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SHORT COMMUNICATION

Fleas from common rodent species are an unlikely source of plague (*Yersinia pestis*) in managed forests of northwestern Oregon, USA

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Abstract

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Anthropogenic environmental change can alter the susceptibility of wildlife hosts to pathogens and provide an opportunity for disease emergence. We explored *Yersinia pestis* prevalence in fleas from three rodent species inhabiting intensively managed forests in Oregon, USA. *Y. pestis* was not detected in the 145 fleas (3 families and 9 species) collected. Our results suggest a low public health threat from plague in this anthropogenically altered landscape and contribute to regional *Y. pestis* monitoring efforts.

KEYWORDS

anthropogenic environmental change, intensive forest management, Microtus oregoni, Peromyscus maniculatus, Tamias townsendii

1 | INTRODUCTION

Anthropogenic environmental change can have detrimental consequences for wildlife populations and drive the dynamics of infectious diseases, including zoonoses (Brearley et al., 2012). Forecasting the emergence, re-emergence or spread of pathogens in relation to environmental change is difficult because complex ecological responses by hosts, vectors and pathogens make the direction and magnitude of shifts in disease dynamics unpredictable (Mills, 2005). As a first step, it is essential to determine existing patterns of disease in wildlife populations and assess the impact of anthropogenic disturbance on pathogen prevalence.

In the western United States, Yersinia pestis is maintained in rodent hosts. Intermittent amplification resulting in plague outbreaks in wild rodent communities significantly increases the risk of human infection (Gage & Kosoy, 2005). Deer mice (Peromyscus maniculatus), and certain species of chipmunks (Tamias spp.) and voles (Microtus spp.), have been implicated in regional plague epizootiology. However, the exact host and flea vector assemblages responsible for maintaining and amplifying Y. pestis differ geographically

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885

and appear dependent on environmental conditions (Ari et al., 2011; Smith et al., 2010).

In the US Pacific Northwest, forests are managed intensively with clearcutting and heavy herbicide use before and after replanting primarily with fast-growing species in monoculture (e.g. Douglas fir, *Pseudotsuga menziesii*). These management practices significantly alter the landscape and simplify community structure (Lehmkuhl & Ruggiero, 1991). Resulting changes in habitat quality can impact host health (e.g. condition, stress and immune function) and modify the susceptibility of wild animals to pathogens (Brearley et al., 2012). For zoonotic diseases with typically low enzootic levels, changes in host susceptibility can provide an opportunity for pathogens to emerge and invade a community with potentially serious implications for human health (Mills, 2005).

Yersinia pestis is enzootic in Oregon with sporadic reports of human disease (Hopkins & Gresbrink, 1982; Oregon Health Authority Public Health Division, 2015). Our objectives were to determine whether Y. pestis was present in fleas collected from common rodent species inhabiting differentially managed forest plots in the Oregon Coast Range and to explore whether infection prevalence changed with increasing management intensity.

2 | MATERIALS AND METHODS

Every 3-4 weeks between June and September 2011-2013, we sampled deer mice, Townsend chipmunks (Tamias townsendii) and creeping voles (Microtus oregoni) in the coastal mountain range of northwestern Oregon. Twelve study plots (12–16 ha) were located along Oregon Route 6, approximately 50 miles west of Portland, Oregon (within a 15-mile radius of 45.5889°N, -123.5369°W). Plots were replicated across three levels of forest management intensity. Four plots each were either clearcut and sprayed with a mixture of herbicides, clearcut only (not sprayed), or consisted of 40-50-year-old mixed hardwood and Douglas fir stands not recently managed (Betts et al., 2013; Supporting Information 1 in Appendix S1). At time of first capture, each rodent was identified with a unique ear tag (Supporting Information 2 in Appendix S1). The presence of fleas was noted for each animal, and fleas were collected opportunistically when observed during handling. The number of captured rodents with fleas and the number of infested animals from which fleas were collected were determined for each rodent species and forest management level. Upon collection, fleas were immediately stored in ethanol and refrigerated until submission to the USGS National Wildlife Health Center in Madison, Wisconsin for identification to species (or genus) (Furman & Catts, 1982; Hubbard, 1947; Stark, 1958; Supporting Information 3 in Appendix S1). For each rodent host, fleas of the same species were pooled, DNA was extracted (Quick g-DNA MiniPrep kit, Zymo Research, Irivine, CA) and screened for the presence of Y. pestis DNA by SYBR green-based real-time PCR targeting 112 bp of the pesticin immunity protein (pim) gene on the pPCP1 plasmid (Genbank AL109969) of Y. pestis (Bron et al., 2019;

Impacts

- Anthropogenic environmental change can be detrimental for wildlife health, change host susceptibility to pathogens, and drive infectious disease dynamics.
- Establishing enzootic patterns of disease in wildlife populations is fundamental to assessing the impact of anthropogenic disturbance on pathogen prevalence and predicting transmission risk for domestic animals and humans.
- Our results indicate low risk for infection with Y. pestis from wildlife in disturbed forests of northwest Oregon. However, recreational activities throughout the region provide opportunity for future zoonotic pathogen transmission and warrant continued monitoring of wildlife and flea vectors for Y. pestis to protect public health.

Supporting Information 4 in Appendix S1). All animal procedures were approved by the Oregon Department of Fish and Wildlife and the Institutional Animal Care and Use Committee at Oregon State University (ACUP #4195).

3 | RESULTS

Over 9668 trapnights, we captured 347 deer mice (2011–2013) and 233 Townsend chipmunks and 58 creeping voles (2011–2012), representing 220, 157 and 51 unique animals respectively. Recapture rates were 36.6% for deer mice, 32.6% for Townsend chipmunks and 12.1% for creeping voles. Considering all captures, we noted the presence of fleas on 40 deer mice (11.7%), 83 chipmunks (36.1%) and 18 voles (31.6%) across all three forest management levels and collected and tested fleas from 27 (67.5%), 43 (51.8%) and 15 (83.3%) animals respectively (Table 1). Of 145 fleas tested (89 flea pools representing 3 families/9 species; Table 2), none were positive for Y. *pestis* (95% CI: 0.0–2.5% prevalence, Supporting Information 5 in Appendix S1).

4 | DISCUSSION

We aimed to explore the presence of *Yersinia pestis* in rodents inhabiting the coastal mountains of Oregon and to examine potential differences in prevalence across three levels of forest management. We tested fleas collected from three abundant rodent species for the evidence of the plague bacterium but found none at any of the study sites regardless of forest management level. *Y. pestis* has been identified in Oregon wildlife species other than rodents, including coyotes, raccoons, badgers and striped skunks from 20 of 36 counties (Hopkins & Gresbrink, 1982). Between 1934 and 2015, 20 human cases of plague were reported in 10 counties (Oregon

HANSELMANN ET AL.

| | Cleard | cut + spra | yed | | Clearc | ut | | | Uncut | | | | |
|----------------------------|------------|------------|------|-------|--------|------|----|-------|-------|------|----|-------|-------------|
| | М | F | U | Total | М | F | U | Total | М | F | U | Total | Grand Total |
| Deer mice (95% Cl: 0.0-7.9 | 9%) | | | | | | | | | | | | |
| Captures | 70 | 44 | 1 | 115 | 95 | 82 | 1 | 178 | 32 | 21 | 1 | 54 | 347 |
| Unique animals | 45 | 28 | | 74 | 59 | 44 | | 104 | 28 | 13 | | 42 | 220 |
| Ave. mass (g) | 17.8 | 19.1 | 18.5 | 18.3 | 17.8 | 20.1 | ND | 18.9 | 17.2 | 19.6 | 12 | 18 | 18.5 |
| Captures w/fleas | 8 | 1 | ND | 9 | 11 | 11 | 0 | 22 | 6 | 3 | ND | 9 | 40 |
| Captures tested | 5 | 1 | | 6 | 7 | 7 | | 14 | 5 | 2 | | 7 | 27 |
| Fleas tested | 11 | 2 | | 13 | 10 | 7 | | 17 | 10 | 5 | | 15 | 45 |
| Townsend chipmunks (95% | 6 CI: 0.0- | 4.6%) | | | | | | | | | | | |
| Captures | 17 | 20 | 1 | 38 | 42 | 37 | 1 | 80 | 58 | 57 | 0 | 115 | 233 |
| Unique animals | 11 | 16 | | 28 | 35 | 25 | | 61 | 35 | 33 | | 68 | 157 |
| Ave. mass (g) | 70.3 | 75.7 | 68 | 73.1 | 70.3 | 76.2 | 64 | 72.9 | 73.1 | 78.5 | | 75.4 | 74.2 |
| Captures w/fleas | 5 | 5 | 0 | 10 | 18 | 14 | 0 | 32 | 25 | 16 | 0 | 41 | 83 |
| Captures tested | 2 | 3 | | 5 | 9 | 8 | | 17 | 14 | 7 | | 21 | 43 |
| Fleas tested | 2 | 3 | | 5 | 23 | 19 | 0 | 42 | 18 | 14 | | 32 | 79 |
| Creeping voles (95% Cl: 0. | 0-16.1%) | | | | | | | | | | | | |
| Captures | 6 | 10 | 1 | 17 | 6 | 16 | 1 | 23 | 8 | 9 | 1 | 18 | 58 |
| Unique animals | 6 | 9 | | 16 | 6 | 11 | | 18 | 8 | 8 | | 17 | 51 |
| Ave. mass (g) | 16.3 | 16.5 | 20 | 16.6 | 15.5 | 16.4 | 17 | 16.2 | 15.9 | 17.2 | 19 | 16.7 | 16.5 |
| Captures w/fleas | 1 | 2 | 0 | 3 | 1 | 9 | 1 | 11 | 3 | 1 | 0 | 4 | 18 |
| Captures tested | 1 | 2 | | 3 | 1 | 7 | 1 | 9 | 3 | 0 | | 3 | 15 |
| Fleas tested | 2 | 2 | | 4 | 1 | 11 | 1 | 13 | 4 | | | 4 | 21 |

^aU, unknown host sex; ND, no data available.

886

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^bClopper-Pearson exact two-sided 95% confidence intervals were computed for each rodent species to provide a conservative estimate of the true prevalence of Y. *pestis* in our study populations (Supporting Information 5 in Appendix S1).

TABLE 2 Number of flea pools tested for Yersinia pestis (and the total number of fleas tested), arranged by rodent host, flea family and species, and forest management level

| Host species | Flea family | Flea species | Clearcut + sprayed | Clearcut | Uncut |
|--|--------------------|---------------------------------|--------------------|----------|---------|
| Deer mice (Peromyscus maniculatus) | Ceratophyllidae | Ceratophyllus ciliatus protinus | 0 | 4 (4) | 1 (2) |
| | | Opisodasys keeni | 5 (12) | 10 (13) | 6 (14) |
| | Hystrichopsyllidae | Delotelis telegoni | 0 | 0 | 1 (1) |
| | | Epitedia scapani | 0 | 0 | 1 (2) |
| | | Rhadinopsylla spp | 1 (1) | 0 | 0 |
| Townsend chipmunks (Tamias townsendii) | Ceratophyllidae | Monopsyllus ciliatus protinus | 5 (5) | 16 (41) | 21 (32) |
| | | Opisodasys keeni | 0 | 1 (1) | 0 |
| Creeping voles (Microtus oregoni) | Ceratophyllidae | Monopsyllus ciliatus protinus | 0 | 1 (1) | 0 |
| | | Megabothris abantis | 0 | 2 (3) | 0 |
| | Hystrichopsyllidae | Atyphloceras multidentatus | 0 | 0 | 1 (1) |
| | | Epitedia jordani | 2 (3) | 4 (4) | 1 (2) |
| | | Hystrichopsylla dippiei | 0 | 0 | 1 (2) |
| | Leptopsyllidae | Peromyscopsylla selenis | 2 (3) | 2 (5) | 1 (1) |

Health Authority Public Health Division, 2015). That we did not find Y. *pestis* in the fleas tested parallels the absence of reports of human cases of plague in our study area and suggests that risk for infection with Y. *pestis* from wildlife in northwest Oregon is likely low. However, outdoor recreation, including hunting, is common in this area, which increases the transmission potential of zoonotic infections such as plague to domestic animals and humans, either directly or via flea vectors (Smith et al., 2010). Thus, the continued monitoring of wildlife and flea vectors for Y. *pestis* throughout Oregon and the US Pacific Northwest, especially in areas affected by high levels of anthropogenic disturbance, is of interest for public health.

AUTHOR CONTRIBUTIONS

RH and AEJ conceived the study design and developed the methodology; RH and LJD collected the samples; GMB identified fleas and performed the Yersinia pestis analyses; RH analysed and interpreted the data; RH and LJD led the writing of the manuscript. All authors contributed critically to the drafts and gave final approval for the publication.

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CONFLICT OF INTEREST

The authors declare no competing financial or other interests.

DATA AVAILABILITY STATEMENT

The data that support the findings of this study are available from the corresponding author upon reasonable request.

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SUPPORTING INFORMATION

Additional supporting information may be found in the online version of the article at the publisher's website.

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