# COCCIDIOSIS IN CHICKENS AND TURKEYS

**Slide Study Set #7**

A Continuing Education Program Prepared by

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## Introduction

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 homoxenous 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.

## The Disease

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 coccidia 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 of 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 sporulation 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 all 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 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 generation 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 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.

## 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 is 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.

## Clinical Signs and Diagnosis

Poultry suffering from coccidial infections may exhibit many outward signs of the disease. Unfortunately, few, if any, of these signs can be considered pathognomonic for the disease. The infective species, the severity of the challenge or number of organisms ingested, and the stage of the infection (initial, acute, recovery) will influence the incidence, appearance and pathogenicity of the disease. Initial signs of coccidiosis usually include anorexia and lethargy. As the disease progresses signs such as huddling, ruffled feathers, and dehydration are observed. Loss of pigmentation may occur in birds consuming pigmented feeds. Mucoid, watery, or bloody diarrhea can be seen in the litter and (or) on soiled vent feathers. Bloody dropping are usually confined to birds infected with either *E. tenella* or *E. necatrix*. None of the turkey species are associated with bloody droppings. Except for commercial layers, even during severe coccidial infections, mortality is not usually significant. If present, mortality is usually seen only in chickens infected with *E. maxima, E. tenella, E. necatrix*, or *E. brunetti* or turkeys infected with *E. adenoeides* or *E. meleagrimitis*. Since individual birds respond differently to coccidial challenges, flocks exposed to a significant coccidial challenge may exhibit a great deal of variation in size. Such severe infections may ultimately result in reduced final weight and feed efficiency for the flock.

To confirm coccidial infections or identify infective species, an examination of the intestinal tract is required. It is best to select 5 to 10 birds per flock for necropsy. Do not select sick or cull birds since these birds may not be true indicators of overall flock health. Coccidiosis, as with many poultry diseases, must be diagnosed and treated on a flock basis. Since health programs are usually implemented on a complex basis (across many farms in a specific geographical area) it is best to examine birds of several ages. Coccidial challenge is usually most significant between 3 to 5 weeks of age. However, it is best to sample birds from 2 weeks to market to assure that, if the disease is present, it is running a normal course. A complete necropsy of each bird should be conducted to determine all factors that may influence the bird's health.

The most common method of quantifying coccidial infections in chickens was defined by Johnson and Reid (1970) where a number from 0 (no lesions) to 4 (most severe lesions) is given for each species. This lesion scoring system is used extensively to assess coccidial challenge in broilers. Because of the lack of discrete lesions, the presence of non-pathogenic species, and the value of turkeys, coccidial lesion scoring is not a common practice in commercial turkeys. In addition to lesion

scoring, there are other macro and microscopic assessments that can be utilized to adequately confirm, speciate, and quantify coccidial infections. Both the serosal and mucosal layers of the intestine from duodenum to rectum should be examined. Factors such as the incidence and severity of discrete coccidial lesions, location of lesions, microscopic confirmation of life cycle stages, general intestinal condition, intestinal thinning, thickening and ballooning, extent of mucosal sloughing, mortality and morbidity rate, and even the scent and the appearance of the intestinal contents can be used to diagnose and characterize the severity of coccidial infections. Tables 1 and 2 list differential criteria used to speciate chicken and turkey coccidia.

Some species of coccidia produce lesions that are more easily identified by examining the serosal surface of the intestine. For this reason the serosa should be examined prior to incising the intestinal wall. When the intestine is excised the contents should be examined for the presence of mucosal sloughing, blood, undigested feed, mucous, and watery contents. The intestinal contents are then removed and the intestinal tract is examined for the presence of the characteristic lesions produced by the different species of *Eimeria*. If an intestinal lesion is present, a smear may be prepared by scraping the infected area, placing the scraping on a slide, adding a drop of water to the sample, and covering with a cover slip. A microscopic examination of the slide (10 or 20x) will allow easy detection of oocysts or other life stages. The size, shape, and appearance of the oocyst assist in species identification (Table 1 and 2). Although confirmation of a coccidial infection can be made using microscopic visualization, the mere presence of the parasite is not enough to make a diagnosis of clinical coccidiosis. The detection of oocysts in the intestine of apparently normal birds is a common finding, as some degree of coccidial cycling normally occurs in each flock. Clinical coccidiosis should only be diagnosed if gross lesions are severe, production parameters are adversely affected, and oocysts are present under microscopic examination.

## Practical Field Evaluation and Control of Coccidiosis

On-farm posting sessions are one of the best ways to make a proper coccidial assessment. Such sessions allow one the opportunity to observe the flock as a whole and to evaluate farm conditions. These are critical factors in assessing coccidial challenge and anticoccidial management. Bird uniformity, degree of bird activity, appearance of droppings, litter conditions, house temperature, ventilation and air quality, water and feeder management, feed quality and availability, and overall quality of management should be evaluated. Many believe that well managed farms can tolerate a heavier coccidial challenge without a significant loss in flock performance. Data have demonstrated that under some conditions average lesion scores even exceeding 2.5 may have little or no effect on growth performance (Reid and Johnson, 1970).

One of the problems with assessing coccidial challenge and anticoccidial efficacy is the lack of well-defined and predictive evaluation methods. It is relatively easy to diagnose coccidial infections, however, it is extremely difficult to determine whether the severity of the infection is significant enough to influence long-term

performance. Interactions between coccidia, the environment, and numerous management factors further complicate our ability to assess coccidial challenge and to design, implement, and assess anticoccidial programs. The presence of coccidial lesions and oocysts often does no more than confirm the presence of the parasite.

There are more than 20 anticoccidial products available worldwide (Table 3). However, controlling coccidiosis is not as simple as having an arsenal of efficacious products. One must know how to use these products effectively and realize that drugs alone will not control this disease. Proper house management (litter, water, ventilation, temperature, biosecurity) and bird health (chick/poult quality, vaccination, nutrition, feed ingredient quality, medication) must compliment the anticoccidial program in order to provide good long-term control of coccidiosis. The vast majority of anticoccidial programs are ionophore based. Ionophores provide good broad spectrum anticoccidial efficacy and since they allow some parasite cycling, birds develop protective immunity during treatment. While chemicals tend to be very potent anticoccidials, many chemical products interfere with the development of protective immunity. Because of growth performance and uniformity issues, live vaccine programs are not commonly used in broilers and commercial turkeys but are used in replacement birds.

In broilers, anticoccidials are often shuttled and (or) rotated. Shuttling refers to the use of more than one anticoccidial during a specific growout, for example, using a chemical in the starter feed and an ionophore in the grower feed. Rotating refers to the sequential use of more than one anticoccidial over time, for example, using one anticoccidial program in the winter and another anticoccidial program in the spring. The most common shuttle program is a chemical starter with an ionophore grower and finisher. Ionophore ionophore shuttles are also used. A recent survey of the U.S. broiler industry found that 50% of anticoccidial programs were straight ionophore programs, 36% were chemical ionophore shuttle programs and 14% were ionophore ionophore shuttle programs (Chapman, 1996). Most of the U. S. broiler industry uses between two and four anticoccidial programs per year. Shuttling is not routinely practiced in commercial turkeys and a company will usually use only one or two anticoccidial programs per year.

While most chemotherapeutics are very effective at preventing coccidiosis, designing anticoccidial programs to maximizing long-term efficacy is often more an art than a science. Factors such as production objectives, environmental conditions, management practices, drug resistance, and economics must be considered for long-term programming. Each product has advantages and disadvantages and even products within the same therapeutic class exhibit subtle differences. In addition, the direct effect of the drug on the host often has more influence on overall bird performance than the effect of the drug on the parasite. Therefore, information regarding drug side effects and safety must also be considered.

With today's intense poultry production it is an unrealistic goal to raise commercial flocks coccidia-free. Although we may not be able to prevent or eradicate coccidia, we do need to control the disease. Since coccidiosis is a self-limiting disease, the severity of an infection depends upon the number of oocysts ingested by the host. Subclinical and even light clinical infections often go

undetected and may have little or no effect on production efficiency. In the early 1960's, N. D. Levine termed this disease state coccidiasis. Coccidiosis is a coccidial infection that produces morbidity and (or) mortality resulting in reduced bird performance. Coccidiasis can be defined as a coccidial infection producing an insignificant degree of morbidity having little or no effect on bird performance and no mortality. The intense demands of modern poultry production require that we prevent coccidiosis, but current technology probably dictates that we live with coccidiasis. Learning to minimize coccidial pathology while maximizing production efficiency should be our goal. There are essentially three approaches to controlling coccidiosis: environmental, immunological, and chemotherapeutic. Designing health programs that utilize all three approached should provide the best long-term control of the disease.

## Description of Slides

Slide 1. *Eimeria tenella* life cycle.

Slide 2. Sporulated oocyst showing 4 sporocysts with 2 sporozoites each.

Slide 3. Scanning electron micrograph of a sporozoite, the first invasive stage.

Slide 4. First generation schizont showing sickle-shaped first generation merozoites.

Slide 5. Macrogamete

Slide 6. Unsporulated oocysts

Slide 7. Segment of duodenum and anterior jejunum from uninfected chicken. Note healthy appearance of the mucosa.

Slide 8. Location of *E. acervulina* infections in the chicken. *E. acervulina* invades the upper intestinal tract and is characterized by white elongated lesions found on the mucosal surface. Most infections are limited to the duodenal loop but may extend to the yolk-sac diverticulum.

Slide 9. Light *E. acervulina* infection. Scattered, white plaque-like lesions containing developing oocysts are usually confined to the duodenum. Mucosal surface appears relatively normal. Lesions are elongated and transversely oriented on the intestinal walls. Lesions may be seen from either the serosal or mucosal surfaces.

Slide 10. Moderate *E. acervulina* infection. Lesions are much closer together and more numerous, but not coalescent. Lesions may extend below the duodenum. The intestinal wall shows no thickening. Digestive tract contents appear relatively normal.

Slide 11. Heavy *E. acervulina* infection. Lesions are numerous enough to coalesce, lesion size may be reduced, and give the intestine a coated appearance. The intestinal wall may be thickened or thinned and the intestinal contents watery. Lesions and (or) mucosal damage may extend as far posterior as the yolk sac diverticulum.

Slide 12. Severe *E. acervulina* infection. The mucosal wall is pale in color with lesions completely coalescent. Individual lesions are usually indistinguishable. Lesions may appear in the middle part of the intestine. The intestine may be thickened or very thin. The entire intestine is pale and is filled with a creamy or watery exudate.

Slide 13. Location of *E. maxima* infections in the chicken. *E. maxima* typically invades the middle section of the intestine but will migrate throughout the intestine. Lesions appear as small discrete red hemorrhages on the serosal surface of the intestine and orange mucus is often present within the lumen. Since numerous factors can cause intestinal hemorrhaging and mucus accumulation, a microscopic examination is recommended to confirm the presence of oocysts. *E. maxima* is thought to be the most immunogenic of the chicken species (Rose and Long, 1962).

Slide 14. Light *E. maxima* infection. Discrete red petechiae (pin-point hemorrhages) may appear on the serosal side of the intestine at any location. There is little or no ballooning or thickening of the intestine.

Slide 15. Moderate *E. maxima* infection. Serosal surface may be speckled with numerous red petechiae. The intestine may contain orange mucus. The intestinal wall may be thickened but little or no ballooning is evident.

Slide 16. Heavy *E. maxima* infection. Intestinal wall exhibits ballooning and may be thickened. The mucosal surface is roughened. Intestinal contents may contain significant amounts of orange or brown mucus and some specks of blood.

Slide 17. Severe *E. maxima* infection. The intestinal wall may be severely ballooned in one or more areas and the intestinal contents may contain large quantities of mucus, specks of blood and digested red blood cells giving a reddish brown color and putrid odor. The intestinal wall can be greatly thickened or very thinned.

Slide 18. Location of *E. tenella* infection in the chicken. This species is confined to the ceca. Although lesions associated with *E. tenella* can be quite severe, since the ceca do not play a major role in nutrient absorption, growth performance is minimized. Lesions can be seen on the serosa of the ceca and the pouches often contain blood. Observations may be associated with one or both ceca.

Slide 19. Light *E. tenella* infection. A few scattered petechiae are found on the cecal wall. There is no thickening of the cecal walls or intraluminal blood present. Cecal contents appear normal.

Slide 20. Moderate *E. tenella* infection. Petechiae are more numerous and the cecal wall is somewhat thickened. Cecal contents are of normal consistency but may contain streaks of blood.

Slide 21. Heavy *E. tenella* infection. Cecal walls are greatly thickened with scattered petechiae. The ceca may contain scant fecal contents; blood and white caseous material may be present.

Slide 22. Severe *E. tenella* infection. The cecal wall is distended with blood and (or) caseous cores. Normal fecal debris is lacking or included in cores.

Slide 23. Location of *E. brunetti* infection in chickens.

Slide 24. Moderate *E. brunetti* infection. Infections are characterized by red pin point lesions of the lower intestine extending from the mid small intestine into the large intestine. The petechiae become more numerous in severe infections and may be found throughout the intestine. The intestinal contents may be watery, slightly mucoid or empty. The mucosal surface in the lower intestine often takes on a pink to red appearance. This species can have significant adverse effects on performance; however, it is not commonly found in broilers.

Slide 25. Location of *E. necatrix* infection in chickens.

Slide 26. Severe *E. necatrix* infection. Infections are usually midintestinal and characterized by hemorrhage and ballooning. Classic "salt and pepper" lesions which are a result of both petechia and white plaques (large 2nd generation schizonts) are observed. Birds with light infections may only exhibit petechiae. Oocysts only develop in the cecal pouches and will not be found in other areas of the small intestine. This species is rarely found in broilers.

Slide 27. Intestinal tract from uninfected turkey. Note appearance of mucosal surface.

Slide 28. Location of *E. meleagrimitis* infection in turkeys.

Slide 29. *E. meleagrimitis* infection. Note the lack of discrete lesions. The entire intestine may exhibit loss of pigmentation, spotty congestion, dilatation, and casts.

Slide 30. Location of *E. gallopavonis* infection in turkeys.

Slide 31. *E. gallopavonis* infection. The lower intestine is characterized by the presence of soft white to pink caseous material with ulcerations of the mucosa. Infections are confined to the posterior small intestine.

Slide 32. Location of *E. adenoeides* infection in turkeys.

Slide 33. *E. adenoeides* infection. The ceca (both mucosal and serosal surfaces) may appear white in color. Loose or solid white caseous cores in the ceca usually contain large numbers of oocysts.

Slide 34. Location of *E. dispersa* infection in turkeys.

Slide 35. *E. dispersa* infection. Note the lack of discrete lesions. The mucosal surface is usually roughened and sometimes cream colored. The condition can be found throughout the small intestine. This species is mildly pathogenic.

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**Table 1. Differential Identification of Selected Chicken Coccidia[1]**

| Species | Oocyst size (L / W in μ) | Oocyst shape/index | Prepatent period (h) | Primary location of invasion | Pathogenicity | Lesion appearance |
|---|---|---|---|---|---|---|
| *E. acervulina* | 17.7-20.2<br>13.7-16.3 | Ovoid<br>1.25 | 97 | Duodenum to yolk- sac | Moderate | White rounded or transversely oriented ladder-like lesions. |
| *E. brunetti* | 20.7-30.3<br>18.1-24.2 | Ovoid<br>1.31 | 120 | From yolk-sac to rectum, but may migrate throughout small intestine | High | Pin-point hemorrhages on serosal surface. |
| *E. hagani* [2] | 15.8-20.9<br>14.3-19.5 | Broadly ovoid<br>1.08 | 99 | Anterior one-third of small intestine | Low-None | Lesions not adequately described. |
| *E. maxima* | 21.5-42.5<br>16.5-29.8 | Ovoid<br>1.47 | 121 | Middle intestine but may migrate throughout small intestine | High | Pin-point hemorrhages on serosal surface. Intestinal ballooning with orange mucoid contents. |
| *E. mitis* | 11.7-18.7<br>11.0-18.0 | Subspherical<br>1.09 | 93 | From yolk-sac to rectum | Low | There are usually no discrete lesions found. |
| *E. mivati* [2] | 11.1-19.9<br>10.5-16.2 | Ellipsoid<br>1.16 | 93 | Anterior half of the small intestine but may migrate throughout | Low | Lesion descriptions have been inconsistent. |
| *E. necatrix* | 13.2-22.7<br>11.3-18.3 | Oblong ovoid<br>1.19 | 138 | Middle small intestine with oocysts developing in ceca | High | Pin-point red (hemorrhage) and (or) white (2nd generation schizonts) lesions on serosal surface. Blood and mucoid exudate may be present. |
| *E. praecox* | 19.8-24.7<br>15.7-19.8 | Ovoid<br>1.24 | 85 | Anterior one-third of small intestine | Low-None | There are usually no discrete lesions found. Some mucoid enteritis. |
| *E. tenella* | 19.5-26.0<br>16.5-22.8 | Ovoid<br>1.16 | 115 | Cecal pouches (some reports in rectum) | High | Pin-point red (hemorrhage) and (or) white (2nd generation schizonts) lesions on serosal surface of ceca. Cecal may be filled with blood. |

[1]Information compiled from Johnson and Reid (1970), Levine (1973), Long and Reid (1982), Edgar (1987) and personal observations.

[2]Species of doubtful validity.

**Table 2. Differential Identification of Selected Turkey Coccidia**[1]

| Species | Oocyst size (L / W in μ) | Oocyst shape/index | Prepatent period (h) | Primary location of invasion | Pathogenicity | Lesion appearance |
|---|---|---|---|---|---|---|
| *E. adenoeides* | 18.9-31.3<br>12.6-20.9 | Ellipsoidal<br>1.54 | 103 | Ceca and rectum | High | Watery to solid white caseous cecal contents. |
| *E. dispersa* | 21.8-31.3<br>17.7-23.9 | Broadly ovoid<br>1.24 | 120 | Entire small intestine | Low | No discrete lesions found. Serosal surface may appear cream-colored. |
| *E. gallopavonis* | 22.7-32.7<br>15.2-19.4 | Ellipsoidal<br>1.52 | 105 | From yolk-sac to rectum | High | White or pink ulcerations of ileal and (or) rectal mucosa. White caseous material may be found in lower intestine and (or) ceca. |
| *E. innocua* | 18.6-25.9<br>17.3-24.5 | Subspherical<br>1.07 | 114 | Anterior half of small intestine | None | None described. |
| *E. meleagridis* | 20.3-30.8<br>15.4-20.6 | Ellipsoidal<br>1.34 | 110 | From yolk-sac to rectum and maybe ceca | None | Cream-colored serosal surface. Some reports of petechial hemorrhages in posterior small intestine. |
| *E. meleagrimitis* | 15.8-26.9<br>13.1-21.9 | Ovoid<br>1.17 | 103 | Middle intestine but may migrate throughout small intestine | High | Thickened jejunum containing colorless to pink fluid. The remainder of the small intestine may be congested with petechial hemorrhages on mucosal surface. |
| *E. subrotunda* | 16.5-26.4<br>14.2-24.4 | Subspherical<br>1.10 | 95 | Anterior half of small intestine | None | None described. |

[1]Information compiled from Levine (1973), Edgar (1985) and personal observations.

**Table 3. Currenlty approved anticoccidials**

| Compound | Class |
|---|---|
| Amprolium | Chemical |
| Amprolium + Ethopabate | Chemical |
| Amprolium + Ethopabate + Sulpha | Chemical |
| Clopidol | Chemcial |
| 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 | Chemcial |
| Zoalene | Chemical |

*Not available in the United States as of 4/1/97.