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# Modelling Foot and Mouth Disease in the Context of Active Immigrants

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## Abstract

This study employs mathematical modeling to analyze the impact of active immigrants on Foot and Mouth Disease (FMD) transmission dynamics. We calculate the reproduction number ( $\mathcal{R}_0$ ) using the next-generation matrix approach. Applying the Routh-Hurwitz Criterion, we establish that the Disease-Free Equilibrium (DFE) point achieves local asymptotic stability when $\mathcal{R}_0 < 1$ and instability otherwise. Employing the quadratic Lyapunov function, we confirm the global asymptotic stability of the DFE. Numerical simulations were conducted using MATLAB software. The results indicate that infectious immigrants ( $\alpha_1$ and $\alpha_2$ ) are closely associated with reduced susceptibility in animal populations, underscoring the link between immigrants and susceptibility. Furthermore, our findings emphasize the interplay of disease introduction with population response and adaptation, particularly involving incoming infectious immigrants. Swift interventions are vital due to the limited potential for disease establishment and rapid susceptibility decline. This study offers crucial insights into the complexities of FMD transmission with active immigrants, informing effective disease management strategies.

## Keywords

Next-Generation Matrix Approach, Routh-Hurwitz, Quadratic Lyapunov Function, Active Immigrants, Reproduction Number, Analysis

## 1. Introduction

In our interconnected world, the intersection of global health concerns and human mobility has captured the attention of researchers, policymakers, and public health experts [1]. The complex patterns of infectious disease transmission,

particularly in the context of active immigrants, present intricate challenges demanding deep comprehension and innovative solutions [2]. Foot and Mouth Disease (FMD) serves as a prime example, a highly contagious viral infection bearing substantial economic consequences for livestock industries [3] [4]. Simultaneously, the mobility of active immigrants seeking improved lives abroad further complicates disease spread [5]. This introduction explores the interplay of FMD, active immigrants, and the potent role of mathematical modeling in bridging gaps and shaping effective disease management strategies.

Foot and Mouth Disease (FMD) serves as a vivid illustration of the intricate links between animal well-being, economic strength, and global disease propagation [6] [7]. Affecting cloven-hoofed animals like cattle, pigs, sheep, and goats, FMD causes mouth and hoof blisters, inflicting both substantial suffering and economic setbacks [8] [9]. Rapid action is essential due to its rapid transmission, often involving large-scale culling to contain the outbreak [10] [11]. Notably, the impact of animal mobility, especially among active immigrants, in amplifying disease transmission has gained significant research attention.

Immigrants seeking improved lives and economic prospects can unintentionally facilitate the spread of diseases due to their close community ties and cross-border mobility [12] [13]. Recognizing their role is crucial for developing effective strategies to prevent and control diseases like FMD.

Mathematical modeling provides an advanced method to understand how diseases spread with active immigrants [14]. These models quantify interactions between populations, movement, and disease, revealing outbreak possibilities, high-risk areas, and intervention effects [15] [16]. They also fill research gaps by evaluating strategies like vaccination, culling, quarantine, treatment, surveillance, and border controls.

However, despite mathematical modeling's potential, significant research gaps remain regarding the interplay between FMD and active immigrants. These gaps encompass more precise human movement data, inclusion of socio-cultural influences on transmission, and adaptable models for changing disease dynamics [17]. Addressing these requires teamwork among epidemiologists, mathematicians, social scientists, and policymakers to create comprehensive prevention and control strategies [18].

In our study of Foot and Mouth Disease, active immigrants, and mathematical modeling, we aim to enhance our grasp of disease transmission dynamics. Through addressing research gaps and using mathematical models, we intend to offer valuable insights into global health security complexities, presenting creative strategies to reduce the impact of infectious diseases on animals and humans.

## 2. Materials and Methods

### 2.1. Model Formulation

In this section, we outline the mathematical representation of the FMD com-

partmental model using a system of differential equations. The model captures FMD transmission dynamics, categorized into five compartments: Susceptible $S(t)$, Exposed $E(t)$, Asymptomatic $A(t)$, Symptomatic $I(t)$ and Recover $R(t)$. So, $N(t)=S(t)+E(t)+A(t)+I(t)+R(t)$. The corresponding flow diagram is depicted in Figure 1 and the corresponding model system of differential equations is presented in (1).

## 2.2. Model Diagram

From Figure 1, the transition between compartments can now be expressed into five non-linear differential equations defined as follows:

$$\begin{cases} \dfrac{\mathrm{d}S}{\mathrm{d}t} = \pi + \eta R - (\lambda + \mu) S, \\ \dfrac{\mathrm{d}E}{\mathrm{d}t} = \lambda S - (\sigma + \rho + \mu) E, \\ \dfrac{\mathrm{d}A}{\mathrm{d}t} = \rho E + \alpha_1 - (\kappa + \mu) A, \\ \dfrac{\mathrm{d}I}{\mathrm{d}t} = \sigma E + \alpha_2 - (\delta + \mu + \nu) I, \\ \dfrac{\mathrm{d}R}{\mathrm{d}t} = \nu I + \kappa A - (\eta + \mu) R. \end{cases} \tag{1}$$

where $\lambda = \dfrac{\beta(A+\phi I)}{N}$ is the force of infection.

- Animals are recruited at a constant rate $\pi$ and are assumed to be susceptible to infection. Animals in all compartments suffer natural mortality at rate $\mu$ and $\delta$ is a disease induced dearth rate.

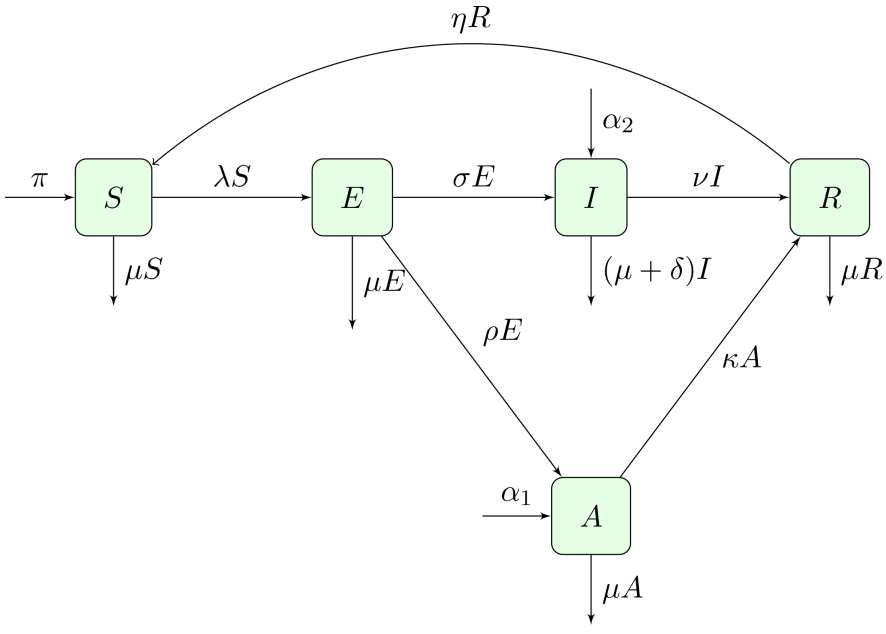


Figure 1. Model flow chart for the dynamics of Foot and Mouth disease with active immigrants.

- $\lambda$ stands for force of infection, where $\beta$ is the effective contact rate and $\phi$ is a modification parameter.
- $\sigma$ and $\rho$ represent progression rates from exposed to symptomatic and asymptomatic classes respectively.
- $\nu$ and $\kappa$ are recovery rates for symptomatic and asymptomatic animals, leading to temporary immunity.
- $\alpha_1$ and $\alpha_2$ are varying influx rates for asymptomatic and symptomatic animals from other regions, with $0 \le \alpha_1, \alpha_2 \le 1$ and $\alpha_1 > \alpha_2$.

The state variables and model parameters for the given model system are presented in Table 1 and Table 2 respectively.

Table 1. The model variables and their representations

| Variables | Description |
|---|---|
| $S$ | Susceptible population. |
| $E$ | Exposed population. |
| $A$ | Asymptomatic population. |
| $I$ | Infected population. |
| $R$ | Recovered population. |
| $N$ | Total population. |

Table 2. Parameters and their descriptions.

| Parameter | Description | Values ($day^{-1}$) | Source |
|---|---|---|---|
| $\mu$ | Natural death rate | 0.02 | [19] |
| $\delta$ | Disease-induced death rate | 0 (0.0 - 0.8) | [19] |
| $\beta$ | Effective contact rate | (0.3 - 0.99) | [20] |
| $\kappa$ | Recovery rate of asymptomatic infected | (0.45 - 0.85) | [20] |
| $\nu$ | Recovery rate of symptomatic infected | (0.45 - 0.85) | [20] |
| $\sigma$ | Exit rate from exposed class | 6 | [20] |
| $\pi$ | Recruitment rate in Susceptible cattle population | 100 | [21] |
| $\rho$ | Proportion of exposed individuals to become asymptomatic infected | (0.15 - 0.5) | [21] |
| $\phi$ | Modification parameter to reduce infectiousness of asymptomatic individual | 0.4 | [22] |
| $\eta$ | Rate at which recovery individual lose immunity | (0.01 - 0.05) | [22] |
| $\alpha_1$ | Immigration rate of asymptomatic individuals | 0.64 | Estimated |
| $\alpha_2$ | Immigration rate of symptomatic individuals | 0.32 | Estimated |

## 2.3. Model Analysis

### Non-Negativity and Boundness of Model System (1)

**Theorem 1.** For the model system (1), there exists a unique solution in $(0,\infty)$, however, the solution is always positive for all value of $t \geq 0$ and remains in $R_+^5$.

*Proof.* From the first equation of the model equations, we have $\frac{\mathrm{d}S}{\mathrm{d}t} = \pi + \eta R - (\lambda + \mu)S \geq -(\lambda + \mu)S$, integrating with initial condition $S = S(0)$ yields:

$$S(t) \geq S(0)\exp\left(-\int_0^t (\lambda + \mu)\mathrm{d}t\right) \geq 0. \tag{2}$$

From the second equation of the model equations, we have:

$$\frac{\mathrm{d}E}{\mathrm{d}t} = \lambda S - (\sigma + \rho + \mu)E,$$

integrating again with initial condition $E = E(0)$ yields:

$$E(t) \geq E(0)\exp\left(-(\sigma + \rho + \mu)t\right) \geq 0. \tag{3}$$

In a similar way, the rest of the equations of the model Equation (1) with initial conditions, $A = A(0)$, $I = I(t)$, and $R = R(0)$ gives:

$$\begin{aligned} A(t) &\geq A(0)\exp\left[-(\kappa + \mu)t\right] \geq 0, \\ I(t) &\geq I(0)\exp\left[-(\delta + \nu + \mu)t\right] \geq 0, \\ R(t) &\geq R(0)\exp\left[-(\eta + \mu)t\right] \geq 0. \end{aligned} \tag{4}$$

Hence, all the solutions of model Equation (1) are non negative for all $t \geq 0$. □

**Theorem 2.** Let $\Phi(t) = N(t)$ be the unique solution of the model system (1) for all $t \geq 0$, then the solution $\Phi(t)$ is bounded above, that is, $\Phi(t) \in \Omega$ where $\Omega$ is the feasible region defined as:

$$\Omega = \left\{N(t) \in \mathbb{R}_+^5 \;\; 0 \leq N(t) \leq C_N\right\}$$

which is interior denoted by int(Ω) and given by:

$$\mathrm{int}(\Omega) = \left\{N(t) \in \mathbb{R}_+^5 \;\; 0 \leq N(t) \leq C_N\right\}$$

*Proof.* Here, we prove that the solutions of model system (1) are bounded for all $t \geq 0$. Biologically, the lowest possible value of each states of the model system (1) is zero. Next we determine the upper-bound of states. Given that, $N(t) = S(t) + E(t) + A(t) + I(t) + R(t)$:

$$\Rightarrow \frac{\mathrm{d}N(t)}{\mathrm{d}t} = \frac{\mathrm{d}S(t)}{\mathrm{d}t} + \frac{\mathrm{d}E(t)}{\mathrm{d}t} + \frac{\mathrm{d}A(t)}{\mathrm{d}t} + \frac{\mathrm{d}I(t)}{\mathrm{d}t} + \frac{\mathrm{d}R(t)}{\mathrm{d}t}$$

substitute the values of differential equations from the model system (1), simplification gives:

$$\frac{\mathrm{d}N(t)}{\mathrm{d}t} = \pi + \alpha_1 + \alpha_2 - \mu S - \mu E - \mu A - \mu I - \mu R - \delta I,$$

$$\frac{\mathrm{d}N(t)}{\mathrm{d}t} = \pi + \alpha_1 + \alpha_2 - \delta I - \mu N \le \pi + \alpha_1 + \alpha_2 - \mu N.$$

$$\frac{\mathrm{d}N(t)}{\mathrm{d}t} + \mu N \le \pi + \alpha_1 + \alpha_2. \tag{5}$$

By integration gives:

$$N(t) \le \frac{\pi + \alpha_1 + \alpha_2}{\mu} + \left( N(0) - \frac{\pi}{\mu} \right) \mathrm{e}^{-\mu t} \tag{6}$$

Since the total population $N(t)$ is positive for all $t \ge 0$. It is well defined that:

$$\limsup_{t \to \infty} N(t) \le \frac{\pi + \alpha_1 + \alpha_2}{\mu}$$

Therefore,

$$0 \le N(t) \le \frac{\pi + \alpha_1 + \alpha_2}{\mu}, \text{ for all } t > 0.$$

This proves that all solutions of the FMD model with initial conditions in Ω remain in Ω for all $t > 0$.□

## 2.4. Disease-Free Equilibrium Point (DFE)

In this section, we present the disease-free equilibrium point of the model system (1). We first set the right-hand side of the model system (1) equal to zero as follows:

$$\begin{cases} \pi + \eta R - (\lambda + \mu) S = 0, \\ \lambda S - (\sigma + \rho + \mu) E = 0, \\ \rho E + \alpha_1 - (\kappa + \mu) A = 0, \\ \sigma E + \alpha_2 - (\delta + \mu + \nu) I = 0, \\ \nu I + \kappa A - (\eta + \mu) R = 0. \end{cases} \tag{7}$$

In the absence of disease in the population, we have $E(t) = I(t) = A(t) = R(t) = 0$ and the model (7) admits a trivial equilibrium point commonly known as the disease-free equilibrium point. Thus, the model (7) has a disease-free equilibrium $E_0$ given by:

$$E_0 = \left( S^0, E^0, A^0, I^0, R^0 \right) = \left( \frac{\pi}{\mu}, 0, 0, 0, 0 \right). \tag{8}$$

The disease-free equilibrium point in disease modeling is important as it is used when computing the basic reproduction number $\mathcal{R}_0$.

## 2.5. Basic Reproduction Number ( $\mathcal{R}_0$ )

Following the next generation matrix approach as used by [23] [24] [25], the non-negative matrix $\mathcal{F}$ that denotes the generation of new infection in the population and non-singular matrix $\mathcal{V}$ that denotes the disease transfer of infected compartments evaluated at the disease-free $E_0$ are given as follows:

$$\mathcal{F}=\begin{pmatrix} 0 & \frac{\beta\pi}{\mu} & \frac{\beta\phi\pi}{\mu} \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{pmatrix}, \mathcal{V}=\begin{pmatrix} \sigma+\rho+\mu & 0 & 0 \\ -\rho & \kappa+\mu & 0 \\ -\sigma & 0 & \delta+\mu+\nu \end{pmatrix} \tag{9}$$

Thus, using the next-generation matrix approach in (9), it can easily be verified that the model system (1) has basic reproduction number $\mathcal{R}_0$ given by:

$$\mathcal{R}_0=\frac{\beta\left(\delta\rho+\kappa\sigma\phi+\mu\rho+\mu\sigma\phi+\nu\rho\right)}{\left(\kappa+\mu\right)\left(\delta+\mu+\nu\right)\left(\mu+\rho+\sigma\right)}. \tag{10}$$

The threshold value $\mathcal{R}_0$ is an important epidemiological quantity in disease modeling. It determines the strength of transmission potential of a disease to invade the community. It has been scientifically demonstrated that when $\mathcal{R}_0<1$ the disease dies out in the population. However, the disease persist when $\mathcal{R}_0>1$.

## 2.6. Local Stability of Disease-Free Equilibrium (DFE)

Here, we establish the local stability of disease-free equilibrium using trace and determinant method and Routh Hurwitz's stability criterion.

**Theorem 3.** The DFE of the model system (1) at DFE ($E_0$) is locally asymptotically stable if $\mathcal{R}_0<1$ and unstable $\mathcal{R}_0>1$.

*Proof.* In proving this theorem, the Jacobian matrix of model system (1) at DFE ($E_0$) is given as:

$$J\left(E_0\right)=\begin{pmatrix} -\mu & 0 & -\beta & -\beta\phi & \eta \\ 0 & -\left(\sigma+\rho+\mu\right) & \beta & \beta\phi & 0 \\ 0 & \rho & -\left(\kappa+\mu\right) & 0 & 0 \\ 0 & \sigma & 0 & -\left(\delta+\nu+\mu\right) & 0 \\ 0 & 0 & \kappa & \nu & -\left(\eta+\mu\right) \end{pmatrix}.$$

We have $-\mu$, $-\left(\eta+\mu\right)$ negative, while the other three eigenvalues can be computed through the following cubic equations. The remaining eigenvalues are obtained from:

$$G=\begin{bmatrix} -a_1 & \beta & \beta\phi \\ \rho & -a_2 & 0 \\ \sigma & 0 & -a_3 \end{bmatrix}$$

where $a_1=\mu+\rho+\sigma$, $a_2=\kappa+\mu$ and $a_3=\delta+\mu+\nu$. The characteristic polynomial for matrix $G$ is given by $G\left(\lambda\right)=d_3\lambda^3+d_2\lambda^2+d_1\lambda^1+d_0$, Whereby; $d_3=1$, $d_2=a_1+a_2+a_3$, $d_1=a_1a_2+a_1a_3+a_2a_3-\beta\rho-\beta\sigma\phi$, $d_0=-a_3\beta\rho-a_2\beta\sigma\phi+a_1a_3a_2$.

Here, we used Routh-Hurwitz criterion, the Routh-Hurwitz Criterion is a mathematical tool used to assess the stability of equilibrium points in a dynamical system, particularly in the context of linear time-invariant systems. In the context of epidemiological models, if all the coefficients in the first column of the Routh array have the same sign, and that sign is positive, then the system is

locally asymptotically stable. Conversely, if there is at least one sign change in the first column, the system is locally unstable. A polynomial $G(\lambda)$ has roots with a negative real part if it satisfies the following necessary and sufficient conditions:

1) $d_3 > 0$, 2) $d_2 > 0$, 3) $d_1 d_2 - d_0 d_3 > 0$ and 4) $d_0 > 0$.

Then, $d_3 = 1 > 0$, $d_3 > 0$. Again, $d_2 = a_1 + a_2 + a_3 > 0$, $d_2 > 0$.

It can be shown that:

$$\begin{aligned} d_1 d_2 - d_0 d_3 = {} & (\delta + \kappa + 3\mu + \nu + \rho + \sigma)(\beta\rho + \beta\sigma\phi + (\kappa + \mu)(\delta + \mu + \nu) \\ & + (\delta + \mu + \nu)(\mu + \rho + \sigma) + (\kappa + \mu)(\mu + \rho + \sigma)) \\ & + \beta\rho(\delta + \mu + \nu) + \beta\sigma\phi(\kappa + \mu) + (\kappa + \mu)(\delta + \mu + \nu)(\mu + \rho + \sigma) \\ & > 0. \end{aligned}$$

The value of $d_0$ is given by:

$$d_0 = (\mu + \rho + \sigma)(\kappa + \mu)(\delta + \mu + \nu)(1 - \mathcal{R}_0).$$

Thus, the condition $d_0 > 0$ holds if $\mathcal{R}_0 < 1$. Therefore, it implies that DFE ($E_0$) is locally asymptotically stable when $\mathcal{R}_0 < 1$ and unstable otherwise.□

## 2.7. Global Stability of DFE

Analysis of the global stability of the disease-free equilibrium solution of the model system (1) is done by using the approach by Castillo-Chavez *et al.* (2002). The model system can be written as:

$$\begin{cases} \dfrac{\mathrm{d}U}{\mathrm{d}t} = F(U, I) \\ \dfrac{\mathrm{d}I}{\mathrm{d}t} = B(U, I), B(U, 0) = 0 \end{cases} \tag{11}$$

From Equation (11), $U = (S, R)$ and $I = (E, A, I)$ indicates the number of uninfected and infected cattle respectively. $F$ and $B$ represent the functions of infected and uninfected individuals respectively. The disease-free equilibrium solutions is denoted by $E_0 = (U^*, 0)$:

$$E^0 = (U^*, 0) = \left(\frac{\pi}{\mu}, 0\right).$$

For DFE to be globally asymptotically stable, the following conditions must be satisfied simultaneously:

$$\begin{cases} 1. \quad \text{For } \dfrac{\mathrm{d}U}{\mathrm{d}t} = F(U, 0), U^* \text{ is globally asymptotically stable (g.a.s)}, & (12) \\ 2. \quad B(U, I) = AI - \hat{B}(U, I), \hat{B}(U, I) \geq 0 \text{ For } (U, I) \in \Omega. & (13) \end{cases}$$

For which, the Jacobian matrix $A$ is a Metzler matrix and Ω is the region where the model system yields a biologically meaningful. Now, if the model system (11) satisfies the above conditions, then Theorem 4 holds.

**Theorem 4.** For $\theta \in (0, 1]$, the disease-free equilibrium point $E^0$ of model system (7) is globally asymptotically stable whenever $\mathcal{R}_0 < 1$.

*Proof.* Condition 1 in (12) is proved as follows:

$$\frac{\mathrm{d}U}{\mathrm{d}t}=\begin{pmatrix}\pi+\eta R-\left(\dfrac{\beta(A+\phi I)}{N}\right)S-\mu S\\ \nu I+\kappa A-(\eta+\mu)R\end{pmatrix}$$

At DFE, $E_0=(U^*,0)$ gives:

$$\frac{\mathrm{d}U}{\mathrm{d}t}=F(U,0)=\begin{pmatrix}\pi-\mu S\\ 0\end{pmatrix}.$$

$$S(t)=\frac{\pi}{\mu}+\left(S(0)-\frac{\pi}{\mu}\right)\mathrm{e}^{-\mu t}$$

It is clear that the solutions $S(t)\to\dfrac{\pi}{\mu}$, and $R(t)=0$ as $t\to\infty$ regardless of the value of $S(0)$ and $R(t)$. Implying that $E_0$ is global asymptotically stable.

Condition 2 in (12) is proved as follows:

$$B(U,I)=\begin{pmatrix}\lambda S-(\sigma+\rho+\mu)E\\ \rho E+\alpha_1-(\kappa+\mu)A\\ \sigma E+\alpha_2-(\delta+\mu+\nu)I\end{pmatrix}$$

At DFE $E^0=(U^*,0)$ gives:

$$A=\begin{pmatrix}-(\sigma+\rho+\mu) & \dfrac{\beta S^0}{N^0} & \dfrac{\beta\phi S^0}{N^0}\\ \rho & -(\mu+\kappa) & 0\\ \sigma & 0 & -(\delta+\nu+\mu)\end{pmatrix}.$$

It is clear that matrix $A$ is Metzler matrix. Then,

$$\hat{B}(U,I)=AI-B(U,I)$$

$$\hat{B}(U,I)=\begin{pmatrix}-(\sigma+\rho+\mu) & \dfrac{\beta S^0}{N^0} & \dfrac{\beta\phi S^0}{N^0}\\ \rho & -(\mu+\kappa) & 0\\ \sigma & 0 & -(\delta+\nu+\mu)\end{pmatrix}\begin{pmatrix}E\\ A\\ I\end{pmatrix}-\begin{pmatrix}\lambda S-(\sigma+\rho+\mu)E\\ \rho E+\alpha_1-(\kappa+\mu)A\\ \sigma E+\alpha_2-(\delta+\mu+\nu)I\end{pmatrix}$$

$$\Rightarrow$$

$$\hat{B}(U,I)=\begin{pmatrix}\beta(A+\phi I)\left(\dfrac{S^0}{N^0}-\dfrac{S}{N}\right)\\ 0\\ 0\end{pmatrix}$$

Since $S^0>S$, it is clear that $\hat{B}(U,I)\geq 0$.

Hence, $E^0$ is globally asymptotically stable.□

## 2.8. Endemic Equilibrium Point

Endemic equilibrium point denoted by $E^*$ is obtained by solving for $\left(S^*, V^*, E^*, A^*, I^*, R^*\right)$ from the model system (14):

$$\begin{cases} S^* = \dfrac{\pi + \eta R^*}{\lambda^* + \mu}, \\ E^* = \dfrac{\lambda^* S^*}{\sigma + \rho + \mu}, \\ A^* = \dfrac{\alpha_1 + \rho E^*}{\kappa + \mu}, \\ I^* = \dfrac{\sigma E^* + \alpha_2}{\delta + \mu + \nu}, \\ R^* = \dfrac{\nu I^* + \kappa A^*}{\eta + \mu}. \end{cases} \tag{14}$$

whereby

$$\lambda^* = \frac{\beta\left(A^* + \phi I^*\right)}{N}$$

### 2.8.1. Bifurcation Analysis.

Bifurcation analysis was done by using Center Manifold theory, let $x_1 = S$, $x_2 = E$, $x_3 = A$, $x_4 = I$, $x_5 = R$ and $x_5 = R$, by using vector notation $x = \left(x_1, x_2, x_3, x_4, x_5\right)^{\mathrm{T}}$ (where T denotes the transpose). Then, the model system (1) can be written in the form $\dfrac{\mathrm{d}x}{\mathrm{d}t} = f\left(x\right)$, with $f\left(x\right) = \left(f_1\left(x\right), f_2\left(x\right), f_3\left(x\right), f_4\left(x\right), f_5\left(x\right), f_6\left(x\right)\right)$. Then,

$$\begin{cases} \dfrac{\mathrm{d}x_1}{\mathrm{d}t} = f\left(x_1\right) = \pi + \eta x_5 - \left(\beta\left(\dfrac{x_3 - \phi x_4}{X}\right) + \mu\right)x_1, \\ \dfrac{\mathrm{d}x_2}{\mathrm{d}t} = f\left(x_2\right) = \beta\left(\dfrac{x_3 - \phi x_4}{X}\right)x_1 - \left(\sigma + \rho + \mu\right)x_2, \\ \dfrac{\mathrm{d}x_3}{\mathrm{d}t} = f\left(x_3\right) = \rho x_2 + \alpha_1 - \left(\kappa + \mu\right)x_3, \\ \dfrac{\mathrm{d}x_4}{\mathrm{d}t} = f\left(x_4\right) = \sigma x_2 + \alpha_2 - \left(\delta + \mu + \nu\right)x_4, \\ \dfrac{\mathrm{d}x_5}{\mathrm{d}t} = f\left(x_5\right) = \nu x_4 + \kappa x_3 - \left(\eta + \mu\right)x_5. \end{cases} \tag{15}$$

Choose $\beta$ as a bifurcation parameter. Consider when $\mathcal{R}_0 = 1$, then solving for $\beta^*$ using $\beta = \beta^*$ gives:

$$\beta^* = \frac{\left(\kappa + \mu\right)\left(\delta + \mu + \nu\right)\left(\mu + \rho + \sigma\right)}{\delta\rho + \kappa\sigma\phi + \mu\rho + \mu\sigma\phi + \nu\rho}.$$

The Jacobian matrix at DFE ($E_0$) and bifurcation parameter $\beta^*$ is given by:

$$J_\beta^* = \begin{pmatrix} -\mu & 0 & -a_1 & -a_2 & \eta \\ 0 & -a_3 & a_1 & a_2 & 0 \\ 0 & \rho & -a_4 & 0 & 0 \\ 0 & \sigma & 0 & -a_5 & 0 \\ 0 & 0 & \kappa & \nu & -a_6 \end{pmatrix}.$$

where $a_1 = \beta^*$, $a_2 = \beta^*\phi$, $a_3 = \sigma + \rho + \mu$, $a_4 = \kappa + \mu$, $a_5 = \delta + \nu + \mu$ and $a_6 = \eta + \mu$.

The right eigenvector associated with the zero eigenvalue of the Jacobian matrix $J_\beta^*$, $w = \left[w_1, w_2, w_3, w_4, w_5, w_6\right]^{\mathrm{T}}$ is defined such that $J\left(E_0\right)\cdot w = 0$ at $\beta = \beta^*$ given by; $w_1 = -\dfrac{a_6\sigma\left(a_1a_5 + a_2a_5\right) + \left(\eta\rho a_5 + \sigma a_4\right)}{a_4a_5a_6}w_2$, $w_2 = w_2$, $w_3 = \dfrac{\rho}{a_4}w_2$, $w_4 = \dfrac{\sigma}{a_5}w_2$, and $w_5 = \dfrac{a_4\sigma + \rho a_5}{a_4a_5a_6}w_2$.

The left eigenvector $v = \left[v_1, v_2, v_3, v_4, v_5, v_6\right]$ such that $v\cdot J_\beta^* = 0$ yields:

$$v_1 = 0, v_2 = v_2, v_3 = \frac{a_1}{a_5}v_2, v_4 = \frac{a_2}{a_5}v_2 \text{ and } v_5 = 0.$$

**Theorem 5.** Consider the general system of ordinary differential equations with a parameter $\beta$ such that:

$$\frac{\mathrm{d}x}{\mathrm{d}t} = f\left(x,\beta\right),\ f:\mathbb{R}^n\times\mathbb{R}\to\mathbb{R}^n \text{ and } f\in\mathbb{C}^2\left(\mathbb{R}^n\times\mathbb{R}\right), \tag{16}$$

such that $f\left(0,\beta\right)\equiv 0$, whereby 0 is an equilibrium point of the system with the following conditions:

1) $M = B_x f\left(0,0\right) = \left(\dfrac{\partial f_i}{\partial x_j}\left(0,0\right)\right)$ is the linearization matrix of the system (3.33) around the equilibrium 0 with $\beta$ evaluated at 0.

2) Zero is a simple eigenvalue of *M*, and the rest eigenvalue of *M* have negative real parts.

3) Matrix *M* has a right eigenvector *w* and a left eigenvector *v* corresponding to the zero eigenvalue.

Let $f_k$ be the $K^{\text{th}}$ component of *f* and

$$\begin{aligned} a &= \sum_{i,j,k=1}^{n} v_k w_i w_j \frac{\partial^2 f_k}{\partial x_i \partial x_j}\left(0,0\right), \\ b &= \sum_{i,k=1}^{n} v_k w_i \frac{\partial^2 f_k}{\partial x_i \partial \beta}\left(0,0\right). \end{aligned} \tag{17}$$

The local dynamics of (3.33) around 0 are totally determined by *a* and *b*.

1) $a > 0$, $b > 0$. When $\beta < 0$ with $\left|\beta\right| \ll 1$, 0 is locally asymptotically stable, and there exists a positive unstable equilibrium; when $0 < \beta \ll 1$, 0 is unstable and there exists a negative and locally asymptotically stable equilibrium.

2) $a < 0$, $b < 0$. When $\beta < 0$ with $\left|\beta\right| \ll 1$, 0 is unstable; when $0 < \beta \ll 1$, 0 is locally asymptotically stable, and there exists a positive unstable equilibrium.

3) $a > 0$, $b < 0$. When $\beta < 0$ with $\left|\beta\right| \ll 1$, 0 is unstable, and there exists a

locally asymptotically stable negative equilibrium; when $0<\beta\ll 1$, 0 is stable, and a positive unstable equilibrium appears.

4) $a<0$, $b>0$. When $\beta$ changes from negative to positive, 0 changes its stability from stable to unstable. Correspondingly a negative unstable equilibrium becomes positive and locally asymptotically stable.

### 2.8.2. Bifurcation Coefficients

It is clear that $v_1=v_5=0$ so, the consideration is on $v_2, v_3$ and $v_4$.

Hence, the second order partial derivatives at disease-free equilibrium and at $\beta^*$ are given by:

$$\frac{\partial^2 f_2}{\partial x_2 \partial x_3}\left(E_0,\beta^*\right)=\frac{\partial^2 f_3}{\partial x_3 \partial x_2}\left(E_0,\beta^*\right)=-\frac{\beta^*\mu}{\pi},$$

$$\frac{\partial^2 f_2}{\partial x_2 \partial x_4}\left(E_0,\beta^*\right)=\frac{\partial^2 f_4}{\partial x_4 \partial x_2}\left(E_0,\beta^*\right)=\frac{\beta^*\mu\phi}{\pi},$$

$$\frac{\partial^2 f_3}{\partial x_3 \partial x_4}\left(E_0,\beta^*\right)=\frac{\partial^2 f_4}{\partial x_4 \partial x_3}\left(E_0,\beta^*\right)=\frac{\beta^*\mu\phi}{\pi}-\frac{\beta^*\mu}{\pi},$$

$$\frac{\partial^2 f_3}{\partial x_3 \partial x_5}\left(E_0,\beta^*\right)=\frac{\partial^2 f_5}{\partial x_5 \partial x_3}\left(E_0,\beta^*\right)=-\frac{\beta^*\mu}{\pi},$$

$$\frac{\partial^2 f_3}{\partial x_3 \partial x_3}\left(E_0,\beta^*\right)=-\frac{2\beta^*\mu}{\pi},\quad \frac{\partial^2 f_4}{\partial x_4 \partial x_4}\left(E_0,\beta^*\right)=\frac{2\beta^*\mu\phi}{\pi},$$

$$\frac{\partial^2 f_2}{\partial x_3 \partial \beta^*}\left(E_0,\beta^*\right)=1,\quad \frac{\partial^2 f_2}{\partial x_4 \partial \beta^*}\left(E_0,\beta^*\right)=\phi.$$

Then,

$$\begin{aligned}a=&-\frac{\mu\beta^*}{\pi}\Big[v_2w_2\left(w_4\phi+w_3\right)+v_3w_3\left(w_4\phi+w_2+2w_3+w_4+w_5\right)\\&+w_4\left(v_5w_5\phi+v_4\left(w_2\phi+w_3\left(\phi+1\right)+w_4\phi+w_5\phi-w_4\right)\right)\Big]\\<&0.\end{aligned}$$

Hence, $a<0$.

Also, for the sign of $b$ given as:

$$b=v_2w_3\frac{\partial^2 f_2}{\partial x_3\partial\beta^*}+v_2w_4\frac{\partial^2 f_2}{\partial x_4\partial\beta^*},$$

$$b=v_2w_2\left[\frac{\rho}{a_4}+\frac{\sigma\phi}{a_5}\right]>0. \tag{18}$$

Therefore, $a<0$ and $b>0$, hence the model system exhibit a forward bifurcation.

### 2.8.3. Global Stability of Endemic Equilibrium Point

To prove the global stability of EEP, we used quadratic Lyapunov function. The endemic equilibrium point is said to be globally asymptotically stable if its derivative is less than 0.

**Theorem 6.** The FMD has a unique endemic equilibrium point $E^*$ for

model system (1) that is globally asymptotically stable if $\mathcal{R}_0 > 1$ otherwise unstable.

*Proof.* Now, consider the quadratic Lyapunov function,

$$V(x_1, \cdots, x_n) = \sum_{i=1}^{n} \frac{1}{2}\left[x_i - x_i^*\right]^2,$$

where $x_i$ is the cattle population and $x_i^*$ is the endemic equilibrium point. Then,

$$V = \frac{1}{2}\left[\left(S - S^*\right) + \left(E - E^*\right) + \left(A - A^*\right) + \left(I - I^*\right) + \left(R - R^*\right)\right]^2. \tag{19}$$

Clearly, $V : \mathbb{R}_+^6 \to \mathbb{R}$ is a continuous and differentiable function. We have:

$$\frac{\mathrm{d}V}{\mathrm{d}t} = \left[\left(S - S^*\right) + \left(E - E^*\right) + \left(A - A^*\right) + \left(I - I^*\right) + \left(R - R^*\right)\right]\frac{\mathrm{d}}{\mathrm{d}t}\left[S + E + A + I + R\right].$$

$$\frac{\mathrm{d}V}{\mathrm{d}t} = \left[S + E + A + I + R - \left(S^* + E^* + A^* + I^* + R^*\right)\right]\frac{\mathrm{d}}{\mathrm{d}t}\left[S + E + A + I + R\right].$$

But,

$$\frac{\mathrm{d}}{\mathrm{d}t}\left(S + E + A + I + R\right) = \pi + \alpha_1 + \alpha_2 - \mu N(t) - \delta I. \tag{20}$$

And

$$\pi + \alpha_1 + \alpha_2 - \mu N^* - \delta I^* = 0 \Rightarrow \pi + \alpha_1 + \alpha_2 - \delta I^* - \mu\left[S^* + E^* + A^* + I^* + R^*\right] = 0$$

$$S^* + E^* + A^* + I^* + R^* = \frac{\pi + \alpha_1 + \alpha_2 - \delta I^*}{\mu} \tag{21}$$

Substitute (20) and (21) into $\frac{\mathrm{d}V}{\mathrm{d}t}$ gives:

$$\frac{\mathrm{d}V}{\mathrm{d}t} = \left[N(t) - \frac{\pi + \alpha_1 + \alpha_2 - \delta I^*}{\mu}\right]\left[\pi + \alpha_1 + \alpha_2 - \delta I - \mu N(t)\right],$$

$$\frac{\mathrm{d}V}{\mathrm{d}t} = \left[N(t) - \frac{\pi + \alpha_1 + \alpha_2 - \delta I^*}{\mu}\right]\left[-\mu\left(N(t) - \frac{\pi + \alpha_1 + \alpha_2 - \delta I}{\mu}\right)\right],$$

$$\frac{\mathrm{d}V}{\mathrm{d}t} = -\mu\left[N(t) - \frac{\pi + \alpha_1 + \alpha_2}{\mu} + \frac{\delta I^*}{\mu}\right]\left[N(t) - \frac{\pi + \alpha_1 + \alpha_2}{\mu} + \frac{\delta I}{\mu}\right],$$

$$\Rightarrow \frac{\mathrm{d}V}{\mathrm{d}t} = -\mu\left[N(t) - \frac{\pi + \alpha_1 + \alpha_2}{\mu} + \frac{\delta I^*}{\mu}\right]\left[N(t) - \frac{\pi + \alpha_1 + \alpha_2}{\mu} + \frac{\delta I}{\mu}\right]$$

$$\frac{\mathrm{d}V}{\mathrm{d}t} \le -\mu\left[N(t) - \frac{\pi + \alpha_1 + \alpha_2}{\mu}\right]\left[N(t) - \frac{\pi + \alpha_1 + \alpha_2}{\mu}\right],$$

That is,

$$\frac{\mathrm{d}V}{\mathrm{d}t} \le -\mu\left[N(t) - \frac{\pi + \alpha_1 + \alpha_2}{\mu}\right]^2 < 0. \tag{22}$$

Therefore, it is clear that $\frac{\mathrm{d}V}{\mathrm{d}t} < 0$ which implies that the Endemic Equilibrium Point ( $E^*$ ) is globally asymptotically stable.□

## 2.9. Numerical Simulations of the Model

This section uses accessible parameter values from the literature as well as estimated ones to simulate the model system (1) numerically. Unless otherwise specified, simulation results will be based on parameter values from Table 2. The fourth-order Runge-Kutta method (RK4) is used to simulate the model system (1) in MATLAB software. This combination ensures accurate representation of the FMD transmission model dynamics. Our simulation aims to fill knowledge gaps about Foot and Mouth Disease and illustrate its spread in a population with active immigrants. The chosen initial conditions are: $S=90$, $E=5$, $A=2$, $I=3$, and $R=0$. These values are deliberately chosen to produce a desired model behavior.

Figure 2 describes the changes in animal population over time. The trend in the graph implies that active immigrants play a crucial role in shaping the dynamics of an FMD outbreak. The rapid decrease in susceptibility, combined with gradual changes in other compartments, suggests a limited disease establishment potential. This emphasizes the need for prompt interventions and highlights the complex interplay of disease spread within the context of active immigrants.

## 2.10. Results and Discussion

Our results can be summarized as follows: Figures 3-7, Figure 9 and Figure 10 demonstrate that, with an increase in infectious immigrants ($\alpha_1$ and $\alpha_2$); the susceptible animal population gradually decreases to a steady state. This suggests

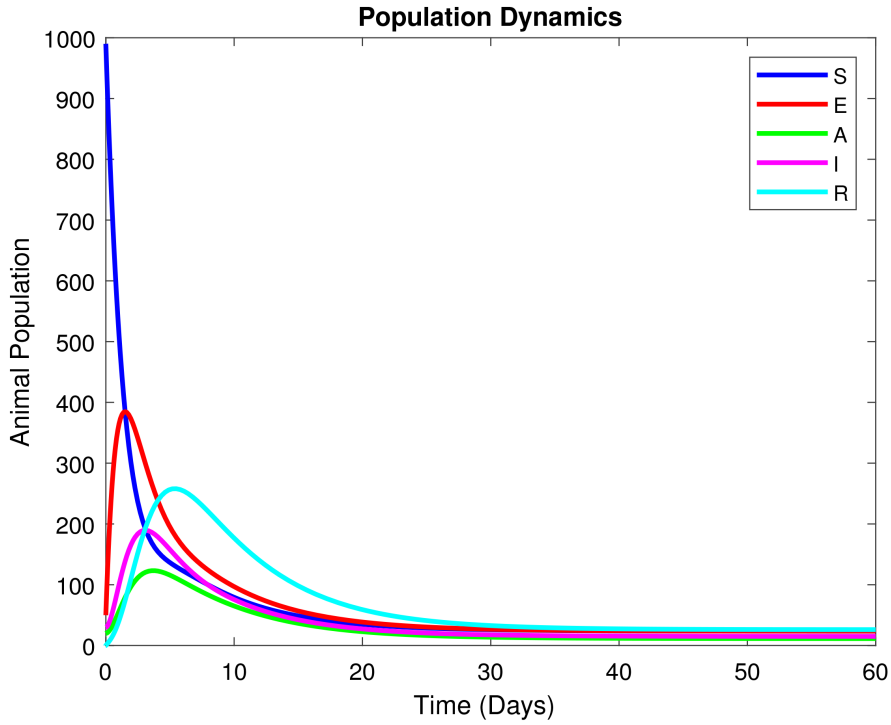


Figure 2. Evolution of animal population with time.

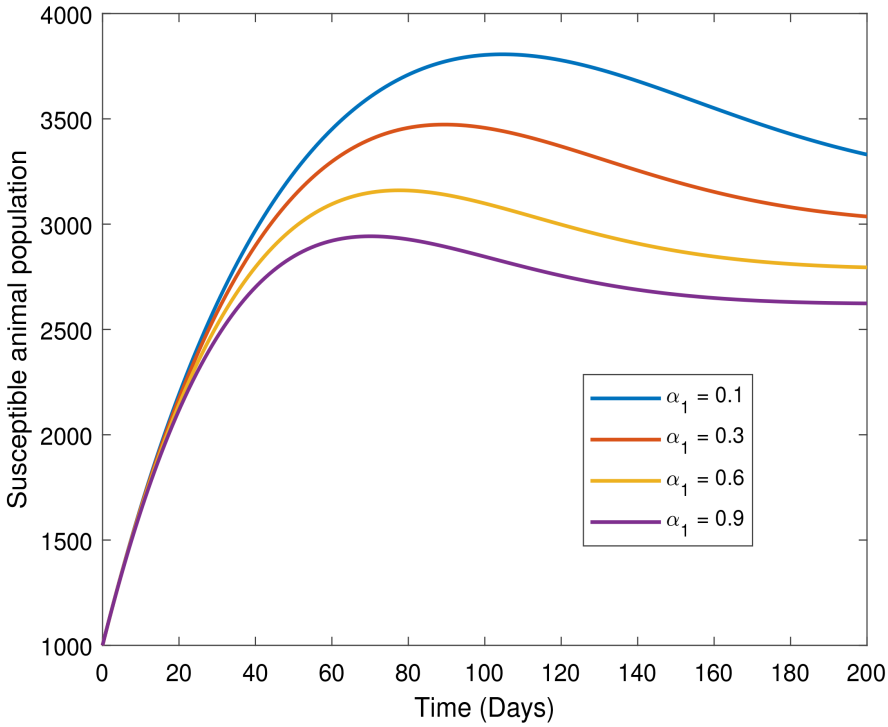


**Figure 3.** Effect of $\alpha_1$ on susceptible animal population.

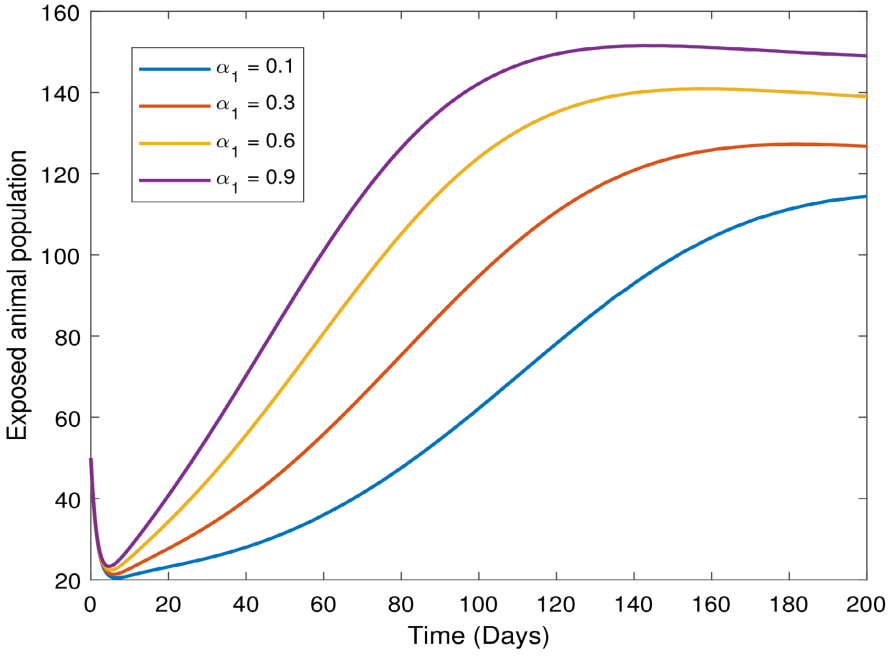


**Figure 4.** Effect of $\alpha_1$ on exposed animal population.

a connection between immigrant influx and susceptible population decline. Infectious immigrants notably contribute to reducing susceptibility through disease transmission. Subsequent gradual decrease to its steady state shows ongoing disease dynamics and the establishment of a stable endemic equilibrium.

Figure 3 shows an initial decline within 10 days followed by a gradual rise with

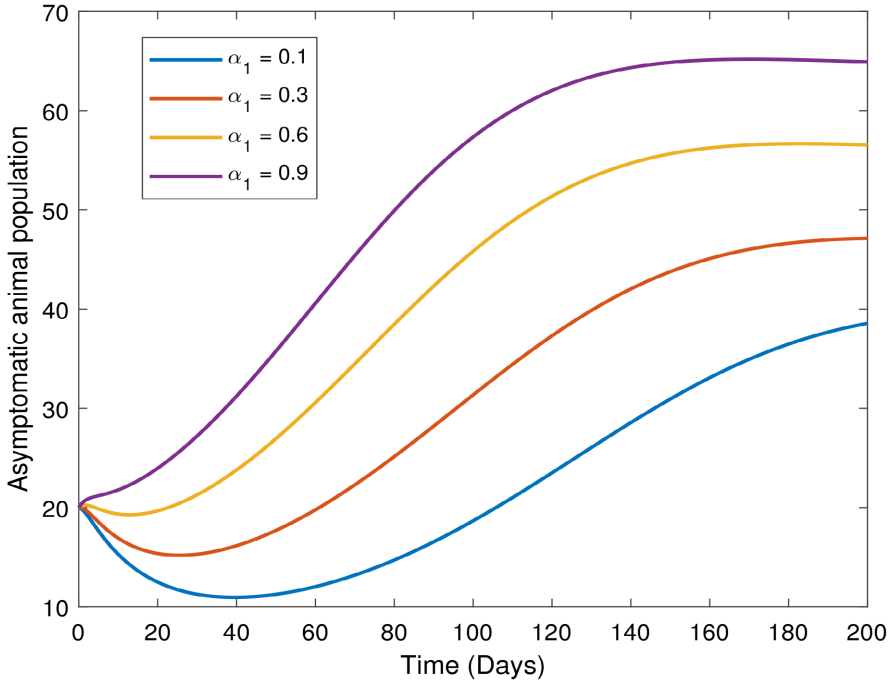


**Figure 5.** Effect of $\alpha_1$ on asymptomatic animal population.

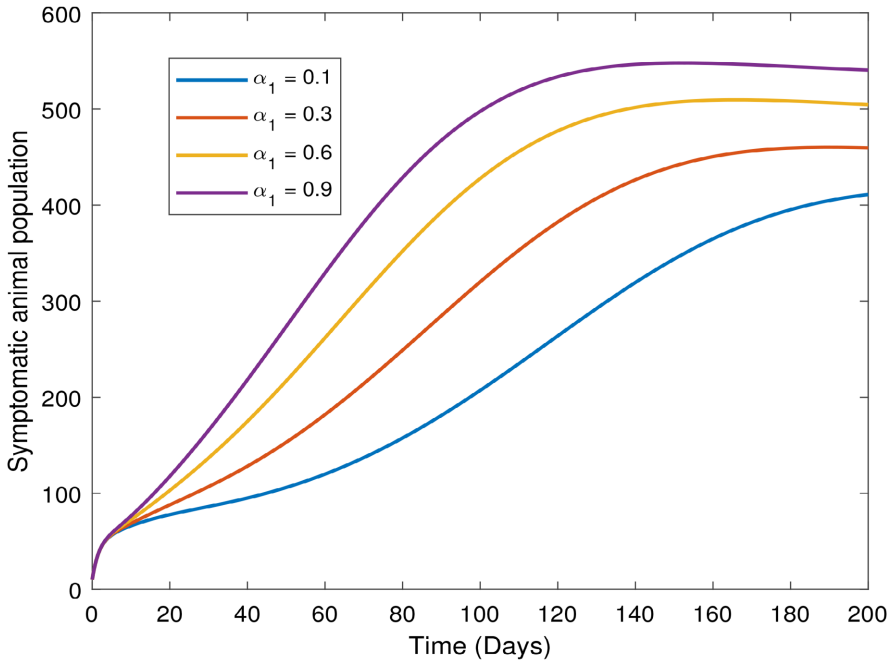


**Figure 6.** Effect of $\alpha_1$ on symptomatic animal population.

an increase in ( $\alpha_1$ ); this might reflect complex exposure-immigrant dynamics. The fluctuations possibly reveal interactions among infection introduction, host responses, and adaptation processes. Figure 7 is characterized by decreasing trend, followed by a sharp increase and gradual decrease before reaching the steady state, this pattern is shaped by the arrival of infectious immigrants ( $\alpha_2$ ), showcasing the dynamic interplay between disease introduction, population response, and

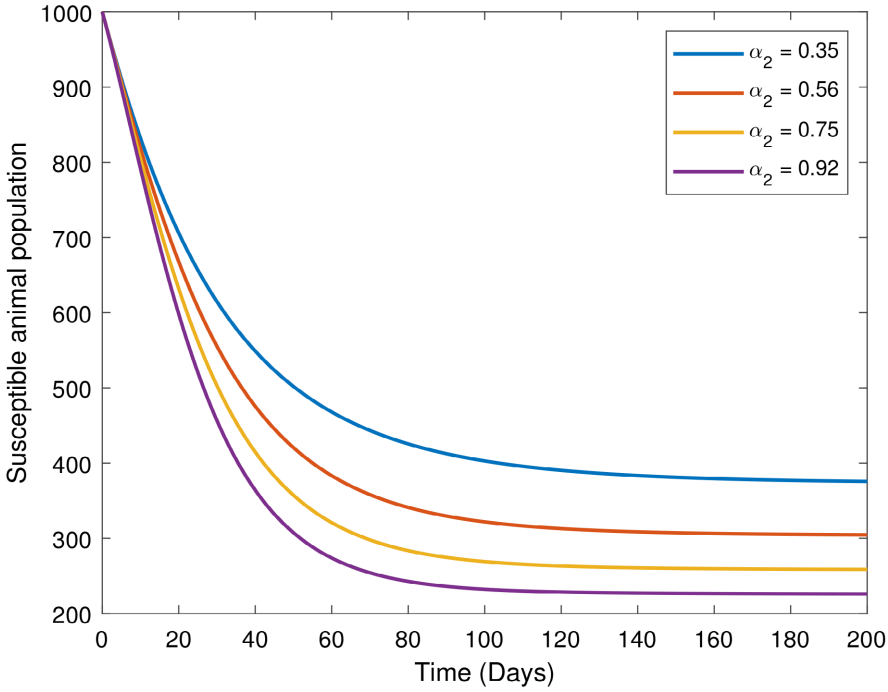


**Figure 7.** Effect of $\alpha_2$ on susceptible animal population.

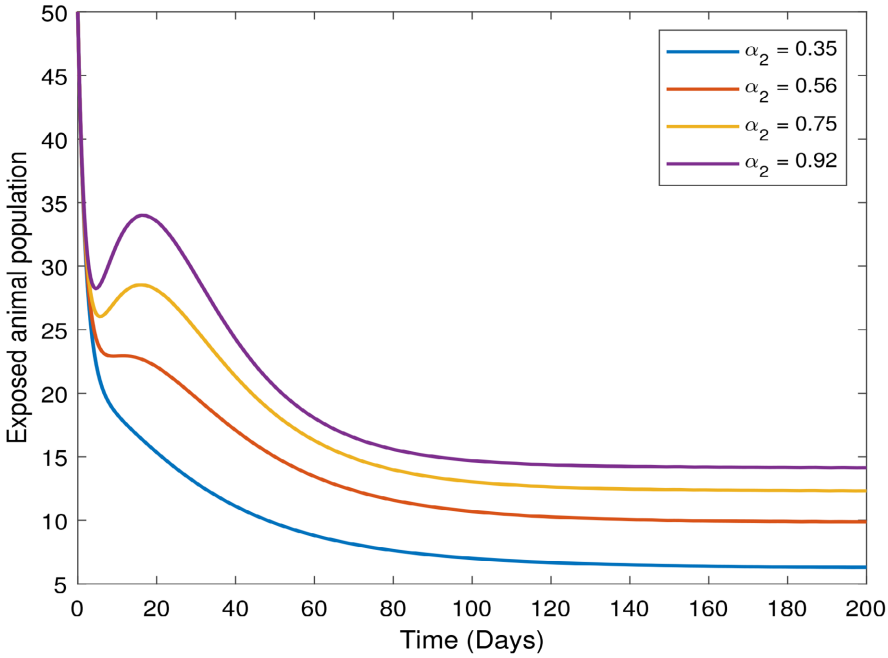


**Figure 8.** Effect of $\alpha_2$ on exposed animal population.

Adaptation (see **Figure 8**). The steady state signifies ongoing disease dynamics and the formation of a stable endemic equilibrium.

In **Figure 5**, rising active immigrants ( $\alpha_1$ ) lead to initial increase, then gradual decrease due to possibly virulent immigrant strains causing higher symptomatic cases and reduce asymptomatic individuals, or new variants by infective immigrants. **Figure 9** displays sharp increase followed by a gradual decrease towards a stable

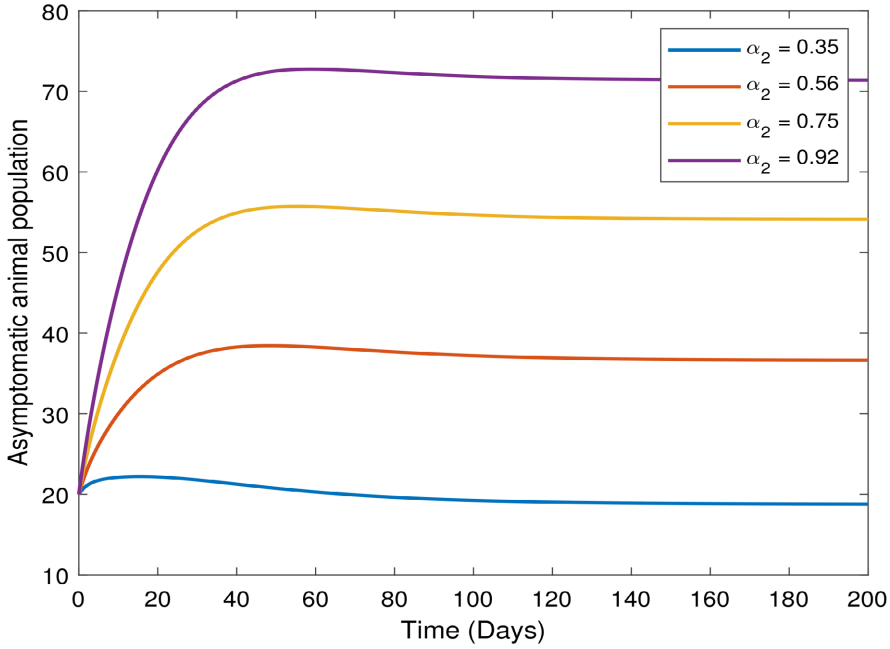


**Figure 9.** Effect of $\alpha_2$ on asymptomatic animal population.

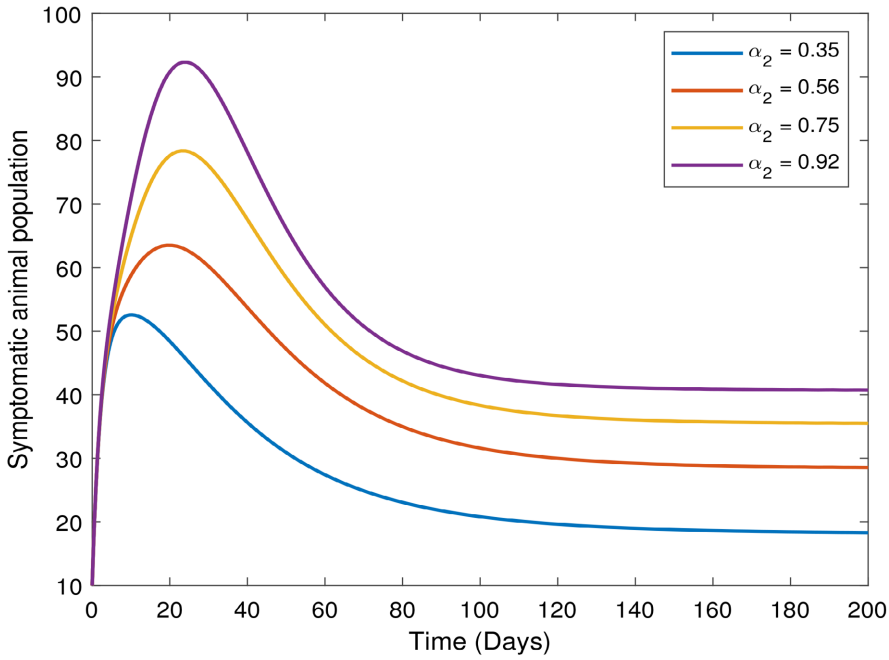


**Figure 10.** Effect of $\alpha_2$ on symptomatic animal population.

state with an increase in ( $\alpha_2$ ); this could be attributed to the fact that infectious immigrants can rapidly spread disease in local animals and shift animals to symptomatic states, reducing asymptomatic numbers. Some animals gain immunity, increasing asymptomatic or resistant individuals. Equilibrium shows disease-immune balance, highlighting dynamic adaptation.

Figure 10 portrays a sharp increase and gradual decrease to stable condition with an increase in ( $\alpha_1$ ); possible factors for this could include, a phase of lo-

cal adaptation to the new agent, during this period, recovery could increase as the agent spreads, also, more animals acquire immunity through infections and recovery. The rapid rise in recoveries signifies initial immunity development to the new agent. Subsequent gradual increase and steady state show ongoing disease dynamics and the establishment of a stable endemic equilibrium.

The key observation is that the presence of infectious immigrants ( $\alpha_1$ and $\alpha_2$ ) is closely associated with increased susceptibility in animal populations. This implies that the local animal population becomes more susceptible to FMD when exposed to these infectious immigrants. This result underscores the complexity of disease transmission dynamics and has important implications for designing targeted interventions and management strategies. The findings provide valuable insights that contribute to a more comprehensive understanding of the factors influencing the establishment and spread of infectious diseases in populations.

## 3. Conclusions

In summary, the figures collectively highlight the intricate interplay of disease dynamics in animal populations, driven significantly by the influx of infectious immigrants ( $\alpha_1$ and $\alpha_2$ ). These immigrants significantly shape susceptibility and recovery populations, causing fluctuations and equilibrium. The observed patterns stress the need to account for immigrant influence in understanding disease transmission, population responses, and stability. These insights have implications for disease control and management strategies. In essence, the figures underscore the dynamic nature of disease processes and the significance of grasping the interaction between immigrant influx, host responses, and disease outcomes for effective management.

While these figures provide valuable insights, there are areas requiring further exploration. Specifically, researching the mechanisms of interactions between immigrants and local populations, understanding strain impacts and transmission dynamics, and investigating genetic, ecological, and epidemiological factors will refine our comprehension. Advancing understanding in these aspects will enhance precise disease management strategies. The study's findings provide a foundation for shaping targeted and adaptive intervention strategies in the management of Foot and Mouth Disease. These recommendations emphasize the need for a multifaceted approach, combining early detection, preventive measures, adaptive management, and international collaboration to effectively address the complexities introduced by active immigrants in disease transmission dynamics. For the future, the study can be extended in a multidisciplinary approach, incorporating insights from epidemiology, immunology, economics, and genomics, which can provide a holistic understanding of the complexities associated with FMD and contribute to the global effort to control and mitigate the impact of this important livestock disease.

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## Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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