Dynamics of Cutaneous Leishmaniasis Infection in the Presence of Bird Reservoirs as a Tool for Vector Surveillance in Ecuador

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Abstract

Leishmaniasis is a neglected tropical disease, transmitted by species of phlebotomus vectors of the genus *Lutzomyia* and is responsible for over 1500 yearly cases in Ecuador. Vector collection studies in Ecuador suggest an association between the ecological diversity of an ecosystem, presence of reservoir host and the abundance of sand fly species. Data collected in a Coastal community suggest that birds are the preferred host when compared to humans and other mammals. There has been limited reporting of leishmaniasis cases as it is non-fatal. Hence, actual incidence of the disease is

unknown. A vector-host infection model was developed and analyzed to understand the complexity of the transmission dynamics in this coastal ecosystem and to estimate its transmission rates. The results of this study highlight the mechanisms responsible for the observed patterns of Leishmaniasis and the need for effective vector surveillance in Ecuador.

1 Introduction

Leishmaniasis is a disease caused by a protozoan that is transmitted by sand flies of the subfamily Phlebotominae of the genus *Lutzomyia* in the Americas. The sand flies females are infected when bite a natural reservoir or human with disease and transmit the parasite in the same way [14]. Ecuador is a country located in north-west South America that is extremely ecological diverse. Ecuador's ecology range from dry forest in the coast, cloudy forest in the Andes to tropical rain forest in the Amazon lowlands [4].

The first case of Leishmaniasis in Ecuador was reported in 1920. For many years the clinical diagnosis was the only method to confirm cases and report them. Cutaneous Leishmaniasis is a public health problem in Ecuador because of its wide distribution, mainly in rural areas in three regions of the country (coast, highlands and lowland). It is present in 23 out of the 24 provinces [21]. Few studies have been conducted to determine possible reservoirs of the leishmaniasis and where it has been reported, in an endemic area, three mammalian species with the parasite [12].

Phlebotomine are insects of the family Psychodidae in the order Diptera, approximately 800 sand fly species have been recorded; of these, fewer than 10% have been confirmed as vector species of leishmaniasis. Serological studies in the Pacific and Andean region, determined the infection in dogs with the same human strain isolated in the respective regions, but nevertheless the study does not distinguish whether dogs are incidental or reservoir hosts, dogs seems to be a victim-host as humans are [4]. For instance, studies in

Brazil indicated, as a potential risk factor for visceral leishmaniasis, the proximity to the breeding of chickens, although transmission between humans and chickens has not been explained, since some birds do not have the ability to keep the parasite on your system [17].

New molecular techniques allowed to estimate the rates of contact between vectors and hosts of this form to determine the preference that influence transmission of the parasite [20]. In Ecuador, blood meal sources in hyper endemic area in the coastal region have been identified using molecular analysis to register preference meals identified on sand flies on birds [2].

The first mathematical model focusing on epidemiology was presented by Ronald Ross in 1902. It explained the malaria life cycle and the relationship between its vector and *Trypanosoma* parasite. Kermack and McKendrick formulated the famous theorem of the threshold ($\mathcal{R}_0$), that states that an epidemic is originated by the introduction of infected individuals into a completely susceptible population [15].

Since re-emergent infectious diseases increase the frequency of outbreaks, it is of an interest to gain understanding of the dynamics of these diseases. In the development of mathematical models for infectious diseases, the model structure is determined by their biological assumptions, which influence the predictability potential of the model [22].

Mathematical models can be used to design control strategies, taking as a reference the decrease of the basic reproductive number ($\mathcal{R}_0$), which is the average number of secondary cases of infection as a result of the introduction of a primary infection into a completely

susceptible population. Finding this number allows to set the threshold of a disease outbreak [8].

The limited studies (five published works) in the area of mathematical modelling focus on the study of transmission dynamics of Cutaneous Leishmaniasis (CL). Chaves *et al.*, [8] suggest models to describe the dynamics of infection among vectors, humans and consider dogs and donkeys as reservoirs of the parasite. In another work, Chaves [5], analyzed the relations in the changes produced by humans and deforestation, and suggested that deforestation increases the risk factors for infection by C.L. A simple mathematical framework was designed to illustrate limitations in the ecological knowledge of the transmission of Leishmania. That are required to understand the best form the life cycle of the parasite ([7]). The same author developed another model for the transmission of C.L. where he included incidental hosts for the parasite, and related it to a species that acts as a reservoir [6]. In The dynamics of the infection in three hosts in two endemic localities for C.L. was analized and the differences in the value of ($\mathcal{R}_0$) at these locations were determined [19]. According to the manual of procedures of the system of surveillance epidemiologic (SIVE in spanish) of the Ministry of Public Health, recommended research of the field to determine possible foci disease once is has reported a case of leishmaniasis [16]. As regards the control, the mentioned manual recommends spraying of dwellings of infected patients and suggests performing a study of possible reservoirs, while to date this has been implemented in the country. These reasons are which lead us to ask ourselves the following questions: What is the role of birds reservoir in the transmission dynamics of ACL?

What are the levels of underreporting of leishmaniasis?

In this study we present a mathematical model to understand the dynamics of Cutaneous Leishmaniasis infections in the presence of birds. The aim of this work is to develop a mathematical mechanism to estimate the rates of parasite infection in sand flies, using the Centers for Disease Control and Prevention (CDC) trap sampling, which can be used as an early warning system for health authorities to take specific actions on vector control as a response to Leishmaniasis outbreaks.

2 Methods

2.1 Data Sources

In Valle Hermoso, Santo Domingo de Los Tsachilas, Ecuador, monitoring of sand flies was conducted. Phlebotominos were collected during the dry season, in July 2013 and during the rain season, in March 2014. The samples were captured with the Centers for Disease Control and Prevention (CDC) miniature light traps. The traps were placed in six transects, 150 meters apart. Specimens collected were killed and stored at -20°C and transported to the laboratory. Specimens were identified, counted and classified into three groups: blood fed, unfed and gravid females. Females whit blood meals were easily recognized by the presence of engorded abdomens. Females abdomens were dissected for DNA analyses. DNA was extracted, amplified and sequenced to identify the potential food source and identify parasitic infection in each sand fly [2]. Epidemiological data of

the Province were obtained from 2009 to 2011, from the Ministry of Public Health (Fig 1).

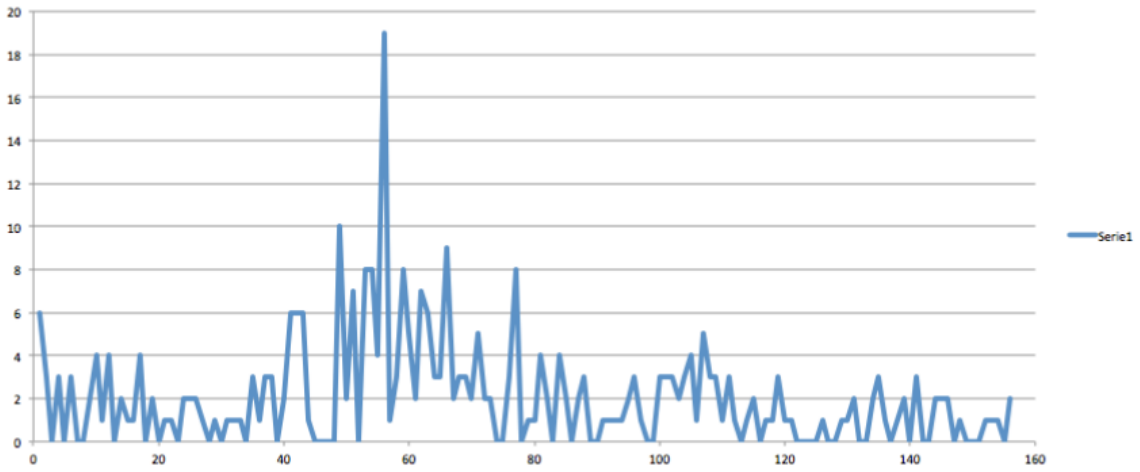

Figure 1: Leishmaniasis incidence by epidemiological week in the Santo Domingo de Los Tsachilas from 2009 to 2011. Data from Ministry of Health

2.2 Model Description

An SIR epidemiological framework is proposed to model zoonotic parasite transmission in a community of two host with a single vector. The model incorporates parasite transmission between the vector, *Lutzomyia* sp., and two categories of hosts: A preferred by the vector (Birds) an the Human population like an alternative host. Our model adds an additional compartment that corresponds to the reported cases, considering that in rural areas, there is no access to conventional treatment and traditional and ancestral knowledge methods are used [4]. The human host is divided into classes of susceptible (S_{h_1}), infected (I_{h_1}),

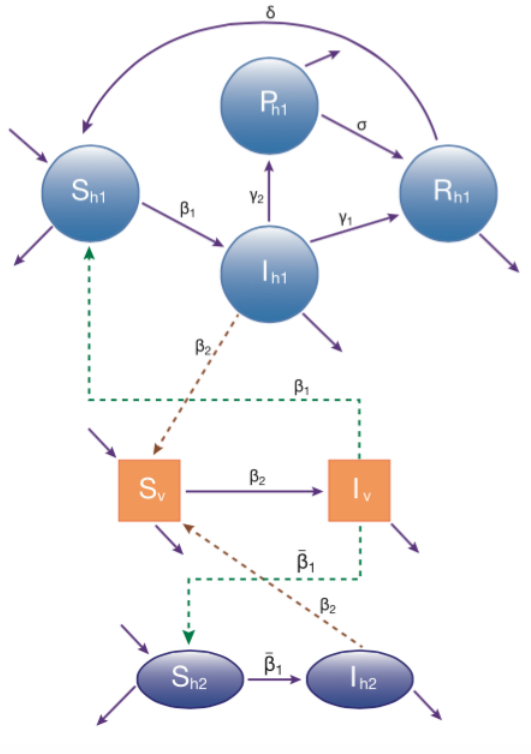

Figure 2: Flowchart for the general model

infected reported (P_{h_1}) and recovered (R_{h_1}) individuals. The size of the total populations is $N_{h_1} = S_{h_1} + I_{h_1} + P_{h_1} + R_{h_1}$. Both vector and birds hosts are divided into classes of susceptible (S) and infected (I) individuals so the total population size is N = S + I. The variables for the vector are indicated with subscript v and birds with subscript h_2.

The model is given by the following ordinal differential equations:

$$
\textbf{Humans:}\begin{cases}
S'_{h_1} &= \mu_{h_1}N_{h_1} - \left(\dfrac{b\beta_1 I_v}{\alpha_v N_{h_2}+N_{h_1}}\right)S_{h_1} + \delta R_{h_1} - \mu_{h_1}S_{h_1} \\
I'_{h_1} &= \left(\dfrac{b\beta_1 I_v}{\alpha_v N_{h_2}+N_{h_1}}\right)S_{h_1} - \gamma_1 I_{h_1} - \gamma_2 I_{h_1} - \mu_{h_1}I_{h_1} \\
P'_{h_1} &= \gamma_2 I_{h_1} - \sigma P_{h_1} - \mu_{h_1}P_{h_1} \\
R'_{h_1} &= \gamma_1 I_{h_1} + \sigma P_{h_1} - \delta R_{h_1} - \mu_{h_1}R_{h_1}
\end{cases}
$$

$$
\textbf{Vectors:}\begin{cases}
S'_v &= \mu_v N_v - \dfrac{b\beta_2(\alpha_v I_{h_2}+I_{h_1})}{\alpha_v N_{h_2}+N_{h_1}}S_v - \mu_v S_v \\
I'_v &= \dfrac{b\beta_2(\alpha_v I_{h_2}+I_{h_1})}{\alpha_v N_{h_2}+N_{h_1}}S_v - \mu_v I_v
\end{cases}
$$

$$
\textbf{Birds:}\begin{cases}
S'_{h_2} &= \mu_{h_2}N_{h_2} - \left(\dfrac{b\tilde{\beta}_1 I_v}{\alpha_v N_{h_2}+N_{h_1}}\right)\alpha_v S_{h_2} - \mu_{h_2}S_{h_2} \\
I'_{h_2} &= \left(\dfrac{b\tilde{\beta}_1 I_v}{\alpha_v N_{h_2}+N_{h_1}}\right)\alpha_v S_{h_2} - \mu_{h_2}I_{h_2}
\end{cases}
$$

Assumptions

The model of the Cutaneous Leishmaniasis transmission dynamics is divided into three groups: Humans, Vector and Birds. Some assumptions are required in each group for the construction of the model.

Human population:

The sand flies bite at a constant rate (b). The people recover from the infected population to become susceptible again. Not all the people are reporting the disease. The people infected don‘t die due to the disease. Population remains constant.

Vector:

Sand flies bite humans and birds at different rates. They can infected humans and birds. The birth and death rates as a result are equal. Sand flies can get infected from humans and birds. Sand flies don‘t die of the disease. Population remains constant.

Birds:

Sand flies bite at a constant rate (b). Birds can get infected due to bites from infected sand flies. Infected birds can transmit the infection to sand flies and there is no disease induced deaths in infected birds. Birds population remains constant.

Table 1: Definition of state variables

Variable	Variable description
N_{h_1}	Total Human Population
S_{h_1}	Susceptible Human Population
I_{h_1}	Infected Human Population
P_1	Reported Cases
R_{h_1}	Recovered Human Population
N_v	Sand Flies Population
S_v	Susceptible Sand Flies Population
I_v	Infected Sand Flies Population
N_{h_2}	Birds Population
S_{h_2}	Susceptible Birds Population
I_{h_2}	Infected Birds Population

Table 2: Parameters definitions and values

Parameter	Parameter Description	Value	Units	Source
μ_{h1}	Human Natural Mortality Rate in the Coastal	26973.5	days	[13]
b	Biting Rate	$0,2856$	day^{-1}	[11]
β_1	Probability Human Host Transmission to Vector	0,08223		[2]
$\tilde{\beta}_1$	Probability Birds Transmission to Vector	0,31399		[2]
β_2	Host to Vector Transmission Probability	0.25		[3]
α_v	Feeding for Preferred Host	0,7924		[2]
δ_c	Rate Immunity Lost	0.0033	day^{-1}	[3]
$\frac{1}{\gamma_1}$	Mean Infection Period	$\frac{1}{450}$	day^{-1}	[9]
$\frac{1}{\gamma_2}$	Mean Incubation Period	$\frac{1}{15}$	days^{-1}	[18]
$\frac{1}{\sigma}$	Rate at Which Confirm Individuals, Move to Recover	$\frac{1}{45}$	day^{-1}	[9]

Simulations will be performed using the parameter values shown in Table 2.

3 Mathematical analysis

3.1 Basic Reproductive Number

It is clear that the Disease Equilibrium Point is

$$E_0 = (N_{h_1}, 0, 0, 0, N_v, 0, N_{h_2}, 0).$$

In order to calculate the basic reproductive number we consider the vector of new infection rates and the vector of all other rates

$$\mathcal{F} = \begin{pmatrix} \left(\dfrac{b\beta_1 I_v}{\alpha_v N_{h_2} + N_{h_1}}\right) S_{h_1} \\ 0 \\ \dfrac{b\beta_2(\alpha_v I_{h_2} + I_{h_1})}{\alpha_v N_{h_2} + N_{h_1}} S_v \\ \left(\dfrac{b\tilde{\beta}_1 \alpha_v I_v}{\alpha_v N_{h_2} + N_{h_1}}\right) S_{h_2} \end{pmatrix} \quad \text{and} \quad \mathcal{V} = \begin{pmatrix} (\gamma_1 + \gamma_2 + \mu_{h_1}) I_{h_1} \\ -\gamma_2 I_{h_1} + (\sigma + \mu_{h_1}) P_{h_1} \\ \mu_v I_v \\ \mu_{h_2} I_{h_2} \end{pmatrix}.$$

Then the derivatives of these vector fields are

$$D\mathcal{F} = \begin{pmatrix} 0 & 0 & \dfrac{b\beta_1 S_{h_1}}{\alpha_v N_{h_2} + N_{h_1}} & 0 \\ 0 & 0 & 0 & 0 \\ \dfrac{b\beta_2 S_v}{\alpha_v N_{h_2} + N_{h_1}} & 0 & 0 & \dfrac{b\beta_2 \alpha_v S_v}{\alpha_v N_{h_2} + N_{h_1}} \\ 0 & 0 & \dfrac{b\tilde{\beta}_1 \alpha_v S_{h_2}}{\alpha_v N_{h_2} + N_{h_1}} & 0 \end{pmatrix}$$

$$D\mathcal{V} = \begin{pmatrix} \gamma_1 + \gamma_2 + \mu_{h_1} & 0 & 0 & 0 \\ -\gamma_2 & \sigma + \mu_{h_1} & 0 & 0 \\ 0 & 0 & \mu_v & 0 \\ 0 & 0 & 0 & \mu_{h_2} \end{pmatrix}$$

Now we evaluate in the disease free equilibrium point and we obtain:

$$F = \begin{pmatrix} 0 & 0 & \frac{b\beta_1 N_{h_1}}{\alpha_v N_{h_2} + N_{h_1}} & 0 \\ 0 & 0 & 0 & 0 \\ \frac{b\beta_2 N_v}{\alpha_v N_{h_2} + N_{h_1}} & 0 & 0 & \frac{b\beta_2 \alpha_v N_v}{\alpha_v N_{h_2} + N_{h_1}} \\ [15pt]0 & 0 & \frac{b\tilde{\beta_1} \alpha_v N_{h_2}}{\alpha_v N_{h_2} + N_{h_1}} & 0 \end{pmatrix}$$

and

$$V = \begin{pmatrix} \gamma_1 + \gamma_2 + \mu_{h_1} & 0 & 0 & 0 \\ -\gamma_2 & \sigma + \mu_{h_1} & 0 & 0 \\ 0 & 0 & \mu_v & 0 \\ 0 & 0 & 0 & \mu_{h_2} \end{pmatrix}$$

It is easy to see that

$$V^{-1} = \begin{pmatrix} \frac{1}{\gamma_1 + \gamma_2 + \mu_{h_1}} & 0 & 0 & 0 \\ \frac{\gamma_2}{(\gamma_1 + \gamma_2 + \mu_{h_1})(\sigma + \mu_{h_1})} & \frac{1}{\sigma + \mu_{h_1}} & 0 & 0 \\ 0 & 0 & \frac{1}{\mu_v} & 0 \\ 0 & 0 & 0 & \frac{1}{\mu_{h_2}} \end{pmatrix},$$

then the next generation matrix is

$$FV^{-1} = \begin{pmatrix} 0 & 0 & \frac{b\beta_1 N_{h_1}}{(\alpha_v N_{h_2} + N_{h_1})\mu_v} & 0 \\ 0 & 0 & 0 & 0 \\ \frac{b\beta_2 N_v}{(\alpha_v N_{h_2} + N_{h_1})(\gamma_1 + \gamma_2 + \mu_{h_1})} & 0 & 0 & \frac{b\beta_2 \alpha_v N_v}{(\alpha_v N_{h_2} + N_{h_1})\mu_{h_2}} \\ 0 & 0 & \frac{b\tilde{\beta_1} \alpha_v N_{h_2}}{(\alpha_v N_{h_2} + N_{h_1})\mu_v} & 0 \end{pmatrix}$$

Now, let's calculate the eigenvalues of FV^{-1}

$$|FV^{-1} - \lambda I| = \begin{vmatrix} -\lambda & 0 & \frac{b\beta_1 N_{h_1}}{(\alpha_v N_{h_2} + N_{h_1})\mu_v} & 0 \\ 0 & -\lambda & 0 & 0 \\ \frac{b\beta_2 N_v}{(\alpha_v N_{h_2} + N_{h_1})(\gamma_1 + \gamma_2 + \mu_{h_1})} & 0 & -\lambda & \frac{b\beta_2 \alpha_v N_v}{(\alpha_v N_{h_2} + N_{h_1})\mu_{h_2}} \\ 0 & 0 & \frac{b\tilde{\beta_1} \alpha_v N_{h_2}}{(\alpha_v N_{h_2} + N_{h_1})\mu_v} & -\lambda \end{vmatrix}$$

$$= -\lambda \begin{vmatrix} -\lambda & \frac{b\beta_1 N_{h_1}}{(\alpha_v N_{h_2} + N_{h_1})\mu_v} & 0 \\ \frac{b\beta_2 N_v}{(\alpha_v N_{h_2} + N_{h_1})(\gamma_1 + \gamma_2 + \mu_{h_1})} & -\lambda & \frac{b\beta_2 \alpha_v N_v}{(\alpha_v N_{h_2} + N_{h_1})\mu_{h_2}} \\ 0 & \frac{b\tilde{\beta_1} \alpha_v N_{h_2}}{(\alpha_v N_{h_2} + N_{h_1})\mu_v} & -\lambda \end{vmatrix}$$

$$= -\lambda \left\{ -\lambda^3 + \lambda \left(\frac{b\beta_2 N_v}{(\alpha_v N_{h_2} + N_{h_1})(\gamma_1 + \gamma_2 + \mu_{h_1})} \right) \left(\frac{b\beta_1 N_{h_1}}{(\alpha_v N_{h_2} + N_{h_1})\mu_v} \right) + \right.$$

$$\left. + \left(\frac{b\tilde{\beta_1} \alpha_v N_{h_2}}{(\alpha_v N_{h_2} + N_{h_1})\mu_v} \right) \left(\frac{b\beta_2 \alpha_v N_v}{(\alpha_v N_{h_2} + N_{h_1})\mu_{h_2}} \right) \right\}$$

$$= \lambda^2 \left\{ \lambda^2 - \left(\frac{b\beta_1 N_{h_1}}{(\alpha_v N_{h_2} + N_{h_1})\mu_v} \right) \left(\frac{b\beta_2 N_v}{(\alpha_v N_{h_2} + N_{h_1})(\gamma_1 + \gamma_2 + \mu_{h_1})} \right) \right.$$

$$\left. - \left(\frac{b\tilde{\beta_1} \alpha_v N_{h_2}}{(\alpha_v N_{h_2} + N_{h_1})\mu_v} \right) \left(\frac{b\beta_2 \alpha_v N_v}{(\alpha_v N_{h_2} + N_{h_1})\mu_{h_2}} \right) \right\}$$

Then the basic reproductive number for this model is

$$\begin{aligned} R_0^2 &= \left(\frac{b\beta_1 N_{h_1}}{(\alpha_v N_{h_2}+N_{h_1})\mu_v}\right)\left(\frac{b\beta_2 N_v}{(\alpha_v N_{h_2}+N_{h_1})(\gamma_1+\gamma_2+\mu_{h_1})}\right)+ \\ &+\left(\frac{b\tilde{\beta_1}\alpha_v N_{h_2}}{(\alpha_v N_{h_2}+N_{h_1})\mu_v}\right)\left(\frac{b\beta_2\alpha_v N_v}{(\alpha_v N_{h_2}+N_{h_1})\mu_{h_2}}\right) \end{aligned} \tag{1}$$

4 Results

In order to analyze the behaviour of the model, we simulate various scenarios of leishmaniasis infection. The data used for these simulations are shown in the table 1.

4.1 Parameter Estimation

Populations

It is assumed that all the populations human, vectors and birds size are constant. According to the National Institute of Statistics and Census, INEC (for its acronym in spanish), the agency responsible for the statistical population. The town of Valle Hermoso had 10000 inhabitants in the year 2010, date of the last survey registered.

Feeding for Preferred Host (α_v)

A study was conducted in Valle Hermoso, Los Tsachilas province, Ecuador, to determine the power supply with blood for phlebotomine sand fly. Valle Hermoso is an area hyper endemic for leishmaniasis. A total of 442 female sand flies were collected and classified

as non-engorded and engorged. The 106 engorded females were identified morphologically and selected for blood meal identification by PCR technique. 84 individuals of these were positive for blood birds. We estimated the proportion of blood meal sand flies in 0.7924.

Probability Human Host Transmission to Vector (β_1)

In such study we found that in the 106 samples of engorded females, 42 were positive for leishmaniasis and 22 were positive for blood of mammals. Thus likelihood that a sand fly is infected by bite on humans is 0.08223

Probability Birds Transmission to Vector ($\tilde{\beta_1}$)

In the same study we found that the 106 samples of engorded females, 84 were positive for blood birds and 42 were positive for leishmaniasis; by what we estimate the likelihood that a sand fly is infected by bites on birds is 0.3139.

4.2 Simulations

Our model has been simulated in three different cases. In the first case we consider the model in the absence of reservoir (no birds). In the second case, we assume that the reservoir population size is very large. Finally, in the third case, it is assumed that both the vector and reservoir population sizes are very low, as it may happen in the time dry conditions.

Simulations show the existence of a peak in the epidemiological week 45. Also, a correlation between reported cases and infected human population has been found, see figure 4.2.

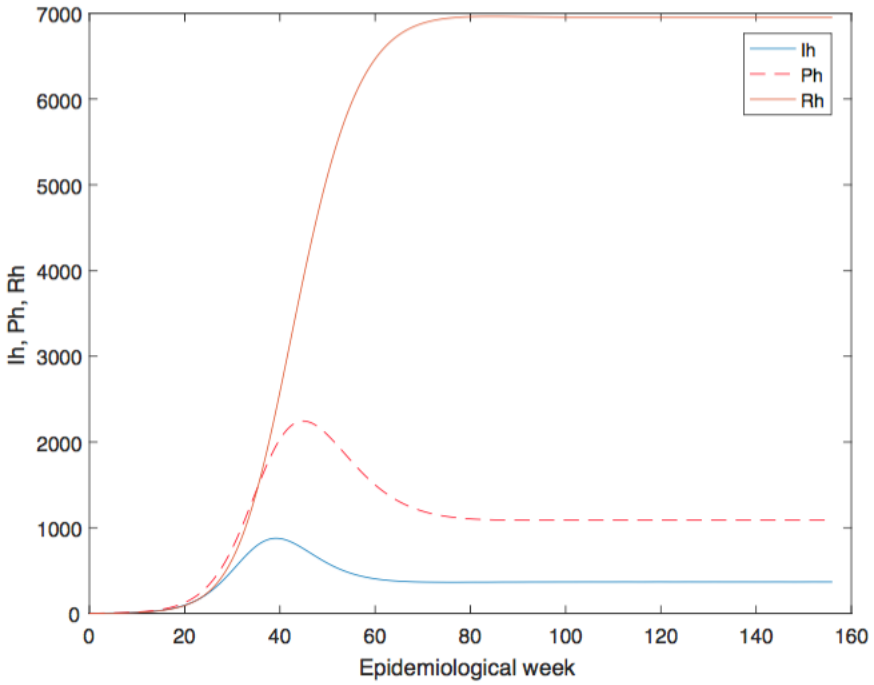

Figure 3: Simulation of the general model with out variations in the parameters

Figure 4 presents the prevalence of leishmaniasis cases in the province. It shows that there is an outbreak of the disease in the weeks 55 to 60.

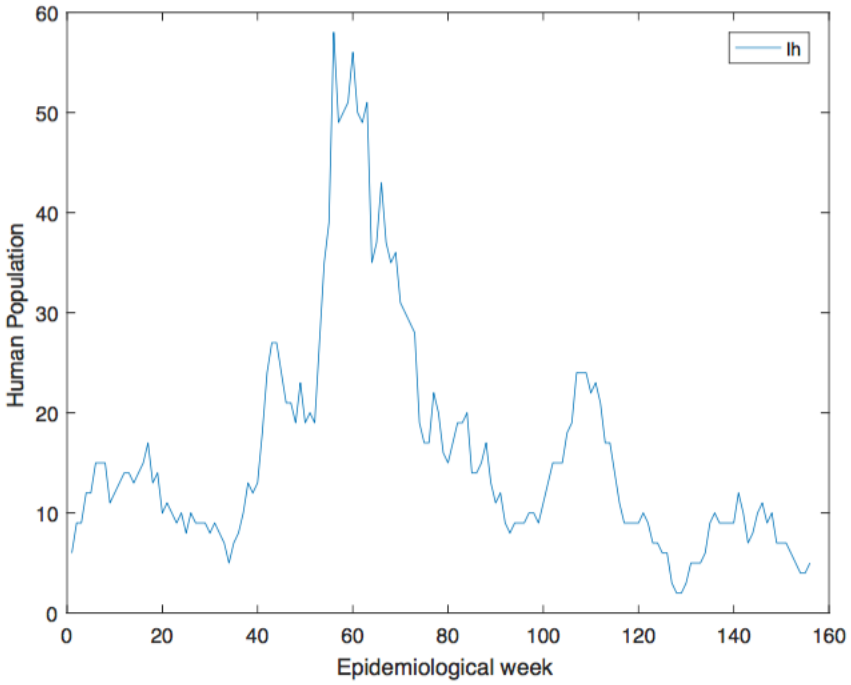

Figure 4: Leishmaniasis prevalence in the province from 2009 to 2011. Data from Ministry of Health

In the initial state we can see the total population of birds step of being susceptible to infected in week 50, while the human subject for the week 50, 80 % population is infected.

Case 1

In this case it was considered a non-existent population of birds.

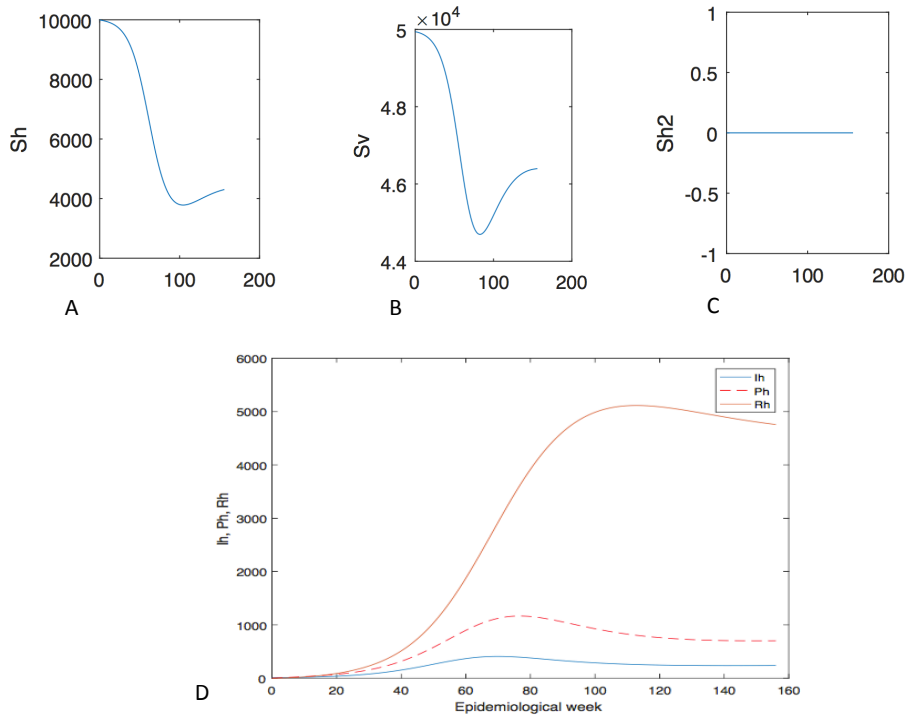

Figure 5: Simulation case 1, system with out birds

In the figure A we can see that there is 60% of the infected human population in the 100 epidemiological week. Figure B shows that the population of susceptible vectors descends rapidly towards the week 80. Figure C indicates the absence of birds. In Figure D, there is a shift of the peak of the epidemic for the week 80 and the number of recovered decreases compared to the initial state.

Case 2

In this case it was considered a high population of birds.

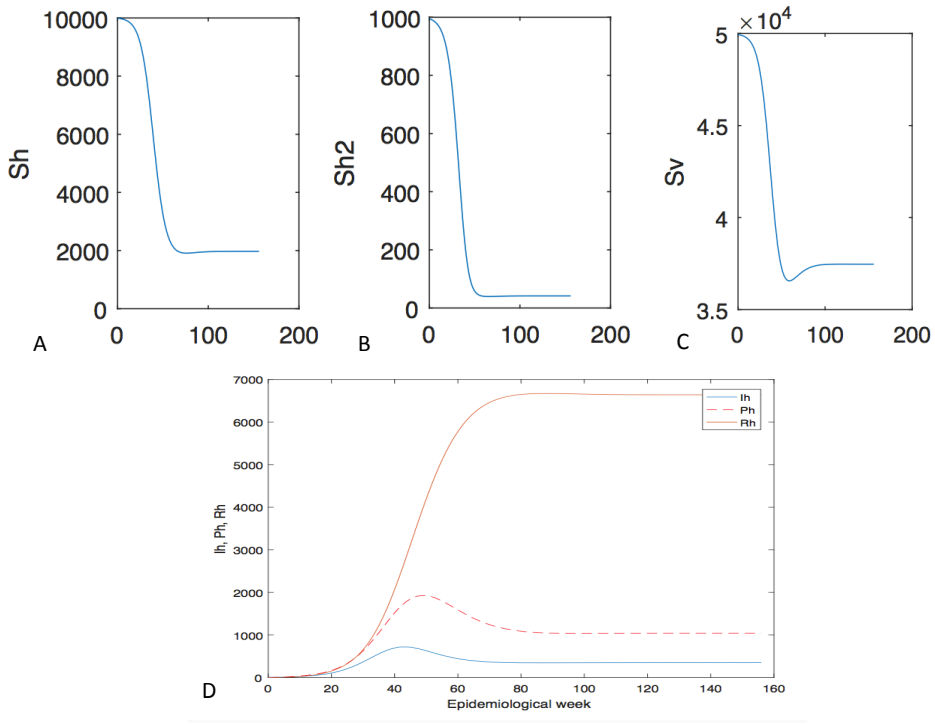

Figure 6: Simulation case 2, system with high birds population

In the figure A can see that there is 80% of the infected human population in the 75 epidemiological weeks. Figure B shows that the population of susceptible vectors descends rapidly towards the week 50. Figure C indicates the bird population decreased in the same week. In Figure D, there is an epidemic, little peak in the week 50.

Case 3

In case 3 is considered a low population of vectors and birds, which is what is expected to happen during the dry season.

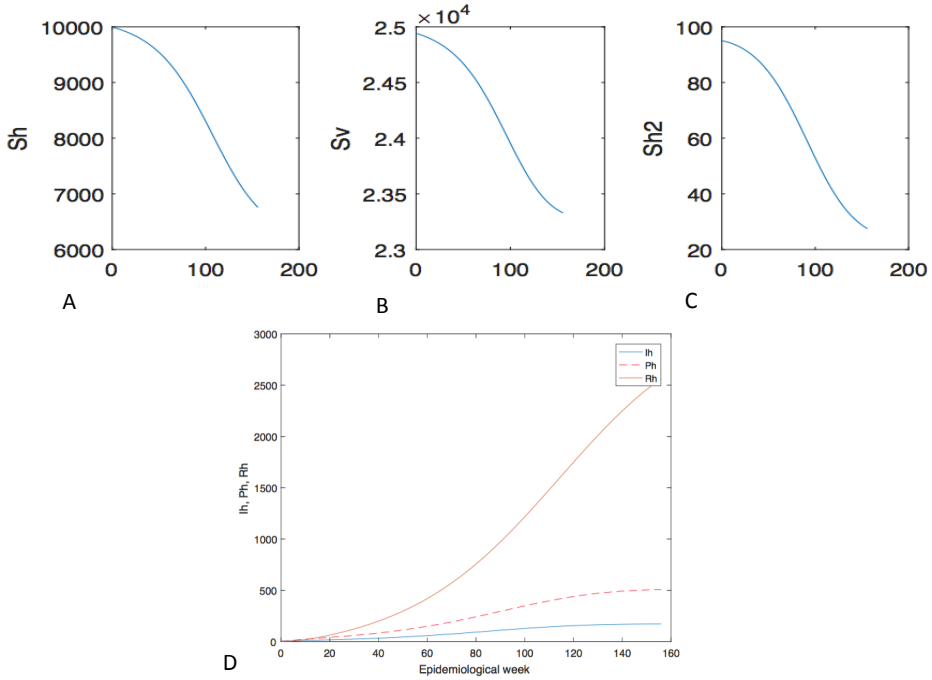

Figure 7: Case 3, system whit simulation of dry conditions, low populations of vectors and birds.

In this figure is observed an infection of the 30% of the population susceptible of human (fig A), this unethical has a tendency similar in them vectors and in the birds (fig B and C). Figure D shows very low levels of infection with a very slight beak towards the latest epidemiological weeks

5 Discussion

A mathematical model can be defined as the set of equations that help to understand and describe the dynamics of infection between vectors and hosts ([8]), the model presented in this paper describes the interactions between the vector responsible for the transmission of Cuteanous Leishmaniasis, and its two main hosts, humans and birds.

Brazil lead the effort in conducting studies on food preference in phlebotominos,their results suggest that *Lutzomyia longipalpis* (the vector for transmission of visceral leishmaniasis) has a preference for birds ([1]). On the other hand, a study carried out in a costal town in Ecuador shows that birds are the preferred food for several species of phlebotomos([2]). At the same time, Anaguanos work serve as evidence that there are differences in the abundance of sand flies species and therefore food preference during the dry and wet season, which would be consistent with the epidemiological data reported by the Ministry of Health.

In the simulations it can be observed that there is a wide gap between the reported (Ph1) and the infected cases (Ih1), confirming our hypothesis that there are an under reporting of the disease. Under reporting is a result of difficult access to endemic areas, registration system failures and patients diagnosed and treated in private medical centers [10].

The simulations under initial conditions and using parameters reported in literature, show

the existence of a peak around the 50th week, coinciding with the outbreak reported by the epidemiological time series data. This result suggest that the system can be adjusted if the field data increase.

The simulations performed with the variability of the populations of birds, show that these hosts have a very important role in the dynamics of the transmission of the disease, so increase the data about infection in birds is a very important point to validate the model. The present work aims to become a tool for decision-making in the control of leishmaniasis in endemic areas of the Ecuador

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6 Appendix

Any Appendix goes here as Appendix A, Appendix B, etc. as separate sections.