Coccidiosis is a disease of the large and small intestines caused by protozoa. The coccidia are very host specific parasites; cattle coccidia affect only cattle, and coccidia from goats, sheep, dogs or cats will not cause disease in cattle. The major damage that occurs from this organism is the damage to the gut cells as the parasite reproduces. Multiplication occurs so rapidly, that the final release of oocysts (eggs) into the manure represents over 23 million times the number of eggs the animal originally swallowed. The eggs are resistant to environmental stress and thus are difficult to completely remove from the environment. They are frequent contaminants of feed and water, and once ingested by other cattle, the life cycle starts again in a new host.

The life cycle of coccidia is complicated. After being passed in the manure, and exposed to the environment for 2-3 days, each oocyst will develop 8 sporozoites. Once ingested by a susceptible victim, the oocyst ruptures releasing the sporozoites. These then penetrate the lining cells of the small intestines and begin a period of multiplication that destroys intestinal cells as asexual reproduction occurs. This multiplication results in the release of millions of merozoites to invade the large intestine. It is there that the final stage of sexual reproduction occurs where male and female parasites unite to produce the oocyst and the fertile “egg” appears (microscopic) in the manure. The clinical signs of coccidiosis generally occur because of this last large intestinal stage, resulting in bloody diarrhea, straining and the passage of clumps of necrotic intestinal lining. However, because damage can also occur in the small intestine if sufficient intestinal lining cells are destroyed, diarrhea, unthriftiness and poor doing cattle can be a result even before oocysts are found in the stool.

It is estimated that as few as 50,000 oocysts ingested by a young susceptible calf can result in severe disease. While this sounds like a ridiculously large number, bear in mind that these oocysts are microscopic and cannot be seen by the naked eye. Fifty thousand would occupy only a tiny amount of space. Furthermore, the eggs are very resistant to environmental conditions such as heat and cold, and thus remain infective for long periods. Contamination of feed or water can increase the likelihood of infection. The severity of the disease is directly related to the number of oocysts ingested. In light infections, the damage to the gut is minimal, and because the intestinal epithelial cells replace themselves at such a rapid rate, any damage is quickly repaired. However, in the case of heavy infections, about 2 weeks after the eggs are consumed, most of the cells at the base of the intestinal glands are filled with merozoites (asexually reproduced life stages) and gametocytes (life stage with both male and female counterparts in the large intestine that finally result in the production of the oocyst). When all these cells die and rupture because of their infestation, damage is severe, there is loss of blood, fluid, electrolytes and proteins. Dead intestinal cells are sloughed and also passed out in the diarrhea.

Immunologically naïve animals consume oocysts from manure contaminated pasture, feed, water or bedding and also by grooming contaminated hair coats of their pasture mates. Most animals infected with coccidia do not show signs of disease. This is due to a normally low dose exposure and subsequent immunity to that particular coccidian species. These animals will appear healthy but feed efficiency may be reduced and lower weight gains could be seen. Subclinical disease is the most common syndrome in growing cattle. Calves may appear unthrifty with manure stained backsides. Clinical disease is also a condition usually of the immature animal due to lack of acquired immunity, compounded by any number of stressful events, including weather changes, overcrowding, long travel, filthy surroundings or other concurrent illnesses such as shipping fever. Calves are most susceptible between 3 weeks and 6 months of age and most clinically sick animals will be less than 12 months old. Weaning with consolidation of calves into close quarters is the perfect place to expect an outbreak.

In mild cases, animals may be asymptomatic, or may have only watery diarrhea, with little or no blood and exhibit a rough hair coat. Severely affected animals may have severe, watery diarrhea with general emaciation and weakness that may cause the calf to dehydrate without rising, thus soiling its hindquarters. Straining with defecation is usually evident and rapid dehydration, weight loss and anorexia (not eating) may also occur in such cases. In these severe cases, diarrhea may contain blood, mucous and stringy masses of tissue due to the presence of the epithelium of the intestines. Severe infections are not common but unfortunately, death can be a result.

Certain species of the many coccidia that affect cattle are more likely to cause severe disease. Eimeria zuernii, E. bovis and E. auburnensis are the main culprits in cattle. Other species may be present but are far less likely to be pathogenic. Susceptibility to infection varies. Some animals are naturally immune. Most others will develop immunity with exposure; however, this is just to the species causing illness and exposure to a new variety can induce disease. Many animals will carry the parasites and coexist reasonably well with the parasite until severe periods of stress. Older cattle may break with coccidiosis when brought to a new herd and exposed to a novel strain for that individual.

Diagnosis is made from a combination of history of illness and sanitation for the herd, clinical signs, gross lesions found at autopsy, and microscopic evaluation of intestinal scraping from the dead animals or microscopic evaluation of the
manure. Diarrhea may precede the heavy discharge of oocysts by several days and diarrhea may continue after the discharge of oocysts, thus finding evidence of coccidia in the manure may be difficult. Differential diagnoses include salmonellosis, Bovine Viral Diarrhea, malnutrition poisons or other intestinal parasites. If coccidiosis is believed to be responsible for cattle deaths, a complete autopsy should be performed to confirm the diagnosis, and stool samples should be examined immediately after diarrhea first appears in sick calves.

Animals can die from coccidiosis; either directly or from secondary diseases. While many sick animals will recover, permanent damage to the gut may occur resulting in poor weight gain or even stunting.

So called “nervous coccidiosis” is associated with heavy coccidia infestations. It is theorized that the coccidia produce a soluble protein exotoxin that is encephalotoxic. Signs include muscle tremors, convulsions, nystagmus (eyes moving back and forth or up and down in a rapid, rhythmic manner) and other central nervous system symptoms. Mortality is very high, in the 80-90% range.

Clinical coccidiosis, that is, an animal showing signs of disease, must be treated as soon as possible. In an outbreak, the clinically affected animals should be isolated and given individual treatment, both specific for the coccidia and supportive as necessary. Mass medication is indicated for those animals in the same grouping. Nervous coccidiosis requires extensive supportive care that may be hard to economically justify for a condition with such a poor prognosis. It is best to treat each sick animal individually but these same treatments can be used in a herd situation. Sulfas drugs such as sulfadimethoxine (Albon), sulfaethazine or the vitamin B1 analog amprolium (Corid) are the mainstay of treatment for clinical coccidiosis. All can be used as a drench or as a water additive. Most sick calves will still continue to drink water but if there is any question of the calf’s ability or desire to drink from a communal water supply, that animal should be dosed individually by body weight and the medication administered as a drench directly into the animal’s mouth. Animals that refuse to drink and or eat will require additional symptomatic treatment such as IV fluids. The rest of the exposed animals should be treated with the addition of these medications in the drinking water. Medicated water should be mixed up once per day. Generally treatment for acute coccidiosis continues for 5-6 days and all calves in a group of animals should be treated even if some are asymptomatic. Sulfas have the advantage of providing antibacterial effects for secondary infections that may be associated with coccidiosis, such as pneumonia or bacterial enteritis (gut infection). Amprolium replaces vitamin B1 (thiamine) in the coccidia, interfering with its life cycle. High doses for prolonged periods beyond those recommended can result in thiamine deficiency in treated animals. Likewise, high doses of thiamine to the animals under treatment can interfere with the efficacy of the drug. Secondary conditions such as dehydration, bacterial pneumonia may require specific treatment.

There are several products available to prevent coccidiosis in a group of cattle, before any of them show signs. These medications must be used for 28 days at a minimum, to ensure that they have a chance to act on the part of the life cycle for which they are effective. Amprolium can be used as a preventative at half the dosage required for treatment. Other useful drugs include the ionophore antibiotics lasalocid (Bovatec) and monensin (Rumensin) and the coccidiostat decoquinate (Deccox). These are all effective preventatives, given well before signs of disease appear, but with the exception of amprolium at the higher dose, none are effective treatments for clinical disease. These products will do no good in animals already showing signs of coccidiosis. The ionophore antibiotics lasalocid and monensin have the advantage of being very good growth promotants. However, they are usually mixed with grain and they have one important disadvantage; they are both poisonous to dogs and horses and can be fatal if ingested by the wrong species. If these products are used, extreme care should be taken to ensure that horses and dogs have no access to the premix or any grain containing them. If you have horses or dogs, decoquinate or amprolium are safer options.

Using preventative may have an unwanted side effect in that withdrawal from them has been reported to result in fatal coccidiosis in some animals, presumably because the drug suppressed the development of immunity with subsequent fatal reexposure. Anecdotally, some animals fed ionophores for the purpose of increased gain and reduction of bloat from “hot” rations have done very poorly when put out to pasture, presumably for the same reason. When these products are used in feedlot situations, slaughter is the end result and re-exposure is a non-issue. In the case of replacement animals, care should be taken when they are first weaned from the products. They should be pastured in clean areas, with minimal previous contamination so that their first introduction to the parasite is at low numbers. This will allow for immunity to develop. Animals should be closely observed for evidence of acute disease and treated accordingly.

Coccidiosis is difficult to control with absolute precision. Overcrowding should be avoided, particularly until animals have a chance to develop immunity to the protozoa in their environment. Calving areas should be well drained and kept as dry as possible. Feeders and waterers should be high enough to avoid heavy fecal contamination. The population density of animals must be kept to a minimum. Facilities allowing no access to pasture must be routinely cleaned on a daily basis to limit infective oocysts in the environment. Keeping this parasite at bay requires an ongoing combination of good husbandry practices, ample space for animal housing and careful use of indicated chemotherapeutics.