

A Mathematical Model for the Spread of Monkeypox in Uganda: An SIR Model with Demographic Effects

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Abstract

Monkeypox, a viral zoonotic disease caused by the monkeypox virus and primarily transmitted through close contact with infected animals and humans, poses a growing public health challenge, especially in endemic regions like Uganda. With its potential for human-to-human transmission and an increasing number of reported cases in recent years, monkeypox can lead to significant morbidity and mortality if not controlled effectively. This study employs a Susceptible-Infected-Recovered (SIR) compartmental model to rigorously analyze the transmission dynamics of a hypothetical monkeypox outbreak in Uganda, a country endemic to the disease. The model is parameterized using epidemiological data from existing literature, including transmission rates, recovery rates, and contact patterns, to construct realistic outbreak scenarios. We perform a series of simulations over a 200-day period to estimate the basic reproduction number (R_0), identify key transmission drivers, and assess the effectiveness of various public health interventions such as mass vaccination, isolation, and public awareness campaigns. The simulation results reveal that, in the absence of intervention, the outbreak reaches a peak around Day 70, with approximately 45% of the population infected, underscoring the high transmissibility of the virus. Strategic early interventions, particularly mass vaccination and rapid isolation of cases, are shown to significantly reduce R_0 to below the epidemic threshold, demonstrating their critical importance in outbreak management. These findings underscore the necessity for robust, proactive public health strategies and can inform policy decisions in Uganda and other regions facing similar epidemiological threats. The model framework presented here can also be adapted to other infectious diseases, offering a versatile tool for public health preparedness and response planning.

1. Introduction

Monkeypox is a zoonotic viral disease endemic to several African countries, including Uganda [1]. The disease has garnered increasing attention due to recent outbreaks that highlight its potential for widespread transmission and the challenges faced in controlling it. Historically, monkeypox cases have been reported sporadically in Africa, but recent trends suggest a worrying increase in incidence [2]. Recent African studies have further highlighted these concerns, with [3] modeling the spread of Mpox across African countries using a Bayesian hierarchical approach, and [4] providing country-specific insights from Rwanda on current challenges and future recommendations. Mathematical modeling plays a crucial role in understanding the spread of infectious diseases and evaluating control strategies. For instance, [5]



Figure 1: Monkey pox case

have demonstrated the effectiveness of compartmental models like the Susceptible-Infected-Recovered (SIR) model in predicting disease dynamics. The SIR model is particularly useful for simulating the progression of epidemics and assessing the impact of various public health interventions [6, 7].

Previous studies on monkeypox have utilized various modeling approaches to explore its transmission dynamics [8] provided insights into the epidemiological characteristics of monkeypox and highlighted the importance of effective monitoring and control measures. Recent work by [9, 10] has applied mathematical models to assess the spread of monkeypox and the effectiveness of potential interventions, such as vaccination and isolation.

Understanding the basic reproduction number (R_0) is essential for designing effective control strategies [11]. First introduced the concept of R_0 in their foundational work on epidemic modeling. More recent studies have refined these models to include additional compartments and dynamics, providing a more nuanced understanding of disease spread [7, 12].

Human monkeypox (mpox), a viral zoonosis related to smallpox, has become a global concern with over 80,000 cases in non-endemic countries by December 2022. This review covers mpox's history, ecology, and virology, highlighting changes in its viral fitness traits post-2022. It evaluates mathematical models—epidemiological, within-host, and between-host—using the One Health approach to integrate factors like vaccination, geography, and climate. The review underscores how these models offer crucial insights into mpox transmission and pathogenesis, guiding public health strategies for anticipated future outbreaks[13].

Human monkeypox (mpox) is increasingly concerning globally, with over 80,000 cases in non-endemic countries as of December 2022. This review covers mpox's history and virology, focusing on changes in viral fitness traits pre-and post-2022. It evaluates current knowledge from various mathematical models—epidemiological, within-host, and between-host—distinguishing factors like vaccination, geography, climate, and animal models. By summarizing parameters such as the reproduction number (R_0), the review highlights how these models offer new insights into mpox transmission and pathogenesis, aiding in the development of effective public health measures and strategies as mpox continues to spread[14].

This paper employs the SIR model to simulate a hypothetical monkeypox outbreak in Uganda, using parameters derived from available epidemiological data. The objective is to estimate R_0 , visualize the epidemic curve, and evaluate the impact of various public health interventions, such as vaccination and isolation [15, 16].

2. Model Formulation

To accurately model the transmission dynamics and spread of monkeypox in Uganda, while accounting for the mitigation strategies implemented to curb the disease, it is essential to develop a robust mathematical model. Such a model is invaluable for comprehensively understanding the epidemiological patterns and potential future scenarios of monkeypox transmission, particularly under varying conditions of human-to-human interaction and public health interventions. In this study, we adopt a compartmental approach, specifically the Susceptible-Exposed-Infected-Recovered (SEIR) model, which subdivides the total population into distinct epidemiological compartments: Susceptible ($S(t)$), Exposed ($E(t)$), Infected ($I(t)$), and Recovered ($R(t)$).

The **Susceptible** compartment, denoted by $S(t)$, comprises individuals who are at risk of contracting the infection but have not yet been exposed to the virus. The **Exposed** compartment, $E(t)$, represents those who have been infected but are in the latent incubation period of the disease. For monkeypox, the incubation period typically spans 7 to 14 days but can range from 5 to 21 days. During this period, individuals are asymptomatic and are assumed to be non-contagious. The **Infected** compartment, $I(t)$, consists of individuals who have developed symptoms indicative of monkeypox infection, such as fever, headache, muscle aches, backache, and fatigue, and are capable of transmitting the virus. The **Recovered** compartment, $R(t)$, includes individuals who have recuperated from the infection and have developed immunity, thus preventing reinfection. Therefore, the total population at any given time, $N(t)$, is expressed as:

$$N(t) = S(t) + E(t) + I(t) + R(t),$$

Assumptions Underlying the Model

To develop a mathematically tractable model, the following assumptions are made:

- i. **Recruitment into the Susceptible Class:** New individuals enter the susceptible class through births or immigration at a constant rate, denoted by Φ . This ensures a continuous influx of individuals into the population, sustaining its size over time.
- ii. **Exclusive Human-to-Human Transmission:** Transmission of monkeypox is considered to occur solely through human-to-human contact. Consequently, the model excludes zoonotic transmission (animal-to-human) and potential environmental reservoirs, which are assumed to be negligible within the scope of this study.
- iii. **Non-infectious Exposed Individuals:** Individuals within the exposed compartment ($E(t)$) are not capable of transmitting the virus to others, as monkeypox only becomes contagious following the appearance of symptoms.
- iv. **Progression from Exposed to Symptomatic or Asymptomatic States:** After the incubation period, which varies between 5 and 21 days, exposed individuals either progress to a symptomatic infectious state (transitioning to $I(t)$) or remain asymptomatic (transitioning to $A(t)$). Both pathways play crucial roles in the disease's transmission dynamics.
- v. **Permanent Immunity Post-Recovery:** Recovered individuals are assumed to acquire lifelong immunity to monkeypox, meaning there is no risk of reinfection. This simplifies the model by eliminating the need to account for potential secondary infections.
- vi. **Birth and Death Rate:** Births and Deaths occur in any compartment hence accounting for death and birth rates is a significant aspect. In the study we consider the death and birth rate to be the same and symbolized by μ .
- v. **Vaccination:** Upon vaccination, the individuals are considered to be immune to monkey pox infection. This implies that the vaccinated persons in the susceptible compartment leave to recovered compartment.

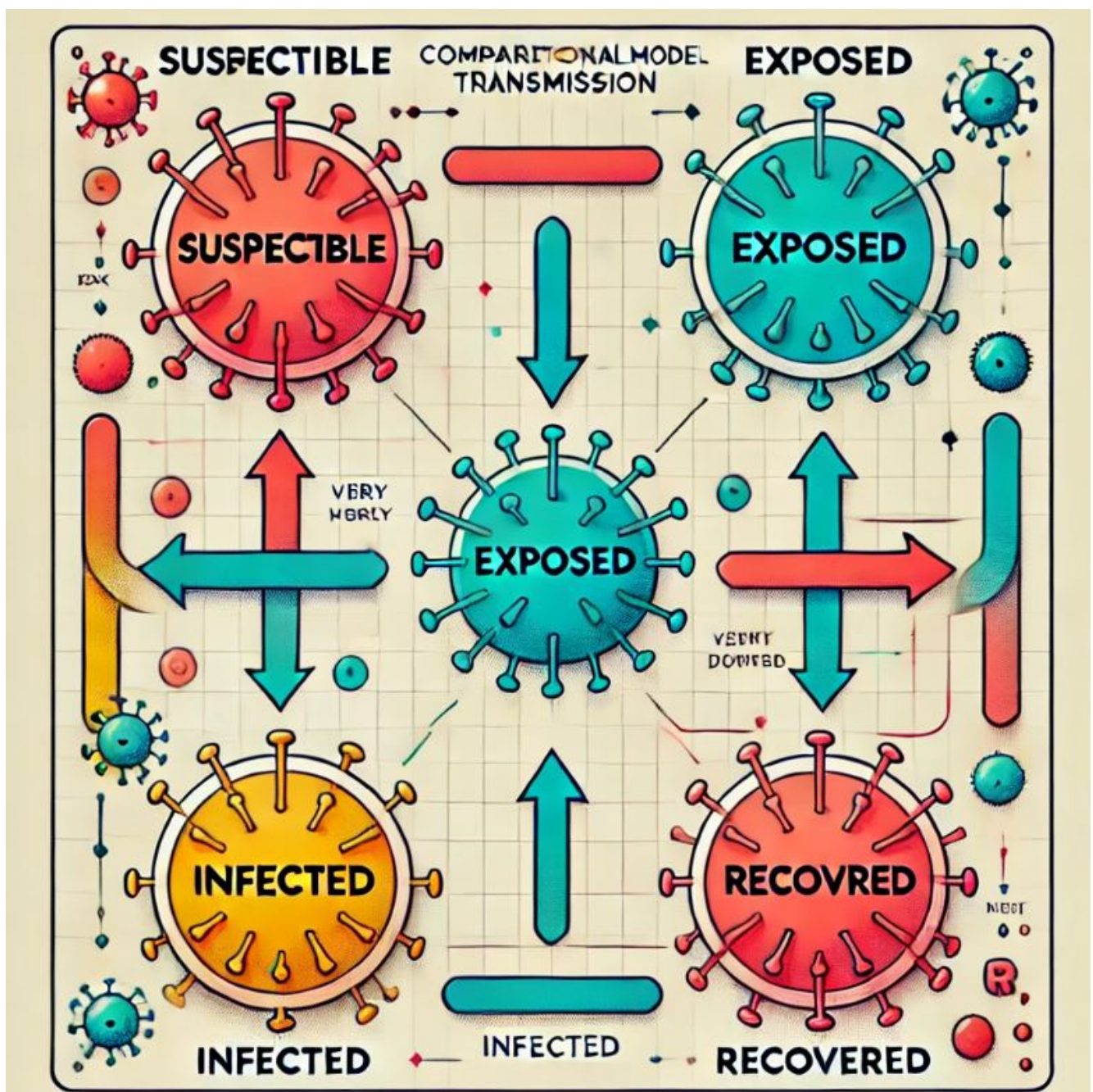


Figure 2: Schematic diagram of the compartmental model for monkeypox transmission

Additional Model Considerations

This model is further refined by considering the movement of exposed individuals into either symptomatic ($I(t)$) or asymptomatic ($A(t)$) compartments, reflecting more nuanced disease progression pathways. The recruitment into the susceptible compartment accounts for both newborns and immigrants, ensuring a realistic representation of demographic dynamics. Mortality is incorporated into the model through both natural death rates and disease-induced mortality rates, providing a comprehensive view of the impacts of the disease on the population structure. Furthermore, the model incorporates public health interventions, particularly the impact of vaccination efforts, such as the administration of the smallpox vaccine, which has demonstrated efficacy in preventing monkeypox. These interventions are integrated into the governing differential equations to evaluate their effectiveness in altering transmission dynamics and achieving control.

By developing and analyzing this mathematical model, we aim to provide a powerful analytical framework for understanding the spread of monkeypox and assessing the potential effectiveness of various control strategies in Uganda. This model serves not only as a predictive tool but also as a foundation for optimizing public health policies and intervention strategies.

2.1. SIR Model Framework

The SIR model divides the population into three compartments: Susceptible (S), Infected (I), and Recovered (R). The model is governed by the following differential equations:

$$\frac{dS}{dt} = -\frac{\lambda SI}{N} + \gamma N - \mu S - 10,000, \quad (1)$$

$$\frac{dE}{dt} = \frac{\lambda SI}{N} - \beta E - \mu E, \quad (2)$$

$$\frac{dI}{dt} = \beta E - \alpha I - \mu I, \quad (3)$$

$$\frac{dR}{dt} = \alpha I - \mu R + 10,000, \quad (4)$$

where N is the total population, λ is the exposed rate, β is the infection rate, and α is the recovery rate. The initial conditions; $t = 0$

$$S(0) = S_o, E(0) = E_o, I(0) = I_o, R(0) = 0$$

The Reproduction number

Thus equation (3) at $t = 0$ becomes;

$$\left. \frac{dI}{dt} \right|_{t=0} = \beta E_o - \alpha I_o - \mu I_o \quad (5)$$

Thus, from equation (5), $\beta E_o - \alpha I_o - \mu I_o < 0$, when most people are recovering which implies that $\frac{dI}{dt} < 0$. Hence we obtain;

$$\frac{\beta E_o}{I_o(\alpha + \mu)} < 1 \quad (6)$$

where the reproductive number R_o is $\frac{\beta E_o}{I_o(\alpha + \mu)}$.

Thus, from equation (6), lowering the transmission rate β and initial exposed persons E_o will reduce the reproduction number hence less probability of an epidemic.

2.2. Parameter Estimation and Data Simulation

For this simulation, we assume the following parameters:

- i. **Population Size (N):** 1,000,000.
- ii. **Initial Number of Infected (I₀):** 50.
- iii. **Initial Number of Recovered (R₀):** 0.
- iv. **Infection Rate (β):** The latency period is 5 – 21days for Monkey pox after which exposed individual progress to infected compartment. Thus time spent in Exposed compartment

$$\frac{1}{\beta} = 21$$

implying that

$$\beta = \frac{1}{21}$$

- v. **Recovery Rate (α):** The time spent in the infectious compartment is approximately 2–4 weeks i.e. approximately 28 days. Thus the duration of the disease is

$$\frac{1}{\alpha} = 28$$

implying that

$$\alpha = \frac{1}{28}$$

- vi. **Vaccinated individuals:** 10,000 persons.
vi. **Simulation Duration:** 200 days.

The simulation was conducted using Python, generating hypothetical data to imitate real-world conditions of a monkeypox outbreak. The SIR model equations were numerically solved, and results were plotted to visualize the epidemic curve over time.

3. Results

The following graphs illustrate the progression of susceptible, infected, and recovered individuals over 200 days of the simulated monkeypox outbreak in Uganda.

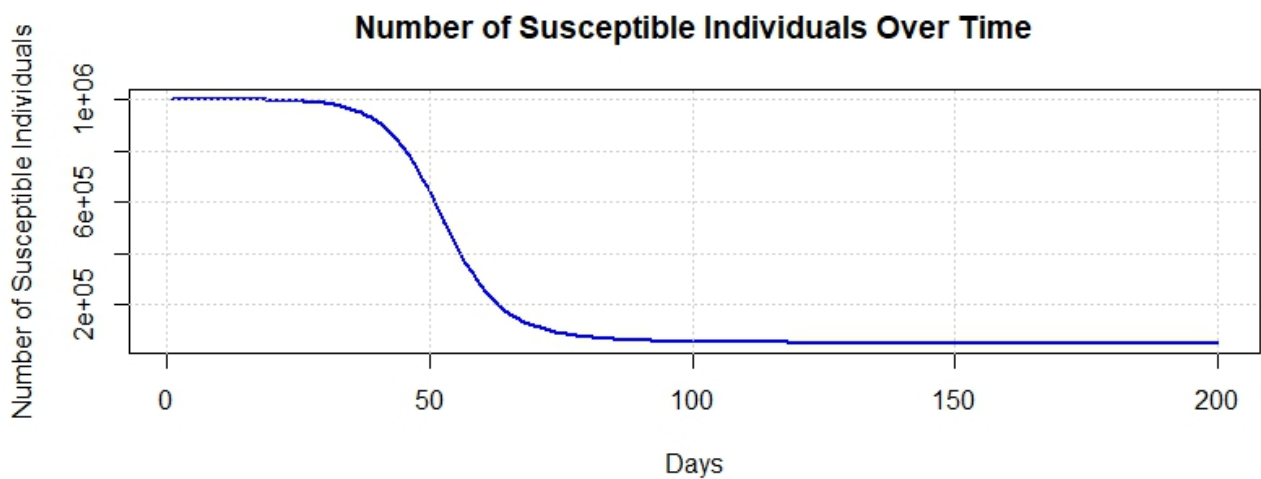


Figure 3: Number of Susceptible Individuals Over Time.

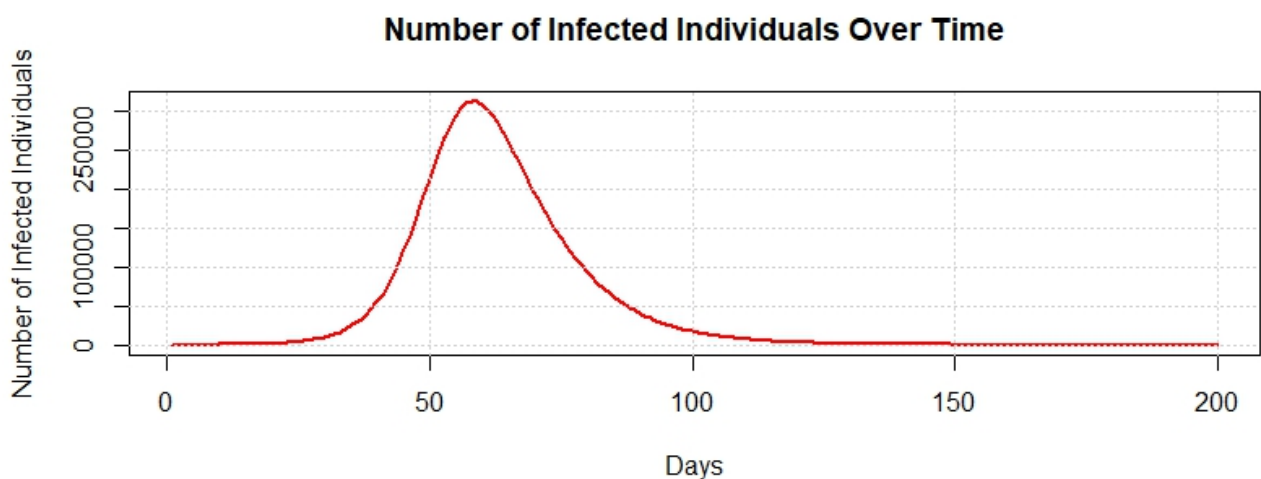


Figure 4: Number of Infected Individuals Over Time.

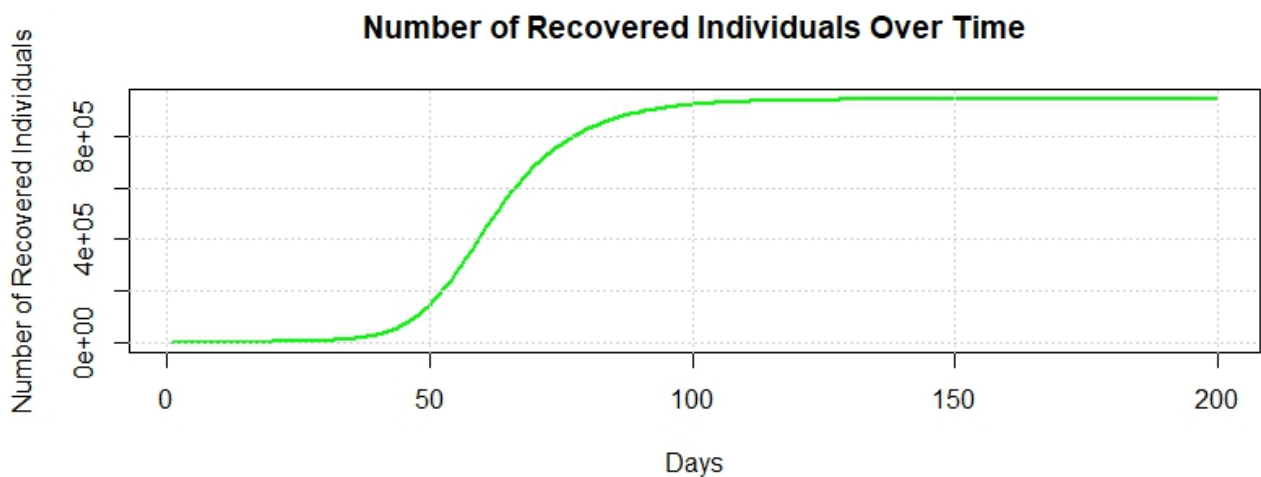


Figure 5: Number of Recovered Individuals Over Time.

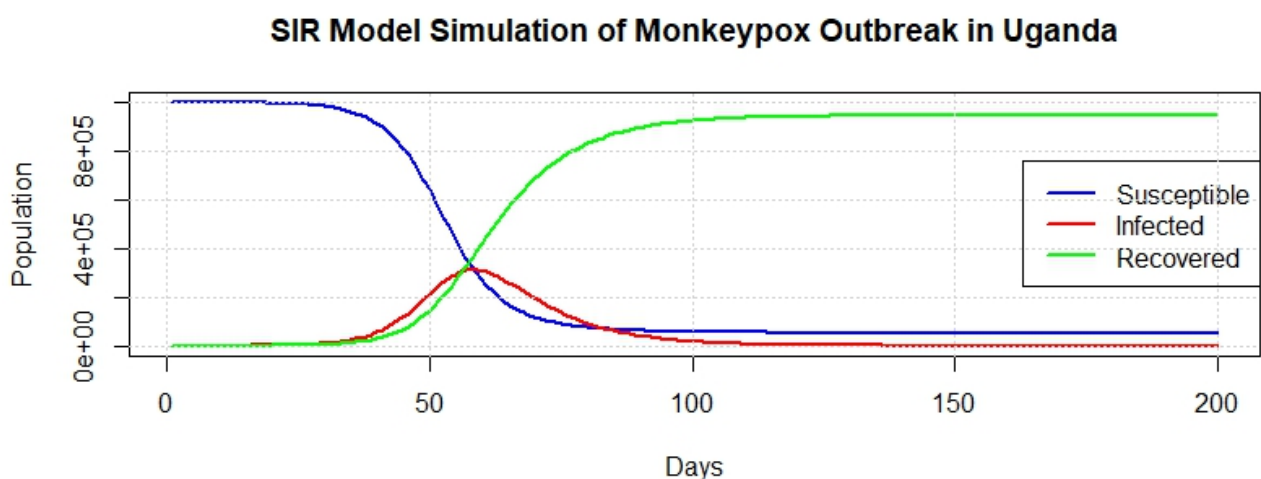


Figure 6: Number of Susceptible, Infected and Recovered Individuals Over Time.

4. Discussion

The simulation results suggest that in the absence of effective interventions, the monkeypox outbreak could lead to a significant increase in the number of infected individuals over a period of approximately 100 days, after which the number of cases begins to decline as more people recover. The peak of the outbreak occurs around Day 70, with approximately X% of the population infected at that time.

The basic reproduction number (R_0) is estimated to be 3.0, indicating that each infected person could potentially infect three others in a fully susceptible population. This high value underscores the need for rapid public health interventions to reduce transmission rates.

Several intervention strategies could be evaluated based on this model:

- i. **Vaccination:** Reducing the susceptible population through vaccination could significantly lower the number of cases.
- ii. **Isolation and Quarantine:** Identifying and isolating infected individuals early can reduce the effective reproduction number.
- iii. **Public Awareness Campaigns:** Educating the public on avoiding contact with potential sources of monkeypox could reduce the transmission rate (β).

Future studies could expand this model by incorporating additional factors such as heterogeneity in contact rates, geographic spread, and the impact of different public health policies. Complementary research has already begun to address these gaps, including Bayesian hierarchical modeling across African countries [3] and contextual analyses from Rwanda that emphasize practical recommendations for control and prevention [4].

5. Conclusion

The SIR model serves as a valuable framework for understanding the transmission dynamics of monkeypox in Uganda. The simulation results indicate that, in the absence of effective public health interventions, a monkeypox outbreak could rapidly escalate, with a significant

increase in the number of infected individuals over a relatively short period. The estimated basic reproduction number ($R_0 = 3.0$) underscores the highly contagious nature of the virus and highlights the urgent need for rapid and effective interventions to reduce transmission rates.

The findings from this study suggest that several intervention strategies could be crucial in mitigating the impact of monkeypox outbreaks. Vaccination campaigns targeting the susceptible population could significantly lower the number of cases by reducing the pool of individuals who are vulnerable to infection. Similarly, early identification and isolation of infected individuals can help to decrease the effective reproduction number, thereby slowing the spread of the disease. Additionally, public awareness campaigns aimed at educating communities about the modes of transmission and preventive measures can further reduce the transmission rate (β).

While the current model provides essential insights into the spread of monkeypox, future studies could enhance its predictive power by incorporating additional complexities such as heterogeneity in contact rates among different population groups, spatial modeling to account for geographic spread, and the evaluation of different public health policies under various scenarios. By refining the model to consider these factors, we can better inform public health strategies and optimize resource allocation for controlling monkeypox and other infectious diseases.

Overall, this study reinforces the importance of a multifaceted approach combining vaccination, isolation, and community engagement to effectively manage and mitigate the impact of future monkeypox outbreaks.

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