

Research

EDITORIAL

Schmallenberg virus: on its way out or due for a comeback?

Nick De Regge

SCHMALLEMBERG virus (SBV) emerged during the summer of 2011 in north-western Europe and was identified for the first time in dairy cattle herds in Germany (Hoffmann and others 2012). The virus belongs to the Simbu serogroup of Orthobunyaviruses and many similarities have been reported with the closely related Akabane virus regarding its transmission, pathogenesis and induced clinical signs. SBV causes mild clinical signs in adult ruminants, ranging from fever, diarrhoea and temporarily reduced milk production. However, infection of pregnant cattle, sheep and goats can cause transplacental transmission leading months later to abortions, stillbirths and congenital malformations characterised by the

arthrogryposis-hydranencephaly syndrome in newborns (Doceul and others 2013).

After its first identification in Germany in 2011, SBV spread rapidly and widely over a large part of Europe, including the UK (EFSA 2014). Indigenous European *Culicoides* biting midges, which were already known to have spread bluetongue virus serotype 8 after its emergence in Europe in 2006 (Mehlhorn and others 2007, Meiswinkel and others 2007), were found to be responsible for the rapid transmission of SBV (De Regge and others 2012, 2015). Seroprevalence studies showed that the majority of domestic ruminants became infected in SBV-affected countries (Méroc and others 2013, 2014, Wernike and others 2014). In 2012, SBV spread further over Europe and evidence for renewed but lower levels of SBV circulation in countries affected in 2011 was found (Conraths and others 2013, Afonso and others 2014, De Regge and others 2014). From spring 2013 onwards, the number

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of reported and confirmed cases dropped drastically, indicating that the peak of the first emergence was over (Afonso and others 2014) and that further extensive circulation of the virus was probably hindered by the high level of induced population immunity. The limited available data on SBV surveillance from 2013 onwards suggests a strongly reduced SBV circulation in Belgium, France, the Netherlands and Germany since 2013 (Dominguez and others 2014, Veldhuis and others 2015, Wernike and others 2015, Poskin and others 2016). Only Germany reported some SBV reappearances during summer and autumn 2014 (Wernike and others 2015).

The results of Stokes and others (2016), summarised on p 435 of this issue of *Veterinary Record*, are in line with the observations in other European countries. After a decline in reported SBV cases in 2014 and 2015 in the UK, no clear indications were found for SBV circulation in the south of England in 2015. Among 1444 samples that were collected at 131 farms from six- to 12-month-old sheep in autumn 2015, only five ELISA-positive samples were found, and these all tested negative in confirmatory virus neutralisation tests. The authors consider that these ELISA results were most likely false positives and conclude that it is unlikely that SBV-specific antibodies were present in the tested sheep. However, the fact that the samples were collected late in the *Culicoides* vector season (November 2015) means the possibility remains that these ELISA-positive sheep are animals that became infected late in the season, possibly by SBV-infected *Culicoides* reaching the UK by wind, when the abundance of local *Culicoides* was already too low to transmit the virus further.

The lack of significant SBV circulation and associated losses during the past few years means governments, veterinarians and farmers may have become less aware of this disease and might think or hope that SBV will disappear. However, epidemiological data available for the closely related Akabane virus predicts otherwise. Since its initial isolation in 1959 (Oya and others 1961) low level Akabane virus circulation has been observed almost every year in Japan using sentinel animal and entomological surveillance (Kurogi and others 1976, Miyazato and others 1989, Yanase and others 2005, Kono and others 2008). Epizootics, however, tend to only occur at four to six years intervals, coinciding with a decrease in population immunity to the virus (Kono and others 2008, CFSPH 2016). No data are currently available on the level of population immunity that is still present against SBV in the ruminant population,



Photograph: CSIRO

One similarity between Schmallenberg virus and Akabane virus is that *Culicoides* species are the vector; the Akabane virus vector, *Culicoides brevitarsis*, is pictured

but it is to be expected that the susceptible population is growing each year due to the constant renewal of immune adults by susceptible newborns that have not been into contact with the virus and are only protected for a limited timespan by the passive immunity (Claine and others 2014, Elbers and others 2014, Veldhuis and others 2015, Poskin and others 2016). Five years after its initial emergence, this could shape ideal conditions for a renewed large scale re-emergence of SBV that would probably be followed by a new abortion storm. After a period of epidemiological silence, some indications pointing in this direction have already been reported. In Belgium, a limited number of SBV confirmed abortions in cattle have been found in spring 2016, evidencing an SBV circulation in autumn 2015. These were the first confirmed SBV cases in three years (Delooz and others 2016). During the summer of 2016, the Belgian reference laboratory for SBV at CODA-CERVA furthermore observed an increase in the number of SBV seropositive animals (unpublished data), suggesting an ongoing SBV circulation. Also Dutch agricultural media (www.nieuweoogst.nu/nieuws/2016/09/01/kans-bestaat-dat-schmallenberg-virus-opnieuw-toeslaat) and the website of Gezondheidszorg voor Dieren (www.gddiergezondheid.nl/actueel/nieuws/2016/08/schmallenbergvirus-vastgesteld-in-rund) have reported that SBV has circulated in 2015 and that it was detected by RT-PCR in blood samples collected from cattle at the end of July 2016 in the Netherlands. The extent of this

ongoing circulation is currently being studied in more detail and the real impact will probably only be seen at the end of this year and during spring of 2017 based on the number of aborted and malformed calves, lambs and goat kids that will be observed.

What we know about Akabane virus teaches us that we should not only be alert for renewed episodes of congenital malformations induced by SBV, but that also new virus variants might arise that are capable of inducing a different range of clinical signs. It has become clear that field isolates of Akabane virus exist that differ considerably in virulence and genetic and antigenic properties (Kono and others 2008). The most remarkable example of this was the emergence of the Iriki strain in 1984, 25 years after the first Akabane virus isolation (Miyazato and others 1989, Kono and others 2008). Compared to other strains that mostly affect unborn ruminants after transplacental transmission, the Iriki strain and

some other recently emerged strains are capable of inducing encephalomyelitis in cattle by postnatal infection (Kono and others 2008, Kamata and others 2009, Oem and others 2012). Since a similar potential for genetic variability has been reported for SBV (Coupeau and others 2013, Fischer and others 2013, Hulst and others 2013), we should remain vigilant for new emerging forms of SBV with an altered pathogenesis.

Despite the limited circulation of SBV during the past few years and the decrease in interest of governments in this disease (Dominguez and others 2014, Poskin and others 2016), it seems advisable to closely monitor the situation via different surveillance techniques to allow timely warnings to veterinarians and farmers and to remind them to remain alert for potential SBV-induced disease. SBV should continue to be included in the differential diagnosis of future epizootic outbreaks of pathologies of unknown origin in previously infected areas to identify new forms of the disease and in naive areas to monitor for SBV expansion.

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