

Mathematical Modeling of Monkeypox Transmission Dynamics: A Review

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Doi: https://doi.org/10.64891/jome.7

Abstract: The Monkeypox (Mpox) outbreak continues to pose a significant public health burden worldwide. This situation necessitates a deeper understanding of the disease's transmission and control mechanisms. Mathematical modeling serves as an effective tool for this purpose. The integration of vaccination, quarantine, isolation, and hospitalization strategies within these models highlights their critical role in mitigating the spread of Mpox. This review presents a comprehensive analysis of mathematical models developed to study the transmission dynamics and control of the Mpox virus, covering a broad spectrum from basic frameworks to more advanced models incorporating vaccination, quarantine, isolation, and hospitalization strategies. Key findings from the reviewed studies suggest that models integrating multiple simultaneous intervention strategies better represent the dynamics, but are relatively rare. Furthermore, few models consider bidirectional transmission routes between humans and animals; these are crucial for accurately characterizing Mpox dynamics. The inclusion of complex features such as fractional-order derivatives, risk group stratification, and optimal control analyses has been limited but demonstrates significant potential for more realistic scenario analysis. By synthesizing these findings, this review aims to inform researchers and policymakers in designing more effective intervention strategies to curb the spread of Mpox.

Keywords: Monkeypox mathematical model; Monkeypox transmission dynamics; Control strategies; Literature review.

AMS Math Codes: 34D20; 37N25; 92D30.

Introduction

Throughout human history, numerous outbreaks of infectious diseases have caused significant disruptions to public health, the economy, society, and the environment. Among the most well-known of these outbreaks are smallpox, HIV, Ebola, SARS, SARS-CoV-2, and, more recently, Monkeypox (Mpox). In recent years, the Mpox outbreak has emerged as a serious global health threat. Mpox is a zoonotic disease triggered by the Mpox virus, which belongs to the genus *Orthopoxvirus* in the *Poxviridae* family. The *Poxviridae* family is characterized by its double-stranded DNA genome [1]. Within this family, there are three additional viruses that are pathogenic to humans: the variola virus, the cowpox virus, and the vaccinia virus [2]. A distinctive clinical feature of Mpox in its early stages is lymphadenopathy [3]. The virus can spread to humans through close contact with an infected individual's body fluids, such as saliva, mucus, or

skin lesions. Common symptoms include fever, headache, muscle aches, skin lesions, and rashes. Swelling caused by the virus may appear on various parts of the body. Currently, there is no specific treatment for Mpox. However, supportive care, including medications to reduce fever and pain, can help manage the symptoms. While the smallpox vaccine offers partial protection against Mpox, its effectiveness is not guaranteed. Therefore, early detection of cases is crucial for controlling the spread of the disease [1].

The Mpox virus was first identified in 1958 in laboratory monkeys used for research at the State Serum Institutes in Copenhagen, Denmark, and Africa [4]. It was not recognized as a distinct human infection until 1970, during the global smallpox eradication campaign, when the virus was isolated from a patient in the Democratic Republic of the Congo who was initially suspected of having smallpox. This delay in recognition was due to the fact that many clinical features of Mpox infection in humans closely resemble those of smallpox [5]. The first outbreak outside of Africa occurred in the United States in 2003. Since 2005, thousands of cases have been reported annually in the Democratic Republic of the Congo. With the increase in international travel by infected individuals, the virus has rapidly spread to different regions. In May 2022, a new outbreak of Mpox affected several countries in Europe, the United States, and elsewhere. Between January 2022 and August 2024, over 100,000 cases were reported across more than 120 countries. On November 28, 2022, the World Health Organization (WHO) announced that the term "Mpox" would be used as the preferred name for the disease [6,7].

Although the Mpox virus has been isolated from several rodents and non-primate animals in Africa, the exact animal reservoir of the virus remains unknown. In addition to animal-to-animal transmission, animal-to-human transmission can occur through direct contact with infected animals, including touching, cleaning cages, hunting, processing meat, bites, or scratches. Human-to-human transmission occurs via respiratory droplets, direct contact, vertical transmission, percutaneous exposure, or indirect contact through fomites [8]. Furthermore, recent reports have presented evidence suggesting that the Mpox virus can also be transmitted from humans to animals. This possibility has raised concerns about the need to isolate pets from infected individuals and has sparked new discussions within the context of public health [9, 10].

Mathematical modeling has become an indispensable tool in the study of infectious disease dynamics. This approach provides a systematic framework to understand disease transmission mechanisms, evaluate the effectiveness of intervention strategies, and inform public health policymaking. However, despite the increasing number of mathematical models for Mpox, the literature has some limitations. Many studies focus on a single intervention such as vaccination, quarantine, isolation, and hospitalization but are not yet sufficient in terms of combined interventions or the examination of richer dynamics. Furthermore, the number of complex models that consider bidirectional transmission routes between humans and animals, which is necessary for the characterization of Mpox transmission, is quite low. Complex structures that include fractional dynamics, discrete dynamics, and risk group stratification are also not very common. By addressing these shortcomings and methodological limitations, the present review aims to provide a solid basis for comparing existing Mpox models and identify aspects that are overlooked in them, as well as inform future research directions in this area.

This study offers a comprehensive review of mathematical modeling approaches developed for Mpox, categorizing and analyzing models based on different methodological perspectives. The present review focuses specifically on the class of deterministic models, which include classical integer and fractional order models, and does not consider stochastic, agent-based, or purely data-driven modeling approaches. Mpox models are classified in Section 2 according to the number of incorporated control strategies, such as vaccination, quarantine, isolation, and hospitalization, grouped as single, double, or multiple intervention models. In Section 3, the key results and a summary of findings from the reviewed studies are presented, highlighting important trends, model features and insights. Section 4 provides a critical discussion of the strengths and limitations of the various modeling approaches, addresses impor-

tant methodological considerations. Finally, Section 5 concludes the study by summarizing the main outcomes and proposing specific, actionable recommendations for future research directions.

2 Classification of Mpox Models on Implemented Control Strategies

Zoonotic diseases often serve as the origin of pathogens that can effectively cross species barriers and transmit from human to human. In a study examining over 1400 pathogens known to cause infections in humans, it was found that 61% of them are zoonotic [11]. Modeling zoonotic diseases generally requires addressing transmission across multiple species. Mathematical models of Mpox typically consider both human and animal or rodent populations. Subsequently, a system of differential equations is developed to represent the transmission dynamics within and between these populations. Fractional-order differential equations have also been frequently employed in Mpox models.

In the literature, Mpox models typically include compartments representing the different disease stages within human and animal populations. These compartments generally consist of susceptible, vaccinated, exposed (infected but not infectious), infectious, quarantined, isolated, hospitalized, and recovered individuals, denoted respectively by the classes S, V, E, I, Q, J, H, and R. Subscripts h and a denote human and animal populations, respectively (e.g., S_h or S_a). Additional compartments have been incorporated in some studies based on further assumptions. In subsequent, the Mpox mathematical models from the literature are examined and categorized according to their number of control strategies included. Descriptions of frequently used parameters and compartments in these models are provided in Table 1. Parameters not included in Table 1 are either defined within the corresponding model descriptions or are used contextually in a self-explanatory manner. In particular, deterministic compartmental models will be presented. The characteristics of the models reviewed are summarized in Tables 2,3, and 4. This table allows a comparison of the models in terms of the compartments used, modeling approach, the control strategies implemented, and conducted analysis. The control strategies column considers vaccination, quarantine, isolation, and hospitalization. Since treatment strategies are addressed in most studies, they are not specified separately. On the other hand, the conducted analyses column includes the identification of the feasible region, the calculation of \mathfrak{R}_0 , stability analyses of equilibrium points, sensitivity analyses, bifurcation analyses, numerical analyses, and optimal control studies. As various simulations are provided in all studies, these are not specified separately. There, E_0 , E^* , and E^{**} denotes disease-free, endemic, and animal-disease-free equilibrium points, respectively.

2.1 Single-Strategy Models

This subsection discusses Mpox models that incorporate only a single control strategy, highlighting the key features and findings of the relevant studies.

Vaccination

Smallpox vaccines play a significant role in combating the Mpox outbreak. However, the effectiveness, side effects, and practical limitations of these vaccines restrict their protective function within certain populations. Therefore, there remains a need for safer and more effective vaccines [12]. Vaccination reduces the susceptible population by inducing immunity. Therefore, in the long run, it reduces epidemic spread and potential and contributes to herd immunity. Numerous mathematical modeling studies have investigated the impact of vaccination programs on the dynamics of the Mpox virus within populations. The first mathematical modeling study examining Mpox dynamics, which includes

Parameter	Description	Compartment	Description
Λ_h	Recruitment rate of human	S_h	Susceptible humans
Λ_a	Recruitment rate of animal	S_{h_1}	Low-risk susceptible humans
μ_h	Natural death rate of human	S_{h_2}	High-risk susceptible humans
μ_a	Natural death rate of animal	V_h	Vaccinated humans
δ_h	Disease-related death rate of human	E_h	Exposed humans
δ_a	Disease-related death rate of animal	I_h	Infected humans
eta_1	Animal-to-human infection rate	I_{h_1}	Asymptomatic infected humans
eta_2	Human-to-human infection rate	I_{h_2}	Symptomatic infected humans
eta_3	Animal-to-animal infection rate	Q_h	Quarantined humans
eta_4	Human-to-animal infection rate	J_h	Isolated humans
p	Proportion of successful vaccinated human	H_h	Hospitalized humans
q	Proportion of unsuccessful vaccinated human	R_h	Recovered humans
$lpha_1$	Transition rate of humans from exposed to infected class	T_h	Treated humans
α_2	Transition rate of animals from exposed to infected class	B	Contaminated environment
α_3	Transition rate of humans from exposed to quarantined class	S_a	Susceptible animals
γ_h	Recovery rate of infected human	E_a	Exposed animals
γ_a	Recovery rate of infected animal	I_a	Infected animals
heta	Recovery rate of quarantined human	R_a	Recovered animals
au	Transition rate of humans from infected to quarantined class		

Table 1: Descriptions of the parameters and compartments included in the models.

vaccination and treatment interventions, was conducted by [13]. They developed the following model to analyze the transmission dynamics of the Mpox virus:

$$\frac{dS_{h}}{dt} = \Lambda_{h} - \frac{\beta_{1}I_{a}S_{h}}{N_{a}} - \frac{\beta_{2}I_{h}S_{h}}{N_{h}} - (\mu_{h} + p)S_{h}, \quad \frac{dS_{a}}{dt} = \Lambda_{a} - \frac{\beta_{3}S_{a}I_{a}}{N_{a}} - \mu_{a}S_{a},
\frac{dV_{h}}{dt} = pS_{h} - \mu_{h}V_{h}, \qquad \frac{dE_{a}}{dt} = \frac{\beta_{3}S_{a}I_{a}}{N_{a}} - (\mu_{a} + \alpha_{2})E_{a},
\frac{dE_{h}}{dt} = \frac{\beta_{1}I_{a}S_{h}}{N_{a}} + \frac{\beta_{2}I_{h}S_{h}}{N_{h}} - (\mu_{h} + \alpha_{1})E_{h}, \qquad \frac{dI_{a}}{dt} = \alpha_{2}E_{a} - (\mu_{a} + \delta_{a} + \gamma_{a})I_{a},
\frac{dI_{h}}{dt} = \alpha_{1}E_{h} - (\mu_{h} + \gamma_{h} + \delta_{h})I_{h}, \qquad \frac{dR_{a}}{dt} = \gamma_{a}I_{a} - \mu_{a}R_{a},
\frac{dR_{h}}{dt} = \gamma_{h}I_{h} - \mu_{h}R_{h}. \tag{2.1}$$

In this model, vaccinated susceptible individuals move to the vaccinated class V_h , where they are assumed to acquire permanent immunity against the disease. The reproduction numbers for the human and animal populations are respectively computed as

$$\mathfrak{R}_{0}^{h} = \frac{\alpha_{1}\beta_{2}\mu_{h}}{(\mu_{h} + \gamma_{h} + \delta_{h})(\mu_{h} + \alpha_{1})(p + \mu_{h})}, \quad \mathfrak{R}_{0}^{a} = \frac{\alpha_{2}\beta_{3}}{(\mu_{a} + \delta_{a} + \gamma_{a})(\mu_{a} + \alpha_{2})},$$

and the overall basic reproduction number is given by $\mathfrak{R}_0 = \max\{\mathfrak{R}_0^h, \mathfrak{R}_0^a\}$. The study concluded that if $\mathfrak{R}_0 < 1$, the disease can be eradicated from both human and animal populations. Sensitivity analyses were performed on the parameters within \mathfrak{R}_0 to quantify the positive impact of vaccination and treatment strategies in reducing the value of reproduction number. It has been stated that with treatment and vaccination measures, it is possible to eliminate the disease from both human and animal populations over time.

Another vaccination-included mathematical model was proposed in [14], where exposure states in humans were

not considered:

$$\begin{split} \frac{dS_h}{dt} &= \Lambda_h - \frac{(\beta_1 I_a + \beta_2 I_h) S_h}{N_h} - (p + \mu_h) S_h + q V_h, & \frac{dS_a}{dt} &= \Lambda_a - \frac{\beta_3 S_a I_a}{N_a} - \mu_a S_a, \\ \frac{dV_h}{dt} &= p S_h - (q + \mu_h) V_h, & \frac{dI_a}{dt} &= \frac{\beta_3 S_a I_a}{N_a} - (\gamma_a + \delta_a + \mu_a) I_a, \\ \frac{dI_h}{dt} &= \frac{(\beta_1 I_a + \beta_2 I_h) S_h}{N_h} - (\gamma_h + \mu_h + \delta_h) I_h, & \frac{dR_a}{dt} &= \gamma_a I_a - \mu_a R_a, \\ \frac{dR_h}{dt} &= \gamma_h I_h + (1 - q) V_h - \mu_h R_h. \end{split}$$

Here, the successful vaccination rate among vaccinated susceptible individuals is represented by 1-q, with a fraction q failing vaccination and reverting to the susceptible class. The basic reproduction number is derived as

$$\mathfrak{R}_0 = \frac{\beta_2 \beta_3 \Lambda_h \lambda_a (q + \mu_h)}{N_h N_a \mu_h (\gamma_h + \mu_h + \delta_h) (p + q + \mu_h) (\delta_a + \mu_a) (\gamma_a + \mu_a)},$$

where N_h and N_a denote the total human and animal population sizes, respectively. The model concluded that when $\mathfrak{R}_0 < 1$, eradication of the disease from both populations is achievable. Sensitivity analyses of parameters were also conducted. It is suggested that individuals with a strong immune system are likely to recover more rapidly than those with a moderate immune system, while individuals with a weak immune system may recover much more slowly. Moreover, it is emphasized that individuals can play an active role in strengthening their immune system by maintaining a healthy weight, engaging in regular physical exercise, ensuring adequate sleep, and consuming healthy food and clean water. Consequently, it was noted that governments have a crucial role to play in supporting these aspects of public health.

A more complex model examining the effect of vaccination was introduced in [15]:

$$\frac{dS_h}{dt} = (1 - f)\Lambda_h - \left(\frac{\beta_1(\varepsilon_a E_a + I_a)}{N_a} + \frac{\beta_2(\varepsilon_h E_h + I_h)}{N_h}\right) S_h \qquad \frac{dS_a}{dt} = \Lambda_a - \frac{\beta_3(\varepsilon_p E_a + I_a) S_a}{N_a} - \mu_a S_a,$$

$$- (p + \mu_h) S_h + q V_h, \qquad \frac{dE_a}{dt} = \frac{\beta_3(\varepsilon_a E_a + I_a) S_a}{N_a} - (\alpha_2 + \mu_a) E_a,$$

$$\frac{dV_h}{dt} = f \Lambda_h + p S_h - (q + \mu_h) V_h, \qquad \frac{dI_a}{dt} = \alpha_2 E_a - (\gamma_a + \mu_a + \delta_a) I_a,$$

$$\frac{dE_h}{dt} = \frac{\beta_1(\varepsilon_a E_a + I_a) S_h}{N_a} + \frac{\beta_2(\varepsilon_h E_h + I_h) S_h}{N_h} - (\alpha_1 + \mu_h) E_h, \qquad \frac{dR_a}{dt} = \gamma_a I_a - \mu_a R_a,$$

$$\frac{dI_h}{dt} = \alpha_1 E_h - (\gamma_h + \mu_h + \delta_h) I_h,$$

$$\frac{dR_h}{dt} = \gamma_h I_h - \mu_h R_h.$$

Unlike the previous models, this one considers the possibility of transmission via interactions between susceptible and exposed individuals. Parameters ε_h and ε_a represent the reduced infectiousness of exposed humans (E_h) and animals (E_a) compared to infectious individuals (I_h) and I_a . Furthermore, a fraction f of recruits entering the population are assumed to be vaccinated and thus enter directly into the vaccinated class V_h . The basic reproduction number of this model is considerably more complex to compared to the previous ones. It is concluded that reducing the basic reproduction number below one will eliminate the disease from the human population.

Recognizing that susceptible individuals may vary in their defense mechanisms and susceptibility to infection, [16]

proposed a model that stratifies susceptible humans into low-risk (S_{h_1}) , and high-risk (S_{h_2}) groups:

$$\begin{split} \frac{dS_{h_1}}{dt} &= \Lambda_{h_1} - \frac{(\beta_1 I_a + \beta_2 I_h) S_{h_1}}{1 + \lambda I_h^2} - (p + \mu_h) S_{h_1} + q_1 V_h, & \frac{dS_a}{dt} &= \Lambda_a - \beta_3 S_a I_a - \mu_a S_a, \\ \frac{dS_{h_2}}{dt} &= \Lambda_{h_2} - \frac{(\beta_5 I_a + \beta_6 I_h) S_{h_2}}{1 + \lambda I_h^2} - (p + \mu_h) S_{h_2} + q_2 V_h, & \frac{dI_a}{dt} &= \beta_3 S_a I_a - (\gamma_a + \mu_a + \delta_a) I_a, \\ \frac{dV_h}{dt} &= p(S_{h_1} + S_{h_2}) - (q_1 + q_2 + \mu_h) V_h, & \frac{dR_a}{dt} &= \gamma_a I_a - \mu_a R_a, \\ \frac{dI_h}{dt} &= \frac{(\beta_1 I_a + \beta_2 I_h) S_{h_1}}{1 + \lambda I_h^2} + \frac{(\beta_5 I_a + \beta_6 I_h) S_{h_2}}{1 + \lambda I_h^2} - (\gamma_h + \mu_h + \delta_h) I_h, \\ \frac{dR_h}{dt} &= \gamma_h I_h + (1 - q_1 - q_2) V_h - \mu_h R_h. \end{split}$$

This model also incorporates imperfect vaccination, with q_1 and q_2 representing the failure rates of vaccination among low-risk and high-risk groups, respectively. The infection rates from animals to low-risk and high-risk humans are denoted by β_1 and β_5 , respectively. Additionally, the incidence rate is assumed to be of the form $\frac{kI}{1+\lambda I^2}$, where the term $1+\lambda I^2$ captures the inhibitory effect-often referred to as a "psychological effect"-induced by government interventions such as isolation, quarantine, and public transportation restrictions. The study conducted a detailed dynamical analysis, examining the existence, uniqueness, positivity, boundedness of solutions, and dependency on initial conditions. The basic reproduction number is computed as

$$\mathfrak{R}_0 = \max \left\{ \frac{\beta_2 + \beta_4}{\gamma_h + \mu_h + \delta_h}, \frac{\beta_5}{\gamma_a + \mu_a + \delta_a} \right\}.$$

Stability and bifurcation analyses were performed, and numerical simulations were presented. It was predicted that an increase in vaccine imperfection rates would lead to a significant decrease in the number of immune individuals capable of resisting the disease. In general, it is found that vaccination programs help establish high immunity levels within populations and reduce the number of individuals susceptible to the disease.

Numerous other studies have investigated the impact of vaccination as a single control strategy on Mpox population dynamics. For instance, [17] extended model (2.1) to include temporary immunity. The same model was also analyzed with fractional derivatives in [18]. Game-theoretic approaches to evaluate vaccination strategies were explored in [19]. Additionally, several studies focused exclusively on the transmission dynamics of Mpox within human populations and the role of vaccination [20,21]. The environmental contribution to Mpox transmission has been investigated in [22]. In addition, an age-structured modeling approach was applied in the study [23].

Quarantine

Several countries have implemented quarantine measures to mitigate the spread of Mpox. Belgium was the first to enforce a mandatory 21-day quarantine for infected individuals. The United Kingdom, on the other hand, has recommended that individuals who have had direct or household contact with confirmed cases self-isolate for 21 days [24]. Quarantine (and isolation) practices immediately break the chain of transmission by isolating infected or exposed individuals from the community. Therefore, a rapid decrease in the number of cases can be achieved in the short term. The effects of quarantine interventions have been incorporated into numerous mathematical models of

Mpox transmission. In [25], a model that accounts for quarantined individuals was proposed as follows:

$$\begin{split} \frac{dS_h}{dt} &= \Lambda_h - (1 - \varepsilon) \frac{(\beta_1 I_a + \beta_2 I_h) S_h}{N_h} - \mu_h S_h, & \frac{dS_a}{dt} &= \Lambda_a - \frac{\beta_3 S_a I_a}{N_a} - \mu_a S_a, \\ \frac{dI_h}{dt} &= (1 - \varepsilon) \frac{(\beta_1 I_a + \beta_2 I_h) S_h}{N_h} - (\mu_h + \delta_h + \tau) I_h, & \frac{dI_a}{dt} &= \frac{\beta_3 S_a I_a}{N_a} - (\mu_a + \delta_a) I_a, \\ \frac{dQ_h}{dt} &= \tau I_h - (\mu_h + (1 - \xi) \delta_h + \theta) Q_h, \\ \frac{dR_h}{dt} &= \theta Q_h - \mu_h R_h. \end{split}$$

In this model, $0 \le \varepsilon \le 1$ represents the effectiveness of awareness campaigns, while $0 \le \xi \le 1$ captures the efficacy of quarantine and treatment. The reproduction numbers for humans and animals are computed as:

$$\mathfrak{R}_0^h = \frac{(1-\varepsilon)\beta_2}{\mu_h + \delta_h + \tau}, \quad \mathfrak{R}_0^a = \frac{\beta_3}{\mu_a + \delta_a}.$$

The disease is expected to be eradicated when both $\mathfrak{R}_0^h < 1$ and $\mathfrak{R}_0^a < 1$. It was concluded that public awareness campaigns and the isolation of infected individuals from susceptibles can significantly reduce the spread of the disease.

The model presented in [26] incorporates the saturation effect in disease transmission:

$$\begin{split} \frac{dS_h}{dt} &= \Lambda_h - \frac{(\beta_1 I_a + \beta_2 I_h) S_h}{1 + \lambda I_h} - \mu_h S_h, & \frac{dS_a}{dt} &= \Lambda_a - \frac{\beta_3 S_a I_a}{1 + \lambda I_a} - \mu_a S_a, \\ \frac{dI_h}{dt} &= \frac{(\beta_1 I_a + \beta_2 I_h) S_h}{1 + \lambda I_h} - (\mu_h + \delta_h + \tau) I_h, & \frac{dI_a}{dt} &= \frac{\beta_3 S_a I_a}{1 + \lambda I_a} - (\mu_a + \delta_a) I_a, \\ \frac{dQ_h}{dt} &= \tau I_h - (\mu_h + \delta_h + \theta) Q_h, \\ \frac{dR_h}{dt} &= \theta Q_h - \mu_h R_h. \end{split}$$

The parameter λ reflects the strength of government interventions such as public restrictions. A detailed dynamical analysis of the model was conducted. Human and animal reproduction numbers were given as:

$$\mathfrak{R}_0^h = \frac{\beta_2 \Lambda_h}{\mu_h (\mu_h + \delta_h + \tau)}, \quad \mathfrak{R}_0^a = \frac{\beta_3 \Lambda_a}{\mu_a (\mu_a + \delta_a)},$$

and the basic reproduction number defined as $\max\{\mathfrak{R}_0^h,\mathfrak{R}_0^a\}$. Three possible equilibrium states were identified: disease eradication in both populations, persistence in the human population only, and persistence in both populations. A comprehensive stability analysis performed, and the presence of forward bifurcation at $\mathfrak{R}_0^h=1$ and $\mathfrak{R}_0^a=1$ established. Sensitivity analysis quantified the influence of parameters on \mathfrak{R}_0 , and all theoretical results supported with simulations. The effectiveness of government interventions in reducing the number of infected humans and animals was highlighted. Moreover, it was emphasized that minimizing contact with infected humans and animals, as well as implementing strategies to reduce migration rates into the population, can have a significant impact on controlling the disease.

In most of the models existing in the literature, Mpox virus transmission has considered thorough human-to-human, animal-to-human, and animal-to-animal routes. However, as previously noted, there are several reports and studies indicating the possibility of human-to-animal transmission of the Mpox virus, particularly from humans to domestic pets [9, 10]. Based on these facts, some mathematical models have been developed to incorporate all possible

transmission routes. For example, in [27], the following mathematical model was proposed:

$$\begin{split} \frac{dS_h}{dt} &= \Lambda_h - (\beta_1 I_a + \beta_2 I_h) S_h - \mu_h S_h - \varphi Q_h, \\ \frac{dI_h}{dt} &= (\beta_1 I_a + \beta_2 I_h) S_h - (\mu_h + \delta_{h1} + \alpha_4 + \gamma_h + \gamma_t) I_h, \\ \frac{dI_a}{dt} &= (\beta_3 I_a + \beta_4 I_h) S_a - \mu_a I_a, \\ \frac{dQ_h}{dt} &= \alpha_4 I_h - (\mu_h + \delta_{h2} + \theta + \varphi) Q_h, \\ \frac{dR_h}{dt} &= (\gamma_h + \gamma_t) I_h + \theta Q_h - \mu_h R_h. \end{split}$$

In this model, infected individuals recover either through natural immunity at rate γ_h or via treatment at rate γ_t . A fractional-order dynamical analysis performed, and results validated through numerical simulations. With this model, which includes all routes of transmission, the authors aimed to prove more realistic predictions about the spread of the disease.

In [28], different disease-induced mortality rates for infected and quarantine individuals (δ_{h1} and δ_{h2} , respectively) considered. The proposed model is given by:

$$\frac{dS_{h}}{dt} = \Lambda_{h} - \frac{\beta_{1}I_{a}S_{h}}{N_{a}} - \frac{\beta_{2}I_{h}S_{h}}{N_{h}} - \mu_{h}S_{h}, \qquad \frac{dS_{a}}{dt} = \Lambda_{a} - \frac{\beta_{3}I_{a}S_{a}}{N_{a}} - \mu_{a}S_{a},$$

$$\frac{dE_{h}}{dt} = \frac{\beta_{1}I_{a}S_{h}}{N_{a}} + \frac{\beta_{2}I_{h}S_{h}}{N_{h}} - (\alpha_{1} + \alpha_{3} + \mu_{h})E_{h}, \qquad \frac{dE_{a}}{dt} = \frac{\beta_{3}I_{a}S_{a}}{N_{a}} - (\mu_{a} + \alpha_{2})E_{a},$$

$$\frac{dI_{h}}{dt} = \alpha_{1}E_{h} - (\mu_{h} + \delta_{h1} + \gamma_{h})I_{h}, \qquad \frac{dI_{a}}{dt} = \alpha_{2}E_{a} - \mu_{a}I_{a},$$

$$\frac{dQ_{h}}{dt} = \alpha_{3}E_{h} - (\theta + \delta_{h2} + \mu_{h})Q_{h},$$

$$\frac{dR_{h}}{dt} = \gamma_{h}I_{h} + \theta Q_{h} - \mu_{h}R_{h}.$$

The study used actual Mpox outbreak data from the United States. Human and animal reproduction numbers calculated as:

$$\mathfrak{R}_0^h = \frac{\beta_2 \alpha_1}{(\alpha_1 + \alpha_3 + \mu_h)(\mu_h + \delta_{h1} + \gamma_h)}, \quad \mathfrak{R}_0^a = \frac{\beta_3 \alpha_2}{\mu_a(\mu_a + \alpha_2)},$$

and the basic reproduction number defined as $\mathfrak{R}_0 = \max\{\mathfrak{R}_0^h, \mathfrak{R}_0^a\}$. Stability analysis of the equilibria performed, and backward bifurcation observed at $\mathfrak{R}_0 = 1$. Sensitivity analysis also conducted. Results indicated that reducing contact between humans and animals by eliminating their food source, water, and shelter, regularly disposing of trash, and decreasing human-to-human transmission can all lead to a decrease in future cases. Avoiding contact with infected animals and humans, washing hands with soap and water after any contact, and separating infected patients from healthy individuals are recommended.

Since individuals quarantined due to suspected Mpox infection may or may not be actually infected, [29] proposed a model that incorporates the diagnostic process. Some suspected cases may test negative and return to the susceptible

class:

$$\begin{split} \frac{dS_h}{dt} &= \Lambda_h - \frac{(\beta_1 I_a + \beta_2 I_h)S_h}{N_h} - \mu_h S_h + \varphi Q_h, & \frac{dS_a}{dt} &= \Lambda_a - \frac{\beta_3 S_a I_a}{N_a} - \mu_a S_a, \\ \frac{dE_h}{dt} &= \frac{(\beta_1 I_a + \beta_2 I_h)S_h}{N_h} - (\alpha_1 + \alpha_3 + \mu_h)E_h, & \frac{dE_a}{dt} &= \frac{\beta_3 S_a I_a}{N_a} - (\mu_a + \alpha_2)E_a, \\ \frac{dI_h}{dt} &= \alpha_1 E_h - (\mu_h + \delta_h + \gamma_h)I_h, & \frac{dI_a}{dt} &= \alpha_2 E_a - (\mu_a + \delta_a)I_a, \\ \frac{dQ_h}{dt} &= \alpha_3 E_h - (\varphi + \theta + \delta_h + \mu_h)Q_h, \\ \frac{dR_h}{dt} &= \gamma_h I_h + \theta Q_h - \mu_h R_h. \end{split}$$

The rate at which individuals in quarantine return to the susceptible class after testing negative is denoted by φ . The basic reproduction number is given by:

$$\mathfrak{R}_0 = \frac{\alpha_1 \beta_2}{(\alpha_1 + \alpha_3 + \mu_h)(\mu_h + \delta_h + \gamma_h)}.$$

The stability of equilibria investigated, and a backward bifurcation identified at $\mathfrak{R}_0 = 1$. Sensitivity analysis demonstrated the impact of various parameters on \mathfrak{R}_0 , highlighting the importance of isolation in controlling disease transmission. It was emphasized that the isolation of infected individuals plays a crucial role in reducing disease spread and in helping to keep the virus under control.

Several other studies have explored the impact of quarantine measures on the transmission dynamics of Mpox. In [30], four different control measures that can be used to prevent the spread of the virus were considered. It was found that an optimal control scheme can help reduce the number of infected, quarantined, and exposed individuals while increasing the number of susceptible and recovered individuals. In [31], it was discovered that the speed at which humans and animals progress from the exposure stage to the infectious stage is also important factor and may pose a significant risk by increasing the overall level of Mpox infection. In [32], a fractional order mathematical model was developed to investigate the co-infection dynamics of Mpox and HIV/AIDS. The findings suggest that an optimal vaccination strategy combined with improved HIV management could significantly reduce co-infection rates. In [33], the importance of reducing contacts not only among susceptible and infected humans but also between rodents is emphasized. The study highlights the necessity of encouraging treatment and medical interventions. The same model was revisited in [34], where an approximate solution was obtained using a mathematical technique known as the Laplace Adomain Decomposition Method. This method enables the prediction of outcomes for various disease management strategies. Additional studies that consider quarantine as a single control strategy can be found in [35–44].

Hospitalization

Hospitalization is another important control strategy that has been incorporated into several mathematical models of Mpox transmission. This subsection summarizes representative studies that have explicitly considered hospitalization as a single control measure. The study given in [45], the following extended model, considering the human-to-animal

transmission, was analyzed:

$$\begin{split} \frac{dS_h}{dt} &= \Lambda_h - \frac{(\beta_1 I_a + \beta_2 I_{h_1} + \beta_3 I_{h_2}) S_h}{N_h} - \mu_h S_h, & \frac{dS_a}{dt} &= \Lambda_a - \frac{\beta_4 I_a S_a}{N_a} - \frac{(\beta_5 I_{h_1} + \beta_6 I_{h_2}) S_a}{N_h} - \mu_a S_a, \\ \frac{dE_h}{dt} &= \frac{(\beta_1 I_a + \beta_2 I_{h_1} + \beta_3 I_{h_2}) S_h}{N_h} - (\mu_h + \alpha_1) E_h, & \frac{dI_a}{dt} &= \frac{\beta_4 I_a S_a}{N_a} + \frac{(\beta_5 I_{h_1} + \beta_6 I_{h_2}) S_a}{N_h} - \mu_a I_a, \\ \frac{dI_{h_1}}{dt} &= \alpha_1 (1 - \epsilon) E - (\mu_h + \omega_1 + \alpha_5) I_{h_1}, \\ \frac{dI_{h_2}}{dt} &= \alpha_1 \epsilon E + \omega_1 I_{h_1} - (\mu_h + \alpha_8 + \delta_h) I_{h_2}, \\ \frac{dH_h}{dt} &= \alpha_5 I_{h_1} + \alpha_8 I_{h_2} - (\mu_h + \eta) H_h, \\ \frac{dR_h}{dt} &= \eta H_h - \mu_h R_h. \end{split}$$

This model investigates the impact of awareness programs and effective treatment strategies aimed at reducing the risk of Mpox transmission. The study focused on optimal control strategies designed to reduce the number of exposed and infected individuals. The effectiveness of the proposed strategies validated thorough numerical simulations, high-lighting their critical role in controlling the spread of the virus. The study highlights the importance of the model for key factors in virus control, such as public health interventions and proactive treatments.

In [46], the human population was divided into high-risk and low-risk groups. Additionally, infection stages were distinguished by asymptomatic, mild symptomatic, and severe symptomatic phases:

$$\begin{split} \frac{dS_{h_1}}{dt} &= (1-\pi)\Lambda_h - \left(\frac{\beta_1 I_a}{N_a} + \frac{\beta_2 (aP + \eta I_{h_1} + I_{h_2})}{N_h}\right) \nu S_{h_1} - \mu_h S_{h_1}, & \frac{dS_a}{dt} &= \Lambda_a - \frac{\beta_3 I_a S_a}{N_a} - \mu_a S_a, \\ \frac{dS_{h_2}}{dt} &= \pi \Lambda_h - \left(\frac{\beta_1 I_a}{N_a} + \frac{\beta_2 (aP + \eta I_{h_1} + I_{h_2})}{N_h}\right) S_{h_2} - \mu_h S_{h_2}, & \frac{dE_a}{dt} &= \frac{\beta_3 I_a S_a}{N_a} - (\mu_a + \alpha_2) E_a, \\ \frac{dE_h}{dt} &= \left(\frac{\beta_1 I_a}{N_a} + \frac{\beta_2 (aP + \eta I_{h_1} + I_{h_2})}{N_h}\right) (\nu S_{h_1} + S_{h_2}) - (\mu_h + \alpha_6) E_h, & \frac{dI_a}{dt} &= \alpha_2 E_a - (\mu_a + \delta_a + \gamma_a) I_a, \\ \frac{dP}{dt} &= \alpha_6 E_h - (\mu_h + \alpha_7) P, & \frac{dR_a}{dt} &= \gamma_a I_a - \mu_a R_a, \\ \frac{dI_{h_1}}{dt} &= \alpha_7 P - (\mu_h + \omega_1 + \alpha_5 + \gamma_1) I_{h_1}, & \\ \frac{dI_{h_2}}{dt} &= \omega_1 I_{h_1} - (\mu_h + \delta_{h_1} + \alpha_8 + \gamma_2) I_{h_2}, \\ \frac{dH_h}{dt} &= \alpha_5 I_{h_1} + \alpha_8 I_{h_2} - (\mu_h + \delta_{h_2} + \eta) H_h, & \\ \frac{dR_h}{dt} &= \gamma_1 I_{h_1} + \gamma_2 I_{h_2} + \eta H - \mu_h R_h. \end{split}$$

In this model, S_{h1} and S_{h2} represent the low-risk and high-risk susceptible humans, respectively. P denotes the asymptomatic infectious class, while I_{h_1} and I_{h_2} represent mild and severe symptomatic individuals. A dynamical analysis, including stability, bifurcation, and sensitivity, conducted. Numerical simulations demonstrated that high-risk regions without adequate intervention face elevated outbreak risks. Contact tracing and early detection in these groups shown to be effective in mitigating Mpox spread. The importance of surveillance in animal populations also emphasized. Additional Mpox models that focus exclusively on hospitalization as a single control measure can be found in [47–49].

Isolation

Isolation is another relevant control strategy that has been incorporated into Mpox transmission models. It aims to reduce human-to-human transmission by identifying and separating infected individuals from the susceptible population. To date, the only mathematical model that exclusively considers isolation is presented in [50]:

$$\begin{split} \frac{dS_h}{dt} &= \Lambda_h - \frac{(\beta_1 I_a + \beta_2 (I_h + \theta_m J_h)) S_h}{N_h} - \mu_h S_h, & \frac{dS_a}{dt} &= \Lambda_a - \frac{\beta_3 I_a S_a}{N_a} - \mu_a S_a, \\ \frac{dE_h}{dt} &= \frac{(\beta_1 I_a + \beta_2 (I_h + \theta_m J_h)) S_h}{N_h} - (\mu_h + \alpha_1) E_h, & \frac{dI_a}{dt} &= \frac{\beta_3 I_a S_a}{N_a} - \mu_a I_a, \\ \frac{dI_h}{dt} &= \alpha_1 E_h - (\mu_h + \delta_h + d\theta_h + k) I_h, \\ \frac{dJ_h}{dt} &= k I_h - (d + \mu_h) J_h, \\ \frac{dR_h}{dt} &= d\theta_h I_h + dJ_h - \mu_h R_h. \end{split}$$

Here k denotes the isolation rate based on surveillance and contact tracing efforts. The parameter d represents the natural recovery rate of infected individuals as well as recovery rate of isolated individuals through treatment. θ_h is a modification parameter for the recovery rate of infected individuals, while θ_m is a modification parameter that adjusts the transmission rate of isolated individuals. The fundamental dynamics of the model were analyzed basically. An optimal control study also carried out by incorporating four control variables aimed at preventing Mpox transmission from animals to humans and from human to human, through preventive measures, isolation of infected individuals via contact tracing, and treatment of isolated individuals. Furthermore, a cost-effectiveness analysis was performed to identify the most-effective control strategy among all possible combinations of these control measures. The results suggest that strategies to prevent animal to human transmission are the most economical and effective approach.

2.2 Double-Strategy Models

In many studies, models have been proposed that incorporate two or more of the control strategies discussed in the previous subsection. For example, in [51], a model is proposed that includes the control strategies of both vaccination and quarantine:

$$\begin{split} \frac{dS_h}{dt} &= \Lambda_h - (\beta_1 I_a + \beta_2 I_h) S_h - (\mu_h + r_1 u_1) S_h + \sigma R_h, & \frac{dS_a}{dt} &= \Lambda_a - \beta_3 S_a I_a + \xi I_a - \mu_a S_a, \\ \frac{dE_h}{dt} &= (\beta_1 I_a + \beta_2 I_h) S_h - (\alpha_1 + \alpha_3 + \mu_h) E_h, & \frac{dI_a}{dt} &= \beta_3 S_a I_a - (\xi + \mu_a + \delta_a) I_a, \\ \frac{dI_h}{dt} &= \alpha_1 E_h - (\gamma_h + r_2 u_2 + \mu_h + \delta_{h1}) I_h, & \\ \frac{dQ_h}{dt} &= \alpha_3 E_h - (\theta + \mu_h + \delta_{h2}) Q_h, & \\ \frac{dR_h}{dt} &= r_1 u_1 S_h + (\gamma_h + r_2 u_2) I_h + \theta Q_h - (\sigma + \mu_h) R_h. & \end{split}$$

Here, u_1 and u_2 are control variables representing vaccination and treatment, respectively. The parameters r_1 and r_2 denote the effectiveness of these two controls. It is assumed that recovered individuals gain temporary immunity, and a fraction σ of them return to the susceptible class after losing immunity. A fractional-order version of the model is also developed and analyzed. The basic reproduction number \mathfrak{R}_0 is computed, and its role as a threshold parameter

in determining system stability is demonstrated. It is shown that transcritical bifurcations may occur at $\mathfrak{R}_0^h = 1$ and $\mathfrak{R}_0^a = 1$. Optimal control theory is employed to derive strategies that minimize both disease prevalence and the cost of implementing controls. Sensitivity analysis reveals the influence of key parameters on \mathfrak{R}_0 . All findings are supported by numerical simulations showing that treatment and vaccination, as well as quarantine measures, significantly reduce Mpox transmission in the human population.

In [52], a mathematical model including vaccination and quarantine compartments is proposed:

$$\begin{split} \frac{dS_h}{dt} &= \Lambda_h - \frac{(\beta_1 I_a + \beta_2 I_h) S_h}{N_h} - (\mu_h + p) S_h, \quad \frac{dS_a}{dt} = \Lambda_a - \frac{\beta_3 S_a I_a}{N_a} - \mu_a S_a, \\ \frac{dV_h}{dt} &= p S_h - \mu_h V_h, \qquad \qquad \frac{dI_a}{dt} = \frac{\beta_3 S_a I_a}{N_a} - \mu_a I_a, \\ \frac{dE_h}{dt} &= \frac{(\beta_1 I_a + \beta_2 I_h) S_h}{N_h} - (\alpha_1 + \mu_h) E_h, \\ \frac{dI_h}{dt} &= \alpha_1 E_h - (\mu_h + \delta_h + \gamma_h + \alpha_4) I_h, \\ \frac{dQ_h}{dt} &= \alpha_4 I_h - (\theta + \mu_h + \delta_h) Q_h, \\ \frac{dR_h}{dt} &= \theta Q_h + \gamma_h I_h - \mu_h R_h. \end{split}$$

A fuzzy fractional-order analysis of this model is conducted. The significance of fuzzy fractional differential equations stems from their capacity to more accurately describe transmission dynamics by accounting for non-local effects, which capture the memory and hereditary characteristics inherent in the spread of infectious diseases. This study highlights the significant role of vaccination in reducing disease transmission, demonstrating the practical usefulness of fuzzy fractional techniques in epidemiological modeling.

In [53], a model is proposed in which susceptible humans can become infected not only through contact with infectious individuals but also through environmental contamination caused by infected humans and animals:

$$\begin{split} \frac{dS_h}{dt} &= \Lambda_h - \frac{\beta_1 I_h S_h}{N_h} - \frac{\beta_2 I_a S_h}{N_a} - \frac{\beta_7 B S_h}{K + B} - (\mu_h + p) S_h + q V_h + \varphi Q_h, & \frac{dS_a}{dt} &= \Lambda_a - \frac{\beta_3 S_a I_a}{N_a} - \mu_a S_a, \\ \frac{dV_h}{dt} &= p S_h - (\mu_h + q) V_h, & \frac{dI_a}{dt} &= \frac{\beta_3 S_a I_a}{N_a} - \mu_a I_a, \\ \frac{dE_h}{dt} &= \frac{\beta_1 I_h S_h}{N_h} - \frac{\beta_2 I_a S_h}{N_a} - \frac{\beta_7 B S_h}{K + B} - (\alpha_1 + \alpha_3 + \mu_h) E_h, & \frac{dB}{dt} &= \rho_1 I_h + \rho_2 I_a - \mu_B B, \\ \frac{dI_h}{dt} &= \alpha_1 E_h + \zeta Q_h - (\mu_h + \delta_h + \gamma_h) I_h, & \frac{dQ_h}{dt} &= \alpha_3 E_h - (\varphi + \zeta + \mu_h) Q_H, \\ \frac{dR_h}{dt} &= \gamma_h I_h - \mu_h R_h. & \end{split}$$

Here, B(t) represents the environmental contamination compartment. The parameter K denotes the concentration of Mpox pathogens in the environment, which increases the transmission rate by 50%. The environmental virus concentration increases when infected humans and rodents shed the virus at rates ρ_1 and ρ_2 , respectively. The model's equilibrium points and basic reproduction number are derived, and it is shown that \Re_0 serves as a threshold for stability. The sensitivity analysis revealed that environmental parameters, such as the environmental transmission rate, the decay rate of Mpox virus in the environment, and the shedding rate of infected humans, play an important role in the spread of Mpox. These findings indicate that quarantine measures alone are insufficient and should be comple-

mented by additional interventions to effectively control the disease. It also suggests that healthcare practitioners and policy-makers should focus on increasing the environmental decay rate of the monkeypox virus while reducing both the environmental transmission rate and the shedding rate of infected individuals.

In [54], vaccination is examined in a broader context. A portion p_1 of new recruits to the human population is assumed to be vaccinated at entry and directly enters the vaccinated class. Additionally, both susceptible and recovered individuals can be vaccinated at rates p_2 and p_3 , respectively. The model equations are given by:

$$\begin{split} \frac{dS_h}{dt} &= \Lambda_h (1-p_1) - \frac{(\beta_1 I_a + \beta_2 I_h) S_h}{N_h} - (\mu_h + p_2) S_h + q V_h + \varphi Q_h, & \frac{dS_a}{dt} &= \Lambda_a - \frac{\beta_3 S_a I_a}{N_a} - \mu_a S_a, \\ \frac{dV_h}{dt} &= \Lambda_h p_1 + p_2 S_h + p_3 R_h - (\mu_h + q) V_h, & \frac{dE_a}{dt} &= \frac{\beta_3 S_a I_a}{N_a} - (\mu_a + \alpha_2) E_a, \\ \frac{dE_h}{dt} &= \frac{(\beta_1 I_a + \beta_2 I_h) S_h}{N_h} - (\alpha_1 + \alpha_3 + \mu_h) E_h, & \frac{dI_a}{dt} &= \alpha_2 E_a - (\mu_a + \delta_a) I_a, \\ \frac{dI_h}{dt} &= \alpha_1 E_h + \zeta Q_h - (\mu_h + \delta_h + \gamma_h) I_h, & \frac{dQ_h}{dt} &= \alpha_3 E_h - (\varphi + \zeta + \mu_h + \delta_h) Q_h, \\ \frac{dR_h}{dt} &= \gamma_h I_h - (\mu_h + p_3) R_h. \end{split}$$

It is also assumed that quarantined individuals become infected at a rate ζ . The equilibrium points are determined, and their stability behavior is analyzed. The basic reproduction numbers is computed, and sensitivity analysis is conducted. The findings demonstrate that vaccination, quarantine, and avoiding contact with infected animals are effective strategies for reducing the spread of the virus. Overall, the study indicates that, within the proposed mathematical model, enhancing vaccination coverage, implementing quarantine measures, and minimizing contact with infected animals can lead to the eradication of the virus. The transmission dynamics of the human population were the focus in [55], with considering the vaccination and quarantine strategies.

In [56], a compartmental model was proposed incorporating individuals under quarantine and those hospitalized:

$$\begin{split} \frac{dS_h}{dt} &= \Lambda_h - \frac{(\beta_1 I_a + \beta_2 I_h)S_h}{N_h} - \mu_h S_h + \varphi Q_h, \qquad \frac{dS_a}{dt} = \Lambda_a - \frac{\beta_3 S_a I_a}{N_a} - \mu_a S_a, \\ \frac{dE_h}{dt} &= \frac{(\beta_1 I_a + \beta_2 I_h)S_h}{N_h} - (\alpha_1 + \alpha_3 + \mu_h)E_h, \qquad \frac{dE_a}{dt} = \frac{\beta_3 S_a I_a}{N_a} - (\mu_a + \alpha_3)E_a, \\ \frac{dI_h}{dt} &= \alpha_1 E_h - (\gamma_h + \mu_h + \delta_h + \alpha_5)I_h, \qquad \qquad \frac{dI_a}{dt} = \alpha_3 E_a - (\delta_a + \mu_a)I_a, \\ \frac{dQ_h}{dt} &= \alpha_3 E_h - (\mu_h + \delta_h + \varphi + \theta)Q_h, \\ \frac{dH_h}{dt} &= \alpha_5 I_h - (\mu_h + \delta_h + \eta)H_h, \\ \frac{dR_h}{dt} &= \eta H_h + \gamma_h I_h + \theta Q_h - (\mu_h + \delta_h)R_h. \end{split}$$

In this model, infected individuals may be hospitalized at a rate α_5 , and recover at a rate η . A fractional-order dynamical analysis conducted, emphasizing the effectiveness of hospitalization in reducing disease transmission. An in-depth analysis of intervention strategies and their potential impact on disease control was conducted. It was demonstrated that hospitalizing infected individuals significantly reduces disease transmission.

In the model proposed by [57], the dynamics of infected individuals transitioning to quarantine, hospitalization, or

recovery were simultaneously considered:

$$\begin{split} \frac{dS_h}{dt} &= \Lambda_h - (\beta_1 I_a + \beta_2 I_h) S_h - \mu_h S_h, & \frac{dS_a}{dt} &= \Lambda_a - \beta_3 I_a S_a - \mu_a S_a, \\ \frac{dE_h}{dt} &= (\beta_1 I_a + \beta_2 I_h) S_h - (\alpha_1 + \mu_h) E_h, & \frac{dE_a}{dt} &= \beta_3 I_a S_a - (\alpha_3 + \mu_a) E_a, \\ \frac{dI_h}{dt} &= \alpha_1 E_h - (\gamma_h + \mu_h + \delta_{h1} + \alpha_4 + \alpha_5) I_h, & \frac{dI_a}{dt} &= \alpha_3 E_a - \mu_a I_a, \\ \frac{dQ_h}{dt} &= \alpha_4 I_h - (\mu_h + \delta_{h2} + \nu + \theta) Q_h, & \\ \frac{dH_h}{dt} &= \alpha_5 I_h + \nu Q_h - (\mu_h + \delta_{h3} + \eta) H_h, & \\ \frac{dR_h}{dt} &= \gamma_h I_h + \theta Q_h + \eta H_h - \mu_h R_h. & \end{split}$$

Individuals in quarantine may either recover or be hospitalized. A fractional-order dynamical analysis performed, and both human and animal reproduction numbers derived. The model was calibrated and validated using weekly Mpox case data from the human population in the United States. It was concluded that simultaneously increasing quarantine and hospitalization rates has a significant impact on reducing the basic reproduction number. The model later extended in [58] using different fractional modeling frameworks.

In another study that simultaneously considers both quarantine and hospitalization strategies [59], the impact of a contaminated environment on the transmission dynamics was also investigated. In addition, readers may also find it useful to look into [60,61] for models that deal with vaccination and isolation, [62,63] for models that include quarantine and isolation, and [64–67] for models that examine the combined effects of vaccination and hospitalization.

2.3 Multiple-Strategy Models

Models incorporating more than two control strategies can provide a more comprehensive framework for analyzing Mpox transmission dynamics. Such models aim to capture the combined effects of multiple interventions, which can lead to more realistic predictions and support the design of integrated control policies. This subsection highlights representative studies that simultaneously implemented three control measures. Only a few studies exist that include three control strategies. In [68], a model introduced including a compartment for severe complications, along with

awareness, vaccination, quarantine, and hospitalization as control measures:

$$\frac{dS_h}{dt} = \Lambda_h - (1 - \kappa)(\beta_1 I_a + \beta_2 I_h) S_h - (\mu_h + p) S_h + q V_h, \quad \frac{dS_a}{dt} = \Lambda_a - \beta_3 S_a I_a - \mu_a S_a,
\frac{dV_h}{dt} = p S_h - (\mu_h + q) V_h, \quad \frac{dI_a}{dt} = \beta_3 S_a I_a - \mu_a I_a,
\frac{dE_h}{dt} = (1 - \kappa)(\beta_1 I_a + \beta_2 I_h) S_h - (\mu_h + \alpha_1) E_h,
\frac{dI_{h_1}}{dt} = \alpha_1 E_h - (\mu_h + \delta_{h_1} + \alpha_4 + \alpha_5 + \omega_1) I_{h_1},
\frac{dQ_h}{dt} = \alpha_4 I_{h_1} - (\mu_h + \delta_{h_2} + \nu + \theta) Q_h,
\frac{dH_h}{dt} = \alpha_5 I_{h_1} + \nu Q_h - (\mu_h + \delta_{h_3} + \omega_2 + \eta) H_h,
\frac{dI_{h_2}}{dt} = \omega_1 I_{h_1} + \omega_2 H_h - (\mu_h + \delta_{h_4} + \gamma_c) I_{h_2},
\frac{dR_h}{dt} = \theta Q_h + \eta H + \gamma_c I_{h_2} - \mu_h R_h.$$

Here, I_{h_2} represents individuals with severe complications, such as those affecting the lungs. The awareness level is represented by κ . The model demonstrates multiple pathways for infected individuals, including quarantine, hospitalization, and progression to severe illness. Human and animal reproduction numbers are derived as:

$$\Re_0^h = \frac{\alpha_1 \beta_2 \Lambda_h (1 - \kappa) (\mu_h + q)}{\mu_h (\mu_h + q + p) (\mu_h + \alpha_1) (\mu_h + \delta_{h1} + \alpha_4 + \alpha_5 + \omega_1)}, \quad \Re_0^a = \frac{p \beta_3 \Lambda_a}{\mu_h (\mu_h + q + p)}$$

and $\mathfrak{R}_0 = \max\{\mathfrak{R}_0^h, \mathfrak{R}_0^a\}$. Transcritical bifurcations observed at the thresholds $\mathfrak{R}_0^h = 1$ and $\mathfrak{R}_0^a = 1$. Simulation results confirmed that awareness, vaccination, quarantine, and treatment significantly reduced Mpox transmission. For additional modeling studies that incorporate three or more of these control strategies, readers may refer to [69, 70].

3 Results

This review identified and analyzed a diverse range of mathematical models developed to study the transmission dynamics and control strategies of Mpox. These models vary in structure and complexity, incorporating features such as vaccination, quarantine, isolation, hospitalization, awareness interventions, risk-group stratification, and optimal control measures. More studies still focus on single or double intervention strategies, whereas models that integrate multiple simultaneous interventions are relatively rare but demonstrate a stronger capacity to capture the complex dynamics of Mpox transmission. While animal-to-human transmission widely modeled, only a limited number of studies explicitly incorporate bidirectional transmission routes between humans and animals, which is crucial given the zoonotic nature of Mpox. In summary, while the existing literature provides valuable insights, it remains relatively limited in combining multiple interventions, bidirectional transmission pathways, and advanced modeling approaches.

In addition to the studies summarized far, several other Mpox modeling studies demonstrate unique approaches that do not directly include the control strategies specified above but still provide important insights. For example, in [71], both exposed and infected human compartments were stratified by risk-groups and incorporated into the model. Control measures representing healthy lifestyle behaviors and antiviral treatments were included to demonstrate the effectiveness of these interventions in reducing Mpox cases. In the thesis presented by [72], an in-depth analysis of a basic model was conducted, and the results support the prediction that if Mpox becomes endemic in the animal

population, it will likely become endemic in the human population as well. In [73], a separate T_h compartment representing treated individuals was added to the model. An optimal control problem was formulated to minimize the number of infected individuals and reduce the costs of prevention and treatment strategies, demonstrating their impact on disease spread. The study [74] considered an age-structured and meta-population model, revealing that removing the age structure increases the estimated basic reproduction number in humans and raising concerns about interventions such as culling. Finally, in [75], a fractional-order model was proposed and analyzed. The existence of an optimal control strategy that minimizes both the number of infected individuals and the costs of treatment and prevention was established, showing that implementing these strategies together is necessary and effective in preventing outbreaks.

4 Discussion

Obtaining analytical solutions for nonlinear differential equation systems are often difficult or even impossible. Consequently, various numerical methods are used to understand the behavior of solutions. In the mathematical epidemiology literature, the most commonly used methods are the standard Euler and Runge-Kutta methods. However, some nonstandard finite difference schemes can also be developed and applied. In addition, a model can be constructed directly as a difference equation system. Discrete-time approaches can be particularly advantageous when data are collected at regular intervals or contact patterns. Continuous models generally assume that changes in the population, such as births and deaths, occur continuously. However, many plant or animal populations have discrete generations and reproduce at specific times of the year, with population counts therefore conducted at certain times. Moreover, discrete models can also be used to approximate continuous dynamics in numerical simulations, providing computationally efficient solutions while preserving the key properties of the continuous model. For these reasons, continuous models can be discretized, or models can be developed directly in discrete form to better capture the transmission dynamics of Mpox. To the best of the author's knowledge, the existing Mpox modeling literature consists entirely of continuous models. Thus, the development of discrete-time Mpox models represents a promising avenue for future research.

When we think about the early stages of an outbreak, it's clear that transmission events can be pretty random. That's why using stochastic modeling approaches can really enhance the realism and predictive accuracy of Mpox transmission models. These methods are especially useful in small populations, emerging hotspots, or situations where spreading occurs, as randomness plays a huge role in these scenarios. Stochastic models are really useful for estimating the changes of an outbreak occurring, the odds of a disease fading away, and the unpredictable nature of an epidemic's trajectory, things that deterministic models often miss. It would be fantastic for future research to combine deterministic frameworks with stochastic simulations. This could really enhance public health preparedness and risk assessment in uncertain situations.

Beyond these methodological considerations, there is clear potential for further development of models that explicitly incorporate human-to-animal transmission pathways of Mpox. Given the complex interactions between humans and animals, including domestic pets, such models can provide a more comprehensive understanding of the disease dynamics and help design more effective control strategies. Finally, extending models to include age-structured populations remains essential, but there is also a clear opportunity to develop gender-structured models. Such models can capture differential susceptibility, contact patterns, and behavioral factors that influence transmission dynamics between different demographic groups, thereby refining intervention strategies.

Despite the richness of existing models, several gaps and avenues for future research remain as discussed in pre-

vious section. This review not only synthesizes the existing mathematical modeling studies of Mpox but also lays the groundwork for advising modeling efforts that are more comprehensive, realistic, and aligned with the nature of Mpox transmission. These enhanced models will be effective in guiding government and health policies, as well as control strategies, aimed at mitigating the impact of Mpox outbreaks globally. By addressing these methodological and structural gaps, future models can provide more robust insights that support the design of effective, evidence-based intervention strategies.

5 Conclusion

This review has provided a comprehensive synthesis and classification of mathematical models developed to understand the transmission dynamics and control strategies of Mpox. While significant progress has been made, the findings highlight clear ares for improvement. Models that combine multiple intervention strategies, include bidirectional human-animal transmission, and use advanced techniques such as fractional calculus, optimal control, discrete-time frameworks, and stochastic approaches remain under represented but hold significant potential.

Future modeling efforts should prioritize:

- Combining multiple simultaneous interventions in unified frameworks.
- Developing discrete-time and stochastic models to better capture real-world uncertainty.
- Explicitly modeling bidirectional transmission routes between humans and animals.
- Expanding demographic structures, such as age and gender stratification.

Environmental factors as well as risk groups based on awareness levels or immune status can also be incorporated into models to better capture the complexity of real-world dynamics. Addressing these directions will advance the understanding of Mpox dynamics and provide a stronger scientific basis for designing effective public health policies to mitigate the impact of future outbreaks. This review not only synthesizes the existing mathematical modeling studies of Mpox but also lays the groundwork for advising modeling efforts that are more comprehensive, realistic, and aligned with the nature of Mpox transmission. These enhanced models will be effective in guiding government and health policies, as well as control strategies, aimed at mitigating the impact of Mpox outbreaks globally.

		Modeling Approach	Control Strategies	
				Conducted Analysis
[13]	S_h, V_h, E_h, I_h, R_h S_a, E_a, I_a, R_a	Integer Order	Vaccination	Feasible region, \Re_0 , E_0 LAS and GAS, Sensitivity
[14]	S_h, V_h, I_h, R_h S_a, I_a, R_a	Integer Order	Vaccination	Feasible region, \Re_0 , E_0 LAS and GAS, Sensitivity
[15]	S_h, V_h, E_h, I_h, R_h S_a, E_a, I_a, R_a	Integer Order	Vaccination	Feasible region, \mathfrak{R}_0
[16]	$S_{h_1}, S_{h_2}, V_h, I_h, R_h$ S_a, I_a, R_a	Integer Order	Vaccination	Feasible region, \Re_0 , E_0 LAS, Bifurcation, Risk-group
[17]	S_h, V_h, E_h, I_h, R_h S_a, E_a, I_a, R_a	Integer Order	Vaccination	Feasible region, \mathfrak{R}_0 , E_0 LAS and GAS, E^* LAS, Sensitivity
[18]	S_h, V_h, E_h, I_h, R_h S_a, E_a, I_a, R_a	Fractional Order	Vaccination	Feasible region, \mathfrak{R}_0 , E_0 and E^* LAS, Sensitivity
[19]	S_h, V_h, E_h, I_h, R_h S_a, E_a, I_a, R_a	Game-theoretic	Vaccination	Existence of equilibria, Herd immunity and Nash equilibrium vacc. rates, Sensitivity
[20]	S_h, V_h, E_h, I_h, R_h	Game-theoretic	Vaccination	Vacc. game, Analysis of Nash equilibria, Sensitivity, Numerical Analysis
[21]	$S_h, V_h, E_h, I_{h_1}, I_{h_2}, R_h$	Integer Order	Vaccination	Feasible region, \Re_0 , E_0 LAS and GAS, Bifurcation
[22]	S_h, V_h, E_h, I_h, R_h S_a, E_a, I_a, B	Fractional Order	Vaccination	Feasible region, \mathfrak{R}_0 , E_0 LAS, E^* GAS
[23]	$S_h^1, S_h^2, V_h^1, V_h^2, E_h^1, E_h^2, I_h^1, I_h^2, I_{h_1}^1, I_{h_2}^2, R_h^1, R_h^2, S_a, I_a, R_a$	Age-structured	Vaccination	\mathfrak{R}_0,E_0 GAS
[25]	$S_h, I_h, Q_h, R_h \ S_a, I_a$	Integer Order	Quarantine	\mathfrak{R}_0 , E_0 LAS and GAS
[26]	S_h, I_h, Q_h, R_h S_a, I_a	Integer Order	Quarantine	Feasible region, \mathfrak{R}_0 , E_0 , E^* and E^{**} LAS and GAS, Bifurcation, Sensitivity
[27]	$S_h, I_h, Q_h, R_h \ S_a, I_a$	Fractional Order	Quarantine	Feasible region, \mathfrak{R}_0 , Numerical analysis
[28]	S_h, E_h, I_h, Q_h, R_h S_a, E_a, I_a	Integer Order	Quarantine	Feasible region, \mathfrak{R}_0 , E_0 LAS, E^* GAS, Sensitivity
[29]	S_h, E_h, I_h, Q_h, R_h S_a, E_a, I_a	Integer Order	Quarantine	Feasible region, \mathfrak{R}_0 , E_0 GAS, E^* LAS, Bifurcation
[30]	S_h, E_h, I_h, Q_h, R_h S_a, E_a, I_a	Fractional Order	Quarantine	Feasible region, \mathfrak{R}_0 , E_0 and E^* LAS, Optimal control
[31]	S_h, E_h, I_h, Q_h, R_h S_a, E_a, I_a	Fractional Order	Quarantine	Feasible region, \Re_0 , E_0 LAS and GAS, Sensitivity
[32]	$S_{h_1}, S_{h_2}, E_{h_1}, E_{h_2}, I_{h_1}, I_{h_2}, Q_{h_1}, Q_{h_2}, R_{h_1}, R_{h_2}, S_a, I_a$	Fractional Order	Quarantine	Feasible region, \mathfrak{R}_0 , Risk-group, Optimal control
[33]	$S_h, E_h, I_h, Q_h, T_h, R_h$ S_a, E_a, I_a	Integer Order	Quarantine	Feasible region, \mathfrak{R}_0 , E_0 LAS-GAS, E^* stability, Sensitivity
[34]	$S_h, E_h, I_h, Q_h, T_h, R_h$ S_a, E_a, I_a	Fractional Order	Quarantine	Feasible region, \mathfrak{R}_0 , E_0 LAS-GAS, Numerical analysis
[35]	S_h, E_h, I_h, Q_h, R_h S_a, E_a, I_a	Fractional Order	Quarantine	Feasible region, \mathfrak{R}_0 , Numerical analysis
[36]	S_h, E_h, I_h, Q_h, R_h S_a, E_a, I_a	Fractional Order	Quarantine	Feasible region, \Re_0 , E_0 LAS and GAS, E^* LAS
[37]	S_h, E_h, I_h, Q_h, R_h S_a, E_a, I_a	Fractional Order	Quarantine	Feasible region, E_0 LAS, Numerical analysis
[38]	S_h, E_h, I_h, Q_h, R_h S_a, E_a, I_a	Fractional Order	Quarantine	E_0 LAS, Numerical analysis
[39]	S_h, E_h, I_h, Q_h, R_h S_a, E_a, I_a	Fractional Order	Quarantine	Feasible region, \mathfrak{R}_0 , E_0 and E^* LAS, Numerical analysis
[40]	S_h, E_h, I_h, Q_h, R_h S_a, E_a, I_a	Fractional Order	Quarantine	Feasible region, E^* GAS, Numerical analysis

Table 2: Summary of modeling features in the reviewed Mpox literature.

Reference	Compartments	Modeling Approach	Control Strategies	Conducted Analysis
[41]	S_h, I_h, Q_h, R_h S_a, E_a, I_a	Integer Order	Quarantine	\mathfrak{R}_0 , E_0 LAS
[42]	$S_{h_1}, S_{h_2}, E_{h_1}, E_{h_2}, I_{h_1}, I_{h_2}, Q_{h_1}, Q_{h_2}, R_{h_1}, R_{h_2}, S_a, I_a$	Fractional Order	Quarantine	Feasible region, \mathfrak{R}_0 , Risk-group, Optimal control
[43]	S_h, E_h, I_h, Q_h, R_h	Integer Order	Quarantine	Feasible region, E_0 LAS-GAS, E^* LAS-GAS, Sensitivity
[44]	S_h, E_h, I_h, Q_h, R_h S_a, E_a, I_a	Fractional Order	Quarantine	Feasible region, \mathfrak{R}_0 , E_0 LAS-GAS, E^* LAS, Sensitivity, Optimal control
[45]	$S_h, E_h, I_{h_1}, I_{h_2}, H_h, R_h$ S_a, I_a	Integer Order	Hospitalization	Feasible region, Risk-group, Optimal control
[46]	$S_{h_1}, S_{h_2}, E_h, P, I_{h_1}, I_{h_2}, H_h, R_h$ S_a, E_h, I_a, R_a	Integer Order	Hospitalization	Feasible region, \mathfrak{R}_0 , E_0 LAS, E^* GAS, Bifurcation, Sensitivity, Risk-group
[47]	$S_h, E_h, I_{h_1}, I_{h_2}, H_h, R_h$ S_a, I_a, R_a	Integer Order	Hospitalization	Feasible region, \Re_0 , E_0 LAS-GAS, E^* GAS, Bifurcation, Sensitivity
[48]	S_h, E_h, I_h, H_h, R_h S_a, E_a, I_a	Fractional Order	Hospitalization	Feasible region, \Re_0 , E_0 LAS
[49]	S_h, E_h, I_h, H_h, R_h S_a, E_a, I_a	Fractional Order	Hospitalization	Numerical analysis
[50]	S_h, E_h, I_h, J_h, R_h S_a, I_a	Integer Order	Isolation	Feasible region, \mathfrak{R}_0 , E_0 LAS, Sensitivity, Optimal control
[51]	S_h, E_h, I_h, Q_h, R_h S_a, I_a	Fractional Order	Vaccination, Quarantine	Feasible region, \mathfrak{R}_0 , E_0 , E^* , E^{**} LAS-GAS Bifurcation, Sensitivity, Optimal control
[52]	$S_h, V_h, E_h, I_h, Q_h, R_h$ S_a, I_a	Fractional Order	Vaccination, Quarantine	Numerical analysis
[53]	$S_h, V_h, E_h, I_h, Q_h, R_h$ S_a, I_a, B	Integer Order	Vaccination, Quarantine	Fesabile region, \mathfrak{R}_0 , E_0 and E^{**} LAS and GAS, Bifurcation, Sensitivity
[54]	$S_h, V_h, E_h, I_h, Q_h, R_h$ S_a, E_a, I_a	Integer Order	Vaccination, Quarantine	Fesabile region, \mathfrak{R}_0 , E_0 LAS and GAS, E^* LAS, Sensitivity
[55]	$S_h, V_h, E_h, I_h, Q_h, R_h$ S_a, E_a, I_a, R_a	Fractional Order	Vaccination, Quarantine	Feasible region, \mathfrak{R}_0 , E_0 LAS
[56]	$S_h, E_h, I_h, Q_h, H_h, R_h$ S_a, E_a, I_a	Fractional Order	Quarantine, Hospitalization	Feasible region, \mathfrak{R}_0 , Sensitivity
[57]	$S_h, E_h, I_h, Q_h, H_h, R_h$ S_a, E_a, I_a	Fractional Order	Quarantine, Hospitalization	Feasible region, \mathfrak{R}_0 , E_0 , E^* and E^{**} LAS and GAS, Sensitivity
[58]	$S_h, E_h, I_h, Q_h, H_h, R_h$ S_a, E_a, I_a	Fractional Order	Quarantine, Hospitalization	Feasible region, Numerical analysis
[59]	$S_h, E_h, I_{h_1}, I_{h_2}, Q_h, H_h, R_h$ S_a, E_a, I_a, B	Integer Order	Quarantine, Hospitalization	Feasible region, \mathfrak{R}_0 , E_0 and E^{**} GAS, Risk-group, Optimal control
[60]	$S_h, V_h, E_h, I_{h_1}, I_{h_2}, J_h, R_h$ S_a, E_a, I_a, R_a	Integer Order	Vaccination, Isolation	Feasible region, \mathfrak{R}_0 , E_0 LAS-GAS, E^* GAS, Risk-group, Bifurcation, Sensitivity
[61]	$S_{h_1}, S_{h_2}, E_h, I_h, R_h$ S_a, E_a, I_a	Integer Order	Vaccination, Isolation	Feasible region, \mathfrak{R}_0 , E_0 LAS, Risk-group, Sensitivity
[62]	$S_h, E_h, I_h, J_h, Q_h, R_h$ S_a, I_a	Integer Order	Quarantine, Isolation	Feasible region, \mathfrak{R}_0 , E_0 LAS-GAS, Bifurcation, Sensitivity
[63]	$S_h, E_h, I_h, J_h, Q_h, R_h$ S_a, I_a	Fractional Order	Quarantine, Isolation	Feasible region, Stability, Numerical analysis
[64]	$S_h, V_h, E_h, I_h, H_h, R_h$ S_a, E_a, I_a	Integer Order	Vaccination, Hospitalization	Feasible region, \mathfrak{R}_0 , E_0 LAS and GAS, Bifurcation, Optimal control
[65]	$S_h, V_h, E_h, I_h, H_h, R_h$ S_a, E_a, I_a	Integer Order	Vaccination, Hospitalization	Feasible region, E_0 LAS-GAS, Sensitivity Optimal control
[66]	$S_h, V_h, E_h, I_h, H_h, R_h$ S_a, E_a, I_a	Fractional Order	Vaccination, Hospitalization	Feasible region, \mathfrak{R}_0 , E_0 LAS-GAS, E^* GAS, Sensitivity, Optimal control
[67]	$S_h, V_h, I_{h_1}, I_{h_2}, H_h, R_h$ S_a, I_a	Fractional Order	Vaccination, Hospitalization	Feasible region, \mathfrak{R}_0 , Stability, Risk-group
[68]	$S_h, V_h, E_h, I_{h_1}, I_{h_2}, Q_h, H_h, R_h$ S_a, I_a	Integer Order	Vaccination, Quarantine, Hospitalization	Feasible region, \mathfrak{R}_0 , E_0 and E^{**} LAS, E^* GAS, Bifurcation, Risk-group
[69]	$S_h, E_h, I_h, Q_h, H_h, R_h$ S_a, E_a, I_a	Fractional Order	Vaccination, Quarantine, Hospitalization	Feasible region, E_0 , E^* and E^{**} LAS, Optimal control

 Table 3: Summary of modeling features in the reviewed Mpox literature (continued).

Reference	Compartments	Modeling Approach	Control Strategies	
				Conducted Analysis
[70]	$S_h, V_h, E_h, I_h, Q_h, H_h, R_h$ S_a, E_a, I_a	Fractional Order	Vaccination, Quarantine, Hospitalization	Feasible region, \Re_0 , E_0 GAS
[71]	$S_h, E_{h_1}, E_{h_2}, I_{h_1}, I_{h_2}, R_h$ S_a, E_a, I_a	Integer Order	-	Numerical analysis, Optimal control Sensitivity
[72]	$S_h, I_h, R_h S_a, I_a, R_a$	Integer Order	-	Feasible region, \mathfrak{R}_0 , E_0 LAS-GAS, E^* LAS-GAS
[73]	S_h, I_h, T_h, R_h S_a, I_a, R_a	Integer Order	-	Feasible region, \Re_0 , E_0 and E^* LAS, Sensitivity, Optimal control
[74]	$S_h^1, S_h^2, S_{h_1}^1, S_{h_2}^2, I_h^1, I_h^2, R_h^1, R_h^2, S_a, I_a, R_a$	Age-structured	-	\mathfrak{R}_0 , E_0 LAS
[75]	$S_h, I_h, T_h, R_h S_a, I_a, R_a$	Fractional Order	-	Feasible region, \mathfrak{R}_0 , E_0 LAS, E^* LAS, Optimal control

Table 4: Summary of modeling features in the reviewed Mpox literature (continued).

Declarations

Author's Contributions: This article is solely authored by the undersigned.

Availability of Data and Materials: All the data used for the study are available from the corresponding author upon request.

Use of AI tools: The author declares that he has not used Artificial Intelligence (AI) tools in the creation of this.

Funding: This study did not receive any funding.

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References

- [1] A. Letafati and T. Sakhavarz, Monkeypox virus: A review, Microb. Pathog., 176, (2023), 106027.
- [2] N. Sklenovska and M. Van Ranst, *The emergence of monkeypox as the most important orthopoxvirus infection in humans*, Front. Public Health, **6**, (2018), 383729.
- [3] R. Grant, L. B. L. Nguyen, and R. Breban, *Modelling human-to-human transmission of monkeypox*, Bull. World Health Organ., **98**(9), (2020), 638.
- [4] J. Kaler, A. Hussain, G. Flores, S. Kheiri, and D. Desrosiers, *Monkeypox: a comprehensive review of transmission, pathogenesis, and manifestation*, Cureus, **14**(7), (2022).
- [5] A. M. McCollum, and I. K. Damon, *Human monkeypox*, Clin. Infect. Dis., **58**(2), (2014), 260-267.
- [6] World Health Organization, Mpox, Geneva: WHO. Available from: https://www.who.int/news-room/fact-sheets/detail/mpox [Accessed Jun. 19, 2025]

- [7] World Health Organization, WHO recommends monkeypox new name for disease, WHO, 2022 Nov 28. Available from: https://www.who.int/news/item/ Geneva: 28-11-2022-who-recommends-new-name-for-monkeypox-disease [Accessed Jun. 19, 2025]
- [8] O. Mitja, D. Ogoina, B. K. Titanji, C. Galvan, J. J. Muyembe, M. Marks, and C. M. Orkin, *Monkeypox*, The Lancet, **401**(10370), (2023), 60-74.
- [9] World Organisation for Animal Health, *Mpox guidelines*, Paris: WOAH; 2022 Dec. Available from: https://www.woah.org/app/uploads/2022/12/woah-mpox-guidelines-en.pdf [Accessed Jun. 19, 2025]
- [10] S. Seang, S. Burrel, E. Todesco, et al., Evidence of human-to-dog transmission of monkeypox virus The Lancet, **400**(10353), (2022), 658-659.
- [11] M. Martcheva, An Introduction to Mathematical Epidemiology, New York: Springer, (2015).
- [12] X. Wang, Z. Gu, S. Sheng, R. Song, and R. Jin, *The current state and progress of mpox vaccine research*, China CDC Weekly, **6**(7), (2024), 118.
- [13] S. Usman and I. I. Adamu, Modeling the transmission dynamics of the monkeypox virus infection with treatment and vaccination interventions, J. Appl. Math. Phys., 5(12), (2017), 2335-2351.
- [14] P. Emeka, M. Ounorah, F. Eguda, and B. Babangida, *Mathematical model for monkeypox virus transmission dynamics*, Epidemiol. Open Access, **8**(3), (2018), 1000348.
- [15] N. O. Lasisi, N. I. Akinwande, and F. A. Oguntolu, *Development and exploration of a mathematical model for transmission of monkey-pox disease in humans*, Math. Models Eng., **6**(1), (2020), 23-33.
- [16] A. Elsonbaty, W. Adel, A. Aldurayhim, and A. El-Mesady, *Mathematical modeling and analysis of a novel monkeypox virus spread integrating imperfect vaccination and nonlinear incidence rates*, Ain Shams Eng. J., **15**(3), (2024), 102451.
- [17] S. Tiwari, P. Porwal, and N. Mangal, Design and investigation of mathematical model for the vaccination and transmission of monkeypox virus without lifelong immunity, Indian J. Sci. Technol., **16**(39), (2023), 3423-3434.
- [18] S. Paul, A. Mahata, M. Karak, S. Mukherjee, S. Biswas, and B. Roy, *Dynamical behavior of fractal-fractional order monkeypox virus model*, Franklin Open, 7, (2024), 100103.
- [19] S. V. Bankuru, S. Kossol, W. Hou, P. Mahmoudi, J. Rychtar, and D. Taylor, *A game-theoretic model of Monkeypox to assess vaccination strategies*, PeerJ, **8**, (2020), e9272.
- [20] I. B. Augsburger, G. K. Galanthay, J. H. Tarosky, J. Rychtar, and D. Taylor, *Voluntary vaccination may not stop monkeypox outbreak: A game-theoretic model*, PLoS Negl. Trop. Dis., **16**(12), (2022), e0010970.
- [21] B. Bolaji, A. Ibrahim, F. Ani, B. Omede, and G. Acheneje, *A model for the control of transmission dynamics of human monkeypox disease in Sub-Saharan Africa*, J. Niger. Soc. Phys. Sci., (2024), 1800-1800.
- [22] E. Addai, M. Ngungu, M. A. Omoloye, and E. Marinda, Modelling the impact of vaccination and environmental transmission on the dynamics of monkeypox virus under Caputo operator, Math. Biosci. Eng, **20**, (2023), 10174-10199.
- [23] R. L. G. Kakai, Y. Montcho, H. B. Taboe, and K. Traore, Modeling Mpox transmission and burden across various vaccination levels in the DRC using an age-structured model, (2024).
- [24] M. A. Alah, S. Abdeen, E. Tayar, and I. Bougmiza, *The story behind the first few cases of monkeypox infection in non-endemic countries*, 2022, J. Infect. Public Health, **15**(9), (2022), 970-974.

- [25] S. A. Somma, N. I. Akinwande, and U. D. Chado, *A mathematical model of monkey pox virus transmission dynamics*, Ife J. Sci., **21**(1), (2019), 195-204.
- [26] M. Gümüş and K. Türk, Global analysis of a monkeypox virus model considering government interventions, Phys. Scr., 100(4), (2025), 045216.
- [27] A. M. Alzubaidi, H. A. Othman, S. Ullah, N. Ahmad, and M. M. Alam, *Analysis of Monkeypox viral infection with human to animal transmission via a fractional and fractal-fractional operators with power law kernel*, Math. Biosci. Eng., **20**(4), (2023), 6666-6690.
- [28] F. M. Allehiany, M. H. DarAssi, I. Ahmad, M. A. Khan, and E. M. Tag-Eldin, *Mathematical modeling and backward bifurcation in monkeypox disease under real observed data*, Results Phys., **50**, (2023), 106557.
- [29] O. J. Peter, S. Kumar, N. Kumari, F. A. Oguntolu, K. Oshinubi, and R. Musa, *Transmission dynamics of Monkeypox virus: a mathematical modelling approach*, Model. Earth Syst. Environ., (2022), 1-12.
- [30] A. El-Mesady, W. Adel, A. A. Elsadany, and A. Elsonbaty, Stability analysis and optimal control strategies of a fractional-order monkeypox virus infection model, Phys. Scr., 98(9), (2023), 095256.
- [31] M. Ngungu, E. Addai, A. Adeniji, U. M. Adam, and K. Oshinubi, *Mathematical epidemiological modeling and analysis of monkeypox dynamism with non-pharmaceutical intervention using real data from United Kingdom*, Front. Public Health, **11**, (2023), 1101436.
- [32] M. Manivel, A. Venkatesh, and S. Kumawat, *Numerical simulation for the co-infection of Monkeypox and HIV model using fractal-fractional operator*, Model. Earth Syst. Environ., **11**(3), (2025) 157.
- [33] B. C. Agbata, W. Obeng-Denteh, D. Raimonda, P. A. Kwabi, A. A. R. Habeeb, F. Asante-Mensa, ... & S. S. Arivi, *Mathematical Modeling and Analysis of Monkeypox Transmission Dynamics with Treatment and Quarantine Interventions*, Dutse J. Pure Appl. Sci., **10**(4b), (2024), 78-96.
- [34] B. C. Agbata, W. Obeng-Denteh, D. F. Agbebaku, M. M. Shior, D. J. Yahaya, & A. Y. Danjuma, Laplace-Adomian Decomposition method for solving fractional-order mathematical model of Monkeypox transmission dynamics, Int. J. Math. Anal. Model., 7(2), (2024).
- [35] A. El-Mesady, A. Elsonbaty, and W. Adel, *On nonlinear dynamics of a fractional order monkeypox virus model*, Chaos Solitons Fractals, **164**, (2022), 112716.
- [36] M. A. Qurashi, S. Rashid, A. M. Alshehri, F. Jarad, and F. Safdar, New numerical dynamics of the fractional monkeypox virus model transmission pertaining to nonsingular kernels, Math. Biosci. Eng. **20**(1), (2023), 402-436.
- [37] N. H. Sweilam, S. M. Al-Mekhlafi, S. M. Hassan, N. R. Alsenaideh, and A. E. Radwan, A novel hybrid crossover dynamics of monkeypox disease mathematical model with time delay: Numerical treatments, Fractal Fractional, 8(4), (2024), 185.
- [38] N. H. Sweilam, Z. N. Mohammed, and W. A. Kareem, *Numerical approaches for solving complex order monkeypox mathematical model*, Alex. Eng. J., **90**, (2024), 170-182.
- [39] W. Sudsutad, C. Thaiprayoon, J. Kongson, and W. Sae-dan, *A mathematical model for fractal-fractional monkeypox disease and its application to real data*, AIMS Math., **9**(4), (2024), 8516-8563.
- [40] M. Farman, A. Akggl, H. Garg, D. Baleanu, E. Hincal, and S. Shahzeen, *Mathematical analysis and dynamical transmission of monkeypox virus model with fractional operator*, Expert Syst., **42**(1), (2025), e13475.
- [41] S. Onitilo, A. Ajani, D. Daniel, and A. Haruna, *Mathematical modeling of the transmission dynamics of monkeypox with the impact of quarantine and public enlightenment in Nigeria*, J. Innov. Sci. Eng., **8**(1), (2024), 1-17.

- [42] W. Adel, A. Elsonbaty, A. Aldurayhim, and A. El-Mesady, *Investigating the dynamics of a novel fractional-order monkeypox epidemic model with optimal control*, Alex. Eng. J., 73, (2023), 519-542.
- [43] I. Smouni, A. El Mansouri, B. Khajji, A. Labzai, M. Belam, and Y. Tidli, *Mathematical modeling and analysis of a Monkeypox model*, Commun. Math. Biol. Neurosci., 2023, Article ID 85.
- [44] S. Barman, S. Jana, S. Majee, A. Khatua, and T. K. Kar, Complex dynamics of a fractional-order monkeypox transmission system with saturated recovery function, Eur. Phys. J. Spec. Top., (2024). https://doi.org/10.1140/epjs/s11734-024-01283-3
- [45] M. Baroudi, I. Smouni, H. Gourram, A. Labzai, and M. Belam, *Optimizing control strategies for monkeypox through mathematical modeling*, Partial Differ. Equ. Appl. Math., **12**, (2024), 100996.
- [46] M. M. Al-Shomrani, S. S. Musa, and A. Yusuf, Unfolding the transmission dynamics of monkeypox virus: an epidemiological modelling analysis, Mathematics, 11(5), (2023), 1121.
- [47] U. E. Michael, L. O. Omenyi, E. Kafayat, E. Nwaeze, O. A. Akachukwu, G. Ozoigbo, and M. Ekhator, *Monkeypox mathematical model with surveillance as control*, Commun. Math. Biol. Neurosci., 2023, Article ID 6.
- [48] O. J. Peter, F. A. Oguntolu, M. M. Ojo, A. O. Olayinka, R. Jan, and I. Khan, *Fractional order mathematical model of monkeypox transmission dynamics*, Phys. Scr., **97**(8), (2022), 084005.
- [49] S. Suantai, Z. Sabir, M. Umar, and W. Cholamjiak, *Scaled conjugate gradient for the numerical simulations of the mathematical model-based monkeypox transmission*, Fractal Fractional, **7**(1), (2023), 63.
- [50] O. J. Peter, C. E. Madubueze, M. M. Ojo, F. A. Oguntolu, and T. A. Ayoola, *Modeling and optimal control of monkeypox with cost-effective strategies*, Model. Earth Syst. Environ., **9**(2), (2023), 1989-2007.
- [51] S. Majee, S. Jana, S. Barman, and T. K. Kar, *Transmission dynamics of monkeypox virus with treatment and vaccination controls: A fractional order mathematical approach*, Phys. Scr., **98**(2), (2023), 024002.
- [52] A. Venkatesh, M. Manivel, K. Arunkumar, M. Prakash Raj, Shyamsunder, and S. D. Purohit, *A fractional mathematical model for vaccinated humans with the impairment of Monkeypox transmission*, Eur. Phys. J. Spec. Top., (2024), 1-21.
- [53] C. E. Madubueze, I. O. Onwubuya, G. N. Nkem, and Z. Chazuka, *The transmission dynamics of the monkeypox virus in the presence of environmental transmission*, Front. Appl. Math. Stat., **8**, (2022), 1061546.
- [54] L. Leandry and E. W. Mureithi, *An investigation on the monkeypox virus dynamics in human and rodent populations for a deterministic mathematical model*, Inform. Med. Unlocked, **41**, (2023), 101325.
- [55] A. Adom-Konadu, E. Bonyah, A. L. Sackitey, M. Anokye, and J. K. K. Asamoah, A fractional order Monkeypox model with protected travelers using the fixed point theorem and Newton polynomial interpolation, Healthc. Anal., 3, (2023), 100191.
- [56] M. Manivel, A. Venkatesh, K. Arunkumar, M. Prakash Raj, and Shyamsunder, A mathematical model of the dynamics of the transmission of monkeypox disease using fractional differential equations, Adv. Theory Simul., 7(9), (2024), 2400330.
- [57] R. R. Musafir, A. Suryanto, and I. Darti, *Stability analysis of a fractional-order monkeypox epidemic model with quarantine and hospitalization*, J. Biosaf. Biosecur., **6**(1), (2024), 34-50.
- [58] R. R. Musafir, A. Suryanto, I. Darti, and T. Trisilowati, *Comparison of fractional-order monkeypox model with singular and non-singular kernels*, Jambura J. Biomathemat., **5**(1), (2024), 1-9.

- [59] W. Okongo, J. Okelo Abonyo, D. Kioi, S. E. Moore, and S. N. Aguegboh, *Mathematical modeling and optimal control analysis of Monkeypox virus in contaminated environment*, Model. Earth Syst. Environ., **10**(3), (2024), 3969-3994.
- [60] T. T. Ashezua, R. I. Gweryina, and F. S. Kaduna, *Population dynamics of a mathematical model for monkeypox*, Int. J. Math. Anal. Model., **6**(1), (2023).
- [61] O. I. Idisi, T. T. Yusuf, E. Adeniyi, A. A. Onifade, Y. T. Oyebo, A. T. Samuel, and L. A. Kareem, *A new compartmentalized epidemic model to analytically study the impact of awareness on the control and mitigation of the monkeypox disease*, Healthc. Anal., 4, (2023), 100267.
- [62] O. J. Peter, A. Abidemi, M. M. Ojo, and T. A. Ayoola, *Mathematical model and analysis of monkeypox with control strategies*, Eur. Phys. J. Plus, **138**(3), (2023), 242.
- [63] F. A. Wireko, I. K. Adu, C. Sebil, and J. K. K. Asamoah, *A fractal-fractional order model for exploring the dynamics of Monkeypox disease*, Decis. Anal. J., **8**, (2023), 100300.
- [64] O. A. Adepoju and H. O. Ibrahim, An optimal control model for monkeypox transmission dynamics with vaccination and immunity loss following recovery, Healthc. Anal., 6, (2024), 100355.
- [65] S. Ullah, R. Nawaz, and A. Alshehri, *Mathematical modeling of monkeypox infection with optimized preventive control analysis: a case study with 2022 outbreak*, Eur. Phys. J. Plus, **138**(8), (2023), 1-34.
- [66] S. T. Akinyemi, I. O. Idisi, M. Rabiu, V. I. Okeowo, N. Iheonu, E. J. Dansu, et al., and K. Oshinubi, A tale of two countries: optimal control and cost-effectiveness analysis of monkeypox disease in Germany and Nigeria, Healthc. Anal., 4, (2023), 100258.
- [67] R. Alharbi, R. Jan, S. Alyobi, Y. Altayeb, and Z. Khan, *Mathematical modeling and stability analysis of the dynamics of monkeypox via fractional-calculus*, Fractals, **30**(10), (2022), 2240266.
- [68] K. Soni and A. K. Sinha, Modeling and stability analysis of the transmission dynamics of Monkeypox with control intervention, Partial Differ. Equ. Appl. Math., 10, (2024), 100730.
- [69] R. R. Musafir, A. Suryanto, and I. Darti, *Optimal control of a fractional-order monkeypox epidemic model with vaccination and rodents culling*, Results Control Optim., **14**, (2024), 100381.
- [70] K. T. Kubra, S. Gulshan, and R. Ali, An Atangana-Baleanu derivative-based fractal-fractional order model for the monkey pox virus: A case study of USA, Partial Differ. Equ. Appl. Math., 9, (2024), 100623.
- [71] P. N. Ikhsani, T. Usman, and M. Ikhwan, *Optimal control on mathematical model of Mpox disease spread*, Barekeng: J. Ilmu Mat. Terapan, **19**(1), (2025), 477-490.
- [72] R. E. TeWinkel, Stability Analysis for the Equilibria of a Monkeypox Model, Ph.D. thesis, Univ. Wisconsin-Milwaukee, (2019).
- [73] D. Yapiskan, M. Yurtoglu, D. Avci, BB. Iskender Eroglu and E. Bonyah, A novel model for monkeypox disease: System analysis and optimal preventive strategies Iran. J. Sci., 47(5),(2023) 1665-1677.
- [74] J. M. Tchuenche and C. T. Bauch, Can culling to prevent monkeypox infection be counter-productive? Scenarios from a theoretical model, J. Biol. Syst., **20**(03), (2012), 259-283.
- [75] T. Gunasekar, S. Manikandan, V. Govindan, J. Ahmad, W. Emam, and I. Al-Shbeil, *Symmetry analyses of epidemiological model for Monkeypox virus with Atangana-Baleanu fractional derivative*, Symmetry, **15**(8), (2023), 1605.

Journal of Mathematical Epidemiologys (JoME), (J. Math. Epidemiol.) https://mathepidemi.com/index.php/pub/index



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How to cite this article: K. Türk, *Analysis of mathematical Listeriosis model in ready-to-eat food*, J. Math. Epidemiol., **1**(1) (2025), 52-79. DOI 10.64891/jome.7