SCHERING-PLOUGH ANIMAL HEALTH
technical services bulletin

Clinacox™ and Monensin: Impact of Anticoccidials on Development of Immunity and Performance in Turkeys

In the US turkey industry, it is common practice to include anticoccidials in the feed only for the first 10 weeks of growout. Turkey growers rely on age resistance and the development of immunity to protect the flock against coccidiosis from 10 weeks of age until processing.

The recent approval of Clinacox® (diclazuril) for use in turkeys has raised concerns about the impact of an effective chemical when used only through 10 weeks of age. Dr. David Chapman of the University of Arkansas recently completed a pen trial that examined the impact of feeding either Clinacox or monensin on the development of immunity in 10-week-old turkey poults. The study also compared the impact of both anticoccidials on performance at 16 weeks of age.

Monensin was selected for comparison because it is used widely in the industry. Its label includes a warning that use of this ionophore may interfere with the development of immunity to some coccidial species.

Key Points

- **A study compared the impact of feeding either Clinacox™ or monensin on the development of immunity in 10-week-old turkey poults. The study also compared performance at 16 weeks of age.**
- **By 10 weeks of age, birds fed either anticoccidial had not developed immunity to a mixed coccidial challenge.**
- **Birds fed Clinacox had minimal oocysts after withdrawal of the anticoccidial and had numerically better weight gain and feed conversion compared to those fed monensin.**

Study Design

Eighteen floor pens each containing 22 poults were randomly divided between three treatment groups. They received either:

- Clinacox (1 ppm) from placement through 10 weeks of age
- Monensin (60 g/ton) from placement through 10 weeks of age
- No anticoccidial products (controls)

A fourth group of birds was raised in isolation to serve as unexposed (susceptible) controls for the challenge study.

During the first 9 days following placement, the floor pen birds were exposed to a simulated natural challenge of *Eimeria dispersa*, *E. meleagrimitis*, *E. adenoides* and *E. gallopavonis*; these are *Eimeria* species all known to be sensitive to both anticoccidial products. The infection rate was monitored by litter oocyst counts from all pens beginning at week 2 and continued on a weekly basis through termination of the study at 16 weeks. Mean oocyst counts are summarized in Table 1

Table 1: Litter oocyst counts: oocysts/gm of litter (thousands)

Week	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16
Clinacox	0.5	0	0	0.1	0.1	0.1	0	0	0.1	0.15	0	1.6	0	2.15	1.25
Monensin	17.0	91.5	27.6	2.4	3.1	2.9	1.5	0.6	0.6	0.58	0.30	0.50	0.38	0.32	0.50
Non Medicated	166.8	113.3	34.1	8.4	15.5	8.7	5.0	0.5	3.4	0.38	0	0.18	0	0.28	0.25

and demonstrate a substantial early infection rate in non-medicated poults.

At 6 weeks of age, 12 poults per pen were transferred to adjacent pens across the aisle for performance evaluation. They remained on their assigned feeding program after transfer.

At 10 weeks of age, all groups were placed on non-medicated feed, representing common industry practice.

Immunity Study

At 10 weeks of age and after 3 days on non-medicated feed, all remaining birds in the original pens were captured, weighed and wing banded.

Four randomly selected poults per pen were orally challenged with a mixture of *E. dispersa*, *E. meleagrimitis*, *E. adenoides* and *E. gallopavonis*. Four pen-mates remained unchallenged controls. All birds were weighed again at 7 days post-challenge. Immunity was evaluated based on weight gain in challenged vs. unchallenged control birds over the 7-day period.

Any remaining birds in each pen were challenged with the *Eimeria* mixture and necropsied 6 days post-challenge. The poults were evaluated for immunity based on oocysts in intestinal scrapings and gross cecal lesions. The birds raised in isolation served as the challenged and non-challenged susceptible controls for this study.

The weight gain post-challenge and necropsy results are summarized in Tables 2 and 3.

Immunity Study Conclusions and Discussion

Oocyst shedding was almost completely suppressed while Clinacox was fed to the test poults. The poults did not develop immunity during this 10-week period as evaluated by weight gain post-challenge and by lesion and oocyst scores in necropsied birds.

Table 2: Effect of challenge on weight gain at 7 days post 10-week challenge

Treatment	Challenged Weight Gain Day 0-7 (kg)	Not Challenged Weight Gain Day 0-7 (kg)	Challenged Weight As % of Not-Challenged Weight	Immune?
Clinacox	0.577[b]	1.060[a]	54	no
Monensin	0.543[b]	1.047[a]	52	no
Non-Medicated	1.072[a]	1.015[a]	105	yes
Susceptible Control	0.571[b]	1.014[a]	56	no

Different superscripts indicate significantly different results.

Treatment	Challenged	Average Oocyst Score[1]			Cecum Gross[2]	Immune?
		Duodenum	Mid-Intestine	Cecum		
Clinacox	yes	++	+++	++	0.50	no
Monensin	yes	++	+++	++	2.66	no
Non-Medicated	yes	–	–	–	0	yes
Susceptible Control	yes	++	++	++++	2.0	no
Susceptible Control	no	–	+	–	0	N/A

Table 3: Post-challenge necropsy results: cecal lesion and intestinal oocyst scores

[1] Subjective evaluation of the number of oocysts per microscope field.

[2] Presence of white core and/or hemorrhage cecum.

Monensin reduced oocyst shedding compared to non-medicated controls, but still permitted substantial oocyst shedding during the 10-week medicated feed period. Despite the obvious cycling of some coccidial species, substantial immunity to the mixed *Eimeria* challenge failed to develop after 10 weeks. In fact, gross cecal lesion scores were noticeably higher for the monensin test group than for the Clinacox test group, and equaled the gross lesion scores of susceptible controls.

Poults exposed to the simulated natural infection without medicated feed developed substantial immunity to challenge, as evaluated both by weight gain and coccidial scoring post-challenge.

Despite the failure to develop immunity in the two medicated test groups, litter oocyst counts remained minimal from 10 weeks through termination of the study at 16 weeks. Age-related resistance may explain the reduction in oocyst shed.

Comparative Performance Evaluation and Conclusions

Weight gain, feed intake and feed conversion ratios for the remaining poults at 6 weeks, 10 weeks and 16 weeks of age were evaluated. Size limitations of the pens required termination of the study at 16 weeks. Performance is summarized in Tables 4, 5 and 6.

Treatment	Body Weight/Bird (kg)	Feed Intake/Bird (kg)	FCR
Clinacox	2.65[a]	3.75[a]	1.42
Monensin	2.62[a]	3.70[a]	1.41
Non-Medicated	2.39[b]	3.43[b]	1.44

Table 4: Comparative performance of treatments at 6 weeks of age

Different superscripts indicate significantly different results.

The poults fed either Clinacox or monensin demonstrated a significant advantage in weight gain and feed intake at 6 weeks of age compared to nonmedicated controls exposed to the simulated natural coccidial infection. By 10 weeks of age, only the Clinacox flocks demonstrated a significant advantage in weight gain and feed intake over the other treatments.

At the termination of the study, Clinacox pens still demonstrated numerically better body weight and feed conversion compared to poults fed either monensin or non-medicated feed.

Table 5: Comparative performance of treatments at 10 weeks of age

Treatment	Body Weight/Bird (kg)	Feed Intake/Bird (kg)	FCR
Clinacox	3.94[a]	9.08[a]	2.30
Monensin	3.78[ab]	8.76[ab]	2.32
Non-Medicated	3.60[b]	8.02[b]	2.23

Different superscripts indicate significantly different results.

Table 6: Comparative performance of treatments at 16 weeks of age

Treatment	Body Weight/Bird (kg)	Feed Intake/Bird (kg)	FCR
Clinacox	6.13	19.93	3.26
Monensin	5.94	19.70	3.33
Non-Medicated	5.88	18.69	3.19

Clinacox is a trademark of Janssen Pharmaceuticals.