The Effects of Dietary Mannanoligosaccharides on Cecal Parameters and the Concentrations of Enteric Bacteria in the Ceca of Salmonella-Challenged Broiler Chicks

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ABSTRACT The ability of different enteric pathogens and coliforms to trigger agglutination of yeast cells (Saccharomyces cerevisiae,NCYC 1026) and a yeast cell wall preparation (MOS) was examined. Five of seven strains of Escherichia coli and 7 of 10 strains of Salmonella typhimurium and Salmonella enteritidis agglutinated MOS and Sac. cerevisiae cells. Strains of Salmonella choleraesuis, Salmonella pullorum, and Campylobacter did not lead to agglutination. Two strains that agglutinated MOS (S. typhimurium 29E and Salmonella dublin) and one nonagglutinating strain (S. typhimurium 27A) were selected as challenge organisms for in vivo studies in chicks under controlled conditions.

In a series of three trials in which 3-d-old chicks were orally challenged with $10^4$ cfu of S. typhimurium 29E, birds receiving 4,000 ppm of dietary MOS had reduced cecal S. typhimurium 29E concentrations (5.40 vs 4.01 log cfu/g; $P < 0.05$) at Day 10. In a second series of three trials with S. dublin as challenge organism, the number of birds that tested salmonella positive in the ceca at Day 10 was less when MOS was part of the diet (90 vs 56%; $P < 0.05$). To test the effect of MOS on concentrations of bacteria that do not express Type 1 fimbriae, a challenge trial was conducted with S. typhimurium 27A. However, strain 27A did not colonize the birds sufficiently to evaluate whether MOS affected its cecal concentration. Mannanoligosaccharide did not significantly reduce the concentrations of cecal coliforms ($P < 0.10$) although they were numerically lower. It had no effect on cecal concentrations of lactobacilli, enterococci, anaerobic bacteria, lactate, volatile fatty acid, or cecal pH.

(Key words: Salmonella, chicken, mannans, yeast, ceca)

INTRODUCTION

Newly hatched birds lack complex gastrointestinal microflora and are especially prone to colonization of enteric pathogens (Milner and Shaffer, 1952). However, the application of cultures of cecal microflora obtained from healthy adult birds (competitive exclusion cultures) to the newly hatched chick has been shown to increase the complexity of their gastrointestinal microflora and give the newly hatched chick has been shown to increase the complexity of their gastrointestinal microflora and give protection against enteric pathogens (Nurmi and Rantala, 1973; Stavric et al., 1985; Corrier et al., 1995). Many enteric pathogens must attach to the mucosal surface of the gut wall to establish themselves in the gastrointestinal tract (Costerton et al., 1978, Fuller et al., 1981). One mode of action of competitive exclusion (CE) cultures to exclude pathogens is related to their ability to increase the competition for attachment sites (Schneitz et al., 1993). Because attachment is often mediated through binding of bacterial lectins to receptors containing D-mannose (Eshdat et al., 1978), it may be possible to block the lectins with mannose or similar sugars and to inhibit bacterial attachment.

Oyofo et al. (1989a) tested the effect of different sugars on the in vitro adherence of Salmonella typhimurium to epithelial cells from 1-d-old chicks. They reported inhibition of adherence by methyl-a-D-mannoside and mannose by more than 90%. The inhibition of the adherence of S. typhimurium by mannose (Oyofo et al., 1989a) is reflected in decreased colonization of S. typhimurium when mannose is added to the diet of young chicks (Oyofo et al., 1989b). Because relatively high concentrations of mannose are required to control colonization of pathogenic bacteria, the cost of using pure mannose in commercial production is prohibitive even for short periods. However, mannose-based carbohydrates occur naturally...
in many products, such as yeast cell walls or different gums, which are available at reasonable prices. The hypothesis tested in this study was that mannooligosaccharides (MOS) from yeast cell walls are able to decrease the concentrations of enteric pathogens that express Type 1 fimbiae in poultry.

The objectives of these studies were to screen different bacterial strains for their ability to agglutinate MOS in yeast cell preparations and to determine the effect of MOS on cecal fermentation parameters, cecal microflora, and enteric pathogen and coliform colonization in chicks under controlled conditions.

MATERIALS AND METHODS

Mannooligosaccharides

The MOS preparation used in these studies was the commercial product Bio-Mos. The product contains yeast cell wall fragments derived from Saccharomyces cerevisiae. The cell wall fragments are obtained by centrifugation from a lysed yeast culture. The pellet containing the yeast cell wall fragments is then washed and spray dried.

Agglutination of Yeast Products

The ability of the bacteria to adhere to the mannan associated with the yeast cell wall was measured with an agglutination test. Agglutination of MOS (yeast cells) by different enteric pathogens and coliforms in the presence and absence of specific sugars was qualitatively examined (Table 1). These tests were conducted using a modification of the method described by Mirelmann et al. (1980). To standardize the concentration of bacteria, bacterial cells were grown for 24 h on the following media: 10 g/L Bacto-Peptone, 5 g/L yeast extract; and 5 g/L NaCl (Firon et al., 1979). Cells were harvested by centrifugation and suspended in PBS (0.05 M, pH 7.2) to reach an optical density at 660 nm (OD660) of 1.0. Mannooligosaccharide was suspended at 1 g/L in PBS (OD660 = 1.60). A 24-h culture of Sac. cerevisiae NCYC 1026, grown in Tryptic Soy Broth, was harvested by centrifugation, and cells were suspended in PBS to reach an OD similar to the one of the MOS suspension (OD660 = 1.60). The yeast and MOS suspensions were blended for 1 min in a kitchen blender to separate clumping cells and MOS particles. Agglutination tests were then performed with 1.0 mL of the bacterial suspension and 1.0 mL of the MOS or yeast suspension. Agglutination was determined microscopically. To determine the inhibitory effect of carbohydrates on agglutination, each agglutinating strain was first incubated for 5 min in PBS containing 50 mM glucose, mannose, galactose, or fructose.

Animal Trials

Experimental Design. All animal trials were conducted following the animal care protocol IACUC 94-0004A approved by the University of Kentucky Institutional Animal Care and Use Committee. The ability of salmonella to colonize the intestinal tracts of chickens was evaluated in six separate trials. In each of the first two trials, the birds were divided into two control and two treatment groups containing 10 birds each. Trial 3 was conducted with 26 birds per group to study salmonella colonization over time. The fourth and fifth trials also used four groups of 10 birds, but because of low hatchability only 36 birds were assigned to treatment groups (9 birds per group). Birds were hatched and housed in bacterial isolation chambers. All diets met NRC requirements for young birds (NRC, 1994). Gastrointestinal microflora of birds were standardized at the beginning of each experiment by inoculating each bird with 1 × 106 cfu of one of the challenge organisms on Day 3. Cecal parameters were determined on Day 10. Dietary treatments in all trials included an un-supplemented diet (control) and a diet supplemented with 4,000 ppm of dietary Bio-Mos.

Bacterial Isolation Chambers. Five bacterial isolation chambers were used for these studies. One chamber containing the incubator was outfitted with a bacterial trap filled with disinfecting solution; the other four contained plastic housing boxes (3,840 cm2 × 320 cm2 per bird) and were equipped with regular inlet ports. The chambers were sanitized as described by Makin and Tzipori (1980). Feed, water, and 500 g wood shavings were placed in autoclave bags and were placed in the chambers prior to fumigation.

Eggs. Eggs (broiler type, line 248) were sanitized by spraying with a disinfectant solution (2.0% H2O2, 0.8% quaternary ammonium, and 0.25% acetic acid) and were misted daily during incubation. On Day 19, eggs were dipped for 30 s in disinfectant solution (37 C) and were transferred into the sterile incubator, which was placed in one bacterial isolator.

Standard Inoculum. Newly hatched chicks were inoculated with a standard inoculum to provide similar basic microflora for each bird at the beginning of each experiment. The standard inoculum was derived from the hatching debris from a commercial hatchery. The debris were mixed and stored in 100-g aliquots at −80 C. The inoculum was tested to be salmonella-free according to the approved standard method for isolation of salmonellae (Wallace et al., 1992). Microbial counts on the debris revealed the following bacterial concentrations: total bacteria, 2.5 × 107 cfu/g (Viand Levure Agar at pH 6.7; Barnes et al., 1979); coliforms, 8.03 × 106 cfu/g (MacConkey Agar); enterococci, 7.50 × 106 cfu/g (KF Streptococcal Agar); and lactobacilli, <1,000 cfu/g (Rogosa Agar). The defrosted debris was mixed 1:5 with 0.1% peptone water, blended in a sterile kitchen blender, and each bird was given 0.25 mL from the tip of a pipette (1.25 × 106 cfu/bird). Six h after standard inoculation, chicks were randomly as-
TABLE 1. Inhibitory effects of fructose, galactose, glucose and mannose on bacterial agglutination of mannanoligosaccharide preparations (MOS)

<table>
<thead>
<tr>
<th>Strain</th>
<th>Source</th>
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<th>Inhibition of agglutination by</th>
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<tr>
<td></td>
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</tr>
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<td>A 4157</td>
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<tr>
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<td>UKAS5 15R</td>
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</tr>
<tr>
<td>E. coli O157:H7</td>
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<td>Salmonella</td>
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<tr>
<td>C. coli UKAS 234</td>
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<td>−</td>
<td>No</td>
</tr>
</tbody>
</table>

1Culture Collection of Alltech Inc., Nicholasville, KY.
2The MOS particles were clumping together.
3American Type Culture Collection, Rockville, MD.
4No MOS particles were clumping together.
5Culture Collection of the University of Kentucky, Department of Animal Science, Lexington, KY.
6National Collection of Type Cultures, Central Public Health Laboratory, London, U.K.
7A few particles were clumped, but the majority were free.

signed to four groups of 10 chicks each and were transferred into the bacterial isolation chambers.

**Husbandry.** Room temperature was adjusted to 34°C for the first day and was then gradually lowered to 28°C at Day 10. Light was on continuously for the first 24 h after the chicks were transferred into the chamber and for 20 h each of the following days. Feed and water were consumed *ad libitum*. The feed was a corn-soybean meal diet that was formulated to meet NRC recommendations and contained no antimicrobial supplement (NRC, 1994).

**Challenge Cultures and Their Application.** Challenge cultures of *S. typhimurium* (strains 29E and 27A) and *S. dublin* for *in vivo* studies were grown in the basal peptone-yeast extract medium described earlier. The cultures were then diluted in sterile 0.1% peptone solution to a final concentration of $4 \times 10^4$ cfu/mL. Concentrations of salmonellae were determined on brilliant green agar (BGA) using spread-plate technique. One quarter of a milliliter of the diluted culture ($1 \times 10^4$ cfu/bird) was given to each bird from the tip of a pipette.

**Sampling and Sample Analysis.** After 10 d, the chicks from each group were sacrificed by asphyxiation with CO₂ and both ceca were aseptically removed. The content of one cecum was placed in a sterile test tube, weighed, and diluted 1:10 with sterile 0.1% peptone solution. The empty cecum was cut longitudinally and placed in lactose broth. Decimal dilutions of each sample were prepared in 0.1% peptone solution. Salmonellae were enumerated on BGA containing 30 mg naladixic acid/L after 24 h incubation at 37°C. Naladixic acid was added to the media.
to facilitate the selection of the antimicrobial-resistant challenge organisms. The tube containing the empty cecum in lactose broth was incubated at 37°C for 24 h for enrichment of salmonellae (Wallace et al., 1992). Cecal samples that were salmonella-negative on BGA plates, but positive in the enrichment test, were assigned as 1.50 log cfu/g (Nisbet et al., 1993). Samples that were confirmed negative in the enrichment test were assigned a concentration of 1 cfu/g (0 log cfu/g) (Nisbet et al., 1993). Four birds were randomly selected from each group for analysis of the concentrations of different groups of intestinal bacteria. Lactobacilli, enterococci, coliforms, and anaerobic bacteria were enumerated in duplicate using the pour-plate technique on Rogosa SL agar, KF streptococcal agar, McConkey agar, and reinforced clostridial agar, respectively.

**Determination of pH, Volatile Fatty Acid, and Lactate Concentrations.** The content of the second cecum was diluted 1:10 in distilled water, and pH was determined with a combined electrode. Samples were stored at −20°C until analyses of volatile fatty acids and lactate. Volatile fatty acid analysis was conducted on a Hewlett-Packard model 5890 gas chromatograph, fitted with a 180-cm × 4-mm glass column, containing 10% SP 1000 and 1% H3PO4 on 100/120 Chromosorb WAW with a modification of the method described by Erwin et al. (1961). D- and L-lactate were determined spectrophotometrically with D- and L-lactate dehydrogenase, respectively, in the presence of nicotinamide adenine dinucleotide (Brandt et al., 1980).

**Statistical Analysis.** Data were analyzed by ANOVA appropriate for the randomized complete block design; chambers (10 birds) were considered to be the experimental unit. Differences between treatment means were assessed by least significant difference test (Kuehl, 1994). Bacterial concentrations were subject to log 10 transformation prior to analysis. To evaluate the overall effects of MOS supplementation, the data was initially blocked across the trials. Statistical analyses were conducted using the general linear models procedure of SAS® (SAS Institute, 1985).

**RESULTS**

**Agglutination**

Five of seven strains of Escherichia coli and 7 of 10 strains of Salmonella typhimurium and Salmonella enteritidis agglutinated MOS (Table 1). Salmonella montevideo, Salmonella give, Salmonella kedougou, and S. dublin also triggered agglutination. Addition of Salmonella cholerasuis and Salmonella pullorum strains did not lead to agglutination of the MOS particles. Agglutination associated with these Gram-negative bacterial strains could be inhibited with mannose or fructose but not with glucose or galactose. However, it generally took 9 to 16 times more fructose to bring about the inhibitory responses observed with mannose (data not shown). This result suggests that fructose was less effective as a blocking agent. Agglutination patterns of MOS were similar to patterns of S. cerevisiae (data not shown). Different strains of Campylobacter were also tested for their abilities to agglutinate MOS (Table 1). Only two strains showed some agglutination; however, these agglutinations were very weak and were not affected by addition of carbohydrates.

**Challenge Trials**

The concentrations of salmonellae and coliforms in chicks challenged with Salmonella typhimurium 29E are shown in Table 2. The concentrations of salmonellae were consistently lower in birds fed MOS than in birds fed the unsupplemented diet. Salmonellae concentrations were between 4.97 and 5.73 cfu/g for the control and ranged from 3.79 to 4.29 log cfu/g for the MOS treatment. Over the three trials, MOS-supplemented chicks had lower Salmonella typhimurium 29E concentrations by about 25-fold (4.01 vs. 5.40 log cfu/g; P < 0.05). Concentrations of coliforms also tended to be lower (P < 0.1) when MOS was added to the feed (8.47 vs. 8.71 log cfu/g). Concentrations of lactobacilli, enterococci, and anaerobic bacteria were not affected by treatment. Volatile fatty acid concentrations, lactic acid concentrations, and pH were not affected by dietary treatment. Average values were as follows: pH 5.61, acetate 61.7 mM, propionate 5.9 mM, butyrate 12.2 mM, and lactate 9.0 mM. In Trial 3, the cecal Salmonella typhimurium 29E concentrations in the unsupplemented group rose from 3.61 log cfu/g at 2 d to 4.97 log cfu/g at 7 d after challenge (Figure 1). Salmonella typhimurium 29E concentrations in the MOS treatment group were lower (P < 0.05) than those of the unsupplemented control group 7 d after challenge. Birds receiving MOS had average cecal salmonella concentrations of 2.98 log cfu/g at 2 d and 3.79 log cfu/g at 7 d after challenge.

The percentage of birds from which S. dublin could be recovered was lower in the MOS-treated groups than in the control group in all three trials (Table 3). In Trial 6, 90% of the birds in one of the groups receiving MOS were colonized with S. dublin. As a result, the mean reduction in the percentage of colonized birds with MOS addition appears lower in Trial 6 (24%) than in Trial 4 (47%) or Trial 5 (44%). Over the three trials, MOS decreased the number of salmonella-positive birds from 89.8 to 55.7% (P < 0.05). Concentrations of coliforms, lactobacilli, enterococci, and anaerobic bacteria were not different in the two treatment groups. Cecal pH, VFA, and lactate concentrations were very similar in the trials with Salmonella typhimurium and were not affected by treatment (data not shown).

Salmonella typhimurium 27A, a nonagglutinating strain of salmonella, colonized the chicks at a very low rate.
This organism could only be recovered from the ceca of 3 of 40 chicks that were challenged with doses of up to 1 \times 10^4 

Two control chicks and one bird receiving MOS were salmonella positive at 10 d, which made it impossible, using the bacterial isolation chambers, to evaluate the effects of MOS on the colonization of birds by this strain.

**DISCUSSION**

The colonization of bacteria on mucosal tissues is recognized as an important step in the infectious process. To colonize the mucosal surfaces, bacteria must first bind to the epithelial cells of these tissues. One way of binding to epithelial cells is through attachment of Type-1 fimbriae (Ofek et al., 1977). A variety of strains of E. coli, Salmonella, and Campylobacter were tested for the presence of Type-1 fimbriae by means of yeast and MOS agglutination. No differences were observed between agglutination pattern of the two yeast products. The majority of the strains of E. coli and S. typhimurium and S. enteritidis tested agglutinated both the yeast culture and MOS. All agglutination observed in these enteric bacteria was mannose sensitive. This result is in agreement with findings by Duguid (1964) and Duguid et al. (1966) who reported high frequency of Type-1 fimbriae expression in S. typhimurium, Salmonella typhi, and S. enteritidis. However, Mirelmann et al. (1980) only found 4 of 13 strains of S. typhimurium to express Type-1 fimbriae. The poultry-specific salmonella types, Salmonella pullorum and Salmonella gallinarum, did not trigger agglutination in the work of Duguid (1964) or in the study presented here. Four of seven tested strains of E. coli agglutinated MOS. Mirelmann et al. (1980) reported that about half of the tested strains of E. coli expressed Type-1 fimbriae. None of the tested Campylobacter strains caused a mannose-sensitive agglutination of MOS. However, mannose was reported to inhibit adherence of C. jejuni to epithelial cells in vitro (McSweegan and Walker, 1980), and protective effects with dietary mannose were also reported against Campylobacter jejuni colonization in chickens (Schoeni and Wong, 1994). These authors did not determine whether the Campylobacter strains used in their study expressed mannose-sensitive fimbriae. Therefore, it is not possible to say whether inhibition of attachment or other mechanisms were involved in their studies. Fucose is a constituent of the mucus the primary site of Campylobacter colonization, and inhibition of Campylobacter colonization has also been reported with addition of fucose to the diet (Cinco et al., 1984).

Standardization of experimental conditions is important to achieve reproducible results. Initial results obtained from challenge studies with birds from a commercial hatchery were not reproducible. It was hypothesized that the birds might have been exposed to different bacterial flora at the hatchery, which could explain differences between trials. Birds that would have been exposed to a more complex microflora at the hatchery would exert a stronger CE effect in the gastrointestinal tract and would, consequently, be colonized with the challenge organisms at lower rates. Therefore, the experimental conditions were modified to standardize microflora of each bird at the beginning of the experiment by removing the native microflora of the egg through sanitation and then exposing the newly hatched bird to a constant level of microorganisms derived from hatching debris in bacterial isolators. This process allowed for the standardization of the microflora at the beginning of the experiment and allowed the composition of the cecal gastrointestinal microflora, cecal pH, and cecal acid concentrations of the...
birds to be similar in each experiment and each treatment group.

The main goal of these trials was to evaluate the effects of MOS on salmonella colonization in chicks. The focus was on Salmonella strains expressing Type-1 fimbriae. We hypothesized that MOS might affect colonization of such strains through blocking bacterial attachment to the gut mucosa. In a first series of three trials, dietary addition of MOS decreased the cecal concentration of S. typhimurium 29E. To further investigate whether MOS could decrease the concentration of a strain of Salmonella that colonizes birds with a cecal concentration less than 10^4 cfu/g, another series of trials was conducted using S. dublin as a challenge organism. Mannanoligosaccharide treatment also reduced the number of birds from which S. dublin could be isolated in these trials. The challenge trials with S. dublin and S. typhimurium 29E suggest that MOS can lower cecal salmonella colonization of strains that colonize at high or low concentrations. The study conducted over time showed that MOS reduced cecal S. typhimurium 29E concentrations 7 d after challenge. To observe cecal S. typhimurium 29E concentration over a longer period of time, with and without withdrawing MOS, might help to further investigate what effect MOS exerts on Type-1 fimbriated enteric pathogens. An inhibitory effect on salmonella colonization in chicks has also been shown with live yeast culture. Line et al. (1995) reported decreased colonization of S. typhimurium with dietary Saccharomyces boulardii. However, different modes of action might be involved with live yeast cells. Live yeast cells excrete metabolites, which are known to affect the composition of the gastrointestinal microflora (Girard, 1996) and could, therefore, alter the CE effect of the indigenous microflora. In addition, the strain of S. typhimurium used by Line et al. (1995) has not been screened for presence of Type-1 fimbriae. Therefore, it can not be determined whether the observed reduction was due to yeast cell wall mannan or other mechanisms.

The decrease in concentrations of Salmonella expressing Type-1 fimbriae was not accompanied by changes in cecal parameters such as decreases in pH or increase in propionic acid concentrations, which are known to reduce salmonella concentrations, which suggests that other modes of action are responsible for the decrease salmonella concentrations with dietary MOS. In vitro agglutination results suggest that adsorption of salmonellae by MOS, which would keep them from adhering to the intestinal wall, could be a possible mode of action. Oyofe et al. (1989a) tested the effect of different sugars on the adherence of S. typhimurium to epithelial cells of 1-d-old chicks in vitro. They reported inhibition of adherence by methyl-a-D-mannoside and mannose by more than 90%. In different in vivo trials, mannose has also been shown to decrease cecal colonization of S. typhimurium in young chicks (Oyofe et al., 1989b,c). The fact that MOS had no effect on the concentrations of lactobacilli and enterococci, two bacterial species that are known to not express Type-1 fimbriae, further suggests that MOS affects bacterial concentrations in the gastrointestinal tract by adsorbing bacteria and keeping them from adhering to the gut wall.

Dietary MOS has been shown to decrease the prevalence of strains of Salmonella expressing Type-1 fimbriae in young chicks under laboratory-scale conditions. No changes in cecal parameters such as a major shift in bacterial populations or changes in pH or VFA concentrations, which are known to affect salmonella, were observed with MOS addition. Field trials will be required to show how the reduction noticed under small-scale conditions will transfer into commercial production systems. Because MOS can be considered animal, environmental, and consumer friendly, it might offer an additional and interesting tool that can be included in a hazard analysis critical control point program of poultry production.

REFERENCES


