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Stochastic and Deterministic Model for Transmission of Monkeypox Disease

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Abstract
In this paper, a SIR Model is established for Monkey Pox disease. SIR is an acronym which stands for Susceptible, Infectious and Recovered groups in a given population. An equivalent deterministic model which is an auxiliary tool is transformed into a stochastic model. The stochastic model is studied by numerical simulation which is used to analyse the control of transmission of the disease. Numerical simulation of the model shows that an increase in vaccination leads to low disease prevalent in a population. Raising awareness of risk factors and educating people about the measure they can take to reduce exposure to the virus is the main prevention strategy for Monkey Pox.

Introduction
Monkeypox is a viral zoonosis, meaning it is a virus transmitted to humans from animals, sharing symptoms with smallpox but generally presenting less severe clinical manifestations. With the eradication of smallpox in 1980 and the subsequent discontinuation of smallpox vaccination, Monkeypox has become the most significant orthopox virus. It is prevalent in Central and West Africa, often found in close proximity to tropical rainforests (WHO, 2017).

The first documented case of human Monkeypox occurred in 1970 in a nine-year-old boy in the Democratic Republic of Congo, where smallpox had been eliminated in 1968. Subsequently, most cases have been reported in rural, rainforest regions of the Congo Basin, particularly in the Democratic Republic of the Congo, where it is considered endemic (WHO, 2017).

Since 1970, human Monkeypox cases have been reported in eleven African countries, including Nigeria, where the largest documented outbreak occurred in 2017, forty years after the last confirmed case. The outbreak involved 122 confirmed or probable cases with a 6% case fatality rate, affecting individuals aged between 2 days and 50 years, with

a higher prevalence in males. Common symptoms included vesiculopustular rash, fever, pruritus, headache, and lymphadenopathy. In 2019, Nigeria reported 113 suspected cases, with 46 confirmed cases and one death. Four states (Lagos, Delta, Rivers, and Bayelsa) accounted for 85% of the confirmed cases (NCDC, 2019).

Monkeypox infection in humans typically results from direct contact with the blood, bodily fluids, or cutaneous material of infected animals. Evidence of Monkeypox virus infection has been found in various animals, including rope squirrels, tree squirrels, Gambian poached rats, dormice, and different monkey species. The natural reservoir of Monkeypox has not yet been identified, but rodents are considered the most likely source. Inadequately cooked meat and other animal products from infected animals pose a potential risk (WHO, 2017).

While secondary or human-to-human transmission is limited, close contact with respiratory secretions can lead to infection. Droplet respiratory particle transmission usually requires prolonged face-to-face contact, putting health workers and household members of active cases at greater risk. Transmission can also occur via the placenta, leading to congenital Monkeypox. Therefore, prevention strategies focus on raising awareness of risk factors and educating people about measures to reduce virus exposure. Ongoing scientific studies are assessing the feasibility and appropriateness of using vaccines for Monkeypox prevention and control (WHO, 2017).

To complement this research, existing literature on the mathematical study of Monkeypox is briefly outlined. Bhunu and Mushayabasa (2011) developed an SIR mathematical model for the transmission dynamics of pox-like infections in humans and rodents/wild animals. Emeka et al (2018) incorporated an imperfect vaccine compartment in their mathematical model for Monkeypox virus transmission dynamics. Bankuru, Samuel, William, Parsa, Jan and Dewey. (2020) employed a game theoretic model to assess vaccination strategies, suggesting that Monkeypox is controllable but may not be eradicated by vaccination alone in a fully endemic equilibrium. Lasisi, Akinwande and Oguntolo, (2020) developed a mathematical model for the transmission of Monkeypox in humans using ordinary differential equations, providing valuable insights into the effective basic reproduction number of the model. This paper contributes to the field by offering a stochastic analysis of the transmission dynamics of Monkeypox using Allen's Method (2008).

Model Formulation

The Deterministic Model Formulation

A compartment non-linear deterministic Model is introduced here, adapted from Peter *et al* (2018) which was previously used to describe the transmission of infectious disease. The compartmental model sub-divided the human population into three classifications namely:

S: Number of susceptible individuals who can be infected

I: Number of infectious case in the community, who are capable of transmitting the diseases.

R: Number of individuals removed from the chain of transmission (cored or dead and buried)

The flow diagram for the transmission dynamics is shown in Figure 1.

The SIR model can be represented by a system of ordinary differential equations as follows:

$$\left.\begin{aligned} \frac{dS}{dt} &= \beta - \alpha SI - (p+\mu)S + \sigma R \\ \frac{dI}{dT} &= \alpha SI - (\gamma + \delta + \mu)I \\ \frac{dR}{dt} &== \gamma I - (\mu + \sigma)R + \rho S \end{aligned}\right\} \qquad (1)$$

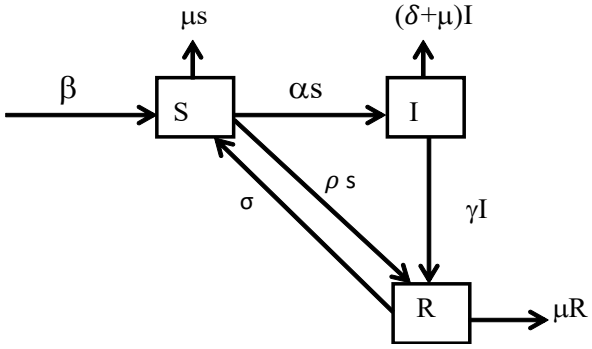

Figure 1: ***Transmission dynamics of Monkeypox***

The parameters used for the model explained are explained on Table 1.

Table 1: Model parameters

Parameter	Description
ó	Rate of loss immunity after recovery/vaccination
γ	Rate of recovery from infection
δ	Disease induced death rate
μ	Natural death rate
α	Contact rate
ρ	Vaccination rate
β	Recruitment rate

The model is based on the following assumptions:

i. Individuals are recruited into the susceptible class through birth
ii. All susceptible individuals can be infected through a direct contact with infectious individuals.
iii. Some newborns are vaccinated at birth while some are not.
iv. Those in each class can die as a result of natural death

Existence and Uniqueness of the solution

Lemma 1: The closed set

$$D = \{S + I + R:/S - S(0)/\leq a,/I - I(0)/\leq b,/R - R(0)/\leq c\}$$

Proof:

Consider the biologically-feasible region D, defined above. The model in (2.1) must be continuous and bounded in D. Therefore,

$/\frac{dxi}{dxj}/$, i, j, = 1, 2, 3 are continuous and bounded. All solution of the (2.1) with initial conditions in D. Hence the model (1) has a unique solution in D, which means that the model (3.1) is epidemiologically and mathematically well posed.

Equilibrium State of the Model Analysis

We now solve the model equations to obtain the equilibrium states. At equilibrium

$$\frac{dS(t)}{dt} = \frac{dI(t)}{dt} = \frac{dR(t)}{dt} = 0$$

Let:

$$S(t) = k,\ I(t) = l,\ R(t) = m$$

Then the equations (2.1) become:

$$\left.\begin{aligned} \beta - \alpha kI - (\rho + \mu)k + \sigma = 0 \\ \alpha kI - (\gamma + \delta + \mu)I = 0 \\ \gamma I - (\mu + \sigma)m + \rho k = 0 \end{aligned}\right\} \quad (2)$$

Existence of Endemic Equilibrium Point (EEP)

Endemic Equilibrium point (EEP) is a steady – state solution where the disease persists in the populations. Therefore, from the model equations, we have the following corresponding to the endemic equilibrium points in the population being infected with Monkey pox.

$$k = \frac{\gamma + \delta + \mu}{\alpha} \quad (3)$$

$$I = \frac{\mu\alpha\beta - \mu\rho\gamma - \mu\rho\delta - \rho\mu^2 - \mu^2\gamma - \mu^2\delta - \mu^3 + \alpha\sigma\beta - \mu\sigma\gamma - \mu\sigma\delta - \mu^2\sigma}{\alpha(\mu\Upsilon + \mu\delta + \mu^2 + \delta\sigma + \mu\sigma)} \quad (4)$$

$$m = \frac{\gamma\alpha\beta - \mu\gamma^2 - \gamma\mu\delta - \gamma\mu^2 + \rho\gamma\delta + \rho\gamma\mu + \rho\delta^2 + 2\rho\delta\mu + \rho\mu^2}{\alpha(\mu\gamma + \mu\delta + \delta\sigma + \mu^2 + \mu\sigma} \quad (5)$$

Disease Free Equilibrium

The disease free equilibrium is defined as the point in which no disease is present in the population, which is represented in the model as I = 0 . The system of equation simplifies to

$$S^1 = \beta - \mu s;\ I^1 = 0;\ R^1 = 0$$

Thus, the disease-free equilibrium lies at the point $(\frac{\beta}{\mu}, 0, 0)$

The Basic Reproduction Number (R_o)

According to Diekmann *et al* (1990), the basic reproduction Number, R_0 is defined as the number of secondary infections that one infectious individual would create over the duration of the infectious period, provided that everyone also is susceptible. $R_0 = 1$, is a threshold below which the generation of secondary cases is insufficient to maintain the infection within human community. If $R_0<1$, each individual produces on average, less than one new infected individuals and hence the disease dies out. If $R_0>1$, each

individual produces more than one new infected individual and hence the disease is capable of invading the susceptible population. It is therefore a useful quantity in the study of a disease as it sets the threshold for its establishment.

In order to compute R_0, it is important to distinguish new infections from all other changes in the population. Let:
F_i be the rate of appearance of new infectious in compartment.
$V_{-I} = V_i - V_i$, be the different between th rate of transfer of individuals out o compartment I, (V_i), by all other mean and the rate of transfer of individual in the compartment, i, (V_i) by all other means.
X_0 be the disease-free equilibrium point
Let:

$$F = \left[\frac{\delta F_i}{\delta x_j}(x_o)\right] \text{ and } V = \left[\frac{\delta V_i}{\delta x_j}(x_o)\right] \quad (7)$$

With $1 \le i, j \le m$.

F is non-negative, V is a non-singular m-Matrix in which both are the m x m matrices, where m stands for the number of infected classes. We use next generation operator approach to define the basic reproduction number of the model as

$$R_o = \frac{\alpha\beta}{\mu(\gamma+\delta+\mu)} \quad (8)$$

Local Stability of the Disease-Free Equilibrium (E_0)

The Local stability of the disease-free equilibrium can be discussed by examining the linearized form of the system (2.1) at the steady state E_0.

Lemma 2: The disease-free equilibrium point E_0 for the system (2.1) is locally asymptotically stable if $R_0<1$ and unstable if $R_0>1$

The Jacobian matrix G is given by:

$$G = \begin{Bmatrix} -\alpha l(\rho+\mu) & -\alpha k & \sigma \\ \alpha l & \alpha k-(\gamma+\delta+\mu) & 0 \\ \rho & \gamma & -(\mu+\sigma) \end{Bmatrix} \quad (9)$$

$$G/(E_0) = \begin{bmatrix} -(\rho+\mu+\lambda) & -\frac{\alpha\beta}{\mu} & \sigma \\ 0 & \frac{\alpha\beta}{\mu}-(\gamma+\delta+\mu)-\lambda & 0 \\ \rho & \gamma & -(\mu+\sigma+\lambda) \end{bmatrix} \quad (10)$$

The characteristic equation of the above matrix is defined by:

$$\det |G - \lambda I| = 0$$

The eigenvalues of $G(E_0)$

$$-(\rho+\mu), -\left[-(\gamma+\delta+\mu)+\frac{\alpha\beta}{\mu}\right] \text{ and } -(\mu+\sigma)$$

From the above, we have all the eigenvalues being real and negative, which shows that the model is locally asymptotically stable.

Formulation of the stochastic Model

Allen, Allen, Arciniega and Greenwood (2008) used a method of discretizing the continuous time stochastic process. We assume in addition to the assumptions of the model in session 2.1 that transition from the susceptible class to the infected class is under some random influence which we modeled as white noise represented by a Wiener process.

Applying this method yields Table 2.

Table 1: Transition table and their probabilities

Change	Probability	Event
$[100]^T$	$P_1 = \beta\Delta t$	Birth of susceptible individuals
$[-100]^T$	$P_2 = \mu s\Delta t$	Susceptible dies natural death
$[-101]^T$	$P_3 = \rho s\Delta t$	Vaccinated susceptible becomes recovered.
$[-110]^T$	$P_4 = \alpha s\Delta t$	Susceptible becomes infected
$[0\text{-}10]^T$	$P_5 = (\delta + \mu)\, I\Delta t$	Infected dies natural death due to the disease.
$[0\text{-}11]^T$	$P_6 = \gamma I\Delta t$	Infected becomes recovered
$[00\text{-}1]^T$	$P_7 = \mu R\Delta t$	Recovered dies natural death
$[10\text{-}1]^T$	$P_8 = \sigma R\Delta t$	Recovered loses immunity

Stochastic differential equation is characterized by the drift (diffusion) coefficient and the diffusion (volatility) where the drift is given by:

$$\vec{f} = \sum_{j=1}^{8} p_j \lambda_j$$

Where λ_j and P_j are the random changes and the transition probabilities defined in the table 1. That is;

$$f = P_1\begin{bmatrix}1\\0\\0\end{bmatrix} + P_2\begin{bmatrix}-1\\0\\0\end{bmatrix} + P_3\begin{bmatrix}-1\\0\\1\end{bmatrix} + P_4\begin{bmatrix}-1\\1\\0\end{bmatrix} + P_5\begin{bmatrix}0\\-1\\0\end{bmatrix} + P_6\begin{bmatrix}0\\-1\\1\end{bmatrix} + P_7\begin{bmatrix}0\\0\\-1\end{bmatrix} + P_8\begin{bmatrix}1\\0\\-1\end{bmatrix}$$

$$= \begin{bmatrix}\beta\\0\\0\end{bmatrix} + \begin{bmatrix}-\mu S\\0\\0\end{bmatrix} + \begin{bmatrix}-\rho S\\0\\\rho S\end{bmatrix} + \begin{bmatrix}-\alpha SI\\\alpha SI\\0\end{bmatrix} + \begin{bmatrix}0\\-(\delta+\mu)I\\0\end{bmatrix} + \begin{bmatrix}0\\-\gamma I\\\gamma I\end{bmatrix} + \begin{bmatrix}0\\0\\-\mu R\end{bmatrix} + \begin{bmatrix}\sigma R\\0\\-\sigma R\end{bmatrix}$$

Hence, the drift vector $\vec{f}$ of order is:

$$f = \begin{bmatrix}\beta - (\alpha I + \rho + \mu)S + \sigma R\\ \alpha SI - (\gamma + \delta + \mu)I\\ \gamma I - (\mu + \sigma)R + \rho S\end{bmatrix} \quad (11)$$

The covariance matrix is defined by:

$$V = \sum_{j=1}^{8} p_j \lambda_j (\lambda_j)^T \quad (12)$$

$$V = P_1\begin{bmatrix}1\\0\\0\end{bmatrix}[100] + P_2\begin{bmatrix}-1\\0\\0\end{bmatrix}[-100] + P_3\begin{bmatrix}-1\\0\\1\end{bmatrix}[-101] + P_4\begin{bmatrix}-1\\1\\0\end{bmatrix}[-110]$$

$$+ P_5\begin{bmatrix}0\\-1\\0\end{bmatrix}[0-10] + P_6\begin{bmatrix}0\\-1\\1\end{bmatrix}[0-11] + P_7\begin{bmatrix}0\\0\\-1\end{bmatrix}[00-1] + P_8\begin{bmatrix}1\\0\\-1\end{bmatrix}[10-1]$$

Multiplying the covariance matrix, we have

$$V = P_1\begin{bmatrix}1&0&0\\0&0&0\\0&0&0\end{bmatrix} + P_2\begin{bmatrix}1&0&0\\0&0&0\\0&0&0\end{bmatrix} + P_3\begin{bmatrix}1&0&-1\\0&0&0\\-1&0&1\end{bmatrix} + P_4\begin{bmatrix}1&-1&0\\-1&1&0\\0&0&0\end{bmatrix} + P_5\begin{bmatrix}0&0&0\\0&1&0\\0&0&0\end{bmatrix}$$

$$+ P_6\begin{bmatrix}0&0&0\\0&1&-1\\0&-1&1\end{bmatrix} + P_7\begin{bmatrix}0&0&0\\0&0&0\\0&0&1\end{bmatrix} + P_8\begin{bmatrix}1&0&-1\\0&0&0\\-1&0&1\end{bmatrix}$$

Which therefore, implies;

$$V = \begin{bmatrix}\beta&0&0\\0&0&0\\0&0&0\end{bmatrix} + \begin{bmatrix}\mu S&0&0\\0&0&0\\0&0&0\end{bmatrix} + \begin{bmatrix}\rho S&0&\rho S\\0&0&0\\-\rho S&0&0\end{bmatrix} + \begin{bmatrix}\alpha SI&-\alpha SI&0\\-\alpha SI&\alpha SI&0\\0&0&0\end{bmatrix}$$

$$+ \begin{bmatrix}0&0&0\\0&(\delta+\mu)I&0\\0&0&0\end{bmatrix} + \begin{bmatrix}0&0&0\\0&\gamma I&-\gamma I\\0&-\gamma I&\gamma I\end{bmatrix} + \begin{bmatrix}0&0&0\\0&0&0\\0&0&\mu R\end{bmatrix} + \begin{bmatrix}\sigma R&0&-\sigma R\\0&0&0\\-\sigma R&0&\sigma R\end{bmatrix}$$

Therefor the covariance matrix V of order 3x3 is given by:

$$V = \begin{bmatrix}\beta + (\mu + \rho + \alpha I)S + \sigma R & -\alpha SI & -\rho S - \sigma R\\ -\alpha SI & \alpha SI + (\delta + \mu)I + \gamma I & -\gamma I\\ -\rho S - \sigma R & -\gamma I & \rho S + \gamma I + \mu R + \sigma R\end{bmatrix} \quad (13)$$

Consequently, the resulting stochastic differential equation model of the equivalent ordinary differential model in equation (1) is:

$$d\vec{X} = f\left(t, \vec{X}(t)\right)dt + \vec{V}^{\frac{1}{2}}\left(t, \vec{X}(t)\right)d\vec{W}(t)$$

$$\vec{X}(0) = [X_1(0)X_2(0)X_3(0)] \quad (14)$$

where F and V are as defined in equation (3.1) and (3.3) respectively which is the stochastic counterpart of

Method of Solution

The Millstein method is used for the simulation of the SDE SIR model for the transmission of Monkey Pox. The Milstein scheme is an extension of the Euler-Maruyama method and it is known to be more accurate and refined method compared to Euler-Maruyama, particularly when dealing with SDEs that have strong or nonlinear stochastic terms. The scheme makes used of the Ito lemma to improve on the accuracy of the Euler-Maruyama methods by adding a second order term to the approximation of the Ito process. Representing σ_x the partial derivative of $\sigma(t,x)$ with respect to x of the Ito type SDE which is given by

$$dX_t = \mu dt + \sigma dW_t$$

The Milstein is given by:

$$Y_{n+1} = Y_i + b(t_i, Y_i)(t_{i+1} - t_i) + \sigma(t_i, Y_i)(W_{i+1} - W_i)$$

$$+\frac{1}{2}\sigma(t_i, Y_i)\sigma_x(t_i, Y_i)\{(W_{i+1} - W_i)^2 - (t_{i+1} - t_i)\} \quad (15)$$

This can be written in a more compact form as:

$$Y_{n+1} = Y_i + b\Delta t + \sigma\Delta W_i + \frac{1}{2}\sigma\sigma_x\{(\Delta W_i)^2 - \Delta t\} \quad (16)$$

The Milstein scheme is known to have both weak and strong convergences of order 1. The algorithm for the method is as follows:

Algorithm for the Milstein Method

Function Milstein Method (a, b, X0, T, dt):

1. Input the following:
   $f(t, X(t))$- Drift term function

   $V(t, X(t))$ - Diffusion term function

   X_0- Initial value of the process

   T- Total simulation time

   dt- Time step size

2. Determine the number of time steps
   $num_{steps} = int(\frac{T}{dt})$

3. Initialize arrays to store the time and process values
   times = [0.0] * (num_steps + 1)

   X = [0.0] * (num_steps + 1)

4. Set initial values
   times[0] = 0.0

   X[0] = X0

5. Perform the Milstein simulation
   for i in range(1, num_steps + 1):

   a. Current time
      t = i * dt

      times[i] = t

   b. Calculate the deterministic and stochastic terms
      drift_term = $f(t, X(t)$

      diffusion_term = $V(t, X(t))$-

   c. Generate a random increment (sampled from N(0, dt))
   d. $dW = sqrt(dt) * rand\_normal(\ \ )$
      Calculate the correction term due to second-order expansion

      correction_term = $0.5 * f(t, X(t_{i-1}) * V(t, X(t_{i-1}) * dW^2 - dt$

      Update the process value using the Milstein scheme

      $X_i = X_{i-1} + drift\_term * dt + diffusion\_term * dW + correction\_term$

6. return times, X

The algorithm was implemented in Python programming language.

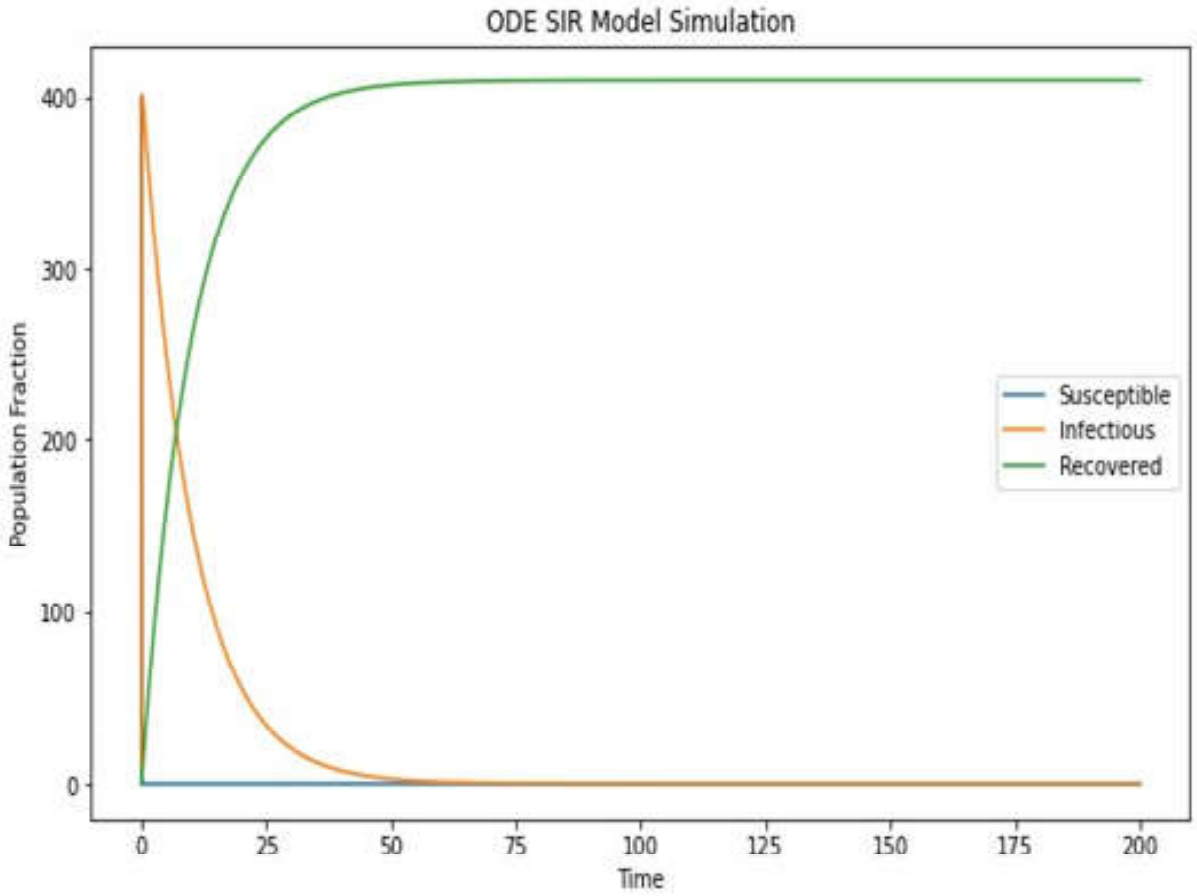

Figure 1:Deterministic model simulation

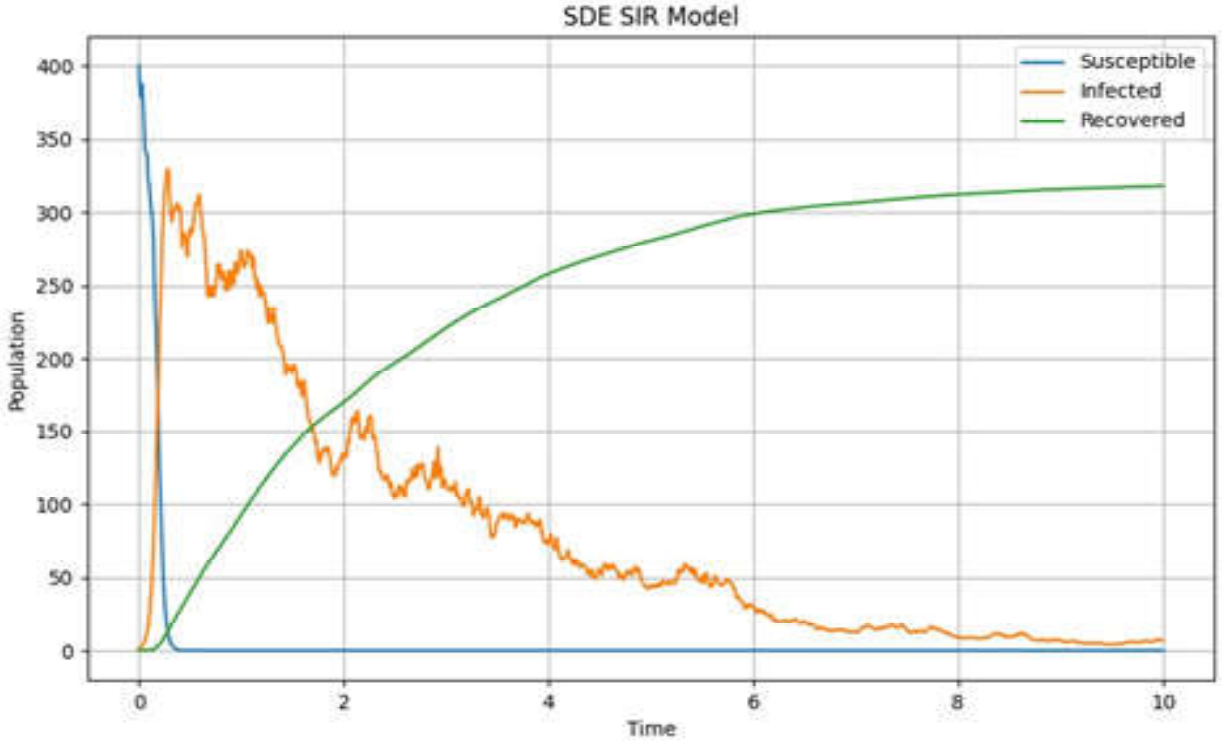

Figure 2: Stochastic model simulation

Conclusion

In conclusion, this work provides a comprehensive overview of Monkeypox, a viral zoonosis that has gained significance since the eradication of smallpox. The study delves into the historical background, epidemiology, and clinical characteristics of Monkeypox, with a focus on the notable outbreaks in Nigeria. The paper emphasizes the importance of raising awareness about risk factors and preventive measures, and highlights ongoing scientific efforts to explore the feasibility of vaccination strategies.

In addition to the descriptive analysis, the research contributes to the field by incorporating and SDE based mathematical modeling approach to better understand the transmission dynamics of Monkeypox. While emphasizing the limited existing literature on mathematical modeling of Monkeypox, the paper underscores the importance of such models in informing public health interventions.

In conclusion, this research not only consolidates existing knowledge on Monkeypox but also contributes novel insights through mathematical modeling, thereby enhancing our understanding the dynamics of the spread of the disease. Continued interdisciplinary efforts, combining epidemiology, clinical studies, and mathematical modeling are recommended as being crucial for addressing the challenges posed by Monkeypox in the future.

Reference

Allen, E.J; Allen L.J.S; Arciniega A; Greenwood P.E (2008).Construction of equivalent stochastic Differential Equation models. Stochastic Analysis Applications 26(2):274-297.

Bankuru S.V; Samuel K, William H; Parsa M; Jan R; Dewey. T(2020). A game – theoretic model of Monkeypox to assess Vaccination Strategies. Peer j,

Vol 8:e9272 Dol:10.7717/peer j. 9272.

Bhunu, C.P; Mushayabasa, S (2011). Modeling the Transmission Dynamics of pox-like infections. International Journal of Applied mathematics, 41,2.

Diekmann, O, Hesterbeek, J.A.P and Metz, J.A.J (1990): On the definition and the computation of the basic reproduction number ratio R_0 in model for infectious diseases in heterogeneous populations .J.Math.Biol.28.365-612.

Emaka, P.C; Onuorah, M.o, Eguda, F.t; Babangida, B.G (2018).Mathematical model for Monkeypox virus transmission Dynamics. Epidemiology (Sunnyvale), Vol 8(3):348.Dol:10.4172/ 2161-1165.1000348.

Lasisi, N.O; Akinwande, N.I; Oguntolo, F.A (2020).Development and exploration of a Mathematical model for the transmission of Monkeypox disease in humans. Mathematics models in Engineering, Vol 6, issue 1, P.23-33.

NCDC (2019).Nigeria Monkeypox monthly situation report.

Retrieved from: http://ncdc.gov.ng/ themes/common/docs/protocols/96-1577798 337.pdf.

Peter, O. J; Akinduko, O. B; Oguntolu, F. A; Ishola, C. Y (2018) Mathematical Model for the Control of Infectious Disease. J. Appl Sci. Environ. Manage. vol.22(4) 447 – 451.

WHO (2017). "Monkeypox Fact Sheet". World Health Organization 21 December 2017. Retrieved 7 April, 2018