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Optimal Control of Monkeypox Transmission Model with the Effect of Hospitalization

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Abstract

Monkeypox, also known as mpox, is a zoonotic illness caused by the Monkeypox Virus (MPV), which belongs to the Orthopoxvirus genus within the Poxviridae family. According to a WHO report as of September 2023, the virus has spread to numerous non-endemic countries, showing a significant number of cases. The United States reported the highest count, with 4,259 cases. In contrast, Indonesia has reported relatively fewer cases compared to other Southeast Asian nations. Nonetheless, the risk of transmission, particularly through close personal contact, remains a public health concern. This study examined the transmission of monkeypox among human populations using the spread model proposed by previous research. The novelty of this research is the enhancement of the model by introducing hospitalization rate as a control mechanism, aiming to determine the optimal hospitalization rate level to minimize the disease's spread. The method used for optimal control is minimum pontryagin principle. The model also consider the asymptomatic and symptomatic infected individuals. There is a transition from asymptomatic to symptomatic individuals. Numerical simulation results show that implementing this control leads to a more rapid decline in the number of symptomatic infected individuals compared to scenarios without control measures.

Keywords: *monkeypox, transmission, optimal control, numerical simulation, hospitalization*

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1. Introduction

Monkeypox, or mpox, is a zoonotic disease caused by Monkeypox Virus (MPV), a member of the Orthopoxvirus genus in the Poxviridae family [1]. The disease was first discovered in humans in the Democratic Republic of the Congo in 1970. Since 2022, there has been a significant increase in mpox cases in various non-endemic countries, such as West Africa and Central Africa, making it a global concern [2]. Monkeypox can be transmitted from animals to humans through direct contact with infected animals. In addition, human-to-human transmission occurs through direct contact with skin lesions, body fluids, and respiratory droplets. The virus can also spread through fomites, such as contaminated clothing and towels [1].

Monkeypox is caused by Monkeypox Virus (MPV), which is genetically different from other viruses in the same family, such as variola (smallpox), vaccinia, camelpox, cowpox, and ectromelia [3]. MPV was first identified in 1958 at the State Serum Institute, Copenhagen, as the cause of pox-like disease in quarantined monkeys [3]. However, the natural reservoir of this virus is not monkeys, but wild rodents, such as squirrels, rats, and dormice found in Central and West Africa. MPV has 96.3 percent genomic similarity to variola (smallpox), but has a wider host range, making it more difficult to eradicate through human vaccination. Significant differences in its genome allow MPV to infect more species than variola [3]. Therefore, although monkeypox has similarities with smallpox, MPV is an independent virus in evolution and is not a direct descendant of variola [3].

Monkeypox is included in the category of zoonotic diseases, which can spread from animals to humans and between humans. Transmission from animals to humans occurs through direct contact with infected animals, either through bites, scratches, or contact with body fluids and skin lesions. Consumption of undercooked wild animal meat can also increase the risk of infection. Transmission between humans can occur through close contact with sufferers' skin lesions, body fluids, and respiratory droplets. In addition, the virus can spread through contaminated objects such as towels, clothing, and personal equipment. Recent studies have also shown that monkeypox can be transmitted through sexual contact, especially through close contact with infected individuals [1, 4].

Most cases of monkeypox are mild and resolve within 2–4 weeks without any specific medical intervention. Primary treatment focuses on supportive care to relieve symptoms and boost the patient's immune system. However, for individuals with weakened immune systems or more severe cases, antivirals such as tecovirimat, brincidofovir, and cidofovir are used to inhibit viral replication. Vaccinia immune globulin intravenous (VIGIV) can also be given in certain conditions to treat severe complications [1].

In some cases, around 40 percent of patients experience complications such as rectal pain, odynophagia (pain when swallowing), penile edema, and skin and anorectal abscesses, requiring additional treatment [4]. NSAIDs or opioids can be given to relieve pain from skin lesions or rectal inflammation, while topical lidocaine and laxatives are used for patients with proctitis. If a secondary bacterial infection occurs, antibiotics need to be given, for patients with extensive anogenital lesions or abscesses, wound drainage and debridement procedures are required. Patients who experience dehydration due to dysphagia or severe diarrhea may require rehydration with intravenous fluids [4].

Based on the WHO report until September 2023, the spread of monkeypox has spread to many non-endemic countries with a fairly high number of cases. The United States recorded the highest number of cases with 4,259 cases and 11–25 deaths, followed by Nigeria (1,484 cases) and Australia (1,680 cases). Several other countries, such as the Central African Republic (263 cases) and Germany (240 cases), have also reported deaths due to monkeypox. Meanwhile, in Southeast Asia, the Philippines (21 cases), Indonesia (14 cases), and Singapore (13 cases) have reported cases of infection, although there have been no reports of deaths [5].

Clade is the most dominant variant, especially in countries such as the United States, Australia, Canada, and Hong Kong, which have experienced a spike in cases since 2022. Meanwhile, Clade is more frequently found in African countries, such as Nigeria, Ivory Coast, and

South Africa. Clade is found in the Central African Republic and the Republic of Congo, which were previously endemic areas. Several countries such as the United Kingdom and Taiwan still have undetermined clade status, possibly due to limitations in genomic testing or suboptimal surveillance systems [5].

In Indonesia, the number of monkeypox cases is still relatively low compared to other countries in Southeast Asia. However, its spread remains a concern, especially because of the potential for transmission through close contact between individuals. The Indonesian government has taken steps such as strengthening the health surveillance system, educating the public, and increasing the vigilance of medical personnel in detecting and handling cases. In addition, the Ministry of Health has issued guidelines regarding the prevention and treatment of monkeypox, including self-isolation for infected patients and monitoring close contacts. However, one of the main challenges in controlling this disease is the limited capacity for genomic testing, which causes the identification of virus variants to be slower than in other countries with more advanced laboratory facilities. Therefore, increasing detection capacity and collaboration with international health organizations is needed to prevent further spread in the future [6].

Mathematics has an important role in participating in handling the spread of disease [11], One of them is by using optimal control theory to reduce the spread of disease [12], optimal control is in the form of quarantine in hospitals [13]. Modeling the spread of monkeypox disease has undergone several developments such as the model of the spread of monkeypox disease in humans [8]. There is also research on the spread of monkeypox disease in humans and rodents [10]. Likewise, the development of the model of the spread of monkeypox disease co-infected with Covid-19 and HIV [9]. This study aims to modify the monkeypox model by changing its parameters to optimal control. The technique used is the minimum pontryagin principle. The parameter modified to be a control is the hospitalization rate.

2. Model Formulation

The monkeypox model is developed by Smouni et al., [7]. The model is divided the human population into five compartments: susceptible individuals (S), asymptomatic individuals infected with monkeypox (E), symptomatic individuals infected with monkeypox (I), hospitalized individuals (Q), and recovered individuals (R). This model considers the effect of monkeypox hospitalization. In this study, the modification made was to replace a constant parameter of the hospitalization rate (δ) with an optimal control (u) that depends on time. The model has the following assumptions:

(1) The recruitment of humans occurs only in the susceptible compartment.
(2) Individuals get infected only with monkeypox.
(3) Only asymptomatic individuals infected can transmit monkeypox (due to the symptomatic human individual being hospitalized with all the procedures, hence it can't transmit monkeypox).
(4) Infected individuals can recover if they get hospitalized.
(5) The death rate caused by monkeypox only occurs in symptomatic individuals infected with monkeypox, but the natural death rate occurs in all compartment.

The compartment of susceptible human individuals (S) increases due to recruitment at rate Λ. This compartment decreases due to natural deaths at rate μ. It also decreases due to monkeypox infections with the rate of β.

The compartment of asymptomatic infected human individuals (E) increases due to monkeypox infections with the rate of β. This compartment decreases due to natural deaths at rate μ. It is also decrease due to asymptomatic infected human individuals develop symptoms at the rate of α.

The compartment of symptomatic infected human individuals (I) increases due to asymptomatic infected human individuals develop symptoms at the rate of α. This compartment decreases due to natural deaths at rate μ. It also decreases due to death caused by monkeypox with the rate of d and monkeypox hospitalization at the rate of δ.

The compartment of hospitalized infected human individuals (Q) increases due to hospitalization at the rate of δ. This compartment decreases due to natural deaths at rate μ. It also decreases due to recovery at the rate of θ. It also decreases due to recovery at the rate of α.

The compartment of recovered human individuals (R) increases due to monkeypox recovery at the rate of θ. This compartment decreases due to natural deaths at rate μ.

According to the assumptions and description of the model, the transmission diagram for the monkeypox infection model is shown in Figure 1.

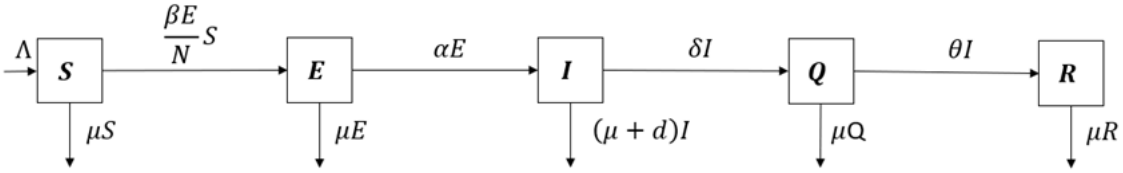

FIGURE 1. Transmission diagram

The system of differential equations for the monkeypox model, based on the given assumptions and descriptions, is as follows:

$$\begin{aligned}
\frac{dS}{dt} &= \Lambda - \frac{\beta S(t)E(t)}{N(t)} - \mu S(t),\\
\frac{dE}{dt} &= \frac{\beta S(t)E(t)}{N(t)} - (\alpha+\mu)E(t),\\
\frac{dI}{dt} &= \alpha E(t) - (\delta + d + \mu)I(t),\\
\frac{dQ}{dt} &= \delta I(t) - (\theta+\mu)Q(t),\\
\frac{dR}{dt} &= \theta Q(t) - \mu R(t).
\end{aligned}$$

Where $N(t) = S(t) + E(t) + I(t) + Q(t) + R(t)$.

The stability of the equilibrium and the basic reproduction number can be seen in [7]. The descriptions of the parameter values are provided in detail in Tables 1. The parameter values have been estimated from the data.

TABLE 1. Description of parameters

Parameter	Description	Value	Source
Λ	Recruitment rate	1500	[7]
β	Infection rate of monkeypox	0.09	[7]
α	Development rate of monkeypox symptoms	0.01	[7]
d	Death rate due to infection of monkeypox	0.5	[7]
μ	Natural death rate of human	0.4	[7]
δ	Hospitalization rate of monkeypox	0.05	[7]
θ	Recovery rate of monkeypox	0.05	[7]

3. RESULT

3.1. **Optimal Control.** The purpose of this optimal control is to minimize the spreading of monkeypox by reducing the symptomatic infected individuals. The hospitalization rate of monkeypox is considered as the control (u) of this model. The objective function as follow:

$$\min_u \int_0^{t_f} \left(AI(t) + Bu(t)^2\right) dt$$

with $0 \leq t \leq t_f$ and $0 \leq u(t) \leq 1$.

s.t

$$\begin{aligned}
\frac{dS}{dt} &= \Lambda - \frac{\beta S(t)E(t)}{N} - \mu S(t),\\
\frac{dE}{dt} &= \frac{\beta S(t)E(t)}{N} - (\alpha+\mu)E(t),\\
\frac{dI}{dt} &= \alpha E(t) - (u(t)+d+\mu)\, I(t),\\
\frac{dQ}{dt} &= u(t)I(t) - (\theta+\mu)Q(t),\\
\frac{dR}{dt} &= \theta Q(t) - \mu R(t),
\end{aligned}$$

where $S(t) \geq 0$, $E(t) \geq 0$, $I(t) \geq 0$, $Q(t) \geq 0$, $R(t)$. A and B are the weight of the asymptomatic and symptomatic infected individuals.

The Hamiltonian function for the optimal control model is given by the following equation:

$$\begin{aligned}
H = {} & AI(t) + Bu(t)^2 + \lambda_1 \left(\Lambda - \frac{\beta S(t)E(t)}{N} - \mu S(t)\right)\\
& + \lambda_2 \left(\frac{\beta S(t)E(t)}{N} - (\alpha+\mu)E(t)\right)\\
& + \lambda_3 \left(\alpha E(t) - (u(t)+d+\mu)I(t)\right)\\
& + \lambda_4 \left(u(t)I(t) - (\theta+\mu)Q(t)\right)\\
& + \lambda_5 \left(\theta Q(t) - \mu R(t)\right)
\end{aligned}$$

with λ_i for $i = 1, 2, \ldots, 5$ is the adjoint variable of $S(t), E(t), I(t), Q(t), R(t)$. The co-state equation of the optimal control model is given by the following equation:

$$\begin{aligned}
\frac{d}{dt}\lambda_1(t) &= -\lambda_1(t)\left(-\frac{\beta E(t)}{S(t)+E(t)+In(t)+Q(t)+R(t)} - \mu\right) - \frac{\lambda_2(t)\beta E(t)}{S(t)+E(t)+In(t)+Q(t)+R(t)}\\
\frac{d}{dt}\lambda_2(t) &= \frac{\lambda_1(t)\beta S(t)}{S(t)+E(t)+In(t)+Q(t)+R(t)} - \lambda_2(t)\left(\frac{\beta S(t)}{S(t)+E(t)+In(t)+Q(t)+R(t)} - \alpha - \mu\right) - \lambda_3(t)\alpha\\
\frac{d}{dt}\lambda_3(t) &= -A - \lambda_3(t)(-u(t)-d-\mu) - \lambda_4(t)u(t)\\
\frac{d}{dt}\lambda_4(t) &= -\lambda_4(t)(-\theta-\mu) - \lambda_5(t)\theta\\
\frac{d}{dt}\lambda_5(t) &= \lambda_5(t)\mu
\end{aligned}$$

The optimal condition of the optimal control for $0 \leq t \leq t_f$ is given as follows:

$$\frac{\partial H}{\partial u} = 0 \qquad \frac{\partial H}{\partial v} = 0$$

Since $0 \leq u(t) \leq 1$ and $0 \leq v(t) \leq 1$, we obtained the optimal control as follows:

$$u^*(t) = \min\left\{1, \max\left\{0, \frac{l(t)(\lambda_3 - \lambda_4)}{2B}\right\}\right\}$$

3.2. **Numerical Simulation.** Numerical simulations are used to observe population dynamics from the monkeypox model. The numerical simulations are categorized into two scenarios: First case is the simulation where there is no control in the model, also there will be two weight of the control, $B = 1$ and $B = 2000$. Second case is the simulation where there is control in the model. The initial values of the compartment are $S(0) = 90000$,$E(0) = 6000$,$I(0) = 4500$,$Q(0) = 4000$,and $R(0) = 3000$. The population dynamic can be seen in Figure 2-5.

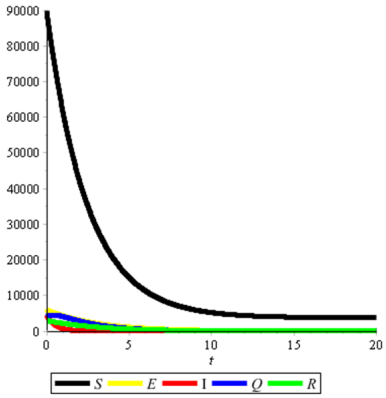

FIGURE 2. Population dynamic of SEIQR with control

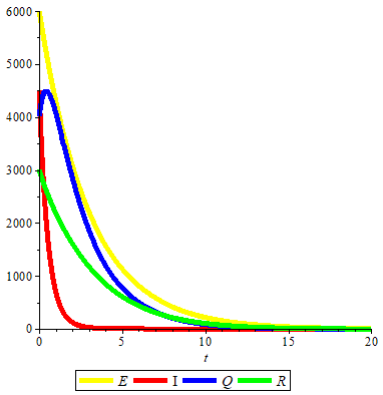

FIGURE 3. Population dynamic of EIQR with control

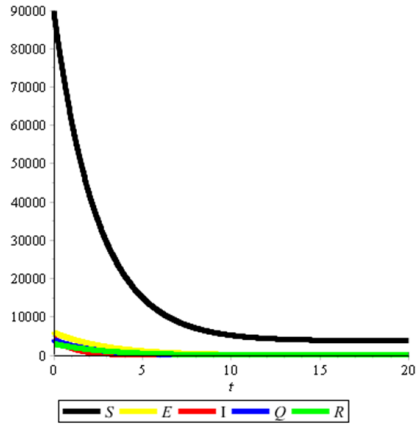

FIGURE 4. Population dynamic of SEIQR without control

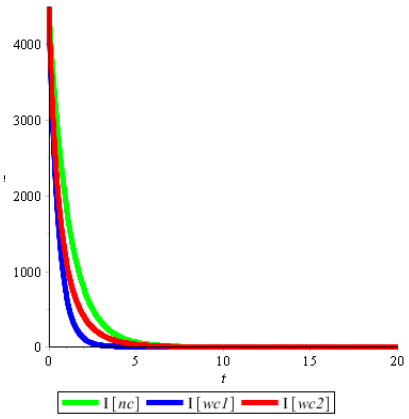

FIGURE 5. Comparison of symptomatic infected (I) with $(I[wc1], I[wc2])$ and without control$(I[nc])$.

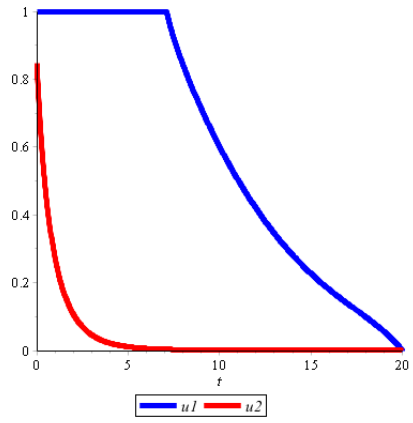

FIGURE 6. Control $u(t)$

Figure 2-3 show the dynamic population of the SEIQR monkeypox model with control. Based on Figure 2, all compartment decreases due to the parameter values which has basic

reproduction number less than one. In other words, the system is experiencing non-endemic conditions. Basic reproduction number is the threshold which show whether the disease will spread or extinct in the population. It can be seen more clearly in Figure 3 without susceptible individuals.

Meanwhile Figure 4 shows the dynamic of monkeypox in human population without control. The difference can be seen more clearly in Figure 5. There are two values of the control weight, $B = 1$ can be seen in control 1 (blue) and $B = 2000$ can be seen in control 2 (red). We can see that the symptomatic infected individuals with control ($I[wc1]$) and ($I[wc2]$) are decreasing faster than symptomatic infected individuals without control ($I[nc]$). It means, for these populations, hospitalization rate as a control (u) is the recommended solution to decrease the spreading of monkeypox in the population. Also, ($I[wc1]$) is decreasing faster than ($I[wc2]$), it means the smaller the control weight, the more effective the control is in reducing the spread of disease. The dynamic of the control can be seen in Figure 6. Control with $B = 1$ is more effective than $B = 2000$ because the control is fully used in the first half and then relaxed in the second half.

4. Conclusion

In this research, we investigated the spread of monkeypox in human populations. The model used is the monkeypox spread model by Smouni et al. We developed this model by modifying the hospitalization rate into a control to obtain the most optimal hospitalization rate level to reduce the spread of monkeypox. Based on the results of numerical simulations, changing the hospitalization parameters into a control can result in a faster reduction in the symptomatic infected compartment compared to without control. Also, by modifying the weight of the control, we found that the smaller the control weight, the more effective the control is in reducing the spread of disease. This model is a simple model with limitations, therefore, by adding various other parameters such as age structure, vaccination, or stochastic effects, the model can be further developed in the future.

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