MATHEMATICAL BIOSCIENCES
AND ENGINEERING
Volume **7**, Number **3**, July **2010**

doi:10.3934/mbe.2010.7.657

pp. **657–673**

ALTERNATIVE TRANSMISSION MODES FOR *TRYPANOSOMA CRUZI*

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(Communicated by Carlos Castillo-Chávez)

Abstract. The parasite *Trypanosoma cruzi*, which causes Chagas' disease, is typically transmitted through a cycle in which vectors become infected through bloodmeals on infected hosts and then infect other hosts through defecation at the sites of subsequent feedings. The vectors native to the southeastern United States, however, are inefficient at transmitting *T. cruzi* in this way, which suggests that alternative transmission modes may be responsible for maintaining the established sylvatic infection cycle. Vertical and oral transmission of sylvatic hosts, as well as differential behavior of infected vectors, have been observed anecdotally. This study develops a model which accounts for these alternative modes of transmission, and applies it to transmission between raccoons and the vector *Triatoma sanguisuga*. Analysis of the system of nonlinear differential equations focuses on endemic prevalence levels and on the infection's basic reproductive number, whose form may account for how a combination of traditionally secondary infection routes can maintain the transmission cycle when the usual primary route becomes ineffective.

1. **Introduction.** The protozoan parasite *Trypanosoma cruzi*, known principally for causing Chagas' disease throughout Latin America, is found in mammalian hosts and insect vectors from the United States south to Argentina and Chile. Chagas' disease affects millions of people throughout Latin America, and remains enzootic in the wild—and widely underdiagnosed among humans—despite recent successes in eradicating vectors in some areas. While transmission by blood transfusion has become a concern in urban areas, including in the United States, where many people have visited areas of Latin America at risk for Chagas' disease infection, transmission in rural areas remains linked to the sylvatic transmission cycle in which the parasite is maintained. In the southeastern United States (from Texas to the Atlantic seaboard), *T. cruzi* has been documented in numerous hosts, including raccoons, opossums, woodrats, skunks, dogs, armadillos, and even lemurs and macaques in a breeding facility on a coastal island in Georgia [23, 30, 31, 32, 33]. To date six autochthonous cases of transmission to humans have been documented in the U.S. [20]. The primary hosts in the southeastern U.S. appear to be raccoons

2000 *Mathematics Subject Classification.* Primary: 92D30, 92D40; Secondary: 92D25.
Key words and phrases. Trypanosoma cruzi, Chagas, oral infection, vertical infection, vector infection.

and opossums, while the primary vector species is *Triatoma sanguisuga* [30, 32] (others include *Triatoma gerstaeckeri* and *Triatoma lecticularia*).

Historically, the primary means of transmission has involved vectors feeding on [usually sleeping] hosts. *T. cruzi* lives in the gut of blood-sucking insect vectors such as *Triatoma* and *Rhodnius* sp. (often called cone-nose bugs or kissing bugs in English, and *chinches* or *vinchucas* in Spanish), and in the bloodstream of domestic and sylvatic hosts, including humans. (*T. cruzi* can also be found in hosts in an intracellular form, the amastigote.) The typical infection cycle transmits the parasite from host to vector during bloodmeals, and from vector to host through the vector defecating on the host following (or during) feeding, and the parasite being rubbed or scratched into the skin or mucous membranes of the host [16]—so-called stercorarian transmission. However, the vectors native to the southeastern U.S., in particular *T. sanguisuga* and *T. gerstaeckeri*, have long been observed to exhibit behavior which appears to make them poor vehicles for transmitting *T. cruzi*. For example, the delay between feeding and defecation, which can be no more than a few minutes in some South American species (e.g., [2, 13, 18, 32]) is more than half an hour in some species found in the southern U.S. [43, 44], by which time the vectors may already have left the host. The vectors, known to be cautious, also avoid climbing entirely onto the host while feeding, making stercorarian transmission to hosts difficult, and likely insufficient to maintain the high levels of prevalence observed in many host species. In addition, the strain of *T. cruzi* associated with raccoons in the U.S., identified in [23, 36] as type IIa, is sometimes described as being less virulent, suggesting a reduced infectivity compared to type I, the primary strain responsible for causing Chagas' disease in Central and South America. And yet the parasite has long been observed to have a fairly high prevalence among raccoons in the southeastern U.S. (reported between 37% and 61% in [45]). Consequently it appears likely that this cycle involves additional transmission modes.

Recent research suggests that sylvatic hosts such as raccoons and opossums may become infected through consumption of infected vectors [12, 33, 37, 47]; oral transmission has also been confirmed in humans in these studies, as well as experimentally (via intragastric injection) in mice, and potentially by macaques as well. A recent mathematical study [26] superimposed a predator-prey structure upon a host-vector SI infection cycle, and found standard disease dynamics and a reproductive number in which the effect of oral transmission was essentially additive in the term for host infection, as well as the possibility of vector consumption creating an Allee effect or two locally stable positive densities (each with a different R_0) in vector density.

This transmission route may be facilitated by the differential behavior of infected vectors. Parasites have been known to affect vector behavior, for instance Añez and East's 1984 study of *T. rangeli* in which they found that parasites blocked vectors' throats, impeding their ability to draw blood and consequently making them bite as much as 25 times as often as an uninfected vector [3]. Parasites may also affect vector mobility. Normally triatomine *T. cruzi* vectors remain hidden in dark areas and do not leave their hosts' den, but anecdotal reports of vectors found wandering in the open and later determined to be infected lend credence to this idea. This increased mobility may increase vectors' availability for consumption by hosts. These behaviors, while disadvantageous for the vector, tend to amplify the efficiency of the biting and oral transmission routes. Mathematically they may also couple vector density dynamics to disease dynamics.

In addition, vertical transmission has been documented in both sylvatic and human hosts. Studies of mothers and children in Mexico, Argentina and Brazil [4, 5, 6, 38] found a vertical transmission rate of 2% to 10%, and a study in Venezuela found a vertical transmission rate among Wistar rats (*R. norvegicus*) of 9.1% for one strain of *T. cruzi* isolated from dogs but none for a different strain isolated from humans [29], which suggests variable adaptation of some strains to vertical transmission. A higher prevalence of *T. cruzi* infection has also been observed in raccoons, which are placental, than in opossums, which are marsupial (and thus not susceptible to vertical transmission). Vertical transmission has been studied in dengue vectors (e.g., [21]), and variable adaptation to it has been studied in directly transmitted infections (e.g., [1, 17, 22]), but not among hosts in a vector-borne disease. Mathematical modeling of *T. cruzi* has heretofore largely been limited to its transmission in humans, including by transfusion, e.g., [34, 42].

In this paper we consider a model for the transmission of *T. cruzi* which incorporates all of these factors, in order to evaluate the relative contribution of each transmission route to sustaining the sylvatic infection cycle. Each of the following sections adds a feature, building up to a model which addresses the full picture.

2. **Saturation in contact processes.**

One of the key issues in modeling biological systems where population sizes may vary significantly is that of saturation in the contact processes which drive mathematical models' dynamics, by manifesting as nonlinearities. The mean number of contacts made by an average individual in one population per unit time with members of a second population depends, in general, on the sizes of the two populations. If the two groups are subsets of the same population, such as infected and uninfected members of the same species, then the average per capita contact rate is usually considered a function of that larger population size; if the groups making contact are not subject to a common bound or resource limitation, then the average per capita contact rate may be considered a function of the ratio of the two populations, as discussed further below. This contact rate, say $c(N)$, is often assumed to increase roughly linearly for small N but to reach a saturation point for large N, beyond which it becomes largely independent of N [7, 14, 24, 41]. This saturation occurs for logistical reasons due to the time required for each contact (in the case of *T. cruzi* transmission, some kind of predation) to be made. Kribs-Zaleta [25, 27] studied ways to incorporate saturation into general contact processes, comparing functional responses introduced by Holling to describe saturation in predation, both sharp (type I, e.g., $c(N) = c_0 \min(N/A, 1)$) and smooth (type II, e.g., $c(N) = c_0 N/(N + A)$), as well as a range of intermediate types. It was observed [27] that type I saturation gives rise to a wider variety of behaviors than type II, and so, in order to capture all possible end behaviors of the transmission cycle, this study will use type I saturation.

There are two relevant host-vector contact processes in this study of *T. cruzi* infection: vector bloodmeals and host predation on vectors, both of which affect disease transmission, and the latter of which affects vector density dynamics. Kribs-Zaleta [26] used a variable contact rate (considering both of the options suggested in [25]) to study vector consumption, and found that, when the saturation is sharp enough, two locally stable vector densities may exist, giving the system "memory". As the vector density decoupled from disease dynamics in the absence of differential behavior by infected vectors, and approached an equilibrium level monotonically, standard incidence was used to describe the biting contact process which drives

what is usually the primary infection pathway. Results found standard simple disease dynamics, driven entirely by the infection's reproductive number (in fact, as will be shown in this paper for a more complicated model, in this case the results are the same if one uses mass action incidence for the biting rate).

In general, these contact processes depend upon the ratio of vector to host population densities. If this ratio is high enough, then vectors are so plentiful that hosts can easily find them, while vectors are limited in their ability to contact hosts. In this case the contact rate is a linear function of host density, and can be said to be saturated in vector density. If, instead, the vector-host ratio is low, then hosts are plentiful, vectors can find them easily, and hosts have a harder time finding vectors, so that the contact rate is driven by (proportional to) vector density, and saturated in host density. (The threshold vector-host density ratio is in general different for each contact process.) We therefore denote the rate at which a single host makes potentially infectious stercorarian contacts by $c_h(N_v/N_h)$, so that the total rate of new stercorarian infections is given by multiplying c_h (with units of 1/time) by both the proportion I_v/N_v of infected vectors and the number of susceptible hosts. (For simplicity the term "potentially", which refers to the need to multiply by I/N, shall henceforth be omitted in discussing infection rates.) We also similarly denote the rate at which a single vector makes infectious bloodmeal contacts by $c_v(N_v/N_h)$ (units 1/time), and the per-host predation (effort) rate by $E_h(N_v/N_h)$ (units vectors/host/time). This is a modification of the model in [26], which used $c_h(N_v)$, $c_v(N_h)$, and $E_h(N_v)$ to consider saturation in each density only in the dynamics of the other species. Otherwise, we retain the general framework of [26]:

$$\begin{aligned} I_h'(t) &= \left(c_h\left(\frac{N_v(t)}{N_h(t)}\right)\frac{I_v(t)}{N_v(t)} + \rho E_h\left(\frac{N_v(t)}{N_h(t)}\right)\frac{I_v(t)}{N_v(t)}\right)(N_h - I_h(t)) - \mu_h I_h(t), \\ I_v'(t) &= c_v\left(\frac{N_v(t)}{N_h}\right)\frac{I_h(t)}{N_h}(N_v(t) - I_v(t)) - \mu_v I_v(t) - E_h\left(\frac{N_v(t)}{N_h(t)}\right)N_h\frac{I_v(t)}{N_v(t)}, \\ N_v'(t) &= b_v(N_v(t)) - \mu_v N_v(t) - E_h(N_v(t)/N_h(t))N_h. \end{aligned} \quad (1)$$

Here I_h and I_v are the respective numbers of infected hosts and vectors, N_h and N_v are the respective total densities of hosts and vectors, μ_h and μ_v the respective natural per capita mortality rates, and $b_v(N_v) = r_v N_v\left(1 - \frac{N_v}{K_v}\right)$ the total vector birth rate, given in terms of a maximum per capita reproductive rate r_v and carrying capacity K_v (assumed constant). Host density N_h is assumed constant since hosts such as raccoons and opossums are opportunistic feeders with many food sources, and as such their survival is not dependent upon finding vectors to eat. ρ is the proportion of cases in which host consumption of an infected vector infects the host (with units of hosts per vector).

In order to incorporate Holling type I saturation as a function of the vector-host density ratio $Q = N_v/N_h$, we must first define the threshold density ratios Q_h for host predation on vectors and Q_v for vector feeding on hosts, above which the given contact process is considered saturated in vectors (and thus dependent upon host density), and below which it is saturated in hosts (and thus dependent upon vector

density). Then we can write

$$\begin{aligned} c_h(Q) &= \beta_h \min(Q/Q_v, 1), \\ c_v(Q) &= \beta_v \min\left(\frac{1}{Q}/\frac{1}{Q_v}, 1\right) = \beta_v \min(Q_v/Q, 1), \\ E_h(Q) &= H \min(Q/Q_h, 1), \end{aligned} \tag{2}$$

where β_h is the (maximum) stercorarian infection rate for one host, β_v is the (maximum) bloodborne vector infection rate, and H is the maximum (preferred) per-host vector consumption rate. Thus the host-related rates c_h and E_h saturate for high vector-host ratios. The vector-related infection rate c_v is written initially as a function of $1/Q$ rather than Q since it saturates in precisely the reverse way as the per-host infection rate c_h: that is, for low vector-host ratios.

Applying ratio-dependent type I saturation (2), the system (1) becomes

$$\begin{aligned} I_h'(t) =& \beta_h \min\left(S_h(t)\frac{I_v(t)}{N_v(t)}, \frac{S_h(t)}{N_h}\frac{I_v(t)}{Q_v}\right) + \rho H \min\left(N_h, \frac{N_v(t)}{Q_h}\right)\frac{S_h(t)}{N_h}\frac{I_v(t)}{N_v(t)} \\ & - \mu_h I_h(t), \\ I_v'(t) =& \beta_v \min\left(Q_v\frac{S_v(t)}{N_v(t)}I_h(t), S_v(t)\frac{I_h(t)}{N_h}\right) - H \min\left(N_h, \frac{N_v(t)}{Q_h}\right)\frac{I_v(t)}{N_v(t)} \\ & - \mu_v I_v(t), \\ N_v'(t) =& r_v N_v(t)\left(1 - \frac{N_v(t)}{K_v}\right) - \mu_v N_v(t) - H \min\left(N_h, \frac{N_v(t)}{Q_h}\right), \end{aligned} \tag{3}$$

where to simplify notation we denote $N_h - I_h(t)$ by $S_h(t)$ and $N_v(t) - I_v(t)$ by $S_v(t)$. Depending on the size of the vector-host ratio $Q(t) = N_v(t)/N_h$, each infection rate takes either the form $kS(t)I(t)$ (species subscripts omitted) which corresponds to an unsaturated contact rate and so-called mass action incidence, or the form $kS(t)I(t)/N(t)$ which corresponds to a saturated contact rate (since the per capita contact rate is then largely independent of the population size) and so-called standard incidence.

By inspection the equation for vector population density $N_v(t)$ decouples from the infection dynamics, and can be studied separately. This is the same as equation (3) in [26] using $E_h = E_{sw}$ (Holling type I saturation) and $A = N_h Q_h$; analysis in that study found complex behavior described in terms of threshold quantities $x = H/(r_v - \mu_v)Q_h$ and $a = N_h Q_h/(1 - \mu_v/r_v)K_v$: The extinction equilibrium $N_v^* = 0$ is locally asymptotically stable (henceforth LAS) for $x > 1$; a LAS equilibrium $N_v^* = (1 - x)K_v(1 - \mu_v/r_v)$ exists for $1 - a < x < 1$; a second LAS equilibrium $\frac{1}{2}\left(1 + \sqrt{1 - 4ax}\right)K_v(1 - \mu_v/r_v)$ exists iff $4ax \leq 1$ and, if $a > 1/2$, $x \leq 1 - a$ also; and an unstable equilibrium exists between the two positive LAS equilibria for $a \leq 1/2$ and $1 - a < x < 1/4a$. The behavior can also be described in terms of the vector population's demographic reproductive number $R_d = r_v/(\mu_v + H/Q_h)$, as $x > 1 \Leftrightarrow R_d < 1$ (see [26] for a full explanation). In summary, depending upon parameter values, there are four possible end behaviors for this equation: globally stable extinction, globally stable survival, an Allee effect (survival only above a certain critical level), and two locally stable positive vector densities (this latter behavior is not seen under Holling type II saturation).

If $N_v \to 0$ and the vector population goes extinct, then of course the infection dies out and $I_h, I_v \to 0$ as well. Otherwise (and field observations suggest otherwise), we can substitute a given equilibrium density $N_v^* > 0$ for N_v and study the reduced

system, to which by a theorem of Thieme [39, 40] the full model's behavior is asymptotic. This yields the simple host-vector infection model

$$I_h'(t) = \tilde{\beta}_h S_h(t) I_v(t) - \mu_h I_h(t),$$
$$I_v'(t) = \tilde{\beta}_v S_v(t) I_h(t) - \tilde{\mu}_v I_v(t),$$

where

$$\tilde{\beta}_h = \frac{\beta_h}{\max(N_v^*, N_h Q_v)} + \frac{\rho H}{\max(N_v^*, N_h Q_h)}, \; \tilde{\beta}_v = \frac{\beta_v}{\max(N_v^*/Q_v, N_h)},$$

$$\text{and } \tilde{\mu}_v = \mu_v + \frac{H}{\max(N_v^*/N_h, Q_h)}.$$

This model has been well-studied and exhibits classical threshold behavior, with the disease-free equilibrium (0,0) globally asymptotically stable (henceforth GAS) for $R_0 < 1$ and the endemic equilibrium

$$\frac{I_h^*}{N_h} = \frac{\tilde{\beta}_v \tilde{\beta}_h - \tilde{\mu}_v \mu_h}{\tilde{\beta}_v(\tilde{\beta}_h + \mu_h)}, \quad \frac{I_v^*}{N_v^*} = \frac{\tilde{\beta}_h \tilde{\beta}_v - \mu_h \tilde{\mu}_v}{\tilde{\beta}_h(\tilde{\beta}_v + \mu_v)}$$

GAS for $R_0 > 1$, where

$$R_0 = \sqrt{\frac{\tilde{\beta}_h \tilde{\beta}_v}{\mu_h \tilde{\mu}_v}}.$$

Note (as pointed out in [26]) that since R_0 is a function of N_v^*, in the case mentioned above where there are two distinct LAS equilibria N_v^* each one will correspond to a different value of R_0, leaving open the possibility that a short-term perturbation in vector density might lead to a sustained difference in it, and consequently cause R_0 to cross the threshold value of 1, in one direction or the other.

As will be discussed further below, it is estimated in [28] that in the southeastern United States, the actual vector-host density ratio Q is on the order of 1000 for raccoons and opossums, far exceeding the likely ranges of Q_h and Q_v and thus clearly casting both contact processes as saturated in vectors and hence proportional to host densities.

3. **Differential behavior.** If we now consider the possibility of behavior changes for infected vectors, the proportion of any given type of host-vector contact involving infected vectors will no longer simply be prevalence I_v/N_v, but rather a weighted proportion $\frac{\gamma I_v}{S_v + \gamma I_v} = \frac{\gamma I_v}{N_v + (\gamma - 1) I_v}$, where infected vectors are γ times as likely as uninfected vectors to be involved (we shall henceforth assume that $\gamma > 1$). If we consider differential biting as well as differential presenting for consumption, we can either consider the same factor γ to apply to both or we can assign γ_1 and γ_2 to the respective phenomena (this includes the possibility that $\gamma_1 > 1$ while $\gamma_2 = 1$ or vice versa, i.e., only one type of differential behavior). If $\gamma_2 \neq 1$, then we may consider the effective (apparent) vector population density to be $N_v^+ = N_v + (\gamma_2 - 1) I_v$ rather than N_v for vector consumption purposes, so that the per-host consumption rate is $E_h(N_v + (\gamma_2 - 1) I_v)$. γ_1 similarly affects c_h and c_v.

These assumptions change our general one-host model (1) to

$$I_h'(t) = \left(c_h\left(\frac{N_v+(\gamma_1-1)I_v}{N_h}\right)\frac{\gamma_1 I_v}{N_v+(\gamma_1-1)I_v} + \rho E_h\left(\frac{N_v^+}{N_h}\right)\frac{\gamma_2 I_v}{N_v^+}\right)(N_h - I_h) - \mu_h I_h, \tag{4}$$

$$I_v'(t) = c_v\left(\frac{N_v+(\gamma_1-1)I_v}{N_h}\right)\frac{I_h}{N_h}(N_v - I_v) - \mu_v I_v - E_h(N_v^+/N_h)N_h\frac{\gamma_2 I_v}{N_v^+}, \tag{5}$$

$$N_v'(t) = r_v N_v\left(1-\frac{N_v}{K_v}\right) - \mu_v N_v - E_h(N_v^+/N_h)N_h. \tag{6}$$

The dependence of E_h on I_v due to differential presenting for consumption couples vector density dynamics to disease dynamics; however, any disease-free equilibrium (DFE) has the same condition on N_v^* as the equation for $N_v'(t)$ in model (3), so that the potential for complex vector density behavior is preserved.

Applying the next-generation operator method [15, 19], we find a basic reproductive number for the infection,

$$R_0 = \sqrt{\frac{c_v\left(\frac{N_v^*}{N_h}\right)}{\mu_h}\frac{\gamma_1 c_h\left(\frac{N_v^*}{N_h}\right)+\gamma_2\rho E_h\left(\frac{N_v^*}{N_h}\right)}{\mu_v+\gamma_2 E_h\left(\frac{N_v^*}{N_h}\right)\frac{N_h}{N_v^*}}}, \tag{7}$$

where N_v^* is the limiting value for the vector density at the DFE. Note that the effect of the differential behavior γ on R_0 is to reduce μ_v by a factor of γ (in the case $\gamma = \gamma_1 = \gamma_2$).

Substituting the type I forms (2) for the contact rates gives

$$\begin{aligned} I_h'(t) =& \beta_h \min\left(\frac{S_h\,\gamma_1 I_v}{N_v+(\gamma_1-1)I_v}, \frac{S_h}{N_h}\frac{\gamma_1 I_v}{Q_v}\right) + \rho H \min\left(N_h, \frac{N_v^+}{Q_h}\right)\frac{S_h}{N_h}\frac{\gamma_2 I_v}{N_v^+} \\ & - \mu_h I_h, \\ I_v'(t) =& \beta_v \min\left(\frac{S_v\,Q_v I_h}{N_v+(\gamma_1-1)I_v}, S_v\frac{I_h}{N_h}\right) - \mu_v I_v - H\min\left(N_h, \frac{N_v^+}{Q_h}\right)\frac{\gamma_2 I_v}{N_v^+}, \\ N_v'(t) =& r_v N_v\left(1-\frac{N_v}{K_v}\right) - \mu_v N_v - H\min\left(N_h, \frac{N_v^+}{Q_h}\right). \end{aligned} \tag{8}$$

As previously noted, estimates in [28] suggest that for raccoons and opossums in the southeastern U.S., $Q > Q_h, Q_v$, in which case each of the minima in (8) reduces to the first argument of each pair. However, we may first wish to see whether it is possible for the increased vector mobility caused by infection, which makes infected vectors γ_2 times as easy for hosts to find during predation (as uninfected vectors), to cause the vector population to go extinct because of an increase in the predation rate. In this case we can instead take the second argument of each minimum given in (8), and write the corresponding equilibrium conditions as follows, where $x_h = I_h/N_h$ and $x_v = I_v/N_v$ are rescaled variables which allow us to distinguish

between extinction of the vector population and "extinction" of the infection:

$$\left(\gamma_1\frac{\beta_h}{Q_v}+\gamma_2\frac{\rho H}{Q_h}\right)(1-x_h)x_vQ-\mu_hx_h=0, \tag{9}$$

$$\beta_v(1-x_v)x_h-\left(\mu_v+\gamma_2\frac{H}{Q_h}\right)x_v=0, \tag{10}$$

$$N_v\left[r_v\left(1-\frac{N_v}{K_v}\right)-\mu_v-\frac{H}{Q_h}(1+(\gamma_2-1)x_v)\right]=0. \tag{11}$$

From (11) $N_v^*=0$ is clearly a possibility, but in this case $Q=0$, which implies in (9) that $x_h^*=0$, which in turn implies in (10) that $x_v^*=0$. That is, the only extinction equilibrium is disease-free, so increased mobility of infected vectors will not cause extinction. We may therefore use the vector-saturated form of (8):

$$\begin{aligned}
I_h' &= \beta_hS_h\frac{\gamma_1I_v}{N_v+(\gamma_1-1)I_v}+\rho HS_h\frac{\gamma_2I_v}{N_v+(\gamma_2-1)I_v}-\mu_hI_h,\\
I_v' &= \beta_vQ_vI_h\frac{S_v}{N_v+(\gamma_1-1)I_v}-\mu_vI_v-HN_h\frac{\gamma_2I_v}{N_v+(\gamma_2-1)I_v},\\
N_v' &= r_vN_v\left(1-\frac{N_v}{K_v}\right)-\mu_vN_v-HN_h.
\end{aligned} \tag{12}$$

As with models (1) and (3), the vector density dynamics of (12) decouple from infection dynamics. The equation for N_v has two equilibria:

$$N_\pm^*=\frac{1}{2}\left(1\pm\sqrt{1-4\frac{HN_hr_v}{K_v(r_v-\mu_v)^2}}\right)K_v\left(1-\frac{\mu_v}{r_v}\right).$$

The greater of these, N_+^*, is quickly seen to be locally asymptotically stable, while the lesser, N_-^*, is unstable. As with other simple constant-yield harvesting models (cf. [8, 9]), the lower equilibrium becomes a threshold value, below which the population goes extinct in finite time and above which the population approaches a constant value (N_+^*). Since this model is not appropriate for studying extinction equilibria, we shall take from it simply the value of the limiting vector density, N_+^*.

We can now again substitute this equilibrium density for N_v and study the reduced system by applying the result of Thieme. The rescaled model for the limiting system of (12) becomes

$$x_h' = \left(\beta_h\frac{\gamma_1x_v}{1+(\gamma_1-1)x_v}+\rho H\frac{\gamma_2x_v}{1+(\gamma_2-1)x_v}\right)(1-x_h)-\mu_hx_h, \tag{13}$$

$$x_v' = \beta_vQ_v\frac{N_h}{N_v^*}x_h\frac{1-x_v}{1+(\gamma_1-1)x_v}-\mu_vx_v-H\frac{N_h}{N_v^*}\frac{\gamma_2x_v}{1+(\gamma_2-1)x_v}, \tag{14}$$

and a straightforward computation shows that the reproductive number (7) takes the particular form

$$R_0=\sqrt{\frac{\beta_vQ_v}{\mu_h}\frac{(\beta_h\gamma_1+\rho H\gamma_2)\frac{N_h}{N_v^*}}{\mu_v+H\frac{N_h}{N_v^*}\gamma_2}}. \tag{15}$$

To find endemic equilibria, we solve (13) with $x_h'=0$ for x_h^*:

$$x_h^*=\frac{(\beta_hy_1^*+\rho Hy_2^*)x_v^*}{\mu_h+(\beta_hy_1^*+\rho Hy_2^*)x_v^*},\quad\text{where}\quad y_i=\frac{\gamma_i}{1+(\gamma_i-1)x_v}.$$

By inspection $0 \le x_h^* < 1$ when $x_v^* \ge 0$. Substituting into (14) with $x_v' = 0$ yields

$$0 = \beta_v Q_v \frac{(\beta_h y_1^* + \rho H y_2^*) x_v^*}{\mu_h + (\beta_h y_1^* + \rho H y_2^*) x_v^*} \frac{1 - x_v^*}{1 + (\gamma_1 - 1) x_v} - \mu_v x_v^* \frac{N_v^*}{N_h} - H y_2^* x_v^*.$$

Then either $x_v^* = 0$ or (multiplying by $(\mu_h + \beta_h y_1^* x_v^* + \rho H y_2^* x_v^*)/x_v^*$)

$$0 = \beta_v Q_v (\beta_h y_1^* + \rho H y_2^*) \frac{1 - x_v^*}{1 + (\gamma_1 - 1) x_v} - \left(\mu_v \frac{N_v^*}{N_h} + H y_2^* \right) (\mu_h + \beta_h y_1^* x_v^* + \rho H y_2^* x_v^*). \tag{16}$$

Making this equation polynomial requires multiplying by $(1+(\gamma_1-1)x_v^*)^2(1+(\gamma_2-1)x_v^*)^2$ and results in a quartic equation with a constant coefficient which is a positive multiple of $(R_0^2 - 1)$; one can therefore show that the number of endemic equilibria changes at $R_0 = 1$. However, for the two special cases $\gamma_2 = 1$ and $\gamma_2 = \gamma_1$, (16) is equivalent to a quadratic which can more easily be shown to have one solution in (0,1) when $R_0 > 1$ and no such solutions otherwise. For instance, if $\gamma = \gamma_1 = \gamma_2$, then (16) becomes $f(x_v^*) = a x_v^{*2} + b x_v^* + c = 0$, where

$$\begin{aligned} a &= -(\gamma - 1)\mu_v \frac{N_v^*}{N_h} \left[\gamma(\beta_h + \rho H) + \mu_h(\gamma - 1) \right], \\ b &= -\beta_v Q_v (\beta_h + \rho H)\gamma - \mu_h \mu_v \frac{N_v^*}{N_h}(\gamma - 1) \\ &\quad - \left(\mu_v \frac{N_v^*}{N_h} + H\gamma \right) [(\beta_h + \rho H)\gamma + \mu_h(\gamma - 1)], \\ \text{and } \ c &= \mu_h \left(\mu_v \frac{N_v^*}{N_h} + H\gamma \right) (R_0^2 - 1). \end{aligned}$$

Since $a, b < 0$, then $-b/2a < 0$. If $R_0 < 1$ then $c < 0$, which implies that $b^2 - 4ac < b^2$, so that any real solutions have the same sign as $-b/2a$, i.e., negative. If instead $R_0 > 1$ then $c > 0$, $b^2 - 4ac > b^2$, and there are two real solutions with different signs. In this case, $f(0) = c > 0$ and $f(1) < 0$, guaranteeing that the positive solution is in (0,1).

We can, finally, show that all solutions to (13)–(14) (and hence (12)) approach an equilibrium (which we can show is unique and GAS for the special cases $\gamma_1 = \gamma_2$ and $\gamma_2 = 1$: disease-free if $R_0 < 1$ and endemic if $R_0 > 1$) by applying the Poincaré-Bendixson Theorem after observing that solutions never leave the unit square and, by Bendixson's Criterion, approach no periodic orbits, since

$$\begin{aligned} \frac{\partial}{\partial x_h}\left(\frac{dx_h}{dt}\right) &= -(\beta_h y_1 + \rho H y_2) x_v - \mu_h < 0, \\ \frac{\partial}{\partial x_v}\left(\frac{dx_v}{dt}\right) &= -\beta_v Q_v \frac{N_h}{N_v^*} x_h \frac{\gamma_1}{[1 + (\gamma_1 - 1)x_v]^2} - \mu_v - H \frac{N_h}{N_v^*} \frac{\gamma_2}{[1 + (\gamma_2 - 1)x_v]^2} < 0. \end{aligned}$$

To evaluate the overall effect of the differential behavior γ on the transmission of *T. cruzi*, we recall that R_0 increases with γ; as γ rises, the proportion of both types of host-vector contact made with infected vectors approaches 1 because infected vectors are so much more active than uninfected ones. In the case $\gamma_1 = \gamma_2$, it increases only up to a fixed maximum (obtained from (7) or (15) by setting $\mu_v = 0$) since the increase in infected vectors' death rate due to predation keeps pace with the increases in both types of infectious contacts. However, if γ_1 (amplified vector feeding) increases more than γ_2 (amplified predation), then R_0 can increase without bound, as the rise in host infections is not matched by a rise in the vector death rate. Note also that although increasing γ_i increases the proportion of contacts

made with infected vectors, it does not necessarily increase endemic prevalence in the case where $R_0 > 1$. For instance, in the case $\gamma_1 = \gamma_2 = \gamma$, since the coefficients a and b in $f(x)$ are quadratic in γ but c is asymptotically linear in γ (since R_0 is asymptotically constant as $\gamma \to \infty$), one can show that as $\gamma \to \infty$, $x_v^* \to 0$. This occurs because increasing γ_2 increases the death rate of infected vectors, and, as will be seen in Section 5 below, increasing γ_1 actually disadvantages new vector infections.

4. **Vertical transmission.** We now incorporate into the model vertical transmission among hosts, occurring with probability p (properly speaking, a proportion), in addition to the horizontal transmission modes addressed in previous sections. With the additional term, (4) becomes

$$I_h'(t) = \left(c_h \left(\frac{N_v + (\gamma_1 - 1)I_v}{N_h} \right) \frac{\gamma_1 I_v}{N_v + (\gamma_1 - 1)I_v} + \rho E_h \left(\frac{N_v^+}{N_h} \right) \frac{\gamma_2 I_v}{N_v^+} \right) S_h(t) + p\frac{I_h}{N_h} b_h(N_h) - \mu_h I_h(t), \tag{17}$$

where $b_h(N_h)$ is the total host birth rate. (Since we do not distinguish host gender here, we may properly limit ourselves to studying female hosts.) Analysis of the system (17), (5), (6) follows closely that of the previous section. The disease-free dynamics and DFE are identical. Taking the host population to be a constant N_h, the vertical transmission term in (17) simplifies to $p\mu_h I_h$, and the rescaled equation (13) changes only in replacing $-\mu_h x_h$ with $-(1-p)\mu_h x_h$. All the analysis of the rescaled systems in the previous section therefore holds here as well [1].

There is, however, a change in the structure of the basic reproductive number, which the next-generation operator approach calculates as

$$R_v = \frac{1}{2}\left(p + \sqrt{p^2 + 4R_h^2} \right),$$

where R_h is the reproductive number for horizontal transmission alone (given as R_0 in (7)). This reflects the combined effects of two fundamentally different types of transmission: vertical, with efficiency $p \leq 1$, and horizontal via vectors, with efficiency R_h. The radical in R_v reflects the two-stage vector transmission cycle, with vertical transmission inextricably linked. We note that $\max(p, R_h) < R_v < p + R_h$, so that even limited vertical transmission together with an inefficient horizontal cycle may sustain the infection.

5. **Numerical analysis.** To apply the model structures developed above to the particular *T. cruzi* transmission cycle between raccoons *Procyon lotor* and the vector *Triatoma sanguisuga* in the southeastern quarter of the United States, we must first estimate the values of the parameters in these models. Numerous field and laboratory studies have been published on different aspects of the sylvatic transmission cycle for *Trypanosoma cruzi* in the United States, but much also remains unknown, especially as regards the rates at which oral and vertical transmission occur in hosts, as stercorarian and bloodborne transmission driven by vector feeding has historically been the focus of study. In addition, particulars vary from place to place, even when host and vector species are the same. [28] used an extensive literature review

[1] The only difference worth noting is that terms which in the previous section included $R_0^2 - 1$ now appear as $R_h^2 - 1$ but with μ_h replaced by $(1-p)\mu_h$, rather than as $R_v^2 - 1$. Such terms are positive iff $R_v^2 > 1$, so the essential dynamics have not changed

Param.	Meaning	Value
μ_h	Natural raccoon mortality rate	0.40/yr
N_h	Raccoon population density	20. racc/km^2
μ_v	Natural *T. sanguisuga* mortality rate	0.271/yr
r_v	*T. sanguisuga* birth rate	33/yr
N_v^*	*T. sanguisuga* population density	31600 vec/km^2
Q_h	Threshold vector-host density ratio for predation	10 vec/racc
Q_v	Threshold vector-host density ratio for bloodmeals	100 vec/racc
p	Probability (proportion) of vertical transmission	0.1
ρ	Probability (proportion) of oral transmission	0.28 racc/vec
γ	Amplification factor for differential behavior	6.5

TABLE 1. Parameter estimates, from [28]

to derive estimates for parameters relevant to modeling *T. cruzi* transmission in the southeastern U.S. between (among others) raccoons and *T. sanguisuga*, and we shall here make use of those estimates. Table 1 summarizes these values.

Some caveats are in order regarding these estimates (for a full discussion, see [28] and references therein): Vector population density is based on a single study in Texas [11] where the landscape was scrub-dominated rather than heavily wooded as in much of the southeastern U.S. Both threshold density ratios Q_h and Q_v are based on very rough estimates, including an estimated 10 bites/night maximum that a raccoon could (or would) sustain ([28] estimated $14 < Q_v < 800$; our value of 100, based on the aforementioned maximum, falls roughly on the geometric mean of this range). The probability p of vertical transmission in raccoons is taken from estimates of 9–10% vertical transmission of certain strains of *T. cruzi* in Wistar rats and humans, as described in [28], since there are no published studies on vertical transmission rates in raccoons. The probability ρ of oral transmission to a host given consumption of one infected vector is based on two very small studies of North American *T. cruzi* hosts, one of which involved only 2 trials of a raccoon being fed 3 vectors infected with the Type IIa *T. cruzi* strain associated with raccoons in the U.S. [37] (both trials resulted in infection), and the other of which involved 11 trials of a Virginia opossum (*Didelphis virginiana*) being fed 2 vectors infected with a Type IIe strain from Chile (3 trials resulted in infection) [47]. (Another study of a few South American opossums, *Didelphis albiventris*, which consumed variable numbers of South American vectors *Triatoma infestans* infected with an unspecified (but not Type IIa) *T. cruzi* strain resulted in an even lower probability estimate of 0.075 [35].) These sample sizes are too small to infer with confidence, but a value of 0.2 (infected raccoons per consumed vector) or lower for ρ makes the 2 successful trials described in [37] more unlikely than likely (that is, the probability of 2 consecutive successful trials is less than 1/2), so the estimate of 0.28 taken from [28] is not unreasonable. Finally, the amplification factor γ for differential behavior of infected vectors is based on the single study involving the triatomine Chagas vector *Rhodnius prolixus* native to South America and the parasite *Trypanosoma rangeli* [3], and will be used only for illustrative purposes in considering the various special cases discussed in Section 3.

The remaining model parameters to be estimated are the basic contact rates β_h, β_v and H. In the absence of data on biting rates and infection probabilities associated with stercorarian and bloodborne transmission, or on predation rates of

γ_1	γ_2	H	β_h	β_v	R_v	% sterc.	% oral	% vert.
1	1	1 v/r/yr	0.122/yr	14.4/yr	1.89	27.4	62.6	10
6.5	1	1 v/r/yr	0.0773/yr	59.1/yr	5.24	27.4	62.6	10
1	1	0.5 v/r/yr	0.262/yr	14.4/yr	1.89	58.7	31.3	10
6.5	1	0.5 v/r/yr	0.166/yr	59.1/yr	6.53	58.7	31.3	10
1	6.5	0.5 v/r/yr	0.181/yr	14.4/yr	3.07	40.4	49.6	10
6.5	6.5	0.5 v/r/yr	0.114/yr	59.1/yr	7.57	40.4	49.6	10
1	1	0.4 v/r/yr	0.290/yr	14.4/yr	1.89	64.9	25.1	10
6.5	1	0.4 v/r/yr	0.183/yr	59.1/yr	6.75	64.9	25.1	10
1	6.5	0.4 v/r/yr	0.225/yr	14.4/yr	2.87	50.3	39.7	10
6.5	6.5	0.4 v/r/yr	0.142/yr	59.1/yr	7.58	50.3	39.7	10

TABLE 2. Computational results for model (17), (5), (6) for various values of γ_1, γ_2 and H. All other parameters are as given in Table 1. The last three columns give percentages of host infections at the endemic equilibrium arising from stercorarian, oral, and vertical transmission.

raccoons upon triatomines, these values must be estimated indirectly, using prevalence data and the equilibrium conditions obtained by setting x_h' and x_v' to 0 in (13) (with μ_h replaced by $(1-p)\mu_h$ as described in Section 4) and (14). It is estimated in [28] that the average prevalence of *T. cruzi* infection in raccoons in the southeastern U.S. is 0.387 (that is, 38.7%, although a recent large study [10] generated values ranging from 33% to 68% across the southeastern quarter of the country), while for *T. sanguisuga* it is 0.565.

Applying the parameter estimates in Table 1 to the equilibrium conditions as described above, we find that, in the case without differential behavior ($\gamma_1 = \gamma_2 = 1$) the infection rate term $\beta_h + \rho H$ must be about 0.4/yr in order to account for the observed prevalence levels. Thus both the host infection rate and the host predation rate must be quite low ($\beta_h \leq 0.402/yr$, $H \leq 1.44/yr$). Without further empirical data on the two rates, it is not possible to determine their individual values, but we can compare results using three benchmark values for H: 1, 0.5, and 0.4 vectors/raccoon/yr. These correspond to an average raccoon eating a triatomine vector once per year, once per 2 years, and once per 2.5 years, respectively; the last value is significant because the average raccoon lifetime is estimated at 2.5 years, so H values above this indicate that raccoons do on average eat a vector during their lifetimes, while H values below this indicate that raccoons do not on average eat a vector. For comparison purposes we also use values of 1 (no differential behavior) and 6.5 (the estimate derived from Añez and East [3]) for γ_1 and γ_2. Results are given in Table 2. Since for $\gamma_2 = 6.5$ it is not possible to obtain the observed prevalence levels for $H = 1$ vec/racc/yr, that combination is not given in the table.

Given the gross uncertainty in the estimate for the probability ρ of oral infection when a raccoon consumes an infected vector, it is worth observing that the H values in Table 2 may need adjusting, but it seems more likely that ρ is an underestimate than an overestimate (the stark difference in experimental oral infection results between raccoons and opossums suggesting rather that ρ is significantly smaller for opossums than for raccoons). Thus any corrections to H would be downward revisions, which make it even more likely that oral transmission predominates among raccoons: for instance, doubling ρ would lead to halving H in order to produce

the results given in the table, an implication of which would be that nearly any predation rate which has most raccoons eating at least one vector in their lifetimes would cause more infections than stercorarian means.

Although β_h and R_v can be seen to be influenced by both γ_1 and γ_2 (and, to a lesser extent, by H), there are more particular patterns to be seen. The primary influence of the biting rate increase γ_1 is on β_v, but perhaps not in the direction expected. An increased biting rate among infected vectors does increase stercorarian transmission to hosts, but actually decreases bloodborne transmission to vectors (the only route to infection for them) by disadvantaging uninfected vectors—an increase in γ_1 increases the proportion of bites made by infected vectors, and since by assumption the biting rate is already vector-saturated (driven by host density), the total number of bites made by all vectors per unit time cannot increase, only the proportion of those bites made by infected vectors. Thus uninfected vectors actually have fewer contacts with hosts (including infected hosts), obliging (as seen in Table 2) β_v to be higher in order to account for the same observed prevalence. It is estimated in [28] that *T. sanguisuga* prefers to feed at a rate of about 0.102 bites/vector/day; converting to years and multiplying by the observed proportion of infected hosts $x_h^* = 0.387$ yields a figure of 14.4/yr, in perfect agreement with the β_v estimates when $\gamma_1 = 1$. This suggests that perhaps *T. sanguisuga* does not exhibit an increased biting rate when infected; if true, this would be in keeping with the many other ways in which this species has been observed to be a poor vector.

One can also observe that, regardless of the values of γ_1, γ_2 and H, the raw host infection rate β_h is as much as two orders of magnitude below the raw vector infection rate β_v, despite the fact that both rates arise from the same type of host-vector contact. In all the example estimates given in Table 2, $\beta_h < 0.3/yr$, which appears to verify that *T. sanguisuga* is indeed a poor vector, especially compared to the vector species found in the tropics.

Finally, the predation-related parameters γ_2 and H both affect the percentages of host infections that arise from each of the three hypothesized transmission routes (but do not appear to affect β_v). More specifically, unless γ_2 and H are both particularly low (no increased vector mobility due to infection, and an average predation rate of well below one vector per year per raccoon), oral transmission does appear to account for more host infection than the traditional stercorarian route, in accordance with the conjectures of several researchers.

6. **Discussion.** This study developed models to evaluate the relative importance of nontraditional (i.e., non-stercorarian) transmission avenues to hosts in sustaining the sylvatic transmission of *Trypanosoma cruzi* in the southeastern United States, in particular between raccoons and the vector *T. sanguisuga*. Both vector feeding and host predation on vectors were modeled as ratio-dependent contact processes with Holling type I saturation. The resulting system allows for complex vector population dynamics, but no infection-driven vector extinction. More specifically, the "sharpness" of the contact process saturation may create two locally stable vector densities, each corresponding to a different value of R_0 (possibly on different sides of 1). As a result, differential behavior of infected vectors may amplify vector consumption to the point that it drives the vector density to a lower level where $R_0 < 1$ and the disease may die out. More generally, however, differential behavior increases R_0 despite the obvious inefficiency in sacrificing vectors to infect hosts

orally (which may reduce any endemic prevalence). An increased biting rate in infected vectors (represented in our model by γ_1) tends to increase parasite prevalence in hosts, but may not increase prevalence in vectors since it tends to reduce the proportion of contacts made by uninfected vectors, in a setting where contact rates are limited by the density of available hosts. On the other hand, if infection does not also increase vector mobility to the point that they are at a higher predation risk (γ_2) than uninfected vectors (which tend to hide in dark places waiting for sleeping hosts), then increased biting can raise R_0 without bound, while increased predation of infected vectors can have only a limited effect on R_0 since it also removes the infected vectors more quickly.

The alternative means of transmitting *T. cruzi* to hosts proposed by biologists and studied here, oral (predation-based) and vertical (congenital) transmission, each have distinct epidemiological characteristics. Vertical transmission, which has been observed at low levels in human hosts and for which some evidence exists in sylvatic *T. cruzi* hosts, cannot sustain an infection cycle by itself, but even at mediocre efficiencies can sustain the infection cycle in conjunction with even very inefficient vector-host transmission modes. At an endemic equilibrium, the vertical transmission "probability" p (here estimated at 10%) becomes the proportion of all host infections which result from vertical transmission, and vertical transmission is seen to have an "almost additive" effect on the infection's basic reproductive number, since $\max(p, R_h) < R_v < p + R_h$. Adaptation to oral transmission, although risky as an evolutionary strategy for the parasite (since infected vectors can infect at most one host this way), appears to be fully as significant as conjectured by some researchers, as numerical analysis suggests that even when each host consumes on average less than one *T. cruzi* vector per year ($H < 1$) oral transmission can be the dominant transmission avenue to hosts. In general, however, numerical analysis also indicates that *T. sanguisuga* is indeed as poor a vector as predicted from its behavior, and increased biting also appears unlikely in infected vectors.

It should be noted that, while most of these conclusions hold even in the absence of good data on oral and vertical transmission, there is a clear need for laboratory and field studies to document the true likelihoods of oral and vertical *T. cruzi* transmissions in raccoons and other sylvatic hosts, as well as the feeding (predation) behavior of hosts with regard to *T. cruzi* vectors. These transmission rates are likely to be heavily strain-dependent, and future modeling work already in progress will examine the role played by the adaptations of one strain (to different transmission avenues) relative to another—in particular, the Type I and IIa strains presently circulating in the southern U.S.—including the cross-immunity which infection with one strain appears to confer against the other, which may highlight the significance of adaptation to vertical transmission as an evolutionary strategy for the parasite. Other work in progress will consider the overlaps between transmission cycles involving distinct hosts on a larger geographical scale, from Mexico north to Texas and east to the Atlantic coast.

Acknowledgments. This research began during a Fulbright-García Robles Fellowship at the Universidad de Colima in Colima, Mexico, and was also partially supported during that time by a grant from the Fondo Ramón Buylla-Álvarez. The author wishes to thank Carlos Moisés Hernández-Suárez and Francisco Espinoza-Gómez, both of the Universidad de Colima, and Christopher Hall of Berry College in Georgia, for many helpful conversations during this time. More recently, this

work was supported by a grant from the Norman Hackerman Advanced Research Program. The author also wishes to thank Michael Yabsley of the University of Georgia for preprints of several articles including [10] and [37].

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Received July 31, 2009; Accepted April 20, 2010.

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