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Analysis of a Mathematical Model of Zoonotic Visceral Leishmaniasis (ZVL) Disease

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Abstract: This research paper attempts to describe the transmission dynamic of zoonotic visceral leishmaniasis with the aid of a mathematical model by considering the asymptomatic stages in humans and animals. The disease is endemic in several countries. Data used in the research are obtained from the literature while some are assumed based on the disease dynamic. The consideration of both asymptomatic and the symptomatic infected individuals is incorporated in both humans and animals (reservoir), as well as lines of treatment for the human population. It is found that the model has two fixed points; the VL-free fixed point and the VL-endemic fixed point. Stability analysis of the fixed points shows that the VL-free fixed point is globally asymptotically stable whenever the basic reproduction number is less than one and the VL-endemic fixed point is globally asymptotically stable whenever the basic reproduction number is greater than one. Sensitivity analysis is conducted for the parameters in the basic reproduction number, and the profile of each state variable is also depicted using the data obtained from the literature and those assumed. The transmission probability from infected sandflies to animals, transmission probability from infected animals to sandflies, per capita biting rate of sandflies of animals, and rate of transfer from symptomatic infected animals to the recovered class are among the most sensitive parameters that have the greatest influence on the basic reproduction number. Moreover, the value of the basic reproduction number is obtained to be 0.98951, which may require further study, as the margin between potential disease control and outbreak is thin.

Keywords: visceral leishmaniasis; basic reproductive number; non-linear differential equations; mathematical model; sensitivity analysis

MSC: 34A34; 92B05; 37N25; 37M05

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1. Introduction

Visceral leishmaniasis (VL) is one of the Neglected Tropical Diseases (NTDs). The protozoan parasites that cause leishmaniasis are spread by the bite of infected female phlebotomine sandflies. The disease has a connection to poverty and is linked to malnutrition, substandard housing, armed conflict, population displacement, illiteracy, gender discrimination, a weakened immune system, and a lack of resources [1,2].

Over 88 countries in the world have some areas where leishmaniasis is present. In these regions, there are about 350 million residents. In the tropics and subtropics are

where most of the countries impacted by leishmaniasis can be found. Leishmaniasis can be found in rain forests in Central and South America as well as in deserts in West Asia. India, Bangladesh, Nepal, Sudan, and Brazil account for more than 90% of the global cases of visceral leishmaniasis. Mexico, Central America, and South America, from northern Argentina to Texas (but not in Uruguay, Chile, or Canada); Asia (but not Southeast Asia); the Middle East; and Africa (mainly East and North Africa, with some cases elsewhere) are the regions where leishmaniasis is found [3].

The primary reservoir of the parasite in these places is the domestic dog, which is spread by the phlebotomine sandfly, Lutzomyia longipalpis [4,5]. Upon introduction into a community, the parasite sustains a dog–insect–dog peri-domestic transmission cycle [6], wherein infected flies sporadically bite humans, resulting in zoonotic visceral leishmaniasis (ZVL). Because of this, the incidence and prevalence of ZVL are crucial epidemiological factors for limiting transmission [7], and the estimates of these parameters rely on the accurate identification of dogs that are affected [8]. Dogs that are affected are therefore frequently eliminated as part of ZVL control operations.

In the Mediterranean, ZVL is a veterinary and medical issue, where dogs are valued as "valuable" animals [9]. Human cases of ZVL are common in Brazil, where 90% of cases reported are in humans, dogs are viewed as "less valuable", and ZVL is primarily regarded as a medical issue. Because of this, 850,000 dogs in Brazil undergo screening each year, and 20,000–25,000 [9,10] of those canines are put to death after receiving a positive test. This official position that dogs are undervalued is held by some governments, such as the Brazilian Health Authorities, which prohibit treating infected dogs with drugs intended for human use and mandate the mandatory elimination of dogs that test positive for drugs. Thousands of dogs are euthanized each year without a counterproof diagnosis because health officials threaten to fine dog owners severely if they refuse to have their animals' serabiological analyses performed. Therefore, rather than having their dogs submitted to serological screening, owners either forbid health agents from entering their houses or move their pets to locations without serological screening. Some proprietors even avoid taking their dogs to veterinary doctors, fearing an examination could result in a death sentence for their animals [11].

The two parts of ethics that need to be stressed as regards leishmanisis are those pertaining to animals and human relations, where human health is at stake and animals and their link with human society are valued more. It is important that these ethic issues are addressed so that they do not impede the effort towards the control of leishmaniasis.

During World War II, leishmaniasis and sandfly fever were highly prevalent among soldiers stationed in the Persian Gulf region. This region saw the deployment of some 697,000 US troops during the Gulf War (1990–1991). In this cohort, only 12 cases of visceral and 19 cases of cutaneous leishmaniasis were found to be diagnosed. The application of insecticides and repellents, reduced summertime transmission rates, and increased urbanization all contributed to the improvement [5,6]. It is estimated that in 2003, approximately 150 American soldiers fighting in Iraq were diagnosed with leishmaniasis, and additional cases are anticipated. Preliminary information on 22 instances of leishmaniasis treated at Walter Reed Army Medical Center that were contracted by US troops in Afghanistan, Kuwait, and Iraq between August 2002 and September 2003 was recently released.

In order to effectively combat visceral leishmaniasis, early detection and proper treatment are essential. Visceral leishmaniasis symptoms and signs are non-specific; thus, a diagnosis is only made by combining clinical symptoms and laboratory testing that are specific to Leishmania. The level of the health system determines the diagnostic approach for medical services in endemic areas. For application in the field in the majority of endemic locations, two serological tests—the Direct Agglutination Test (DAT) and the rK39 antigen-based immunochromatographic tests—have been developed [12]. A Recombinant DNA Technology (RDT) for visceral leishmaniasis is an easy test that may be used both peripherally and centrally. It identifies antibodies. According to a scientific evaluation of published data, the sensitivity of RDTs varies with the eco-epidemiological regions,

particularly in East Africa, where it is low. The drawbacks of all serological tests include their inability to reliably diagnose relapse and the fact that a sizable portion of healthy individuals living in endemic regions without a history of visceral leishmaniasis are positive for antileishmanial antibodies as a result of asymptomatic infections. These limitations are caused by the persistence of antibodies for prolonged periods after cure. Because of this, it is necessary to diagnose visceral leishmaniasis by combining antibody-based testing with a consistent clinical case definition.

Pentavalent antimonials have been the first-line treatment for VL for more than 70 years, although there are currently four medications available for the condition. This treatment takes 20 to 30 days, is toxic (3–5% deaths due to treatment), and is accompanied by increasing failure rates as noted in [13]. The first oral medication against VL is miltefosine, which has been recommended as a first-line medication in the VL elimination initiative; however, because of its long half-life, it is teratogenic and may quickly lead to resistance. Amphotericin B is used in two formulations: "Conventional" amphotericin B and "Liposomal" amphotericin B (AmBisome), and finally, paromomycin (PMM), which was registered in India in 2006, and is currently being tested in a phase IV trial [8,14].

An in vitro point-of-care test is necessary to confirm or rule out active cases for early diagnosis since untreated cases operate as reservoirs for infection and endanger the community to contracting leishmaniasis. Comparably, to ascertain whether therapy for visceral leishmaniasis has been successful, a laboratory test is necessary [15].

Despite the fact that model projections are predicated on precise parameter estimations and scenarios, actual conditions vary greatly, contributing a variable complexity that is difficult to measure. Numerous features of VL disease remain poorly understood, and its elimination has been identified as a public health concern [16]. Specifically, the progression of the disease through many stages, including PKDL and asymptomatic infection, remains unclear, particularly with respect to infectivity and diagnosis. Since recent efforts to manage VL appear to be working, the 2020 deadline for ending VL as a public health concern was rescheduled to 2017 [4,16]. However, modeling and studies of gearbox have indicated a likely scenario. However, modeling and transmission studies have raised the conceivable potential that those who are asymptomatic could obstruct the removal of the infection or hide the true number of leishmaniasis infections. Leishmaniasis infections are a major worry for any group in societies worldwide. Many studies have presented models to understand the disease, as well as the importance of eliminating VL infection independent of population-level asymptomatic infection classifications.

Using ordinary differential equations, a compartment-based mathematical model of zoonotic visceral leishmaniasis transmission and its control across three distinct populations—human, animal, and sandfly—was created in [17]. Asymptomatic, symptomatic, post-kala-azar cutaneous leishmaniasis, and transiently infected individuals made up the human population. The study examined the effects of the asymptomatic stages, but it did not address the treatment phases for the infected human population.

The treatment approach for VL is centered on managing patients who have manifested symptoms of the disease. This implies that since laboratory testing is the only method available to identify those who are infected but have not yet displayed any symptoms, asymptomatic individuals will continue to transmit the disease unchecked. This indicates that the current treatment approach might not be enough on its own to bring the condition under control and sufficiently lower its occurrence. The ratio of symptomatic transmission to sandflies that receive medical attention is the main focus of the argument on disease transmission. If affected people are the main agent transmitting the disease, then treatment will have a major effect on additional transmission. Primary indirect evidence for this disease comes from the long-term cycles seen in many regions and the spatial clustering of leishmaniasis cases; both of these are best explained if Kala-Azar cases are the source of transmission. On the other hand, if asymptomatic infections account for most of the transmission, this means that early treatment of leishmaniasis may not significantly reduce transmission. Currently, there is less evidence to support this scenario, but it is not im-

plausible if multiple asymptomatic infections propagate slowly to sandflies, increasing the rate of transmission to humans and animals. This emphasizes the necessity of conducting additional study and gathering precise data on the VL history and infectiousness relativity of various infection stages in order to enhance control strategies.

This work therefore will contribute to the methodology of vector control in the management of VL disease. Through this research, it is hoped that maximal benefits and new strategies will be developed, especially given that our model considers the critical states of infectiousness of the disease.

Furthermore, the study will lessen the lack of models and data available to address VL disease. It will also be very helpful in understanding how the population of asymptomatic animals, which typically impedes management of the disease, interacts with sandfly populations, which appear to be crucial to the eradication effort. Furthermore, it will advance our knowledge of the biology of sandflies and how transmission might alter over the course of a year. Future studies could make use of the fundamental frameworks established by those who have already studied the seasonality of sandflies in VL and the broader body of knowledge on the seasonal dynamics of other disease vectors.

In this paper, therefore, a comprehensive compartment-based mathematical model that examines the transmission process of VL in three different populations (humans and animals acting as hosts, and sandflies acting as the vector) is considered, taking into account the precise categorization of the human infected population into three distinct clinical classes (symptomatic, asymptomatic, and PKDL infected) and subdividing asymptomatic classes into early and late asymptomatic classes, and considering classes of lines of treatment. The model includes a total of 20 compartments (represented as variables) with regard to various demographic categories, and it represents the rate of disease transmission as a flow from one compartment to another. For this big system, the model is examined both analytically and numerically in order to determine the potential control mechanism and show the impact of the disease transmission amongst the different populations.

The paper is organized in this order: In Section 2, we formulate the model together with the description of the parameters defined in the model. In Section 3, we discuss the basic properties of the model. In Section 4, we obtain the fixed points, compute the basic reproduction number, analyze the local and global stability of the fixed points obtained, and conduct sensitivity analysis on some key parameters. In Section 5, we present the numerical analysis vis-à-vis sensitivity analysis and numerical simulation. Finally, in Section 6, we discuss the results and conclusion.

2. Model Formulation

In this research paper, a model of zoonotic visceral leishmaniasis disease incorporating lines of treatment for the infected human population is considered. Let $N_H(t)$ stand for the total human population, $N_A(t)$ for the total animal population (reservoir), and $N_F(t)$ for the total sandfly population (vectors). In order to represent a biologically realistic complex scenario among populations, the total human population is further divided into twelve compartments: susceptible $S_H(t)$, exposed $E_H(t)$, early asymptomatic infected $I_H^E(t)$, late asymptomatic infected $I_H^L(t)$, early recovered stage who are DAT-positive and not yet LST-positive $R_H^E(t)$, late recovered stage who are DAT-negative but still LST-positive $R_H^L(t)$, symptomatic infected $I_H^S(t)$, infected individuals who are receiving first-line treatment $I_H^1(t)$, infected individuals who are receiving second-line treatment $I_H^2(t)$, PKDL-infected $I_H^P(t)$, recovered humans who have cleared the parasite $R_H^1(t)$, and putative recovered human $R_H^2(t)$. The first and second lines of treatment, which aim to reduce the parasite population within each host, are the only ones that helped the people in this class recover. Similarly, the animal or reservoir populations are divided into five compartments: susceptible animal $S_A(t)$, exposed animal $E_A(t)$, asymptomatic infected animal $I_A^A(t)$, symptomatic infected animal $I_A^S(t)$, and recovered asymptomatic infected animal $R_A(t)$. The sandfly (vector) population is divided into three compartments: susceptible sandflies $S_F(t)$, exposed sandflies $E_F(t)$, and infected sandflies $I_F(t)$. The regular SEIR model is used to

simulate the flow of infection between these compartments in the human and animal populations, whereas the SEI carrier-type model is used to simulate the flow of infection through the vector. Each arrow between the compartments in the model Figure 1 below indicates the passage of infection from one compartment to the next and illustrates the model structure incorporating the numerous components considered in the model. The arrows entering each susceptible population represent the birth rates of humans, animals, and sandflies (Π_H, Π_A, Π_F), respectively, while the arrows moving out from each compartment that do not enter any compartment represent the death rates (μ_H, μ_A, μ_F) of each individual compartment considered in the model. Using a standard incidence function as defined by [18] the interactions of susceptible humans and animals with the sandfly populations are modeled, considering the likelihood that an infected sandfly may spread infection to a susceptible human or animal host (λ_F, η_F) and also that the sandfly populations can contract infection by an infected animal with incidence functions (ρ_A, ξ_A), respectively. A fraction of exposed humans (θ_1) become asymptomatic first at an early stage before late stage at a rate of θ_2. After infection, exposed humans enter the early asymptomatic stage $I_H^E(t)$ and become PCR-positive in peripheral blood. As the specific antibodies develop in the blood serum due to the infection, the fraction of humans in the early asymptomatic stage move to the late asymptomatic stage $I_H^L(t)$, which is characterized by the onset of DAT-positivity. If not dying, humans remain in this asymptomatic stage. Infection with Leishmania parasites proceeds in most cases asymptomatically, with only a minor fraction of cases subsequently developing KA. A major fraction $\tau_1\psi_1$ does not develop disease, becomes PCR-negative in the early recovered stage $R_H^E(t)$, and develops LST positivity $R_H^L(t)$ at a rate of ε. A remaining fraction of $\tau_2\psi_1$ asymptomatic infections become symptomatic $I_H^S(t)$, and a tiny fraction $\tau_3\psi_1$ of putatively recovering humans develop a state of PCR-negativity in peripheral blood, while still harboring a non-detectable number of parasites; this is denoted as $R_H^2(t)$, from where relapse to PKDL ($I_H^P(t)$) follows. A fraction $\tau_2\psi_1$ develops symptomatic KA (I_H^S) whilst staying PCR-positive. Those developing symptoms are eligible for treatment. If not dying, these patients receive first-line treatment (I_H^1). A proportion ω_1 of patients clear parasites under first-line treatment, recover (R_H^1), and finally become LST-positive (R_H^L) like those with asymptomatic infections. A fraction φ of the LST-positive (R_H^L) patients become susceptible. The remaining proportion of patients represent the treatment failures that are split into a PCR-positive proportion of ω_3 patients receiving second-line treatment (I_H^2) and a proportion of ω_2 patients putatively recovering into a state of PCR-negativity. The second proportion still harbors a non-detectable number of parasites (R_H^2, from where relapse to PKDL will follow). For second-line treatment (in state I_H^2), as with first-line treatment, a proportion γ_2 of patients under second-line treatment recover (R_H^1) and become LST-positive (R_H^L). The remaining proportion of γ_1 patients recover putatively into a state of PCR-negativity (R_H^2) from which, again, relapse to PCR-positivity and PKDL (I_H^P) follows for those who do not die. All PKDL patients (I_H^P) are treated until full recovery (R_H^1).

The sandfly population is considered in the susceptible (S_F), exposed (E_F), or infectious (I_F),) stage. Sandflies can become infected by blood meals taken from an infectious animal through a certain interaction. The infection rate of sandflies is determined by the following; the biting rate, the infection probabilities of sandflies dependent on the infection status of the hosts, and the number of infectious hosts. We assume that each blood meal of a susceptible sandfly leads to a sandfly infection if taken from either an asymptomatic or symptomatic infected animal.

The following assumptions are considered in the formulation of the model:

i. Entire human infected class includes PKDL-infected people as well as early asymptomatic infected, late asymptomatic infected, and symptomatic infected people.
ii. According to each person's immunogenic potential, both early and late asymptomatic humans may eventually show symptoms and join the symptomatic infected group, and the symptomatic infected group may eventually become PKDL-infected, or they may gradually recover [19].

iii. The case where humans from the early asymptomatic class recover is not considered in this model.
iv. The capacity of the population of human, animals, and sandflies to fend off infection also plays a role in how smoothly the population moves from one compartment to another.
v. Humans acquire the disease but do not transmit.
vi. Transmission between animals and sandflies is assumed to be indirect.
vii. A fraction of late asymptomatic infected humans that are developing symptoms will receive first treatment.

Through the schematic diagram depicted in Figure 1, a system of non-linear differential equations is obtained and presented below:

$$
\begin{aligned}
\frac{dS_H}{dt} &= \Pi_H - \lambda_F S_H - \mu_H S_H, \\
\frac{dE_H}{dt} &= \lambda_F S_H - (\theta_1 + \mu_H) E_H, \\
\frac{dI_H^E}{dt} &= \theta_1 E_H - (\theta_2 + \mu_H) I_H^E, \\
\frac{dI_H^L}{dt} &= \theta_2 I_H^E - (\psi_1 + \mu_H) I_H^L, \\
\frac{dR_H^E}{dt} &= \tau_1 \psi_1 I_H^L - (\varepsilon + \mu_H) R_H^E, \\
\frac{dR_H^L}{dt} &= \varepsilon R_H^E + \varrho_1 \psi_4 R_H^1 - \mu_H R_H^L, \\
\frac{dI_H^S}{dt} &= \tau_2 \psi_1 I_H^L - (\sigma + \mu_H) I_H^S, \\
\frac{dI_H^1}{dt} &= \sigma I_H^S - (\psi_2 + \mu_H) I_H^1, \\
\frac{dI_H^2}{dt} &= \omega_3 \psi_2 I_H^1 - (\psi_3 + \mu_H) I_H^2, \\
\frac{dI_H^P}{dt} &= \delta R_H^2 - (\phi + \mu_H) I_H^P, \\
\frac{dR_H^1}{dt} &= \omega_1 \psi_2 I_H^1 + \phi I_H^P + \gamma_2 \psi_3 I_H^2 - (\varrho_1 \psi_4 + \mu_H) R_H^1, \\
\frac{dR_H^2}{dt} &= \tau_3 \psi_1 I_H^L + \omega_2 \psi_2 I_H^1 + \gamma_1 \psi_3 I_H^2 - (\delta + \mu_H) R_H^2, \\
\frac{dS_A}{dt} &= \Pi_A - \eta_F S_A - \mu_A S_A, \\
\frac{dE_A}{dt} &= \eta_F S_A - (\zeta_1 + \mu_A) E_A, \\
\frac{dI_A^A}{dt} &= \varpi_1 \zeta_1 E_A - (\zeta_2 + \mu_A) I_A^A, \\
\frac{dI_A^S}{dt} &= \varpi_2 \zeta_1 E_A + \nu_2 \zeta_2 I_A^A - (\kappa + \mu_A) I_A^S, \\
\frac{dR_A}{dt} &= \nu_1 \zeta_2 I_A^A + \kappa I_A^S - \mu_A R_A, \\
\frac{dS_F}{dt} &= \Pi_F - (\rho_A + \xi_A + \chi_A) S_F - \mu_F S_F, \\
\frac{dE_F}{dt} &= (\rho_A + \xi_A + \chi_A) S_F - (\pi + \mu_F) E_F, \\
\frac{dI_F}{dt} &= \pi E_F - \mu_F I_F,
\end{aligned}
\tag{1}
$$

subject to the following initial conditions:

$$\begin{aligned}
&S_H(0) = S_{H_0} > 0,\ E_H(0) = E_{H_0} \geq 0,\ I_H^E(0) = I_{H_0}^E \geq 0,\ I_H^L(0) = I_{H_0}^L \geq 0,\\
&R_H^E(0) = R_{H_0}^E \geq 0,\ R_H^L(0) = R_{H_0}^L \geq 0, I_H^S(0) = I_{H_0}^S \geq 0,\ I_H^1(0) = I_{H_0}^1 \geq 0,\\
&I_H^2(0) = I_{H_0}^2 \geq 0,\ I_H^P(0) = I_{H_0}^P \geq 0,\ R_H^1(0) = R_{H_0}^1 \geq 0,\ R_H^2(0) = R_{H_0}^2 \geq 0,\\
&S_A(0) = S_{A_0} > 0,\ E_A(0) = E_{A_0} \geq 0,\ I_A^A(0) = I_{A_0}^A \geq 0,\ I_A^S(0) = I_{A_0}^S \geq 0,\\
&R_A(0) = R_{A_0} \geq 0,\ S_F(0) = S_{F_0} > 0, E_F(0) = E_{F_0} \geq 0,\ I_F(0) = I_{F_0} \geq 0.
\end{aligned}$$

where:

$$\lambda_F = \frac{\beta_H g_H I_F}{N_H + N_A}, \eta_F = \frac{\beta_A g_A I_F}{N_H + N_A}, \rho_A = \frac{\beta_F g_A I_A^A}{N_H + N_A}, \xi_A = \frac{\beta_F g_A I_A^S}{N_H + N_A}, \chi_A = \frac{\beta_F g_A h_A E_A}{N_H + N_A}$$

$$\tau_1 + \tau_2 + \tau_3 = 1, \omega_1 + \omega_2 + \omega_3 = 1, \gamma_1 + \gamma_2 = 1, \varpi_1 + \varphi_2 = 1, \nu_1 + \nu_2 = 1.$$

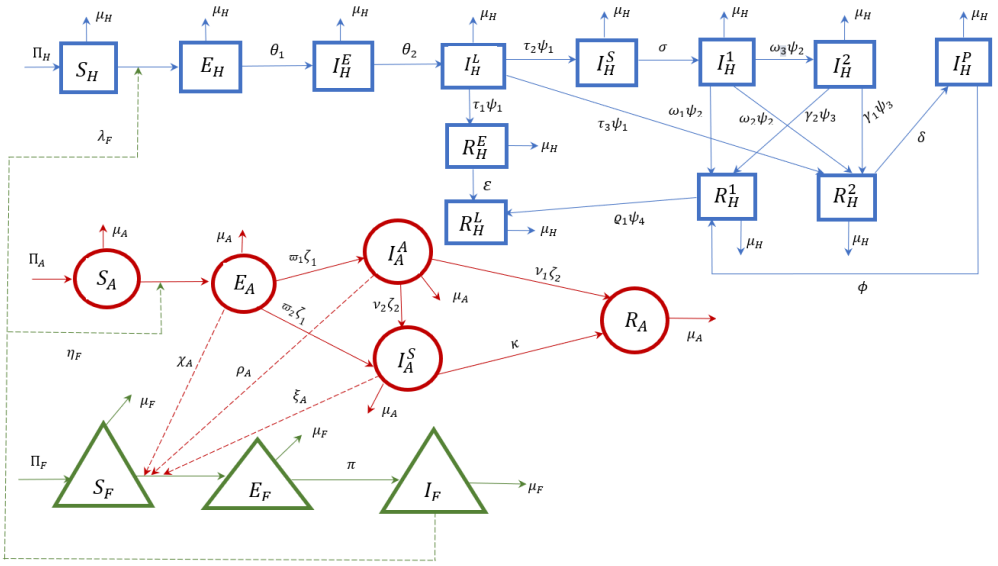


Figure 1. Schematic diagram of the VL model with early and late asymptomatic infected classes.

3. Basic Properties of the Integer Model

Here, we explore the basic properties of the model. All state variables are dependent on time, t. The total populations of humans (N_H), sandflies (N_F), and animals (N_A) are given by

$$\begin{aligned}
\frac{N_H}{dt} &= \Pi_H - \mu_H N_H,\\
\frac{N_A}{dt} &= \Pi_A - \mu_A N_A,\\
\frac{N_F}{dt} &= \Pi_F - \mu_F N_F,
\end{aligned} \tag{2}$$

with the initial conditions

$$\begin{aligned}
&S_H(0) = S_{H_0} > 0,\ E_H(0) = E_{H_0} \geq 0,\ I_H^E(0) = I_{H_0}^E \geq 0,\ I_H^L(0) = I_{H_0}^L \geq 0,\\
&R_H^E(0) = R_{H_0}^E \geq 0,\ R_H^L(0) = R_{H_0}^L \geq 0,\ I_H^S(0) = I_{H_0}^S \geq 0,\ I_H^1(0) = I_{H_0}^1 \geq 0,\\
&I_H^2(0) = I_{H_0}^2 \geq 0,\ I_H^P(0) = I_{H_0}^P \geq 0,\ R_H^1(0) = R_{H_0}^1 \geq 0,\ R_H^2(0) = R_{H_0}^2 \geq 0,\\
&S_A(0) = S_{A_0} > 0,\ E_A(0) = E_{A_0} \geq 0,\ I_A^A(0) = I_{A_0}^A \geq 0, I_A^S(0) = I_{A_0}^S > 0,\\
&R_A(0) = R_{A_0} \geq 0,\ S_F(0) = S_{F_0} \geq 0,\ E_F(0) = E_{F_0} \geq 0,\ I_F(0) = I_{F_0} \geq 0.
\end{aligned}$$

Therefore, in order for the solutions to the model (1) with non-negative initial data to have biological significance, it is necessary to demonstrate that they will always remain non-negative $t > 0$.

Positivity of Solution

Since the model (1) describes the human population, here, we show that all the state variables that are non-negative for all $t \geq 0$ solutions are all bounded. Before analyzing the model, it is important to show that the state variables of the model remain non-negative for all non-negative initial conditions. We claim the following result.

Theorem 1. *Let $S_H > 0, E_H \geq 0, ..., I_F \geq 0$. The solutions $S_H, E_H, ..., I_F$ of the model system (1) for $t \geq 0$ are positive. For the model system (1), the region Ω is positively invariant and all solutions starting in Ω approach, enter, or stay in Ω, where $\Omega = \Omega_H \cup \Omega_A \cup \Omega_F \in \mathbb{R}^{12}_{+} \times \mathbb{R}^{5}_{+} \times \mathbb{R}^{3}_{F+}$:*

$$\begin{aligned}
&\Omega_H = (S_H, E_H, I_H^E, I_H^L, I_H^S, R_H^E, R_H^L, I_H^1, I_H^2, I_H^P, R_H^1, R_H^2) \in \mathbb{R}^{12}_{H+},\\
&\Omega_A = (S_A, E_A, I_A^A, I_A^S, E_A, R_A) \in \mathbb{R}^{5}_{A+},\\
&\Omega_F = (S_F, E_F, I_F) \in \mathbb{R}^{3}_{F+}.
\end{aligned}$$

Proof. We use the method of contradiction as in [20] to prove Theorem (1). Under the given initial conditions, it is straightforward to prove that the components of the solutions of the model system (1) are always positive; otherwise, we assume a contradiction. We claim there exists a first time, t_1

$$\begin{aligned}
t_1\text{: } &S_H(t_1) = 0, \tfrac{dS_H}{dt_1} < 0, E_H(t) > 0, I_H^E(t) > 0, ..., I_F(t) > 0 \text{ for } 0 < t < t_2 \text{ or claim } \exists\\
t_2\text{: } &E_H(t_2) = 0, \tfrac{dE_H}{dt_2} < 0, S_H(t) > 0, I_H^E(t) > 0, ..., I_F(t) > 0 \text{ for } 0 < t < t_3 \text{ or claim } \exists\\
t_3\text{: } &I_H^E(t_3) = 0, \tfrac{dI_H^E}{dt_3} < 0, S_H(t) > 0, E_H(t) > 0, ..., I_F(t) > 0 \text{ for } 0 < t < t_1 \text{ or claim } \exists\\
t_4\text{: } &I_H^L(t_4) = 0, \tfrac{dI_H^L}{dt_4} < 0, S_H(t) > 0, E_H(t) > 0, ..., I_F(t) > 0 \text{ for } 0 < t < t_4 \text{ or claim } \exists\\
t_5\text{: } &R_H^E(t_5) = 0, \tfrac{dR_H^E}{dt_5} < 0, S_H(t) > 0, E_H(t) > 0, ..., I_F(t) > 0 \text{ for } 0 < t < t_5 \text{ or claim } \exists\\
t_6\text{: } &R_H^L(t_6) = 0, \tfrac{dR_H^L}{dt_6} < 0, S_H(t) > 0, E_H(t) > 0, ..., I_F(t) > 0 \text{ for } 0 < t < t_6 \text{ or claim } \exists\\
t_7\text{: } &I_H^S(t_7) = 0, \tfrac{dI_H^S}{dt_7} < 0, S_H(t) > 0, E_H(t) > 0, ..., I_F(t) > 0 \text{ for } 0 < t < t_7 \text{ or claim } \exists\\
t_8\text{: } &I_H^1(t_8) = 0, \tfrac{dI_H^1}{dt_1} < 0, S_H(t) > 0, E_H(t) > 0, ..., I_F(t) > 0 \text{ for } 0 < t < t_8 \text{ or claim } \exists\\
t_9\text{: } &I_H^2(t_9) = 0, \tfrac{dI_H^2}{dt_8} < 0, S_H(t) > 0, E_H(t) > 0, ..., I_F(t) > 0 \text{ for } 0 < t < t_9 \text{ or or claim } \exists\\
t_{10}\text{: } &I_H^P(t_{10}) = 0, \tfrac{dI_H^P}{dt_{10}} < 0, S_H(t) > 0, E_H(t) > 0, ..., I_F(t) > 0 \text{ for } 0 < t < t_{10} \text{ or claim } \exists\\
t_{11}\text{: } &R_H^1(t_{11}) = 0, \tfrac{dR_H^1}{dt_{11}} < 0, S_H(t) > 0, E_H(t) > 0, ..., I_F(t) > 0 \text{ for } 0 < t < t_{11} \text{ or claim } \exists\\
t_{12}\text{: } &R_H^2(t_{12}) = 0, \tfrac{dR_H^2}{dt_{12}} < 0, S_H(t) > 0, E_H(t) > 0, ..., I_F(t) > 0 \text{ for } 0 < t < t_{12} \text{ or claim } \exists\\
t_{13}\text{: } &S_A(t_{13}) = 0, \tfrac{dS_A}{dt_{13}} < 0, S_H(t) > 0, E_H(t) > 0, ..., I_F(t) > 0 \text{ for } 0 < t < t_{13} \text{ or claim } \exists\\
t_{14}\text{: } &E_A(t_{14}) = 0, \tfrac{dE_A}{dt_{14}} < 0, S_H(t) > 0, E_H(t) > 0, ..., I_F(t) > 0 \text{ for } 0 < t < t_{14} \text{ or claim } \exists\\
t_{15}\text{: } &I_A^A(t_{15}) = 0, \tfrac{dI_A^A}{dt_{15}} < 0, S_H(t) > 0, E_H(t) > 0, ..., I_F(t) > 0 \text{ for } 0 < t < t_{15} \text{ or claim } \exists\\
t_{16}\text{: } &I_A^S(t_{16}) = 0, \tfrac{dI_A^S}{dt_{16}} < 0, S_H(t) > 0, E_H(t) > 0, ..., I_F(t) > 0 \text{ for } 0 < t < t_{16} \text{ or claim } \exists\\
t_{17}\text{: } &R_A(t_{17}) = 0, \tfrac{dR_A}{dt_{17}} < 0, S_H(t) > 0, E_H(t) > 0, ..., I_F(t) > 0 \text{ for } 0 < t < t_{17} \text{ or claim } \exists\\
t_{18}\text{: } &S_F(t_{18}) = 0, \tfrac{dS_F}{dt_{18}} < 0, S_H(t) > 0, E_H(t) > 0, ..., I_F(t) > 0 \text{ for } 0 < t < t_{18} \text{ or claim } \exists\\
t_{19}\text{: } &E_F(t_{19}) = 0, \tfrac{dE_A}{dt_{19}} < 0, S_H(t) > 0, E_H(t) > 0, ..., I_F(t) > 0 \text{ for } 0 < t < t_{19} \text{ or claim } \exists\\
t_{20}\text{: } &I_F(t_{20}) = 0, \tfrac{dI_A}{dt_{20}} < 0, S_H(t) > 0, E_H(t) > 0, ..., E_F(t) > 0 \text{ for } 0 < t < t_{20}.
\end{aligned}$$

In the first claim, we have

$$\frac{S_H}{dt_1} = \Pi_H > 0, \tag{3}$$

which is a contradiction to the assumption. That is, S_H remains positive.

In the second claim, we have

$$\frac{dE_H}{dt_2} = \lambda_F S_H > 0, \tag{4}$$

which is a contradiction to the assumption. That is, E_H remains positive.

In the third claim, we have

$$\frac{dI_H^E}{dt_3} = \theta_1 E_H > 0, \tag{5}$$

which is a contradiction to the assumption. That is, I_H^E remains positive.

In the fourth claim, we have

$$\frac{dI_H^L}{dt_4} = \theta_2 I_H^E > 0, \tag{6}$$

which is a contradiction to the assumption. That is, I_H^L remains positive.

In the fifth claim, we have

$$\frac{dR_H^E}{dt_5} = \tau_1 \psi_1 I_H^L > 0, \tag{7}$$

which is a contradiction to the assumption. That is, R_H^E remains positive.

In the sixth claim, we have

$$\frac{dR_H^L}{dt_6} = \varepsilon R_H^E + \varrho_1 \psi_4 R_H^1 > 0, \tag{8}$$

which is a contradiction to the assumption. That is, R_H^L remains positive.

In the seventh claim, we have

$$\frac{dI_H^S}{dt_7} = \tau_2 \psi_1 I_H^L > 0, \tag{9}$$

which is a contradiction to the assumption. That is, I_H^S remains positive.

In the eighth claim, we have

$$\frac{dI_H^1}{dt_8} = \sigma I_H^S > 0, \tag{10}$$

which is a contradiction to the assumption. That is, I_H^1 remains positive.

In the ninth claim, we have

$$\frac{dI_H^2}{dt_9} = \omega_3 \psi_2 I_H^1 > 0, \tag{11}$$

which is a contradiction to the assumption. That is, I_H^2 remains positive.

In the tenth claim, we have

$$\frac{dI_H^P}{dt_{10}} = \delta R_H^2 > 0, \tag{12}$$

which is a contradiction to the assumption. That is, I_H^P remains positive.

In the eleventh claim, we have

$$\frac{dR_H^1}{dt_{11}} = \omega_1\psi_2 I_H^1 + \phi I_H^P + \gamma_2\psi_3 I_H^2 > 0, \tag{13}$$

which is a contradiction to the assumption. That is, R_H^1 remains positive.

In the twelfth claim, we have

$$\frac{dR_H^2}{dt_{12}} = \tau_3\psi_1 I_H^L + \omega_2\psi_2 I_H^1 + \gamma_1\psi_3 I_H^2 > 0, \tag{14}$$

which is a contradiction to the assumption. That is, R_H^2 remains positive.

In the thirteenth claim, we have

$$\frac{dS_A}{dt_{13}} = \Pi_A > 0, \tag{15}$$

which is a contradiction to the assumption. That is, S_A remains positive.

In the fourteenth claim, we have

$$\frac{dE_A}{dt_{14}} = \eta_F S_A > 0, \tag{16}$$

which is a contradiction to the assumption. That is, E_A remains positive.

In the fifteenth claim, we have

$$\frac{dI_A^A}{dt_{15}} = \varpi_1\zeta_1 E_A > 0, \tag{17}$$

which is a contradiction to the assumption. That is, I_A^A remains positive.

In the sixteenth claim, we have

$$\frac{dI_A^S}{dt_{16}} = \varpi_2\zeta_1 E_A + \nu_2\zeta_2 I_A^A > 0, \tag{18}$$

which is a contradiction to the assumption. That is, I_A^S remains positive.

In the seventeenth claim, we have

$$\frac{dR_A}{dt_{17}} = \nu_1\zeta_2 I_A^A + \kappa I_A^S > 0, \tag{19}$$

which is a contradiction to the assumption. That is, R_A remains positive.

In the eighteenth claim, we have

$$\frac{dS_F}{dt_{18}} = \Pi_F, \tag{20}$$

which is a contradiction to the assumption. That is, S_F remains positive.

In the nineteenth claim, we have

$$\frac{dE_F}{dt_{19}} = (\rho_A + \xi_A + \chi_A)S_F > 0, \tag{21}$$

which is a contradiction to the assumption. That is, E_F remains positive.

In the twentieth claim, we have

$$\frac{dI_F}{dt_{20}} = \pi E_F > 0, \tag{22}$$

which is a contradiction to the assumption. That is, I_F remains positive.

Therefore, all solutions of the model (1) remain positive for all non-negative initial conditions as required. □

Also, since $\frac{dN_H}{dt} = \Pi_H - \mu_H N_H$, $\frac{dN_A}{dt} = \Pi_A - \mu_A N_A$, $\frac{dN_F}{dt} = \Pi_F - \mu_F N_F$, this means that $N_H \longrightarrow \frac{\Pi_H}{\mu_H}$, $N_A \longrightarrow \frac{\Pi_A}{\mu_A}$ and $N_F \longrightarrow \frac{\Pi_F}{\mu_F}$ are bounded. Based on biological considerations, the model system (1) is studied in the following feasible region:

$$\begin{aligned}\Omega = \{&(S_H,\ E_H,\ I_H^E,\ I_H^L,\ R_H^E,\ R_H^L,\ I_H^S,\ I_H^1,\ I_H^2,\ I_H^P,\ R_H^1,\ R_H^2,\ S_A,\ E_A,\\ &I_A^A,\ I_A^S,\ R_A,\ S_F,\ E_F,\ I_F)\\ \in \mathbb{R}_+^{20} = \mathbb{R}_{H+}^{12} \times \mathbb{R}_{A+}^{5} \times \mathbb{R}_{F+}^{3} &: N_H(t) \leq \frac{\Pi_H}{\mu_H},\ N_A(t) \leq \frac{\Pi_A}{\mu_A},\ N_F(t) \leq \frac{\Pi_F}{\mu_F}\}. \end{aligned} \tag{23}$$

This region is positively invariant with respect to the model system (1). This also mean that, all solutions of the model (1), with initial conditions in Ω, will remain in Ω for all $t \geq 0$. We can conveniently consider the solutions of the model in Ω. (see also, [21]). Combining this result and Theorem (1), we have the following lemma:

Lemma 1. *The region Ω is positively-invariant for the model 1 with initial conditions in $\mathbb{R}_+^{20} = \mathbb{R}_{H+}^{12} \times \mathbb{R}_{A+}^{5} \times \mathbb{R}_{F+}^{3}$.*

Additionally, it is easy to see that each of the differential equations of the model system (1) is Lipschitz continuous with the given initial conditions and has solutions. Moreover, the solution is unique and since Ω is a positively invariant region, the solution exist for any time $t \geq 0$ (see, [22]).

4. Fixed Points of the Model and Their Stability Analysis

In this section, we explore the existence and stability of fixed points of the model (1).

4.1. VL-Free Fixed Point

Let $x = x^*$ be the VL-free fixed point for the system (1). Then, $f(x^*) = 0$, $\frac{dx}{dt} = f(x)$, $x = (x_1, x_2, \ldots, x_{20})^T$, $x_1 = S_H, x_2 = E_H, \ldots, x_{20} = I_F$ in order of the model variables. This implies that $\frac{dx^*}{dt} = f(x^*)$, $x^* = (x_1^*, x_2^*, \ldots, x_{20}^*)^T$, $x_1^* = S_H^*, x_2^* = E_H^*, \ldots, x_{20}^* = I_F^*$.

Thus, at fixed point $f(x^*) = 0$, implies

$$\Pi_H - \lambda_F S_H^* - \mu_H S_H^* = 0, \tag{24}$$
$$\lambda_F S_H^* - (\theta_1 + \mu_H) E_H^* = 0, \tag{25}$$
$$\theta_1 E_H^* - (\theta_2 + \mu_H) I_H^{E*} = 0, \tag{26}$$
$$\theta_2 I_H^{E*} - (\psi_1 + \mu_H) I_H^{L*} = 0, \tag{27}$$
$$\tau_1 \psi_1 I_H^{L*} - (\varepsilon + \mu_H) R_H^{E*} = 0, \tag{28}$$
$$\varepsilon R_H^{E*} + \varrho_1 \psi_4 R_H^{1*} - \mu_H R_H^{L*} = 0, \tag{29}$$
$$\tau_2 \psi_1 I_H^{L*} - (\sigma + \mu_H) I_H^{S*} = 0, \tag{30}$$
$$\sigma I_H^{S*} - (\psi_2 + \mu_H) I_H^{1*} = 0, \tag{31}$$
$$\omega_3 \psi_2 I_H^{1*} - (\psi_3 + \mu_H) I_H^{2*} = 0, \tag{32}$$
$$\delta R_H^{2*} - (\phi + \mu_H) I_H^{P*} = 0, \tag{33}$$
$$\omega_1 \psi_2 I_H^{1*} + \phi I_H^{P*} + \gamma_2 \psi_3 I_H^{2*} - (\varrho_1 \psi_4 + \mu_H) R_H^{1*} = 0, \tag{34}$$
$$\tau_3 \psi_1 I_H^{L*} + \omega_2 \psi_2 I_H^{1*} + \gamma_1 \psi_3 I_H^{2*} - (\delta + \mu_H) R_H^{2*} = 0, \tag{35}$$
$$\Pi_A - \eta_F S_A^* - \mu_A S_A^* = 0, \tag{36}$$
$$\eta_F S_A^* - (\zeta_1 + \mu_A) E_A^* = 0, \tag{37}$$
$$\omega_1 \zeta_1 E_A^* - (\zeta_2 + \mu_A) I_A^{A*} = 0, \tag{38}$$

$$\omega_2\zeta_1 E_A^* + \nu_2\zeta_2 I_A^{A*} - (\kappa + \mu_A) I_A^{S*} = 0, \tag{39}$$
$$\nu_1\zeta_2 I_A^{A*} + \kappa I_A^{S*} - \mu_A R_A^* = 0, \tag{40}$$
$$\Pi_F - (\rho_A + \xi_A + \chi_A) S_F^* - \mu_F S_F^* = 0, \tag{41}$$
$$(\rho_A + \xi_A + \chi_A) S_F^* - (\pi + \mu_F) E_F^* = 0, \tag{42}$$
$$\pi E_F^* - \mu_F I_F^* = 0. \tag{43}$$

At the VL-free fixed point, it means there is no presence of the disease, meaning $E_H = 0$, $I_H^E = 0$, $I_H^L = 0$, $R_H^E = 0$, $R_H^L = 0$, $I_H^S = 0$, $I_H^1 = 0$, $I_H^2 = 0$, $I_H^P = 0$, $R_H^1 = 0$, $R_H^2 = 0$, $E_A = 0$, $I_A^A = 0$, $I_A^S = 0$, $R_A = 0$, $E_F = 0$, $I_F = 0$.

Thus, we have

$$\begin{aligned}
\Pi_H - \mu_H S_H^* = 0 &\iff S_H^* = \tfrac{\Pi_H}{\mu_H}, \\
\Pi_A - \mu_A S_A^* = 0 &\iff S_A^* = \tfrac{\Pi_A}{\mu_A}, \\
\Pi_F - \mu_F S_F^* = 0 &\iff S_F^* = \tfrac{\Pi_F}{\mu_F}.
\end{aligned}$$

Therefore, the VL-free fixed point is given by

$$\begin{aligned}
x^* &= (x_1^*, x_2^*, \ldots, x_{20}^*)^T, \\
&= (S_H^*, E_H^*, \ldots, I_F^*)^T.
\end{aligned}$$

Hence, the VL-free fixed point, denoted by E^0, is given by

$$E^0 = \left(\frac{\Pi_H}{\mu_H}, 0, 0, 0, 0, 0, 0, 0, 0, 0, 0, \frac{\Pi_A}{\mu_A}, 0, 0, 0, \frac{\Pi_F}{\mu_F}, 0, 0\right). \tag{44}$$

4.2. Basic Reproduction Number R_0

The number of secondary infections caused by a single infectious individual over the course of their whole infectious period is known as the basic reproduction number. The basic reproduction number can be expressed mathematically as a spectral radius. The number of new infections caused by a single infected person in a community that is totally susceptible is defined by the spectral radius R_0, a threshold parameter for disease control [23]. The number of VL infections caused by an active VL case is what we refer to as the basic reproduction number, or R_0, in this instance. To ascertain the model system's basic reproductive number, we employ the method of next generation matrix described in [23] to determine the basic reproductive number of the system (1). The matrices $\mathcal{F}$ and $\mathcal{V}$, for the new infection terms and the remaining transfer terms, are, respectively, given by

$$\mathcal{F} = \begin{pmatrix} \lambda_F S_H \\ 0 \\ 0 \\ 0 \\ 0 \\ 0 \\ 0 \\ \eta_F S_A \\ 0 \\ 0 \\ (\rho_A + \xi_A + \chi_A) S_F \\ 0 \end{pmatrix}, \mathcal{V} = \begin{pmatrix} (\theta_1 + \mu_H) E_H \\ -\theta_1 E_H + (\theta_2 + \mu_H) I_H^E \\ -\theta_2 I_H^E + (\psi_1 + \mu_H) I_H^L \\ -\tau_2 \psi_1 I_H^L + (\sigma + \mu_H) I_H^S \\ -\sigma I_H^S + (\psi_2 + \mu_H) I_H^1 \\ -\omega_3 \psi_2 I_H^1 + (\psi_3 + \mu_H) I_H^2 \\ -\delta R_H^2 + (\phi + \mu_H) I_H^P \\ (\zeta_1 + \mu_A) E_A \\ -\omega_1 \zeta_1 E_A + (\zeta_2 + \mu_A) I_A^A, \\ -\omega_2 \zeta_1 E_A - \nu_2 \zeta_2 I_A^A + (\kappa + \mu_A) I_A^S \\ (\pi + \mu_F) E_F \\ -\pi E_F + \mu_F I_F \end{pmatrix}.$$

We then obtain the matrices F and V at the VL-free fixed point, E^0. Thus, we have

$$F = \begin{pmatrix} 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & b_1 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & b_2 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & b_3 & b_4 & b_4 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \end{pmatrix}, \tag{45}$$

$b_1 = \frac{\beta_H g_H \Pi_H \mu_A}{\Pi_H \mu_A + \Pi_A \mu_H}, b_2 = \frac{\beta_A g_A \Pi_A \mu_H}{\Pi_H \mu_A + \Pi_A \mu_H}, b_3 = \frac{\beta_F g_A \Pi_F \mu_A \mu_H}{\mu_F(\Pi_H \mu_A + \Pi_A \mu_H)}, b_4 = \frac{\beta_F g_A h_A \Pi_F \mu_A \mu_H}{\mu_F(\Pi_H \mu_A + \Pi_A \mu_H)}$

$$V = \begin{pmatrix} a_{11} & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ -\theta_1 & a_{12} & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & -\theta_2 & a_{13} & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & -a_{22} & a_{14} & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & -\sigma & a_{15} & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & -a_{23} & a_{16} & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & a_{17} & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & a_{18} & 0 & 0 & 0 & \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & -a_{24} & a_{19} & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & -a_{25} & -a_{26} & a_{20} & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & a_{21} & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & -\pi & \mu_F \end{pmatrix}, \tag{46}$$

where

$$\begin{aligned} a_{11} = \theta_1 + \mu_H, a_{12} = \theta_2 + \mu_H, a_{13} = \psi_1 + \mu_H, a_{14} = \sigma + \mu_H, a_{15} = \psi_2 + \mu_H, \\ a_{16} = \psi_3 + \mu_H, a_{17} = \phi + \mu_H, a_{18} = \zeta_1 + \mu_A, a_{19} = \zeta_2 + \mu_A, a_{20} = \kappa + \mu_A, \\ a_{21} = \pi + \mu_A, a_{22} = \tau_2\psi_1, a_{23} = \omega_2\psi_2, a_{24} = \varpi_1\zeta_1, a_{25} = \varpi_2\zeta_1, a_{26} = \nu_2\zeta_2. \end{aligned}$$

the determinant of V is obtained to be

$$det(V) = a_{11}a_{12}a_{13}a_{14}a_{15}a_{16}a_{17}a_{18}a_{19}a_{20}a_{21}\mu_F.$$

$$V^{-1} = \begin{pmatrix} \frac{1}{a_{11}} & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ A_1 & \frac{1}{a_{12}} & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ A_2 & A_3 & \frac{1}{a_{13}} & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ A_4 & A_5 & A_6 & \frac{1}{a_{14}} & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ A_7 & A_8 & A_9 & A_{10} & -a_{15} & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ A_{11} & A_{12} & A_{13} & A_{14} & A_{15} & \frac{1}{a_{16}} & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & \frac{1}{a_{17}} & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & \frac{1}{a_{18}} & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & A_{16} & \frac{1}{a_{19}} & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & A_{17} & A_{18} & \frac{1}{a_{20}} & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & \frac{1}{a_{21}} & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & A_{19} & \frac{1}{\mu_F} \end{pmatrix} \tag{47}$$

$$A_1 = \frac{\theta_1}{a_{11}a_{12}},\ A_2 = \frac{\theta_1\theta_2}{a_{11}a_{12}a_{13}},\ A_3 = \frac{\theta_2}{a_{12}a_{13}},\ A_4 = \frac{\theta_1\theta_2 a_{22}}{a_{11}a_{12}a_{13}a_{14}}, A_5 = \frac{\theta_2 a_{22}}{a_{12}a_{13}a_{14}},$$
$$A_6 = \frac{a_{22}}{a_{13}a_{14}}, A_7 = \frac{\theta_1\theta_2\sigma a_{22}}{a_{11}a_{12}a_{13}a_{14}a_{15}}, A_8 = \frac{\theta_2\sigma a_{22}}{a_{12}a_{13}a_{14}}, A_9 = \frac{\sigma a_{22}}{a_{13}a_{14}}, A_{10} = \frac{\sigma}{a_{14}},$$
$$A_{11} = \frac{\theta_1\theta_2\sigma a_{22}a_{22}}{a_{11}a_{12}a_{13}a_{14}a_{16}}, A_{12} = \frac{\theta_2\sigma a_{22}a_{29}}{a_{12}a_{13}a_{14}a_{16}}, A_{13} = \frac{\sigma a_{22}a_{23}}{a_{13}a_{14}a_{16}}, A_{14} = \frac{\sigma a_{23}}{a_{14}a_{16}}, A_{15} = \frac{a_{29}}{a_{16}},$$
$$A_{16} = \frac{a_{24}}{a_{18}a_{25}}, A_{17} = \frac{a_{19}a_{25}+a_{24}a_{26}}{a_{18}a_{15}a_{20}}, A_{18} = \frac{a_{26}}{a_{25}a_{20}}, A_{19} = \frac{\pi}{\mu_F a_{21}}.$$

Thus, we have

$$FV^{-1} = \begin{pmatrix} 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & \frac{\pi b_1}{\mu_F a_{21}} & \frac{b_1}{\mu_F} \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & \frac{\pi b_2}{\mu_F a_{21}} & \frac{b_2}{\mu_F} \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & B_1 & B_2 & \frac{b_4}{a_{20}} & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \end{pmatrix}, \tag{48}$$

where

$$B_1 = \frac{b_4 a_{24}(a_{20}+a_{26}) + a_{25}b_4(a_{20}+a_{25})}{a_{18}a_{25}a_{20}}, B_2 = \frac{a_{20}+a_{26}}{a_{25}a_{20}}.$$

The spectral radius $\rho(FV^{-1}) = R_0$, the basic reproduction number to the system, which is the dominant eigenvalue, after simplifying, is given as

$$R_0 = \sqrt{\frac{\pi(a_{20}b_3 + b_2b_4a_{24}a_{25}) + \pi b_4 a_{24}(a_{26}+a_{20}b_2)}{\mu_F a_{18}a_{20}a_{21}a_{25}}}. \tag{49}$$

4.3. Local Stability Analysis of VL-Free Fixed Point

To analyze the stability of the VL-free fixed point, we linearize the non-linear system (1) by taking a small perturbation about the fixed points.

Since the decomposed matrices $\mathcal{F}$ and $\mathcal{V}$ exist, which satisfy conditions (A1)–(A5) in [23], it follows from Theorem 2 of the same [23] that we have the following.

Lemma 2. *The VL-free E^0 of the model given by (1) is locally asymptotically stable (LAS) whenever $R_0 < 1$, and unstable when $R_0 > 1$.*

The epidemiological implication of Lemma 2 is that a small influx of infectious individuals will not generate large outbreaks in the population if $R_0 < 1$. Basically, R_0 calculates the typical number of secondary cases in a population that is fully susceptible that are caused by a single infectious individual. According to Lemma 2, if $R_0 < 1$, then a modest excess of infectious individuals will not cause significant outbreaks in the community. For the VL-free E^0 of the model when $R_0 < 1$, a global asymptotic stability (GAS) property needs to be provided for the disease elimination to be independent of the initial sizes of the populations of the model. We examine now the global asymptotic stability (GAS) of the VL-free fixed point, E^0.

4.4. Global Stability Analysis of the VL-Free Fixed Point

The basic reproduction number of the model is the threshold quantity R_0 as obtained in (49). It measures how many new infected cells, on average, are produced [21,23]. It is implied by Lemma 1 that the disease may die out when $R_0 < 1$ and the initial sizes of the

model's subpopulations are within Ω. It is crucial to demonstrate that the VL-free fixed point is globally asymptotically stable to guarantee the independence of disease elimination when $R_0 < 1$.

We explore the global asymptotic stability of the VL-free fixed point E^0 of the model using a Lyapunov functional.

Theorem 2. *Suppose that* $\mu_F a_{18} a_{19} a_{20} a_{21} \geq \pi \beta_A \beta_F g_A^2 [a_{24}(a_{20} + a_{26}) + a_{19}(a_{20} h_A + a_{25})]$ *and* $\pi a_{24} b_2 b_4 > \mu_F a_{18} a_{20} a_{21}$, *the VL-free fixed point* (E^0) *is globally asymptotically stable in the region* Ω *whenever* $R_0 < 1$.

Proof. Consider the Lyapunov function

$$V = g_1 E_H + I_H^E + I_H^L + I_H^1 + I_H^2 + I_H^S + g_2 I_H^P + g_3 E_A + g_4 I_A^A + g_5 I_A^S + g_6 E_F + g_7 I_F, \quad (50)$$

where

$$\begin{aligned}
g_1 &= \tfrac{\mu_F a_{18} a_{19} a_{20} a_{21} - \pi \beta_A \beta_F g_A^2 [a_{24}(a_{20}+a_{26}) + a_{19}(a_{20} h_A + a_{25})]}{a_{18} \pi \beta_H g_H}, \\
g_2 &= \pi a_{20} b_3 + \pi a_{24} a_{26} b_4 + \pi a_{20} a_{24} b_2 b_4 + a_25(\pi a_{24} b_2 b_4 - \mu_F a_{18} a_{20} a_{21}), \\
g_3 &= \tfrac{\beta_F g_A [a_{24}(a_{20}+a_{26}) + a_{19}(a_{20} h_A + a_{25})]}{a_{18}}, \\
g_4 &= \beta_F g_A (a_{20} + a_{26}), \\
g_5 &= a_{19} \beta_F g_A, \\
g_6 &= a_{19} a_{20}, \\
g_7 &= \tfrac{a_{19} a_{20} a_{21}}{\pi},
\end{aligned}$$

with the Lyapunov derivative along the solution curve

$$\dot{V} = g_1 \dot{E_H} + \dot{I_H^E} + \dot{I_H^L} + \dot{I_H^1} + \dot{I_H^2} + \dot{I_H^S} + g_2 \dot{I_H^P} + g_3 \dot{E_A} + g_4 \dot{I_A^A} + g_5 \dot{I_A^S} + g_6 \dot{E_F} + g_7 \dot{I_F},$$

$$\begin{aligned}
\dot{E}_H = \tfrac{dE_H}{dt},\ \dot{I}_H^E = \tfrac{dI_H^E}{dt},\ \dot{I}_H^L = \tfrac{dI_H^L}{dt}\ \dot{I}_H^1 = \tfrac{dI_H^1}{dt},\ \dot{I}_H^2 = \tfrac{dI_H^2}{dt},\ \dot{I}_H^S = \tfrac{dI_H^S}{dt},\ \dot{I}_H^P = \tfrac{dI_H^P}{dt}, \\
\dot{E}_A = \tfrac{dE_A}{dt}, \dot{I}_A^A = \tfrac{dI_A^A}{dt},\ \dot{I}_A^S = \tfrac{dI_A^S}{dt},\ \dot{E}_F = \tfrac{dE_F}{dt}\ \dot{I}_F = \tfrac{dI_F}{dt}.
\end{aligned}$$

Substituting the derivatives of each from the system (1) in $\dot{V}$, we have

$$\begin{aligned}
\dot{V} &= g_1[\lambda_F S_H - (\theta_1 + \mu_H) E_H] + [\theta_1 E_H - (\theta_2 + \mu_H) I_H^E] + [\theta_2 I_H^E - (\psi_1 + \mu_H) I_H^L] \\
&+ [\sigma I_H^S - (\psi_2 + \mu_H) I_H^1] + [\omega_3 \psi_2 I_H^1 - (\psi_3 + \mu_H) I_H^2] + [\tau_2 \psi_1 I_H^L - (\sigma + \mu_H) I_H^S] \\
&+ g_2[\delta R_H^2 - (\phi + \mu_H) I_H^P] + g_3[\eta_F S_A - (\zeta_1 + \mu_A) E_A] + g_4[\varpi_1 \zeta_1 E_A - (\zeta_2 + \mu_A) I_A^A] \\
&+ g_5[\varpi_2 \zeta_1 E_A + \nu_2 \zeta_2 I_A^A - (\kappa + \mu_A) I_A^S] + g_6[(\rho_A + \xi_A + \chi_A) S_F - (\pi + \mu_F) E_F] \\
&+ g_7[\pi E_F - \mu_F I_F], \\
&= g_1 \lambda_F S_H + g_3 \eta_F S_A + g_6(\rho_A + \xi_A + \chi_A) S_F + [\theta_1 - (\theta_1 + \mu_H)] E_H + [\theta_2 \\
&- (\theta_2 + \mu_H)] I_H^E + [\tau_2 \psi_1 - (\psi_1 + \mu_H)] I_H^L + [\sigma - (\sigma + \mu_H)] I_H^S + [\omega_3 \psi_2 \\
&- (\psi_2 + \mu_H)] I_H^1 - (\psi_3 + \mu_H) I_H^2 - g_2(\phi + \mu_H) I_H^P + g_2 \delta R_H^2 + [g_4 \varpi_1 \zeta_1 \\
&- g_3(\zeta_1 + \mu_A) + g_5 \varpi_2 \zeta_1] E_A + [g_5 \nu_2 \zeta_2 - g_4(\zeta_2 + \mu_A)] I_A^A - g_5(\kappa + \mu_A) I_A^S \\
&+ [g_7 \pi - g_6(\pi + \mu_F)] E_F - g_7 \mu_F I_F. \qquad (51)
\end{aligned}$$

Now,

$$\begin{aligned}
g_1 \lambda_F S_H &= \tfrac{g_1 \beta_H g_H I_F}{N_H + N_A} S_H < g_1 \beta_H g_H I_F, \\
g_3 \eta_F S_A &= \tfrac{g_3 \beta_A g_A I_F}{N_H + N_A} S_A < g_3 \beta_A g_A I_F, \\
g_6(\rho_A + \xi_A + \chi_A) S_F &= \tfrac{g_6(\beta_F g_A I_A^A + \beta_F g_A I_A^S + \beta_F g_A h_A E_A)}{N_H + N_A} S_F < g_6(\beta_F g_A I_A^A + \beta_F g_A I_A^S + \beta_F g_A h_A E_A).
\end{aligned}$$

Also, using the fact that if $a,\ b,\ c > 0$, then $a + b - c < a + b$, dropping $g_2(\phi + \mu_H)I_H^P$, and simplifying the term according to the variable, (51) becomes

$$\begin{aligned}
\leq\ & [\theta_1 - (\theta_1 + \mu_H)]E_H + [\theta_2 - (\theta_2 + \mu_H)]I_H^E + [\tau_2\psi_1 - (\psi_1 + \mu_H)]I_H^L + [\sigma - (\sigma + \mu_H)]I_H^S \\
+\ & [\omega_3\psi_2 - (psi_2 + \mu_H)]I_H^1 - (\psi_3 + \mu_H)I_H^2 + g_2\delta R_H^2 + [g_4\omega_1\zeta_1 - g_3(\zeta_1 + \mu_A) + g_5\omega_2\zeta_1 \\
+\ & g_6\beta_F g_A h_A]E_A + [g_5\nu_2\zeta_2 - g_4(\zeta_2 + \mu_A) + g_6\beta_F g_A]I_A^A + [g_6\beta_F g_A - g_5(\kappa + \mu_A)I_A^S \\
+\ & [g_7\pi - g_6(\pi + \mu_F)]E_F + [g_1\beta_H g_H + g_3\beta_A g_A - g_7\mu_F]I_F, \\
=\ & -\mu_H E_H - \mu_H I_H^E - (\tau_1\psi_1 + \tau_3\psi_1 + \mu_H)I_H^L - \mu_H I_H^S - (\omega_1\psi_2 + \omega_2\psi_2 + \mu_H)I_H^1 \\
-\ & (\psi_3 + \mu_H)I_H^2 + g_2\delta R_H^2 + [g_4\omega_1\zeta_1 - g_3(\zeta_1 + \mu_A) + g_5\omega_2\zeta_1 + g_6\beta_F g_A h_A]E_A \\
+\ & [g_5\nu_2\zeta_2 - g_4(\zeta_2 + \mu_A) + g_6\beta_F g_A]I_A^A + [g_6\beta_F g_A - g_5(\kappa + \mu_A)I_A^S + [g_7\pi - g_6(\pi + \mu_F)]E_F \\
+\ & [g_1\beta_H g_H + g_3\beta_A g_A - g_7\mu_F]I_F. \qquad (52)
\end{aligned}$$

Substituting $g_1,\ g_2,\ g_3,\ g_4,\ g_5,\ g_6,\ g_7$ in (52), we have

$$\begin{gathered}
[g_4\omega_1\zeta_1 - g_3(\zeta_1 + \mu_A) + g_5\omega_2\zeta_1 + g_6\beta_F g_A h_A]E_A = 0, \\
[g_5\nu_2\zeta_2 - g_4(\zeta_2 + \mu_A) + g_6\beta_F g_A]I_A^A = 0, \\
[g_6\beta_F g_A - g_5(\kappa + \mu_A)I_A^S = 0, \\
[g_7\pi - g_6(\pi + \mu_F)]E_F = 0,
\end{gathered}$$

$[g_1\beta_H g_H + g_3\beta_A g_A - g_7\mu_F]I_F = 0$ and (52) becomes

$$\begin{aligned}
=\ & -\mu_H E_H - \mu_H I_H^E - (a_{27} + a_{32} + \mu_H)I_H^L - \mu_H I_H^S - (\omega_1\psi_2 + a_{23} + \mu_H)I_H^1 \\
-\ & (\psi_3 + \mu_H)I_H^2 + [\pi a_{20}b_3 + \pi a_{24}a_{26}b_4 + \pi a_{20}a_{24}b_2b_4 + a_{25}(\pi a_{24}b_2b_4 \\
-\ & \mu_F a_{18}a_{20}a_{21})]\delta R_H^2, \\
=\ & -\mu_H E_H - \mu_H I_H^E - (a_{27} + a_{32} + \mu_H)I_H^L - \mu_H I_H^S - (\omega_1\psi_2 + a_{23} + \mu_H)I_H^1 \\
-\ & (\psi_3 + \mu_H)I_H^2 + [\pi(a_{20}b_3 + a_{24}a_{25}) + \pi a_{24}b_4(a_{26} + a_{20}b_2) - \mu_F a_{18}a_{20}a_{21}a_{25})]\delta R_H^2, \\
=\ & -\mu_H E_H - \mu_H I_H^E - (a_{27} + a_{32} + \mu_H)I_H^L - \mu_H I_H^S - (\omega_1\psi_2 + a_{23} + \mu_H)I_H^1 \\
-\ & (\psi_3 + \mu_H)I_H^2 + \left(\frac{\pi(a_{20}b_3 + a_{24}a_{25}) + \pi a_{24}b_4(a_{26} + a_{20}b_2)}{\mu_F a_{18}a_{20}a_{21}a_{25}} - 1\right)\delta\mu_F a_{18}a_{20}a_{21}a_{25}R_H^2, \\
=\ & -\mu_H E_H - \mu_H I_H^E - (a_{27} + a_{32} + \mu_H)I_H^L - \mu_H I_H^S - (\omega_1\psi_2 + a_{23} + \mu_H)I_H^1 \\
-\ & (\psi_3 + \mu_H)I_H^2 + (R_0^2 - 1)\delta\mu_F a_{18}a_{20}a_{21}a_{25}R_H^2, \\
<\ & 0.
\end{aligned}$$

Clearly, $\dot{V} \leq 0$ for $R_0 < 1$ with $V = 0$ only at the VL-free fixed point, E^0. Thus, it follows that by LaSalle's invariance principle (as stated in [22]), E^0 is globally asymptotically stable. □

Theorem 2 holds biological significance, as it states that VL can be eliminated from an infected individual whenever $R_0 < 1$. Specifically, for the system (1), $R_0 < 1$ is both necessary and sufficient for VL elimination. Additionally, in a large population, the disease dies out.

4.5. Existence of VL Endemic Fixed Point

In order to determine the presence of a non-zero (endemic) model fixed point, we let

$$\begin{aligned}
E^{**} =\ & (S_H^{**}, E_H^{**}, I_H^{E**}, I_H^{L**}, R_H^{E**}, R_H^{L**}, I_H^{S**}, I_H^{1**}, I_H^{2**}, I_H^{P**}, R_H^{1**},\ R_H^{2**}, S_A^{**}, \\
& E_A^{**}, I_A^{A**}, I_A^{S**}, R_A^{**}, S_F^{**}, E_F^{**}, I_F^{**}). \qquad (53)
\end{aligned}$$

denote any arbitrary fixed point of the model. We then solve the equations of the model in terms of the associated forces of infection $(\lambda_F, \eta_F, \rho_A, \zeta_A, \xi_A)$. It follows from (24)–(43), that the system has a unique endemic fixed point, E^{**}, where

$$
\begin{aligned}
S_H^{**} &= \frac{\Pi_H}{\lambda_F^{**} + \mu_H}, \\
E_H^{**} &= \frac{\Pi_H \lambda_F^{**}}{a_{11}(\lambda_F^{**} + \mu_H)}, \\
I_H^{E**} &= \frac{\theta_1 \Pi_H \lambda_F^{**}}{a_{11} a_{12}(\lambda_F^{**} + \mu_H)}, \\
I_H^{L**} &= \frac{\theta_1 \theta_2 \Pi_H \lambda_F^{**}}{a_{11} a_{12} a_{13}(\lambda_F^{**} + \mu_H)}, \\
R_H^{E**} &= \frac{a_{27} \theta_1 \theta_2 \Pi_H \lambda_F^{**}}{a_{11} a_{12} a_{13} a_{35}(\lambda_F^{**} + \mu_H)}, \\
I_H^{1**} &= \frac{a_{22} \theta_1 \theta_2 \sigma \Pi_H \lambda_F^{**}}{a_{11} a_{12} a_{13} a_{14} a_{15}(\lambda_F^{**} + \mu_H)}, \\
I_H^{2**} &= \frac{a_{22} a_{23} \theta_1 \theta_2 \sigma \Pi_H \lambda_F^{**}}{a_{11} a_{12} a_{13} a_{14} a_{15} a_{16}(\lambda_F^{**} + \mu_H)}, \\
R_H^{2**} &= \frac{c_1 \psi_1 \theta_1 \theta_2 \Pi_H \lambda_F^{**}}{a_{11} a_{12} a_{13} a_{14} a_{15} a_{16} a_{38}(\lambda_F^{**} + \mu_H)}, \\
I_H^{S**} &= \frac{a_{22} \theta_1 \theta_2 \Pi_H \lambda_F^{**}}{a_{11} a_{12} a_{13} a_{14}(\lambda_F^{**} + \mu_H)}, \\
I_H^{P**} &= \frac{c_1 \delta \psi \theta_1 \theta_2 \Pi_H \lambda_F}{a_{11} a_{12} a_{13} a_{14} a_{15} a_{16} a_{17} a_{38}(\lambda_F^{**} + \mu_H)}, \\
R_H^{1**} &= \frac{(c_2 + c_1 \delta \phi + c_3) \psi_1 \theta_1 \theta_2 \Pi_H \lambda_F^{**}}{a_{11} a_{12} a_{13} a_{14} a_{15} a_{16} a_{17} a_{37} a_{38}(\lambda_F^{**} + \mu_H)}, \\
R_H^{L**} &= \frac{c_4 \lambda_F^{**} + c_5 \lambda_F^{**}(c_2 + c_1 \delta \phi + c_3)}{\mu_H a_{11} a_{12} a_{13} a_{14} a_{15} a_{16} a_{17} a_{37} a_{38}(\lambda_F^{**} + \mu_H)}, \\
S_A^{**} &= \frac{\Pi_A}{\eta_F^{**} + \mu_A} \\
E_A^{**} &= \frac{\Pi_A \eta_F^{**}}{a_{18}(\eta_F^{**} + \mu_A)}, \\
I_A^{A**} &= \frac{b_{14} \Pi_A \eta_F^{**}}{a_{18} a_{19}(\eta_F^{**} + \mu_A)}, \\
I_A^{S**} &= \frac{(a_{19} b_{15} + b_{14} b_{16}) \Pi_A \eta_F^{**}}{a_{18} a_{19} a_{20}(\eta_F^{**} + \mu_A)}, \\
R_A^{**} &= \frac{[a_{20} a_{24} b_{19} + \kappa(a_{19} b_{15} + b_{14} b_{16})] \Pi_A \eta_F^{**}}{a_{18} a_{19} a_{20} \mu_A(\eta_F^{**} + \mu_A)}, \\
S_F^{**} &= \frac{\Pi_F}{\rho_A^{**} + \zeta_A^{**} + \xi_A^{**} - \mu_F}, \\
E_F^{**} &= \frac{(\rho_A^{**} + \zeta_A^{**} + \xi_A^{**}) \Pi_F}{a_{21}(\rho_A^{**} + \zeta_A^{**} + \xi_A^{**} - \mu_F)}, \\
I_F^{**} &= \frac{(\rho_A^{**} + \zeta_A^{**} + \xi_A^{**}) \pi \Pi_F}{\mu_F a_{21}(\rho_A^{**} + \zeta_A^{**} + \xi_A^{**} - \mu_F)},
\end{aligned}
\tag{54}
$$

where,

$$
\begin{aligned}
&a_{27} = \tau_1\psi_1, a_{28} = \varrho_1\psi_4, a_{29} = \omega_3\psi_2, a_{30} = \omega_1\psi_1, a_{31} = \gamma_2\psi_3,\\
&a_{32} = \tau_3\psi_1, a_{33} = \gamma_1\psi_3, a_{34} = \nu_1\zeta_1, a_{35} = \epsilon + \mu_H, a_{36} = \phi + \mu_H,\\
&a_{37} = \varrho_1 + \mu_H, a_{38} = \delta + \mu_H\\
&c_1 = \tau_3 a_{14}a_{15}a_{16} + a_{23}\tau_2\sigma + a_{23}a_{33}\tau_2\sigma,\\
&c_2 = \psi_2\sigma a_{16}a_{17}a_{22}a_{38},\\
&c_3 = a_{23}a_{31}\tau_2\sigma.\\
&c_4 = \epsilon\psi_1\theta_1\theta_2 a_{14}a_{15}a_{16}a_{17}a_{37}a_{38}\Pi_H,\\
&c_5 = a_{28}\psi_1\theta_1\theta_2\Pi_H.
\end{aligned}
$$

4.6. Local Stability Analysis of VL Endemic Fixed Point

We rely on the linearization approach to establish the local asymptotic stability of the VL endemic fixed point, that is, finding the eigenvalues of the linearized system around the fixed point.

Theorem 3. *If $R_0 > 1$, then the VL endemic fixed point E^{**} of system (1) is locally asymptotically stable (LAS).*

Proof. Linearize the model system around the VL endemic fixed point. Also, to contain the elements of the matrix of the Jacobian, we redefine the parameter representations. Thus, we have the Jacobian matrix at the endemic fixed point E^{**} denoted by $J_{E^{**}}$ as

$$
J_{E^{**}} = \begin{pmatrix}
e_1 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & e_{11}\\
\lambda_F^{**} & e_2 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & m_4\\
0 & \theta_1 & e_3 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0\\
0 & 0 & \theta_2 & e_4 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0\\
0 & 0 & 0 & a_{27} & e_5 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0\\
0 & 0 & 0 & 0 & \epsilon & e_6 & 0 & 0 & 0 & a_{28} & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0\\
0 & 0 & 0 & a_{22} & 0 & 0 & e_7 & 0 & 0 & 0 & a_{28} & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0\\
0 & 0 & 0 & 0 & 0 & 0 & \sigma & e_8 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0\\
0 & 0 & 0 & 0 & 0 & 0 & 0 & a_{29} & e_9 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0\\
0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & e_{10} & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0\\
0 & 0 & 0 & 0 & 0 & 0 & a_{30} & a_{31} & \phi & e_{12} & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0\\
0 & 0 & 0 & a_{32} & 0 & 0 & 0 & a_{23} & a_{32} & 0 & 0 & e_{13} & 0 & 0 & 0 & 0 & 0 & 0 & 0\\
0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & e_{14} & 0 & 0 & 0 & 0 & 0 & 0\\
0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & \eta_F^{**} & e_{15} & 0 & 0 & 0 & 0 & 0\\
0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & a_{24} & e_{16} & 0 & 0 & 0 & 0\\
0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & a_{33} & a_{26} & e_{17} & 0 & 0 & 0\\
0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & a_{34} & \kappa & e_{18} & 0 & 0\\
0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & e_{22} & e_{23} & e_{23} & 0 & e_{19} & 0 & 0\\
0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & b_3 & b_2 & b_2 & 0 & 0 & e_{20} & 0\\
0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & \pi & e_{21}
\end{pmatrix}, \tag{55}
$$

where

$$
\begin{aligned}
&e_1 = -m_1, e_2 = -a_{11}, e_3 = -a_{12}, e_4 = -a_{13}, e_5 = -a_{35}, e_6 = -\mu_H, e_7 = -a_{14},\\
&e_8 = -a_{15}, e_9 = -a_{16}, e_{10} = -a_{17}, e_{11} = -m_4, e_{12} = -a_{37}, e_{13} = -a_{38},\\
&e_{14} = -m_2, e_{15} = -a_{18}, e_{16} = -a_{19}, e_{17} = -a_{20}, e_{18} = -\mu_A, e_{19} = -m_3,\\
&e_{20} = -a_{21}, e_{21} = -\mu_F, e_{22} = -b_3, e_{23} = -b_2.
\end{aligned}
$$

The characteristic equation associated with the model system (1) is given by $|J_{E^{**}} - \lambda I| = 0$, where $\lambda (= \lambda_i,\ i = 1, 2, 3, \ldots, 20)$ denotes the eigenvalues of the $J_{E^{**}}$, $I = I_{20\times 20}$ identity matrix.

This implies

$$|J_{E^*} - \lambda I| = 0. \tag{56}$$

Thus,

$$(e_1-\lambda_1)(e_2-\lambda_2)(e_3-\lambda_3)(e_4-\lambda_4)(e_5-\lambda_5)(e_6-\lambda_6)(e_7-\lambda_7)(e_8-\lambda_8)(e_9-\lambda_9)(e_{10}-\lambda_{10})$$
$$(e_{12}-\lambda_{11})(e_{13}-\lambda_{12})(e_{14}-\lambda_{13})(e_{15}-\lambda_{14})(e_{16}-\lambda_{15})(e_{17}-\lambda_{16})(e_{18}-\lambda_{17})(e_{19}-\lambda_{18})$$
$$(e_{20}-\lambda_{19})(e_{21}-\lambda_{20}) = 0.$$

This implies

$$\begin{aligned}
\lambda_1 = e_1 = -m_1 = -(\lambda_F^{**} + \mu_H) < 0, \quad & \lambda_{11} = e_{12} = -a_{37} = -(\varrho_1\psi_4 + \mu_H) < 0,\\
\lambda_2 = e_2 = -a_{11} = -(\theta_1 + \mu_H) < 0, \quad & \lambda_{12} = e_{13} = -a_{38} = -(\delta + \mu_H) < 0,\\
\lambda_3 = e_3 = -a_{12} = -(\theta_2 + \mu_H) < 0, \quad & \lambda_{13} = e_{14} = -m_2 = -(\eta_F^{**} + \mu_H) < 0,\\
\lambda_4 = e_4 = -a_{13} = -(\psi_1 + \mu_H) < 0, \quad & \lambda_{14} = e_{15} = -a_{18} = -(\zeta_1 + \mu_A) < 0,\\
\lambda_5 = e_5 = -a_{35} = -(\epsilon + \mu_H) < 0, \quad & \lambda_{15} = e_{16} = -a_{19} = -(\zeta_2 + \mu_A) < 0,\\
\lambda_6 = e_6 = -\mu_H < 0, \quad & \lambda_{16} = e_{17} = -a_{20} = -(\kappa + \mu_A) < 0,\\
\lambda_7 = e_7 = -a_{14} = -(\sigma + \mu_H) < 0, \quad & \lambda_{17} = e_{18} = -\mu_A < 0,\\
\lambda_8 = e_8 = -a_{15} = -(\psi_2 + \mu_H) < 0, \quad & \lambda_{18} = e_{19} = -m_3 < 0,\\
\lambda_9 = e_9 = -a_{16} = -(\psi_3 + \mu_H) < 0, \quad & \lambda_{19} = e_{20} = -a_{21} = -(\pi + \mu_A) < 0,\\
\lambda_{10} = e_{10} = -a_{17} = -(\phi + \mu_H) < 0, \quad & \lambda_{20} = e_{21} = -\mu_F < 0,
\end{aligned}$$

$\lambda_i, i = 1, 2, 3, \ldots, 20$ are all negative.

We therefore conclude that the VL endemic fixed point E^{**} is locally asymptotically stable. Hence, the proof is complete. □

This result indicates that the VL disease is likely to spread quickly and the number of infected individuals may rise exponentially if R_0 remains greater than unity for some time $t > 0$.

4.7. Global Stability Analysis of VL Endemic Fixed Point

Here, we explore the global stability of the VL endemic fixed point E^{**} to help understand the spread of the VL disease in a population.

Theorem 4. *There is no periodic orbit for the system* (1).

Proof. Applying the Dulac's criterion as used in [24], let

$$X = (S_H, E_H, I_H^E, I_H^L, R_H^E, R_H^L, I_H^S, I_H^1, I_H^2, I_H^P, R_H^1, R_H^2, S_A, E_A, I_A^A, I_A^S, R_A, S_F, E_F, I_F).$$

Taking the Dulac's function as

$$G = \frac{1}{I_F},$$

from the system (1) equations, we have

$$\begin{aligned}
G\frac{dS_H}{dt} &= \frac{\Pi_H}{I_F} - \lambda_F\frac{S_H}{I_F} - \mu_H\frac{S_H}{I_F},\\
G\frac{dE_H}{dt} &= \lambda_F\frac{S_H}{I_F} - (\theta_1 + \mu_H)\frac{E_H}{I_F},\\
G\frac{dI_H^E}{dt} &= \theta_1\frac{E_H}{I_F} - (\theta_2 + \mu_H)\frac{I_H^E}{I_F},\\
G\frac{dI_H^L}{dt} &= \theta_2\frac{I_H^E}{I_F} - (\psi_1 + \mu_H)\frac{I_H^L}{I_F},\\
G\frac{dR_H^E}{dt} &= \tau_1\psi_1\frac{I_H^L}{I_F} - (\varepsilon + \mu_H)\frac{R_H^E}{I_F},
\end{aligned}$$

$$
\begin{aligned}
G\frac{dR_H^L}{dt} &= \varepsilon\frac{R_H^E}{I_F} + \varrho_1\psi_4\frac{R_H^1}{I_F} - \mu_H\frac{R_H^L}{I_F},\\
G\frac{dI_H^S}{dt} &= \tau_2\psi_1\frac{I_H^L}{I_F} - (\sigma+\mu_H)\frac{I_H^S}{I_F},\\
G\frac{dI_H^1}{dt} &= \sigma\frac{I_H^S}{I_F} - (\psi_2+\mu_H)\frac{I_H^1}{I_F},\\
G\frac{dI_H^2}{dt} &= \omega_3\psi_2\frac{I_H^1}{I_F} - (\psi_3+\mu_H)\frac{I_H^2}{I_F},\\
G\frac{dI_H^P}{dt} &= \delta\frac{R_H^2}{I_F} - (\phi+\mu_H)\frac{I_H^P}{I_F},\\
G\frac{dR_H^1}{dt} &= \omega_1\psi_2\frac{I_H^1}{I_F} + \phi\frac{I_H^P}{I_F} + \gamma_2\psi_3\frac{I_H^2}{I_F} - (\varrho_1\psi_4+\mu_H)\frac{R_H^1}{I_F},\\
G\frac{dR_H^2}{dt} &= \tau_3\psi_1\frac{I_H^L}{I_F} + \omega_2\psi_2\frac{I_H^1}{I_F} + \gamma_1\psi_3\frac{I_H^2}{I_F} - (\delta+\mu_H)\frac{R_H^2}{I_F},\\
G\frac{dS_A}{dt} &= \frac{\Pi_A}{I_F} - \eta_F\frac{S_A}{I_F} - \mu_A\frac{S_A}{I_F},\\
G\frac{dE_A}{dt} &= \eta_F\frac{S_A}{I_F} - (\zeta_1+\mu_A)\frac{E_A}{I_F},\\
G\frac{dI_A^A}{dt} &= \varpi_1\zeta_1\frac{E_A}{I_F} - (\zeta_2+\mu_A)\frac{I_A^A}{I_F},\\
G\frac{dI_A^S}{dt} &= \varpi_2\zeta_1\frac{E_A}{I_F} + \nu_2\zeta_2\frac{I_A^A}{I_F} - (\kappa+\mu_A)\frac{I_A^S}{I_F},\\
G\frac{dR_A}{dt} &= \nu_1\zeta_2\frac{I_A^A}{I_F} + \kappa\frac{I_A^S}{I_F} - \mu_A\frac{R_A}{I_F},\\
G\frac{dS_F}{dt} &= \frac{\Pi_F}{I_F} - (\rho_A+\xi_A+\chi_A)\frac{S_F}{I_F} - \mu_F\frac{S_F}{I_F},\\
G\frac{dE_F}{dt} &= (\rho_A+\xi_A+\chi_A)\frac{S_F}{I_F} - (\pi+\mu_F)\frac{E_F}{I_F},\\
G\frac{dI_F}{dt} &= \pi\frac{E_F}{I_F} - \mu_F.
\end{aligned}
$$

Thus,

$$
\begin{aligned}
\frac{dGX}{dt} &= \frac{\partial}{dS_H}\left(G\frac{dS_H}{dt}\right) + \frac{\partial}{dE_H}\left(G\frac{dE_H}{dt}\right) + \frac{\partial}{dI_H^E}\left(G\frac{dI_H^E}{dt}\right) + \frac{\partial}{ddI_H^L}\left(G\frac{dI_H^L}{dt}\right)\\
&\quad + \frac{\partial}{dR_H^E}\left(G\frac{dR_H^E}{dt}\right) + \frac{\partial}{dR_H^L}\left(G\frac{dR_H^L}{dt}\right) + \frac{\partial}{dI_H^S}\left(G\frac{dI_H^S}{dt}\right) + \frac{\partial}{dI_H^1}\left(G\frac{dI_H^1}{dt}\right)\\
&\quad + \frac{\partial}{dI_H^2}\left(G\frac{dI_H^2}{dt}\right) + \frac{\partial}{dI_H^P}\left(G\frac{dI_H^P}{dt}\right) + \frac{\partial}{dR_H^1}\left(G\frac{dR_H^1}{dt}\right) + \frac{\partial}{dR_H^2}\left(G\frac{dR_H^2}{dt}\right)\\
&\quad + \frac{\partial}{dS_A}\left(G\frac{dS_A}{dt}\right) + \frac{\partial}{dE_A}\left(G\frac{dE_A}{dt}\right) + \frac{\partial}{dI_A^A}\left(G\frac{dI_A^A}{dt}\right) + \frac{\partial}{dI_A^S}\left(G\frac{dI_A^S}{dt}\right)\\
&\quad + \frac{\partial}{dR_A}\left(G\frac{dR_A}{dt}\right) + \frac{\partial}{dS_F}\left(G\frac{dS_F}{dt}\right) + \frac{\partial}{dE_F}\left(G\frac{dE_F}{dt}\right) + \frac{\partial}{dI_F}\left(G\frac{dI_F}{dt}\right),\\
&= \frac{\partial}{dS_H}\left(\frac{\Pi_H}{I_F} - \lambda_F\frac{S_H}{I_F} - \mu_H\frac{S_H}{I_F}\right) + \frac{\partial}{dE_H}\left(\lambda_F\frac{S_H}{I_F} - (\theta_1+\mu_H)\frac{E_H}{I_F}\right)\\
&\quad + \frac{\partial}{dI_H^E}\left(\theta_1\frac{E_H}{I_F} - (\theta_2+\mu_H)\frac{I_H^E}{I_F}\right) + \frac{\partial}{dI_H^L}\left(\theta_2\frac{I_H^E}{I_F} - (\psi_1+\mu_H)\frac{I_H^L}{I_F}\right)
\end{aligned}
$$

$$
\begin{aligned}
&+\frac{\partial}{dR_H^E}\left(\tau_1\psi_1\frac{I_H^L}{I_F}-(\varepsilon+\mu_H)\frac{R_H^E}{I_F}\right)+\frac{\partial}{dR_H^L}\left(\varepsilon\frac{R_H^E}{I_F}+\varrho_1\psi_4\frac{R_H^1}{I_F}-\mu_H\frac{R_H^L}{I_F}\right)\\
&+\frac{\partial}{dI_H^S}\left(\tau_2\psi_1\frac{I_H^L}{I_F}-(\sigma+\mu_H)\frac{I_H^S}{I_F}\right)+\frac{\partial}{dI_H^1}\left(\sigma\frac{I_H^S}{I_F}-(\psi_2+\mu_H)\frac{I_H^1}{I_F}\right)\\
&+\frac{\partial}{dI_H^2}\left(\omega_3\psi_2\frac{I_H^1}{I_F}-(\psi_3+\mu_H)\frac{I_H^2}{I_F}\right)+\frac{\partial}{dI_H^P}\left(\delta\frac{R_H^2}{I_F}-(\phi+\mu_H)\frac{I_H^P}{I_F}\right)\\
&+\frac{\partial}{dR_H^1}\left(\omega_1\psi_2\frac{I_H^1}{I_F}+\phi\frac{I_H^P}{I_F}+\gamma_2\psi_3\frac{I_H^2}{I_F}-(\varrho_1\psi_4+\mu_H)\frac{R_H^1}{I_F}\right)\\
&+\frac{\partial}{dR_H^2}\left(\tau_3\psi_1\frac{I_H^L}{I_F}+\omega_2\psi_2\frac{I_H^1}{I_F}+\gamma_1\psi_3\frac{I_H^2}{I_F}-(\delta+\mu_H)\frac{R_H^2}{I_F}\right)\\
&+\frac{\partial}{dS_A}\left(\frac{\Pi_A}{I_F}-\eta_F\frac{S_A}{I_F}-\mu_A\frac{S_A}{I_F}\right)+\frac{\partial}{dE_A}\left(\eta_F\frac{S_A}{I_F}-(\zeta_1+\mu_A)\frac{E_A}{I_F}\right)\\
&+\frac{\partial}{dI_A^A}\left(\varpi_1\zeta_1\frac{E_A}{I_F}-(\zeta_2+\mu_A)\frac{I_A^A}{I_F}\right)+\frac{\partial}{dI_A^S}\left(\varpi_2\zeta_1\frac{E_A}{I_F}+\nu_2\zeta_2\frac{I_A^A}{I_F}-(\kappa+\mu_A)\frac{I_A^S}{I_F}\right)\\
&+\frac{\partial}{dR_A}\left(\nu_1\zeta_2\frac{I_A^A}{I_F}+\kappa\frac{I_A^S}{I_F}-\mu_A\frac{R_A}{I_F}\right)+\frac{\partial}{dS_F}\left(\frac{\Pi_F}{I_F}-(\rho_A+\xi_A+\chi_A)\frac{S_F}{I_F}-\mu_F\frac{S_F}{I_F}\right)\\
&+\frac{\partial}{dE_F}\left((\rho_A+\xi_A+\chi_A)\frac{S_F}{I_F}-(\pi+\mu_F)\frac{E_F}{I_F}\right)+\frac{\partial}{dI_F}\left(\pi\frac{E_F}{I_F}-\mu_F\right),\\
&=\left(-\lambda_F\frac{1}{I_F}-\mu_H\frac{1}{I_F}\right)+\left(-(\theta_1+\mu_H)\frac{1}{I_F}\right)+\left(-(\theta_2+\mu_H)\frac{1}{I_F}\right)\\
&+\left(-(\psi_1+\mu_H)\frac{1}{I_F}\right)+\left(-(\varepsilon+\mu_H)\frac{1}{I_F}\right)+\left(-\mu_H\frac{1}{I_F}\right)+\left(-(\sigma+\mu_H)\frac{1}{I_F}\right)\\
&+\left(-(\psi_2+\mu_H)\frac{1}{I_F}\right)+\left(-(\psi_3+\mu_H)\frac{1}{I_F}\right)+\left(-(\phi+\mu_H)\frac{1}{I_F}\right)\\
&+\left(-(\varrho_1\psi_4+\mu_H)\frac{1}{I_F}\right)+\left(-(\delta+\mu_H)\frac{1}{I_F}\right)+\left(-\eta_F\frac{1}{I_F}-\mu_A\frac{1}{I_F}\right)\\
&+\left(-(\zeta_1+\mu_A)\frac{1}{I_F}\right)+\left(-(\zeta_2+\mu_A)\frac{1}{I_F}\right)+\left(-(\kappa+\mu_A)\frac{1}{I_F}\right)\\
&+\left(-\mu_A\frac{1}{I_F}\right)+\left(-(\rho_A+\xi_A+\chi_A)\frac{1}{I_F}-\mu_F\frac{1}{I_F}\right)+\left(-(\pi+\mu_F)\frac{1}{I_F}\right)\\
&+\left(-\pi E_F I_F^{-2}\right),\\
&=-(\lambda_F+\eta_F+\rho_A+\xi_A+\chi_A+\delta+\kappa+12\mu_H+5\mu_A+2\mu_F+\theta_1+\theta_2\\
&+\pi+\psi_1+\psi_2+\psi_3+\rho_1\psi_4+\varepsilon+\sigma+\phi+\zeta_1+\zeta_2)\frac{1}{I_F}-\pi\frac{E_F}{I_F^2},\\
&<0.
\end{aligned}
$$

Hence, there exists no periodic solution for the system (1). □

With Ω being positively invariant, all solutions to the system (1) originate and remain in Ω for all t. This is supported by the Poincaré–Bendixson theorem. Consequently, the theorem follows.

Theorem 5. *The VL endemic fixed point E^{**} for the system (1) is globally asymptotically stable whenever $R_0 > 1$.*

This result indicates that the disease may not die out whenever the threshold $R_0 > 1$.

4.8. Sensitivity Analysis

This subsection presents the analytical sensitivity analysis of some of the important parameters in R_0 (i.e., β_A, β_F, g_A, and κ) using the differential approach as applied by [25] to quantify their impact on the model dynamic. Moreover, the R_0 important parameters' numerical and analytical values are produced using precise assumptions based on parameter values. Some insight on tracking the model's onset at variant places is given by the analytical expressions produced. By reducing the value to less than unity, the threshold value R_0 is acknowledged as the primary method of stopping the disease's progress. Positively signed parameters are considered highly and proportionally sensitive to R_0, whereas negatively signed parameters are not declining R_0. This is obtained using partial derivatives of R_0 with respect to the key parameter. The following are valid based on the expression for R_0 provided $D_8D_{10} < D_9D_{11}$:

$$\begin{aligned}
\frac{\partial R_0}{\partial \beta_A} &= \frac{D_1\sqrt{(\beta_A D_1 + D_2)D_3D_4}}{2(\beta_A D_1 + D_2)D_3D_4} > 0,\\
\frac{\partial R_0}{\partial \beta_F} &= \frac{D_5\sqrt{\beta_F D_3D_4D_5}}{2\beta_F D_3D_4} > 0,\\
\frac{\partial R_0}{\partial g_A} &= \frac{(D_6 + 2g_A D_7)\sqrt{(g_A D_6 + g_A^2 D_7)D_3D_4}}{2D_3D_4(g_A D_6 + g_A^2 D_7)} > 0,\\
\frac{\partial R_0}{\partial \kappa} &= \frac{k(D_8D_{10} - D_9D_{11})\sqrt{D_3(\kappa D_9 + D_{10})(\kappa D_8 + D_{11})}}{(\kappa D_8 + D_{11})(\kappa D_9 + D_{10})} < 0.
\end{aligned} \tag{57}$$

$$\begin{aligned}
D_1 &= \beta_F g_A^2 \mu_A \mu_H^2 \Pi_A Pi_F,\\
D_2 &= \beta_F g_A h_A \kappa \mu_A \Pi_F(\zeta_1 \mu_F \Pi_H + \zeta_1 \mu_H \Pi_A + mu_A^2 \Pi_H + \mu_A \mu_H \Pi_A)\\
&\quad + \beta_F g_A h_A \mu_A^3 \mu_F \Pi_F \Pi_H \zeta_1 + \beta_F g_A h_A \mu_A^3 \mu_H \Pi_A \Pi_F \zeta_1 + \beta_F g_A h_A \mu_A^4 \mu_F \Pi_F \Pi_H\\
&\quad + \beta_F g_A h_A \mu_A^3 \mu_F \mu_H \Pi_A \Pi_F \zeta_1 + \beta_F g_A \mu_A^2 \mu_H^2 \nu_2 \Pi_F \varrho_1 \zeta_1 \zeta_2,\\
D_3 &= \mu_A^2 \mu_F \Pi_H^2 + 2\mu_A \mu_F \mu_H \Pi_A \Pi_H + \mu_F \mu_H,\\
D_4 &= \kappa \mu_F \pi \zeta_1 \zeta_2 + \kappa \mu_A \mu_F \zeta_1 \zeta_2 + \kappa \mu_A \mu_F \pi \zeta_1 + \kappa \mu_A^2 \mu_F \zeta_1 + \kappa \mu_A \pi \zeta_2 + \kappa \mu_A^2 \mu_F \zeta_2\\
&\quad + \kappa \mu_A^2 \mu_F \pi + \kappa \mu_A^3 \mu_F + \mu_A \mu_F \pi \zeta_1 \zeta_2 + \mu_A^2 \mu_F \zeta_1 \zeta_2 + \mu_A^2 \mu_F \pi \zeta_1 + \mu_A^3 \mu_F \zeta_1 \zeta_2\\
&\quad + \mu_A^2 \mu_F \pi \zeta_2 + \mu_A^3 \mu_F \zeta_2 + \mu_A^3 \mu_F \pi + \mu_A^4 \mu_F,\\
D_5 &= \kappa g_A h_A \mu_A \mu_F \Pi_F \Pi_H \zeta_1 + \kappa g_A h_A \mu_A \mu_F \Pi_A \Pi_F \zeta_1 + \beta_F \kappa g_H h_A \mu_A^3 \Pi_F \Pi_H\\
&\quad + \kappa g_A h_A \mu_A^2 \mu_H \Pi_A \Pi_F + g_A h_A \mu_A^3 \mu_F \Pi_F \Pi_H \zeta_1 + \beta_F g_A h_A \mu_A^3 \mu_H \Pi_A \Pi_F \zeta_1\\
&\quad + g_A h_A \mu_A^4 \mu_F \Pi_F \Pi_H + g_A h_A \mu_A^3 \mu_F \mu_H \Pi_A \Pi_F + \beta_A g_A^2 \mu_A \mu_H^2 \Pi_A \Pi_F\\
&\quad + g_A \mu_A^2 \mu_H^2 \nu_2 \Pi_F \varrho_1 \zeta_1 \zeta_2 + g_A \mu_A \mu_H^2 \nu_2 \Pi_F \varrho_1 \zeta_1 \zeta_2,\\
D_6 &= \beta_F \kappa h_A \mu_A \mu_F \Pi_F \Pi_H \zeta_1 + \beta_F \kappa h_A \mu_A \mu_F \Pi_A \Pi_F \zeta_1 + \beta_F \kappa h_A \mu_A^2 \Pi_A \Pi_F\\
&\quad + \beta_F \kappa h_A \mu_A^3 \Pi_F \Pi_H + \beta_F h_A \mu_A^4 \mu_F \Pi_F \Pi_H + \beta_F h_A \mu_A^3 \mu_F \mu_H \Pi_A \Pi_F\\
&\quad + \beta_F h_A \mu_A^3 \mu_F \mu_H \Pi_A \Pi_F \zeta_1 + \beta_F h_A \mu_A^2 \mu_F \mu_H \Pi_A \Pi_F \zeta_1\\
&\quad + \beta_F \mu_A^2 \mu_H^2 \nu_2 \Pi_F \varrho_1 \zeta_1 \zeta_2 + \beta_F \mu_A \mu_H^2 \nu_2 \Pi_F \varrho_1 \zeta_1 \zeta_2,\\
D_7 &= \beta_A \beta_F \mu_A \mu_H^2 \Pi_A \Pi_F,\\
D_8 &= \beta_F g_A h_A \mu_A \mu_F \Pi_F \Pi_H \zeta_1 + \beta_F g_A h_A \mu_A \mu_H \Pi_A \Pi_F \zeta_1 + \beta_F g_A h_A \mu_A^3 \Pi_F \Pi_H\\
&\quad + \beta_F g_A h_A \mu_A^2 \mu_H \Pi_A \Pi_H,\\
D_9 &= \mu_F \pi \zeta_1 \zeta_2 + \mu_A \mu_F \zeta_1 \zeta_2 + \mu_A \mu_F \pi \zeta_1 + \mu_A^2 \mu_F \zeta_1 + \mu_A \pi \zeta_2 + \mu_A^2 \mu_F \zeta_2\\
&\quad + \mu_A^2 \mu_F \pi + \mu_A^3 \mu_F,\\
D_{10} &= \mu_A \mu_F \pi \zeta_1 \zeta_2 + \mu_A^2 \mu_F \zeta_1 \zeta_2 + \mu_A^2 \mu_F \zeta_1 + \mu_A^3 \mu_F \zeta_1 \zeta_2 + \mu_A^2 \mu_F \pi \zeta_2 + \mu_A^3 \mu_F \zeta_2
\end{aligned}$$

$$
\begin{aligned}
&+\mu_A^3\mu_F\pi+\mu_A^4\mu_F,\\
D_{11} &= \beta_F g_A h_A \mu_A^3 \mu_F \Pi_F \Pi_H \zeta_1 + \beta_F g_A h_A \mu_A^3 \mu_H \Pi_A \Pi_F \zeta_1 + \beta_F g_A h_A \mu_A^4 \mu_F \Pi_F \Pi_H\\
&+\beta_F g_A h_A \mu_A^4 \mu_H \Pi_A \Pi_F + \beta_A \beta_F g_A^2 \mu_A \mu_H^2 \Pi_A \Pi_F + \beta_F g_A \mu_A^2 \mu_H^2 \nu_2 \Pi_F \varrho_1 \zeta_1 \zeta_2\\
&+\beta_F g_A \mu_A \mu_H^2 \nu_2 \Pi_F \varrho_1 \zeta_1 \zeta_2.
\end{aligned}
$$

The equations in (57) show that decreasing the transmission probability from infected sandflies to susceptible animals (β_A), the transmission probability from infected animals to susceptible sandflies (β_F), and the per capita biting rate of sandflies of animals (g_A) will help reduce the transmission of VL infection. Again, from (57), increasing the fraction of rate of transfer of symptomatic infected animals to the recovered class (κ) will also reduce the transmission of VL disease in a population.

The elasticity indices for all the R_0 parameters which is given by

$$\gamma_q^{R_0} = \frac{\partial R_0}{\partial q}.\frac{q}{R_0}. \tag{58}$$

where R_0 denotes the basic reproduction ratio and q is a parameter are analyzed numerically using Variable Precision Arithmetic (VPA) in MATLAB in the section for numerical simulation.

5. Numerical Simulation

In this section, the numerical outcomes of the various compartments of the model are observed using parameter values from the literature and some assumptions. All values of the parameters in Table 1 are given in Table 2. This is to gain insight into the behavior of the numerical solutions of the model.

Table 1. The description of parameters in the ZVL model.

Parameter	Description
Π_H	Recruitment rate of humans
Π_A	Recruitment rate of animals
Π_F	Recruitment rate of sandflies
θ_1	Transfer rate of exposed humans to early VL infection stage
θ_2	Transfer rate of early asymptomatic VL-infected humans to late VL infection stage
τ_1	Proportion of late asymptomatic VL-infected humans moving to R_H^E
τ_2	Proportion of late asymptomatic VL-infected humans moving to I_H^S
τ_3	Proportion of late asymptomatic VL-infected humans moving to R_H^2classes
ψ_1	Rate of transfer of late asymptomatic infected humans to symptomatic infected class
ψ_2	Rate of transfer of infected humans receiving first-line treatment to second-line treatment, to recovered humans who have cleared the parasite and to putative recovered human classes
ψ_3	Rate of transfer of infected humans receiving second-line treatment to recovered humans who have cleared the parasite and to putative recovered human classes
ω_1	Proportions of infected humans receiving first-line treatment moving to recovered class who have cleared the parasite
ω_2	Proportions of infected humans receiving first-line treatment moving to putative recovered class
ω_3	Proportions of infected humans receiving first-line treatment moving to infected class receiving second-line treatment after first-line treatment failure
γ_1	Proportion of infected humans receiving second-line treatment moving to putative recovered class
γ_2	Proportion of infected humans receiving second-line treatment moving to recovered class who have cleared the parasite
ϱ_1	Proportion of recovered humans who have cleared the parasite moving to recovered class who are DAT-positive and LST-positive

Table 1. *Cont.*

Parameter	Description
ϱ_2	Proportion of recovered humans who have cleared the parasite moving to putative recovered class
μ_H	Natural death rates of humans
μ_A	Natural death rates of animals
μ_F	Natural death rates of sandflies
δ	Rate of transfer from putative recovered humans to PKDL-infected class
ε	Rate of transfer from recovered humans who are DAT-positive and not yet LST-positive to recovered humans who are DAT-positive and still LST-positive
ϕ	Rate of transfer of PKDL-infected humans to putative recovered class
φ	Rate of transfer from recovered humans who are DAT-positive and but still LST-positive susceptible humans
σ	Rate of transfer of humans from symptomatic infected class to infected humans receiving first-line treatment class
ϖ_1	Proportion of exposed animals moving to asymptomatic infected class
ϖ_2	Proportion of exposed animals moving to symptomatic infected class
ζ_1	Rate of transfer of exposed animals to asymptomatic infected class and symptomatic infected class
ζ_2	Rate of transfer of asymptomatic infected animals to symptomatic infected class and recovered class
ν_1	Proportion of asymptomatic infected animals moving to symptomatic infected class
ν_2	Proportion of asymptomatic infected animals moving to recovered class
κ	Rate of transfer from symptomatic infected animals to recovered class
π	Rate of transfer of exposed sandflies to infected sandflies
β_H	Transmission probability from infected sandflies to susceptible humans
β_A	Transmission probability from infected sandflies to susceptible animals
β_F	Transmission probability from infected animal to susceptible sandflies (kept constant for both asymptomatic and symptomatic infected class)
g_H	Per capita biting rate of sandflies of humans
g_A	Per capita biting rate of sandflies of animals (kept constant for both asymptomatic and symptomatic infected classes)
h_A	Modification parameter for the relative infectiousness of an animal

Table 2. Parameter values used for the simulation result.

Parameter	Value	Unit	Source
Π_H	19.5	day^{-1}	[17]
Π_A	8.33	day^{-1}	[26]
Π_F	210.62	day^{-1}	Assumed
θ_1	0.0111	day^{-1}	[27]
θ_2	0.01667	day^{-1}	[19]
τ_1	0.001	day^{-1}	Assumed
τ_2	0.699	day^{-1}	$1-(\tau_1+\tau_3)$
τ_3	0.3	day^{-1}	[28,29]
ψ_1	0.083	day^{-1}	[19]
ψ_2	0.033	day^{-1}	[19]
ψ_3	0.333	day^{-1}	[19]
ω_1	0.92	day^{-1}	$1-(\omega_2+\omega_3)$
ω_2	0.03	day^{-1}	[19]
ω_3	0.05	day^{-1}	[19]
γ_1	0.03	day^{-1}	[19]
γ_2	0.97	day^{-1}	$1-\gamma_2$
ϱ_1	0.0135	day^{-1}	[19]

Table 2. *Cont.*

Parameter	Value	Unit	Source
μ_H	0.00795	day^{-1}	Assumed
μ_A	0.0169	day^{-1}	[17]
μ_F	0.14	day^{-1}	Assumed
δ	0.00397	day^{-1}	[19]
ε	0.0135	day^{-1}	[19]
ϕ	0.00556	day^{-1}	[19]
σ	1	day^{-1}	[19]
ω_1	0.79	day^{-1}	Assumed
ω_2	0.21	day^{-1}	Assumed
ζ_1	0.69	day^{-1}	Assumed
ζ_2	0.211	day^{-1}	Assumed
ν_1	0.97	day^{-1}	Assumed
ν_2	0.03	day^{-1}	$1-\nu_1$
κ	0.115	day^{-1}	[17]
π	0.2	day^{-1}	[17]
β_H	1	day^{-1}	[19]
β_A	1	day^{-1}	[19]
β_F	1	day^{-1}	[19]
g_H	0.02856	day^{-1}	[30,31]
g_A	0.2856	day^{-1}	[17]
h_A	1.39	day^{-1}	[18]

The initial states considered for the model variables given in Table 3 are $S_H = 250,000$, $E_H = 3000$ $I_H^E = 2000$ $I_H^L = 1000 I_H^S = 900$, $R_H^E = 200$, $R_H^L = 150$, $I_H^1 = 150 I_H^2 = 200$, $I_H^p = 120$, $R_H^1 = 100$, $R_H^2 = 110$, $S_A = 2500$, $E_A = 700$, $I_A^A = 200$, $I_A^S = 190$, $R_A = 180$, $S_F = 10,000$ $E_F = 3500$, $I_F = 2000$. The elasticity indices of the parameters of R_0 are obtained numerically in MATLAB2014a using Variable Precision Arithmetic (VPA) for precision. By employing high-precision arithmetic to reduce errors in the crucial computations, this will ensure precise assessment of the basic reproduction number's sensitivity to changes in a particular parameter. It is adjudged that VPA makes the computations more precise, which is important when little adjustments to the parameters have a big impact on R_0. The elasticity index is computed more precisely using the VPA than using the standard floating-point arithmetic [32–34]. This is required because normal floating-point arithmetic may not be accurate enough to handle the model's numerous parameters and intricate relationships, which could result in errors that may happen during computation. Additionally, the Variable Precision Arithmetic (VPA) used for the sensitivity analysis is a global sensitivity analysis method, as it considers the entire input space, making it more robust for complex, multi-dimensional models like in this study. Table 4 provides the numerical values indicating R_0's relative significance. It is discovered that some parameters have a negative elasticity index, while others are positive, and a few zero. When the parameters show a positive relationship, it means that raising the values of that parameter will significantly impact how frequently the disease spreads. Conversely, if there is a negative relationship, raising these parameters would aid in slowing the disease's spread.

The numerical values from Table 4, provides a comprehensive overview of which parameters are critical for controlling the disease; that is the impact of parameter changes on R_0. Understanding the elasticity indices helps in strategic planning for disease control, allowing public health officials to focus on the most influential parameters. This targeted approach can optimize resource allocation and improve the effectiveness of intervention strategies, ultimately contributing to better disease management and control efforts.

To gain more insight, the contribution of some of the parameters in R_0, some experiments were conducted by varying the values of the key parameters (using the basic reproduction number R_0 as response function). The transmission probability from in-

fected sandflies to animals (β_A), transmission probability from infected animals to sandflies (β_F), per capita biting rate of sandflies of animals (g_A), and rate of transfer from symptomatic infected animals to the recovered class (κ) are among the parameters that have greatest influence on R_0. In particular, the range of $R_0 \in (0.98951, 1.1138)$ as the values of the critical parameters (β_A, β_F, g_A and κ) are varied from $(0.8, 0.8, 0.01856, 0.115)$ to $(1.3, 1.3, 0.02856, 0.415)$, respectively. These numerical results corroborate the analytical results obtained for the critical parameters. It is worth mentioning that attention should be given to the most sensitive parameters identified so that the value of R_0 remains less than one, to avoid disease outbreak.

Table 3. The description of variables in the VL model.

Variable	Description
S_H	Population of susceptible humans
E_H	Population of exposed humans
I_H^E	Population of infected humans at early asymptomatic stage
I_H^L	Population of infected humans at late asymptomatic stage
I_H^S	Population of symptomatic infected humans
R_H^E	Population of recovered humans who are DAT-positive and not yet LST-positive
R_H^L	Population of recovered humans who are DAT-positive and but still LST-positive
I_H^P	Population of infected humans at the PKDL stage
I_H^1	Population of infected humans who are receiving first-line treatment
I_H^2	Population of infected humans who are receiving second-line treatment
R_H^1	Population of recovered humans who have cleared the parasite
R_H^2	Population of putative recovered humans
S_A	Population of susceptible animals
E_A	Population of exposed animals
I_A^A	Population of asymptomatic infected animals
I_A^S	Population of symptomatic infected animals
R_A	Population of recovered animals
S_F	Population of susceptible sandflies (vector)
E_F	Population of exposed sandflies
I_F	Population of infected sandflies

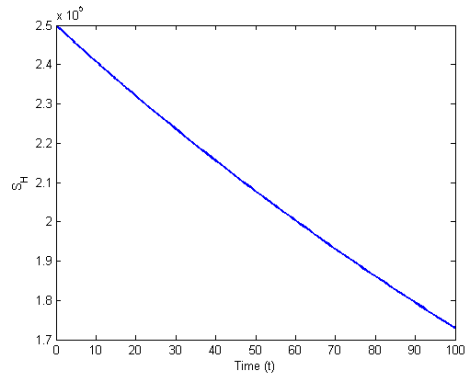
Table 4. Elasticity indices of $R_0 = 0.98951$ to the parameters of the model.

Parameter	Value	Elasticity Index
Π_H	19.5	-0.58423
Π_A	8.33	-0.11367
Π_F	210.62	0.6979
μ_H	0.00795	0.58423
μ_A	0.0169	0.03997
μ_F	0.14	-1.3958
ω_1	0.79	0.029476
ω_2	0.21	-0.69521
ζ_1	0.69	-1.3472
ζ_2	0.211	0.024342
ν_2	0.03	0.024342
κ	0.115	-0.023604
π	0.2	-0.39048
β_H	1	0
β_A	1	0.0051338
β_F	1	0.6979
g_H	0.02856	0
g_A	0.02856	0.70304
h_A	1.39	0.029476

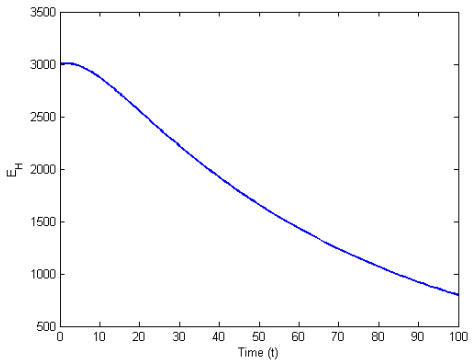
Thus, this study shows that effective disease control entails a multi-faceted approach based on minimizing the transmission probability from infected sandflies to animals, transmission probability from infected animals to sandflies, and per capita biting rate of sandflies of animals; increasing the rate of transfer from symptomatic infected animals to the recovered class; and the early diagnosis of ZVL cases in animals (reservoirs), among others.

The simulations also show that ZVL modeling studies in communities may require the implementation of an insecticide-based treatment strategy of infected animals (reservoirs) to reduce disease transmission and burden, and increase sandfly mortality, which is the main source of ZVL transmission.

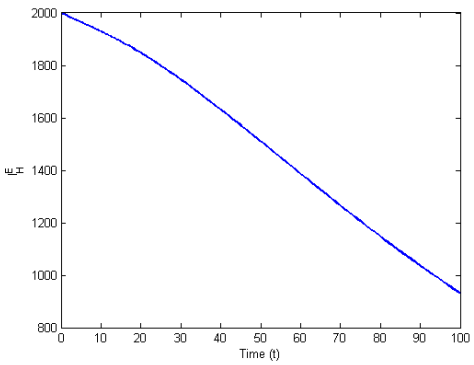
The profiles of the state variables are given in Figure 2a–t:



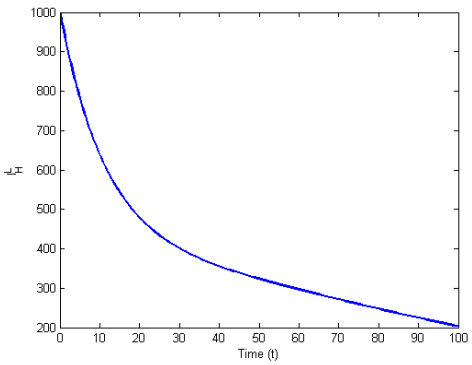
(**a**) Profile of S_H



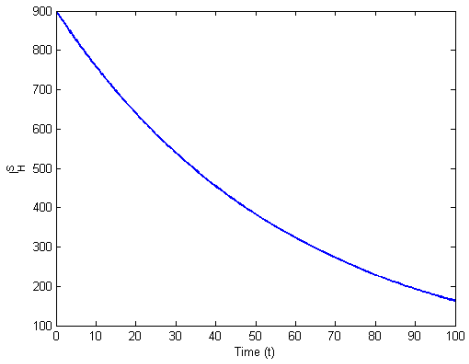
(**b**) Profile of E_H



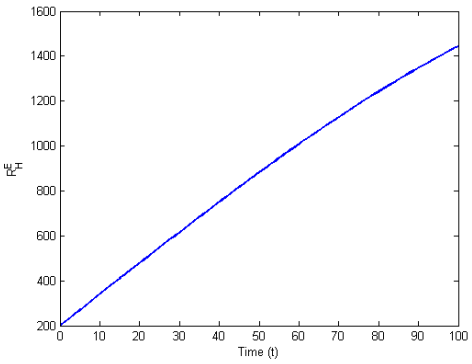
(**c**) Profile of I_H^E



(**d**) Profile of I_H^L

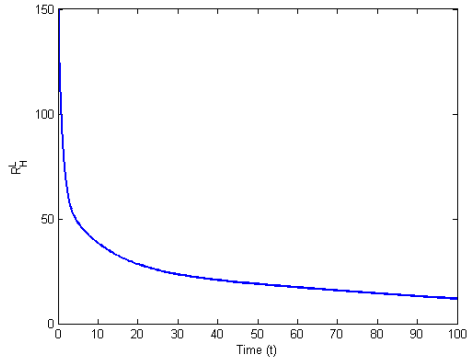


(**e**) Profile of I_H^S

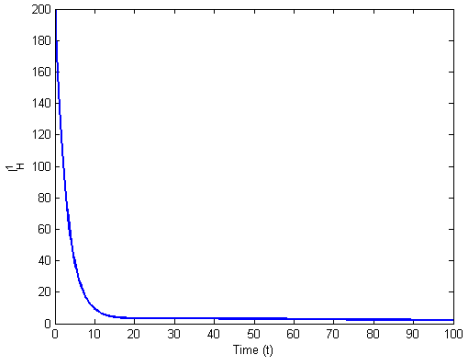


(**f**) Profile of R_H^E

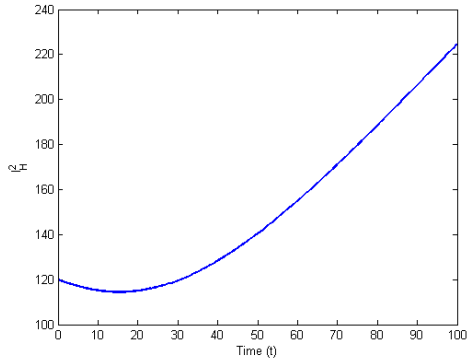
Figure 2. *Cont.*



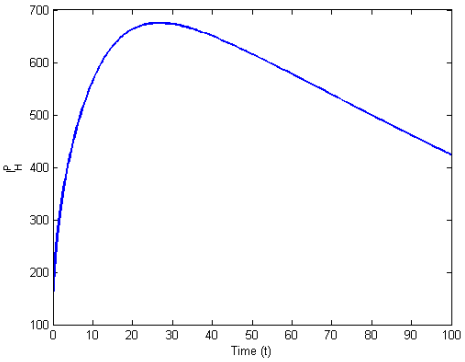
(**g**) Profile of R_H^L



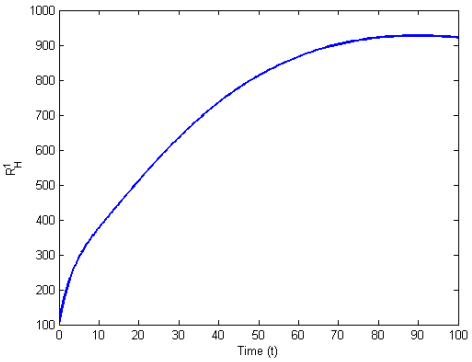
(**h**) Profile of I_H^1



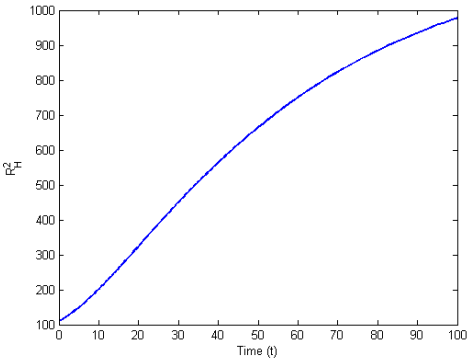
(**i**) Profile of I_H^2



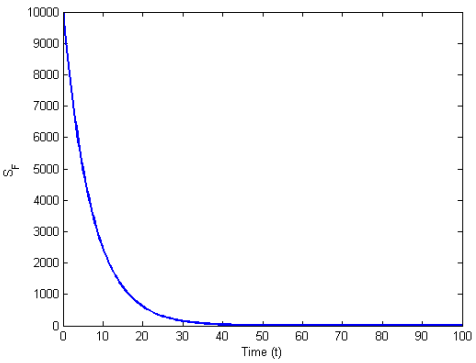
(**j**) Profile of I_H^P



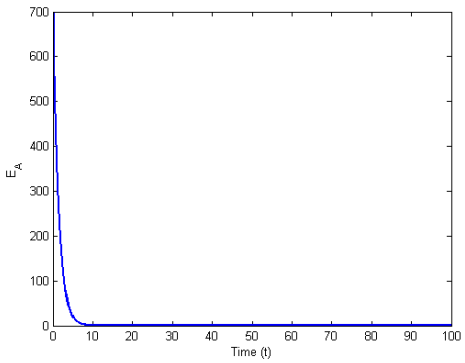
(**k**) Profile of R_H^1



(**l**) Profile of R_H^2

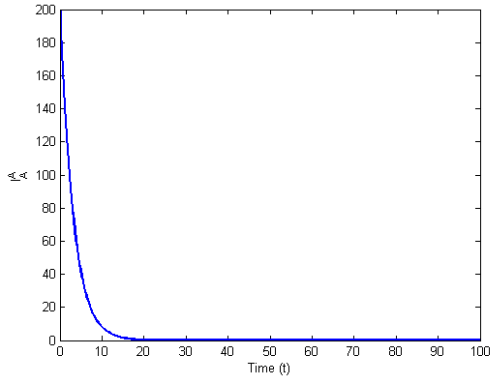


(**m**) Profile of S_A

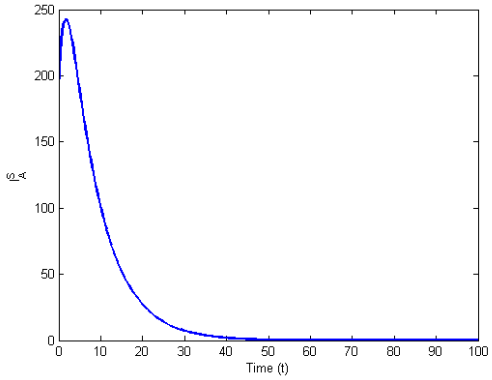


(**n**) Profile of E_A

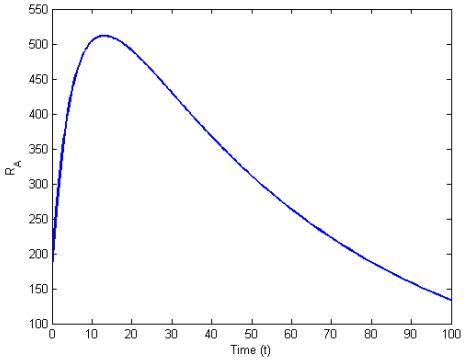
Figure 2. *Cont.*



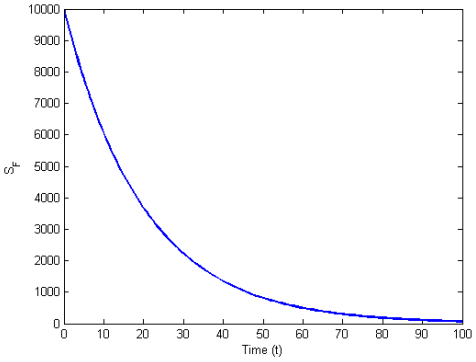
(**o**) Profile of I_A^A



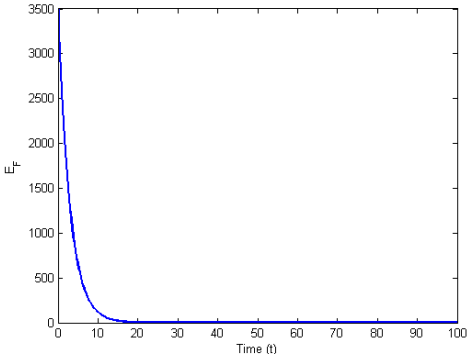
(**p**) Profile of I_A^S



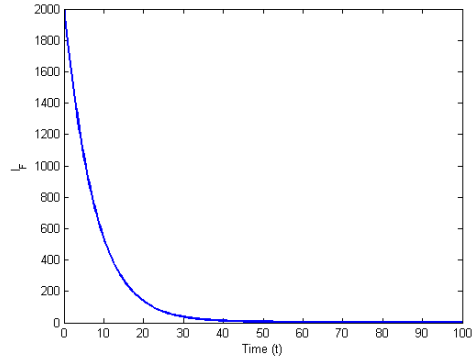
(**q**) Profile of R_A



(**r**) Profile of S_F



(**s**) Profile of E_F



(**t**) Profile of I_F

Figure 2. Profiles for behavior of each state variable of the model.

6. Conclusions

In this paper, a mathematical model of the transmission dynamic of ZVL incorporating several additional complexities was considered. These complexities include lines of treatment (first and second lines of treatment) to reflect the healthcare interventions for visceral leishmaniasis, depending on the progression of the disease and the response to initial treatments, and asymptomatic and symptomatic stages in animals to differentiate between asymptomatic and symptomatic stages in animal hosts, which is crucial because animals, particularly canines, play a significant role in the transmission cycle of zoonotic visceral leishmaniasis. Asymptomatic animals can still be infectious, which complicates control efforts.

Providing precise instructions for disease control, the stability analysis demonstrates that the stability of the VL-endemic and VL-free fixed points globally will make the de-

velopment of strategies for the eradication of the disease easy. Public health actions can be made simpler by emphasizing the necessity of maintaining R_0 below one in order to eradicate the disease.

Furthermore, the research's findings are consistent with the previous findings in the literature, particularly regarding the significance of an integrated strategy that addresses several elements of transmission, prioritizes vector (sandfly) control, and regulates reservoirs (animals). The research's other distinctive features include an emphasis on the targeted increase in sandfly mortality as determined by the analysis, the recommendation for insecticide-based treatment specifically for infected animals, and a clear understanding of the various stages of infection in both humans and animals. These elements reflect new approaches to resource optimization, even though they are in line with more general ZVL management techniques.

The model can be used for forecasting the disease outcomes and directing management methods because of its analytical and numerical consistency, which increases its credibility. This is further improved by the sensitivity analysis, which identifies crucial variables that have a major impact on ZVL dynamics and helps to come up with more successful intervention plans.

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Data Availability Statement: All data analyses are included in this article.

Conflicts of Interest: The authors declare no conflicts of interest.

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