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Mathematical model for the transmission dynamics of Leptospirosis in human population

Felix Yakubu Eguda[1,3*], Ali Maianguwa Shuaibu[1], Ocheme Christian Ameh[1], Samuel Eneojo Abah[2,3,4,5,], Sule Mustapha Mohammed[1] and Ibrahim Sulaiman Mohammed[6]

[1]Department of Mathematics, Federal University, Dutse, Jigawa State, Nigeria.
[2]Department of Applied Biology, Coventry University, United Kingdom.
[3]British Community Hospital and Outpatient Clinics Ipaja, Lagos, Nigeria.
[4]British Pathodiagnostic and Biotechnology Laboratory, Ipaja, Lagos, Nigeria.
[5]The Francis Crick Institute, London, United Kingdom.
[6]Institute of Strategic Industrial Decision Modelling (ISIDM), School of Quantitative Science, Universtit Utara Malaysia, Malaysia.

**Correspondence: felyak_e@yahoo.co.uk*

Abstract

Leptospirosis infection is a contractable disease which is caused by bacteria known as *Leptospira*. It can lead to serious complications and can pose serious health challenge if not treated promptly and effectively. This paper considered the formulation of a model with eight compartments for leptospirosis transmission in human population to study and analyze the dynamics of the infection. The threshold parameter known as basic reproduction number, a vital quantity for estimating the trends of the spread of the disease was derived. It was discovered from the analysis that model (1) exhibits a disease-free equilibrium point. This was further verified as being both locally and globally asymptotically unchanging whenever the effective basic reproduction number is less than one. Model (1) has an endemic equilibrium point which was as well proved to be both locally and globally asymptotically unchanging whenever the effective basic reproduction number is greater than unity. The study extends its analysis to verify backward bifurcation phenomenon of leptospirosis infection and was ascertained to exist due to loss of temporary immunity as human continue to interact with domestic animals, rodents found in stores and the use of recreational centers such as swimming pool after treatment. Finally, the work suggested the incorporation of vaccination as a control measure to prevent re-infection to human from leptospirosis disease.

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1.0 Introduction

Leptospirosis disease is known global as zoonotic disease triggered by bacteria called leptospira (WHO, 2006). Human and other animals, commonly livestock are mostly infected from this bacteria infection. Human contracted leptospirosis infection through drinking or interacting with water and other surfaces in which an infected rat (dead rats) is found, while cattle that drink this water becomes infectious (Muhammad *et al.*, 2016). Leptospirosis disease is transmitted by many animals such as rodents, skunks, opossums, raccoons, foxes, and other vermin. Human being acquired leptospirosis infection after interacting with contaminated surfaces by either of the secondary host organisms (Pongsumpun, 2012). It is also reported that people living in the cities are mostly infected by this disease which leads to liver infections (Pongsumpun, 2012). Leptospirosis infection is known and called different names at the early times as regards the people, occupation and days of its

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manifestation. Few of such is: Weil's disease, canicola fever, canefield fever, 7-day fever, nanukayami fever (Muhammad *et al.,* 2012). Leptospirosis infection indices are in various forms in human population to the extent that it can cause acute febrile illness and patient having this form of illness can recover naturally without medical treatment. The severe forms of this disease are usually referred to as Weil's disease. This disease can occur in various forms depending on the severity and observed symptoms such as acute renal failure, haemorrhagic diathesis and aseptic meningitis. Previous studies revealed that there is high mortality rate when leptospirosis infection deteriorates to severe pulmonary haemorrhagic form. It is on record that over 200 serovars of pathogenic leptospires are prevalent in human population. The different species of leptospira can further be divided into serovars groups as analyzed by a well-defined agglutination after cross-absorption with homologous antigen (Monahan *et al*., 2009).

Much attention has not been given to leptospirosis diseases in most tropical region and this has resulted to adverse effects on human and livestock health (Garba *et al*., (2018). This important negligence, especially in the technologically advanced regions of the world is partly due to the relatively low number of reported human cases, less unembellished complications and truncated incidence in animals especially in the temperate climate zones. On the other way, there is increase in cases of severe incidence of this infection in under-developed tropical countries (Garba *et al.,* 2018). The true incidence and global distribution of the disease is unknown (Muhammad *et al.,* 2014). Clinical analysis of the disease is quite problematic since similar symptoms can be observed in other infections like rickettsioses, dengue fever, malaria and yellow fever (Bharti *et al.,* 2003). Outbreak of leptospirosis infection depends on the season and may often be linked to environmental factors involving animal activities, agricultural activities and occupational migration (Allan, 2016). Researchers asserted that in case of vector-borne disease, focus should be given to eradication of the causative bacteria population as a way for controlling the disease. Others previous work considered administration of vaccine on the affected organism (Muhammad *et al*., 2012).

Mathematical models have gained wide acceptance as a vital tool for studying the dynamics of the transmission of infectious diseases. In the past decades, scholastic researchers and mathematicians including (Muhammad *et al*., 2012; Bharti *et al.,* 2003; Monahan *et al*., 2009) have accepted mathematical models to study leptospirosis fever (Weil's disease) and other related diseases. The present study extends the work of (Muhammad *et al*., 2014) by adding vaccination compartment as a control strategy in the leptospirosis model to determine the nature of stability of the disease, and further investigate vaccination as an important control measure that will aid in reducing the disease prevalence in human population.

2.0 Materials and methods

2.1 Formulation of Leptospirosis infection model

A model of total human population denoted as (N_h) and total vector population as (N_v) was formulated to describe the dynamics of leptospirosis infection. The human population at time, (t) is sub-divided into five (5) classes. Vector population at time (t) is sub-divided into three (3) classes. Susceptible (S), Expose (E_L), Active (infected) human population (I_L), Recovered human (R_L) and Vaccinated human (V); Susceptible vector (S_v), Infectious vector (I_v), and Recovered vector (R_v). $N_h = S + E_L + I_L + R_L + V$ and $N_v = S_v + I_v + R_v$.

Susceptible humans are recruited at a rate $\Lambda_h(1-f)$ [where $(1-f)$ are proportion of human not vaccinated], while susceptible vector are recruited at a rate Λ_v .

Susceptible human contract leptospirosis from infected vectors at a rate: $\lambda_{VL} = \frac{\beta_{VL} I_v (1-\theta)}{N_h}$

Susceptible vectors (domestic animals and rodents) acquire leptospirosis from infected human at a rate:

$$\lambda_{LV} = \frac{B_{LV}(\eta_A E_L + \eta_B I_L)}{N_h} \quad ,$$

where $\eta_A < \eta_B$ denotes relative infectiousness of human with expose leptospirosis compared to human infective (active) leptospirosis disease.

2.2 Equations of the model

$$
\begin{aligned}
\dot{V} &= f\Lambda_h - k_1 V \\
\dot{S} &= \Lambda_h(1-f) + \psi_L V + \chi_L R_L - \lambda_L S - \mu_h S \\
\dot{E}_L &= \lambda_L S - k_2 E_L \\
\dot{I}_L &= \alpha_L E_L - k_3 I_L \\
\dot{R}_L &= \tau_L I_L - k_4 R_L \\
\dot{S}_v &= \Lambda_v + \gamma_v R_v - \lambda_v S_v - \mu_v S_v \\
\dot{I}_v &= \lambda_v S_v - k_6 I_v \\
\dot{R}_v &= \alpha_v I_v - k_7 R_v
\end{aligned}
\tag{1}
$$

where, $k_1 = (\psi_L + \mu_h)$, $k_2 = (\alpha_L + \mu_h)$, $k_3 = (\tau_L + \mu_h + \delta_L)$, $k_4 = (\chi_L + \mu_h)$, $k_5 = (\mu_v + \alpha_v + \delta_v)$ and $k_6 = (\mu_v + \gamma_v)$

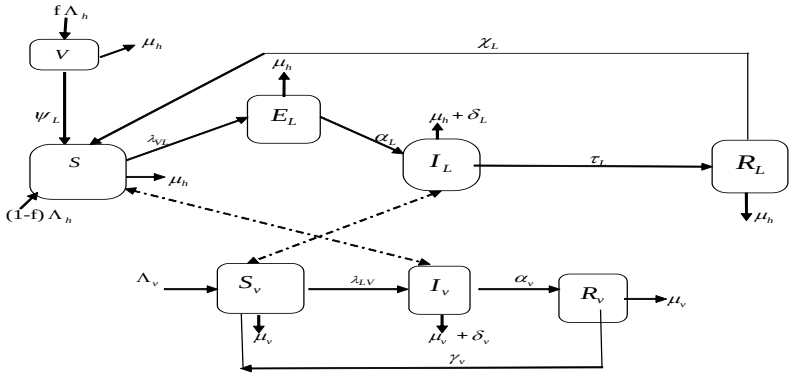

Figure 1. Flow diagram of Leptospirosis infection

Table 1: Description of state variables for equation (1)

Symbols	Description
V	Vaccinated human population against leptospirosis infection
S	Susceptible human population
E_L	Exposed human (asymptomatic) with potential leptospirosis causing bacteria
I_L	Infected (active) human with leptospirosis disease
R_L	Treated human population of leptospirosis disease
S_V	Susceptible vector population
I_V	Infectious (active) vector population with leptospirosis disease
R_V	Recovered vector population from leptospirosis disease

2.3 Basic analysis of the model (1) equation

2.4 Boundedness of solutions of model (1) equations

We shall consider the region $\Omega_2 = \left\{(V, S, \mathrm{E_L}, \mathrm{I_L}, R_L, S_v, I_v, \mathrm{R_v}) \in \mathbb{R}_+^8 : N_h \le \frac{\Lambda_h}{\mu_h},\ N_v \le \frac{\Lambda_v}{\mu_v}\right\}$. We prove that the region Ω_2 is positively invariant and an attractor of all positive solutions of the model (1).

Table 2: Description of parameters for leptospirosis disease

Symbol	Description	Value (Year^{-1})	Reference
Λ_h	Birth or recruitment rate of human into the susceptible population	6000	Assumed
μ_h	Natural death rate of all human subclasses	0.00046	(Okosun, *et al*. 2016)
ψ_L	Waning rate of temporary immunity of the vaccinated human	0.75	(Okosun, *et al.* 2016)
τ_L	Treatment rate of infected human with leptospirosis	0.25	(Okosun, *et al.* 2016)
χ_L	Rate at which recovered human from leptospirosis lose immunity	0.0004	(Okosun, *et al.* 2016)
α_L	Progression rate of carrier human into infective subclass	0.0027	(Okosun, *et al.* 2016)
δ_L	Disease induced death rate of human due to leptospirosis	0.001	(Okosun, *et al.* 2016)
Λ_v	Birth or recruitment rate of vector into the susceptible population	600	Assumed
μ_v	Natural death rate of the vector	0.0018	(Khan, *et al.* 2012)
α_v	Recovery rate of infected vector	0.05	(Okosun, *et al.* 2016)
δ_v	Death rate of vector due to leptospirosis disease	0.0018	(Khan, *et al.* 2012)
λ_{LV}	Force of infection of vector with leptospirosis from human	0.074	(Khan, *et al* . 2012)
γ_v	Waning rate of temporal immunity of recovered vectors	0.004	(Khan, *et al* . 2012)
f	Proportion of human population vaccinated against leptospirosis disease	0.8	Assumed
β_{LV}	Effective contact rate for leptospirosis from vector to human	0.05	(Khan, *et al* . 2012)
β_{LV}	Effective contact rate for leptospirosis from human to vector	0.000078	(Khan, *et al* . 2012)
η_A	Relative infectiousness of human with latent leptospirosis at exposed class	0.05	Assumed
η_B	Relative infectiousness of human with latent leptospirosis at infected class	0.8	Assumed
θ	Compliance rate to public awareness campaign	0.25	Assumed

Lemma 1: The region Ω_2 is positively invariant for the model (1)

Proof: The rate of change of the total human population is given thus:

$$\dot{N}_h = \dot{V} + \dot{S} + \dot{E}_L + \dot{I}_L + \dot{R}_L \tag{2}$$

$$\dot{N}_h = \Lambda_h - \mu_h N_h - \delta_L I_L \tag{3}$$

By standard comparison theorem

$$\dot{N}_h \leq \Lambda_h - \mu_h N_h \tag{4}$$

Integrating both sides by integrating factor method gives:

$$N_h(t) \leq N_h(0)^{-u_h t} + \frac{\Lambda_h}{\mu_h}\left(1 - e^{-\mu_h t}\right) \tag{5}$$

Also, for the vector compartment,

$$\dot{N}_v = \Lambda_v - \mu_v N_v - \delta_v I_v \tag{6}$$

By standard comparison theorem

$$\dot{N}_v \leq \Lambda_v - \mu_v N_v \tag{7}$$

Similarly, solving (7) by integrating factor method we obtain

$$N_v(t) \leq N_v(0)^{-u_v t} + \frac{\Lambda_v}{\mu_v}\left(1 - e^{-\mu_v t}\right) \tag{8}$$

In particular, $N_h(t) \le \frac{\Lambda_h}{\mu_h}$ if $N_h(0) \le \frac{\Lambda_h}{\mu_h}$ and $N_v(t) \le \frac{\Lambda_v}{\mu_v}$ if $N_v(0) \le \frac{\Lambda_v}{\mu_v}$, respectively.

Hence Ω_2 is a positively invariant set and the solution enters Ω_2 in finite time or $N_h(t) \to \frac{\Lambda_h}{\mu_h}$ and $N_v(t) \to \frac{\Lambda_v}{\mu_v}$ as $t \to \infty$. Therefore, it becomes relevant to study the dynamics of the model (1) in the region, Ω_2. In this space, model (1) is considered scientifically (mathematically) and epidemiologically well posed.

2.5 Positivity of solutions of model (1)

Lemma 2: Let the initial data for model (1) be:
$S(t)>0, E_L(t)>0, I_L(t)>0, R_L(t)>0, S_v(t)>0, I_v(t)>0, \text{and} R_v(t)>0$, then the solution
$\{ S(t), E_L(t), I_L(t), R_L(t), S_v(t), I_v(t) \text{and} R_v(t) \}$ with real positive number initial data will remain positive at all time $t>0$.

Proof: Let $\Omega_2 = \sup \{t>0; S(t)>0, E_L(t)>0, I_L(t)>0, R_L(t)>0, S_v(t)>0, I_v(t)>0, R_v(t)>0\} > 0$ for the model (1), and assuming ($\psi_L = \chi_L = 0$)

$$\dot{S} \ge \Lambda_h(1-f) - (\lambda_{\lambda_{VL}} + \mu_h)S \tag{9}$$

Solving by integrating factor method

$$\text{I.F} = exp(\mu_h t) + exp\left(\int_0^t \lambda_{VL}(\tau)d\tau\right) \tag{10}$$

$$\frac{dS}{dt}\left[S(t)\left(exp(\mu_h t) + exp\left(\int_0^t \lambda_{VL}(\tau)d\tau\right)\right)\right] \ge \Lambda_h(1-f)\left[exp(\mu_h t) + exp\left(\int_0^t \lambda_{VL}(\tau)d\tau\right)\right]$$

Integrating both sides results to

$$S(t)exp\left\{\mu_h t + \int_0^t \lambda_{VL}(\tau)d\tau\right\} - S(0) \ge \int_0^t \Lambda_h(1-\text{f})\left[exp\left\{\mu_h y + \int_0^y \lambda_{VL}(\tau)d\tau\right\}\right]dy$$

$$S(t)exp\left\{\mu_h t + \int_0^t \lambda_{VL}(\tau)d\tau\right\} \ge S(0) + \int_0^t \Lambda_h(1-\text{f})\left[exp\left\{\mu_h y + \int_0^y \lambda_{VL}(\tau)d\tau\right\}\right]dy$$

$$S(t) \ge S(0)exp\left\{-\mu_h t - \int_0^t \lambda_{VL}(\tau)d\tau\right\} + exp\left\{-\mu_h t - \int_0^t \lambda_{VL}(\tau)d\tau\right\} \times \int_0^t \left[\Lambda_h(1-\text{f})exp\left\{\mu_h y + \int_0^y \lambda_{VL}(\tau)d\tau\right\}\right]dy \ > 0 \tag{11}$$

It is therefore accomplished that all the state variables of model (1) are real number whenever $t=0$, That is; $S(t)>0, E_L(t)>0, I_L(t)>0, R_L(t)>0, S_v(t)>0, I_v(t)>0, \text{and} R_v(t)>0$ at all $t>0$. This completes the proof.

2.6 Local stability of Disease-Free Equilibrium (DFE) of model (1)

We solved and obtained the disease-free equilibrium of model (1) by setting the left-hand side of model (1) equations to zero and setting the classes with infection to zero as shown below.

$$\xi_L{}^* = \left(V^*, S^*, E_L{}^*, I_L{}^*, R_L{}^*, S_v{}^*, I_v{}^*, R_v{}^*\right) = \left(\frac{f\Lambda_h}{k_1}, \frac{\Lambda_h(1-f)k_1 + \psi_L f\Lambda_h}{\mu_h k_1}, 0, 0, 0, \frac{\Lambda_v}{\mu_v}, 0, 0\right) \tag{12}$$

We would establish the steady state of ξ_L^* by next generation operator approach as shown in Van Den Driessche and Watmough, (2002). We do this by computing matrices F and V^{-1} thus:

$$F = \begin{bmatrix} 0 & 0 & \frac{B_{VL}(1-\theta)S^*}{N_h^*} \\ 0 & 0 & 0 \\ \frac{B_{LV}\eta_A S_v^*}{N_h^*} & \frac{B_{LV}\eta_B S_v^*}{N_h^*} & 0 \end{bmatrix} \tag{13}$$

$$V^{-1}=\begin{bmatrix} \frac{1}{k_2} & 0 & 0 \\ \frac{\alpha_L}{k_2k_3} & \frac{1}{k_3} & 0 \\ 0 & 0 & \frac{1}{k_5} \end{bmatrix} \tag{14}$$

$$FV^{-1}=\begin{bmatrix} 0 & 0 & \frac{B_{VL}(1-\theta)S^*}{N_h^*k_5} \\ 0 & 0 & 0 \\ \frac{B_{LV}(\eta_Ak_3+\eta_B\alpha_L)S_v^*}{N_h^*k_2k_3} & \frac{B_{LV}\eta_BS_v^*}{N_h^*k_3} & 0 \end{bmatrix} \tag{15}$$

The eigenvalue is obtained as:

$$R_{EL}=\sqrt{\frac{B_{VL}(1-\theta)S^*B_{LV}(\eta_Ak_3+\eta_B\alpha_L)S_V^*}{\mathrm{N}_h^{*2}k_2k_3k_5}} \tag{16}$$

$$R_{EL}=\sqrt{R_H.R_V} \tag{17}$$

$$R_H=\sqrt{\frac{B_{VL}(1-\theta)S^*}{\mathrm{N}_h^*k_2k_3}} \tag{18}$$

$$R_V=\sqrt{\frac{B_{LV}(\eta_Ak_3+\eta_B\alpha_L)S_V^*}{\mathrm{N}_h^*k_5}} \tag{19}$$

Where, $k_2=(\alpha_L+\mu_h), k_3=(\tau_L+\mu_h+\delta_L), \mathrm{k}_5=(\alpha_v+\mu_v+\delta_v)$

Theorem 1: The disease-free equilibrium, (DFE) of model (1) is locally asymptotically stable if $R_{EL}<1$ and unstable if $R_{EL}>1$.

Proof: By theorem 1, epidemiologically and mathematically, leptospirosis infection would be eradicated from human population when $R_{EL}<1$ whenever the initial population size of subhuman of model (1) is within the region of attraction of ξ_L^*.

2.7 Global stability of disease-free equilibrium of model (1)

Theorem 2: Disease-free equilibrium ξ_L^* is globally asymptotically stable, (GAS) in the region Ω_2 whenever $R_{EL}<1$.

Proof: We would show this theorem 2 by developing a Lyapunov function with positive constants technically chosen as D_1, D_2 and D_3 such that $D_1>0$, $D_2>0$ and $D_3>0$.

$$\begin{aligned} V=&(\mathrm{S}-S^o-S^o\ln\frac{S}{S^o})+D_1E_L+D_2I_L+(R_L-R_L^o-R_L^o\ln\frac{R_L}{R_L^o})+(V-V^o-V^o\ln\frac{V}{V^o}) \\ &+(\mathrm{S}_v-S_v^o-S_v^o\ln\frac{S_v}{S_v^o})+D_3I_v+(\mathrm{R}_v-R_v^o-R_v^o\ln\frac{R_v}{R_v^o}) \end{aligned} \tag{20}$$

The derivative of (20) with respect to time t, is obtained as:

$$\frac{dV}{dt}=(1-\frac{S^o}{S})\frac{dS}{dt}+D_1\frac{dE_L}{dt}+D_2\frac{dI_L}{dt}+(1-\frac{R_L^o}{R_L})\frac{dR_L}{dt}+(1-\frac{V^o}{V})\frac{dV}{dt}+(1-\frac{S_v^o}{S_v})\frac{dS_v}{dt}+D_3\frac{dI_v}{dt}+(1-\frac{R_v^o}{R_v})\frac{dR_v}{dt} \quad (21)$$

Substituting the values for the derivatives of model equation (1) into (21) gives:

$$\begin{aligned}\frac{dV}{dt}=&(1-\frac{S^o}{S})\left[\Lambda_h(1-f)+\psi_L V+\chi_L R_L-(\lambda_{VL}+\mu_h)S\right]+D_1\left[\lambda_{VL}S-(\alpha_L+\mu_h)E_L\right]\\&+D_2\left[\alpha_L E_L-(\tau_L+\mu_h+\delta_L)I_L\right]+(1-\frac{R_L^o}{R_L})\left[\tau_L I_L-(\chi_L+\mu_h)R_L\right]\\&+(1-\frac{V^o}{V})\left[f\Lambda_h-(\psi_L+\mu_h)V\right]+(1-\frac{S_v^o}{S_v})\left[\Lambda_v+\gamma_v R_v-(\lambda_{LV}+\mu_v)S_v\right]\\&+D_3\left[\lambda_{LV}S_v-(\mu_v+\alpha_v+\delta_v)I_v\right]+(1-\frac{R_v^o}{R_v})\left[\alpha_v I_v-(\mu_v+\gamma_v)R_v\right]\end{aligned} \quad (22)$$

After forming the Lyapunov function over the eight state variables, ($S,V,\ E_L,I_L,R_L,S_v,I_v,\ R_v$), we introduce the idea from (Martcheva, 2015). It is clear that if $(E_L(t),I_L(t),\ I_v(t))$ at DFE are globally stable, thus

($I_L=E_L=I_v=0$)

This implies that $S(t)=\Lambda_h(1-f)+\psi_L V/\mu_h$, $V(t)=f\Lambda_h/(\psi_L+\mu_h), S_v(t)=\Lambda_v/\mu_v$ (23)

By Standard comparison theorem

$$S\le S^o=\Lambda_h(1-f)+\psi_L V/\mu_h, V\le V^o=f\Lambda_h/(\psi_L+\mu_h), S_v\le S_v^o=\Lambda_v/\mu_v \quad (24)$$

Substituting (24) into (22) gives:

$$\frac{dV}{dt}\le D_1\left[\lambda_{VL}S^*-(\alpha_L+\mu_h)E_L^*\right]+D_2\left[\alpha_L E_L^*-(\tau_L+\mu_h+\delta_L)I_L^*\right]+D_3\left[\lambda_{LV}S_v^*-(\mu_v+\alpha_v+\delta_v)I_v^*\right] \quad (25)$$

$$\le D_1\left[\frac{B_{VL}(1-\theta)\mathrm{I}_v}{N_h^*}S^*-k_2E_L^*\right]+D_2\left[\alpha_L E_L^*-k_3I_L^*\right]+D_3\left[\frac{B_{LV}(\eta_A k_3+\eta_B\alpha_L)}{N_A^*}S_v^*-k_5I_v^*\right] \quad (26)$$

$$\begin{aligned}\le\ &+D_1\left[\frac{B_{VL}(1-\theta)\Lambda_h((1-\mathrm{f})k_1+\psi_L\,\mathrm{f})}{N_h^*\mu_h k_1}\mathrm{I}_v-k_2E_L^*\right]\\&+D_2\left[\alpha_L E_L^*-k_3I_L^*\right]\\&+D_3\left[\frac{B_{LV}(\eta_A k_3+\eta_B\alpha_L)\Lambda_v}{N_h^*\mu_v}-k_5I_v^*\right]\end{aligned} \quad (27)$$

$$
\begin{aligned}
\leq\ & + D_1\left(\frac{B_{VL}(1-\theta)\Lambda_h((1-\mathrm{f})k_1+\psi_L\,\mathrm{f})}{N_h^*\mu_h k_1}\right)\mathrm{I}_v^* \\
& - D_1 k_2 E_L^* \\
& + D_2\alpha_L E_L^* \\
& - D_2 k_3 I_L^* \\
& + D_3\frac{B_{LV}\eta_A\Lambda_v}{N_h^*\mu_v}E_L^* \\
& + D_3\frac{B_{LV}\eta_B\Lambda_v}{N_h^*\mu_v}I_L^* \\
& - D_3 k_5 I_v^*
\end{aligned} \tag{28}
$$

Collecting like terms gives:

$$
\begin{aligned}
\frac{dV}{dt} \leq\ & \left(\frac{D_1 B_{VL}(1-\theta)\Lambda_h((1-\mathrm{f})k_1+\psi_L\,\mathrm{f})}{N_h^*\mu_h k_1} - D_3 k_5\right)\mathrm{I}_v^* \\
& + \left(D_2\alpha_L - D_1 k_2 + D_3\frac{B_{LV}\eta_A\Lambda_v}{N_h^*\mu_v}\right)E_L^* \\
& + \left(D_3\frac{B_{LV}\eta_B\Lambda_v}{N_h^*\mu_v} - D_2 k_3\right)I_L^*
\end{aligned} \tag{29}
$$

Equating the coefficient of $I_v^*, E_L^*, I_L^* = 0$, and solving gives

$$D_2 = \frac{B_{LV}\eta_B\Lambda_v}{N_h^*\mu_v} \tag{30}$$

$$D_3 = k_3$$

$$D_1 = \frac{B_{LV}\Lambda_v(\eta_A k_3 + \eta_B\alpha_L)}{N_h^*\mu_v k_3} \tag{31}$$

$$\frac{dV}{dt} \leq \left(\frac{D_1 B_{VL}(1-\theta)\Lambda_h((1-\mathrm{f})k_1+\psi_L\,\mathrm{f})}{N_h^*\mu_h k_1} - D_3 k_5\right) \tag{32}$$

Substituting for D_1 and D_3 into (32) gives

$$\frac{dV}{dt} \leq \left(\frac{B_{LV}\Lambda_v B_{VL}(1-\theta)\Lambda_h((1-\mathrm{f})k_1+\psi_L\,\mathrm{f})(\eta_A k_3+\eta_B\alpha_L)}{N_h^{*2}\mu_v\,\mu_h k_1 k_2} - k_3 k_5\right)I_L$$

$$\frac{dV}{dt} \leq k_3 k_5\left[\frac{B_{VL}(1-\theta)S^* B_{LV}(\eta_A k_3+\eta_B\alpha_L)\mathrm{S}_v^*}{N_h^{*2}k_2 k_3 k_5} - 1\right]I_L \tag{33}$$

$$\frac{dV}{dt} \leq k_3 k_5\left[R_{EL}^2 - 1\right]I_L \text{ , if } R_{EL} \leq 1. \tag{34}$$

Also, $\frac{dV}{dt}=0$ if and only if $I_L=0$. Therefore, for $E_L=I_L=I_v=0$ it shows that $S(t)\Rightarrow\Lambda_h(1-f)+\psi_L V/\mu_h, V(t)\Rightarrow f\Lambda_h/\psi_L+\mu_h, S_v(t)\Rightarrow\Lambda_v/\mu_v$ whenever $t\to\infty$. Hence, the principal compact invariant set in the set $(S,V,E_L,I_L,R_L,S_v,I_v,R_v)\in\Omega_2: dV/dt\le 0$. Therefore, from LaSalle's invariance principle, we conclude that ξ_0^* is globally asymptotically stable in Ω_2 if $R_{EL}<1$.

2.8 Existence of endemic equilibrium point of model (1)

We represent the endemic equilibrium point (EEP) of model (1) as:

$\xi_2^{**}=\left(V^{**},S^{**},E_L^{**},I_L^{**},R_L^{**},S_v^{**},I_v^{**},R_v^{**}\right)$ and solving the model equations (1) in relationship with the force of infection at equilibrium state gives the following (assumption: let $\chi_L=\gamma_v=0$).

$$\left(V^{**},S^{**},E_L^{**},I_L^{**},R_L^{**},S_v^{**},I_v^{**},R_v^{**}\right)=\left\{\begin{array}{l}\frac{f\Lambda_h}{k_1},\frac{\Lambda_h(1-f)k_1+\psi_L f\Lambda_h}{(\lambda_{VL}^{**}+\mu_h)k_1},\frac{(\Lambda_h(1-f)k_1+\psi_L f\Lambda_h)\lambda_{VL}^{**}}{(\lambda_{VL}^{**}+\mu_h)k_1k_2},\frac{\alpha_L(\Lambda_h(1-f)k_1+\psi_L f\Lambda_h)\lambda_{VL}^{**}}{(\lambda_{VL}^{**}+\mu_h)k_1k_2k_3},\\ \frac{\alpha_L(\Lambda_h(1-f)k_1+\psi_L f\Lambda_h)\lambda_{VL}^{**}}{(\lambda_{VL}^{**}+\mu_h)k_1k_2k_3k_4},\frac{\Lambda_v}{(\lambda_{LV}^{**}+\mu_v)},\frac{\Lambda_v\lambda_{LV}^{**}}{(\lambda_{LV}^{**}+\mu_v)k_5},\frac{\alpha_v\Lambda_v\lambda_{LV}^{**}}{(\lambda_{LV}^{**}+\mu_v)k_5k_6}\end{array}\right\}$$

Recall that:

$$\lambda_{VL}^{**}=\frac{B_{VL}(1-\theta)I_v^{**}}{N_h^{**}},\quad \lambda_{LV}^{**}=\frac{B_{LV}(\eta_A E_L^{**}+\eta_B I_L^{**})}{N_h^{**}} \tag{35}$$

Substituting the expression for I_v^{**} in (35) gives:

$$\lambda_{VL}^{**}=\frac{B_{VL}(1-\theta)\Lambda_v\lambda_{LV}^{**}}{N_h^{**}(\lambda_{LV}^{**}+\mu_v)k_5} \tag{36}$$

Substituting for I_L^{**} *and* E_L^{**} in (35) gives:

$$\lambda_{LV}^{**}=\frac{B_{LV}\left(\frac{\eta_A\cdot(\Lambda_h(1-f)k_1+\psi_L f\Lambda_h)\lambda_{VL}^{**}}{k_1k_2(\lambda_{VL}^{**}+\mu_h)}+\frac{\eta_B\cdot(\alpha_L(1-f)k_1+\psi_L f\Lambda_h)\lambda_{VL}^{**}}{k_1k_2k_3(\lambda_{VL}^{**}+\mu_h)}\right)}{N_h^{**}}$$

$$\lambda_{LV}^{**}=\frac{B_{LV}(\Lambda_h(1-f)k_1+\psi_L f\Lambda_h)\left[\eta_A k_3+\eta_B\alpha_L\right]\lambda_{VL}^{**}}{N_h^{**}k_1k_2k_3(\lambda_{VL}^{**}+\mu_h)} \tag{37}$$

Substituting (37) into (36) gives:

$$\lambda_{VL}^{**}=\frac{\frac{B_{VL}(1-\theta)\Lambda_v B_{LV}\left[k_3\eta_A\Lambda_h((1-f)k_1+\psi_L f)+\alpha_L\eta_B\Lambda_h((1-f)k_1+\psi_L f)\right]\lambda_{VL}^{**}}{k_1k_2k_3(\lambda_{VL}^{**}+\mu_h)}}{k_5N_h^{**}\left[\frac{B_{LV}\left[k_3\eta_A\Lambda_h((1-f)k_1+\psi_L f)+\alpha_L\eta_B\Lambda_h((1-f)k_1+\psi_L f)\right]\lambda_{VL}^{**}}{k_1k_2k_3(\lambda_{VL}^{**}+\mu_h)}+\mu_v N_h^{**}\right]}$$

Simplifying the above expression gives

$$\lambda_{VL}^{**}=\frac{A\Lambda_v B_{LV}M_9\lambda_{VL}^{**}}{k_5N_h^{**}B_{LV}M_9\lambda_{VL}^{**}+k_5N_h^{**2}\mu_v P\lambda_{VL}^{**}+k_5N_h^{**2}\mu_v P\mu_h} \tag{38}$$

$$A_1\lambda_{VL}^{**2}+A_2\lambda_{VL}^{**}=0 \tag{39}$$

$$\lambda_{VL}^{**}(A_1\lambda_{VL}^{**}+A_2)=0$$

$$\lambda_{VL}^{**}=-\frac{A_2}{A_1} \tag{40}$$

Where $A_1=k_5N_h^{**}B_{LV}\Lambda_h((1-f)k_1+\psi_L f)[\eta_A k_3+\eta_B\alpha_L]+k_5N_h^{**2}\mu_v k_1k_2k_3$

$$A_2=N_h^{**2}k_1k_2k_3k_5\mu_h\mu_v-B_{VL}(1-\theta)\Lambda_v B_{LV}\Lambda_h((1-f)k_1+\psi_L f)[\eta_A k_3+\eta_B\alpha_L]$$

$$= N_h^{**2} k_1 k_2 k_3 k_5 \mu_h \mu_v \left[1 - \frac{B_{VL}(1-\theta)\Lambda_v B_{LV} \Lambda_h ((1-f)k_1 + \psi_L f)[\eta_A k_3 + \eta_B \alpha_L]}{N_h^{**2} k_1 k_2 k_3 k_5 \mu_h \mu_v} \right]$$

$$N_h^{**2} k_1 k_2 k_3 k_5 \mu_h \mu_v \left[1 - R_{EL}{}^2 \right] \quad (41)$$

$A_2 < 0$ if $R_{EL} > 1$

Therefore, model equation (1) has a distinctive (stable) endemic equilibrium if $R_{EL} > 1$ since $A_2 < 0$ for $R_{EL} > 1$.

2.9 Local stability of endemic equilibrium of model (1)

The Jacobian expressed in terms of force of infection is obtained thus:

$$J(\xi_2^{**}) = \begin{bmatrix} -k_1 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ \psi_L & -(\lambda_{VL}^{**} + \mu_h) & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & \lambda_{VL}^{**} & -k_2 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & \alpha_L & -k_3 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & \tau_L & -k_4 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & -(\lambda_{LV}^{**} + \mu_v) & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & \lambda_{LV}^{**} & -k_5 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & \alpha_v & -k_6 \end{bmatrix}$$

Resolving the $J(\xi_2^{**})$ using upper triangular matrix, we obtain thus:

$$J(\xi_2^{**}) = \begin{bmatrix} -k_1 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & -(\lambda_{VL}^{**} + \mu_h) & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & -k_2 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & -k_3 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & -k_4 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & -(\lambda_{LV}^{**} + \mu_v) & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & -k_5 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & -k_6 \end{bmatrix} \quad (42)$$

the eigenvalues are:

$$\lambda_1 = -k_1 < 0, \lambda_2 = -(\lambda_{VL}^{**} + \mu_h), \lambda_3 = -k_2 < 0, \lambda_4 = -k_3 < 0, \lambda_5 = -k_4 < 0, \lambda_6 = -(\lambda_{LV}^{**} + \mu_v), \lambda_7 = -k_5 < 0, \lambda_8 = -k_6 < 0$$

Recall that:

$$\lambda_{VL}^{**} = -\frac{A_2}{A_1} = \frac{k_1 k_2 k_3 k_5 \mu_v \mu_h N_h^{**2} \left[R_{EL}{}^2 - 1 \right]}{k_5 N_h^{**} B_{LV} \Lambda_h ((1-f)k_1 + \psi_L f) + k_5 N_h^{**} \mu_v k_1 k_2 k_3} > 0$$

If $R_{EL} > 1$, therefore $\lambda_2 = -(\lambda_{VL}^{**} + \mu_h) < 0$

Also, $\lambda_6 = -(\lambda_{LV} + \mu_v)$

$$\lambda_{LV}^{**} = \frac{B_{LV} \Lambda_h ((1-f)k_1 + \psi_L f)[\eta_A k_3 + \eta_B \alpha_L]}{N_h^{**} k_1 k_2 k_3 (\lambda_{VL}^{**} + \mu_h)} > 0 \quad \text{if} \quad R_{EL} > 1$$

then, $\lambda_6 = -(\lambda_{LV}^{**} + \mu_v) < 0$, if $R_{EL} > 1$.

We then conclude that the endemic equilibrium of leptospirosis infection is shown to be locally asymptotically stable (LAS), whenever $R_{EL} > 1$.

2.10 Global stability of endemic equilibrium of model (1)

Theorem 3: The endemic equilibrium point of model (1) is globally asymptotically stable if $R_{EL} > 1$, otherwise unstable if $R_{EL} < 1$

Proof: Let verify this using Lyapunov function and substituting equations of model (1).

We assumed that:

The simplified model equation (1) is thus:

$$\begin{aligned}
\dot{S} &= \Lambda_h - \bar{B}_1 I_v S - \mu_h S \\
\dot{E}_L &= \bar{B}_1 I_v S - k_2 E_L \\
\dot{I}_L &= \alpha_L E_L - k_3 I_L \\
\dot{S}_L &= \Lambda_v - \bar{B}_2 I_L S_v - \mu_v S_v \\
\dot{I}_v &= \bar{B}_2 I_L S_v - k_5 I_v
\end{aligned} \tag{43}$$

$$\begin{aligned}
F = &(S - S^{**} - S^{**} \ln \frac{S}{S^{**}}) + (E_L - E_L^{**} - E_L^{**} \ln \frac{E_L}{E_L^{**}}) + (I_L - I_L^{**} - I_L^{**} \ln \frac{I_L}{I_L^{**}}) \\
&+ (S_v - S_v^{**} - S_v^{**} \ln \frac{s_v}{S_v^{**}}) + (I_v - I_v^{**} - I_v^{**} \ln \frac{I_v}{I_v^{**}})
\end{aligned} \tag{44}$$

Differentiating (44) gives:

$$\dot{F} = (\dot{S} - \frac{S^{**}}{S}\dot{S}) + (\dot{E}_L - \frac{E_L^{**}}{E_L}\dot{E}_L) + (\dot{I}_L - \frac{I_L^{**}}{I_L}\dot{I}_L) + (\dot{S}_v - \frac{S_v^{**}}{\dot{S}_v}\dot{S}_v) + (\dot{I}_v - \frac{I_v^{**}}{I_v}\dot{I}_v) \tag{45}$$

Substituting (43) into (44) gives:

$$\begin{aligned}
\dot{F} = &([\Lambda_h - \bar{B}_1 I_v S - \mu_h S] - \frac{S^{**}}{S}[\Lambda_h - \bar{B}_1 I_v S - \mu_h S]) + ([\bar{B}_1 I_v S - k_2 E_L] - \frac{E_L^{**}}{E_L}[\bar{B}_1 I_v S - k_2 E_L]) \\
&+ ([\alpha_L E_L - k_3 I_L] - \frac{I_L^{**}}{I_L}[\alpha_L E_L - k_3 I_L]) + ([\Lambda_v - \bar{B}_1 I_L S_v - \mu_v S_v] - \frac{S_v^{**}}{\dot{S}_v}[\Lambda_v - \bar{B}_1 I_L S_v - \mu_v S_v]) \\
&+ ([\bar{B}_1 I_L S_v - k_5 I_v] - \frac{I_v^{**}}{I_v}[\bar{B}_1 I_L S_v - k_5 I_v])
\end{aligned} \tag{46}$$

$$\begin{aligned}
\dot{F} = &\Lambda_h - \bar{B}_1 I_v S - \mu_h S - \frac{S^{**}}{S}\Lambda_h + \bar{B}_1 I_v S^{**} + \mu_h S^{**} + \bar{B}_1 I_v S - k_2 E_L - \frac{E_L^{**}}{E_L}\bar{B}_1 I_v S + k_2 E_L^{**} \\
&+ \alpha_L E_L - k_3 I_L - \frac{I_L^{**}}{I_L}\alpha_L E_L + k_3 I_L^{**} + \Lambda_v - \bar{B}_1 I_L S_v - \mu_v S_v - \frac{S_v^{**}}{S_v}\Lambda_v + \bar{B}_1 I_L S_v^{**} + \mu_v S_v^{**} \\
&+ \bar{B}_1 I_L S_v - k_5 I_v - \frac{I_v^{**}}{I_v}\bar{B}_1 I_L S_v + k_5 I_v^{**}
\end{aligned} \tag{47}$$

At steady state:

$$\left.\begin{aligned}
\Lambda_h &= \bar{B}_1 I_v^{**} S^{**} + \mu_h S^{**} \\
\Lambda_v &= \bar{B}_2 I_L^{**} S_v^{**} + \mu_v S_v^{**}
\end{aligned}\right\} \tag{48}$$

Substitute (48) into (47) results:

$$\begin{aligned}
\dot{F} = &(\bar{B}_1 I_v^{**} S^{**} + \mu_h S^{**}) - \bar{B}_1 I_v S - \mu_h S - \frac{S^{**}}{S}(\bar{B}_1 I_v^{**} S^{**} + \mu_h S^{**}) + \bar{B}_1 I_v S^{**} + \mu_h S^{**} + \bar{B}_1 I_v S - k_2 E_L - \frac{E_L^{**}}{E_L}\bar{B}_1 I_v S \\
&+ k_2 E_L^{**} + \alpha_L E_L - k_3 I_L - \frac{I_L^{**}}{I_L}\alpha_L E_L + k_3 I_L^{**} + (\bar{B}_2 I_L^{**} S_v^{**} + \mu_v S_v^{**}) - \bar{B}_1 I_L S_v - \mu_v S_v - \frac{S_v^{**}}{S_v}(\bar{B}_2 I_L^{**} S_v^{**} + \mu_v S_v^{**}) \\
&+ \bar{B}_1 I_L S_v^{**} + \mu_v S_v^{**} + \bar{B}_1 I_L S_v - k_5 I_v - \frac{I_v^{**}}{I_v}\bar{B}_1 I_L S_v + k_5 I_v^{**}
\end{aligned} \tag{49}$$

$$\dot{F} = \bar{B}_1 I_v^{**} S^{**} + \mu_h S^{**} - \mu_h S - \frac{S^{**2}}{S}\bar{B}_1 I_v^{**} - \mu_h S^{**2} + \bar{B}_1 I_v S^{**} + \mu_h S^{**} - k_2 E_L - \frac{E_L^{**}}{E_L}\bar{B}_1 I_v S$$
$$+ k_2 E_L^{**} + \alpha_L E_L - k_3 I_L - \frac{I_L^{**}}{I_L}\alpha_L E_L + k_3 I_L^{**} + \bar{B}_2 I_L^{**} S_v^{**} + \mu_v S_v^{**} - \mu_v S_v - \frac{S_v^{**2}}{S_v}\bar{B}_2 I_L^{**} - \mu_v S_v^{**2} \quad (50)$$
$$+ \bar{B}_1 I_L S_v^{**} + \mu_v S_v^{**} - k_5 I_v - \frac{I_v^{**}}{I_v}\bar{B}_1 I_L S_v + k_5 I_v^{**}$$

Substituting for $k_2, k_3 \text{ and } k_5$, simplifying and rearranging (50) gives:

$$\dot{F} = 2\bar{B}_1 I_v^{**} S^{**} - \bar{B}_1 I_v^{**}\frac{S^{**2}}{S} - \bar{B}_1 I_v^{**} S^{**}\frac{E_L}{E_L^{**}} + \bar{B}_1 I_v S^{**} - \bar{B}_1 I_v S\frac{E_L^{**}}{E_L} + 2\mu_h S^{**} - \mu_h S - \mu_h S^{**2} + \alpha_L E_L\left(1 - \frac{I_L^{**}}{I_L}\right)$$
$$+ \alpha_L E_L^{**}\left(1 - \frac{I_L}{I_L^{**}}\right) + 2\bar{B}_2 I_L^{**} S_v^{**} - \bar{B}_2 I_L^{**}\frac{S_v^{**2}}{S_v} - \frac{\bar{B}_2 I_L^{**} S_v^{**} I_v}{I_v^{**}} + \bar{B}_2 I_L S_v^{**} - \frac{\bar{B}_2 I_v^{**} S_v I_L}{I_v} + 2\mu_v S_v^{**} - \mu_v S_v - \mu_v\frac{S_v^{**2}}{S_v} \quad (51)$$

$$\dot{F} = \bar{B}_1 I_v^{**} S^{**}\left(2 - \frac{S^{**}}{S} - \frac{E_L}{E_L^{**}}\right) + \bar{B}_1 I_v S^{**}\left(1 - \frac{S}{S^{**}} - \frac{E_L^{**}}{E_L}\right) + \mu_h S^{**}\left(2 - \frac{S}{S^{**}} - \frac{S^{**}}{S}\right) + \alpha_L E_L\left(1 - \frac{I_L^{**}}{I_L}\right)$$
$$+ \alpha_L E_L^{**}\left(1 - \frac{I_L}{I_L^{**}}\right) + \bar{B}_2 I_L^{**} S_v^{**}\left(2 - \frac{S_v^{**}}{S_v} - \frac{I_v}{I_v^{**}}\right) + \bar{B}_2 I_L S_v^{**}\left(1 - \frac{I_v^{**} S_v^{**}}{I_v S_v}\right) + \mu_v S_v^{**}\left(2 - \frac{S_v}{S_v^{**}} - \frac{S_v^{**}}{S_v}\right) \quad (52)$$

We therefore conclude that it is evident that the arithmetic mean (AM) exceeds the geometric mean (GM) then, these inequalities hold.

$$2 - \frac{S^{**}}{S} - \frac{E_L}{E_L^{**}} \le 0;\ 1 - \frac{S}{S^{**}}\frac{E_L^{**}}{E_L} \le 0;\ 2 - \frac{S}{S^{**}} - \frac{S^{**}}{S} \le 0; 1 - \frac{I_L^{**}}{I_L} \le 0; 1 - \frac{I_L}{I_L^{**}} \le 0;$$
$$2 - \frac{S_v^{**}}{S_v} - \frac{I_v}{I_v^{**}} \le 0; 1 - \frac{I_v^{**} S_v}{I_v S_v^{**}} \le 0;\ 2 - \frac{S_v}{S_v^{**}} - \frac{S_v^{**}}{S_v} \le 0$$

$\dot{F} \le 0$ for all $R_{EL} > 1$ hence, F is a Lyapunov function in Ω_2 and the EEP is globally asymptotically stable, (G.A.S) (for special case $d = 0$) based on the LaSalles invariance principle (LaSalles,1969).

2.11 Bifurcation analysis of model (1) equations

We would examine the reality of backward bifurcation phenomenon of the model (1) equations when $R_{EL} < 1$. We use centre manifold theorem as presented by (Augusto, 2017; Castillo-Chavez and Song, 2004).

Theorem 4: The model (1) equations undergo backward bifurcation phenomenon whenever $R_{EL} = 1$ under special condition that ($\chi_L = 0$).

Proof: The proof is based on the Centre Manifold theorem. From model (1) equations,

Let $x_1 = V, x_2 = S, x_3 = E_L, x_4 = I_L, x_5 = R_L, x_6 = S_v, x_7 = I_v$ and $x_8 = R_v$

The following transformed equation is obtained for the model (1) equations.

$$\begin{aligned}
\dot{x}_1 &= f\Lambda_h - k_1 x_1 \\
\dot{x}_2 &= \Lambda_h(1-f) + \psi_L x_1 + \chi_L x_5 - \frac{B_{VL}(1-\theta)x_7 x_2}{N_h} - \mu_h x_2 \\
\dot{x}_3 &= \frac{B_{VL}(1-\theta)x_7 x_2}{N_h} - k_2 x_3 \\
\dot{x}_4 &= \alpha_L x_3 - k_3 x_4 \\
\dot{x}_5 &= \tau_L x_4 - k_4 x_5 \\
\dot{x}_6 &= \Lambda_v + \gamma_L x_8 - \frac{B_{LV}(\eta_A x_3 + \eta_B x_4)x_6}{N_h} - \mu_v x_6 \\
\dot{x}_7 &= \frac{B_{LV}(\eta_A x_3 + \eta_B x_4)x_6}{N_h} - k_5 x_7 \\
\dot{x}_8 &= \alpha_v x_7 - k_6 x_8
\end{aligned} \tag{53}$$

The Jacobian of the equation (53) estimated at DFE is given as:

$$J(\xi_0^*) = \begin{bmatrix}
-k_1 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\
\psi_L & -\mu_h & 0 & 0 & \chi_L & 0 & -\frac{B_{VL}(1-\theta)x_2^*}{N_h^*} & 0 \\
0 & 0 & -k_2 & 0 & 0 & 0 & \frac{B_{VL}(1-\theta)x_2^*}{N_h^*} & 0 \\
0 & 0 & \alpha_L & -k_3 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & \tau_L & -k_4 & 0 & 0 & 0 \\
0 & 0 & -\frac{B_{LV}\eta_A x_6^*}{N_h^*} & -\frac{B_{LV}\eta_B x_6^*}{N_h^*} & 0 & -\mu_v & 0 & \gamma_v \\
0 & 0 & \frac{B_{LV}\eta_A x_6^*}{N_h^*} & \frac{B_{LV}\eta_B x_6^*}{N_h^*} & 0 & 0 & -k_5 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 & \alpha_v & -k_6
\end{bmatrix} \tag{54}$$

We considered the case when $B_{VL}{}^*$ is selected as the bifurcation parameter at $R_{VL}=1$. We therefore have:

$$B_{VL}{}^* = \frac{N_h^{*2}k_2k_3k_5}{B_{LV}(1-\theta)S^*[\eta_A k_3 + \eta_B \alpha_L]S_v^*} \tag{55}$$

The right eigenvector of $J(\xi_0^*)_{B_{VL}=B_{VL}^*}$ is assumed as: $w=(w_1,w_2,w_3,w_4,w_5,w_6,w_7,w_8)$ where:

$$\begin{aligned}
&w_1=0; w_2=\frac{B_{VL}(1-\theta)x_2^*\left[\chi_L\alpha_L\tau_L - k_2k_3k_4\right]w_7}{N_h^*k_2k_3k_4\mu_h};\ w_3=\frac{B_{VL}(1-\theta)x_2^*w_7}{N_h^*k_2};\ w_4=\frac{B_{VL}\alpha_L(1-\theta)x_2^*w_7}{N_h^*k_2k_3}; \\
&w_5=\frac{B_{VL}\alpha_L\tau_L(1-\theta)x_2^*w_7}{N_h^*k_2k_3k_4};\ w_6=\frac{\left(N_h^*\gamma_v\alpha_v k_2k_3 - B_{LV}B_{VL}(1-\theta)x_2^*x_6^*\left[\eta_A k_3k_6 - \eta_B\alpha_L k_6\right]\right)w_7}{N_h^{*2}k_2k_3k_6\mu_v};\ w_8=\frac{\alpha_v w_7}{k_6};\ w_7=w_7>0.
\end{aligned} \tag{56}$$

The above eigenvectors were obtained by solving equation (57) below:

$$
\begin{aligned}
&-w_1k_1=0\\
&w_1\psi_L-w_2\mu_h+w_5\chi_L-\frac{B_{VL}(1-\theta)x_2^*w_7}{N_h^*}=0\\
&-w_3k_2+\frac{B_{VL}(1-\theta)x_2^*w_7}{N_h^*}=0\\
&w_3\alpha_L-k_3w_4=0\\
&w_4\tau_L-k_4w_5=0\\
&-\frac{B_{LV}\eta_Ax_6^*w_3}{N_h^*}-\frac{B_{LV}\eta_Bx_6^*w_4}{N_h^*}-\mu_vw_6+\gamma_vw_8=0\\
&\frac{B_{LV}\eta_Ax_6^*w_3}{N_h^*}+\frac{B_{LV}\eta_Bx_6^*w_4}{N_h^*}-k_5w_7\\
&\alpha_vw_7-k_6w_8=0
\end{aligned}
\tag{57}
$$

Likewise, the $J(\xi_0^*)_{B_{VL}=B_{VL}^*}$ has left eigenvector as: $v=(v_1,v_2,v_3,v_4,v_5,v_6,v_7,v_8)$ where;

$$
v_1=v_2=v_5=v_6=v_8=0;\ v_3=\frac{B_{LV}x_6^*\left[\alpha_L\eta_B+k_3\eta_A\right]v_7}{N_h^*k_2k_3};\ w_4=\frac{B_{LV}\eta_Bx_6^*v_7}{N_h^*k_3};\ v_7=v_7>0. \tag{58}
$$

The above eigenvectors were obtained by solving these equation (59)

$$
\begin{aligned}
&-k_1v_1+\psi_Lv_2=0\\
&-\mu_hv_2=0\\
&-k_2v_3+\alpha_Lv_4-\frac{B_{LV}\eta_Ax_6^*v_6}{N_h^*}+\frac{B_{LV}\eta_Ax_6^*v_7}{N_h^*}=0\\
&-k_3v_4+\tau_Lv_5-\frac{B_{LV}\eta_Bx_6^*v_6}{N_h^*}+\frac{B_{LV}\eta_Bx_6^*v_7}{N_h^*}=0\\
&\chi_Lv_2-k_4v_5=0\\
&\mu_vv_6=0\\
&-\frac{B_{VL}(1-\theta)x_2^*v_2}{N_h^*}+\frac{B_{VL}(1-\theta)x_2^*v_3}{N_h^*}-k_5v_7+\alpha_vv_8=0\\
&\gamma_vv_6-k_6v_8=0
\end{aligned}
\tag{59}
$$

2.12 Computation of the Bifurcation Coefficient a and b for the model equation (53)

We follow the Castillo-Chavez and Song deduction in Castillo-Chavez and Song, (2004) and Andrawus *et al*. (2017). We considered the accompanying non-zero partial derivatives of the transformed model equation (53) required to calculate the bifurcation coefficients a at (DFE) is expressed as:

$$
a=\sum_{k,i,j=1}^{n}v_kw_iw_j\frac{\partial^2f_k}{\partial x_i\partial x_j}(0,0) \tag{60}
$$

We obtained the expression for the bifurcation coefficient a as:

$$
a=2v_3w_2w_7\frac{\partial^2f_2}{\partial x_2\partial x_7}(0,0)+2v_7w_3w_6\frac{\partial^2f_7}{\partial x_3\partial x_6}(0,0)+2v_7w_4w_7\frac{\partial^2f_2}{\partial x_4\partial x_6}(0,0) \tag{61}
$$

$$
\begin{aligned}
a=&\frac{2v_7B_{LV}B_{VL}(1-\theta)x_2^*x_6^*\left(\eta_B\alpha_L+\eta_Ak_3\right)\left(\chi_L\alpha_L\tau_L-k_2k_3k_4\right)w_7^2}{k_2^2k_3^2N_h^{*2}}\\
&+\frac{2v_7B_{LV}B_{VL}(1-\theta)x_2^*\left(N_h^*\alpha_v\gamma_vk_2k_3-B_{LV}B_{VL}(1-\theta)x_2^*x_6^*[\eta_Ak_3k_6+\eta_B\alpha_Lk_6]\right)w_7^2}{k_2^2k_3k_6N_h^*\mu_v}+\frac{2v_7B_{LV}\eta_BB_{VL}\alpha_L(1-\theta)x_2^*w_7^2}{k_2k_3N_h^{*2}}
\end{aligned}
\tag{62}
$$

which leads to:

$$
a=G_1-(G_2+G_3), \tag{63}
$$

$$G_1 = \frac{2\nu_7 B_{LV} B_{VL}(1-\theta)x_2^*}{N_h^* k_2 k_3}\left[\frac{B_{VL}x_6^*(\eta_B\alpha_L+\eta_A k_3)\chi_L\alpha_L\tau_L}{\mu_h k_3 k_2}+\frac{\gamma_v N_h^* k_2 k_3\alpha_v}{\mu_v k_6 k_2}+\eta_B\alpha_L\right]$$

$$G_2 = \frac{2\nu_7 B_{LV} B_{VL}^2(1-\theta)x_2^* x_6^*(\eta_B\alpha_L+\eta_A k_3)k_4 w_7^2}{N_h^{*2}\mu_h k_2 k_3} \quad (64)$$

$$G_3 = \frac{2\nu_7 B_{LV} B_{VL}(1-\theta)x_2^* x_6^*(\eta_B\alpha_L+\eta_A k_3)w_7^2}{N_h^{*2}\mu_h k_2^2 k_3^2}$$

$$b = \sum_{k,i,=1}^{n} v_k w_i \frac{\partial^2 f_k}{\partial x_i \partial B_{LV}^*}(0,0) \quad (65)$$

$$b = v_3 w_7 \frac{\partial^2 f_3}{\partial x_7 \partial B_{VL}^*}(0,0)$$

$$= \frac{B_{LV}(1-\theta)x_6^*(\eta_B\alpha_L+\eta_A k_3)\mathrm{v}_7 w_7}{N_h^{*2}k_2 k_3} > 0 \quad (66)$$

$b > 0$

It follows from (65) that the bifurcation coefficient, a, is positive whenever,

$$G_1 > G_2 + G_3. \quad (67)$$

We therefore conclude that the model (53) equations undergo a backward bifurcation phenomenon at $R_{EL} = 1$ whenever the inequality (67) holds.

3.0 Numerical simulation

We illustrate the results of the analysis by simulating the model (1) equations using parameters in Table 2. Figure 2 demonstrates the impact of public awareness/education (θ) on the dangers of leptospirosis to susceptible class. Here, we observe that an increase on (θ) may not necessarily lead to an increase or decrease on the susceptible class, since there is no outbreak/incidence of the disease yet. Figure 3 shows the impact of public awareness/education (θ) on the dangers of leptospirosis to the exposed class. Here, we observe that an increase on (θ) may lead to a reduction on the exposed human as indicated by decrease in the number of humans from the graph. Figure 4 indicates the result of infected class showing zero level leptospirosis incidence. Here, we see that there is a scenario in human population where there may not be cases of leptospirosis infection as a result of effective adherence to public awareness/educational campaign strategy.

Figures 5, 6 and 7 indicates the impacts of vaccination on the control of leptospirosis transmission dynamics. These figures show that vaccination administered to (susceptible individuals in Figure 5), Figure 6 (exposed individuals), and Figure 7 (infected individuals), respectively. Comparing the results displayed on Figure 5, Figure 6 and Figure 7, we observe that early vaccination of susceptible has a more significant impact on reducing new leptospirosis cases compared to vaccinating individual at exposed and infectious classes. However, this does not suggest that treating infectious leptospirosis is not necessary, but the study may be stressing the importance of early vaccination against leptospirosis as a more effective intervention strategy.

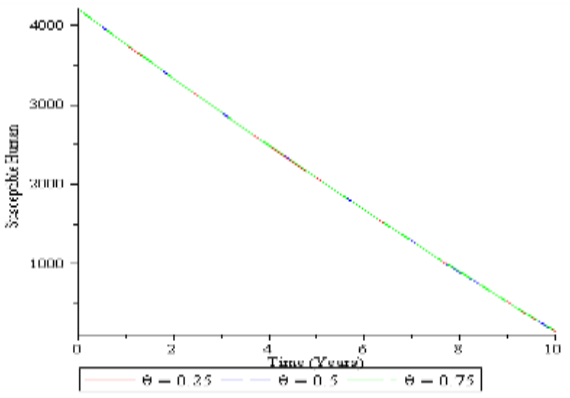

Figure 2: varying public awareness campaign on susceptible human

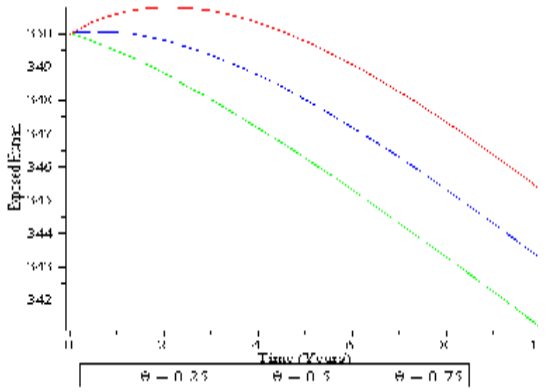

Figure 3: Varying public awareness campaign on exposed human

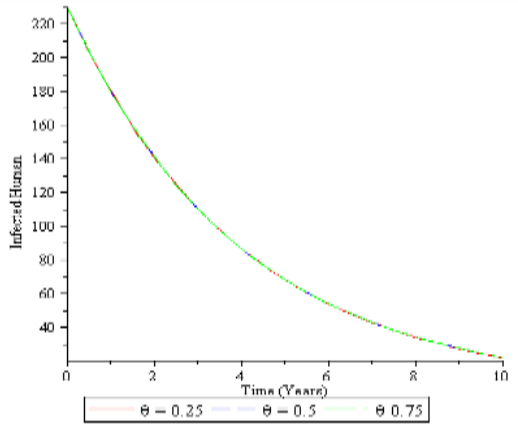

Figure 4: Varying public awareness campaign infected human

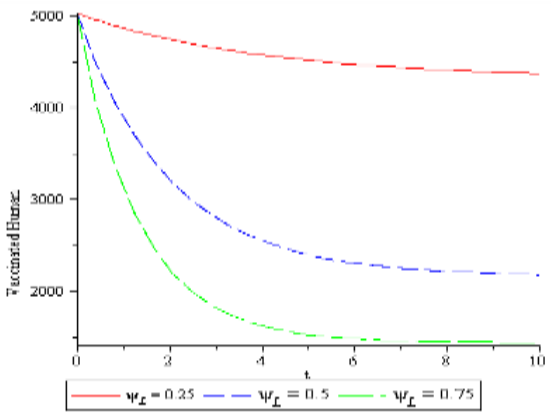

Figure 5: Varying vaccination rate on susceptible human

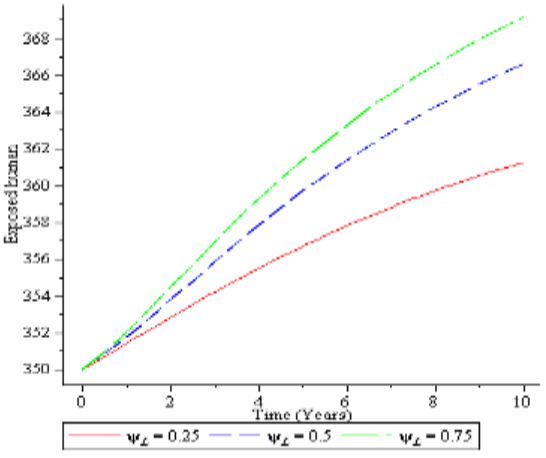

Figure 6: Varying vaccination rate on exposed human

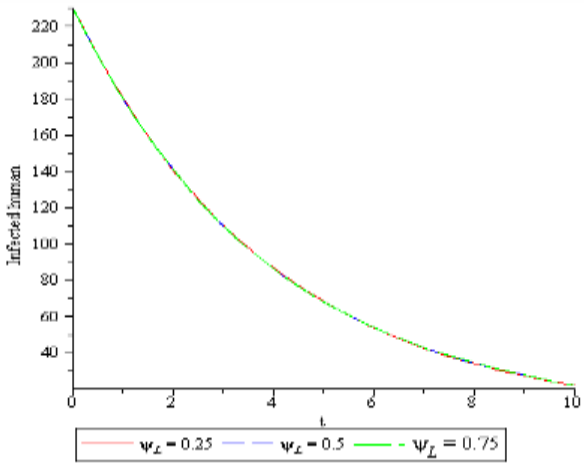

Figure 7: Varying vaccination rate on infected human

Figures 8 and 9 indicate the impact of treatment on the control of leptospirosis transmission dynamics. These figures show that right treatment may lead to increased recovery rate of infected individuals as shown in (Figure 8). Also, Figure 9 shows that treatment strategy given to infected individuals at varying treatment levels indicates reduction of the disease transmission dynamics, respectively.

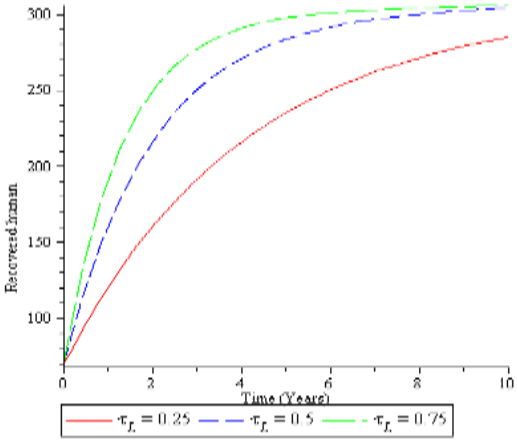

Figure 8: Varying rate of treatment on recovery in human

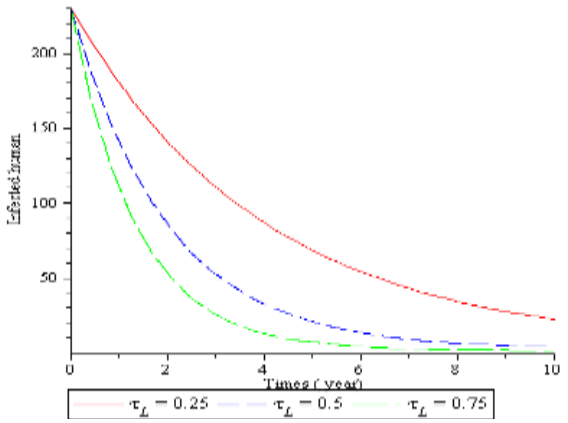

Figure 9: Varying rate of treatment on infected human

4.0 Results and discussion

Epidemiologically, leptospirosis can be controlled in human population if the initial size of the bacteria concentration and the secondary hosts (rodents and domestic animals) can be reduced nearly to zero (small enough), such that the basic reproduction number can be brought to unity, (i.e. $R_T < 1$). Also, preventative measures such as public awareness campaign on healthy handling of the secondary hosts and routine maintenance of recreational centres such as swimming pools are suggested to reduce the disease outbreak. The model (1) undergoes the phenomenon of backward bifurcation whenever a stable disease-free equilibrium point coexists with a stable endemic equilibrium point and the accompanying basic reproduction number is equal to unity (Gumel, 2012). The epidemiological consequence of the backward bifurcation phenomenon of the model (1) is that the necessary condition for the basic reproduction number to be less than one becomes only a necessity, but not sufficient condition for leptospirosis disease control (Albert *et al*. 2009). Therefore, this research discovers that there was loss of acquired temporary protection from treatment with the use of antibiotics (penicillin) in human population for leptospirosis disease since the vaccine is still under laboratory investigation. Loss of temporal immunity of recovered humans (χ_L) is the cause of backward bifurcation in the leptospirosis transmission dynamics in human population as it portrays why leptospirosis still persist in human population despite control measures being adopted.

5.0 Conclusion

In this research work, we formulate and analyzed eight (8) compartmental model for leptospirosis transmission dynamics incorporating vaccination as a control measure in human. The quantitative analysis of the models indicates that the solutions of the model is bounded and positive. This study obtained the reproduction number (R_{EL}) for leptospirosis transmission dynamics and established that the disease-free equilibrium is locally and globally asymptotically stable if $R_{EL} < 1$, and unstable when $R_{EL} > 1$. The endemic equilibrium was proved to exist, which are locally and globally asymptotically stable if $R_{EL} > 1$, and unstable when $R_{EL} < 1$. This analysis discovered that the model will undergo the phenomenon of backward bifurcation for a special case when ($\chi_L = 0$, $R_{EL} = 1$), i.e. DFE coexists with endemic equilibrium. The study has shown that when there is adequate adherence to environmental health public awareness on use of recreational centres and routine proper treatment culture cultivated by environmental health workers, then, the medium for transmitting leptospira will be totally checked. The study also indicated that effective control of leptospirosis disease in human will result when experimental vaccination is confirmed effective.

Declarations

Ethics approval and consent to participate

Not Applicable

Consent for publication

All authors have read and consented to the submission of the manuscript.

Availability of data and material
Not Applicable.
Competing interests
All authors declare no competing interests.
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