

JOHNE'S DISEASE (PARATUBERCULOSIS)

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WHAT IS JOHNE'S DISEASE?

Johne's disease is a worldwide problem causing clinical disease in many ruminants including goats, cattle, sheep and deer managed intensively or semi-intensively. Many wildlife species including rabbits are able to carry and thus maintain localised infections, and can also spread the organism from unit to unit. It is now recognised that there are specific cattle and sheep isolates, with most goat infections identified as cattle strains. Epidemiologically, goats will pick up infection from other goats or from cattle kept in close association (or from faecal contamination of their environment). Sheep strains have been identified in goats kept in close association with infected sheep. It is important to point out that the author has encountered problems in large commercial dairy herds housed all year – BUT ALSO in small groups of goats including Pygmy goats

WHAT IS IT CAUSED BY?

The main causative organism of Johne's disease in goats is the cattle strain now referred to as *Mycobacterium avium* subsp *paratuberculosis* (often abbreviated to MAP). Once introduced into a group of goats, uncontrolled infection progressively becomes established and can be very difficult to eradicate.

Infection is mainly transmitted to young kids <6 months old, by ingestion of the organism following faecal contamination of feed and / or water supplies and also potentially from faecal contamination of the teat and udder while suckling. Contamination of colostrum and specifically the contamination of pooled colostrum can readily transmit new infection to a wider group of susceptible kids. There is also confirmed intra-uterine infection of kids, from dams that are heavily infected in late pregnancy. Goats > 6months of age become progressively more resistant to new infection, although lateral spread even in adult goats may occur if the environment becomes heavily infected.

Although susceptible to sunlight and desiccation, MAP organisms can reportedly survive for up to one year on pasture (not forgetting potential maintenance in local wildlife).

WHAT HAPPENS NEXT?

Following infection, there is then an extended incubation period during which the infected goat will appear fit and healthy. Clinical signs may develop as early as 12 months of age, but more commonly from around 2.5 years old. Before clinical signs develop, these infected but apparently healthy goats may be shedding organisms in their faeces, thus adding to the environmental challenge. The stimulus for infection to become clinical is still poorly understood, but may be linked to stressful insults

such as kidding, transportation, poor nutrition or concurrent disease such as endoparasitism.

After infection, MAP localises in the wall of the gut and associated lymph nodes. This in turn stimulates a local immune response resulting in a progressive thickening of the gut wall and lymph node enlargement. Depending on a number of factors, a number of things can happen:

- The infection may be controlled with the goat becoming resistant to infection with no further shedding or clinical disease.
- The infection may progress to intermittent shedding and sub-clinical disease.
- The disease may become clinical with heavy shedding.

In an endemically infected herd, it is likely that all three manifestations may be apparent – thus further complicating its control. It is for these reasons that Johne's disease is referred to as an "iceberg infection" - I.e. for every goat showing clinical disease, there may be many more appearing perfectly healthy, but carrying the infection and potentially shedding it.

Serum antibody responses in infected goats prior to the development of clinical signs are poor, becoming stronger as clinical disease develops. It follows therefore that serological testing of goats using currently available tests such as the commercially available ELISA test will effectively result in "false negative" results during this long period of dormant infection – but an increasingly positive result as clinical disease develops.

WHAT ARE THE CLINICAL SIGNS?

Once triggered to develop, clinical signs may go unnoticed initially, but include a progressive weight loss and reduction in milk yield if lactating, although appetite may be unaffected. As the condition develops, anaemia and a lack-lustre coat may become apparent together with sub-mandibular oedema (a feature of the progressive protein loss that occurs). The diarrhoea associated with disease in cattle is not a feature of the condition in goats (although it may develop in the terminal stages).

WHAT OTHER INFECTIONS PRODUCE THESE SAME CLINICAL SIGNS?

Differential diagnoses for the combined signs of weight loss, reduced milk yield, and anaemia in the absence of diarrhoea include the parasites fluke and haemonchosis.

DIAGNOSIS ?

There is no single reliable test that can be used to confirm infection due to the complexity of the immune response, and the long period of latent infection in clinically healthy goats.

Live clinically healthy goats - most available tests are ineffective during this phase of disease, there will be little if any antibody response detectable, and the sporadic shedding of organisms in faeces may be below detectable limits.

Live infected goats in early clinical phase – antibody levels begin to rise, and may be detectable by e.g. an ELISA test, but false negative results may be widespread during this phase. Goats may give a positive result on one day, then a negative result when retested – these goats will invariably retest as positive in time. Faecal examination using a variety of tests will progressively become more reliable as clinical signs progress.

Live clinically infected goats – all available tests become more reliable, as the antibody response becomes stronger and faecal shedding increases.

At post mortem examination, the gut wall thickening evident in the lower jejunum and ileum is more subtle than that seen in cattle in which “corrugation” and apparent folding of the mucosal surface is evident. Gut lymph nodes may be grossly enlarged and oedematous, with caseation and even calcification - a feature of later stage infections. These lymph node changes are similar to those seen in TB – which must be considered.

Bulk milk monitoring is becoming more widely available in cattle, and may in time be available when validated for milking goat herds.

MANAGEMENT/TREATMENT / CONTROL?

Effective control is difficult to achieve, because of the long incubation period during which goats are clinically healthy, shedding by these infected goats, and the low sensitivity of the currently available tests.

Eradication may be a long term aim, but will need considerable commitment by the herd owner and attending veterinarian. Such an approach would be based predominantly on regular whole herd testing using available serological tests supplemented by faecal monitoring – and culling those goats giving a positive result. This may need to be continued for a number of years to be successful, if two consecutive negative clear tests are the ultimate aim. An alternative to culling is to separate positives and negatives into a “clean” and “dirty” herd. Health Schemes aimed at Johne’s Control are available.

Such a control programme must be supported by:

- Prompt identification and removal of clinical cases.
- Minimising faecal contamination of feed and bedding – particularly for young kids.
- Snatching kids and rearing away from adults, feeding colostrum from “known negative” dams, or using artificial colostrum.
- Avoiding the use of pooled colostrum
- Culling kids born to does developing disease in late pregnancy (due to risk of in-utero infection).

Control can also be achieved in endemically infected herds by the use of vaccination, widely available and licensed for use in goats in many countries. It should be used following manufacturers recommendations in young kids.

Vaccination will result in far fewer clinical cases, increased productivity – but will not eradicate infection – effectively allowing a unit to continue in production. Vaccination may interfere with any tuberculosis control programme based on intradermal testing.

There is no known treatment for Johne's disease