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Describing Chagas Disease Spread by Traveling Waves

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Abstract. We propose a mathematical model using a system of partial differential reaction-diffusion equations to study and to describe the spread of Chagas disease in the human population in spatial short scale. We determine the speed of the disease propagation by using the traveling wave solutions of the model. We determine the wave speed variation as function of the diffusion coefficient.

Key-words. Chagas disease, Triatomines, Reaction-Diffusion Equation, Traveling waves, Wave speed.

1 Introduction

Chagas disease, discovered by the Brazilian physician and epidemiologist Carlos Chagas at the beginning of the last century, is caused by the flagellated protozoan *Trypanosoma cruzi*, of the *Trypanosomatidae* family. *Trypanosoma cruzi* may infect more than 100 species of mammals from different orders and exists in populations of vertebrate hosts, including wild and domestic animals and humans. This parasite is also found in invertebrate populations, such as insects. In humans, *Trypanosoma cruzi* transmission mainly occurs through a vector, triatomines, commonly known in Brazil as Barbeiro (the kissing bug or barber bug).

Triatomines can only transmit parasites if they are infected, and they must feed on the blood of a mammalian host with the parasite to become infected. Thus, if a specific location has high *Trypanosoma cruzi* infection rates, the vector will likely become infected and in turn infect humans or other mammals, thereby completing its cycle.

Human infection with *Trypanosoma cruzi* occurs in two phases: acute and chronic. In the acute phase, the parasite circulates in the bloodstream in considerable quantities. In this phase, signs and symptoms may spontaneously disappear, progressing to the chronic phase or to death. In the chronic phase, few parasites circulate in the blood stream.

We propose a model to study the spatial dynamics of Chagas disease that considers the population of vectors (barbeiros) and the population of host individuals (humans).

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For the temporal dynamic, we modified in [5] a model proposed in 2005 by Das and Mukherjee [3] because, based on the references to the disease, [1], we noted that the transition from the acute to the chronic phase does not occur through a second infected bite. Some individuals in the acute phase progress to the chronic phase and others recover upon treatment. We modeled the spatial dynamics of the spread of the disease through the diffusion and advection of the vectors (triatomines). We present this model for studying the spatial spread of Chagas disease in the next section.

2 Spatial Model for Chagas disease

In this section , we propose a model that considers a population of vectors $\overline{N}(x,t)$ divided into susceptible $\overline{V}(x,t)$ and infected $\overline{W}(x,t)$ vectors and a population of hosts $\overline{H}(x,t)$, divided into susceptible individuals $\overline{S}(x,t)$ and individuals infected in the acute $\overline{A}(x,t)$ and chronic $\overline{C}(x,t)$ phases of the disease. We describe the spatial spread of the disease by the diffusion and advection of the vectors (triatomines) only, disregarding the spatial locomotion of the population of individuals on a large scale. We consider that susceptible and infected vectors have the same constant of diffusion D, because we assume that the disease has no effect on vector locomotion, [4].

The class of susceptible individuals acquires the disease at a rate $b\alpha$ through the bite of an infected vector, or at a rate $p\mu$ through vertical transmission. The parameter b is the daily mean number of bites per vector and α is the probability of disease transmission from vector to human. Therefore, $b\frac{\overline{N}}{\overline{H}}$ is the average number of bites per human, and the probability of being stung by an infected vector also depends on the ratio of that subpopulation $\frac{\overline{W}}{\overline{N}}$. Thus, the infection rate from vector to human is given by: $b\alpha\frac{\overline{N}}{\overline{H}}\frac{\overline{W}}{\overline{N}}$.

We consider the rate of recovery of individuals in the acute phase, $q\gamma$, through treatment by medication, and therefore, $(1-q)\gamma$ is the rate of individuals in the acute phase who do not recover and instead progress to the chronic phase of the disease. The mortality rate of the disease for individuals in the chronic phase is denoted by θ, and the birth rate and natural mortality rate of humans is denoted by μ.

We consider η to be the birth rate and natural mortality rate of the vectors. The subpopulation of susceptible vectors acquires the disease at a rate $b\beta_1$ by biting infected individuals in the acute phase and at a rate $b\beta_2$ by biting infected individuals in the chronic phase, wherein β_1 and β_2 are the probabilities of transmission from a human in the acute and chronic phases to the vector, respectively. Thus, the infection rate of susceptible vectors will depend on the population of infected humans in the acute phase, which is $b\beta_1\frac{\overline{A}}{\overline{H}}$, and on the population of infected humans in the chronic phase, $b\beta_2\frac{\overline{C}}{\overline{H}}$.

The following model expresses the spatiotemporal dynamics of disease:

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$$\begin{aligned}
\frac{\partial \overline{S}}{\partial t} &= \mu\overline{H} + \theta\overline{C} - p\mu\overline{C} - \frac{b\alpha}{\overline{\overline{H}}}\overline{S}\ \overline{W} + q\gamma\overline{A} - \mu\overline{S}, && (1)\\
\frac{\partial \overline{A}}{\partial t} &= \frac{b\alpha}{\overline{\overline{H}}}\overline{S}\ \overline{W} + p\mu\overline{C} - \gamma\overline{A} - \mu\overline{A}, && (2)\\
\frac{\partial \overline{C}}{\partial t} &= (1-q)\gamma\overline{A} - \theta\overline{C} - \mu\overline{C}, && (3)\\
\frac{\partial \overline{V}}{\partial t} &= D\frac{\partial^2 \overline{V}}{\partial x^2} + \eta\overline{N} - b\left(\frac{\beta_1}{\overline{\overline{H}}}\overline{A} + \frac{\beta_2}{\overline{\overline{H}}}\overline{C}\right)\overline{V} - \eta\overline{V}, && (4)\\
\frac{\partial \overline{W}}{\partial t} &= D\frac{\partial^2 \overline{W}}{\partial x^2} + b\left(\frac{\beta_1}{\overline{\overline{H}}}\overline{A} + \frac{\beta_2}{\overline{\overline{H}}}\overline{C}\right)\overline{V} - \eta\overline{W}. && (5)
\end{aligned}$$

Here, D is the coefficient of diffusion of the vector population. We aim to determine the spatial spread of Chagas Disease. The total population is $\overline{H} = \overline{S} + \overline{A} + \overline{C}$, and by adding the first three equations of the above system, (1), (2) and (3), we obtain

$$\frac{\partial \overline{H}}{\partial t} = \frac{\partial \overline{S}}{\partial t} + \frac{\partial \overline{A}}{\partial t} + \frac{\partial \overline{C}}{\partial t} = 0, \tag{6}$$

that is, the total population $\overline{H}$ of host individuals is constant over time.

Adding equations (4) and (5), we obtain

$$\frac{\partial \overline{V}}{\partial t} + \frac{\partial \overline{W}}{\partial t} = D\frac{\partial^2 \overline{V}}{\partial x^2} + D\frac{\partial^2 \overline{W}}{\partial x^2}, \tag{7}$$

where $\overline{N} = \overline{V} + \overline{W}$, then

$$\frac{\partial \overline{N}}{\partial t} = D\frac{\partial^2 \overline{N}}{\partial x^2}. \tag{8}$$

Then $\overline{N} = N$, with N constant, is a solution for the Partial Differential Equation (8). Assuming that the human and triatomine populations are already established in the region and that the total population of vectors $\overline{N}$ is constant, similar to the total population of individuals $\overline{H}$, we can normalize the system of equations writing: $S = \dfrac{\overline{S}}{\overline{\overline{H}}}$, $A = \dfrac{\overline{A}}{\overline{\overline{H}}}$, $C = \dfrac{\overline{C}}{\overline{\overline{H}}}$, $V = \dfrac{\overline{V}}{\overline{\overline{N}}}$ e $W = \dfrac{\overline{W}}{\overline{\overline{N}}}$.

Considering that the populations are constant, $S = 1 - A - C$ and $V = 1 - W$, and that we may uncouple the variables S and V from the system, then the system (1) - (5) is equivalent to the following system of equations:

$$\begin{aligned}
\frac{\partial A}{\partial t} &= e(1-A-C)W + p\mu C - (\gamma+\mu)A,\\
\frac{\partial C}{\partial t} &= (1-q)\gamma A - (\theta+\mu)C, && (9)\\
\frac{\partial W}{\partial t} &= D\frac{\partial^2 W}{\partial x^2} + (b_1 A + b_2 C)(1-W) - \eta W,
\end{aligned}$$

where $e = b\alpha\frac{\overline{\overline{N}}}{\overline{\overline{H}}}$, $b_1 = b\beta_1$ and $b_2 = b\beta_2$,

2.1 Traveling wave solutions

Let us assess the rate of spatial spread of Chagas disease when some infected individuals are introduced into a region with a fully susceptible population. We attempt to find a traveling wave linking equilibrium and determine the conditions for existence and the propagation speed of such a wave.

Traveling wave solutions are of the form:

$A(x,t) = a(z)$, $C(x,t) = c(z)$, $W(x,t) = w(z)$, where $z = x - \nu t$ and ν is the constant wave speed, which will be determined. Replacing this in the equation system (9), we obtain the following system of first-order equations

$$\begin{aligned} \frac{da}{dz} &= -\frac{e}{\nu}(1-a-c)w - \frac{p\mu}{\nu}c + \frac{(\gamma+\mu)}{\nu}a, \\ \frac{dc}{dz} &= -\frac{(1-q)\gamma}{\nu}a + \frac{(\theta+\mu)}{\nu}c, \\ \frac{dw}{dz} &= u, \\ \frac{du}{dz} &= -\frac{\nu}{D}u - \frac{(b_1 a + b_2 c)}{D}(1-w) + \frac{\eta}{D}w, \end{aligned} \tag{10}$$

where $\dfrac{du}{dz} = w''(z)$.

There are two equilibrium points of the equation system (10): the first $Q_1 = (0,0,0,0)$, which represents the disease-free equilibrium, and the second, which is the only non-trivial equilibrium that corresponds to infection, $Q_2 = (A^*, C^*, W^*, 0)$, where A^*, C^* and W^* are non-negative, given by:

$$C^* = \frac{(1-q)\gamma A^*}{\theta+\mu} \text{ and } W^* = \frac{(b_1(\theta+\mu) + b_2(1-q)\gamma)A^*}{b_1(\theta+\mu)A^* + b_2(1-q)\gamma A^* + \eta(\theta+\mu)}, \tag{11}$$

and A^* is given by:

$$A^* = \frac{e(\theta+\mu)[R_0 - 1]}{R_0[e(\theta+\mu+(1-q)\gamma) + (\gamma+\mu)(\theta+\mu) - p\mu(1-q)\gamma]}, \tag{12}$$

where R_0 the basic reproduction number given by:

$$R_0 = \frac{e}{\eta}\left[\frac{b_1(\theta+\mu) + b_2(1-q)\gamma}{(\gamma+\mu)(\theta+\mu) - p\mu(1-q)\gamma}\right].$$

Considering that $k = (\gamma+\mu)(\theta+\mu) - p\mu(1-q)\gamma > 0$ then $A^* > 0$ when $R_0 > 1$. In this case $C^* > 0$ and $W^* > 0$.

We want to find the conditions for a solution with a positive wave propagation speed ν and with non-negative w, a and c that meet the following boundary conditions

$$\begin{aligned} w(-\infty) = W^* \quad &\text{and} \quad w(+\infty) = 0, \\ a(-\infty) = A^* \quad &\text{and} \quad a(+\infty) = 0, \\ c(-\infty) = C^* \quad &\text{and} \quad c(+\infty) = 0, \\ u(-\infty) = 0 \quad &\text{and} \quad u(+\infty) = 0. \end{aligned}$$

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As the variables used are populations, we searched for solutions in the positive region, that is, such that in the solution, $w(z) > 0$, $a(z) > 0$, $c(z) > 0$ and the solution cannot oscillate near the origin. Then, the eigenvalues must be real and non-complex values, otherwise $w(z) < 0$, or $a(z) < 0$, or $c(z) < 0$, for some z. We will linearize the system (10) at equilibrium Q_1 to analyze the eigenvalues. This is not necessary for the equilibrium Q_2 as long as it belongs to the positive region, which is invariant in the system. Linearizing the system (10) at point $Q_1 = (0, 0, 0, 0)$, we obtain the Jacobian matrix of the system at Q_1, given by

$$Dg_{(0,0,0,0)} = \begin{pmatrix} \frac{(\gamma+\mu)}{\nu} & -\frac{p\mu}{\nu} & -\frac{e}{\nu} & 0 \\ -\frac{(1-q)\gamma}{\nu} & \frac{(\theta+\mu)}{\nu} & 0 & 0 \\ 0 & 0 & 0 & 1 \\ -\frac{b_1}{D} & -\frac{b_2}{D} & \frac{\eta}{D} & -\frac{\nu}{D} \end{pmatrix}, \tag{13}$$

where
$g(a, c, w, u) = (-\frac{e}{\nu}(1 - a - c)w - \frac{p\mu}{\nu}c + \frac{(\gamma+\mu)}{\nu}a, -\frac{(1-q)\gamma}{\nu}a + \frac{(\theta+\mu)}{\nu}c, u, -\frac{\nu}{D}u - \frac{(b_1a+b_2c)}{D}(1 - w) + \frac{\eta}{D}w)$.

The corresponding eigenvalues are the roots of the polynomial

$$p(\lambda) = \lambda^4 + d_3\lambda^3 + d_2\lambda^2 + d_1\lambda + d_0, \tag{14}$$

where

$$\begin{aligned} d_0 &= \frac{\eta[(\gamma+\mu)(\theta+\mu) - p\mu\gamma(1-q)]}{D\nu^2}[R_0 - 1], \\ d_1 &= \frac{(\gamma+\mu)(\theta+\mu) + \eta(\gamma+\theta+2\mu) - \gamma p\mu(1-q) - eb_1}{D\nu}, \\ d_2 &= \frac{-\nu^2(\gamma+\eta+\theta+2\mu) + D[(\gamma+\mu)(\theta+\mu) - \gamma p\mu(1-q)]}{D\nu^2}, \\ d_3 &= \frac{\nu^2 - D(\gamma+\theta+2\mu)}{D\nu}. \end{aligned}$$

As $R_0 > 1$, $d_0 > 0$, that is, $p(0) = d_0 > 0$. Furthermore, $lim_{\lambda\to\pm\infty}p(\lambda) = +\infty$. Thus, we concluded that this polynomial may have 4 complex roots, or, 2 conjugated complex roots and 2 real roots, or, to provide the minimum wave propagation speed, 4 real roots. This provides a method to calculate the minimum wave speed.

For the parameters: $b = 0.04\ days^{-1}$, $\alpha = 0.0009$, $\beta_1 = 0.03$, $\beta_2 = 0.03$, $\eta = 0.005\ days^{-1}$, $\theta = 0.00013\ days^{-1}$, $\mu = 0.000042\ days^{-1}$, $p = 2\%$, $q = 0.04$ and $1/\gamma = 56\ days$, found in Cruz-Pacheco *et al.* [2], when the dispersal coefficient considered is $D = 0.125\ km^2/day$, [4], the polynomial $p(\lambda)$ always has two different positive real roots and two roots that determine the wave propagation speed ν, which may be real or complex. Thus, the lowest value of speed ν such that $p(\lambda)$ has all real roots and, therefore, such that there is a stable traveling wave solution is $\nu_{min} = 0.0074\ km/day$, Figure 1.

For different values of D (diffusion coefficient), we obtained the minimum wave propagation speed using the polynomial $p(\lambda)$ as shown in Table 1.

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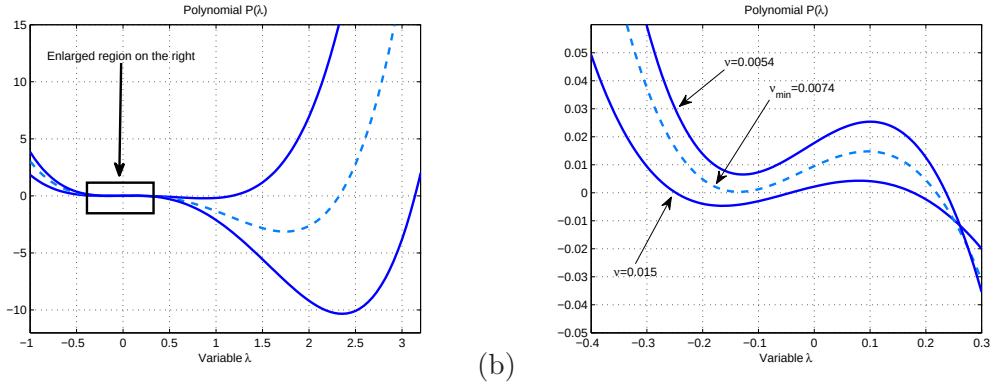

(a) (b)

Figure 1: (a) Graph of the polynomial $p(\lambda)$ for the parameters: $b = 0.04\ days^{-1}$, $\alpha = 0.0009$, $\beta_1 = 0.03$, $\beta_2 = 0.03$, $\eta = 0.005\ days^{-1}$, $\theta = 0.00013\ days^{-1}$, $\mu = 0.000042\ days^{-1}$, $p = 2\%$, $q = 0.04$ and $1/\gamma = 56\ days$, considering $D = 0.125\ km^2/day$, for three speeds: $\nu = 0.0054\ km/day < \nu_{min}$, $\nu = 0.0074\ km/day = \nu_{min}$ e $\nu = 0.015\ km/day > \nu_{min}$. (b) Zoomed-in view of the rectangular region drawn in the Figure 1 (a), showing that all roots of $p(\lambda)$ are real for $\nu \geq 0.0074\ km/day$.

Table 1: Wave speed for different diffusion coefficients calculated through the polynomial $p(\lambda)$.

Diffusion coefficient D	Wave speed ν_{min}
$0.005\ km^2/day$	$0.0015\ km/day$
$0.020\ km^2/day$	$0.0030\ km/day$
$0.045\ km^2/day$	$0.0045\ km/day$
$0.080\ km^2/day$	$0.0060\ km/day$
$0.125\ km^2/day$	$0.0074\ km/day$
$0.180\ km^2/day$	$0.0089\ km/day$
$0.245\ km^2/day$	$0.0104\ km/day$

Note that the minimum speed increases with the diffusion coefficient and that for $D \in [0.0005, 0.245]\ km^2/day$, the minimum wave propagation speed $\nu_{min} \in [0.0015, 0.0104]$ km/day. The fit function $\nu(D) = 0.021\sqrt{D}$ describe this wave speed as shown in Figure 2.

3 Conclusion

We study the spread of the disease in a human population using the spatial model. We conducted the analysis of the model under the condition $R_0 > 1$. When this condition hold, infection is established in the population and there is a trajectory linking the disease-free equilibrium and the endemic equilibrium, enabling the determination of the minimum disease spread rate. We determine the wave speed as function of the diffusion movement.

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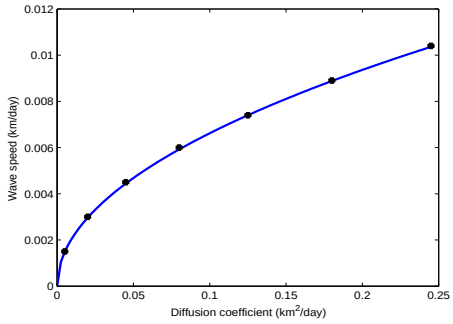

Figure 2: Graph of the wave speed as a function of the diffusion coefficient D for the values given in Table 1 (dots) and the fit function for these values given by $\nu(D) = 0.021\sqrt{D}$ (solid line). The values used for the other parameters are $b = 0.04\ days^{-1}$, $\alpha = 0.0009$, $\beta_1 = 0.03$, $\beta_2 = 0.03$, $\eta = 0.005\ days^{-1}$, $\theta = 0.00013\ days^{-1}$, $\mu = 0.000042\ days^{-1}$, $p = 2\%$, $q = 0.04$ and $1/\gamma = 56\ days$, found in Cruz-Pacheco *et al.* [2].

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