Eimeria spp
Common Coccidians of Hoof stock and Poultry
Many Eimeria spp. with high host specificity

A. Morphology
   • Oocyst
     o Species-specific size, shape, outer coat, presence or absence of polar cap, etc.
     o Single-cell embryo when passed
     o Sporulated oocyst contains 4 sporocysts with 2 sporozoites each = 8 sporozoites total
   • Intracellular parasites of enterocytes.

B. Life Cycle
   1. Transmission
      a. Direct life cycle – fecal-oral, ingestion of oocyst
   2. Invasion
      a. Sporozoites excyst from oocyst and invade enterocyte
   3. Asexual reproduction
      a. Merogony (schizogony) [multi-nuclear division followed by cytoplasmic division]
      b. Merozoites exit the enterocyte and infect other enterocytes and goes through merogony again.
      c. Number of asexual cycles and number of merozoites per merogony is species-specific.
   4. Sexual reproduction
      a. Final generation of merozoites exit the enterocyte, infect other enterocytes, and go through
gametogony (production of gametes)
      b. Macrogamete (egg)
         i. Some final merozoites remain a single cell and become a macrogamete (egg) within a
            macrogamont.
      c. Microgametes (sperm)
         i. Other final merozoites go through multi-nuclear division, cytoplasmic division, and develop
            2 flagella (bi-flagellate); thus forming a microgamont
         ii. Exflagellation – when microgametes exit the microgamont in search of a macrogamete.
      d. Fertilization – a microgamete fuses with a macrogamete forming a zygote
      e. A cyst wall forms around the zygote and the immature oocyst exits the macrogamont into the
         lumen of the host’s gut and is passed in the feces.
   5. Dissemination
      a. Oocysts (unsporulated) exit the host in the feces and contaminate the environment.
   6. Sporogony (= Sporulation)
      b. Sporogony occurs in the environment.
         i. Appropriate temperature, moisture, and oxygen are required for sporogony.
         ii. Some species can take as little as 1 day to sporulate in optimal conditions
      c. After sporulation, the oocyst is ready for transmission to the next host.

C. Pathogenesis
   1. Exponential destruction of enterocytes with each merogonic cycle, thus causing malabsorption,
destruction of epithelial lining and hemorrhagic ulcers.
   2. Traumatic permeability, with loss of fluids and blood in to the gut lumen
   3. Hypersecretion due to immune response.
   4. Pathogenicity depends upon dose, host health status, immunological competence
D. Clinical Disease  
1. Complaint -- Mild to severe diarrhea (bloody, mucoid, or watery)
   a. most often reported in young or naïve animals.
2. Range of Pathology  
   a. Hemorrhagic diarrhea / dysentery, tenesmus, fever, anemia, weakness, weight loss, death.
   b. Location of pathologic lesions is coccidian-species specific.
3. Manifestation of Coccidiosis varies  
   a. Individual animal  
      i. Non-clinical, but large numbers of oocysts in feces  
      ii. Acute, severe, fatal, bloody diarrhea, but no oocyst in feces – prior to prepatency  
      iii. Disease caused by 1) An overwhelming dose of oocyst OR 2) a moderate dose + stress  
   b. Herd or flock  
      i. Regularly recurring diarrhea issues with each successive cohort of young animals.

E. Diagnosis  
1. Clinical Signs  
2. Fecal Float Centrifugation or McMaster Slide  
3. Diarrhea may occur prior to oocyst excretion  

F. Treatment  
1. Ionophores (Monensin, Lasalocid, etc.) although often used, are not effective for the treatment of acute disease.
2. Treatment is mainly to eliminate incoming coccidial organisms; not to eliminate the ones already causing pathology. i.e. treatment does not stop occurring pathology.
3. Give supportive fluid-therapy for symptoms  
4. Treatment difficult as feed & water consumption is depressed.
5. Treat prophylactically for control – medicated feed or water with coccidiostats.

G. Control  
1. Sanitation, especially for young and naïve animals  
   a. Keep susceptible animals out of moist area, where oocysts will sporulate.  
   b. Hutch system for dairy calves  
   c. Direct sunlight and dryness best disinfectants
2. Good nutrition important
3. Coccidiostats (won’t cure an infected animal, rather the goal is to limit infection of newly exposed animals so they don’t get sick but develop natural immunity)  
   a. Coccidiostats act to limit the number of successful coccidial organisms, especially in young hosts.  
      i. Kills or inhibits growth of most entering organisms, but not all.  
         1. Allows for the development of immunity without disease. A “natural vaccine”.  
   b. Extremely important in systems of intense and / or confinement rearing of poultry, ruminants.  
   c. Prophylaxis -- Decoquinate (Deccox), Monensin (Rumensin), Lasalocid (Bovatec)  
   d. Treatment -- Amprolium (Corid), “Sulfia Drugs”: Sulfadimethoxine, Sulfamethazine, Sulfamethoxine  
   e. Concern for the development of resistance – rotate coccidiostats.  
   f. **WARNING -- IONOPHORES (MONENSIN, LASALOCID, ETC.) ARE HIGLY TOXIC TO HORSES**
4. Requires a coordinated control strategy (must have all these components)  
   a. Coccidiostats  
   b. Sanitation  
   c. Good Nutrition  
   d. Low Stress  
   e. Don’t mix age groups  
      i. Adults source of environment al contamination and source of infection for young animals.  
   f. At first sign of disease  
      i. Separate sick animals for supportive care  
      ii. Begin treatment of whole herd / flock.
5. Vaccines – used in Poultry coccidiosis  
   a. Oocyst cocktails, irradiated, mutated – ex. Inovocox vaccine
H. Epidemiology
   a. Ubiquitous
   b. Very, very host specific (thus no cross-species infection or zoonosis)
   c. Each host species may have many *Eimeria* species, but few are pathogenic.

2. Host risk factors
   a. Immunodeficient: young, stressed, poor nutrition
   b. Immunologically naïve
   c. Immunity is coccidian-species specific.
      i. *Eimeria bovis* infection does not confer protection against *Eimeria zurneii*
   d. Immunological experience provides incomplete or complete protection.
      i. Incomplete = Reinfestation usually leads to asymptomatic shedding of oocyst.

3. Environmental risk factors
   a. Primary infective dose – pathology proportional to infecting dose.
   b. Moist, cool habitats promote sporulation of oocysts
      i. Spring, Fall higher risks
   c. Crowded conditions
      i. Can quickly become highly contaminated with oocysts
      1. Pathogenesis proportional to infecting dose.
      ii. Stresses hosts thus decrease immune-competence

4. Consider Immunity and Environmental factors: Which is more likely to have a serious coccidian outbreak? Confinement chickens or Free-range chickens.

I. Host & pathogenic *Eimeria* species.
1. Bovine -- *Eimeria bovis, Eimeria zurneii* (differentiated by oocyst size)
   a. Once oocysts appear in feces it is too late
   b. Supportive therapy against dehydration is most important
2. Sheep -- *Eimeria ovinoidalis*
3. Goats -- *E. ninakohlyakimovae, Eimeria arloingi*
4. Swine -- 8 *Eimeria spp.* but low pathogenicity
5. Horse -- *Eimeria leuckarti* -- non-pathogenic
6. Poultry
   a. Massive destruction of epithelial cells - hemorrhage, malabsorption.
      i. often prior to patency
      ii. young birds at greatest risk
   b. Sanitation & prophylaxis with coccidiostats
      i. resistance a problem, rotate through a variety of coccidiostats
   d. Turkeys -- *E. adenoides, E. meleagrimitis*
   e. Clinical signs: bloody feces, pale combs, ruffle feathers, low appetite, high mortality, necropsy shows coagulated blood in ceca