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Effects of encapsulated and non-encapsulated compound acidifiers on gastrointestinal pH and intestinal morphology and function in weaning piglets*

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ABSTRACT

The experiment was conducted to study the effects of the addition of encapsulated and non-encapsulated compound acidifiers in a diet based on maize-soyabean meal-extruded soyabean on gastrointestinal pH, growth performance, villus height and crypt depth of jejunum, intestinal digestive enzymes activities, microbial population and intestinal mucosal secretory immunoglobulin A in weaning piglets. Sixty-four 28-day-old, crossbred piglets (Landrace×Large White), weighing an average of 7.00±0.10 kg, were randomly assigned to four treatments with four replicates and four piglets (2 male and 2 female) per pen, according to single-factor design principle. The feeding trial lasted 35 days. The results showed that encapsulated compound acidifiers significantly reduced the gastrointestinal pH ($P<0.01$), and improved the average daily gain and the feed conversion ratio ($P<0.05$), but they had no significant effect on the average daily feed intake. In addition, encapsulated compound acidifiers significantly increased the ratio between the villus height and crypt depth of jejunum ($P<0.01$), and stimulated the sucrase activity and lactase activity ($P<0.05$) as well; during the later weaning period, encapsulated compound acidifiers significantly increased the counts of *Lactobacillus* and decreased the counts of *Escherichia coli* in the caecum and the colon ($P<0.01$); it was also noted that there was an insignificant tendency of lower secretion of intestinal mucosal secretory IgA ($P>0.05$). These results indicate that the encapsulated compound acidifiers improve the intestinal morphology and function by reducing the gastrointestinal pH, so as to enhance the intestinal adaptation and immunity, and consequently improve the growth performance of weaning piglets.

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KEY WORDS: piglets, encapsulated compound acidifiers, gastrointestinal pH, intestinal morphology, intestinal function

INTRODUCTION

Extensive studies have shown the post-weaning stress syndrome in piglets is related to their increased gastrointestinal pH values (Cranwell and Moughan, 1989). In recent years, there have been many research reports on solving the difficult problem of post-weaning stress syndrome by means of the addition of acidifiers in piglets' diets. Most acidifiers referred to in these reports were unprotected, and so could be buffered easily by some feedstuffs, or absorbed in the digestive tract before serving as acidification (Canibe et al., 2001). Therefore, the addition of these acidifiers mainly increased stomach acidity, but no further effect was found in the intestinal tract. Research showed that higher pH of the intestinal tract in weaning piglets would reduce the activity of intestinal digestive enzymes and cause the proliferation of pathogens, which resulted in the weaning piglets' trophism diarrhoea (Sutton et al., 1996). For this reason, some scholars proposed that the acidifiers should be protected by encapsulating so that the acidification could be extended to the intestinal tract. To date, there hasn't been any research reported on the effects of encapsulated acidifiers in weaning piglets. The present experiment was conducted to study the effects of microcapsule slow-release compound acidifiers encapsulated by fatty acids on growth performance and intestinal function in weaning piglets, and hence provides some evidence for regulating the piglets' intestinal adaptation and intestinal health by more effective use of acidifiers.

MATERIAL AND METHODS

Design of the trial

Altogether 64 crossbred (Landrace × Large White) pigs weaned at the age of 28 days, weighing an average of 7.00±0.10 kg, were randomly assigned to four treatments with four replicates and four pigs (2 male and 2 female) per pen, according to the single-factor design principle. The pigs were raised for 5 weeks after 3 days' adaptation. The weaning piglets were fed different diets at the stage of 1-3 and of 4-5 weeks, respectively. The design of the trial is shown in Table 1.

Table 1. The design of the trial

Treatment	1-3 weeks
control group 1	Basal diet
control group 2	Basal diet + 0.10% control acidifiers
trial group 1	Basal diet + 0.10% encapsulated acidifiers
trial group 2	Basal diet + 0.05% encapsulated acidifiers
Treatment	4-5 weeks
control group 1	Basal diet
control group 2	Basal diet + 0.10% control acidifiers
trial group 1	Basal diet + 0.05% encapsulated acidifiers
trial group 2	Basal diet + 0.03 % encapsulated acidifiers

Materials

The encapsulated acidifier used for weaning piglets in trial groups was a microcapsule compound, and the active ingredients were fumaric acid, lactic acid, citric acid and malic acid. These were encapsulated by fatty acids, so that the microcapsule compound acidifiers had a slow-release effect. The acidifier used for weaning piglets in control group 2 was a non-encapsulated compound but the main components were the same as the trial acidifier.

Diets and feeding managament

The basal diets used in the experiment on maize-soyabean meal-extruded soyabean basis , for both the earlier stage (1-3 weeks) and the later stage (4-5 weeks), were formulated to meet nutrient requirements of piglets (NRC, 1998), feeding standard of pig of China in 2004. The composition and nutrient levels of diets are presented in Table 2.

Pigs were raised in nursery pens with expanded metal floors. The nurseries were kept at a controlled temperature (28°C at the beginning and 25°C at the end of the experiment, with a decrease of 1°C every week during the first 3 weeks), and the relative humidity remained between 50-70% throughout the experiment. Piglets were fed at 8.00, 12.00, 16.00 and 20.00 every day. During the experimental period, the piglets were allowed *ad libitum* access to feed and water. The nurseries were cleaned every day and disinfected every four days.

Slaughter and sample collection

On d 14 and 35, one pig (the closest to the mean body weight in the pen) was selected from each replicate, allowed access to their meal for 1 h and then excluded from the trough for the last hour before killing. Piglets were bled, the abdomen

Table 2. Composition and nutrient levels of diets, (air-dry basis), %

Ingredient	1-3 weeks	4-5 weeks	Nutrient	Nutrient levels 1-3 wks	4-5 wks
Maize	59.91	63.40	Digestible enery	14.23	14.18
Soyabean meal (sol.,44%)	12.00	14.00	Crude protein	20.33	19.05
Extruded soyabean	15.00	11.00	Ca	0.81	0.81
Fish meal	6.00	3.00	Available P	0.41	0.40
Wheat bran	2.50	3.00	D-lysine	1.19	1.10
Soyabean oil	1.50	2.00	D-methionine	0.42	0.37
L-lysine-HCl	0.30	0.38	D-tryptophan	0.22	0.20
DL-methionine	0.10	0.08	D-threonine	0.74	0.69
Threonine	0.07	0.07	D-leucine	0.67	0.60
Limestone	0.95	0.95			
Calcium phosphate tribasic	0.50	0.95			
Choline chloride	0.06	0.06			
Vitamin premix	0.05	0.05			
Salt	0.30	0.30			
Trace mineral premix	0.50	0.50			
Feed antibiotics	0.06	0.06			
Acidifiers	0.20	0.20			
Total	100.00	100.00			

[1] fish meal is made in China. Calcium phosphate tribasic contains two molecules of crystal water. The purity of choline chloride 50%; [2] the stage of 1-3 weeks, provided per kg of diet, IU: vit. A 2200, vit D_3 220, vit. E 16.00; mg: vit. K_3 0.50, vit. B_1 1.50, vit. B_2 4.00, vit. B_6 2.00, niacin 20.00, calcium pantothenic12.00, folic acid 0.30, biotin 0.08; µg: vit. B_{12} 20.00. The stage of 4-5 weeks, provided per kg of diet, IU: vit. A 1800, vit. D_3 200, vit. E 11.00; mg; vit. $K_3$0.50, vit. B_1 1.00, vit. B_2 3.50, vit. B_6 1.50, niacin 15.00, calcium pantothenic10.00, folic acid 0.30; biotin 0.05; µg; vit. B_{12} 17.50; | [3] provided per kg of diet; mg: Fe 105, Cu 6.00, Mn 4.00, Zn 110, I 0.14, Se 0.30; [4] the two kinds of feed antibiotics are against gram-positive bacteria and gram-negative bacteria, respectively; [5] the acidifiers are compound acidifiers used by each treatment and the maize was supplemented

opened immediately from sternum to pubis, and the whole gastrointestinal tract was removed, tied off, and sampled. Samples of the gut content were collected from the stomach, duodenum, jejunum and ileum, and stored at -20°C for pH and enzyme activity measurement. Samples for histological study were obtained from the proximal jejunum wall, 75 cm from the stomach. The samples were cut open longitudinally along the mesenteric attachment and fixed by immersing in 10% buffered formalin immediately after slaughter. Three intestinal samples (50 cm in length) were taken from the duodenum, jejunum and ileum, respectively, the mucosa of which were scraped with glass microscope slides and the secretions were stored at -70°C for the IgA measurements. A caecum portion and a colon portion (25 cm in length), were tied off and collected for *Lactobacilli* and *Escherichia coli* counts. The intestinal portions were stored at 4°C until the culture was completed.

Measurements and analysis

Pigs were weighed before feeding and feed consumption was measured at the beginning and at the end of the experiment. Feed wastage was collected each day and taken into account in the calculation of feed consumption and feed conversion ratio (FCR). FCR was calculated by the average daily gain (ADG) and the average daily feed intake (ADFI) of the piglets.

The pH of gastrointestinal content was measured by pH meter (PHS-3C, Shanghai; degree of accuracy ±0.01).

Jejunum samples for histological study were dehydrated and embedded in paraffin wax, sectioned at 5 μm, and stained with haematoxylin and eosin. Ten villous height (VH) and 10 crypt depth (CD) measurements were taken from each section. The average of the 10 VH and CD measurements was treated as a single experimental unit.

For analysis of sucrase and lactase, the mucosal sample (0.2-1.0 g) was diluted by distilled water (m/V=1/9), and then centrifugated at 2000 rpm for 10 min at 4°C, taking the supernatant fraction upon completion. The supernate was stored at -20°C until measured by a kit purchased from Nanjing Jian Cheng Bioengineering Institute.

For bacterial counts, one gram of sample was weighed, serially diluted, and 100 μl aliquots were plated in MRS for *Lactobacilli* counts (dilutions 10^{-5} to 10^{-7}) and in EMB for *Escherichia coli* counts (dilutions 10^{-4} to 10^{-6}). *Lactobacilli* were counted after a 48 h incubation (37°C, 5% CO_2) and *Escherichia coli* after a 24 h incubation (37°C).

Phosphate buffered solution (0.01 mol/l, pH=7.2) was added to the mucosal sample (m/V=1/3) for 24 h at 4°C, and then the sample was centrifuged at 10000 rpm for 5 min at 4°C, taking the supernatant fraction upon completion (Mukiibi-Muka and Jones, 1999). The supernate was stored at -70°C until measured of IgA by transmission turbidimetry kit.

Statistical analysis

The obtained data were statistically analysed by using SPSS11.5 software. Differences among means were tested by using Duncan's multiple range test. The results are shown by mean ± SD.

RESULTS

The growth performance of weaning piglets is shown in Table 3. The average daily gain (ADG) was increased by 12.75% and the feed intake/gain (F/G) was

Table 3. Effects of compound acidifiers on growth performance in weaning piglets, g

Items	Control group 1	Control group 2	Trial group 1	Trial group 2
Initial weight	6989 ± 9	7028 ± 8	7001 ± 11	6971 ± 9
Final weight	17699 ± 700	15918 ± 918^{Aa}	19076 ± 924^{B}	18451 ± 1286^{b}
1-5 weeks ADG	306 ± 7^{Aa}	254 ± 14^{B}	345 ± 17^{Ab}	328 ± 8^{Aa}
1-5 weeks ADFI	465 ± 56	442 ± 12	497 ± 12	487 ± 6
1-5 weeks F/G	1520 ± 15^{Aa}	1740 ± 14^{B}	1441 ± 11^{Ab}	1485 ± 8^{Aa}

in the same row, values with the same letter superscripts indicate no difference (P>0.05), but different small-letter superscripts indicate significant difference (P<0.05), and different capital superscripts indicate highly significant difference (P<0.01), the same is used below; ADG - average daily gain; ADFI - average daily feed intake; F/G - feed intake/ gain (FCR)

reduced by 5.20% for piglets in trial group 1 when compared to the piglets in control group 1 (P<0.05). The ADG and F/G in trial group 2 showed an increase of 7.19% and a reduction of 2.30%, respectively, to those in control group 1 (P>0.05). Control acidifiers caused a greater reduction in ADG and FCR of piglets as compared to no acidifiers (P<0.01). However, there were no differences in feed intake of piglets among the four treatments (P>0.05).

Two weeks and five weeks after weaning, the pH of gastrointestinal content (Table 4) was significantly lower for piglets in trial groups than that for piglets in control groups (P<0.01). The pH measurements showed differences between control groups only in the stomach and duodenum but not in the jejunum and ileum after weaning for 2 weeks (P<0.01).

Table 4. Effects of compound acidifiers on gastrointestinal pH in weaning piglets

Items	Control group 1	Control group 2	Trial group 1	Trial group 2
2-week				
stomach	4.17 ± 0.02^{A}	3.59 ± 0.21^{Ba}	3.37 ± 0.05^{Bb}	3.46 ± 0.01^{Bb}
duodenum	5.02 ± 0.04^{A}	4.81 ± 0.06^{Ba}	4.68 ± 0.04^{Bb}	4.79 ± 0.01^{Ba}
jejunum	6.17 ± 0.02^{A}	6.10 ± 0.04^{A}	5.87 ± 0.03^{Ba}	5.95 ± 0.04^{Bb}
ileum	6.25 ± 0.07^{A}	6.24 ± 0.06^{A}	6.02 ± 0.02^{Ba}	6.11 ± 0.03^{Bb}
5-week				
stomach	4.50 ± 0.04^{A}	4.48 ± 0.04^{A}	3.50 ± 0.09^{B}	3.60 ± 0.04^{B}
duodenum	5.55 ± 0.03^{A}	5.57 ± 0.02^{A}	4.89 ± 0.06^{Ba}	5.05 ± 0.01^{Bb}
jejunum	6.50 ± 0.03^{A}	6.57 ± 0.01^{A}	6.08 ± 0.08^{Ba}	6.20 ± 0.01^{Bb}
ileum	6.63 ± 0.04^{A}	6.68 ± 0.04^{A}	6.15 ± 0.04^{Ba}	6.25 ± 0.01^{Bb}

^{a,b} - P<0.05; ^{A,B} P<0.01

The villus height (VH), crypt depth (CD) and the ratio between the villus height and crypt depth of jejunum in weaning piglets are shown in Table 5. Two weeks and five weeks after weaning, trial acidifiers caused a greater improvement in VH, CD and VH/CD of piglets than no acidifiers (P<0.01). But control acidifiers caused a worse effect in VH, CD and VH/CD of piglets as compared to no acidifiers (P<0.05).

Table 5. Effects of compound acidifiers on villus height (VH) and crypt depth (CD) of jejunum in weaning piglets, μm

Items	Control group 1	Control group 2	Trial group 1	Trial group 2
2-week				
VH	279.4 ± 37.0^{a}	274.2 ± 11.7^{a}	305.9 ± 26.7^{b}	291.9 ± 18.1^{b}
CD	147.4 ± 14.7^{a}	152.4 ± 10.4^{a}	118.6 ± 13.3^{b}	129.3 ± 19.6^{b}
VH/CD	1.9 ± 0.2^{Aa}	1.8 ± 0.2^{Ab}	2.6 ± 0.4^{Ba}	2.3 ± 0.2^{Bb}
5-week				
VH	247.8 ± 28.4^{Aa}	230.7 ± 17.8^{Ab}	281.9 ± 12.5^{Ba}	265.9 ± 10.4^{Bb}
CD	166.5 ± 31.2^{Aa}	178.2 ± 26.5^{Ab}	129.5 ± 19.6^{Ba}	142.6 ± 22.0^{Bb}
VH/CD	1.5 ± 0.1^{Aa}	1.3 ± 0.2^{Ab}	2.2 ± 0.2^{Ba}	1.9 ± 0.1^{Bb}

^{a,b} - $P<0.05$; ^{A,B} - $P<0.01$

Two weeks and five weeks after weaning, when compared to no acidifiers, the addition of trial acidifiers increased the intestinal sucrase activity and lactase activity for piglets in trial groups ($P<0.05$), while the intestinal sucrase activity and lactase activity for piglets in control group 2 were unaffected by the addition of control acidifiers ($P>0.05$) (Table 6).

Table 6. Effects of compound acidifiers on intestinal enzymes activities in weaning piglets, U/mg protein

Items	Control group 1	Control group 2	Trial group 1	Trial group 2
2-week				
sucrase activity	391.4 ± 37.0^{a}	382.8 ± 51.7^{a}	447.9 ± 56.7^{b}	428.9 ± 48.1^{b}
lactase activity	436.4 ± 31.7^{a}	413.4 ± 56.4^{a}	481.6 ± 39.3^{b}	468.6 ± 19.6^{b}
5-week				
sucrase activity	1872.8 ± 98.4^{Aa}	1776.7 ± 107.8^{Ab}	2165.9 ± 92.5^{Ba}	2088.9 ± 100.4^{Bb}
lactase activity	140.5 ± 24.2^{a}	122.2 ± 30.5^{a}	179.5 ± 28.6^{b}	152.4 ± 22.0

^{a,b} - $P<0.05$; ^{A,B} - $P<0.01$

Five weeks after weaning, the *Lactobacillus* counts in caecum and colon for piglets in trial groups were more than that for piglets in control group 1 and the *E. coli* counts in caecum and colon were less than that for piglets in control group 1 ($P<0.01$) (Table 7).

The secretion of intestinal mucosal secretory IgA (sIgA) in weaning piglets is shown in Table 8. Two weeks and five weeks after weaning, the addition of trial acidifiers resulted in the increase of secretion of sIgA in duodenum, jejunum and ileum as compared to no acidifiers ($P>0.05$). While 5 weeks after weaning, the addition of control acidifiers resulted in the reduction of secretion of sIgA in intestine as compared to no acidifiers ($P>0.05$).

Table 7. Effects of compound acidifiers on microbial population in weaning piglets

Items	Control group 1	Control group 2	Trial group 1	Trial group 2
2-week				
caecum *Lactobacillus* /10^9 CFU/g	1.65 ± 0.03^{a}	1.34 ± 0.47^{a}	3.93 ± 1.02^{b}	3.12 ± 1.25
colon *Lactobacillus* /10^9CFU/g	2.51 ± 0.69	1.94 ± 0.43^{a}	4.27 ± 0.81^{b}	4.21 ± 0.76^{b}
caecum *E. coli*/10^7CFU/g	1.79 ± 0.08	2.85 ± 0.71^{a}	0.95 ± 0.07^{b}	1.39 ± 0.62^{b}
colon *E. coli*/10^7CFU/g	1.40 ± 0.14^{a}	2.82 ± 0.74^{a}	0.48 ± 0.21^{b}	0.56 ± 0.32^{b}
5-week				
caecum *Lactobacillus* /10^9CFU/g	1.20 ± 0.03^{A}	0.98 ± 0.01^{A}	2.25 ± 0.14^{Ba}	1.94 ± 0.21^{Bb}
colon *Lactobacillus* (10^9CFU/g	1.34 ± 0.03^{A}	1.09 ± 0.01^{A}	2.84 ± 0.06^{Ba}	2.27 ± 0.05^{Bb}
caecum *E. coli* /10^6CFU/g	5.89 ± 0.04^{A}	6.14 ± 0.07^{A}	2.31 ± 0.04^{Ba}	2.94 ± 0.03^{Bb}
colon *E. coli* /10^6CFU/g	3.15 ± 0.13^{Aa}	3.77 ± 0.03^{Ab}	1.04 ± 0.02^{B}	1.14 ± 0.02^{B}

^{a,b} - $P<0.05$; ^{A,B} - $P<0.01$

Table 8. Intestinal mucosal secretory IgA in 5-week, g/l

Items	Control group 1	Control group 2	Trial group 1	Trial group 2
2-week				
duodenum	0.24 ± 0.02	0.25 ± 0.06	0.26 ± 0.02	0.26 ± 0.05
jejunum	0.25 ± 0.03	0.25 ± 0.03	0.28 ± 0.01	0.26 ± 0.06
ileum	0.25 ± 0.02	0.25 ± 0.01	0.28 ± 0.03	0.27 ± 0.02
5-week				
duodenum	0.30 ± 0.04	0.28 ± 0.04	0.33 ± 0.05	0.32 ± 0.01
jejunum	0.25 ± 0.04	0.25 ± 0.03	0.28 ± 0.02	0.26 ± 0.02
ileum	0.25 ± 0.03	0.25 ± 0.07	0.28 ± 0.05	0.27 ± 0.04

^{a,b} - $P<0.05$; ^{A,B} - $P<0.01$

DISCUSSION

The appropriate acidity within the digestive tract is an essential factor to maintain the normal function of digestive systems for weaning piglets. It is generally demonstrated that the digestive systems are immature and the neuroendocrine regulation mechanisms are not established completely in the early weaning piglets, thus the pH of gastrointestinal tract in piglets weaned for 3-4 weeks is higher than that in piglets before weaning (Funderburke and Seerley, 1990). Research shows that the addition of acidifiers in diet causes the reduction of gastrointestinal pH in weaning piglets. Burnell et al. (1988) reported that the addition of 1% citric acid in diet resulted in a lower pH of the stomach content and jejunum content in weaning piglets as compared to no acidifiers. Risley et al. (1992) obtained the same results when 1.5% fumaric acid was supplemented to the diet of the weaning piglets. But Manzanilla et al. (2004) found the addition

of acidifiers did not affect the pH of gastrointestinal tract in weaning piglets. In this experiment as well, the addition of control acidifiers did not increase the acidity of gastrointestinal pH in weaning piglets. One possible reason is that most acidifiers could be buffered easily by some feedstuffs, or absorbed in the stomach before reaching the intestinal tract. An unhealthy intestinal tract often causes the problem of post-weaning stress syndrome characterized by diarrhoea and seriously restricts the production potential of the piglets. In order to extend the acidity to the intestinal tract, the acidifiers can be encapsulated by lipids. Bosi et al. (1999) reported that the addition of acidifiers encapsulated by free fatty acids could reduce the pH of the stomach, ileum and caecum and increase the ADG and ADFI in weaning piglets. In this experiment, the addition of microcapsule compound acidifiers significantly reduced the gastrointestinal pH and increased the ADG and FCR in weaning piglets. These results demonstrate that the use of microcapsule compound acidifiers, as compared to other free acids, results in greater growth performance of weaning piglets by way of adjusting the gastrointestinal acidity.

At weaning time, the small intestine of piglets generally showed a reduction in villous height and an increase in crypt depth because of physical damage by solid diet. Both VH and CD are important indicators of the digestive health of the piglet and directly related to the absorptive capacity of the mucous membrane (Buddle and Bolton, 1992). From a theoretical point of view, VH reflects a balance between the mitotic activity of the crypt enteric cells and the desquamation produced principally by external aggressors (Nabuurs, 1995). Lupton et al. (1999) asserted that cell growth was affected by acidity and a higher pH was beneficial to the mitotic activity of the cells. In this experiment, the addition of microcapsule compound acidifiers significantly improved the VH and CD of jejunum, and significantly increased the ratio between the villus height and crypt depth. This may contribute to the reduction of intestinal pH. Others have pointed out that the VH and CD of the intestine are related to the sucrase activity and lactase activity of the striated border (Hampson and Kidder, 1986). Sucrase and lactase are the most important carbohydrases in the intestines of piglets. The acidity of intestinal content affects the secretion and activities of digestive enzymes. Many digestive enzymes are most active in an acidic environment. For example, the optimum pH of disaccharidase activities in intestine of piglets is usually between 6.1 and 6.2. As shown in this experiment, the addition of microcapsule compound acidifiers significantly increased the gastrointestinal acidity. The microcapsule compound acidifiers could stimulate the sucrase activity and lactase activity as well. Therefore, it can be inferred that the addition of microcapsule compound acidifiers increases the activities of digestive enzymes and improves the intestinal morphology by means of reducing the gastrointestinal pH in weaning piglets.

It is generally accepted that the largest microbial population of the pig

is localized in the large intestine. There are different reports on the effects of acidifiers on intestinal microbial population in weaning piglets. Franco et al. (2005) reported that addition of acidifiers resulted in a reduction of pathogenic bacteria in the intestine of piglets. This experiment showed that microcapsule compound acidifiers increased the counts of *Lactobacillus* and decreased the counts of *Escherichia coli* in caecum and colon of piglets during the later weaning period and the effects in trial group 1 were better than that in trial group 2. This may due to the fact that the addition of microcapsule compound acidifiers reduced the stomach pH and intestine pH to 3.5-6.0, which could stunt the growth of pathogenic bacteria, but allow the growth of *Lactobacilli* in the intestinal tract. It is generally accepted that *Lactobacilli* can inhibit the colonization and proliferation of *E. coli* by blocking the sites of adhesion or by producing lactic acid and other metabolites which lower the pH and inhibit *E. coli* (Fuller, 1977). Moreover, nondissociated (non-ionized, more lipophilic) organic acids can penetrate the bacterial cell wall and disrupt the normal physiology of certain types of bacteria (Gauthier, 2002). But Bolduan et al. (1988) found the addition of acidifiers did not affect the counts of intestinal microbial population in weaning piglets. In this experiment, control acidifiers did not improve the microflora of intestine in weaning piglets either. This was possibly because the addition of control acidifiers did not reduce pH value in intestinal tract.

The stimulation of the secretory IgA (sIgA) system and the local production of IgA antibody are recognized as one of the first line of defense against mucosal infections (Schat and Myers, 1991). sIgA, as the principal antibody in the secretions that bathes the mucosal surfaces, can provide immune protection to offspring and may promote development of neonatal immune competence (Kelleher and Lonnerdal, 2001). It is resistant to proteolytic enzymes and plays an essential role in the immunological defense against gastrointestinal infections. Bosi et al. (2007) found the addition of fat protection of calcium formate did not affect the mucosal secretion of sIgA in jejunum of weaning piglets. In this experiment, the addition of microcapsule compound acidifiers increased the intestinal mucosal secretion of sIgA in weaning piglets. One possible reason is that the microcapsule compound acidifiers significantly reduces the intestinal tract pH and improves the microflora of intestine in weaning piglets as well, thus it stimulates the secretion of sIgA of the IgA plasma cells in the intestine. But the control acidifiers caused a reduction of the secretion of sIgA in weaning piglets. All of the different study results suggest that there exist complicated intestinal mucosal immune systems in weaning piglets and the concrete mechanism of action on the secretion of sIgA remains to be further investigated.

CONCLUSIONS

Encapsulated compound acidifiers play an important part in reducing the pH value in the intestinal tract. The slow-release compound acidifiers improve the intestinal morphology and function by increasing the acidity in the gastrointestinal tract, so as to enhance the intestinal adaptation and immunity, and improve the growth performance in weaning piglets.

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