COCCIDIOSIS IN CHICKENS AND TURKEYS

Slide study set #7

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By: KEVIN L. WATKINS and KEN OPENGART

The coccidia are in the phylum Apicomplexa and may be grouped into numerous genera consisting of more than a thousand species; however, this discussion will be restricted to the genus Eimeria, which infects chickens and turkeys. These homogenous intracellular protozoans possess several characteristics that make disease control and eradication extremely difficult under commercial conditions. Coccidia are ubiquitous to commercial poultry production. Although nearly all commercial birds receive some form of preventative therapy, field cases of coccidiosis are still common. Several techniques can be used to diagnose coccidiosis. However, we will describe only those macro- and microscopic techniques routinely used during field evaluations.

The greatest impact of coccidiosis on the poultry industry is the cost of prophylaxis and the production losses associated with clinical disease. Coccidia are very easy to identify. However, the presence of the parasite does not confirm the presence of clinical disease or a reduction in growth performance. Therefore, the clinician must be able to differentiate between coccidiosis (true clinical disease) and coccidiasis (mild infections not associated with significant economic loss). Although mortality can occur during severe outbreaks, it is rare and most economic losses are attributed to a reduction in production efficiency.

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Etiology. Coccidiosis is unique in that it affects almost every animal species, yet individual species of coccidia are host specific. In other words, each species of Eimeria, for all practical purposes, infects only one species of poultry . In addition to species specificity, there is immunogenic specificity. Although coccidia tend to be very immunogenic, exposure to one species will not provide significant protection against other species. Although there are exceptions, the life cycle of c occidian associated with commercial poultry is basically confined to the enteric system of the host and the disease is transmitted by the fecal-oral pathway. There is no intermediate host and infections are self-limiting since in the absence of reinfection oocyst shedding will cease. Under the right conditions (lack of reinfection or because of the development of protective immunity) birds surviving a coccidial infection can recover very rapidly. Intestinal tissue will be repaired to the point that within a couple of weeks it may be difficult to identify even severely infected birds. A compensatory growth phase results from rapid rehydration and improved nutrient utilization. However, body weight and overall feed efficiency does not usually return to that o f uninfected contemporaries. Layers and breeders surviving coccidial infections usually return to normal production within a month.

Life Cycle. Coccidia usually complete their life cycle in 6 to 8 days in the chicken and turkey. The life cycle can be divided into three discrete stages (sporogony, schizogony or merogony, and gamogony. Sporogony is the only life phase to occur outside the host. Under ideal environmental conditions sporulation may occur within a day or two. Ideal conditions for coccidia spor ulation include a moderate temperature (ranging from 28 to 31 C), high litter moisture (approximately 50 to 75%), and an adequate supply of oxygen (not less than 10% below normal oxygen tension). If conditions are unfavorable, the unsporulated oocysts will sporulate at a slower rate or will lie dormant until favorable conditions exist. Oocysts are resistant to adverse environmental conditions and may remain viable for many months or even years. However, if oocysts are exposed to extreme conditions some or a ll of the oocysts may die. Oocysts appear to be most susceptible to desiccation by high dry heat. Environmental conditions will not only influence the rate of sporulation but will influence the percentage

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of the oocyst population that sporulates. Oocysts must be sporulated to be infective. Although the oocyst population can be dramatically reduced with good health, litter, and ventilation management, it is impractical and perhaps impossible to eliminate all parasites from a commercial poultry facility.

The asexual life phase (schizogony or merogony) results in an explosion in parasite numbers. Infections begin when a bird ingests viable sporulated oocysts. A sporulated oocyst contains four sporocysts, each containing two sporozoites (eight sporozoites per oocyst). Through mechanical and biochemical action of the gastrointestinal system, sporozoites will be released from the oocysts. Within minutes excystation occurs releasing the sporozoites into the intestinal lumen. Once free in the intestinal lumen a sporozoite will actively penetrate a host epithelial cell on the intestinal villi. The location within the intestine where invasion occurs will depend upon the infective species and can range from duodenum to cecum and rectum. The parasite then goes through a series of asexual generations to produce the final generation merozoites. The asexual phase of the life cycle results in the exponential increase in parasite numbers. The number of asexual generations ranges from two to four and varies by coccidial species. The final generation merozoites develop into a sexual stage becoming either microgametocytes (male) or macrogametocytes (female).

In the sexual life phase (gamogony), microgametes released from the microgametocyte will fertilize the macrogametes. The fertilized macrogamete will develop a protective cell wall becoming an oocyst. As the fertilized oocyst matures the epithelial cell membrane ruptures releasing the oocyst. The unsporulated oocyst enters the intestinal lumen to be shed in the host's fecal material. The time between the ingestion of a sporulated oocyst and the initial appearance of an unsporulated oocyst in the feces is known as the pre-patent period. The pre-patent period will average between four and eight days depending upon the infective species. Shedding may continue for many days to more than a week after the appearance of the first oocyst (patent period).

Physiological Effects. Many of the physiological manifestations of coccidia are related to the enteric nature of the disease. Direct effects on nutritional status are known to influence protein, vitamin, carbohydrate, lipid and mineral utilization. Indirect effects

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resulting from secondary infections or immune system stimulation are known to confound these interactions. As a result, coccidia-infected birds may exhibit anorexia, lethargy, reduced body temperature and huddling, nutrient deficiencies or toxicities, dehydration, depigmentation, anemia, loose droppings, bloody droppings, poor feed conversion, reduced growth rate, and decreased egg production. While coccidiosis alone does not usually cause clinical nutritional disorders, it often contributes to fat soluble vitamin deficiencies (rickets, encephalomalacia) and will exacerbate nutritional inadequacies (protein, vitamins, Ca, Mg, Se, and Zn) or excesses (Co and Cu). Although severe infections, particularly with *E. brunetti, E. maxima, E. necatrix*, and *E. tenella*, may cause death, an increase in mortality rate is not usually associated with field cases in the United States.

In addition to nutritional effects, interactions between coccidiosis and other diseases have been identified. The interactions include diseases of bacterial and viral origin and diseases attributed to feedstuff associated toxins. Coccidial infections will alter the intestinal environment (pH, transit time, osmotic characteristics, composition, viscosity, and microflora). One of the more evident interactions is the relationship between coccidiosis and necrotic enteritis. Coccidial infections have also been shown to influence Salmonella and *E. coli* shedding.

The interaction between coccidiosis and Marek's disease may have been one of the first parasitic-viral disease interactions described. Viral diseases such as Marek's and infectious bursal disease interfere with the development of immunity to coccidiosis, exacerbating the adverse effects of the disease and reducing the development of protective immunity. Interactions between coccidiosis and reovirus, and coccidiosis and reticuloendothelial virus have also been reported.

There have been several reports on the interactions between coccidiosis and mycotoxins (aflatoxin, DON, ochratoxin). Interactions between coccidiosis and other feedstuff associated toxins such as biogenic amines and tannins are suspect.

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IMPORTANT SPECIES

Chicken Species. There have been nine species of *Eimeria* identified in chickens. Two of these species are of questionable validity (*E. hagani* and *E. mivati*). Probably only six species (*E. acervulina, E. brunetti, E. maxima, E. mitis, E. necatrix,* and *E. tenella*) cause significant pathology in chickens and of these, only four (*E. acervulina, E. maxima, E. mitis* and *E. tenella*) are routinely identified in broiler chickens in the United States. While not common in broilers, *E. necatrix* and *E. brunetti* can be found in broiler breeders, broiler breeder replacements, and layers in the United States or in broilers in other parts of the world. Some consider *E. mitis* and *E. praecox* nonpathogenic although morbidity and reduced growth performance has been reported. The great majority of economic losses from coccidial infections in broilers in the United States are caused by *E. acervulina, E. maxima*, and *E. tenella*. Although, severe *E. maxima* and *E. tenella* infections can cause mortality, rarely if ever is mortality associated with *E. acervulina* infected birds. While considered less pathogenic than *E. maxima* and *E. tenella, E. acervulina*'s prevalence, reproductive potential, and adverse effects on production efficiency, may make it the single most economically impactive parasite in the chicken industry in some geographical areas.

Turkey Species. Of the seven species of *Eimeria* found in turkeys, four are considered to be significantly pathogenic. While *E. dispersa* is less pathogenic than *E. meleagrimitis*, *E. adenoeides*, and *E. gallopavonis*, it too can reduce growth performance. For the most part, coccidial infections in turkeys do not produce discrete lesions to the extent of those produced in chickens. In addition, the presence of non-pathogenic species (*E. innocua, E. melagridis*, and *E. subrotunda*) of turkey coccidia can make diagnosis and microscopic speciation difficult. *Eimeria adenoeides* and *E. meleagrimitis* appear to be the most common species found in commercial turkeys.

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Table 3 Currently approved anticoccidials

Compound	Class
Amprolium	Chemical
Amprolium + Ethopabate	Chemical
Amprolium + Ethopabate + Sulpha	Chemical
Clopidol	Chemical
Clopidol + Methyl Benzoate*	Chemical
Coccivac	Live Vaccine
Decoquinate	Chemical
Diclazuril*	Chemical
Halofuginone	Chemical
Immucox	Live Vaccine
Lasalocid	Ionophore
Maduaramicin	Ionophore
Monensin	Ionophore
Narasin	Ionophore
Narasin + Nicarbazin	Potentiated Ionophore
Nicarbazin	Chemical
Paracox	Live Vaccine
Robenidine	Chemical
Roxarsone	Chemical
Salinomycin	Ionophore
Semduramicin	Ionophore
Sulfonamides	Chemical
Zoalene	Chemical

*Not available in the United States as of 4/1/9

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REFERENCES

1. Chapman, H. D., 1996. Anticoccidial drug programs in the United States. Poultry Sci. 75 (Suppl. 1): 90.

2. Edgar, S. A., 1986. Coccidiosis in turkeys: Biology and incidence. In: Research in Avian Coccidiosis. Proceedings of the Georgia Coccidiosis Conf. Nov. 1985, University of GA, Athens.

3. Edgar, S. A., 1977. Field Diagnosis of Coccidiosis in Chickens. Alabama Agriculture Experiment Station, Auburn, AL.

4. Gregory, M. W., 1990. Pathology of coccidial infections. In: Coccidiosis of Man and Domestic Animals. Ed. P. L. Long, CRC Press, Boca Raton, FL.

5. Johnson, J., and W. M. Reid, 1970. Anticoccidial drugs: Lesion scoring techniques in battery and floor-pen experiments with chickens. Exp. Parasitology 28: 30-36.

6. Levine, N. D., 1973. Protozoan Parasites of Domestic Animals and Man. 2nd ed. Burgess Publishing Co., Minneapolis, MN.

7. Long, P. L. and B. J. Millard, 1977. Coccidiosis in turkeys: Evaluation of infection by the examination of turkey broiler litter for oocysts. Avian Pathol. 6: 227-233.

8. Long, P. L. and W. M. Reid, 1982. A guide for the diagnosis of coccidiosis in chickens. Research Report 404. The University of Georgia College of Agriculture Experiment Stations. Athens, GA.

9. Reid, W. M., 1989. Recommending sanitary practices for coccidiosis control. In: Coccidia and Intestinal Coccidiomorphs. Proceedings of the Vth International Coccidiosis Conference. October 1989, Tours, France.

10. Reid, W. M., and J. Johnson, 1970. Pathology of Eimeria in light and heavy coccidial infections. Avian Diseases 14: 166-171.

11. Rose, M. E., and P. L. Long, 1962. Immunity to four species of Eimeria in fowls. Immunology 5: 79-92.

12. Ruff, M. D., 1986. Reasons for inadequate nutrient utilization during avian coccidiosis: A review. In: Research in Avian Coccidiosis. Proceedings of the Georgia Coccidiosis Conf., Nov. 1985, University of GA, Athens.

13. Ruff, M. D., 1989. Interaction of avian coccidiosis with other diseases: A review. In: Coccidia and Intestinal Coccidiomorphs. Proceedings of the Vth International Coccidiosis Conference. October 1989, Tours, France.

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14. Ruff, M. D., and P. C. Allen, 1990. Pathophysiology. In: Coccidiosis of Man and Domestic Animals. ed. P. L. Long, CRC Press, Boca Raton, FL.

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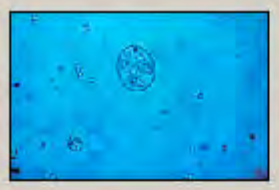
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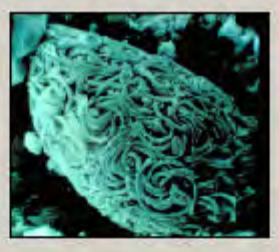
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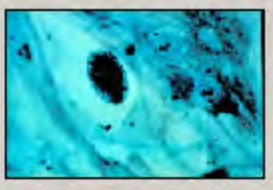
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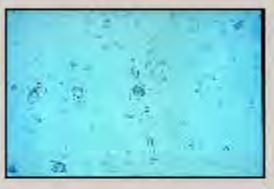
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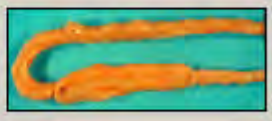
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