A MATHEMATICAL STUDY ON MEDIA EFFICIENCY IN COMBATING LEISHMANASIS VIA AWARENESS CAMPAIGNS

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Abstract

Leishmaniasis is a complicated vector-borne illness caused by protozoa from the genus Leishmania. Infected female Phlebotominae sandflies bite humans or animals, transmitting the illness. Awareness campaigns can prevent the spread of cutaneous leishmaniasis. Our mathematical model of cutaneous leishmaniasis (CL) includes susceptible and infected human populations, as well as vectors. Increased awareness among the populace may reduce ineptitude towards environmental problems. Our analysis and numerical data show that regular advocacy reduces illness prevalence. The awareness campaign improves the system, leading to a healthier and cleaner environment.

Keywords: Cutaneous Leishmaniasis (CL), Impulsive Approach. Vector-Borne Disease, Awareness Programme

1. Introduction.

According to the World Health Organization (WHO, 2009), leishmaniasis is among the top seventeen neglected tropical diseases (NTDs) worldwide. Leishmania parasites infect humans and animals by bites from infected female sand flies of the Phlebotominae subfamily (Kendrick, 1999). According to Langer et al. (2012), the illness affects over two million people and causes over 50,000 fatalities annually. After ten days to two years of incubation, symptoms may include fever, diarrhea, weight loss, lymphadenopathy, hepatomegaly, and splenomegaly. The sickness begins with an erythematous papule at the sand-fly bite site on the outer body.

The papule develops into a nodule with a defined edge (Bathena, 2009; Reithinger et al. 2007).

However, there is still room for improvement in understanding parasite metabolism and illness development.

The transmission dynamics of cutaneous leishmaniasis have been explored using several mathematical models. In 2004, Rabinovich and Feliciangeli developed a probabilistic model for calculating the frequency of infective bites by sand flies.

Lysenko and Beljaev (1987) devised a mathematical model for cutaneous leishmaniasis to explain parasite transmission. Chaves and Hernandez (2004) proposed a mathematical model for American cutaneous leishmaniasis that considers the vector, host, and reservoir populations. Marcos and Moreira (2007) created a mathematical model for immune response in cutaneous leishmaniasis.

Biswas et al. (2016) found that delaying treatment may lead to infection spreading during the human macrophagic phase. Das et al. (2007) investigated the transmission of American Cutaneous Leishmaniasis with a delay effect. Bacaer and Guernaoui (2006) provided a mathematical model for the seasonality of vector populations.

The disease's restricting mechanisms have previously been investigated from both a mathematical and clinical perspective. However, there is little research on how awareness campaigns may effectively reduce cutaneous leishmaniasis. Our mathematical approach incorporates modern awareness campaign thinking for model development.

To manage the condition, we have included social media-driven awareness efforts in our study. Awareness campaigns may help establish behavioral designs in the general community to combat disease spread. It may increase receptivity among the human population. Effective awareness campaigns may significantly reduce the spread of illness. It reduces the risk of disease transmission. Social media initiatives aim to raise awareness and limit interaction between healthy and diseased populations by sharing information about disease prevention strategies. Once awareness programs are delivered, individuals might respond to the strategy. Media initiatives focus on spreading understanding about disease transmission. Campaigns may reduce the risk of infection by speeding up activities. Numerous studies have studied the impact of social media awareness campaigns (Nyabadza et al., 2010; Funk et al., 2009; Liu and Cui, 2008; Misra et al., 2011) using mathematical analysis. Awareness programs create a new demographic segment known as the aware population, which is distinct from the vulnerable population. Media efforts are believed to safeguard the conscious class against infection. According to Misra et al. (2011), social and environmental variables might cause a population to lose consciousness over time.

This research paper builds on Bacaer and Guernaoui's (2006) work by including an awareness campaign to reduce cutaneous leishmaniasis within a mathematical environment.

We analyzed the geographic region in which social media is present. Our main goal is to quickly manage the illness via awareness campaigns and determine the most effective campaigning methods.

2. Model Formulation with Assumptions

This model examines disease transmission between two distinct populations: the host (human) population and the vector (sand-fly) population. The host population is categorized into two groups: susceptible individuals SH(t)S_H(t)SH(t) and infected individuals IH(t)I_H(t)IH(t). The reservoir class is not considered in this model, as the primary goal is to prevent direct contact between the host and the vector. It is assumed that the reservoir population remains in a steady state.

The recruitment rates of humans and sand-flies, denoted as λH\lambda_HλH and λV\lambda_VλV respectively, are considered constant. The natural death rates for humans and sand-flies are represented by μH\mu_HμH and μV\mu_VμV. A susceptible human becomes infected through **mass action transmission** upon interaction with an infected vector. Here, β\betaβ represents the per capita biting rate of the vector on humans, while π\piπ is the transmission probability per bite per human (Bacaer & Guernaoui, 2006).

It is assumed that the total human population remains constant, i.e., P(t)=SH(t)+IH(t)P(t) = S_H(t) + I_H(t)P(t)=SH(t)+IH(t), and the vector population consists of **susceptible vectors** sV(t)s_V(t)sV(t) and infected vectors iV(t)i_V(t)iV(t), denoted as V(t)=sV(t)+iV(t)V(t) = s_V(t) + i_V(t)V(t)=sV(t)+iV(t). The disease can be transmitted within the vector population from parent (female) to offspring, but for simplicity, it is assumed that all newborn vectors are initially susceptible, and vertical transmission is neglected.

Susceptible vectors become infected when they bite an infected human at a rate β\betaβ, with a transmission probability \pî per bite from human to sand-fly. Thus, the number of newly infected vectors is determined by the **mass action term** \beta \pî s_V(t) I_H(t). Unlike the host population, once vectors are infected, they remain carriers of the pathogen permanently.

They carry the micro parasite throughout their lives (Yang et al., 2010). The illness dynamics may be described using the following system, based on the assumptions above:

$$\dot{s}_V = \lambda_V - \beta\hat{\pi}s_V(t)\frac{I_H(t)}{P(t)} - \mu_V s_V(t),$$
$$\dot{i_V} = \beta\,\hat{\pi}\,s_V(t)\frac{I_H(t)}{P(t)} - \mu_V i_V(t). \qquad (1)$$

It is evident that the vector population remains constant, i.e., limt→∞λVμV\lim_{t \to \infty} \frac{\lambda_V}{\mu_V}limt→∞μVλV for all t>0t > 0t>0, provided that the initial population satisfies sV(0)+iV(0)=λVμVs_V(0) + i_V(0) = \frac{\lambda_V}{\mu_V}sV(0)+iV(0)=μVλV. The dynamics of disease transmission in both the human and sand-fly populations are illustrated in Fig. 1.

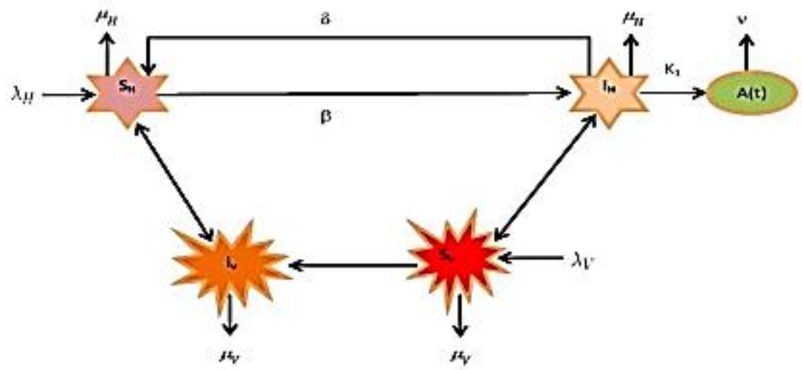


Fig. 1. Compartmental diagram of the model system (2)

We also suppose that A(t) represents the aware class. The natural recovery rate from infected to vulnerable humans is δ, whereas the decreasing rate of infected individuals owing to knowledge is represented by k1. The populace is becoming more aware at a pace of k2, but awareness initiatives are being reduced owing to incompetence (v). Awareness efforts are projected to increase proportionally with the number of afflicted persons. Awareness efforts have led to a separation of vulnerable persons from infectious people. It is also assumed that k2 ≫ ν. The aforementioned picture yielded the entire system of equations shown below:

$$\begin{aligned}
\dot{S}_H &= \lambda_V - \beta\pi i_V(t)\frac{S_H(t)}{P(t)} - \mu_H S_H(t) + \delta I_H(t),\\
\dot{I_H} &= \beta\pi i_V(t)\frac{S_H(t)}{P(t)} - \mu_H I_H(t) - \delta I_H(t) - k_1 A(t) I_H(t),\\
\dot{i_V} &= \beta\hat{\pi}\left(\frac{\lambda_V}{\mu_V} - i_V(t)\right)\frac{I_H(t)}{P(t)} - \mu_V i_V(t),\\
\dot{A} &= k_2 I_H(t) - \nu A(t).
\end{aligned} \tag{2}$$

We have established that the system is bounded for the conditions $0 < P(t) \leq \frac{\lambda_H}{\mu_H}, V(t) \leq \frac{\lambda_V}{\mu_V}$ and $A(t) \leq \sqrt{\frac{k_2\lambda_H}{k_1\nu}}$ as $t \to \infty$.

3. Theoretical Analysis of the System

3.1 Existence Condition

In this system, there are two equilibrium points such as disease-free equilibrium $E0$ $(SH\ ,0,0,0) \equiv$

In this system, there are two equilibrium points such as disease-free equilibrium $E_0(\bar{S}_H\ ,0,0,0) \equiv$ $E_0\left(\frac{\lambda_H}{\mu_H},0,0,0\right)$ and another is endemic equilibrium $E^*(S_H^*, I_H^*, i_V^*, A^*)$, where $S_H^* = \frac{-k_1 k_2 {I_H^*}^2 - \mu_H \nu I_H^* + \lambda_H \nu}{\mu_H \nu}$, $i_V^* = \frac{\frac{\beta\hat{\pi}\lambda_V}{P\ \mu_V} I_H^*}{\mu_V + \frac{\beta\hat{\pi}}{P} I_H^*}$, $A^* = \frac{k_2 I_H^*}{\nu}$ and I_H^* is determined from the equation $a{I_H^*}^2 + bI_H^* + c = 0$, where $a = \frac{\beta\hat{\pi} k_1 k_2}{P\nu}, b = \left(\frac{\mu_V k_1 k_2}{\nu} + \frac{\beta\hat{\pi}\mu_H}{P} + \frac{\delta\beta\hat{\pi}}{P}\right)$ and $c = \mu_V \mu_H + \delta\mu_V - \frac{\beta^2 \pi\hat{\pi}\lambda_V}{P^2 \mu_V}$.

Now, the endemic equilibrium E^* exists if $\beta > P\mu_V \sqrt{\frac{(\mu_H + \delta)}{\lambda_V \pi\hat{\pi}}}$.

Biological interpretation: If the bite rate surpasses the critical value, the system becomes endemic and sickness continues. Increasing the bite rate leads to an increase in the infected human population at a certain transmission rate. Simultaneously, As transmission rates grow, the infected population outnumbers the vulnerable population in terms of preferred bite rates.

3.2 Stability of the System

The model system (2)'s Jacobian matrix at disease-free equilibrium is provided by

$$J|_{(\bar{S}_H\ ,0,0,0)} = \begin{pmatrix} -\mu_H & \delta & -\frac{\beta\pi\bar{S}_H}{P} & 0 \\ 0 & -\mu_H - \delta & \frac{\beta\pi\bar{S}_H}{P} & 0 \\ 0 & \frac{\beta\hat{\pi}\lambda_V}{P\mu_V} & -\mu_V & 0 \\ 0 & k_2 & 0 & -\nu \end{pmatrix}.$$

The characteristic equation for the disease-free equilibrium E0 is stated as

$$\lambda^2 + (\mu_H + \mu_V + \delta)\lambda + (\mu_H + \delta)\mu_V - \frac{\beta^2 \pi\hat{\pi}\lambda_H \lambda_V}{P^2 \mu_H \mu_V} = 0. \qquad (3)$$

The threshold condition is determined by the sign of constant term. It follows that the basic reproduction number

$$R_0 = \frac{\beta^2 \pi \hat{\pi} \mu_H \lambda_V}{\lambda_H \mu_V^2 (\mu_H + \delta)}.$$

Thus, if $R0 < 1$, then the disease-free equilibrium is stable, while if $R0 > 1$, then the disease-free equilibrium is unstable and the system moves to its endemic state.

Finally the Jacobian matrix at the endemic equilibrium $E * (SH *, IH *, iV *, A *)$ is furnished by,

$$J|_{(S_H^*, I_H^*, i_V^*, A^*)} = \begin{pmatrix} -\frac{\beta\pi i_V^*}{P} - \mu_H & \delta & -\frac{\beta\pi S_H^*}{P} & 0 \\ \frac{\beta\pi i_V^*}{P} & -\mu_H - \delta - k_1 A^* & \frac{\beta\pi S_H^*}{P} & -k_1 I_H^* \\ 0 & \frac{\beta\hat{\pi}}{P}\left(\frac{\lambda_V}{\mu_V} - i_V^*\right) & -\frac{\beta\hat{\pi} I_H^*}{P} - \mu_V & 0 \\ 0 & k_2 & 0 & -\nu \end{pmatrix}.$$

The characteristic equation is given by,

$$\lambda^4 + a_1\lambda^3 + a_2\lambda^2 + a_3\lambda + a_4 = 0,$$

Where

$$\begin{aligned} a_1 &= -(x_{11} + x_{22} + x_{33} + x_{44}), \\ a_2 &= x_{11}x_{22} + x_{11}x_{33} + x_{11}x_{44} + x_{22}x_{33} + x_{22}x_{44} \\ &\quad + x_{33}x_{44} - x_{24}x_{42} - x_{12}x_{21} - x_{23}x_{32}, \\ a_3 &= x_{11}x_{24}x_{42} + x_{12}x_{21}x_{44} + x_{24}x_{33}x_{42} + x_{12}x_{21}x_{33} + x_{11}x_{23}x_{32} + x_{23}x_{32}x_{44} \\ &\quad - x_{11}x_{22}x_{33} - x_{11}x_{22}x_{44} - x_{22}x_{33}x_{44} - x_{13}x_{21}x_{32} - x_{11}x_{33}x_{44}, \\ a_4 &= x_{11}x_{22}x_{33}x_{44} + x_{13}x_{21}x_{32}x_{44} - x_{11}x_{23}x_{32}x_{44} - x_{12}x_{21}x_{33}x_{44} - x_{11}x_{24}x_{33}x_{42}, \end{aligned}$$

$x_{11} = -\frac{\beta\pi i_V^*}{P} - \mu_H$, $x_{12} = \delta$, $x_{13} = -\frac{\beta\pi S_H^*}{P}$, $x_{21} = \frac{\beta\pi i_V^*}{P}$, $x_{22} = -\mu_H - \delta - k_1 A^*$, $x_{23} = \frac{\beta\pi S_H^*}{P}$, $x_{24} = -k_1 I_H^*$, $x_{32} = \frac{\beta\hat{\pi}}{P}\left(\frac{\lambda_V}{\mu_V} - i_V^*\right)$, $x_{33} = -\frac{\beta\hat{\pi} I_H^*}{P} - \mu_V$, $x_{42} = k_2$ and $x_{44} = -\nu$.

Hence the endemic equilibrium point $E^*(S_H^*, I_H^*, i_V^*, A^*)$ is said to be stable if $a_1 a_2 - a_3 > 0$ and $a_1 a_2 a_3 - a_1^2 a_4 - a_3^2 > 0$.

4. Awareness Programme via Impulsive Mode

This section examines the impact of awareness campaigns on impulsive behavior over a certain time period. It helps to avoiding interaction between humans and diseased vectors. We have picked a unique time span for campaigning. The program increases environmental awareness by a certain percentage (ρ). This reduces both the number of sick individuals and vectors. Our study of the model in impulsive mode indicates that awareness campaigns do not negatively effect system dynamics. The impulsive differential equation has the following form:

$$\frac{d(A(t))^2}{dt} = \xi - \nu'(A(t))^2, t \neq t_k$$

$$\Delta A = \rho A, \qquad t = t_k, \tag{4}$$

where

$$\frac{2k_2\lambda_H}{k_1} = \xi \text{ (say)}.$$

For single impulsive cycle $tk-1 \leq t \leq tk$, the general solution is given by,

$$A_{k+1}^+ = \frac{\xi'}{\nu'}\{1 - e^{-\nu'(t_k - t_{k-1})}\} + A_k^- e^{-\nu'(t_k - t_{k-1})}, \text{ using } \frac{\xi'}{\nu'} = \sqrt{\frac{k_2\lambda_H}{k_1\nu}},$$

where $A(t_k^+) = A_k^+$ and $A(t_k^-) = A_k^-$. Now,

$$A_1^+ = \frac{\xi'}{\nu'}, \ A_1^- = (1-\rho)\frac{\xi'}{\nu'}, \ A_2^+ = (1-\rho)\frac{\xi'}{\nu'}e^{-\nu'(t_2-t_1)} + \frac{\xi'}{\nu'}\{1 - e^{-\nu'(t_2-t_1)}\},$$

$$A_2^- = (1-\rho)A_2^+ = (1-\rho)^2 \frac{\xi'}{\nu'}e^{-\nu'(t_2-t_1)} + (1-\rho)\frac{\xi'}{\nu'}\{1 - e^{-\nu'(t_2-t_1)}\},$$

$$A_3^+ = \frac{\xi'}{\nu'}[(1-\rho)^2 e^{-\nu'(t_3-t_1)} + (1-\rho)e^{-\nu'(t_3-t_2)} + 1 - (1-\rho)e^{-\nu'(t_3-t_1)} - e^{-\nu'(t_3-t_2)}],$$

$$A_4^+ = \frac{\xi'}{\nu'}[(1-\rho)^3 e^{-\nu'(t_4-t_1)} + (1-\rho)^2 e^{-\nu'(t_4-t_2)} + (1-\rho)e^{-\nu'(t_4-t_3)} + 1 - (1-\rho)^2 e^{-\nu'(t_4-t_1)} - (1-\rho)e^{-\nu'(t_4-t_2)} - e^{-\nu'(t_4-t_3)}].$$

Therefore the general solution becomes,

$$A_n^+ = \frac{\xi'}{\nu'}[(1-\rho)^{(n-1)}e^{-\nu'(t_n-t_1)} + (1-\rho)^{(n-2)}e^{-\nu'(t_n-t_2)} + \cdots + (1-\rho)e^{-\nu'(t_n-t_{n-1})} + 1 - (1-\rho)^{(n-2)}e^{-\nu'(t_n-t_1)} - (1-\rho)^{(n-3)}e^{-\nu'(t_n-t_2)} - \cdots - e^{-\nu'(t_n-t_{n-1})}].$$

4.1 Awareness for Fixed Time Interval

For fixed time interval, i.e., $\tau = tn - tn-1$ is constant, we have

$$A_n^+ = \frac{\xi'}{\nu'}[1 + (1-\rho)e^{-\nu'\tau} + (1-\rho)^2 e^{-2\nu'\tau} + \cdots + (1-\rho)^{n-1}e^{-\nu'(n-1)\tau} - e^{-\nu'\tau}\{1 + (1-\rho)e^{-\nu'\tau} + \cdots + (1-\rho)^{(n-2)}e^{-\nu'(n-2)\tau}] = \frac{\xi'}{\nu'}[\frac{1-(1-\rho)^n e^{-\nu' n\tau}}{1-(1-\rho)e^{-\nu'\tau}} - e^{-\nu'\tau}\frac{1-(1-\rho)^{(n-1)}e^{-\nu'(n-1)\tau}}{1-(1-\rho)e^{-\nu'\tau}}].$$

Therefore,

$$\lim_{n\to\infty} A_n^+ = \frac{\xi'}{\nu'}[\frac{1}{1-(1-\rho)e^{-\nu'\tau}} - e^{-\nu'\tau}\frac{1}{1-(1-\rho)e^{-\nu'\tau}}] = \frac{\xi'}{\nu'}[\frac{1-e^{-\nu'\tau}}{1-(1-\rho)e^{-\nu'\tau}}]. \tag{5}$$

This is maximum number of human present in the environment that have to aware. To keep this under the threshold value $\tilde{A}$ we have,

$$\frac{\xi'}{\nu'}\left[\frac{1-e^{-\nu'\tau}}{1-(1-\rho)e^{-\nu'\tau}}\right] < \tilde{A},$$

$$\tau < \frac{1}{\nu'}\ln\left[\frac{\xi'-\tilde{A}\,\nu'(1-\rho)}{\xi'-\tilde{A}\,\nu'}\right] = \tau_{max}(\rho) \text{ (say).}$$

$$\text{i.e., } \tau < \frac{1}{2\nu}\ln\left[\frac{\sqrt{\frac{k_2\lambda_H}{k_1\nu}}-\tilde{A}\,2\nu(1-\rho)}{\sqrt{\frac{k_2\lambda_H}{k_1\nu}}-\tilde{A}\,2\nu}\right] = \tau_{max}(\rho) \text{ (say).}$$

which implies

Biological Interpretation: If the time interval τ be less than some predetermined quantity (τmax), then we can reduce the number of infected human population through awareness campaign under the threshold value $\tilde{A}$ in a real-life scenario.

5. Numerical Simulation

Parameter	Range	Default Value (day^{-1})
λ_H	300-318	317
Λ_V	14950-15000	14950
μ_H	0.3-0.4	0.3
μ_V	0.189-0.195	0.189
β	0.21-0.29	0.25
π	0.22-0.3	0.3
$\hat{\pi}$	0.071428-0.1	0.1
K_1	0.0284-0.0324	0.03
K_2	0.0378-0.401	0.04
ν	0.0028-0.00034	0.003
δ	0.00281-0.042	0.03
ρ	2-9	2,8

Table 1. List of parameters used in the model equation (2)

The recruitment rate of the human population is denoted as λH\lambda_HλH, while that of the sand-fly population is represented by λV\lambda_VλV. The natural death rate of sand-flies is given by μV\mu_VμV, and β\betaβ denotes the per capita biting rate of vectors on humans. The values for these parameters have been sourced from Biswas et al. (2014) and Elmojtaba et al. (2010).

The transmission probability per bite per human (π\piπ) and the transmission probability per bite from human to sand-fly (\pî) are taken from Bathena (2009) and Elmojtaba et al. (2010). The reduction rate of awareness programs due to incompetence (ν\nuν) is referenced from Misra et al. (2011). Additionally, the natural death rate of humans (μH\mu_HμH), the reduction rate of infected humans due to awareness programs (k1k_1k1), and the growth rate of the aware population (k2k_2k2) have been estimated. The natural recovery rate from the infected to the susceptible human class (δ\deltaδ) and the increase in the number of aware individuals by a proportion ρ\rhoρ have also been estimated.

To analyze the mathematical model (2), a numerical simulation has been conducted. The values of the model parameters used in the simulation are listed in Table 1.

From Fig. 2, it is evident that when the biting rate remains below its threshold value (β<0.25\beta < 0.25β<0.25), the system reaches a disease-free equilibrium. However, if the biting rate surpasses the threshold (β>0.25\beta > 0.25β>0.25), the system transitions into an endemic state, leading to the persistence of the disease (Fig. 3).

The impact of awareness campaigns is illustrated in Fig. 4 and Fig. 6. In Fig. 6, different levels of awareness efforts are examined by considering ρ=2\rho = 2ρ=2 and ρ=8\rho = 8ρ=8 with τ=2\tau = 2τ=2. It is observed that conducting awareness campaigns twice daily at an interval of two days helps the system move towards a disease-free state. Moreover, increasing the frequency of campaigns per day results in a more effective reduction of infections.

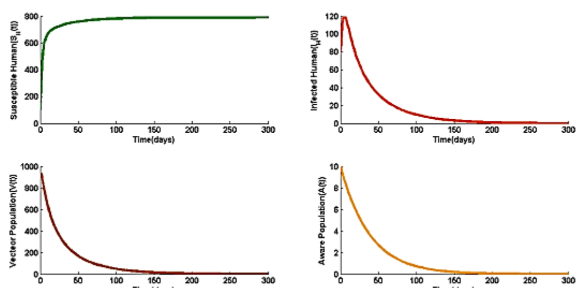


Fig. 2. Behavior of different population for R0 < 1, when the system attains its disease-free state

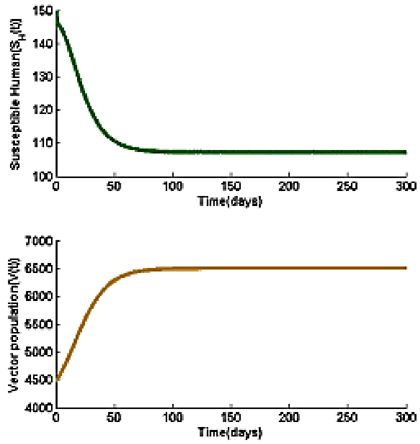


Fig. 3. Behavior of different population for R0 > 1, when the system attains its endemic state

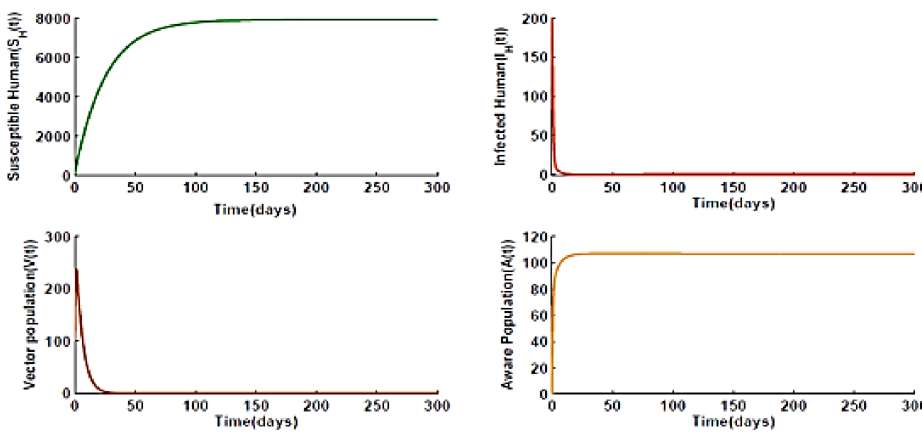


Fig. 4. Population density as a function of time when awareness campaigning is circulated by media

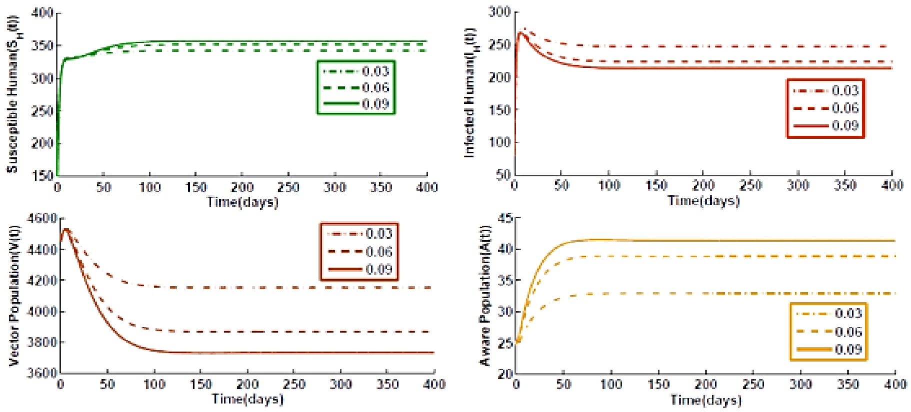


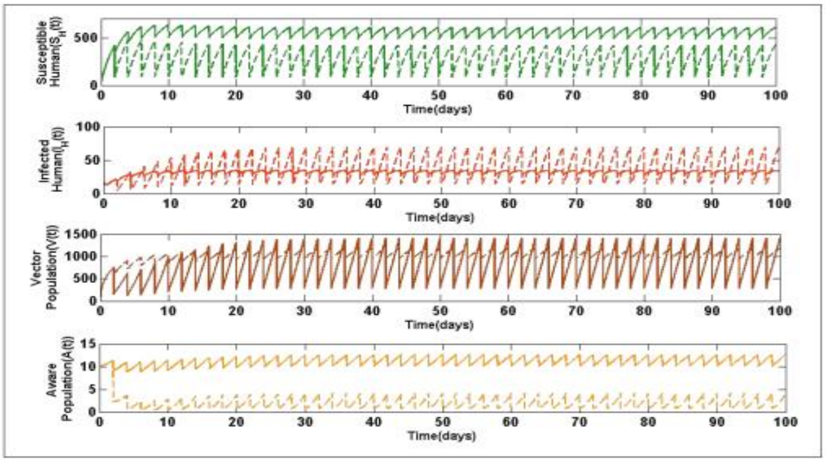
Fig. 5. Density of four populations for different values of □2

6. Discussion

It is critical to understand the interaction mechanism between humans and diseased vectors. If the contact process can be kept to a minimal, the illness may be eradicated. In this regard, raising awareness may play a significant role in disease management. We examined the system in the absence of awareness programs. If R0 < 1, there is a disease-free scenario. If R0 > 1, the disease-free condition becomes unstable and the system shifts towards an endemic situation. When the illness develops, the susceptible host population is dramatically reduced for 45 days (about), whereas the infected host population steadily increases for 50 days or so. Furthermore, sandfly bite rates are thought to be more crucial for disease propagation. Similarly, the growth rate of the conscious population is a highly important metric in mathematical terms. The shift in the system's behavioral structure is mostly determined by the bite rate of sandflies and the growth rate of the conscious population. So, if we can manage the connection between humans and sandflies via periodic awareness campaigns, sickness will be automatically controlled.

7. Conclusion.

Implementing an effective awareness campaign may help eliminate disease transmission in a timely manner. Awareness efforts, mostly via social media, may effectively manage the condition. Proper frequency and spacing of campaigns are crucial. Successfully eradicating cutaneous leishmaniasis is challenging due to its many vectors and reservoirs. Our model-based approach implies that an impulsive awareness campaign may provide valuable insights for combating cutaneous leishmaniasis.



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References

1. Bacaër, N., & Guernaoui, S. (2006). The epidemic threshold of vector-borne diseases with seasonality. Journal of Mathematical Biology, 53(3), 421-436.
2. Bathena, K. (2009). A Mathematical model of cutaneous leishmaniasis. Thesis. Rochester Institute of Technology.
3. Biswas, D., Kesh, D. K., Datta, A., Chatterjee, A. N., & Roy, P. K. (2014). A mathematical approach to control cutaneous leishmaniasis through insecticide spraying. Sop Transactions on Applied Mathematics, 1(2), 44-54.
4. Biswas, D., Roy, P. K., Li, X. Z., Basir, F. A., & Pal, J. (2016). Role of macrophage in the disease dynamics of cutaneous Leishmaniasis: a delay induced mathematical study. Communications in Mathematical Biology and Neuroscience, 2016(4), pp. 1-31.
5. Chaves, L. F., & Hernandez, M. J. (2004). Mathematical modelling of American cutaneous leishmaniasis: incidental hosts and threshold conditions for infection persistence. Acta Tropica, 92(3), 245-252.
6. Das, P., Mukherjee, D., & Sarkar, A. K. (2007). Effect of delay on the model of American cutaneous leishmaniasis. Journal of Biological Systems, 15(02), 139-147.

7. ELmojtaba, I. M., Mugisha, J. Y. T., & Hashim, M. H. (2010). Mathematical analysis of the dynamics of visceral leishmaniasis in the Sudan. Applied Mathematics and Computation, 217(6), 2567-2578.
8. Funk, S., Gilad, E., Watkins, C., & Jansen, V. A. (2009). The spread of awareness and its impact on epidemic outbreaks. Proceedings of the National Academy of Sciences, 106(16), 6872-6877.
9. Killick-Kendrick, R. (1999). The biology and control of phlebotomine sand flies. Clinics in Dermatology, 17(3), 279-289.
10. Länger, B. M., Pou-Barreto, C., González-Alcón, C., Valladares, B., Wimmer, B., & Torres, N. V. (2012).
11. Modeling of leishmaniasis infection dynamics: novel application to the design of effective therapies. BMC Systems Biology, 6(1), 1.
12. Liu, Y. & Cui, J. (2008). The impact of media convergence on the dynamics of infectious diseases, International Journal of Biomathematics, 1, 65-74.
13. Lysenko, A. J., Beljaev, A. E., Peters, W., & Killick-Kendrick, R. (1987). Quantitative approaches to epidemiology. The leishmaniases in biology and medicine. Volume I. Biology and epidemiology, 263- 290.
14. Marcos, C. D. & Moreira, H. N. (2007). A mathematical model of immune response in cutaneous leishmaniasis. Journal of Biological Systems, 15(3), pp. 313 – 354
15. Misra, A. K., Sharma, A., & Shukla, J. B. (2011). Modeling and analysis of effects of awareness programs by media on the spread of infectious diseases. Mathematical and Computer Modelling, 53(5), 1221- 1228.
16. Misra, A. K., Sharma, A., & Singh, V. (2011). Effect of awareness programs in controlling the prevalence of an epidemic with time delay. Journal of Biological Systems, 19(02), 389-402.
17. Nyabadza, F., Chiyaka, C., Mukandavire, Z., & Hove-Musekwa, S. D. (2010). Analysis of an HIV/AIDS model with public-health information campaigns and individual withdrawal. Journal of Biological Systems, 18(2), 357-375.
18. Rabinovich, J. E., & Feliciangeli, M. D. (2004). Parameters of leishmania braziliensis transmission by indoor Lutzomyia ovallesi in Venezuela. The American Journal of Tropical Medicine and Hygiene, 70(4), 373- 382.
19. Reithinger, R., Dujardin, J. C., Louzir, H., Pirmez, C., Alexander, B., & Brooker, S. (2007). Cutaneous leishmaniasis. The Lancet Infectious Diseases, 7(9), 581-596.
20. World Health Organization. (2009). Leishmaniasis: magnitude of the problem. World Health Organization, Geneva.
21. Yang, H., Wei, H., & Li, X. (2010). Global stability of an epidemic model for vector-borne disease. Journal of Systems Science and Complexity, 23(2), 279-292.