# BGS Disease Leaflet

## CAE (Caprine Arthritis Encephalitis)

Caprine Arthritis Encephalitis (CAE) is a viral disease of goats. The CAE virus belongs to the same family of viruses as that which causes Maedi Visna (MV) in sheep, and they are collectively termed small ruminant lentivirus. Cross-species infection is possible with either of these viruses.

### Clinical Signs

Many goats are asymptomatic carriers of the virus. This may be the case for months or years before clinical disease presents itself.

CAE has five varied clinical presentations:

Arthritis

Arthritis is seen more commonly in goats over one year old, and most commonly affects the carpal (knee) joints, but other joints may also be affected in the later stages of the disease. The affected joints will usually be enlarged but may not always be painful. Its development may be rapid and sudden or occur more slowly, ranging from slight lameness to restricted movement. Other signs that may occur along with arthritis include varying degrees of lameness, and so increased laying time and an unwillingness to move, reduced appetite, and reduced milk production in lactating dairy goats.

Mastitis (Hard Udder)

The form of mastitis typical of CAE is also known as 'hard udder' and is distinct from other forms of mastitis. The affected half of the udder becomes progressively firm and shrinks, gradually reducing milk production as this occurs. The milk appears normal, with no indications of bacterial mastitis.

Interstitial pneumonitis

Interstitial pneumonitis is a progressive condition in which the lungs become scarred. It develops slowly, and may initially be mild, with animals showing only exercise intolerance and a dry cough. As the disease progresses, goats will show chronic difficulty breathing and weight loss. Interstitial pneumonitis is often seen with other forms of CAE.

Encephalitis

Encephalitis (inflammation of the brain) is rarely seen in CAE in the UK. When it is seen, it mainly affects young kids from 1-6 months of age but can also be seen in adults. The clinical signs vary depending on the age of the goat affected. The most common signs seen in kids are tremors, lameness, and lack of coordination, progressing to include other central nervous system signs such as, blindness, loss of use of some or all the limbs, and circling. In adults, neurological signs often appear after other presentations of CAE. Without the presence of arthritis, these signs may resemble listeriosis. Signs include knuckling of fetlocks and circling, with weakness and paralysis in the later stages.

Progressive weight loss

Progressive weight loss is often seen in CAE. It may be in conjunction with other signs, but can occur independently.

### Transmission

Transmission of CAE predominantly occurs through feeding infected colostrum or milk to kids. Many other important routes of infection exist, however, such as direct contact between individuals, and sharing equipment such as needles, tattooing equipment, or drenching guns. Transmission to kids in-utero is likely to occur but at a low level. Embryo transfer and artificial insemination are thought to be low risk procedures for transferring infection, but precautions should be taken to minimise this risk. Once infected, goats remain carriers for life, many without any signs of disease, meaning disease spread can occur without the owner's knowledge of the presence of CAE.

### Testing

Testing forms an important part of wider control measures for CAE prevention. A positive serology result indicates an infected goat that is able to shed the virus. A negative result either means that the goat is not infected or has

been infected too recently for antibody levels to be detectable. Kids under 6 months of age are not routinely tested, as they have not had time to develop antibodies.
Goats infected with CAE virus can be identified by blood testing before they show any clinical signs of disease. Some animals are infected and able to shed the virus for years before visible signs develop, capable of infecting others throughout this asymptomatic period. This highlights the importance of routine testing in all herds, not just those with animals showing signs of disease.

### Prevention

No treatment or commercial vaccine exists for CAE, and at present, it is contained at low levels due to routine control measures. These consist of routine testing of every animal over 12 months of age, every 6-12 months. Any sheep in contact with the goats should also be tested at routine tests. If there have been no indications of infection for 5 years or more, then a herd is 'CAE free'. No unpasteurised milk or colostrum should be fed to kids, other than from their dam. Pooled milk should not be fed to kids, as if one doe is shedding virus, she is able to infect a whole group of kids. All adult animals, or the dam of kids, should have a negative blood test before entering the herd. No milk from any other herd should be fed to kids.

## Johne's Disease

Johne's disease is caused by a bacterium, *Mycobacterium avium* subspecies *paratuberculosis*. Goats can be infected by the cattle and sheep strains, but the cattle strain is much more serious in goats. Once infection is established in a herd, it is difficult to eradicate. Johne's is a major cause of death or culling of adult goats in commercial herds.

### Clinical Signs

Johne's has an extended incubation period. Signs can develop from 12 months of age, but more commonly from 2.5 years. During the incubation period infected goats will appear healthy, but will be shedding bacteria in their faeces, adding to the contamination of the environment. Clinical signs may be subtle initially and can include progressive weight loss and a reduction in milk yield.
Depending on a number of host-pathogen factors, infection with the *M. avium* subsp. *paratuberculosis* may progress in a number of ways. The infection may be controlled if the goat becomes resistant and stops shedding bacteria. Alternatively, the infection may progress to become subclinical with intermittent shedding of bacteria, or become clinical with visible signs and heavy shedding of bacteria. In an infected herd it is likely that all three stages are present at any one time, complicating control measures.
As the infection develops to become clinical, common signs include anaemia, a lacklustre coat, and submandibular oedema, due to low blood protein. Diarrhoea is not a major feature of Johne's disease in goats until terminal stages, unlike in cattle.

### Transmission

Transmission of Johne's occurs mostly by the faecal-oral route. Goats may become infected directly, from other goats, cattle, and more rarely sheep, kept in close association to them. The bacteria are very persistent in the environment, so transmission often occurs indirectly, via faecal contamination of their environment. This may occur by ingestion of faecally contaminated feed or water supplies, from contaminated teats or udder while kids are sucking, or from contaminated pooled colostrum. Intrauterine infection of unborn kids is possible in dams that are heavily infected in late pregnancy.
Johne's disease is mainly transmitted to young kids, below 6 months of age. Goats older than 6 months become progressively more resistant to new infection, although transmission may occur between these individuals if the environment becomes heavily contaminated. Wildlife, such as birds and rabbits, may act as a reservoir of the bacteria.

### Testing

No single reliable test exists that is able to identify all animals infected with Johne's disease, due to the complex development and latent period of the disease. For this reason, Johne's disease is likely underdiagnosed.

Some tests that can be performed to detect Johne's disease include:

- **Post-mortem examination**: Looking for the bacteria which can be found in the lymph nodes of the lower bowel.
- **Faecal sampling:** Looking for the bacteria which can be found in faeces. In clinical healthy animals, however, shedding of the bacteria in faeces is sporadic and may be below detectable limits. For this reason, some true positive cases may be missed. Despite this, faecal sampling remains the most reliable test in live animals.
- **Serum antibody testing:** Looking for the evidence of an immune response to the presence of bacteria. Before development of clinical signs, goats show a poor antibody response, so false negatives are common in clinically healthy animals. A test for serum antibodies is more likely to be positive as the disease advances.
- **Biopsy of lymph nodes:** Looking at tissue from the lymph nodes in the gut to identify presence of bacteria. This is an invasive procedure, and so only a viable option for high value or pet goats.

### Prevention & Control

Most of the risk of introducing Johne's disease into a herd comes from the movement of new livestock into the herd. This risk should be considered when buying in new animals.

There is no known treatment for Johne's disease, meaning bringing it under control is difficult. Once it has established itself in a herd, the aim is generally to reduce the spread within the herd, rather than to aim to eradicate it. Complete eradication of Johne's disease from a herd is difficult and involves several years of commitment by the owner and their vet. Control strategies are primarily based on whole herd testing, with culling of positive animals, as well as culling of any animals showing clinical signs; minimising contamination of feed and bedding with faeces, especially for young kids; snatching kids at birth or removing them from their dams as soon as possible after birth; and avoiding the use of pooled colostrum, only feeding colostrum from dams who have tested negative, possibly combined with pasteurisation.

There is a vaccine for Johne's disease available and licensed for use in goats in the UK. Vaccination can be used as part of a control plan in infected herds, to bring the disease under control by reducing the number of cases which go on to show clinical signs. Vaccination won't eradicate infection, but it will allow an infected herd to continue productively.

## Scrapie

Scrapie is a transmissible spongiform encephalopathy (TSE) of sheep and goats. It is a fatal degenerative disease of the central nervous system, caused by the expression of an abnormal form of a naturally occurring protein, which accumulates in the nervous and lymphoid tissues (liver, spleen, lymph nodes). Scrapie is a notifiable disease in the UK, meaning that if you or your vet suspect it, it must be reported to the Animal and Plant Health Agency (APHA), who will then investigate further.

### Clinical Signs

Scrapie has a long incubation period, so clinical signs are rarely seen in animals younger than 2 years old. Once present, signs will progress very slowly over several weeks, and result in death within weeks or months.
Two forms of Scrapie with different clinical signs are observed in goats, but there is a big overlap between the two, with many animals showing signs associated with both types. The clinical signs are split into those related to the nervous system, and pruritic (itching) signs.
Initially, signs may be very non-specific, and, therefore, easily overlooked. These include behavioural changes, increased irritability, increased excitability, lethargy, weight loss without a decreased appetite. In some animals, these signs may not progress further for a long period. Most progress to show more obvious signs. Neurological signs seen in Scrapie include a progressive lack of coordination; difficulty placing legs and stumbling whilst walking; changes on posture or gait; changes in behaviour; increased sensitivity to pain; tremors; pricked ears and holding the tail up and over the back; and excessive salivation.
Pruritic signs are thought to be rarer in goats compared to sheep, where it is a common presentation. These signs include scratching using the hind feet; rubbing of their back, poll and withers; nibbling of sides, abdomen or udder; and biting of limbs.

### Transmission

The main source of new infections is likely to be the placenta and uterine fluids of infected animals. The abnormal infectious protein is known to be present at high levels in these tissues, so ingestion of these by kids or other goats will result in infection. The infective protein is very resistant in the environment, including on pasture, so infection from contamination of an animals' environment is possible.

### Testing

In live animals, diagnosis of Scrapie is based on clinical signs. At postmortem, it can be definitively diagnosed by looking at the brain tissue for the sponge-like appearance, and analysing it for presence of the infectious protein itself. Several tests are being used experimentally to try to diagnose Scrapie in live animals, such as biopsy of the rectal lymphoid tissue.

### Prevention

No treatment currently exists for Scrapie, meaning the only method of control involves culling of any goats showing clinical signs, and their offspring. Other measures exist that can be put into place to limit the amount of disease spread in a herd, such as thorough disinfection of buildings in which infected goats have been housed, especially after kidding. Genetic selection for Scrapie resistance is also a possible control strategy, and has proved useful in sheep, where it is better understood compared to in goats. Research into the genetics of Scrapie resistance in goats is ongoing, as it appears to be quite different to that of sheep.

## CLA (Caseous Lymphadenitis)

Caseous lymphadenitis (CLA) is a common disease in the UK. It is a chronic bacterial disease, primarily of sheep and goats, caused by *Corynebacterium pseudotuberculosis*.

### Clinical Signs

CLA causes lymph nodes to enlarge, as they form abscesses, particularly the parotid (on the side of the face, below the ear), the submandibular (behind lower jaw), and prescapular (at the base of the neck, in front of the shoulder) lymph nodes. Occasionally other lymph nodes are affected depending on where the bacteria entered the body. These abscesses can rupture spontaneously, drain the creamy white or yellow pus, heal, and appear to resolve. The goat, however, is unlikely to ever be free of infection, which may flare up at any time. In some cases, internal abscesses form, and so clinical signs will vary depending on the site of these. The incubation period between infection and superficial lymph node abscesses is typically 2-3 months, although it can be up to 6 months. In many animals, CLA has only minimal or no impact on their overall health or productivity, because it is often only the superficial lymph nodes that are affected.

### Transmission

The main route of transmission between animals is through contamination of open wounds with the bacteria. The bacteria are discharged from abscesses and contaminate the environment. They are able to survive there for several months. Flies are able to spread infection by transporting the bacteria from a ruptured abscess or contaminated area to an existing open wound on an uninfected animal. The infection may also be spread by inhalation in the case of particularly heavily infected animals, and kids may become infected if the bacteria is present in milk they are being fed. Spread of infection between herds may occur by buying in infected animals, or through shared equipment or facilities.

### Testing

Diagnosis of CLA is based on clinical signs, looking for the presence of abscesses at the superficial lymph nodes, and culture of the contents of the abscesses to look for presence of bacteria. Serum tests, looking for evidence of an immune response and, therefore, exposure to the bacteria can be used in management of outbreaks, but are likely to give some false negative results for those animals in the early stages of infection.

## Prevention and Control

Due to the persistent nature of the infection, control of CLA is based on identifying the infected goats, and culling these animals, which seems to be a successful approach.

To minimise the potential for disease spread within a herd, care should be taken to stop the abscesses bursting naturally, and, therefore, avoid contamination of the environment with pus. Removing or surgically draining the superficial lymph nodes will remove the visible abscessation but will not prevent abscesses appearing elsewhere in the body.

Vaccines for CLA are available, but not in the UK, and do not serve to eradicate the disease from the herd, only to reduce the level of abscesses, and so reduce the chance of further disease spread.

In order to prevent new infections arising, it is advisable to remove or minimise anything in the goats' environment that could potentially cause open wounds, like exposed nails or barbed wire. Control of ectoparasites, like flies, lice, and mites, serves to prevent infection, as these make the goats less likely to rub on things, break the skin and enable bacteria to enter. Thorough and regular disinfection of any facilities or equipment shared between herds or 'clean' and 'dirty' groups within a herd, is essential to prevent spread of any disease, especially where known infection is present.

Quarantine of any new animals entering the herd is key, especially monitoring any incoming goats for signs of enlarged lymph nodes during this time. An effective quarantine period for CLA is two to three months, which may not be feasible. Incoming animals could also be serologically tested, but this has a chance of giving a false negative result. To pose the lowest possible risk of introducing CLA to a herd, new goats should be purchased from herds with no history of CLA.

# Sources

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