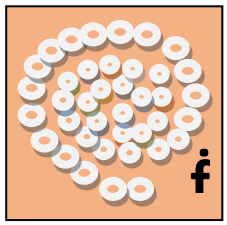

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Mathematical analysis for the dynamics of Feline immunodeficiency virus in cats population

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Abstract

In this paper, we present an extended version of the deterministic model of feline immunodeficiency virus (FIV) within cats population to carry out the stability analysis with regards to the total population of cats and disease prevalence. For the extension, we incorporate the compartment of latently infected domestic cats since it is evident that such cats transmit the virus as well. Stability analysis of the presented model is carried out. The analysis reveals that the retrovirus cannot invades the population at carrying capacity state if the threshold parameter, $\mathcal{R}_0$ is less than unity. Indeed, we obtain an additional equilibrium point known as semi-trivial state for which the disease exterminate the host population. Moreover, the carrying capacity state can exchange stability with non-trivial equilibrium at $\mathcal{R}_0 = 1$ so that the latter will be the only attractor when $\mathcal{R}_0 > 1$, a phenomenon called transcritical bifurcation.

Keywords and phrases: Feline immunodeficiency virus; FIV latency; domestic cats.

1 Introduction

The feline immunodeficiency virus (FIV) is detected worldwide and is among the most common infectious diseases of domestic cats, other small felids and wild cats. It belongs to a Lentivirus subfamily which was first isolated and described by Pedersen, 1987 in the city of Petaluma, USA [1]. The FIV distinct subtypes are designated A through E [2]. The immunodeficiency disease from FIV infection in domestic cats is similar to human acquired immune deficiency syndrome (AIDS) [3]. This is an important Lentivirus that causes immune disorders in both domestic and nondomestic cats. The high incidence of the virus is associated with density of cat population and males have high risk of infection than females since transmission mode is through bites inflicted during fights and biting is more apt to occur between male cats [4]. Therefore, the risk of FIV transmission is low in socially well-adapted cats [2, 5]. Veneral transmission from infected males to females is also possible as experimental studies shows that infection occurs not only via a vaginal route, but also via rectal mucous membrane [6]. The seroprevalence of FIV is highly variable between geographic locations ranging from 2% in Germany and 16% in the United States to 33% in the United Kindom to 44% in Japan [7].

Feline immunodeficiency virus infection can be divided into five stages similar to those of human immunodeficiency virus (HIV) infection namely: acute phase which begins about four weeks after infection and lasts for four month, asymptomatic carrier (AC), persistent generalized lymphadenopathy (PGL), AIDS-related complex (ARC) and AIDS stages [5]. Although many cats exhibit no clinical signs during acute infection but fever, leucopenia, gingivitis, lethargy, signs of enteritis, stomatitis, dermatitis, conjunctivitis, respiratory tract disease, neutropenia, and generalized lymphadenopathy may be observed. There are no clinical signs in AC stage which lasts several months to years but there is a progressive decline in CD4+ T lymphocyte numbers. The PGL phase is associated with unspecific clinical signs including apathy, weight loss, lymphadenopathy, fever, anorexia, depression, chronic

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gingivostomatitis (difficulty eating), chronic rhinitis, lymphadenopathy, immune-mediated glomerulo nephritis, leucopenia and behavioral abnormalities. Even though the ARC and AIDS phases of disease are not clearly defined, cats with ARC usually experience chronic respiratory, gastrointestinal, and skin disorders, accompanied by PGL. While some signs that signal a progression to AIDS include more opportunistic infections, severe emaciation, neoplasia, lymphoid depletion and seizures, and neurological abnormalities [2, 5, 8].

A deterministic mathematical model of susceptible-infectious (SI) type is proposed in [9] to study the circulation of FIV within populations of domestic cats. The model analysis reveals that FIV is endemic in domestic feline populations. In 1997, a deterministic model for the transmission dynamics of two retrovirus, namely: a Feline Immunodeficiency Virus (FIV) and Feline Leukaemia Virus (FeLV) simultaneously infecting a single host (domestic cat Felis catus) was presented in [10]. Stability analysis and simulations of the model reveal that FIV persists and is maintained in the population when introduced, while FeLV is either persists or goes extinct. Further, the combined impact of the diseases is observed to be higher than the sum of the impact of the two individual diseases. This is perhaps due to a higher mortality rate when both viruses infect a single individual. A generic SEIRS model with application to cat retroviruses and fox rabies is developed in [11]. An explicit criterion for the stability of the largest disease-free stationary state is provided. The result is applied to the Feline Immunodeficiency Virus and to the Feline Leukemia Virus, two retroviruses of domestic cats (Felis catus), and to the rabies virus for red foxes (Vulpes vulpes).

In the last decade, a stochastic dynamics of FIV in cats populations was studied in [12] to get insight on the effects of environmental driving forces on its dynamics. The analysis reveals that FIV outbreak can be suppressed by large environmental fluctuations and a threshold parameter R_s^h governs the virus distributions. Moreover, susceptible-free dynamics is possible by white noise perturbations of the birth rate for infected cats. Recently, a discrete-time zoonotic infectious disease model with strong Allee effect was proposed in [13]. They shown that the presence of the strong Allee effect in the disease-free equation rendered the model bistable. The authors examined the structures of the coexisting attractors using the threshold parameter $\mathcal{R}_0$ and the spectral radius, λ_k. Numerical simulations reveals that the disease can enlarge and shrink the basic of attraction of the catastrophic extinction state as well as breaking it into several disjoint sets.

In this study, we extend the susceptible-infectious (SI) model proposed in [9] by incorporating the compartment of latently infected (as FIV supports latent infection both *in vitro* and *in vivo*, [14]) cats to carry out the stability analysis with regards to the total population of cats and disease prevalence. In fact, after a prolonged latent period that last for years, the progressive loss of T-lymphocytes results in an immunodeficiency syndrome characterised by chronic and recurrence infections. The analysis reveals that in addition to the three equilibrium points reported in [9], an additional equilibrium point exists for which the disease exterminate the host population known as semi-trivial equilibrium [15, 16]. We show that disease persistence depends on two threshold parameters $\mathcal{R}_0$ and $\mathcal{R}_1$. In fact, the uniqueness of the non-trivial equilibrium if it exists leads to occurrence of a transcritical bifurcation at $\mathcal{R} = 1$. This phenomenon results in the exchange of stability between the carrying capacity state and the nontrivial equilibrium at $\mathcal{R}_0 = 1$. That is, if $\mathcal{R}_0 < 1$, the only local asymptotic stable equilibrium is the carrying capacity state, whereas the non-trivial equilibrium will be the only attractor for $\mathcal{R}_0 > 1$ and $\mathcal{R}_1 > 1$.

We organised the remaining part of this paper as follows: In Section 2, we describe the proposed model. Stability analysis of the model equilibria is carried out in Section 3. Then we discuss on the model results and give a concluding remarks in the last section.

2 Model formulation

We denote by $N(t)$, the total number of cats at time t. Let b be the natural birth rate and m be the natural death rate in the absence of infection. Thus, the intrinsic growth-rate of the cat population in the absence of resource limitation and epidemic is $r = b - m$. We assume that resource limitation acts through density-dependence on the death rate, while the birth rate remains constant [9]. Therefore,

we split the Logistic growth rate, $G(N) = r(1 - \frac{N}{K})$ as

$$G(N) = B(N) - D(N),$$

with $B(N) = b$ is the birth rate which is constant and $D(N) = m + r\frac{N}{K}$ is the death rate which is linearly related to N, where m is the natural death rate, K is the carrying capacity of the habitat, $r = b - m \geq 0$ is the population growth rate in absence of resource limits which adjusts the maximum per capita growth rate. That is resource limitation is assumed to act through density-dependence on the death rate.

When the population is free from FIV infection, the dynamics of the total population is governed by the logistic model.

$$\frac{dN}{dt} = rN\left(1 - \frac{N}{K}\right). \tag{1}$$

To describe the dynamics of the FIV through cats population, we divide the population into three classes of susceptibles denoted by $S(t)$, latent denoted by $L(t)$ and infectious denoted by $I(t)$, so that $S(t) + L(t) + I(t) = N(t)$. Although there are several stages of illness, for the sake of simplicity we assume that there is only one stage of illness combining acute, seropositivity, persistent generalized lymphadenopathy (PGL), Aids-related complex (ARC) and AIDS stages. Furthermore, we assume that the latently infected cats transmit the disease in a reduced rate (i.e., there is a modification parameter $0 < \epsilon < 1$, accounting for the reduction of infectiousness of latently infected individuals in comparison to the infectious ones) since there is a clinical evidence of FIV latent infection [17]. As in [9], we also assume that:

(i) FIV transmission is via proportionate mixing and a cat's infection status does not affect the probability of having direct contacts with other cats,

(ii) The frequency of an aggressive contacts resulting in a bite when an encounter occurs is β, and c is the efficiency of the FIV transmission by biting,

(iii) A cat comes into contact with others in the population at a constant rate ρ,

(iv) Of all the contacts had by a single susceptible cat, a proportion $\frac{\epsilon L+I}{N}$ is with infected cats. Therefore the rate at which contacts between susceptibles and infected cats occur is $\rho\frac{S(\epsilon L+I)}{N}$ so that the force of infection is $c\rho\beta\frac{S(\epsilon L+I)}{N}$, i.e., $\sigma\frac{S(\epsilon L+I)}{N}$,

(v) There is no vertical transmission and there is no recovery of infectious cats by any means.

The respective rates of transfer between the three compartments are presented in Figure 1. Then combining the above assumptions the model equations describing the dynamics of cats population infected with FIV are given by the following system of ordinary differential equations.

$$\begin{cases} \frac{dS}{dt} = b(S + L + I) - \left(m + r\frac{N}{K}\right)S - \frac{\sigma(\epsilon L + I)}{N}S, \\ \frac{dL}{dt} = \frac{\sigma(\epsilon L + I)}{N}S - \left(m + r\frac{N}{K}\right)L - \gamma L, \\ \frac{dI}{dt} = \gamma L - \left(m + r\frac{N}{K}\right)I - \alpha I \\ \frac{dN}{dt} = rN\left(1 - \frac{N}{K}\right) - \alpha I, \end{cases} \tag{2}$$

The parameter σ is the rate of effective bites for FIV transmission. The disease related mortality rate is α and so, $\frac{1}{\alpha}$ is the rate of infectious period.

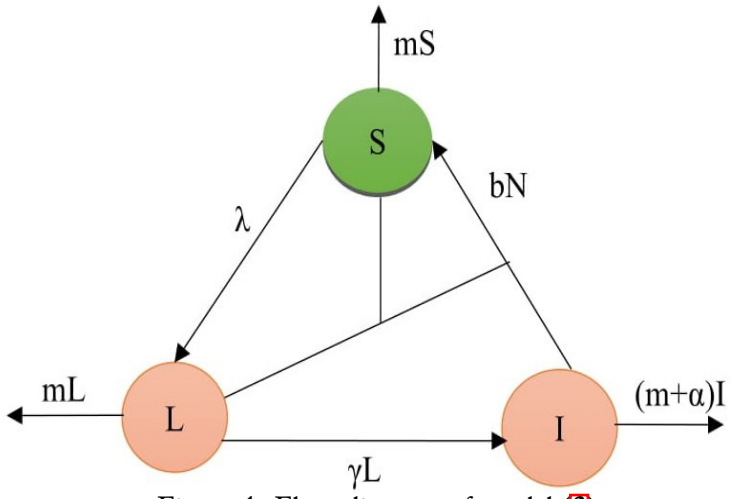

Figure 1: Flow diagram of model (2)

Introducing the prevalence $i(t)$, we have

$$s = \frac{S}{N}, e = \frac{L}{N}, i = \frac{I}{N}, \text{ and } p = \frac{N}{K}, \tag{3}$$

so that model (2) is rescaled to

$$\begin{cases} \dfrac{dp}{dt} = [r(1-p) - \alpha i]p, \\ \dfrac{de}{dt} = \sigma\epsilon e + \sigma i(1-i) - [b + \gamma + (\sigma - \alpha)i + \sigma\epsilon(e+i)]e, \\ \dfrac{di}{dt} = \gamma e - [b + \alpha(1-i)]i, \end{cases} \tag{4}$$

with $s = 1 - e - i$.

Theorem 2.1. *The system of equations (4) defines a dynamical system in the biologically feasible region*

$$\mathcal{D} = \{(p, e, i) \in \mathbb{R}^3_+ : 0 \leq e, i, e + i \leq p \leq 1\}.$$

In the following analysis, we assume that $\sigma > \alpha$ as in [9]. This is because when the effective contact rate σ is less than or equal to the disease-induced death rate α, the influx of infectious compartment is lower than the out flux as the number of latently infected cats decreases, resulting in the reduction of the number of infectious cats. This scenario can be seen from the second and third equations of (2).

All the model parameters are assumed to be non-negative and are described in Table 2.

Table 1: Parameter values

Parameter	Description	Nominal value	Reference
b	Natural birth rate	2.4	[9]
m	Natural death rate	1.8	[9]
α	disease-induced death rate	0.2	[9]
σ	Rate of effective bite	3	[9]
ϵ	Modification parameter	(0, 1]	Assumed
γ	Progression rate	0.3	Assumed

3 Analysis of the model

3.1 Trivial equilibria

In the absence of infection, model (4) exhibits the following equilibrium points.

(i) The trivial equilibrium: $E_0 = (p_0, e_0, i_0) = (0, 0, 0)$,

(ii) The carrying capacity state: $E_1 = (p_1, e_1, i_1) = (1, 0, 0)$,

It is well known that a disease can be controlled by either one of the two ways for disease transmission models with varying population size due to demographic effects [18, 19]. The first one requires that the proportion of infected individuals $i(t)$ goes to zero, while the second requirement is that the absolute number of infectives $I(t)$ approaches to zero. Therefore, the conditions for the linear stability of disease free equilibria and for the endemic proportion equilibria are required. The pertinent threshold parameter is as follows

$$\begin{aligned} \mathcal{R}_0 &= \frac{\sigma\gamma}{(b+\alpha)(b+\gamma)} + \frac{\sigma\epsilon}{(b+\gamma)} \\ &= \frac{\sigma[\gamma + \epsilon(b+\alpha)]}{(b+\alpha)(b+\gamma)}. \end{aligned} \tag{5}$$

Theorem 3.1. *The equilibrium point of model (4)*

(i) *E_0 is unstable if $\mathcal{R}_0 < 1$,*

(ii) *E_1 is locally asymptotically stable if $\mathcal{R}_0 < 1$ and is unstable when $\mathcal{R}_0 > 1$.*

Proof. By linearization, we obtain the Jacobian matrix of system (4) as

$$J(p, e, i) = \begin{pmatrix} r(1-2p) - \alpha i & 0 & -\alpha p \\ 0 & Q & \sigma(1-2i) - [\sigma\epsilon + (\sigma - \alpha)]e \\ 0 & \gamma & -[b + \alpha(1-2i)] \end{pmatrix}, \tag{6}$$

$Q = \sigma\epsilon(1 - 2e - i) - (b + \gamma) - (\sigma - \alpha)i$. Thus, the Jacobian matrix evaluated at trivial equilibrium, E_0, is given by

$$J(E_0) = \begin{pmatrix} r & 0 & 0 \\ 0 & \sigma\epsilon - (b+\gamma) & \sigma \\ 0 & \gamma & -(b+\alpha) \end{pmatrix}.$$

Then, the eigenvalues of $J(E_0)$ are: r and the eigenvalues of the matrix $J_0(E_0)$ which is obtained by deleting the first row and the first column of $J(E_0)$. It can be verified that the trace and determinant of $J_0(E_0)$ are respectively,

$$tr[J_0(E_0)] = -\left[(b+\gamma)\left(1 - \frac{\sigma\epsilon}{b+\gamma}\right) + b + \alpha\right] < 0$$

and

$$\det[J(E_0)] = (b+\gamma)(b+\alpha)(1 - \mathcal{R}_0) > 0$$

if $\mathcal{R}_0 < 1$. Then the eigenvalues of the matrix $J_0(E_0)$ have negative real parts. Hence, E_0 is unstable whenever $\mathcal{R}_0 < 1$.

Similarly, the eigenvalues of $J(E_1)$ are: $-r$ and the eigenvalues of the matrix $J_1(E_1)$, which is equivalent to $J_0(E_0)$. Therefore, all the eigenvalues of $J(E_1)$ have negative real parts. Hence E_1 is locally asymptotically stable. ■

3.2 Semi-trivial and non-trivial equilibrium points

Setting the right hand sides of (4) to zero, we have from the first and third equations of (4) that

$$p = 0 \text{ or } i = \frac{r(1-p)}{\alpha} \text{ and } e = \frac{b+\alpha(1-i)}{\gamma} i, \tag{7}$$

respectively. Moreover, if $p \neq 0$ then $p = \frac{r-\alpha i}{r}$. Thus, i satisfies $f(i) = 0$, with

$$f(i) = a_3 i^3 + a_2 i^2 + a_1 i + a_0, \tag{8}$$

where

$$\begin{aligned}
a_3 &= \alpha^2 \sigma \epsilon, \\
a_2 &= -\alpha\{\sigma\epsilon[\gamma + 2(b+\alpha) + \gamma(\sigma - \alpha)], \\
a_1 &= \sigma\{\epsilon(b+\alpha)^2 + \gamma[b(1+\epsilon) + \alpha + \gamma]\} + 2\alpha\gamma(b+\gamma)\left(\frac{\sigma\epsilon}{b+\gamma} - 1\right), \\
a_0 &= \gamma(b+\alpha)(b+\gamma)(1-\mathcal{R}_0).
\end{aligned}$$

It can be seen that the coefficient a_3 is always positive and a_2 is always negative. While the sign of the coefficients a_1 and a_0 depend on the value of $\mathcal{R}_0$. Then by Descartes' rule of sign, we have the following results.

(i) If $\mathcal{R}_0 > 1$ such that $\frac{\sigma\epsilon}{b+\gamma} > 1$, then $a_1 > 0, a_0 < 0$ and so, there are three positive roots or one positive root of (8);

(ii) If $\mathcal{R}_0 > 1$ such that $\frac{\sigma\epsilon}{b+\gamma} < 1, a_1$ can be positive or negative and $a_0 < 0$. Thus, if $a_1 > 0$ two positive roots exist while there are three positive roots or one positive root when $a_1 < 0$;

(iii) If $\mathcal{R}_0 < 1, a_1$ can be positive or negative and $a_0 > 0$. Then (8) has two or no positive roots.

It follows that, the possible number of non-trivial equilibria of system (4) could be one or two or three.

Let $E^s = (p^s, e^s, i^s)$ be an arbitrary semi-trivial equilibrium point, then from (7) and (8), we have

$$p^s = 0, e^s = \frac{b+\alpha(1-i^s)}{\gamma} i^s$$

and i^s solves $f(i) = 0$ as defined in equation (8). In a similar note, let $E^* = (p^*, e^*, i^*)$ with $p^* = \frac{r-\alpha i^*}{r}, e^* = \frac{b+\alpha(1-i^*)}{\gamma} i^*$ and i^* solves $f(i) = 0$ as defined in equation (8) be an endemic equilibrium point of (4). Then, we have the following stability results.

Theorem 3.2. *A unique semi-trivial equilibrium, E^s if it exist, is locally asymptotically stable when $\mathcal{R}_1 < 1$ and $\mathcal{R}_0 > 1$ such that $\frac{\sigma\epsilon}{b+\alpha} < 1$. It is unstable when $\mathcal{R}_1 > 1$.*

Proof. The Jacobian matrix of the linearized system of (4) evaluated at E^s is obtained from (9) as

$$J(E^s) = \begin{pmatrix} r - \alpha i^s & 0 & 0 \\ 0 & Q & \sigma(1-2i^s) - [\sigma\epsilon + (\sigma - \alpha)]e^s \\ 0 & \gamma & -[b + \alpha(1-2i^s)] \end{pmatrix}, \tag{9}$$

$Q = \sigma\epsilon(1 - 2e^s - i^s) - (b+\gamma) - (\sigma - \alpha)i^s$. Then the eigenvalues of $J(E^s)$ are:

$$r - \alpha i^s = b - (m + \alpha i^s) = (m + \alpha i^s)(\mathcal{R}_1 - 1) < 0,$$

if $\mathcal{R}_1 = \frac{b}{m+\alpha i^s} < 1$ and the eigenvalues of the matrix $J_0(E^s)$ which is obtained by deleting first row and the first column of $J(E^s)$. Then the trace and determinant of $J_0(E^s)$ are respectively, given by

$$\begin{aligned} tr[J_0(E^s)] &= \sigma\epsilon - (b+\gamma) - \sigma\epsilon(2e^s + i^s) - (\sigma-\alpha)i^s - b - \alpha(1-i^s) + \alpha i^s \\ &= -\left\{(b+\gamma)\left(1 - \frac{\sigma\epsilon}{b+\gamma}\right) + \sigma\epsilon(2e^s + i^s) + (\sigma-\alpha)i^s + b - \alpha i^s + \alpha(1-i^s)\right\} \\ &< 0 \end{aligned}$$

and

$$\begin{aligned} \det[J_0(E^s)] &= \left[(b+\gamma)\left(1 - \frac{\sigma\epsilon}{b+\alpha}\right) + \sigma\epsilon(2e^s + i^s) + (\sigma-\alpha)i^s\right][b - \alpha i^s + \alpha(1-i^s)] \\ &\quad + \gamma[\sigma\epsilon + (\sigma-\alpha)e^s + \sigma(2i^s - 1)] \\ &> 0, \end{aligned}$$

if $\mathcal{R}_1 < 1$ so that $m > 0 > b - \alpha i^s$ and $\mathcal{R}_0 > 1$ such that $\frac{\sigma\epsilon}{b+\alpha} < 1$. Therefore, the eigenvalues of $J_0(E^s)$ have negative real parts. Hence $J(E^s)$ have eigenvalues with negative real parts and so, E^s is locally asymptotically stable. ■

Theorem 3.3. *The non-trivial equilibrium E^* of system (4) if it exists is stable locally asymptotically in the interior of the domain $\mathcal{D}$, when $\mathcal{R}_0 > 1$ such that $\frac{\sigma\epsilon}{b+\gamma} > 1$ and $\mathcal{R}_1 > 1$.*

Proof. Linearizing model (4) around E^*, we obtain the Jacobian matrix

$$J(E^*) = \begin{pmatrix} r(1-2p^*) - \alpha i^* & 0 & -\alpha p^* \\ 0 & j_{22} & \sigma(1-i^*) - \sigma i^* - [\sigma\epsilon + (\sigma-\alpha)] \\ 0 & \gamma & \alpha i^* - b - \alpha(1-i^*) \end{pmatrix}$$

where $j_{22} = \sigma\epsilon - b - \gamma - (\sigma-\alpha)i^* - \sigma\epsilon(e^* + i^*) - \sigma\epsilon e^*$. One can see by inspection that the first eigenvalue of this matrix is

$$\begin{aligned} -rp^* + r(1-p^*) - \alpha i^* &= -(b-m)(2p^* - 1) - \alpha i \\ &= -[2p^* r - (m + \alpha i^*)(1 - \mathcal{R}_1)] < 0, \end{aligned}$$

for $\mathcal{R}_1 > 1$. For the remaining two eigenvalues, it suffices to show that the trace and determinant of the 2×2 sub-matrix $\tilde{J}(E^*)$ of $J(E^*)$ which is obtained by deleting the first row and the first column of $J(E^*)$ are negative and positive respectively.

$$\begin{aligned} \text{tr}(\tilde{J}) &= \sigma\epsilon - b - \gamma - (\sigma-\alpha)i^* - \sigma\epsilon(e^* + i^*) - \sigma\epsilon e^* + \alpha i^* - b - \alpha(1-i^*) \\ &= (b+\gamma)\left(\frac{\sigma\epsilon}{b+\gamma} - 1\right) - (\sigma-\alpha)i^* - \sigma\epsilon(2e^* + i^*) - m \\ &\quad + (m + \alpha i)(1 - \mathcal{R}_1) - \alpha(1-i) \\ &= -\left[(\sigma-\alpha)i^* + \sigma\epsilon(2e^* + i^*) + (m + \alpha i)(\mathcal{R}_1 - 1) + \alpha(1-i)\right] \\ &\quad - m + (b+\gamma)\left(\frac{\sigma\epsilon}{b+\gamma} - 1\right) \end{aligned}$$

$$\begin{aligned} \det(\tilde{J}) &= \left[(\sigma-\alpha)i^* + \sigma\epsilon(2e^* + i^*) + (b+\gamma)\left(1 - \frac{\sigma\epsilon}{b+\gamma}\right)\right] \\ &\quad \times \left[\alpha i^*\left(\frac{b}{\alpha i^*} - 1\right) + \alpha(1-i^*)\right] \\ &\quad + \gamma\{\sigma i^* + [\sigma\epsilon + (\sigma-\alpha)]e^* - \sigma(1-i^*)\} \end{aligned}$$

If $\mathcal{R}_0 > 1$ and $\mathcal{R}_1 > 1$ then these trace and determinant are negative and positive, respectively. Thus the eigenvalues of $\tilde{J}$ have negative real parts. Hence, the eigenvalues of $J(E^*)$ have negative real parts and so, the equilibrium point E^* locally stable asymptotically whenever $\mathcal{R}_0 > 1$ and $\mathcal{R}_1 > 1$. ■

3.3 Bifurcation analysis

The uniqueness of the non-trivial equilibrium point E^* if it exist, indicates that there is an exchange of stability between E^* and the carrying capacity state E_1 when $\mathcal{R}_0 = 1$ or equivalently $\sigma = \sigma^* = \frac{(b+\alpha)(b+\gamma)}{\epsilon(b+\gamma)+\gamma}$ (a phenomenon known as transcritical/forward bifurcation). That is, there is an exchange of stability between E_1 and E^* when $\sigma > \sigma^*$. Suppose such an equilibrium exists, then we establish the following result.

Theorem 3.4. *The system of equations (4) exhibits a transcritical bifurcation at E_1 if the bifurcation parameter $\sigma = \sigma^* = \frac{(b+\alpha)(b+\gamma)}{\epsilon(b+\gamma)+\gamma}$.*

Proof. Let f be the vector field defined by the right hand side of system (4). Then

$$f(p,e,i;\sigma) = \begin{pmatrix} [r(1-p)-\alpha i]p \\ \sigma\epsilon e + \sigma i(1-i) - [b+\gamma+(\sigma-\alpha)i + \sigma\epsilon(e+i)]e \\ \gamma e - [b+\alpha(1-i)]i \end{pmatrix}. \tag{10}$$

The Jacobian matrix evaluated at E_1, denoted by J_1 is

$$J_1 = \begin{pmatrix} -r & 0 & 0 \\ 0 & \sigma\epsilon - (b+\gamma) & \sigma \\ 0 & \gamma & -(b+\alpha) \end{pmatrix}.$$

The determinant of the matrix J_1 is

$$\det J_1 = r(b+\gamma)(b+\alpha)(\mathcal{R}_0 - 1),$$

which is zero if $\mathcal{R}_0 = 1$ or equivalently $\sigma = \sigma^* = \frac{(b+\alpha)(b+\gamma)}{\epsilon(b+\gamma)+\gamma}$. The trace of J_1 is

$$\mathrm{tr}J_1 = -\left[m + \alpha + (b+\gamma)\left(-\frac{\sigma\epsilon}{b+\gamma}\right)\right] \neq 0.$$

This guarantees that the Jacobian J_1 has a simple zero eigenvalue. Therefore the existence of transcritical bifurcation at E_1 can be established using Sotomayor theorem [20]. To verify the conditions for transcritical bifurcation given in this theorem, let $U = (u_1, u_2, u_3)^T$ be an eigenvector of the matrix J_1 corresponding to the eigenvalue $\lambda = 0$. Then using the standard computation of an eigenvector, we obtain

$$U = \begin{pmatrix} -\frac{\alpha}{r} & \frac{b+\alpha}{\gamma} & 1 \end{pmatrix}^T. \tag{11}$$

Next, let $V = (v_1, v_2, v_3)^T$ be an eigenvector of the transpose of the Jacobian matrix J_1. The eigenvector V corresponding to the eigenvalue $\lambda = 0$ is then given by

$$V = \begin{pmatrix} 0 & 1 & \frac{\sigma}{b+\alpha} \end{pmatrix}^T.$$

The derivative of the vector field $f(p,e,i;\sigma)$ in (10) with respect to the bifurcation parameter σ at the equilibrium point E_1 is

$$f_\sigma(E_1;\sigma) = \begin{pmatrix} 0 & 0 & 0 \end{pmatrix}^T$$

and so,

$$U^T f_\sigma(E_1;\sigma) = \begin{pmatrix} -\frac{\alpha}{r} & \frac{b+\alpha}{\gamma} & 1 \end{pmatrix} \begin{pmatrix} 0 \\ 0 \\ 0 \end{pmatrix} = 0. \tag{12}$$

The expression $Df_\sigma(p,e,i;\sigma)U$ as defined in [20] is

$$Df_\sigma(p,e,i;\sigma)U = \frac{\partial f_\sigma}{\partial p}u_1 + \frac{\partial f_\sigma}{\partial e}u_2 + \frac{\partial f_\sigma}{\partial i}u_3.$$

Evaluating this expression at E_1 and using (11) gives

$$Df_\sigma\left(E_1;\sigma\right)U = \begin{pmatrix} 0 & \epsilon\frac{b+\alpha}{\gamma} & 0 \end{pmatrix}^T.$$

Thus,

$$\begin{aligned} V^T\left[Df_\sigma\left(E_1;\sigma\right)\right] &= \begin{pmatrix} 0 & 1 & \frac{\sigma}{b+\alpha} \end{pmatrix}\begin{pmatrix} 0 \\ \epsilon\frac{b+\alpha}{\gamma} \\ 0 \end{pmatrix} \\ &= \epsilon\frac{b+\alpha}{\gamma} \neq 0. \end{aligned} \tag{13}$$

Also $D^2f(x;\sigma)(U,U)$ is defined in [20] as follows:

$$D^2f(x;\sigma)(U,U) = \sum_{k,i,j=1}^{3}\frac{\partial^2 f_k}{\partial x_i \partial x_j}u_i u_j.$$

Expanding and evaluating this using (11) at the point E_1, we obtain

$$D^2f(E_1;\sigma)(U,U) = \begin{pmatrix} 0 \\ -\frac{1}{\gamma^2}[2\sigma\alpha^2 + (\sigma-\alpha)\gamma + 2\sigma\epsilon(b+\alpha)(b+\alpha+\gamma)] \\ 0 \end{pmatrix}.$$

Hence,

$$\begin{aligned} V^T[D^2f(E_1;\sigma)(U,U)] &= \begin{pmatrix} 0 & 1 & \frac{\sigma}{b+\alpha} \end{pmatrix}\begin{pmatrix} 0 \\ -\frac{1}{\gamma^2}[2\sigma\alpha^2 + (\sigma-\alpha)\gamma + \varpi] \\ 0 \end{pmatrix} \\ &= -\frac{1}{\gamma^2}[2\sigma\alpha^2 + (\sigma-\alpha)\gamma + Q] \neq 0, \end{aligned} \tag{14}$$

with $\varpi = 2\sigma\epsilon(b+\alpha)(b+\alpha+\gamma)$.

It follows from [20, Theorem 1] together with conditions (12), (13) and (14) that system (4) exhibits a transcritical bifurcation at E_1 (see Figure 2). ■

This result indicates that FIV cannot invade cats population at the carrying capacity state when $\mathcal{R}_0 < 1$. While, there will be an endemic persistence of the virus for $\mathcal{R}_0 > 1$ at the non-trivial equilibrium E^* if $\mathcal{R}_1 > 1$. Moreover, Theorem 3 reveals that if E^* does not exists then the virus will exterminate the host population and persists at semi-trivial equilibrium E^s when $\mathcal{R}_1 < 1$.

4 Discussions and conclusion

In this paper, we extend the model of feline immunodeficiency virus of susceptible-infectious (SI) proposed in [9], by adding the compartment of latently infected cats. The stability analysis is carried out and it reveals that stability results of the disease persistence equilibria does not depend solely on the basic reproduction number,$\mathcal{R}_0$. In fact, we show that the disease could exterminate the host population when the virus persists in the population for ($\mathcal{R}_0 > 1$) provided the other threshold parameter, $\mathcal{R}_1 < 1$. More precisely, if $\mathcal{R}_0 < 1$ the only stable equilibrium point is the carrying capacity state E_1, while either the semi-trivial equilibrium E^s or the non-trivial equilibrium E^* is asymptotically

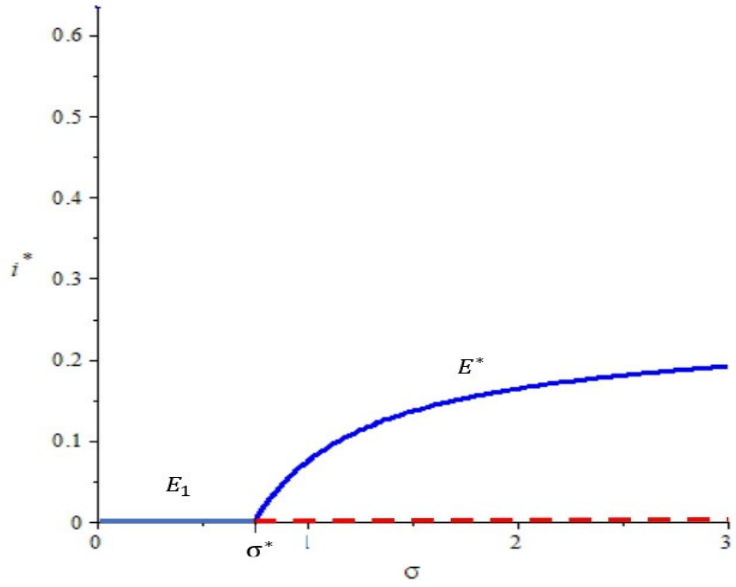

Figure 2: Transcritical bifurcation diagram showing the exchange of stability between the carrying capacity state, E_1 and the endemic equilibrium E^* at $\mathcal{R}_0 = 1$. E_1 is stable when $\mathcal{R}_0 < 1$ (Solid line) and becomes unstable if $\mathcal{R}_0 > 1$ (Dash line) so that E^* is stable. Parameter values used are as given in Table 1

stable if $\mathcal{R}_0 > 1$ depending on whether $\mathcal{R}_1 < 1$ or $\mathcal{R}_1 > 1$, respectively. It is to be noted that the semi-trivial extinction state (E^s) coincides with trivial equilibrium, E_0 when expressed in the (N, E, I) or (S, E, I) state variables. The non-trivial equilibrium, E^* if it exists, leads to an exchange of stability between it and the carrying capacity state (E_1) at $\mathcal{R}_0 = 1$ or equivalently $\sigma = \sigma^* = \frac{(b+\alpha)(b+\gamma)}{\epsilon(b+\gamma)+\gamma}$. Such a phenomenon is known as transcritical bifurcation (or forward bifurcation). That is, the equilibrium point E_1 which is stable when $\mathcal{R}_0 < 1$ ($\sigma < \sigma^*$) losses stability when $\mathcal{R}_0 > 1$ ($\sigma > \sigma^*$), leading to the emergence of a locally stable endemic equilibrium E^* (Figure 2). The occurrence of this phenomenon indicates that FIV cannot invades cats population at the carrying capacity state when $\mathcal{R}_0 < 1$, rather the disease persist if $\mathcal{R}_0 < 1$.

Complex dynamics of the presented model can be investigated when multiple non-trivial equilibrium points exist for further research. As the existence of such equilibria in disease transmission models leads to complex dynamics of a system [16, 21, 22].

The following provides a sample reference list with entries for journal articles [1], conference proceeding [2], a URL [3], as well as a book [4]. [Note that the reference list should be arranged in the order in which the articles are cited in the work, and not in alphabetical order]

References

[1] Pedersen N. C., Ho E. W., Brown M. L. and Yamamoto J. K. (1987), Isolation of a T-lymphotropic virus from domestic cats with an immunodeficiency-like syndrome. Science 235 , 790-793.

[2] Alves F. and Pimenta dos Reis J. K., Feline Immunodeficiency, http://dx.doi.org/10.5772/51631

[3] Elder J. H., Lin Y. C., Fink E., Grant C. K. (2010), Feline immunodeficiency virus (FIV) as a model for study of lentivirus infections: parallels with HIV. Curr HIV Res. 8 (2010), 73-80.

[4] Yamamoto J. K., Hansen H., Ho E. W. *et al.* (1989), Epidemiologic and clinical aspects of feline immunodeficiency virus infection in cats from the continental United States and Canada and

possible mode of transmission, Journal of American Veterinary Medecine Association, 194, 213-220.

[5] Margaret C. (1994), Feline retrovirus infections, Condensed from "Feline Viral Diseases," Textbook of Veterinary Internal Medicine, 4th Edition

[6] Moench T. R., Whaley K. J., Mandrell T. D., Bishop B. D. and Witt C. J. (1993), The cat/feline immunodeficiency virus model for transmucosal transmission of AIDS: nonoxynol-9 contraceptive jelly blocks transmission by an infected cell inoculum, AIDS 7, 797-802.

[7] Hartmann K. (1998), Feline immunodeficiency virus infection: An overview, Veterinary Journal, 155, 123-137.

[8] Ishida T., and Tomoda I. (1990), Clinical staging of feline immunodeficiency virus infection. Japanees Journal of Vetenary Sciences, 52, 645-648.

[9] Courchamp F., Pontier D., Langglais M. and Artois M. (1995), Population dynamics of Feline immunodeficiency virus within cat populations, Journal of Theoretical Biology, 175, 553-560.

[10] Courchamp F., Suppo Ch., Fromont E. and Bouloux C. (1997), Population dynamics for two feline retroviruses (FIV and FeLV) within one population of cats, Proc. R. Sot. Land. B 264 , 785-794.

[11] Langlais M. and Suppo CH. (2000), A remark on a generic SEIRS Model and application to cat retroviruses and fox rabies, Mathematics and Computer Modeling, 31, 117-124

[12] Jingli L., Shan M., Barnerjee M. and Wang W. (2016), Stochastic dynamics of feline immunodeficiency virus within cat populations, Preprint submitted to Journal of Franklin Institute, https://www.elsevier.com/open-access/userlicence/1.0/

[13] Yakubu A. and Ziyadi N. (2022), Strong Allee effect and basins of attraction in a discrete-time zoonotic infectious disease model, Natural resource modeling, 35:e12310. https://doi.org/10.1111/nrm.12310

[14] Samantha J. M., Ellen E. S., and Brian G. M. (2013), Feline immunodeficiency virus latency, Retrovirology, 10(69) https://dx.doi:10.1186/1742-4690-10-69

[15] Roberts M. G. and Jowett J. (1996), An SEI model with density-dependent demographics and epidemiology, Journal of Mathematics Applied in Medicine and Biology, 13, 245-257

[16] Usaini S., Lloyd A. L., Anguelov R. and Garba S. M. (2017), Dynamical behavior of an epidemiological model with a demographic Allee effect, Math. Comp. Simul. 133, 311-325.

[17] McDonnel S. J., Sparger E. E. and Murphy B. G. (2013), Feline immunodeficiency virus latency, Retrovirology, 10 (69), 1-8. doi:10.1186/1742-4690-10-69.

[18] Busenberg S., Cooke K.L. and Thieme H. (1991), Demographic change and persistence of HIV/AIDS in a heterogeneous population, SIAM Journal of Applied Mathematics, 51 (4), 1030-1052.

[19] Busenberg S. and Van den Driessche P. (1990), Analysis of a disease transmission model in a population with varying size, Journal of Mathematical Biology, 28, 257-270.

[20] Perko L. (2001), *Differential Equations and Dynamical Systems*. Third Edition, New York.

[21] Buonomo B. and Lacitignolo D. (2011), On the backwrd bifurcation of a vaccination model with nonlinear incidence, Nonlinear Analysis: Modelling and Control, 16 (1), 30-46.

[22] Hilker F. M., Population collapse to extinction: the catastrophic combination of parasitism and Allee effect, Journal of Biological dynamics, 4 (1), 86-101.