

SCHMALLEMBERG VIRUS

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Schmallenberg virus was discovered in November 2011 and epidemiological, immunological and virological investigations are on-going in several European countries. The information presented in this technical factsheet reflects the epidemiological observations and research done to date (May 2012), together with data extrapolated from genetically similar viruses of the same genus and serogroup.

AETIOLOGY

Classification of the causative agent

The “Schmallenberg virus” is an enveloped, negative-sense, segmented, single-stranded RNA virus. It belongs to the *Bunyaviridae* family, within the *Orthobunyavirus* genus. The Schmallenberg virus is a member of the Simbu serogroup viruses, which includes Shamonda, Akabane, and Aino viruses.

Field and laboratory studies indicate a causal relationship between Schmallenberg virus infection and the reported clinical signs. .

Resistance to physical and chemical action

From extrapolation from the California serogroup of Orthobunyaviruses:

- Temperature:** Infectivity lost (or significantly reduced) at 50–60°C for at least 30 minutes.
- Chemicals/Disinfectants:** Susceptible to common disinfectants (1 % sodium hypochlorite, 2% glutaraldehyde, 70 % ethanol, formaldehyde)
- Survival:** Does not survive outside the host or vector for long periods

EPIDEMIOLOGY

According to the epidemiological investigations, reinforced by what is already known about the genetically related Simbu serogroup viruses, Schmallenberg virus affects ruminants. Serological studies indicate that it is not zoonotic. Transmission in animals is by insect vectors and then vertically *in utero*.

Hosts

- Confirmed by PCR or virus isolation:
 - Cattle, sheep, goats
 - Bison
- Confirmed by serology only:
 - Red deer
 - Roe deer
 - Alpaca
 - Mouflons
- *Humans*: Epidemiological and virological studies of human populations considered to be at risk did not demonstrate evidence of zoonotic potential.

Transmission

- Epidemiological investigations indicate insect vector transmission.
- Vectors: Schmallenberg virus genome was detected in several Culicoides species. Further information is required to determine whether mosquitoes play a role. Vertical transmission across the placenta is proven.
- Direct transmission from animal to animal is very unlikely.
- Further research is still needed to confirm these transmission routes and to determine the competent insect species.

Viraemia and incubation period

Experimental infection in cattle and sheep showed no clinical signs or mild symptoms at 3 to 5 days post-inoculation with an incubation period of between 1 and 4 days and viraemia lasting for 1 to 5 days.

Sources of virus

Material found to be positive in virus isolation (up to May 2012):

- Blood from affected adults and brain from infected fetus.

Material found PCR positive (up to May 2012):

- Organs and blood of infected fetus, placenta, amniotic fluid, meconium.

Occurrence

Only some Orthobunyaviruses had been reported in Europe but viruses from the Simbu serogroup had never been isolated in Europe before 2011.

Schmallenberg virus was first detected in November 2011 in Germany from samples collected in summer/autumn 2011 from diseased (fever, reduced milk yield) dairy cattle. Similar clinical signs (including diarrhoea) were detected in dairy cows in the Netherlands where the presence of Schmallenberg virus was also confirmed in December 2011.

Since early December 2011, congenital malformations were reported in newborn lambs in the Netherlands, and Schmallenberg virus was detected in and isolated from the brain tissue. Up to May 2012, The Netherlands, Belgium, Germany, United Kingdom, France, Luxembourg, Spain and Italy have reported stillbirth and congenital malformations with PCR positive results.

For detailed information on the occurrence of this disease worldwide, see the *OIE World Animal Health Information Database (WAHID)* interface [<http://www.oie.int/wahis/public.php?page=home>].

DIAGNOSIS

Clinical diagnosis

Manifestation of clinical signs varies by species: bovine adults have shown a mild form of acute disease during the vector season, congenital malformations have affected more species of ruminants (to date: cattle, sheep, goat and bison). Some dairy sheep and cow farms have also reported diarrhoea.

- Adults (cattle)
 - Probably often inapparent, but some acute disease during the vector-active season
 - Fever (>40°C)
 - Impaired general condition
 - Anorexia
 - Reduced milk yield
 - Diarrhoea
 - Recovery within a few days for the individuals, 2–3 weeks at the herd scale
- Malformed animals and stillbirths (calves, lambs, kids)
 - Arthrogryposis/ Hydranencephaly
 - Brachygnathia inferior
 - Ankylosis
 - Torticollis
 - Scoliosis

The exact rate of malformation is not known and varies depending on the stage of gestation at the time of infection.

Lesions

In malformed newborn:

- Hydranencephaly
- Hypoplasia of the central nervous system
- Porencephaly
- Subcutaneous oedema (calves)

The symptoms can be summarised as arthrogryposis and hydranencephaly syndrome (AG/HE)

Differential diagnosis

For the acute infection of the adults:

The symptoms are not specific. All possible causes of high fever, diarrhoea and milk reduction should be taken into account.

For the malformation of calves, lambs and kids:

- Other Orthobunyaviruses
- Bluetongue
- Pestiviruses
- Genetic factors
- Toxic substances

Laboratory diagnosis

Samples

Samples should be transported cooled or frozen

From live animals for the detection of acute infection:

- EDTA blood
- Serum
 - At least 2 ml, transported cooled

From stillborns and malformed calves, lambs and kids:

- Virus detection:
 - Tissue samples of brain (cerebrum and brainstem)
 - Amniotic fluid
 - From live newborn:
 - Amniotic fluid and placenta
 - (Meconium)
- Antibody detection:
 - Pericardial fluid
 - Blood(preferably pre-colostral)
- Histopathology:
 - Fixed central nervous system, including spinal cord

Procedures

Identification of the agent

- Real-time RT-PCR (Bilk et al., 2012); commercial PCR kits are available
- Cell culture isolation of the virus: insect cells (KC), hamster cells (BHK), monkey kidney cells (VERO)

Serological tests on serum samples

- ELISA: commercial kit available
- Indirect Immunofluorescence
- Neutralization test

For further information, reference material and advice, refer to Dr Martin Beer (Martin.Beer@fli.bund.de), Institute of Diagnostic Virology, Friedrich-Loeffler-Institut, Federal Research Institute for Animal Health, Greifswald-Insel Riems, Germany.

PREVENTION AND CONTROL

There is currently no specific treatment or vaccine for Schmallenberg virus.

Sanitary prophylaxis

Control of potential vectors during the vector-active season may decrease the transmission of virus.

Reschedule of breeding outside the vector season should decrease the number of fetal malformations.

REFERENCES AND OTHER INFORMATION

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The OIE will update this Technical Factsheet when relevant

Additional Information

MEAT

Relevant knowledge: Only clinically healthy animals should be slaughtered. The viraemic period is very short. Transmission of the virus is most likely by vectors.

Risk of transmission to humans and animals: Negligible

MILK

Relevant knowledge: Milk should only be collected from clinically healthy animals. The viraemic period is very short. Transmission of the virus is most likely by vectors.

Risk of transmission to humans and animals: Negligible

SEMEN

Relevant knowledge: The viraemic period is very short. Semen should be collected from clinically healthy animals. From 8 bulls experimentally infected with Akabane virus, virus was not found in semen even during the viraemic period (*Experimental infection of bulls with Akabane virus*, Parsonson IM, Della-Porta AJ, Snowdon WA, O'Halloran ML, Res Vet Sci. 1981 Sep; 31(2):157-60.).

Risk of transmission to animals: Negligible for sero-positive bulls; negligible for sero-negative and PCR negative bulls.

EMBRYOS

Relevant knowledge: The viraemic period is very short. Embryos should be collected from clinically healthy animals. Akabane virus is classified under the category 4 (diseases or pathogenic agents for which studies have been done or are in progress that indicate that either no conclusions are yet possible with regard to the level of transmission risk; or the risk of transmission via embryo transfer might not be negligible even if the embryos are properly handled between collection and transfer).

Recommendation: Safety measures applicable to Akabane virus should be followed.

Risk of transmission: According to the current knowledge, the risk from sero-negative donor animals is negligible. Sero-positive and PCR-negative donor animals at the day of insemination should be also considered with negligible risk.

LIVE NON-PREGNANT ANIMALS

Relevant knowledge: The viraemic period is very short. Mild clinical signs might occur. Transmission is most likely by vectors.

Risk of transmission: Negligible for the following animals:

- PCR-negative after 7 days in a vector-free environment or,
- Sero-positive and PCR-negative.

LIVE PREGNANT ANIMALS

Relevant knowledge: The virus can persist in the fetus; this may result in the birth of virus positive calves, lambs and kids. The relevant pregnancy time to induce viraemic newborns is not exactly known.

Risk of transmission:

- Negligible for the offspring of sero-negative animals tested twice in a vector-free environment (within 28 days),
- Negligible for the offspring of animals sero-positive before insemination,
- Undetermined for the offspring of all animals not covered by the previous bullets.