SCHMALLENBERG VIRUS

Schmallenberg virus was discovered recently (November 2011) and epidemiological, immunological and microbiological investigations are still on-going in several European countries. The information presented in this technical disease card describes the epidemiological observations and research done during the first months following its discovery, and data extrapolated from genetically similar viruses of the same genus and serogroup.

AETIOLOGY

Classification of the causative agent

The provisionally named "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 related to the Simbu serogroup viruses, in particular Shamonda, Akabane, and Aino virus. So far, sequence data suggests the closest relationship to Shamonda virus. This classification has to be confirmed with further sequence data and investigations e.g. about the serological relationship to other Simbu sero-group viruses.

Even though the exact role of Schmallenberg virus needs to be further investigated, first inoculation experiments as well as the diagnostic data from malformed lambs and calves strongly suggest a causal relationship between the presence of the virus 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 domestic ruminants. It is unlikely to be zoonotic. The spatial and temporal distribution suggests that the disease is first transmitted by insect vectors and then vertically *in utero*.

Hosts

- Cattle, sheep, goats
- Bison
- No information on the susceptibly of exotic ruminants (camelids, llamas, etc.), or other wild ruminants, or on other species. It is worth noting that other viruses of the Simbu serogroup affect wild ruminants and that antibodies to Akabane virus have been found in horses, donkeys, buffalo, deer, camels and even in pigs. Some viruses of the Simbu serogroup (Mermet, Peaton and Oropouche viruses) have also been detected in birds. Mice and hamsters can be infected experimentally.
- *Humans:* No human disease related to Schmallenberg virus have been reported in the affected zone so far, and the genetically most related Orthobunyaviruses do not cause disease in humans. Thus current risk assessments conclude that the virus is unlikely to cause disease in humans even if it cannot be fully excluded at this stage. Nevertheless, close collaboration between public health and animal health services is recommended for the early detection of potential human cases, particularly in farmers and veterinarians in close contact with potentially infected animals, and especially during interventions for dystocia.

Transmission

The transmission of Schmallenberg virus needs to be confirmed but hypotheses have been developed through recent epidemiological investigations and comparison with other Orthobunyaviruses:

- It is likely to be transmitted via insects vectors (biting midges and/or mosquitoes)
- Vertical transmission across placenta is proven

- Direct contamination from animal to animal or animal to human is very unlikely but needs further investigation (first experiments have been started)

Further research is still needed to confirm these transmission routes and to determine the competent insect species.

Viraemia and incubation period

Experimental infection in 3 calves showed mild clinical signs of acute infection at 3 to 5 days post-inoculation and viraemia at 2 to 5 days post-inoculation. No data are available for sheep and goats up to February 2012.

Sources of virus

Source of transmission:

- Likely to be infected insect vectors

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

- Virus has been isolated from blood from affected adults and infected foetus and brain from infected foetus.

Material found PCR positive (up to February 2012)

- Organs and blood of infected foetuses, placenta, amniotic fluid, meconium

All these findings have to be further investigated for their role in transmission.

Occurrence

Only some Orthobunyaviruses had been reported in Europe: e.g. Tahyna virus from the California serogroup, but viruses from the Simbu serogroup had never been isolated in Europe before.

First phase: 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.

Second phase: In 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 February 2012, Belgium, Germany, United Kingdom and France have reported stillbirth and congenital malformations with PCR positive results.

For more recent, 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 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 (by up to 50%)
  - Diarrhoea
  - Recovery within a few days for the individuals, 2–3 weeks at the herd scale
- Malformed animals and stillbirths (calves, lambs, kids)
  - Arthrogryposis
  - Hydrocephaly
  - Brachygnathia inferior
  - Ankylosis
  - Torticollis
  - Scoliosis

The exact rate of malformation is not known up to February 2012. Some sheep farms have reported in a period related to acute infection in Summer and Autumn 2011 more than 25% malformed lambs.

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 (AHS)

Differential diagnosis

For the acute infection of the adults:

- Bluetongue
- Epizootic haemorrhagic disease (EHD) virus
- Foot and mouth disease (FMD) virus
- Bovine viral diarrhoea (BVD) virus, border disease and other pestiviruses
- Bovine herpesvirus 1 and other herpesviruses
- Rift Valley fever virus
- Bovine ephemeral fever virus
- Toxic substances

The symptoms are not specific. Other sources of diarrhoea and milk reduction could be taken into account.

For the malformation of calves, lambs and kids:

- Toxic substances
- Genetic factors
- Bluetongue
- Pestiviruses
- Other viruses of the Simbu serogroup (Akabane)

Laboratory diagnosis

Samples

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:

- From necropsy: Tissue samples of brain (cerebrum and cerebellum), additional samples: central nervous system, spleen and blood
- From live newborn: blood, (preferably pre-colostral) serum and meconium
  - Samples should be transported cooled or frozen
- Placenta and amniotic fluids

Procedures

Identification of the agent

- Real-time RT-PCR
- Cell culture isolation of the virus

Serological tests on serum samples

- Indirect Immunofluorescence
- Neutralization test
- ELISA to be developed

PREVENTION AND CONTROL

- There is no specific treatment or vaccine for Schmallenberg virus

Sanitary prophylaxis

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

Delay of breeding may decrease the number of foetal malformations.

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The OIE will update this Technical Factsheet when relevant