species. However, the new spirochete is distinct from all other known Borrelia spp. with an available 16S rDNA sequence in the GenBank database. Its partial 16S rDNA sequence differed by 3.3% to 4.2% from 9 LB group species and 2.4% to 3.4% from 15 relapsing fever group species. For comparison, intragroup sequence differences were ≤1.9%. On this basis, as well as the finding of partial IGS sequences (GenBank accession nos. AY668955 and AY668956) that were unique among all Borrelia spp. studied to date (3,5), we propose that this is a new species of Borrelia, provisionally named Borrelia davisi in honor of Gordon E. Davis for his contributions to Borrelia research and taxonomy.

While the new species was detected in 8 of 131 Peromyscus leucopus blood samples by using PCR for the IGS, the assays for this organism in the DNA extracts of 282 I. scapularis nymphs (4) from the same geographic site were uniformly negative (p = 0.0003, 2-sided Fisher exact test). This finding suggests that the new spirochete has another vector. The only other documented tick species that has been found feeding in small numbers on P. leucopus in Connecticut is Dermacentor variabilis (7). Holden et al. reported the presence of Borrelia in D. variabilis ticks in California by using PCR with genus-specific primers, but the species in these ticks was not identified by sequencing (8).

Although how B. miyamotoi and B. davisi affect the health of humans and other animals remain to be determined, our finding of 3 Borrelia species with overlapping life cycles in the same host in the same area shows that the ecology of Borrelia is more complex than was imagined. The presence of species other than B. burgdorferi in a major reservoir will have to be considered in future surveys and interventions.

Acknowledgments

We thank Hany Mattaous and Lili Sheibani for technical assistance and Jean Tsao, Klara Hanincova, and Durland Fish for discussions and ongoing collaborative studies.

This work was supported by grants to AGB (AI37248 from the National Institutes of Health) and to JB (Cooperative Agreement 919558-01 from the Centers for Disease Control and Prevention).

Jonas Bunikis* and Alan G. Barbour*

*University of California Irvine, Irvine, California, USA

References

1. Levine JF, Wilson ML, Spielman A. Mice as reservoirs of the Lyme disease spirochete. Am J Trop Med Hyg. 1985;34:355–60.
2. Scoles GA, Papero M, Beati L, Fish D. A relapsing fever group spirochete transmitted by Ixodes scapularis ticks. Vector Borne Zoonotic Dis. 2001;1:21–34.
3. Bunikis J, Tsao J, Garpmo U, Berglund J, Fish D, Barbour AG. Typing of Borrelia relapsing fever group strains. Emerg Infect Dis. 2004;10:1661–4.
4. Tsao JT, Wootton JT, Bunikis J, Luna MG, Fish D, Barbour AG. An ecological approach to preventing human infection: vaccinating wild mouse reservoirs intervenes in the Lyme disease cycle. Proc Natl Acad Sci U S A. 2004;101:18159–64.
5. Bunikis J, Garpmo U, Tsao J, Berglund J, Fish D, Barbour AG. Sequence typing reveals extensive strain diversity of the Lyme borreliosis agents Borrelia burgdorferi in North America and Borrelia afzelii in Europe. Microbiology. 2004;150:1741–55.
6. Barbour AG, Maupin GO, Teltow GJ, Carter CJ, Piesman J. Identification of an uncultivable Borrelia species in the hard tick Amblyomma americanum: possible agent of a Lyme disease-like illness. J Infect Dis. 1996;173:403–9.
7. Stafford KC, III, Bladen VC, Magnarelli LA. Ticks (Acarai: Ixodidae) infesting wild birds (Aves) and white-footed mice in Lyme, CT. J Med Entomol. 1995;32:453–66.
8. Holden K, Boothby JT, Anand S, Massung RF. Detection of Borrelia burgdorferi, Ehrlichia chaffeensis, and Anaplasma phagocytophilum in ticks (Acarai: Ixodidae) from a coastal region of California. J Med Entomol. 2003;40:534–9.

Address for correspondence: Jonas Bunikis, Department of Microbiology and Molecular Genetics, B240 Medical Sciences I, University of California Irvine, Irvine, CA 926974025, USA; fax: 949-824-6452; email: jbunikis@uci.edu
LETTERS

selected on the basis of International Classification of Diseases, Ninth Revision, Clinical Modification (ICD-9-CM) codes (4) as defined by the Department of Defense (5). Laboratory data were not part of SIDRs and SADRs but were part of CHCS.

For records with diagnostic codes relating to any of the 4 diseases of interest, laboratory records were searched to determine: 1) whether the provider ordered an appropriate test or tests and 2) if these were ordered, were the test results confirmatory (positive). Appropriate and confirmatory test results were determined by using published references (5–7) and local laboratory practices. For malaria, a blood smear was considered an appropriate test with a positive blood smear accepted as confirmatory (5,6). We considered both nontreponemal and treponemal tests to be appropriate for syphilis but only a positive treponemal test as confirmatory (5,6). For acute hepatitis B, we considered hepatitis B surface antigen or immunoglobulin (Ig) M anti-hepatitis B core (anti-HBc) to be an appropriate test, but only a positive IgM anti-HBc was accepted as confirmatory (5,6). We considered enzyme immunoassay total antibody screens or Western blot (WB) IgG or IgM tests to be appropriate for Lyme disease and accepted any positive test as confirmatory (5–7). \( \chi^2 \) calculations were conducted \((\alpha = 0.05)\).

Twenty-one SIDRs and 155 SADRs met the selection criteria (Table). While 61.9% of SIDRs studied had appropriate laboratory tests ordered, only 19.0% had associated confirmatory results in CHCS. For outpatient records, 64.5% had appropriate tests ordered, and 15.5% had confirmatory results. Among the SADRs, the proportions of appropriate laboratory tests for the diseases studied differed significantly (summary \( \chi^2 = 11.5, p = 0.01 \)). These results suggest that tracking electronic SADR and SIDR datasets for the selected reportable diseases could produce a high number of false-positive reports; in this study, 81.0% of inpatient and 84.5% of outpatient reports would lack a confirmatory laboratory test result.

This initial evaluation is limited but supports the need to evaluate electronic datasets before using them for medical surveillance. We examined only ICD-9-CM coded records of selected diseases from 1 geographic area, with resulting small samples. Therefore, our results may not be generalizable. This study was restricted to laboratory, inpatient, and outpatient data recorded within 1 coordinated military system. Laboratory testing or clinical visits may have occurred outside of this network and may not have been captured in this study. Laboratory data were not recorded or stored in a standardized format in CHCS, increasing the likelihood of misclassification. We did not evaluate all related sources of data, including the hard-copy clinical records, so we do not know the completeness of the ICD-9-CM codes or the extent of ICD-9-CM code misclassification. Additionally, local clinical practices in terms of both ordering laboratory tests and coding diagnoses for the diseases studied were not defined.

Future studies would benefit from comparing reported medical events, paper medical records, and electronic datasets to include determination of sensitivity as well as positive predictive value (2,8,9). Discordance in these data sources should be investigated for miscoding, incomplete data, and unexpected clinical practices.

Efforts to improve medical record coding at military medical treatment facilities are under way (10). Additionally, standardization of CHCS laboratory test files, including adoption of the Logical Observation Identifiers Names and Codes system for standardized reporting of test names, is under way (available from http://www.ha.osd.mil/policies/2003/03-023.pdf). However, a documented, complete, reliable, and closely monitored single source of data for medical surveillance and disease reporting does not currently exist. Therefore, surveillance programs for infectious diseases in the US military should include monitoring of multiple, related sources of data and information (e.g., electronic inpatient and

| Disease | Inpatient records (SIDR) | Outpatient records (SADR) |
|---------|--------------------------|--------------------------|
|         | No. records selected | No. tests ordered (%) | No. confirmatory results (%) | No. records selected | No. tests ordered (%) | No. confirmatory results (%) |
| Malaria | 3 | (100.0) | 1 (33.3) | 17 | 8 (47.1) | 1 (5.9) |
| Syphilis | 1 | (100.0) | 1 (100.0) | 44 | 31 (70.4) | 12 (27.3) |
| Acute hepatitis B | 16 | 8 (50.0) | 1 (6.3) | 39 | 32 (82.1) | 5 (12.8) |
| Lyme disease | 1 | 1 (100.0) | 1 (100.0) | 55 | 29 (52.7) | 6 (10.9) |
| Total | 21 | 13 (61.9) | 4 (19.0) | 155 | 100 (64.5) | 24 (15.5) |

*This table presents Standard Inpatient Data Records (SIDR) and Standard Ambulatory Data Records (SADR) studied and percentages with appropriate laboratory tests ordered and confirmatory laboratory test results.

| Disease | Inpatient records (SIDR) | Outpatient records (SADR) |
|---------|--------------------------|--------------------------|
|         | No. records selected | No. tests ordered (%) | No. confirmatory results (%) | No. records selected | No. tests ordered (%) | No. confirmatory results (%) |
| Malaria | 3 | (100.0) | 1 (33.3) | 17 | 8 (47.1) | 1 (5.9) |
| Syphilis | 1 | (100.0) | 1 (100.0) | 44 | 31 (70.4) | 12 (27.3) |
| Acute hepatitis B | 16 | 8 (50.0) | 1 (6.3) | 39 | 32 (82.1) | 5 (12.8) |
| Lyme disease | 1 | 1 (100.0) | 1 (100.0) | 55 | 29 (52.7) | 6 (10.9) |
| Total | 21 | 13 (61.9) | 4 (19.0) | 155 | 100 (64.5) | 24 (15.5) |

#Summary \( \chi^2 = 11.5; p = 0.01 \).

#Summary \( \chi^2 = 7.0; p = 0.07 \).
outpatient encounters, laboratory results, and pharmacy data). All of these sources should be evaluated for completeness and accuracy.

Acknowledgments
We thank the Army Medical Surveillance Activity and TriCare Management Activity for data support; and Shilpa Hakre, Danielle Dell, Cara Olsen, and Julie Pavlin for manuscript review.

This study was supported in part by the Department of Defense Global Emerging Infections Surveillance and Response System, Silver Spring, Maryland.

Asha J. Riegodedios,* Anuli Ajene,* Mark A. Malakooti,* Joel C. Gaydos,† Victor H. MacIntosh,‡ and Bruce K. Bohnker*†

*Department of Defense Global Emerging Infections Surveillance and Response System, Silver Spring, Maryland, USA; †Department of Defense Global Emerging Infections Surveillance and Response System, Silver Spring, Maryland, USA; ‡Naval Environmental Health Center, Portsmouth, Virginia, USA.

References
1. Thomas RJ, MacDonald MR, Lenart M, Calvert WB, Morrow R. Moving toward eradication of syphilis. Mil Med. 2002;167:489–95.
2. Bond MM, Yates SW. Sexually transmitted disease screening and reporting practices in a military medical center. Mil Med. 2000;165:470–2.
3. Meyer GS, Krakauer H. Validity of the Department of Defense standard inpatient data record for quality management and health service research. Mil Med. 1998;163:461–5.
4. International Classification of Diseases, Ninth Revision, Clinical Modification. Dover (DE): American Medical Association; 1997.

Concurrent Dengue and Malaria

To the Editor: A 37-year-old woman, a logistics director for a non-government organization, returned to France in March 2004 from an 18-day trip to Guinea, Senegal, and Sierra Leone. Fever, chills, and myalgia developed in the woman 3 days before she returned to France, and she treated herself with aspirin and paracetamol (acetaminophen). Malaria prophylaxis was taken neither during nor after the trip.

The day after returning to France, the woman’s condition progressively worsened; diarrhea and extreme weakness that led to the inability to walk developed. Ten days after her return, she was admitted to the local hospital and treated with intravenous quinine and oral doxycycline (2 g per day) after thick and thin blood films showed 3% parasitemia with *Plasmodium falciparum*. Three days later, she was still febrile and had conjunctival jaundice, vomiting, insomnia, and moderate hemorrhagic manifestations (epistaxis, blood in urine and feces). Three days after initial hospitalization, the patient was transferred to the Infectious Diseases Unit in Marseille; fever (39.5°C) continued, and hepatosplenomegaly developed. Biologic analyses showed disseminated intravascular coagulation with platelet count of 22,000/µL, an elevated prothrombin time (54% higher than the control value), a longer activated clotting time (51 seconds versus a control value of 34 seconds), a fibrinogen level of 0.9 g/dL, exaggerated plasma fibrin formation and degradation, and hepatic cytolysis with both aspartate aminotransferase and alanine aminotransferase levels of 80 U/L.

Although acute malaria had been diagnosed, viral serologic tests were performed because the patient had returned from a tropical country with a fever. Persons in these circumstances are systematically administered a series of tests to determine the cause of their fever. Serologic tests for dengue performed on the acute-phase serum (collected 13 days after onset of symptoms) and convalescent-phase serum (collected 23 days after onset of symptoms) showed the presence of immunoglobulin (Ig) M (titers 1:800 and 1:3,200, respectively) and IgG (titers 1:400 and 1:3,200, respectively), which suggested that the patient had dengue fever and malaria concurrently. These results were obtained by using the Dengue Duo IgM-capture and IgG-indirect enzyme-linked immunosorbent assay (Biotrin,