Modeling Chagas Disease and Control Measures

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Abstract

Chagas disease, also known as American trypanosomiasis, is a potentially life-threatening illness caused by the protozoan parasite, Trypanosoma cruzi (T. cruzi). Chagas disease is considered the most important vector borne infection in Latin America, with an estimation of 16 to 18 millions infected persons, and about 50,000 deaths each year [5].

The main mode of transmission of Chagas disease in endemic areas is through an insect vector called a triatomine bug. A triatomine becomes infected with T. cruzi by feeding on the blood of an infected person or animal. Chagas disease may also be spread through blood transfusion and organ transplantation, ingestion of food contaminated with parasites, and from a mother to her fetus. The rate of trans-placental transmission from mothers with chronic T cruzi infection to their newborns is 2-10\% [4].

In the early stage of the infection, acute stage, the symptoms are mild and usually produce no more than local swelling at the site of infection, usually around the eyes in children. Anti parasitic treatment with benznidazol and nifurtimox in the acute phase may result in cure rates between 60 and 90 percent. Usually, after 48 weeks, individuals with active infections enter the chronic phase of Chagas disease that is asymptomatic for 60 to 80 percent of chronically infected individuals through their lifetime. The anti parasitic treatments also appear to delay or prevent the development of disease symptoms during the chronic phase of the disease, but 20 to 40 percent of chronically infected individuals will still eventually develop life-threatening heart and digestive system disorders [1].

Control measures include insecticides to kill the vector, screening blood donors, and treatment to patients in the acute phase. Recently, a controversial strategy, Zooprophylaxis, has been proposed for the control of vector transmitted diseases [7]. This controversial technique refers to the control of vector-borne diseases by attracting vectors to domestic animals in which the pathogen cannot amplify (a dead-end host).
In this work we formulate a model to study the transmission of this illness among the vector, humans and some mammals. Our main objective is to assess the effectiveness of some control measures for the infection. We attain this through a sensitivity analysis of the basic reproductive number $R_0$ with respect to the epidemiological and demographic parameters.

1 Formulation of the model

We consider transmission by triatomine bites, and vertical transmission since these are the most common routes of infection.

We will consider the following populations:

- Humans
- Transmitters: mammals that can be infected by the Triatome bugs, and can transmit the infection (like dogs, cats, etc.)
- Non-transmitters: animals that can be bitten by the Triatome bugs, but can not be infected, and in consequence do not transmit the infection (like hens, birds, etc.)
- Vectors: Triatome bugs

The infected human population is divided into infected humans in the acute phase, $I_a$, and infected humans in the chronic phase, $I_c$. The infected transmitters, and infected vector populations are denoted by $I_t$, and $I_v$, respectively.

The dynamics of the disease is modeled by the following system of differential equations

\[
\begin{align*}
\frac{dI_a}{dt} &= p\mu_h I_c + \frac{b_h \beta_h}{N_h + N_t + N_{nt}} (N_h - I_a - I_c)I_v - (\gamma + \mu_h)I_a \\
\frac{dI_c}{dt} &= (1 - q)\gamma I_a - (\sigma + \mu_h)I_c \\
\frac{dI_t}{dt} &= \frac{b_h r_t \beta_t}{N_h + N_t + N_{nt}} (N_t - I_t)I_v - \mu_t I_t \\
\frac{dI_v}{dt} &= \frac{b_h \alpha_a I_a + b_h \alpha_c I_c + b_h r_t \alpha_t I_t}{N_h + N_t + N_{nt}} (N_v - I_v) - \mu_v I_v. 
\end{align*}
\]
In the model, $N_h$, $N_t$, $N_{nt}$, $N_v$ are the humans, transmitters, non-transmitters, and vector population sizes, respectively, $\mu_h$, $\mu_t$, and $\mu_v$ their corresponding mortality rates; $p$ denotes the proportion of newborns from chronic infected humans that are acute infected; $b_h$, $b_t$, $b_{nt}$ the biting rate of humans, transmitters and non-transmitters, respectively; $\beta_h$, $\beta_t$, $\alpha_a$, $\alpha_c$, and $\alpha_t$ the transmission probabilities from humans and transmitters to vector and vector to human and transmitters, respectively; $1/\gamma$ is the mean residence in the acute phase; $\sigma$ is the daily disease-induced death rate in the chronic phase; $q$ is the percentage of acute infected that are treated and cure.

2 Results

Using the next generation operator approach ([6]), we obtain the Basic reproductive number, $R_0$, in terms of the epidemiological and demographic parameters:

$$R_0 = \sqrt{\frac{b_h^2 \beta_h N_h N_v}{K \mu_v N^2} \left[ (\mu_h + \sigma) \alpha_a + (1 - q) \gamma \alpha_c \right] + \frac{b_t^2 \beta_t \alpha_t N_t N_v}{\mu_v \mu_t N^2}}, \quad (2)$$

where $K = (\gamma_h + \mu_h)(\sigma + \mu_h) - p(1-q)\gamma \mu_h$. The following result is established.

**Theorem.** The disease-free equilibrium, $E_0 = (0, 0, 0, 0)$, of model (1) is globally-asymptotically stable (GAS) if $R_0 \leq 1$. When $R_0 > 1$, $E_0$ becomes unstable, and emerges an endemic equilibrium $E_1 = (I^*_a, I^*_c, I^*_t, I^*_v)$ which is globally asymptotically stable.

We analyze the effect on $R_0$ and prevalence of the disease of the following control measures:

1. Reduction of the population of vectors
2. Early treatment of the disease
3. Use of non-transmitters

The results can be summarized as:

- Elimination of Triatomines is the control measure that has more impact on the diminishing of $R_0$. 

• Treatment of disease in the acute phase is effective if people is isolated from other transmitters.

• Zooprophylaxis has little impact on the reduction of the infection transmission, and in some cases can even increases it.

• A combination of elimination of Triatomines, early treatment, and keeping the transmitters animals out of the houses can be considered the most effective control measure.

References


