Modelling the influence of host community composition in a sylvatic *Trypanosoma cruzi* system

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SUMMARY

Species composition of wild reservoir hosts can influence the transmission and maintenance of multi-host vector borne pathogens. The 'pace of life' hypothesis proposes that the life history strategy of reservoir hosts can influence pathogen transmission of vector borne generalist pathogens. We use empirical data to parameterize a mathematical model that investigates the impacts of host life history traits on vector transmission dynamics of the vector-borne multi-host parasite *Trypanosoma cruzi* in habitats characterized by different degrees of deforestation and varying host community structure. The model considers susceptible and infected vector and host populations. When comparing the proportion of vectors infected with *T. cruzi* predicted by the model with empirical data, we found a trend of increasing vector infection as anthropogenic landscape disturbance increases for both data and model output. The model's vector infection rates were significantly lower than empirical results, but when incorporating host congenital transmission in the model, vector infection approaches field data. We conclude that intervened habitats associated with r-selected host species communities predict higher proportions of infected vectors.

Key words: Multi-host vector borne pathogen, mathematical model, Chagas disease, *Trypanosoma cruzi*, host life history strategy, host community composition, deforestation.

INTRODUCTION

Species composition of wild reservoir hosts can influence the transmission and maintenance of multi-host vector borne pathogens. In theory, life history characteristics among species within a community can be an important driver of variation in infectious disease transmission (Lochmiller and Deerenberg, 2000; Martin *et al*. 2006; Miller and Sinervo, 2007; Johnson *et al*. 2012). Relative to infectious disease transmission, the 'pace of life' hypothesis posits that r-selected species, those that have high reproductive rates and with relatively short lifespans, are expected to invest less in acquired immunity compared with species that are more 'k-selected', with lower reproductive rates and longer life spans (Martin *et al*. 2006; Lee *et al*. 2008). Furthermore, highly reproductive (r-selected) species tend to have higher litter sizes and give birth more frequently, leading to an increased number of susceptible individuals, which can lead to increased rates of infectious disease transmission in a population (Poulin and Morand, 2004; Blackwell *et al*. 2010). Therefore, among a community of hosts, 'fast-paced' species may be more competent reservoirs of infection than 'slow-living'/'longer-lived' species (Ostfeld *et al*. 2014).

The 'pace of life' hypothesis in nature has been evidenced in a variety of wildlife-parasite systems. In amphibians, Johnson *et al*. (2013), observed that 'fast-lived' amphibian species were more susceptible to infection and had more severe pathology associated with trematode infections (Johnson *et al*. 2013). Regarding vector-borne diseases, *Borellia burgdorferi* (Lyme disease agent) reservoir competence (the host ability to infect uninfected ticks) was highest in the most r-selected species and lower in more slowly reproducing species (Previtali *et al*. 2012; Ostfeld *et al*. 2014). Models evaluating reservoir host traits show that host life history traits such as lifetime reproductive output, are also an important predictor of zoonotic disease reservoir potential (Han *et al*. 2015).

In this study, we model how species composition in five different habitats characterized by different degrees of anthropogenic disturbance impacts vector and host infection with *Trypanosoma cruzi*, the cause of Chagas disease in humans. Chagas disease is a neglected zoonotic, vector-borne tropical disease that infects over 8 million humans worldwide and is a significant cause of morbidity and mortality throughout Central and South America (Bern *et al*. 2011). This parasite cycles between a wide range of mammal reservoirs and hematophagous triatomine vectors by multiple routes of transmission. Vectorial transmission is

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the most known route; however, vector ingestion by hosts seems to be the most efficient dispersion strategy of *T. cruzi* in sylvatic habitats (Yoshida, 2008, 2009; Jansen *et al*. 2015). The congenital route is relevant in humans (Carlier *et al*. 2015) and under experimental conditions in multiple mammal species infection (Alkmim-Oliveira *et al*. 2013). Finally, laboratory studies have reported direct transmission between triatomines (Ryckman, 1951; Marinkelle, 1965; Añez, 1982; Schaub, 1988) and positive serological tests in hyper-carnivorous Felidae suggest transmission between hosts by predation (Rocha *et al*. 2013); although both infection routes remain a matter of debate (Roellig *et al*. 2009).

The most important host and vector species responsible for *T. cruzi* transmission varies in different geographical, ecological and epidemiological contexts. Regardless, in many *T. cruzi* transmission systems, sylvatic cycles can dominate parasite transmission, particularly in rural landscapes or forest-urban boundaries. Our mathematical model of *T. cruzi* transmission is based on previous data collected from rural landscapes of Panama (Gottdenker *et al*. 2012), where evidence suggests that habitat type (degree of deforestation) and host community composition are important drivers for vector infection prevalence with *T. cruzi*. Specifically, we incorporate biologically realistic aspects of sylvatic infection into our model, including acute and chronic infection status in hosts, host predation on vectors, and host vertical transmission to better understand their relative importance in *T. cruzi* transmission and to identify critical parameters to measure in field research.

METHODS

We developed a mathematical model that assumes the basic dynamics of sylvatic *T. cruzi* transmission cycle, with five coupled ordinary differential equations. We considered vector and mammal hosts, both partitioned into susceptible and infected populations. Additionally, we included a chronically infected population for hosts. We maintained a constant population size where birth rate equals death rate. Figure 1 shows the populations considered in the model with their interactions.

Vector dynamics

The vector population is defined by N_v and is divided into two compartments: susceptible (S_v) and infected (I_v). Vectors are born at a *per capita* rate λ_v that is equal to death rate. S_v moves into I_v by biting infected hosts (I_h) or chronically infected hosts (C_h), and is described by a Holling type II functional response (Table 1);

$$g(I_h) = \frac{\varepsilon I_h}{\rho + I_h} \tag{1}$$

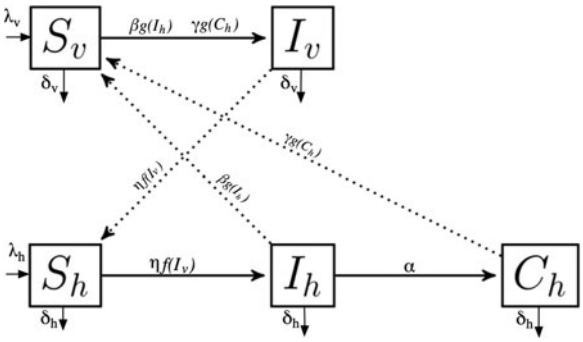

Fig. 1. Model diagram of *Trypanosoma cruzi* sylvatic system. The model considers vector (S_v, I_v) and host populations (S_h, I_h, C_h), with their vital dynamics and interactions. For parameters description see Table 2.

Table 1. Variables of the model

Symbol	Name
S_v	Susceptible vector population
I_v	Infected vector population
S_h	Susceptible host population
I_h	Infected host population
C_h	Chronically infected host population

where ε is the maximum number of hosts bitten per week per vector and ρ is the proportion of hosts when host bitten is ε/2.The infection probability of the susceptible vector population is β if the encounter is with infected host class and γ if the encounter is with the chronically infected host population. Parasite transmission from chronically infected hosts to vectors is less likely compared with transmission from the acutely infected host population, thus $\beta \gg \gamma$. Biological parameters with their values and units are summarized in Table 2.

$$\frac{dS_v}{dt} = \lambda_v(S_v + I_v) - \beta g(I_h)S_v - \gamma g(C_h)S_v - \lambda_v S_v \tag{2}$$

$$\frac{dI_v}{dt} = \beta g(I_h)S_v + \gamma g(C_h)S_v - \lambda_v I_v \tag{3}$$

Host dynamics

We considered that host population N_h is constant, thus birth rate λ_h is equal to death rate. In the model the main transmission route by which wild hosts become infected is ingestion of an infected vector. Therefore, susceptible hosts (S_h) get infected by feeding on infected vectors (I_v) and the probability of infection by this route is defined by η. Host predation on vector population is described by a Holling Type II functional response;

$$f(I_v) = \frac{\phi I_v}{\sigma + I_v} \tag{4}$$

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Table 2. Model parameters

Symbol	Name	Units	Value	References
λ_v	Vector birth rate	1/time	0·21	Rabinovich and Nieves (2011)
λ_h	Host birth rate (community)	1/time	See Fig. 2	Carey and Judge (2002), Ernest (2003), Weigl (2005) and Tacutu *et al*. (2012)
ε[a]	Maximum number of hosts bitten per unit time per vector	1/time	1	
ρ[a]	Proportion of hosts when host biting is $\varepsilon/2$		0.5	
β	Probability of infection from I_h to S_v		0·465	Perlowagora-Szumlewicz *et al*. (1990)
β_{high}	Highest probability of infection from I_h to S_v		1	
γ	Probability of infection from C_h to S_v		0·026	Gurtler *et al*. (1996)
γ_{high}	Highest probability of infection from C_h to S_v		0·1	
α	Movement rate from I_h to C_h	1/time	See Fig. 2	Jansen *et al*. (1991) and Añez *et al*. (2011)
η	Probability that intake infects the host		0·177	Kribs-Zaleta (2010)
ϕ[a]	Maximum number of vectors eaten per unit time per host	1/time	[0·1–2] species diet[b]	See Supplementary material
σ[a]	Proportion of vector when vector consumption is $\phi/2$		0·5	
κ	Infection rate between vectors $I_h - S_v$	1/time	[0–1]	

[a] These parameters correspond to the Holling Type II functional response.
[b] Insectivorous hosts: 1–2 ind/week×ind;depending on preference for insects *vs*. other food sources, non-insectivorous hosts: 0·1 ind/week×ind; to reflect only vectorial transmission.

where ϕ is the maximum number of vectors eaten per week per host and σ represents the proportion of vectors when vector consumption is $\phi/2$. Therefore, $\sigma = 0.5$ means that host consumption rate on infected vectors is the half ($\phi/2$) when 50% of the vectors are infected. In addition, the model assumes that non-insectivorous hosts become infected only by a vectorial route using the same expression, but modifying ϕ to reflect the adequate rate of transmission, thus ϕ depends on species diet.

After an infectious period (α), the infected host population (I_h) moves into a chronically infected class (C_h). Parameters are described in Table 2. λ_h and α depend on the particular host species life history strategy (see Supplementary material).

$$\frac{dS_h}{dt} = \lambda_h(S_h + I_h + C_h) - \eta f(I_v)S_h - \lambda_h S_h \quad (5)$$

$$\frac{dI_h}{dt} = \eta f(I_v)S_h - \alpha I_h - \lambda_h I_h \quad (6)$$

$$\frac{dC_h}{dt} = \alpha I_h - \lambda_h C_h \quad (7)$$

Host community composition: life history strategy and diet parameters

We modelled the epidemiological dynamics of five habitat types across different degrees of anthropogenic disturbance common in neotropical landscapes, namely contiguous forest, early secondary forest fragment, mid secondary forest remnant, pasture and peridomicile. For each habitat we know about the host species composition based on insect blood meal data collected in the Panama Canal (Gottdenker *et al*. 2012) (Fig. 2). Thus, for each habitat we had different model parameters.

We considered that host birth rate (λ_h), the rate at which infected hosts become chronically infected (α) and diet (ϕ) are the most relevant parameters in our epidemiological model when moving from one species to another. λ_h and α are associated with species life history strategy, where α is a measure of the host ability to generate an acquired immune response and it is correlated with a particular species life history strategy. ϕ depends on whether insects have been reported to be part of host dietary intake, thus the parameter varies from one host species to another (for information about host species diet and ϕ values see Supplementary material). Therefore, insectivorous hosts were assumed to ingest 1–2 insects per week depending on preference for insects over other food sources and non-insectivorous hosts 0·1 insects per week to reflect only vectorial transmission. The transmission probability per contact with an infected triatomine has been reported to be between: $10^{-4} - 10^{-2}$ (Rabinovich *et al*. 1990, 2001; Catala *et al*. 1992; Basombrío *et al*. 1996; Nouvellet *et al*. 2013).

Therefore, the infection probability by the vectorial route is lower compared with the oral route.

The product of species litter size and breeding frequency results in λ_h (see Supplementary material). α for all species is difficult to obtain from the literature. However, based on the average parasitaemia reported for *Didelphis marsupialis* and mice after *T. cruzi* experimental inoculation, and considering

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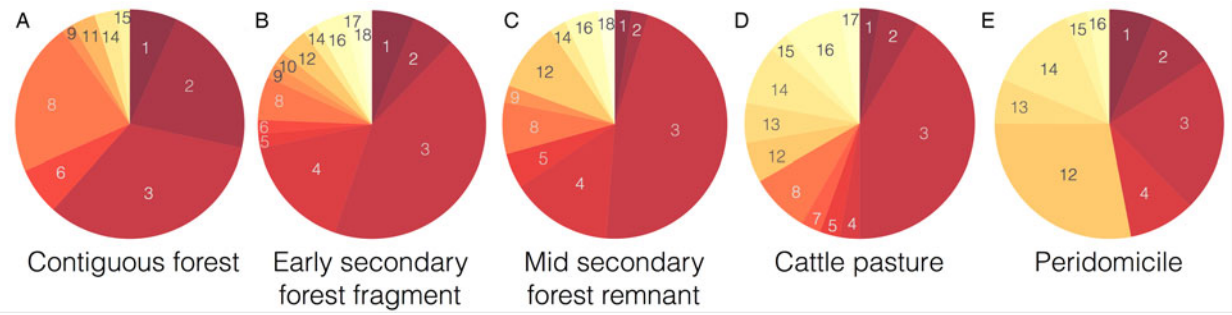

Fig. 2. Proportion of blood meals taken from different species per habitat from Gottdenker *et al*. (2012). Each number inside the pie chart represents a species coded as follows: 1 – *Alouatta palliata*, 2 – *Cebus capucinus*, 3 – *Choloepus hoffmanni*, 4 – *Bos taurus*, 5 – *Coendou* sp., 6 – *Potos flavus*, 7 – *Cyclopes didactylus*, 8 – *Tamandua tetradactyla*, 9 – *Sciurus* sp, 10 – *Heteromyidae* sp, 11 – *Mustela* sp, 12 – *Metachirus nudicaudatus*, 13 – *Sus scrofa*, 14 – *Didelphis marsupialis*, 15 – *Philander opossum*, 16 – *Canis familiaris*, 17 – *Marmosa sp* and 18 – *Mus musculus*. Species within the pie charts are ordered and colored from the lowest (1 dark red – *A. palliata*) to the largest birth rate (18 light yellow – *M. musculus*). Note how the proportion of blood meal coming from species that are more prone to spend time close to dwellings increases as ones moves from the sylvatic to domestic cycle.

a more robust acquired immunity for longer-lived hosts, we assumed α values for all the species. Host species with birth rates higher than $0 \cdot 09$/week, such as *D. marsupialis* and *Mus musculus*, were considered as r-selected in a host community and the period of moving from infected (high parasitemia) to chronically infected (low parasitemia) is 12 weeks, or α equals to 1/12 weeks (Jansen *et al*. 1991; Añez *et al*. 2011). On the other hand, host species with birth rate values less than $0 \cdot 01$/week correspond to K-selected and α equals to 1/4 weeks. If λ_h is between 0·01 and 0·09/week, α equals 1/8 weeks. See Supplementary material for values and references on species individual parameters.

To simulate species composition for each habitat, we estimated λ_h, α and ϕ by bootstrapping the distributions reported by Gottdenker *et al*. (2012) illustrated in Fig. 2. In particular per habitat, we generated 1000 samples by re-sampling with replacement the original distribution. Bootstrapping was conducted to increase variability and compensate for possible field sample bias. Thus, every habitat has an average composite life history strategy based on the species present; this average is marked on Fig. 3 as the mean of the distributions. For each sample distribution we solved the model to steady state (260 weeks/5 years) and recorded the values of the state variables (i.e. proportion of infected hosts and vectors). The initial conditions of the model were: $S_v = 0 \cdot 9$, $I_v = 0 \cdot 1$, $S_h = 0 \cdot 9$, $I_h = 0 \cdot 1$ and $C_h = 0$; however, the steady-state results are independent from the initial conditions. The distribution of the parameters empirically obtained after the simulations for each habitat are shown on Fig. 3.

Model variation: including host vertical transmission route

We incorporated congenital transmission in hosts. The equations describing host population dynamics are amended as follows:

$$\frac{dS_h}{dt} = \lambda_h S_h - \eta f(I_v) S_h - \lambda_h S_h \quad (8)$$

$$\frac{dI_h}{dt} = \lambda_h (I_h + C_h) + \eta f(I_v) S_h - \alpha I_h - \lambda_h I_h \quad (9)$$

$$\frac{dC_h}{dt} = \alpha I_h - \lambda_h C_h \quad (10)$$

Note that if an infected (I_h) or chronically infected host (C_h) produces offspring in the infected compartment, $\lambda_h(S_h + C_h)$. A total of 1000 resamples were generated per habitat type and the model was run until steady state (260 weeks/5 years). The initial conditions of the model were: $S_v = 0 \cdot 9$, $I_v = 0 \cdot 1$, $S_h = 0 \cdot 9$, $I_h = 0 \cdot 1$ and $C_h = 0$, and the proportion of infected populations per habitat was recorded.

Finally, considering that the probability of infection of a S_v by a I_h is 0·465 (β) and it is an estimate for *Rhodnius prolixus* feeding on acute guinea pigs (Perlowagora-Szumlewicz *et al*. 1990) and similarly the probability of infection of a S_v by a C_h is 0·026 (γ) and was calculated from a experiment in which *Triatoma infestans* feeds on seropositive humans (Gurtler *et al*. 1996), we decided to also test the model with increased probabilities (β_{high}, γ_{high}) to account for more competent hosts (Orozco *et al*. 2016) see Table 2.

The model was implemented using R version 3.3.2 (R Development Core Team, 2016) and the RStudio Integrated Development Environment (IDE). The ordinary differential equations were solved using the 'deSolve' package (Soetaert *et al*. 2010) and bootstrapping was conducted using the sample function, 'base' package version 3.3.2.

RESULTS

Analysis of the model

The basic reproduction number R_0 is the average number of infections produced by the contribution

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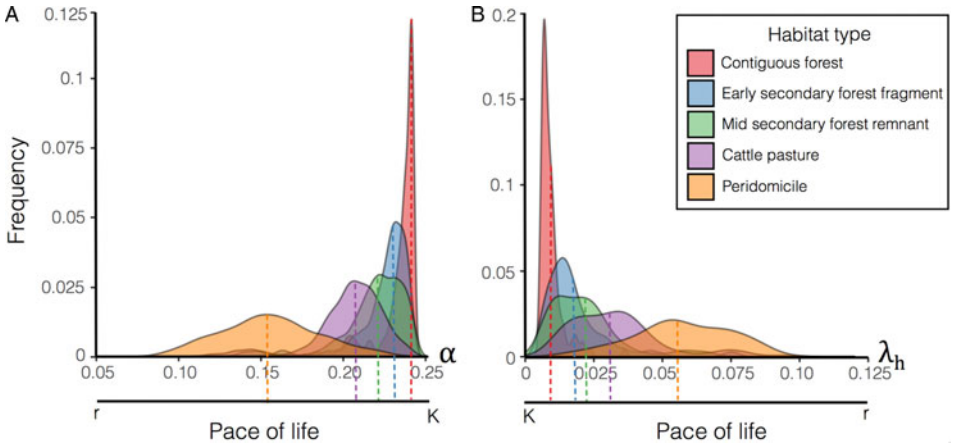

Fig. 3. Distribution of host life history strategy related parameters: α (movement rate from acute to chronically infected) and λ_h (birth rate). The frequency distribution for α and λ_h is plotted colour-coded per habitat for 1000 samples. Bootstrapping the proportion of blood meals taken from different species (Fig. 1), we obtained 1000 different arrangements per habitat. Each species $1/\alpha$ and λ_h estimates are based on previous reports and life history strategy (see Supplementary material), therefore for each model simulation we used the average value of α and λ_h obtained from the resampled ensemble. α is higher in forests compared with other habitats because host community tends to last shorter periods of time in the acute state probably due to a more robust acquired immunity of the longer lived hosts, similarly λ_h is higher in peridomicile due to higher litter size and shorter inter-birth intervals.

of the whole network. It is a valuable quantity because it tells whether the infection is going to prevail ($R_0 \geqslant 1$) or disappear and can be used to compare different scenarios. We found R_0 by identifying the largest eigenvalue of Next Generation Matrix method (NGM) (Diekmann *et al*. 1990, 2010). The NGM has the number of new infected individuals of a class (columns) generated by the introduction of an infected individual of every other class (rows). Thus, for building the NGM, we only consider three of five compartments of our system belonging to infected stages (I_v,I_h and C_h).

$$\text{NGM} = \begin{pmatrix} 0 & \dfrac{\beta\varepsilon}{\rho(\alpha+\lambda_h)} + \dfrac{\alpha\varepsilon\gamma}{\lambda_h\rho(\alpha+\lambda_h)} & \dfrac{\varepsilon\gamma}{\lambda_h\rho} \\ \dfrac{\phi\eta}{\sigma\lambda_h} & 0 & 0 \\ 0 & 0 & 0 \end{pmatrix} \tag{11}$$

See Supplementary material for more information about NGM construction.

From this product, the greatest eigenvalue is the basic reproductive number.

$$R_0 = \sqrt{\frac{\varepsilon\phi\eta(\beta\lambda_h + \alpha\gamma)}{\sigma\rho\lambda_v\lambda_h(\alpha+\lambda_h)}} \tag{12}$$

To understand the parameter effects on model output, we performed a sensitivity analysis for R_0 using the Latin Hypercube method (LH). Negative values indicate that an increase in a particular parameter determines a decrease in R_0 and positive values an increase in R_0. Therefore, increases in β, γ, η, ϕ and ε generate increments in R_0 (Fig. 4). η(PRCC = 0·7), ϕ(PRCC = 0·7) and ε (PRCC = 0·7) have the higher effect on R_0. In addition, the transmission rate from infected hosts to susceptible vectors (β) has a significant effect (PRCC = 0·5). On the other hand, the parameter that has the most significant effect on lowering R_0 is the host birth rate (λ_h, PRCC = –0·5).

Effect of species composition on the proportion of infected individuals

Each habitat blood meal distribution was resampled 1000 times. We ran the model until it reached steady state (260 weeks/5 years) and record the proportion of infected individual (host and vectors) per habitat. The proportion of infected vectors has the lowest prevalence in the contiguous forest with an $I_v = 0 \cdot 121 (0 \cdot 118 - 0 \cdot 124)$. Whereas in the early secondary forest fragment ($I_v = 0 \cdot 142, 0 \cdot 138 - 0 \cdot 146$), mid-secondary forest remnant ($I_v = 0 \cdot 16,\ 0 \cdot 155 - 0 \cdot 165$), pasture ($I_v = 0 \cdot 224, 0 \cdot 219 - 0 \cdot 229$) and peridomicile ($I_v = 0 \cdot 36, 0 \cdot 355 - 0 \cdot 365$), the proportion is significantly higher ($P < 0 \cdot 05$) compared with the contiguous forest (Welch two sample *t*-test 95% CI, Fig. 5).

Similarly, I_h has the same tendency as I_v, being the contiguous forest the lowest I_h ($0 \cdot 021$, $0 \cdot 019 - 0 \cdot 022$) and the peridomicile the highest among all habitats ($I_v = 0 \cdot 163,\ 0 \cdot 159 - 0 \cdot 167$). The proportion of infected hosts in the early secondary forest ($I_h = 0 \cdot 031,\ 0 \cdot 029 - 0 \cdot 033$), mid secondary forest ($I_h = 0 \cdot 039,\ 0 \cdot 036 - 0 \cdot 041$) and pasture ($I_h = 0 \cdot 066, 0 \cdot 063 - 0 \cdot 069$) is significantly different ($P < 0 \cdot 05$) from the I_h in the contiguous forest (not shown).

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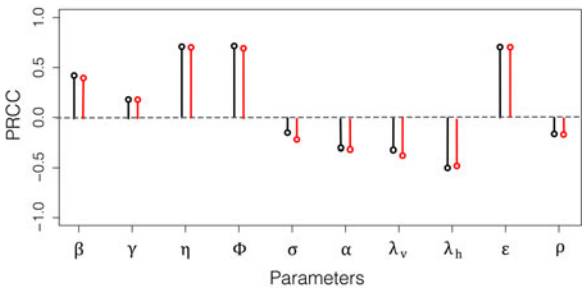

Fig. 4. Latin Hypercube Sampling using the global reproductive number R_0 as model output. PRCC: Partial Rank Correlation Coefficient. Black coloured circles correspond to the model without horizontal transmission and red to the model including host vertical transmission. For information about each parameter's meaning see Table 2. Note that parameters related to host predation on vectors (η and ϕ), vector biting on hosts (ε), and host life history strategy (λ_h) have significant and opposite contributions to R_0.

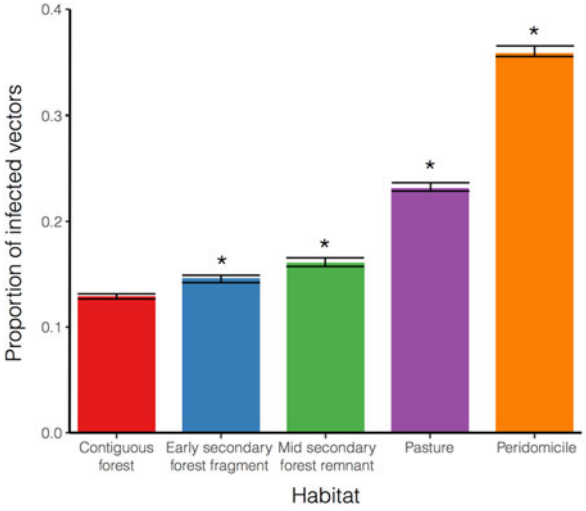

Fig. 5. Proportion of infected vector population (I_v) per habitat at steady state. Results are shown for contiguous forest, early secondary forest fragments, mid secondary forest remnant, pasture and peridomicile. Bar heights represent the average for the 1000 runs and the error bar shows the 95% of confidence interval. *Significant differences compared to contiguous forest (Welch Two Sample *t*-test, 95% CI). Habitats with forest components (three bars on the left) have an I_v less than 20%, being the contiguous forest the lowest I_v, while the more intervened (two on the right) are well above 20%. Note also that infected vector population has an increasing trend as habitat alteration increases.

Vertical transmission in hosts

The model including vertical transmission showed higher proportion of infected vectors for each habitat compared with the model without this transmission mode. As before, the contiguous forest showed the lowest proportion of infected vectors ($I_v = 0 \cdot 199$, $0 \cdot 195 - 0 \cdot 203$) and the peridomicile the highest proportion of infected vectors($I_v = 0 \cdot 445$, $0 \cdot 441 - 0 \cdot 449$). Consequently, regarding host population infection, the lowest proportion was reported for the contiguous forest ($I_h = 0 \cdot 045$, $0 \cdot 043 - 0 \cdot 047$) and the highest proportion for the peridomicile ($I_h = 0 \cdot 271$, $0 \cdot 265 - 0 \cdot 277$).

By increasing the values for β and $\gamma(\beta_{high}, \gamma_{high})$ the model including vertical transmission better approached field data. Similarly to previous results, the contiguous forest presented the lowest proportion of infected vectors ($I_v = 0 \cdot 402$, $0 \cdot 398 - 0 \cdot 406$); followed by the secondary forests (early secondary forest fragment $I_v = 0 \cdot 460$, $0 \cdot 455 - 0 \cdot 465$; mid secondary forest remnant $I_v = 0 \cdot 482$, $0 \cdot 477 - 0 \cdot 487$) pasture ($I_v = 0 \cdot 552$, $0 \cdot 548 - 0 \cdot 556$) and peridomicile ($I_v = 0 \cdot 646$, $0 \cdot 642 - 0 \cdot 650$). Infected host population exhibited the same pattern, the proportion of infected hosts in the contiguous forest was ($I_h = 0 \cdot 046$, $0 \cdot 044 - 0 \cdot 048$) and in the peridomicile ($I_h = 0 \cdot 271$, $0 \cdot 265 - 0 \cdot 277$).

DISCUSSION

Here, we present an epidemiological model that investigates the transmission dynamics of *T. cruzi* in five different sylvatic environments in the Panama Canal with different anthropogenic intervention levels influencing host communities. Our model shares many features with previous theoretical studies. For instance, studies like the one by Spagnuolo *et al.* (2011) that investigate the effect of spraying dwellings on disease endemicity (Spagnuolo *et al.* 2011), or other research teams studying the role of dogs (Fabrizio *et al.* 2016) and synanthropic animals in human infection (Peterson *et al.* 2015), also use traditional compartmental epidemiological models. Other studies like Coffield in 2013 and Kribs-Zaleta in 2010 used Holling functions to simulate host predation on vectors (Kribs-Zaleta, 2010; Coffield *et al.* 2013). Therefore, we feel confident that the model proposed here can be a simple, yet powerful tool to understand the role of host community composition in sylvatic *T. cruzi* transmission cycles.

In addition to being a good tool, the model also adjusts to real data. When comparing the proportion of *Rhodnius pallescens* infected with *T. cruzi* predicted by the model without vertical transmission, with the proportion actually found (Gottdenker *et al.* 2012), we note that there is a trend of increasing infection as the anthropogenic landscape disturbance increases for both data and model output (see Fig. 5). Varying parameters on host life history strategy based on habitat's host community composition and blood meal distribution (Gottdenker *et al.* 2012), altered the infected vector and host populations. More specifically, habitats with a higher proportion of r-selected species within the host community, such as peridomicile sites, presented higher vector infection rates in the field and simulations (see Fig. 6). For instance, opossum blood meals

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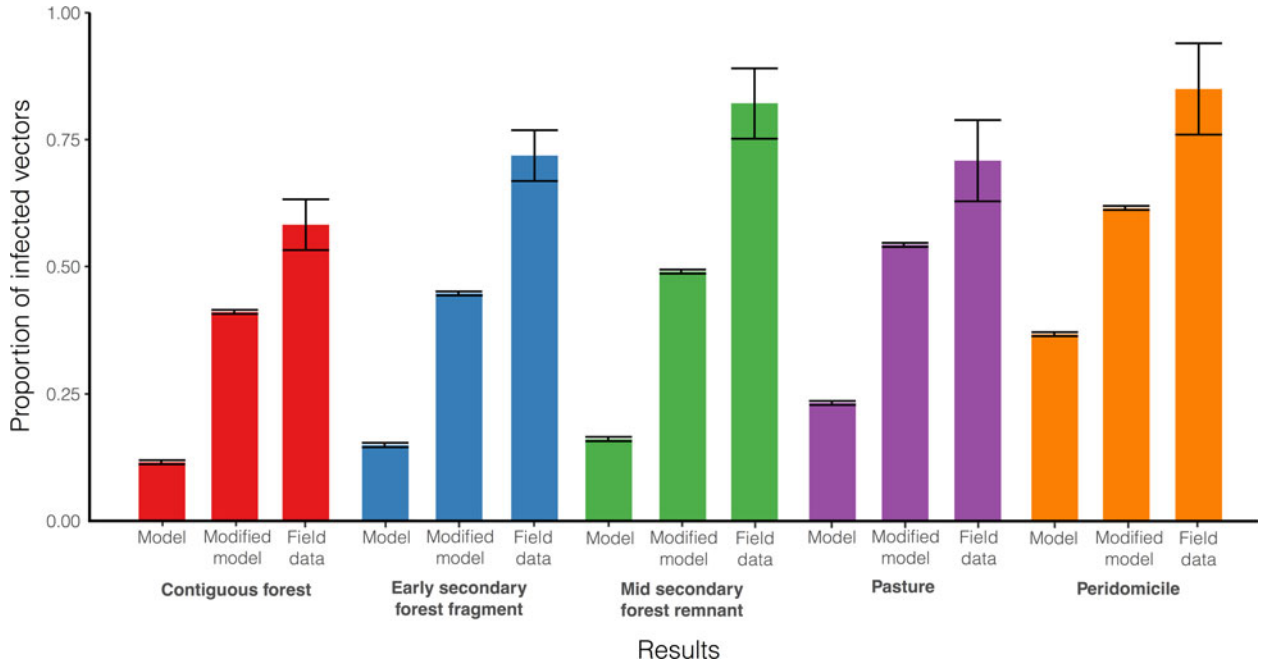

Fig. 6. Infected vector population (I_v): comparison between the model without host vertical transmission, including host vertical transmission with the highest vector transmission probabilities (β_{high}, γ_{high}) and field results. Results are shown for the five selected habitats, for each bars set grouped by colour, the left and middle bars correspond to model results and the bar on the right to field results (Gottdenker *et al*. 2012). Host vertical transmission dynamics are represented by equations (8)–(10). The model including vertical transmission considering high probabilities of transmission in vectors better approaches field data compared with the model accounting only for oral and vectorial transmission.

showed a noticeable increase in disturbed habitats and domestic species were only detected in anthropogenically altered habitats (Gottdenker *et al*. 2012).

However, field data showed values that are much higher compared with simulation results. While the proportion of infected vectors found in the contiguous forest is the lowest with 58% (54 – 63%, 95% CI) and in the peridomicile is the highest with 85% (76 – 94%, 95% CI), the model without host vertical predicted 12 and 36%, respectively. The same pattern occurs in intermediately disturbed habitats. We argue that this discrepancy could point out previously unconsidered mechanisms involved in parasite transmission. In other words, the model suggests that vectorial and oral routes are not sufficient to explain the high proportion of infected vectors found in the Panama Canal. Therefore, we decided to incorporate host vertical transmission in the model. Congenital transmission, under experimental studies, impacts infection in host populations (Alkmim-Oliveira *et al*. 2013) and currently, the only observation of congenital *T. cruzi* transmission in nature was reported in bats (Añez *et al*. 2009). Furthermore, in opossums (*Didelphis marsupialis*) it is possible that offspring may become infected from the perineal region during birth, as infectious stages of *T. cruzi* have been found in the anal glands (Deane *et al*. 1984). When we incorporate vertical transmission in hosts accompanied by an increase in the probability of infection from the infected host populations (I_h and C_h) to the susceptible vector population (I_v), our results are more similar to the empirical data from the Panama Canal (Fig. 6) than when we do not include this mechanism in the model.

Another *T. cruzi* transmission mechanism that we do not simulate here, but could be important, is vector direct transmission. Previous laboratory studies have reported 'kleptohemodeipnonism' or the act of stealing blood, as a means of infection between triatomines (Ryckman, 1951; Marinkelle, 1965; Añez, 1982; Schaub, 1988). In addition, Gottdenker *et al*. (2012) reported nymphs collected from palms that contain blood from domestic animals, suggesting that they may have fed from engorged adults who fed on these terrestrial mammals and then returned to the palm (Gottdenker *et al*. 2012). To the authors' knowledge, there are no reported observations of this process in the wild, as it can be very difficult to observe wild vector behaviour '*in situ*' and requires further research.

Although useful, our modelling approach has some limitations. The first limitation is considering the whole set of different host species in a particular habitat as an average ensemble of host populations. An alternative approach to study host community composition could be to model each host population independently; however, the lack of data on host species parameters related to *T. cruzi* transmission makes this a highly hypothetical approach. Another limitation is that the assumption of host species ingesting 1–2 vectors per week on average ignores that predation is opportunistic and many

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other insects besides vectors might be available to hosts (Kribs-Zaleta, 2010). Another model limitation relies in using *Rhodnius neglectus* intrinsic rate instead of *R. pallescens*. Both species belong to the same genus, but different clade (Monteiro *et al*. 2000); suggesting potential discrepancies in their life cycles. In addition, the model does not include spatial aspects of disease transmission, which may differ between discrete locations impacting the proportion of infected vectors. For instance, palm trees, the primary habitat of *R. pallescens* (De Vasquez *et al*. 2004) and most of *Rhodnius* species (Abad-Franch *et al*. 2015), could vary on their physiognomy, shaping bug population density and age distribution (Urbano *et al*. 2015). In addition, palm spatial location could determine triatomine blood meals affecting epidemiological indices (Suarez-Davalos *et al*. 2010). Thus, in an endemic area, certain palm trees might play an important role as spatial 'source' foci where enhanced transmission could be taking place. The preceding considerations are even more relevant if one acknowledges the importance of host predation on vectors based on the results of the sensitivity analysis.

Finally, infected host populations could be studied using the model; however, for the Panama Canal area we were not able to contrast our results to field data yet. Comparing our results with an experimental study conducted in a protected and a disturbed area in the Argentine Chaco, the model supports that *T. cruzi* infection within host species is significantly higher in disturbed habitats (Orozco *et al*. 2016). A similar pattern was also reported for an Atlantic Rain Forest landscape in Brazil, where patch size was studied as a means of habitat fragmentation and smaller patches showed higher *T. cruzi* prevalence in small mammals as compared with larger patches (Vaz *et al*. 2007). Additionally, the range values for the infected host population (2–27%) found with the models are comparable with the results reported by both studies, which were 5·7–22% (Orozco *et al*. 2016) and 6–25% (Vaz *et al*. 2007). We suggest that conducting more detailed field studies, including extensive sampling of mammal hosts potentially involved in *T. cruzi* transmission in habitats varying in the levels of disturbance, will help clarify the dynamics of transmission in anthropogenically altered habitats.

SUPPLEMENTARY MATERIAL

The supplementary material for this article can be found at https://doi.org/10.1017/S0031182017001287

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