Combined Effects of Dietary Carbohydrates and Preslaughter Flushing with Magnesium Sulfate on Cecal Coliform Colonization in Chicks

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ABSTRACT

Day-old broiler chicks were fed a corn-soybean meal based diet containing either 2% lactose or 0.05% mannanoligosaccharide (MOS) per kg of feed. Lactose-fed chicks at 7 and 14 d of age had significantly fewer cecal coliforms (log_{10} 3.90 cfu/mL) than MOS-fed (log_{10} 4.57 cfu/mL) and untreated control chicks (log_{10} 5.88 cfu/mL). Chicks at 21 d of age were given magnesium sulfate (MgSO₄) in the drinking water for 12 h prior to sacrifice. Chicks given MOS followed by MgSO₄ in the drinking water had fewer cecal coliforms (cfu/mL) than chicks in the remaining treatment groups. Compared to the other treatments the combination of MOS and MgSO₄ resulted in greater reduction in cecal bacterial population in pre-slaughter chicks at 21 d of age.

KEYWORDS: Lactose, Mannanoligosaccharide, Coliforms, Magnesium sulfate, Chicks

INTRODUCTION

Control of intestinal foodborne pathogens continues to be of interest to poultry producers and researchers. Among the foodborne pathogens Salmonella and Campylobacter spp (especially Campylobacter jejuni) are among the most important pathogenic organisms (White et al., 1999; Saleha et al., 1998). Salmonella contamination of processed poultry and meat products has been shown in some cases to be as high as 35% (Houston, 1987). In 1998, 849 cases of campylobacteriosis were reported in Georgia (Georgia Department of Human Resources, 1999). Campylobacter has been associated with poultry carcasses and poultry products that have been processed further (White et al., 1999; Saleha et al., 1998). Despite advances in processing and storage practices, consumer concerns have not diminished in recent years regarding the contamination of processed poultry meat by Salmonella typhimurium and Campylobacter.

Studies conducted with pigs and mice stated that in order for enteric pathogens to colonize the gastrointestinal (GI) tract, they must first be able to gain attachment to mucosal epithelial cell surfaces within the GI tract where they can grow to sufficient numbers to produce clinical signs of the disease (Oyofo et al., 1989).
Interference with the ability of bacteria to adhere to epithelial cells has been shown, \textit{in vitro} as well as \textit{in vivo}, to prevent bacterial colonization and subsequent pathogenesis (Fuller, 1978). If attachment does not occur, the organisms are expelled by mucus secretions and by peristalsis (Morton, 1993). The adherence of pathogens to the intestinal wall can be inhibited by different sugars (Oyofu et al., 1989). Lactose (Corrier et al., 1990), mannanoligosaccharide (MOS) (Sisak, 1994) and mannose (Oyofu et al. 1989) as feed additives or in the drinking water have been shown to reduce enteric pathogen colonization.

The movement of the unattached bacteria from the GI tract depends on the viscosity of the contents. Bedford et al. (1991) demonstrated that weight gain in broiler chicks correlated closely with the reduction in intestinal viscosity. Magnesium sulfate, a mild and inexpensive laxative, reduces intestinal viscosity and is closely correlated with the reduction of coliforms in cecal and colon contents of broiler chicks (Stanley et al., 1991). The objective of the present study was to examine the combined effects of lactose and MOS, followed by pre-slaughter flushing of the GI tract with MgSO\textsubscript{4}, on the reduction of cecal coliform bacterial population in broiler chicks.

**MATERIALS AND METHODS**

A total of 360, 1-day-old male broiler chicks (Cornish Rocks) were obtained from a local commercial hatchery. The chicks were divided into three treatment groups of 40 chicks per group with each treatment group replicated three times. The chicks were maintained in wire cages with incandescent lighting and were fed a commercial, unmedicated corn-soybean meal-based diet. The diet contained or exceeded levels of critical nutrients recommended by the National Research Council (NRC, 1994). The birds were not inoculated with \textit{Salmonella}; however, evaluation for \textit{Salmonella} was made on the cecal contents. MOS (Alltech Biotechnology Laboratory, Lexington, Kentucky) was added at 0 and 0.05%/kg of feed for three treatment replications. Lactose was applied at 0 and 2%/kg of feed to three additional replicate groups. Chicks in the three replicates remaining (control) did not receive either lactose or MOS in the feed.

Ten chicks, 7 and 14 d of age, respectively, were randomly selected from each treatment group and sacrificed by cervical dislocation. Ceca were removed aseptically and the contents were collected, weighed wet, serially diluted in 9 mL of bacterial free distilled water, and plated. Coliform counts were obtained by using pour plates of plate count agar (PCA) that were overlaid with violet red bile agar [Food and Drug Administration (FDA), 1984]. The numbers of colony forming units (CFU) were determined after the plates were incubated aerobically at 35°C for 12 h. Isolation and identification of coliform bacteria were completed using methods described by the FDA (1984).

At 21 d of age the remaining 20 chicks in each group were divided into two groups of 10 and given MgSO\textsubscript{4} in the drinking water at 0 or 5.5 g/liter for 12 h before the chicks were sacrificed (Stanley et al., 1991). Chicks receiving the MgSO\textsubscript{4}-free water in each treatment group were used as control groups. The chicks were weighed before being treated with MgSO\textsubscript{4} and before they were sacrificed to evaluate the effect of MgSO\textsubscript{4} on body weight.

Data on cecal bacterial population were transformed to logarithms and subjected to a one-way analysis of variance using general linear models procedures as programed by SAS software (SAS Institute, 1988). Duncan’s multiple range test (Duncan, 1955) was conducted to detect differences between means (P< 0.05).
RESULTS AND DISCUSSION

At 7 d of age chicks given a diet containing 2% lactose/kg of feed had 1.20 log units fewer (P < 0.05) cecal coliforms than did ceca from control chicks, whereas chicks fed 0.05% MOS per kg of feed had 1.3 log fewer (P < 0.05) coliform population, compared to the control chicks (Table 1). The mean log_10 cecal (cfu/g) of cecal contents increased at 14 d and again at 21 d of age in chicks in the lactose and MOS treatment groups. These values were not different from those obtained for chicks in the control group.

Table 1. Mean cecal coliform population after dietary MOS and lactose in broiler chicks at 7, 14, and 21 d of age.

<table>
<thead>
<tr>
<th>Treatments</th>
<th>7(^{1})</th>
<th>14(^{1})</th>
<th>21(^{1})</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control</td>
<td>5.88a</td>
<td>5.59a</td>
<td>6.51a</td>
</tr>
<tr>
<td>Lactose(^{2})</td>
<td>3.89b</td>
<td>4.80a</td>
<td>5.37a</td>
</tr>
<tr>
<td>MOS(^{3})</td>
<td>4.57b</td>
<td>5.10a</td>
<td>5.85a</td>
</tr>
<tr>
<td>SEM</td>
<td>1.65</td>
<td>1.37</td>
<td>1.51</td>
</tr>
</tbody>
</table>

\(^{3}\)Means with no common superscript differ significantly (P>0.05).
\(^{1}\)Three replications, forty chicks per replication (n=30).
\(^{2}\)Lactose was fed continuously at 2% kg of feed.
\(^{3}\)MOS=Mannanoligosaccharide, fed continuously at 0.05%/kg of feed.

Lactose is a disaccharide sugar found in milk and when added to broiler feed has considerable beneficial effects in preventing the growth of harmful bacteria (Schaible, 1970; Corrier et al., 1994). MOS derived from yeast is a source of mannose (Sisak, 1994). Administration of mannose to chicks resulted in a significant reduction in Salmonella typhimurium colonization in broiler chickens (Oyofu et al., 1989). Many species of cecal bacteria have lectin-like appendages (fimbriae) that bind the receptors on to epithelial cells (Oyofu et al., 1989). Ofek et al. (1977) stated that epithelial cell receptors are probably coated with mannose. Dietary mannose, and mannose-containing feed additives, block the adherence of bacteria that have mannose-sensitive fimbriae (Ofek et al., 1977; Eshdat et al., 1978). MOS appears to have an additional mode of action in the reduction of intestinal bacteria in chicks. Bedford et al. (1991) indicated that increased dietary fiber in MOS is related to reduction in intestinal viscosity. Because of its high fiber content of 30%, MOS is thought to decrease the viscosity of the contents of the GI tract, as it is slow to degrade and passes intact through the GI tract (Sisak, 1994).

Lactose-fed chicks at 21 d of age had significantly (P<0.05) lower cecal coliforms than chicks in the control group or MOS-fed chicks. The addition of MgSO\(_4\) to the drinking water at 21 d during the last 12 h prior to sacrifice resulted in a significant (P<0.05) decrease in the cecal coliforms in all treatment groups (Table 2). Ceca taken from chicks fed diet...
containing MOS followed by MgSO₄ in the drinking water 12 h before sacrifice had the lowest cecal coliforms when compared with the untreated or the chicks given only MgSO₄.

Table 2. Mean cecal coliform populations before and after MgSO₄ of chicks at 21 d of age treated with MOS and lactose.

<table>
<thead>
<tr>
<th>Treatments</th>
<th>Coliform (log₁₀ cfu/g) of cecal content</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Without¹ MgSO₄</td>
</tr>
<tr>
<td>Control</td>
<td>6.51a</td>
</tr>
<tr>
<td>Lactose2</td>
<td>5.32a</td>
</tr>
<tr>
<td>MOS3</td>
<td>5.85a</td>
</tr>
<tr>
<td>SEM</td>
<td>1.15</td>
</tr>
</tbody>
</table>

¹ Means within row with no common superscript differ significantly (P<0.05).
² Three replications, twenty chicks per replications (n=30).
³ Lactose was fed continuously at 2%/kg of feed.
⁴ MOS=Mannanoligosaccharide, fed continuously at 0.05%/kg of feed.

Chicks given lactose also had their coliforms lowered after receiving MgSO₄. Magnesium sulfate has been demonstrated to decrease the viscosity of the ingesta in the GI tract of chickens (Stanley et al., 1991). Therefore, MgSO₄ and MOS, had a combined effect on the coliform population in the GI tract suggesting that the increase of the ingesta viscosity and the inability of the bacteria to bind the receptor in the epithelial cells were responsible for the reduction in the bacteria count. Being trapped in the ingesta, the bacteria failed to attach to the epithelial cells and were expelled (Morton, 1993). By increasing the persisstic movement of the GI tract with MgSO₄ less fecal contents were recovered 12 h after the administering MgSO₄ (Stanley et al., 1991).

Mannanoligosaccharide of lactose added to the feed had no significant effect on the pH levels of the cecal contents from chicks in all groups. The pH of the cecal contents was relatively the same for the control, lactose, and MOS-fed groups (5.51, 4.77, and 5.38), respectively, indicating that the reduction in the cecal coliforms from the combined effect of MOS and MgSO₄ was not due to the changes in the pH of cecal contents but due to the combined effect of increased viscosity and the blocking effect of MOS. Additionally, MgSO₄ administered 12 h prior to processing did not adversely affect the BW of the chicks in all groups (477 vs 470g for control, 451 vs 449 g for lactose, and 462 vs 460 g for MOS). Stanley et al. (1991) reported that the coliform population in the ceca was lowered significantly when the level of MgSO₄ exceeded 5.5 g/L of drinking water without any significantly effect on body weight of the broiler chickens. Also, they reported that a positive correlation existed between cecal coliform reduction in broiler chickens and decreased bacterial population on the carcass when MgSO₄ was administered to the drinking water 12 h before the birds were sacrificed. Further, no withdrawal period is necessary for MgSO₄. Magnesium sulfate is a low-cost additive and is relatively simple to administer. Treatment of chicks with MOS fed continuously followed with pre-slaughter flushing of the G.I. tract with MgSO₄, 12 h prior to sacrifice was highly effective in reducing cecal


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coliforms population. The combined use of MOS followed by MgSO₄ 12 h prior to processing could be a useful integrated management practice in reducing the levels of bacteria in the digestive tracts of pre-slaughter chickens and the potential for reducing carcass contamination during processing.

REFERENCES