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# Persistence Aspects of an Epidemic Model of Chagas Disease*

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**Abstract**

This article concentrates on the study of persistence using an epidemic model of Chagas Disease.Threshold conditions of disease persistence have been derived.Results are developed by using the differential inequality theorem.

**Keywords and Phrases:** *Chagas disease, Mathematical model, Persistence, Vector transmission,Thresholds.*

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# 1. Introduction

Chagas disease is a major disease in South America,it is a disease of the poor [9] bibitemAber1980.It is very frustrating that in one of the most authoritative books on the subject of transmission of infectious diseases published, Trypanosoma cruzi is not mentioned [3].Chagas disease is one of the major helth problems in the South American continent which is caused by the protozoan parasite Trypanosoma cruzi.The population in danger reached around 64 million in 1981 (Zeledon and Rabinovich [18]) with around 24 million of infected individuals.Carlos Chagas [9] first identified this vertically transmitted disease in human.The parasite Trypanosoma cruzi also affects a large number of mammalian species.The life cycle involves transmission by blood-sucking reduviid insects that carry infective (metacyclic trypomasgate) forms of Trypanosoma cruzi in their fecal fluids and deposit them on the stein at the time of procuring a blood meal.After entering the body through skin lessions or mucosal surfaces, the parasite may penetrate a variety of host cells in whose cytoplasm it transform into the amastigote form, capable of intracellular replication.Eventually further intracellular transformation gives rise to a trymomatigote form commonly found in tissue fluids and in the blood, through which it can be disseminated to other cells and tissues or from where it could be ingested by an insect vector.

In endemic areas, individuals are usually infected during childhood.In cases in which the acute disease can develop in a few weeks and present with high fever.Mortality among acute patients is not common and is usually due to central nervous tissue or cardiac involvement.The chronic progresses relatively slowly,leading to death in a period of time ranging from a few years to decades.Cardiovascular disease develops in a majority of the patients and death from this condition is probably the most frequent cause of cardiovascular mortality in the South American continent.Some patients prevent serious gastrointestinal pathology, including massive enlargement of the esophagus and /or colon (megaviscera).

The disease has several forms, the two main ones living a long-term low-level chronic form and a clinical one (see Salazar et.al.[11]).In its clinical form, the disease is often fatal, usually, because of serious cardiac, lung and digestive tract autonomic degeneration (Billencourt et.al.[5],Billencourt[6]).The chronic form of the diseases eventually leads to similar complications and consequences as the clinical form, but it can last in a subclinical state for many years.

For detailed information on the epidemiology of the Chagas disease we refer to [7,8,12,16,17].There is no therapy to treat this chronic disease.Some medicines have been used in the treatment of this disease with little success but there is no established cure.Therefore it will be a great problem due to vertical and horizontal transmission by immigrants from endemic region to unaffected regions (Thesis et.al.[13]).So study of appropriate mathematical models is necessary for the encoming danger of this infection.Busenberg and Vargas [8] and Velasco-Hernandez [16,17] have analyzed such models. Busenberg and Vargas [8] have obtained partial analytical results on stability.They showed that there are exponentially stable solutions and no oscillatory phenomena are possible when approaching the endemic equilibrium.They consider the case where the initial population consists of only susceptible individuals and some chronically infected ones.Since $g$ is very small they assumed it to be zero.They showed that if the horizontal transmission is strong enough, even when the vector transmission is absent, the disease would reach an endemic level if silent infectives are introduced in an otherwise healthy population.Velasco-Hernandez [16] studied the infection under the assumption of a constant host population introducing vector population dynamics.In another paper [17] he analyzed the basic population dynamics and transmission mechanisms.He also investigated plausible density dependent mechanisms of fluctuations in bug populations.

Persistence theory has so far focused rather on ecological than epidemiological models.In the epidemiology of infectious disease persistence has two faces: persistence (or endemicity) of the disease and survival of the host population.In this paper we established conditions for the disease and /or the host population to persist, as well as for the disease to limit the growth of the population.We are mainly interested to study the persistence of the disease.A component $x(t)$ of a given ordinary differential equation system is said to be persistent (uniformly strongly) if there exists a constant $k > 0$ such that $lim_{t\to\propto} minx(t) > k$ whenever $x(0) > 0$ , whereas persistence (uniformly weak) means the existence of a constant $m > 0$ such that.A system is said to be uniformly strongly persistent if each component is uniformly persistent.Many results on persistence have been developed in ecological problems (see [10] and the references therein).It indicates the long-term survival of certain (if not all) components in ecological systems by suitably applying the results of dynamic systems theory.In the models of microparasites and macroparasites Anderson and May [1,2] have analyzed how the threshold phenomena for the persistence of epidemics are modified when population size is variable.Thieme and Castillo-

Chavez [15] studied uniform persistence for an HIV/AIDS model.Thieme [14] obtained persistence under relaxed point dissipativity in an epidemic model and derived conditions for both host and disease persistence and for host limitation by the disease.

In this paper we are able to show the persistence of the disease of an epidemic model analyzed previously in [8] by simply applying the theory of differential inequality.

In the next section we present the model.Section 3 deals with sufficient conditions for persistence. Finally we represent a brief discussion of our results together with some possible implications.

## 2. Mathematical Model

We consider the model of Chagas disease due to Busenberg and Vargas [8].We take a human host population of susceptible denoted by $S(t)$.In this model the infection has a chronic low level form and a clinical form, thus we obtain two distinct epidemiological classes $I_1$ and $I_2$, denoting the chronically ill and the clinically infected individuals respectely.

The dynamics obey the following equations:

$$\begin{aligned}
\frac{dS}{dt} &= (b-r-v)S + (b_1^{'}p_1 + c_1)I_1 + (b_2^{'}p_2 + c_2)I_2 - S\frac{k_1I_1 + k_2I_2}{N} \\
\frac{dI_1}{dt} &= (b_1^{'}q_1 - r_1^{'} - c_1)I_1 + vS + S\frac{k_1I_1 + k_2I_2}{N} - gl_1 \qquad (2.1) \\
\frac{dI_2}{dt} &= (b_2^{'}q_2 - r_2^{'} - c_2)I_2 + gl_1
\end{aligned}$$

Where

$$p_i + q_i = 1, \quad i = 1, 2$$

$$N = S + I_1 + I_1$$

Therefore

$$\frac{dN}{dt} = (b-r)S + (b_1^{'} - r_1^{'})I_1 + (b_2^{'} - r_2^{'})I_2$$

Here $b, b_1^{'}, b_2^{'}, r, r_1^{'}$ and $r_2^{'}$ denote the birth rates of susceptibles, the chronically and clinically ill individuals and their death rates respectively. $c_1, c_2, q_1$ and $q_2$ denote the cure rates of the chronically and the clinically ill individuals and

their probabilities of vertical transmission.$v$ is the vector transmission rate, $k_1$ and $k_2$ represent the rates of transmission through blood transfusion by which the chronically and clinically ill classes move to the clinically ill classes and $g$ is the rate by which the chronically ill move into the clinically ill class.For Chagas disease, $g$ would be very small since $\frac{1}{g}$ is the mean period of stay in the chronic classes which is estimated to be in the order of ten to fifteen years. So $g$ may be assumed to be zero.Also, in the case of Chagas disease, the chronically ill individuals are normally not detected and if detected, their probability of being cured is very low.All the parameters in system (2.1) are all constants. We are interested in studying this model where the population $N(t)$ is not stationary.

**Reformulation of the model:**

To proceed with the analysis, we consider the proportions of individuals in the epidemiological classes, namely

$$s = \frac{S}{N}, i_1 = \frac{I_1}{N}.i_2 = \frac{I_2}{N} \tag{2.2}$$

The dynamical system (2.1) becomes

$$\frac{ds}{dt} = (b-r-v)s+(b_1^{'}p_1+c_1)i_1+(b_2^{'}p_2+c_2)i_2-(b-r)s^2-(k_1+b_1^{'}-r_1^{'})si_1-(k_2+b_2^{'}-r_2^{'})si_2$$

$$\frac{di_1}{dt} = (b_1^{'}q_1-r_1^{'}-c_1-g)i_1+vs+(k_1-b+r)si_1+k_2si_2-(b_1^{'}-r_1^{'})i_2^1-(b_2^{'}-r_2^{'})i_1i_2 \tag{2.3}$$

$$\frac{di_2}{dt} = (b_2^{'}q_2 - r_2^{'} - c_2)i_2 + gi_1 - (b-r)si_2 - (b_1^{'} - r_1^{'})i_1i_2 - (b_2^{'} - r_2^{'})i_2^2$$

$$\frac{dN}{dt} = [(b-r)s + (b_1^{'} - r_1^{'})i_1 + (b_2^{'} - r_2^{'})i_2]N$$

Equations (2.2) imply

$$s + i_1 + i_2 = 1$$

The feasibility region is $\{B = (s, i_1, i_1) : s \geq 0, i_1 \geq 0, i_2 \geq 0, s + i_1 + i_2 = 1\}$

# 3. Persistence of Disease

In this section we shall show the disease persistence in the chronically and clinically ill classes under certain conditions.Also we shall derive the threshold

condition on limitation of these classes by the disease. There are different ways in which disease persistence can be interpreted in our model.We have chosen to call the disease to be persistent or endemic in the population, if the fraction of ill or infective individuals $i_1$ or $i_2$ is bounded away from zero.If the population dies out and the fraction of infectives remains bounded away from zero, we still say that the disease is persistent in the population. Through this section, for a real-valued function $h$ on $[t_0, \propto)$ we define

$$h_\propto = lim_{t\to\propto} inf h(t), h^\propto = lim_{t\to\propto} sup h(t)$$

**Theorem 3.1.** *Let* $q_1 > \frac{r_1^{'}+c_1+g}{b_1^{'}}, b < k_1 + r$ *and* $b_2^{'} < r_2^{'}$. *Then the disease is uniformly weakly persistent in so far as* $i_1^\propto = lim_{t\to\propto} sup i_1(t) \geq \epsilon$ *where constant* $\epsilon > 0$ *being independent of the initial data, provided that* $i_1(0) > 0$.
**Proof.** Consider the $i_1$ equation in (2.3)

$$\frac{di_1}{dt} = (b_1^{'} - r_1^{'} - c_1 - g)i_1 + vs + (k_1 - b + r)si_1 + k_2 si_2 - (b_1^{'} - r_1^{'})i_1^2 - (b_2^{'} - r_2^{'})i_1 i_2$$

Since $b < k_1 + r, b_2^{'} < r_2^{'}$

$$\frac{di_1}{dt} \geq (b_1^{'} q_1 - r_1^{'} - c_1 - g)i_1 - (b_1^{'} - r_1^{'})i_1^2$$

which implies

$$\frac{\frac{di_1}{dt}}{i_1} \geq (b_1^{'} q_1 - r_1^{'} - c_1 - g)i_1 - (b_1^{'} - r_1^{'})i_1$$

Hence

$$lim_{t\to\propto} inf \frac{\frac{di_1}{dt}}{i_1} \geq (b_1^{'} - r_1^{'} - c_1 - g) - (b_1^{'} - r_1^{'})i_1^\propto$$

If possible, let

$$i_1^\propto < \frac{(b_1^{'} q_1 - r_1^{'} - c_1 - g)}{(b_1^{'} - r_1^{'})}$$

We have $lim_{t\to\propto} inf \frac{\frac{di_1}{dt}}{i_1} > 0$ which implies that $i_1(t) \to \propto, t \to \propto$ in contradiction to the fact that $i_2$ is bounded by one.Therefore we must have

$$i_1^\propto \geq \frac{(b_1^{'} q_1 - r_1^{'} - c_1 - g)}{(b_1^{'} - r_1^{'})}$$

**Theorem 3.2** *Let the assumptions of Theorem 3.1 hold. Then the disease is uniformly strongly persistent in so far as* $i_{1\propto} = lim_{t\to\propto} inf i_1(t) \geq \epsilon$ *with a constant* $\epsilon$ *being independent of the initial data, provided that* $i_1(0) > 0$.
**Proof.** Clearly $\frac{di_1}{dt} \geq i_1[b_1^{'}q_1 - r_1^{'} - c_1 - g - (b_1^{'} - r_1^{'})i_1]$ as $t \geq t_0$ becomes large enough.
Note that $b_1^{'}q_1 - r_1^{'} - c_1 - g > 0$ and $b_1^{'} > r_1^{'}$.
From the comparison theorem we have

$$lim_{t\to\propto} i_1(t) > \frac{(b_1^{'}q_1 - r_1^{'} - c_1 - g)}{(b_1^{'} - r_1^{'})}$$

For any $i_1(t)$ with $i_1(0) > 0$.

**Theorem 3.3** *Let* $q_2 > \frac{(r_2^{'}+c_2)}{b_2^{'}}, b < r$ *and* $b_1^{'} < r_1^{'}$. *Then the disease is uniformly weakly persistent in so far as* $i_2^{\propto} = lim_{t\to\propto} supi_2(t) \geq C$ *where constant* $C > 0$ *being independent of the initial data provided that* $i_2(0) > 0$.
**Proof.** The proof of Theorem 3.3 is similar to that of Theorem 3.1 and is therefore omitted.

**Theorem 3.4** *Let the assumptions of Theorem 3.3 hold.Then the disease is uniformly strongly persistent in so far as* $i_{2\propto} = lim_{t\to\propto} inf i_2(t) \geq C$ *with constant* $C > 0$ *being independent of the initial data, provided that* $i_2(0) > 0$.
**Proof.** The proof of Theorem 3.4 is similar to that of Theorem 3.2 and is omitted.

**Theorem 3.5** *Let* $b < r, r_1^{'} > b_1^{'} + k_1$ *and* $r_2^{'} < b_2^{'} < \frac{(r_2^{'}+c_2)}{q_2}$. *Then* $i_{1\propto} = lim_{t\to\propto} inf i_1(t) \leq \eta$ *with constant* $\eta > 0$ *not depending on the initial data provided that* $i_1(0) > 0$.
**Proof.** Since $s, i_1, i_2 <\leq 1$ we have

$$\frac{di_1}{dt} \leq g + r - b + r_1^{'} - b_1^{'} - (r_2^{'} + c_2 - b_2^{'}q_2)i_2$$

From which it follows by a standard differential inequality argument [4] that

$$i_2(t) \leq \frac{g + r - b + r_1^{'} - b_1^{'}}{r_2^{'} + c_2 - b_2^{'}q_2} + [i_2(0) - \frac{g + r - b + r_1^{'} - b_1^{'}}{r_2^{'} + c_2 - b_2^{'}q_2}]e^{-(r_2^{'}+c_2-b_2^{'}q_2)t}$$

Thus

$$lim_{t\to\propto} supi_2(t) < \frac{g + r - b + r_1^{'} - b_1^{'}}{r_2^{'} + c_2 - b_2^{'}q_2} = m$$

Again as $b < r$, we have

$$\frac{ds}{dt} \geq (b - r - v)s - (k_1 + b_1^{'} - r_1^{'})si_1 - (k_2 + b_2^{'} - r_2^{'})si_2$$

Hence

$$lim_{t\to\propto} inf \frac{\frac{ds}{dt}}{s} \geq (b - r - v) + (r_1^{'} - b_1^{'} - k_1)i_{1\propto} - (b_2^{'} - r_2^{'} + k_2)m$$

If possible, let $i_{1\propto} > \frac{r+v-b+(b_2^{'}-r_2^{'}+k_2)m}{r_1^{'}-b_1^{'}-k_1}$

We have $lim_{t\to\propto} inf \frac{\frac{ds}{dt}}{s} > 0$ which implies that $s(t) \to\propto, t \to\propto$ in contradiction to the fact that $s$ is bounded by one.

Therefore we must have

$$i_{1\propto} \leq \frac{r + v - b + (b_2^{'} - r_2^{'} + k_2)m}{r_1^{'} - b_1^{'} - k_1}$$

**Theorem 3.6** *Let* $b < r, r_1^{'} < b_1^{'} < \frac{r_1^{'}+c_1+g}{q_1}$ *and* $k_2 + b_2^{'} < r_2^{'}$. *Then* $i_{2\propto} = lim_{t\to\propto} inf i_2(t) \leq \eta$ *with constant* $\eta > 0$ *not depending on the initial data provided* $i_2(0) > 0$.

**Proof.** The proof of Theorem 3.6 is similar to that of Theorem 3.5 and is therefore omitted.

**Theorem 3.7** *Let* $0 < b - r < v$ *and* $r_i^{'} > k_i + b_i^{'}, i = 1, 2$. *Then* $i_{j\alpha} = lim_{t\to\propto} inf i_j(t) \leq \epsilon_j, \quad j = 1, 2$ *with constants* $\epsilon_j, \quad j = 1, 2$ *not depending on the initial data provided that* $i_j(0) > 0, j = 1, 2$.

**Proof.** Proof is obvious.

# 4. Discussion

Charles Chagas identified triatominae insects as carriers of the parasite around 1909 in the state of Minas Gerai's, Brazil. In this paper we have studied the basic population dynamics and transmission mechanisms.We observed that the disease persistent for chronically ill individuals depend on the magnitude of birth rate of susecptable and clinically ill individuals and these are in probability of vertical transmission of the chronically ill individuals.It is to be noted that uniform weak persistence implies persistence in our system.Under

the stated conditions in Theorem 3.1, the infected population does not go to extinction.

Again it is observed that when the probability of vertical transmission of the clinically ill individuals exceeds a certain lower threshold value and the birth rates of susceptible and chronically ill individuals are less than their corresponding death rates then the disease is persistent in clinically ill individuals.Lastly we have obtained the threshold conditions on the disease persistence in the two distinct infective classes, which are mainly dependent on a certain range of the parameters namely the birth rates of susceptibles, the chronically and the clinically ill individuals, respectively.

We mention some of the factors important in disease transmission that have not been considered here. Perhaps the most important one is the neglation of host age-structure. Also vector transmission is assumed to be constant in the model system although it depends on so many factors like biting rate, probability of producing an infection in the host, the ratio of vector members of host numbers and the total vector population.It would be interesting to know the persistence behaviour for a constant vector transmission and also age/stage structure of the vector population.

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