Assessment of The Invasion Speed of Triatomine Populations, Vectors of Chagas Disease

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Assessment of the invasion speed of triatomine populations, vectors of Chagas disease*

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Abstract.
Spraying insecticides to control triatomine populations, the vectors of Chagas disease, does not prevent the reemergence of the disease in infested areas. Mathematical models attempt to explain this reemergence by identifying the factors involved in sylvatic transmission of the parasite *T. cruzi*. The presence of reservoir hosts such as woodrats is essential to an understanding of the infection’s geographical spread. The biological vector-host system is modeled by applying integrodifference equations to incorporate dispersal as well as host-vector interactions. These equations capture, simultaneously, the three processes taking place between successive generations: demography, infection and spatial dispersal. The travelling waves, solutions of the integrodifference equations thus derived, allow one to calculate numerically the invasion speed of the disease. The application of Neubert-Caswell’s theorem to calculate the analytical invasion speed seems feasible.

Keywords: Chagas disease, vector, host, integrodifference equations, travelling waves, invasion speed

1. Introduction.
Chagas disease, or American trypanosomiasis, is a disease that currently affects an estimated human population of 10 million people [1]. In 2008, approximately 14,000 deaths were attributed to this disease which does not remain confined to Latin America. Its spread, mainly due to the high frequency of human mobility, organ donation, blood transfusions and vertical transmission (from infected mother to child), is such that the United States of America, Canada, parts of Europe (particularly Spain) and many Asian countries including Japan, are among the affected areas [2].

Although the causative agent of the disease, *Trypanosoma cruzi*, was discovered one hundred years ago, there is no vaccine to stop the spread of the disease, and the only struggle advocated by the WHO is the fight against its vectors, which are blood-sucking bugs of the genus *Triatoma*. If these bugs live in the nests of mammals or birds, then they are said to be sylvatic. If they live in shelters neighboring human habitations, then they are domestic. In endemic areas, international control programs are based on the mass spraying of insecticides in domestic and peridomestic areas, whereas prevention programs are based on blood transfusion security by systematic epidemiological testing [3].

These programs objectives have been only partially achieved because the disease has re-emerged, supported by sylvatic transmission of the parasite. Not captured by means of laboratory studies, these transmission processes are an important focus for mathematical modeling studies.
To the life cycle of *T. cruzi* are associated intermediate hosts, or vectors, and definitive hosts, which are usually mammals. In the south of the United States, two vectors are dominant: *Triatoma sanguisuga* and *Triatoma gerstaeckeri*, associated, respectively, with raccoons (and opossums) and with woodrats [4].

Spatial dispersion plays an important role in triatomines’ life cycle as it is essentially linked to foraging. The frequency of blood meals, which is very variable, has a major impact on the demography of the population. Indeed, it is established that spawning takes place after the blood meal. It is therefore appropriate to assume that between two successive generations three separate processes take place: demographics, infection and spatial dispersal.

This study develops a mathematical model which incorporates vector and host dispersal (as well as demography and infection) in order to describe the speed with which sylvatic *T. cruzi* infection spreads geographically.

2. Mathematical model.
Triatomines’ life cycle is composed of seven stages: an egg stage, five instars, and an adult stage. Adults’ longevity is usually longer than the accumulated times of the egg stage and larval stages [5]. We consider then vectors in two stages: juveniles and adults while, no class structure is taken on the hosts. Two stages of infection are considered: susceptible and infected.

2.1. Population status before the infection.
Before the onset of the disease, the population is characterized by the state vector $N_s(x, t) = (V_{js}, V_{as}, H_s)'(x, t)$ which represents respectively the juvenile vectors, adult vectors and hosts.

![Figure 1: Vectors’ and hosts’ life cycle, between two successive generations, during the time interval $[t, t + 1]$, before the disease onset.](image)
with, respectively, survival probabilities of $\sigma_{js}$, $\sigma_{as}$ and $\sigma_{hs}$. The transition probability from juvenile to adult is $\tau_s$. Adult vector and host fertilities are denoted by $f_{vs}$ and $f_{hs}$ respectively (Figure 1).

In the simplest case, the spatial dispersal can be described by linear movement along the real line $\mathbb{R}$. Demographic equations of the biological system are written thus, for any habitat point $x$ and for any generation $t$:

$$V_{js}(x, t + 1) = V_{js}(x, t) \sigma_{js}(1 - \tau_s) + V_{as}(x, t) \sigma_{as}f_{vs}$$

$$V_{as}(x, t + 1) = V_{js}(x, t)\sigma_{js} \tau_s + V_{as}(x, t) \sigma_{as}$$

$$H_s(x, t + 1) = H_s(x, t)\sigma_{hs}(1 + f_{hs})$$

Healthy populations (vectors and hosts) are assumed to be in steady state. This steady state requires that vectors’ and hosts’ fertility are density dependent, so that we take:

$$f_{vs} = f_{vs}(V_{js}, V_{as}) = f_{vs}^{\text{max}} \exp \left[ -(V_{js} + V_{as}) \right]$$

$$f_{h} = f_{h}^{\text{max}} \exp(-H_s)$$

where $f_{vs}^{\text{max}}$ and $f_{h}^{\text{max}}$ are the respective maximum fecundities. Steady state is achieved if demographic parameters satisfy the equations:

$$V_j^* = \frac{1 - \sigma_{as}}{1 - \sigma_{as} + \sigma_{js} \tau_s} \ln \frac{\sigma_{js} \sigma_{as} \tau_s f_{vs}^{\text{max}}}{(1 - \sigma_{js} + \sigma_{js} \tau_s)(1 - \sigma_{as})}$$

$$V_a^* = \frac{\sigma_{js} \tau_s}{1 - \sigma_{as} + \sigma_{js} \tau_s} \ln \frac{\sigma_{js} \sigma_{as} \tau_s f_{vs}^{\text{max}}}{(1 - \sigma_{js} + \sigma_{js} \tau_s)(1 - \sigma_{as})}$$

$$H_s^* = \ln \frac{\sigma_{hs} f_{hs}^{\text{max}}}{1 - \sigma_{hs}}$$

### 2.2. Population status after the infection’s onset.

After the infection’s onset, three new classes appear: they are infected juvenile vectors, infected vector adults and infected hosts, whose densities are denoted, respectively, by $V_{ji}(x, t)$, $V_{ai}(x, t)$ and $H_i(x, t)$. The total population is described then by the state vector $N(x, t) = (V_{js}, V_{as}, H_s, V_{ji}, V_{ai}, H_i)'(x, t)$ (Figure 2).

The biological system now, consists of six classes. It is always assumed to go through three processes: the demographic process between $[t, t + 1/3]$; the infection process between $[t + 1/3, t + 2/3]$ and the dispersal processes between $[t + 2/3, t + 1]$. Dividing the interval $[t, t + 1]$ into three equal intervals is formal: one process can be longer than another. The order of the process is the natural order: after the blood meal, the triatomines initiate their
demography and transmit or receive *T. cruzi*. After that, they will disperse until the next blood meal.

### 2.3. Demographic process.

Fecundity, which is a major life history trait, is initiated after the blood meal. We will assume in this study, that infection affects demographic parameters of neither vectors nor hosts. If \( \sigma_{ji} \), \( \sigma_{ai} \) and \( \sigma_{hi} \) represent respectively the survival probabilities of infected juvenile vectors, adult vectors and hosts, then we have the equalities: \( \sigma_{ji} = \sigma_{js} = \sigma_{j} \), \( \sigma_{ai} = \sigma_{as} = \sigma_{a} \), and \( \sigma_{hi} = \sigma_{hs} = \sigma_{h} \). The same hypothesis is applied to the transition probability of infected juvenile vectors to adulthood, so \( \tau_i = \tau_s = \tau \). Also, fecundity of infected adult vectors and hosts is not affected by infection: \( f_{vi} = f_{vs} = f_v \) and \( f_{hi} = f_{hs} = f_h \).

To highlight the parental effect, we distinguish between the new offspring (to vectors or hosts) by putting them in new temporary classes: new births to the vectors are placed in the class \( V^1_j \) (resp \( V^2_j \)) if they come from susceptible parents (resp. infected parents). Similarly \( H_1 \) (resp \( H_2 \)) are the new classes of the new births to hosts if they come from susceptible parents (resp. infected).

If we assume that the rate of vertical transmission of the disease in the vectors is null, then the classes \( V^1_j \) and \( V^2_j \) will furnish the juvenile susceptible class \( V_{js} \).

Assuming that vertical transmission among hosts affects a proportion \( \nu \) of newborns, then \( \nu H_2 \) individuals go to the class \( H_i \) and the other, non-infected, go to the class of susceptible hosts. Newborns to susceptible hosts are susceptible.

### 2.4. Infection process.

Formally, the infection process takes place during the time interval \([t + 1/3, t + 2/3]\). Let \( \Lambda_j \), \( \Lambda_a \) and \( \Lambda_h \) denote the probabilities of infection of juvenile vectors, adult vectors and hosts, respectively. It is natural to assume that these probabilities depend on the whole population state vector \( N(x, t) \) and transmission rates of the disease between different classes which are entries of the WAIFW matrix (who acquires infection from whom).

If \( \beta_{hi}, \beta_{ha}, \beta_{jh} \) and \( \beta_{ah} \) represent the rate of transmission of infection from hosts to juvenile vectors, from hosts to adult vectors, from juvenile vectors to hosts and from adult vectors to hosts, respectively, then these probabilities of infection are:

\[
\begin{align*}
\Lambda_j(N) &= 1 - \exp(-\beta_{hi}H_i) \\
\Lambda_a(N) &= 1 - \exp(-\beta_{ha}H_i) \\
\Lambda_h(N) &= 1 - \exp(-\beta_{jh}V_ji - \beta_{ah}V_{ai})
\end{align*}
\]
For simplicity, we consider that: $\beta_{hj} = \beta_{jh} = \beta_1$ and $\beta_{ha} = \beta_{ah} = \beta_2$.

At the end of the infection process, the balance, at each habitat point $x$, can be obtained via the transition matrix:

Figure 2: Schema of the demographic and infection processes. Parental effects were highlighted.
\[ (\sigma_j(1-\tau)(1-\Lambda_j) \quad \sigma_{af}(1-\Lambda_j) \quad 0 \quad 0 \quad \sigma_{af}(1-\Lambda_j) \quad 0 ) \\
\sigma_j(1-\Lambda_a) \quad 0 \quad 0 \quad 0 \quad 0 \quad 0 \\
\sigma_j(1-\Lambda_j) \quad \sigma_{af}\Lambda_j \quad \sigma_j(1-\tau) \quad \sigma_{af}\Lambda_i \quad 0 \quad 0 \\
\sigma_j\tau\Lambda_j \quad 0 \quad 0 \quad 0 \quad 0 \quad \sigma_h(1+f_h)(1-\Lambda_h) \] 

(5)

2.5. Dispersal process.

The dispersal process, corresponding to the search of blood meals for adult vectors, concerns also juvenile vectors. Laboratory results show that triatomine larvae are also able to disperse. On the other hand, dispersion among hosts is also permitted.

We assume that all species displace in one-dimensional space and the probability of far dispersal distance decreases with distance. The Laplace kernel, defined by:

\[ K(x) = \frac{1}{2\alpha} \exp \left( -\frac{|x|}{\alpha} \right) \] 

(6)

where \( \alpha \) is the mean dispersal ability, expressed as a (distance)/(step of time), fits correctly with this biological property. Let \( j \), \( a \) and \( h \) respectively denote the mean dispersal abilities of juveniles, adults and hosts. If we denote \( K_j(x), K_a(x) \) and \( K_h(x) \) the dispersal kernels of juveniles, adults and hosts respectively and assume that the disease does not affect these abilities, the densities are written as:

\[ V_{js}(x, t + 1) = \sigma_j(1-\tau) \int_{-\infty}^{+\infty} \left( 1-\Lambda_j \right) K_j(|x-y|) V_{js}(y, t) dy \]

\[ + \sigma_a \int_{-\infty}^{+\infty} f_a(1-\Lambda_j) K_a(|x-y|) V_{as}(y, t) dy \]

\[ + \sigma_a \int_{-\infty}^{+\infty} f_a(1-\Lambda_j) K_a(|x-y|) V_{ai}(y, t) dy \]  

(7a)

\[ V_{as}(x, t + 1) = \sigma_j \tau \int_{-\infty}^{+\infty} \left( 1-\Lambda_a \right) K_j(|x-y|) V_{js}(y, t) dy + \sigma_a \int_{-\infty}^{+\infty} \left( 1-\Lambda_a \right) K_a(|x-y|) V_{as}(y, t) dy \]

(7b)

\[ H_{s}(x, t + 1) = \sigma_h \int_{-\infty}^{+\infty} \left( 1+f_h(1-\Lambda_h) K_h(|x-y|) H_s(y, t) dy \right) \]

\[ + \sigma_h(1-\nu) \int_{-\infty}^{+\infty} f_h(1-\Lambda_h) K_h(|x-y|) H_t(y, t) dy \]  

(7c)
$V_j(x, t + 1) = \sigma_j (1 - \tau) \int_{-\infty}^{+\infty} \Lambda_j K_j(|x - y|) V_{j\ell}(y, t) dy + \sigma_a \int_{-\infty}^{+\infty} f_v \Lambda_j K_a(|x - y|) V_{as}(y, t) dy$

$\quad + \sigma_j (1 - \tau) \int_{-\infty}^{+\infty} K_j(|x - y|) V_{ij}(y, t) dy + \sigma_a \int_{-\infty}^{+\infty} f_v \Lambda_j K_a(|x - y|) V_{ai}(y, t) dy$

$(7d)$

$V_{ai}(x, t + 1) = \sigma_j \tau \int_{-\infty}^{+\infty} \Lambda_a K_j(|x - y|) V_{as}(y, t) dy + \sigma_a \int_{-\infty}^{+\infty} \Lambda_a K_a(|x - y|) V_{as}(y, t) dy$

$\quad + \sigma_j \tau \int_{-\infty}^{+\infty} K_j(|x - y|) V_{ij}(y, t) dy + \sigma_a \int_{-\infty}^{+\infty} \Lambda_a K_a(|x - y|) V_{ai}(y, t) dy$

$(7e)$

$H_i(x, t + 1) = \sigma_h \int_{-\infty}^{+\infty} (1 + f_h) \Lambda_h K_h(|x - y|) H_s(y, t) dy$

$\quad + \sigma_h \int_{-\infty}^{+\infty} [f_h \Lambda_h (1 - v) + f_h v + 1] K_h(|x - y|) H_i(y, t) dy$

$(7f)$

But each point of the habitat is supposed to be at its equilibrium state, defined by equations (3) so:

$\forall x \quad \forall t \quad V_{js}(x, t) + V_{j\ell}(x, t) = V_j^*$

$V_{as}(x, t) + V_{ai}(x, t) = V_a^*$

$H_s(x, t) + H_i(x, t) = H^*$

Equations (8) allow to reduce equations (7) to infected populations only, so we finally obtain:

$V_j(x, t + 1) = \sigma_j (1 - \tau) V_j^* \int_{-\infty}^{+\infty} \Lambda_j K_j(|x - y|) dy + \sigma_a V_a^* \int_{-\infty}^{+\infty} f_v \Lambda_j K_a(|x - y|) dy$

$\quad + \sigma_j (1 - \tau) \int_{-\infty}^{+\infty} (1 - \Lambda_j) K_j(|x - y|) V_{ij}(y, t) dy$

$(9a)$

$V_{ai}(x, t + 1) = \sigma_j \tau V_j^* \int_{-\infty}^{+\infty} \Lambda_a K_j(|x - y|) dy + \sigma_a V_a^* \int_{-\infty}^{+\infty} \Lambda_a K_a(|x - y|) dy$

$\quad + \sigma_j \tau \int_{-\infty}^{+\infty} (1 - \Lambda_a) K_j(|x - y|) V_{ij}(y, t) dy + \sigma_a \int_{-\infty}^{+\infty} (1 - \Lambda_a) K_a(|x - y|) V_{ai}(y, t) dy$

$(9b)$

$H_i(x, t + 1) = \sigma_h H^* \int_{-\infty}^{+\infty} (1 + f_h) \Lambda_h K_h(|x - y|) dy$

$\quad + \sigma_h \int_{-\infty}^{+\infty} (1 + f_h) (1 - \Lambda_h) K_h(|x - y|) H_i(y, t) dy$

$(9c)$

Equations (9) are the major results of this section: they describe the three processes that occur between two successive generations. Equations (9) will be analyzed numerically and analytically.
This section presents some numerical results using sample parameter values to illustrate the roles played by certain quantities in the advance of the infection. Results are shown for positive values beginning at 0, with the wave moving in the positive direction from one time step to the next. In each resulting “generation” the infection spreads through the dispersal of infected hosts and vectors, which then begin the infection process in their new locations. In this way the maximal accumulated dispersal increases with each generation. Graphs in this section show the sizes of infected populations only; comparing results from different scenarios shows that model parameters affect not only the amplitude and speed of the traveling wave solutions, but also which population (juvenile vectors, adult vectors, or hosts) carries the leading edge of the infection.

Table 1 presents a representative set of results for a sample parameter set including a time step of 2 weeks. Here, as seen in the superposed graphs on the right showing the waves in the three populations after 20, 26, and 30 generations, the host population carry the wavefront, followed closely by the juvenile vectors despite their lower dispersal ability, and finally the adult vectors.

We can investigate the role played by the maturation rate of the vectors by increasing the proportion of juveniles which mature at each time step. Table 2 presents results based on a transition probability of 0.9, an order of magnitude higher than the baseline provided by Table 1, where only 0.09 (9%) mature each time step. The most immediately evident result is a marked change in the shape of the traveling wave for the juvenile vectors, as seen in the first graph in Table 2; after an initial rise, the infection levels among juveniles level off, since the vast majority of infected juveniles in each generation become infected adults in the following generation. As illustrated in the comparative graphs on the right side of the table, the resulting depression in the levels of juvenile vector infection compared to those for adult vectors and hosts makes the waves in these latter two populations essentially identical.

We can also investigate the effects of changing the time step or generation time; however, since those model parameters representing probabilities or proportions of events (birth, death, infection, etc.) occurring within a single generation, a change in the time step necessitates changes in many of the other parameters. For a proportion \( p \) derived from an average rate \( r \) (or, inversely, average waiting time \( 1/r \)), the conversion to a proportion or probability uses an exponential law: \( p = 1 - \exp(-rT) \), where \( T \) is the time step. So, for example, if juvenile vectors mature at a rate of \( r = 0.047/\text{week} \), then when \( T = 2\text{wk} \), the maturation proportion \( \tau = 1 - \exp(-0.047\times 2) = 0.09 \). When \( T = 4\text{wk} \), however, we have \( \tau = 1 - \exp(-0.047\times 4) = 0.17 \). The change in generation time therefore changes several parameters (see Table 3), but in this case produces results consistent with those in Table 1. This is reassuring since the
change in time step is largely a modeling decision in this case, and the consistency in results shows that the decision does not have a major impact on predictions.

Demographic parameters:

- $d_j = 0.94$
- $\sigma_a = 0.88$
- $\sigma_h = 0.90$
- $j_{\text{max}} = 100$
- $f_{\text{max}} = 1$
- $\tau = 0.09$

Infection parameters:

- $v = 0.5$
- $\beta_1 = 0.001$
- $\beta_2 = 0.001$

Dispersal parameters:

- $j = 0.5$
- $a = 1.0$
- $h = 1.0$

**Table 1:** Juvenile vectors, adult vectors and hosts travelling wave, when varying the number of generations.
**Demographic parameters:**

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>$\sigma_j$</td>
<td>0.94</td>
</tr>
<tr>
<td>$\sigma_a$</td>
<td>0.88</td>
</tr>
<tr>
<td>$\sigma_h$</td>
<td>0.90</td>
</tr>
<tr>
<td>$f_{\text{max}}$</td>
<td>100</td>
</tr>
<tr>
<td>$f_{\text{max}}$</td>
<td>1</td>
</tr>
<tr>
<td>$\tau$</td>
<td>0.9</td>
</tr>
</tbody>
</table>

**Time step**: 2 weeks

**Infection parameters:**

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>$\nu$</td>
<td>0.5</td>
</tr>
<tr>
<td>$\beta_1$</td>
<td>0.001</td>
</tr>
<tr>
<td>$\beta_2$</td>
<td>0.001</td>
</tr>
</tbody>
</table>

**Dispersal parameters:**

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>$j$</td>
<td>0.5</td>
</tr>
<tr>
<td>$a$</td>
<td>1.0</td>
</tr>
<tr>
<td>$h$</td>
<td>1.0</td>
</tr>
</tbody>
</table>

**Table 2**: Travelling wave of juveniles, adults and hosts obtained by varying the number of generations. The difference with Table 1 is that the value of the transition probability from the juvenile class to the adult class is greatly increased (from 0.09 to 0.9)
### Table 3

<table>
<thead>
<tr>
<th>Demographic parameters:</th>
<th>Infection parameters:</th>
<th>Dispersal parameters:</th>
</tr>
</thead>
<tbody>
<tr>
<td>( \sigma_j )</td>
<td>( \nu )</td>
<td>( j )</td>
</tr>
<tr>
<td>( \sigma_a )</td>
<td>( \beta_1 )</td>
<td>( a )</td>
</tr>
<tr>
<td>( \sigma_h )</td>
<td>( \beta_2 )</td>
<td>( h )</td>
</tr>
<tr>
<td>( f_{\text{max}}^j )</td>
<td></td>
<td></td>
</tr>
<tr>
<td>( f_{\text{max}}^a )</td>
<td></td>
<td></td>
</tr>
<tr>
<td>( \tau )</td>
<td></td>
<td></td>
</tr>
<tr>
<td>0.89</td>
<td>0.5</td>
<td>0.5</td>
</tr>
<tr>
<td>0.76</td>
<td>0.001</td>
<td>1.0</td>
</tr>
<tr>
<td>0.80</td>
<td>0.001</td>
<td>1.0</td>
</tr>
</tbody>
</table>

Table 3: Travelling waves of the juveniles, adults and hosts obtained by varying the number of generations for a time step of 4 weeks.
Finally, Tables 4 and 5 investigate the role played by vertical transmission in the spread of infection. The graphs in these tables show results for a full spectrum of values for the vertical transmission probability $\nu$, from 0 to 1. Table 4 shows that, predictably, the infection spreads faster as the vertical transmission proportion increases. The comparative graphs in Table 5, however, show a less obvious result: when vertical transmission is unlikely (say $\nu < 0.3$), the wavefront is carried by the vectors (with juveniles and vectors in close alignment, and much higher incidence than in the host population), but when vertical transmission is more likely it is instead the hosts that carry the wavefront, as the vertical transmission amplifies the spread of infection in the host population so much that it becomes a much stronger source of infection (for vectors) than the vectors are (for hosts).
Table 5: Travelling waves of juveniles, adults and hosts obtained by varying the rate of vertical transmission of disease from 0% to 100%.

4. Analytical results.

Equations (9) can be rewritten using Neubert-Caswell notation; indeed, if we put:

\[
A_{N_i} = \begin{pmatrix}
\sigma_j (1 - \tau) \Lambda_j & \sigma_a f_h \Lambda_j & 0 \\
\sigma_j \tau \Lambda_a & \sigma_a \Lambda_a & 0 \\
0 & 0 & \sigma_h (1 + f_h) \Lambda_h
\end{pmatrix}
\]  \hspace{1cm} (10a)

\[
B_{N_i} = \begin{pmatrix}
\sigma_j (1 - \tau) (1 - \Lambda_j) & 0 & 0 \\
\sigma_j \tau (1 - \Lambda_a) & \sigma_a (1 - \Lambda_a) & 0 \\
0 & 0 & \sigma_h (1 + v f_h) (1 - \Lambda_h)
\end{pmatrix}
\]  \hspace{1cm} (10b)

and the dispersal kernels matrix:

\[
K(x) = \begin{pmatrix}
K_j(x) & K_a(x) & K_h(x) \\
K_j(x) & K_a(x) & K_h(x) \\
K_j(x) & K_a(x) & K_h(x)
\end{pmatrix}
\]  \hspace{1cm} (10c)

we obtain the equation:
\[ N_i(x, t + 1) = \int_{-\infty}^{+\infty} [K(|x - y|) \circ A_{N_i}] N^* dy + \int_{-\infty}^{+\infty} [K(|x - y|) \circ B_{N_i}] N_i(y, t) dy \quad (11) \]

where the state vector is \( N_i(x, t) = (V_{ji}, V_{ai}, H_i)'(x, t) \) and \( N^* = (V^*_j, V^*_a, H^*)' \) and " \( \circ \) " is the product term to term.

We define:

\[
\begin{align*}
\varphi(V_{ji}, V_{ai}, H_i) &= \sigma_j (1 - \tau) A_j V_j^* + \sigma_a f_a A_j V_a^* \\
\psi(V_{ji}, V_{ai}, H_i) &= \sigma_j \tau A_a V_j^* + \sigma_a A_a V_a^* \\
\chi(V_{ji}, V_{ai}, H_i) &= \sigma_h (1 + f_h) A_h H^* 
\end{align*}
\]

(12a) (12b) (12c)

It is clear that these three functions are of class \( C^1(\mathbb{R}^3) \), Taylor expansion of the vectorial function \( \Phi = (\varphi, \psi, \chi)' \) and in the neighborhood of \((0,0,0)\) gives:

\[ A_{N_i} N^* = \Phi |_{(0,0,0)} + J_\Phi |_{(0,0,0)} (V_{ji}, V_{ai}, H_i)' + (R_1, R_2, R_3)' |_{(V_{ji}, V_{ai}, H_i)} \]

(13)

where the \( \lim_{(V_{ji}, V_{ai}, H_i) \to (0,0,0)} R_k (V_{ji}, V_{ai}, H_i) = 0 \) for \( k = 1, 2, 3 \) and \( J_\Phi \) is the jacobian of \( \Phi \) defined by:

\[
J_\Phi = \begin{pmatrix}
-\sigma_a f_a V_a^* A_j & -\sigma_a f_a V_a^* A_j & \beta_1 [\sigma_j (1 - \tau) V_j^* + \sigma_a f_a V_a^*] (1 - A_j) \\
0 & 0 & \beta_2 [\sigma_j \tau V_j^* + \sigma_a V_a^*] (1 - A_a) \\
\beta_1 \sigma_h (1 + f_h) H^* (1 - A_h) & \beta_2 \sigma_h (1 + f_h) H^* (1 - A_h) & -\sigma_h f_h H^* A_h 
\end{pmatrix}
\]

(14)

But:

\[ \Phi |_{(0,0,0)} = 0 \]

(15a)

and:

\[
J_\Phi |_{(0,0,0)} = \begin{pmatrix}
0 & 0 & \beta_1 [\sigma_j (1 - \tau) V_j^* + \sigma_a f_a V_a^*] \\
0 & 0 & \beta_2 [\sigma_j \tau V_j^* + \sigma_a V_a^*] \\
\beta_1 \sigma_h (1 + f_{max}) H^* & \beta_2 \sigma_h (1 + f_{max}) H^* & 0
\end{pmatrix}
\]

(15b)

By the linear conjecture, the invasion speed of the system described by the integrodifference equation (11) is the same as that of the system:

\[ N_i(x, t + 1) = \int_{-\infty}^{+\infty} K(|x - y|) \circ [J_\Phi |_{(0,0,0)} + B_{(0,0,0)}] N_i(y, t) dy \]

(16)

Following the Neubert-Caswell theorem [6], the matrix of the moment generating functions can be written via (10c) and (6):
\[
M(s) = \begin{pmatrix}
1 & 1 & 1 \\
1 - j^2 s^2 & -\alpha^2 s^2 & 1 \\
1 & 1 - h^2 s^2 & 1
\end{pmatrix}
\]

The asymptotic invasion speed depends on the largest eigenvalue of the matrix \( H(s) = [J_\phi|_{(0,0,0)} + B_{(0,0,0)}] \circ M(s) \), which is defined by the relation:

\[
H(s) = \begin{pmatrix}
\frac{\sigma_f(1-\tau)}{1-j^2 s^2} & 0 & \frac{\beta_1 [\sigma_f(1-\tau) \nu_j + \sigma_a h_{as} \nu_a]}{1-h^2 s^2} \\
\frac{\sigma_j^*}{1-j^2 s^2} & \frac{\sigma_a}{1-\alpha^2 s^2} & \frac{\beta_2 [\sigma_f \nu_j + \sigma_a \nu_a]}{1-h^2 s^2} \\
(\beta_1 \sigma_h (1+f_{h_{max}}) H^*) & (\beta_2 \sigma_h (1+f_{h_{max}}) H^*) & \sigma_h (1+\nu_{h_{max}}) H^*
\end{pmatrix}
\]

Indeed, if \( \rho(s) \) is the largest eigenvalue of the matrix \( H(s) \), then the asymptotic invasion speed of the biological system described by equations (11) satisfies:

\[
c^* = \min_{0 < s < \hat{s}} \left[ \frac{1}{\hat{s}} \ln \rho(s) \right]
\]

where \( \hat{s} = \min \left( \frac{1}{j}, \frac{1}{\alpha}, \frac{1}{h} \right) \) in order to avoid singularities at \( s = j, s = \alpha \) and \( s = h \). This expression can be evaluated numerically for specific parameter values.

A question arises from this calculation, however, which is: to which wave does this speed correspond, i.e., in which of the three populations does the infection spread with this speed? Or does it represent some combination of the three, as might be suggested by an eigenvector of \( H(s) \)? Asked another way, what contribution does each species make to the invasion speed? The Neubert-Caswell measure was originally developed for single populations [6], and the extension developed here for multiple populations leaves this question open and deserving of further study.

5. Conclusion.

This report develops and illustrates a spatially explicit model for the sylvatic transmission of the parasite \( Tryptasoma cruzi \). The model can easily be adapted to represent other vector-borne infections. The model, consisting of three coupled integrodifference equations, allows one to describe the spread (in a single spatial dimension) of \( T. cruzi \) infection among dispersing
populations of juvenile and adult vectors, and hosts, and to develop an expression for the asymptotic invasion speed.

The numerical results presented here using sample parameter values show the influences of some of the demographic processes on the invasion speed, most notably how vertical (transplacental) transmission in hosts changes which species carries the wavefront. It remains to develop more precise estimates of the processes under study in order to derive an estimate of invasion speed which can be compared with other studies presently underway using other modeling approaches. These results will also permit the calculation and study of spatial as well as temporal variation in the infection's basic reproductive number, $R_0$.


