



PROTOZOOSIS OF THE CALF: *GIARDIA*, *CRYPTOSPORIDIUM*, *EIMERIA*, *SARCOCYSTIS*, *NEOSPORA*

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1. INTRODUCTION

Protozoal parasites are ubiquitous within domestic cattle. In fact, infections of cattle with *Giardia duodenalis*, *Cryptosporidium* spp., *Eimeria* spp., *Sarcocystis* spp., and *Neospora caninum* are so widespread that eradication of these organisms is seldom a wise or practical goal. The good news is that practical steps are available for the prevention or reduction of clinical disease by these parasites. These organisms share several important traits. All of them have fecal-oral transmission by encysted egg-like structures that survive for prolonged periods in the environment and resist chemical disinfection. After the initial infection, these organisms cause permanent or repetitive subclinical infections that are held in check by the development of specific immunity (i.e. “premunition”). A few management practices can reduce the risk of disease from these organisms, including ingestion of high quality colostrum, reducing parasite burdens in the farm environment, protecting feedstuffs from contamination, providing good nutrition, and prophylactic or therapeutic antimicrobial therapy.

Eimeria, *Cryptosporidium*, and to a lesser degree *Giardia*, are enteric pathogens that may cause diarrhea, resulting in decreased rate of gain or mortality in calves. In contrast, *Sarcocystis* and *Neospora* are systemic pathogens that are transmitted in the feces of dogs and some wild canids. Additionally, *Neospora* may be transmitted transplacentally from one generation of cattle to the next.

2. SYSTEMIC PROTOZOAL PARASITES

2.1 Sarcocystis

Sarcocystis cruzi is the most common *Sarcocystis* sp. of cattle. “Sporocysts,” which are shed in feces of acutely infected dogs or wild canids, are able to survive in the environment for prolonged periods. Canids become infected by ingesting meat from cattle, water buffalo, or some species of deer (Wee & Shin, 2001). Cattle become infected by grazing pasture or ingesting feed or water that are contaminated with sporocysts. Most surveys find that the great majority of adult cattle are

infected with *S. cruzi*, even cattle on farms that do not have dogs, which suggests that sporocysts may spread to distant sites by wind. After a period of intravascular dissemination and endothelial proliferation, *Sarcocystis* organisms, as their name implies, encyst within striated muscle. These cysts are called “sarcocysts.” Fortunately, *S. cruzi* forms only microscopic sarcocysts that do not affect the appearance or taste of meat and are not infectious to people.

Clinical disease from *Sarcocystis* infection is rarely noticed, and may only occur when unusually high numbers of sporocysts are ingested by previously uninfected cattle. Experiments to induce sarcocystosis have often used administration of a bolus containing tens or hundreds of thousands of *S. cruzi* sporocysts per calf (Johnson *et al.* 1975), but this probably doesn't reflect the typical situation in nature in which a trickle exposure may be more common. After a long incubation period (several weeks), experimental ingestion of sporocysts causes fever and inappetance, elevated muscle enzymes (lactate dehydrogenase and aldolase), and can cause salivation, enlarged lymph nodes, weakness, or death. At necropsy, the carcass may be pale (anemia) and there may be pale streaks in muscles. Histologic examination reveals immature stages of protozoal organisms within muscle and endothelium. Pregnant cows may abort (Lopes *et al.* 2005), but this is a rare event, in part because most cattle are exposed to *Sarcocystis* and develop protective immunity before they reach breeding age. An aborted fetus may or may not have organisms visible in endothelium of the brain and other organs.

It is conceivable that *Sarcocystis* infections may commonly induce nonspecific signs of transitory fever and reduced feed intake, but that the occurrence is unnoticed or the cause is undetermined. Alopecia of the tail switch (“rat tail”) has been noted in naturally occurring unthriftiness attributed to sarcocystosis (Giles *et al.* 1980).

Specific control programs for *Sarcocystis* infections are not warranted, other than to avoid egregious breaches of good hygiene. A risk factor for clinical sarcocystosis is to raise calves or pregnant cows in uncleaned pens that previously were or concurrently are used to keep dogs or litters of puppies (Carrigan, 1986), especially dogs that are fed bovine carcasses or raw meat.

Infection with *S. hominis* causes formation of sarcocysts that can be nearly 1 mm long, which if observed could potentially result in condemnation of the slaughtered carcass. Because people are the definitive host of this parasite (Fayer, 2004), preventive measures are obvious. Infections in cattle are associated with ingestion of raw or very rare beef by farmers or workers, and dispersal of raw human sewage in fields or defecation by workers onto cattle feedstuffs.

Cattle can also be infected with *Sarcocystis hirsuta*, which is shed by cats after consuming infected beef. These cysts may be large enough to be visible in the carcass, especially in the esophagus.

Eosinophilic myositis (EM) is an uncommon, sporadic, and potentially fatal condition. It is most often discovered at slaughter, resulting in partial or complete condemnation of the carcass. Affected meat contains scattered green-tinted foci, which histologically consist of necrotic and degenerating myocytes with intense infiltration by eosinophilic leukocytes. Several authors have noticed ruptured or degenerating sarcocysts within inflammatory foci, and it has been speculated that the condition is caused by a florid inflammatory reaction to these organisms (Jensen *et al.* 1986). Normally there is no inflammation associated with sarcocysts, so the circumstances that may create an intense eosinophilic reaction are speculative. Infection with an unknown species of *Sarcocystis* was observed in one EM-affected cow that also was infected with *S. cruzi* and *S. hirsuta* (Gajadhar *et al.* 1987), so perhaps EM may be caused by an inflammatory reaction to an aberrant species of *Sarcocystis*, or perhaps EM is predisposed by infection with multiple species that result in cross-reacting hypersensitivities.

2.2 *Neospora*

Neospora caninum is a common infection in cattle, although the parasite is not as omnipresent as *S. cruzi*. Owners of infected herds may not notice any problem. Usually, most congenitally infected calves are clinically healthy and are indistinguishable from uninfected herdmates, except by serology. Nevertheless, not all congenitally infected calves fare well. Neosporosis is one of the most common causes of bovine abortion, and is also a cause of congenital neurological defects (weak or “dummy” calves) including poor suckle reflex, poor balance, improper positioning of rear fetlocks (knuckling,” a proprioceptive deficit), inability to stand, and seizures. A few studies have also investigated the possible association of *N. caninum* serologic status with subclinical production losses, but the conclusions have varied between no subclinical effect or modest reductions in average milk production or rate of gain (Barling *et al.* 2000; Thurmond & Hietala, 1997; Waldner *et al.* 2004).

Endemic transmission is common within chronically infected herds. Epidemic transmission has been reported in numerous articles that show outbreaks of abortion, premature births, and neurologically impaired calves. Transplacental transmission may occur from a cow that first becomes infected during pregnancy as a result of ingesting oocysts shed in the feces of infected canids (“exogenous transplacental transmission”) (Gondim *et al.* 2004), or transplacental transmission may occur from a congenitally infected dam to her own offspring (“endogenous transplacental transmission”) (Anderson *et al.* 1997; Björkman *et al.* 1996; Schares *et al.* 1998). Well-studied abortion epidemics were caused by exogenous transmission, while in contrast the majority of congenital infections in endemically infected herds result from endogenous transmission.

On a national or international level, endemic abortions account for more losses than do abortion epidemics, and this may be why many articles state that endogenous transplacental transmission is more important than is transmission by canids. However, in all instances endemic transmission must have been preceded at some point in the past by ingestion of oocysts. Without periodic transmission from canids the parasite would gradually disappear from any endemically-infected herd. Consistent with this, epidemiologic studies from many different countries have associated the prevalence of neosporosis in cattle with the presence and number of dogs or other canids (Bartels *et al.* 1999; Dijkstra *et al.* 2002; Mainar-Jaime *et al.* 1999; Otranto *et al.* 2003; Ould-Amrouche *et al.* 1999; Paré *et al.* 1998; Rinaldi *et al.* 2005; Sanchez *et al.* 2003; Sawada *et al.* 1998).

Fortunately, practical steps can be taken to reduce the risk of widespread transmission of *N. caninum* from dogs to cattle. I do not recommend removing dogs from farms for several reasons:

- many farmers would reject such a recommendation and might then refuse to listen to more moderate advice,
- in some circumstances a guard dog may actually reduce the risk of infections spread by other canids, and,
- there are other practical control methods.

I have studied 3 severe abortion outbreaks, involving a dairy with 1200 cows, a dairy with 60 cows, and a beef herd with 200 cows. Each of these herds suffered a point source exposure, each herd was feeding a mixed ration to the pregnant cows, and each farm had dogs with access to feed piles. Stored feedstuffs should be protected so that dogs and other canids cannot defecate in them (which they commonly will do). Small farms may be able to keep feedstuffs within silos, grain bins, and enclosed sheds. However, modern large dairies consume too much feed to keep it all within

containers. Feedstuffs are frequently kept in large open piles. These feedstuffs are attractive to dogs and wild canids such as North American coyotes (*Canis latrans*). Dogs and coyotes consume silage, hunt rodents, and sleep and defecate in hay. If a dog is actively shedding *N. caninum* in its feces, then contamination of any feedstuff can cause rapid, widespread dissemination of the parasite to a large number of cattle that consume the mixed ration. This is to be avoided. Large dairies can erect a dog-proof fence around the area where feedstuffs are stored. A gate can be designed to open automatically when machinery approaches, or some farmers may prefer to leave the gate open during the day when human activity is high and visits by feral or wild canids are less likely. Infections acquired from grazing may be unavoidable, but these infections may occur sporadically in individuals rather than infecting a large number of cows at once.

Steps can also be taken to reduce the risk of transmitting *N. caninum* from cattle to dogs, thereby inhibiting dogs from shedding oocysts. Carcasses of cattle may harbor this parasite in muscle and nervous tissue, regardless of the reason that the animal has died. Therefore, dead stock or offal should be disposed of in a manner that keeps dogs from eating the carcass. Burying carcasses, using a rendering service for removal, or placing fences or electric fences around carcasses until they have decomposed, will prevent dogs from eating dead stock. Dogs may also become infected by consuming cattle placentas (Dijkstra *et al.* 2001), which will not always be possible to prevent. Keep in mind that the goal is to reduce the risk of transmission, but complete elimination of this risk is not possible even when no dogs are on the farm; for example, feral and wild canids may defecate in pastures, or a feedstuff may be purchased that is already contaminated.

In herds with long-term, persistently elevated, endemic abortions attributable to neosporosis (other contributing causes must be ruled out!), then selective culling of heifers in infected lines may be considered (Larson *et al.* 2004). For example, seropositive heifers may be sold or fed for slaughter, while seronegative replacement heifers may be retained for breeding. Or seropositive dairy cows may be inseminated with semen from a beef breed, thus ensuring that replacement heifers come only from seronegative dams. Embryos may be transferred from particularly valuable seropositive cows to seronegative surrogates, thus breaking the cycle of endogenous transplacental transmission and helping the next generation to be free of this parasite (Baillargeon *et al.* 2001). These strategies speed the decline of *N. caninum* seroprevalence in a herd, which may reduce endemic abortions in herds in which this is a problem. Not all infected herds have abortion problems, so I do not recommend going to the trouble and expense of culling seropositive animals in herds that do not have a reproductive problem associated with *N. caninum* serologic status. There are several additional important considerations when considering a selective culling program to control bovine neosporosis. First, *Neospora* serologic status is only one of many important factors when making culling decisions. Second, there is no ideal serological cut-off to reliably distinguish between infected and uninfected cattle. Third, reinfection of the herd is possible, thereby negating efforts to eradicate the parasite. Fourth, as the seroprevalence rate in a herd decreases, the susceptibility of the herd to a neosporosis abortion outbreak may actually increase, because chronically infected cows may be immune to exogenous transplacental transmission, while naive cows are more likely to abort if they should ingest *Neospora* oocysts during pregnancy (McAllister *et al.* 2000). Who wants to eradicate neosporosis from their herd, and then have an abortion epidemic? Therefore, in my opinion any attempt to reduce the prevalence of endogenous neosporosis in a herd by selective culling must also include management practices to reduce the risk of exogenous transmission by canids.

A commercial vaccine for bovine neosporosis has been sold in the US for about 8 years. There is still a great need for well-designed and adequately funded studies to test the effect of this vaccine on endogenous and exogenous transplacental transmission and abortion. These studies need to be published whether the results are positive or negative.

3. ENTERIC PROTOZOAL PARASITES

3.1 *Eimeria* (coccidiosis)

Bovine coccidiosis was recently reviewed by Dauschies and Najdrowski (2005). There are many species of *Eimeria* that infect cattle, but most cases of clinical coccidiosis are caused by *E. bovis* and *E. zurnii*. Diarrhea caused by *E. alabamensis* has been reported in Sweden and Germany (Svensson *et al.* 1994; von Samson-Himmelstjerna *et al.* 2006). Other species of *Eimeria* seldom cause diarrhea, and mixed infections are common. All bovine *Eimeria* have a fecal-oral transmission cycle, with cattle as the sole hosts. Almost all cattle shed *Eimeria* oocysts at one time or another, most without clinical signs of disease. Nevertheless, coccidiosis is a common and important cause of diarrhea, reduced weight gain, and mortality in calves. The occurrence of diarrhea depends upon the interaction of many factors, including: the species of coccidia; the density of *Eimeria* oocysts in the environment and the related rate of exposure of naive calves to oocysts; environmental temperature, humidity, and sunlight, which affect the development and survival of oocysts; and stressors such as weaning, poor nutrition, or severe weather extremes (Dauschies & Najdrowski, 2005).

Clinical coccidiosis most frequently affects calves between 3 weeks and 6 months of age, or soon after weaning. The onset of diarrhea may slightly precede fecal shedding of oocysts, but many diarrheic calves will be shedding large numbers of oocysts. Although *Eimeria* oocysts are smaller than helminth ova, they are nevertheless easy to observe in routine fecal floats when they are shed in large numbers. Counts of less than 5000 oocysts per gram of feces may not be clinically significant, while counts well above 100,000 per gram may occur in disease outbreaks. Diarrheic feces may contain mucous, red blood, and shreds of sloughed mucosa, and calves exhibit tenesmus. Milder cases may exhibit only soft stools, rough hair coats, and poor growth.

Cattle of all ages shed *Eimeria*, but calves with primary infections shed the greatest number. Massive infections can create massive contamination of a premises, thus leading to a vicious cycle of overwhelming exposure, severe infections, and further environmental contamination. Fecal contamination is of course a simple fact of life in all cattle operations, but certain management techniques may reduce the level of contamination of the environment and the rate of exposure of naive young stock to the parasites. After being passed in feces, coccidial oocysts typically take 2-4 days to "sporulate" and become infectious, so frequent cleaning of calf pens is an important technique to help limit the rate of exposure. Individual calf hutches, when cleaned and moved between calves, are superior to group pens. Sunlight and dessication are the best disinfectants for protozoal oocysts, while chemical disinfection is difficult. In dairy cattle, indoor pens that receive little sunlight, moist bedding with infrequent changes, and crowding all increase the build-up and survival of oocysts.

In beef cattle, clinical coccidiosis may occur in the autumn shortly after calves are stressed by weaning. Certain pastures may be associated with coccidiosis, possibly because they are shaded and moist (favoring long survival of oocysts), were recently fertilized with fresh manure, or because of disadvantageous pasture rotation practices. Hay or other feeds should not be spread on the ground, which increases fecal-oral transmission, but should be placed in elevated feeders. If practical, locations that feeds are provided may be moved frequently to avoid excessive concentration of feces and to move cattle away from oocysts before they have sporulated. Coccidial diarrhea is a common problem in calves entering feedlots, unless prophylactic coccidiostats are used such as monensin, decoquinate, or amprolium.

Nervous coccidiosis is an uncommon clinical manifestation associated with coccidial diarrhea, most frequently observed in feedlot cattle (Isler *et al.* 1987). Clinical signs include diarrhea with heavy

Eimeria oocyst counts, plus CNS signs including opisthotonus, horizontal nystagmus, and muscle fasciculations. The pathogenesis may be related to a poorly characterized neurotoxin elaborated from the enteric parasites. The differential diagnosis includes polioencephalomalacia and other CNS diseases. Mortality is very high.

3.2 *Cryptosporidium*

As is true for all of the protozoal organisms discussed here, most infections with *Cryptosporidium* are subclinical. Different species of *Cryptosporidium* are morphologically identical, and speciation has only recently become possible by using genetic analyses. Cryptosporidial diarrhea occurs in young calves infected with *C. parvum*, approximately between the ages of 5 days and 4 weeks. Cryptosporidiosis often occurs in combination with other neonatal diarrheic pathogens, but *C. parvum* can be the sole pathogen, causing a yellow mucoid diarrhea. Cryptosporidiosis is more common in dairy than beef calves, because of differences in stocking density, feeding, and calving seasons. In studies in western Canada, cryptosporidiosis with high mortality in beef herds has been associated with the introduction of dairy calves (probable carriers) and with selenium deficiency (micronutrient stress) (Olson *et al.* 2004).

Prevention of clinical disease relies on sanitation and secondary factors such as good colostrum management. Risk factors are similar to those described for *Eimeria*, except that the period of risk is limited to the first month of life, after which the risk of coccidiosis is just beginning. There are many similarities between *C. parvum* and *Eimeria*, including fecal-oral transmission and long-lived oocysts that are resistant to chemical disinfectants. Again, sunlight and dessication are among the most effective methods of killing the oocysts. Individual calf hutches can reduce the spread of this organism between young calves, and moving calf hutches whenever calves are changed will allow sunlight to help reduce the level of contamination. One important difference from *Eimeria* is that *Cryptosporidium* oocysts are immediately infectious when passed in the feces.

Cryptosporidium oocysts are quite tiny and difficult for untrained examiners to observe. A common diagnostic test is acid-fast staining of a fecal smear, but fecal floats and immunofluorescent tests may also be performed, and the organism can be seen histologically in the small intestine, especially the ileum. Until recently there were no effective antimicrobial treatments, but now drugs such as azithromycin, paromomycin, and halofuginone appear to be helpful as preventives or treatments, and may be available in some countries.

As many veterinary students can attest, *C. parvum* is a zoonotic pathogen. Until recent molecular analyses, *C. parvum* was believed to be the sole cause of cryptosporidiosis of people, but now it is only one of several species known to infect humans, including *C. meleagridis* first discovered in turkeys, and most importantly *C. hominis* of people and other primates (Caccio *et al.* 2005). Organisms that are still named *C. parvum* have been divided into various subgroups, some that appear to be specific for humans, some that are specific for cattle, and some that are shared between cattle, humans, and other animals. A cattle-specific subgroup of *C. parvum* was recently proposed to be a separate species, *C. bovis* (Fayer *et al.* 2005). In a recent study, *C. bovis* was more prevalent in calves between the ages of 1 and 11 months (not associated with diarrhea), while *C. parvum* was more prevalent in calves less than 1 month old (when cryptosporidial diarrhea may occur) (Fayer *et al.* 2005).

Cryptosporidium hominis is more frequently found in cases of human diarrhea in the Americas, Australia, and Africa, while *C. parvum* is more common in parts of Europe (Caccio *et al.* 2005). Interestingly, the incidence of human cryptosporidiosis in Great Britain decreased in association with the epidemic of foot and mouth disease (Smerdon *et al.* 2003). This has been attributed to the associated mass culling of cattle and sheep and restricted access of people to rural areas, hence

secondarily reducing the zoonotic spread of *C. parvum*. Although cattle should no longer be blamed as the single major source of human cryptosporidiosis, zoonotic transmission from cattle to people is nevertheless an important public health concern. More studies are needed to address concerns about public health and such things as manure fertilization, water run-off from farms, and contamination of surface waters by livestock feces.

Cryptosporidium andersoni infects the abomasal glands of feedlot and adult cattle (Olson *et al.* 2004). Some sources identify this organism as *C. muris*, but *C. muris* of mice is genetically distinct. *Cryptosporidium andersoni* causes persistent infections of abomasal glands, and may reduce milk production or rate of growth. When detected in fecal exams, *C. andersoni* oocysts are slightly larger than *C. parvum* and *C. bovis*, and the diagnosis can also be achieved using histologic examination of the abomasum.

3.3 *Giardia*

As with *Cryptosporidium*, recognition of different species of *Giardia* was quite problematic before genetic analyses. Recent molecular information is now changing the nomenclature and concepts about zoonotic transmission. Cattle may be infected with *G. duodenalis*, which had previous synonyms of *G. intestinalis* and *G. lamblia*. Although *G. duodenalis* is an important cause of diarrhea in people, and transmission from livestock has long been suspected, current genetic evidence suggests that cattle and people are infected by different subtypes (Caccio *et al.* 2005; Olson *et al.* 2004).

Similar to *Eimeria*, *Giardia duodenalis* infects nearly all cattle at one time or another, and may cause of diarrhea in calves older than 1 month. *Giardia* cysts are infectious at the time they are passed in feces. Although the cysts are larger than *Cryptosporidium* oocysts, they are still quite tiny, much smaller than *Eimeria*, only about 14 microns long. Benzimidazoles such as fenbendazole are effective treatments for *G. duodenalis* (Olson *et al.* 2004). There aren't many articles about clinical giardiasis in cattle, but it isn't clear if this is because it is an infrequent problem, or if the condition is frequently overlooked because the organism is small and difficult to observe.

4. SUMMARY

Protozoal parasites of cattle are extremely common. *Eimeria bovis*, *E. zurnii*, and *E. alabamensis* may cause bloody diarrhea in calves between the ages of 3 weeks and 6 months, or shortly after weaning. *Cryptosporidium parvum* may cause diarrhea in calves between 5 days and 4 weeks of age. In addition, *C. parvum* can cause diarrhea in people. *Giardia duodenalis* is a common parasite of cattle and may be associated with diarrhea in calves older than 1 month, although there are only a small number of reports in the literature. Bovine isolates of *Giardia* appear to be unlikely to infect people. All of these enteric parasites have fecal-oral transmission among cattle, and control measures are discussed. *Sarcocystis cruzi* is a ubiquitous systemic parasite that is transmitted in canine feces; after ingestion by cattle the organism encysts in striated muscle, but rarely causes clinical illness. *Neospora caninum* is a common cause of abortion, and has two important methods of transmission to cattle; ingestion of oocysts that are shed by acutely infected dogs, or endogenous transplacental transmission from a congenitally infected dam to her own offspring. Control measures are discussed.

5. KEY WORDS

Protozoal parasites, diarrhea, cattle.

6. RESUME

Les protozoaires parasites sont couramment rencontrés chez les bovins. *Eimeria bovis*, *E. zurnii*, et *E. alabamensis* peuvent être à l'origine d'une diarrhée sanguinolente chez les veaux entre 3 semaines et 6 mois d'âge, ou peu après le sevrage. *Cryptosporidium parvum* peut causer une diarrhée chez les veaux âgés de 5 jours à 4 semaines. De plus, *C. parvum* peut être responsable de diarrhée chez l'homme. *Giardia duodenalis* est un parasite commun des bovins qui peut être associé à des épisodes de diarrhée chez les veaux de plus d'un mois, bien que très peu de données soient disponibles dans la littérature. Les isolats bovins de *Giardia* ne semblent pas infecter l'homme. Tous ces parasites intestinaux se transmettent chez les bovins selon un cycle fécal-oral et les moyens de contrôle sont discutés dans cet article. *Sarcocystis cruzi* est un parasite systémique, ubiquitaire qui se transmet par ingestion de fèces d'origine canine. Après ingestion, le parasite s'enkyste dans les muscles striés mais provoque rarement des signes cliniques. *Neospora caninum* est une cause commune d'avortement. Ce parasite se transmet aux bovins selon deux possibilités, l'ingestion d'oocystes qui sont excrétés par les chiens lors d'infection aigüe, ou une transmission transplacentaire de la mère infectée à sa propre descendance. Les moyens de lutte contre *Neospora caninum* sont présentés dans cet article.

7. MOTS CLES

Protozoaires parasites, diarrhée, bovin.

8. REFERENCES

- Anderson ML, Reynolds JP *et al.* Evidence of vertical transmission of *Neospora* sp. infection in dairy cattle. JAVMA, 1997; 210:1169-72.
- Baillargeon P, Fecteau G *et al.* Evaluation of the embryo transfer procedure proposed by the International Embryo Transfer Society as a method of controlling vertical transmission of *Neospora caninum* in cattle. JAVMA, 2001; 218:1803-6.
- Barling KS, McNeill JW *et al.* Association of serologic status for *Neospora caninum* with postweaning weight gain and carcass measurements in beef calves. JAVMA, 2000; 217:1356-60.
- Bartels CJ, Wouda W, Schukken YH. Risk factors for *Neospora caninum*-associated abortion storms in dairy herds in The Netherlands (1995 to 1997). Theriogenology, 1999; 52:247-57.
- Björkman C, Johansson O *et al.* *Neospora* species infection in a herd of dairy cattle. JAVMA, 1996; 208:1441-4.
- Caccio SM, Thompson RC *et al.* Unravelling *Cryptosporidium* and *Giardia* epidemiology. Trends Parasitol, 2005; 21:430-7.
- Carrigan MJ. An outbreak of sarcocystosis in dairy cattle. Aust Vet J, 1986; 63:22-4.
- Dauguschies A, Najdrowski M. Eimeriosis in cattle: current understanding. J Vet Med B Infect Dis Vet Public Health, 2005; 52:417-27.
- Dijkstra T, Barkema HW *et al.* Natural transmission routes of *Neospora caninum* between farm dogs and cattle. Vet Parasitol, 2002; 105:99-104.
- Dijkstra T, Eysker M *et al.* Dogs shed *Neospora caninum* oocysts after ingestion of naturally infected bovine placenta but not after ingestion of colostrum spiked with *Neospora caninum* tachyzoites. Int J Parasitol, 2001; 31:747-52.
- Fayer R. *Sarcocystis* spp. in human infections. Clin Microbiol Rev, 2004; 17:894-902.
- Fayer R, Santin M, Xiao L. *Cryptosporidium bovis* n. sp. (Apicomplexa: Cryptosporidiidae) in cattle (*Bos taurus*). J Parasitol, 2005; 91:624-9.

- Gajadhar AA, Yates WD, Allen JR. Association of eosinophilic myositis with an unusual species of *Sarcocystis* in a beef cow. *Can J Vet Res*, 1987; 51:373-8.
- Giles RC, Jr., Tramontin R *et al.* Sarcocystosis in cattle in Kentucky. *JAVMA*, 1980; 176:543-8.
- Gondim LFP, McAllister MM *et al.* Transplacental transmission and abortion in cows administered *Neospora caninum* oocysts. *J Parasitol*, 2004; 90:1394-1400.
- Isler CM, Bellamy JE, Wobeser GA. Pathogenesis of neurological signs associated with bovine enteric coccidiosis: a prospective study and review. *Can J Vet Res*, 1987; 51:261-70.
- Jensen R, Alexander AF *et al.* Eosinophilic myositis and muscular sarcocystosis in the carcasses of slaughtered cattle and lambs. *Am J Vet Res*, 1986, 47:587-93.
- Johnson AJ, Hildebrandt PK, Fayer R. Experimentally induced sarcocystis infection in calves: pathology. *Am J Vet Res*, 1975; 36:995-9.
- Larson RL, Hardin DK, Pierce VL. Economic considerations for diagnostic and control options for *Neospora caninum*-induced abortions in endemically infected herds of beef cattle. *JAVMA*, 2004; 224:1597-604.
- Lopes CW, de Sa WF, Botelho GG. [Lesions in cross-breed pregnant cows, experimentally infected with *Sarcocystis cruzi* (Hasselmann, 1923) Wenyon, 1926 (*Apicomplexa: Sarcocystidae*)]. *Rev Bras Parasitol Vet*, 2005; 14:79-83.
- Mainar-Jaime RC, Thurmond MC, *et al.* Seroprevalence of *Neospora caninum* and abortion in dairy cows in northern Spain. *Vet Rec*, 1999; 145:72-5.
- McAllister MM, Björkman C, *et al.* Evidence of point-source exposure to *Neospora caninum* and protective immunity in a herd of beef cows. *JAVMA*, 2000; 217:881-7.
- Olson ME, O'Handley RM *et al.* Update on *Cryptosporidium* and *Giardia* infections in cattle. *Trends Parasitol*, 2004; 20:185-91.
- Otranto D, Llazari A *et al.* Seroprevalence and associated risk factors of neosporosis in beef and dairy cattle in Italy. *Vet Parasitol*, 2003; 118:7-18.
- Ould-Amrouche A, Klein F *et al.* Estimation of *Neospora caninum* seroprevalence in dairy cattle from Normandy, France. *Vet Res*, 1999; 30:531-8.
- Paré J, Fecteau G *et al.* Seroepidemiologic study of *Neospora caninum* in dairy herds. *JAVMA*, 1998; 213:1595-8.
- Rinaldi L, Fusco G *et al.* *Neospora caninum* in pastured cattle: determination of climatic, environmental, farm management and individual animal risk factors using remote sensing and geographical information systems. *Vet Parasitol*, 2005; 128:219-230.
- Sanchez GF, Morales SE *et al.* Determination and correlation of anti-*Neospora caninum* antibodies in dogs and cattle from Mexico. *Can J Vet Res*, 2003; 67:142-5.
- Sawada M, Park CH *et al.* Serological survey of antibody to *Neospora caninum* in Japanese dogs. *J Vet Med Sci* 1998, 60: 853-4.
- Schares G, Peters M *et al.* The efficiency of vertical transmission of *Neospora caninum* in dairy cattle analysed by serological techniques. *Vet Parasitol*, 1998; 80:87-98.
- Smerdon WJ, Nichols T *et al.* Foot and mouth disease in livestock and reduced cryptosporidiosis in humans, England and Wales. *Emerg Infect Dis*, 2003; 9:22-8.
- Svensson C, Ugglå A, Pehrson B. *Eimeria alabamensis* infection as a cause of diarrhoea in calves at pasture. *Vet Parasitol*, 1994; 53:33-43.
- Thurmond MC, Hietala SK. Effect of *Neospora caninum* infection on milk production in first- lactation dairy cows. *JAVMA*, 1997; 210:672-4.
- von Samson-Himmelstjerna G, Epe C *et al.* Clinical and epidemiological characteristics of *Eimeria* infections in first-year grazing cattle. *Vet Parasitol*, 2006; 136:215-21.

Waldner C, Wildman BK *et al.* Determination of the seroprevalence of *Neospora caninum* in feedlot steers in Alberta. Can Vet J, 2004; 45:218-24.

Wee SH, Shin SS. Experimental induction of the two-host life cycle of *Sarcocystis cruzi* between dogs and Korean native calves. Korean J Parasitol, 2001; 39:227-32.