



The Real Story: Distinctions Between Deccox[®], Bovatec[®], and Rumensin[®] That Impact Product Selection and Efficacy

Introduction

Information distributed by Elanco addresses differences that exist between decoquinatate (Deccox), monensin, (Rumensin) and lasalocid (Bovatec). While differences do exist, a full understanding of those differences and how they affect product selection and efficacy needs to be understood.

Items in Elanco Article* Needing Clarification *(quotes are italicized)*

“After invasion into the mucosa, decoquinatate may hold sporozoite development in the cycle.”

Decoquinatate **does** hold sporozoite development in the cycle, but evidence suggests this may not be the sole action of the product (detailed in other sections of this paper).

“The ionophores, monensin and lasalocid are thought to act in a similar manner. However, it is accepted that monensin is a more potent ionophore than lasalocid.”

Monensin **is not** more potent than lasalocid.

- Lasalocid and monensin express their anticoccidial effects at the same point in the coccidia life cycle; sporozoite, 1st and 2nd generation merozoite.¹

* Thomas E. Coccidiosis control in cattle. Elanco Animal Health.

^a based on total of *E. bovis*, *E. zuernii* and other oocyst, sq. root, 1000/g feces in non-medicated challenge group divided by the same total for each treatment group; exact time period not specified

^b based on sum of non-medicated challenge group total oocyst counts 3 weeks after challenge divided by the same total for each treatment group at the same time period

- Watkins et al. has shown monensin to reduce total fecal oocyst counts by 69.4, 89.6 and 95.3% when fed to challenged calves at 0.4, 0.8 and 1.2 mg/kg bodyweight respectively.² In contrast, lasalocid has been shown to reduce total oocyst shedding by 97.8% at a dosage 20% lower (the lasalocid label claim of 1 mg/kg BW) than the maximum dosage utilized in the Watkins study (see bullet point below).
- Stromberg et al., in a separate study but in the same laboratory as the Watkins study, found lasalocid to reduce total fecal oocyst counts by 90, 97.6, 97.8 and 99.8%^b when fed to challenged calves at 0.5, 0.75, 1.0 and 3.0 mg/kg bodyweight respectively.³
- Lasalocid has been shown to be more effective ($P < 0.05$) than monensin in reducing chick intestinal lesion scores when tested at several concentration levels against three out of five *Eimeria* species, and significantly ($P < 0.05$) reduced lesion scores in mixed *Eimeria* species.⁴ In that same study, chicks receiving lasalocid had numerically higher 7-day weight gains than those receiving monensin.
- Monensin is only more potent than lasalocid in its toxic ability.
 - Equine LD₅₀:⁵
 - lasalocid = 21.5 mg/kg BW
 - monensin = 2-3 mg/kg BW
 - Cattle lethal dose range:¹²
 - lasalocid = 50-100 mg/kg BW
 - monensin = 22.4-39.8 mg/kg BW.

“Decoquinatate - Action is due to reversible inhibition of electron transport and respiration in the mitochondria.”

It is true that decoquinatate has a static effect. However, what is unclear is the possible “cidal” effect of inhibiting mitochondrial respiration for extended periods of time.

- Miner (1976) found oocyst production and pathological effects of coccidiosis in calves decreased the longer decoquinatate is fed. In those same studies, clinical signs of coccidiosis did not recur for 23 days (total length of observation) after treatment was discontinued.⁶

The positive effects of decoquinatate in controlling coccidia migration are documented.

- At necropsy on post-infection day 29 of experimentally infected calves given decoquinatate at 0.5 mg/kg on a continuous basis, only one degenerate schizont was seen in histologic section from the lower portion of the ileum. Other tissues appeared normal.⁷
- An additional study by Fitzgerald⁸ revealed no oocysts in tissue samples from calves experimentally challenged with *E. zuernii*, treated with decoquinatate at 0.5 mg/kg from day 13 - 20, and necropsied on day 20.

“Ionophores and amprolium exhibit a “cidal” control of coccidiosis in which the organism is “killed.” This is in contrast to “static” control compounds (decoquinatate) in which coccidial development within the intestinal mucosa is prevented but the parasite is not killed.”

While the primary action of decoquinatate is “static,” it can also exhibit “cidal” properties.

- Many antibiotics with static action become cidal at increased dosages. Decoquinatate, fed to calves at 1.5 mg/kg BW was found to not only to arrest development and release of merozoites from schizonts, but also to kill sporozoites.⁹
- Poultry studies suggest quinolones exert primary static effect on sporozoites once they have entered intestinal epithelial cells, a secondary cidal effect against early schizontous stages, and a tertiary

effect against gametocytes.¹⁰

- In poultry, the “cidal” effects of decoquinatate may be overridden by the “static” effect.¹⁰ Therefore clinically, it may be difficult to determine if the product is static or cidal.

“Once use of the static product is discontinued, coccidial development may continue through the life cycle.”

“When Deccox is removed from either milk replacer or feed, the parasite located in the intestinal tissues resumes development and may result in a coccidiosis break.”

Extreme stress levels, management errors, and overwhelming challenge can precipitate breaks in coccidiosis control, regardless of the product involved.

Anytime product is discontinued, it is reasonable to expect those oocysts not yet contacted by the product to continue to develop. This is true with any anticoccidial. Since monensin is less efficacious than lasalocid in reducing oocyst shedding,^{2,3} one might expect greater shedding of oocysts when monensin is discontinued as opposed to lasalocid.

- Poultry studies suggest that if a quinolone (i.e., decoquinatate) is administered before infection at a concentration high enough to inhibit sporozoites, and is later withdrawn, infection will develop but be ameliorated because some of the first-stage schizonts will be killed by lower drug concentration as the tissue levels become depleted.¹⁰

“Deccox may allow continued development of the sporozoite through the schizont state with subsequent release of merozoites into the lumen of the intestines. At that point, Rumensin has the opportunity to contact the merozoites and kill them. However, in some cases, the number of merozoites may be so great that some are able to re-invade the tissues before Rumensin can kill them. That situation can lead to a possible cocci break in the situations where there is a significant coccidiosis challenge.”

- As previously stated, the ability of **any** anticoccidial product to control clinical coccidiosis can be overcome by overwhelming challenge. The ability to combat challenges is greatly dependent on drug intake and efficacy of the drug in the gut lumen. Deccox is

very palatable, which helps ensure correct drug intake and efficacy.

- Coccidia-infected calves typically express intake depression, and to truly measure an anticoccidial's effect on intake, the study must be conducted without experimental challenge. In the only reported Elanco calf starter study involving natural (no experimental) coccidia challenge, monensin-fed calves experienced numerically depressed intake when compared to natural controls.¹¹ Therefore, any coccidiosis break that occurs when a calf starter containing monensin follows a calf starter containing decoquinatate may have less to do with cidal or static control and more to do with feed intake. Decreased intake of the monensin-containing feed results in less monensin available for coccidiosis control. This, coupled with less than optimal efficacy as evidenced by Watkins,² may be factors contributing to the break.
- If there is a desire to switch diets from one containing decoquinatate to one containing an ionophore, lasalocid is the logical choice for the ionophore component. Both intake and efficacy data at varying dosages (Watkins, Stromberg) would suggest a smoother transition in intake and more effective coccidiosis control.

“Two courses of action can help prevent breaks during that time. First, use amprolium (Corid®) to kill the parasites at the schizont stage following removal of Decco from the diet. That will reduce the number of merozoites rupturing into the intestines and thus, reduce the number that Rumensin must kill.”

Amprolium is coccidiacidal and is indicated for the treatment of coccidiosis. In effect, what Elanco is saying is that they would like to have the calves cleared of oocysts prior to implementing Rumensin in the ration. Is this a lack of faith in Rumensin's ability to handle a cocci load for reasons cited above?

How does this recommendation apply to other Rumensin applications (i.e., feedlot starter rations) where stressed calves can be subject to extreme levels of coccidia in a natural challenge? Does Elanco exhibit that same lack of confidence?

Conclusions

Effective coccidiosis control involves proper product selection and implementation, based on cattle type and management goals. Quality scientific data, when presented to and understood by producers, results in the proper positioning of products within a management scheme. The end result is an elevation of cattle health and a contribution to overall profitability.

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Alharma Inc.
One Executive Drive
Fort Lee, NJ 07024 USA
1-888-897-8657

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