Borrelia garinii in Seabird Ticks (Ixodes uriae), Atlantic Coast, North America

Robert P. Smith, Jr,* Sabir Bin Muzaffar,† Jennifer Lavers,† Eleanor H. Lacombe,* Bruce K. Cahill,* Charles B. Lubelczyk,* Allen Kinsler,* Amy J. Mathers,* and Peter W. Rand*

*Borrelia garinii is the most neurotropic of the genospecies of B. burgdorferi sensu lato that cause Lyme disease in Europe, where it is transmitted to avian and mammalian reservoir hosts and to humans by Ixodes ricinus. B. garinii is also maintained in an enzootic cycle in seabirds by I. uriae, a tick found at high latitudes in both the Northern and Southern Hemispheres. To determine whether B. garinii is present in seabird ticks on the Atlantic Coast of North America, we examined 261 I. uriae ticks by polyclonal antiborrelial fluorescent antibody. Ten of 61 ticks from Gull Island, Newfoundland, were positive for borreliae by this screen. Amplicons of DNA obtained by PCR that targeted the B. garinii rrs-rrla intergenic spacer were sequenced and matched to GenBank sequences for B. garinii. The potential for introduction of this agent into the North American Lyme disease enzootic is unknown.

In Europe, Lyme disease is caused by 3 genospecies of Borrelia burgdorferi (i.e., B. afzelii, B. garinii, B. burgdorferi sensu stricto), while in North America only B. burgdorferi sensu stricto, the most neurotropic of these 3 genospecies, causes most neurologic Lyme disease in Europe, including cases of meningopolyneuritis and, rarely, encephalomyelitis (1–3). The presence of multiple pathogenic genospecies that cause Lyme disease in Europe complicates serodiagnostic testing (4).

In Eurasia, B. garinii is transmitted to avian and rodent hosts and to humans by I. ricinus, the sheep or forest tick, and I. persulcatus, the taiga tick (5–9). I. uriae, the seabird tick, also maintains this agent in a “silent” enzootic cycle in seabirds at their nesting sites over a wide but discontinuous area (10–13). Although these 2 enzootic cycles are generally geographically and ecologically separate, interchange of B. garinii strains may occur at sites where both vectors coexist (14). The risk for seabird-associated strain types of B. garinii to cause Lyme disease, however, is not known (15).

Although B. garinii is present in seabird ticks in a nearly circumpolar distribution in both the Northern and Southern Hemispheres (12,13), including Alaska, the presence of B. garinii in I. uriae ticks at sites on the North Atlantic Coast has not previously been documented. We sought to determine whether B. garinii is present in ticks obtained from colonial seabird nesting sites on the Atlantic Coast of North America.

Methods

Seabird researchers working at several sites on the Atlantic Coast in the United States and Canada (Figure 1) submitted I. uriae ticks to our laboratory for analysis (16). Ticks were identified to species, stage, and degree of engorgement (17). A subset of ticks was dissected, and midguts were screened for spirochetes by fluorescent microscopy by using a polyclonal antiborrelial antibody (18).

DNA was extracted from Borrelia-positive ticks by using the Qiagen DNeasy Tissue Kit (Qiagen, Valencia, CA, USA). DNA amplification was performed in a designated room with genus-specific primers that include the partial sequence of rrs-rrla intergenic spacer region as described by Bunikis et al. (19) with use of negative con-
trols. Amplification products were viewed on a 1% agarose gel containing 0.5 µg/mL ethidium bromide. At a second laboratory, ticks positive by fluorescent antibody screen were prepared as above for DNA extraction, and PCR was performed by using primers directed at the 16S ribosomal DNA. Sequences of amplicons obtained at both laboratories were confirmed to be *B. garinii* by comparison with known sequences in the GenBank database.

**Results**

*I. uriae* ticks submitted from 6 seabird colony sites and several hosts in the United States and Canada were processed (Table). Through 2005, 880 *I. uriae* ticks recovered primarily from Atlantic puffin chicks or their nests, were submitted from Maine sites, and another 383 were submitted from sites on the Atlantic Coast of Canada. Over 200 ticks from Maine sites and 61 ticks from Canadian sites off the coast of Newfoundland and Labrador were screened for borreliae by fluorescent microscopy. Spirochetes were detected only in ticks from Gull Island, Newfoundland (47° 15′ N, 52° 46′ W), where 9 of 22 adults and 1 of 39 nymphal ticks were positive. DNA was extracted from 2 of these ticks, and PCR showed a 1,900-base amplicon of the rrs-rrls intergenic spacer region that matched with *B. garinii* on comparison with GenBank sequences. Two additional ticks were examined in the laboratory of Sam Telford (Tufts University School of Veterinary Medicine, Grafton, MA, USA) by means of PCR targeting of 16S ribosomal DNA and again confirmed a match for *B. garinii* (GenBank bankit no. 800902 DG463373). Figure 2 shows the sequence from one of the ticks shown in an alignment with sequences from *B. burgdorferi* strain B31 and a representative *B. garinii* sample from GenBank.

**Discussion**

The finding of *B. garinii* in *I. uriae* ticks from Gull Island, Newfoundland, adds to the known distribution of this agent and increases the likelihood that this agent is present in other colonial seabird nesting sites on the Atlantic Coast of North America, as is the case in Europe. The recent emergence of *I. scapularis* in coastal Maine and some Maritime Canadian sites (20,21) brings these 2 enzootic cycles of different genospecies of *B. burgdorferi* into proximate areas, although their ecologic settings differ. The public health importance of this finding depends on the probability of the introduction of *B. garinii* into emergent *I. scapularis*-vectored *B. burgdorferi* s.s. cycles and its potential maintenance in this cycle. The public health effects also depend on the pathogenic potential for human disease caused by seabird-associated strains of *B. garinii* (15).

The remote geographic and, at some sites, isolated topographic location of colonial seabird colonies provide significant barriers to the introduction of *B. garinii* into other vector ticks and their reservoir hosts. *I. scapularis* is

---

**Table. Submissions of *Ixodes uriae* from coastal Maine (USA) and Canada, 1996–2005**

<table>
<thead>
<tr>
<th>Site</th>
<th>Host species</th>
<th>Atlantic puffin (Fratercula arctica)</th>
<th>Murre (uria aalage/Razorbill (Alca torda)</th>
<th>Black guillemot (Cephus grille)</th>
<th>Herring gull (Larus argentatus)</th>
<th>Common eider (Somateria mollissima)</th>
<th>On humans</th>
<th>Flag/drug sampling</th>
<th>Soil/litter sample</th>
</tr>
</thead>
<tbody>
<tr>
<td>Machias Seal Island</td>
<td></td>
<td>218</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Matinicus Rock*</td>
<td></td>
<td>258</td>
<td>23</td>
<td>25</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Petit Manan Island*</td>
<td></td>
<td>56</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Seal Island*</td>
<td></td>
<td>46</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Gannet Island†</td>
<td></td>
<td>111</td>
<td>88</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Gull Island‡</td>
<td></td>
<td>22</td>
<td></td>
<td>22</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*Maine, USA.
†Labrador, Canada.
‡Newfoundland, Canada.
dispersed to remote coastal islands of the North American Atlantic Coast during bird migration (22,23), but its establishment at these sites requires the presence of deer (24–26). With rare exception, deer are absent from sites with large seabird colonies, which are usually limited to offshore islands. Dispersal of infected I. uriae to proximate coastal areas by seabird hosts is unlikely because most species of seabirds parasitized by I. uriae are highly philopatric and forage at sea. One exception might be gulls, which may move between coastal and island sites. Passerine birds, which may forage near seabird colonies, provide another potential mechanism for dispersal of B. garinii, either through movement of infected I. uriae ticks or by serving as reservoir hosts of this agent. However, the frequency of parasitism of passerine birds by I. uriae ticks is unknown.

If B. garinii was introduced into I. scapularis ticks, its maintenance in this cycle would depend on the vector competence of I. scapularis for B. garinii, the reservoir competence of available hosts, and perhaps the population genetics and strain diversity of B. garinii (14,27). Although I. scapularis is vector-competent for transmission of B. garinii to rodents, its efficiency of transmission appears lower than for B. burgdorferi s.s. (28). In addition, the vector competence of I. scapularis for seabird-associated strains of B. garinii has not been tested. The presence of similar ribotypes of B. garinii in I. ricinus ticks on a European island suggests that interchange of different B. garinii strains in different ecologic cycles may occur (14).

To determine the public health importance of B. garinii in seabird colonies along the North Atlantic coast, additional studies on the issues of dispersal, vector competence, and reservoir host competence are needed. All strain types of B. garinii may not be pathogenic for humans, and future studies should also address the potential for seabird-associated strains to cause disease.

Acknowledgments
We thank Anthony Diamond of the University of New Brunswick, Linda Welch of the Maine Coastal Islands National Wildlife Refuge, and Stephen Kress of the National Audubon Society, and their staffs, for the collection of I. uriae from Machias Seal Island, Petit Manan Island, Seal Island, and Matinicus Rock during the past 12 years. We are also indebted to Jonas Bunikis, Sam R. Telford III, Heidi K. Goethert, Barbara Conley, and Cal Vary for their assistance in confirming the genetic identity of B. garinii.

Dr Smith is an infectious disease physician and codirector of the Vector-borne Disease Laboratory at the Maine Medical Center Research Institute. His research interests include the ecology of Lyme disease emergence and the strain diversity of B. burgdorferi.

References


Address for correspondence: Robert P. Smith, Vector-borne Disease Laboratory, Maine Medical Center Research Institute 75 John Roberts Rd, Suite 9B, South Portland, ME 04106, USA; email: smithr@mmc.org

The opinions expressed by authors contributing to this journal do not necessarily reflect the opinions of the Centers for Disease Control and Prevention or the institutions with which the authors are affiliated.