Use of Monensin Sodium in Caged Layer Replacements for Control of Coccidiosis

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Primary Audience: Veterinarians, Live Production (Layers) Managers

SUMMARY

Two, paired-house trials were conducted to determine the relative efficacy of monensin sodium for control of coccidiosis in caged layer replacements. Parameters evaluated included clinical diagnosis, weight gain, feed conversion, uniformity, and mortality. Two genetic lines of pullets were included, one on each of two farms. One farm had a history of coccidiosis, whereas it had not been diagnosed on the second farm. One farm had two incidences of coccidiosis, clinically diagnosed, in the amprolium-treated house. Birds on this farm that were fed monensin had lower mortality; feed conversion was improved; the birds were more uniform and weighed approximately 28 g more than birds not receiving monensin. No coccidiosis was diagnosed on the second farm. In caged pullets, inclusion of monensin sodium in feed at levels consistent with the label claim adequately controlled coccidiosis, as evidenced in the parameters evaluated.

Key words: monensin, coccidiosis, pullet


DESCRIPTION OF PROBLEM

Coccidiosis in poultry, caused by species of the protozoan Eimeria, is responsible for significant economic loss [1]. Numerous performance factors may be impacted as a result of infection by this parasite. Although significant attention has been given to the economics of controlling the disease in meat production birds, the laying industry has not received similar focus. Mortality affects both equally; however, uniformity, feed efficiency, and weight gain are assessed somewhat differently [2]. In the laying industry, uniformity is critical to future performance factors such as time and level of peak lay, persistence, egg grade, and bird health. Feed efficiency is assessed for both bird weight and egg quality and quantity [3]. Coccidiosis may increase mortality and reduce feed efficiency, and recovered birds may demonstrate lack of uniformity throughout the life of the flock [4].

Coccidiosis is not commonly considered in caged birds; however, it is present, and bird strain susceptibility differences may alter the apparent incidence within a given complex. Coccidiosis is traditionally diagnosed by increased mortality and postmortem identification of lesions but may not include more subtle signs such as variation in uniformity or reduced feed efficiency. Routine intestinal monitoring, common in the broiler industry for lesions indicative of coccidiosis, is less frequently applied to the laying industry.

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A disparity exists between results noted in research-facility trials and those measurable in field situations. It is often beneficial to validate research trials by conducting field evaluations with the realization that repetition may be minimal and variation less controllable. This trial was designed to investigate the role of two anticoccidial programs in a commercial setting.

Control measures in layers have traditionally relied on immunity via vaccination or through treatment programs that allow the disease to progress until clinical signs are evident and then a drug is administered [5]. Live vaccine strains rely solely on immunity and may be pathogenic, causing mild flushing and lesions, which can require chemotherapeutic intervention [4, 6]. Effective treatment programs disrupt the cycling coccidia, which are needed to maintain adequate immunity. Vaccination programs are, in essence, controlled exposure to disease and are subject to many management factors recognized as contributors to severity of coccidiosis [6, 7]. Inattention to these factors can result in severe coccidiosis. While immunity develops, loss of uniformity, reduced feed efficiency, and mortality may occur.

Treatment programs rely solely on control of the disease, and leave birds completely at risk from an immune perspective. Because treatment is administered in response to clinical signs of disease, uniformity, feed efficiency, and mortality are all affected. The ionophore monensin is recognized for utility in balancing control with immunity [8]. The mode of action is considered to be coccidiocidal; however, it does not affect the total coccidial population equally. Some of the parasites progress, in a debilitated form, through subsequent life cycle stages and elicit an immune response from the host. By changing the level of drug administered, the poultry producer can manage and control immunity based on evaluation of the birds. Routine health checks, including intestinal lesion scoring, and monitoring performance parameters will provide accurate assessment of an effective program. This report presents data from paired-house trials of pullets raised using monensin vs. an amprolium treatment program.

**METHODS AND MATERIALS**

**Trial Site**

Farms (identified as TH and EB) were selected that had two houses of similar design and holding capacity and had been actively raising replacement pullets for several years. Historical information was used to develop a background of flock performance and health concerns over several growing periods. The paired house design was selected to minimize husbandry variables and farm-to-farm variation that make interpretation between treated farms difficult. The use of the paired-house design compares two treatments at a single location and is beneficial when evaluating treatments under field conditions.

**Birds**

Commercial white layer pullets representing two distinct genetic lines were purchased, and a single genetic line was placed in cages in each house on a farm from day of hatch through 17 wk.

**Standard of Care**

All birds were beak-trimmed at 4 wk and vaccinated with live vaccine as follows: 2.0 wk Newcastle disease (ND), bronchitis, and infectious bursal disease (IBD); 3.4 wk IBD; 4.6 wk ND, bronchitis and IBD; 7.0 wk pox and laryngotracheitis (LT); 9.0 wk ND and bronchitis; and 13.0 wk LT. Six birds, randomly selected from each house, were euthanized and necropsied at 4-wk intervals, starting at approximately 6 wk of age. Intestinal lesions were noted and scored using the technique of Johnson and Reid [9].

**Anticoccidial Programs**

One house on each farm was given feed containing monensin sodium at 90 g/tonne beginning at 2 wk of age and continuing through approximately 10 wk of age or when the first new load of feed was delivered after that date. The other house received amprolium per label instructions in the feed at 2 wk, at 6 to 7 wk, and as needed thereafter if evidence of coccidiosis was present.

**Evaluation Parameters**

Accumulated mortality, body weight, body weight uniformity, and feed consumption data were collected when the birds were moved to the layer facilities at 17 wk. Feed conversion was calculated from the weight and feed consumed data. Uniformity was calculated by weighing 1% of the birds from each house and determining the percentage that were within 10% of the house...
mean. Necropsy and histopathology at the state diagnostic laboratory, of birds found dead, were used to confirm clinically apparent coccidiosis.

RESULTS AND DISCUSSION

Performance parameters evaluated are presented in Table 1. No evidence of clinically significant coccidiosis was noted in birds placed on the EB farm, which had a prior history of coccidiosis. Mortality associated with coccidiosis was noted in birds in the amprolium-treated house at the TH farm at 8 and 12 wk of age and was confirmed by necropsy and histopathology. Treatment was initiated in the 9th and 12th wk in the amprolium-treated house, reducing mortality and evidence of lesions.

Although direct challenge with coccidial oocysts was not part of this evaluation and limited the interpretive value, repeat incidence of coccidiosis at the TH farm amprolium-treated house but not at the monensin house, suggests that monensin provided adequate control of coccidia. Necropsy of euthanatized birds from all houses revealed occasional lesions, scored as two or less, and suggested that coccidia were cycling through the flocks.

All birds from the TH farm had mild airsacculitis at the initial necropsy session (6 wk), which was resolved in remaining birds at subsequent visits. Two of six birds in the amprolium-treated and one of six in the monensin-treated house had mild enteritis, but the cause was not determined. During the same session, one of six birds in each house at the TH farm had lesions consistent with *Eimeria acervulina*. At the EB farm, enteritis was noted in one of six birds in the amprolium-treated house at the initial necropsy session. At the second session, 2 wk posttreatment with soluble amprolium, no lesions were noted in the house treated with amprolium, whereas two of six birds had lesions, score of 1, consistent with *Eimeria maxima* in the house receiving monensin.

CONCLUSIONS AND APPLICATIONS

1. Effective coccidiosis control can be achieved when using monensin sodium in replacement pullets. Reductions in mortality, improved feed efficiency, and uniformity were noted and are a result of good health practices, including a quality coccidiosis control program.

2. Routine monitoring of bird health, including intestinal lesions, can be helpful in assessing the effectiveness of the control program, but overall performance is the key criterion that should be considered. No program will completely eliminate coccidial infection, but good management practices must be used to maintain a successful program.

3. Improved weight gain, feed conversion, and mortality was noted in the houses receiving monensin at 90 g/tonne provided to pullets from 2 to 10 wk of age. An anticoccidial program built around inclusion of monensin in the feed for pullet replacements may aid in the control of coccidiosis thus improving performance.

### TABLE 1. Comparison of selected parameters from birds fed either monensin sodium or amprolium at 17 wk

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Breeder</th>
<th>TH 1</th>
<th>TH 2</th>
<th>Breeder</th>
<th>EB 1</th>
<th>EB 2</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mortality, %</td>
<td>4.30</td>
<td>2.88</td>
<td>1.10</td>
<td>4.15</td>
<td>1.74</td>
<td>1.25</td>
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<td>Weight, g</td>
<td>1,229</td>
<td>1,222</td>
<td>1,254</td>
<td>1,102</td>
<td>1,094</td>
<td>1,103</td>
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<tr>
<td>Uniformity, %</td>
<td>NA</td>
<td>78.70</td>
<td>84.40</td>
<td>NA</td>
<td>91.00</td>
<td>87.20</td>
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<tr>
<td>Feed consumption, kg</td>
<td>5.52</td>
<td>4.80</td>
<td>4.79</td>
<td>4.23</td>
<td>4.09</td>
<td>4.11</td>
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<tr>
<td>Feed conversion, kg</td>
<td>NA</td>
<td>3.93</td>
<td>3.82</td>
<td>NA</td>
<td>3.74</td>
<td>3.73</td>
</tr>
</tbody>
</table>

| Farm identifiers; AMP = house receiving amprolium; MON = house receiving monensin; NA = not available.

| Birds on the TH farm experienced increased mortality at 8 and 12 wk of age. Lesions consistent with coccidiosis, and microscopic evidence of coccidial oocysts were used to confirm a diagnosis of coccidial infection. |
REFERENCES AND NOTES


