for infection appear to be immunodeficiency and age. The prevalence of infection in immunodeficient foals with diarrhoea has been 80 to 100%, compared to 0 to 100% in studies of immunocompetent foals with diarrhoea.[3,4] In this report, the humoral immune status of the stallion appeared normal, based on IgG enzyme immunoassay of two separate serum samples. The cell-mediated immune status of the stallion was presumed to be normal on the basis of its normal lymphocyte count. However, more definitive tests to assess immune status were not performed.

Prevalence of cryptosporidiosis appears to decrease with age,[3,4] and cryptosporidiosis is not a recognized cause of diarrhoea in adult horses. In this case, diarrhoea appeared to be associated with cryptosporidial oocyst shedding in an adult horse, and no other enteropathogens or aetiological agents were identified after extensive diagnostic testing.

Cryptosporidium infection is diagnosed antemortem by finding oocysts in faeces. Faecal oocyst excretion is intermittent, therefore multiple fresh samples must be examined to increase the chances of accurate diagnosis.[5] The most widely used diagnostic technique is acid-fast staining, but this method has low sensitivity and specificity.[6,7] The sensitivity and specificity of oocyst detection can be greatly increased by using immunofluorescent staining and fluorescent microscopy.[6] Immunofluorescent assay, used in the present case, has been estimated to be at least 10 times more sensitive than acid-fast staining.[8]

Treatment of cryptosporidiosis is mainly supportive. Replacement of lost fluids and electrolytes and maintenance of adequate hydration are of paramount importance.[2-4] Various chemotherapeutic and immunotherapeutic drugs have been used in humans and other animals. No consistently effective treatment has been found.[3] The clinical improvement in this case may have been due to intravenous fluid therapy and the self-limiting nature of the disease.

Control measures for cryptosporidiosis involve reducing environmental oocyst contamination and bolstering the immune status of potential hosts through vaccination and adequate nutrition.[2] The zoonotic potential of the disease must also be addressed. Minimizing human exposure to infected animals through isolation and basic hygiene is essential to prevent zoonotic infection.[3]

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Yersiniosis and trace element deficiency in a dairy herd

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Yersinia pseudotuberculosis infection in cattle in Australia has previously been reported in Victoria and in New South Wales as a disease affecting cattle of all ages and with a distinct seasonal incidence.[1,2] Presentations have included 'found dead', acute and chronic enteritis and subclinical infection. The seasonal incidence probably reflects the ability of the organism to survive moist conditions.[1]

Trace element deficiency limiting the productivity of grazing cattle in southern Australia is well-documented, but leads to an ill-defined syndrome in southeast Queensland.[3,4] We report here losses associated with *Y pseudotuberculosis* infection and trace element deficiency in a dairy herd.

Case report

A dairy farmer maintained a mixed, predominantly Friesian, herd in hilly country near Dayboro in southeast Queensland. In July 1990, 10 Jersey cows of mixed ages were introduced to the farm. The cows were purchased from Kenilworth, about 80 km to the north of Dayboro.

At the same time the farmer noticed that several replacement heifers were growing poorly and had harsh, starey coats. Some of these animals were also scouring and had submandibular swellings. Veterinary attention was sought when two recently calved cows, one Friesian and one Jersey, became recumbent and were unable to rise. The farm was visited by one of us (DRK) and the following observations were recorded. The recumbent cows, calved 6 and 7 days respectively and recumbent for 24 h, showed increased temperatures (39.6 and 39.9°C), increased heart rates and pale

[a]Deceased

mucous membranes, and both had foul-smelling watery faeces tinged with blood. Both cows had been inappetent and listless prior to becoming recumbent.

A tentative diagnosis of salmonellosis was made and both cows were treated with short-acting oxytetracycline by intravenous injection. One of these cows died within 24 h but was unavailable for necropsy.

Because of a previous history of trace element deficiency on this farm, samples were collected from eight replacement heifers and the two recumbent cows: heparinised blood was submitted for copper and selenium estimation, and faecal samples were sent for worm egg counts and microbial culture. The results of these tests are shown in Table 1.

Two days later, a 4-year-old Jersey cow became recumbent and died after a short illness. The cow had calved 10 days previously and had also shown inappetence, dullness, submandibular swelling, decreased milk production and bloody diarrhoea.

The cow was necropsied in the field, with the following findings. A clear yellow fluid infiltrated sub-mandibular tissues. The abomasal wall was oedematous, thickened to twice normal size and ecchymoses were present in the mucosa. The small intestinal contents were watery and brown-green and the mucosa showed ecchymotic haemorrhages. Haemorrhages and mucosal oedema were present in the wall of the caecum. The mesenteric lymph nodes appeared normal. Samples of the gastrointestinal tract submitted for histological examination revealed marked submucosal oedema of the abomasum and small intestine, and severe erosive purulent enteritis of the small intestine (Figure 1). Numerous bacterial microcolonies (of small Gram- negative rod-shaped bacteria) were present in the purulent exudate (Figure 2). A fresh liver sample from the cow submitted for analysis revealed a liver copper content of 12 mg/kg dry matter (reference 40-1000).

Blood samples collected at the first visit revealed low glutathione peroxidase (GSH-Px, used to assess selenium status) values in both recumbent cows and a low plasma copper value in one of these cows (Animals 1 and 2, Table 1). A low GSH-Px and/or plasma copper value was also recorded in six of the eight heifers, although low concentrations of both were recorded in only one heifer (animal 4, Table 1).

Table 1. Blood glutathione peroxidase activity (GSH-Px) and plasma copper concentrations, faecal egg count and faecal culture results for 10 animals of mixed age and breed sampled at the time of the first visit.

Animal number	Age months	Breed J=Jersey F=Friesian	Blood GSH-Px[a] IU/g Hb	Plasma copper mg/L	Faecal egg count eggs/g	Faecal culture[b]
1	48	J	27	372	< 25	*Y pseudotuberculosis*
2	50	F	43	693	30	*Y pseudotuberculosis*
3	15	F	200	411	< 25	–
4	15	J	35	424	200	–
5	12	J	44	725	50	*Y pseudotuberculosis*
6	16	F	265	646	150	–
7	14	F	31	442	< 25	–
8	17	F	44	674	50	–
9	14	F	235	410	100	–
10	15	F	210	587	< 25	–

[a]Reference ranges: GSH-Px 60-400 IU/g Hb, plasma copper 500-1100 mg/L (Animal Research Institute, Queensland Department of Primary Industries, Moorooka, Queensland 4105)
[b]Faecal culture (–) indicates no *Salmonella* sp or *Yersinia* sp isolated

These findings suggested that two conditions coexisted on the property, *Yersinia* enteritis in adult cattle and trace element deficiency affecting cattle of all ages, with young cattle more severely affected. All replacement heifer stock 12 months of age or older were dosed concomitantly with copper capsules and selenium pellets (Cuprax Sustained Release Copper Supplement for Cattle and Permasel Selenium Pellets for Cattle, Mallinckrodt Veterinary [Coopers Brand]). The condition of the heifers was then monitored visually by one of us (DRK) and was observed to improve, although actual heifer growth rates were not recorded. No further cases of enteritis occurred in adult cows in the herd.

Discussion

Three cases of enteritis associated with *Y pseudotuberculosis* occurred in cattle on this farm with clinical and pathologic features similar to those already described in Australia.[1,2] The occurrence during the winter months is consistent with epidemiological features described in these reports, however yersiniosis has not previously been described in cattle in Queensland.

Subclinical infection with *Y pseudotuberculosis* is also well-documented.[1,2,5,6] It was not possible to measure the extent of subclinical infection in cattle on this farm, but the organism was isolated from one heifer that was not scouring (animal 5, Table 1). The association between parturition and *Yersinia* enteritis does not appear to have been documented but may be related to alteration in immune responsiveness during the peripartum period.[7] It is possible that the infection in these parturient cows was introduced to the farm when cattle were purchased from Kenilworth.

Copper and selenium deficiency have previously been diagnosed in young stock on this property.[4] At that time affected cattle were treated with sodium selenate drench and copper glycinate injection but the treatment was stopped when blackleg was induced in treated unvaccinated cattle (unpublished). The introduction of selenium bullets and copper oxide needles has reduced the likelihood of toxicity associated with treatment, but an apparent antagonistic effect of selenium on hepatic copper concentration was noted in cattle dosed with copper oxide plus selenium bullets in South Australia.[8,9]

On this farm two of three cows that developed acute yersiniosis had low blood selenium concentrations and it is possible that these low concentrations, combined with recent calving, predisposed them to infection. An association between selenium deficiency, impaired polymorphonuclear bactericidal activity and increased incidence of clinical mastitis has been noted by several

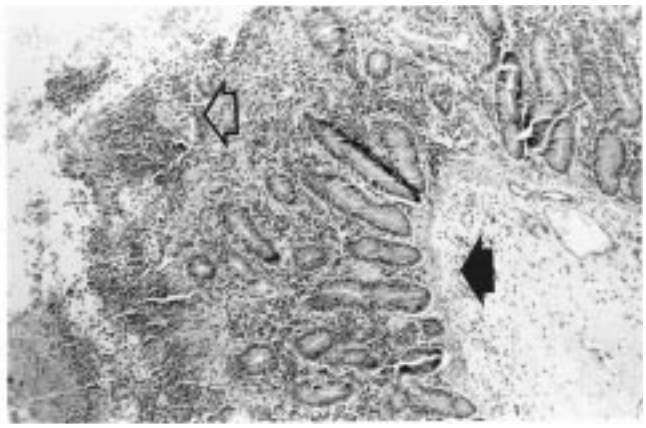

Figure 1. Marked submucosal oedema of the small intestine (solid arrow) and inflammation involving the mucosa (hollow arrow) of the small intestine. Haematoxylin and eosin x 100.

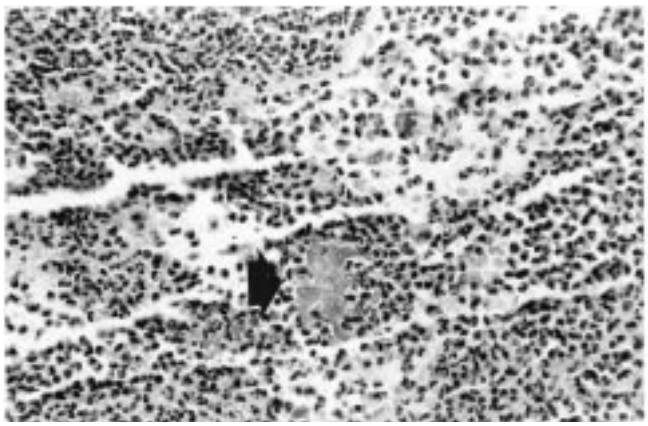

Figure 2. Close-up view of the small intestine showing a bacterial microcolony (solid arrow) embedded in the purulent exudate. Haematoxylin and eosin x 400.

workers.[10-12] Other studies have linked selenium supplementation to reduced severity and duration of coliform mastitis, to an increased rate of uterine involution and to a decreased incidence of retained placenta, although an increased susceptibility to toxic metritis in selenium, deficient cows was also noted recently.[13-16] An association between intestinal yersiniosis and copper deficiency has also been observed recently in beef herds in northern New South Wales (J Boulton personal communication).

We conclude that in, south-eastern Australia, yersiniosis should be considered in the differential diagnosis of cattle that are recumbent and scouring shortly after calving and that this condition can be seen concurrently with trace element deficiency.

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