Increasing Incidence of Zoonotic Visceral Leishmaniasis on Crete, Greece

Maria Antoniou, Ippokratis Messaritakis, Vasiliki Christodoulou, Ioanna Ascoksilaki, Nikos Kanavakis, Andrew J. Sutton, Connor Carson, and Orin Courtenay

To determine whether the incidence of canine leishmaniasis has increased on Crete, Greece, we fitted infection models to serodiagnostic records of 8,848 dog samples for 1990–2006. Models predicted that seroprevalence has increased 2.4% (95% confidence interval 1.61%–3.51%) per year and that incidence has increased 2.2- to 3.8-fold over this 17-year period.

Zoonotic visceral leishmaniasis (ZVL) caused by *Leishmania infantum* is a disease of humans and domestic dogs (the reservoir) transmitted by phlebotomine sandflies. According to the World Health Organization (1), ZVL was first recorded on Crete in 1907, after which it featured prominently in medical literature as a serious public health problem. In Chania, Crete, the annual incidence in the 1930s was 50 cases/30,000 population (2), and reports of canine ZVL were recorded on Crete (3,4) during 1946–1949 compared with unsprayed villages (3). Dur-
To assess the change in infection incidence, our principal aim, we used 3 standard epidemiologic models (9–11; online Technical Appendix) to calculate infection rates accounting for time, dog age, and potential loss of infection. The first method (model 1) used the cross-sectional age-prevalence data (IFAT cutoff titer 160), in which the proportion of seropositive dogs in each age class is fitted by logistic confidence limits over the 17-year study equated to a mean prevalence increase of –ln(1 – 0.321)/16 = 2.4% (95% CI 1.61%–3.51%) per year.

Table 1. Variation in incidence over time estimated from cross-sectional data for 1,205 dogs with accompanying demographic records, Crete, Greece, 1990–2006*

<table>
<thead>
<tr>
<th>Period</th>
<th>Incidence/mo</th>
<th>95% CI</th>
<th>Loss of infection/mo</th>
<th>95% CI</th>
<th>Incidence/mo</th>
<th>95% CI</th>
<th>No. dogs</th>
</tr>
</thead>
<tbody>
<tr>
<td>1999–2000</td>
<td>0.016</td>
<td>0.0107–0.0206</td>
<td>0.045</td>
<td>0.0260–0.0645</td>
<td>0.015</td>
<td>0.0093–0.0213</td>
<td>237</td>
</tr>
<tr>
<td>2001–2002</td>
<td>0.029</td>
<td>0.0114–0.0455</td>
<td>0.071</td>
<td>0.0201–0.1211</td>
<td>0.023</td>
<td>0.0166–0.0298</td>
<td>219</td>
</tr>
<tr>
<td>2003–2004</td>
<td>0.030</td>
<td>0.0216–0.0381</td>
<td>0.049</td>
<td>0.0320–0.0667</td>
<td>0.029</td>
<td>0.0221–0.0367</td>
<td>401</td>
</tr>
<tr>
<td>2005–2006</td>
<td>0.059</td>
<td>0.0233–0.0946</td>
<td>0.106</td>
<td>0.0383–0.17430</td>
<td>0.032</td>
<td>0.0205–0.0477</td>
<td>348</td>
</tr>
</tbody>
</table>

*Only model 1 is designed to estimate loss of infection (serorecovery). CI, confidence interval. A full description of these models is available in the online Technical Appendix (available from www.cdc.gov/EID/content/15/6/932-Techapp.pdf).

To the continual increase in canine seroprevalence during the latter part of the intervention (Figure) suggests that the culling policy was unsuccessful in reducing transmission: estimated to be 2.20–3.78-fold higher during 2005–2006 than during 1999–2000. The models differed in approach and age of dogs considered by necessity of the model, number of estimated parameters, or model reduction. Inclusion of a parameter describing loss of infection (Table 1, model 1) did not significantly lower the infection rate estimates as might be expected compared with a single parameter (Table 1, model 2) or longitudinal (Table 2, model 3) model, both of which identified younger (<2 years of age) dogs to be at substantially greater risk for infection (p<0.0001).

Conclusions

The potential contribution of any improvements in diagnostic test sensitivity or vigilance to the increasing incidence of ZVL infection is unclear. The difference in cutoff titers between data sources minimally shifted the absolute prevalence values, but not relative prevalence slopes, with time (Figure). Any loss of infection with age (Table 1, model 1) could result from nonmutually exclusive biologic processes including recovery from infection, death, or reduced past exposure (9,11). The latter possibility is unlikely on Crete because of the higher risk identified in young dogs in all biannual periods. Disproportionate numbers of deaths of seropositive dogs is not suggested by a decline in ZVL clinical signs in older dogs in this study (data not shown) or elsewhere (11,12). Loss of detectable Leishmania-specific antibody is the more likely explanation because the observed rates of serorecovery are not dissimilar to those (e.g., 0.062/month) estimated by cohort studies elsewhere (12).

Actual infection rates are likely to be higher than those shown here because IFAT sensitivity is <100%. Similarly, absolute prevalences, particularly low values for 1990–1991, should be treated with caution because the official leishmaniasis control program on Crete (1984–1995) began before this period when infection was presumably sufficient on the island to warrant intervention. The intervention comprised elimination of IFAT-seropositive dogs (cutoff titer 400) but did not include insecticide spraying (DDT spraying ceased in 1950; V. Chatzistefanou, pers. comm.).

The continual increase in canine seroprevalence during the latter part of the intervention (Figure) suggests that the culling policy was unsuccessful in reducing transmission:
likely reasons for the low efficacy of dog culling in other leishmaniasis-endemic regions have been described (13). Officially, destruction of seropositive dogs is still required today unless the owner agrees to veterinary treatment of the dog or to keep the dog under sandfly-proof netting. However, there is no current policy on Crete to combat vectors. We conclude that the results of our study are consistent with a postwar reemergence and current increasing incidence of ZVL infection on Crete.

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Dr Antoniou is an assistant professor of parasitology at the Medical School of the University of Crete. Her research interests are Leishmania and Toxoplasma parasites, the epidemiology of zoonoses, and development of strategies for disease control.

References


Address for correspondence: Orin Courtenay, Populations and Disease Research Group, Department of Biological Sciences, University of Warwick, Coventry CV4 7AL, UK; email: orin.courtenay@warwick.ac.uk