Modeling Chagas disease vector infection prevalence: incorporating life history characteristics and community composition

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BACKGROUND

- Multihost vector-borne pathogens are transmitted between arthropod vectors and multiple host species. They have a significant impact on human, domestic animal, and wildlife health (e.g. Chagas disease, leishmaniasis, West Nile Virus, Lyme disease).
- The variety of potential hosts with varying degrees of reservoir competence complicates transmission.
- In many cases, anthropogenic land use change (urbanization, forest fragmentation) is linked to increased transmission of vector-borne multihost pathogens. Proposed mechanisms of increased vector-borne disease transmission include altered host diversity and host community composition.[3]
- In theory, host life history may also affect reservoir competence. Host species that 'live fast and die young' (r-selected) may drive increased transmission compared to 'slow-paced'(K-selected) species because of 1) increased production of susceptible individuals and 2) less investment in acquired immunity.[4-6]

OBJECTIVES

To create a model that evaluates how host life history and host community structure impact transmission of a vector-borne multihost pathogen and compare results to empirical data.

STUDY SYSTEM

Chagas disease, caused by the kinetoplastid protozoan *Trypanosoma cruzi* and transmitted to mammal reservoir hosts by reduviid bug vectors.[7-9]

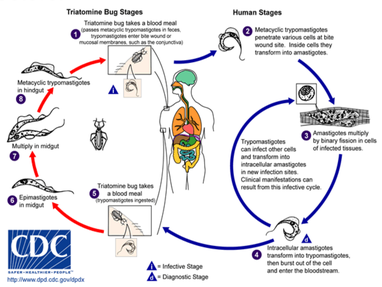

Figure 1 (right). *T. cruzi* life cycle

Figure 2 (left). In Panama, *T. cruzi* cycles between the triatomine vector *Rhodnius pallescens* and a suite of potential mammal reservoir host species. Host species composition differs across anthropogenic gradients.

METHODS

Deterministic SI model of Chagas transmission (Fig. 3, right, and Fig. 4, below)

- **Compartments**:
  - Vector: Susceptible (S_v) and Infected vectors (I_v)
  - Host community: Susceptible (S_h), Infected (I_h), and Partially Immune (P_h)
- **Inputs/Outputs:** b_v: birth rate of vector; b_h: birth rate of host community; d_v: death rate of vector; dh: death rate of host community (assumed to be at equilibrium, b=d)
- **Controls: β**: transmission from host community to vector after initial host infection; **γ**: transmission from host community to vector after subsequent infections, **λ**: transmission from vector to hosts.
- **β > γ:** Hosts infected by vector after initial infection assumed to have acquired immunity and will be less likely to transmit infection to vector.[10-12] Degree of immunity depends on host life history characteristics.

β and **γ** calculated by estimating host competence for transmission after a single infection and multiple infections, respectively, using literature data and constructing a GLM to relate r_{max} and competence. Scaled up to host community level.

5 'model' communities with different host community structure: 1 composed only of opossum (r-selected), 1 only primate hosts (K-selected), and 3 (contiguous forest, forest fragment, peridomestic) with host community structure estimated from blood meal data. Competence averaged for each host community, weighted by the relative 'contact' of species from blood meal data.

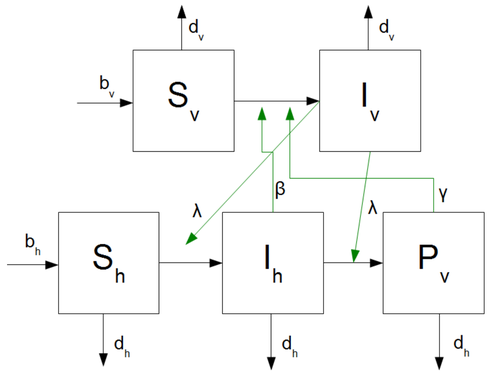

Figure 3. Compartment model of vector and multiple hosts.

$$\frac{dS_v}{dt} = b_v(S_v + I_v) - \beta S_v I_h - \gamma S_v P_h - d_v S_v$$
$$\frac{dI_v}{dt} = \beta S_v I_h + \gamma S_v P_h - d_v I_v$$
$$\frac{dS_h}{dt} = b_h(S_h + I_h + P_h) - \lambda S_h I_v - d_h S_h$$
$$\frac{dI_h}{dt} = \lambda S_h I_v - \lambda I_h I_v - d_h I_h$$
$$\frac{dP_h}{dt} = \lambda I_h I_v - d_h P_h$$

Figure 4. Equations used in dynamical model.

***r_{max}**. Intrinsic rate of population increase (data for species in community taken from literature). 'r' selected species higher rmax than 'K selected'.

RESULTS

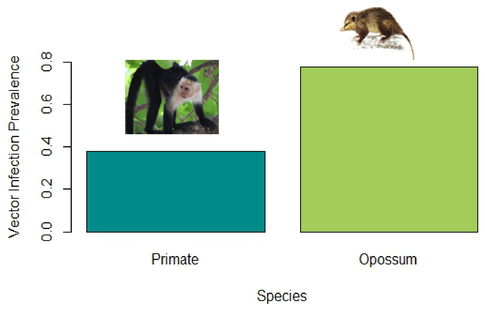

Figure 5. Proportion of vectors predicted to be infected by Chagas given a theoretical host community. The theoretical habitat comprised solely of *Cebus* monkeys (K-selected) results in the lowest proportion of infected vectors (0.38). The theoretical community consisting solely of opossums (r-selected) results in a higher proportion of infected vectors (0.78).

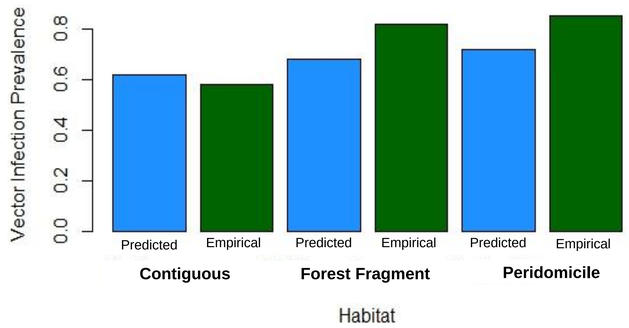

Figure 6. Proportion of vectors infected with Chagas in different habitat types. Blue: model predicted values, green: empirical values. Model predicted proportion of infected vectors: contiguous forests (0.62), forest fragments (0.68), peridomicile (0.72).

CONCLUSION

Model results fit the general trend observed in the study system, but empirical vector infection prevalence is higher than model predictions. This higher transmission may be due to:

- Effects of relative abundance between hosts and vectors
- Transmission between vectors due to feeding on each other (kleptohemodeipnonism)
- Lack of data on wildlife reservoir competence leading to inaccurate estimation of relationship between r_{max} and competence
- Coinfections in either hosts or vectors affect susceptibility to the pathogen
- Other still unknown drivers of transmission

FUTURE DIRECTIONS

- Incorporating one or more of the missing factors into the model
- Scaling up the model to include spatial heterogeneity effects; create a network of transmission between habitats
- Using model to predict infection prevalence in places hard to sample

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