CAPRINE ARTHRITIS ENCEPHALITIS (CAE)

David Harwood BVetMed, FRCVS
Hon Vet British Goat Society
Senior Vice President Goat Vet Society.

This article taken from Chapter 13
"The Veterinary Guide to Goat Health and Welfare"
David Harwood (Crowood Press).

Cause:

A lentivirus in the family Retroviridae, a genus causing a group of slowly developing insidious conditions such as CAE and Maedi Visna (MV) of sheep, the two diseases often referred to collectively as SLRV (small ruminant lentivirus). Although the two viruses cause different clinical presentations in their respective host species, cross species infection can occur, and is an important factor in CAE control programmes, that have been developed on a compulsive / voluntary basis in many countries around the world.

Spread of infection

An important part of any disease control or preventative programme, is an understanding of how the infectious agent is picked up and spread within a population. This knowledge is vital if this condition is to be contained.

CAE is transmitted predominantly via the ingestion of infected colostrum or milk from infected does. There are however many other important routes of infection such as via nose to nose contact and aerosol transfer, via "infected milk impacts" at the teat end in the milking parlour, or through shared use of equipment such as drenching guns or tattooing equipment. Transplacental infection can occur but is thought to be at low levels, and although both embryo transfer and artificial insemination may pose only a minimal risk of transferring infection, both practices need to be risk managed if eradication is being attempted. Increased infection rates will occur in housed and intensively managed goats because of close contact. The practice of feeding pooled colostrum and milk in dairy herds can also lead to increased infection rates as one infected doe can infect the pool, and hence potentially many kids – and this is also true for Johne's disease.

The development of any clinical disease after infection will be slow and protracted, and as such is associated with a high prevalence of latent inapparent infection, in which goats remain fit, healthy and productive. The incubation period between infection and the development of clinical signs can be very variable. Infection induces a strong humoral response, but the antibody produced is not protective – and an infected goat is essentially both virus and antibody positive – and it is the antibody test that forms the basis of any testing / monitoring programme. The colostrum from infected dams will contain both virus and antibody, the latter affording no maternal protection – **thus unlike other infections – colostrum can actually be the cause of a new infection in the kid, rather than its prevention.**

Signs:

CAE infection results in a range of clinical signs, some of which have already been discussed. The main clinical presentation is lameness linked to arthritic change in the joints. Other signs include encephalitis in kids (Chapter 12), lung infection (Chapter 10), mastitis and udder change – udder shrinks and becomes more firm in consistency (Chapter 16) or non-specific weight loss.

Arthritis is seen in goats of usually six months of age and older. It tends to be chronic and develops slowly, with the carpal (knee) joints most commonly affected, followed by, in descending order, the tarsal (hock), stifle, fetlock, neck and hip joints. One joint may be affected, or several joints, and although joint involvement may be acutely painful in one goat, another goat may show very ill-defined signs of mild discomfort including stiffness, difficulty getting up or reluctance to move. The joint may be visibly swollen or show only mild change externally.

Infection can lie dormant in a group of goats for many years before clinical disease develops, and the full spectrum of clinical signs already described may not be seen until a high proportion of goats is infected. If you suspect disease, contact your veterinary surgeon and arrange for laboratory tests to be undertaken.

Treatment:

There is no treatment, and currently no vaccine available to prevent disease.

Control:

This is based on a full knowledge and understanding of the way infection is picked up and spread within a population of goats and will also depend on the level of infection in your own herd. Once infected with CAE, a goat remains infected throughout the remainder of its life – not all goats will develop disease during their lifetime, and furthermore many goats can be infected with virus but be unrecognized by the owner, as they remain fit and healthy!

The first step in control must be to evaluate the CAE status of any herd under investigation, and to also ask the question as to what your aspirations are as an owner. If infection is confirmed in a commercial herd, then a test and cull programme may be an economically sound decision. If on the other hand, the infected goats are essentially pets – then a programme devised at living with the problem is more likely to be an option. The CAE herd status can then be broken down into three main categories:

1. Free of infection – i.e. naïve

2. Low/moderate level of infection – no or minimal clinical disease apparent, but blood test positive goats identified.

3. Heavily infected – increasing number and spectrum of clinical cases being identified.

If the herd is truly naïve, the control is based predominantly on keeping infection out by a programme of testing incoming animals, also by ensuring that adequate quarantine measures are in place, and that all other biosecurity measures referred to in this book are adhered to.

Infected herds (low, moderate or heavy): Control measures should be drawn up depending on the level of infection, the type and size of the unit, the available finances etc.. Each unit is different, and options may vary from doing nothing and living with the problem, to developing a ruthless test and cull policy. Measures to consider include:

- A whole herd bleed, thus having a population of goats of known status (within the limitations of the tests available, and the number of tests undertaken), as a starting point for devising your next steps.

- More limited and targeted testing of, for example, breeding females, yearlings due to breed or any goats showing the spectrum of clinical signs described..

- A culling policy (dependent on the results of blood testing), depending on what the unit can tolerate with regard to finance, production requirements and sentiment.

- If the prevalence of disease is high in a herd and culling of the seropositive animals is not possible (or the affected goats are not being kept commercially), the herd can be separated into two groups (one “dirty” containing seropositive animals and one “semi-clean” containing seronegative animals). Those that have tested as seronegative cannot be guaranteed to be uninfected as some could be in the early stages of infection and not yet positive in the blood test. The “semi- clean” group should therefore be tested on a six to twelve monthly basis and any seropositive animals moved to the “dirty” group. Any animals showing clinical signs of CAE should be culled as these animals are likely to be excreting high levels of virus.

- Snatching kids at birth and rearing them away from the dam environment.

- Avoiding the use of pooled colostrum if there is evidence of CAE in the herd, and using either colostrum supplements, colostrum from known negative donors, feeding cow or sheep colostrum, or feeding pasteurized colostrum. Heat-treated or ‘pasteurized’ colostrum has been held at 56°C for one hour. This time or temperature must not be exceeded, or the colostral antibody protein could be destroyed.