

# Akabane Disease

*Congenital Arthrogryposis–Hydranencephaly Syndrome, A–H Syndrome, Akabane Disease, Congenital Bovine Epizootic A–H Syndrome, Acorn Calves, Silly Calves, Curly Lamb Disease, Curly Calf Disease, Dummy Calf Disease, Bovine Epizootic Encephalomyelitis*

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## Importance

Akabane disease is an arthropod-borne viral disease that can affect cattle, sheep and goats. In the predominant syndrome, inapparent infections in adults can lead months later to abortions, stillbirths and congenital defects in newborns. Most affected neonates die or must be euthanized. Before vaccines were developed, Akabane disease caused significant economic losses in some countries. Between 1972 and 1975, this virus resulted in the birth of more than 42,000 abnormal calves in Japan. A few strains of Akabane virus can also cause outbreaks of encephalomyelitis in calves and adult cattle. In the past 20 years, these strains have become endemic in Japan and Korea, where they periodically cause outbreaks affecting anywhere from a few to hundreds of animals. There is no treatment for Akabane disease.

## Etiology

Akabane virus is an arbovirus in the genus *Orthobunyavirus* of the family Bunyaviridae. Some closely related, named viruses including Tinaroo and Sabo virus are now considered to be strains or isolates of Akabane virus. Serologically, Akabane virus belongs to the Simbu serogroup of the Bunyaviridae. This serogroup contains some other viruses that are also teratogenic in ruminants, but are considered to be distinct viral species, such as Aino virus and Schmallenberg virus, as well as some viruses that seem to be nonpathogenic. A potential complication in distinguishing Simbu serogroup viruses is that its members, including Akabane virus, can exchange gene segments (reassort) with each other.

Strains of Akabane virus can differ considerably in virulence. Although most of these viruses only affect unborn ruminants, a few variants, like the Iriki strain, can cause neurological signs in postnatal cattle. This syndrome has been called bovine epizootic encephalomyelitis.

## Species Affected

Akabane virus affects cattle, sheep and goats. One report suggests that this virus can also cause clinical signs in pigs. Antibodies to Akabane virus have been found in horses, donkeys, buffalo, deer, camels, and wild boar, but there are currently no descriptions of Akabane-associated illnesses in these species. Serological evidence of infection has also been reported in wild ruminants and other ungulates. Some wildlife that have a high seroprevalence rate (e.g., saiga antelope, *Saiga tatarica tatarica*, in Kazakhstan) have been suggested as potential reservoir hosts. There have been no reports of disease in wild ruminants; however, clinical cases could occur infrequently and be missed if animals are not usually infected for the first time during pregnancy. Mice and hamsters can be infected experimentally.

## Zoonotic potential

There are no indications that humans are susceptible to Akabane virus.

## Geographic Distribution

Akabane virus is common in many tropical and subtropical areas in the Eastern Hemisphere, including parts of Asia, Africa, the Middle East and Australia. However, outbreaks tend to occur mainly in the far northern and far southern ranges of its distribution. Some countries that have reported Akabane disease include Japan, Korea, Taiwan, Australia, Israel and Turkey. In Australia, the virus is endemic in the northern half of the country, but occasional outbreaks occur in southern Australia when conditions are favorable for it to spread. The viral strains associated with bovine epizootic encephalomyelitis (postnatal encephalomyelitis) have, to date, been reported in Japan, Chinese Taipei (Taiwan) and Korea.

## Transmission

Akabane virus is transmitted primarily by biting midges (gnats) in the genus *Culicoides*. The specific vectors differ between regions; some of these species include *Culicoides oxystoma* in Japan, *C. brevitarsis* in Australia, and *C. milnei* and *C. imicola* in Africa. Additional *Culicoides* species have been infected in the laboratory, including



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*C. variipennis*, which is common in North America. Akabane virus has been detected in various mosquitoes, such as *Aedes vexans*, *Culex tritaeniorhynchus*, *Anopheles funestu* and *Anopheles vagus*; however, their role in transmission (if any) is thought to be minor.

Vertical transmission is important in the epidemiology of this disease. In animals that are not immune to Akabane virus, it is transmitted across the placenta to the fetus, and causes congenital defects. This virus does not appear to be contagious by casual contact; horizontal transmission has only been reported via insect vectors. It has not been found in the semen of bulls. Ruminants do not become long-term carriers of Akabane virus.

## Disinfection

Akabane virus is not transmitted between animals by direct contact. If disinfection is necessary, some agents listed for other Bunyaviridae include hypochlorite, glutaraldehyde, 70% alcohol, hydrogen peroxide, peracetic acid and iodophors. Bunyaviridae are also sensitive to heat and UV light.

## Incubation Period

Akabane virus infections are asymptomatic in most adult animals, but viremia usually occurs 1 to 6 days after infection, and Akabane virus is transmitted across the placenta to the fetus. Fetal infections do not become evident until the animal is either born or aborted due to severe defects.

## Clinical Signs

Most strains of Akabane virus infect non-pregnant animals subclinically, but a few can cause encephalomyelitis in calves and adult cattle. Neurological signs that have been reported in these animals include tremors, ataxia, lameness, paralysis, nystagmus, opisthotonus and hypersensitivity. Although some individual animals have been febrile, fever was absent in most cases with CNS signs.

More often, Akabane disease is characterized by asymptomatic infections in postnatal animals, and abortions, stillbirths, premature births and congenital defects (arthrogryposis and congenital malformations of the brain) in fetuses and newborns. Birth complications may cause injuries to the dam that result in infertility or death, particularly when the fetus has malformed joints.

Because Akabane virus has different effects at each stage of gestation, an ordered sequence of events tends to be seen. This is particularly evident in cattle, which have a longer gestation period than small ruminants. In cattle, abortions, stillbirths and premature births may be the first sign of an Akabane outbreak. Aborted fetuses can appear normal on first examination, but fixation of the joints may be detected on careful examination, and severe hydranencephaly may be found if the skull is opened. Some strains of Akabane virus can cause nonsuppurative encephalomyelitis in calves that were infected late in

gestation. These calves may have a variety of neurological signs including flaccid paralysis, exaggerated movements and hyperexcitability. Many cannot stand, and those that can rise with assistance are ataxic. The next calves to be born, which were infected during an earlier stage of their gestation, usually have arthrogryposis at birth. One or more joints are rigid and fixed in flexion (or, less often, in extension), and the associated muscles are often atrophied. The first calves tend to have less severe defects than calves born later. Torticollis, scoliosis, kyphosis and spina bifida may also be seen occasionally. Affected calves born late in the outbreak, which were affected during an early stage of gestation, have congenital lesions in the brain ranging from small cavitations to severe hydranencephaly. Although these animals can usually stand and walk, they have behavioral abnormalities. Many are blind, depressed or dull, deaf and unaware of their environment; they may wander aimlessly. The suckle reflex can be slow or absent. Other neurological signs can also be seen, and the gestation is often extended. A few calves may have both arthrogryposis and CNS defects. Most affected neonates die or must be euthanized soon after being born.

The range of fetal and neonatal defects seen in sheep and goats is similar, but there is more overlap. Arthrogryposis and CNS lesions are seen at the same time during the outbreak, and often occur in the same animals. Additional defects including pulmonary hypoplasia have been reported in small ruminants.

The abstract of a recent article in a Japanese veterinary journal describes isolating Akabane virus from a suckling pig with neurological signs, during an outbreak in cattle. It also indicates that this virus caused "abnormal deliveries (malformed fetuses)" on three swine farms during this outbreak. In a previous report from Japan, both Akabane virus and porcine teschovirus were recovered from three 14-week-old pigs with convulsions and diarrhea. Experimental infection of one-month-old pigs with this isolate did not result in clinical signs or gross lesions, although some inoculated pigs had microscopic lesions of mild nonsuppurative encephalitis and vasculitis.

## Post Mortem Lesions

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Fetuses and newborns may have arthrogryposis, hydranencephaly or both syndromes. In animals with arthrogryposis, one or more joints are affected in one or multiple legs. These joints are fixed by abnormalities in the soft tissues, and cannot be straightened. However, if the tendons are cut, the joints may move freely. The muscles may appear fibrotic and gray. CNS lesions can include hydranencephaly (thinning or disintegration of the cerebral cortex), hydrocephalus, agenesis of the brain, microencephaly, porencephaly (small cystic defects) and cavitations. The cerebellum and brainstem do not usually have gross lesions even when the cerebral hemispheres are absent. Torticollis, scoliosis, kyphosis, spina bifida and brachyggnathism can sometimes be seen, especially in lambs and kids. Hypoplasia of the lungs, thymus and spinal cord may also be observed in small ruminants. Cataracts and

ophthalmia have been reported. Aborted or stillborn fetuses may appear to be normal until they are examined carefully.

Calves infected late in gestation, or calves and adult cattle infected postnatally by certain strains, can have lymphohistiocytic encephalomyelitis. Marked gross lesions are absent in the brains of these animals. Nonsuppurative lymphohistiocytic encephalomyelitis is found on histological examination. Although lesions can also be found elsewhere, they are most common in the pons and medulla oblongata, and the ventral horn gray matter of the spinal cord.

## Diagnostic Tests

Congenital Akabane disease is often diagnosed by serology, using serum samples or body fluids (pericardial, pleural or peritoneal) from the fetus or presuckle neonate. Most affected fetuses and full term calves have mounted an antibody response to this virus, although fetuses that were infected before they became immunocompetent may be seronegative. Serum samples from the dam are most useful in areas where Akabane virus is not endemic. Where the virus does circulate, a seronegative dam rules out Akabane infection in the fetus, but an antibody titer is inconclusive, as it could have been acquired before the pregnancy. Serology may be useful occasionally in cases of bovine epizootic (postnatal) encephalomyelitis. Virus neutralization and enzyme-linked immunosorbent assays (ELISAs) are the most commonly used serological tests. Other assays that have been employed include agar gel immunodiffusion, hemagglutination inhibition and hemolysis inhibition assays. Low titers in unpaired serum samples may be due to cross-reactions with related viruses, particularly those in the Simbu serogroup.

Akabane virus, its antigens or nucleic acids may be detected in the CNS of cattle with bovine epizootic encephalomyelitis. These tests can also be useful sometimes in fresh fetuses aborted before they have developed an immune response. Samples should be taken from the placenta and fetal skeletal muscle, brain and spinal cord. In cattle, the sampled fetus should have been aborted before 5 months of gestation and soon after it was infected. Tests to detect Akabane virus are generally unsuccessful in most other animals infected before they were born, or the dam, as they have usually cleared the infection before its effects are seen. However, real-time RT-PCR may sometimes find residual nucleic acids in neonatal tissues or the placenta. Akabane virus can be isolated in a number of cell lines (e.g., Vero, HmLu-1, BHK-21 cells), and the virus can be identified by immunofluorescent or immunohistochemical staining or virus neutralization. Suckling mice were also used for virus recovery in the past, but animal inoculation is generally discouraged if other techniques are available. Antigens can be detected directly in tissues by immunofluorescent or immunohistochemical staining. Cross-reactivity with other Simbu serogroup viruses can be an issue. A number of reverse transcription polymerase chain reaction (RT-PCR) assays have been described, and may be available in some laboratories. Some

published assays are reported to distinguish Akabane virus from Aino and Schmallenberg viruses, which also cause reproductive disorders in livestock. A reverse transcription loop-mediated isothermal amplification assay (RT-LAMP) has been published. The ability of Simbu serogroup members to exchange gene segments may complicate the development and interpretation of some diagnostic tests.

## Treatment

There is no treatment for animals affected by Akabane virus.

## Control

### Disease reporting

A quick response is vital for containing outbreaks in disease-free regions. Veterinarians who encounter or suspect an Akabane virus infection should follow their national and/or local guidelines for disease reporting. In the U.S., state or federal veterinary authorities should be informed immediately.

### Prevention

Akabane virus does not appear to be transmitted between animals except by arthropods. If this virus is introduced into an area where it is not endemic, care should be taken to prevent it from infecting potential vectors, especially *Culicoides* spp. gnats.

Vaccines are available in some countries, and can prevent fetal losses. There are, however, reports of antigenic differences between vaccine strains and the viruses that cause postnatal encephalomyelitis in Japan and Korea, and some authors suggest that new vaccines should be developed for these strains. Akabane disease can also be controlled in pregnant animals by moving them into an endemic area in time to develop immunity before they are first bred. Changing herd management to avoid infections during the most susceptible period of pregnancy may be helpful. Insect control techniques, including the use of repellents, can be effective for a few days, but cannot control the disease in the long term.

## Morbidity and Mortality

Akabane disease is seasonal in temperate regions, and occurs year-round but tends to peak at certain times of the year in tropical areas (i.e., when vector numbers increase due to factors such as rainfall). Reproductive disease is only seen when an immunologically naive animal is exposed to Akabane virus during pregnancy. Because animals usually become infected before sexual maturity where this virus is constantly present, outbreaks generally occur at the limits of the virus's geographic range. Such outbreaks can occur during environmental conditions that favor the multiplication of the *Culicoides* vectors, such as a mild, moist autumn, or perhaps when infected midges are blown long distances by the wind. Clinical cases can also be seen when these midges have been absent for a period, usually as the result of drought, or when unvaccinated pregnant animals are moved

from nonendemic to endemic areas. Epizootics tend to occur at 4-6-year intervals in some areas, probably when immunity to previous viruses has waned. Subsequent pregnancies are not affected.

Cattle are affected more often than sheep and goats, due to factors that limit small ruminants' exposure to Akabane virus during their first pregnancy, such as the timing of the breeding season and the environments where they are raised. The effects of Akabane virus depend on the stage of the pregnancy. In cattle, the fetus can be affected any time after the first two months, but the most severe defects occur when the animal is infected at approximately 80-150 days of gestation. The peak incidence of defects is seen during the third and fourth months of gestation. Depending on the viral strain, up to 40% of calves infected at this time can have congenital abnormalities. Sheep and goats are most susceptible between 28 and 56 days of gestation, particularly at 28-36 days. Few abnormalities are generally seen during the last 2 months of gestation in cattle or after 60 days in small ruminants. Morbidity also varies with the strain of the virus. Highly virulent strains can affect as many as 80% of infected animals, while other strains may cause clinical signs in fewer than 20%, even when the dam is infected at the most susceptible stage of the pregnancy. The mortality rate is very high in affected newborns: most animals die soon after birth or must be euthanized.

Bovine epizootic (postnatal) encephalomyelitis appears to be uncommon in most regions. However, the strains that cause this syndrome seem to have become endemic in Japan and South Korea, where such outbreaks have been reported regularly among cattle since 2000. While some outbreaks have affected limited numbers of animals, approximately 180 cattle developed CNS signs in Japan between the end of August and mid-December, 2006, and an epizootic in Korea affected more than 500 animals in 2010. The morbidity rate for this syndrome is still unclear. In 2000, an outbreak on five farms in Korea affected approximately 30% of the animals. However, other studies suggest that only small numbers of infected cattle develop encephalomyelitis.

## Internet Resources

[The Merck Veterinary Manual](#)

[United States Animal Health Association. Foreign Animal Diseases](#)

[World Organization for Animal Health \(WOAH\)](#)

[WOAH Manual of Diagnostic Tests and Vaccines for Terrestrial Animals](#)

[WOAH Terrestrial Animal Health Code](#)

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