**Sarcocystis cruzi**  
Canine coccidian of bovine deaths

A. Morphology  
- Sporocyst  
  - Thin-walled oocyst sporulates and ruptures before exiting in the feces, thus sporocysts are seen in the feces  
  - small, oval, smooth coat, no polar cap; 4 sporozoites

B. Life Cycle Rather Complex  
1. Cattle infects Dog  
   a. Obligatory Indirect Life Cycle (heteroxenous) --- Definitive host – Dog (other canids)  
   b. Transmission -- carnivorism, dog ingests sarcocyst in cattle muscle  
   c. Invasion -- Bradyzoites from sarcocyst from muscle invades intestinal cells  
   d. (Asexual reproduction – none occurs in the dog.)  
   e. Sexual reproduction (only occurs in Dogs)  
      i. Zoites go through gametogony (production of gametes)  
      ii. Macrogamete (egg)  
         1. Some final zoites remain a single cell and become a macrogamete (egg), within a macrogamont.  
      iii. Microgametes (sperm)  
         1. Other final zoites go through multi-nuclear division, cytoplasmic division, and develop 2 flagella (bi-flagellate); thus forming a microgamont  
         2. Exflagellation – when microgametes exit the microgamont in search of a macrogamete.  
   iv. Fertilization – a microgamete fuses with a macrogamete forming a zygote  
   v. A cyst wall forms around the zygote and the immature oocyst exits the macrogamont into the lumen of the host’s gut  
   vi. The oocyst sporulates within the gut lumen, then ruptures, releasing its 2 sporocysts into the gut lumen.  
   f. Dissemination  
      i. Sporocysts exit the host in the feces and contaminate the environment.  
         1. Dogs are the only hosts to pass sporocysts.  
         2. Sporocysts are infectious when shed, are very resistant and remain infectious for several months if kept cool and moist.

2. Dog to Cattle  
   a. Intermediate host – Cattle  
   b. Transmission  
      i. Cattle ingests a sporocyst from the feces of a dog (the definitive host)  
   c. Invasion -- Sporozoites enter vascular endothelial cells and eventually muscle cells  
   d. Asexual reproduction – vascular endothelial cells and muscle cells  
      i. Zoites go through a few cycles of schizogony, then disperse throughout the body to muscle cells.  
         1. The destruction of host cells during the tachyzoite phase causes the acute/severe disease with immune reaction and inflammation.  
      ii. Bradyzoites go through schizogony  
         1. Sarcocysts (muscle cysts), full of bradyzoites develop within muscle cells and remain viable for the life of the intermediate host.

C. Pathogenesis  
1. Intestinal Phase in Canids – no pathology  
2. Systemic Disease (Endothelial and Muscle Phase) in cattle – Zoites cause destruction of host cells, also acute immune response.  
3. Multisystemic dz due to zoites throughout the vascular endothelium and muscles, w/ immune reaction and inflammation
D. Clinical Disease
1. Intestinal Sarcocystosis – no pathology in the dog
2. Immune status of the host and the dose of sporocysts important factors of development of clinical dz
3. Systemic Sarcocystosis -- cattle only (4-6 weeks post ingestion of sporocyst)
   i. Protracted fever, lymphadenopathy, anorexia, cachexia, muscle spasms, myositis, hyper-excitability, diarrhea, hyper-salivation, weakness, hair loss around eyes, neck and tail switch,
   ii. Pregnant cows – abortions, still births
   iii. Acute disease, more severe in calves
   iv. Condemnation of carcass at inspection due to Sarcocysts

E. Diagnosis
1. Dogs – sporocysts found on a routine fecal exam
2. Cattle – serology, necropsy

F. Treatment
1. Dogs
   a. No drugs available to kill tissue forms.
2. Cattle
   a. Amprolium may provide some prophylactic protection.
   b. Treatment against sarcocysts is ineffective

G. Epidemiology and Control
1. Distributed worldwide
2. Control wild and domestic canid populations
3. don’t let dogs have access to raw meat, offal or dead animals

H. Zoonosis  --- Is not zoonotic

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Sarcocystis neurona
Opossum coccidian of horse neuropathy (Equine Protozoal Myeloencephalitis –EPM)

A. Morphology
   • Sporocyst (not oocyst in opossum feces)
     o Thin walled oocyst sporulates and rupture before exiting in the feces, thus sporocysts are seen
       in the feces
     o small, oval, smooth coat, no polar cap; 4 sporozoites

B. Life Cycle
1. Sylvatic Life Cycle
   a. Obligatory Indirect Life Cycle (heteroxenous)
      i. Opossum -- definitive host; sexual / intestinal stages, passes sporocysts
      ii. Other small mammals and birds – intermediate host (IH), asexual / muscle stages,
         sarcocysts in muscles
      iii. IH ingests sporocysts form opossum feces and opossum ingests sarcocyst in bird muscle.
2. Accidental Host  -- Aberrant, dead-end host
   a. Horse
      i. ingests sporocyst from opossum feces (contaminated food or water)
      ii. Asexual stages / systemic stages
      iii. Organism disseminates throughout body., especially to neural tissue, and muscle tissue

C. Pathogenesis
1. Systemic Disease but with a predilection for neural tissue (neurons and leukocytes of the brain and
   spinal cord) in horses – Zoites cause destruction of host cells, and acute immune response /
   inflammation.
D. Clinical Disease
   1. Equine Protozoal Myeloencephalitis (EPM) - Know the 3 A’s (Asymmetry, Ataxia and Atrophy)
      a. Asymmetry = a symptom that is worse on one side of the body than the other side (usually used related to muscle development)
      b. Ataxia = the inability to coordinate movement; often see legs crossing over
      c. Atrophy = muscle deterioration; will see this with asymmetry where one side of the horse has muscle atrophy and the other side is normal
      d. Other neurological signs include: gait abnormalities, myopathy, demarcated spontaneous sweating, loss of reflexes, cutaneous hyper-sensation, seizures, visual defects, behavioral changes, depression, paralysis, dysphagia.
      e. Without treatment, may progress to recumbency and death

E. Diagnosis
   1. Horses
      a. Observation of Clinical Signs, mostly neurologic and muscle atrophy.
         i. Unfortunately there is a broad spectrum of disease agents that induce similar clinical signs
         ii. Horses are not painful and rarely febrile from this infection
      b. Serology
         i. Serum IgG against S. neurona indicates exposure; may also give a false-positive due to cross-reactivity to another non-pathogenic Sarcocystis species
            1. Many horses exposed to S. neurona do not develop EPM
            2. Seropositive results in non-neurological horse has low positive predictive value; thus do not use serology to screen healthy horses
         ii. Negative serology has a high negative predictive value (helps you rule out EPM)
         iii. Seropositive + neurological signs strongly supportive of EPM. Don’t use serology to diagnose EPM in horses only with gait abnormalities.
         iv. Paired serology testing with CSF and serum with a end-point titer CSF:serum ratio < 100 indicated intrathecal antibody presence predictive of active infection
         v. Western Blotting and ELISAs use S. neurona specific antigens (less cross-reactivity)
            1. snSAG2 and snSAG3/4 (surface antigen) ELISAs gold standard
            vi. IFA uses whole cell antigen (more cross-reactivity with other Sarcocystis spp.)
            vii. Parasite antigen or antibody in spinal fluid is diagnostic.
      c. PCR detects S. neurona DNA (= active infection) but not reliable (high false negative).
      d. Post-mortem demonstration of organism in CNS lesions.

F. Treatment
   1. Horses
      a. Ponazuril, diclazuril, pyrimethamine, sulfadiazine (all very expensive in horse to treat for 2 months).
      b. Treat until clinical signs resolve, usually a long treatment period (1-2 mo). Be vigilant of side-effects.
      c. Improvement in 60-70%, complete recovery in up to 20%, relapse in 20%.

G. Epidemiology
   a. no breed predilections
   b. confined to the Americas (range of opossum distribution)
   c. Estimated 60-90% of horses have been exposed to S. neurona but <1% develop EPM

H. Control
   1. Prevent access of opossums to horse-feeding / watering areas
   2. Prevent access of opossums to stored horse feed
   3. Fallen fruit should be removed from horse pastures. Why?

I. Zoonosis --- Is not zoonotic

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