Epidemiology of Schmallenberg virus (SBV) infection

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I am concerned that some reports in the media give an imprecise explanation of SBV epidemiology and tend to suggest that SBV is transmitting between lambs in affected flocks and spreading between flocks by this means; I would suggest that more needs to be done to ensure that the public is better informed on the epidemiology of this disease. I would also like to make some comments about Europe's vulnerability to virus invasions.

As is common knowledge, the epidemiology of SBV infection seems to be similar to that of Akabane virus and other orthobunyaviruses of the Simbu Group which affect ruminants. Useful insights can be gained from a global view of Akabane disease over the last 40 years or so since its 1st description.
Akabane virus infection is endemic in many tropical and subtropical areas. In these, susceptible ruminant species become infected at an early age when fed on by ubiquitous midge and mosquito vectors and develop a long-lasting protective immunity by the time of breeding; thus, congenital abnormalities are seldom seen in endemic areas even though cases can occur when naive, susceptible animals are introduced into such areas. Under favourable environmental conditions, the vectors (and hence the virus) may spread beyond their usual range, and outbreaks of congenital disease then occur in areas where the disease process has rarely, or never been experienced before.

Essentially it is at the interface between free and endemic areas that severe outbreaks are to be expected. Acute infection following postnatal exposure, accompanied by unremarkable clinical signs, is usually not recognised to have occurred until malformed lambs and calves are born. By this time the virus is no longer present in either dam or offspring; antibodies disclose the presence of earlier infection. Akabane viruses vary in their virulence but only rarely has pathology been associated with post-natal infection (ref 1).

Outbreaks at the interface tend to occur infrequently but are recognised to have occurred in Japan, Korea, Taiwan, Australia, Israel, and Turkey (ref 1) and southern Africa to the north and south of the endemic zone. Repeated outbreaks in Turkey and Israel over the last 40 years indicate clearly that, at least from 1970 until 2010 or 2011, the eastern Mediterranean was situated at the interface between endemic and free areas.

Now that a Simbu Group virus, like bluetongue viruses in 2006 to 2010, has made the leap northwards in Europe, and without understanding the determinants that favoured the change, it is impossible to predict what will happen next. Will the virus "burn out" and the earlier status be re-established (whatever that was); are we to see a permanent shift in the interface between endemic and free zones; or, alternatively, will the epidemic wave move on as vector range extends or previously free populations of vectors become infected for the 1st time? It has been reported that SBV is most closely related to Shamonda virus, one of a number of Simbu Group viruses detected in Nigeria in the 1960s. Having not been seen since, emergence of the virus in Japan in 2002 (ref 3) illustrated that we know little of the epidemiology of such potential pathogens. One thing is clear – our vision of the threat posed by the presence of these viruses at the edge of Europe was too complacent by far.
Several other viruses are in a similar situation to that of SBV and pose an immediate threat to the livestock of northern Europe. Not least of these are the orbiviruses causing epizootic haemorrhagic disease [EHD] of ruminants, African horse sickness [AHS], and equine encephalosis [EE]. Enhanced surveillance and applied research are surely essential at this time to prepare for the introduction of new vectorborne diseases which now seems inevitable.

References

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The following passage, derived from chapter 2.9.1 of OIE's terrestrial manual which addresses Akabane and other bunyaviruses, falls in line with Dr Roeder's appreciated commentary and adds supportive background:

"In endemic areas, antibody in the female animal prevents fetal infection, but Akabane virus is capable of establishing a long-term infection of the placenta in susceptible cattle and sheep. This takes place between 30 and 70 days gestation in the ewe and between 30 and 150 days gestation in the cow. Akabane virus has a predilection for brain, spinal cord and muscle cells where non-inflammatory necrosis interferes with morphogenesis.

"Akabane virus infection has been studied experimentally in sheep and goats with the production of arthrogryposis/hydroencephaly, kyphosis, scoliosis, micro- and porencephaly, stillbirths and abortions.

"Experimental Akabane virus studies have been carried out in pregnant cattle and it was shown that the type of abnormality is dependent on the gestational age of the fetus with hydroencephaly seen at 76-104 days and arthrogryposis at 103-174 days gestation. This time differential in appearance of abnormalities is clearly seen in bovine fetuses, whereas in sheep with a shorter gestation period, brain and skeletal lesions appear concurrently in the same fetus. The sequence of events during an epizootic of Akabane virus-induced fetal loss are the birth of uncoordinated calves, followed by those with arthrogryposis and dysplastic muscle changes, and lastly those with hydrocephalus and other severe CNS lesions. These events may be preceded by stillbirths and abortions." (For additional data and references, see http://www.oie.int/fileadmin/Home/eng/Health_standards/tahm/2.09.01_BUNYAVIRAL_DISEASES.pdf)