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DYNAMICAL ANALYSIS OF SVEIR MODEL IN SPREAD OF THE MONKEYPOX DISEASE WITH TIME DELAY

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ABSTRACT. *Monkeypox is an infectious disease whose spread is influenced by various epidemiological factors, one of which is the incubation period. This study developed a mathematical model of Monkeypox, SVEIR, by incorporating a time delay in the transition from an exposed individual to an infectious individual, representing the incubation period. Model analysis shows that the system has two equilibrium points: a disease-free state and an endemic state. Based on the simulation results, a threshold value (τ^*) is obtained, which plays a crucial role in disease control. If the incubation period is below the threshold ($\tau < \tau^*$), then $R_0 > 1$, and the disease will spread endemically. Conversely, if the incubation period exceeds the threshold ($\tau > \tau^*$), then $R_0 < 1$, and the disease will disappear from the population.*

Keywords*: monkeypox, SVEIR model, time delay, incubation period.*

ABSTRAK. Monkeypox merupakan penyakit menular yang penyebarannya dipengaruhi oleh berbagai faktor epidemiologis, salah satunya masa inkubasi. Penelitian ini mengembangkan model matematika SVEIR pada penyakit *Monkeypox* dengan memasukkan waktu tunda pada transisi dari individu terpapar ke infeksius sebagai representasi dari masa inkubasi. Analisis model menunjukkan bahwa sistem memiliki dua titik ekuilibrium, yaitu bebas penyakit dan endemik. Berdasarkan hasil simulasi, diperoleh nilai ambang batas (τ^*) yang berperan penting dalam pengendalian penyakit. Jika masa inkubasi berada di bawah ambang batas ($\tau < \tau^*$), maka $R_0 > 1$, dan penyakit akan menyebar secara endemik. Sebalinya, jika masa inkubasi melebihi ambang batas ($\tau > \tau^*$), maka $R_0 < 1$, dan penyakit akan menghilang dari populasi.

Kata Kunci: *monkeypox*, model SVEIR, waktu tunda, masa inkubasi.

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1. INTRODUCTION

Monkeypox is a zoonotic disease caused by the monkeypox virus (MPXV) of the Orthopoxvirus genus. The disease was first discovered in laboratory monkeys in Denmark in 1958, while the first human case was reported in 1970 in the Democratic Republic of the Congo (DRC). Since then, the disease has been endemic in several central and west African countries, with thousands of cases reported annually in the DRC, and several surges, including an outbreak in Nigeria in 2017 (Zahmatyar et al., 2023). Monkeypox began to attract global attention after a large, transnational outbreak outside Africa, particularly in Europe, the Americas, and Asia, was discovered in May 2022. This global outbreak marked the first massive spread outside endemic areas, leading the WHO to declare monkeypox a Public Health Emergency of International Concern in July 2022 (WHO, 2025). The first case of monkeypox in Indonesia was confirmed on August 20, 2022, in a person who had recently returned from abroad. Based on data from the Ministry of Health as of August 17, 2024, there were 88 confirmed cases of Monkeypox in Indonesia.

Monkeypox can spread through direct contact between humans and animals (especially primates and rodents) and can also spread between humans through close contact (Qelina & Graharti, 2019). However, human-to-human transmission of the virus is now more common, particularly through direct contact with the blood, skin lesions, or bodily fluids of infected individuals, as well as through contaminated objects. The virus can enter the body through breaks in the skin, the respiratory tract, or mucous membranes such as the eyes, nose, and mouth (Alakunle et al., 2020). Since the World Health Organization (WHO) declared the disease outbreak a global health emergency in 2022, studies on the dynamics of monkeypox transmission have progressed, particularly through mathematical modeling (Ludji & Buan, 2022).

In mathematical modeling, the SEIR (Susceptible–Exposed–Infectious–Recovered) model has been widely used to analyze the dynamics of infectious disease transmission, including monkeypox. One example of the application of the SEIR model is shown in the study by Byrne & Rodrigues (2025), who discussed

the use of the SEIR model to study the spread of monkeypox in the United States. As disease control strategies evolve, model expansion is needed to represent relevant interventions, including vaccination. Several studies have modified the SEIR model by adding a vaccination compartment to the SVEIR model (Ahaya et al., 2020). Furthermore, other studies have shown that models with delay differential equations can more accurately capture the dynamics of biological systems (Dehingia et al., 2021). Delays are commonly used to represent the incubation period or delay in disease transmission. For example, the study by Röst & Wu (2008) developed a disease transmission model with a delay in the incubation period and showed that the presence of a delay can cause changes in the stability of the equilibrium point.

Based on the description above, this study will develop a SEIR model for the spread of Monkeypox by adding a vaccination compartment and incorporating a time delay in the transition from exposed individuals (E) to infectious individuals (I). This time delay represents the disease incubation period, which is the time interval between exposure to the virus and the appearance of clinical symptoms. According to WHO (2024), the incubation period for Monkeypox ranges from 5 to 21 days, with a median of approximately 14 days. Meanwhile, a clinical study conducted by Adler et al (2022) on Monkeypox patients in the UK showed that the average incubation period was approximately 12 days. Based on this, it is important to analyze how changes in the duration of the incubation period can affect the spread of the disease, because the time of symptom onset significantly determines the potential for transmission to other individuals. Furthermore, the addition of a vaccination compartment to the model also plays a crucial role, considering that vaccines are one of the main steps in disease prevention and control efforts. By considering these two aspects, the developed model is expected to provide a more comprehensive picture of the dynamics of Monkeypox spread and assist in designing more effective intervention strategies.

2. RESULTS AND DISCUSSIONS

2.1 Mathematical Model

The model of Monkeypox spread in the human population discussed in this study will be carried out using the SVEIR approach using time delay, where the time delay denotes Monkeypox incubation period. The model is the development of SEIR model by including vaccined population. For the formulation process, we use several assumption such as the population is a closed one and every birth will enter the susceptible population, and of course there are natural births and deaths. Then, the vaccination is only given to susceptible individuals and is 100% effective. For the spread of the disease, it only occurs when there is direct contact between a susceptible individual and an infected individual, while the infected susceptible individuals will become the exposed ones first. In this point, the incubation period occurs when an individual changes from exposed to infected state and explicity modeled with time delay τ. The individuals do not transmit the disease in this period. The movement of individuals from exposed to infected state is influenced by an exponential decay factor due to natural mortality during the incubation period ($e^{-\mu\tau}$). The exponential function is used to express the probability of an individual surviving the incubation period τ. Furthermore, infected individuals can recover and develop permanent immunity. From the assumptions above, a compartment diagram of the SVEIR Monkeypox disease spread model can be formed as follows.

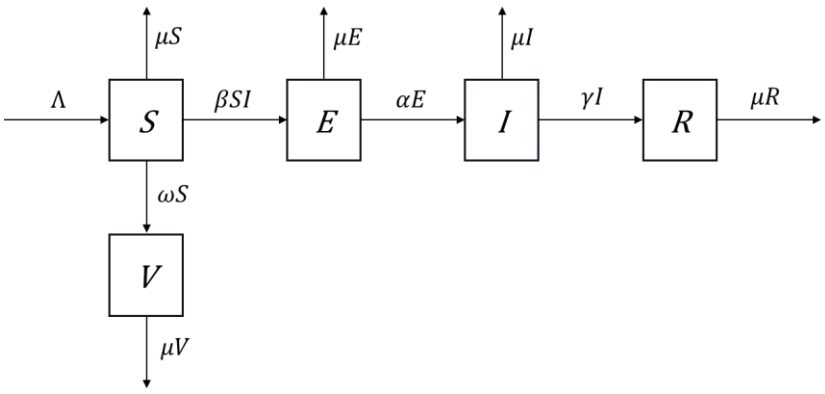

Figure 1. Compartment diagram of the SVEIR Monkeypox disease spread model without time delay

To simplify the model formulation, we will use the notation as a symbol for the compartments and parameters presented in Table 1.

Table 1. Symbols of the compartments and parameters used

Compartments/ Parameters	**Explanation**	**Conditions**
$S(t)$	The number of individuals susceptible to the disease at time t.	$S(t) \geq 0$
$E(t)$	The number of individuals exposed to the disease (yet not infectious) at time t.	$E(t) \geq 0$
$I(t)$	The number of individuals infected to the disease at time t.	$I(t) \geq 0$
$V(t)$	The number of vaccinated individuals at time t.	$V(t) \geq 0$
$R(t)$	The number of individuals recovered to the disease at time t.	$R(t) \geq 0$
Λ	Birth rate (individuals/time)	$\Lambda > 0$
μ	Natural death rate (1/time)	$\mu > 0$
β	Infectious rate (1/(individuals × time))	$\beta > 0$
ω	Vaccinated rate (1/time)	$\omega > 0$
α	Transition rate (1/time)	$\alpha > 0$
γ	Recovery rate (1/time)	$\gamma > 0$
τ	Incubation period (1/time)	$\tau > 0$

Based on the assumptions and the condition of each compartment and parameter in Table 1, the following system of delayed differential equations (DDE) is obtained for the Monkeypox disease spread model :

$$\begin{aligned} \frac{dS}{dt} &= \Lambda - \beta SI - \mu S - \omega S \\ \frac{dV}{dt} &= \omega S - \mu V \\ \frac{dE}{dt} &= \beta SI - \mu E - \alpha E \\ \frac{dI}{dt} &= \alpha e^{-\mu\tau} E(t-\tau) - \mu I - \gamma I \\ \frac{dR}{dt} &= \gamma I - \mu R. \end{aligned} \tag{1}$$

2.2 Disease Free Equilibrium Point

The first equilibrium point is a condition in which there are no individuals infected with Monkeypox disease in a population ($I = 0$). In this case, we will obtain the disease free equilibrium point by solving for

$$\frac{dS}{dt} = \frac{dV}{dt} = \frac{dE}{dt} = \frac{dI}{dt} = \frac{dR}{dt} = 0$$

in System (1) where $E(t-\tau) = E(t)$ for a long period of time and $I = 0$, that is

$$E_{BP} = (S^*, V^*, E^*, I^*, R^*) = \left(\frac{\Lambda}{\mu+\omega}, \frac{\omega\Lambda}{\mu(\mu+\omega)}, 0, 0, 0\right).$$

Since Λ, μ, and ω are positive, the disease free equilibrium point will remain.

2.3 Basic Reproductive Number

The possibility of a disease epidemic occurring in a population in the future can be seen by using the basic reproduction number (R_0). Each infected individual can transmit the disease to more than one susceptible individual and cause the disease to spread rapidly in the population (endemic) when $R_0 > 1$. Whereas for $R_0 < 1$ resulting in each infected individual is unable to produce a single new case of infection that survives to become infectious. As a result, the number of infected individuals will continue to decrease, and the disease will eventually disappear from the population (disease-free state).

The value of R_0 can be obtained by using the method of next generation matrix towards System (1). We can form the matrix M (new infection) and the matrix D (transition between infectious compartments) as follows.

$$M = \begin{bmatrix} 0 & \frac{\beta\Lambda}{\mu+\omega} \\ 0 & 0 \end{bmatrix}, D = \begin{bmatrix} \mu+\alpha & 0 \\ -\alpha e^{-\mu\tau} & \mu+\gamma \end{bmatrix}$$

Then the value of MD^{-1} is sought, namely

$$MD^{-1} = \begin{bmatrix} \frac{\alpha\beta\Lambda e^{-\mu\tau}}{(\mu+\alpha)(\mu+\omega)(\mu+\gamma)} & \frac{\beta\Lambda(\mu+\alpha)}{\mu+\omega} \\ 0 & 0 \end{bmatrix}$$

The value of R_0 is obtained from the spectral radius matrix MD^{-1}, namely the largest positive eigenvalue from matrix MD^{-1}. Furthermore, we will have

$$R_0 = \rho(MD^{-1}) = \frac{\alpha\beta\Lambda e^{-\mu\tau}}{(\mu+\alpha)(\mu+\omega)(\mu+\gamma)}.$$

2.4 Endemic Equilibrium Point

The second equilibrium point occurs when $I \neq 0$. By using the same procedure as in the previous discussion, we will have an endemic equilibrium point

$$E_{Edmk} = (S^*, V^*, E^*, I^*, R^*)$$

where

$$S^* = \frac{\Lambda}{(\mu+\omega)R_0},\ V^* = \frac{\omega\Lambda}{\mu(\mu+\omega)R_0},\ E^* = \frac{(\mu+\omega)(\mu+\gamma)}{\alpha\beta e^{-\mu\tau}}(R_0-1),$$

$$I^* = \frac{(\mu+\omega)}{\beta}(R_0-1),\ R^* = \frac{\gamma(\mu+\omega)}{\mu\beta}(R_0-1).$$

The endemic equilibrium point will exist if $R_0 > 1$.

2.5 The Stability of Equilibrium Points

The stability of the equilibrium point of System (1) is obtained by linearizing the system around it, then continuing by finding the eigenvalues of the characteristic equation resulting from the linearization process. The linearization results of System (1) around the equilibrium point x = $(S^*, V^*, E^*, I^*, R^*)$ are given as follows.

$$\frac{d\mathbf{x}}{dt} = A_0\mathbf{x}(t) + A_1\mathbf{x}(t-\tau) \tag{2}$$

where

$\mathbf{x}(t) = (S(t), V(t), E(t), I(t), R(t))^T$,

$$A_0 = \begin{bmatrix} -(\mu+\omega)-\beta I^* & 0 & 0 & -\beta S^* & 0 \\ \omega & -\mu & 0 & 0 & 0 \\ \beta I^* & 0 & -(\mu+\alpha) & \beta S^* & 0 \\ 0 & 0 & 0 & -(\mu+\gamma) & 0 \\ 0 & 0 & 0 & \gamma & -\mu \end{bmatrix},$$

$$A_1 = \begin{bmatrix} 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & \alpha e^{-\mu\tau} & 0 \\ 0 & 0 & 0 & 0 & 0 \end{bmatrix}.$$

The characteristic equation of System (2) can be obtained by zeroing the determinant of $\lambda I - A_0 - A_1 e^{-\lambda\tau}$ such that

$$(\lambda+\mu)(\lambda+\mu)\Big((\lambda+\mu+\gamma)(\lambda+\mu+\alpha)(\lambda+\mu+\omega+\beta I^*) - \alpha\beta e^{-\mu\tau}e^{-\lambda\tau}(\lambda+\mu+\omega)S^*\Big) = 0 \tag{3}$$

2.5.1 Stability Analysis of Disease-Free Equilibrium Point

Theorem 1. *The disease-free equilibrium point of System (1) is locally asymptotically stable if* $R_0 < 1$. *On the other hand, it is unstable if* $R_0 > 1$.

Proof. Based on Equation (3), the characteristic equation for the disease-free equilibrium point can be written as follows

$$(\lambda+\mu)^2(\lambda+\mu+\omega)\left((\lambda+\mu+\alpha)(\lambda+\mu+\gamma)-(\mu+\alpha)(\mu+\gamma)R_0e^{-\lambda\tau}\right)=0 \tag{4}$$

Three eigenvalues can be obtained directly from Equation (4) as below

$$\lambda_{1,2}=-\mu<0,\ \lambda_3=-(\mu+\omega)<0$$

Whereas the remaining eigenvalues can be obtained from Equation (5) as follows

$$\Delta_{BP}(\lambda,\tau)=(\lambda+\mu+\alpha)(\lambda+\mu+\gamma)-(\mu+\alpha)(\mu+\gamma)R_0e^{-\lambda\tau} \tag{5}$$

It will be shown that the real parts of all eigenvalues of the equation $\Delta_{BP}(\lambda,0)=0$ are negative. We will have

$$\lambda_{4,5}=\frac{-A\pm\sqrt{A^2-4B}}{2}$$

where $A=2\mu+\alpha+\gamma$ and $B=(\mu+\alpha)(\mu+\gamma)(1-R_0)$. We know that $B>0$ when $R_0<1$ and A is always positive. Thus, the real part of the eigenvalues λ_4 and λ_5 are negative because $A>\sqrt{A^2-4B}$. We can say that all eigenvalues of $\Delta_{BP}(\lambda,0)=0$ have a negative real part when $R_0<1$.

Further, it will be shown that for any real number p and $\tau\geq 0$, it holds that $\Delta_{BP}(ip,\tau)\neq 0$ or equivalently say that for any $\tau\geq 0$, $\Delta_{BP}(\lambda,\tau)=0$ has no purely imaginary eigenvalues. Note again that the real parts of all eigenvalues of the equation $\Delta_{BP}(\lambda,0)=0$ are negative. It means that for $\tau=0$, equation $\Delta_{BP}(\lambda,0)=0$ does not have purely imaginary eigenvalues. Furthermore, suppose there is $\tau>0$ which satisfies $R_0<1$ so that equation $\Delta_{BP}(\lambda,\tau)=0$ has purely imaginary eigenvalues, that is $\lambda=ip$ where p is any real number. Substitute $\lambda=ip$ into $\Delta_{BP}(\lambda,\tau)=0$, by separating the real and imaginary parts, we will obtain

$$Re:\ p^2-C=-CR_0\cos p\tau \tag{6}$$

$$Im:\ Ap=-CR_0\sin p\tau \tag{7}$$

where $C=(\mu+\alpha)(\mu+\gamma)$. From Equations (6) and (7), we can obtain

$$p^4 + Q_1 p^2 + Q_2 = 0 \tag{8}$$

where $Q_1 = A^2 - 2C$ dan $Q_2 = C^2\left(1 - {R_0}^2\right)$. Suppose $k = p^2$, we will have

$$k_{1,2} = \frac{-Q_1 \pm \sqrt{{Q_1}^2 - 4Q_2}}{2}.$$

By using mathematical calculations, we obtain $Q_1 > 0$ and $Q_2 > 0$ if $R_0 < 1$, and $Q_1 > \sqrt{{Q_1}^2 - 4Q_2}$. Consequently, the real part of $k_{1,2}$ is negative. Furthermore, $k_{1,2} < 0$ so that $p^2 < 0$ (does not satisfy). This means that there is no real number p that satisfies $\Delta_{BP}(ip, \tau) = 0$. This contradicts the assumption. The assumption must be rejected, which is true for any $\tau > 0$ which satisfy $R_0 < 1$, $\Delta_{BP}(\lambda, \tau) = 0$ does not have a purely imaginary eigenvalue. Based on the two proofs above, it can be said that if $R_0 < 1$ then the disease-free equilibrium point of System (1) is locally asymptotically stable.

Next, to show that the disease-free equilibrium point of System (1) is unstable if $R_0 > 1$, the following procedure is used. Since $\Delta_{BP}(0, \tau) = (\mu + \alpha)(\mu + \gamma)(1 - R_0)$, we have $\Delta_{BP}(0, \tau) < 0$ if $R_0 > 1$. Furthermore, $\Delta_{BP}(\lambda, \tau) \to \infty$ when $\lambda \to +\infty$. Since $\Delta_{BP}(\lambda, \tau)$ is continuous in λ for every $\lambda \in \mathbb{R}$, there exist $\lambda_0 > 0$ such that $\Delta_{BP}(\lambda_0, \tau) = 0$. That is, $\Delta_{BP}(\lambda, \tau) = 0$ has at least one eigenvalue whose real part is positive. Thus, if $R_0 > 1$, then the disease-free equilibrium point of System (1) is unstable.

2.5.2 Stability Analysis of Endemic Equilibrium Point

Theorem 2. *If $R_0 > 1$, endemic equilibrium point of System (1) is locally asymptotically stable.*

Proof. Based on Equation (3), the characteristic equation for the endemic equilibrium point can be written as follows.

$$(\lambda + \mu)^2\big[(\lambda + \mu + \alpha)(\lambda + \mu + \gamma)\big(\lambda + \mu + \omega + (\mu + \omega)(R_0 - 1)\big) - (\mu + \alpha)(\mu + \gamma)e^{-\lambda\tau}(\lambda + \mu + \omega)\big] = 0 \tag{9}$$

From Equation (9), we will have to eigenvalues

$$\lambda_{1,2} = -\mu < 0,$$

while the remaining eigenvalues can be obtained from Equation (10) as follows

$$\Delta_{Edmk}(\lambda,\tau) = (\lambda+\mu+\alpha)(\lambda+\mu+\gamma)\big(\lambda+\mu+\omega+(\mu+\omega)(R_0-1)\big) - (\mu+\alpha)(\mu+\gamma)e^{-\lambda\tau}(\lambda+\mu+\omega). \quad (10)$$

It will be shown that the real parts of all eigenvalues of the equation $\Delta_{Edmk}(\lambda,0)=0$ are negative. We will have

$$\lambda^3 + (A+B+CR_0)\lambda^2 + \big((A+B)CR_0\big)\lambda + ABC(R_0-1) = 0 \quad (11)$$

where $A=\mu+\alpha$, $B=\mu+\gamma$, and $C=\mu+\omega$. Using the Routh-Hurwitz criterion, if $R_0>1$ then all eigenvalues of $\Delta_{Edmk}(\lambda,0)=0$ have negative real parts.

Furthermore, we will show that for any real number p and $\tau\geq 0$, $\Delta_{Edmk}(ip,\tau)\neq 0$ holds, or equivalently, that for any $\tau\geq 0$, the equation $\Delta_{Edmk}(\lambda,\tau)=0$ has no purely imaginary eigenvalues. Note again that the real part of all eigenvalues of $\Delta_{Edmk}(\lambda,0)=0$ is negative. This means that for $\tau=0$, $\Delta_{Edmk}(\lambda,0)=0$ has no purely imaginary eigenvalues. Furthermore, suppose there exists $\tau>0$ satisfying $R_0>1$ such that the equation $\Delta_{Edmk}(\lambda,\tau)=0$ has a purely imaginary eigenvalue, namely, $\lambda=ip$ with p an arbitrary real number. Substitute $\lambda=ip$ into the equation $\Delta_{Edmk}(\lambda,\tau)=0$, by separating the real and imaginary parts, we will obtain

$$Re:\ L-Mp^2 = ABp\sin p\tau + ABp\cos p\tau \quad (12)$$

$$Im:\ Tp-p^3 = ABp\cos p\tau - ABp\sin p\tau. \quad (13)$$

where $M=A+B+CR_0$, $T=(A+B)CR_0+AB$, and $L=ABCR_0$. From Equations (12) and (13), we can obtain

$$p^6 + Z_1p^4 + Z_2p^2 + Z_3 = 0 \quad (14)$$

where $Z_1=M^2-2T$, $Z_2=T^2-2LM-A^2B^2$, dan $Z_3=L^2-A^2B^2C^2$. Suppose $k=p^2$, then Equation (14) can be written as

$$k^3 + Z_1k^2 + Z_2k + Z_3 = 0 \quad (15)$$

Using the Routh-Hurwitz criterion, if $R_0>1$ then the roots of Equation (15) have negative real parts. Furthermore, $k_{1,2,3}<0$ so that $p^2<0$ (does not satisfy). Thus, there is no real number p that satisfies $\Delta_{Edmk}(ip,\tau)=0$. This contradicts the assumption. The assumption must be rejected, what is true is that for any $\tau>0$ that satisfies $R_0>1$, the equation $\Delta_{Edmk}(\lambda,\tau)=0$ does not have a purely

imaginary eigenvalue. Based on the two proofs above, it can be said that if $R_0 > 1$ then the endemic equilibrium point of System (1) is locally asymptotically stable.

2.6 Model Simulation

In this section, the SVEIR monkeypox disease spread model will be numerically analyzed using a Python program. This analysis is performed by substituting predetermined parameter values as shown in Table 2.

Table 2. Parameter values

Parameter	Value	Source
Λ	10	Assumed for simulation purposes
β	0,007	Bragazzi et al., 2022
ω	0,015	Estimated based on data
μ	0,085	Estimated based on data
γ	0,0476	Estimated based on data
α	0,143	Estimated based on data

Now, consider the equation R_0. If $R_0 = 1$, we will have

$$\tau^* = \frac{1}{\mu}\ln\left(\frac{\alpha\beta\Lambda}{(\mu+\alpha)(\mu+\omega)(\mu+\gamma)}\right). \tag{16}$$

By substituting parameter values in Table 2 into Equation (16), we obtain $\tau^* = 14{,}09$ days. The parameter τ has a significant influence on R_0 (see Figure 2).

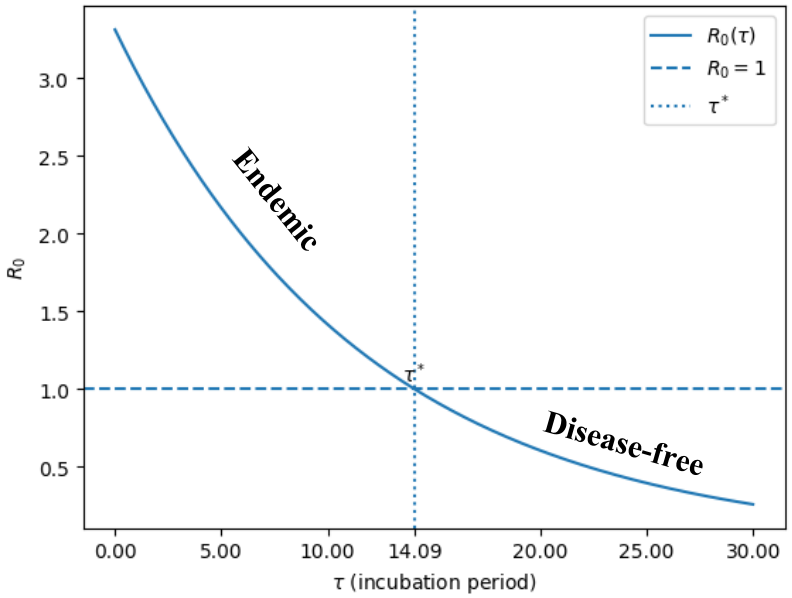

Figure 2. The effect of the time-delay parameter τ on the basic reproduction number (R_0)

Based on Figure 2, it can be observed that an increase in the time-delay parameter τ leads to a decrease in the basic reproduction number R_0. Furthermore, it is found that when $\tau < \tau^* = 14.09$, the value of $R_0 > 1$, indicating that monkeypox can persist and spread within the population. Conversely, when $\tau > \tau^* = 14.09$, the value of $R_0 < 1$, implying that the disease will gradually die out. This result indicates that τ^* acts as a threshold value separating endemic and disease-free conditions.

The simulation is divided into three cases with varying values of τ (incubation period), namely two values of τ that are less than τ^* and one value of τ that is more than τ^*. The simulation is carried out by taking the initial values of $S(0) = 10000$, $V(0) = 500$, $E(0) = 200$, $I(0) = 100$, and $R(0) = 100$. The simulation results presented in Figures 3 and 4.

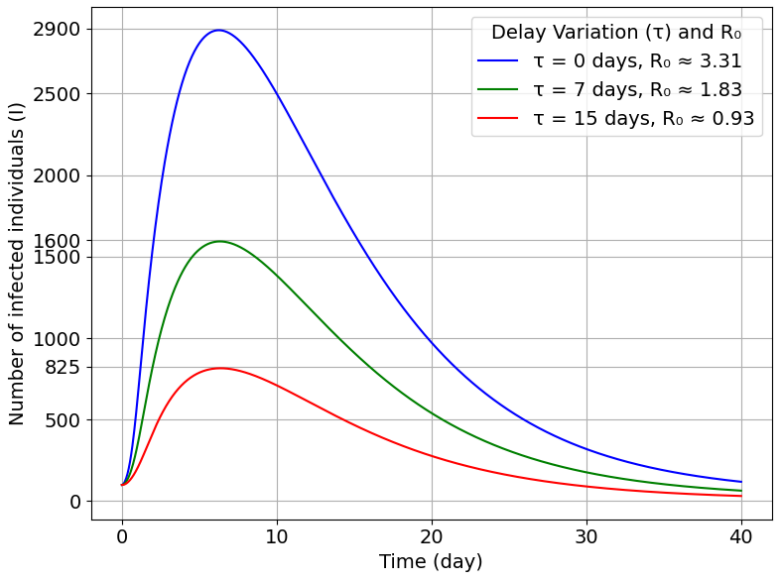

Figure 3. Visualization of infected population $I(t)$ with several $\tau = \{0, 7, 15\}$.

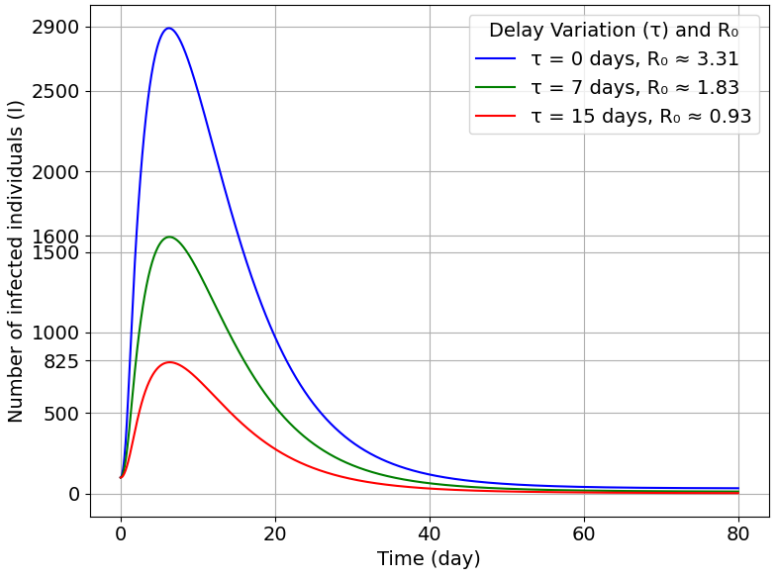

Figure 4. Visualization of infected population $I(t)$ with several $\tau = \{0, 7, 15\}$ for a long period of time.

Based on the simulation results in Figure 3, it can be seen that the number of infected individuals increases simultaneously for different values of τ then decreases simultaneously around the 7th day. Furthermore, it can also be seen that the greater the value of τ or in this case the longer the incubation period, the lower the peak of the Monkeypox disease outbreak. This means that the longer the incubation period for monkeypox, the fewer individuals are infected. This indicates that an increase in the incubation period reduces the maximum number of infected individuals, thereby significantly influencing the transmission dynamics of the disease. In particular, when $\tau = 0$, meaning no incubation period, the epidemic peak is the highest, reflecting a faster and more intense disease transmission. As τ increases, the delay in progression to the infectious stage slows down the transmission process, resulting in a reduced outbreak intensity.

Furthermore, based on the simulation results in Figure 4, it can be seen that for the values of $\tau = 7$, where $R_0 > 1$, the number of infected individuals converges to an endemic equilibrium over a long time period, stabilizing at approximately 11 individuals. Meanwhile, for $\tau = 15$, where $R_0 < 1$, the number of infected individuals gradually decreases and approaches the disease-free equilibrium, eventually tending to zero. This result is in accordance with what has

been explained in Theorems 1 and 2 in the previous sub-chapter, namely if $R_0 < 1$ hen for a long period of time the disease will disappear (towards a disease-free state), while for $R_0 > 1$ then the disease will remain (endemic state).

3. CONCLUSION AND SUGGESTION

Based on the discussion above, it is obtained that the Monkeypox disease spread model in System (1) has two equilibrium points, namely the disease-free and endemic equilibrium points. The stability analysis of the equilibrium points was carried out to determine the long-term dynamics of the spread of Monkeypox disease. Based on the simulation results, a threshold value (τ^*) was obtained which plays an important role in efforts to control the spread of this disease. If the incubation period is below the threshold ($\tau < \tau^*$), which causes $R_0 > 1$, then the disease is expected to continue to spread in the long term. Meanwhile, if the incubation period exceeds the threshold ($\tau > \tau^*$), so that $R_0 < 1$, then for a long period of time the disease will disappear from the population. These results indicate that small changes in the incubation period can have a significant impact on the dynamics of the spread of Monkeypox disease. Therefore, special attention to this parameter is very important in designing interventions that can suppress the spread of Monkeypox disease effectively.

In this study, it was assumed that vaccination against susceptible individuals has a 100% effectiveness rate. In reality, vaccine effectiveness can vary depending on several factors such as vaccine type, individual age, viral mutation, etc. Therefore, in future studies, it is recommended to consider vaccine effectiveness levels that are less than 100%, namely in the interval $[0,1]$ in the model.

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