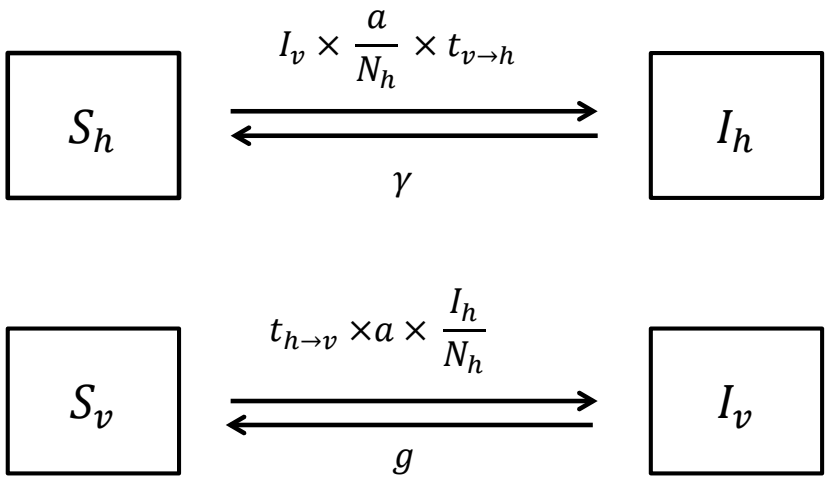
*Stochastic simulation of Ross-MacDonald model with host bottlenecks:* To more easily consider the effects of fluctuations in host populations, we developed a stochastic simulation based on the Ross-MacDonald model (Supplementary material). As the simulation tracks the numbers of infected hosts and vectors rather than their proportions, we follow convention and use the SIR (susceptible, infectious, removed/recovered) notation to track the number of hosts and vectors in each category. Susceptible hosts ($S_h$) are infected at a rate based on the proportion of infected vectors ($I_v$ /$N_v$ ), the daily number of bites on a host per vector (a), the total number of available hosts ($N_h$), and the infectivity of vectors to hosts (b). Infectious hosts ($I_h$) may revert to an uninfectious state at a rate (1/r). Similarly, susceptible vectors ($S_v$) are infected at a rate based on the daily number of bites on a host per vector (a), the infectivity of hosts to vectors (c), and the proportion of hosts infected ($I_h$/$N_h$).




**A compartmental representation of the stochastic Ross-MacDonald model to describe *T. cruzi* transmission within a vector colony. Parameters of the model are described in Table 1.**

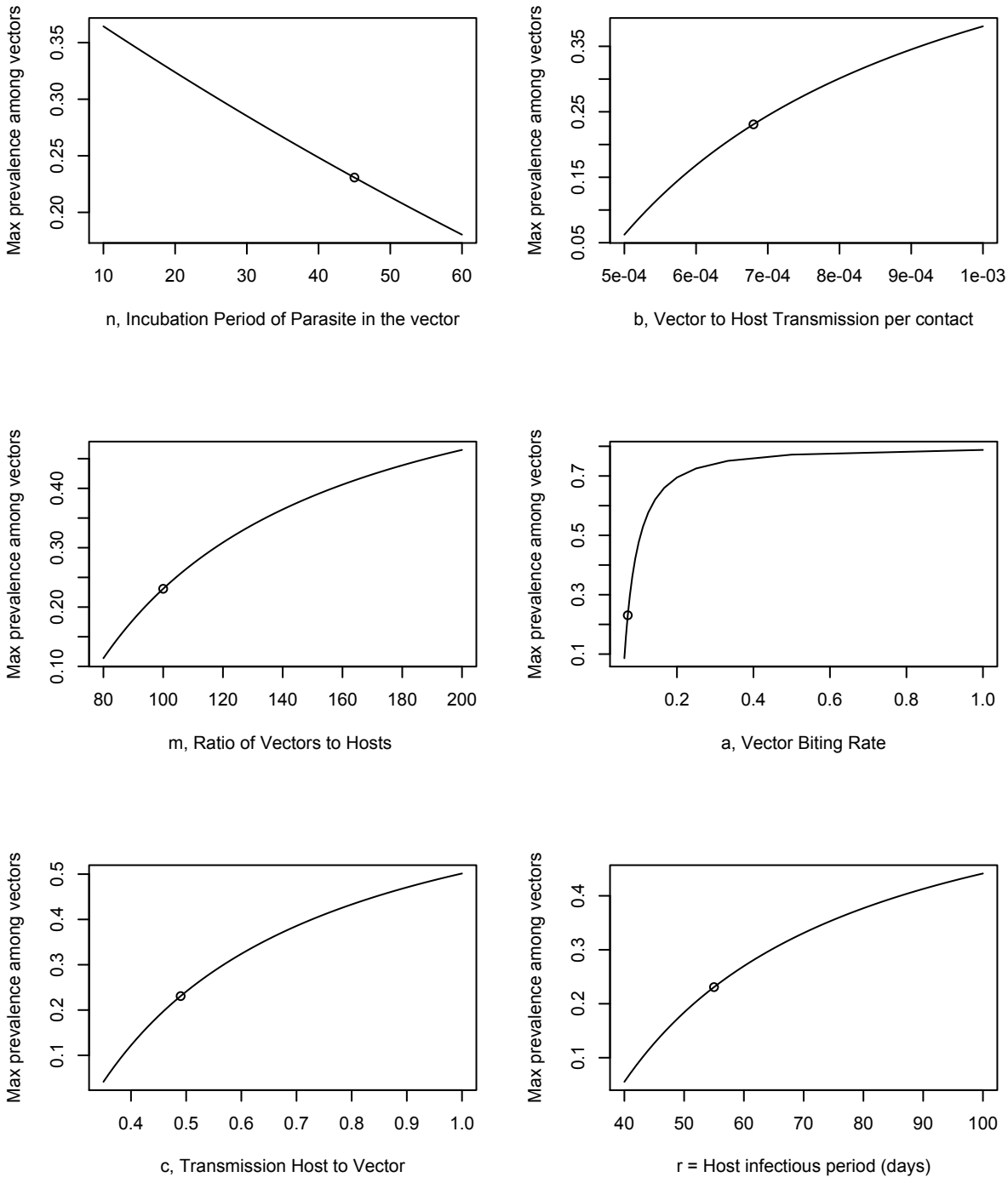



**Figure S1.** Sensitivity of the deterministic Ross-MacDonald model to changes in model parameters. In each case a single parameter, noted on the x-axis is varied while the additional parameters are held fixed at the values described in Table 1 of the main text.

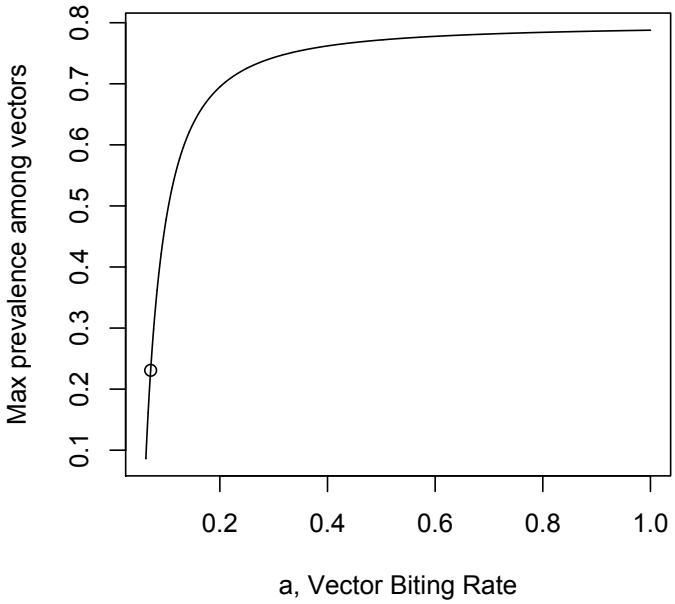



**Figure S1 continued.** Sensitivity of the deterministic Ross-MacDonald model to changes the vector biting rate.

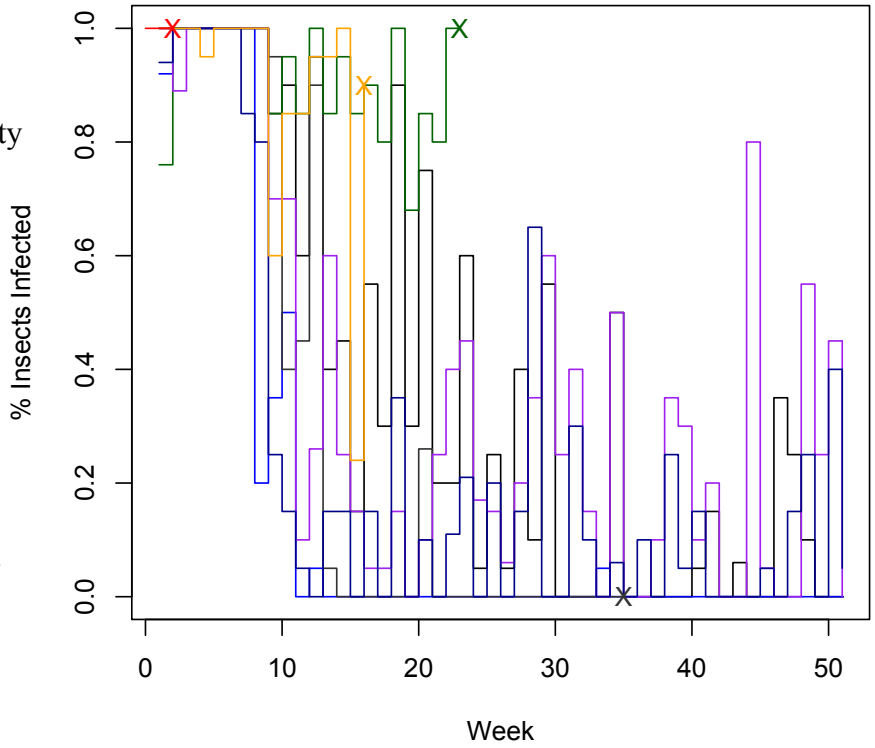



**Figure S2.** Observed variability in the percentage of triatomines infected (out of sets of 20 exposed insects) after feeding on eight experimentally-infected guinea pigs over the course of a year. Different colours represent separate animals. Guinea pigs were initially inoculated with $10^6$/100µl of a local strain of *T. cruzi*.

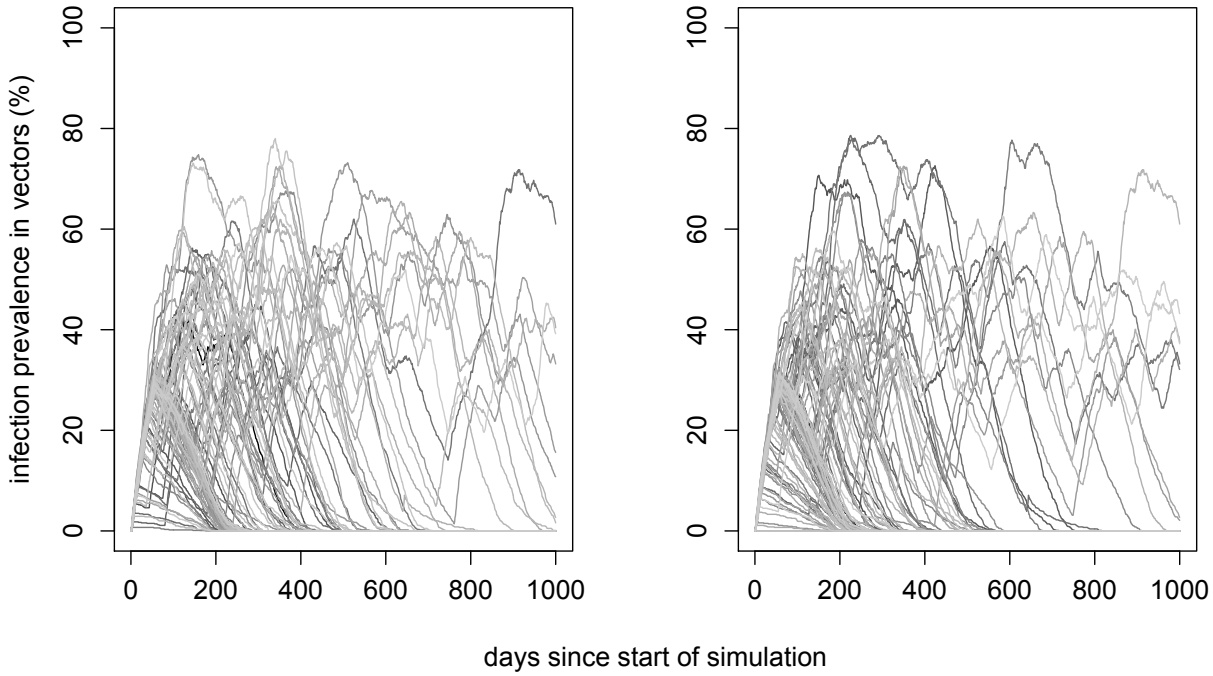



**Figure S3**. Simulated dynamics of *T. cruzi* through colonies of *T. infestans* in guinea pig enclosures in which the guinea pigs have uniform and short durations of infectiousness (left) or heterogenous infectiousess. For the heterogenous infectiousess simulation guinea pigs were drawn, with replacement, from the experimental animals, and their level of infectiousness, as a funtion of time since initial infection, was drawn from the experimental data shown in Figure 4 of the main text.

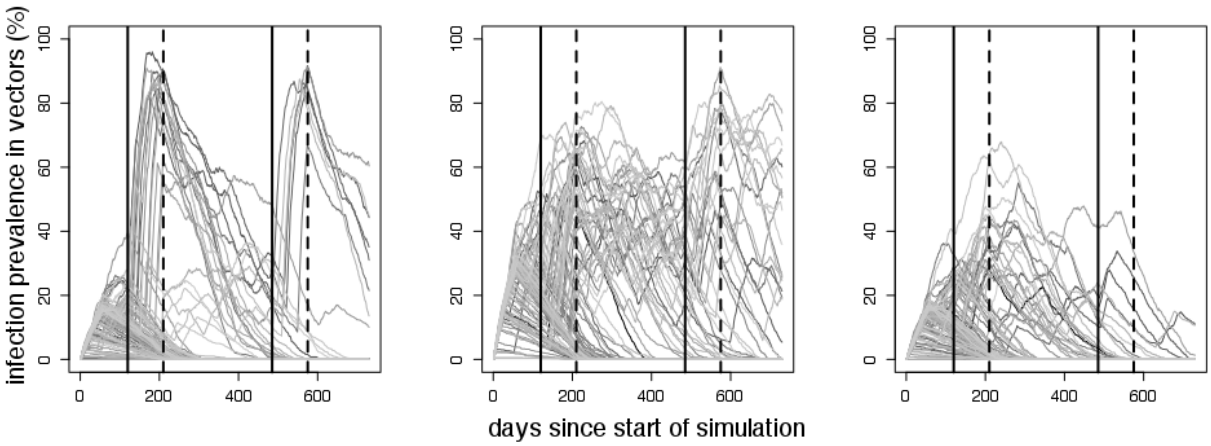



**Figure S4.** Simulated time course of *Trypanosoma cruzi* in colonies of *Triatoma infestans* in Guinea Pig Enclosures when hosts are subject to seasonal bottlenecks of varying sizes. A host bottleneck from 20 to 2 individuals (Left); from 10 to 5 (Center); and, from 20 to 10 (Right). Bottleneck periods are delimited by the solid and dashed vertical lines

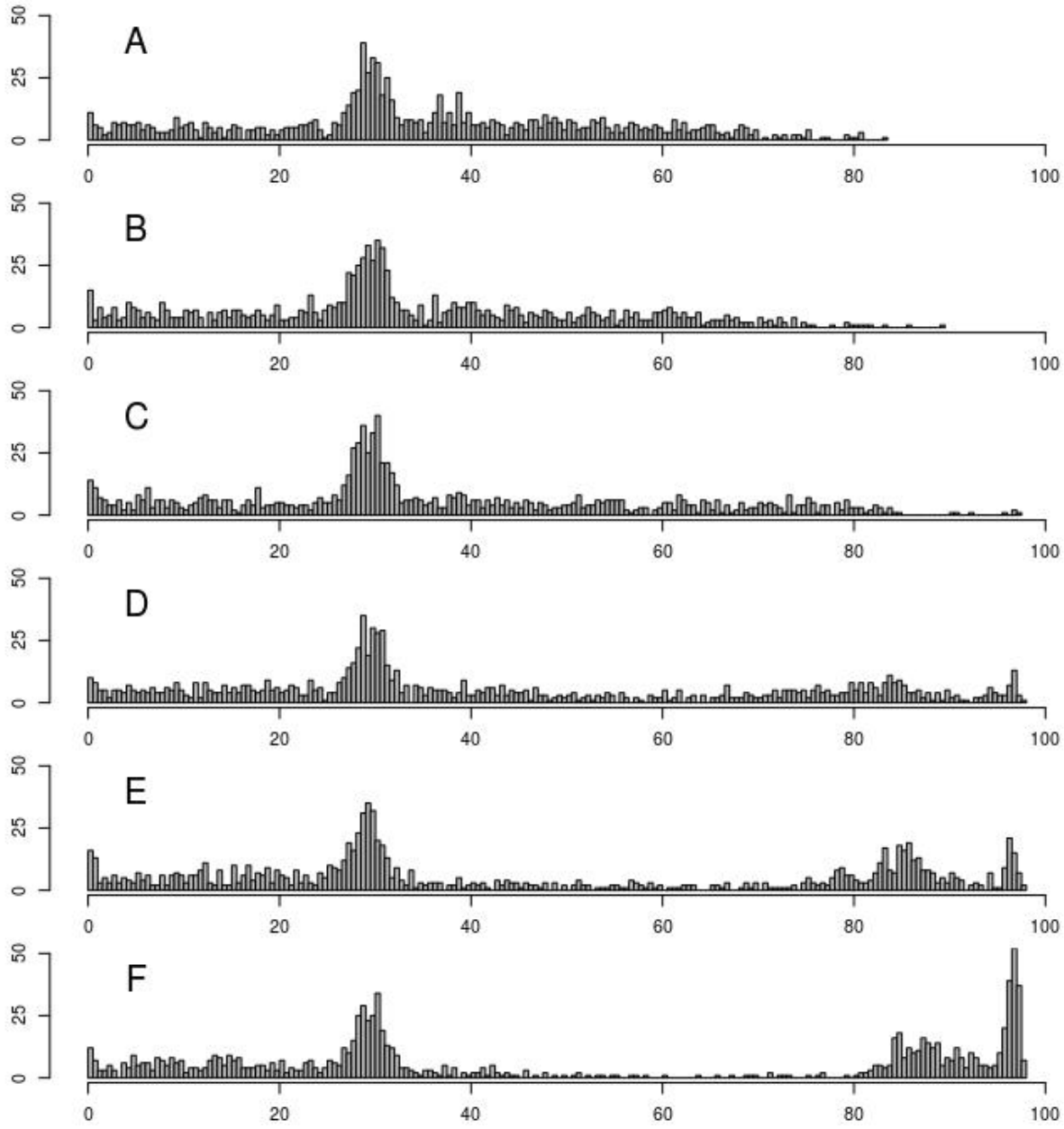



**Figure S5.** The effect of host bottlenecks on the prevalence of *Trypanosoma cruzi* in colonies of *Triatoma infestans*. Simulations tracked 1,000 vectors and a host population of ten guinea pigs that declined to two hosts during bottleneck periods. A total of 1,000 replicate simulations were run for each bottleneck length. The x-axis of each histogram shows the *maximum* infection prevalence reached in vectors over the time course of the simulation. The y-axis shows the frequency (number of simulations). Panel A: No bottleneck. Panels B-F: Bottleneck durations of 14, 30, 60, 90, and 180 days, respectively.

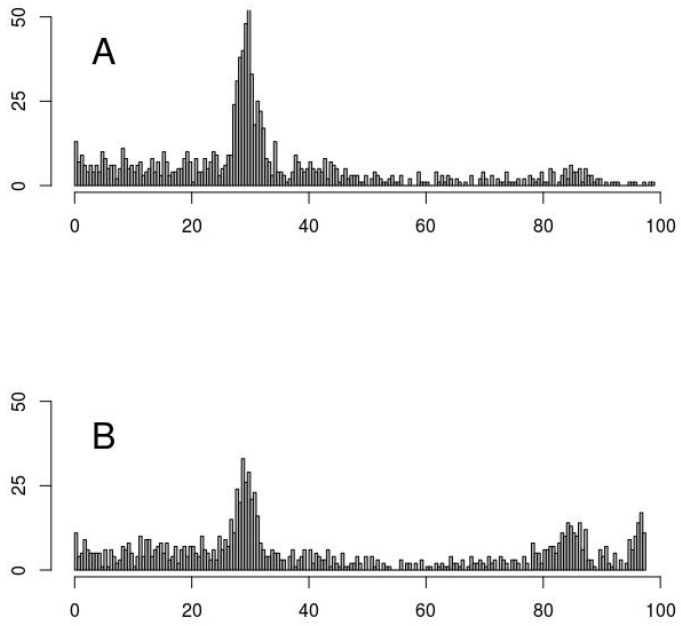



**Figure S5b.** Effects of Bottlenecks in the Vector population. In a separate sensitivity analysis, we allowed the number of vectors to fall from 1,000 to 200 (a decline of 80%, identical to the percentage drop in the guinea pig population) concurrently with host bottlenecks. During the bottleneck, the median peak prevalence in vectors declined from 22% to 18%, and the 75$^{th}$ percentile of the peak prevalence dropped from 79% to 35%, compared to the case in which the vector population size was constant. Post-bottleneck peak prevalence was also lower; the median declined from 4.7% to 0.3% and the 75$^{th}$ percentile declined from 72% to 2.3%. Importantly, there were still some cases in which prevalence of infection in the vector population exceeded 85% even when the insect populations declined with the hosts. These were, however, much less frequent than in the case when the vector population did not decline with the hosts, highlighting the importance of the delay (Cohen & Gurtler 2001, citation in the main text), in the functional response of *T. infestans* population sizes to changes in host availability.

Simulations from the Ross MacDonald model with a 90 day bottleneck in guinea pig populations with (A) and without (B) a contemporaneous bottleneck in the vector populations. Even in the presence of bottlenecks in the vector populations *T. cruzi* can infect almost all insects; though the frequency of such occurrences is lower.

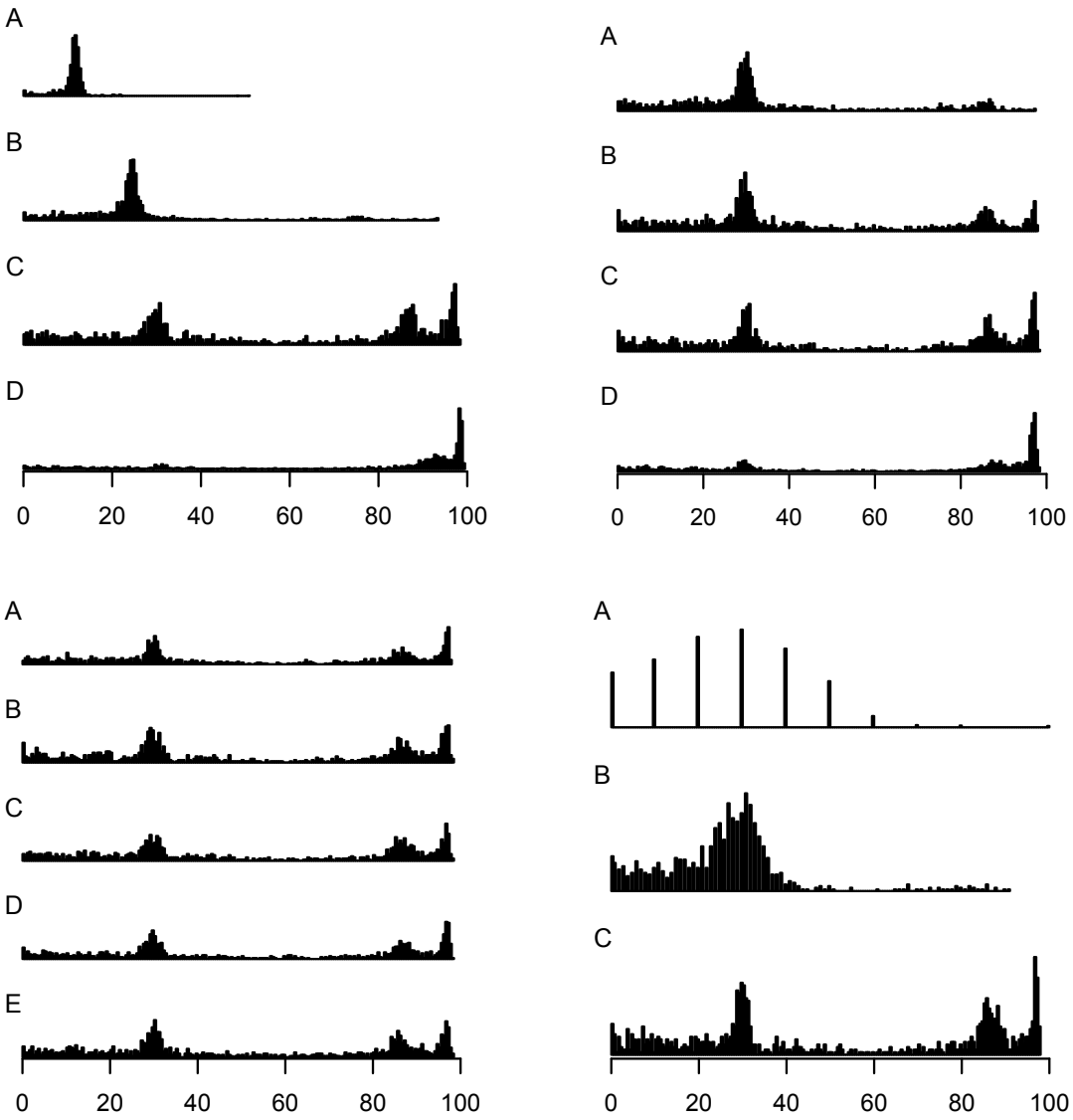



**Figure S6.** Sensitivity of the Stochastic Ross-MacDonald simulations to the effect of host bottlenecks on the prevalence of *Trypanosoma cruzi* in colonies of *Triatoma infestans*. A total of 1,000 replicate simulations were run as described in the main text. In each sensitivity analysis one parameter was varied; the other parameters were held at the values described in the main text. Host bottlenecks were set at 90 days. The x-axis of each histogram shows the *maximum* infection prevalence reached in vectors over the time course of the simulation. The y-axis shows the frequency (number of simulations). **Top left:** Average vector lifetime in days, **1/g**. A. 30; B. 90; C. 200*; D. 365. **Bottom left:** Length of incubation period of parasite in vector (in days) (**n)** A. 10; B. 30; C. 45*; D. 60; E. 90. **Top Right:** Infected vector to susceptible host infection probability, (**b)** A. 0.0025; B. 0.0005; C. 0.0068*; D. 0.001. **Bottom Right:** Vector population size **(m)** A. 10; B. 100; C. 1000

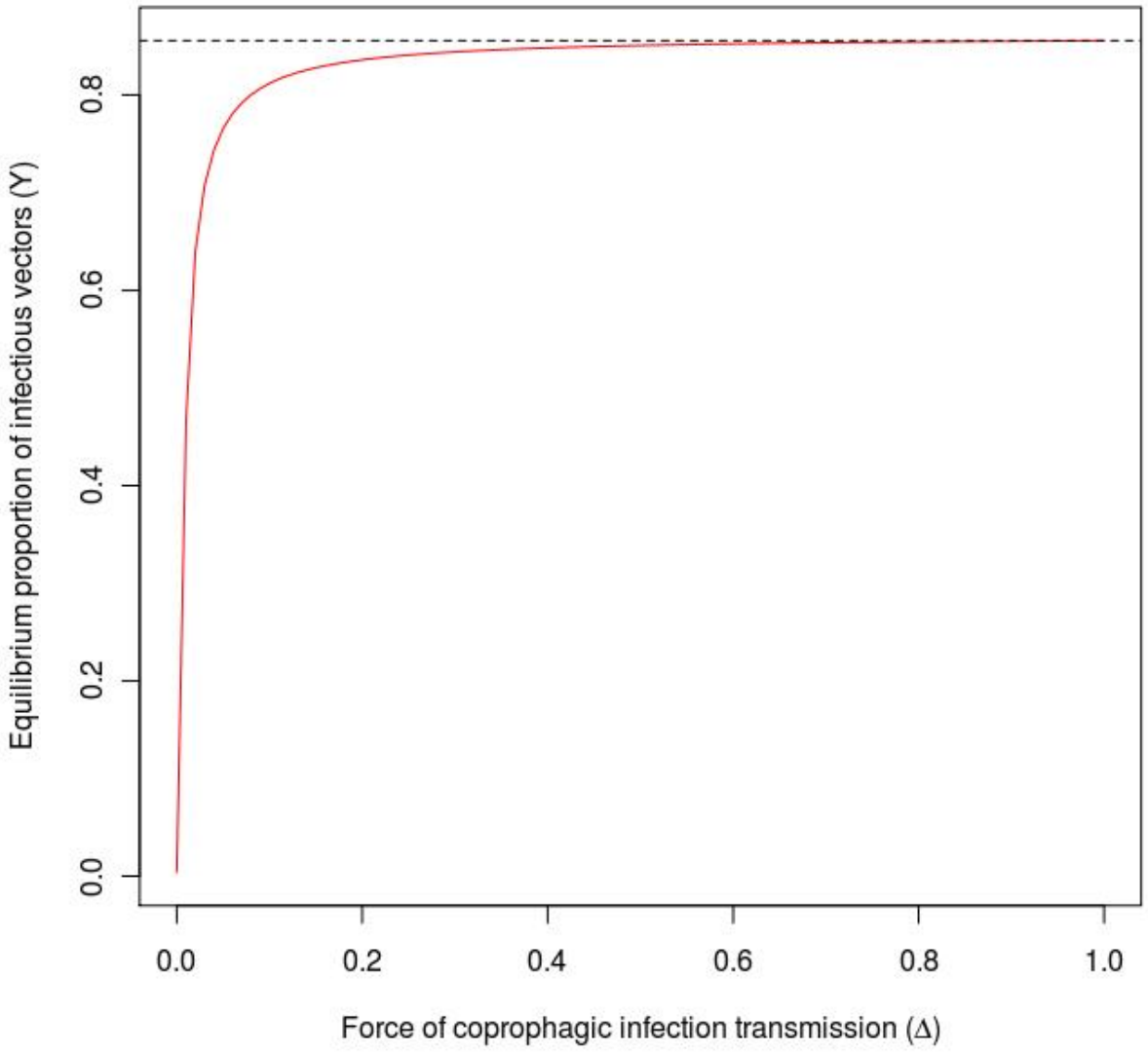



**Figure S7.** Results of Extended Ross-MacDonald Model including coprphagy (Eq. 6 in the main text). As the force of coprophagic transmission increases, the infection prevalence approaches an asymptote whose value is related to the probability that the vector will survive the parasite's incubation period.

**Table S2.** Identification of host DNA from the blood meals of *Triatoma infestans* collected from guinea pig pens harbouring large numbers of infected vectors. All insects were collected between August and November of 2008 from the district of La Joya, Arequipa, Peru

| **Site of collection** | | **Nutritional Status** | ***T. cruzi*** | | **Blood meal PCR products** | | | | |
|---|---|---|---|---|---|---|---|---|---|
| Guinea Pig pen code | Nymphal Stage | **4**= fully fed **3**= nearly full | **Microscopy** | **PCR** (Z1/Z2) | Human | Dog | Guinea Pig | Mouse | Chicken |
| 212B | 2 | 3 | + | + | . | . | + | . | . |
| | 2 | 3 | . | + | . | . | + | . | . |
| | 2 | 3 | . | + | . | . | + | . | . |
| | 2 | 3 | + | + | . | . | + | . | . |
| | 2 | 3 | + | . | . | . | + | . | . |
| | 2 | 3 | . | + | . | . | + | . | . |
| 244A | 2 | 4 | + | + | . | . | + | . | . |
| | 2 | 4 | . | . | . | . | . | . | . |
| | 2 | 4 | . | . | . | . | + | . | . |
| | 2 | 4 | + | + | . | . | + | . | . |
| | 2 | 4 | + | + | . | . | + | . | . |
| | 2 | 3 | . | + | . | . | + | . | . |
| 238B | 2 | 3 | + | + | . | . | + | . | . |
| | 2 | 3 | + | + | . | . | + | . | . |
| | 2 | 3 | + | + | . | . | + | . | . |
| | 2 | 3 | . | . | . | . | . | . | . |
| | 2 | 3 | + | + | . | . | + | . | . |
| | 2 | 3 | + | + | . | . | + | . | . |
| | 2 | 3 | + | + | . | . | + | . | . |
| | 2 | 3 | + | + | . | . | + | . | . |
| | 2 | 3 | + | + | . | . | + | . | . |
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| | 2 | 3 | . | + | . | . | + | . | . |
| | 2 | 3 | . | + | . | . | + | . | . |