### CAE: CAPRINE ARTHRITIS ENCEPHALITIS

In 1974, a scientific report described a previously unreported nervous disease of goats which was believed to be caused by a virus. This was followed by a report of an arthritic disease in goats presumably caused by the same agent. Shortly afterward the viral cause of these conditions was confirmed and the virus characterized. The development of a diagnostic serologic test was soon reported along with the observation that a high percentage of goats in the United States appeared to have experienced the virus. Investigation into the transmission of the disease suggested that virus was spread through colostrum and milk of infected dams and that virus free herds might be maintained by raising kids in isolation.

## Nomenclature

The nervous disease first reported in 1974, was named Viral Leukoencephalomyelitis of Goats (VLG). When it became apparent that arthritis could also result from the same virus infection, the name of the disease was changed to Caprine Arthritis Encephalitis Syndrome (CAE). It is now apparent that the virus also produces changes in the lung and udder. The name CAE however still remains in place.

## The Causative Virus

Knowledge of how a disease is transmitted is often the key to developing a successful program for preventing the spread of infection. To date, all published reports suggest that goats become infected with CAE virus as newborn kids. Experimental evidence for this is persuasive. Kids delivered either naturally or taken by cesarean-section, but deprived of colostrum and fed cow milk remain free of the virus despite the fact that their dams are infected. If taken by cesarean-section or delivered naturally, but allowed to nurse colostrum or milk from an infected doe, kids will show evidence of virus infection. These findings indicate that kids are not infected in utero or during passage through the birth canal; but do pick up infection when nursing colostrum or milk from infected dams. This suggests that control of the spread of new infections might be achieved by separation and artificial rearing of kids at birth.

### Transmission of CAE Virus

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## Prevalence of CAE

One aspect of the CAE syndrome which has proven most troubling to the US goat industry was a published report that a high percentage of goats tested from all over the United States showed serological evidence of infection with CAE virus. Of 1160 goats tested from 24 states, 81 showed antibody to CAE virus using the agar gel immunodiffusion (AGID) test. It can be assumed that animals with antibody to CAE virus have been exposed to and infected by the virus. The only exception to this could be young kids with detectable antibody picked up from the dam's co lostrum. Unlike most bacterial diseases, where a strong antibody response means that the animal has cleared itself of the invading organism, infections with CAE and other retroviruses are likely to persist in the animal despite a high antibody titer. Therefore, it is probably true that a large percentage of antibody positive goats carry persistent infections. However, it does not necessarily follow that the majority of these goats are likely to show clinical signs of the CAE syndrome. The factors which contribute to the onset of clinical signs in animals infected with the virus are unknown.

The major problem associated with this high prevalence of infected US goats is not the actual incidence of clinical disease so much as the negative perception of prospective goat buyers and regulatory officials confronted with a positive AGID test. Already some countries importing US goats, like Kenya, have refused or destroyed shipments of goats which turned out to be antibody positive. Economic restraints such as this increase pressure on the goat industry to aggressively tackle the CAE problem.

### The Clinical Signs of CAE

Two separate distinct syndromes are caused by the CAE virus, a neurological disease in the spinal cord and brain of young kids and a joint infection of older goats resulting in arthritis. How individual animals infected with CAE virus escaped one or the other or both syndromes remains a mystery. The clinical signs of the two syndromes are as follows.

### The Nervous Form of CAE

The nervous form of CAE was the first to be described. All breeds of goats can be affected as can both sexes, and most individuals first show signs between one and four months of age. The problem is one of progressive weakness (paresis) of the hind limbs leading to eventual paralysis. The early paresis may be perceived as lameness, incoordination or weakness in one or both rear legs. Knuckling over of the feet and difficulty in rising may follow until such time that the animal is unable to rise at all. The course of the disease is from several days to several weeks. Despite the progressive paralysis, the kid will usually remain bright and alert and continue to eat and drink. Mild pneumonia may be present. If the correct diagnosis is made, the animal is often euthanized since there is no known treatment for the condition.

The development of these signs results from inflammation in the spinal cord induced by the virus. Nerves which control motor function of the hind limbs are progressively destroyed. In spite of the ongoing inflammation, there is little or no change observed in the cerebrospinal fluid on CSF tap nor in the complete blood count (CBC). Diagnosis is based on recognition of the clinical signs and confirmation depends on observation of the characteristic changes seen microscopically in the spinal cord at the time of postmortem examination.

In older goats, a clinical variation of the nervous form of CAE has been observed which is clinically indistinguishable from Listeriosis. Signs include circling, head tilt and facial nerve paralysis. On postmortem examination, the characteristic lesions of CAE virus are found in the brain stem rather than the cervical spinal cord.

# The Arthritic Form of CAE

The joint form of CAE most often appears clinically between one and two years of age. There can be great variability in the progression and severity of signs. Some goats can be severely crippled within a few months while others may show only intermittent lameness or stiffness for years without ever becoming completely debilitated. A "typical" case would fall somewhere in between. The disease is usually first recognized as a gradually developing lameness accompanied or followed by swelling of the joints. Swelling is most often noted in the front knees (carpi) and can also be seen in the hock and stifle joints. As the condition progresses, joint pain and stiffness become more apparent. The animal may spend a good deal of time lying down, will begin to lose weight and develop a rough hair coat. In severely affected joints, the range of motion may become limited and goats are forced to walk around on their carpi. No specific cure is known for CAE arthritis. The well-being of affected goats may be improved by proper foot trimming, extra bedding and administration of anti-inflammatory drugs such as aspirin.

As in the nervous form, the complete blood count in goats with CAE arthritis will most likely be normal. Fluid taken from affected joints, however, may show changes suggestive of CAE. These include a reddish brown discoloration, increased volume, low viscosity and an increase in mononuclear cells. All joint fluid aspirates should be cultured for bacteria, chlamydia and mycoplasma since these organisms can also cause arthritis in goats. In addition to these infectious causes, traumatic injury and poor conformation can also lead to joint problems. Keep in mind, that not all swollen joints or stiff limbs are CAE arthritis.