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Modeling and Stability Analysis of the African Swine Fever Epidemic Model

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Abstract: In this paper, a mathematical model for the transmission dynamics and control of African swine fever with recruitment of susceptible, exposed and infected domestic pigs into the population is studied using a system of ordinary differential equations. The basic reproduction number R_0 for the model was obtained and its dependence on model parameters discussed. Without the inflow of exposed and infected pigs into the pig population, the model exhibits the disease-free equilibrium E_0 and the endemic equilibrium E_1. The disease-free equilibrium E_0 is globally stable if the basic reproduction number $R_0 < 1$ and the disease will be wiped out of the population. If $R_0 > 1$, the endemic equilibrium E_1 is asymptotically globally stable and the disease persists in the pig population. With the influx of exposed and infective domestic pigs, the model has only a unique endemic equilibrium E_e that is globally asymptotically stable and the disease persists.

Numerical simulation is carried out to verify the analytical results. It is revealed that with the influx of the exposed and infected pigs, the disease is maintained at endemic equilibrium.

Keywords: African swine fever; endemic equilibrium; global stability; Lyapunov function; reproduction number.

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I. Introduction

African swine fever is a devastating haemorrhagic fever of domestic pigs that causes up to 100% mortality of affected pigs [22], and is a major threat to the pig industry worldwide. African swine fever virus is the causative agent of African swine fever. The organism which causes ASF is a DNA virus classified within the genus *Asfivirus, Asfarviridae* family [7] that naturally infects domestic and wild pigs. It is highly contagious and is transmitted by direct contact between infectious and susceptible domestic pigs or by indirect contact with or inges- tion of infectious secretions and excretions. It is endemic in most sub-Saharan African countries where wild pigs hosts and soft ticks vectors of the genus *Or- nithodoros* act as biological reservoirs for the ASFV [4,

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based on limiting these transports rather than focusing on controlling the virus in the natural disease reservoir hosts, such as the wild pigs and wart hogs [22]. This means that all actors in the pig value chain (farmers, middle men, butchers, restaurant owners and consumers) are to some extent responsible and involved in continued spread of the disease.

Compartmental models to study the spread of infectious diseases consider the population to consist of different epidemiological classes. The suscepti- ble individuals are assumed to first go through a latent (exposed) period be- tween being infected and becoming infective (infectious). This results into the Susceptible-exposed-infective-removed (SEIR) type of epidemic model. Some SEIR epidemic models with influx of infective individuals for bilinear mass ac- tion or standard incidence for horizontal disease transmission have been studied (see [16, 17, 18, 25] for example and the references there in).

A simulation model to study the spread of ASF within a pig unit and impact of unit size on the spread of ASF was proposed by [12]. The model incorporated the effects of residues from dead animals in an exponential fading out pattern. They found out that emergency vaccination against classical swine fever can be equally effective and safe as pre-emptive culling. A stochastic individual-based simulation model to estimate the probability of releasing ASFV-infected pigs via emergency sale was considered in [6]. In Barongo *et al.* [2] and Guinat *et al.* [11], stochastic mathematical models were designed to simulate the transmission dynamics of ASFV in a free-ranging pig population under various interventions and to estimate quantitative pig-to-pig transmission parameters for the circu- lating ASFV strain, respectively.

In the present paper a deterministic model is presented. The pig population is divided into four epidemiological classes based on the disease status. In the SEIR epidemic model a population that consists of susceptible (S), exposed (E), infective (I) and removed (R) is presented. Susceptible pigs become exposed, that is, infected but not yet infective. They remain in the exposed stage for a certain period before they become infective. The infective pigs are infectious and capable of transmitting the ASF to susceptible pigs. The model is used to describe the transmission dynamics and explore control strategies for the African swine fever epidemic in domestic pigs.

II. Model formulation

The model consists of four compartments categorizing domestic pigs based on their status with respect to the disease. The following are the assumptions and definitions of variables and parameters definitions used in the model formulation.

Assumptions

(i) The model assumes homogeneous mixing of individuals in the population, that is, all domestic pigs have equal likelihood of getting infected if there are effective contacts with infective individuals.
(ii) Domestic pigs do not recover from the disease.
(iii) Total domestic pig population change through reproduction and immigra- tion.
(iv) Individuals can only be infected through contacts with infectious pigs.

Variables and parameters

The model definitions of variables and parameters are given as follows:
$S(t)$: susceptible population size of domestic pigs at time t
$E(t)$: latent (exposed) population size of domestic pigs at time t $I(t)$: infective population size of domestic pigs at time t
$R(t)$:removed population size of domestic pigs at time t
μ : per capita natural mortality rate
v : disease related mortality rate
Λ : per capita recruitment rate of domestic pig population
$1 - p - k$: proportion of domestic pigs that enter the susceptible class
p : proportion of domestic pigs that enter the exposed class k : proportion of domestic pigs that enter the infective class ρ : removal rate of infective domestic pigs
α : disease transmission rate
c : the average contact rate
β : transfer rate between the exposed and the infective

Equations of the model

Using descriptions of variables, parameters and assumptions, the following cou- pled system of ordinary differential equations which describe the progress of the disease is obtained.

$$\begin{aligned}
\frac{dS}{dt} &= \Lambda(1 - p - k) - acS\frac{I}{N} - \mu S, \\
\frac{dE}{dt} &= p\Lambda + acS\frac{I}{N} - (\beta + \mu)E, \\
\frac{dI}{dt} &= k\Lambda + \beta E - (\rho + v + \mu)I, \\
\frac{dR}{dt} &= \rho I - \mu R,
\end{aligned} \tag{1}$$

where $N(t) = S(t) + E(t) + I(t) + R(t)$ together with

$$\frac{dN}{dt} = \Lambda - \mu N - vI.$$

System (1), can be studied in the closed set

$$\Omega = \{S, E, I, R) \in \mathbb{R}^4_+ : 0 \leq S + E + I + R = N \leq \Lambda/\mu\},$$

where $\mathbb{R}^4_+$ denotes the non negative cone of $\mathbb{R}^4$ including its lower dimensional faces.

It can be shown that Ω is positively invariant with respect to the system (1).

We denote the boundary and the interior of Ω by $\partial\Omega$ and $\mathring{\Omega}$ respectively.

III. Analysis of the Model

We consider the equations of the normalized quantities. For convenience, we rewrite system (1) in terms of proportions of the indiyidual pigs in each class. We make the transformation $s = \frac{S}{N}, e = \frac{E}{N}, i = \frac{I}{N}$ and $r = \frac{R}{N}$ as the proportions for the epidemiological classes $S(t)$, $E(t)$, $I(t)$ and $R(t)$ respectively. Differentiating with respect to time t, it is clear that s, e, i and r satisfy the following system of differential equations

$$\begin{aligned}
\frac{ds}{dt} &= \frac{\Lambda}{N} - acsi - \mu s - s\left[\frac{\Lambda}{N} - \mu - vi\right], \\
\frac{de}{dt} &= acsi - (\beta + \mu)e - e\left[\frac{\Lambda}{N} - \mu - vi\right], \\
\frac{di}{dt} &= \beta e - (\rho + v + \mu)i - i\left[\frac{\Lambda}{N} - \mu - vi\right], \\
\frac{dr}{dt} &= \rho i - \mu r - r\left[\frac{\Lambda}{N} - \mu - vi\right],
\end{aligned} \tag{2}$$

together with

$$\frac{dN}{dt} = \square \frac{\Lambda}{N} - \mu - vi \square N. \tag{3}$$

From Eq. (3), $\frac{dN}{dt} = (\wedge \frac{}{N} \mu - vi)N$, setting $\frac{dN}{dt} = 0$, for $N \neq 0$ implies that $\frac{\Lambda}{N} = \mu + vi$.

Substituting for $\frac{\Lambda}{N}$ into system (2) and simplifyingyields;

$$\begin{aligned}
\frac{ds}{dt} &= (1 - p - k)(\mu + vi) - acsi - \mu s, \\
\frac{de}{dt} &= p(\mu + vi) + acsi _ (\beta + \mu)e, \\
\frac{di}{dt} &= k(\mu + vi) + \beta e _ (\rho + v + \mu)i, \\
\frac{dr}{dt} &= \rho i _ \mu r, \\
s + e + i + r &= 1.
\end{aligned} \tag{4}$$

In the absence of influx of infected pigs into the population, ($p = k = 0$). Substituting for r of s, e and i, that is, $r = 1 \_ s \_ e \quad i$, we reduce system (4) to the following 3-dimension system

$$\begin{aligned}
\frac{ds}{dt} &= \mu + vi _ acsi _ \mu s, \\
\frac{de}{dt} &= acsi _ (\beta + \mu)e, \\
\frac{di}{dt} &= \beta e _ (\rho + v + \mu)i.
\end{aligned} \tag{5}$$

System (5), can be studied in the closed set

$$T = \{(s, e, i) \in \mathbb{R}^3_+ : 0 \leq s + e + i \leq 1\},$$

where $\mathbb{R}^3_+$ denotes the non negative cone of $\mathbb{R}^3$ including its lower dimensional faces.

It can be shown that T is positively invariant with respect to the system (5).

We denote the boundary and the interior of T by ∂T and $\overset{\circ}{T}$ respectively. Thus, system (5) is bounded.

Equilibria of the model

To compute the equilibrium points of the system (5), we set the right-hand side of system (5) equal to zero and obtain the disease-free and endemic equilibrium $E_0(1, 0, 0)$ and

$$E_1 = \left(\frac{(\beta+\mu)(\rho+\mu+\nu)}{\beta ac}, \frac{\mu(\rho+\mu+\nu)}{ac\beta}, \frac{(\beta+\mu)(\rho+\mu+\nu) - ac\beta}{\beta\nu - (\beta+\mu)(\rho+\mu+\nu)}, \frac{\mu(\beta+\mu)(\rho+\mu+\nu) - ac\beta\mu}{ac\beta\nu - ac(\beta+\mu)(\rho+\mu+\nu)}\right)$$

respectively.

Local and global stability of the disease free equilib- rium E_0

The Jacobian matrix for the system (5) is given by

$$J = \begin{bmatrix} -(aci+\mu) & 0 & \nu - acs \\ aci & -(\beta+\mu) & acs \\ 0 & \beta & -(\rho+\mu+\nu) \end{bmatrix}. \quad (6)$$

The local stability of the disease free equilibrium $E_0(1, 0, 0)$ is obtained by evaluating the Jacobian matrix (6) at $E_0(1, 0, 0)$ to give

$$J_{E_0} = \begin{bmatrix} -\mu & 0 & \nu - ac \\ 0 & -(\beta+\mu) & ac \\ 0 & \beta & -(\rho+\mu+\nu) \end{bmatrix}. \quad (7)$$

It is clear from the first column that the Jacobian matrix (7) has a negative eigenvalue $-\mu$. The other two eigenvalues can be obtained by reducing the Jacobian matrix (7) into a 2×2 matrix given by

$$J_{E_0} = \begin{bmatrix} -(\beta+\mu) & ac \\ \beta & -(\rho+\mu+\nu) \end{bmatrix}. \quad (8)$$

Using the trace-determinant method for stability analysis, we need to show that $tr(J_{E_0}) < 0$ and $det(J_{E_0}) > 0$ if the equilibrium point $E_0(1, 0, 0)$ is stable.
Thus, from the Jacobian matrix (8), it is clearly seen that

$$tr(J_{E_0}) = -(\beta + \nu + \rho + 2\mu) < 0.$$

Thus, for stability we seek $det(J_{E_0}) > 0$.
This gives the expression

$$det(J_{E_0}) = (\rho + \mu + \nu)(\beta + \mu) - ac\beta > 0,$$

$$\Rightarrow (\rho + \mu + \nu)(\beta + \mu) > ac\beta,$$

which is the same as

$$\frac{ac\beta}{(\rho + \mu + \nu)(\beta + \mu)} < 1,$$

where

$$R_0 = \frac{ac\beta}{(\rho + \mu + \nu)(\beta + \mu)}$$

.

It should be noted that the expression for the basic reproduction R_0 iscomprised of combinations of parameters that can be interpreted as follows:
a is the transmission rate between susceptible domestic pigs and infective ones and c is the contact rate.
$\frac{1}{\rho+\mu+\nu}$ is the effective infectious period.

$\frac{ac}{\rho+\mu+\upsilon}$ is the number of exposed domestic pigs produced by one infectious domestic pig during the effective infectious period.
$\frac{\beta}{\beta+\mu}$ is the exposed stage into the infective stage.

Thus the endemic equilibrium $E_1(s, e, i)$ expressed in terms of the basic reproduction number R_0 is given by

$$E_1 = \left(\frac{1}{R_0}, \frac{\mu(\rho+\mu+\upsilon)(R_0-1)}{R_0[\beta(\rho+\mu)+\mu(\rho+\mu+\upsilon)]}, \frac{\beta\mu(R_0-1)}{R_0[\beta(\rho+\mu)+\mu(\rho+\mu+\upsilon)]} \right).$$

The following lemma is used to establish stability of E_0. it is easy to see that $tr(J_{E_0}) < 0$ and $det(J_{E_0}) > 0$ if $R_0 < 1$. Therefore, we have established the following lemma.

Lemma 3.1. *The disease free equilibrium $E_0(1, 0, 0)$ is locally stable if $R_0 < 1$ and unstable if $R_0 > 1$.* R_0 is called the basic reproduction number [1], defined as the number of secondary infective cases produced by one primary case introduced into an entirely susceptible population at the disease-free equilibrium. It is an important parameter that plays a big role in the control of the disease. The effort to eliminate the disease from the population targets the parameters that will bring its value to less than one. When the basic reproduction number is less than unity, the disease-free equilibrium is locally asymptotically stable, and there is a possibility that the disease will be wiped out of the population.

The global stability of the disease free equilibrium E_0 is established from the following theorem below:

Theorem 3.2. *Suppose all the pigs that enter into the pig population are susceptible, that is, $p + k = 0$, then;*

(a) the disease free equilibrium $E_0(1, 0, 0) \in T$ exists for all non-negative values of its parameters and it is globally asymptotically stable when $R_0 \leq 1$ and unstable when $R_0 > 1$.

(b) if $R_0 > 1$, solutions to the system (5) starting sufficiently close to disease-free equilibrium $E_0(1, 0, 0) \in T$ move away from the disease-free equilibrium E_0 except those starting close to the invariant s–axis which approach E_0 along this axis.

(c) if $R_0 > 1$, then system (5) has a unique endemic equilibrium $E_1(s, e, i)$.

Proof. We note that, when all new recruits into the pig population are susceptible, that is, p=k=0, we obtain $s = 1$ for $i = 0$ from the first equation of system (5) . From the second and third equations of system (5), we get $e = 0$ for $i = 0$. Thus, we have the disease free equilibrium $E_0(1, 0, 0) \in T$.

When $R_0 \leq 1$, Consider the Lyapunov function $L = \beta e + (\beta + \mu)i$. Its

derivative along the solutions of system (5) is

$$\begin{aligned} \dot{V} &= \beta\frac{de}{dt} + (\beta+\mu)\frac{di}{dt}, \\ &= (\beta acsi - (\beta+\mu)(\rho+\mu+\upsilon)i) + 2\beta(\beta+\mu)e, \\ &\leq (\beta acs - (\beta+\mu)(\rho+\mu+\upsilon))i, \\ &\leq \left(\frac{\beta acs}{(\beta+\mu)(\rho+\mu+\upsilon)} - 1\right)(\beta+\mu)(\rho+\mu+\upsilon)i, \\ &= (R_0 s - 1)(\beta+\mu)(\rho+\mu+\upsilon)i, \text{ for } s = 1, \\ &\leq (R_0 - 1)(\beta+\mu)(\rho+\mu+\upsilon)i, \\ &\leq 0 \text{ if } R_0 < 1. \end{aligned}$$

□

It is shown that $\dot{V} \leq 0$, if $R_0 \leq 1$ and the equality, $\dot{V} = 0$ holds when $R_0 = 1$ and $e = i = 0$. For $R_0 > 1$, then $\dot{V} > 0$ when s is sufficiently close to 1 except when $e = i = 0$. The maximum invariant set in $\{(s, e, i) \in T : \dot{V} = 0\}$ is the singleton E_0. The global stability of E_0 when $R_0 \leq 1$ follows from the Lyapunov-Lassalle theorem [13]. Thus all solutions paths in T approach the disease-free equilibrium E_0 as $t \to \infty$. This proves that the disease free equilibrium E_0 is globally asymptotically stable.

Local and global stability of the endemic equilibrium E_1

E_1

From the endemic equilibrium $E_1(s, e, i)$ expressed in terms of the basic reproduction number R_0 given by

$$E_1 \left(\frac{1}{R_0}, \frac{\mu(\rho+\mu+\upsilon)(R_0-1)}{R_0[\beta(\rho+\mu)+\mu(\rho+\mu+\upsilon)]}, \frac{\beta\mu(R_0-1)}{R_0[\beta(\rho+\mu)+\mu(\rho+\mu+\upsilon)]}\right),$$

it is evident that when $R < 1$, e and i assume negative values which is not biologically realistic. This implies that the system has no positive endemic equilibrium when $R < 1$. The positive endemic equilibrium E_1 is only possible when $R > 1$. In order to establish the local stability of the endemic equilibrium E_1, the Jacobian matrix (6) is evaluated at the endemic equilibrium E_1 to give

$$J_{E_1} = \begin{bmatrix} -\frac{ac\beta\mu(R_0-1)}{R_0[\beta(\rho+\mu)+\mu(\rho+\mu+\upsilon)]} + \mu & 0 & \upsilon - \frac{\beta c}{R_0} \\ \frac{ac\beta\mu(R_0-1)}{R_0[\beta(\rho+\mu)+\mu(\rho+\mu+\upsilon)]} & -(\beta+\mu) & \frac{\beta c}{R_0} \\ 0 & \beta & -(\rho+\mu+\upsilon) \end{bmatrix}. \quad (9)$$

The Jacobian matrix (9) has the $tr(J_{E_1})$ and $det(J_{E_1})$ given by

$$tr(J_{E_1}) = -\frac{ac\beta\mu(R_0-1)}{R_0[\beta(\rho+\mu)+\mu(\rho+\mu+\upsilon)]} + 3\mu + \beta + \rho + \upsilon .$$

$$det(J_{E_1}) = \frac{ac\beta\mu(R_0-1)}{R_0[\beta(\rho+\mu)+\mu(\rho+\mu+\upsilon)]} + \mu)(\beta+\mu)(\rho+\mu+\upsilon) + \left(\frac{a^2c^2\beta^2\mu(R_0-1)}{R_0^2[\beta(\rho+\mu)+\mu(\rho+\mu+\upsilon)]} + ac\beta\mu\right) + \left(\frac{a^2c^2\beta^2\mu(R_0-1)(\upsilon-\rho)}{R_0[\beta(\rho+\mu)+\mu(\rho+\mu+\upsilon)]}\right).$$

From the above expressions, it can be noted that $R_0 > 1$ implies that $tr(J_{E_1}) < 0$ and $det(J_{E_1}) > 0$. Thus, conditions for trace and determinant hold for the endemic equilibrium E_1 to be locally asymptotically stable.

The global stability of the endemic equilibrium E_1 can be established by applying the theory of competitive systems [14, 23] , and additive compound matrices and differential equations [20] for the analysis of the system.

The Jacobian matrix system (5) can be written as

$$J = \begin{bmatrix} -(aci+\mu) & 0 & \upsilon - acs \\ aci & -(\beta+\mu) & acs \\ 0 & \beta & -(\rho+\mu+\upsilon) \end{bmatrix} = \begin{bmatrix} a_{11} & a_{12} & a_{13} \\ a_{21} & a_{22} & a_{23} \\ a_{31} & a_{32} & a_{33} \end{bmatrix}, \quad (10)$$

where a_{ij} is the corresponding entry of the matrix J.

The second additive compound matrix of the Jacobian matrix (10) is calculated as follows:

$$J_{E_1}^{[2]} = \begin{bmatrix} a_{11}+a_{22} & a_{23} & -a_{13} \\ a_{32} & a_{11}+a_{33} & a_{12} \\ -a_{31} & a_{21} & a_{22}+a_{33} \end{bmatrix}. \quad (11)$$

$$J_{E_1}^{[2]} = \begin{bmatrix} -(aci+\beta+2\mu) & acs & -\upsilon+acs \\ \beta & -(aci+\upsilon+\rho+2\mu) & 0 \\ 0 & aci & -(\beta+\upsilon+\rho+2\mu) \end{bmatrix}. \quad (12)$$

Deftnition: Competitive system. Let $x \to f(x)$ be a smooth vector field defined for x in an open set $D \subset R^n$. The differential equation

$$x' = f(x), x \in D, \quad (13)$$

is said to be competitive in D if, for some diagonal matrix $H = diag(u_1, u_2, \ldots, u_n)$, where each u_i is either 1 or -1, $H\frac{\partial f}{\partial x}H$ has non- positive off-diagonal elements for all $x \in D$. If D is convex, the flow of a competitive system (5) preserves, for $t < 0$, the partial ordering in R^n defined by the orthant $K = \{x_1, \ldots, x_n) \in R^n : u_i x_i \geq 0\}$

Let the matrix H be chosen as

$$H = \begin{bmatrix} 1 & 0 & 0 \\ 0 & -1 & 0 \\ 0 & 0 & 1 \end{bmatrix}. \quad (14)$$

Then from the matrix H and the Jacobian matrix (6), the following matrix is obtained

$$H(J_{E_1})H = \begin{bmatrix} -(aci+\mu) & 0 & v-acs \\ -aci & -(\beta+\mu) & -acs \\ 0 & -\beta & -(\rho+\mu+v) \end{bmatrix}. \quad (15)$$

It is observed that the system is competitive in ω with respect to the partial ordering defined by the orthant $K = \{(s, e, i) \in \mathbb{R}^3 : s \geq, e \leq, i \geq 0\}$. In Hirsch [14] and Smith [23], it is proved that the three-dimensional competitive systems that live in convex sets have the Poincaré-Bendixson Property. That is to say, any non-empty compact omega set that contains no equilibrium must be a closed orbit. Following [23], the following theorem is stated to help in establishing the global stability of the endemic equilibrium E_1.

Let $p(t)$ with minimal period ω and orbit $\Gamma = p(t) : 0 \leq t \leq \omega\}$ be the periodic solution of the competitive system. The definitions stated in [13] are used to establish the stability of the orbit.

Theorem 3.3. *Assume $n = 3$ and D is convex. Suppose that Eq.(13) is competitive in D. Then it satisfies the Poincaré-Bendixson Property.*

The endemic equilibrium point E_1 is globally asymptotically stable in the interior of T so that the disease remains endemic. This is established by proving the Theorem (3.4) below.

Theorem 3.4. *If $p + k = 0$, and $R_0 > 1$, then the endemic equilibrium point E_1 of the system (5) is locally asymptotically stable in $\mathring{T}$. All solutions with initial data $(1, 0, 0)$ approach the disease free equilibrium E_0.*

Proof. It can easily be seen that all trajectories starting from the boundary ∂T of T enter $\mathring{T}$ except those on the s-axis which converge to E_0 along this invariant axis. Therefore, E_0 is the only T limit point in the boundary of T. It is sufficient to show that E_1 is globally asymptotically stable in $\mathring{T}$. Since system (5) is competitive, as long as $R_0 > 1$ and E_1 is locally asymptotically stable, the result follows from Theorem (3.3) that system (5) has the property of stability of periodic orbits. This can be obtained from [20] for the asymptotic orbital stability of a periodic stability of a periodic orbit in a general autonomous system. Thus, it suffices to prove that the linear non-autonomous system

$$\omega'(t) = (J^{[2]}_{E_1}(p(t)))\omega(t), \quad (16)$$

is asymptotically stable where $J^{[2]}_{E_1}$ is the second additive compound matrix (12). From Eq.(16), a linear system with respect to the solution $p(t) = (s(t), e(t), i(t))$ is obtained and is given by

$$\begin{aligned} w_1'(t) &= -(aci(t) + \beta + 2\mu)w_1(t) + acs(t)w_2(t) + (acs(t) - v)w_3(t), \\ w_2'(t) &= \beta w_1(t) - (aci(t) + \beta + v) + \rho + 2\mu)w_2(t), \\ w_3'(t) &= aci(t)w_2(t) - (\beta + \rho + v + 2\mu)w_3(t). \end{aligned} \quad (17)$$

To prove the asymptotic stability of the system (17), we consider the following Lyapunov function:

$$V(w_1(t), w_2(t), w_3(t); s(t), e(t), i(t)) = \sup\left\{|w_1(t)|, \frac{e(t)}{i(t)}(|w_2(t)| + |w_3(t)|)\right\}. \tag{18}$$

Let the left hand derivative of $V(t)$ be denoted by $D_+V(t)$, then the following inequalities are obtained:

$$\begin{aligned} D_+|w_1(t)| &\leq -(aci(t) + \beta + 2\mu)|w_1(t)| + acs(t)|w_2(t)| + (acs(t) - v)|w_3(t)| \\ &\leq -(aci(t) + \beta + 2\mu)|w_1(t)| + \frac{acs(t)i(t)}{e(t)}\frac{e(t)}{i(t)}(|w_2(t)| + |w_3(t)|) \end{aligned} \tag{19}$$

$$D_+|w_2(t)| \leq \beta|w_1(t)| - (aci(t) + \beta + v + \rho + 2\mu)|w_2(t)| \tag{20}$$

$$D_+|w_3(t)| \leq aci(t)|w_2(t)| - (\beta + v + \rho + 2\mu)|w_3(t)|. \tag{21}$$

Adding Eqs. (20) and (21) gives

$$\begin{aligned} &D_+(|w_2(t)| + |w_3(t)|) \\ &\leq \beta|w_1(t)| - (\beta + v + \rho + 2\mu)|w_2(t)| - (\beta + v + \rho + 2\mu)|w_3(t)|, \\ &\leq \beta|w_1(t)| - (\beta + v + \rho + 2\mu)(|w_2(t)| + |w_3(t)|), \end{aligned} \tag{22}$$

We also have the following expression

$$\begin{aligned} &D_+\frac{e(t)}{i(t)}(|w_2(t)| + |w_3(t)|), \\ &= \frac{e'(t)}{e(t)} - \frac{i'(t)}{i(t)}\frac{e(t)}{i(t)}(|w_2(t)| + |w_3(t)|) + \frac{e(t)}{i(t)}D_+(|w_2(t)| + |w_3(t)|), \end{aligned} \tag{23}$$

Substituting Eq. (22) into Eq. (23) gives

$$\begin{aligned} &D_+\frac{e(t)}{i(t)}(|w_2(t)| + |w_3(t)|) \\ &\leq \frac{e(t)}{i(t)}\beta|w_1(t)| + \frac{e'(t)}{e(t)} - \frac{i'(t)}{i(t)} - (\beta + v + \rho + 2\mu)\frac{e(t)}{i(t)}(|w_2(t)| + |w_3(t)|). \end{aligned} \tag{24}$$

(25)

From Eqs. (19) and (25), we have

$$D_+V(t) \leq \sup\{h_1(t), h_2(t)\}V(t).$$

$$h_1(t) = -(aci(t) + \beta + 2\mu) + \frac{acs(t)i(t)}{e(t)} \tag{26}$$

$$h_2(t) = \beta\frac{e(t)}{i(t)} + \frac{e'(t)}{e(t)} - \frac{i'(t)}{i(t)} - (\beta + v + \rho + 2\mu) \tag{27}$$

From the second and third equations of system (5), we have

$$\frac{acs(t)i(t)}{e(t)} = \frac{e^{l}(t)}{e(t)} + \beta + \mu, \tag{28}$$
$$\beta\frac{e(t)}{i(t)} = \frac{i^{l}(t)}{i(t)} + \rho + v + \mu.$$

Substituting into Eqs. (26) and (27) gives

$$h_1(t) = \frac{e^{l}(t)}{e(t)} - aci(t) - \mu \tag{29}$$
$$h_2(t) = \frac{e^{l}(t)}{e(t)} - \beta - \mu \tag{30}$$

Let $\delta = min\{aci + \mu, \beta + \mu\}$ so that

$$sup\{(h_1(t),\ h_2(t))\} = \frac{e^{l}(t)}{e(t)} - \delta \tag{31}$$

Taking ω as a limit, the integral of Eq. (31) is evaluated as follows:

$$\int_0^{\omega} sup\{h_1(t),\ h_2(t)\}dt \leq \Sigma \ln e(t) \Sigma_0^{\omega} - \delta\omega = -\mu\omega < 0.$$

This shows that the periodic solution $(s(t),\ e(t),\ i(t))$ is asymptotically stable. This establishes the fact that the endemic equilibrium is globally asymptotically stable. □

If there is influx of infected pigs into the population, that is, $p + k = 0$, we analyze system (4) and investigate the existence of equilibrium points. System (4) is reduced to a 3-dimensional system of equations by eliminating r since $r = 1 - s - e - i$. This gives

$$\begin{aligned}
\frac{ds}{dt} &= (1 - p - k)(\mu + vi) - acsi - \mu s,\\
\frac{de}{dt} &= p(\mu + vi) + acsi - (\beta + \mu)e,\\
\frac{di}{dt} &= k(\mu + vi) + \beta e - (\rho + v + \mu)i.
\end{aligned} \tag{32}$$

System (32) can be studied in the closed set $T = \{(s, l, i) \in R^3_+ : 0 \leq s + e + i \leq 1\}$ where R^3_+ denotes the non negative cone of R^3 including its lower dimensional faces. It can be verified that D is positively invariant with respect to system (32). We denote the boundary and the interior of T by ∂T and $\overset{\circ}{T}$ respectively. Thus, system (32) is bounded. The equilibrium of system (32) is obtained as follows:

$$\begin{aligned}
&(1 - p - k)(\mu + vi) - acsi - \mu s = 0,\\
&p(\mu + vi) + acsi - (\beta + \mu)e = 0,\\
&k(\mu + vi) + \beta e - (\rho + v + \mu)i = 0.
\end{aligned} \tag{33}$$

From the last equation of system (33), we have

$$\beta e = (\rho + v + \mu)i - k(\mu + vi),$$
$$e = \frac{(\rho + v + \mu)i - k(\mu + vi)}{\beta}. \quad (34)$$

Substituting for e in Eq. (34) into the second equation of system (33) gives

$$p\mu + pvi + acsi - (\beta + \mu)\frac{(\rho + v + \mu)i - k(\mu + vi)}{\beta} = 0. \quad (35)$$

Simplifying Eq. (35) gives

$$acsi = [((\beta + \mu)(\rho + v + \mu) - p\beta v - (\beta + \mu)kv)i - (p\beta\mu + (\beta + \mu)k\mu]/\beta,$$
$$\Rightarrow s = \frac{[(\beta + \mu)(\rho + v + \mu) - p\beta v - (\beta + \mu)kv]i - (p\beta\mu + (\beta + \mu)k\mu)}{ac\beta i}. \quad (36)$$

Substituting for s in Eq. (36) into the first equation of system (33) gives

$$(1 - p - k)(\mu + vi) - \frac{[(\beta+\mu)(\rho+v+\mu)-p\beta v-(\beta+\mu)kv]i-(p\beta\mu+(\beta+\mu)k\mu)}{\beta}$$
$$- \mu\frac{[(\beta+\mu)(\rho+v+\mu)-p\beta v-(\beta+\mu)kv]i-(p\beta\mu+(\beta+\mu)k\mu)}{ac\beta i} = 0,$$

$$H(i) = ac[(\beta + \mu)(\rho + v + \mu) - (\beta + \mu k)v]i^2$$
$$+\mu[(\beta + \mu)(\rho + v + \mu) - a\beta c + (p\beta + (\beta + \mu)k)v - ac\mu k)]\, i - \mu^2[p\beta + (\beta + \mu)k] \quad (37)$$

It is noted for $p = k = 0$, one root is $i = 0$, and a second root

$$i = \frac{\mu\,[a\beta c - (\beta + \mu)(\rho + v + \mu)]}{ac[(\beta + \mu)(\rho + v + \mu) - \beta v]} \quad (38)$$

which is positive if and only if
$\sigma = a\beta c - (\beta + \mu)(\rho + v + \mu) > 0$
For $0 < p + k < 1$, the quadratic Eq. $H(i) = 0$, that is Eq. (37) gives two values. The negative value that is biologically meaningless and the positive solution given by

$$i = \frac{\mu[\sigma-(p\beta+(\beta+\mu)k)v-ac\mu k)]+\mu\sqrt{([\sigma-(p\beta+(\beta+\mu)k)v-ac\mu k)]^2+4ac[(\beta+\mu)(\rho+v+\mu)-(\beta+\mu k)v][p\beta+(\beta+\mu)k]}}{2ac[(\beta+\mu)(\rho+v+\mu)-(\beta+\mu k)v]}.$$

For p, k sufficiently small, we use the binomial approximation $(1 + x)^{1/2} \approx 1 + \frac{x}{2}$ (see Brauer and van den Driessche [3]). From the equation above, we note that as $p \to 0, k \to 0$, the positive root i becomes

$$\lim_{p,k\to 0} i = \frac{\mu\sigma + \mu|\sigma|}{2ac[(\beta + \mu)(\rho + v + \mu) - \beta v]}$$
$$= \begin{cases} 0, \text{ for } \sigma < 0 \\ \frac{\mu\sigma}{ac[(\beta+\mu)(\rho+v+\mu)-\beta v]}, \text{ for } \sigma > 0 \end{cases}. \quad (39)$$

From the expression above, we have

$$\approx \mu\sigma + \mu|\sigma| \left[1+\frac{2ac[(\beta+\mu)(\rho+v+\mu)-(\beta+\mu k)v][p\beta+(\beta+\mu)k]}{\sigma^2}\right] \quad (40)$$

From Eq.(40), it is clear that
if $R_0 < 1$, so that $\sigma < 0$, we have

$$i \approx \frac{\mu[p\beta+(\beta+\mu)k]}{|\sigma|}, \quad (41)$$

while if $R_0 > 1$, so that $\sigma > 0$, then

$$i \approx \frac{\mu\sigma}{ac[(\beta+\mu)(\rho+v+\mu)-\beta v]} + \frac{\mu[p\beta+(\beta+\mu)k]}{|\sigma|}. \quad (42)$$

We note that the model has a threshold value $R_0 = 1$ for the values of p and k close to zero. In addition to that, for $0 < p + k < 1$, system (32) has exactly one endemic equilibrium for all parameter values for which the disease will always persist in the population. There is no disease free-equilibrium and thus the model has no basic reproduction number R_0 for $0 < p + k < 1$. However, from Eqs. (41) and (42) there is a threshold-like behavior in the sense as the values of p and k go to zero. If $R_0 < 1$, endemic equilibrium approaches a disease-free equilibrium as the p and k go to zero, otherwise if $R_0 > 1$, then for $p \geq 0$ and $k \geq 0$, the model has a unique endemic equilibrium E_e.

Local stability of the endemic equilibrium E_e

The Jacobian matrix of system (32) evaluated at endemic equilibrium $E_e(s,e,i)$ is given by

$$J = \begin{bmatrix} -(aci+\mu) & 0 & (1-p-k)v-acs \\ ai & -(\beta+\mu) & pv+acs \\ 0 & \beta & -(\rho+v+\mu-kv) \end{bmatrix}. \quad (43)$$

The characteristic equation for the Jacobian matrix (43) is

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

where

$$a_1 = \rho+\beta+3\mu+aci+(1-k)v$$
$$a_2 = (aci+\mu)[(\beta+\rho+2\mu+(1-k)v]+[(\beta+\mu)(\rho+\mu+(1-k)v-\beta(pv+acs)]$$
$$a_3 = (aci+\mu)[(\beta+\mu)(\rho+\mu+(1-k)v)-\beta(pv+acs)]-a\beta i[(1-k-p)v-acs]$$

It is clear that the constants a_1, a_2 and a_3 are positive. Now, we compute

$$\begin{aligned}
&a_1a_2 - a_3\\
&= (aci + \mu)[\rho + \beta + 3\mu + aci + (1 - k)][(\beta + \rho + 2\mu + (1 - k)v]\\
&+[\rho + \beta + 3\mu + aci + (1 - k)v][(\beta + \mu)(\rho + \mu + (1 - k)v - \beta(pv + acs)]\\
&-\{(aci + \mu)[(\beta + \mu)(\rho + \mu + (1 - k)v) - \beta(pv + acs)] - a\beta i[(1 - k - p)v - acs]\}\\
&> 0.
\end{aligned}$$

The Routh-Hurwitz conditions ($a_1 > 0$, $a_2 > 0$, $a_3 > 0$, $a_1a_2 > a_3$) for a polynomial of degree three are satisfied and hence the unique endemic equilibrium $E_e(s, e, i)$ for $0 < p + k < 1$ for system (32) is locally asymptotically stable.

3.5 Global stability of the endemic equilibrium E_e

In the following, the geometrical approach of Li and Muldowney [18] is used to obtain the necessary and sufficient conditions that the endemic equilibrium E_e is globally asymptotically stable. We first give a brief outline of this geometrical approach.

Let $f(x) \to x \in R^n$ be a C^1 function for x in an open set $D \subset R^n$. Consider the differential equation

$$\dot{x} = f(x). \tag{44}$$

Denote by $x(t, x_0)$ the solution to 44 such that $x(0, x_0) = x_0$. The following three assumptions are made

(H_1) D is simply connected;

(H_2) There exists a compact absorbing set $K \subset D$.

(H_3) Eq. (44) has a unique equilibrium $\tilde{x}$ in D.

The equilibrium $\tilde{x}$ is said to be globally stable in D if it is locally stable and all trajectories in D converge to $\tilde{x}$. For $n \geq 2$, by a *Bendixson* criterion we mean a condition satisfied by f which precludes the existence of non-constant periodic solutions of (44). The classical Bendixson's condition, $\operatorname{div} f(x) < 0$ for $n = 2$ is robust under C^1 local perturbations of f. For higher dimensional systems, the C^1 robust properties are discussed in Li and Muldowney [18].

A point $x_0 \in D$ is wandering for (44) if there exists a neighborhood of U of x_0 and $T > 0$ such that $U \cap x(t, \cdot)$ is empty for all $t > T$. Thus, for example, all the equilibrium and limit points are non-wandering. The following global-stability principle is established in Li and Muldowney [18] for finite systems in any finite dimension.

Theorem 3.5. . *Suppose that assumptions* (H_1), (H_2) *and* (H_3) *hold. Assume that (44) satisfies a Bendixson criterion that is robust under* C^1 *local perturbations of* f *at non-equilibrium non-wandering points for (44). Then* $\tilde{x}$ *is globally stable in* D *provided it is stable.*

The following *Bendixson* criterion is given in Li and Muldowney [18] and shown to have the robustness required by Theorem (3.6). Let $x \to P(x)$ *bean* $(\frac{n}{2})\times(\frac{?}{2})$ matrix-valued function that is C^1 for $x \in D$. Assume that $P^{-1}(x)$ exists and is continuous for $x \in K$, the compact absorbing set. A quantity $\tilde{q_2}$ is defined as

$$\tilde{q_2} = \lim \sup_{t\to\infty} \sup_{x_0\in K} \frac{1}{t} \int_0^t \mu(B(x(s,x_0)))\, ds \tag{45}$$

where

$$B = P_f P^{-1} + PJ^{[2]}P^{-1}, \tag{46}$$

the matrix P_f is obtained by replacing each entry p_{ij} of P by its derivative in the direction of f, p_{ij}, and the quantity $\mu(B)$ is the Lozinski˘ measure of B with respect of a vector norm $|.|$ in $\mathbf{R}^N$, $N = (_2{}^n)$, and is defined by

$$\mu(B) = \inf_{h\to 0^+} \frac{|I + hB| - 1}{h}.$$

For a simply connected, D, the condition $\tilde{q_2} < 0$ rules out the presence of any orbit that gives rise to a simple closed rectifiable curve that is invariant for (44), such as closed orbits, homoclinic orbits, and heteroclinic cycles as shown in Li and Muldowney [18]. Moreover, it is robust under C^1 local perturbations of f near any non-equilibrium point that is non-wandering. The global stability analysis follows from the following global-stability result proved in Li and Muldowney [18].

Theorem 3.6. . *Assume that the assumptions* (H_1), (H_2) *and* (H_3) *hold. Then the unique endemic equilibrium* $\tilde{x}$ *of system (44) is globally stable in* D *if* $\tilde{q_2} < 0$.

Let $x = (s, e, i)$ and let $f(x)$ denote the vector field of system (32). The Jacobian matrix $J = \partial f/\partial x$ associated with a general solution $x(t)$ of system (32) is given by

$$J = \begin{bmatrix} -(aci + \mu) & 0 & (1-p-k)v - acs \\ ai & -(\beta+\mu) & pv + acs \\ 0 & \beta & -(\rho + v + \mu - kv) \end{bmatrix}. \tag{47}$$

and its second compound matrix $J^{[2]}$ as follows (see Muldowney, 1990)

$$J^{[2]} = \begin{bmatrix} J_{11} & pv + acs & -(1-p-k)v + acs \\ \beta & J_{22} & 0 \\ 0 & ai & J_{33} \end{bmatrix} \tag{48}$$

where

$$\begin{aligned} J_{11} &= -(aci + \beta + 2\mu) \\ J_{22} &= -(aci + \rho + 2\mu + (1-k)v) \\ J_{33} &= -(\beta + \rho + 2\mu + (1-k)v) \end{aligned}$$

The proof of the theorem consists of choosing a suitable vector norm $|\cdot|$ in $\mathbf{R}^3$ and a 3×3 matrix-valued function $P(x)$, such that the quantity $q_2 < 0$. We set P as the following diagonal matrix

$$P(s,e,i) = diag\left(1, \frac{e}{i}, \frac{e}{i}\right).$$

Then $P_f P^{-1} = diag\left(0, \frac{e'}{e} - \frac{i'}{i}, \frac{e'}{e} - \frac{i'}{i}\right)$

and the matrix $B = P_f P^{-1} + PJ^{[2]}P^{-1}$ is given by

$$B = \begin{pmatrix} J_{11} & \frac{(pv+acs)i}{e} & \frac{(acs-(1-p-k)v)i}{e} \\ \frac{\beta e}{i} & J_{22} + \frac{e'}{e} - \frac{i'}{i} & 0 \\ 0 & ai & J_{33} + \frac{e'}{e} - \frac{i'}{i} \end{pmatrix} \tag{49}$$

This can be written in the following block form

$$B = \begin{pmatrix} B_{11} & B_{12} \\ B_{21} & B_{22} \end{pmatrix} \tag{50}$$

where

$$B_{11} = -(aci + \beta + 2\mu)$$

$$B_{12} = \left(\frac{(pv+acs)i}{e}, \frac{(acs-(1-p-k)v)i}{e}\right), B_{21} = \begin{pmatrix} \frac{\beta e}{i} \\ 0 \end{pmatrix}$$

$$B_{22} = \begin{pmatrix} \frac{e'}{e} - \frac{i'}{i} + J_{22} & 0 \\ ai & \frac{e'}{e} - \frac{i'}{i} + J_{33} \end{pmatrix}.$$

The following method stated in Li and Muldowney [18], and Fan *et al.* [8] is used. We let (u,v,w) to denote the vectors in $\mathbf{R}^3 = \mathbf{R} \sim \mathbf{R}^2$ for the norm $|.|$ in $\mathbf{R}^3$ chosen as

$$|(u,v,w)| = \max\{|u|, |v| + |w|\},$$

and let μ denote the Lozinskiĭ measure with respect to this norm. The method is used to estimate the Lozinskiĭ measure $\mu(B)$ with respect to $|.|$ as follows

$$\mu(B) \leq \sup\{g_1, g_2\},$$

where

$$g_1 = \mu_1(B_{11}) + |B_{12}|,$$
$$g_2 = |B_{21}| + \mu_1(B_{22}).$$

Note that $|B_{12}|$ and $|B_{21}|$ are operator norms of B_{12} and B_{21} with respect to the l_1 vector norm when they are regarded as mappings from $\mathbf{R}^2$ to $\mathbf{R}$, and $\mathbf{R}^2$ to $\mathbf{R}$, respectively. $\mu_1(B)$ denotes the Lozinskiĭ measure of the 2×2 matrix B_{22} with respect to the l_1 norm in $\mathbf{R}^2$. To compute $\mu_1(B_{22})$, we add the absolute

value of the off-diagonal elements to the diagonal one in each column of B_{22}, and then take the maximum of two sums. The resulting expression is as follows

$$\mu_1(B_{11}) = -(aci + \beta + 2\mu),\ \mu_1(B_{22}) = \frac{e'}{e} - \frac{i'}{i} - (aci + \rho + 2\mu + (1-k)v),$$
$$|B_{12}| = \max\left\{\frac{(pv+acs)i}{e}, \frac{(acs-(1-p-k)v)i}{e}\right\} = \frac{(pv+acs)i}{e},\ |B_{21}| = \frac{\beta e}{i}.$$

Therefore, for $t > \tilde{t}$,

$$g_1 = -(aci + \beta + 2\mu) + \frac{(pv+acs)i}{e}, \tag{51}$$

$$g_2 = \frac{e'}{e} - \frac{i'}{i} - (aci + \rho + 2\mu + (1-k)v) + \frac{\beta e}{i}, \tag{52}$$

Rewriting the last two equations of system (32), we obtain

$$\frac{e'}{e} + \beta + \mu - \frac{p\mu}{e} = \frac{(pv+acs)i}{e} \tag{53}$$

$$\frac{i'}{i} + (\rho + v + \mu) - \frac{k\mu}{i} - kv = \frac{\beta e}{i} \tag{54}$$

Substituting Eq.(53) into Eq.(51) and Eq.(54) into Eq.(52) gives

$$\mu(B) = \frac{e'}{e} - \mu + \sup\left\{\frac{-p\mu}{e} - aci, -\frac{k\mu}{i} - aci\right\} = \frac{e'}{e} - \tilde{M},$$

Substituting Eq.(53) into Eq.(51) and Eq.(54) into Eq.(52) gives

$$\mu(B) = \frac{e'}{e} - \mu + \sup\left\{\frac{-p\mu}{e} - aci, -\frac{k\mu}{i} - aci\right\} = \frac{e'}{e} - \tilde{M},$$

for $t \geq \tilde{t}$, where $\tilde{M} = min\left\{\mu + \frac{p\mu}{e} + aci, \mu + \frac{k\mu}{i} + aci\right\} = \mu + \frac{p\mu}{e} + aci.$
Since the epidemic persists in the community when $R_0 > 1$, then the solution $(s(t), e(t), i(t))$ to (32) with $(s(0), e(0), i(0)) \in D$, where D is a compact absorbing set, we have

$$\frac{1}{t}\int_0^t \mu(B)\,dt = \frac{1}{t}\int_0^{\tilde{t}} \mu(B)\,dt + \frac{1}{t}\int_{\tilde{t}}^t \mu(B)\,dt \leq \frac{1}{t}\log\frac{e(t)}{e(\tilde{t})} - \tilde{M},$$

which implies that $\tilde{q}_2 \leq -\tilde{M}/2 < 0$, which completes the proof of Theorem (3.6).

IV. Numerical simulation

In this section, numerical simulations are used to verify the analytical results for the parameter values using Matlab computer software program. Some of the parameter values have been gleaned from epidemiological literature while other parameters have been allowed to vary within the possible intervals of time. Estimate of the relevant model parameters are given in Table 1 below.

Table 1: Parameter estimates for ASF model (per day)

Symbol	Biological meaning	Value	Source
μ	Per capita natural mortality rate of domestic pigs	0.0035	[2]
v	Disease related mortality rate	0.25	[7]
Λ	Per capita recruitment rate into susceptible population	9.275	[Estimated]
ρ	Rate of progress from infective to the removed class	0.2	Estimated
α	Disease transmission rate	0.5	[5]
c	Contact rate	10	Estimated
β	Transfer rate between the exposed and the infective	0.35	[2]
p	Proportion of exposed pigs that enter into the population	0.3	[Estimated]
k	Proportion of infective pigs that enter into the population	0.02	[Estimated]

With influx of infective pigs into the population, the results in Figure 1 show that the number of susceptible pigs decreases rapidly while the number of exposed and infective pigs decreases. However, the exposed and infective populations never go to zero and thus its not possible to attain the disease-free equilibrium. Thus there is an endemic equilibrium as shown in Figure 2 when there is influx of infected pigs.

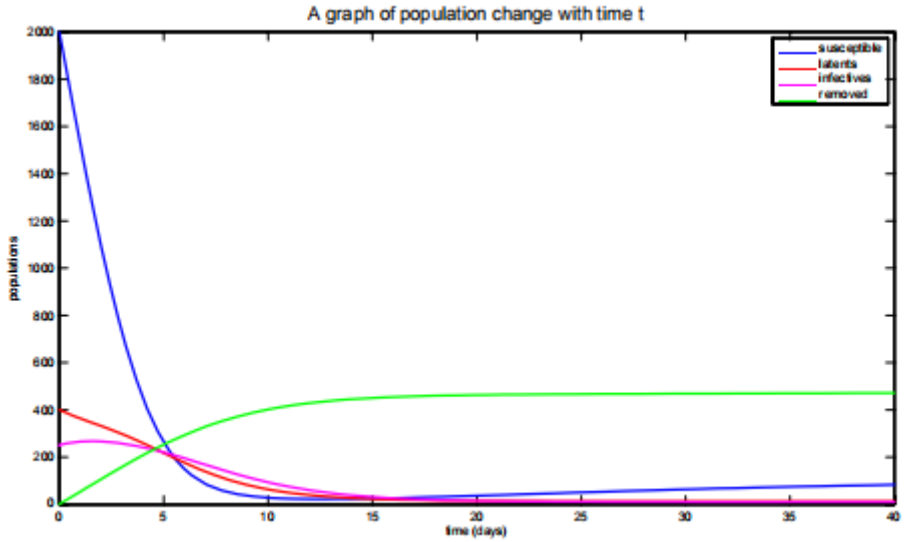

Figure 1: Population size against time with influx of infected population.

Without influx of infected pigs, the model results shown in Figure 3 indicate a sharp decrease in susceptible population in the early stages of the epidemic but later increase gradually. There is a decline in both the exposed and infec- tive population classes since all the recruits are susceptible. There is an increase in the removed population but later attains a disease-free equilibrium as pre-

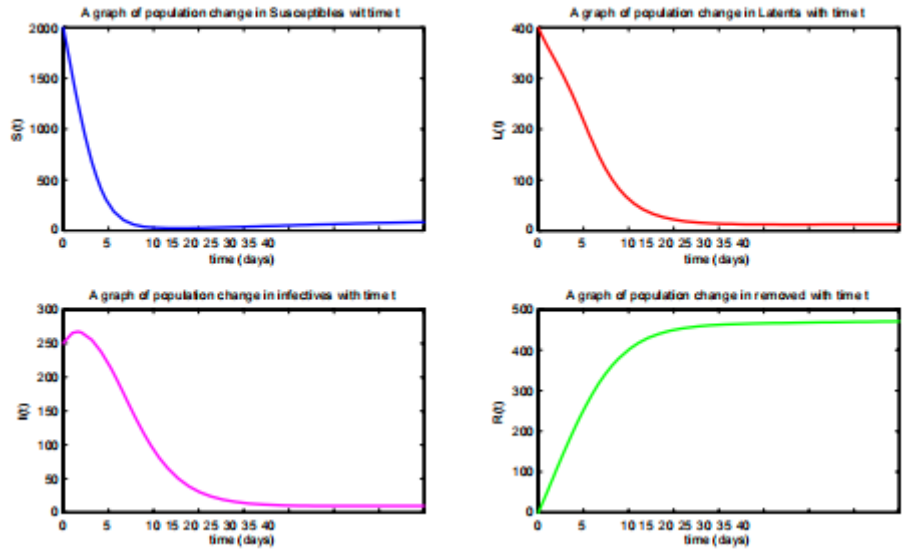

Figure 2: Population size against time for ASF with influx of infected pigs.

sented in Figure 4. In this case, it is possible to contain the disease within the population.

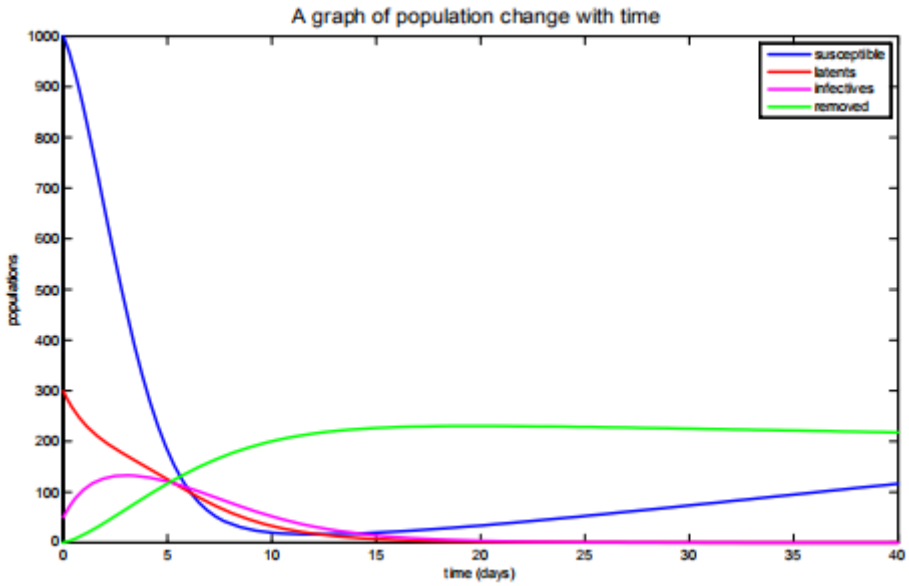

Figure 3: Population size against time for ASF with influx of only susceptible pigs.

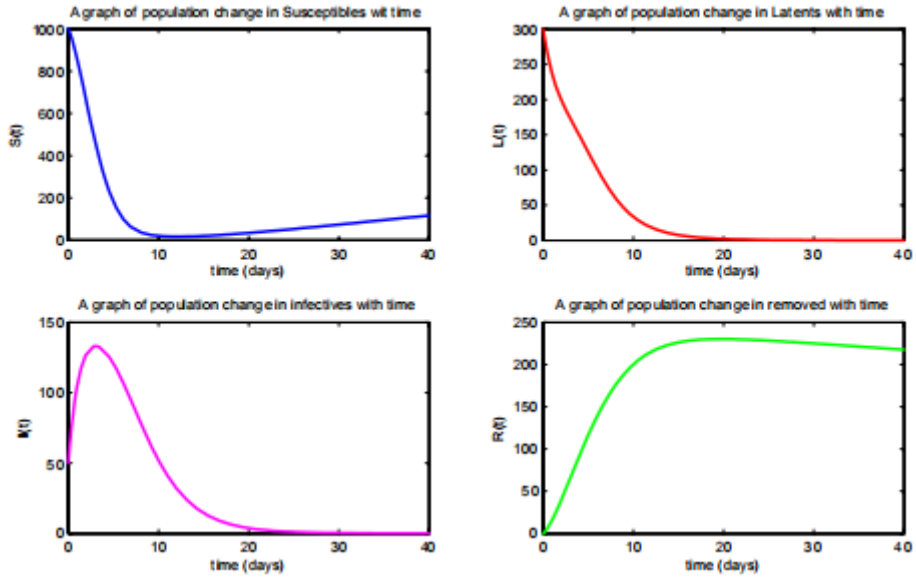

Figure 4: Population size against time for ASF with influx of only susceptible pigs.

V. Discussion of results

The model is first studied without the influx of exposed and infective domestic pigs, that is, $p = k = 0$. The model results reveal that the threshold value for which the disease can be eliminated from the community is attainable. The Equilibria of the model (E_0 and E_1) are obtained and their stability established. The global stability for the disease-free equilibrium is established by constructing a suitable Lyapunovfunction. It is found out that the disease-free equilibrium is globally asymptotically stable if $R_0 \leq 1$ and unstable if $R_0 > 1$. This means that on average if an infectious pig produces less than one new infectious individual in its entire life time as an infectious, then the infection cannot grow. The endemic equilibrium E_1 is found to be locally asymptotically stable if $R > 1$. Using the theory of compound matrices, competitive systems and periodic orbits, it is established that if $R_0 > 1$, the endemic equilibrium E_1 is globally stable and the disease persists in the community.

The model is then considered with the influx of exposed and infective individuals. It is revealed that there is no disease-free equilibrium for $p + k = 0$ and only the endemic equilibrium E_e exists, indicating that the disease persists in the community. The model results show that the disease cannot be eliminated because of the constant influx of the exposed and infective pigs. In this case, the basic reproduction number is not a good basis for the discussion for elimination of the disease unless the fraction of domestic pigs that enter into the population when exposed and infective becomes zero.

In order to reduce the disease, it is imperative that the basic reproduction number $\mathcal{R}$ should be less than unity. Public health interventions should be focused on the epidemiological parameters that constitute the basic reproduction number. There is need for early detection of any epidemic outbreak and isolation of the exposed and infective pigs. Isolation of the exposed and infective domestic pigs from the rest of the herd will lower the contact rate c and transmission rate a, and also increase the value of the removal rate of infective pigs ρ. This will reduce the value of the basic reproduction number $\mathcal{R}_0$ to less than unity, a necessary condition for disease control.

Numerical simulation reveal that with influx of exposed and infective pigs, the disease is maintained at an endemic equilibrium. The susceptible, exposed and infective populations reduce sharply as the number of the removed grows. In the absence of the influx of exposed and infective pigs into the population, the susceptible population reduces in the early stages of the epidemic but later gradually grows. The number of the exposed and infective pigs continuously reduces to zero and thus a disease-free equilibrium can be attained.

VI. Conclusion

In conclusion, the significance of influx of exposed and infective domestic pigs, and their products from one region to another needs to be recognized in the spread and control of ASF, and should be given special attention by veterinary service providers to ensure that the disease is reduced and eventually eradicated in the community. Despite the efforts to control the spread of ASF, there are still challenges for vaccine development and ASF has remained a threat worldwide. The control and prevention of the disease needs a combination of strategies. It is established that if a population has an influx of exposed and infective pigs, the population can never have a disease-free equilibrium.

The disease can be controlled based on the appropriate model parameters that make the value of the basic reproduction number R_0 less than unity. Thus, policies on preventive measures should be strengthened in such a way that influx of pigs within and across regional boundaries is restricted and ASF tests should be carried out.

Furthermore, since ASF eradication remains a challenge to most communi- ties, there is need to strengthen control strategies at hand as well as looking for more new ones since currently the incidence rate of ASF is at a high increase. Thus, from the results of the study, we recommend that:

(a) Efforts to reduce close contacts among the exposed and infective and the susceptible pigs in the population should be put in place as this would reduce the transmission and contact rates.
(b) Restrictions on the inflow of pigs and their products from affected ASF areas to non-affected ASF areas should be emphasized by policy makers.
(c) Good disease awareness and campaigns in communities should be empha- sized for early detection and management of the disease so that early im- plementation of effective control measures is put in place.
(d) Properly constructed pig pens should be encouraged to ensure that there are no contacts between exposed and infective scavenging or free-roaming pigs coming into contact with healthy domestic pigs.

Competing interests

The authors declare that there are no competing interests regarding the publi- cation of this paper.

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