

Use of Bio-Mos[®] to Control Salmonella and Campylobacter in Organic Poultry

Dr. Curtis Novak and Catalina Troche
Dept. Animal and Poultry Sciences Virginia Tech
3300 Litton-Reaves Hall (0306)
Blacksburg, VA 24061
cnovak@vt.edu

Introduction

The USDA defines organic farming as: A production system that is managed in accordance with the Organic Foods Production Act and regulation in this part to respond to site-specific conditions by integrating cultural, biological, and mechanical practices that foster cycling of resources, promote ecological balance, and conserve biodiversity (National Organic Program). Nearly all aspects of production and processing are dictated by these recommendations. Birds must be fed certified organic feed and have access to the outdoors, including direct sunlight and fresh air when temperature allows. Waste must be managed in such a way as to prevent soil, crop or water contamination. Poultry meat and eggs must be processed in certified organic plants (Dimitri and Greene, 2001). Chicks must be raised organically from the second day of life. Preventative care and dietary supplements, i.e. vaccines, vitamins and minerals are permitted in production schemes. Animals are not allowed growth promotants, which in the poultry industry refers solely to antibiotics.

Organic farming is becoming more prevalent in the United States with sales of organic eggs and meat in natural foods stores and conventional supermarkets increasing by 80% between 1999 and 2000 (NFM, 2001). Increased organic sales in the past 16 years may reflect increasing U.S. concerns regarding antibiotic resistance. Similar concern in Europe forced The Commission of the European Communities to phase out, and ultimately ban, the marketing and use of antimicrobials as growth promoters (AGP) in animal feed. The final steps of this phase-out were implemented on January 1 of this year. Though no such ban has been implemented in the United States many large companies are voluntarily doing away with AGP. As of January 24, four of the top ten poultry companies (Tyson Foods, Gold Kist, Perdue Farms and Foster Farms) which account for roughly 38% of the broilers raised in the U.S. voluntarily removed feed grade antibiotics for growth promotion (Weise, 2006).

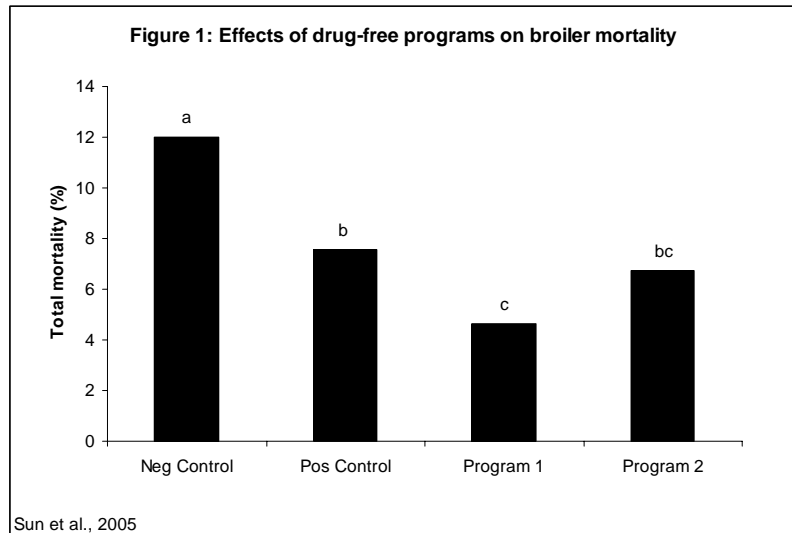
The recent ban of AGP in Europe and consumer pressures in the United States has presented the poultry industry with new challenges concerning bird health and consumer safety. Traditionally, microbial populations were considered a ruminant issue; however, regulations are forcing pig and poultry producers to take microbial populations into consideration. Normal microbial populations provide protection to their host through the production of volatile fatty acids and competition with non-indigenous organisms for both, limited nutrient supply and intestinal attachment sites (Verstegen and Williams, 2002). On the other hand, over proliferation of indigenous microflora could also negatively impact growth by competing with the animal for nutrients and producing toxic metabolites which increase mucosal gut turnover, i.e. ammonia (Anderson et al., 1999). Antibiotics are thought to promote growth by keeping microbial populations in check. They effectively reduce competition; resulting in increased nutrient availability, reduced gastrointestinal maintenance costs or both. Therefore, a feed additives designed to replace an AGP would also be expected to fulfill these functions.

Antibiotic replacement strategies

Probiotics, prebiotics, organic acids and enzymes/microbial modifiers have all been identified as potential antibiotic replacements. Ceylan and Ciftci (2003) compared Cylactin (*Enterococcus faecium*) as a probiotic, Bio-Mos[®] as a prebiotic, and a humic-acid treatment all compared against the performance of Avilamycin. All of the antibiotic alternative treatments had improved feed conversion over the non-

supplemented controls, with antibiotic and alternative groups having similar performance. This suggested that antibiotic alternatives may indeed be a viable option. A large body of research supports these findings, maintaining that antibiotic alternatives may promote growth. When seven levels of mannanoligosaccharide (MOS) were fed to broilers, live weight was improved through 21 days and feed conversion continued to improve through 42 days (Kumprecht et al., 1997). Similarly, 18 wk old turkeys responded to MOS supplementation with improved feed:gain ratio over that of the negative control (Parks et al., 2001).

These findings suggest that non-antibiotic feed additives may be able to replace antimicrobials in terms of growth. Several studies have used various challenge models to demonstrate how antibiotic alternatives may prevent disease. Sun et al. (2005) conducted a trial which attempted to determine the effects of pre- and probiotics on broiler performance. Four dietary treatments were utilized: a negative control basal diet and a positive control diet which was the basal supplemented with lincomycin. The two remaining drug-free treatments were termed PG1 and PG2. PG1 was the basal diet supplemented with Bio-Mos[®] (prebiotic), All-Lac XCL (probiotic), Vegpro (vegetable enzyme), MTB-100 (anti-mycotoxin) and Acid Pak 4-way (acidifier). PG2 was a simplified version of PG1 using only Bio-Mos[®] and All-Lac XCL. On day 14 3 birds per pen were presented with a mixed coccidia challenge. Birds on both antibiotic and PG2 treatments demonstrated improvements in feed conversion ratio over the negative control.



Mortality was relatively high (12%) throughout the duration of the trial with necropsy revealing the presence of necrotic enteritis from the over proliferation of *Clostridia perfringens*. PG1 reported the least mortality (4.68%) followed by PG2 (6.74%) and the positive control (7.58%) (Figure 1). Cecal lamina propria was reported to be thinner in PG1 and PG2, as compared to the negative control. This suggests that these alternative treatments had reduced inflammation due to a reduced pathogenic bacterial load. Additional studies have attempted to derive additive effects of AGP use in conjunction with MOS. Fairchild et al. (2001) compared the effects of Bio-Mos[®] and Flavomycin on *E. coli* challenge. Day old poults were initially challenged through oral gavage and challenge was kept continuous through weekly water doses. Overall the presence of Bio-Mos[®] or Flavomycin improved body weight gain during challenge. However, these improvements did not correlate with feed conversion ratios, nor did these treatments improve production in non-challenged birds. When looking at coliform count Bio-Mos[®] decreased the total liver coliforms present on day 7. These studies demonstrate that while anti-biotic alternatives appear promising, there is still much to learn concerning their interactions with indigenous microflora.

Prebiotics

Prebiotics are described as, non-digestible substances that when consumed provide a beneficial physiological effect on the host by selectively stimulating the favorable growth or activity of a limited number of indigenous bacteria (Gibson and Roberfroid, 1995). In other words, prebiotics are meant to provide a substrate for beneficial gastrointestinal microbes. Large amounts of bacteria present in the monogastric small intestine and are potentially capable of utilizing these indigestible carbohydrate

sources for energy. In pigs it has been estimated that 40-60% of non-digestible oligosaccharides and up to 20% of other non-starch polysaccharides (NSP) are fermented in the small intestine of pigs (Bach Knudsen and Hausen, 1995). A stable supply of NSP to the last part of the gastrointestinal tract may contribute to maintenance energy of the gastrointestinal tract. However, the balance between helpful and harmful fermentation loads is a precarious one. If substrate is fermented too quickly large intestinal bacteria are forced to use protein as an energy source, leading to increased production of branched-chain fatty acids and ammonia. A study designed to test the effects of fermentation rate on maintenance energy compared two feeding strategies in pigs (Verstegen and Williams, 2002). Two sources of carbohydrates were fed a mixture of rapidly (fructooligosaccharide) and slowly (sugarbeet pulp) fermentable carbohydrates versus only slow (sugarbeet pulp). Both treatments were compared against a control diet which was free of fermentable carbohydrates. Stress was induced in the pigs through rearing temperatures; a standard temperature (25°C) was compared against a low temperature (15°C). As expected maintenance requirements were increased at the lower temperature. Interestingly, the treatment with the range of fermentation rates had the lowest maintenance energy requirement at both respective temperatures. Prebiotics such as fructo-oligosaccharides, inulin and lactulose behave similarly in that they alter the microbial balance in favor of beneficial bacteria. Mannan oligosaccharide is different in that it appears to impact bacterial adhesion properties. These adhesion properties suggest that the primary function of MOS may be that of immune activation vs. fermentable nutrient source (Spring et al., 2000; Zdunczyk et al., 2005).

Mannan oligosaccharides

Mechanism of action

Many gram-negative bacteria use type-1 fimbriae (or pilli) to attach to the intestinal lining. These bacteria primarily recognize mannose and to a lesser extent, fructose, moieties on glycoproteins attached to the endothelial wall (Hooge, 2003). The exact mechanism through which pathogenic bacteria are inhibited by mannose is unclear, though two theories have been presented. One being that MOS may adsorb bacteria containing type-1 fimbriae inhibiting them from binding to the carbohydrate moieties of the intestinal lining (Hooge, 2003). The other being one of agglutination, that MOS causes pathogenic cells with type-1 fimbriae to aggregate or clump, brining them out of solution (Spring et al., 2000). Table 1 depicts different adhesion rates of several different bacterial genera (Finucane et al., 1999b). Strains of *E. coli* and *Salmonella* were screened to determine the incidence of strains possessing mannose sensitive adhesions. Authors found that 80% of *Salmonella enteritidis* and 67% of *Salmonella typhimurium* freely agglutinated with MOS. It is interesting to note that adhesion appears to not occur with *Clostridium* or *Helicobacter pylori*, though production improvements have been observed with the use of MOS products. This may implicate other mechanisms of intestinal modification beyond simple type-1 agglutination.

Table 1: Adhesion properties of different bacterial genera

Organism	Number Tested	Number with mannose sensitive adhesion
<i>E. coli</i>	77	51
<i>Salmonella</i> spp.	30	16
<i>Clostridium</i> spp.	4	0
<i>Vibrio cholerae</i>	3	0
<i>Helicobacter pylori</i>	2	0
<i>Pasteurella</i> spp.	2	0
<i>Pseudomonas</i> spp.	3	0

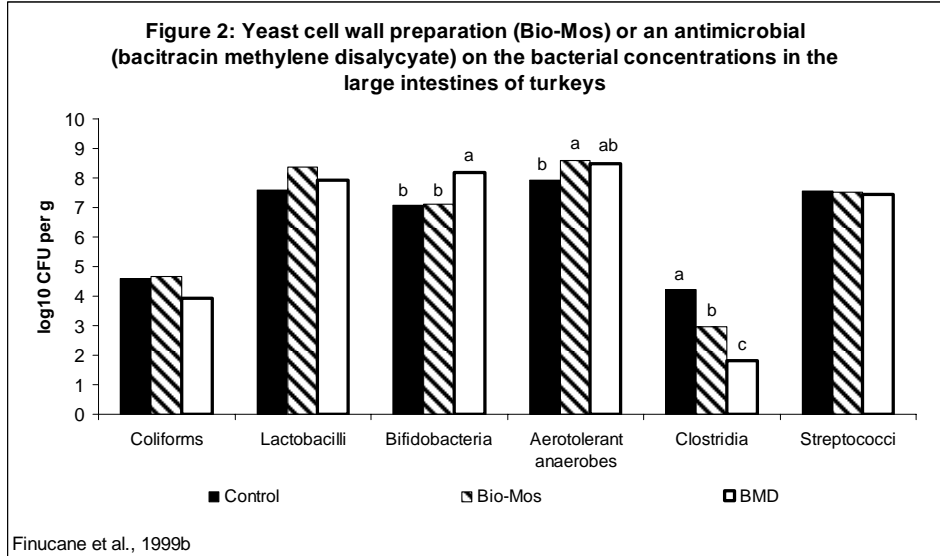
Finucane et al., 1999a

Modification of intestinal microflora

Mannan

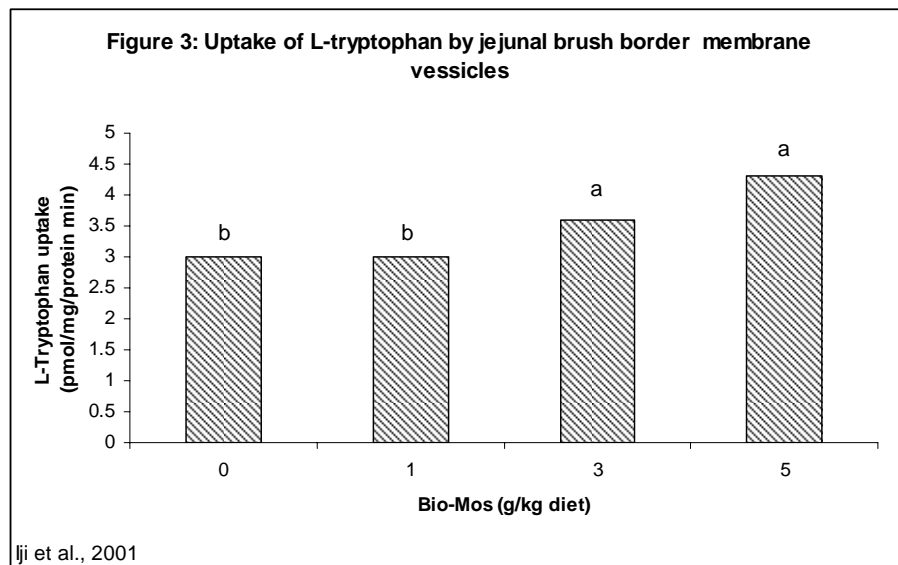
oligosaccharides have been shown to have indirect effects on bacterial populations which are not type-1 specific. Those populations specifically involved in the development of necrotic enteritis (coccidia and *C. perfringens*) and *Campylobacter* are of particular interest. In a study designed to test the effects of MOS on

various bacteria groups within the intestine four dietary treatments were utilized; a negative control, a MOS treatment, a BMD treatment and a MOS + BMD treatment (Finucane et al., 1999a). Coliforms, *E. coli*, lactobacilli, bifidobacteria, total anaerobes, streptococci, and *Clostridium perfringens* were all cultured and quantified using on a log of colony forming units/gram basis (Figure 2). At 6 wks of age MOS treatment group had a higher total anaerobe count (8.61 vs. 7.92) and a lower level of *Clostridium perfringens* (2.98 vs. 4.22). Again, suggesting that MOS may influence the capabilities of total (vs. type-1 only) microflora populations within the digestive tract.



Modification of mucosal lining

An increasing number of reports have suggested that MOS may influence the physical properties of the epithelial lining itself. Histological examination of the duodenal loop (mid-distal) and the jejunum (proximal to the Meckels) revealed an increase in the number of goblet cells with an inclusion level of 0.33% Bio-Mos® (Savage et al., 1997). A lower level of inclusion (0.11%) did not reveal an increase in



goblet cell numbers but did show a decrease in crypt size and villus width, suggesting a potential reduction in mucosal turnover rate. A study by Iji et al. (2001) produced similar results. Authors determined the effect of Bio-Mos® on structural and functional aspects of the gut. With high levels of supplementation jejunal villi height increased. Protein/RNA and RNA/DNA ratios in the ileal

homogenates were significantly influenced by the presence of Bio-Mos[®] in the diet. Birds on MOS had a higher expression of digestive enzyme activities. Uptake of L-tryptophan by brush border membrane vesicles also increased with increased levels of Bio-Mos[®] (Figure 3). These findings suggest further mechanisms through which MOS improves host ability to absorb nutrients and fight pathogens.

Immunomodulation

Perhaps one of Bio-Mos[®] most complex effects is its ability to influence the systemic immune reaction. Cotter and Weiner (1997) qualified Bio-Mos[®] immunomodulatory properties by testing its influence on delayed wattle reaction (DWR). Phytohemmagglutinin-P (PHA), a kidney bean lectin, was used to induce an inflammatory response in wattles. Primary responses between the Bio-Mos[®] and control treatments were similar. However, inflammatory reactions in the secondary (8 wk post challenge) and tertiary responses (10wk post challenge) were greatly reduced when compared to control groups. The inclusion of Bio-Mos[®] resulted in the modulation of cell-mediated immune function, in this case, the PHA wattle reaction. Singbootra et al. (2003) investigated the influence of Bio-Mos[®] on apoptosis and inflammation using an immortal chicken macrophage cell line (MQ-NCSU) which is used as the model for antigen-presenting cells in the chicken immune system. High concentrations of Bio-Mos[®] (20 mg/ml) assayed with MQ-NCSU (5x10³ cells/well) initiated processes which lead to cell death. Authors theorized that cell death activated MQ-NCSU surface receptors to release cytokines, subsequently causing apoptosis. In fact, a wide variety of cells express mannan binding lectins, which are involved in apoptosis and production of inflammatory cytokines, i.e. TNF, IL-1, IL-2, and IL-6. Increasing MOS in the diet may positively influence the strength of the immune reaction. There is also some evidence that MOS may have antioxidant properties, possibly leading to improvement in activity of antioxidants such as glutathione peroxidase (GSH-Px) and superoxide desmutase (SOD). In a study performed on broiler breeders, egg traits (production, hatchability, fertility) and bird immunity were measured in both cage and deep litter trials (Shashidhara and Devegowda, 2003).

In both trials, sperm density as well as antibody responses against infectious bursal disease virus were higher in the MOS group when compared to control birds. Maternal antibody titers in progeny were also positively influenced by MOS supplementation. Authors suggested that the antioxidant properties of MOS protected developing spermatozoa from the high peroxide

Table 2: Mannan oligosaccharide (MOS) on sperm count, proportion of live sperm and infectious bursal disease virus (IBDV) antibody titers

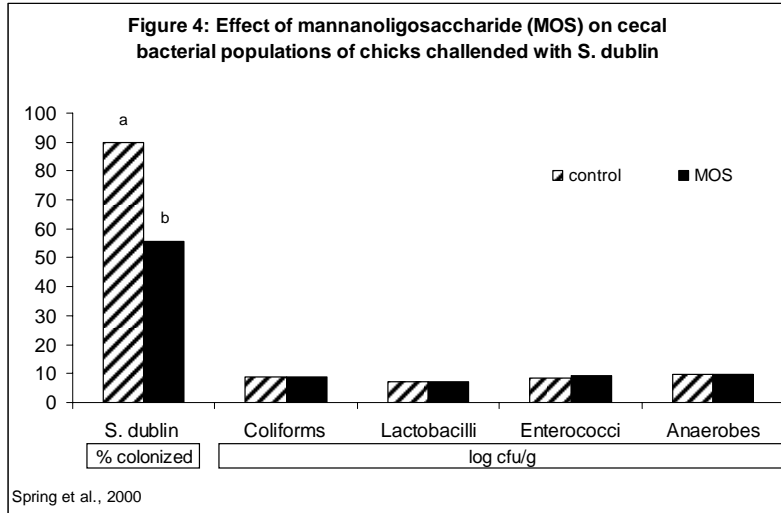
<i>Cage trial</i>	n	Control	MOS
Sperm count (million/mL)	20	2,155.5 ^b	2,540 ^a
Live sperm	20	91.45	91.0
IBDV antibody titer	20	1,758.50 ^b	2,062.30 ^a
<i>Deep litter trial</i>	n	Control	MOS
Breeder: IBDV Antibody titer	24	2,044 ^b	2,351 ^a
Progeny: IBDV Antibody titer	30	658.78 ^b	969.18 ^a

Shashidhara and Devegowda, 2003

concentration in the testes. It is important to take all of these effects of MOS on both microflora populations and the immune system when looking at its effects on disease status of birds. Buzby and Roberts (1997) reported that microbial pathogens in food cause 6.3-33 million cases of human illness and up to 9,000 deaths in the U.S. each year. Four of the most common food borne pathogens, *Salmonella*, *Campylobacter jejuni*, *Escheria coli O157:H7*, and *Listeria monocytogenes*, are believed to account for \$1.1-4.1 billion in human illness costs (a statistic which combines medical expenses, lost individual earnings and employer productivity losses) in the U.S. each year. The potential of MOS to effectively replace antibiotic treatments lies in its ability to inhibit these economically detrimental pathogens.

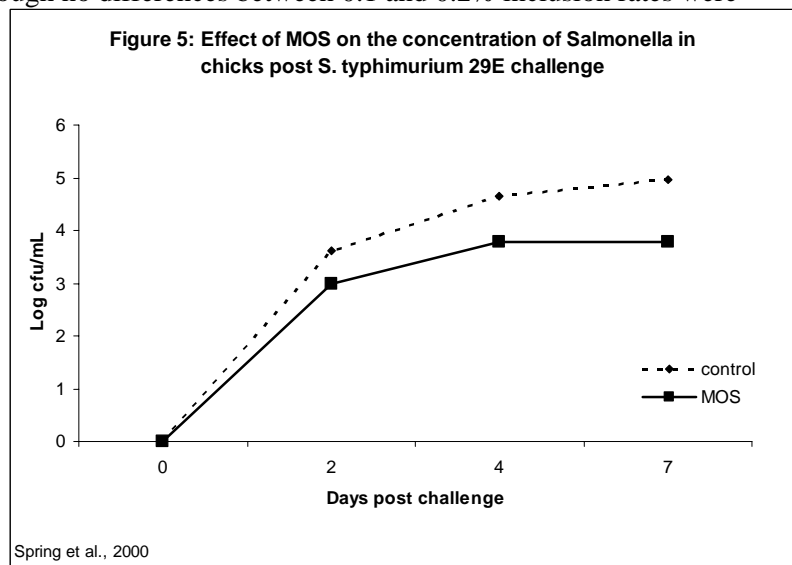
Salmonella

Salmonella is a term used to describe a large group of bacteria which often cause gastrointestinal disease within animals and humans. Generally, most of the *Salmonella* strains dealt with in research are serovars of *Salmonella enterica*. Three serovars typically discussed in research are *S. Typhi*, *S. Typhimurium* and *S. Enteritidis*. *S. Typhi* is species specific to humans and is the causative agent of typhoid fever. *S. Typhimurium* is a common source of food poisoning in humans and causes typhoid like symptoms in mice. *S. Enteritidis* has become the prevalent cause of food poisoning in the U.S. and often exists at sub-clinical levels in poultry flocks. Similar to necrotic enteritis, sub-clinical levels of *Salmonella* can result in a loss in production



Spring et al. (2000) utilized bacterial isolation chambers to detect the effects of MOS on *Salmonella* inoculated birds. Eggs were sanitized and upon hatch chicks were inoculated with a solution derived from a commercial hatchery in an effort to standardize endogenous microflora populations. In keeping with the sterilized design feed, water and litter were autoclaved in preparation for chicks. Upon challenge with *S. typhimurium* 29E concentrations in MOS treatment group were lower than the control group (Figure 4). In a

similar series of trials performed with *S. dublin*, which typically colonizes the intestine at low concentrations, MOS decreased the number of salmonella-positive birds from 89.8 to 55.7% (Figure 5). No differences in concentrations of coliforms, *lactobacilli*, *enterococci* and anaerobic bacteria were observed with the addition of MOS in any of the performed trials. The same was true of cecal pH, VFA and lactate concentrations. Similar effects were seen in a study which determined that the incidence of *Salmonella* was reduced with the use of Bio-Mos[®]. *Salmonella* counts in the cecum, liver and spleen were all lower with the use of Bio-Mos[®] though no differences between 0.1 and 0.2% inclusion rates were observed (Yamaguchi and Tanaka, 2001). Fernandez et al. (2000) set out to determine the protective effects of feeding 9 day old chicks cecal contents from adult hens fed dietary carbohydrates (mannose, MOS, or palm kernel meal). Authors reported that both carbohydrates provided optimal protection when chicks were supplemented with the same oligosaccharides as the hens. Feeding cecal contents remained protective until 10⁶ fold dilution, where protective capabilities were then lost.



Campylobacter jejuni

Campylobacter is one of the primary causes of bacterial gastroenteritis worldwide, with poultry being the major disease vector. The Center for Disease Control estimated that over 1 million people in the U.S. are infected annually (CDC, 2005). *Campylobacter* adhesion mechanism differs from that of *Salmonella* and *E. coli* in that it utilizes glycosylated proteins on its cell surface, including its flagellin, as a means of attachment (Guerry, 1997; Moser et al., 1997). Mutants deficient in the N-linked general protein glycosylation pathway (Pgl) were unable to adhere and invade human epithelial Caco-2 cells (Karlyshev et al., 2004). These results provide strong evidence that glycosylation is essential to the adherence capabilities of *C. jejuni*. Several studies have shown that oligosaccharides are capable of prohibiting *Campylobacter* attachment. Alpha 1,2-linked fucosylated glycans are the dominant glycan structure found in human breast milk and are believed to constitute the major innate immunological mechanism which protects infants against pathogenic infections. In vivo and in vitro binding studies have linked these fucosylated glycans with inhibition of *campylobacter*, cholera, enterotoxigenic *E. coli*, and major strains of caliciviruses (Morrow et al., 2005). A study with poultry utilized fermented feed with high concentrations of *lactobacilli* and lactic acid with low pH. Although the fermented feed source did not change *Campylobacter* colonization levels within the ceca, it did reduce the likelihood of bacterial shedding by infected birds (Heres et al., 2002). Authors theorized that fermented feed increased crop and gizzard pH, reducing the likelihood of *campylobacter* to reach the lower gut. It has been suggested that mannose may reduce *C. jejuni* adherence by 50 to 60% (McSweegan and Walker, 1986). Mechanisms through which inhibition occurs remain unknown given the inability of *Campylobacter* to exhibit mannose-sensitive

agglutination of MOS. Despite this, several studies have reported protective effects of dietary mannose on *C. jejuni* colonization. Schoeni and Wong (1994) found that a competitive exclusion culture (*Citrobacter diversus*, *Klebsiella pneumoniae* and *E. coli*) combined with mannose caused a 62% reduction in the

Table 3: Prevention studies: ability of CE cultures and Mannose treatments administered on day 1 to prevent colonization by *C. jejuni* administered on day 3.

Treatment	No. of trials	No. of chicks	% Colonization	Infection Factor	Protection factor
Control	6	37	61.6	2.2	NA
Competitive Exclusion culture (CE)	5	27	20.2	<1.1	>2.7
Mannose with:					
No CE	4	25	12.9	<0.3	>5.0
CE	3	25	0.0	<0.2	>13.2

Schoeni and Wong, 1994

rate of colonization (Table 3). A more recent trial determined that birds supplemented with Bio-Mos[®] and challenged with *Campylobacter* (10^8 cfu) had reduced cecal colony counts and higher cecal pH as compared to *Campylobacter* only treatments (Anderson et al., 2005). Another study observed carcass contamination found that *Campylobacter* presence was lower in birds fed Bio-Mos[®] (1kg/tonne of feed). Overall cloacal infection was reduced from 3.51 to 1.87 on a log scale (Tucker et al., 2005).

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