Deer Ticks (*Ixodes scapularis*) and the Agents of Lyme Disease and Human Granulocytic Ehrlichiosis in a New York City Park

Rodent trapping and drag sampling in Van Cortlandt Park, New York City, yielded all stages of *Ixodes scapularis*, the deer tick vector of Lyme disease and human granulocytic ehrlichiosis (HGE). Polymerase chain reaction analyses of the ticks showed *Borrelia burgdorferi* and the *Ehrlichia* sp. that causes HGE.

Lyme disease, a tick-borne spirochete transmitted by the deer tick (*Ixodes scapularis* Say), was reported from 46 states in the United States in 1996; for the past 6 years, an average of 20% of those cases have been from Westchester and Suffolk Counties, New York (1). While spread of the deer tick population in New York State has been documented north and west of Westchester County (2), movement of ticks southward toward New York City has been largely ignored, despite rising Lyme disease case numbers in southern Westchester and a relatively high incidence of human parasitism by vector ticks. The discovery in Westchester County of human granulocytic ehrlichiosis (HGE), a second, potentially fatal, tick-borne disease (3), and of the causative *Ehrlichia equi*-like rickettsial agent in *I. scapularis* (4) highlights the significance of defining the geographic range of the deer tick. This is particularly important in urban areas, where residents may not be familiar with tick-borne diseases common in nearby suburban and rural areas. Foci of Lyme disease can occur in forested urban areas, as well as in rural sites, if ticks and their hosts are present (5).

Because of the proximity of Van Cortlandt Park to areas of southern Westchester where *I. scapularis* have been collected (Falco, unpub. data), the park’s relatively large wooded area (approximately 60% of 468 ha), and the wide range of vertebrate hosts on which this tick feeds, we examined rodents live-trapped in the park to determine if deer ticks were present.

For one night in August 1995, during the period of larval *I. scapularis* activity, trapping was conducted on five study grids, each 50 m by 50 m. Fifty Sherman mouse traps (H.B. Sherman, Tallahassee, FL) and nine Tomahawk traps (Tomahawk Live Traps, Tomahawk, WI) for larger mammals were baited and placed on each grid. Mean distance between neighboring grids was 400 m, and all were located in the northern half of the park, where woodland is concentrated. Captured animals were lightly anesthetized with methoxyflurane (Metofane, Pitman-Moore, Mundelein, IN); they were examined, and their age, sex, and weight were determined. All ectoparasites were collected, identified, and counted. Animals were released at the capture site after recovery from anesthesia.

The presence of this tick species in the park might lead to a Lyme disease or HGE focus and, therefore, the need for additional surveillance efforts. To further evaluate the risk for park visitors from infected ticks, host-seeking ticks were sampled in the summer (July 1996), when nymphal *I. scapularis* were active. Drag sampling, in which a 1 m² panel of white corduroy cloth is pulled along the ground and over vegetation to collect host-seeking ticks, was conducted. Any ticks found on the drag cloth or on researchers were removed with forceps, placed in a glass vial, and held live until identification. Specimens were stored in 70% ethanol until testing.

Polymerase chain reaction (PCR) analysis was conducted on all ticks collected in 1995 and 1996 (6). For nymphal and adult ticks, each specimen was dissected with sterile needles, and DNA was extracted by the Isoquick DNA extraction kit (ORCA Research, Bothell, WA), according to manufacturer’s directions. Final DNA pellets were suspended in 50 µl of sterile water. Each tick extract was tested for *B. burgdorferi* and the HGE agent by PCR amplification of a 10 µl aliquot.

Even though neither *B. burgdorferi* nor other ehrlichiae are efficiently transovarially
transmitted (7,8), white-footed mice are competent reservoirs of both agents (9). Given the likelihood that transovarial transmission of the Ehrlichia sp. causing HGE is extremely low, larval *I. scapularis* collected from mice might have acquired either agent while feeding. Seven of the nine larvae removed from hosts were tested in pools of two (n = 2) or three (n = 1) specimens; the remaining two larvae were tested individually. Larvae were pooled only with ticks that had been removed from the same host animal. Larval specimens were likewise dissected in a tube, and DNA was extracted as described above.

B. burgdorferi-specific PCR targeted the spacer region between duplicated 23S rRNA genes with primer IS1 and IS2 (10). Amplified products were electrophoresed on a 1.5% agarose gel, and DNA was transferred to nylon membranes hybridized with a B. burgdorferi-specific probe (P19) (10). The HGE agent was detected by amplifying a 151 bp fragment of 16S rDNA with primers GER3 and GER4 (11). PCR products were resolved by electrophoresis on 2% agarose gels and visualized by staining with ethidium bromide.

Of 33 captured mammals examined in the summer of 1995, 19 were white-footed mice (*Peromyscus leucopus*), the primary reservoir of *B. burgdorferi*. Four (21%) mice hosted *I. scapularis*; two mice each hosted a single larva, one hosted two larvae, and one mouse hosted five larvae. Mice that hosted ticks were captured on three of the five trapping grids. Examination of the nine larvae removed from hosts was negative for *B. burgdorferi* or other tick species.

To evaluate the relative density of host-seeking *I. scapularis*, i.e., unattached ticks available to parasitize a passing host, 5,840 m² of woodland habitat was drag sampled on or adjacent to the five trapping grids. One nymphal and two adult male *I. scapularis* were collected, along with a single *I. dentatus* nymph.

Results of PCR analyses indicated that one pool of two larvae, removed from a white-footed mouse that hosted five *I. scapularis*, was positive for the Ehrlichia agent of HGE. Of the four host-seeking ticks examined, two male *I. scapularis* were infected with *B. burgdorferi*; the single *I. dentatus* nymph was not infected with either agent. No specimens were infected with both agents. The primers used to amplify the HGE agent DNA would also yield PCR product with the closely related *E. platys* (an agent of canine ehrlichiosis) and a recently described Ehrlichia species from white-tailed deer (12). However, neither of these bacteria has been reported in hosts in the northeastern United States, nor are the invertebrate vectors known (although *Amblyomma americanum* is a suspected vector of the deer *Ehrlichia*) (12,13). Since the prevalence rate of the HGE agent in *I. scapularis* collected in Westchester County, New York, is approximately 20% (14), it is reasonable to conclude that in the current study, positive PCR results reflect the presence of the HGE agent.

Although anecdotal reports of Lyme disease by New York City residents who have not traveled to disease-endemic sites have previously suggested the presence of *I. scapularis* within city limits, to our knowledge, this is the first instance in which the deer tick has been confirmed on wildlife hosts resident in the city. Examinations of tick collections at both the American Museum of Natural History, New York (L.N. Sorkin, pers. comm.), and the U.S. National Tick Collection at the Institute of Arthropodology and Parasitology, Georgia Southern University (L.A. Durden, pers. comm.), also indicate that no specimens of *I. scapularis* previously collected from wildlife in New York City have been deposited.

These findings have several implications. First, the distribution of infested hosts suggests at least three potential tick population foci within Van Cortlandt Park. From these, a growing tick population may develop. Second, the larvae collected in this study were likely derived from eggs laid by replete females in the park. Thus, it is probable that host-seeking adults had successfully found medium- to large-sized mammals on which to feed during the previous adult season. The collection of nymphs and adult ticks further supports our conclusion that a population of *I. scapularis* is established in Van Cortlandt Park, though at a low level. By comparison, average drag densities at a woodland site in central Westchester County typically are one nymph per approximately 16 m² during a comparable period in the nymphal activity cycle (Daniels and Falco, unpub. data). Third, the potential exists for increased exposure to the agents of Lyme disease and HGE by park visitors. Rather than creating the peri-domestic exposure that marks suburban habitats, tick populations in urban areas will likely result in more focal exposure, restricted to woodland habitat "islands."
which exist primarily as parkland. The presence of white-tailed deer (*Odocoileus virginianus*) in such parks, even if it occurs on an intermittent, seasonal basis (as appears to be the case in Van Cortlandt Park), may serve to introduce new ticks into the park from adjoining disease-endemic areas. In this case, Westchester County is the apparent source of the sporadic deer migration. In addition, the presence of deer in the park can feed host-seeking ticks active at that time and thereby help increase the tick population. Even in the absence of white-tailed deer, the preferred host of adult *I. scapularis*, small populations of deer ticks may be maintained by medium-sized mammals such as raccoons (*Procyon lotor*). Therefore, these ticks may be overlooked by physicians unaware of the potential risk to their patients, resulting in undiagnosed cases of Lyme disease and HGE.

The *I. scapularis* population in Van Cortlandt Park may have been present for many years, though at such low densities as to be unnoticed. Moreover, other wooded parks in the city, which can provide a refuge for urban wildlife and ticks, may pose a risk of encountering ticks infected with either or both of these tick-borne disease agents. Further surveillance that measures the extant tick population is needed to assess the risk for Lyme disease and ehrlichiosis in this urban area.

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Thomas J. Daniels,*† Richard C. Falco,*† Ira Schwartz,† Shobha Varde,† and Richard G. Robbins‡

*Fordham University, Armonk, New York, USA; †New York Medical College, Valhalla, New York, USA; ‡Walter Reed Army Medical Center, Washington, D.C., USA

References

1. Centers for Disease Control and Prevention. Lyme disease surveillance summary 1997;8:2.
2. White DJ, Chang HG, Benach J L, Bosler EM, Meldrum SC, Means RG, et al. The geographic spread and temporal increase of the Lyme disease epidemic. JAMA 1991;266:1230-6.
3. Centers for Disease Control. Human granulocytic ehrlichiosis—New York, 1995. MMWR Morb Mortal Wkly Rep 1995;44:593-5.
4. Bakken JS, Dumler JS, Chen SM, Eckman MR, Van Etta LL, Walker DH. Human granulocytic ehrlichiosis in the upper midwest United States. JAMA 1994;272:212-8.
5. Magnarelli LA, Denicola A, Stafford KC, Anderson J F. Borrelia burgdorferi in an urban environment: white-tailed deer with infected ticks and antibodies. J Clin Microbiol 1995;33:541-4.
6. Schwartz I, Varde S, Nadelman RB, Wormser GP, Fish D. Inhibition of efficient polymerase chain reaction amplification of Borrelia burgdorferi DNA in blood-fed ticks. Am J Trop Med Hyg 1997;56:339-42.
7. Magnarelli LA, Anderson J F, Fish D. Transovarial transmission of Borrelia burgdorferi in *Ixodes dammini* (Acari: Ixodidae). J Infect Dis 1987;156:234-6.
8. Dumler JS, Bakken JS. Ehrlichial diseases of humans: emerging tick-borne infections. Clin Infect Dis 1995;20:1102-10.
9. Telford III SR, Dawson JE, Katavolos P, Warner CK, Kolbert CP, Persing DH. Perpetuation of the agent of human granulocytic ehrlichiosis in a deer tick-rodent cycle. Proc Natl Acad Sci U S A 1996;93:6209-14.
10. Schwartz I, Wormser GP, Schwartz JJ, Cooper D, Weissensee P, Gazumyan A, et al. Diagnosis of early Lyme disease by polymerase chain reaction amplification and culture of skin biopsies from erythema migrans lesions. J Clin Microbiol 1992;30:3082-8.
11. Munderloh UG, Madigan MJ, Dumler JS, Goodman JL, Hayes SF, Barlough JE, et al. Isolation of the equine granulocytic ehrlichiosis agent, Ehrlichia equi, in tick cell culture. J Clin Microbiol 1996;34:664-70.
12. Dawson JE, Warner CK, Baker V, Ewing SA, Stallknecht DE, Davidson WR, et al. Ehrlichia-like 16S rDNA sequence from wild white-tailed deer (*Odocoileus virginianus*). J Parasitol 1996;82:52-8.
13. Rikihisa Y. The tribe Ehrlichiae and ehrlichiosis diseases. Clin Microbiol Rev 1994;4:286-308.
14. Schwartz I, Fish D, Daniels TJ. Prevalence of the rickettsial agent of human granulocytic ehrlichiosis in ticks from a hyperendemic focus of Lyme disease. N Engl J Med 1997;337:49-50