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MONKEYPOX MATHEMATICAL MODEL WITH SURVEILLANCE AS CONTROL

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Abstract. A compartmental mathematical model of the transmission dynamics of the monkeypox virus (MPXV) was developed and analyzed. The model incorporates proper surveillance and contact tracing as effective controls. The equilibrium states of the model were obtained and analyzed both locally and globally. The effective reproduction number, R_m was obtained and the sensitivity of the model parameters were studied using R_m as the threshold of transmission. When the infection becomes endemic, $R_m \approxeq 1$, the model exhibits a backward bifurcation but $R_m < 1$ which means that the interventions tend to MPXV containment. Numerical simulations to bespeak our findings and discussions are provided. Our result shows that surveillance and contact tracing are effective for the containment of MPXV in the absence of a perfect vaccine.

Keywords: mathematical modeling; monkeypox; reproduction number; stability; surveillance.

2020 AMS Subject Classification: 92C60.

1. INTRODUCTION

Among the epidemic rampaging the world lately, monkeypox is the one with disturbing reemergence. Since the first recorded case of monkeypox virus (MPXV) on human in 1970 in the Democratic Republic of Congo (DRC), regardless that it was initially discovered in Denmark in 1958 [16, 3, 13], it has been trending in Central and West African nations [10, 15]. The

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burden of this epidemic is increasing substantially and has eventually became a global concern. Between 1st January and 4th October 2022, 69,244 cases have been confirmed in around 107 different countries of which the United Kingdom has the highest number of confirmed cases of 26,194 and Nigeria has the highest in Africa with 400 confirmed cases including over 26 deaths [15, 27]. Monkeypox is predominantly found in regions/states that are closed to tropical rain-forests in the remote area where humans interact with the forest and its game, hence it is a zoonotic disease. The virus that causes monkeypox is a double-stranded DNA virus of the family *Poxviridae* that belongs to the genus *Orthopoxvirus* [17] which has two genetic clades: central African clade and west African clades of which the former is believed to be more transmissible [15]. Transmission of MPXV is through animal hosts including but not limited to a range of rodents, squirrels, pets, monkeys, chimpanzees, and other non-human pirates. Zoonotic transmission often occurs from direct contact with the blood, bodily fluids, cutaneous or mucosal lesions of an infected animal, and likewise human to human transmission [18]. Human-to-human transmission is thought to occur predominately through respiratory droplets that can not travel more than a few feet, and thus prolonged face-to-face contact is always a reveille to the infection, body fluids and lesion-infected materials are other red - flags. MPXV is the most common orthodox virus currently since the eradication of smallpox in 1980 [19]. Regardless that prolonged face-to-face contact is needed for the transmission of monkeypox virus, the longest chain of community transmission recently is from 6 to 9 successive person-to-person infection [15]; which may be a result of the cessation of smallpox vaccination which has proved potent in boosting the immune system against monkeypox.

Monkeypox has symptoms that are clinically similar to those seen in smallpox viral infection patients, although it is clinically less severe [20, 27]. When in contact with MPXV, the incubation period is often between 6 to 13 days but can range from 5 - 21 days [27, 20, 15]. MPXV infection within the invasion period, 0 - 5 days is characterized by headache, fever, back pain, asthenia, myalgia, and lymphadenopathy. Smallpox, Measles, scabies, and chickenpox have similar symptoms during the incubation period, what differentiates monkeypox from others is the swelling of the lymph nodes called lymphadenopathy. Between 1 - 3 days of the onset of fever, maculopapular rashes appear with small fluid blisters that are pus-filled and crust over in

about ten days [21]. The rashes affect the face, palms of the hands, soles of the feet, oral mucous membranes, and sometimes the genitalia, conjunctivae, or cornea [15, 27, 12]. Like any other viral infection, monkeypox is a self-limited viral infection with symptoms lasting from 2 - 4 weeks. The severity of the infection is determined by the health status of the patient before the invasion, virus exposure, and the nature of the virus strand.

The present therapeutic method for monkeypox is clinical care, which is targeted squarely at symptom alleviation, management of possible complications, that includes encephalitis, pneumonia, etc, and prevention of long-term sequelae like keloid scars [31]. A healthy diet and fluid are used to maintain adequate nutritional status and regulation and optimality of internal organs. Vaccination against defeated smallpox has shown through a series of observational studies that it is 85% effective in the prevention of monkeypox disease except that it is no more accessible after the eradication of smallpox in 1980. There are other variants of vaccines going through trials and some countries have started administering modified attenuated vaccinia virus - Ankara strain which is a two dose vaccine but the availability remains limited; which means that most of the countries cannot access it at present [27, 12]. As a result of this gap, the most effective prevention strategy currently for monkeypox is constructed awareness of the risk factors and education on effective measures to curtail the exposure to the virus [7]. To contain the monkeypox outbreak, swift surveillance and rapid identification of the new cases are key; unprotected contact with wild animals especially sick or dead ones should be avoided and animal trading should also be monitored.

Mathematical models have been instrumental in understanding the dynamics of different diseases: HIV [23], Malaria [24], conjunctivitis [28] and tuberculosis [25] etc and other areas of endeavors like heat transfer [2], psychiatric [11], bio-medical engineering [1] and other countless fields of life. Considerable researchers have applied mathematical modeling in the study of transmission dynamics of monkeypox. The study [6] showed that with treatment intervention monkeypox will be eradicated from human and rodents population, [14] studied the transmission dynamics of monkeypox virus which they concluded that isolating infected individuals in the human population helps to reduce the disease transmission. Treatment and vaccination interventions were considered by [8] as an effective measure for the containment of monkeypox,

[16] incorporates impact vaccine on the human population as a means of boosting the immune system of the people, [26] modeled human to human transmission, [29] used game theory to model monkeypox vaccination assess strategies and likewise [4]. There is other priced work on monkeypox transmission but to the best of our knowledge, none has considered infection cautioned by infected individuals who are out of radar and surveillance subpopulation. United kingdom as of 12 July 2022 has a total of 1735 confirmed cases just between 6 May to 11 July of which most of the patients are traced to men without documented history of travel to endemic countries [30]. We aim to study the effect of effective surveillance in containing the transmission dynamics of monkeypox whether the suspected or probable case is symptomatic or asymptomatic; to buttress the importance of public health awareness on how the disease is transmitted, its symptoms, preventive measures, and action points when the infection is suspected or confirmed.

The remaining part of the paper is structured as follows: model formulation in section 2 and model analysis in section 3 which includes stability of the equilibrium points and the bifurcation analysis. Section 4 consists of the numerical results which include sensitivity analysis of the model parameters and numerical simulation and result of the model. Finally, in section 5, the discussion and conclusion are presented.

2. Model Formulation

A deterministic compartmental model is used to represent the transmission dynamics of monkeypox virus infection in two populations; human and host animals. The human population is partitioned into the susceptible human population, S_h, which are humans that have the potency of contacting MPXV when they come in contact with infected individual; exposed human population, E_h, are persons who have been in contact with at least an infected person either directly or indirectly; population of humans under surveillance, H_h, are individuals who have been exposed to MPXV and through contact tracing are under watch of health worker(s); Isolated infected human population, I_h, are individuals that have been proved to be infected by MPXV after medical diagnosis and have been isolated using standard procedure. Unidentified infected human population, I_u, are individuals who have been infected by MPXV with symptoms that

are outside radar; recovered human population, R_h, are individuals that were infected previously with Monkeypox virus but have recovered. The animal host population is sub-divided into Susceptible animals, S_a, infected animals, I_a and recovered animal population, R_a. The influx of humans into the population which is either by birth or immigration is at the rate Λ_h. The level of health awareness carried out within the population is represented with θ, while the probability of an individual being infected with the virus per contact with unidentified infected human and isolated infected human are β and β_1 respectively. β_2 is the product of effective contact rate and the probability of a human being infected by an infectious animal or its product. The proportion of the exposed individuals that the virus overcomes their immunity and hence making such a person infectious is ω, and the level of contact tracing implored in a community to isolate all infected humans is ρ. Human inherent recovery rate without any help from a health worker is k and γ when a health worker guides the infected human to recovery. The disease-induced death for unidentified infected human population is d_1 and the induced death rate for the isolated infected human which is predominantly children with a history of terminal disease is d_2. The proportion of the exposed that are under surveillance which is either passively, actively, or directly is represented as δ, under this condition, collection and dispatch of specimens for monkeypox laboratory examination is carried out. When the laboratory results are out, the rate of humans under surveillance that is negative to MPXV is τ, and they go back to the susceptible population. The rate of individuals whose diagnosis comes out positive is α, and these are isolated completely. The natural death rates of humans and non-human primates are μ and μ_a respectively. The recruitment of the animal host population is at the rate Λ, the rate of disease-induced death in the animal is d_3, the natural recovery rate of animals is b_a and a_a is the effective contact rate with the probability of an animal being infected per contact with an infected animal. Standard incidence function is used as the force of infection because monkeypox is a frequency dependent infection, then $\lambda = (1-\theta)\frac{\beta I_u + \beta_1 I_h + \beta_2 I_a}{N}$ where $N = S_h + E_h + H_h + I_h + I_u + R_h, N_a = S_a + I_a + R_a$ and all the state variables are function of time, t is in months/years. The schematic representation of the above assumptions and illustrations is shown in Figure 1.

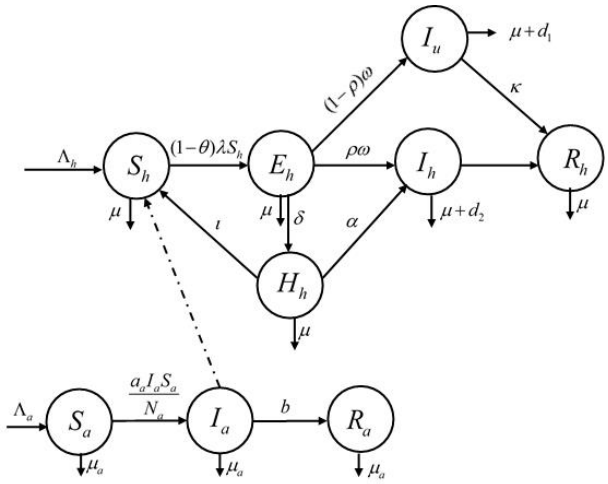

FIGURE 1. Schematic illustration of the model.

Taking into consideration the above assumptions, the model is governed by the following system of differential equations:

$$\left.\begin{aligned}
\frac{dS_h}{dt} &= \Lambda_h + \tau H_h - \lambda S_h - \mu S_h,\\
\frac{dE_h}{dt} &= \lambda S_h - (\omega + \delta + \mu)E_h,\\
\frac{dH_h}{dt} &= \delta E_h - (\tau + \alpha + \mu)H_h,\\
\frac{dI_h}{dt} &= \rho\omega E_h + \alpha H_h - (\gamma + d_2 + \mu)I_h,\\
\frac{dI_u}{dt} &= (1-\rho)\omega E_h - (\kappa + d_1 + \mu)I_u,\\
\frac{dR_h}{dt} &= \kappa I_u + \gamma I_h - \mu R_h,\\
\frac{dS_a}{dt} &= \Lambda_a - \frac{a_a I_a}{N_a} S_a - \mu_a S_a,\\
\frac{dI_a}{dt} &= \frac{a_a I_a}{N_a} S_a - (b + d_3 + \mu_a)I_a,\\
\frac{dR_a}{dt} &= bI_a - \mu_a R_a
\end{aligned}\right\}. \tag{2.1}$$

subject to the following initial conditions

(2.2)

$$S_h(0) > 0, E_h(0) \geq 0, H_h(0) \geq 0, I_h(0) \geq 0, I_u(0) \geq 0, R_h(0) \geq 0, S_a(0) \geq 0, I_a(0) \geq 0, R_a(0) \geq 0,$$

such that $N_h(0) \geq S_h(0) + E_h(0) + H_h(0) + I_h(0) + I_u(0) + R_h(0)$ and $N_a(0) \geq S_a(0) + I_a(0) + R_a(0)$. The epidemiological interpretation of all the parameters found in model (2.1) is given in Table 1; all the parameters are assumed to have non - negative numerical value(s).

TABLE 1. Epidemiological Interpretation of parameters involved in the model system (2.1).

Parameters	**Description**	**Value(s)**	**Source**
Λ_h	Influx of human population	321554	[5]
θ	Level of community awareness of monkeypox	$(0,1]$	Estimated
β	Transmission rate through contact with unidentified infected human	0.0008	Assumed
β_1	Transmission rate through contact with isolated infected human	0.00006	[10]
β_2	Transmission rate through contact with inflected animals	0.0027	[10]
ω	Proportion of exposed human that become infectious	0.095	[8]
ρ	Level of contact tracing of the infected human in the community	$(0,1]$	Estimated
κ	Human innate recovery rate with help form a health professional	0.83	[6]
d_1	Disease induced death rate for unidentified infected human	0.17	[15]
γ	Recovery rate of the isolated human	0.93	[10]
δ	Proportion of the exposed that are under surveillance	0.481	Assumed
τ	Proportion of human under surveillance that test negative after diagnosis	0.283	Assumed
α	Proportion of human under surveillance that test positive after diagnosis	0.172	Assumed
d_2	Disease induced death rate for the isolated human	0.05	[7]
μ	Natural death rate of human	0.01794	[5]
Λ_a	influx of animal	0.2	[10]
a_a	Animal to animal contact rate	0.027	[6]
b	Recovery rate of animal	0.6	[14]
d_3	Disease induced death of animal	0.4	[6]
μ_a	Natural death rate of animal	0.006	[14]

3. Model Analysis

3.1. Model Properties.

3.1.1. *Invariant Region.*

Theorem 3.1. *The feasible region* $\Omega = \Omega_h \times \Omega_a$ *with* $\Omega_h = \left\{S_h, E_h, H_h, I_h, I_u, R_h \in \mathbb{R}^6_+ : N_h \leq \frac{\Lambda_h}{\mu}\right\}$ *and* $\Omega_a = \left\{S_a, I_a, R_a \in \mathbb{R}^3_+ : N_a \leq \frac{\Lambda_a}{\mu_a}\right\}$ *is positively invariant set for model* (2.1).

Proof. By definition $N_h = S_h + E_h + H_h + I_h + I_+ R_h$, hence $\frac{dN_h}{dt} = \Lambda_h - \mu N_h - d_1 I_u - d_2 I_h \leq \mu N_h$ and $N_a = S_a + I_a + R_a$, then $\frac{dN_a}{dt} = \Lambda_a - \mu_a N_a - d_3 I_a \leq \Lambda_a - \mu_a N_a$. Hence we have that

$$\begin{cases} H_h(t) \leq \frac{\Lambda_h}{\mu} + \left(N_h(0) - \frac{\Lambda_h}{\mu}\right) e^{-\mu t}, \\ N_a(t) \leq \frac{\Lambda_a}{\mu_a} + \left(N_a(0) - \frac{\Lambda_a}{\mu_a}\right) e^{-\mu_a t}. \end{cases}$$

As $t \to \infty$, we have that $\limsup_{t\to\infty} N_h(t) \leq \dfrac{\Lambda_h}{\mu}$ and $\limsup_{t\to\infty} N_a(t) \leq \dfrac{\Lambda_a}{\mu_a}$. Hence Ω is a feasible solution set for the model (2.1), which implies that all solution sets of model (2.1) are bounded for $t \in [0, \infty)$. □

3.1.2. *Positivity of Solution.*

Theorem 3.2. *All solutions of model* (2.1) *remains non - negative for all* $t \in [0,\infty)$ *when* (2.2) *is satisfied.*

Proof. Let

$$W = \sup\{t > 0 : S_h(\hat{t}) \geq 0, E_h(\hat{t}) \geq 0, H_h(\hat{t}) \geq 0, I_h(\hat{t}) \geq 0, I_u(\hat{t}) \geq 0, R_h(\hat{t}) \geq 0,$$

$$S_a(\hat{t}) \geq 0, I_a(\hat{t}) \geq 0, R_a(\hat{t}) \geq 0, \forall\, \hat{t} \in [0,t]\}.$$

Clearly $W > 0$ and also $W < \infty$. From (2.1) we have that

$$\frac{dS_h}{dt} = \Lambda_h + \tau H_h - (\Lambda_h + \mu) S_h.$$

By method of integrating factor, we have that

$$\frac{d}{dt}\left[S_h(t) \exp\left\{\mu t + \int_0^t \lambda(\zeta) d\zeta\right\}\right] = (\Lambda_h + \tau H_h(t))\left[\exp\left\{\mu t + \int_0^t \lambda(\zeta) d\zeta\right\}\right]$$

$$\Rightarrow S_h(t_1) \exp\left\{\mu t + \int_0^t \lambda(\zeta) d\zeta\right\} - S_h(0) = \int_0^t (\Lambda_h + \tau H_h(y))\left[\exp\left\{\mu y + \int_0^y \lambda(\zeta) d\zeta\right\}\right] dy,$$

so that

$$S_h(t_1) = S_h(0)\exp\left\{-\mu t_1 - \int_0^{t_1}\lambda(\zeta)d\zeta\right\} + \left[\exp\left\{-\mu t_1 - \int_0^{t_1}\lambda(\zeta)d\zeta\right\}\right] \times \int_0^{t_1}(\Lambda_h + \tau H_h(y))\left(\exp\left\{\mu y + \int_0^{y}\lambda(\zeta)d\zeta\right\}\right)dy > 0.$$

Using the same approach on ther state variables in (2.1), we can easily show that

$$\begin{aligned} E_h'(t) &\geq -(\omega+\delta+\mu)E_h \Rightarrow E_h(t) \geq 0, \\ H_h'(t) &\geq -(\tau+\alpha+\mu)H_h \Rightarrow H_h(t) \geq 0, \\ I_h'(t) &\geq -(\gamma+d_2+\mu)I_h \Rightarrow I_h(t) \geq 0, \\ I_u'(t) &\geq -(\kappa+d_1+\mu)I_u \Rightarrow I_u(t) \geq 0, \\ R_h'(t) &\geq -\mu R_h \Rightarrow R_h(t) \geq 0. \end{aligned}$$

Also $\frac{dS_a}{dt} = \Lambda_a - (\lambda_a + \mu_a)S_a$, then

$$\frac{d}{dt}\left[S_a(t)\exp\left\{\mu_a t + \int_0^t \lambda_a(\zeta)d\zeta\right\}\right] = \Lambda_a \exp\left\{\mu_a t + \int_0^t \lambda_a(\zeta)d\zeta\right\}$$
$$\Rightarrow S_a(t_1)\exp\left\{\mu_a t + \int_0^t \lambda_a(\zeta)d\zeta\right\} - S_a(0) = \int_0^t \Lambda_a \exp\left\{\mu_a y + \int_0^y \lambda_a(\zeta)d\zeta\right\}dy,$$

so that

$$\begin{aligned} S_a(t_1) &= S_a(0)\exp\left\{-\mu_a t_1 - \textstyle\int_0^{t_1}\lambda_a(\zeta)d\zeta\right\} + \left[\exp\left\{-\mu_a t_1 - \textstyle\int_0^{t_1}\lambda_a(\zeta)d\zeta\right\}\right] \\ &\quad \times \textstyle\int_0^{t_1}\Lambda_a\left(\exp\left\{\mu_a y + \int_0^{y}\lambda_a(\zeta)d\zeta\right\}\right)dy > 0. \end{aligned}$$

Similarly, we can easily show that

$$I_a' \geq -(b+d_3+\mu_a)I_a \Rightarrow I_a(t) \geq 0, R_a' \geq -\mu_a R_a \Rightarrow R_a(t) \geq 0.$$

Therefore any solution of model (2.1) when (2.2) holds is non - negative for $t \in [0,\infty)$ and buttress the epidemiological meaningfulness. □

3.2. Model Equilibrium Points. At equilibrium, $\frac{dS_h}{dt} = \frac{dE_h}{dt} = \frac{dH_h}{dt} = \frac{dI_h}{dt} = \frac{dI_u}{dt} = \frac{dR_h}{dt} = \frac{dS_a}{dt} = \frac{dI_a}{dt} = \frac{dR_a}{dt} = 0$.

From this we obtain that

(3.1)

$$\left.\begin{aligned} S_h = \frac{k_1k_2\Lambda_h}{k_1k_2(\lambda+\mu)-\tau\delta\lambda},\ E_h = \frac{k_2\Lambda_h\lambda}{k_1k_2(\lambda+\mu)-\tau\delta\lambda},\ H_h = \frac{\delta\Lambda_h\lambda}{k_1k_2(\lambda+\mu)-\tau\delta\lambda},\\ I_h = \frac{(\rho\omega k_2+\alpha\delta)\Lambda_h\lambda}{k_3\Big(k_1k_2(\lambda+\mu)-\tau\delta\lambda\Big)},\ R_h = \frac{\lambda\Lambda_h\Big(\kappa(1-\rho)\omega k_2+\gamma(\rho\omega k_2+\alpha\delta)\Big)}{\mu(k_3+k_4)\Big(k_1k_2(\lambda+\mu)-\tau\delta\lambda\Big)},\ S_a = \frac{\Lambda_a}{\lambda_a+\mu_a},\\ I_u = \frac{(1-\rho)\omega k_2\Lambda_h\lambda}{k_4\Big(k_1k_2(\lambda+\mu)-\tau\delta\lambda\Big)},\ I_a = \frac{\lambda_a\Lambda_a}{k_5(\lambda_a+\mu_a)},\ R_a = \frac{b\lambda_a\Lambda_a}{k_5\mu_a(\lambda_a+\mu_a)}; \end{aligned}\right\}$$

where $k_1 = \omega+\delta+\mu$, $k_2 = \tau+\alpha+\mu$, $k_3 = \gamma+d_2+\mu$, $k_4 = \kappa+d_1+\mu$, $k_5 = b+d_3+\mu_a$ and $\lambda_a = \frac{a_aI_a}{N_a}$. From this steady state of the model we can observe the following types of equilibrium point:

(a) Monkeypox Free Equilibrium (MFE), E_0: This is a state where there are no infected persons or non-human primates in the community. In this regard $I_h(t) = I_u(t) = I_a(0) = 0$ and also $\lambda = \lambda_a = 0$ which means eradication of monkeypox pathogen that leads to reduction of the worldwide incidence of MPXV to zero by deliberate efforts, obviating the necessity for further control. The state of MFE is made possible by the following eradication strategies: an effective intervention that can truncate the transmission of monkeypox, practical and sensitive diagnostic kits that will be available with the ability to determine the level of infection that is transmissible, and public health awareness. The MFE of model (2.1) is obtain when $\lambda = \lambda_a = 0$ in the above steady-state to obtain

$$E_0 = (S_h^0, E_h^0, H_h^0, I_h^0, I_u^0, R_h^0, S_a^0, I_a^0, R_a^0) = \Big(\frac{\Lambda_h}{\mu}, 0, 0, 0, 0, 0, \frac{\Lambda_a}{\mu_a}, 0, 0\Big).$$

(b) Monkeypox Endemic Equilibrium (MEE), E_1: At this steady state, the force of infection $\lambda > 0, \lambda_a > 0$ which entails that the infection will persist in the community hence, there will be at least one of the infected human or infected reservoir that must pass it on to one other human or non - human pirate on average. The MEE of model (2.1) is given as

$$E_1 = (S_h^*, E_h^*, H_h^*, I_h^*, I_u^*, R_h^*, S_a^*, I_a^*, R_a^*)$$

where the state variables are defined as in (3.1). The MEE exists when $k_1k_2(\lambda+\mu) > \tau\delta\lambda, I_h^* \geq 1, I_u^* \geq 1$ and $I_a^* \geq 1$ at any given time.

3.3. Effective Reproduction Number, R_m. Reproduction number is a threshold used to study the prevalence and long-term behaviour of a given disease. It is the number of secondary cases that emanates from a single infected person in the course of its infectious stage. The next generation matrix as defined in [9] is used to compute the reproduction index which we term as an effective reproduction number because of the involvement of awareness and surveillance parameters used as controls in the model. Using this method, f_i is defined as the influx of the infection into the compartments and v_i is the transfer of infection into other compartments as the disease pathogen progresses. Hence

$$f_i = \begin{pmatrix} \frac{(1-\theta)(\beta I_u+\beta_1 I_h+\beta_2 I_a)}{N_h} S_h \\ 0 \\ 0 \\ 0 \\ \frac{a_a I_a}{N_a} S_a \end{pmatrix} \text{ and } v_i = \begin{pmatrix} k_1 E_h \\ -\delta E_h + k_2 H_h \\ -\rho\omega E_h - \alpha H_h + k_3 I_h \\ -(1-\rho)\omega E_h + k_4 I_u \\ k_5 I_a \end{pmatrix}.$$

Therefore, the transmission matrix F and transition V evaluated at E_0 is given as

$$F = \begin{pmatrix} 0 & 0 & (1-\theta)\beta_1 & (1-\theta)\beta & (1-\theta)\beta_2 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & a_a \end{pmatrix} \text{ and } V = \begin{pmatrix} k_1 & 0 & 0 & 0 & 0 \\ -\delta & k_2 & 0 & 0 & 0 \\ -\rho\omega & -\alpha & k_3 & 0 & 0 \\ -(1-\rho)\omega & 0 & 0 & k_4 & 0 \\ 0 & 0 & 0 & 0 & k_5 \end{pmatrix}$$

respectively. From [9], $R_m = \rho(FV^{-1})$, where ρ is the spectral radius. Then

$$V^{-1} = \frac{1}{k_1k_2k_3k_4k_5} \begin{pmatrix} k_2k_3k_4k_5 & 0 & 0 & 0 & 0 \\ \delta k_3k_4k_5 & k_1k_3k_4k_5 & 0 & 0 & 0 \\ k_2k_5(\alpha\delta + k_2\rho\omega) & \alpha_1k_1k_4k_5 & k_1k_2k_4k_5 & 0 & 0 \\ k_2k_3k_5\omega(1-\rho) & 0 & 0 & k_1k_2k_3k_5 & 0 \\ 0 & 0 & 0 & 0 & k_1k_2k_3k_4 \end{pmatrix}.$$

After further simplification, we obtain that

$$R_m = \rho(FV^{-1}) = \frac{(1-\theta)\Big[\beta_1(\alpha\delta + k_2\rho\omega) + \beta\omega(1-\rho)\Big]}{(\omega+\delta+\mu)(\gamma d_2\mu)(\kappa+d_2+\mu)}.$$

3.4. Stability of Monkeypox - Free Equilibrium.

Theorem 3.3. *Local stability of MFE: The monkeypox - free equilibrium is locally asymptotically stable when* $R_m < 1$ *and unstable when* $R_m > 1$.

Proof. Using the next generation matrix to obtain R_m and the linearization of (2.1) at E_0 taking (2.2) into consideration satisfies the five conditions in theorem 2 of [9]. Hence, the MFE is locally asymptotically stable whenever $R_m < 1$ but unstable if $R_m > 1$. The epidemiological interpretation is that monkeypox will be gradually eliminated from the population whenever $R_m < 1$ given that the initial size of the subpopulations is within the manifold of attraction Ω. By implication diminutive influx of monkeypox infectious individuals into the population will not loom large the possibility of a monkeypox outbreak, and the disease dies out in due time.

□

Theorem 3.4. *Global Stability of MFE: If* $R_m \leq 1$, *the MFE of the model* (2.1) *is globally asymptotically stable in* Ω.

Proof. Rewriting model (2.1) in the form

$$\frac{dX_1}{dt} = F_1(X_1, X_2), \frac{dX_2}{dt} = F_2(X_1, X_2) \text{with } F_2(X_1, 0) = 0,$$

where $X_1 = (S_h, R_h, S_a, R_a) \in \mathbb{R}^4$ the non - disease compartments and $X_2 = (E_h, H_h, I_h, I_u, I_a) \in \mathbb{R}^5$ is the latent/infected compartments. The following conditions must be satisfied for E_0 to be globally asymptotically stable:

l_1: $\frac{dX_1}{dt} = F_1(X_1, 0)$ if $F_1(E_0, 0)$ then E_0 is globally asymptotically stable.

l_2: $F_2(X_1, X_2) = RX_2 - \hat{F}_2(X_1, X_2), \hat{F}_2(X_1, X_2) > 0$ for $(X_1, X_2) \in \Omega$, where $R = D_{X_2}F_2(E_0, 0)$ is a M - matrix.

At MFE, we have

$$\frac{dX_1}{dt} = \begin{pmatrix} \Lambda_h - \mu S_h \\ 0 \end{pmatrix} \tag{3.2}$$

of which solution is $S_h(t) = \frac{\Lambda_h}{\mu} + \left(S_h(0) - \frac{\Lambda_h}{\mu}\right) e^{-\mu t}$ such that $\lim_{t\to\infty} S_h(t) = \dfrac{\Lambda_h}{\mu}$; which shows global convergence of (3.2) in Ω. From (2.1) we have

$$F_2(X_1,X_2) = \begin{pmatrix} -k_1 & 0 & (1-\theta)\beta_1 & (1-\theta)\beta & (1-\theta)\beta_2 \\ \delta & -k_2 & 0 & 0 & 0 \\ \rho\omega & \alpha & -k_3 & 0 & 0 \\ (1-\rho 0\omega & 0 & 0 & -k_4 & 0 \\ 0 & 0 & 0 & 0 & a_a - k_5 \end{pmatrix} \times \begin{pmatrix} E_h \\ H_h \\ I_h \\ I_u \\ I_a \end{pmatrix} - \begin{pmatrix} \frac{(1-\theta)(\beta I_u + \beta I_h \beta_2 I_a}{N_h} S_h \\ 0 \\ 0 \\ 0 \\ \frac{a_a I_a}{N_a} S_a \end{pmatrix}.$$

Clearly $\frac{S_h}{N_h} \geq 0$ and $\frac{S_a}{N_a} \geq 0$ which clearly lie within the manifold Ω and $\hat{F}_2(X_1,X_2) \geq 0$. Observe that R is actually a $M-$ matrix, hence conditions l_1 and l_2 are satisfied and is sufficient for the global asymptotic stability of E_0 when $R_m < 1$. □

3.5. Stability of the Monkeypox Endemic Equilibrium.

Theorem 3.5. *The system* (2.1) *has an endemic equilibrium, E_1 that is globally asymptotically stable whenever $R_m > 1$.*

Proof. Since $R_m > 1$, E_0 loses its stability and a unique endemic equilibrium emerges in Ω and is locally asymptotically stable. Using the method employed in [11] we show that model (2.1) has no limit cycles. Let the vector $M = (S_h, E_h, H_h, I_h, I_u, R_h, S_a, I_a, R_a)$, we define a Dulac's function $F = \frac{1}{E_h H_h}$. Then

$$F\frac{dS_h}{dt} = \frac{\Omega_h}{E_h H_h} + \frac{\tau}{E_h} - \frac{\lambda_h}{E_h H_h} S_h - \frac{\mu}{E_h H_h} S_h, F\frac{dE_h}{dt} = \frac{\lambda_h}{E_h H_h} S_h - \frac{k_1}{H_h},$$

$$F\frac{dH_h}{dt} = \frac{\delta}{H_h} - \frac{k_2}{E_h}, F\frac{dI_h}{dt} = \frac{(1-\rho)\omega}{H_h} + \frac{\alpha}{E_h} - \frac{k_3 I_h}{E_h H_h},$$

$$F\frac{dI_u}{dt} = \frac{\rho\omega}{H_h} - \frac{k_4}{E_h H_h} I_u, F\frac{dR_h}{dt} = \frac{\kappa I_u}{E_h H_h} + \frac{\gamma I_h}{E_h H_h} - \frac{\mu R_h}{E_h H_h},$$

$$F\frac{dS_a}{dt} = \frac{\Lambda_a}{E_h H_h} - \frac{\lambda_a}{E_h H_h} - \frac{\mu_a S_a}{E_h H_h}, F\frac{dI_a}{dt} = \frac{\lambda_a S_a}{E_h H_h} - \frac{k_5 I_a}{E_h H_h}, F\frac{dR_a}{dt} = \frac{bI_a}{E_h H_h} - \frac{\mu_a R_a}{E_h H_h}.$$

$$
\begin{aligned}
\therefore \quad & \frac{dFM}{dt} = \frac{\partial}{\partial S_h}\left(F\frac{dS_h}{dt}\right) + \frac{\partial}{\partial E_h}\left(F\frac{dE_h}{dt}\right) + \frac{\partial}{\partial H_h}\left(F\frac{dH_h}{dt}\right) + \frac{\partial}{\partial I_h}\left(F\frac{dI_h}{dt}\right) + \frac{\partial}{\partial I_u}\left(F\frac{dI_u}{dt}\right) \\
+ \quad & \frac{\partial}{\partial R_h}\left(F\frac{dR_h}{dt}\right) + \frac{\partial}{\partial S_a}\left(F\frac{dS_a}{dt}\right) + \frac{\partial}{\partial I_a}\left(F\frac{dI_a}{dt}\right) + \frac{\partial}{\partial R_a}\left(F\frac{dR_a}{dt}\right). \\
= \quad & \frac{\partial}{\partial S_h}\left(\frac{\Omega_h}{E_h H_h} + \frac{\tau}{E_h} - \frac{\lambda_h}{E_h H_h}S_h - \frac{\mu}{E_h H_h}S_h\right) + \frac{\partial}{\partial E_h}\left(\frac{\lambda_h}{E_h H_h}S_h - \frac{k_1}{H_h}\right) + \frac{\partial}{\partial H_h}\left(\frac{\delta}{H_h} - \frac{k_2}{E_h}\right) \\
+ \quad & \frac{\partial}{\partial I_h}\left(\frac{(1-\rho)\omega}{H_h} + \frac{\alpha}{E_h} - \frac{k_3 I_h}{E_h H_h}\right) + \frac{\partial}{\partial I_h}\left(\frac{\rho\omega}{H_h} - \frac{k_4}{E_h H_h}I_u\right) + \frac{\partial}{\partial R_h}\left(\frac{\kappa I_u}{E_h H_h} + \frac{\gamma I_h}{E_h H_h} - \frac{\mu R_h}{E_h H_h}\right) \\
+ \quad & \frac{\partial}{\partial S_a}\left(\frac{\Lambda_a}{E_h H_h} - \frac{\lambda_a}{E_h H_h} - \frac{\mu_a S_a}{E_h H_h}\right) + \frac{\partial}{\partial I_a}\left(\frac{\lambda_a S_a}{E_h H_h} - \frac{k_5 I_a}{E_h H_h}\right) + \frac{\partial}{\partial R_a}\left(\frac{bI_a}{E_h H_h} - \frac{\mu_a R_a}{E_h H_h}\right). \\
= \quad & -F\left[\frac{\lambda_h(E_h + H_h + I_h + I_u + R_h)}{N_h} + \frac{\lambda_h S_h}{E_h} + \frac{\lambda_a(I_a + R_a)}{N_a} + k_3 + k_4 + k_5 + 2(\mu + \mu_a)\right]. \\
< \quad & 0.
\end{aligned}
$$

$\frac{FM}{dt} < 0$ implies that E_1 will stay non-positive for all $t \in [0,\infty)$ and confined in Ω. Hence by Bendixson - Dulac criterion, there will be no limit cycle in Ω and E_1 is globally asymptotically stable. □

3.6. Bifurcation Analysis. To study the stability of the equilibrium points, it is needful to check for simultaneous and consecutive occurrence of the two existing steady states. To investigate the coexistence of the monkeypox - free equilibrium and endemic equilibrium we implore theorem 4.1 in [22] and center manifold theory. Assuming that p and q represent the dynamic of the center manifold such that $\beta^* = \beta_1$ is the bifurcation parameter with critical value at $R_m = 1$, then

$$\beta^* = \beta_1 = \frac{(\omega+\delta+\mu)(\gamma d_2+\mu)(\kappa+d_1+\mu) - \beta\omega(1-\theta)(1-\rho)}{(1-\theta)(\alpha\delta+\rho\omega(\tau+\alpha\mu))}.$$

Let $S_h = x_1, E_h = x_2, H_h = x_3, I_h = x_4, I_u = x_5, R_h = x_6, S_a = x_7, I_a = x_8, R_a = x_9$; if $V = (x_1,x_2,x_3,x_4,x_5,x_6,x_7,x_8)^T$, then model (2.1) can be transformed as $\frac{dV}{dt} = (f_1,f_2,f_3,f_4,f_5,f_6,f_7,f_8,f_9)^T$ where

$$
\left.\begin{aligned}
f_1 &= \Lambda_h + \tau x_3 - \tfrac{(1-\theta)(\beta x_5+\beta_1 x_4+\beta_2 x_8)}{\bar{N}_h} x_1 - \mu x_1,\\
f_2 &= \tfrac{(1-\theta)(\beta x_5+\beta_1 x_4+\beta_2 x_8)}{\bar{N}_h} x_1 - k_1 x_2,\\
f_3 &= \delta x_2 - k_2 x_3,\\
f_4 &= \rho\omega x_2 + \alpha x_3 - k_3 x_4,\\
f_5 &= (1-\rho)\omega x_2 - k_4 x_5,\\
f_6 &= \kappa x_5 + \gamma x_4 - \mu x_6,\\
f_7 &= \Lambda_a - \tfrac{a_a x_8}{\bar{N}_a} x_7 - \mu_a x_7,\\
f_8 &= \tfrac{a_a x_8}{\bar{N}_a} x_7 - k_5 x_8,\\
f_9 &= b x_8 - \mu_a x_9,
\end{aligned}\right\} \tag{3.3}
$$

where $\bar{N}_h = x_1+x_2+x_3+x_4+x_5+x_6, \bar{N}_a = x_7+x_8+x_9$. Linearizing system (3.3) around E_0 evaluated at β_1^* gives

$$
J_{E_0} = \begin{pmatrix}
-\mu & 0 & \tau & -(1-\theta)\beta^* & -(1-\theta)\beta & 0 & 0 & -(1-\theta)\beta_2 & 0\\
0 & -k_1 & 0 & (1-\theta)\beta^* & (1-\theta)\beta & 0 & 0 & (1-\theta)\beta_2 & 0\\
0 & \delta & -k_2 & 0 & 0 & 0 & 0 & 0 & 0\\
0 & \rho\omega & \alpha & -k_3 & 0 & 0 & 0 & 0 & 0\\
0 & (1-\rho)\omega & 0 & 0 & -k_4 & 0 & 0 & 0 & 0\\
0 & 0 & 0 & \gamma & \kappa & -\mu & 0 & 0 & 0\\
0 & 0 & 0 & 0 & 0 & 0 & -\mu_a & -a_a & 0\\
0 & 0 & 0 & 0 & 0 & 0 & 0 & -k_5 & 0\\
0 & 0 & 0 & 0 & 0 & 0 & 0 & b & -\mu_a
\end{pmatrix}.
$$

Observe that E_0 is a non - hyperbolic equilibrium, hence J_{E_0} has zero eigenvalue while others have negative real part. If $w = (w_1, w_2, w_3, w_4, w_5, w_6, w_7, w_8, w_9)^T$ is the eigenvector corresponding to zero eigenvalues, we have

$$
w_1 = \frac{\tau\delta k_3 k_4 - (1-\theta)\left(\kappa\beta^*(\rho\omega k_2 + \delta\alpha) + \beta k_2 k_3 \omega(1-\rho) + \beta_2 \delta k_3 k_4 w_8\right)}{\mu\delta k_3 k_4},
$$

$$
w_2 = \frac{k_2}{\delta}, w_3 = 1, w_4 \frac{\rho\omega k_2 + \delta\alpha}{\delta k_3}, w_5 = \frac{(1-\rho)\omega k_2}{\delta k_4},
$$

$$
w_6 = \frac{\gamma k_4(\rho\omega k_2 + \delta\alpha) + \kappa k_3}{\mu\delta k_3 k_4}, w_7 = -\frac{a_a}{\mu_a} w_8, w_8 > 0, w_9 \frac{b}{\mu_a} w_8.
$$

Let the associated left eigenvector that corresponds to the zero eigenvalue be
$v = (v_1, v_2, v_3, v_4, v_5, v_6, v_7, v_8, v_9)$ such that $v.w = 0$. Solving $J_{E_0} v = 0$ to obtain that
$v_1 = v_6 = v_7 = v_9 = 0$,

$$v_2 = \frac{k_3}{\beta^*(1-\theta)} v_4, v_3 \frac{\alpha}{k_2} v_4, v_4 > 0, v_5 = \left(\frac{k_2 k_3}{\beta^* \omega (1-\theta)(1-\rho)} - \frac{\alpha\delta + \rho\omega k_2}{k_2 \omega (1-\rho)} \right), v_8 = \frac{\beta_2 k_3}{\beta^* k_5}.$$

From theorem 4.1 in [22] we have that

$$p = \sum_{k,i,j=1}^{9} v_k w_i w_j \frac{\partial^2 f_k}{\partial x_i \partial x_j}(E_0, \beta^*) \text{and } q = \sum_{k,i=1}^{9} v_k w_i \frac{\partial^2 f_k}{\partial x_i \partial \beta_1}(E_0, \beta^*).$$

Since $v_1 = v_6 = v_7 = v_9 = 0$, the second order partial derivative of f_1, f_6, f_7 and f_9 will give zero, hence we obtain the second - order partial derivatives of f_2, f_3, f_4, f_5, and f_8. We have that

$$\frac{\partial^2 f_2}{\partial x_1 \partial x_4} = \frac{\partial^2 f_2}{\partial x_4 \partial x_1} = (1-\theta)\beta_1 \left(\frac{\mu}{\Lambda_h} - 1 \right), \frac{\partial^2 f_2}{\partial x_1 \partial x_5} = \frac{\partial^2 f_2}{\partial x_5 \partial x_1} = (1-\theta)\beta \left(\frac{\mu}{\Lambda_h} - 1 \right),$$
$$\frac{\partial^2 f_2}{\partial x_1 \partial x_8} = \frac{\partial^2 f_2}{\partial x_8 \partial x_1} = (1-\theta)\beta_2 \left(\frac{\mu}{\Lambda_h} - 1 \right), \frac{\partial^2 f_8}{\partial x_7 \partial x_8} = \frac{\partial^2 f_8}{\partial x_8 \partial x_7} = \alpha_2 \left(\frac{\mu_a}{\Lambda_a} - 1 \right),$$

and $\frac{\partial^2 f_2}{\partial x_4 \partial \beta_1} = 1 - \theta$, all the other second - order derivatives gives a zero. Therefore,
$p = 2v_2 w_1 (1-\theta) \left(\frac{\mu}{\Lambda_h} - 1 \right) \left(w_4 \beta_1 + w_5 \beta + w_8 \beta_2 \right) + 2v_8 w_7 w_8 a_2 \left(\frac{\mu_a}{\Lambda_a} - 1 \right)$
and $q = v_2 w_4 (1-\theta) > 0$.

Observe that $w_1 < 0$ and $\frac{\mu}{\Lambda_h - 1} < 0$, then $p > 0$ at $\beta^* = \beta_1$. From theorem 4.1 in [22], model (2.1) exhibits backward bifurcation at $R_m = 1$ since $p > 0$ and $q > 0$. Epidemiologically, it means that factors that help in making $R_m < 1$ are only necessary conditions but not sufficient for eradicating monkeypox in the community. Monkeypox occurs predominantly in remote villages of central and West Africa close to tropical rain forests which mounts a challenge of how to reduce the burden of the viral disease. As a result, the endemic equilibrium co-exists with the monkeypox-free equilibrium when $R_m < 1$ which explains why the viral infection resurfaces in various countries when it appears that it has been eradicated.

4. Numerical Analysis

4.1. Sensitivity Analysis. Studying the transmission dynamics of monkeypox, it is observed that it has a reemergence tendency because it is a zoonotic viral disease that the main reservoir is yet to be identified and humans come in contact with animals daily either in the form of food,

pet or game. We apply a normalized forward sensitivity index to the reproduction number to identify the parameter that is sensitive to the transmission and prevalence of the viral infection. The elasticity sensitivity index of R_m concerning the parameter p_i is given by

$$\xi_{p_i}^{R_m} = \frac{\partial R_m}{\partial p_i} \times \frac{p_i}{R_m}.$$

Hence

$$\xi_{\beta_1}^{R_m} = \frac{\beta_1(\alpha\delta+\rho\omega(\tau+\alpha+\mu))}{\beta_1(\alpha\delta+\rho\omega(\tau+\alpha+\mu))+\beta\omega(1-\rho)}, \xi_{\beta}^{R_m} = \frac{\beta\omega(1-\rho)}{\beta_1(\alpha\delta+\rho\omega(\tau+\alpha+\mu))+\beta\omega(1-\rho)},$$

$$\xi_{\delta}^{R_m} = -\frac{\delta\{\beta_1(\alpha\delta+\rho\omega(\tau+\alpha+\mu))+\beta\omega(1-\rho)\alpha\beta_1(\omega+\delta+\mu)\}}{(\omega+\delta+\mu)(\beta_1(\alpha\delta+\rho\omega(\tau+\alpha+\mu))+\beta\omega(1-\rho))},$$

$$\xi_{\omega}^{R_m} = -\frac{\omega(1-\theta)\{\beta_1(\alpha\delta+\rho\omega(\tau+\alpha+\mu))+\beta\omega(1-\rho)-(\omega+\delta+\mu)(\beta_1\rho(\tau+\alpha+\mu)+\beta(1-\rho))\}}{(\omega+\delta+\mu)(\beta_1(\alpha\delta+\rho\omega(\tau+\alpha+\mu))+\beta\omega(1-\rho))},$$

$$\xi_{\rho}^{R_m} = \frac{\rho\omega(\beta_1(\tau+\alpha+\mu)-\beta)}{\beta_1(\alpha\delta+\rho\omega(\tau+\alpha+\mu))+\beta\omega(1-\rho)}, \xi_{\alpha}^{R_m} = \frac{\alpha\beta_1(\delta+\rho\omega}{\beta_1(\alpha\delta+\rho\omega(\tau+\alpha+\mu))+\beta\omega(1-\rho)},$$

$$\xi_{\tau}^{R_m} = \frac{(1-\theta)\beta_1\rho\omega}{(\omega+\delta+\mu)(\gamma+d_2+\mu)(\kappa+d_1+\mu)}, \xi_{\theta}^{R_m} = -\frac{\theta}{1-\theta}.$$

The sensitivity index of our model is given below (Table 2)

TABLE 2. Sensitivity index of parameters in the effective reproduction number.

Parameters	**Value**
$\xi_{\beta_1}^{R_m}$	0.9993
$\xi_{\beta}^{R_m}$	11.586
$\xi_{\theta}^{R_m}$	-1.1739
$\xi_{\delta}^{R_m}$	-9.2783
$\xi_{\rho}^{R_m}$	-2.3214
$\xi_{\alpha}^{R_m}$	0.9450
$\xi_{\omega}^{R_m}$	4.4429
$\xi_{\tau}^{R_m}$	7.47×10^{-7}.

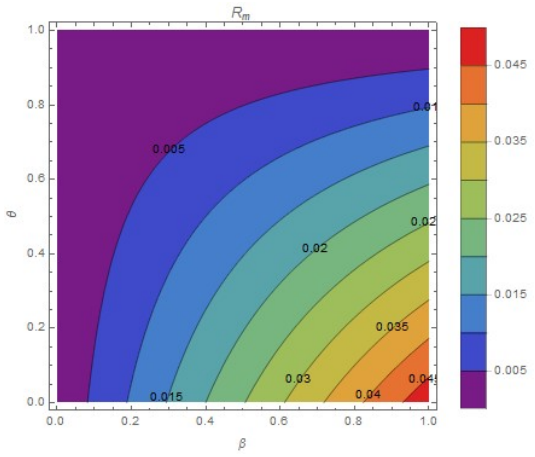

FIGURE 2. Contour plots of R_m versus transmission rate through the unidentified infected population and coordinated awareness on MPXV.

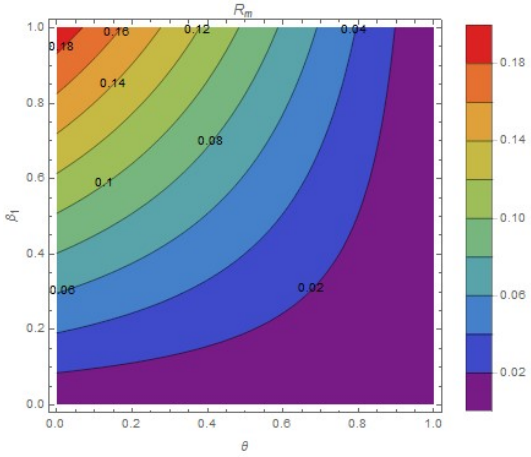

FIGURE 3. Contour plots of $R_m$versus transmission rate through the isolated infected population and coordinated awareness on MPXV.

Parameters with positive indices have a huge impact on the transmission and prevalence of monkeypox in a society, i.e. an increase in such a parameter increases the multiplication of MPXV cases in society while the parameters with negative indices have a minimizing effect on the burden of MPXV in the society as their values increase; thus, they are used in the control of the spread of MPX within the community. Using contour plots, we investigate the effect of the parameters in the model (2.1) on the effective reproduction number R_m, which we used as a divorce threshold in society. Minimization of the burden of MPXV in any community starts with intensified public awareness through risk communication on risk factors and measures to be taken to reduce exposure to *Orthopoxvirus*. It is observed in Figure 2 that when public awareness is intensified, coordinated, and carried out strictly; the transmission rate through unidentified infected persons will be reduced drastically because the community will be aware of all the flag-offs of MPXV which leads to proper guidance individually and collectively. Awareness also reduces the transmission through the isolated infected population as shown in Figure 3 because health workers and household members will apply standard infection control precautions in handling patients and the specimen that emanates from them. The awareness will also be

extended to how humans come in contact with animals especially the sick or dead ones, meat that is not thoroughly cooked, blood, and other lesions because most human infections is a s a result of animals.

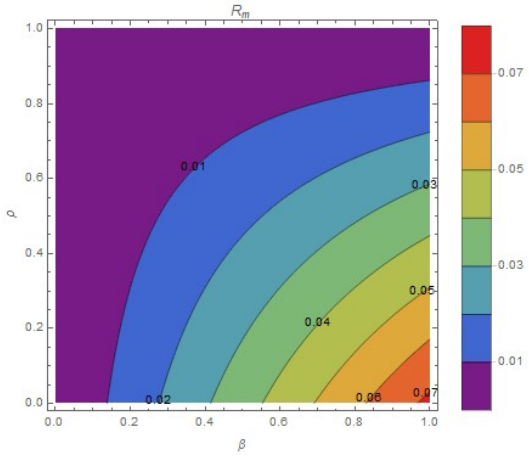

FIGURE 4. Contour plots of R_m versus transmission rate through the unidentified infected population and level of coordinated case investigation with contact tracing.

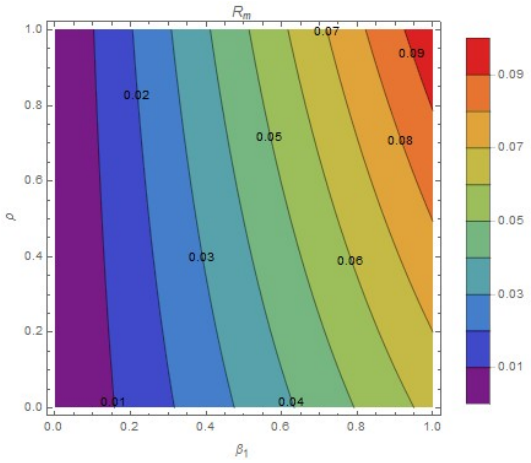

FIGURE 5. Contour plots of R_m versus transmission rate through the isolated infected population and level of coordinated case investigation with contact tracing.

As a control measure, the main objective of surveillance and case investigation is to swiftly identify cases and clusters to provide optimal clinical care; to isolate cases to avert further transmission; to identify, manage and follow up contacts to be able to recognize early signs of infection; to protect front-line health workers; to identify risk groups; to tailor effective control and prevention measures [15]. Figure 4 shows that increase of contact tracing, ρ; reduces the transmission rate cautioned by infected persons outside the radar and hence reduces the reproduction number. Case investigation will also help in containing the transmission of MPXV by increasing the number of patients that will be isolated and given clinical care as shown in Figure 5.

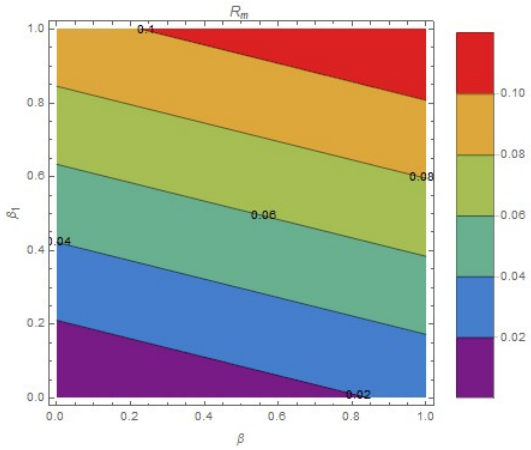

FIGURE 6. Contour plots of R_m versus transmission rate through the unidentified infected population and level of exposed that become infectious.

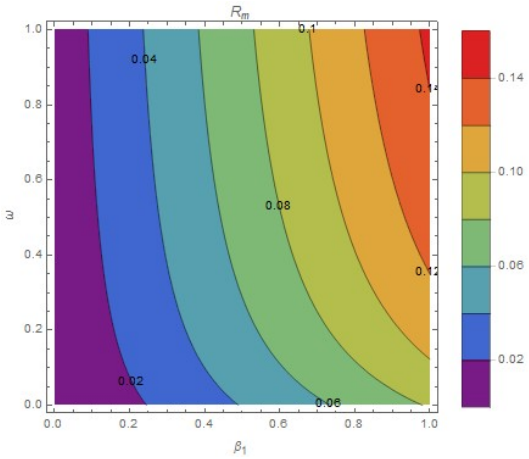

FIGURE 7. Contour plots of R_m versus transmission rate through the isolated infected population and level of exposed that become infectious.

As case investigation and contact tracing are amplified, the human-to-human transmission will be reduced, and hence exposed individuals will be under surveillance regardless of associated symptoms of their absence, in other to categorize pre-symptomatic, pauci - symptomatic or asymptomatic infection as shown in Figure 6 and 7. In Figure 8 and 9 it is observed that when a greater population of the exposed individuals is under surveillance, the reproduction number reduces considerably which is a green - flag that the epidemic will be contained and eradicated in due time.

When these control measures are not in place, the transmission rate caused by unidentified infected individuals will make the containment of the infection difficult as shown in Figure 9. Figure 10 shows that to reduce MPXV transmission human - to - human contact through skin or mucocutaneous lesions, respiratory droplets, and indirect contact with contaminated objects or materials; especially with a sick person should be reduced generally by applying the preventive measures as advised by WHO. Regardless that transmission of monkeypox is more pronounced among isolated patients and under surveillance, care should be applied when nursing any sick one, either as a health worker or household member.

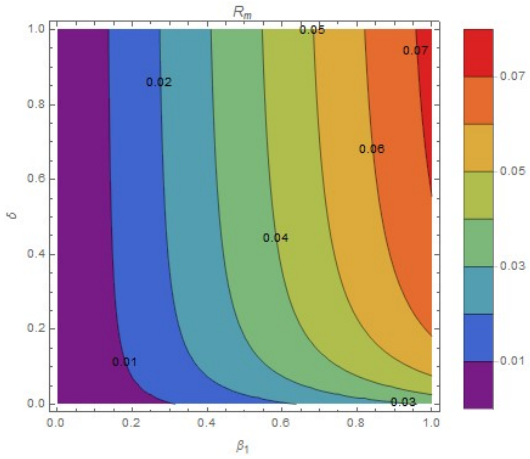

FIGURE 8. Contour plots of R_m versus transmission rate through the isolated infected population and level of exposed that are under surveillance

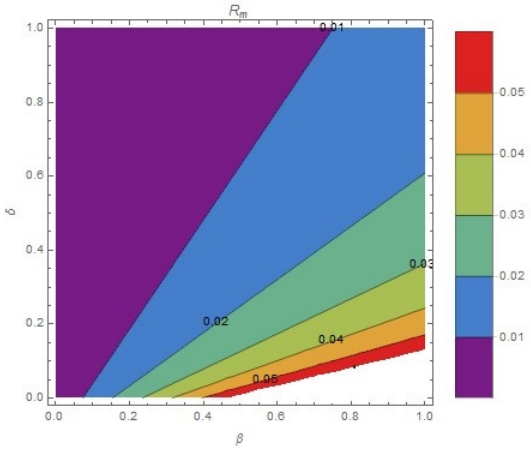

FIGURE 9. Contour plots of $R_m$versus transmission rate through the unidentified infected population and level of exposed that are under surveillance.

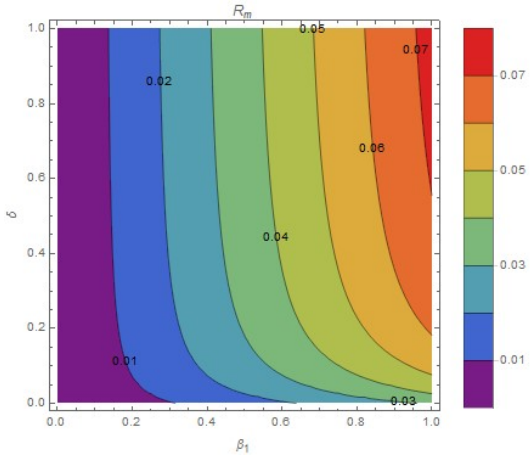

FIGURE 10. Contour plots of R_m versus transmission rate through the isolated infected population and unidentified infected population.

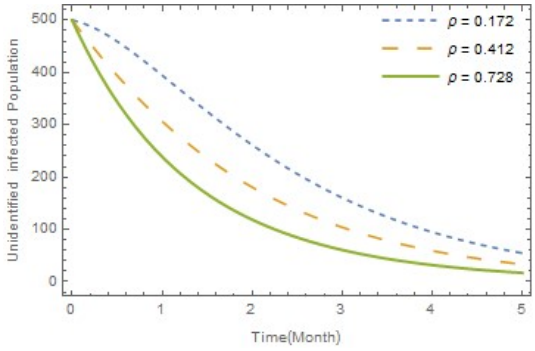

FIGURE 11. Unidentified infected population under varying value of ρ, level of contact tracing.

4.2. Numerical Simulation. In this section, the parameters of the model (2.1) are considered using secondary data and numerical simulation performed to support the analytical results. The values of parameters used in the simulation is shown in Table 1 with some estimated, assumed, calculated and obtained from literature, initial conditions used is follows: $S_h(0) = 8000, E_h(0) = 5000, H_h(0) = 3000, I_h(0) = 1500, I_u(0) = 500, R_h(0) = 800, S_a(0) = 250, I_a(0) = 75$and $R_a(0) = 50$. The value of the effective reproduction number obtained is $R_m = 0.000126$ which means that the viral infection will be dwindling over time if the controls are adhered to and possibly eradicated. During case investigation and contact tracing, clinical examination of the patients should be conducted using appropriate personal protective equipment, questioning the patient on suspected sources of infection and safe collection and dispatch of specimens for monkeypox virus laboratory examination. This process when carried out dutifully will reduce the population of the unidentified infected individuals as shown in Figure 11, thereby localizing the transmission which can be easily contained; in addition, it will increase the population of the isolated infected patients' population as shown in Figure 12 where proper clinical care will be given to the patients.

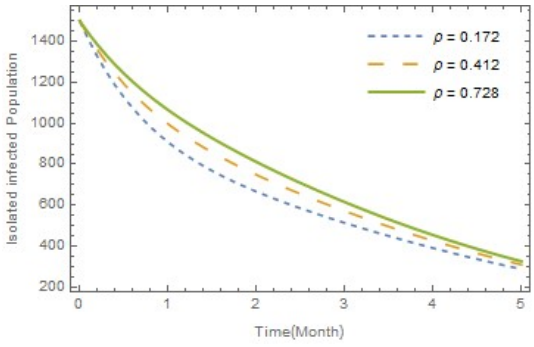

FIGURE 12. Isolated infected population under varying value of ρ, level of contact tracing

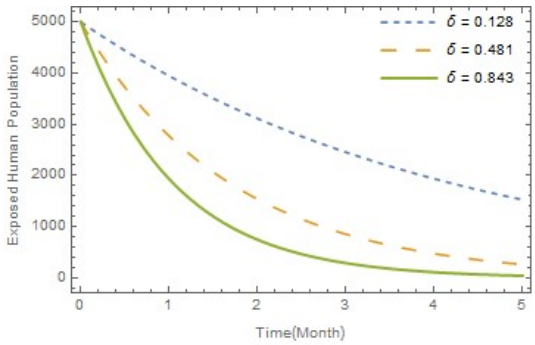

FIGURE 13. Exposed human population under varying value of δ, level of surveillance.

Coordinated surveillance makes sure that any contact with animal or human confirmed to have MPXV is under surveillance for a minimum of 21 days from the day of exposure, hence increase in the surveillance activities reduces the population of the exposed as shown in Figure 13; which means that the population of the isolated patients under clinical watch will increase

as shown in Figure 14 and population of the unidentified infected patients will reduce as shown in Figure 15.

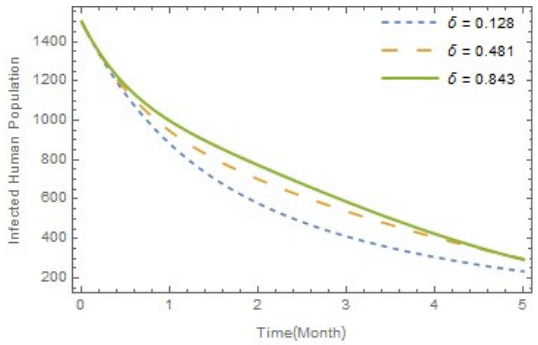

FIGURE 14. Isolated infected human population under varying value of δ, level of surveillance.

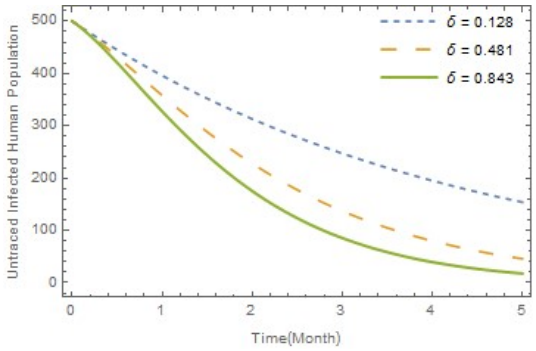

FIGURE 15. Unidentified infected human population under varying value of δ, level of surveillance.

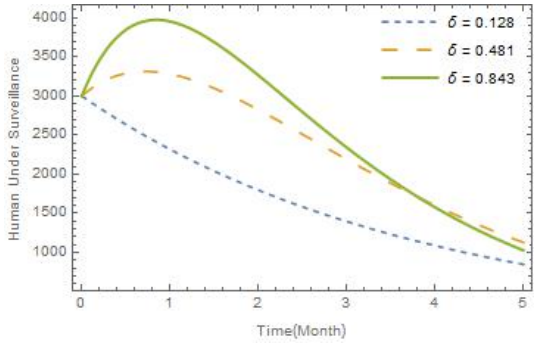

FIGURE 16. Isolated infected human population under varying value of δ, level of surveillance.

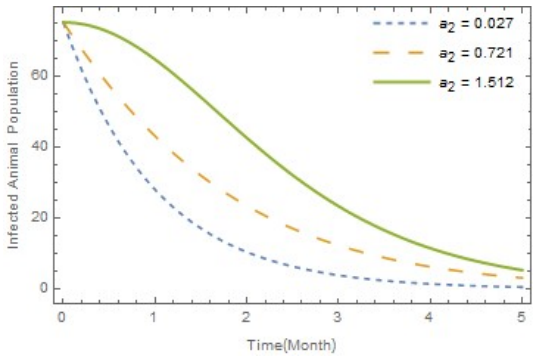

FIGURE 17. Unidentified infected human population under varying value of δ, level of surveillance.

An increase in the level of surveillance will consequently lead to an upsurge of coordinated public awareness, case investigation, and contact tracing which will accidentally increase the population under surveillance, and hence the eradication of monkeypox in any community will be possible as shown in Figure 16. Since the monkeypox vaccine is still in the developmental stage in most countries, therefore proper surveillance and case investigation which will lead to early diagnosis and isolation are the most effective means of containing the outbreak of

monkeypox at present. In addition to this control, strategic caution should be taken on how humans come in contact with animals without protection, and awareness should be directed to abattoirs, slaughters, herdsmen, and any other person that deals directly with animals on the outbreak and the need to be always vigilant of symptoms of monkeypox. Figure 17 shows that actions should be taken to reduce the number of infected animals in the community because they are the primary reservoir of which the main host is yet to be discovered.

5. CONCLUSION

A deterministic mathematical model was developed to understand the role of surveillance in the containment of the transmission dynamics of the monkeypox virus. The model consists of nine mutually exclusive compartments categorized into two subpopulations: Human and non-human primates or animals. Surveillance/case investigation and public awareness of monkeypox are deployed as strategies for endemic containment. The epidemiological and mathematical correctness of the model was established, thereafter, the equilibrium points of the model were obtained and their stability analyzed. We also showed that when monkeypox is endemic, the system exhibits backward bifurcation of which we showed the condition for this existence. Furthermore, a numerical analysis was conducted in which we used the standard sensitivity index to study the sensitivity of model parameters using the effective reproduction number as a threshold. We obtained that contact with infected humans who are out of the radar is what sustains the burden of MPXV but with coordinated contact tracing and awareness it can be contained because of the absence of a perfect vaccine at present. Numerical simulation was carried out to underscore the role of monkeypox awareness, surveillance, and contact tracing in the containment of the transmission dynamics of MPXV. Conclusively, proper surveillance and case investigation comprising of early diagnosis, isolation, clinical care, and contact tracing are strategic for effective control of the monkeypox outbreak at present.

AUTHOR CONTRIBUTIONS

All authors contributed equally to the writing of this paper. All authors read and approved the final manuscript.

CONFLICT OF INTERESTS

The authors declare that there is no conflict of interests.

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