters in the jejunums of laying hens. Poult. Sci. 84:928–932.

Breves, G., L. Szentkuti, and B. Schroder. 2001. Effects of oligosaccharides on functional parameters of the intestinal tract of growing pigs. Dtsch. Tierarztl. Wochenschr. 108:246–248.

Bruggencate, S. J. M. T., I. M. J. Bovee-Oudenhoven, M. L. G. Lettink-Wissink, and R. van der Meer. 2005. Dietary fructooligosaccharides increase intestinal permeability in rats. J. Nutr. 135:837–842.

Crittenden, G. R. 1999. Prebiotics. Pages 141–156 in Probiotics: A Critical Review. G. W. Tannock, ed. Horizon Scientific Press, Dunedin, New Zealand.

Dyer, J., A. Ritzhaupt, I. S. Wood, C. de la Horra, A. A. Ilundain, and S. P. Shirazi-Beechey. 1997. Expression of the Na^+/D-glucose co-transporter (SGLT1) along the length of the avian intestine. Biochem. Soc. Trans. 25:480S.

Garcia-Amado, M. A., J. R. Del Castillo, M. E. Perez, and M. G. Dominguez-Bello. 2005. Intestinal D-glucose and L-alanine transport in Japanese quail (*Coturnix coturnix*). Poult. Sci. 84:947–950.

Garriga, C., N. Rovira, M. Moretó, and J. M. Planas. 1999. Expression of Na^+-D-glucose cotransporter in brush-border membrane of the chicken intestine. Am. J. Physiol. 276:R627–R631.

Iji, P. A., A. A. Saki, and D. R. Tivey. 2001. Intestinal structure and function of broiler chickens on diets supplemented with a mannanoligosaccharide. J. Food Sci. Agric. 81:1186–1192.

Kleessen, B., L. Hartmann, and M. Blaut. 2003. Fructans in the diet cause alterations of intestinal mucosal architecture, released mucin and mucosa-associated bifidobacteria in gnotobiotic rats. Br. J. Nutr. 89:597–606.

Lin, J., S. M. Nafday, S. N. Chauvin, M. S. Magid, S. Pabbatireddy, I. R. Holzman, and M. W. Babyatsky. 2002. Variable effects of short chain fatty acids and lactic acid in inducing intestinal mucosal injury in newborn rats. J. Pediatr. Gastroenterol. Nutr. 35:545–550.

Mineo, H., M. Amano, H. Chiji, N. Shigematsu, F. Tomita, and H. Hara. 2004. Indigestable disaccharides open tight junctions and enhance net calcium, magnesium, and zinc absorption in isolated rat small and large intestinal epithelium. Dig. Dis. Sci. 49:122–132.

Nancy, J., C. Matos, S. M. Donovan, R. E. Isaacson, H. R. Gaskins, B. A. White, and K. A. Tappenden. 2003. Fermentable fiber reduces recovery time and improves intestinal function in piglets following *Salmonella typhimurium* infection. J. Nutr. 133:1845–1852.

Pappenheimer, J. R. 1993. On the coupling of membrane digestion with intestinal absorption of sugar and amino acids. Am. J. Physiol. 265:G409–G417.

Pluske, J. R., M. J. Tompson, C. S. Atwood, P. H. Bird, I. H. Williams, and P. E. Hartmann. 1996. Maintenance of villus height and crypt depth, and enhancement of disaccharide digestion and monosaccharide absorption, in piglets fed on cows' whole milk after weaning. Br. J. Nutr. 76:409–422.

Rada, V., D. Duskova, M. Marounek, and J. Petr. 2001. Enrichment of *Bifidobacteria* in the hen caeca by dietary inulin. Folia Microbiol. (Praha) 46:73–75.

Rehman, H., W. A. Awad, I. Lindner, M. Hess, and J. Zentek. 2006a. *Clostridium perfringens* α toxin affects electrophysiological properties of isolated jejunal mucosa of laying hens. Poult. Sci. 85:1298–1302.

Rehman, H., J. Böhm, and J. Zentek. 2006b. Effects of diets with sucrose and inulin on the microbial fermentation in the gastrointestinal tract of broilers. Page 155 in Proc. Soc. Nutr. Physiol., Göttingen, Germany. DLG-Verlag GmbH, Frankfurt, Germany.

Rémésy, C., M. A. Levrat, L. Gamet, and C. Demigné. 1993. Cecal fermentations in rats fed oligosaccharides (inulin) are modulated by dietary calcium level. Am. J. Physiol. 264:G855–G862.

Shimada, T., and T. Hoshi. 1986. A comparative study of specificity of the intestinal Na^+/sugar cotransport among vertebrates. Comp. Biochem. Physiol. A 84:365–370.

Sonmez, N. W., and M. Eren. 1999. Effects of supplementation of zinc bacitracin, mannan-oligosaccharides and probiotic into the broiler feeds on morphology of the small intestine. Vet. Fak. Dergisi Uludag Univ. 18:125–138.

Soriano, E., and J. M. Planas. 1998. Developmental study of methyl-D-glucoside and L-proline uptake in the small intestine of the White Leghorn chicken. Poult. Sci. 77:1347–1353.

Soriano-Garcia, J. F., M. Torras-Llort, M. Moretó, and R. Ferrer. 1999. Regulation of L-methionine and L-lysine uptake in chicken jejunal brush border by dietary methionine. Am. J. Physiol. 277:R1654–R1661.

Wright, E. M., B. A. Hirayama, D. D. F. Loo, E. Turk, and K. Hager. 1994. Intestinal sugar transport. Pages 1751–1766 in Physiology of the Gastrointestinal Tract. 3rd ed. L. R. Johnson, ed. Raven Press, New York, NY.

Xu, Z. R., C. H. Hu, M. S. Xia, X. A. Zhan, and M. Q. Wang. 2003. Effects of dietary fructooligosaccharide on digestive enzyme activities, intestinal microflora and morphology of male broilers. Poult. Sci. 82:1030–1036.

Yason, C. V., B. A. Summers, and K. A. Schat. 1987. Pathogenesis of rotavirus infection in various age groups of chickens and turkeys: Pathology. Am. J. Vet. Res. 6:927–938.

Yusrizal, Y., and T. C. Chen. 2003. Effect of adding chicory fructans in feed on faecal and intestinal microflora and excretory volatile ammonia. Int. J. Poult. Sci. 2:188–194.