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FEEDING STRATEGIES TO REDUCE MALABSORPTION AND ENTERIC DISORDERS IN POULTRY

Hector M. Cervantes, DVM, MS, Dip. ACPV
Manager, Technical Services
Phibro Animal Health
Poultry Business Team
Watkinsville, Georgia

hector.cervantes@phibroah.com

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INTRODUCTION

Due to the complexity and volume of information currently available, this presentation will be limited to a review of malabsorption syndrome of broiler chickens and turkeys. The malabsorption syndrome (MAS) of broiler chickens was first reported in the United States by Dr. Duane E. Olsen in 1977 at the 26th Western Poultry Disease Conference. A year later, the same syndrome was reported in broiler chickens in Europe (B. Kouwenhoven, *et al*., 1978a). The first report of MAS in southeastern U.S. broilers was made by Dr. Robert K. Page, *et al.*, in 1982. The first description of MAS in turkeys occurred in Europe in 1984 by C.D. Bracewell and C.J. Randall, this syndrome in turkeys is known as infectious stunting syndrome (ISS).

ONE SYNDROME WITH MANY NAMES

MAS and ISS are ill-defined infectious enteric disorders of chickens and turkeys that cause maldigestion and malabsorption and result in poor flock performance and a variety of clinical signs. Over the years, MAS and ISS have received many descriptive names primarily related to the clinical signs observed in chicken and turkey flocks, some of these are: stunting–diarrhea complex (D.E. Olsen, 1977), helicopter disease (B. Kouwenhoven, *et al*., 1978a), runting and leg weakness (B. Kouwenhoven, 1978a), infectious proventriculitis (B. Kouwenhoven, *et al*., 1978b), brittle bone disease (van der Heide *et* al., 1981) runting and stunting (M.F. McLoughlin, *et al*., 1987), pale bird syndrome (R.E. Good, 1982), and infectious stunting syndrome (C.D. Bracewell and C.J. Randall, 1984; R.L. Reece and J.A. Frazier, 1990). The problem with all the names used to describe MAS and ISS in chickens and turkeys is that they identify clinical signs rather than a specific disease. More than likely this is the result of the complex and multi-factorial etiology of MAS and ISS.

CLINICAL SIGNS OF MAS/ISS

Many symptoms have been associated with MAS/ISS in chickens and turkeys, some of these are: Stunting, elevated feed conversions, mucoid to watery diarrhea with yellow to orange droppings, cloacal pasting, pale shanks and skin, poor feathering, increased incidence of lameness and leg weakness, secondary lipid-soluble vitamin deficiencies, increased mortality (especially in turkeys).

LESIONS OF MAS/ISS

Following is a list of lesions most frequently reported in chickens and turkeys affected by MAS/ISS: Enlargement of the proventriculus, decreased size of the gizzard, distended intestines filled with mucoid to watery contents, poorly digested feed and orange-tinged

mucous, poor calcification of the skeletal system, atrophy of the pancreas, atrophy of the thymus and bursa of Fabricius, increased amount of pericardial fluid.

ETIOLOGY OF MAS/ISS

MAS/ISS was originally thought to be caused by reovirus since this virus was often isolated from birds exhibiting typical clinical signs (D.E. Olsen, 1977; R.K. Page, *et al*., 1981; L. van der Heide and M. Horzinek, 1981). However, subsequent reports have implicated other enteric viruses, such as; enterovirus (M.S. McNulty *et al*., 1979); calicivirus (P.J. Wyeth, *et al*., 1981); parvovirus (J. Kisary, *et al*., 1984); torovirus (D.L. Reynolds); astrovirus (D.L. Reynolds and Y.M. Saif, 1986; M.L. Thouvenelle, *et al*., 1992). Other reports have also suggested the involvement of bacteria (B. Kouwenhoven, 1978a) and long segmented filamentous organisms (LSFOs; C.R. Angel, *et al*., 1990b). However, although a syndrome nearly identical to MAS/ISS can be reproduced experimentally by feeding ground intestines from birds affected by clinical MAS/ISS to either chickens (D.E. Olsen, 1977; B. Kouwenhoven, *et* al., 1978a; R.K. Page, *et* al., 1981) or turkeys (M.F., McLoughlin, *et* al., 1987; C.R. Angel, *et al*., 1990a), experimental attempts to reproduce the syndrome with individual viruses or bacteria have failed. This has led other researchers (B. Kouwenhoven, *et al*., 1983) to conclude that a combination of viruses and bacteria is required for the successful experimental reproduction of the syndrome, although to this date not one combination of bacteria and viruses has been found that reproduces MAS/ISS like the feeding of crude intestinal homogenates.

ADVERSE EFFECTS OF MAS/ISS ON PERFORMANCE

Many field reports and controlled experiments have documented the adverse effects of MASS and ISS on performance of broiler and turkey flocks (D.E. Olsen, 1977; B. Kouwenhoven, *et al*., 1978a,b; C.D. Bracewell and P.J. Wyeth, 1981; R.K. Page, *et al*., 1982; C.D. Bracewell, 1982; G. Asdrubali, *et al*., 1983; C.D. Bracewell and C.J. Randall, 1984; R.L. Reece and J.A. Frazier, 1990; C.R. Angel, *et al*., 1990a,b,c; C.R. Angel, *et al*., 1992; H.A. Al-Batshan, *et al*., 1992; D.W. Trampel and J.L. Sell, 1994).

Tables 1 and 2 demonstrate the adverse effects of MAS inoculation on weight gain of broiler chicks, the tables come from the first reported cases of MAS in broiler chickens in the United States. Table 3 summarizes the results of a series of experiments with ISS in turkey poults published in 3 papers (C.R. Angel, *et al*., 1990a,b,c), that perfectly illustrates the adverse effects of ISS on turkey poult performance as measured by body weight gain and feed efficiency.

Table 1. MAS Effects on Broiler Chick Weight Gain[1]

INOCULA	14 DAY WEIGTH (G)	21 DAY WEIGHT (G)	28 DAY WEIGHT (G)
0.5 ml. Gut suspension	134	236	405
0.1 ml. Embryo propagated virus	196	332	534
Controls	243	410	661

[1] From D.E. Olsen, 1977.

Table 2. MAS Effects on Broiler Chick Weight Gain[1]

GROUP	MALE WEIGHT[2] (G)	FEMALE WEIGHT (G)
Controls	1235	950
Inoculated (Isolate 1)	1028	786
Inoculated (Isolate 2)	860	799

[1] From R.K. Page, *et al*., 1982.
[2] Average weight for 2 replicates, 10 chicks per replicate.

Table 3 summarizes the effects of ISS on performance of turkey poults.

Table 3. ISS Effects on Early Weight Gain, Feed Consumption and Feed/Gain Ratio of Turkey Poults.

RESEARCHER	GAIN (G/DAY) 1-13 DAYS		FEED CONS. (G/DAY) 1-13 DAYS		FEED/GAIN RATIO 1- 13 DAYS	
C. R. Angel, *et al*., 1990a	CONT* 39.5	INOC 17.8	CONT 56.8	INOC 36.1	CONT 1.438	INOC 2.028
C.R. Angel, *et al*., 1990b	CONT 40.62	INOC 16.08	CONT 54.3	INOC 31.02	CONT 1.337	INOC 1.929
C.R. Angel, *et al*., 1990c	CONT 37.1	INOC 21.1	CONT 57.6	INOC 41.3	CONT 1.552	INOC 1.957

* CONT = Uninoculated group; INOC = Inoculated group

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NUTRITION-RELATED RESEARCH ON MAS/ISS

In 1982 M. S. Lilburn, *et al*., conducted studies on MAS, lipid absorption and energy utilization with broiler chickens naturally infected in the field and compared them to uninfected chickens raised at The University of Georgia. Lilburn's research clearly showed impaired absorption of dietary lipids and energy utilization in the MAS-affected birds. The authors concluded that the differences in lipid absorption accounted for only a portion of the energy not utilized by the MAS-affected chickens and suggested that absorption of amino acids, carbohydrates and other energy yielding compounds must also be impaired in MAS-affected chickens.

Also in 1982, G.L. Colnago, *et al*., while conducting research studies with broiler chickens on the effect of supplemental vitamin E and selenium on the immune response to vaccination against coccidiosis confronted a natural outbreak of MAS. The experiment switched from the original purpose to study the effects of dietary supplementation with vitamin E and selenium on MAS. In the MAS-affected chickens mortality, primarily due to encephalomalacia, was reduced in those fed the diets supplemented with either vitamin E or selenium, also the chickens fed the diet supplemented with vitamin E and selenium were significantly heavier than the ones fed the unsupplemented (basal) diet. The authors concluded that the supplemental vitamin E and selenium was beneficial to the MAS-affected birds and probably reduced mortality and improved weight gain by increasing tissue levels of vitamin E and preventing deaths by encephalomalacia.

In another study conducted in 1985, J.R. Veltmann, Jr. *et al*., found an interaction between supplemental dietary vitamin A and MAS-affected broiler chickens. Increasing supplemental levels of vitamin A exacerbated the effects of MAS. The authors reported higher mortality and lower body weights, feed consumption, bone ash, and serum concentrations of calcium and phosphorus, in the MAS-affected chickens. In addition, the researchers observed a significant interaction for bone ash in the MAS-affected chickens, where bone ash was significantly lower in those chickens fed the higher levels of supplemental vitamin A in the diets with a high level of vitamin D. The lower bone ash values of the MAS-affected chickens correlated with an increased incidence of rickets that, between the 3rd and 4th week, shifted from rickets to tibial dyschondroplasia. The authors concluded that a nutritional antagonism between excess vitamin A and vitamin E or vitamin D may account for the exacerbative effect of vitamin A on MAS.

In nutritional studies conducted on ISS of turkeys and reported in 1992 by C.R. Angel, *et al*., feeding a complex diet containing fish meal and sunflower meal as the main protein sources as opposed to soybean meal, completely prevented in one experiment the adverse effects of ISS on performance, and partly overcame the adverse effects of ISS on another

two experiments. The authors were unable to conclude what properties of the complex diet made it effective in ameliorating the deleterious effects of ISS.

In another series of studies (C.R Angel, *et* al., 1990c) conducted on ISS of turkeys it was shown that the activities of disaccharidases (maltase and sucrase) in the jejunum and ileum of ISS-affected poults were less than in control ISS-free poults. In the same studies, the activity of pancreatic enzymes (trypsin, amylase and lipase) was not affected by ISS. In the same studies, decreased nutrient retention (dry matter, protein, ash, fat and gross energy) in ISS-affected poults only accounted for a small portion of the growth depression not accounted for by a reduction in feed intake, leading the authors to conclude that rather than maldigestion and malabsorption, impaired metabolism of retained nutrients causing inefficient use of nutrients for growth may serve to explain the severe growth depression and poor feed efficiency of ISS-affected poults.

In subsequent studies by the same research group (H.A. Al-Batshan, *et al*., 1992), the feed additive antibiotic Virginiamycin (Stafac®) fed at 22 ppm significantly improved body weights and feed efficiencies of uninoculated control turkey poults and ISS-inoculated poults. According to the authors, Virginiamycin helped ISS-inoculated poults by facilitating a notable recovery when compared to ISS-inoculated poults not fed Virginiamycin. Additional research demonstrated that Virginiamycin increased specific activities of maltase and sucrase in uninoculated control poults, however, this effect was not observed in the ISS-inoculated poults. The authors concluded that the beneficial effect of Virginiamycin on ISS-inoculated poults did not seem to be mediated by changes in physical or functional properties of the small intestine, as judged by jejunal weight, length or density or by jejunal maltase and sucrase activities.

The effect of another feed additive antibiotic, Bacitracin Methylene Disalicylate (BMD) on ISS was reported 2 years later (D.W. Trampel and J.L. Sell, 1994). BMD was fed at 55 and 220 ppm. BMD at 55 or 220 ppm did not improve significantly body weights or feed efficiency of uninoculated control turkey poults. However, at the highest level (220 ppm) BMD significantly improved body weights and hydration efficiency in the ISS-inoculated poults. Activities of intestinal disaccharidases (maltase and sucrase) were not determined in this study.

FEEDING STRATEGIES TO AMEILORATE MAS/ISS

From the research studies previously reviewed it appears clear that there is not one single strategy that could reliably and completely prevent the adverse effects of MAS/ISS on performance of broiler chicken and turkey flocks. However, from the same research studies several recommendations or strategies can be used when confronting an outbreak of MAS/ISS in the field. These recommendations may not completely prevent the

adverse effects of MAS/ISS but should help in minimizing its adverse effects on poultry performance.

- Increase the levels of vitamin E in the ration, in the original study by G.L. Colnago, *et al*., an additional level of vitamin E of 45 I.U. (100 I.U./Kg) proved effective in ameliorating the adverse effects of MAS in broiler chickens. Lower levels may also be beneficial but were not tested as the original study was not designed for MAS.

- Maintain a level of supplemental selenium in the ration of at least 0.25 ppm. The original paper by G.L. Colnago, *et al*., supplemented the basal ration with 0.25 ppm of selenium. At that time the FDA-approved level of supplemental selenium was limited to 0.1 ppm, since then it has been increased to 0.3 ppm.

- Maintain levels of supplemental vitamin A as low as possible (3500 I.U./lb or 7700 I.U./Kg) to support good growth in broiler chickens. In the original paper by J.R. Veltmann, Jr., *et al*., supplemental vitamin A caused a further reduction in body weight and bone ash in MAS-affected chickens.

- Feed a more complex ration containing alternate ingredients (fish meal and sunflower meal) as the main sources of protein. In the original paper by C.R. Angel, *et al*., a complex ration was completely effective in preventing the adverse effects of ISS in turkey poults inoculated with a moderate ISS challenge, and partly effective in preventing the adverse effects of a severe ISS challenge.

- Include a feed additive antibiotic in the ration. Either Virginiamycin (Stafac®) fed at 22 ppm or bacitracin methylene disalicylate (BMD) fed at 220 ppm have shown to enhance the performance of ISS-affected turkey poults.

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