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MATHEMATICAL MODELING OF HUMAN–RODENT MONKEYPOX INFECTIOUS DISEASE USING A HIERARCHICAL APPROACH

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Abstract. Monkeypox is a resurging zoonotic disease in Nigeria, posing increasing public health concerns. To capture its transmission dynamics, this study develops a hierarchical human–rodent model. Biological validity is ensured by proving the positivity of solutions and identifying an invariant region. Epidemic threshold analysis is conducted through the derivation of the disease-free equilibrium (DFE). The model combines theoretical analysis and numerical simulation in a hierarchical framework. Using the adaptive fourth–fifth order Runge–Kutta method (RK45), the nonlinear system is solved to investigate epidemic trajectories in Nigeria under realistic demographic and epidemiological settings. Results highlight the sensitivity of epidemic waves to transmission parameters and

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emphasize the role of rodent reservoirs in sustaining outbreaks. The study provides computational and analytical insights that may inform public health interventions to control monkeypox spread.

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2020 AMS Subject Classification: 92D30, 34A34, 65L06.

1. INTRODUCTION

Monkeypox is a rare, contagious viral disease caused by the monkeypox virus, a member of the *Orthopoxvirus* family, the same family that includes smallpox. The disease was first identified in Denmark in 1958 when a smallpox-like illness appeared in laboratory monkey colonies, hence the name "monkeypox." Subsequent studies, however, revealed that monkeys were not the natural reservoir of the virus; instead, African rodents are believed to be the primary hosts [1].

The first human case was reported in a 9-month-old child in the Democratic Republic of the Congo in 1970. Since then, cases have been recorded across Central and West Africa, particularly in countries such as Nigeria, Cameroon, and the Central African Republic [2]. Between 1970 and 1990, hundreds of human cases were documented, mainly in rural areas near tropical forests. Researchers observed that vaccination against smallpox provided cross-immunity, which significantly reduced monkeypox infections. However, following the cessation of global smallpox vaccination campaigns after 1980, the incidence of monkeypox gradually increased. In 2003, the first cases outside Africa were recorded in the United States when the virus spread from rodents imported from Ghana to pet prairie dogs, resulting in more than 70 human infections.

A notable resurgence occurred between 2017 and 2019, when Nigeria experienced its largest outbreak in decades, with over 200 confirmed cases. This raised international concern due to the rising number of infections and the emergence of more transmissible strains. In May 2022, hundreds of cases were reported in Europe and North America without direct links to travel in Africa. In July 2022, the World Health Organization (WHO) declared monkeypox a public health emergency of international concern. By 2023, the number of cases began to decline,

aided by vaccination campaigns and increased public awareness. More detailed reviews of monkeypox can be found in [3, 4, 5].

Recent advances in applied mathematics and computational analysis have demonstrated the growing impact of fractional calculus, fuzzy modeling, and numerical algorithms in describing nonlinear and real-world phenomena. Several studies have focused on developing new algorithms for solving linear, nonlinear, and fractional integro-differential equations, highlighting their efficiency and convergence properties in complex systems [6, 7, 8, 9]. In parallel, modern analytical and numerical investigations have explored inequalities in matrix theory [10], nanomaterial performance optimization [11], and the construction of reliable approximate schemes for fuzzy and stochastic models [9]. Within epidemiological and biological modeling, fractional-order and reaction–diffusion frameworks have been successfully employed to capture dynamics that are not adequately described by classical integer-order systems [12, 13]. In particular, fractional-order and discrete-time epidemic models have provided deeper insights into the finite-time stability, synchronization, and numerical behavior of infectious disease propagation [14, 15, 16]. These recent contributions form a strong mathematical foundation and motivate the present study, which aims to analyze the fractional-order monkeypox transmission dynamics through robust analytical and numerical techniques.

In this study, we develop and analyze a hierarchical epidemiological model for the transmission of monkeypox between humans and rodents in Nigeria. The model's biological feasibility is established by proving the positivity of solutions and identifying an invariant region. To understand the system's threshold behavior, stability analysis and the disease-free equilibrium (DFE) are examined. Furthermore, numerical simulations are conducted under realistic demographic and epidemiological assumptions using the adaptive Runge–Kutta method of order 4(5) (RK45). The results highlight the role of rodent reservoirs in sustaining the disease and demonstrate how variations in transmission parameters can drive epidemic waves. This theoretical and computational framework provides insights that may guide effective monkeypox control strategies.

2. Model Development

Monkeypox is a viral zoonotic disease whose progression in humans must be described by accounting for its modes of transmission. Previous studies have identified three main pathways through which the virus spreads [17, 18]. First, **animal-to-animal transmission** occurs when an infected animal comes into contact with a susceptible one through bites, scratches, direct skin contact, or exposure to body fluids, respiratory droplets, and contaminated environments. Second, **animal-to-human transmission** can arise from contact with the blood, body fluids, or infected skin of animals carrying the virus (such as rodents and monkeys). Third, **human-to-human transmission** is possible through respiratory droplets during close contact, direct contact with infected skin or mucous membranes, or indirect contact with contaminated objects (e.g., clothing, bedding, or surfaces). Recently, monkeypox has disseminated across numerous countries, showing pandemic-like characteristics. Several studies have focused on disease dynamics in human populations [19, 20, 21, 22], while others have highlighted the role of animals as reservoirs for human infection [23, 24, 25]. The overall transmission pathways of monkeypox are summarized in Figure 1.

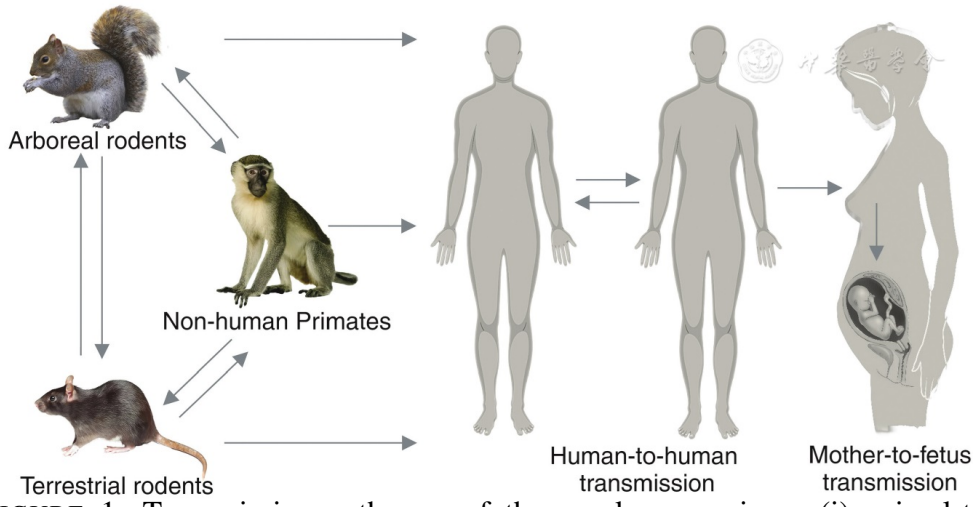

FIGURE 1. Transmission pathways of the monkeypox virus: (i) animal-to-animal, (ii) animal-to-human, and (iii) human-to-human.

We propose a compartmental model to capture the dynamics of monkeypox transmission in both human and rodent populations, as illustrated in Figure 2. In addition, the model incorporates the natural loss of immunity following recovery and the incubation period characteristic of the disease [2], by explicitly including the exposed compartment E.

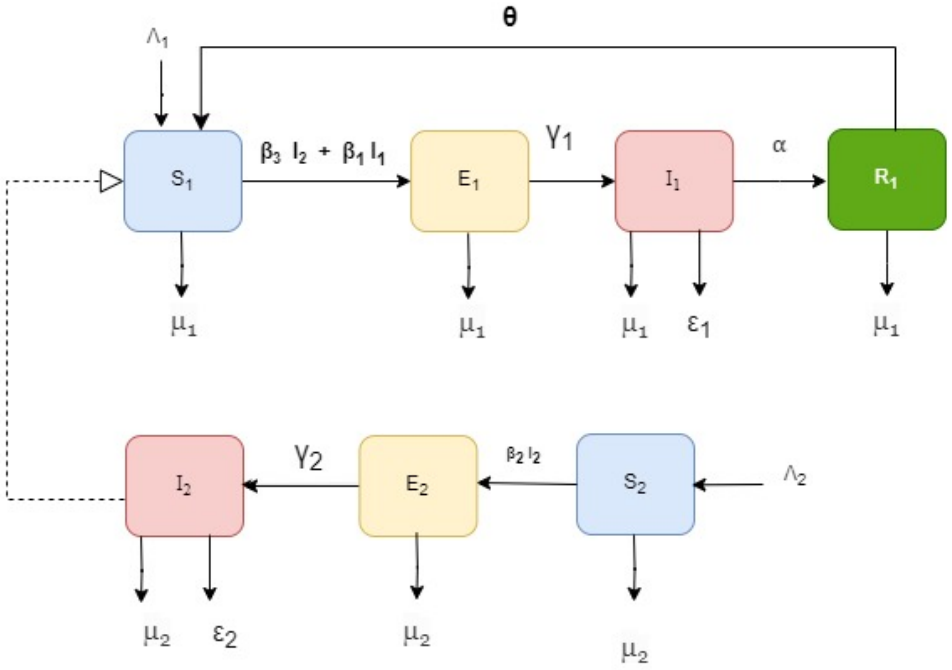

FIGURE 2. Compartmental diagram of the proposed monkeypox model, including human and rodent populations.

The proposed epidemiological model for monkeypox is given by the following system of differential equations:

$$S_1'(t) = -\frac{S_1(t)\big(\beta_1 I_1(t) + \beta_3 I_2(t)\big)}{N_1} + \Lambda_1 + \theta R_1(t) - \mu_1 S_1(t),$$

$$E_1'(t) = \frac{S_1(t)\big(\beta_1 I_1(t) + \beta_3 I_2(t)\big)}{N_1} - (\gamma_1 + \mu_1) E_1(t),$$

$$I_1'(t) = \gamma_1 E_1(t) - (\alpha + \varepsilon_1 + \mu_1) I_1(t),$$

$$R_1'(t) = \alpha I_1(t) - (\theta + \mu_1) R_1(t),$$

$$S_2'(t) = -\frac{\beta_2 I_2(t) S_2(t)}{N_2} + \Lambda_2 - \mu_2 S_2(t),$$

$$E_2'(t) = \frac{\beta_2 I_2(t) S_2(t)}{N_2} - (\gamma_2 + \mu_2) E_2(t),$$

$$I_2'(t) = \gamma_2 E_2(t) - (\varepsilon_2 + \mu_2) I_2(t). \tag{1}$$

It is important to identified all parameters and definitions of the above model, which are given in Table 1.

TABLE 1. Parameters and definitions of the epidemiological model for monkeypox (system (1)).

Symbol	**Definition**	**Value**	**Reference**
S_1	Susceptible human population	–	Region dependent
E_1	Exposed human population	–	Region dependent
I_1	Infected human population	–	Region dependent
R_1	Recovered human population	–	Region dependent
S_2	Susceptible rodent population	–	Region dependent
E_2	Exposed rodent population	–	Region dependent
I_2	Infected rodent population	–	Region dependent
Λ_1	Recruitment rate of humans	–	Region dependent
μ_1	Human natural death rate	–	Region dependent
β_1	Human-to-human transmission rate	$[0,1]$	Assumed
β_3	Transmission rate due to human–rodent contact	$[0,1]$	Region dependent
γ_1	Progression rate ($E_1 \to I_1$)	$\left[\frac{1}{17}, \frac{1}{7}\right]$	[2]
α	Recovery rate ($I_1 \to R_1$)	$\left[\frac{1}{28}, \frac{1}{14}\right]$	[26]
θ	Loss of infection-acquired immunity	–	–
ε_1	Disease-induced mortality in humans	–	–
Λ_2	Recruitment rate of rodents	–	Assumed
μ_2	Rodent natural death rate	–	Assumed
ε_2	Disease-induced mortality in rodents	–	–
β_2	Rodent-to-rodent transmission rate	–	–
γ_2	Progression rate ($E_2 \to I_2$)	–	–

Here, N_1 denotes the total human population, expressed as

$$N_1(t) = S_1(t) + E_1(t) + I_1(t) + R_1(t). \tag{2}$$

Similarly, N_2 denotes the total rodent population, given by

$$N_2(t) = S_2(t) + E_2(t) + I_2(t). \tag{3}$$

3. Mathematical Analysis of the Monkeypox Model

The system given in (1) is analyzed to establish the biological feasibility of the model, investigate its fundamental properties, and derive the basic reproduction number R_0.

3.1. Invariant Region and Positivity of the Solutions. For an epidemiological model to be biologically meaningful, its solutions must remain non-negative and bounded for all time. In this subsection, we show that the system (1) admits an invariant region in which all state variables are non-negative, thereby ensuring the positivity and feasibility of the model.

Theorem 3.1. *The system* (1) *with non-negative parameters admits a biologically feasible region* $\Omega \subset \mathbb{R}^7_+$*, defined as*

$$\Omega = \Big\{ (S_1(t), E_1(t), I_1(t), R_1(t), S_2(t), E_2(t), I_2(t)) \in \mathbb{R}^7_+ : \\ (S_1(t), E_1(t), I_1(t), R_1(t), S_2(t), E_2(t), I_2(t)) \geq 0,\ \forall t \in [0, \infty) \Big\}.$$

Proof. Let $\Omega = \Omega_1 \times \Omega_2$, where Ω_1 denotes the feasible region of the human population

$$(S_1(t), E_1(t), I_1(t), R_1(t)) \in \mathbb{R}^4_+$$

and Ω_2 denotes the feasible region of the rodent population $(S_2(t), E_2(t), I_2(t) \in \mathbb{R}^3_+)$. For the human population, from equation (2), adding the first four equations of system (1) gives

$$N_1'(t) \leq \Lambda_1 - \mu_1 N_1(t). \tag{4}$$

Multiplying by the integrating factor $e^{\mu_1 t}$ and solving (4) yields

$$0 \leq N_1(t) \leq N_1(0) e^{-\mu_1 t} + \frac{\Lambda_1}{\mu_1}\big(1 - e^{-\mu_1 t}\big).$$

For the rodent population, from equation (3), adding the last three equations of system (1) gives

$$N_2'(t) \leq \Lambda_2 - \mu_2 N_2(t). \tag{5}$$

Multiplying by the integrating factor $e^{\mu_2 t}$ and solving (5) yields

$$0 \leq N_2(t) \leq N_2(0)e^{-\mu_2 t} + \frac{\Lambda_2}{\mu_2}\left(1 - e^{-\mu_2 t}\right).$$

Thus, both regions Ω_1 and Ω_2 are positively invariant over time, and hence the whole region Ω is positively invariant. □

3.2. Disease-Free Equilibrium (DFE) and Basic Reproduction Number (R_0). The disease-free equilibrium (DFE) corresponds to the state in which the system is entirely free of infection, meaning that no individuals are exposed or infected in either the human or rodent populations. For system (1), the DFE is given by

$$DFEP = \left(\frac{\Lambda_1}{\mu_1}, 0, 0, 0, \frac{\Lambda_2}{\mu_2}, 0, 0\right). \tag{6}$$

That is,

$$S_1 = \frac{\Lambda_1}{\mu_1}, \quad E_1 = 0, \quad I_1 = 0, \quad R_1 = 0, \quad S_2 = \frac{\Lambda_2}{\mu_2}, \quad E_2 = 0, \quad I_2 = 0.$$

The basic reproduction number, R_0, is a key epidemiological parameter that quantifies the transmissibility or potential spread of an infectious disease [27]. It provides a threshold criterion: if $R_0 < 1$, the infection will eventually die out, while $R_0 > 1$ indicates the potential for an epidemic outbreak.

To evaluate R_0, we employ the Next Generation Matrix (NGM) approach, a widely used framework in epidemiological modeling. This method was first introduced by Diekmann et al. (1990) [28] and later formalized by Van den Driessche and Watmough (2002) [29]. In applying the NGM to the proposed model, the following assumptions are made:

- The infectious compartments are E_1, I_1, and I_2.
- $\mathscr{Z}$ represents the rate of appearance (gain) of new infections in the infectious compartments.

- $\mathscr{M}$ represents the transfer (loss) of individuals from the infectious compartments due to recovery or disease-induced mortality.

Then we have

$$\frac{dX}{dt} = \begin{bmatrix} \frac{dE_1}{dt} \\ \frac{dI_1}{dt} \\ \frac{dE_2}{dt} \\ \frac{dI_2}{dt} \end{bmatrix}, \quad \mathscr{Z} = \begin{bmatrix} \frac{S_1(t)\left(\beta_1 I_1(t) + \beta_3 I_2(t)\right)}{N_1} \\ 0 \\ \frac{\beta_2 I_2(t) S_2(t)}{N_2} \\ 0 \end{bmatrix}, \quad \mathscr{M} = \begin{bmatrix} (\gamma_1 + \mu_1)E_1(t) \\ -\gamma_1 E_1(t) + (\alpha + \varepsilon_1 + \mu_1)I_1(t) \\ (\gamma_2 + \mu_2)E_2(t) \\ -\gamma_2 E_2(t) + (\varepsilon_2 + \mu_2)I_2(t) \end{bmatrix}.$$

Evaluating the Jacobian matrices of $\mathscr{Z}$ and $\mathscr{M}$ at the disease-free equilibrium point (6) gives

$$Z = \begin{bmatrix} 0 & \beta_1 & 0 & \beta_3 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & \beta_2 & 0 \\ 0 & 0 & 0 & 0 \end{bmatrix}, \qquad M = \begin{bmatrix} \gamma_1 + \mu_1 & 0 & 0 & 0 \\ -\gamma_1 & \alpha + \varepsilon_1 + \mu_1 & 0 & 0 \\ 0 & 0 & \gamma_2 + \mu_2 & 0 \\ 0 & 0 & -\gamma_2 & \varepsilon_2 + \mu_2 \end{bmatrix}.$$

Assuming the next-generation matrix is defined as $F = ZM^{-1}$, the basic reproduction number R_0 is given by the spectral radius of F. This leads to two components corresponding to the human and rodent populations, denoted by R_{01} and R_{02}, respectively:

$$R_0 = \rho(ZM^{-1}), \qquad R_{01} = \frac{\beta_1\left(\gamma_1\gamma_2 + \gamma_1\mu_2\right)}{(\gamma_1 + \mu_1)(\alpha + \varepsilon_1 + \mu_1)(\gamma_2 + \mu_2)}, \qquad R_{02} = \frac{\beta_2}{\gamma_2 + \mu_2}.$$

4. NUMERICAL SIMULATION

To investigate the dynamics of the monkeypox epidemic model defined in system (1), we consider Nigeria as the primary case study. Since the 1970s, monkeypox has been present in Central and West Africa, but Nigeria has experienced the largest and most recent outbreaks (2017–2022). Epidemiological data reported by the Centers for Disease Control and Prevention (CDC) and the World Health Organization (WHO) provide estimates for model parameters. Nigeria also offers a suitable environment for study due to its high level of human–rodent contact (e.g., in food markets, storage areas, and rural communities) and its large population of more than 220 million people, which facilitates the analysis of widespread transmission and regional variations. The parameters may also be adjusted to fit other target regions (e.g., North

Africa or the Gulf states), where transmission could be predominantly human-to-human with fewer rodent reservoirs.

At the national level, applying the model to Nigeria has both advantages and disadvantages. Its main advantages lie in comprehensiveness and its potential to support nationwide recommendations and public health strategies. However, it also requires extensive datasets (such as state-level population, mobility, and environmental variation) and more complex calculations, and it risks over-approximation when data are incomplete. By contrast, applying the model at the city level provides localized data that are easier to collect and approximate, produces clearer results, and reduces computational burden. Although city-level modeling is suitable for calibration, it does not capture intercity movements or imported cases from other regions.

To balance these considerations, we adopt a **hierarchical approach** [30, 31, 32], beginning with a city-level model and then extending it to the national level. This strategy enables phased calibration and validation of assumptions while maintaining accuracy and efficiency. We select Lagos city as the most suitable location for simulation because it has a large and dense population (approximately 17 million), a documented history of epidemics and surveillance, and serves as both an economic center and an international hub with a major port and airport. To investigate the dynamics of the human–rodent epidemiological model of monkeypox in Lagos, we consider three different scenarios that reflect varying levels of health interventions:

- **Scenario A:** Low transmission with weak control measures.
- **Scenario B:** Moderate transmission (reference case).
- **Scenario C:** High transmission with weak or absent interventions.

Since the proposed epidemiological model is formulated as a system of nonlinear ordinary differential equations (1) without a closed-form analytical solution, we employ the adaptive Runge–Kutta method of order 4(5) (RK45) [33, 34, 35]. This method is widely applied in epidemiological modeling because it combines high accuracy with computational efficiency, while its adaptive step-size control ensures stable integration over long simulation horizons (365 days in our case).

Table 2 summarizes the numerical values of the parameters used for the simulation of Scenario A in Lagos city. This scenario corresponds to a low-transmission setting, reflecting precautionary measures and weak rodent–human interaction. The table includes demographic parameters for both humans and rodents, epidemiological rates such as transmission, recovery, and disease-induced mortality, as well as the initial population sizes for each compartment. These values serve as the baseline for the simulation, enabling us to analyze the disease dynamics under conditions of limited spread and modest control measures. Figure 3 illustrates the temporal dynamics of the human–rodent monkeypox model in Lagos under Scenario A, which represents a low-transmission setting with precautionary measures in place. For the human population (left panel), the number of susceptible individuals S_1 decreases gradually over time, while the exposed E_1 and infected I_1 classes remain relatively small but exhibit noticeable peaks around the mid-simulation period. The recovered population R_1 increases steadily, eventually comprising a significant portion of the total population by the end of the year. For the rodent population (right panel), the susceptible class S_2 declines sharply in the early phase of the outbreak, accompanied by rapid growth in both exposed E_2 and infected I_2 individuals. After reaching their peaks, the exposed and infected rodent populations decrease gradually due to recovery and mortality. These dynamics highlight the critical role of rodents as reservoirs of infection, initiating strong early outbreaks that subsequently influence the epidemic trajectory in the human population.

TABLE 2. Numerical values of parameters under Scenario A for the Lagos city case

Parameter	**Value**	**Note**
N_1	1,000,000	Human population (studied package)
μ_1	5.0735667×10^{-5}	Natural death rate (per day)
Λ_1	50.735667	$\simeq \mu_1 \times N_1$
β_1	0.1	Low human transmission (precautionary measures)
β_3	0.005	Weak rodent–human transmission
γ_1	$\frac{1}{12}$	Transition $E_1 \to I_1$
α	$\frac{1}{21}$	Recovery $I_1 \to R_1$
θ	0.0	Neglected immunity
ε_1	0.0005	Low disease-induced human mortality
N_2	20,000	Estimated rodent population
μ_2	0.0013698630	Natural death rate (per day)
Λ_2	27.397260	$\simeq \mu_1 \times N_2$
β_2	0.2	Low–moderate rodent–rodent transmission
γ_2	$\frac{1}{8}$	Transition $E_2 \to I_2$
ε_2	0.005	Rodent disease-induced mortality
Initial conditions:		
Human	$S_1 = 999{,}990$, $E_1 = I_1 = 5$, $R_1 = 0$	Small imported focus
Rodent	$S_2 = 19{,}990$, $E_2 = I_2 = 5$	Small hotspot
Simulation time	365 days	One year of monitoring

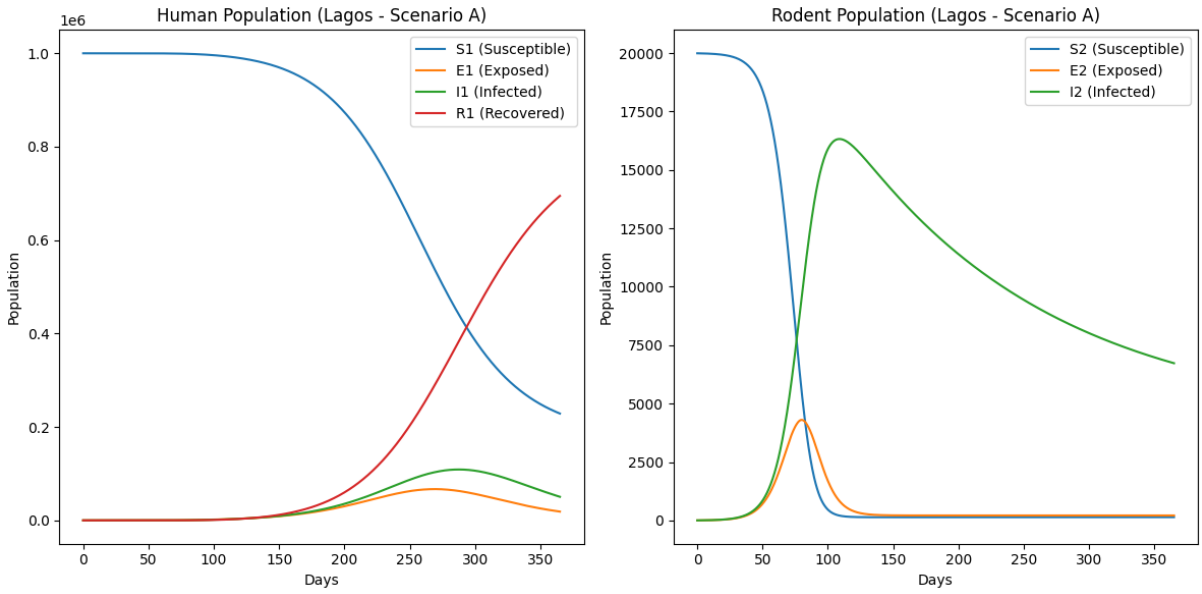

FIGURE 3. Dynamics of the human–rodent monkeypox model in Lagos under Scenario A

Table 3 presents the numerical values of the parameters used in Scenario B for the Lagos city case. This scenario corresponds to a moderate level of transmission, where human-to-human spread ($\beta_1 = 0.3$) is higher than in Scenario A, and rodent-to-human spillover ($\beta_3 = 0.02$) is clearly present though limited. The rodent population is assumed to transmit more efficiently among themselves ($\beta_2 = 0.5$), and both human and rodent mortality rates due to disease are slightly elevated compared to Scenario A. Initial conditions reflect a larger outbreak, with 50 exposed and 50 infected individuals in both the human and rodent populations. These parameter values allow us to investigate epidemic dynamics under conditions of more noticeable transmission while still maintaining partial control interventions. Figure 4 illustrates the epidemic dynamics of the human–rodent monkeypox model in Lagos under Scenario B. In the human population (left panel), the susceptible class S_1 declines steadily, while the exposed E_1 and infected I_1 groups rise to higher peaks than in Scenario A, reflecting the larger initial outbreak and stronger transmission rates. The recovered population R_1 increases more sharply, indicating wider spread but also significant recovery by the end of the simulation. In the rodent population (right panel), the susceptible class S_2 drops rapidly, while exposed E_2 and infected I_2 individuals grow quickly to larger peaks before gradually declining. Compared with Scenario A, these

results show more intense epidemic waves in both humans and rodents, highlighting the impact of stronger transmission parameters and a larger initial seeding of cases.

TABLE 3. Numerical values of parameters under Scenario B for the Lagos city case

Parameter	**Numeric value**	**Note**
N_1	1,000,000	–
μ_1	5.0735667×10^{-5}	–
Λ_1	50.735667	–
β_1	0.3	Moderate human transmission
β_3	0.02	Clear but limited rodent–human transmission
γ_1	$\frac{1}{12}$	–
α	$\frac{1}{21}$	–
θ	0.0	–
ε_1	0.001	Low disease mortality
N_2	20,000	–
μ_2	0.0013698630	–
Λ_2	27.397260	–
β_2	0.5	Medium–high rodent transmission
γ_2	$\frac{1}{8}$	–
ε_2	0.01	Slightly higher mortality rate
Initial conditions:		
Human	$S_1 = 999{,}900$, $E_1 = I_1 = 50$, $R_1 = 0$	Larger initial outbreak
Rodent	$S_2 = 19{,}900$, $E_2 = I_2 = 50$	–
Simulation time	365 days	One year of monitoring

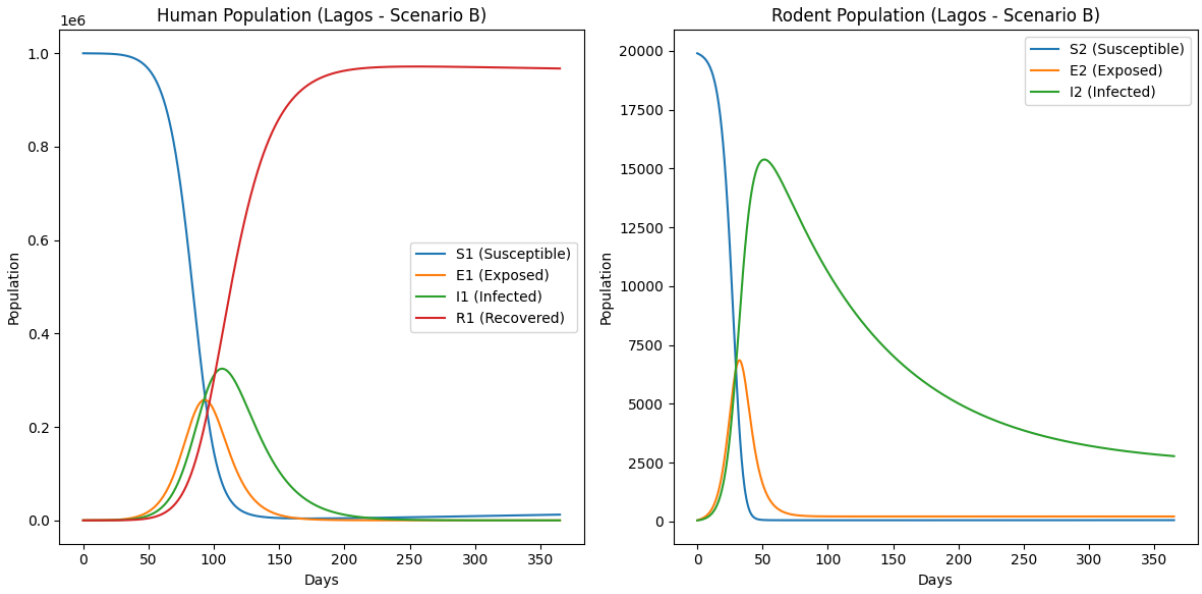

FIGURE 4. Dynamics of the human–rodent monkeypox model in Lagos under Scenario B

Table 4 summarizes the parameter values for Scenario C, which represents a high-transmission setting for monkeypox in Lagos. In this case, human-to-human spread is assumed to be strong ($\beta_1 = 0.6$), with noticeable rodent-to-human spillover ($\beta_3 = 0.05$). The incubation period is shorter ($\gamma_1 = \frac{1}{10}$ for humans, $\gamma_2 = \frac{1}{6}$ for rodents), and mortality rates are higher in both species. Initial conditions are set to reflect a large outbreak, with 500 exposed and 500 infected individuals in both human and rodent populations, simulating either an intense local outbreak or widespread importation. This scenario provides a worst-case baseline for analyzing uncontrolled epidemic spread.

TABLE 4. Numerical values of parameters under Scenario C for the Lagos city case

Parameter	**Numeric value**	**Note**
N_1	1,000,000	–
μ_1	5.0735667×10^{-5}	–
Λ_1	50.735667	–
β_1	0.6	High human transmission (no precautions)
β_3	0.05	Noticeable rodent–human transmission
γ_1	$\frac{1}{10}$	Shorter incubation period
α	$\frac{1}{18}$	Faster recovery
θ	0.0	–
ε_1	0.005	Higher human mortality
N_2	20,000	–
μ_2	0.0013698630	–
Λ_2	27.397260	–
β_2	0.8	High rodent–rodent transmission
γ_2	$\frac{1}{6}$	Shorter incubation in rodents
ε_2	0.02	Higher rodent mortality
Initial conditions:		
Human	$S_1 = 999{,}000$, $E_1 = I_1 = 500$, $R_1 = 0$	Large outbreak or widespread importation
Rodent	$S_2 = 19{,}000$, $E_2 = I_2 = 500$	–
Simulation time	365 days	One year of monitoring

Figure 5 illustrates the epidemic dynamics of the human–rodent monkeypox model in Lagos under Scenario C. In the human population, the susceptible class S_1 decreases steeply, while the exposed E_1 and infected I_1 groups rise to high peaks, reflecting strong human-to-human transmission. The recovered class R_1 grows rapidly, showing that a large proportion of the population becomes infected and eventually recovers within the one-year horizon. In rodents, the

susceptible class S_2 collapses early, while exposed E_2 and infected I_2 surge dramatically before declining due to recovery and mortality. Compared with Scenarios A and B, this case produces the most severe epidemic waves, highlighting how high transmission rates and widespread initial infections drive persistent and intense outbreaks.

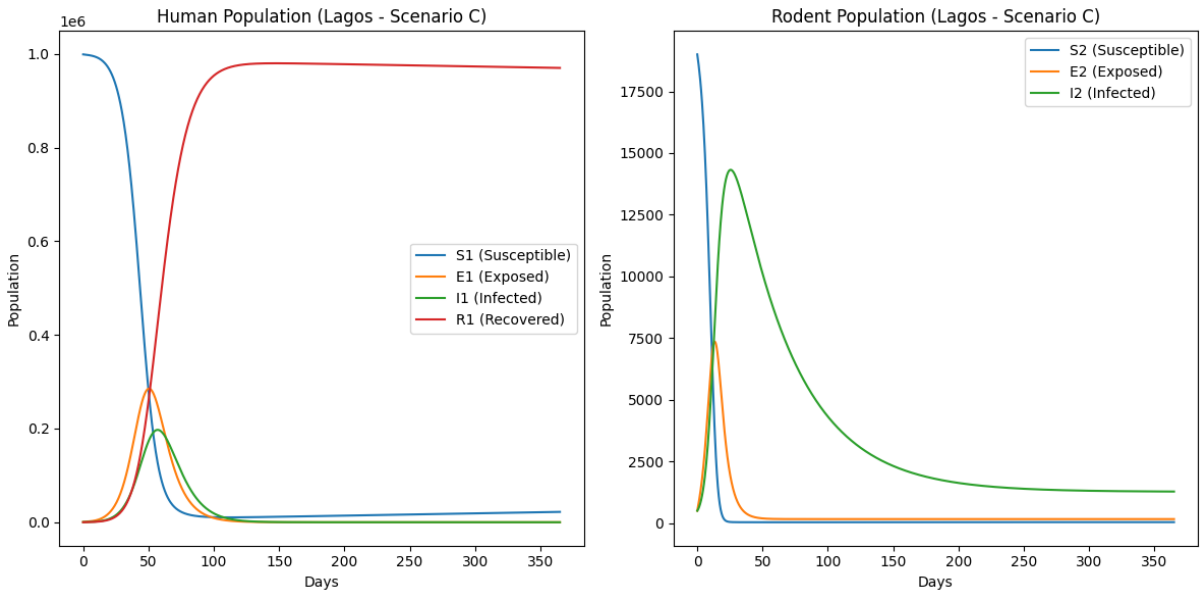

FIGURE 5. Dynamics of the human–rodent monkeypox model in Lagos under Scenario C

Table 5 reports the parameter values adopted for Scenario A at the national level in Nigeria. This scenario reflects a low-transmission setting with precautionary measures and limited spillover from rodents. The human population size is set to $N_1 = 232{,}679{,}478$ based on World Bank data (2024), while the rodent population is approximated at 10 million. Human-to-human transmission is assumed to be weak ($\beta_1 = 0.1$), and rodent-to-human spillover is minimal ($\beta_3 = 0.005$). Incubation and recovery periods are set to average values consistent with reported ranges, and disease-induced mortality is kept low for both species. Initial conditions assume a small number of imported or seeded cases (10 exposed and 10 infected individuals in each population). This scenario therefore represents a mild outbreak that is unlikely to escalate significantly without changes in epidemiological conditions.

TABLE 5. Numerical values of parameters under Scenario A for the Nigeria case

Parameter	**Numeric value**	**Note**
N_1	232,679,478	Population of Nigeria (World Bank, 2024)
μ_1	5.0736×10^{-5}	Natural death rate (daily) $\approx 1/(54\,\text{years} \cdot 365)$
Λ_1	11,805	$\simeq \mu_1 \times N_1$ (population equilibrium, per day)
β_1	0.1	Human-to-human transmission (low)
β_3	0.005	Rodent–human transmission (weak)
γ_1	$\frac{1}{12}$	Incubation period $\sim$ 12 days (within range 3–17/5–21)
α	$\frac{1}{21}$	Average recovery $\sim$ 21 days
θ	0.0	Neglected immunity
ε_1	0.0005	Few human disease deaths
N_2	10,000,000	Estimated rodent population
μ_2	0.00136986	Rodent daily mortality $\approx 1/(2\,\text{years})$
Λ_2	13,699	$\simeq \mu_2 \times N_2$
β_2	0.2	Rodent-to-rodent transmission (moderate–low)
γ_2	$\frac{1}{8}$	Rodent incubation $\sim$ 8 days
ε_2	0.005	Rodent disease mortality
Initial conditions:		
Human	$S_1 = N_1 - 20, E_1 = I_1 = 10, R_1 = 0$	Imported cases/small focus
Rodent	$S_2 = N_2 - 20, E_2 = I_2 = 10$	Humble beginning
Simulation time	365 days	One year of monitoring

Figure 6 illustrates the epidemic dynamics of the human–rodent monkeypox model in Nigeria under Scenario A. In the human population, the susceptible class S_1 declines only slightly, while the exposed E_1 and infected I_1 groups remain small, showing low-level peaks. The recovered class R_1 increases marginally, indicating limited disease spread. In the rodent population, the susceptible class S_2 decreases slowly, while exposed E_2 and infected I_2 rise moderately but do not escalate to severe levels. Overall, the dynamics confirm that with weak transmission and

few initial cases, the epidemic remains controlled and does not lead to large-scale outbreaks in either population.

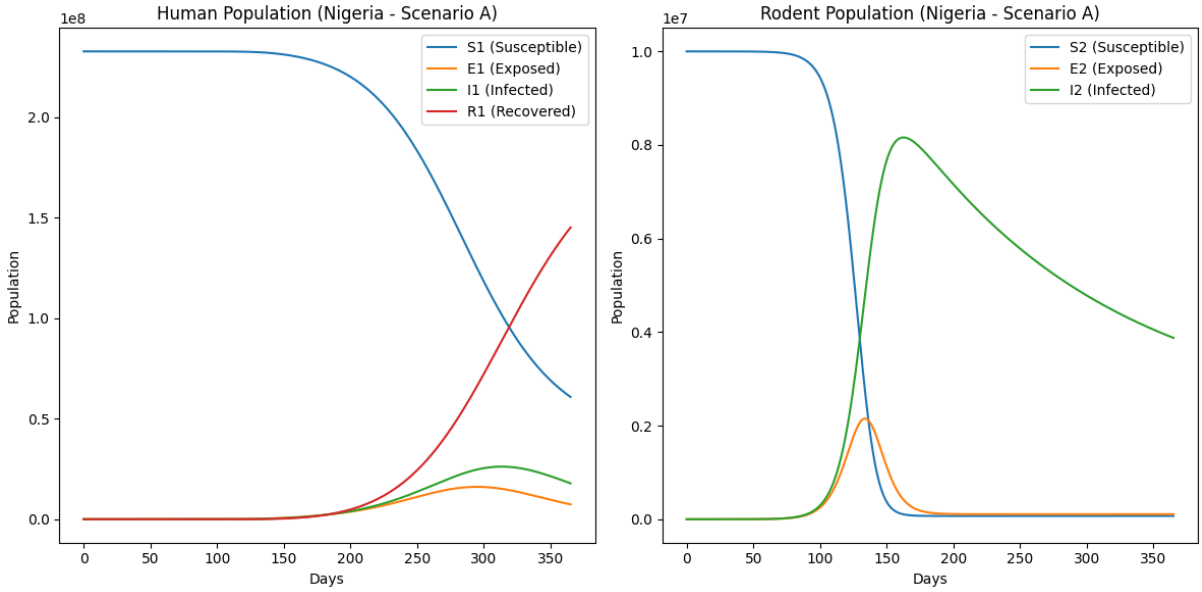

FIGURE 6. Dynamics of the human–rodent monkeypox model in Nigeria under Scenario A

Table 6 provides the parameter values for Scenario B at the national level in Nigeria. This scenario represents a moderate transmission setting, where human-to-human spread is more pronounced ($\beta_1 = 0.3$) due to social contact and lack of precautions, while rodent-to-human transmission ($\beta_3 = 0.02$) is noticeable but remains limited. Compared with Scenario A, the rodent-to-rodent transmission rate ($\beta_2 = 0.5$) is higher, and mortality rates are slightly elevated in both populations. Initial conditions include 50 exposed and 50 infected individuals in each population, representing a modest but more substantial outbreak than in Scenario A. This setup allows for the analysis of an epidemic under intermediate risk levels where spread is observable but not maximal.

TABLE 6. Numerical values of parameters under Scenario B for the Nigeria case

Parameter	Numeric value	Note
N_1	232,679,478	Population of Nigeria (World Bank, 2024)
μ_1	5.0736×10^{-5}	Daily natural death rate
Λ_1	11,805	$\simeq \mu_1 \times N_1$
β_1	0.3	Moderate human transmission (lack of precautions/social contact)
β_3	0.02	Rodent-to-human transmission clear but limited
γ_1	$\frac{1}{12}$	Incubation $\sim$ 12 days
α	$\frac{1}{21}$	Recovery $\sim$ 21 days
θ	0.0	–
ε_1	0.001	Low human mortality
N_2	10,000,000	Assumed rodent population
μ_2	0.00136986	Daily mortality
Λ_2	13,699	$\simeq \mu_2 \times N_2$
β_2	0.5	Rodent-to-rodent transmission (higher than Scenario A)
γ_2	$\frac{1}{8}$	–
ε_2	0.01	Slightly higher rodent mortality
Initial conditions:		
Human	$S_1 = N_1 - 100$, $E_1 = I_1 = 50$, $R_1 = 0$	Slightly higher starting outbreak (imported/primary)
Rodent	$S_2 = N_2 - 100$, $E_2 = I_2 = 50$	–
Simulation time	365 days	One year of monitoring

Figure 7 illustrates the epidemic dynamics of the human–rodent monkeypox model in Nigeria under Scenario B. In the human population, the susceptible class S_1 decreases more noticeably than in Scenario A, while the exposed E_1 and infected I_1 classes rise to moderate peaks before declining. The recovered class R_1 increases steadily, showing wider infection spread compared to Scenario A. In the rodent population, the susceptible class S_2 declines more sharply, while exposed E_2 and infected I_2 reach higher peaks before eventually decreasing due to recovery and mortality. Overall, Scenario B reflects a more intense epidemic than Scenario A, highlighting how moderate increases in transmission parameters and a larger seeding of cases can generate stronger waves in both humans and rodents.

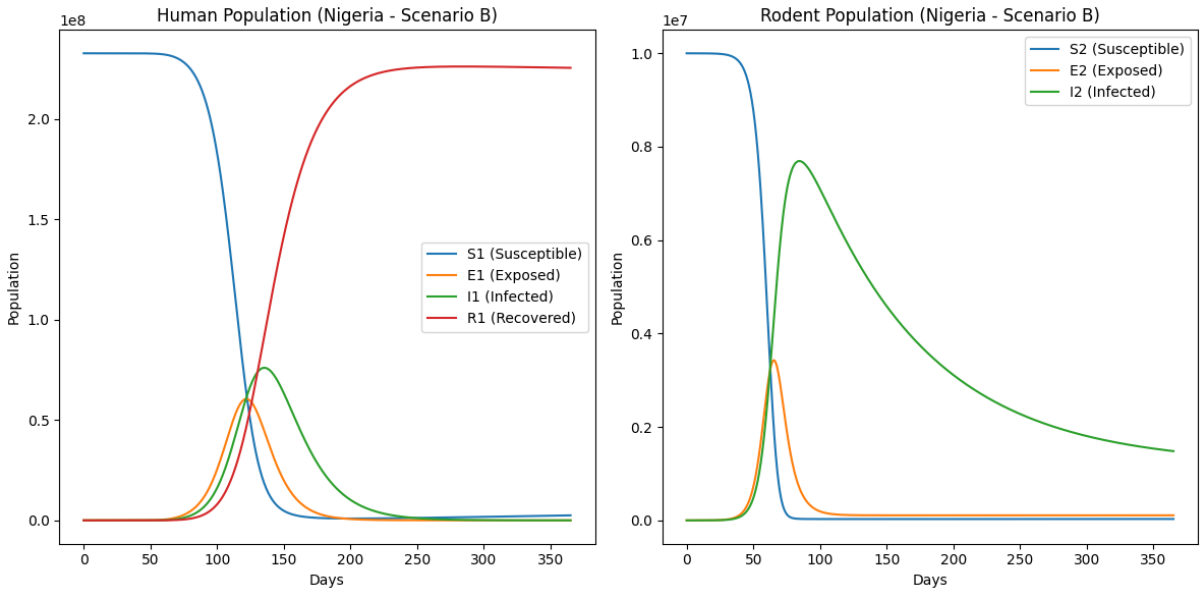

FIGURE 7. Dynamics of the human–rodent monkeypox model in Nigeria under Scenario B

Table 7 presents the parameter values for Scenario C at the national level in Nigeria. This scenario represents a high-transmission setting that mimics a worst-case outbreak. Human-to-human spread is strong ($\beta_1 = 0.6$), while rodent-to-human contact is also significant ($\beta_3 = 0.05$), reflecting conditions of high exposure. The incubation period is shorter ($\gamma_1 = \frac{1}{10}$ for humans, $\gamma_2 = \frac{1}{6}$ for rodents), while recovery is slightly faster, and mortality rates are notably higher in both species ($\varepsilon_1 = 0.005$, $\varepsilon_2 = 0.02$). Initial conditions assume a large outbreak with 500 exposed and 500 infected individuals in each population, combined with reduced susceptible numbers. This setup captures a severe epidemic wave and provides insight into the dynamics of uncontrolled spread in a densely populated country like Nigeria.

TABLE 7. Numerical values of parameters under Scenario C for the Nigeria case

Parameter	Numeric value	Note
N_1	232,679,478	Population of Nigeria (World Bank, 2024)
μ_1	5.0736×10^{-5}	Daily natural death rate
Λ_1	11,805	$\simeq \mu_1 \times N_1$
β_1	0.6	High human transmission (lack of precautions/social contact)
β_3	0.05	High rodent–human transmission (high-contact environment)
γ_1	$\frac{1}{10}$	Shorter incubation period (CDC ref. 3–17/5–21 days)
α	$\frac{1}{18}$	Slightly faster recovery (different clinical course)
θ	0.0	–
ε_1	0.005	Higher human mortality (worst-case scenario)
N_2	10,000,000	Assumed rodent population
μ_2	0.00136986	Daily mortality
Λ_2	13,699	$\simeq \mu_2 \times N_2$
β_2	0.8	High rodent-to-rodent transmission
γ_2	$\frac{1}{6}$	Shorter rodent incubation
ε_2	0.02	Higher rodent mortality
Initial conditions:		
Human	$S_1 = N_1 - 1000$, $E_1 = I_1 = 500$, $R_1 = 0$	Large outbreak or widespread importation
Rodent	$S_2 = N_2 - 1000$, $E_2 = I_2 = 500$	–
Simulation time	365 days	One year of monitoring

Figure 8 shows the epidemic dynamics of the human–rodent monkeypox model in Nigeria under Scenario C. In the human population, the susceptible class S_1 declines rapidly, while the exposed E_1 and infected I_1 classes rise steeply, indicating a large epidemic wave. The recovered class R_1 grows substantially, showing that a significant proportion of the population becomes infected and later recovers. In rodents, the susceptible population S_2 collapses early, while the exposed E_2 and infected I_2 classes reach very high peaks before gradually declining due to recovery and mortality. Compared with Scenarios A and B, this scenario generates the most severe epidemic dynamics, highlighting the risks of high transmission and widespread initial infections in both species.

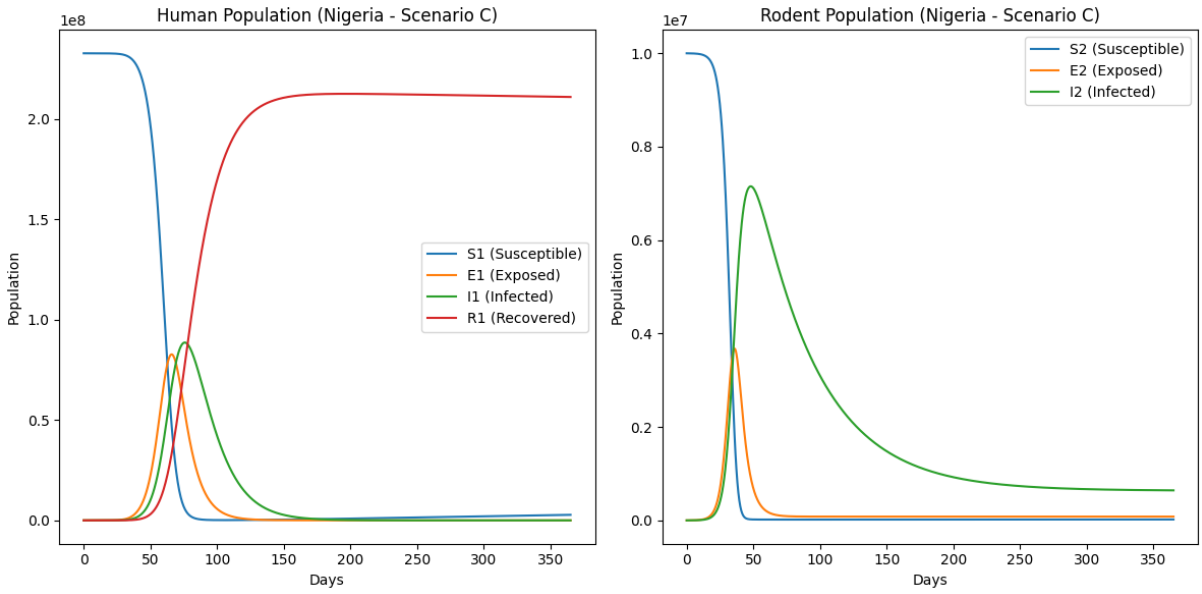

FIGURE 8. Dynamics of the human–rodent monkeypox model in Nigeria under Scenario C

5. CONCLUSION

This study investigated the dynamics of monkeypox transmission between humans and rodents in Nigeria through a hierarchical SEIR-type model. The mathematical analysis confirmed the biological feasibility of the model by establishing positivity and boundedness of solutions, and by identifying an invariant region together with the disease-free equilibrium (DFE). These results provided a rigorous theoretical basis for understanding the long-term behavior of the system. Numerical simulations using the adaptive Runge–Kutta method of order 4(5) (RK45) demonstrated that rodent populations act as a persistent reservoir, sustaining the infection even when human-to-human transmission is relatively weak. The results also revealed the high sensitivity of monkeypox dynamics to both ecological and epidemiological parameters, showing that small variations in transmission rates may be sufficient to trigger epidemic waves. From a public health perspective, the findings suggest that effective control strategies must combine rodent population management with human-focused interventions, such as awareness campaigns, surveillance programs, and vaccination. The hierarchical modeling framework employed here has proven valuable in capturing the complexity of zoonotic diseases and may be extended to other infectious diseases where animal reservoirs play a crucial role.

CONFLICT OF INTERESTS

The authors declare that there is no conflict of interests.

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