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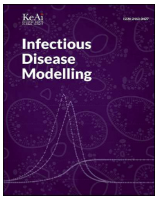

Quantifying mpox transmission and control: A regional analysis of vaccination strategies in East Africa

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ABSTRACT

Africa is home to the endemic mpox disease, especially in the tropical rain-forest regions of Central and West Africa. Although it is mostly found in the Democratic Republic of the Congo, reports of it have also come from other neighboring African nations. To understand the dynamics of mpox, we studied its spread in Burundi, Uganda, Rwanda, Congo, and Kenya before and after the implementation of interventions. Using a Bayesian framework, a simple mathematical model of Susceptible-Infected-Recovered type was calibrated and fitted to the 2022 mpox data covering the period before the introduction of intervention strategies. The model was then re-stratified to incorporate key epidemiological features, including vaccination with imperfect efficacy, partial immunity, exposure, and demographics. The transmission of mpox varied throughout East Africa, with Uganda exhibiting the highest basic reproduction number $\mathcal{R}_0 = 2.51$, suggesting the possibility of a rapid spread. Despite having the highest initial infection count and the lowest $\mathcal{R}_0$ (1.23), Congo may have had delayed detection. The moderate $\mathcal{R}_0$ values (1.35 and 1.88) in Rwanda and Burundi have implications for prompt intervention to control epidemics. Transmission and vaccination rates have a non-linear relationship with the thresholds required to contain mpox outbreaks. Our model shows that in high-transmission settings, substantially higher vaccination coverage (exceeding 80 % at an effectiveness of 70 %) is required to reduce the control reproduction number below unity, whereas in moderate-transmission contexts, coverage above 40 % may suffice. These quantitative thresholds provide actionable guidance for tailoring vaccination strategies to different epidemiological conditions. In particular, sustained vaccination strategies that achieve coverage above the threshold predicted by our model (approximately 80 %) can guarantee mpox eradication, even in situations with strong transmission rates. While real-world complexities such as heterogeneous risk groups and behavioral factors may affect outcomes, these findings shed light on potential quantitative thresholds and provide a foundation for more detailed, population-specific modeling of mpox interventions.

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1. Introduction

Mpox, previously known as monkeypox, is a recurrent emerging zoonosis that causes sporadic human infections. It was first discovered in 1958 in monkeys used for research. The virus was later identified in humans in 1970 in the Democratic Republic of Congo (DRC) (Mitja et al., 2023). Since then, Mpox has remained endemic in parts of Central and West Africa, with occasional outbreaks outside the continent, largely due to international travel (Hakim & Widyaningsih, 2023).

The zoonotic viral disease caused by the Mpox virus, belongs to the Orthopoxvirus genus within the Poxviridae family and the Chordopoxvirinae subfamily. It causes a rash that can be itchy, uncomfortable, and painful (Srivastava et al., 2023). The virus shares genetic and physical similarities with the Variola virus, which causes smallpox. There are three distinct classifications of the Mpox virus-Clade I, from the Congo Basin which has a mortality rate of approximately 10 % in humans, is primarily transmitted by rodents with minimal human-to-human transmission. Clade IIa, from West Africa, has a lower mortality rate of less than 1 % and is zoonotic in nature. Clade IIb is currently spreading around the world with primary transmission from human to human (Gao et al., 2023; Gashema et al., 2024).

Primarily transmitted through direct physical contact with an infected individual's skin, saliva, or mucous membranes, it can also spread through contact with infected hosts. These include humans, domestic or wild animals, the consumption of contaminated animal products or contact with infected materials. Although men who have sex with men (MSM) are considered a key demographic in current transmission dynamics, other risk factors include crowded living conditions and the sharing of personal items or utensils (Gashema et al., 2024).

In recent years, there has been a resurgence of Mpox in Africa, particularly Burundi, Uganda, Rwanda, Congo, and Kenya (collectively known as BURCK countries) (Harris, 2024). The outbreak, which began in late April 2022, has distinct epidemiological and clinical characteristics compared to previous ones (Kumar et al., 2022). 95 % of the 528 reported cases of Mpox were likely transmitted by sexual contact, with the MSM community being disproportionately affected (Pitcock et al., 2023; Rabiu et al., 2024). Sexual transmission has been proposed as the main mode of spread in this current outbreak (Mohapatra et al., 2024).

1.1. Mpox in selected East African (BURCK) countries

Since 2022, East Africa has experienced a resurgence of Mpox cases, with both the West African Clade II and the Central African Clade I identified in the region. This renewed outbreak has drawn significant global attention due to its spread across the BURCK countries, where the management of the virus has been challenging.

1.1.1. Burundi

Burundi, a small, yet densely populated country in East Africa, reported its first case of Mpox on 25 July 2022 (Gashema et al., 2024). The case involved a 36-year-old man who had traveled from the Democratic Republic of Congo (DRC). The predominant strain of the virus in Burundi is Clade II, from West Africa. Human-to-human transmission, primarily through close contact with infected individuals, has been the main route of spread. This initial case highlighted the risk of cross-border transmission, particularly due to Burundi's proximity to Mpox-endemic regions. The estimated basic reproduction number ($R0$) for Mpox in Burundi had not been reported (World Health Organization, 2024a).

Following the first case, Burundi saw a gradual but steady increase in Mpox cases. Despite initial concerns, the country maintained a fatality rate of 0 %. This was largely due to the government's early detection protocols and efficient quarantine measures in August 2022, with international support at the end of 2022 and early 2023, aimed at effectively reducing its spread (World Health Organization, 2024b). In September 2022, Burundi launched a vaccination campaign, prioritizing healthcare workers and people with known exposure to confirmed cases.

In 2023, Burundi reported its first confirmed case of Mpox on 25 July. At the end of 2023, Burundi had reported a total of 40 Mpox cases, mainly in Bujumbura, the region with the most significant case burden. Plans for immunization were underway by December 2023, with commitments from Japan and other donors to assist with vaccine supply to high-risk populations. The country managed to keep the mortality rate at zero, with approximately 75 % of high-risk populations vaccinated by early 2024 (Pal et al., 2024). All reported cases recovered with appropriate medical care.

However, a recent increase in the Clade IIb strain of Mpox has been observed in Burundi (Health Notices, 2024). In mid-October 2024, the country had reported 564 confirmed cases since the resurgence began in July 2024, of which 474 suspected cases (86.9 %) had been investigated and validated. Of 358 suspected cases tested, 142 (39.7 %) tested positive for Mpox (Gashema et al., 2024; World Health Organization, 2024c). Confirmed cases have been reported in 26 of the 49 districts (53.1 %) of Burundi. Bujumbura remaining the epicenter, an urban area, with 54 of the 142 confirmed cases (38 %) (World Health Organization, 2024a).

This outbreak, which is part of a wider resurgence across the continent, has significantly affected both children and adults. Male adults account for 55.6 % of the cases, and female adults for 44.4 %. In particular, approximately one-third of cases have been recorded among school-age children (5–19 years). Children under the age of five years make up 28.9 % of the cases, followed by those aged 11 to 20 (20.4 %), and those aged 21 to 30 (18.3 %) (World Health Organization, 2024c).

Although no specific case fatality ratio for Mpox has been widely reported in Burundi, efforts are ongoing to control the spread, particularly in schools, as the country addresses the increased transmission rates (Unicef, 2024).

1.1.2. Uganda

Uganda, with a history of Mpox dating back to the 1980s, experienced a significant increase in cases. 37 cases were confirmed in May 2022, mainly in rural areas near the border of the DRC (Gao et al., 2023). Most of these cases were related to direct contacts with infected individuals or animals, a situation worsened by human-wildlife interactions. The more virulent Central African Clade I of the Mpox virus is the dominant strain in Uganda (Gashema et al., 2024). The country's public health system faced considerable strain as the virus spread from rural areas to urban centers such as Kampala.

In response, Uganda launched a vaccination campaign in September 2022, initially focusing on border regions and healthcare workers in high-risk areas. This was after the Africa Center for Disease Control and Prevention (ACDCP) allocated 2000 doses of the modified Vaccinia Ankara Bavarian Nordic vaccine (MVA-BN) (Gao et al., 2023). By 2023, the campaign expanded to other parts of the country to help reduce mortality. The government emphasized public health education, raising awareness about Mpox symptoms, the importance of early medical intervention, vaccination, and the risks associated with the virus. At the end of 2023, Uganda had reported a total of 359 confirmed cases of Mpox in 35 districts, with one recorded death, resulting in a fatality rate of approximately 0.3 %. These cases were concentrated in areas with overcrowding and limited healthcare resources (World Health Organization, 2024d).

However, Uganda recorded the highest case fatality rate (CFR) of 4.5 % among East African countries. Despite efforts to improve early diagnosis and treatment, the country confirmed 6 suspected cases of the Mpox virus on 11 July 2024, 2 of which tested positive for Clade Ib strain on 15 July (World Health Organization, 2024c). 39 suspected cases had been reported as of August 12, 2024 and by mid-October 2024, Uganda had recorded a total of 104 confirmed cases of Mpox, with 34 new cases reported in the previous week. The Key affected areas included Kampala (13 cases), Nakasongola (7 cases), Wakiso (5 cases), and 2 confirmed cases among inmates in the Masindi prison (World Health Organization, 2024d). The Ongoing efforts of Uganda's Ministry of Health and its partners focus on surveillance, case management, and public awareness campaigns to manage the outbreak.

1.1.3. Rwanda

Rwanda confirmed its first Mpox case in August 2022. The index case involved a 24-year-old man with no recent travel history, indicating local transmission (Gashema et al., 2024). Although sporadic cases were reported in rural regions, the West African Clade II prevalent in the country was mainly concentrated in urban areas (Harris, 2024). In response to the upsurge, Rwanda's health authorities quickly acted. They launched an investigation into the source of the infection, initiated contact tracking, and identified several suspected cases in Kigali and surrounding areas. Isolation centers were also established, and public awareness campaigns were intensified to limit the spread of the disease (Gashema et al., 2024).

Soon after the first case was detected, Rwanda began its vaccination campaign in November 2022. The campaign targeted healthcare workers and vulnerable populations, such as individuals living with the Human Immunodeficiency Virus (HIV) and other immunosuppressed conditions. This campaign resulted in 60 % vaccination coverage. During the resurgence of the disease in 2023, Rwanda officially reported 85 Mpox cases as part of the larger multi-country outbreak affecting several regions of Africa, including East Africa (Harris, 2024).

In early 2024, approximately 60 % of the target population had been vaccinated, significantly reducing the spread of the virus. Despite this, Rwanda's mortality rate was 1.2 %, with the R0 estimated at 1.5. The majority of fatalities were among patients with pre-existing conditions or those who experienced delayed access to healthcare (World Health Organization, 2024a).

On 27 July 2024, the Ministry of Health declared an outbreak of mpox in the country. The cases included a 33-year-old woman (case 1) who frequently travels to the DRC and a 34-year-old man (case 2) with a recent travel history to the DRC. By 7 August 2024, the country had cumulatively reported 4 confirmed cases of Mpox and zero deaths. Among the two new cases, one was a 34-year-old man who had returned from Burundi on 12 July 2024 (World Health Organization, 2024c).

As of October 2024, Rwanda had reported 6 confirmed Mpox cases during the latest multi-country outbreak. The strain responsible for this surge is Clade IIb, which has been the predominant strain in Africa during this period (World Health Organization, 2024a).

Rwanda's relatively low mortality rate is attributed to early diagnosis, a strong healthcare infrastructure, and effective vaccination strategies. However, despite the government and health authorities' efforts to improve healthcare infrastructure and timely medical interventions, challenges remained. Issues such as the accessibility of vaccines in remote areas and the general capacity of the healthcare system continue to pose obstacles to fully curtail the spread of the virus (Pal et al., 2024).

1.1.4. Congo

The DRC has long been the epicenter of Mpox, experiencing continuous human-to-animal and human-to-human transmission since the 1970s. The Central African Clade I, known for higher fatality rates and more severe symptoms, remains dominant in the country (Gao et al., 2023). This has presented a persistent public health challenge, especially in remote regions with weak healthcare infrastructure.

In 2022, the DRC experienced one of its most severe outbreaks in recent years, reporting more than 1200 cases and a CFR of approximately 3.6 %. The basic R0 of the virus is estimated at 2.1, with outbreaks occurring primarily in remote provinces such as Equateur and Tshuapa, where access to quality healthcare is limited (World Health Organization, 2024e). This underscores the severity of the situation in these regions.

The cases of Mpox have been steadily increasing and spreading in the DRC region since 2022. A year later, the cases increased sharply while neighboring countries, including Burundi, Uganda, Rwanda, and Kenya, declared mpox outbreaks in July 2024. In an attempt to control the outbreak since August 2022, the Congolese government has renewed its effort, in partnership with the World Health Organization (WHO), to vaccinate high-risk populations (Gashema et al., 2024). However, vaccine distribution has faced significant challenges due to the large size of the country and complex geography, particularly in rural areas.

By early 2024, approximately 40 % of the target population had been vaccinated. Although vaccination campaigns have shown some success in reducing transmission rates, logistical issues, such as limited infrastructure and access in remote regions, continue to hinder broader immunization efforts. Despite these efforts, the DRC remains significantly affected by the Clade Ib strain of the Mpox virus, which has a CFR ranging from 1.4 % to more than 10 %. From January to October 2024, around 41 % of suspected cases in the DRC have been tested, with a test positivity of approximately 46 % and had a travel history to the DRC (World Health Organization, 2024a).

Since January 20, 2023, more than 20,000 suspected cases have been reported, with more than 1000 deaths. This higher mortality rate can be attributed to several factors, including delayed treatment, lack of access to adequate healthcare in remote areas, limited diagnostic capabilities, and the prevalence of comorbidities such as malnutrition and HIV.

1.1.5. Kenya

Kenya's first case of Mpox, reported in June 2022, involved a traveler returning from West Africa, where there had been a surge in Mpox infections (Kumar et al., 2022). Although the patient was quarantined upon arrival, subsequent cases appeared in major urban centers, particularly in Nairobi and Mombasa. Like Rwanda and Burundi, Kenya's Mpox cases were mainly of the West African Clade II. The spread of the virus was driven by high population density in urban areas and human-to-human contact, as well as interactions between animal reservoirs in rural and urban regions (Harris, 2024).

Kenya launched its vaccination campaign in October 2022, prioritizing healthcare workers and high-risk populations. With the support of international organizations, the government managed to acquire vaccines, achieving a 55 % vaccination rate by 2024 (Gashema et al., 2024). Public health messages emphasized hygiene, minimizing risks of zoonotic transmission, and the use of personal protective equipment (PPE) in hospitals and laboratories. The country maintained a CFR of 2 %, with prompt isolation and treatment playing a crucial role in controlling mortality. Kenya's relatively moderate mortality rate is attributed to early isolation of patients and the availability of medical care in urban centers (World Health Organization, 2024e).

By early 2024, Kenya had recorded 150 Mpox cases, making it the second-highest in East Africa, after the DRC and Uganda, but higher than Rwanda's. The country's CFR remained at 2 %. In 2024, Kenya had reported a total of 14 confirmed Mpox cases; one case tested positive for Clade Ib, 12 cases tested negative, and the test result for one case was pending, including one death (Gashema et al., 2024; World Health Organization, 2024e). The strain circulating in Kenya belongs to Clade I, which is known for higher virulence and is endemic to several African countries.

The Kenyan government has implemented various measures to contain the outbreak, including deploying rapid response teams for investigations, establishing public health emergency coordination centers, and conducting public sensitization campaigns to combat stigma and promote prevention. However, challenges such as the distribution of vaccines in rural areas continue to hinder the fight against the virus.

These shared experiences (healthcare infrastructure, public awareness, cross-border transmission dynamics, and the persistent issue of vaccine availability) between these East African BURCK countries highlight the critical need for a study of sexual transmission dynamics, continued vigilance, and investment in public health measures to control the spread of Mpox and other infectious diseases. Table 1 consists of the list of BURCK countries, the number of their reported confirmed mpox cases, the date of their first confirmed index case, including past and current interventions implemented against Mpox in the countries with dates.

1.2. Modeling mpox transmission in BURCK countries

Over the years, mathematical modeling (MM) has become an indispensable tool for understanding the transmission dynamics of infectious diseases. Various mathematical models have been used to predict disease spread and assess the impact of control measures, dating back to Bernoulli's work in 1760 (Choisy et al., 2007). Since then, numerous studies have focused on the spread and control of Mpox worldwide, including East Africa, using MM approaches (see (Peter et al., 2022)-(Nia et al., 2023) and the references therein).

Although specific models targeting East African BURCK countries remain limited, some relevant studies can be adapted to the region (Peter et al., 2024; Rabiu et al., 2024). With the resurgence of Mpox in 2022, existing models investigate key aspects of the dynamics of Mpox, offering frameworks to simulate outbreaks, evaluate control strategies, and predict the impact of interventions such as vaccination (Zhang et al., 2024a). This approach provides valuable information on the management of Mpox in East Africa.

For example, a recent study developed a model that incorporates four transmission routes, including sexual behaviors in the MSM and women who have sex with women (WSW) communities, to capture trends in Mpox transmission during its outbreak in the United States. The model disaggregated the data into male and female populations using data analysis techniques, while Bayesian inference was used to fit the model to the daily reported Mpox data. In addition, the study

Table 1
List of BURCK countries, number of their reported confirmed mpox cases, date of their first confirmed index case, including past and current interventions implemented against Mpox in the countries with dates.

Country	Confirmed cases	Date of First confirmed index case	Interventions implemented with date
Burundi	2	25-July-2022 (Gashema et al., 2024)	**Early Aug:** Early detection protocols, efficient quarantine measures, and contact tracing effected (World Health Organization, 2024d). **Dec 1–5:** Vaccination Campaign Launch and International support by WHO and Africa CDC (World Health Organization, 2024b)
	40	25-July-2023 (Pal et al., 2024)	**Late Jan:** Immunization supply, intensified public awareness campaigns and High-Risk populations vaccination (Pal et al., 2024).
	564	July-2024 (Gashema et al., 2024; World Health Organization, 2024c)	**Nov 1–24:** Ongoing vaccine allocation and implementation efforts (Unicef, 2024).
Uganda	37	12-May-2022 (Gao et al., 2023)	**Sept 22:** Vaccination Campaign Launch (Gao et al., 2023).
	359	1-Dec-2023 (World Health Organization, 2024d)	**Late Dec:** Public health education and awareness; medical intervention and vaccination (World Health Organization, 2024c).
	138	12-Oct-2024 (World Health Organization, 2024c)	**Early Nov:** Efforts on Surveillance, case management and public awareness campaign ongoing (World Health Organization, 2024c).
Rwanda	1	12-Aug-2022 (Gashema et al., 2024)	**Mid Nov:** Contact tracing initiated; Isolation centers set up; Public Campaigns intensified; and Vaccination campaign began (Gashema et al., 2024).
	85	23-July-2023 (Harris, 2024)	**Late July:** Declaration of a Public Health Emergency; implementation of increased surveillance (Harris, 2024).
	10	31-Aug-2024 (World Health Organization, 2024a, 2024c)	**Aug 7-Oct 31:** Surveillance and contact tracing; patients' isolation; community engagement and public sensitization campaigns raised (World Health Organization, 2024c).
Congo	1200	6-May-2022 (World Health Organization, 2024e)	**Early July - Late Nov:** Vaccine allocation; contact tracing and surveillance; public awareness campaign; health workers training (World Health Organization, 2024c).
	20,000	16-July-2024 (Gashema et al., 2024)	**Aug 5 -Late Oct:** High-risk population Vaccination still ongoing (Gashema et al., 2024).
Kenya	1	3-June-2022 (Harris, 2024; Kumar et al., 2022)	**Early Oct:** Vaccination campaign and international support received; isolation and treatment began [5, 18].
	150	29-July-2024 (Gashema et al., 2024; World Health Organization, 2024e)	**Aug 2 -Nov 6:** Emergency coordination centers set up; Sensitization campaigns started, vaccine doses allocated (World Health Organization, 2024e).

performed scenario analysis on R0 and proposed control strategies, such as condom use, with simulations to validate the results (Rabiu et al., 2024).

Although numerous studies have used MM to explore the dynamics of Mpox (Rabiu et al., 2024),(Peter et al., 2022)-(Zhang et al., 2024a), only a few have directly targeted BURCK countries or adapted these studies for the East African context (Peter et al., 2024). To date, no research has studied the interplay between the basic reproduction number before the implementation of intervention strategies (such as vaccination, condom use and social distancing) and after the launching of vaccination strategies in the BURCK countries.

In this study, we present in section 1, the historical and current dynamics of Mpox transmission across selected BURCK East African countries, highlighting outbreak trends before and after public health interventions from its emergence in 1958 to the present. The next section develops and calibrates a basic Susceptible-Infectious-Recovered (SIR) model using 2022 outbreak data within a Bayesian inference framework. This model is later re-stratified to incorporate key epidemiological features, including partial immunity, imperfect efficacy vaccination, exposure, and demographic factors. Section 3 estimates critical parameters such as the basic $\mathcal{R}_0$, the control reproduction number $\mathcal{R}_t$ and epidemic final size. In Section 4, numerical simulations illustrate the results of the model and assess the impact of varying vaccination coverage and effectiveness. Section 5 concludes with implications for context-sensitive public health responses in resource-limited settings, limitation of the study and suggestion for future direction.

2. Materials & methods

2.1. Data

In this work, the current demographic data Table 1 and the daily reported mpox data (available on *Our World in Data* website (Our World in Data)) for the BURCK countries were used. The mpox data contains a 7-day rolling average of mpox cases from the date when case reporting started in those countries to date. The data have several variables such as new case, total case, death, etc. We extracted cases reported from the beginning to the time just before the implementation of control intervention strategies. This is to understand the initial spread of the new mpox strain and also to estimate its basic R0. In Burundi, we extracted the new_cases_smoothed data from 21/07/2024 to 13/10/24 which is equivalent to 85 days. In Uganda, the new_cases_smoothed data was extracted from 30/06/2024 to 12/10/24, equivalent to 106 days. In Rwanda, we extracted the total_cases_smoothed data from 14/07/2024 to 13/10/24 which is equivalent to 92 days. In Congo, the total_cases_smoothed data from 02/08/2023 to 23/03/24 which is equivalent to 235 days was extracted. In Kenya, we extracted the total_cases_smoothed data from 21/07/2024 to 13/10/24 which is equivalent to 85 days. We used the total_cases_reported in Rwanda, Kenya, and Congo because the newly reported cases in those countries are too small to be caliberated. Compared to the previous strain, clade IIb, the new mpox strain, clade Ib, is more transmissible and has a higher fatality rate. It has been found in several nations and is spreading throughout Central and East Africa.

2.2. Methods

To determine the basic R0 of the novel mpox strain in BURCK, we employ a simple susceptible-exposed-infectious-recovered (SEIR) compartmental model. Unvaccinated susceptible (S_u), exposed (E), infectious (I), and recovered/removed (R) are the four components of this model. Although infectious people in I can spread the disease to their contacts to some extent, people in the exposed compartment do not spread the disease (Ganyani et al., 2020; Iyaniwura et al., 2022; Li et al., 2020; Liu et al., 2020). According to (Rossotti et al., 2023), there are both symptomatic and asymptomatic people in our infectious compartment I. We make the assumption that people who have recovered will not contract the disease again throughout the time frame we considered.

2.3. Epidemic form of the model

The baseline model is given by (which is an extension of the classical SIR model by [?])

$$\begin{aligned}\frac{dS_u}{dt} &= -\frac{\beta I}{N}S_u,\\ \frac{dE}{dt} &= \frac{\beta I}{N}S_u - \alpha E,\\ \frac{dI}{dt} &= \alpha E - \gamma I,\\ \frac{dR}{dt} &= \gamma I,\end{aligned} \tag{1}$$

where β is the rate of transmission, α is the rate of transitioning from the exposed compartment E to the infectious compartment I and γ is the recovery rate. By inspection, the basic reproduction number $\mathcal{R}_0$ of (1) is given by

$$\mathcal{R}_0 = \frac{\beta}{\gamma}. \tag{2}$$

2.4. Endemic form of the model

The model with control intervention (vaccination and condom usage) is given by

$$\begin{aligned}\frac{dS_u}{dt} &= \Lambda - \frac{\beta(1-\alpha_1\alpha_2)I}{N}S_u - (\tau+\mu)S_u,\\ \frac{dS_v}{dt} &= \tau S_u - (1-\epsilon)\frac{\beta(1-\alpha_1\alpha_2)I}{N}S_v - \mu S_v,\\ \frac{dE}{dt} &= \frac{\beta(1-\alpha_1\alpha_2)}{N}(S_u + (1-\epsilon)S_v)I - (\alpha+\mu)E,\\ \frac{dI}{dt} &= \alpha E - (\gamma+\eta+\mu)I,\\ \frac{dR}{dt} &= \gamma I - \mu R,\end{aligned} \tag{3}$$

where α_1 is the condom-usage rate, α_2 is the condom efficacy, τ is the vaccination rate, Λ is the recruitment rate, μ is the natural death rate, η is the disease-induced death rate and ϵ is the vaccine efficacy. It can be easily observed, using the Next Generation Matrix (Diekmann et al., 1990; Van den Driessche & Watmough, 2002), that the control reproduction number $\mathcal{R}_t$ (under the implementation of condom usage and vaccination) for model (3) is given by

$$\mathcal{R}_t = \frac{\beta\alpha}{\gamma+\mu+\eta}\,\frac{(1-\alpha_1\alpha_2)(\mu+(1-\epsilon)\tau)}{(\alpha+\mu)(\tau+\mu)}. \tag{4}$$

The control reproduction number $\mathcal{R}_t$ captures the risk of mpox transmission in the context of demographic trends and public health interventions such as vaccination and condom use. The transmission rate β, the progression rate from exposure to infection α, and a behavioral adjustment term $(1-\alpha_1\alpha_2)$, which symbolizes the decrease in transmission caused by condom use α_1 and efficacy α_2, make up the numerator of $\mathcal{R}_t$. In addition, the natural mortality rate μ and the poor protection provided by vaccination are both taken into account by the term $\mu + (1 - \epsilon)\tau$. The term $(1 - \epsilon)\tau$ is small if the efficacy of the vaccine ϵ is high, which lowers $\mathcal{R}_t$. On the other hand, this term increases and the effective reproductive number increases in the event of low efficacy or low vaccination coverage. Together, these components imply that sub-optimal vaccination and poor behavioral compliance can considerably prolong the spread of mpox.

The terms that represent demographic changes of the population and the removal of people from contagious and exposed classes make up the denominator. The product of recovery, natural death, and disease-induced death rates $(\gamma + \mu + \eta)$, which decreases the infectious period and consequently the potential for transmission, is $(\gamma + \mu + \eta)(\alpha + \mu)(\tau + \mu)$.

$(\tau + \mu)$ denotes the rate at which susceptibles are eliminated by vaccination, while $(\alpha + \mu)$ controls the rate at which exposed individuals leave the latent class, either by becoming infectious or passing away. The reproductive number is suppressed as these removal rates increase. This framework emphasizes how crucial an effective transition from infectious stages and robust intervention coverage is to mpox control. The fact that $\mathcal{R}_t$ is sensitive to several factors highlights the need for a mix of tactics, such as high vaccination rates, efficient condom use, and rapid case recovery, to sustainably lower $\mathcal{R}_t$ below unity.

2.5. Bayesian inference

As previously mentioned, the model (1) was fitted to the provided daily mpox data. The posterior mean was calculated using the Bayesian Inference Framework in the RStan package (Stan Development Team, 2020) in R version 4.4.1 (R R Core Team et al., 2013). Using this probabilistic framework, we evaluate the probabilistic claims made about the data inside the framework of the model and estimate the model parameters while taking into account our past knowledge known as prior. The sampling distribution, sometimes referred to as likelihood, involves the generation of data based on the model parameters. We use the posterior distribution to predict suitable parameter values based on the data. The effective sample size, standard deviation, mean, diagnostic plots, and other useful information are instrumental in evaluating the correctness of the estimated parameters and the data generating results. The degree of convergence of the iteration is indicated by R-hat. The closeness of $\widehat{R}$ to 1 indicates convergence of chains. In most cases, 4 chains are essential to ensure convergence. In general, the Bayesian framework is expressed as

$$T(\Gamma|\mathcal{K}) \propto T(\mathcal{K}|\Gamma)T(\Gamma), \tag{5}$$

where $T(\Gamma|\mathcal{K})$ is the likelihood, $T(\mathcal{K}|\Gamma)$ is the posterior distribution, and $T(\Gamma)$ is the prior. The expression of the likelihood is

$$\text{daily\_mpox\_reported\_cases}(t) \sim \text{NegBin}(\text{predict\_cases}(t),\ \phi), \tag{6}$$

where daily_mpox_reported_cases is the reported mpox cases in BURCK at time t (measured in days), predict_cases(t) is the predicted daily mpox cases computed from the model (1), NegBin($\cdot$) is the negative binomial distribution function in RStan, and ϕ is the over-dispersion parameter. The estimation of parameters and fitting process were done using the adaptive Hamiltonian Monte Carlo method No-U-Turn sampling (NUTS) in RStan with 10,000 iterations (where 5000 iterations were discarded for warm-up). For more information on this method, see (Cole et al., 2018; Hoffman Gelman et al., 2014). We used uniform distribution for the estimation of β, normal distribution for the estimation of γ and ϕ while lognormal distribution was used to estimate the initial conditions.

2.6. Model fitting and parameter estimation

In this section, we present the results of how our model (1) fits the mpox data in BURCK which can be seen in Table 2 and thses were used in Table 4 for numerical analysis. The fits can be found in Figs. 1–3. All the dates were extracted from the available mpox data in (Our World in Data) and information on Table 1. We further estimated the transmission rate β, recovery rate γ, overdispersion parameter ϕ and initial condition I_0. In the model equation (1), αE was used to model the simulated 7-day rolling average of confirmed mpox cases. The parameter values are presented in Table 5.

The estimated parameter values and $\mathcal{R}_0$ of all provinces are presented in Table 5. The parameters were estimated because of their inassessibility in the literature. The values of the fixed parameter values are given in Table 3.

The estimated parameters and basic reproduction numbers $(\mathcal{R}_0)$ shown in Table 5 allow for a comparative study of the dynamics of mpox transmission in Burundi, Uganda, Rwanda and Congo, revealing unique epidemiological trends and difficulties (see Table 4). A highly transmissible epidemic is indicated by Uganda's highest basic reproduction number, which is $\mathcal{R}_0 = 2.51$ (95 % CrI: 2.02–3.22). The highest estimated transmission rate ($\beta = 0.25$) among countries is consistent with this high transmission. Congo, on the other hand, has the lowest $\mathcal{R}_0 = 1.23$ (95 % CrI: 1.17–1.30), indicating a slower rate of spread, although there were many more infectious persons at the beginning ($I(0) = 296.43$), which may indicate earlier under-reporting or delayed detection. Variations in mpox control measures, response to the health system, and social interaction patterns between these countries are reflected in the variances in $\mathcal{R}_0$ the values.

A relatively low $\beta = 0.12$ supports Rwanda's moderate transmission intensity, with $\mathcal{R}_0 = 1.35$ (95 % CrI: 1.22–1.53). It is interesting to note that Rwanda's low estimated partial immunity ($\phi = 0.02$) may indicate weak historical vaccination coverage or low natural exposure to related orthopoxviruses such as smallpox. With $\mathcal{R}_0 = 1.88$ and a transmission rate

Table 2
Description of variables and parameters.

Variable	Description
S_u	Unvaccinated Population
S_v	Vaccinated population
E	Exposed population
I	Infectious population
R	Recovered or removed population

Parameter	Description
β	Transmission rate
Λ	Recruitment rate
μ	Natural death rate
η	Disease-induced death rate
τ	Vaccination rate
α_1	Proportion of people using condom
α_2	Condom efficacy
ϵ	Vaccine efficacy
γ	Recovery rate
α	Incubation period

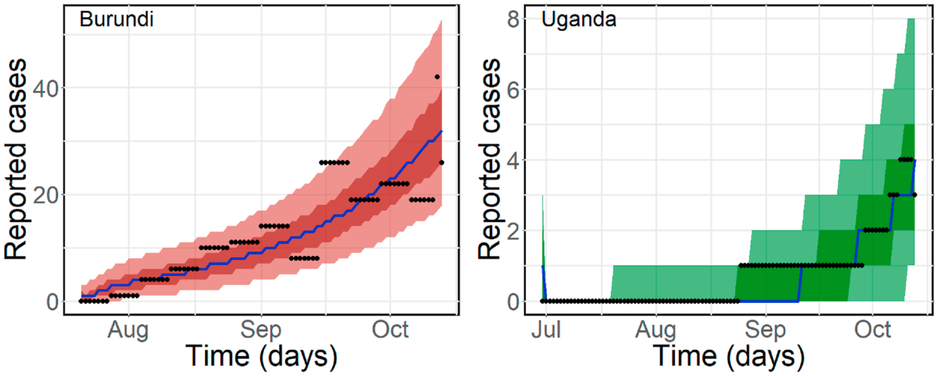

Fig. 1. Model fit using the daily reported mpox cases in Burundi from 21/07/2024 to 13/10/24 equivalent to 85 days. In Uganda, we extracted the data from 30/06/2024 to 12/10/24 which is equivalent to 106 days. The black line indicates the reported cases data while the blue line indicates the model fit. The deeply coloured region represents 50 % CrI while the lightly coloured region represents 95 % CrI.

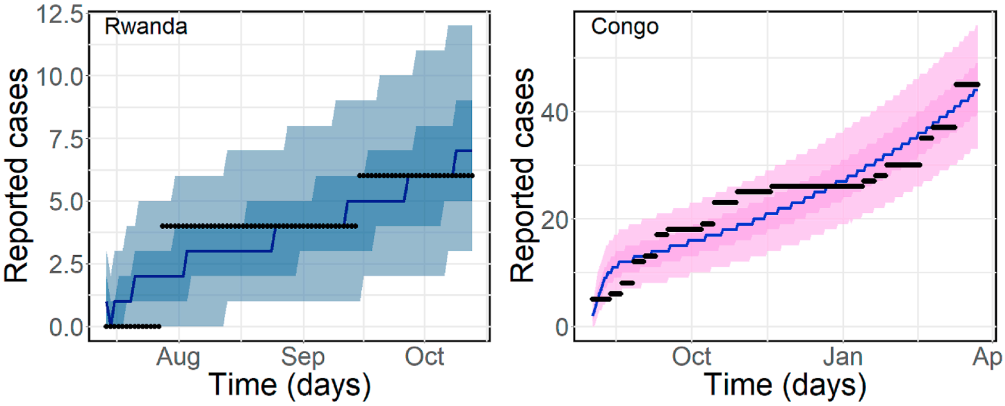

Fig. 2. Model fit using the daily reported mpox cases in Rwanda from 14/07/2024 to 13/10/24 equivalent to 92 days. In Congo, we extracted the data from 02/08/2023 to 23/03/24 which is equivalent to 235 days. The black line indicates the reported cases data while the blue line indicates the model fit. The deeply coloured region represents 50 % CrI while the lightly coloured region represents 95 % CrI.

$\beta = 0.15$, Burundi exhibits a similar pattern, suggesting a stronger potential for spread than Rwanda, but a lower potential than Uganda. In particular, the close $I(0)$ values of 51.27 and 44.40 for Burundi and Rwanda, respectively, suggest that

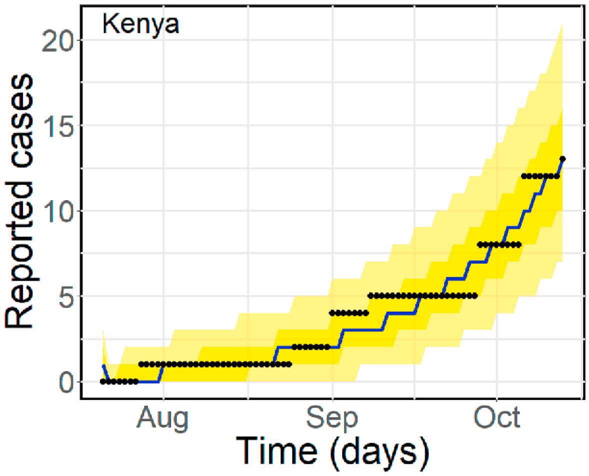

Fig. 3. Model fit using the daily reported mpox cases in Kenya from 21/07/2024 to 13/10/24 which is equivalent to 92 days. The deeply coloured region represents 50 % CrI while the lightly coloured region represents 95 % CrI.

transmission parameters, not initial burden, are the main cause of variations in $\mathcal{R}_0$. These dynamics demonstrate the need for nation-specific control measures, as well as the variation in outbreak trajectories within the region.

Uganda's low initial case count and high $\mathcal{R}_0$ indicate the possibility of a rapidly spreading outbreak from a public health point of view, provided that early interventions are not maintained or accelerated. Due to this, Uganda is especially susceptible to resurgences in the future, particularly if public education, contact tracking, and vaccine campaigns are insufficient. In contrast, Congo's low $\mathcal{R}_0$ is positive, but the high initial number of infections requires monitoring. It implies that even though the virus might not spread quickly, it might already have infected a sizable vulnerable population, in which case strong containment and monitoring measures would be required. In high-contact societies, the difference between low β and high $I(0)$ may also indicate a slower but sustained spread or underestimation in the past.

Rwanda and Burundi are in the middle, where prompt action could greatly reduce the spread of the disease. Their reasonable $\mathcal{R}_0$ values suggest that community involvement and focused vaccination could reduce the reproduction number below 1, which could stop outbreaks. However, the comparatively greater degree of uncertainty in their credible interval ranges suggests that better data quality and surveillance are required. Furthermore, both nations benefit from closer regional collaboration with their neighbors, especially Uganda and Congo, where people's mobility and porous borders raise the risk of cross-border transmission. In such a coordinated regional effort, border health checks and vaccinations will be crucial.

In summary, these results highlight the fact that the dynamics of mpox transmission vary throughout East Africa. For outbreak control to be effective, customized interventions that take into account demographic characteristics, historical immunity, and the possibility of local transmission are essential. High $\mathcal{R}_0$ countries, such as Uganda, should focus on aggressive containment, while countries, such as Congo, should improve continuing case management and early detection. In order to stop a larger regional spread, it is essential to maintain diligent observation and strengthen cross-border coordination. For the purpose of organizing vaccination plans, forecasting resource requirements and assessing the possible effects of public health interventions in various epidemiological scenarios, these estimates provide an essential quantitative basis.

3. Final size of the mpox model

The total number of people in the population who contract the disease during an epidemic is known as the final size of the epidemic. It is critical to comprehend the final size for a number of reasons: a gauge of the overall impact of the epidemic on the populace is given by the final size. Provides public health officials with an estimate of the number of people who are likely to contract the disease at the end of the outbreak. This helps in planning medical resources such as hospital beds, prescription drugs, and medical personnel. Considering our model equation (1), we need to note that

$$N = S + E + I + R. \tag{7}$$

In the final state of the epidemic ($t \to \infty$), we have the following conditions:

- $I \to 0$ (no infectious individuals remain),
- $E \to 0$ (no exposed individuals remain),

Table 3
Table of fixed parameters.

Parameter	Value	Reference
τ	$0.45 \in [0,1]$	Assumed
α_1	$0.25 \in [0,1]$	Assumed
α_2	$0.65 \in [0,1]$	Assumed
ϵ	$0.76 \in [0,1]$	(World Health Organization)
α	$\frac{1}{12} \in [\frac{1}{5}, \frac{1}{20}]$	(Matusali et al., 2023; Zhang et al., 2024b)
Λ	$\mu * N(0)$	Computed
μ	$\frac{1}{60}$	(Galal)
η	0.001	Assumed

Table 4
Model initial conditions. These initial conditions are based on the average population of East African countries. The unvaccinated susceptible population was computed using $S_u(0) = N(0) - (S_v(0) + I(0) + R(0))$.

Variable	Initial population	Value
$N(0)$	Total population	15, 897, 567
$S_u(0)$	Unvaccinated susceptible population	13, 492, 671
$S_v(0)$	Vaccinated susceptible population	1, 535, 256
$E(0)$	Exposed population	508,050
$I(0)$	Infectious population	9, 030
$R(0)$	Recovered population	352, 560

Table 5
Table of estimated parameters, initial condition, Basic R0 $\mathcal{R}_0$ and their 95 % credible interval (CrI).

Parameter/Initial Condition	Value	Reference	Country
α	$\frac{1}{12}$	(Matusali et al., 2023; Zhang et al., 2024b)	Burundi
γ	0.08 (95 % CrI:0.04–0.13)	Estimated	
β	0.15 (95 % CrI: 0.10–0.21)	Estimated	
ϕ	0.07 (95 % CrI:0.03–0.13)	Estimated	
$\mathcal{R}_0$	1.88 (95 % CrI: 1.63–2.27)	Estimated	
$I(0)$	51.27 (95 % CrI: 35.20–74.48)	Estimated	
$N(0)$	14,047,786	Worldometer (2025)	
α	$\frac{1}{12}$	(Matusali et al., 2023; Zhang et al., 2024b)	Uganda
γ	0.10 (95 % CrI:0.06–0.14)	Estimated	
β	0.25 (95 % CrI: 0.18–0.33)	Estimated	
ϕ	0.07 (95 % CrI:0.01–0.21)	Estimated	
$\mathcal{R}_0$	2.51(95 % CrI: 2.02–3.22)	Estimated	
$I(0)$	27 (95 % CrI: 24–74.48)	Estimated	
$N(0)$	50,015,092	Worldometer (2025)	
α	$\frac{1}{12}$	(Matusali et al., 2023; Zhang et al., 2024b)	Rwanda
γ	0.09 (95 % CrI:0.05–0.13)	Estimated	
β	0.12 (95 % CrI: 0.07–0.17)	Estimated	
ϕ	0.02 (95 % CrI:0.00–0.05)	Estimated	
$\mathcal{R}_0$	1.35 (95 % CrI: 1.22–1.53)	Estimated	
$I(0)$	44.40 (95 % CrI: 29.07–67.21)	Estimated	
$N(0)$	14,256,567	Worldometer (2025)	
α	$\frac{1}{20}$	(Matusali et al., 2023; Zhang et al., 2024b)	Congo
γ	0.13 (95 % CrI:0.11–0.16)	Estimated	
β	0.16 (95 % CrI: 0.13–0.18)	Estimated	
ϕ	0.00 (95 % CrI:0.00–0.00)	Estimated	
$\mathcal{R}_0$	1.23 (95 % CrI: 1.17–1.30)	Estimated	
$I(0)$	296.43 (95 % CrI: 287.58–299.90)	Estimated	
$N(0)$	102,521,710	Worldometer (2025)	
α	$\frac{1}{12}$	(Matusali et al., 2023; Zhang et al., 2024b)	Kenya
γ	0.10 (95 % CrI:0.06–0.14)	Estimated	
β	0.20 (95 % CrI: 0.14–0.27)	Estimated	
ϕ	0.02 (95 % CrI:0.00–0.0.05)	Estimated	
$\mathcal{R}_0$	2.07 (95 % CrI: 1.81–2.48)	Estimated	
$I(0)$	8 (95 % CrI: 4.77–11.96)	Estimated	
$N(0)$	56,432, 944	Worldometer (2025)	

- S_∞ is the final number of susceptible individuals,
- R_∞ is the final number of recovered individuals.

The equation for $\frac{dS}{dt}$ is:

$$\frac{dS}{dt} = -\beta \frac{SI}{N}.$$

Dividing both sides by S gives:

$$\frac{dS}{S} = -\frac{\beta}{N} I \, dt.$$

Using the equation $\frac{dR}{dt} = \gamma I$, we express $I \, dt$ as:

$$I \, dt = \frac{1}{\gamma} \, dR.$$

Substitute this into $\frac{dS}{S}$:

$$\frac{dS}{S} = -\frac{\beta}{N\gamma} \, dR.$$

Now, we need to integrate the above equation from the initial state ($S(0) = S_0$, $R(0) = 0$) to the final state ($S(\infty) = S_\infty$, $R(\infty) = R_\infty$). In this way, we have

$$\int_{S_0}^{S_\infty} \frac{dS}{S} = -\frac{\beta}{N\gamma} \int_0^{R_\infty} dR.$$

This results to:

$$\ln\left(\frac{S_\infty}{S_0}\right) = -\frac{\beta}{N\gamma} R_\infty. \tag{8}$$

According to equation (2), the basic R0 R_0 is defined as:

$$R_0 = \frac{\beta}{\gamma}.$$

Substituting equation (2) into equation (8), we have

$$\ln\left(\frac{S_\infty}{S_0}\right) = -\frac{R_0}{N} R_\infty.$$

Since $R_\infty = N - S_\infty$, we substitute R_∞ into the equation:

$$\ln\left(\frac{S_\infty}{S_0}\right) = -R_0\left(1 - \frac{S_\infty}{N}\right).$$

We take the exponential of both sides to get

$$\frac{S_\infty}{S_0} = e^{-R_0\left(1 - \frac{S_\infty}{N}\right)}.$$

The final size equation is:

$$S_\infty = S_0 e^{-R_0\left(1 - \frac{S_\infty}{N}\right)}.$$

The total number of infected individuals during the mpox outbreak is:

$$\text{Total Cases} = N - S_{\infty}.$$

4. Numerical simulations

In this section, we numerically simulate the potential effects of control interventions (such as vaccination and condom use) in controlling the spread of mpox.

Using a deterministic compartmental model calibrated to realistic epidemiological parameters. Fig. 4 shows how the vaccination rate (τ) and the effectiveness of the vaccine (ϵ) affect the temporal evolution of the infected population $I(t)$ and the number of control reproductions $\mathcal{R}_t$ while the transmission rate β remains constant. While changing the vaccination rate from $\tau = 0.2$ to 1.0, the dynamics of infection is simulated in the left panel assuming a fixed vaccine efficacy of $\epsilon = 0.76$ and a transmission rate $\beta = 0.67$. The results demonstrate a clear inverse relationship between the vaccination rate and the maximum number of infections. As τ increases, the epidemic curve becomes increasingly flattened and the infection peak is reduced and occurs sooner. The associated reproduction numbers for the various vaccination rates gradually decline from $\mathcal{R}_t = 1.1281$ to $\mathcal{R}_t = 0.9542$, highlighting the importance of accelerating vaccine rollout speed to bring $\mathcal{R}_t$ below the critical threshold of one, which is required for epidemic control. This behavior illustrates the effect of increasing vaccination coverage in reducing the susceptible pool and thus mitigating disease transmission.

The right panel of the figure, on the other hand, fixes the vaccination rate at $\tau = 0.85$ and examines the effect of vaccine efficacy (ϵ). The findings show that the epidemic curve is dramatically suppressed as the efficacy of the vaccine increases. Although a highly effective vaccination ($\epsilon = 1.0$) reduces the peak and stops sustained transmission, poor-efficacy vaccines ($\epsilon = 0.2$) cause infections to rise to a peak that exceeds 2 million people. It is evident from the steep drop in equivalent control reproduction numbers from $\mathcal{R}_t = 3.0382$ to $\mathcal{R}_t = 0.0727$ that vaccine-induced immunity significantly lowers the risk of subsequent transmission. These factors highlight how important vaccination quality is in containing an outbreak, not just the number of people immunized. Even when baseline transmission is high, a vaccination that offers robust protection can significantly change the course of the epidemic.

Collectively, our results highlight the synergistic advantages of maximizing vaccine efficacy and vaccination rate and offer strong quantitative support for the strategic use of vaccination in epidemic suppression. These effects are captured by the reproduction number $\mathcal{R}_t$, which is determined using the next-generation matrix technique and depends on both τ and ϵ. In particular, the number of control reproduction (2) shows that a high vaccination rate, together with a higher efficacy ($\epsilon \to 1$), significantly lowers the force of infection.

However, increasing vaccine coverage might not be enough to push $\mathcal{R}_t$ below unity if efficacy is low. These observations are extremely pertinent to practical vaccination plans, especially in situations where vaccine supplies are scarce or if novel vaccine variations jeopardize the efficacy of currently available vaccines. The model illustrates that managing an outbreak requires both widespread vaccination and maintaining the vaccine's high immunogenic quality over time.

Fig. 5 examines how behavioral interventions, specifically condom efficacy (α_2) and condom use (α_1), affect the control reproduction number $\mathcal{R}_t$ and the course of mpox infections. The transmission rate $\beta = 0.67$ and the efficacy of the condom $\alpha_2 = 0.795$ are stable in the left panel, while the condom use rate α_1 fluctuates between 0.2 and 1.0. The trends make it

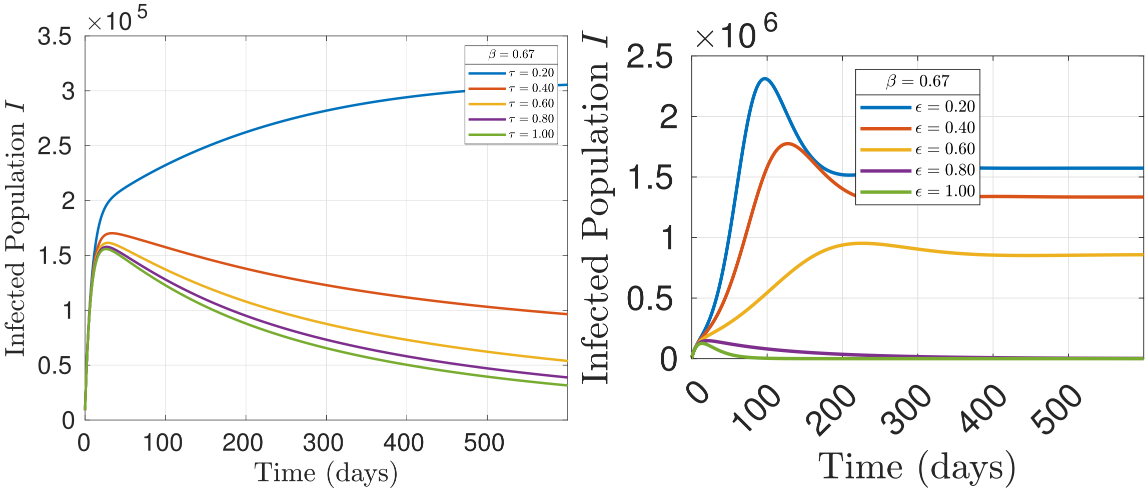

Fig. 4. Effect of vaccination rate and vaccine efficacy on the disease dynamics. The dynamics of the infected population over time for different vaccination rates (left panel) and vaccine efficacy (right panel). In the left panel, for the vaccination rates $\tau = 0.2$, 0.4, 0.6, 0.8, 1.0, the corresponding control reproduction numbers are $\mathcal{R}_t = 1.1281, 1.0220, 0.9847, 0.9657$, and 0.9542, respectively, while in the right panel, $\mathcal{R}_t = 3.0382, 2.2968, 1.5555, 0.8141$, and 0.0727 for $\epsilon = 0.2$, 0.4, 0.6, 0.8, 1.0. Other parameter values are given in Tables 3 and 5

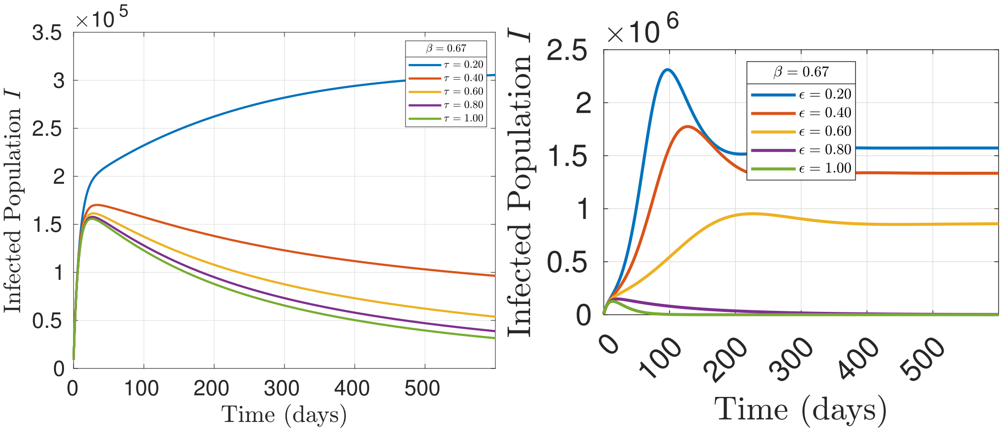

Fig. 5. Effect of condom usage and condom efficacy on the disease dynamics. The dynamics of the infected population over time for different condom usage (left panel) and condom efficacy (right panel). In the left panel, for the condom usage rates $\alpha_1 = 0.2$, 0.4, 0.6, 0.8, 1.0, the corresponding control reproduction numbers are $\mathcal{R}_t = 1.2602, 1.0219, 0.7837, 0.5454$ and 0.3072, respectively, while in the right panel, $\mathcal{R}_t = 1.3635, 1.2287, 1.0938, 0.9590$ and 0.8241 for $\alpha_2 = 0.2$, 0.4, 0.6, 0.8, 1.0. Other parameter values are given in Tables 3 and 5

abundantly clear that using condoms more frequently results in a significant decrease in both the peak and the total number of infections over time. In particular, infections increase and the maximum number of infected people exceeds $\times$ 10^5 when only 20 % of the population uses condoms ($\alpha_1 = 0.2$).

The epidemic is repressed more and more as usage increases, leading to a severely flattened curve for $\alpha_1 = 1.0$, where the maximum number of infections stays below 1×10^5. The matching values of the control reproduction number $\mathcal{R}_t$ indicate a change from uncontrolled to controlled epidemic dynamics, with a decreasing trend from 1.2602 in low usage to 0.3072 at 100 % condom adherence. This emphasizes how important it is to use condoms widely and consistently to prevent the spread of mpox, particularly in groups with high-risk sexual networks.

By separating the impact of condom efficacy (α_2) under a fixed high usage rate of $\alpha_1 = 0.85$, the right panel enhances this analysis. Here, the infection curves once again demonstrate a surprising sensitivity to the effectiveness of condoms in preventing transmission. Infection rates stay high and persistent over time at low efficacy levels (e.g., $\alpha_2 = 0.2$), with maximums near 7×10^5, which closely resembles the situation without intervention. The number of infections is greatly reduced as the efficacy increases, and the epidemic peak not only is lowered, but also occurs sooner.

Interestingly, the infection curve flattens significantly when condom efficiency hits 100 %, indicating that transmission is almost completely eliminated. The control reproduction number, which decreases from 1.3635 at $\alpha_2 = 0.2$ to 0.8241 at maximum efficacy, reflects this pattern. These findings firmly imply that, even in cases where condom use is widespread, the effectiveness of these interventions is just as important in stopping the transmission of diseases. Therefore, in addition to promoting condom use, public health initiatives must ensure the supply of high quality items.

All things considered, Fig. 5 highlights how crucial behavioral tactics are to mpox containment, especially in sexually active groups where human-to-human transmission is common. The results show that condom use and efficacy work together, with neither element being adequate on its own if the other is deficient. High use of condoms that don't work well provides just slight advantages, whereas very effective condoms will not make much of an impression if they aren't extensively used.

These findings highlight the need for focused awareness programs that encourage the distribution of medically approved and effective products, as well as the regular and proper use of condoms. Crucially, the two panels of the figure restate a fundamental epidemiological finding: that in order to stop the epidemic, the control reproduction number $\mathcal{R}_t$ must be less than unity. This data supports condom-based interventions as a useful addition to vaccination and case isolation tactics in the context of mpox, which has demonstrated patterns of sexual transmission during recent outbreaks. The model-driven insights presented here offer quantitative support for laws intended to strengthen the infrastructure for sexual health in order to stop the spread of mpox.

Fig. 6 analyzes the influence of altering the transmission rate β on the trajectory of mpox infections over time, assuming a fixed and reasonably high vaccination rate ($\tau = 0.85$). The increased transmissibility and the intensity of the epidemic are clearly and strongly correlated, as the figure shows. Small and transient outbreaks that rapidly fade away are characteristic of infection curves for lower values of β (e.g., 0.20 and 0.40), which represent low control reproduction numbers $\mathcal{R}_t$ of 0.2873 and 0.5745, respectively, well below the crucial threshold of 1. As a result, the disease quickly fades away because it cannot survive in the population.

However, the system experiences a qualitative change as β increases to 0.60 and above: not only do infection levels increase dramatically, but the epidemic also lasts longer, peaking later and supporting larger infected populations for longer

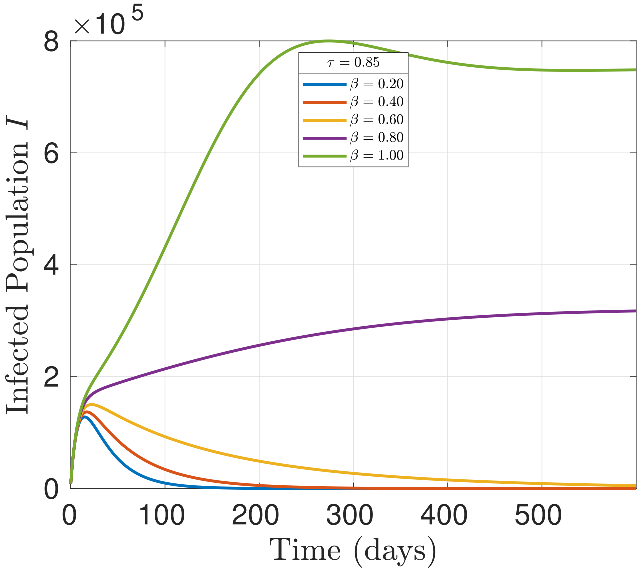

Fig. 6. Effect of change in transmission rate on the control reproduction number. The dynamics of the infected population over time for different transmission rates with constant vaccination rate $\tau = 0.85$ shows that the corresponding control reproduction numbers are $\mathcal{R}_t = 0.2873, 0.5745, 0.8618, 1.1491$ and 1.4363. Other parameter values are given in Tables 3 and 5

periods of time. As an illustration, when $\beta = 1.00$, $\mathcal{R}_t$ increases to 1.4363, resulting in a widespread outbreak that peaks at around 800,000 infected people.

This result indicates that even with a successful vaccination program, high transmission rates can still generate a significant epidemic, underscoring the non-linear and compounding consequences of transmission dynamics on disease control. The findings emphasize the need to reduce β through public health initiatives, including contact tracking, behavioral changes, and isolation because vaccination alone may not be enough in high-transmission environments. The results highlight how crucial it is to combine vaccination plans with measures to reduce transmission in order to successfully contain mpox epidemics, especially in situations where there is still a significant risk of disease spread. Performing and maintaining $\mathcal{R}_t < 1$, the threshold below which epidemics decline, requires this multifaceted approach.

The impact of the rate of condom usage (α_1, left panel) and condom efficacy (α_2, right panel) on the control reproduction number $\mathcal{R}_t$ for mpox, under a fixed high vaccination rate ($\tau = 0.85$) and vaccine efficacy ($\epsilon = 0.76$), is completely contoured in Fig. 7. According to the contour plots, α_1 and α_2 have a clear inverse connection with $\mathcal{R}_t$: at all transmission rate levels β, the control reproduction number significantly decreases when either parameter increases.

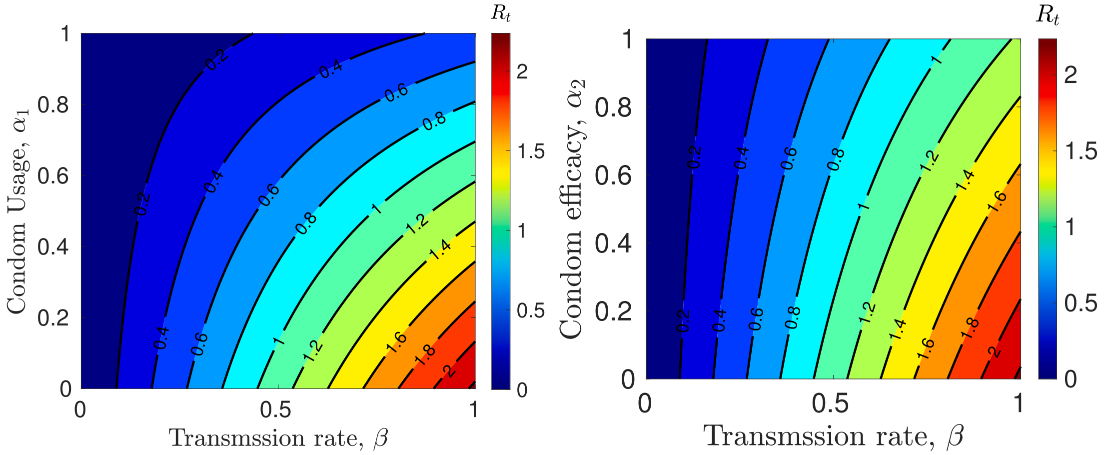

Fig. 7. Effect of condom usage and condom efficacy on the control reproduction number. Vaccination rate, $\tau = 0.85$ and vaccine efficacy, $\epsilon = 0.76$. Other parameter values are given in Tables 3 and 5

In the left panel, for example, even moderate values of β (e.g., $\beta = 0.5$) produce $\mathcal{R}_t$ values well over 1.2, showing sustained transmission, even at low condom usage (e.g., $\alpha_1 = 0.2$). However, $\mathcal{R}_t$ falls below 1 for almost all β values as α_1 rises toward full compliance ($\alpha_1 = 1.0$), highlighting the powerful mitigating effect of universal condom use in decreasing disease spread. This implies that public health initiatives focused on condom uptake can dramatically reduce the dynamics of mpox transmission, particularly in sexually active groups.

Similarly, the right panel highlights how crucial condom effectiveness is in determining the course of epidemics. Even with substantial use, the impact on $\mathcal{R}_t$ is restricted when condom efficacy is poor ($\alpha_2 = 0.2$), and the disease may still spread. However, $\mathcal{R}_t$ drops precipitously as α_2 rises near 1.0, indicating that efficient barrier protection greatly reduces the probability of transmission per contact. This suggests that condom quality and application, in addition to condom prevalence, are critical factors in reducing mpox transmission.

These findings collectively demonstrate the synergistic advantages of integrating immunological tactics such as immunization with behavioral treatments like quality control and condom use. Controlling mpox outbreaks can be greatly aided by such coordinated public health initiatives, especially in situations where close or intimate contact is the primary mode of transmission.

Taking into account fixed moderate values for the condom usage rate ($\alpha_1 = 0.45$) and the condom efficacy ($\alpha_2 = 0.79$), Fig. 8 shows the effects of the vaccination rate (τ, left panel) and vaccine efficacy (ϵ, right panel) on the control reproduction number $\mathcal{R}_t$ for mpox. The contour lines on the left panel show that even with minimal vaccination, the disease is effectively contained at low transmission rates ($\beta < 0.4$), as the control reproduction number stays below 1 for all values of τ.

However, $\mathcal{R}_t$ becomes extremely sensitive to changes in τ as β increases by 0.5. For example, if τ is less than 0.2 at $\beta = 0.8$, $\mathcal{R}_t$ surpasses 2; however, when vaccination rates increase, this value dramatically reduces and falls below the epidemic threshold ($\mathcal{R}_t = 1$) when $\tau \geq 0.8$. This pattern highlights the necessity of widespread vaccination efforts in outbreak response tactics and shows that increasing vaccine coverage dramatically reduces the possibility of transmission, particularly when dealing with highly transmissible strains of mpox.

Fig. 8's right panel shows a similar pattern in terms of vaccine effectiveness. $\mathcal{R}_t$ rapidly increases with increases in β when ϵ is low ($\epsilon < 0.2$), surpassing crucial thresholds and suggesting a significant risk of widespread infection. However, the contours change dramatically as the efficacy of the vaccine increases ($\epsilon > 0.6$), with $\mathcal{R}_t$ staying below 1 over a larger range of values of β. Even with increased transmission rates, the disease can be controlled with full efficacy ($\epsilon = 1$), as long as additional therapies are implemented.

This highlights the importance of both the quality (effectiveness) and quantity (coverage) of the vaccine in mpox control. When combined, the two panels indicate that improving the efficacy and coverage of vaccination programs is crucial to reducing $\mathcal{R}_t$ and stopping the spread of mpox, particularly in situations where condom-related treatments offer only limited protection.

5. Conclusion

In this work, we calibrated and fitted a simple SIR model to the 2022 daily reported mpox data using the current demographic data for the BURCK countries in Africa. The mpox data contains a 7-day rolling average of mpox cases from the date when case reporting started in those countries to date. We extracted cases reported from the beginning to the time just before the implementation of control intervention strategies. The reason for this is that it will give us the opportunity to

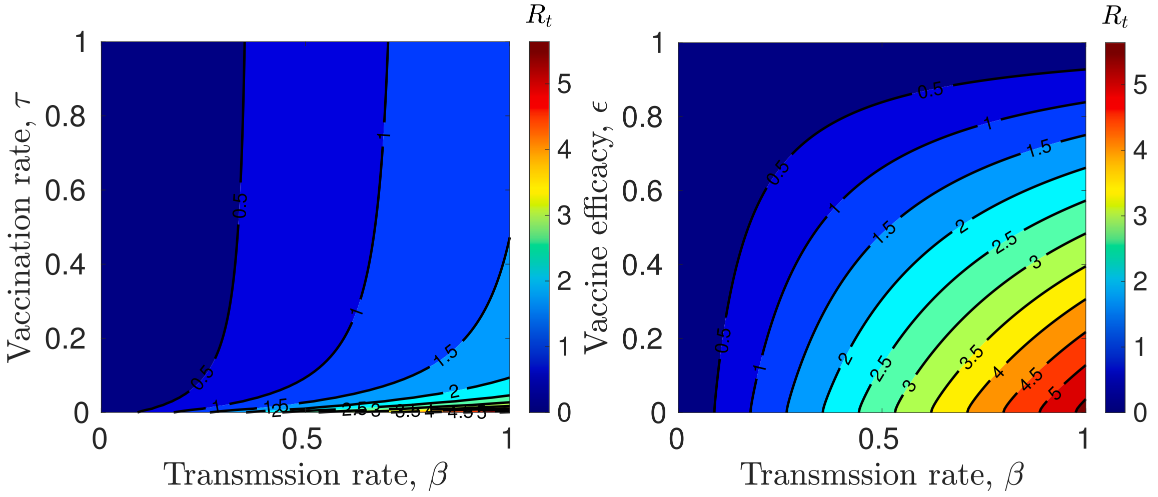

Fig. 8. Effect of vaccination rate and vaccine efficacy on the control reproduction number. Condom usage rate, $\alpha_1 = 0.45$ and condom efficacy rate, $\alpha_2 = 0.79$. Other parameter values are given in Tables 3 and 5

understand the initial spread of the new mpox strain and also estimate its basic reproduction number $\mathcal{R}_0$. We employed a very sophisticated fitting approach known as Bayesian Statistical Inference, which allows us to incorporate our prior knowledge in the fitting and calibration process. After fitting the model to data, our model was able to estimate the relevant parameters and the basic reproduction number of mpox for each countries. Other fixed parameters are assumed, computed or taken from relevant literature. In addition, we calculated the final size of the outbreak. Understanding the final size is important to public health professionals for several reasons: it provides an indication of the overall effect of the epidemic on the population, it provides a rough estimate of the population that is expected to get the disease at the end of the outbreak.

The simple SIR model was further modified to S_uS_vEIR in order to investigate the effects of pharmaceutical (such as vaccination) and non-pharmaceutical interventions (such as condoms). The numerical simulations showed that considerable decreases in the maximum number of infections and $\mathcal{R}_t$ are caused by increases in vaccination rate, vaccine efficacy, condom usage and condom efficacy. The findings emphasize how crucial multifaceted intervention tactics are to successfully stop the spread of disease.

This study emphasizes the importance of vaccination as the most effective mpox control measure. When paired with high vaccination rates, even low vaccine effectiveness can successfully manage disease by bringing the reproduction number below unity. The results also show that, although not as effective as vaccination, behavioral interventions such as increased condom use and efficacy—offer a significant added benefit. These results are essential for directing public health initiatives, particularly in environments with limited resources where optimizing the effectiveness of existing interventions is crucial.

From a public health perspective, these results highlight the need for governments and interested parties to prioritize vaccination campaigns and awareness initiatives as the top priority. Important tactics include promoting behavioral changes, expanding access to vaccination, and educating the public about safe procedures. These treatments are especially crucial in areas where the public health infrastructure may be strained and where mpox is reemerging or emerging. To guarantee maximum efficacy, these tactics must also be modified to fit the regional epidemiological, social, and cultural circumstances.

The applicability of this study is especially noteworthy on the African continent, especially in countries such as Burundi, Kenya, Uganda, Rwanda, and Congo. These countries are still at risk due to their porous borders, inadequate healthcare systems, and low vaccination rates, which have caused an endless increase in mpox. Policymakers in these areas can use the model's insights to assess the possible effects of expanding vaccination and behavioral interventions. Proactively implementing such safeguards can help prevent epidemics from worsening and lessen the impact on already overburdened healthcare systems.

In summary, the mathematical modeling and simulation results provide a solid basis for managing and predicting mpox transmission. The report offers evidence-based recommendations for creating all-encompassing public health responses by measuring the impacts of crucial control measures. In addition to improving our scientific knowledge on mpox transmission, these revelations aid in the creation of affordable, region-specific methods for disease prevention and control throughout Africa and beyond.

Despite the insightful results this study offered, a number of limitations must be noted. First, the model makes the assumption that all people use condoms, are vaccinated, and are mixed into the population. This may not be an accurate representation of the diverse character of populations in the real world. Transmission dynamics can be affected by behavioral, cultural, and socioeconomic variations, particularly in the varied environments of East Africa. Second, the model depends on theoretical presumptions and parameter estimations from earlier research, which may not accurately reflect the changing epidemiological features of mpox in certain areas. Moreover, issues such as decreasing immunity, breakthrough infections, co-infections with other diseases, and changes in viral transmissibility due to mutations were not explicitly incorporated in the model. Another limitation of our study is that the vaccination thresholds derived from our simple SEIR framework cannot be generalized across all settings. The design and implementation of immunization strategies in practice must account for local epidemic conditions, the availability of public health resources, and the acceptability of interventions within the target population. In addition, model parameters may need to be adjusted to reflect these context-specific factors. Therefore, our findings should be viewed as providing baseline insights rather than prescriptive recommendations for all contexts.

To fill in these gaps, future research should take spatial heterogeneity into account by grouping people according to risk categories, locations, or migration patterns to more accurately represent real transmission networks. More precise prediction and uncertainty quantification would also be possible with the inclusion of stochastic components and real-time surveillance data. Future models should also assess the logistical and financial viability of initiatives, such as the relative costs of different vaccination and condom distribution plans. The usefulness of the model for guiding public health decision-making would be further increased by incorporating human behavior dynamics, media influence, and the ability to react to health systems. The predictive ability of the model and the relevance of the policy would ultimately be greatly increased by working together with local health authorities to calibrate it using data unique to the area.

CRediT authorship contribution statement

Musa Rabiu: Writing – review & editing, Writing – original draft, Visualization, Validation, Software, Investigation, Formal analysis, Data curation, Conceptualization. **Bosede Fagbemigun:** Writing – review & editing, Writing – original draft,

Visualization, Validation, Software, Methodology, Investigation, Formal analysis, Conceptualization. **Sunday Fadugba:** Writing – review & editing, Writing – original draft, Visualization, Validation, Software, Methodology, Formal analysis, Conceptualization. **Michael Shatalov:** Writing – review & editing, Writing – original draft, Visualization, Validation, Supervision, Project administration, Investigation, Funding acquisition, Formal analysis, Conceptualization. **Kekana Malesela:** Writing – review & editing, Writing – original draft, Visualization, Validation, Supervision, Resources, Project administration, Methodology, Investigation, Formal analysis, Data curation, Conceptualization. **Adejimi Adeniji:** Writing – review & editing, Writing – original draft, Visualization, Validation, Supervision, Software, Project administration, Methodology, Investigation, Formal analysis, Data curation, Conceptualization.

Data availability statement

The authors should be contacted for the data.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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