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Mathematical modeling of Visceral Leishmaniasis and control strategies

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ABSTRACT

In this article, the author studies the dynamics of Visceral Leishmaniasis disease to analyze the seasonal VL incidence data from South Sudan during the period January, 2011 to December, 2011. The seasonality is consolidated in the model as sandfly growing rate, which is presumed to be time periodic. The basic reproduction number (R_0) has been derived and estimated. On the premise of statistical and epidemiological information of South Sudan, parameters in the model are estimated. The author discusses an optimal control strategy among the two preventive measures namely use of vaccination and possible treatment of infective humans. Numerical findings indicate that the mass treatment is not sufficient to control the outbreak of VL in the population, additional control programs (such as vaccination) with treatment are required to control the VL disease outbreak.

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1. Introduction

Leishmaniasis is world's second largest vector borne disease parasitic to humans and is caused specifically by protozoan parasites belonging to the *Leishmania* genus. Visceral leishmaniasis (VL), the post-kala-azar dermal leishmaniasis (PKDL), cutaneous leishmaniasis (CL) and cutaneous leishmaniasis with involvement of lesions of the mucous membranes, which is also called mucocutaneous leishmaniasis (MCL) are four different clinical demonstration of the disease. Visceral leishmaniasis (VL) or "kala-azar", is responsible for 20,000–40,000 deaths worldwide [40] with 200,000–400,000 new cases in every year [40]. According to WHO, in Bangladesh, India, Nepal, Sudan, Ethiopia and Brazil most of the cases of VL have been reported [54]. Fever, weight loss, splenomegaly, and anaemia are few clinical symptoms of kala-azar (KA). VL is a systemic infection of the phagocytic and reticulo-endothelial system; this infection includes the lymph nodes, spleen and liver. In addition, some VL treated patients (6 months to several years after the treatment regimen) show a macular, maculopapular, and nodular rash that contain dormant parasites [3,10,54]. These individuals are themselves recovered, but serve as an active source of new infection when exposed to vectors. Such individuals are referred to as post-kala-azar dermal leishmaniasis (PKDL) infected.

Visceral leishmaniasis (VL) has been focused by the WHO for elimination as it is lethal, if left untreated. It demands an efficient implementation of disease intervention strategies that can limit the spread of the infection among human populations. Recent studies on VL intervention strategies can be broadly classified under three groups; studies relating to-animal reservoir control, vector population control and human reservoir control [43]. The issues of implementation of strategies, and the associated problems with each of these strategies, further emphasizes the requirement of novel intervention strategies or the use of effective combinations of the existing strategies to target the elimination of the disease. All these require the advancement of an effective and cost-effective VL vaccine [53]. Since vaccination can be used as an important strategy to control the occurrence and prevalence of VL, we need to consider vaccination as a control strategy in the mathematical modeling of VL to reduce the growth of the disease.

Recently, researchers explored rich dynamics such as bistability, limit cycle oscillations, period doubling oscillations & chaos [5,6,8,20,21,30,32,33]. On the other hand, diffusive models explored Turing instability and/or pattern formation in mathematical biology. Recently, some researchers investigated the Turing instability, the phenomena of pattern formation in the ecological and epidemiological system [44–46]. Different types of mathematical models (ODE, PDE, DDE, SDE, etc.) are developed to investigate different biological processes (like, species interactions, disease, dispersal, pattern formation, environmental fluctuations, etc.). Here, we particularly interested to observe the dynamics of VL disease transmission and further contribute in decision-making processes regarding intervention mechanisms using mathematical models, which consider as an important tool to capture the real scenario [47,48,55]. The inter-epidemic periods between 1875 and 1950 in

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Assam, India was studied by Dye [17] using a deterministic model to describe the dynamics of VL. This model was extended in Malta [15] to explain the efficacy of various control methods [16]. After this pioneering work, many mathematical models have been studied to analyze the VL transmission dynamics [2,9,13,14,18,29,40–42] but only few articles describe the VL transmission [2,18,29,40–42]. Transmission of VL can affect animals also. High levels of infection observed in dog populations (canine or CVL) [31]. Through anthroponotic medium or the zoonotic medium; visceral leishmaniasis can be transmitted between human to human or between animal and human respectively [3], and is usually transmitted indirectly between hosts by sandflies of the genera *Phlebotomus* and *Lutzomyia* [26]. Burattini et al. [11], developed an SEIR type model between sandfly, animal and human populations for zoonotic transmission of visceral leishmaniasis. Following up, many other models [18,24,40–42] consider potential PKDL progression rate in humans via the addition of another infective stage. Except few authors [29,40–42], the models are rarely validated with recent data. The aim of this study is to investigate the seasonal fluctuation of VL infection by using mathematical modeling approach with treatment on infected population. The author applies the model to control the seasonal variation of VL in South Sudan.

The rest of the article is organized as follows, Section 2, deals with the model formulation of VL. In Section 3, the author discusses the mathematical analysis of the VL model. The author fits the model outcomes with the number of new VL cases in Section 4. In Section 5, the author uses Pontryagin's Maximum Principle to find the optimal control strategy of the disease. In Section 6, the effect of various control strategies has been studied numerically. The article ends with a conclusions.

2. Model formulation

Here, the author considers a simple and basic SIR type model with respect to history of infection to describe human population. The model comprises of the human, reservoir and sandfly populations with seasonally forced biting rates on the sandfly population. The human population $N_H(t)$ is divided into four sub populations namely S_H, I_H, P_H and R_H.

$$N_H(t) = S_H(t) + I_H(t) + P_H(t) + R_H(t).$$

Similarly, let the reservoir host population be divided into two categories, susceptible reservoir, $S_R(t)$, and infected reservoir, $I_R(t)$, such that

$$N_R(t) = S_R(t) + I_R(t).$$

The total vector (sandfly) population, denoted by N_V is subdivided into susceptible sandflies, $S_V(t)$, and infected sandflies, $I_V(t)$, such that

$$N_V(t) = S_V(t) + I_V(t).$$

All the humans initially remain susceptible to infection (S_H) and are assumed to grow in number with a constant birth Λ_H and death μ_h rate. After being bitten by an infectious sandfly, susceptible humans are considered to become infected (I_H) with force of infection $ab\frac{I_V}{N_H}$ where a is the mean rate of bites per sandfly and b is the sandfly to human (reservoir) transmission probability. Due to short incubation period [16], the author does not consider it in the model. Infected humans expire due to VL at an average rate δ, or treated at an average rate α_1, and a fraction σ of those recover, and the remaining fraction develop PKDL (P_H). PKDL infected humans get treated at an average rate α_2, or recover naturally at an average rate β, and acquire immunity ($R_H(t)$). A per capita natural death rate μ_h is present in all human sub-compartments. Following recovery from KA (I_H) via drug treatment, some individuals (5–10%) develop PKDL (P_H), which is characterised by a nodular or papular skin rash. It is non life-threatening. The probability and timing of PKDL following KA are key assumptions for interpreting surveillance data and in using models to make projections. The occurrence of PKDL is thought to be governed by several factors, including the choice of drugs and adherence to treatment.

Susceptible reservoirs are enlisted into the population at a fixed rate Λ_R, and procure disease after contacts with infected sandflies at a rate $ab\frac{I_V}{N_H}$. The natural mortality rate for reservoirs is μ_r.

Susceptible sandflies are recruited at a constant rate Λ_V, and infected at an average rate equal to $ac\frac{I_H}{N_H} + ac\frac{P_H}{N_H} + ac\frac{I_R}{N_R}$, where c is the transmission probability for sandfly infection. The natural mortality rate for sandfly is μ_v.

The author assumes the biting rate in the following form: $a(t) = a_0(1 + \delta_r \sin\frac{2\pi t}{12})$. The biting rate $a(t)$ of the sandfly population varies periodically with different temperatures which is assumed to be time periodic with period 12 months. a_0 denote the average bitting rate and δ_r denotes the amplitude of seasonality [4,7,36]. With this assumption and the description of the terms, we get the following system of differential equations:

$$\begin{aligned}
S_H' &= \Lambda_H - a(t)bI_V\frac{S_H}{N_H} - \mu_h S_H\\
I_H' &= a(t)bI_V\frac{S_H}{N_H} - (\alpha_1 + \delta + \mu_h)I_H\\
P_H' &= (1-\sigma)\alpha_1 I_H - (\alpha_2 + \beta + \mu_h)P_H\\
R_H' &= \sigma\alpha_1 I_H + (\alpha_2 + \beta)P_H - \mu_h R_H\\
S_R' &= \Lambda_R - a(t)bI_V\frac{S_R}{N_R} - \mu_r S_R\\
I_R' &= a(t)bI_V\frac{S_R}{N_R} - \mu_r I_R\\
S_V' &= \Lambda_V - a(t)cS_V\frac{I_H}{N_H} - a(t)cS_V\frac{P_H}{N_H} - a(t)cS_V\frac{I_R}{N_R} - \mu_v S_V\\
I_V' &= acS_V\frac{I_H}{N_H} + acS_V\frac{P_H}{N_H} + acS_V\frac{I_R}{N_R} - \mu_v I_V
\end{aligned}$$

with

$$\begin{aligned}
N_H' &= \Lambda_H - \mu_h N_H - \delta I_H\\
N_R' &= \Lambda_R - \mu_r N_R\\
N_V' &= \Lambda_V - \mu_v N_V
\end{aligned}$$

3. Basic mathematical properties

The model (2.1) is biologically well defined in $\Omega = \{(S_H, I_H, P_H, R_H, S_R, I_R, S_V, I_V) \in R_+^8 : S_H, I_H, P_H, R_H, S_R, I_R, S_V, I_V \geq 0, N_H \leq \frac{\Lambda_H}{\mu_h}, N_R \leq \frac{\Lambda_R}{\mu_r}, N_V \leq \frac{\Lambda_V}{\mu_v}\}$. Ω is a positive invariant region under the flow induced by (2.1). All the parameters of the model are positive.

Detail description is given in Appendix section.

3.1. Basic reproduction number

Let, for each $s \in \mathbb{R}$, the 4×4 matrix $Y(t, s)$, $\forall t \geq s$, satisfies the ω-periodic system

$$\frac{dy}{dt} = -V(t)y. \tag{3.1}$$

and $Y(s, s) = I$, where I is the 4×4 identity matrix.

Let, C be the ordered Banach space of all ω-periodic functions from $\mathbb{R}$ to $\mathbb{R}^4$, with maximum norm $||.||$. The linear operator L: $C \to C$ can be defined as

$$(L\phi)(t) = \int_0^\infty Y(t, t-a)F(t-a)\phi(t-a)\mathrm{d}a, \quad \forall t \in \mathbb{R}, \quad \phi \in C. \tag{3.2}$$

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