

Testing South American Camelids for Disease

There are four species of South American camelids, but only two are largely domesticated; the llama and the alpaca. A third, the guanaco, can be tamed and some herds are present in the UK.

Alpacas and llamas are generally kept as extensively managed herds at grass all year round with access to shelter. Herds may be on holdings with or without other species of livestock and llamas also may be co-grazed with sheep as 'guard' animals.

In terms of disease, generally they are susceptible to the same parasitic and bacterial diseases as other livestock species but have more limited susceptibility to the common viral diseases of livestock, with the notable exception of BVD. They are also susceptible to nutritional disease associated with imbalances in energy, protein, macrominerals and trace elements and notable additions to this list are zinc and vitamin D deficiencies.

BIOCHEMISTRY

Metabolic and hepatic disease

Camelids have higher blood glucose levels and lower BHB levels than domestic ruminants. Unlike ruminants, in which alimentary ketogenesis is the primary mechanism for ketone body production, in camelids it appears to be hepatic ketogenesis. Camelids also appear able to absorb more glucose from their forestomachs than ruminants, have a weak insulin response and slower uptake of glucose. Disturbances of energy metabolism have similarities with both ruminants (ketosis) and monogastric species (hyperlipidaemia).

Abnormal carbohydrate metabolism in camelids can result in hepatic lipidosis, pancreatitis and diabetes mellitus. Potential precipitating factors for hepatic lipidosis include infectious disease, parasitism, stress, poor nutrition and possibly obesity. Disease onset may be acute. In one study, the most common biochemical abnormalities were raised bile acids, GGT, ALP, AST, glucose, BHB and NEFA, and low Total Protein.

Other causes of hepatocellular damage in camelids apart from lipidosis include bacterial hepatitis, copper poisoning, lymphoma, fasciolosis, tuberculosis and anoxia (such as with pronounced anaemia associated with e.g. haemonchosis). In the USA, SDH is the most commonly measured hepatocellular enzyme, but this is not routinely available in the UK. However, the activity of GLDH is found to parallel that of SDH and tends to be less labile; it therefore appears to be a useful alternative. GGT is the most widely used biliary enzyme.

Renal disease

Camelids appear to be more prone to renal disease than ruminants and reference ranges for urea, sodium and potassium tend to be higher. Azotaemia may be pre-renal, renal or post-renal. Electrolyte abnormalities (e.g. hyperkalaemia, hyperphosphataemia, hyponatraemia, hypocalcaemia) may also be seen in cases of renal disease. Reported infectious causes include pyelonephritis, tuberculosis and leptospirosis.

GI disease

Camelids with GI obstructions tend to have hypochloreaemia without hyponatraemia. Scour may be associated with hypochloreaemia and hyponatraemia. Protein losing enteropathy will lead to hypoalbuminemia.

Assessment of colostral antibody transfer

There are reports suggesting that ZST levels may not correlate well with colostral antibody transfer. Total protein > 55 g/l and globulin > 23 g/l were considered consistent with adequate colostral antibody transfer in one report.

Trace element deficiencies

Copper deficiency: normal levels are lower in alpacas than in other species. Reports of copper deficiency in camelids have mostly been anecdotal, following response to supplementation. Ascending paralysis in a six-month-old llama was associated with very low liver and kidney copper levels.

Selenium/vitamin E deficiency: evidence for selenium deficiency appears to be largely circumstantial, signs including myopathy, weak neonates, ill thrift, infertility and neuropathy. Primary vitamin E deficiency associated with myopathy has been reported in a llama.

Vitamin B12 deficiency: this has not been reported in camelids.

Vitamin D deficiency; rickets: there is an increased risk of vitamin D deficiency in camelids kept at very high and low latitudes, due to reduced exposure to ultraviolet radiation, and this is exacerbated in the spring when grass is relatively low in vitamin D. Crias born in late summer and autumn with little supplementary feeding are at high risk of developing rickets, signs including reduced growth rate, reluctance to move, lameness, recumbency and angular limb deformities. Low vitamin D and phosphorus levels are seen.

Zinc deficiency: Camelids have lower zinc levels than domestic ruminants. Zinc-responsive skin disease, non-pruritic hyperkeratosis, has been reported in young adult camelids, although direct association of such skin lesions with zinc deficiency has not been absolutely confirmed. Histopathology is also important in diagnosis.

Testing:

- Axiom camelid profile - additional tests can be added e.g. Na, Cl, K, glucose, triglycerides
- Total protein or globulin for assessment of colostral antibody transfer.
- Trace element profile (cattle) - GSH-Px, copper
- Vitamin D3 (referral test) and phosphorus
- Zinc - referral test.

HAEMATOLOGY

Erythrocytes

Erythrocytes are smaller and counts are higher than in ruminants; they also are uniquely shaped (flat ellipsoids). Reticulocytosis, anisocytosis and polychromasia are not consistent findings in regenerative anaemias in camelids. GI parasites, in particular *Haemonchus contortus*, ectoparasites and *Mycoplasma haemolamae* may all cause regenerative anaemia. Profound non regenerative anaemia may be seen with bone marrow neoplasia and other disorders. Anaemia

of chronic inflammation also may be seen and anaemia has also been reported with copper responsive disorders in llamas.

Leucocytes

The neutrophil count is higher in camelids than in domestic ruminants. Stress neutrophilias are not uncommon and can significantly elevate counts; neutrophils are typically mature. Immature and toxic neutrophils (bands) may be seen with acute inflammation. Eosinophilia may be seen with endoparasitism and ectoparasitism.

Available tests:

- Axiom haematology - screen or comprehensive
- *Mycoplasma haemolamae* PCR - referral test

Coagulation assays

- PT: 6 – 11 seconds; APTT: 8 – 22 seconds

SCOUR TESTS

Diseases that may cause scour:

Rotavirus: reported as a cause of scour in crias (7 days +) and possibly in adults. There is antigenic variation between strains which are not species specific. Rotavirus PAGE is recommended by APHA.

Coronavirus: alpaca coronavirus infection is considered important in alpacas in the USA and has been reported in all age groups (9 days +). Diagnosis in the USA is carried out by electron microscopy. There has been only one report of enteric coronavirus infection in the UK in 2008, which was picked up on a series of 'near pan' coronavirus PCR tests at APHA Weybridge. Alpaca coronavirus is a Group 2 coronavirus with greater than 99.5% homology to bovine coronavirus. It is unclear how effective lateral immunochromatography, the test used for bovine coronavirus at Axiom, would be in detecting coronavirus. A coronavirus antigen ELISA used at APHA did not prove to be useful. The Moredun Institute have a PCR test for bovine coronavirus but it is unclear how effective this would be for alpaca coronavirus. Histopathology of the small and large intestine may provide a diagnosis in cases that go to post mortem examination.

Cryptosporidiosis: infection is common in crias of 7- 21 days of age and has been recorded in animals up to eight months of age. Signs reported include diarrhoea, dehydration and recumbency, weight loss and inappetence. Asymptomatic carriers have also been reported and may pose an infection risk to both other alpacas and to their handlers. MZN staining of faeces can be used for diagnosis.

Giardiasis: Giardia cysts have been detected in 10 to 60 day old scouring crias, but most typically in crias up to 21 days. They have also been detected in healthy crias and therefore their significance is not always clear. Giardia cysts can be picked up on faecal microscopy.

Coccidiosis: all five reported species of *Eimeria* in camelids have been detected in the UK: *E. macusaniensis*, *E. punoensis*, *E. lamae*, *E. alpaca* and *E. ivitaensis*. Prepatent periods vary with the species; *E. punoensis* is as short as 10 days, *E. macusaniensis* may exceed 30 days. *Eimeria* species other than *E. macusaniensis* rarely cause clinical disease in camelids older than one year of age. *Eimeria macusaniensis* is also associated with the greatest severity of disease. Diagnosis of clinical cases is complicated by the potential for prepatent infections with *E. macusaniensis*, the similarity in size between the large camelid coccidia, *E. ivitaensis* and *E. macusaniensis* and because many asymptomatic alpacas shed coccidia. Therefore, any coccidial oocysts detected in faeces need to be considered in light of clinical signs. These most frequently include bloody diarrhoea and/or ill thrift, with dehydration and tenesmus. Sudden death also has

been reported. Axiom uses a centrifugation method with saturated sugar solution, similar to the Modified Stoll's Method, for camelid worm egg and coccidial oocyst counts.

Parasitic gastroenteritis: this is a significant disease entity in camelids reared in the UK. Many of the nematode species encountered in domesticated camelids are species also encountered in domestic ruminants and some species considered of little significance in ruminants are pathogenic in camelids, e.g. *Trichuris*. Of particular note is *Haemonchus contortus*, as infections are a significant cause of anaemia in camelids. There are also a number of species unique to camelids, e.g. *Camelostrongylus mentulatus* and *Nematodirus lamae*, which have been detected in the UK. There is some suggestion in the literature that low strongyle egg counts may be associated with significant disease in camelids and egg counting methods should be particularly sensitive and use solutions that have a high specific gravity to ensure flotation of *Trichuris* eggs (and *Eimeria macusaniensis* oocysts). As indicated above, Axiom uses a centrifugation method with saturated sugar solution, similar to the Modified Stoll's Method, for camelid worm egg and coccidial oocyst counts.

Escherichia coli infection: typically presents as colisepticaemia rather than colibacillosis and is ubiquitous in faeces. Isolation of a pure growth of *E. coli* on aerobic culture of faeces may be more likely to be consistent with colibacillosis.

Salmonella infection: this is rare but reportedly on the increase. This often presents primarily as septicemia and any scour is relatively mild. Selective enrichment culture of faeces for *Salmonella* can be carried out.

Clostridial enterotoxaemia: this is often seen secondary to other diseases. It is rapidly fatal. Faeces are unlikely to be a useful sample; distal small intestinal contents from animals that die are likely to be more useful in diagnosis and can be tested using the *Clostridium perfringens* toxin ELISA test.

Johne's disease: the bovine strain of MAP is most common in camelids, but ovine strains also have been detected. Primary signs are weight loss and progressive weakness, with scour only tending to occur in the terminal stages of disease. Animals tend to succumb relatively quickly (within 2 to 3 months) once clinical signs are apparent. ELISA tests are unlikely to be effective in detecting Johne's seroconversion as they are based on anti-ruminant conjugates and AGIDT is no longer available from the APHA. PCR on faeces is the most appropriate test but variability in degree of shedding and delays in shedding by infected animals may be an issue, as in ruminant species.

Fasciolosis: camelids appear to be particularly susceptible to *Fasciola hepatica* infection, reasons suggested including a deficient immune response, their relatively small liver size and grazing close to the ground. Chronic fasciolosis has been more commonly reported in alpacas and llamas than the acute presentation, clinical signs being primarily ill thrift but also lethargy, anorexia, diarrhoea, recumbency, ascites, subcutaneous oedema, anaemia and sudden death. Fluke egg microscopy and antigen ELISA on faeces samples can be used in detection of infection.

Suggested tests in different age groups

- **< 7 days:** *Salmonella* culture, aerobic culture
- **7 - 14 days:** *Salmonella* culture, rotavirus (PAGE), cryptosporidia (MZN), *Giardia* (microscopy) (coronavirus – electron microscopy)
- **14 - 60 days:** *Salmonella* culture, rotavirus (PAGE), cryptosporidia (MZN), *Giardia* (microscopy) camelid WEC (coronavirus – electron microscopy)
- **60 days – 1 year:** *Salmonella* culture, cryptosporidia (MZN), camelid WEC (coronavirus – electron microscopy)

- **Over 1 year:** Fluke egg count (or coproantigen ELISA), camelid WEC, Johne's (PCR or ZN), *Salmonella* culture

Histopathology on cases submitted for post mortem examination may help to corroborate the results of faecal examination.

Other selected infectious diseases

Bovine Viral Diarrhoea

BVD infection was first reported in camelids in the UK in 2005. Clinical signs associated with infection have included respiratory disease, abortion, ill thrift and scour. Contact with viraemic ruminants is the most likely source of infection in camelids, but bought in viraemic camelids are also potential sources.

There appears to be three different types of infection in camelids:

1. Typical transient infection similar to that seen in cattle, with a short period of viraemia (10-14 days) and subsequent seroconversion. Signs: anorexia, lethargy, pyrexia, scour, abortion.
2. Prolonged transient infection. Viraemia may last two months or more (reportedly up to eight months) and infected animals also seroconvert. Signs: similar to PI but resolve when viraemia resolves.
3. Persistent infection – seen in foetuses infected at 64-114 days' gestation; these remain viraemic with no seroconversion. PI crias may be underweight or normal weight at birth, some have a coarse, thin fleece and some have neurological deficits. Growth may be poor, they are prone to secondary infections and two thirds die by one year of age. Some may show few clinical signs and live for much longer.

Testing:

- a) screening for evidence of exposure in a herd – BVD SNT serology (referral test) is most appropriate.
- b) finding viraemic animals – BVD PCR. If viraemic animals also seroconvert, it is more likely to be a case of prolonged transient infection than persistent infection. To confirm persistent infection, it is advisable to repeat the PCR test at least 56 days after the first positive PCR result.
- c) screening crias born into an infected herd: BVD PCR at birth and, if negative, again when 12 weeks of age (It has been reported that maternal antibody may affect PCR results in crias until 12 weeks of age).

Alpaca fever - *Streptococcus equi* subspecies *zooepidemicus*

This is an important disease of camelids in South America and it has been reported in the UK. Morbidity may be as low as 5-10% but mortality of affected animals is usually between 50 and 100%. Predisposing factors include stressors such as malnutrition and unfavourable weather. Acute and subacute infections tend to occur in young animals, with systemic infections often resulting in polyserositis and sometimes meningitis. Localised infections can also occur and chronic abscessation is more common in adults.

Testing: post mortem examination and bacteriology.

Louping Ill

Louping Ill is a neurotropic virus causing a polioencephalomyelitis of sheep, characterised by ataxia, posterior paralysis, and incoordination progressing to coma and death. It is transmitted by the sheep tick, *Ixodes ricinus* and several other species also may be infected, including camelids, with cases reported in both llamas and alpacas.

Testing: HAIT serology (referral test); post mortem examination and brain histopathology.

Bluetongue

BTV is an orbivirus transmitted by *Culicoides* midges and a number of incursions to the UK have occurred since 2007, with the virus most recently being detected in cattle and sheep imported from France. Lethal bluetongue virus infection was reported in an alpaca in Germany in 2007; signs in the affected animal before dying included acute 'hiccup-like' breathing and stertor, followed by inappetence, lethargy, coughing and disorientation.

Testing: as this disease is notifiable, if disease is suspected, APHA should be contacted immediately and they will organise testing if considered appropriate.

Tuberculosis

Mycobacterial infections other than Johne's disease have been reported in camelids in the UK; these include *M. bovis* and *M. microti*. Camelids originally were believed to be a spill over, dead end host, but there is now evidence of transfer of infection within a herd. Clinical signs may include respiratory distress, tachypnoea and/or weight loss, or even just death.

Testing: as this disease is notifiable, if disease is suspected, APHA should be contacted immediately and they will organise testing if considered appropriate.

SKIN DISEASE

Parasitic causes include mites and (uncommonly) lice.

Mite infestations include:

Chorioptes – mild pruritus, alopecia and scaling, particularly of the feet and tail base.

Psoroptes – pruritus, crusting, papules and alopecia, particularly of the pinna and ear canal.

Sarcoptes – severe pruritus, hyperaemia, papules, pustules and crusting, particularly of the limbs; may become widespread and potentially fatal if not treated.

Lice infestations include both sucking and biting lice, causing pruritus and, in heavy infestations, matted hair and alopecia.

Insect-mediated hypersensitivity has been suggested for some seasonal cases.

Bacterial causes include:

Staphylococcal infection - may be associated with pruritus, alopecia, crusting and exudation of the head and distal limbs.

Mixed infection with *Staphylococcus* and *Corynebacterium* species – ulcerative pododermatitis.

Actinobacillus – cutaneous lesions may be seen.

Other causes:

Dermatophytosis (ringworm) and dermatophilosis are considered rare in camelids. Pemphigus-like immune mediated disease has rarely been reported. Various hyperkeratotic conditions including zinc responsive disease (see earlier) may also be seen.

Testing:

- Skin microscopy –mites, fungal elements, lice
- *Dermatophilus* – selective culture
- Dermatophytes – selective culture
- Aerobic and anaerobic culture
- Skin cytology

- Histopathology on skin biopsies
- Axiom skin packages

ABORTION

Reported causes of abortion in camelids include:

Viral: BVD

Bacterial: *Chlamydophila abortus*, *Listeria monocytogenes*, *Campylobacter fetus*, *Escherichia coli*, *Trueperella pyogenes* and *Pseudomonas* species. There is circumstantial evidence also for *Leptospira* species.

Protozoal: *Neospora*, *Toxoplasma* (rare).

Testing:

Initial testing:

- Aerobic, *Salmonella*, *Campylobacter* and *Listeria* culture – foetal stomach contents (or liver)
- BVD PCR – spleen, thymus
- *Chlamydophila* MZN (or PCR) – placenta
- *Neospora* PCR – brain (placenta)

Additional testing:

- *Toxoplasma* PCR – brain, placenta
- *Leptospira* PCR – kidney
- Histopathology (+/- IHC for *Neospora*) of brain, heart, other foetal tissues and placenta

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