were seropositive. Because antibodies may be detectable 10 days–3 weeks after experimental infection for the first time (8), the presumed period of infection was between mid-January and mid-February. At this time, the highest temperatures again rose above 6°C for a few days (Figure 1, panel A).

Although the within-herd seroprevalence was >90% in ewes after confirmed or suspected SBV infection in 2011 (9), in this study, conducted during the cold season, only 12 (13%) of 90 tested sheep were positive by ELISA. Three animals seroconverted between mid-January and mid-February. Thus, SBV transmission appears to be possible at a low level, most likely because of the low activity of the involved insect vectors.

In addition to the SBV cases found on the sheep holding in Mecklenburg–Western Pomerania, an additional 52 confirmed SBV cases (defined as virus detection by qRT-PCR or isolation in cell culture) in adult ruminants were reported to the German Animal Disease Reporting System from January 1 through February 20, 2013 (Figure, panel B). Most affected animal holdings were located in Bavaria, but cases were also reported from Thuringia, Saxony, Brandenburg, Mecklenburg–Western Pomerania, Hesse, and Lower Saxony. In conclusion, transmission of SBV by hematophagous insects seems possible, even during the winter in central Europe, if minimum temperatures rise above a certain threshold for several consecutive days.

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Kerstin Wernike, Mareen Kohn, Franz J. Conraths, Doreen Werner, Daniela Kameke, Silke Hechinger, Helge Kampen, and Martin Beer

Author affiliations: Friedrich-Loeffler-Institut, Insel Riems, Germany (K. Wernike, M. Kohn, D. Kameke, S. Hechinger, H. Kampen, M. Beer); Friedrich-Loeffler-Institut, Wusterhausen, Germany (F.J. Conraths); and Leibniz Centre for Agricultural Landscape Research, Müncheberg, Germany (D. Werner)

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References

1. Hoffmann B, Scheuch M, Höper D, Jungblut R, Holsteg M, Schirrmier H, et al. Novel orthobunyavirus in cattle, Europe, 2011. Emerg Infect Dis. 2012;18:469–72. http://dx.doi.org/10.3201/eid1803.111905
2. Beer M, Conraths FJ, van der Poel WH. ‘Schmallenberg virus’—a novel orthobunyavirus emerging in Europe. Epidemiol Infect. 2013;141:1–8.
3. De Regge N, Deblauwe I, De Deken R, Vantieghem P, Madder M, Gysen D, et al. Detection of Schmallenberg virus in different Culicoides spp. by real-time RT-PCR. Transbound Emerg Dis. 2012;59:471–5. http://dx.doi.org/10.1111/tbed.12000
4. Rasmussen LD, Kristensen B, Kirkeby C, Rasmussen TB, Belsham GJ, Bodker R, et al. Culicoides vectors of Schmallenberg virus. Emerg Infect Dis. 2012;18:1204–6. http://dx.doi.org/10.3201/eid1807.120385
5. 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
6. Wernike K, Eschbaumer M, Breithaupt A, Hoffmann B, Beer M. Schmallenberg virus challenge models in cattle: infectious serum or culture-grown virus? Vet Res. 2012;43:84. http://dx.doi.org/10.1186/1297-9716-43-84
7. Viennet E, Garros C, Rakotoarivony I, Allene X, Gardes L, Lhoir J, et al. Host-seeking activity of bluetongue virus vectors: endo/exophagy and circadian rhythm of Culicoides in Western Europe. PLoS ONE. 2012;7:e48120. http://dx.doi.org/10.1371/journal.pone.0048120
8. Wernike K, Eschbaumer M, Schirrmier H, Blohm U, Breithaupt A, Hoffmann B, et al. Oral exposure, reinfection and cellular immunity to Schmallenberg virus in cattle.

Address for correspondence: Martin Beer, Friedrich-Loeffler-Institut, Suedufer 10, 17493 Greifswald, Insel Riems, Germany; email: martin.beer@fli.bund.de.
he was admitted to the hospital with fever (38.6°C) and chills.

Clinical examination showed extended cellulitis; the left leg was bright red, hot, shiny, swollen, and non-pitting. The patient’s leukocyte count was 23 × 10^9/L (reference <10 × 10^9/L) and C-reactive protein level was 332 mg/L (reference <5 mg/L). IV clindamycin and ceftriaxone were administered. Fever and other symptoms improved rapidly. Two consecutive blood cultures carried out before antimicrobial drug treatment were positive for *B. holmesii*, according to biochemical characteristics and molecular detection of the specific *B. holmesii* recA gene (1). Isolates in both cultures were susceptible to amoxicillin, macrolide antimicrobial drugs, cefoxitin, nalidixic acid, and ciprofloxacin and were resistant to cefotaxime and trimethoprim/sulfamethoxazole (Table; blood isolate, day 1). The antimicrobial drug regimen was changed to amoxicillin (6 g/day) for 14 days; the cellulitis resolved, and the patient was discharged.

Cellulitis in the right leg was diagnosed 2 weeks after the end of the previous treatment. Pristinamycin (3 g/day) was prescribed by the man’s physician but was ineffective. He was readmitted, and *B. holmesii* was again isolated in 2 new blood cultures; the organism was now resistant to cefoxitin (Table; blood isolate, day 24). Oral amoxicillin was initiated (6 g/day), without success, and after 1 week, IV ceftriaxone (2 g/day) was administered. *B. holmesii* was again isolated (isolate blood, day 33) from blood cultures despite amoxicillin treatment, and the antibiogram had the same resistance profile, except for amoxicillin (which was not determined). Because the patient was improving, IV ceftriaxone was maintained for 18 days, and he was discharged 5 days after the beginning of efficient antimicrobial drug therapy.

Two weeks after the end of the treatment, the patient was admitted to the hospital for bilateral pneumonia. Treatment with piperacillin/tazobactam and ciprofloxacin for 14 days (750 mg 2×/day) was initiated. *B. holmesii* was again isolated from blood; the bacterium had now acquired resistance to amoxicillin and nalidixic acid (Table; isolate blood, day 74). Nevertheless, ciprofloxacin treatment was continued. By real-time PCR targeting of IS481, *Bordetella* DNA was detected in nasopharyngeal swab (NPS) specimens (1), but the species could not be identified because of an insufficient amount of DNA. One month after the end of the treatment, the patient was recovering. Although the patient was asymptomatic, *B. holmesii* was isolated in a second NPS specimen. The isolate was sensitive to amoxicillin and macrolides and resistant to cefotaxime, nalidixic acid, trimethoprim, and trimethoprim/sulfamethoxazole (Table; isolate blood, day 105). Rituximab was discontinued, and relapse had not occurred after >1 year of follow-up.

*B. holmesii* was first described in 1995 (2); it was primarily isolated from the blood of immunocompromised patients, especially those with spleen dysfunction. Since 1999, *B. holmesii* has been detected during pertussis outbreaks in NPS specimens of patients with pertussis-like signs and symptoms (3–6). To our knowledge, the association between *B. holmesii* infection and rituximab treatment has been reported only once, in a renal transplant recipient, and *B. holmesii* nasal carriage was not tested for (7).

In this patient, the *B. holmesii* infection relapses definitively stopped after rituximab treatment was interrupted, which suggests a relationship between the 2 events and that patients receiving rituximab are at increased risk for severe infection (8). Interpretations of antimicrobial drug resistance are difficult because no breakpoints have been defined for this species, but MICs of the drugs showed changes in the resistance profile between infectious episodes (Table). These observations strongly suggest a heterogeneous population of bacteria and that resistance was acquired after antimicrobial drug treatment as described in the United Kingdom (9). The patient improved while receiving ceftriaxone, although, in vitro; the bacterium was found resistant to this antimicrobial drug family as reported (10). Thus, in vitro susceptibility testing and in vivo efficacy were discordant.

In conclusion, the patient’s nasal carriage and rituximab treatment may explain the recurrent infection. That the nasal carriage was the primary mode of transmission could not be proven because NPS specimens were not taken early enough. More studies

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**Table. Antimicrobial resistance profile of *Bordetella holmesii* isolates in vitro, France, December 2010–March 2011**

| Antimicrobial agent | Blood, d 1 | Blood, d 24 | Blood, d 33 | Blood, d 74 | NPS, d 105 |
|---------------------|------------|------------|------------|------------|------------|
| Amoxicillin         | <2         | <2         | NA#        | 16         | <2         |
| Cefoxitin           | <8         | >256       | >256       | >256       | >256       |
| Cefotaxime          | >32        | >32        | >32        | >32        | >32        |
| Nalidixic acid      | <16        | <16        | <16        | 64         | >256       |
| Trimethoprim        | >32        | >32        | >32        | >32        | >32        |
| Sulfamethoxazole    | >512       | >512       | >512       | >512       | >512       |

*MICs* corresponding to a drug resistance, which may reflect the general interpretation for nonfermenting bacteria, are in **boldface**. NPS, nasopharyngeal swab; NA, no available data.

†MICs were determined by E-test on Bordet–Gengou agar.

‡Site and day (d) of collection of isolate.
are needed to determine the role of nasal carriage in *B. holmesii* bacteremia. That no *B. holmesii* infections occurred after rituximab was stopped suggests that rituximab played a role in the recurrent infections. In cases of recurrent infection or bacteremia, nasal carriage should be assessed, and the interruption of rituximab should be considered by physicians.

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**Rickettsia africana in Amblyomma variecatum Ticks, Uganda and Nigeria**

To the Editor: *Rickettsia africana* is the most widespread spotted fever group (SFG) rickettsia in sub-Saharan Africa, where it causes African tick-bite fever (1), an acute, influenza-like syndrome. The number of cases in tourists returning from safari in sub-Saharan Africa is increasing (1). In western, central, and eastern sub-Saharan Africa, *R. africana* is carried by *Amblyomma variecatum* (Fabricius, 1794) ticks (2); usually associated with cattle, this 3-host tick also can feed on a variety of hosts, including humans (2). *R. africana* has not been reported in Uganda and rarely reported in Nigeria (3,4). Our objective was to determine the potential risk for human infection by screening for rickettsial DNA in *A. variecatum* ticks from cattle in Uganda and Nigeria.

In February 2010, ticks were collected from zebu cattle (*Bos indicus*) from 8 villages in the districts of Kabararamo (Adektar [1°81′N–33°22′ E], Awimon [1°66′N–33°04′ E], Kalo [9°38′ N–9°38′ E]), from 8 villages in the districts of Kabararamo (Adektar [1°81′N–33°22′ E], Awimon [1°66′N–33°04′ E], Kalo [9°38′ N–9°38′ E]), and from 3 villages (Mangar [9°14′N–8°93′ E], Odikara [1°91′N–33°30′ E], Angeta [1°87′N–33°10′ E]) in Uganda and, in June 2010, in 3 villages (Mangar [9°14′N–8°93′ E], Buff [9°43′N–9°10′ E], Tambes [9°38′N–9°38′ E]) in the Plateau State in Nigeria (Figure). This convenience sample was obtained as part of other ongoing research projects in both countries. Ticks were preserved in 70% ethanol and identified morphologically to the species level by using taxonomic keys (5). Because the anatomic features do not enable an objective assessment of the feeding status of adult male ticks, engorgement level was determined only in female tick specimens and nymphs.

**LETTERS**

**References**

1. Njamkepo E, Bonacorsi S, Debruyne M, Gibaud SA, Guillot S, Guiso N. Significant finding of *Bordetella holmesii* DNA in nasopharyngeal samples from French patients with suspected pertussis. J Clin Microbiol. 2011;49:4347–8. http://dx.doi.org/10.1128/JCM.01272-11

2. Weyant RS, Hollis DG, Weaver RE, Amin MFM, Steigerwalt AG, O’Connor SP, et al. *Bordetella holmesii* sp. nov., a new gram-negative species associated with septicaemia. J Clin Microbiol. 1995; 33:1–7.

3. Rodgers L, Martin SW, Cohn A, Budd J, Marcon M, Terranella A, et al. Epidemiologic and laboratory features of a large outbreak of pertussis-like illnesses associated with co-circulating *Bordetella holmesii* and *Bordetella pertussis*—Ohio, 2010–2011. Clin Infect Dis. 2013;56:322–31. http://dx.doi.org/10.1093/cid/cis888

4. Mooi FR, Bruisten S, Linde I, Reubsaat F, Heuvelman K, van der Lee S, et al. Characterization of *Bordetella holmesii* isolates from patients with pertussis-like illness in the Netherlands. FEMS Immunol Med Microbiol. 2012;64:289–91. http://dx.doi.org/10.1111/j.1574-695X.2011.00911.x

5. Kamiya H, Otsuka N, Ando Y, Odairez F, Yoshino S, Kawano K, et al. Transmission of *Bordetella holmesii* during pertussis outbreak, Japan. Emerg Infect Dis. 2012;18:1166–9. http://dx.doi.org/10.3201/eid1807.120130

6. Yih WK, Silva EA, Ida J, Harrington N, Lett SM, George H. *Bordetella holmesii*-like organisms isolated from Massachusetts patients with pertussis-like symptoms. Emerg Infect Dis. 1999;5:441–3. http://dx.doi.org/10.3201/eid0503.990317

7. Chambaraud T, Dickson Z, Ensergueix G, Barraud O, Essig M, Lacour C, et al. *Bordetella holmesii* bacteremia in a renal transplant recipient: emergence of a new pathogen. Transpl Infect Dis. 2012;14:E134–6. http://dx.doi.org/10.1111/tid.12009

8. Vidal L, Gaffar-Gvili A, Salles G, Dreyling MH, Ghielmini M, Hsu Schmitz SF, et al. Rituximab maintenance for the treatment of patients with follicular lymphoma: an updated systematic review and meta-analysis of randomized trials. J Natl Cancer Inst. 2011;103:1799–806. http://dx.doi.org/10.1093/jnci/djr418

9. Fry NK, Duncan J, Pike R, Harrison TG. Emergence of *Bordetella holmesii* infections in the United Kingdom 2010. In: Abstracts of the 9th International *Bordetella* Symposium, Baltimore, Maryland, USA; 2010 Sep 30–Oct 3. Abstract 98.