JOURNAL OF CLINICAL MICROBIOLOGY, June 2008, p. 2115–2118 Vol. 46, No. 6
0095-1137/08/$08.00+0 doi:10.1128/JCM.00394-08

Lyme Disease in Oregon▽

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Received 27 February 2008/Returned for modification 28 March 2008/Accepted 21 April 2008

The incidence of Lyme disease in Oregon is calculated from cases reported to the Oregon State Health Division. We reviewed the exposure history of reported cases of Lyme disease and performed field surveys for infected *Ixodes pacificus* ticks. The incidence of Lyme disease correlated with the distribution of infected *I. pacificus* ticks.

The average reported annual incidence of Lyme disease per 100,000 population during 2003 to 2005 in 10 northeastern and upper Midwest reference states was 29.2 (3). During the same period, the annual incidences for Oregon were 0.45, 0.31, and 0.08 based on 16, 11, and 3 reported cases, respectively (3). Several of the cases were reported from counties comprising the populous greater Portland area in Oregon. However, previous entomologic investigations found *Ixodes pacificus* ticks only in the western third of Oregon with few ticks of any species in the greater Portland area (Fig. 1) (1, 2, 4). We hypothesized that the incidence of Lyme disease reported in the Portland area was greater than the incidence of infection acquired in the Portland area.

County health department nurses routinely attempt to interview reported cases of suspected Lyme disease using a standardized questionnaire that includes questions about relevant travel and exposure to ticks. We reviewed the questionnaires of the confirmed and presumptive cases of Lyme disease reported to the Oregon State Health Division from 1999 to 2004 (8). We calculated a "geographical incidence" of Lyme disease in Oregon by excluding cases with significant tick exposure and development of symptoms outside Oregon and by reassigning cases to the county where patients had tick exposure.

An entomologist collected adult questing *I. pacificus* ticks in April and May of 2003 and 2004. High-use public parks and recreational areas were sampled. The same entomologist had collected ticks from the same locations in southwestern Oregon in 1982 to 1984 and 1997.

Ticks were macerated and then ground in 180 μl of Qiagen buffer ATL in a sterile 1.5-ml microcentrifuge tube (Fisher Scientific, Pittsburgh, PA). Instruments were cleaned with bleach, alcohol, and sterile water between ticks. Nucleic acid was extracted with Qiagen (Valencia, CA) QIAamp DNA blood mini kit reagents and procedures.

Ticks were assayed for the 16S rRNA gene of *Borrelia burgdorferi* using real-time PCR (9). The forward primer was 5′-GCG AAG CGA AAC AGT GAT GTG AAG-3′. The reverse primer was 5′-GTA CAA GGC CCG AGA ACG TAT TCA-3′ (5). The probe employed was 5′/56-carboxyfluorescein/AAG CAG GTC TCA GTC CGG ATT GAA GT/3-Black Hole Quencher (BHQ)_1/-3′. *I. pacificus* 28S rRNA multiplexed with the *B. burgdorferi* 16S rRNA PCR was used as an inhibition control. The forward primer for *I. pacificus* 28S rRNA was 5′-AAG TGG GAG GCC GTT GAA TC-3′. The reverse primer was 5′-GTA AAG AAA CGA TGA AAG TAG TGG TAT TTC-3′ (5). The inhibition assay was validated (data not shown). The probe sequence, developed with Visible OMP software from DNA Software (Madison, WI) was 5′-5-tetrachlorofluorescein/TCG TGG TTG TGT CGT GCC GAC A/3 BHQ_1/-3′. All primers and probes were purchased from Integrated DNA Technologies (Coralville, IA). PCR was performed with Cepheid Smart Cycler and Omnimix beads. The positive control for *B. burgdorferi* was an extract of *B. burgdorferi* isolate ATCC 35210 from the American Type Culture Collection (ATCC) (Rockville, MD). The negative control was PCR-grade water.

The results of our 2003 and 2004 field surveys are shown in Table 1. Of 359 ticks collected at sites in southwestern Oregon in 2003, 6 were positive for *B. burgdorferi* by PCR (1.7%). This result, although using a different and presumably more sensitive PCR methodology, is similar to earlier studies that detected *B. burgdorferi* using a monoclonal antibody method, i.e., 2% reported from surveys conducted in 1982 to 1984 and 3% found in 1997 (1, 2).

Of our 161 ticks collected from the western end of the Columbia Gorge near the mouth of the Deschutes River in Oregon in 2004, the PCR for *B. burgdorferi* was positive in 7 (4.3%) (Fig. 1). By comparison, of our 161 ticks collected in the coastal mountains of northwestern Oregon, all were negative, as were all 47 *I. pacificus* ticks collected from 13 high-use recreational sites in the greater Portland area.

The importance of adjusting for exposure to ticks within or outside Oregon is summarized in Table 2. Of the 34 patients with Lyme disease reported from southwestern counties, 3 described exposure to ticks in areas outside Oregon where Lyme disease is endemic. This discrepancy was most evident in clinical cases reported from the greater Portland area; of the 33 reported patients, 14 had exposure

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▽ Published ahead of print on 30 April 2008.

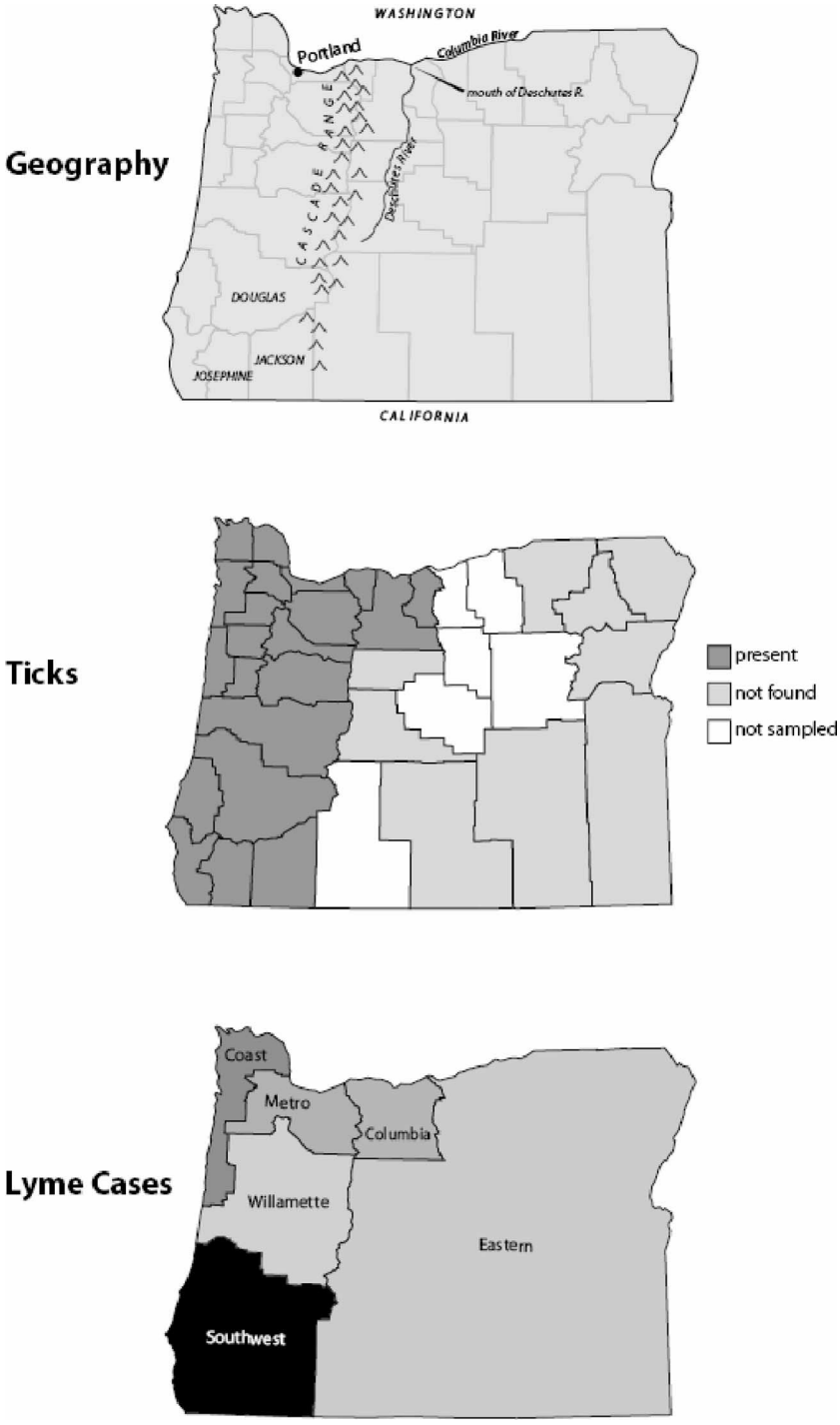

FIG. 1. Three maps of Oregon that summarize the epidemiology of Lyme disease in Oregon. The top map highlights the pertinent geography of Oregon. Note the location of Portland, the Columbia River, and the three counties in southwestern Oregon that have the highest density of *I. pacificus* ticks. The middle map displays the known distribution of *Ixodes pacificus* ticks. The bottom map summarizes the Oregon adjusted annual case rate. The highest rate of 1.28/100,000 population is found in the southwestern portion of the state (in black). The lowest rate of 0.23/100,000 runs north to south through the Willamette Valley (light gray).

to ticks in other areas where Lyme disease is known to be endemic. It is noteworthy that 17 of the 19 remaining Portland area cases did not have tick exposure information on their questionnaires. After adjusting for likely exposure to ticks outside Oregon, the case rate per 100,000 population is 4.7 times greater in the southwestern portion of the state than in the Portland area. By deleting cases exposed to ticks outside Oregon, the incidence per 100,000 population decreases the state-wide incidence from 0.45 case per 100,000 people per year to 0.34 case per 100,000 people per year.

TABLE 1. Prevalence of *Borrelia burgdorferi*-infected ticks in Oregon based on six field surveys conducted from 1982 to 2004

Yr of field survey (reference)	Oregon collection site	No. of *Ixodes pacificus* ticks collected	No. of ticks positive for *B. burgdorferi* (%)
1982 to 1984 (1)	Southwestern counties	715	14 (2)
1997 (2)	Southwestern counties	246	8 (3)
2003	Southwestern counties	359	6 (1.7)
2004	Greater Portland metropolitan area	47	0
	Northwestern counties (coastal)	161	0
	Western Columbia River Gorge	161	7 (4.3)

TABLE 2. Adjustment of the reported number of clinical Lyme disease cases from 1999 to 2004 based on probable tick exposure within or outside Oregon

Region of Oregon	Population of region (2000 census)	No. of cases of Lyme disease reported	No. of cases with likely tick exposure outside Oregon	No. of cases/ 100,000 population with exposure to ticks only in Oregon
Southwestern counties	442,750	34	3	1.09
Northwestern counties (coast)	148,300	5	0	0.67
Greater Portland metropolitan area	1,537,150	33	14	0.23
Western Columbia River Gorge	46,300	1	0	0.36
Eastern Oregon	407,650	9	3	0.25
Willamette Valley	854,600	12	3	0.12
Total	3,436,750	94	23	0.34

Overall, 23 of the 94 Oregon resident cases described significant tick exposure or development of symptoms outside the state prior to their presentation. Correlating Tables 1 and 2, the higher percentage of infected ticks in southwestern Oregon is associated with a higher incidence of reported cases (Fig. 1). In contrast, even though 4.3% of the *I. pacificus* ticks in the western Columbia Gorge are infected, only one Lyme disease patient was identified.

I. pacificus is the tick vector for *B. burgdorferi* in the western United States. The distribution of *I. pacificus* in Oregon has been studied multiple times since 1967 (1, 2, 4). The tick surveys consistently demonstrate a distribution that extends from the western slope of the Cascade mountain range to the Pacific Ocean (Fig. 1). In addition, *I. pacificus* ticks are found roughly 50 miles to the east of Portland.

Cases are reported each year from the counties that constitute the greater Portland metropolitan area, despite a low prevalence of *I. pacificus* ticks in urban recreational sites. None of the 47 ticks collected in the urban area were positive for *B. burgdorferi*. In contrast, large numbers of ticks were recovered from southwestern Oregon, and small numbers of ticks were recovered from the Columbia River Gorge. Consistent with earlier reports, 2 to 4% of these ticks were positive for the marker genes of *B. burgdorferi* (Table 1). We postulate that Lyme disease cases reported in the greater Portland area were either acquired by exposure to endemic foci in southwestern Oregon, in popular recreational sites in the Columbia River Gorge, or in areas in the eastern United States where Lyme disease is endemic.

In areas of northeastern and north central United States where Lyme disease is endemic, the white-footed mouse is the principal reservoir for *B. burgdorferi*. The larval and nymphal forms of *Ixodes scapularis* feed on mice. In contrast, in Oregon and northern California, *B. burgdorferi* bacteria are maintained in nature by the dusky-footed wood rat and *Ixodes neotomae* ticks. *I. neotomae* ticks do not feed on humans. *I. pacificus* ticks prefer to feed on the Western fence lizard, which has a borreliacidal complement system that kills the organism and does not allow the lizard to be a reservoir for *B. burgdorferi* (7, 12). The source of human infection is exposure to the few *I. pacificus* ticks that have fed on infected wood rats.

The risk of clinical disease correlates with the percentage of infected ticks. In Westchester County of New York, 52% of adult ticks and 26% of nymphs were reported infected with *B. burgdorferi* (6, 10, 11). These dramatic figures are in sharp contrast to the roughly 2 to 4% of infected ticks in Oregon.

In summary, the low incidence of Lyme disease in Oregon correlates with a low prevalence of infected ticks in areas far removed from major population centers. The risk of Lyme disease is low in patients who have had no exposure to ticks in the counties of southwestern Oregon, a small area near the mouth of the Deschutes River in north central Oregon, or the areas of the Midwestern and eastern United States where Lyme disease is endemic.

We thank William Keene of the Oregon State Health Division for providing maps, epidemiologic data, and helpful comments.

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