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DYNAMICS OF FOOT-AND-MOUTH DISEASE SPREAD MODEL FOR CATTLE WITH CARRIER, VACCINATION, AND ENVIRONMENTAL TRANSMISSION

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Abstract. In this article, we discuss the dynamics of Foot-and-Mouth Disease (FMD) spread model by considering direct infections from infected and carrier population, indirect infections from patogen population in environment, and the intervention such as vaccination, culling, and environment sanitation. The proposed model contains six subpopulations: susceptible (S), vaccinated (V), exposed (E), infected (I), carrier (I_C), and patogen (P). For the dynamics of proposed model, we first show the non-negativity and boundedness of solutions. The equilibrium point, basic reproduction number, local and global stability of equilibrium points are also investigated analytically. The proposed model has disease-free equilibrium point always exists and endemic equilibrium point exists when $R_0 > 1$. The disease-free equilibrium point is locally asymptotically stable when $R_0 < 1$ and fulfills the Routh-Hurwitz criterion, and globally asymptotically stable when $R_0 < 1$. While the endemic equilibrium point is locally asymptotically stable when Lienard-Chipart criterion is satisfied, and globally asymptotically stable when $R_0 > 1$ and one of the following conditions (i) If $c = 0$ and $\varphi = 0$, or (ii) If $N \leq \frac{\Pi}{\mu}$, is satisfied. Numerical simulations are performed to verify the analytical result. The simulation results demonstrate the local and global stability of equilibrium point.

Keywords: FMD epidemic model; equilibrium point; local stability; global stability.

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1. INTRODUCTION

Foot-and-Mouth Disease (FMD) is transboundary animal disease that highly contagious or transmissible, epidemic diseases, have the potential for rapid spread regardless across the globe, cause substantial socioeconomic losses, and result in negative public health outcomes [5], [11]. FMD attacks cloven-hoofed animals, namely ruminants (cattle, buffalo, goats, sheep), pigs, and similar species caused by Foot and Mouth Disease Virus (FMDV) [5], [8], [17]. Indonesia is one of the countries in the Asian region affected by FMD. In the last few months, since April 2022, FMD has started to reoccur widely and infect livestock, especially cattle [8]. As of July 2022, the Government of Indonesia through the Indonesian Disaster Management Authority or Badan Nasional Penanggulangan Bencana (BNPB) reported 233,370 cases of FMD in 246 districts in 22 provinces in Indonesia. According to data from the Ministry of Agriculture's Crisis Center, Central Java province is the first province with the most FMD with 133460 reported cases. Next is West Nusa Tenggara province with 48246 cases, East Java with 33178 cases, Aceh province with 32330 cases and West Java with 32178 cases [2].

Direct or indirect contact with FMDV infected animals can result in susceptible animals becoming diseased or sub-clinically infected [6], [10]. The main route of virus entry, in natural infections, is the respiratory tract through inhalation of airborne virus and in ruminants, but not pigs, complete clearance of virus from the pharynx to persistently infected carriers [6],[12]. Other animals and humans can also pose a risk of transmission if they become contaminated with the virus (for example, from aerosolization, feces and clothing) [16], [5], [4].

An effective control of FMD is prevention through vaccination [13]. In addition to vaccination, culling infected animals is also an effort to suppress the spread of FMD virus to the environment [10]. In addition to induce immunity in animals and eliminating sources of transmission by culling infected animals, virus transmission in the environment can also be minimized by decontaminating cages, equipment, vehicles and other contaminated materials that can transmit the disease [8]. Culling is also done on carrier animals because after the clinical phase of FMD, most ruminants (cattle, buffalo, goats and sheep) still excrete the virus even though they look clinically healthy [3]. Until now, there has been no known specific treatment

for FMD. Therefore, it is also important to understand the spread of FMD so that prevention and treatment can be optimized.

Based on the history of the spread of FMD, it is necessary to study the spread of FMD. Mathematical modeling is an approach to understanding the dynamics of FMD spread [9]. Mathematical modeling can be applied to represent the phenomenon of change so that it can be used to understand the dynamics of FMD spread. In [15], Mushayabasa and Tapedzesa constructed the SVE_VII_C model by considering vaccination and direct transmission method caused by contact with infected and carrier. Furthermore, in [10], Gashirai et al. stated that the transmission can occur by direct and indirect method, and consider pathogen in the environment. The transmission rates of both are different. In [18], Sseguya et al. constructed the $SVEII_C$ (Susceptible-Vaccinated-Exposed-Infected-Carrier) model that also consider vaccination, carrier properties, and FMD transmission caused by contact with exposed and infected.

The various characteristics of FMD disease and the intervention are important to consider in the mathematical model. In this study, we propose the FMD model by considering direct infections from infected and carrier population, indirect infections from patogen population in environment, carrier properties, and the intervention such as vaccination, culling, and enviromental sanitation. First, in Section 2 we construct $SVEII_CP$ model. Based on the model that has been constructed, we analyze the dynamic of proposed model containing basic properties in Section 3 (non-negativity and boundedness of solutions), equilibrium points and basic reproduction number in Section 4, stability of equilibrium points, both locally in Section 5 and globally in Section 6. We showed a numerical simulations to confirm the result. Furthermore, we conclude in Section 7.

2. Model Formulation

The model of FMD transmission in this study describe the interaction between six subpopulations, that is, S, V, E, I, I_C, and P, which represent the subpopulations size of susceptible, vaccinated, exposed, infected, carrier, and patogen, respectively. The model assumes that susceptible individual (S) may become exposed if there are contact between susceptible individual with infected or carrier individual by direct contact transmission, or by indirect transmission between susceptible individual with patogen in environment. Vaccinated subpopulation (V)

is susceptible individuals who get vaccination and vaccinated subpopulation can return to be susceptible individuals due to the decay of the vaccination effect. Carrier subpopulation (I_C) is infected individuals that have recovered clinically but can still excrete the virus into the environment from the pharynx and cannot be cured by treatment. Patogen subpopulation (P) is FMD virus population that spread in the environment due to excretion from infected and carrier individuals. In this model, we consider three intervention including vaccination, culling, and environmental sanitation. The interaction between six subpopulations of the proposed model are shown by compartment diagram in Figure 1.

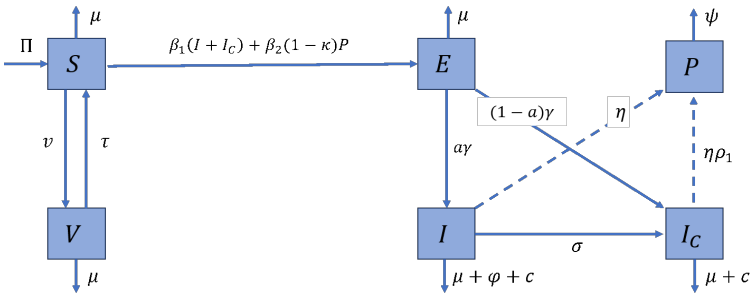

FIGURE 1. The compartment diagram of proposed FMD spread model.

The proposed model is expressed in a first-order differential equations system (1).

$$
\begin{aligned}
\frac{dS}{dt} &= \Pi + \tau V - (\nu + \mu)S - (\beta_1(I + I_C) + \beta_2(1-\kappa)P)S, \\
\frac{dV}{dt} &= \nu S - (\tau + \mu)V, \\
\frac{dE}{dt} &= (\beta_1(I + I_C) + \beta_2(1-\kappa)P)S - (\mu + \alpha\gamma + (1-\alpha)\gamma)E, \\
\frac{dI}{dt} &= \alpha\gamma E - (\sigma + \mu + \varphi + c)I, \\
\frac{dI_C}{dt} &= (1-\alpha)\gamma E + \sigma I - (\mu + c)I_C, \\
\frac{dP}{dt} &= \eta I + \eta\rho_1 I_C - \psi P.
\end{aligned} \tag{1}
$$

with non-negative initial values $S(0) = S_0$, $V(0) = V_0$, $E(0) = E_0$, $I(0) = I_0$, $I_C(0) = I_{C_0}$, $P(0) = P_0$. The definition of parameters in model (1) could be seen in Table 1.

TABLE 1. Model parameter and their description

Parameter	Descriptions	Value	Source
Π	Number of cattle recruitment	100	Gashirai et al. (2020)
ν	Vaccination rate of S	$0,25$	Mushayabasa & Tapedzesa (2015)
τ	Rate of loss of vaccine-induced immunity	$0,011$	Gashirai et al. (2020)
μ	Natural mortality rate	$0,002$	Gashirai et al. (2020)
β_1	Direct disease transmission rate	10^{-6}	Gashirai et al. (2020)
β_2	Indirect disease transmission rate	10^{-10}	Gashirai et al. (2020)
κ	Proportion of environmental sanitation	$0,2$	Assumed
α	Proportion of E that become persistently infected	$0,85$	Gashirai et al. (2020)
γ	Rate of FMD progression	$0,25$	Gashirai et al. (2020)
σ	Rate at which symptomatic cattle become carriers	$0,14$	Gashirai et al. (2020)
φ	Death rate due to disease	$0,1$	Gashirai et al. (2020)
c	Culling rate	$0,1$	Assumed
η	Environmental decontamination rate	10^4	Gashirai et al. (2020)
ρ_1	Proportion of carrier decontamination	$0,7$	Assumed
ψ	Environmental patogen decay rate	$0,07$	Gashirai et al. (2020)

To simplify the System (1), we introduce new symbols $\xi_1 = \nu + \mu$, $\xi_2 = \tau + \mu$, $\xi_3 = \mu + \alpha\gamma + (1-\alpha)\gamma$, $\xi_4 = \sigma + \mu + \varphi + c$, $\xi_5 = \mu + c$, and $\xi_6 = \psi$ expressed in System (2).

$$
\begin{aligned}
\frac{dS}{dt} &= \Pi + \tau V - (\beta_1(I + I_C) + \beta_2(1-\kappa)P)S - \xi_1 S, \\
\frac{dV}{dt} &= \nu S - \xi_2 V, \\
\frac{dE}{dt} &= (\beta_1(I + I_C) + \beta_2(1-\kappa)P)S - \xi_3 E, \\
\frac{dI}{dt} &= \alpha\gamma E - \xi_4 I, \\
\frac{dI_C}{dt} &= (1-\alpha)\gamma E + \sigma I - \xi_5 I_C, \\
\frac{dP}{dt} &= \eta I + \eta\rho_1 I_C - \xi_6 P.
\end{aligned} \tag{2}
$$

3. Non-Negativity and Boundedness of Solutions

Since model (2) describes the interaction of animal and patogen subpopulations, the solution of the system must be non-negative and ultimately bounded. The following theorem has guaranteed the non-negativity and boundedness of solution of Model (2).

Theorem 1. *All solutions of the FMD Model* (2) *subject to non-negative initial values are non-negative and bounded.*

Proof. We first prove that $S(t)$ and $V(t)$ are non-negative. Assume the contrary; then let t_1 and t_2 be the first time such they are equal to zero at t_1 and t_2, respectively. From first and second equations of Model (2), we get

$$
\begin{aligned}
\frac{dS(t)}{dt}\Big|_{t=t_1} &= \Pi + \tau V(t_1), \\
\frac{dV(t)}{dt}\Big|_{t=t_2} &= \nu S(t_2).
\end{aligned} \tag{3}
$$

Since the right-hand side of equation (3) depends on t_1 and t_2, we separate this proof cases.

If $t_1 \leq t_2$, then $V(t_1) \geq 0$. We have

$$
\frac{dS(t)}{dt}\Big|_{t=t_1} = \Pi + \tau V(t_1).
$$

This means that $S(t) > 0$ on $(t_1, t_1+\varepsilon_1)$ for arbitrary small positive constant ε_1. This leads to a contradiction. As a result, $S(t) \geq 0$ for all $t \geq 0$. Consequently, $\frac{dV(t)}{dt}\Big|_{t=t_2} = \nu S(t_2) > 0$. Similarly, $V(t) \geq 0$ on $(t_2, t_2+\varepsilon_2)$ for arbitrary small positive constant ε_2. This leads to a contradiction. As a result, $V(t) \geq 0$ for all $t \geq 0$.

If $t_1 > t_2$, then $S(t_2) > 0$. We have

$$\frac{dV(t)}{dt}\Big|_{t=t_2} = \nu S(t_2).$$

This means that $V(t) > 0$ on $(t_2, t_2+\varepsilon_2)$ for arbitrary small positive constant ε_2. This leads to a contradiction. As a result, $V(t) \geq 0$ for all $t \geq 0$. Consequently, $\frac{dS(t)}{dt}\Big|_{t=t_1} = \Pi + \tau V(t_1) > 0$. Similarly, $S(t) > 0$ on $(t_1, t_1+\varepsilon_1)$ for arbitrary small positive constant ε_1. This leads to a contradiction. As a result, $S(t) \geq 0$ for all $t \geq 0$.

The non-negativity of $E(t), I(t), I_C(t), P(t)$ can also been shown in similar way. Therefore, all solutions of Model (2) are non-negative. □

Proof. We next let $N = S+V+E+I+I_C$. Based on Model (2), we have

$$\begin{aligned}
\frac{dN}{dt} &= \Pi - \mu S - \mu V - (\mu+\varphi+c)I - \mu E - (\mu+c)I_C \leq \Pi - \mu N, \\
\frac{dP}{dt} &= \eta I + \eta\rho_1 I_C - \psi P \leq (\eta+\rho_1\eta)\frac{\Pi}{\mu} - \psi P.
\end{aligned}$$

It is easy to show that N and P satisfy

$$N \leq \frac{\Pi}{\mu} + \left(N(0) - \frac{\Pi}{\mu}\right)\exp(-\mu t), \text{and thus } \lim_{t\to+\infty} N \leq \frac{\Pi}{\mu}$$

and

$$P \leq \frac{\eta+\rho_1\eta}{\psi}\frac{\Pi}{\mu} + \left(P(0) - \frac{\eta+\rho_1\eta}{\psi}\frac{\Pi}{\mu}\right)\exp(-\psi t), \text{and thus } \lim_{t\to+\infty} P \leq \frac{\eta+\rho_1\eta}{\psi}\frac{\Pi}{\mu}.$$

Hence, the feasible region of Model (2) is

$$\Omega = \left\{(S,V,E,I,I_C,P) \in \mathbb{R}^6_+ \cup \{0\} \middle| N \leq \frac{\Pi}{\mu}, P \leq \frac{\eta+\rho_1\eta}{\psi}\frac{\Pi}{\mu}\right\}.$$

Therefore, all solutions of Model (2) are bounded. □

4. Equilibrium Point and Basic Reproduction Number

We first, let $X_1 = \beta_1\left(\frac{\alpha\gamma}{\xi_4} + \frac{((1-\alpha)\xi_4+\sigma\alpha)\gamma}{\xi_4\xi_5}\right) + \beta_2(1-\kappa)\left(\left(\frac{\eta\alpha\gamma\xi_5+((1-\alpha)\xi_4+\sigma\alpha)\eta\rho_1\gamma}{\xi_4\xi_5\xi_6}\right)\right.$, and $\vec{x} = (S,V,E,I,I_C,P)$. By setting the right-hand side of equation Model (2) to be zero, we get the solutions as equilibrium points. We see the third equation of Model (2):

$$(X_1S - \xi_3)E = 0.$$

It is clear that either $E = 0$ or $X_1S = \xi_3$, from which we obtain two equilibrium points of Model (2), that is, disease-free equilibrium point x^0 and endemic equilibrium point x^*. The disease-free equilibrium point is $x^0 = \left(\frac{\xi_2\Pi}{\xi_1\xi_2-\nu\tau}, \frac{\nu\Pi}{\xi_1\xi_2-\nu\tau}, 0, 0, 0, 0\right)$ which is always exists.

We next determine the basic reproduction number (R_0) of Model (2). First, we define $\vec{z} = (E,I,I_C,P)$, which is vector of infected compartment. The expression

$$\mathscr{F}(\vec{z}) = \begin{pmatrix} (\beta_1(I+I_C)+\beta_2(1-\kappa)P)S \\ 0 \\ 0 \\ 0 \end{pmatrix} \text{ and } \mathscr{V}(\vec{z}) = \begin{pmatrix} \xi_3E \\ -\alpha\gamma E + \xi_4 I \\ -(1-\alpha)\gamma E - \sigma I + \xi_5 I_C \\ -\eta I - \eta\rho_1 I_C + \xi_6 P \end{pmatrix}$$

The Jacobian matrices of $\mathscr{F}$ and $\mathscr{V}$ at x^0 are respectively

$$F = \begin{pmatrix} 0 & \beta_1S^0 & \beta_1S^0 & \beta_2(1-\kappa)S^0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{pmatrix} \text{ and } V = \begin{pmatrix} \xi_3 & 0 & 0 & 0 \\ -\alpha\gamma & \xi_4 & 0 & 0 \\ -(1-\alpha)\gamma & -\sigma & \xi_5 & 0 \\ 0 & -\eta & -\eta\rho_1 & \xi_6 \end{pmatrix}$$

The next generation matrix is

$$FV^{-1} = \begin{pmatrix} R_{01}+R_{02} & b_1 & c_1 & d_1 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{pmatrix}$$

where $R_{01} = \beta_1S^0\left(\frac{\alpha\gamma}{\xi_3\xi_4} + \frac{((1-\alpha)\xi_4+\sigma\alpha)\gamma}{\xi_3\xi_4\xi_5}\right)$ and $R_{02} = \beta_2(1-\kappa)S^0(\frac{\eta\alpha\gamma\xi_5+((1-\alpha)\xi_4+\sigma\alpha)\eta\rho_1\gamma}{\xi_3\xi_4\xi_5\xi_6})$. The basic reproduction number is the radius spectral ρ of the next generation matrix, which in

our case is given by

$$R_0 = \rho(FV^{-1}) = R_{01} + R_{02} = \frac{X_1}{\xi_3}\Big(\frac{\xi_2\Pi}{\xi_1\xi_2 - \nu\tau}\Big).$$

We notice that the basic reproduction number has two terms which are from direct transmission and indirect transmission, respectively [10].

The second equilibrium point is endemic equilibrium point $x^* = (S^*, V^*, E^*, I^*, I_C^*, P^*)$ with

$$S^* = \frac{1}{R_0}\Big(\frac{\xi_2\Pi}{\xi_1\xi_2 - \nu\tau}\Big), V^* = \frac{1}{R_0}\Big(\frac{\xi_2\Pi}{\xi_1\xi_2 - \nu\tau}\Big)\frac{\nu}{\xi_2}, E^* = \frac{\Pi}{\xi_3}\Big(1 - \frac{1}{R_0}\Big), I^* = \frac{\alpha\gamma}{\xi_4}\frac{\Pi}{\xi_3}\Big(1 - \frac{1}{R_0}\Big),$$

$$I_C^* = \Big(\frac{((1-\alpha)\xi_4 + \sigma\alpha)\gamma}{\xi_4\xi_5}\Big)\frac{\Pi}{\xi_3}\Big(1 - \frac{1}{R_0}\Big), P^* = \Big(\frac{\eta\alpha\gamma\xi_5 + ((1-\alpha)\xi_4 + \sigma\alpha)\eta\rho_1\gamma}{\xi_4\xi_5\xi_6}\Big)\frac{\Pi}{\xi_3}\Big(1 - \frac{1}{R_0}\Big).$$

The endemic equilibrium point x^* exists if $R_0 > 1$.

5. Local Stability

In this section, we investigate the local stability of equilibrium points of non-linear Model (2) with linearization around equilibrium point. In this linierization, the Jacobian matrix at equilibrium point x^k is given by

$$J(\bar{x^k}) = \begin{pmatrix} -(\beta_1(I^k + I_C^k) + \beta_2(1-\kappa)P^k) - \xi_1 & \tau & 0 & -\beta_1 S^k & -\beta_1 S^k & -\beta_2(1-\kappa)S^k \\ \nu & -\xi_2 & 0 & 0 & 0 & 0 \\ \beta_1(I^k + I_C^k) + \beta_2(1-\kappa)P^k & 0 & -\xi_3 & \beta_1 S^k & \beta_1 S^k & \beta_2(1-\kappa)S^k \\ 0 & 0 & \alpha\gamma & -\xi_4 & 0 & 0 \\ 0 & 0 & (1-\alpha)\gamma & \sigma & -\xi_5 & 0 \\ 0 & 0 & 0 & \eta & \eta\rho_1 & -\xi_6 \end{pmatrix}$$

By evaluating the real part of all eigenvalues of the Jacobian matrix, we get the following stability conditions for the disease-free equilibrium point and the endemic equilibrium point.

Theorem 2. *The disease-free equilibrium point x^0 of Model* (2) *is locally asymptomatically stable if $R_0 < 1$, $a_1a_2 - a_3 > 0$, and $a_3(a_1a_2 - a_3) - a_1^2a_4 > 0$.*

Proof. Assume that $R_0 < 1$. By evaluating $det(\lambda I_{id} - J(x^0)) = 0$, we have the characteristic equation of Jacobian matrix J at x^0 as follow.

$$((\lambda + \xi_1)(\lambda + \xi_2) - \nu\tau)(\lambda^4 + a_1\lambda^3 + a_2\lambda^2 + a_3\lambda + a_4) = 0, \tag{4}$$

where

$$a_1 = \xi_3 + \xi_4 + \xi_5 + \xi_6,$$

$$a_2 = \xi_3\xi_4 + \xi_3\xi_5 + \xi_3\xi_6 + \xi_4\xi_5 + \xi_4\xi_6 + \xi_5\xi_6 - \gamma\beta_1 S^0,$$

$$a_3 = \xi_3\xi_5\xi_6 + \xi_4\xi_5\xi_6 + \xi_3\xi_4\xi_5 + \xi_3\xi_4\xi_6 - \beta_1 S^0(\alpha\gamma(\xi_5 + \xi_6 + \sigma) + (1-\alpha)\gamma(\xi_4 + \xi_6))$$
$$- \beta_2(1-\kappa)S^0(\alpha\gamma\eta + (1-\alpha)\gamma\eta\rho_1),$$

$$a_4 = \xi_3\xi_4\xi_5\xi_6(1 - R_0).$$

It is clear that the first two eigenvalues are determined by $((\lambda + \xi_1)(\lambda + \xi_2) - \nu\tau) = 0$ or equivalently by $\lambda^2 + (\xi_1 + \xi_2)\lambda + (\xi_1\xi_2 - \nu\tau) = 0$. Since $(\xi_1 + \xi_2) > 0$ and $\xi_1\xi_2 - \nu\tau = \mu^2 + (\nu + \tau)\mu > 0$, the real parts of eigenvalues λ_1 and λ_2 are negative. Based on the well-known Routh-Hurwitz criterion, the solutions of

$$\lambda^4 + a_1\lambda^3 + a_2\lambda^2 + a_3\lambda + a_4 = 0$$

have negative real parts if and only if $a_1 > 0$, $a_4 > 0$, $a_1a_2 - a_3 > 0$, and $a_3(a_1a_2 - a_3) - a_1^2a_4 > 0$. We see that a_1 is always positive, $a_4 = \xi_3\xi_4\xi_5\xi_6(1 - R_0) > 0$ if $R_0 < 1$ and other conditions will be proven by numerical simulations. Therefore all solutions of the characteristic Equations (4) have negative real parts if $R_0 < 1$, $a_1a_2 - a_3 > 0$, and $a_3(a_1a_2 - a_3) - a_1^2a_4 > 0$. In other word, the disease-free equilibrium point is locally asymptotically stable. □

Theorem 3. *Let the endemic equilibrium point x^* of Model* (2) *exists. The point x^* is locally asymptotically stable if $w_1 > 0$, $w_3 > 0$, $w_5 > 0$, $w_6 > 0$, $\Delta_3^* > 0$ and $\Delta_5^* > 0$,.*

Proof. By evaluating $det(\lambda I_{id} - J(x^*)) = 0$, the characteristic equation of Jacobian matrix J at x^* can be written as

$$\lambda^6 + w_1\lambda^5 + w_2\lambda^4 + w_3\lambda^3 + w_4\lambda^2 + w_5\lambda + w_6 = 0, \tag{5}$$

where

$$w_1 = \xi_1 + \xi_2 + \xi_3 + \xi_4 + \xi_5 + \xi_6 + \beta_1(I^* + I_C^*) + \beta_2(1-\kappa)P^*,$$

$$w_2 = \xi_6(\xi_1 + \xi_2 + \xi_3 + \xi_4 + \xi_5) + \xi_1(\xi_2 + \xi_3 + \xi_4 + \xi_5) + \xi_2(\xi_3 + \xi_4 + \xi_5) + \xi_3(\xi_4 + \xi_5) + \xi_4\xi_5$$
$$+ (\beta_1(I^* + I_C^*) + \beta_2(1-\kappa)P^*)(\xi_2 + \xi_3 + \xi_4 + \xi_5 + \xi_6) - \gamma\beta_1 S^* - \nu\tau,$$

$$
\begin{aligned}
w_3 &= \xi_1\xi_2(\xi_3+\xi_4+\xi_5)+\xi_1\xi_3(\xi_4+\xi_5)+\xi_4\xi_5(\xi_1+\xi_3)+\xi_2(\xi_3\xi_4+\xi_3\xi_5+\xi_4\xi_5)+\xi_1\xi_6(\xi_2\\
&+\xi_3+\xi_4+\xi_5)+\xi_2\xi_6(\xi_3+\xi_4+\xi_5)+\xi_3\xi_6(\xi_4+\xi_5)+\xi_4\xi_5\xi_6+(\beta_1(I^*+I_C^*)+\beta_2(1-\kappa)P^*)\\
&(\xi_2\xi_3+\xi_2\xi_4+\xi_2\xi_5+\xi_2\xi_6+\xi_3\xi_4+\xi_3\xi_5+\xi_3\xi_6+\xi_4\xi_5+\xi_4\xi_6+\xi_5\xi_6)-\beta_1S^*\gamma(\xi_1+\xi_2\\
&+(1-\alpha)\xi_4+\alpha(\xi_5+\sigma)+\xi_6)-\beta_2S^*\gamma((1-\alpha)(1-\kappa)\eta\rho_1+\alpha(1-\kappa)\eta)\\
&-\nu\tau(\xi_3+\xi_4+\xi_5+\xi_6),\\
w_4 &= \xi_1\xi_2\xi_6(\xi_3+\xi_4)+\xi_1\xi_5\xi_6(\xi_2+\xi_4)+\xi_1\xi_3\xi_6(\xi_4+\xi_5)+\xi_2\xi_5\xi_6(\xi_3+\xi_4)+\xi_1\xi_2\xi_3(\xi_4+\xi_5)\\
&+\xi_1\xi_4\xi_5(\xi_2+\xi_3)+\xi_3\xi_4(\xi_5\xi_6+\xi_2\xi_6+\xi_2\xi_5)+(\beta_1(I^*+I_C^*)+\beta_2(1-\kappa)P^*)(\xi_2\xi_3\xi_4\\
&+\xi_2\xi_3\xi_5+\xi_2\xi_3\xi_6+\xi_2\xi_4\xi_5+\xi_2\xi_4\xi_6+\xi_2\xi_5\xi_6+\xi_3\xi_4\xi_5+\xi_3\xi_4\xi_6+\xi_3\xi_5\xi_6+\xi_4\xi_5\xi_6)\\
&-\beta_1S^*\gamma((1-\alpha)\xi_4\xi_6+(1-\alpha)\xi_2\xi_4+\xi_1(\xi_2+\xi_6)+(1-\alpha)\xi_1\xi_4+\alpha\sigma(\xi_1+\xi_2+\xi_6)\\
&+\alpha(\xi_1\xi_5+\xi_2\xi_5+\xi_5\xi_6)+\xi_2\xi_6-\nu\tau)-\beta_2S^*\gamma((1-\kappa)(1-\alpha)\eta\rho_1\xi_4+(1-\kappa)(\alpha\sigma\eta\rho_1\\
&+\alpha\eta\xi_5)+(1-\kappa)(\xi_1+\xi_2)(\alpha\eta+(1-\alpha)\eta\rho_1))-\nu\tau(\xi_3\xi_4+\xi_3\xi_5+\xi_3\xi_6+\xi_4\xi_5+\xi_4\xi_6+\xi_5\xi_6);\\
w_5 &= (\xi_1\xi_2\xi_3\xi_4\xi_5)+(\xi_1\xi_3\xi_4\xi_5\xi_6)+(\xi_2\xi_3\xi_4\xi_5\xi_6)+(\xi_1\xi_2\xi_3\xi_4\xi_6)+(\xi_1\xi_2\xi_3\xi_5\xi_6)+(\xi_1\xi_2\xi_4\xi_5\xi_6)\\
&+(\beta_1(I^*+I_C^*)+\beta_2(1-\kappa)P^*)(\xi_2\xi_3\xi_4\xi_6+\xi_2\xi_3\xi_5\xi_6+\xi_2\xi_4\xi_5\xi_6+\xi_3\xi_4\xi_5\xi_6+\xi_2\xi_3\xi_4\xi_5)\\
&-\nu\tau(\xi_3\xi_4\xi_5+\xi_3\xi_4\xi_6+\xi_3\xi_5\xi_6+\xi_4\xi_5\xi_6)+\beta_1S^*(\alpha\gamma(\xi_1\xi_2\xi_4+\nu\tau\xi_5+\nu\tau\sigma)+\gamma(\nu\tau\xi_6\\
&+\nu\tau\xi_4))+\beta_2S^*((1-\kappa)\nu\tau\gamma\eta\rho_1+(1-\kappa)\nu\tau\alpha\gamma\eta)-\beta_1S^*(\alpha\gamma(\xi_1\xi_5\xi_6+\xi_2\xi_5\xi_6-\xi_1\xi_4\xi_6\\
&-\xi_2\xi_4\xi_6+\xi_1\xi_2\xi_5+\sigma\xi_1\xi_6+\sigma\xi_2\xi_6+\sigma\xi_1\xi_2+\nu\tau\xi_4)+\gamma(\xi_1\xi_2\xi_4+\xi_1\xi_2\xi_6+\xi_1\xi_4\xi_6\\
&+\xi_2\xi_4\xi_6))-\beta_2S^*((1-\kappa)(1-\alpha)\gamma\eta\rho_1(\xi_1\xi_2+\xi_1\xi_4+\xi_2\xi_4)+(1-\kappa)\alpha\gamma\eta\xi_1\xi_2\\
&+(1-\kappa)\nu\tau\alpha\gamma\eta\rho_1+(1-\kappa)\alpha\gamma\eta(\xi_1\xi_5+\xi_2\xi_5)+(1-\kappa)\sigma\alpha\gamma\eta\rho_1(\xi_1+\xi_2));\\
w_6 &= \xi_1\xi_2\xi_3\xi_4\xi_5\xi_6+(\beta_1(I^*+I_C^*)+\beta_2(1-\kappa)P^*)\xi_2\xi_3\xi_4\xi_5\xi_6+\beta_1S^*\gamma(\nu\tau\alpha\xi_5\xi_6+\nu\tau\alpha\sigma\xi_6\\
&+\alpha\sigma\xi_1\xi_2\xi_6+\alpha\xi_1\xi_2\xi_4\xi_6+\nu\tau\xi_4\xi_6)+\beta_2S^*(\alpha\gamma\eta\rho_1\kappa\sigma\xi_1\xi_2+\nu\tau\alpha\gamma\eta\rho_1\kappa\xi_4\\
&+\alpha\gamma\eta\kappa\xi_1\xi_2\xi_5+\alpha\gamma\eta\rho_1\xi_1\xi_2\xi_4+\gamma\eta\rho_1\kappa\xi_1\xi_2\xi_4+\nu\tau\alpha\gamma\eta\xi_5+\nu\tau\gamma\eta\rho_1\xi_4)\\
&-\beta_1S^*\gamma(\nu\tau\alpha\xi_4\xi_6+\alpha\sigma\xi_1\xi_2\xi_6+\xi_1\xi_2\xi_4\xi_6+\alpha\xi_1\xi_2\xi_5\xi_6)-\beta_2S^*\gamma(\alpha\eta\rho_1\kappa\xi_1\xi_2\xi_4\\
&+\eta\rho_1\xi_1\xi_2\xi_4+\nu\tau\alpha\eta\rho_1\kappa\sigma+\alpha\eta\rho_1\sigma\xi_1\xi_2+\nu\tau\alpha\eta\kappa\xi_5+\nu\tau\alpha\eta\rho_1\xi_4+\nu\tau\eta\rho_1\kappa\xi_4\\
&+\alpha\eta\xi_1\xi_2\xi_5)-\nu\tau\xi_3\xi_4\xi_5\xi_6;
\end{aligned}
$$

The endemic equilibrium point x^* is locally asymptotically stable if all characteristic roots of (5) have negative real parts. Based on Lienard-Chipart Criterion [7], this condition is achieved when $w_1 > 0$, $w_3 > 0$, $w_5 > 0$, $w_6 > 0$, $\Delta_3^* > 0$ and $\Delta_5^* > 0$, where

$$\Delta_3^* = w_3(w_1w_2 - w_3) - w_1(w_1w_4 - w_5),$$

and

$$\begin{aligned}\Delta_5^* =& w_5[w_4\Delta_3^* - w_2(w_1(w_2w_5 - w_1w_6) - w_3w_5) + w_1(w_4w_5 - w_3w_6) - w_5^2] \\ & - w_6[w_3\Delta_3^* - w_1(w_1(w_2w_5 - w_1w_6)w_3w_5)].\end{aligned}$$

Since the forms of $w_i,\ i = 1,...,6$ are too complicated, the Lienard-Chipart Criterion will be evaluated numerically. □

6. Global Stability

In this section, we investigate the global stability of equilibrium points by introducing suitable Lyapunov functions. The conditions for global stability of the equilibrium point of Model (2) are given by the following theorems.

Theorem 4. *The disease-free equilibrium point $x^0(S^0,V^0,0,0,0,0)$ of Model (2) is globally asymptotically stable if $R_0 < 1$.*

Proof. We assume that $R_0 < 1$ and define a Lyapunov function

$$\mathfrak{L}_1(\vec{x}) = g_1\Big(S - S^0 - S^0 ln\Big(\frac{S}{S^0}\Big)\Big) + g_2\Big(V - V^0 - V^0 ln\Big(\frac{V}{V^0}\Big)\Big) + g_3E + g_4I + g_5I_C + g_6P,$$

where $\vec{x} = (S,V,E,I,I_C,P), g_1 = \frac{X_1}{\xi_3}$, $g_2 = \frac{\tau X_1}{\xi_2\xi_3}$, $g_3 = \frac{X_1}{\xi_3}$, $g_4 = \frac{\beta_1}{\xi_4}$, $g_5 = \frac{\beta_1}{\xi_5}$, $g_6 = \frac{\beta_2(1-\kappa)}{\xi_6}$.

Since the geometric mean is less than or equal to the arithmetic mean [14], we get

$$\begin{aligned}\frac{d\mathfrak{L}_1(\vec{x})}{dt} &= \frac{X_1}{\xi_3}\Big(1 - \frac{S^0}{S}\Big)\frac{dS}{dt} + \frac{\tau X_1}{\xi_2\xi_3}\Big(1 - \frac{V^0}{V}\Big)\frac{dV}{dt} + \frac{X_1}{\xi_3}\frac{dE}{dt} + \frac{\beta_1}{\xi_4}\frac{dI}{dt} + \frac{\beta_1}{\xi_5}\frac{dI_C}{dt} + \frac{\beta_2(1-\kappa)}{\xi_6}\frac{dP}{dt} \\ &= \frac{X_1}{\xi_3}\Big(1 - \frac{S^0}{S}\Big)(\Pi + \tau V - (\beta_1(I + I_C) + \beta_2(1-\kappa)P)S - \xi_1S) \\ &\quad + \frac{\tau X_1}{\xi_2\xi_3}\Big(1 - \frac{V^0}{V}\Big)(\nu S - \xi_2V) + \frac{X_1}{\xi_3}((\beta_1(I + I_C) + \beta_2(1-\kappa)P)S - \xi_3E) \\ &\quad + \frac{\beta_1}{\xi_4}(\alpha\gamma E - \xi_4I) + \frac{\beta_1}{\xi_5}((1-\alpha)\gamma E + \sigma I - \xi_5I_C) + \frac{\beta_2(1-\kappa)}{\xi_6}(\eta I + \eta\rho_1I_C - \xi_6P)\end{aligned}$$

$$\begin{aligned} &= \frac{X_1}{\xi_3}\Big(\Pi\Big(2-\frac{S}{S^0}-\frac{S^0}{S}\Big)+\tau V^0\Big(2-\frac{S^0V}{SV^0}-\frac{V^0S}{VS^0}\Big)\Big) \\ &\quad +(R_0-1)(\beta_1(I+I_C)+\beta_2(1-\kappa)P). \end{aligned}$$

Therefore, $\frac{d\mathfrak{L}_1(\vec{x})}{dt} \leq 0$ if $R_0 \leq 1$. Moreover, $\frac{d\mathfrak{L}_1(\vec{x})}{dt} = 0$ is achieved if and only if $\vec{x} = x^0$. The LaSalle's Invariance Principle [14] guarantees that the disease-free equilibrium point is globally asymptotically stable. □

Theorem 5. *Let the endemic point x^* of Model* (2) *exists. The point x^* is globally asymptotically stable if $R_0 > 1$ and one of the following conditions holds*

(i). If $c = 0$ and $\varphi = 0$,

(ii). If $N \leq \frac{\Pi}{\mu}$.

Proof. First we assume that $R_0 > 1$ such that the endemic point x^* exists. Then we consider a Lyapunov function

$$\mathfrak{L}_2(\vec{x}) = \frac{1}{2}\Big((S-S^*)+(V-V^*)+(E-E^*)+(I-I^*)+(I_C-I_C^*)\Big)^2+\frac{1}{2}(P-P^*)^2,$$

where $\vec{x} = (S,V,E,I,I_C,P)$. It is easy to show that

$$\begin{aligned} \frac{d\mathfrak{L}_2(\vec{x})}{dt} &= \frac{\partial\mathfrak{L}_2}{\partial S}\frac{dS}{dt}+\frac{\partial\mathfrak{L}_2}{\partial V}\frac{dV}{dt}+\frac{\partial\mathfrak{L}_2}{\partial E}\frac{dE}{dt}+\frac{\partial\mathfrak{L}_2}{\partial I}\frac{dI}{dt}+\frac{\partial\mathfrak{L}_2}{\partial I_C}\frac{dI_C}{dt}+\frac{\partial\mathfrak{L}_2}{\partial P}\frac{dP}{dt} \\ &= \Big[(S-S^*)+(V-V^*)+(E-E^*)+(I-I^*)+(I_C-I_C^*)\Big]\frac{d}{dt}(S+V+E+I+I_C) \\ &\quad +(P-P^*)\frac{dP}{dt} \\ &= (N-N^*)\frac{dN}{dt}+(P-P^*)\frac{dP}{dt} \\ &\leq \Big(N-\Big(\frac{\Pi}{\mu}-\frac{(\varphi+c)}{\mu}I^*-\frac{c}{\mu}I_C^*\Big)\Big)(\Pi-\mu N-(\varphi+c)I-cI_C) \\ &\quad +(P-P^*)((\eta+\rho_1\eta)\frac{\Pi}{\mu}-\psi P) \\ &\leq \mu\Big(\frac{(\varphi+c)}{\mu}I^*+\frac{c}{\mu}I_C^*\Big)\Big(\frac{\Pi}{\mu}-N\Big)+\Big(\frac{(\varphi+c)}{\mu}I+\frac{c}{\mu}I_C\Big)\Big(\frac{\Pi}{\mu}-N\Big)-\Big(\frac{(\varphi+c)}{\mu}I+\frac{c}{\mu}I_C\Big) \\ &\quad \Big(\frac{(\varphi+c)}{\mu}I^*+\frac{c}{\mu}I_C^*\Big)+\mu\Big(\frac{\Pi}{\mu}-N\Big)\Big(N-\frac{\Pi}{\mu}\Big)-\psi(P-P^*)^2. \end{aligned}$$

Therefore,

(i). If $c = 0$ and $\varphi = 0$ then $\frac{d\mathfrak{L}_2(\vec{x})}{dt} \leq 0$,

(ii). If $N \leq \frac{\Pi}{\mu}$ then $\frac{d\mathfrak{L}_2(\vec{x})}{dt} \leq 0$.

Furthermore, $\frac{d\mathfrak{L}_2(\vec{x})}{dt} = 0$ is satisfied if only if $\vec{x} = x^*$. By applying the LaSalle's Invariance Principle [14], the endemic equilibrium x^* is globally asymptotically stable. □

7. Numerical Simulations

In this section, we present results of numerical simulations to illustrate the dynamics of FMD. We solve the Model (2) with parameters values shown in Table 1 numerically using the fourth-order Runge-Kutta scheme with the step size $h = 0.01$. Here, the initial values are set to be $N_1(0) = (750, 1000, 400, 200, 15000, 20000)$, $N_2(0) = (500, 10000, 150, 150, 20000, 15000)$, and $N_3(0) = (100, 3000, 100, 400, 10000, 10000)$. Here, we obtain two equilibrium points, namely the disease-free equilibriun point $x^0(2472, 47529, 0, 0, 0, 0)$ and the endemic equilibrium point $x^*(386, 7434, 334, 293, 26852, 2685687068)$. Moreover, the basic reproduction number is $R_0 = 0.1661 < 1$. In addition, we also get Routh-Hurwitz Criteria, $a_1a_2 - a_3 = 0.1309 > 0$ and $a_3(a_1a_2 - a_3) - a_1^2a_4 = 0.0021 > 0$. This means that the disease-free equilibrium point x^0 is locally and globally asymptotically stable. The visualization for this stability is shown in Figure 2.

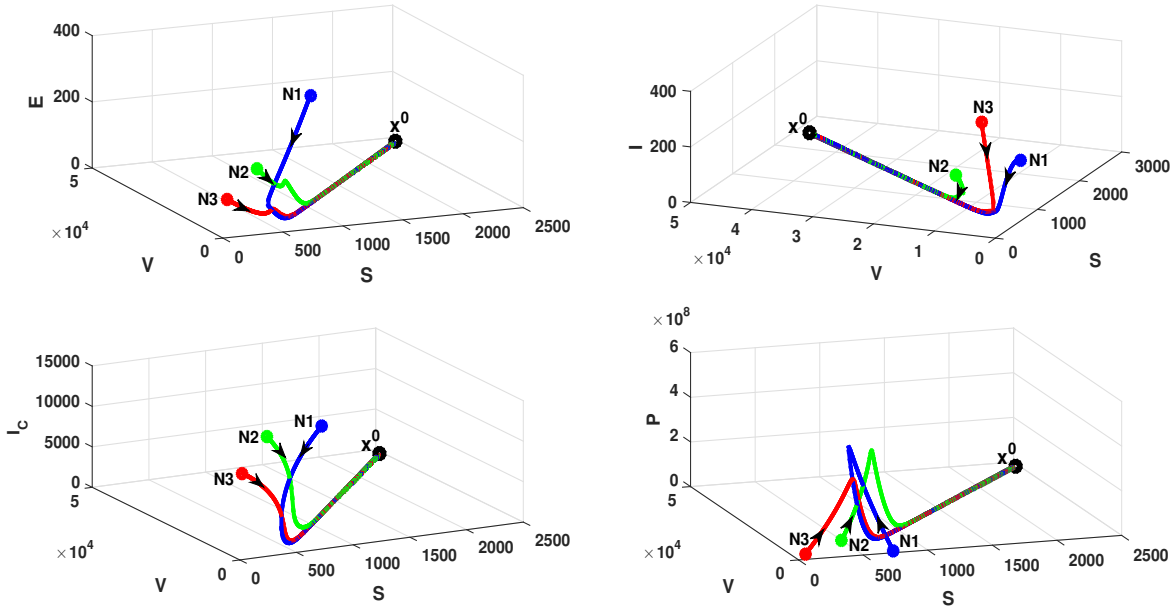

FIGURE 2. The disease-free equilibrium point x^0 is asymptotically stable

Based on the given initial values, where $R_0 < 1$, the solution curve appears to flatten towards the disease-free equilibrium point x^0 as t approaches infinity. It can be observed that from the different given initial values, the solution orbits in the S,V,E space, the S,V,I space, the S,V,I_C space and the S,V,P space converge towards the disease-free equilibrium point x^0. The simulation results support the previous analysis. Therefore, it can also be concluded that if $R_0 < 1$, the disease-free equilibrium point x^0 is locally and globally asymptotically stable.

Furthermore, when no culling cattle rate $(c = 0)$, the basic reproduction number is $R_0 = 6.3927 > 1$. The endemic equilibrium point is exist when $R_0 > 1$ which means that the average number of new exposed individual by one infected individual in susceptible subpopulation is 6.3927. This indicates that the infection of FMDV will continue to exist. The endemic equilibrium point is exist when $R_0 > 1$. In addition, we also get Lienard-Chipart Criteria $w_3 = 0.05671 > 0$, $w_5 = 0.0000279 > 0$, $w_6 = 2.3913x10^{-}8 > 0$, $\Delta_3^* = 0.0172x10^{-}9 > 0$, and $\Delta_5^* = 1.2156 > 0$. This means that the endemic equilibrium point x^* is locally asymptotically stable. The simulation results is presented in Figure 3, and it can be observed that from the different given initial values, the solution orbits in the S,V,E space, the S,V,I space, the S,V,I_C space and the S,V,P space converge towards the endemic equilibrium point x^*. This support the previous analysis that when $R_0 > 1$ and one of the following conditions (i) If $c = 0$ and $\varphi = 0$, or (ii) If $N \leq \frac{\Pi}{\mu}$, is satisfied, the endemic equilibrium point exists and globally asymptotically stable.

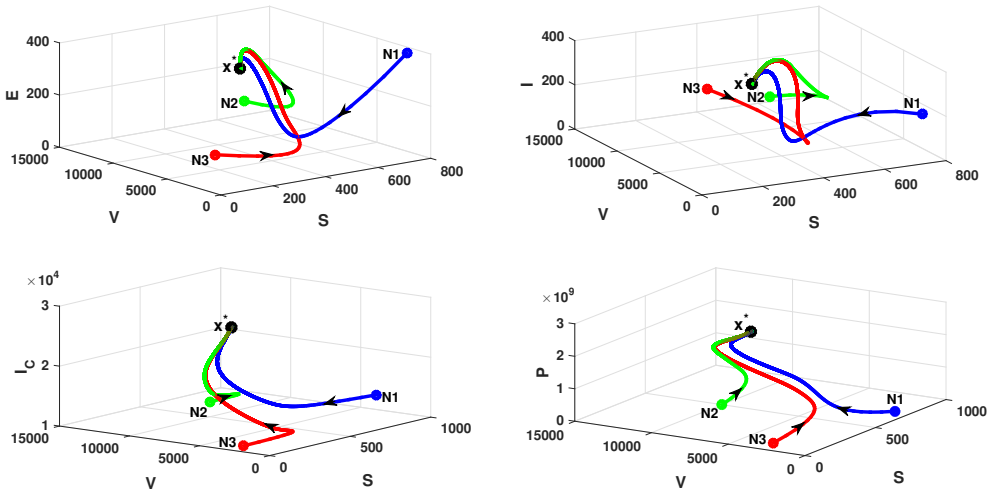

FIGURE 3. The endemic equilibrium point x^*

8. CONCLUSIONS

In this article, FMD model by considering direct infections from infected and carrier population, indirect infections from patogen population in environment, and the intervention such as vaccination, culling, and enviromental sanitation is constructed. The non-negativity and boundedness of solutions of the proposed model have been proven. The model (2) has two equilibrium points, that are, the disease-free equilibrium point (x^0) and the endemic equilibrium point (x^*). The disease-free equilibrium point always exists and is asymptotically stable, both locally and globally, if $R_0 < 1$. The endemic equilibrium exists if $R_0 > 1$. If the endemic equilibrium exists, then it is global asymptotically stable if one of the following conditions applies: (i) $c = 0$ and $\varphi = 0$, or (ii) $N \leq \frac{\Pi}{\mu}$. Such properties have been confirmed by our numerical simulations.

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CONFLICT OF INTERESTS

The authors declare that there is no conflict of interests.

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