Reemerging Schmallenberg Virus Infections, Germany, 2012

To the Editor: In 2011, Schmallenberg virus, a novel orthobunyavirus of the Simbu serogroup, emerged in Germany and the Netherlands and spread rapidly over large parts of central and western Europe (1-5). The infection primarily affects ruminants but affects camelids as well (1.6). So far, evidence has not shown that humans are susceptible to Schmallenberg virus infection (7). Although the infection in adult animals causes only mild symptoms (1) or remains clinically inapparent, in pregnant animals, transplacental transmission during a limited period can lead to the birth of severely malformed progeny (1,2). Acute infections of adult ruminants or malformed Schmallenberg virus-positive offspring have been detected on >5,000 farms in Austria, Belgium, Denmark, Finland, France, Ireland, Germany, Italy, Luxembourg, Norway, Poland, Spain, Sweden, Switzerland, the Netherlands, and the United Kingdom. Also, a high proportion of adult ruminants were seropositive for antigens of the virus in the core region affected by Schmallenberg virus in the Netherlands, Germany, and Belgium (2,4,5). Schmallenberg virus caused the first known outbreak of an infection with a virus of the Simbu serogroup in Europe. Schmallenberg virus infections are notifiable in Germany. Biting midges seem to play a key role in the transmission of the infection (8), and this transmission led to seasonal spread of the infection in summer and autumn 2011.

We report the recurrence of Schmallenberg virus infection in adult cattle, sheep, and a goat in Germany in 2012. Veterinary authorities at the county or town level report the animal holdings where laboratory-confirmed Schmallenberg virus infections are found to the central national database for notifiable animal diseases (Tierseuchennachrichtensystem), which is maintained by the Friedrich-Loeffler-Institut; the reports are made online. This database was analyzed for reported holdings with Schmallenberg virus infections that had been detected in adult animals from June 1, 2012 through October 31, 2012, and confirmed by PCR (9) or virus isolation. In total, 82 infections were reported in adult cattle, 8 in adult sheep, and 1 in a goat (Figure). Forty-five of the cattle holdings and 4 sheep holdings submitted samples for testing because the affected animals had shown clinical signs. One case was detected in a sheep flock, and 5 cases were detected in cattle in trade examinations. For the remaining cases, no specific reason for testing was reported.

Although some cases were reported from the region in western and northern Germany where the epidemic had its center in 2011 (Figure, panel A), several new infections occurred in regions in southern Germany where no cases or only few cases of Schmallenberg virus infection had been detected before (Figure, panel B). This phenomenon may have occurred because of a high level of protective immunity at the population level in the region affected before transmission resumed in 2012, although a substantial proportion of the animals at the margin of the affected area remained susceptible. Schmallenberg virus that has overwintered in these areas may thus be transmitted to naive animals and has apparently spread to regions in southern Germany that were not affected or were less affected by the previous Schmallenberg virus epidemic. Schmallenberg virus could also be introduced into neighboring countries through infected arthropods. Although the respective reports may not have been formally published, indications were that Schmallenberg virus had spread at least to Austria, Ireland, Finland, Norway, Poland, Sweden, and Switzerland by summer/autumn 2012.

Schmallenberg virus infection is often mild or clinically inapparent in adult animals and leads only to a short viremic period of $\approx 4-5$ days (1). Because a substantial proportion of new infections in adult animals are likely not recognized, the new cases reported in Germany starting in June

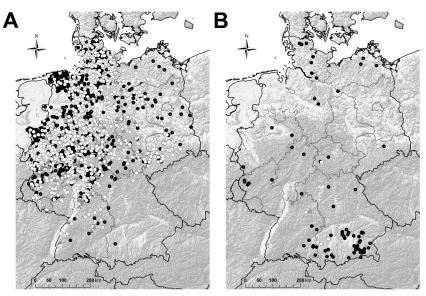


Figure. PCR-confirmed cases of Schmallenberg virus-infections in Germany in A) cattle (black circles, 791), sheep (white circles, 860), and goat holdings (triangles, 47) from August 1, 2011, through May 31, 2012, and B) cattle (black circles, 82), sheep (open circles, 8), and goat holdings (triangle, 1) from June 1, 2012, through October, 31, 2012. A color version of this figure is available online (wwwnc.cdc.gov/eiod/article/10/3/12-1324-F1.htm).

LETTERS

2012 probably represent only the socalled tip of the iceberg. Nevertheless, PCR analysis to detect Schmallenberg virus in samples from animals with clinical signs is a valuable method for identifying first cases in areas where Schmallenberg virus infections have not previously been found.

Acknowledgment

We gratefully acknowledge the farmers, diagnosticians in the regional laboratories, and veterinary officials who provided data on reemerging Schmallenberg virus cases.

Franz J. Conraths, Doris Kämer, Kathrin Teske, Bernd Hoffmann, Thomas C. Mettenleiter, and Martin Beer

Author affiliations: Friedrich-Loeffler-Institut, Greifswald-Insel-Riems, Germany

DOI: http://dx.doi.org/10.3201/eid1903.121324

References

- Hoffmann B, Scheuch M, Höper D, Jungblut R, Holsteg M, Schirrmeier H, et al. Novel orthobunyavirus in cattle, Europe, 2011. Emerg Infect Dis. 2012;18:469–72 http://dx.doi.org/10.3201/eid1803.111905.
- Garigliany MM, Bayrou C, Kleijnen D, Cassart D, Desmecht D. Schmallenberg virus in domestic cattle, Belgium, 2012. Emerg Infect Dis. 2012;18:1512–4 http:// dx.doi.org/10.3201/eid1809.120716.
- Muskens J, Smolenaars AJ, van der Poel WH, Mars MH, van Wuijckhuise L, Holzhauer M, et al. [Diarrhea and loss of production on Dutch dairy farms caused by the Schmallenberg virus]. [in Dutch] Tijdschr Diergeneeskd. 2012;137:112–5.
- Elbers AR, Loeffen WL, Quak S, de Boer-Luijtze E, van der Spek AN, Bouwstra R, et al. Seroprevalence of Schmallenberg virus antibodies among dairy cattle, the Netherlands, winter 2011–2012. Emerg Infect Dis. 2012;18:1065–71 http://dx.doi. org/10.3201/eid1807.120323.
- European Food Safety Authority. "Schmallenberg" virus: analysis of the epidemiological data and impact assessment. EFSA Journal. 2012;10: 2768. doi:10.2903/j.efsa.2012.2768. www.efsa. europa.eu.efsajournal.
- World Organisation for Animal Health. OIE technical factsheet, May 2012 [cited 2013 Jan 3]. 1. Schmallenberg virus. www. oie.int/.../A_Schmallenberg_virus.pdf

- Ducomble T, Wilking H, Stark K, Takla A, Askar M, Schaade L, et al. Lack of evidence for Schmallenberg virus infection in highly exposed persons, Germany, 2012. Emerg Infect Dis. 2012;18:1333–5 http:// dx.doi.org/10.3201/eid1808.120533.
- Rasmussen LD, Kristensen B, Kirkeby C, Rasmussen TB, Belsham GJ, Bødker R, et al. Culicoids as vectors of Schmallenberg virus. Emerg Infect Dis. 2012;18:1204–6 http://dx.doi. org/10.3201/eid1807.120385.
- Bilk S, Schulze C, Fischer M, Beer M, Hlinak A, Hoffmann B. Organ distribution of Schmallenberg virus RNA in malformed newborns. Vet Microbiol. 2012;159:236–8. http://dx.doi. org/10.1016/j.vetmic.2012.03.035

Address for correspondence: Franz J. Conraths, Friedrich-Loeffler-Institut, Federal Research Institute for Animal Health–Institute of Epidemiology, Seestrasse 55, Wusterhausen 16868, Germany; email: Franz.Conraths @fli.bund.de

Peritoneal Tuberculosis in a Pregnant Woman from Haiti, United States

To the Editor: A 29-year-old woman at 23 weeks' gestation during her first pregnancy came to our hospital's obstetrics clinic after 6 days of vaginal bleeding and abdominal pain. She had not experienced fever, sweats, weight loss, contractions, or other symptoms. She was otherwise healthy; she was taking no medications, but was taking iron and multivitamin supplements. She had legally immigrated to the United States from Haiti 8 months previously and had no known tuberculosis contacts. Physical examination disclosed brown vaginal discharge and a closed cervix. Obstetric ultrasound was normal, and vaginal swab samples were negative for Neisseria gonorrhea and Chlamydia trachomatis.

Over the ensuing 2 weeks, her vaginal bleeding and abdominal pain worsened. She was admitted to the hospital. Physical examination revealed vaginal bleeding, but her condition was otherwise unchanged. Routine laboratory studies were normal. Repeat obstetric ultrasound showed a viable fetus, ascites, and a 15×15 × 3-cm rind of echogenic material anterior to the uterus. This abnormality was in the upper abdomen, an area not imaged on her previous ultrasound. Abdominal magnetic resonance imaging revealed moderate ascites and a 21 \times 14 \times 3–cm omental mass of intermediate intensity on T1 and T2 sequences; there was no lymphadenopathy (Figure). A tiny left pleural effusion was seen on chest radiograph. Routine HIV and tuberculin skin test results had been negative 4 months previously, and pre-immigration examination results and chest radiograph had been normal

Fine-needle aspiration of the omental mass was nondiagnostic. The patient's vaginal bleeding and abdominal pain persisted, and her cervix dilated. She had an oral temperature of 38.9° Celsius. Exploratory laparotomy demonstrated a friable omental mass with implants on the small bowel; a partial omentectomy was performed at 26 weeks' gestation. During this procedure, the patient gave birth to a male infant.

Multiple granulomata, some containing acid-fast bacilli, were identified upon histologic examination of the omentum (Technical Appendix Figure, wwwnc.cdc.gov/EID/ article/19/3/12-1109-Techapp1.pdf). Transcription-mediated amplification of the specimen was positive for Mycobacterium tuberculosis rRNA; cultures later grew M. tuberculosis susceptible to all first-line antituberculosis medications. Sputum smears and cultures were not performed. The patient's treatment began with isoniazid, rifampin, ethambutol, and pyrazinamide; her fevers and abdominal pain