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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Address for correspondence: Martin Beer, Friedrich-Loeffler-Institut, Suedufer 10, 17493 Greifswald, Insel Riems, Germany; email: martin.beer@fli.bund.de.

Recurrent Bordetella holmesii Bacteremia and Nasal Carriage in a Patient Receiving Rituximab

To the Editor: We report a case of recurrent Bordetella holmesii bacteremia with 4 clinical manifestations: 3 episodes of cellulitis and 1 episode of pneumonia. The patient, a 67-year-old man, was admitted to the Pitie-Salpetriere hospital in Paris, France, in December 2010, for recurrent cellulitis in his left leg. Eleven years earlier, diffuse large B-cell lymphoma had been diagnosed, and he had undergone 7 chemotherapy courses. He also had received 2 autologous stem cell transplants. He was receiving maintenance treatment with intravenous (IV) rituximab every 3 months and IV immunoglobulin for hypogammaglobulinemia.

The first episode of cellulitis had occurred in his left leg 2 months before admission; the condition was treated with pristinamycin (3 g/day for 14 days), and the leg healed completely. Cellulitis recurred in his left leg 2 months later; it was again treated with pristinamycin (3 g/day) for 4 days in conjunction with fusidic acid. The cutaneous lesions worsened, and...
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 cefotaxime 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 NPS, 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, the 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***

<table>
<thead>
<tr>
<th>Antimicrobial agent</th>
<th>Blood, d 1</th>
<th>Blood, d 24</th>
<th>Blood, d 33</th>
<th>Blood, d 74</th>
<th>NPS, d 105</th>
</tr>
</thead>
<tbody>
<tr>
<td>Amoxicillin</td>
<td>&lt;2</td>
<td>&lt;2</td>
<td>NA#</td>
<td>16</td>
<td>&lt;2</td>
</tr>
<tr>
<td>Cefoxitin</td>
<td>&lt;8</td>
<td>&gt;256</td>
<td>&gt;256</td>
<td>&gt;256</td>
<td>&gt;256</td>
</tr>
<tr>
<td>Cefotaxime</td>
<td>&gt;32</td>
<td>&gt;32</td>
<td>&gt;32</td>
<td>&gt;32</td>
<td>&gt;32</td>
</tr>
<tr>
<td>Nalidixic acid</td>
<td>&lt;16</td>
<td>&lt;16</td>
<td>&lt;16</td>
<td>32</td>
<td>&gt;256</td>
</tr>
<tr>
<td>Trimethoprim</td>
<td>&gt;32</td>
<td>&gt;32</td>
<td>&gt;32</td>
<td>&gt;32</td>
<td>&gt;32</td>
</tr>
<tr>
<td>Sulfamethoxazole</td>
<td>&gt;512</td>
<td>&gt;512</td>
<td>&gt;512</td>
<td>&gt;512</td>
<td>&gt;512</td>
</tr>
</tbody>
</table>

*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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Liem Binh Luong Nguyen, Loïc Epelboin, Jean Gabarre, Marylin Lecso, Sophie Guillot, François Bricaire, Eric Caumes, and Nicole Guiso


12. Barraud O, Essig M, Lacour C, et al. Emerging infection by screening for rickettsial DNA in *A. variegatum* 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 Kameramado (Adektar [1°81′N–33°22′E], Awimon [1°66′N–33°04′E], Kalobo [1°88′N–33°25′E], Odidip [1°90′N–33°30′E], Ollilomo [1°75′N–33°38′E], and Dokolo (Atele [2°09′N–33°16′E], and Angeta [1°87′N–33°10′E]) in Uganda and, in June 2010, in 3 villages (Mangar [9°14′N–8°93′E], Ruff [9°43′N–9°10′E], and 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 anatonic 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.

Rickettsia africana in Amblyomma variegatum 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 (*A. variegatum*), 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 variegatum* (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. variegatum* 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 Kameramado (Adektar [1°81′N–33°22′E], Awimom [1°66′N–33°04′E], Kalobo [1°88′N–33°25′E], Odidip [1°90′N–33°30′E], Odikara [1°91′N–33°30′E], and Ollilomo [1°75′N–33°38′E], and Dokolo (Atele [2°09′N–33°16′E], and Angeta [1°87′N–33°10′E]) in Uganda and, in June 2010, in 3 villages (Mangar [9°14′N–8°93′E], Ruff [9°43′N–9°10′E], and 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 anatonic 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.