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SHORT COMMUNICATION

Spatial distribution of an infectious disease in a small mammal community

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Abstract Chagas disease is a zoonosis caused by the parasite *Trypanosoma cruzi* and transmitted by insect vectors to several mammals, but little is known about its spatial epidemiology. We assessed the spatial distribution of *T. cruzi* infection in vectors and small mammals to test if mammal infection status is related to the proximity to vector colonies. During four consecutive years we captured and georeferenced the locations of mammal species and colonies of *Mepraia spinolai*, a restricted-movement vector. Infection status on mammals and vectors was evaluated by molecular techniques. To examine the effect of vector colonies on mammal infection status, we constructed an infection distance index using the distance between the location of each captured mammal to each vector colony and the average *T. cruzi* prevalence of each vector colony, weighted by the number of colonies assessed. We collected and evaluated *T. cruzi* infection in 944 mammals and 1976 *M. spinolai*. We found a significant effect of the infection distance index in explaining their infection status, when considering all mammal species together. By examining the most abundant species separately, we found this effect only for the diurnal and gregarious rodent *Octodon degus*. Spatially explicit models involving the prevalence and location of infected vectors and hosts had not been reported previously for a wild disease.

Keywords Chagas disease · *Mepraia spinolai* · Prevalence · Wild reservoirs

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Introduction

Non-random distribution of animals is a key factor when studying host-parasite interactions such as the spread of infectious diseases. These are mediated by complex interactions among pathogens, vectors, hosts and the environment, which operate simultaneously in time and space (Pfeiffer et al. 2008). Regarding pathogens transmitted by vectors, climate, landscape and host community elements can be modifying transmission probabilities in space (Peterson 2009). However, little is known about the spatial components of infectious diseases transmitted by vectors.

Chagas disease is a zoonosis caused by the parasite *Trypanosoma cruzi* and transmitted by triatomine insects to several mammalian species. *T. cruzi* multiplies and differentiates in the vector's digestive tract, and the main infection route of sylvatic mammal hosts occurs by contamination of mucous membranes or open wounds with insect-infected faeces (Kollien and Schaub 2000). Triatomines are generally associated with blood sources (hosts) or specific habitats (rocky outcrops, bromeliads and palm trees) with high chance of finding blood meals (Gorla and Noireau 2010). In rural, *Triatoma infestans* is clustered around blood sources such as goat corrals and chicken coops (Cecere et al. 2006), whereas *Mepraia spinolai*, a movement-restricted vector, is usually

found in rocky outcrops and bromeliads (Bacigalupo et al. 2006; Botto-Mahan et al. 2005a).

In hyper-endemic areas of Chagas disease in Chile, the main wild vector *M. spinolai* can reach *T. cruzi* infection levels up to 46 % and native host mammals up to 61 % (Botto-Mahan et al. 2005b; Rozas et al. 2007). Several mammal species may act as reservoir hosts (Botto-Mahan et al. 2012; Botto-Mahan et al. 2010). *M. spinolai* is a diurnal species which can be aggregated in space, using rocky outcrops, nearby human settlements or animal pens (Canals et al. 1998). Small mammals and *M. spinolai* exhibit spatial and temporal variation in their abundances, which may be partially explained by climatic variables (Botto-Mahan et al. 2005a; Lima et al. 2003). However, the potential effect of the proximity of this movement-restricted vector on host infection status remains largely unknown.

Here, we evaluate if the distribution of *T. cruzi* infection in small mammals can be explained by the proximity of potential hosts to infected vector colonies, using a spatially explicit approach to the distribution of vectors and hosts, aiming to test if the distance between them would be a key factor explaining the infection risk to mammals, varying among host species.

Methods

This study was carried out in a semiarid area, Las Chinchillas National Reserve (31° 30′ S, 71° 06′ W; Chile, Appendix 1 in Supplementary Material), a hyper-endemic zone of Chagas disease (Botto-Mahan et al. 2010). *M. spinolai* is the only vector of Chagas disease in the reserve (Botto-Mahan et al. 2005b). Several native small mammal species inhabit the study site and are infected with *T. cruzi*, including rodents and a marsupial (Botto-Mahan et al. 2010).

During January of 2010 to 2013, we investigated nine (2010) to 12 (2011–2013) *M. spinolai* colonies from three sites within the reserve (Appendix 1). In each colony, kissing bugs were manually collected during 1-h spans at the highest activity time (Botto-Mahan et al. 2005a). At the laboratory, insects were killed and the abdomen compressed to obtain rectal contents. Whole genomic DNA was isolated from faecal samples and stored at −20 °C until molecular analyses. See protocol for PCR of faeces in Appendix 2.

Small mammal trapping was performed with folding wire mesh live-animal-traps in the same three sites where vectors were captured. Traps were baited with rolled oats and equipped with cotton bedding. Traps were geo-referenced and placed in two lines per site; each line consisted of 50 traps set 10 m apart. The capture procedure was carried out for four to five nights during January of 2010 to 2013. See blood extraction procedure in Appendix 2. Whole genomic DNA was isolated from blood samples and stored at −20 °C until molecular analyses. See protocol for PCR of blood in Appendix 2.

We geo-referenced small mammal traps and kissing bug colonies in UTM coordinates (precision: ±3 m) using a GPS device. With each geographic coordinate, we first located traps and colonies on a map to examine the proximity of traps to the colonies using a geographic information system (QGIS 2.0.1). Then, we constructed an infection distance index (IDI) calculated within each site for all mammals using the average *T. cruzi* prevalence level of the colony weighted by the distance between the first trapping location of the mammal and the edge of a 12.13-m buffer of each vector colony. In each site, there were three colonies in 2010 and four colonies in 2011–2013, so the average denominator was the number of colonies assessed. IDI was calculated as follows:

$$\mathrm{IDI} = \frac{\sum_{i=1}^{n} \left(\frac{\mathrm{colony}_i\ \mathrm{prevalence}}{\mathrm{distance\ to\ colony}_i} \right)}{\mathrm{number\ of\ colonies}}$$

As defined, IDI values increase with the prevalence and proximity of the colonies to each trap. To assess the relationship between those factors and the status of *T. cruzi* infection in mammals, we fitted generalized linear mixed models (GLMM) with a binomial distribution, using infection condition as response variable (infected/not infected) and IDI values as explanatory variable, and the body condition index (BCI= mass/(total length – tail length)^2) as a covariate, included because it had a significant effect in a previous study (Jiménez et al. 2015). We included the capture site and year as random factors to account for the spatial and temporal variability, which included the small mammals with known *T. cruzi* infection status. The GLMM were conducted in R 2.15 (R Development Core Team 2013) with the package lme4.

Results

Seven small mammal species were collected during this study (2010–2013): *Octodon degus*, *Phyllotis darwini*, *Abrocoma bennetti*, *Oligoryzomys longicaudatus*, *Thylamys elegans* and *Abrothrix* spp. A total of 944 mammals and 1974 *M. spinolai* were collected and examined for *T. cruzi* infection status, obtaining an overall infection of 35.6 and 29.9 %, respectively. Detailed information by species and year is shown in Appendix 3.

We found a significant effect of the infection distance index (GLMM estimate 0.03±0.01, $p<0.01$, $N=944$) when considering all small mammal species together. Considering that *O. degus*, *P. darwini* and *Abrothrix olivaceus* constituted 97 % of the relative small mammal abundances, we examined the effect of the IDI separately for these species (Fig. 1). We found a significant effect for *O. degus* (GLMM estimate 0.06

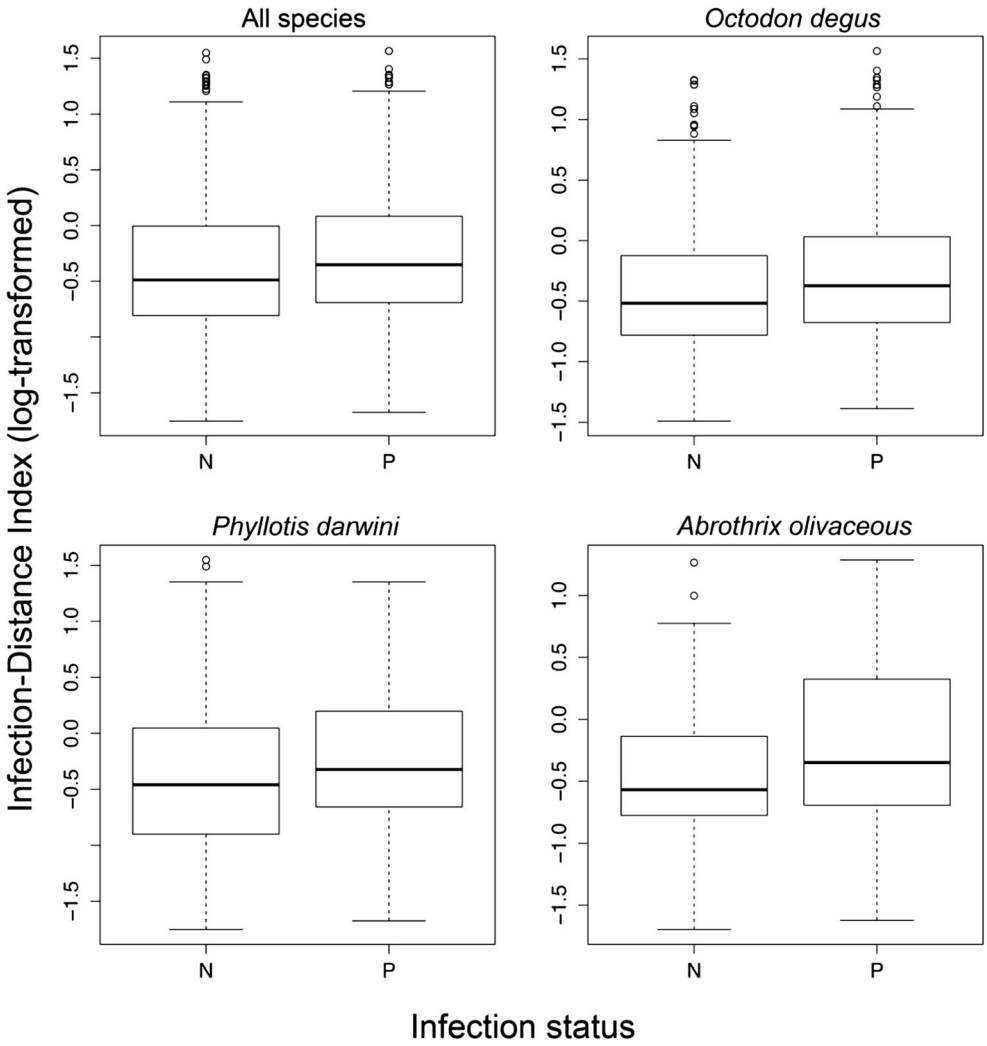

Fig. 1 Effects of the combined distance and colony prevalence (expressed as the infection distance index) on infection status. *P* positive, *N* negative)

±0.02, $p<0.01$, $N=454$), but not for *P. darwini* (GLMM estimate 0.01±0.02, $p=0.62$, $N=379$) or *A. olivaceus* (GLMM estimate 0.06±0.06, $p=0.35$, $N=90$). The BCI covariate had a positive and significant effect considering all species together (GLMM estimate 1.85±0.54, $p<0.01$, $N=944$) and also for *O. degus* (GLMM estimate 1.80±0.90, $p=0.04$, $N=454$) and *P. darwini* (GLMM estimate 11.08±1.94, $p<0.01$, $N=379$), but not for *A. olivaceus* (GLMM estimate 5.44±3.21, $p=0.09$, $N=90$).

Discussion

The IDI, combining proximity between hosts and vector colonies as well as *T. cruzi* prevalence in vector colonies, strongly explained the overall infection status of small mammal hosts. This index takes into account the *T. cruzi* prevalence in vector colonies, providing a more comprehensive assessment of this phenomenon than a distance-only approach. These results may be explained from the fact that *M. spinolai* colonies are dispersal restricted because of a highly philopatric behaviour, not dispersing farther than 12.13 m (Botto-Mahan et al. 2005a). To our knowledge, this is the first report of the spatial relevance of a dispersal-restricted colonial vector, differing from other study models in two aspects: (1) *M. spinolai* dwells in discrete colonies and has a sit-and-wait strategy (Botto-Mahan et al. 2005a), unlike mosquitoes (Chao et al. 2013), and (2) *M. spinolai* only pierces the host for a few minutes while feeding, unlike ticks and fleas (Cadiergues et al. 2001; Falco et al. 1996). Therefore, *M. spinolai* colonies represent discrete and fixed points in time and space that can be treated as single study units characterizing their infection levels, abundances and their variation across time, which could be used to estimate their effect on small mammal hosts.

Infection status in *O. degus* was explained by the IDI, but not for *P. darwini* and *A. olivaceus*. This species-specific response may emerge from the fact that *O. degus* is a diurnal and social rodent, being the second most abundant rodent species in the study site. This rodent exhibits the highest infection level and probably some of their ecological traits, such as

relatively high longevity and communal nursing (Previtali et al. 2010), with potential vertical transmission of *T. cruzi* (Oda et al. 2014), lead to a cumulative infection at the population level. Relating this to the IDI result, the significant effect detected may be caused by *O. degus* philopatric colony persistence in time and space, as what happens with vector colonies. *P. darwini* was the most abundant rodent species in the study site, highly infected with *T. cruzi* and a previous study documented that is one of the main blood sources for *M. spinolai* populations (Oda et al. 2014). *A. olivaceus* was also highly infected, but it much less abundant. Contrary to *O. degus*, *P. darwini* and *A. olivaceus* are solitary, nocturnal and short-lived rodents potentially less exposed to *M. spinolai* attacks, constituting a less easy target than *O. degus*. Notwithstanding, for future studies it would be necessary to assess host reservoir competence, considering parasitic loads to determine host infectivity (Oda et al. 2014; Brunner et al. 2008).

We found evidence that spatially explicit models involving the infection prevalence, location of hosts and philopatric colony vectors may explain, at least in some degree, how the wild cycle of *T. cruzi* works under field conditions. Little is known yet about how parasite infections are maintained over large temporal and spatial scales. Future studies should assess the spatial dynamics of different vector species (sylvatic *Triatoma* species), as well as to evaluate small mammal movements and space use behaviour with more sophisticated tracking techniques, allowing estimation of contact probabilities with vector colonies in wider temporal frames.

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References

Bacigalupo A, Segura JA, García A, Hidalgo J, Galuppo S, Cattan PE (2006) First finding of Chagas disease vectors associated with wild bushes in the metropolitan region of Chile. Rev Med Chile 134: 1230–1236

Botto-Mahan C, Cattan PE, Canals M, Acuña M (2005a) Seasonal variation in the home range and host availability of the blood-sucking insect *Mepraia spinolai* in wild environment. Acta Trop 95:160–163. doi:10.1016/J.Actatropica.2005.05.001

Botto-Mahan C, Ortiz S, Rozas M, Cattan PE, Solari A (2005b) DNA evidence of *Trypanosoma cruzi* in the Chilean wild vector *Mepraia spinolai* (Hemiptera: Reduviidae). Mem Inst Oswaldo Cruz 100: 237–239. doi:10.1590/S0074-02762005000300003

Botto-Mahan C, Campos R, Acuña-Retamar M, Coronado X, Cattan PE, Solari A (2010) Temporal variation of *Trypanosoma cruzi* infection in native mammals in Chile. Vector-Borne Zoonot 10:317–319. doi: 10.1089/Vbz.2009.0006

Botto-Mahan C, Bacigalupo A, Correa JP, Oda E, Solari A (2012) Field assessment of *Trypanosoma cruzi* infection and host survival in the native rodent *Octodon degus*. Acta Trop 122:164–167. doi:10.1016/J.Actatropica.2011.12.003

Brunner JL, Logiudice K, Ostfeld RS (2008) Estimating reservoir competence of *Borrelia burgdorferi* hosts: prevalence and infectivity, sensitivity, and specificity. J Med Entomol 45:139–147

Cadiergues MC, Santamarta D, Mallet X, Franc M (2001) First blood meal of *Ctenocephalides canis* (Siphonaptera: Pulicidae) on dogs: time to initiation of feeding and duration. J Parasitol 87:214–230

Canals M, Bustamante RO, Ehrenfeld M, Cattan PE (1998) Assessing the impact of disease vectors on animal populations. Acta Biotheor 46: 337–345

Cecere MC, Vasquez-Prokopec GM, Gürtler RE, Kitron U (2006) Reinfestation sources for Chagas disease vector, *Triatoma infestans*, Argentina. Emerg Infect Dis 12:1096–1102

Chao DL, Longini IM, Halloran ME (2013) The effects of vector movement and distribution in a mathematical model of dengue transmission. PLoS ONE 8:e76044

Falco RC, Fish D, Piesman J (1996) Duration of tick bites in a Lyme disease-endemic area. Am J Epidemiol 143:187–192

Gorla DE, Noireau F (2010) Geographic distribution of Triatominae vectors in America. In: Telleria J, Tibayrenc M (eds) American Trypanosomiasis Chagas disease: one hundred years of research. Elsevier, Burlington MA, pp 209–223

Jiménez C, Fontúrbel FE, Oda E, Ramírez PA, Botto-Mahan C (2015) Parasitic infection alters rodent movement in a semiarid ecosystem. Mamm Biol 80:255–259. doi:10.1016/j.mambio.2015.01.006

Kollien AH, Schaub GA (2000) The development of *Trypanosoma cruzi* in Triatominae. Parasitol Today 16:381–387. doi:10.1016/S0169-4758(00)01724-5

Lima M, Stenseth NC, Leirs H, Jaksic FM (2003) Population dynamics of small mammals in semiarid regions: a comparative study of within-year demographic variability in two rodent species. Proc R Soc B 270:1997–2007

Oda E, Solari A, Botto-Mahan C (2014) Effect of mammal host diversity and density on the infection level of *Trypanosoma cruzi* in sylvatic kissing bugs. Med Vet Entomol 28:384–390. doi:10.1111/mve.12064

Peterson AT (2009) Shifting suitability for malaria vectors across Africa with warming climates. BMC Infect Dis 9:59. doi:10.1186/1471-2334-9-59

Pfeiffer DU, Robinson TP, Stevenson M, Stevens KB, Rogers DJ, Clements ACA (2008) Spatial analysis in epidemiology. Oxford University Press, Oxford, UK

Previtali MA, Meserve PL, Kelt DA, Milstead WB, Gutierrez JR (2010) Effects of more frequent and prolonged El Niño events on life-history parameters of the Degu, a long-lived and slow-reproducing rodent. Conserv Biol 24:18–28. doi:10.1111/J.1523-1739.2009.01407.X

R Development Core Team (2013) R: a language and environment for statistical computing, reference index version 2.15.3. Foundation for Statistical Computing, Viena, Austria

Rozas M, Botto-Mahan C, Coronado X, Ortiz S, Cattan PE, Solari A (2007) Coexistence of *Trypanosoma cruzi* genotypes in wild and periodomestic mammals in Chile. Am J Trop Med Hyg 77:647–653