Anaplasmosis is an infectious Rickettsial disease of cattle caused by *Anaplasma marginale*. The organisms infects the red blood cells of cattle and produces a round structure that can be seen at the periphery of infected erythrocytes. The organism can be transmitted by ticks or by blood transfer between animals. All ages of animals can be infected, but the severity of clinical signs increase with age with animals over 3 years of age showing more severe clinical signs.

**Phases of disease**

Anaplasmosis goes through 4 distinct phases in the cow including incubation, developmental, convalescent, and carrier. Once infected, animals will incubate the organism without showing clinical signs for approximately 4-8 weeks. Once approximately 1% of the animal’s erythrocytes are infected the animal enters the developmental stage and will begin to show early clinical signs of infection. Early signs are often indistinct. Fever, weakness, and anorexia, along with a drop in production often predominate at this stage.

Approximately 15% of the erythrocytes must be parasitized to show clinical signs associated with anemia. Anemia occurs in cases of anaplasmosis due to the removal of infected erythrocytes by the immune system. The majority of the clinical signs in the cow can be attributed to anemia caused by the extravascular hemolysis. These signs include pale mucus membranes, tachypnea, and tachycardia. As an extravascular hemolysis hemoglobinuria and hemoglobinemia are absent in cases of bovine anaplasmosis. As the disease progresses and animals begin to process heme pigments pale membranes may become jaundiced. Mania, likely induced by cerebral hypoxia is also commonly associated with anaplasmosis in cattle. If left untreated, death may result.

The animal enters the convalescent stage once regenerative changes appear in the blood. And continues until the blood smear appears normal. It is still possible to have animals showing severe clinical signs early in the convalescent stage. In addition some animals may have a phase of chronic unthriftyness as they recover from a clinical anaplasmosis episode.

The carrier stage begins once organisms can no longer be found in the peripheral blood. This phase likely extends for the life of the animal. Carrier animals may be clinically normal with low levels of parasitemia which precludes diagnosis via blood smear. These animals remain infectious to their herdmates and are a lifelong source of the organism.

**Diagnosis**

Diagnosis of anaplasmosis can be made using several different testing methodologies. It is important to recognize that no one test will detect all of the potential stages of disease.

Blood Smears have utility in animals during the developmental phase of the infection. Animals with clinical signs consistent with anaplasmosis that have evidence of organisms on a blood smear are considered to be clinically affected. Early in disease (incubation) smears may be negative because a critical load of erythrocytes may not have been reached allowing them to be detected on blood smear. Similarly animals in the carrier state will typically not have enough infected erythrocytes to be detectable by this method. During the convalescent phase organisms may be detectable early. Signs consistent with regenerative anemia (anisocytosis and basophilic stippling) will be present during this phase and may be suggestive of anaplasmosis, but are not considered diagnostic.

Serologic tests (cELISA) will be positive in animals during the developmental and convalescent stages of infection. It will also be positive in many animals in the carrier state of disease. As such these tests will determine exposure of the animal to the organism, but alone it can not adequately separate active infections from carrier animals. A positive serologic test in the presence of clinical signs is suggestive of active infection. The major limitations to the cELISA are detection of early infection prior to the animal mounting an adequate immune response, prolonged recognition of the immune response in animals that have been chemosterilized, and cross reactivity with other Anaplasma species.

Polymerase chain reaction based diagnostics have the ability to pick up very small amounts of Rickettsial DNA and have proven helpful in identifying carrier animals. Some assays have been reported to detect as few as 30 infected cells per milliliter of blood. These assays lack formal validation and results may vary depending on laboratory protocol.

**Treatment**

Treatment is centered on antibiotic therapy and supportive care. Many treatment protocols have been published, but most center on oxytetracyline therapy. Most animals are suffering from significant anemia by the time they are presented for treatment. Minimizing stress and exertion are important to prevent decompensation from the anemia. Oxytetracycline 200 mg/ml can be administered at 9 mg/lb SQ every 72 hours. Blood transfusion may be indicated in some patients with severe life threatening anemia.
When an animal is diagnosed or in the case of a herd outbreak do not forget that other animals are at risk. At risk animals can also be treated with injectable oxytetracycline, oral chlortetracycline at a rate of 0.5mg/lb BW, or combinations of both injectable and oral oxytetracycline.

Prevention and control
Vector control – Tick and fly control is imperative in preventing movement of anaplasmosis through a herd. Insecticide impregnated ear tags, fly blocks, sprays, back scratchers, etc can be used to decrease the potential for vector transmission. It is important that these methods be reapplied, replaced, or recharged intermittently throughout the vector season for them to remain effective.

Medicated feed and mineral supplements, pulse dosing antibiotics – these methods can be used to prevent clinical disease in endemic herds. It is important to recognize that animals must eat the feed or use the mineral for them to be effective.

Vaccine – In some states a killed vaccine is available for use. It is currently sold as an experimental vaccine and is not licensed by the USDA. However, the USDA has approved the sale and use of the vaccine in multiple states including Missouri, Kansas, and Iowa.

Eliminate carriers – many treatment regimens to eliminate carriers have been suggested. Most of these have failed to hold up in recent evaluations using more stringent test methodology. Several new protocols have shown promise, but have yet to stand up to rigorous reevaluation. Chlortetracycline fed at 4.4; 11; and 22mg/kg/day for 80 days were all effective at eliminating the carrier state in tested animals. An additional study reported success treating with a single SQ injection of oxytetracycline followed by 30 days of chlortetracycline fed at 4.4 mg/kg/day. An additional study showed failure of the 4.4 mg/kg/day chlortetracycline to chemosterilize animals when fed for only 45 days.

Instruments – Blood contaminated instruments including needles, dehorners, tattooing equipment, ear taggers, and surgical equipment are effective at moving anaplasmosis from infected to naïve animals.

Animal movement – It is important to recognize that in some areas of the country and within some herds anaplasmosis is endemic. Care should be taken when moving animals from endemic areas to non-endemic herds. Introduction of a carrier animal to a naïve herd may result in significant numbers of clinically ill cattle since the herd has no background immunity. Conversely, moving a naïve animal to an endemic herd may result in clinical disease in the new introduction.

Recommended reading and resources
anaplasmosisvaccine.com
Reinbold, J. B., J. Coetzee, and R. Ganta, 2009a: Comparison of three tetracycline antibiotic treatment regimens for carrier clearance of persistent Anaplasma marginale infection derived under field conditions. Proceedings of the 42nd Annual Conference of the American Association of Bovine Practitioners (AABP), Omaha, NE