POULTRY/EGGS

EFFICIENCY OF PROBIOTICS, PREBIOTICS AND SYNBIOTICS ON WEIGHT INCREASE OF CHICKENS (*GALLUS DOMESTICUS*)

(G. Zhang, L. Ma, and M.P. Doyle)

Preventing *Salmonella* contamination of poultry and poultry products remains a major challenge. With increasing concerns regarding antibiotics use in animals, probiotics [also called competitive exclusion bacteria (CE) in animals], prebiotics and synbiotics, which are natural and environmental friendly, are attracting greater attention. The use of probiotics to reduce *Salmonella* infection of poultry was first applied in 1973. At that time, it was determined that in newly hatched chicks, the rapid establishment of an adult-type intestinal microflora, by administration of an undefined culture of cecal microorganisms via the oral route, was protective against colonization by *Salmonella*. Since then, the protective effect of indigenous intestinal microflora against *Salmonella* colonization in young chicks has been confirmed by many studies. In previous research, the effect of four suspensions of hen caecal content diets supplemented with 2.5% D-mannose, 2.5% mannose-oligosacchiride, 2.5% palm kernel meal or unsupplemented mash have been compared. Results revealed that feeding caecal content diets supplemented with 2.5% mannose-oligosacchiride or 2.5% palm kernel meal were more effective against *S*. Enteritidis colonization in chicks than feeding caecal content diets alone. In another study, it has been reported that addition of 4g fructooligosaccharide/kg to chick feed enhanced the growth of *Bifidobacterium* spp. and *Lactobacillus* spp., but inhibited *E. coli* in the small intestine and the cecum. Our previous research also revealed that probiotics were effective in reducing *Salmonella* colonization of chickens. The question remains, do probiotics, prebiotics and synbiotics affect body weight gains of chickens? The objective of this study was to determine the effect of feeding probiotic bacteria, prebiotics, and synbiotics on weight gain of chickens.

Three trials have been completed by now. In all trials, day-of-hatch Ross x Ross chicks was used. Each treatment had 20 chicks. Two probiotic bacteria (CE) cocktails were tested. CE 1 was composed of Salm-9, List40-18 and List40-41 (all *Lactobacillus salivarius*); CE 2 was CE1 plus List40-13 (*Streptococcus cristatus*). Two prebiotics [fructooligosaccharide (FOS) and lactose] were mixed into chicken feed at 2.5% throughout the entire trials for prebiotic and synbiotic treatments. Chicks at 3 days old were challenged perorally with *Salmonella* Typhimurium (resistant to nalidixic acid) at 10^6 CFU/chick. In Trials 1 and 2, probiotic bacteria were administered to day-of-hatch chicks at 10^8 CFU/chick perorally. Body weight was determined at 26 days (Trial 1), and 12 and 19 days (Trial 2). In Trial 3, probiotic bacteria were added to drinking water (final concentration 10^6 CFU/ml) for the first week except day 3. Body weight was determined at 9 and 15 days.

In Trial 1, probiotics treatments CE 1 and CE 2, synbiotics treatment CE 2 plus FOS, and prebiotics lactose significantly ($\alpha = 0.05$) increased body weight by 83.0 to 100.5 g/chick at 26 days compared to controls. Although other prebiotics and synbiotics treatments also increased chicken body weight by 32.6 g/chick or more, this was not statistically different from the control group.

In Trial 2, synbiotics CE 2 plus lactose and prebiotics CE 2 significantly ($\alpha = 0.05$) increased body weight by 25.6 to 27.7 g/chick at 12 days; whereas synbiotics CE 1 plus lactose significantly increased body weight by 42.3 g/chick at 19 days. All other treatments at both 12 and 19 days had slightly higher body weights than controls except for two treatments (lactose only and CE 2 plus FOS at 19 days), which were 1.69 and 0.84g/chick lighter than controls, respectively.

In Trial 3, at 9 days of age all treatments except for lactose only increased body weight significantly; however, there was no significant difference between treatments and control in body weight at 15 days, although chickens in all treatment groups except one had heavier body weights than controls.

In conclusion, some probiotics or synbiotics were effective in increasing the body weight of chickens. Prebiotics alone (FOS or lactose) did not consistently affect body weight gain of chickens. The probiotics and synbiotics studied could be used to increase weight gain of chickens. Additional studies are needed to determine the influence of these probiotics and synbiotics on weight gain of broilers in commercial operations throughout their growing cycle.

CROSS-CONTAMINATION OF *LISTERIA MONOCYTOGENES* BETWEEN PROCESSING EQUIPMENT AND DELI MEATS

(L. Ma, G. Zhang, C-M. Lin, and M.P. Doyle)

Contamination of ready-to-eat meats by *Listeria monocytogenes* has resulted in outbreaks of listeriosis and major product recalls. Food processing equipment such as slicers can serve as a potential contamination source. This study was conducted to determine (i) the dynamics of cross-contamination of *Listeria monocytogenes* from slicing equipment to two different types of turkey meat, cured and non-cured, (ii) the role of the conveyor belt in the transfer event, (iii) the fate of *L. monocytogenes* on contaminated samples during storage at 4°C for up to 90 days, and (iv) the efficacy of the BAX-PCR and USDA conventional enrichment culture assays in detecting *L. monocytogenes* on turkey meats. A five-strain mixture of *L. monocytogenes* was inoculated at ca. 500 CFU onto the blade of a commercial slicer. Five consecutive meat slices were packed per package, vacuum sealed, stored at 4°C, and sampled (entire package) at 1, 30, 60, and 90 days postslicing. Of the two types of deli meats, a larger number of *L. monocytogenes*-positive samples were obtained from non-cured turkey meat, 48 of 800 samples compared to 14 of 800 of cured turkey meat. Most of the *L. monocytogenes*-positive samples in cured turkey meat were detected at 30 days postslicing, whereas the largest number of *L. monocytogenes*-positive samples for non-cured turkey meat was recovered at 90 days postslicing. Slightly more (48 vs. 43 of 800 samples) *L. monocytogenes*-positive meat samples were obtained when the conveyor belt was used and the positive meat samples obtained at the middle or near the end of sliding were likely from contamination of the conveyor belt. For cured turkey meat, *L. monocytogenes* was detected in 5 meat samples by both the enrichment culture and BAX-PCR assays and in 9 samples only by enrichment culture assay. For non-cured turkey meat, *L. monocytogenes* was detected in 23 samples by both assays in 13 samples only by the enrichment assay, and in 10 samples only by the BAX-PCR assay. *L. monocytogenes* cell numbers were generally very low when detected on either type of turkey meat contaminated during processing. The results indicate that *L. monocytogenes* can be transferred from a contaminated slicer onto deli meats and survive storage at 4°C. The enrichment culture and BAX-PCR assays are complementary to each other in the detection of *L. monocytogenes*, especially for non-cured turkey meat.