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**Akabane**

*Congenital Arthrogryposis-Hydranencephaly Syndrome, Congenital Bovine Epizootic A-H Syndrome, Acorn calves, Silly calves, Curly Lamb Disease, Curly Calf Disease, Dummy Calf Disease*



Akabane is also known as congenital arthrogryposis-hydranencephaly syndrome, congenital bovine epizootic A-H syndrome, acorn calves, silly calves, curly lamb disease, curly calf disease, and dummy calf disease. It is a viral disease of ruminants that has the potential for serious economic losses.

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**Overview**

- Organism
- Economic Impact
- Epidemiology
- Transmission
- Clinical Signs
- Diagnosis and Treatment
- Prevention and Control
- Actions to take




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In today's presentation we will cover information regarding the organism that causes Akabane and its epidemiology. We will also talk about the economic impact the disease has had in the past and could have in the future. Additionally, we will talk about how it is transmitted, the species it affects, clinical signs, necropsy findings, and diagnosis and treatment of the disease. Finally, we will address prevention and control measures for the disease as well as actions to take if Akabane is suspected.

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**The Organism**



The Akabane virus is an arbovirus (transmitted by arthropods) in the family Bunyaviridae, genus Orthobunyavirus. The single-stranded RNA virus causes intrauterine infection of the fetus in pregnant cattle, sheep and goats by invading the endothelial cells of the placenta, replicating in the trophoblastic cells and finally in the fetus itself. The result is abortions, stillbirths, premature births and deformities of the fetus or newborn with no clinical signs in the dam during pregnancy.

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**Akabane virus**

- Family Bunyaviridae
  - Genus Orthobunyavirus
    - Single stranded RNA virus
- Intrauterine infection of fetus
- Sheep, goats and cattle
- No clinical signs in the dam

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**Importance**



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### History

- 1959
  - First isolated from mosquitoes in Japan
- 1969-70
  - Israel outbreak
  - 3000 calves, 700 lambs, 600 kids
- 1972
  - First reported in Australia

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Akabane is named after the Japanese village where the virus was first isolated from mosquitoes (*Aedes vexans*, *Culex tritaeniorhynchus*) in 1959. In 1969-70, a major epizootic of Akabane occurred in Israel affecting 3,000 dairy calves, 700 lambs, and 600 kids. In Australia, the Akabane virus was first identified in 1972, however sporadic undiagnosed outbreaks (resembling the disease) had been observed as early as the mid-1940's.

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### Economic Impact

- High mortality to newborn and fetal cattle, sheep and goats
- U.S. livestock greatly susceptible
- Potential vectors found in U.S.
- 2002 U.S. livestock statistics
- Calves: 38.2 million head
- Lambs: 4.36 million head

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To date, Akabane has not occurred in the U.S. Therefore our livestock population is highly susceptible to the virus. Based on the severe mortality that can occur with Akabane disease in naïve populations, the economic impact of the disease can be great. Additionally, the potential vectors for the disease are found in the U.S. In 2002, the calf-crop for the U.S. was reported at an estimated 38.2 million head while the lamb-crop was 4.36 million head. [No specific data on goats or kids in the U.S. could be found].

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### Epidemiology

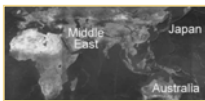


Akabane is common in the tropics and subtropics between latitude 35°N and 35°S (indicated by the red shaded box). It is endemic to northern Australia and occasional outbreaks have occurred in southern Australia, Asia, the Middle East and South Africa when conditions for virus transmission are favorable. Akabane virus is considered exotic to North America, however an endemic Bunyavirus, Cache Valley virus, produces similar clinical signs in lambs and has been isolated from both mosquitoes and biting midges in North America. Incidence for the disease and the potential for epizootics is correlated with climatic factors, a distinct seasonal pattern, the geographic distribution of competent vectors and the availability of susceptible ruminant populations.

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### Geographic Distribution

- Tropics and subtropics
- Australia, Japan, Israel, Korea
- Occasionally in Asia, the Middle East and South Africa



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### Morbidity/ Mortality

- Endemic areas
  - Immunity by sexual maturity
  - Seroprevalence 80%
- Greatest risk
  - Naïve and susceptible animals
  - Favorable environmental conditions
- High mortality in newborns
  - Most die soon after birth or must be euthanized

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In endemic areas, most animals are immune to Akabane virus by the time they reach sexual maturity. Surveys indicate that more than 80% of adult cattle in an endemic area are seropositive for the virus. However, following years of drought or times of reduced vector populations, native livestock may not be exposed prior to breeding age and therefore become susceptible. This can lead to an epizootic in an endemic areas. Data from Japanese and Australian outbreaks suggest that the virus subsequently may invade the fetus in approximately 30-40% of infected pregnant cows. Outbreaks typically occur in areas where naïve and susceptible animals are located and when environmental conditions are favorable for the disease. Most epizootics occur at the northern and southern periphery of the endemic band or susceptible animals are introduced into endemic areas. Outbreaks usually occur in late winter, indicating that the peak of virus activity and fetal infections occurs during the previous late summer and early autumn period. The greatest mortality is in affected newborns and the rate is very high. Most die soon

after birth or must be euthanized.

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Transmission



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**Animal Transmission**

- Vectors
  - Mosquitoes
  - Biting midges
- NOT transmitted by
  - Direct contact
  - Infected tissues, exudates, body fluids
  - Fomites
- Ruminants are not long-term carriers




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The vector for Akabane has not been proven, however, epidemiological evidence suggests that the virus is spread by mosquitoes and gnats. The virus has been isolated from a number of mosquito species (*Aedes*, *Culex*, *Anopheles*) as well as *Culicoides* species (biting midges). *Culicoides brevitarsis* is the only insect in Australia from which Akabane virus has been isolated. Akabane is **not** transmitted by direct contact, infected tissues, exudates, body fluids or fomites. Ruminants do not appear to become long-term carriers of this virus.

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Animals and Akabane



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**Clinical Signs**

- Cattle, sheep, goats
  - Adults are typically asymptomatic
  - Pregnant ruminants
    - Abortion and stillbirths
    - Premature births
    - Dystocia
  - Congenital abnormalities
    - Varies with stage of gestation



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Akabane disease only occurs in cattle, sheep and goats. However, antibodies to the virus have been found in horses, buffalo, deer, camels, dogs, monkeys and most recently in pigs. Adult ruminants with Akabane infection are typically asymptomatic. Viremia usually occurs 1 to 6 days after infection and lasts for 1 to 9 days. Only during this limited time are viral titers sufficient for potential vectors. Long term carriers of the disease are not believed to occur. Manifestation of the disease is not typically noticed until pregnant ruminants abort, have stillbirths, premature births or dystocia. Recently, a report of five adult cattle in Korea exhibiting neurological signs in Korea were later diagnosed with Akabane virus disease. This is the first report of observable clinical signs of Akabane virus in adult cattle.

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### Congenital Abnormalities

- Late first trimester
  - Bright and alert, but unable to stand
  - Ataxia, paralyzed limbs, muscle atrophy
- Second trimester
  - Arthrogryposis, rigidly flex joints
  - Severe muscle atrophy
  - Torticollis, scoliosis, kyphosis
- Late pregnancy
  - Can stand and walk
  - Behavioral abnormalities
  - Skull deformities

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Congenital abnormalities of the fetus or newborns are a hallmark of this disease. Effects on the young varies depending on the stage of gestation reached at the time of infection. When infected late in the first trimester, animals are usually born bright and alert but are unable to stand, are ataxic, and may have one or more paralyzed limbs. Muscle atrophy, limb rotation, exophthalmos (protuding eye), and abnormal vocalization may also occur. Animals infected during the second trimester have arthrogryposis at birth; most cannot stand. The joints are rigid and fixed in flexion and muscles are severely atrophied. Torticollis, scoliosis and kyphosis may also be seen. When infected late in pregnancy, animals can usually stand and walk, but have behavioral abnormalities, such as slow or absent suckle reflex, depression, dullness, periodic hyperexcitability, incoordination and blindness. Skull deformities can be common. Most affected neonates die or must be euthanized soon after being born. Animals with mild symptoms may gradually become more mobile, but most die by 6 months.

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### Post Mortem Lesions

- Fetuses or Newborns
  - Arthrogryposis
- Hydranencephaly



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Post mortem lesions of the fetuses and newborns follow the previously mentioned clinical signs. Arthrogryposis and hydranencephaly are the most commonly noted lesions. Most of the affected joints are ankylosed and cannot be straightened even by force. [Arthrogryposis (persistent flexion of joints) [top photo] is the most frequently observed lesion. Hydranencephaly (absence of the cerebral hemispheres and their normal site being occupied by cerebrospinal fluid) [bottom photo] can also be seen with Akabane infection. Photos courtesy of USAHA Grey book at [http://www.vet.uga.edu/vpp/gray\\_book/fad.pdf](http://www.vet.uga.edu/vpp/gray_book/fad.pdf).]

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### Post Mortem Lesions

- Other congenital abnormalities
  - Hydrocephalus, microencephaly, spinal cord agenesis or hypoplasia, torticollis, scoliosis, brachygnathism
  - Cataracts, ophthalmia
  - Hypoplastic skeletal muscles and lungs
  - Fibrinous polyarticular synovitis

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Central nervous system lesions may include hydrocephalus, agenesis of the brain, microencephaly, proencephaly, cerebellar cavitation. Other abnormalities may include fibrinous leptomenigitis or ependymitis, spinal cord agenesis or hypoplasia, torticollis, scoliosis, brachygnathism, cataracts, ophthalmia [severe inflammation of the eye], hypoplastic skeletal muscles and lungs, fibrinous polyarticular synovitis.

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### Differential Diagnosis

- Cache Valley virus
- Bluetongue
- Bovine viral diarrhea
- Border disease
- Wesselsbron virus
- Nutritional, genetic or toxic diseases

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In the U.S., Akabane disease must be differentiated from infection with Cache Valley virus (which is widespread in the western US). Other differentials to be considered include bluetongue, bovine virus diarrhea, Border disease, Wesselsbron virus. Additionally, nutritional, genetic or toxic diseases should be considered.

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**Sampling**

- Before collecting or sending any samples, the proper authorities should be contacted
- Samples should only be sent under secure conditions and to authorized laboratories to prevent the spread of the disease

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Before collecting or sending any samples from animals with a suspected foreign animal disease, the proper authorities should be contacted. Samples should only be sent under secure conditions and to authorized laboratories to prevent the spread of the disease. Samples of the placenta, muscle, cerebrospinal fluid and nervous tissue should be collected from the fetus for virus isolation. Serum samples should be taken from the dam, the fetus or the neonate before it is allowed to suckle. Pieces of liver, spleen, kidney, heart, lung, lymph nodes, spinal cord, brain and affected muscle should be placed in 10 percent buffered formalin for histopathology. The samples should be delivered (on ice) to a laboratory within 24 hours.

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**Diagnosis**

- Clinical
  - Fetal arthrogryposis and hydranencephaly
- Laboratory Tests
  - Serology
    - Serum samples from fetus or neonate
    - Cerebrospinal fluid
    - Adults: antibody titer or seroconversion
  - Virus isolation and identification
  - Immunofluorescent staining

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A presumptive diagnosis of Akabane disease can be made on the basis of clinical and post mortem examination. The diagnosis of Akabane disease is often confirmed by serology. Antibodies can be found in serum, and sometimes from the cerebrospinal fluid or other body fluids, from the fetus or unsuckled neonate. However, the absence of antibodies does not rule out this disease. In adult ruminants, a rise in antibody titer or seroconversion indicates infection. Serological tests available include microtiter neutralization, agar gel immunodiffusion, hemagglutination inhibition, hemolysis inhibition assays. Virus isolation is rarely successful unless the fetus and placenta were aborted before the fetus developed an immune response. Akabane virus cannot be isolated from maternal tissues by the time the affected fetuses are born. Immunofluorescent staining can also be used to detect virus antigens in the brain and muscle of aborted fetuses.

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**Treatment**


- No effective treatment
- Affected neonates should be euthanized
- Subsequent pregnancies unaffected

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There is no effective treatment for Akabane virus. Most affected neonates die or must be euthanized soon after being born. Animals with mild symptoms may gradually become more mobile, but most die by 6 months. Subsequent pregnancies of the infected dam will not be affected.

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**Akabane in Humans**



Human infections by Akabane virus have not been reported. There is no public health consequence of this disease.

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**Prevention and Control**



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**Recommended Actions**

- Notification of Authorities
  - Federal:
    - Area Veterinarian in Charge (AVIC)
    - [www.aphis.usda.gov/vs/area\\_offices.htm](http://www.aphis.usda.gov/vs/area_offices.htm)
  - State veterinarian
    - [www.aphis.usda.gov/vs/sregs/official.htm](http://www.aphis.usda.gov/vs/sregs/official.htm)

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If you suspect a case or outbreak of Akabane, contact your state and/or federal veterinarian immediately.

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**Prevention**


- Protection from vectors
  - Mosquitoes and biting midges
- Vaccination
  - Inactivated and attenuated vaccine
  - Killed vaccine
  - Not currently available in U.S.

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Prevention of Akabane disease includes vector control measures (mosquitoes and midges) and vaccination. Akabane virus does not appear to be transmitted between animals except by arthropods (mosquitoes and biting midges). Prevention efforts should include elimination of vector breeding sites, repellants or screened housing for animals. An inactivated and an attenuated vaccine have been developed and used in Japan. An effective killed vaccine has been developed but not marketed in Australia. The vaccines must be used prior to exposure to the infected vectors. A vaccine is not currently available in the U.S.

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**Additional Resources**



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**Internet Resources**


- World Organization for Animal Health (OIE) International Animal Health Code
  - [www.oie.int](http://www.oie.int)
- USAHA Foreign Animal Diseases – “The Gray Book”
  - [www.vet.uga.edu/vpp/gray\\_book/index](http://www.vet.uga.edu/vpp/gray_book/index)

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