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OPTIMIZING VACCINATION STRATEGIES TO REDUCE CONJUNCTIVITIS TRANSMISSION: MATHEMATICAL MODELING INSIGHTS FROM KENYA

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ABSTRACT

Conjunctivitis is a widespread condition with significant public health implications, but its potential impact on transmission patterns due to vaccination programs, particularly in Kenya, remains underexplored. The main aims of this study to investigate the role of vaccination in preventing conjunctivitis spread and related complications. A deterministic mathematical model was developed in an attempt to simulate conjunctivitis incidence, considering factors like population size, contact rates, and vaccination efficacy. The basic reproduction number (R_0) was calculated using the next-generation matrix method. Stability analysis of the disease-free equilibrium (DFE) showed stability will occur when $R_0 < 1$ and instability when $R_0 > 1$. Numerical computations using the MATLAB ode45 solver indicated that increased vaccination campaigns reduce the infected population. This implies that vaccination strengthens the immune response against the infection, lowering the risk of severe outcomes like vision loss. This study is vital for understanding the potential impact of effective vaccination programs on conjunctivitis transmission in Kenya, aiding policy-makers and public health practitioners in developing effective disease control measures.

Keywords: Conjunctivitis, Mathematical Modeling, Next Generation Matrix, Transmission Dynamics, Vaccination Campaigns.

I. INTRODUCTION

Pink eye, also known as conjunctivitis, is an inflammation of the conjunctiva. The conjunctiva is a thin, transparent tissue that covers the inner surface of the eyelid and the white section of the eye. It is a highly infectious disease that causes redness, irritation, abundant weeping, and a yellow discharge. Bacterial and viral infections are common causes of pink eye, but it can also be caused by allergies and irritants, as noted by [1]. Several bacteria may lead to bacterial conjunctivitis: *Streptococcus pneumoniae*, *Haemophilus influenzae*, and *Staphylococcus aureus*.

But this type of conjunctivitis often does not have a latent phase in an infectious way and usually shows symptoms quite fast after being around those bacteria, like yellow or green stuff coming out, eyelids getting crusty, and feeling uncomfortable [2,3].

For that purpose, to effectively prevent and control bacterial conjunctivitis, it is crucial to understand how it spreads. Vaccine activities have played an essential part in reducing the global load of infectious diseases [4]. By considering conjunctivitis spreading in Kenya, beginning a vaccine campaign has the potential to significantly decrease the disease's spread. Mathematical models are a powerful tool to check the potential impacts of these vaccine efforts on disease spreading patterns.

Mathematical models, which involve features such as how many people get vaccinated, how well the vaccines work, and population demos, can give useful thoughts about how vaccination might affect conjunctivitis spreading in Kenya. Numerous research areas have used math modeling to better grasp how bacterial conjunctivitis gets passed on and to evaluate ways to stop it. For example, [5] checked if closing schools could prevent bacterial conjunctivitis from spreading among kids. [1] studied how rotavirus vaccines affect epidemiology to stop bacterial conjunctivitis outbreaks in healthcare areas. Moreover, [6] looked at how environmental factors affect the spreading of bacterial conjunctivitis, while [4] tried to find the best ways to vaccinate people to stop infections from happening again.

Our objective in this study is to make available better research by using mathematical models to improve vaccination plans to lessen bacterial conjunctivitis spreading in Kenya based on weather stuff like temp and humidity impacts. We aim to create vaccine tech to stop conjunctivitis from passing on in Kenya. We check into mass vaccinations, where all folks in an area get vaccines, and create the right conditions and resources so people can get vaccines in a certain time frame.

We shall check if lots of existing mathematical models work well in predicting how a disease moves on and how different vaccination schedules can protect the area from getting sick, before using the model for conjunctivitis in Kenya. Specifically, we shall use the plan given by [7], thinking about how education efforts affect the results including vaccines, to see how different vaccination efforts can impact how many people get sick from bacterial conjunctivitis, focusing on Kenya.

This article will be structured as follows: In Section 1, we will briefly express bacterial conjunctivitis and how it spreads. After that, in Section 2, we will share a mathematical model that keeps in mind how the vaccine plans and its features affect things. Section 3 will use some differential equations to show how the disease sticks around, figure out an important number that shows how fast it can spread, and check if our model makes sense. Section 4 will show what our computer tests say, which will support our original thoughts. In Section 5, we will think about what all this means for vaccine plans in Kenya. Lastly, in Section 6, we shall wrap up our study's conclusions.

II. METHODOLOGY

In this phase, a mathematical model is developed and qualitative analysis is done. Our model is based on the assumptions that human population does not change, with the birth rate equals to the mortality rate. This assumption ensure that the overall population remain somewhat steady during our inquiry, setting the foundation to investigate the relationships between susceptibility, infection, and recovery.

In this SIR model, the populations split into three compartments: susceptibly (S), infected (I), and recovered (R). This separation allows us to oversee transitions between these compartments, exposing the complicated dynamics of illness transmission and recovery in our mathematical world.

Additionally, we ameliorate our model by fusing vaccination (v) dynamics and rainfall ($g(T)$) into our framework. Vaccination serves as a crucial intervention tactic, impacting individual susceptibility and disease spread. Altitude, represented by the function $g(T)$, introduces spatial heterogeneity, reflecting environmental conditions that may affect disease dynamics.

The model is designed based on the following assumptions;

- a) All individuals are susceptible to conjunctivitis.
- b) Conjunctivitis recovered individual evolve immunity and cannot catch Conjunctivitis again.
- c) The recovered (R) populations cannot pass away of Conjunctivitis due to the bacteria load has been suppressed.

Table 1: The considered State variables

S	Susceptible population
I	Conjunctivitis affected population
R	Conjunctivitis recovered population

Total population, $N = S + I + R$

Table 2: Used parameters

π	Recruitment rate into the susceptible population
β	Incidence of conjunctivitis in any individual
μ	Natural mortality rate of human population
v	Vaccination campaign Effectiveness

$g(T)$	Measuring rainfall during wet and dry seasons
γ	Recovery rate of infected human populations,

Diagram of three population groups and important parameters showing the model Dynamics of conjunctivitis, as shown in Fig. 1 below.

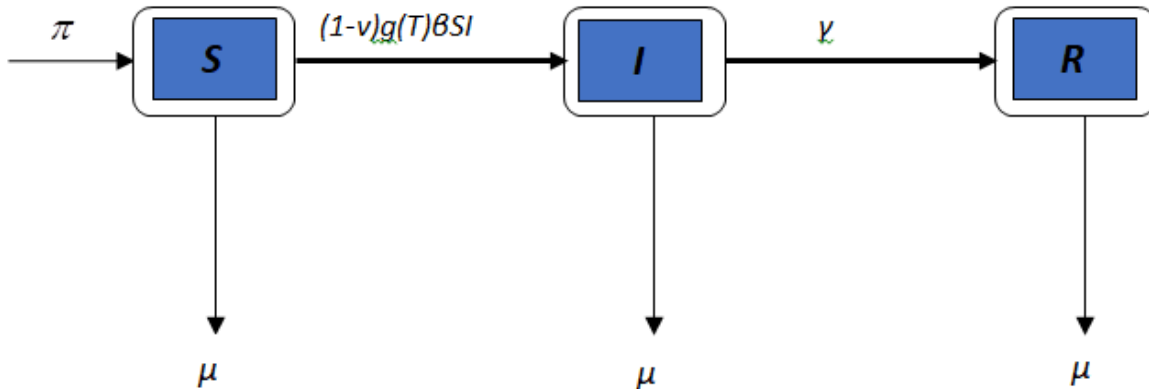


Figure 1: The flow diagram of the model of Conjunctivitis transmission dynamics

The Model representing transmission dynamics consists of a system of nonlinear differential equations given as.

$$\left. \begin{aligned} \frac{dS}{dt} &= \pi - \mu S - (1-v)g(T)\beta SI \\ \frac{dI}{dt} &= (1-v)g(T)\beta SI - (\mu + \gamma)I \\ \frac{dR}{dt} &= \gamma I - \mu R \end{aligned} \right\} \quad (1)$$

All parameters lie between 0 and 1, that is, $0 < \pi, \beta, \mu, v, g(T), \gamma < 1$.

III. MODELING AND ANALYSIS

Positivity of the solutions

States that the total population N can be expressed by the following equation $N = S + I + R$ with the following time derivatives

$$\begin{aligned} \frac{dN}{dt} &= \frac{dS}{dt} + \frac{dI}{dt} + \frac{dR}{dt} \\ \frac{dN}{dt} &= \pi - \mu(S + I + R) \leq \pi - \mu N \end{aligned} \quad (2)$$

$\Rightarrow \frac{dN}{dt} = 0$, this means the population is constant as per our assumption

$\varphi = \left(S, I, R \in R_+^3 \mid N(t) \leq \frac{\pi}{\mu} \right)$, which is both bounded and positively invariant.

By employing the technique of separating variables for integration and comparison theorem, as time approaches infinity ($t \rightarrow \infty$), the total population $N(t)$ tends towards $\frac{\pi}{\mu}$. Consequently, the system solutions

(1) enters and stays in the feasible region φ ,

$\varphi = \left(S, I, R \in R_+^3 \mid N(t) \leq \frac{\pi}{\mu} \right)$, which is both bounded and positively invariant.

Disease-free equilibrium (DFE) and local epidemics points(EDE)

The points of equilibrium are going to be studied and analyzed using our model's standard way of analyzing. The system has two equilibrium points which are: disease-free equilibrium (DFE) along with disease equilibrium points (EDE).

The DFE occurs when there are no infected individuals in the population, with the susceptible folks remaining stable. This balance means a balanced state where no disease is all about, no transmission is happening actively and everyone's staying disease-free presumably with no conjunctivitis infections around. It's achieved by setting;

$I = R = 0$ and from system (1) we obtain $S = N$ and the $DFE = (S^0, I^0, R^0)$

Thus, we have DFE as $E_0 = \left(\frac{\pi}{\mu}, 0, 0 \right)$.

Differing from the absence of disease in DFE, the Disease-Free Equilibrium depicts a situation where the disease is present consistently at a fairly steady level. In the situation of the disease being present, is the EDE that is; $I^* > 0$ the EDE $E_1 = (S^*, I^*, R^*)$ which is obtained by solving the system (3) below

$$\left. \begin{aligned} \pi - \mu S^* - (1 - \nu)g(T)\beta S^* I^* &= 0 \\ (1 - \nu)g(T)\beta S^* I^* - (\mu + \gamma)I^* &= 0 \\ \gamma I^* - \mu R^* &= 0 \end{aligned} \right\} \tag{3}$$

The EDE, $E_1 = (S^*, I^*, R^*)$ is implicitly obtained from the system such as (3) in terms of I^* ;

$$S^* = \frac{\mu + \gamma}{(1 - \nu)g(T)\beta}, I^* = \frac{(1 - \nu)g(T)\beta\pi N - \mu(\mu + \gamma)}{(1 - \nu)g(T)\beta(\mu + \gamma)}, R^* = \frac{\gamma I^*}{\mu} \tag{4}$$

Basic reproduction number

The fundamental epidemiological concept of basic reproduction number represents the average number of infected individuals produced by an infection in a fully population that is susceptible. [3]. It acts as a key indicator of the spread ability and potential of an infectious disease to spread among a population.

The subsequent-generation matrix, introduced by [4], is a mathematical framework used to analyze the dynamicity of infectious diseases in compartmentalized models. It represents the transmission of infections from one group of individuals (e.g., susceptible) to another (e.g., infectious) and plays a crucial role in determining the fundamental reproduction number and the disease equilibria stabilities. Mathematically, it is defined as the most prominent eigenvalue of the next-generation matrix, which embodies the transmission frequencies from various compartments in an epidemiological model.

From the equations shown above, the system can be put in the form of a matrix form.

$$x = \begin{bmatrix} S \\ I \\ R \end{bmatrix} \text{ and } \frac{dx}{dt} = F(x) - V(x) = \begin{bmatrix} \pi - \mu S - (1 - \nu)g(T)\beta SI \\ (1 - \nu)g(T)\beta SI - (\mu + \gamma)I \\ \gamma I - \mu R \end{bmatrix}$$

$$F(x) = \begin{bmatrix} 0 \\ (1 - \nu)g(T)\beta SI \\ 0 \end{bmatrix} \text{ and } V(x) = \begin{bmatrix} -\pi + \mu S + (1 - \nu)g(T)\beta SI \\ (\mu + \gamma)I \\ -\gamma I + \mu R \end{bmatrix}$$

The reproduction number is evaluated by next generation matrix method where F_i and V_i is defined as a Jacobian matrix that represents the new transmission rate and the transition rate of individuals out of the infected compartment, respectively.

$$F_i = \begin{bmatrix} 0 & 0 & 0 \\ (1-v)g(T)\beta I & (1-v)g(T)\beta \frac{\pi}{\mu} & 0 \\ 0 & 0 & 0 \end{bmatrix} \text{ and } V_i = \begin{bmatrix} \mu + (1-v)g(T)\beta I & (1-v)g(T)\beta \frac{\pi}{\mu} & 0 \\ 0 & \mu + \gamma & 0 \\ 0 & -\gamma & \mu \end{bmatrix}$$

Where $F(E_0)$ and $V(E_0)$ are the Jacobian matrix of F_i and V_i at DFE respectively such that;

$$F(E_0) = \begin{bmatrix} 0 & 0 & 0 \\ 0 & (1-v)g(T)\beta \frac{\pi}{\mu} & 0 \\ 0 & 0 & 0 \end{bmatrix} \text{ and } V(E_0) = \begin{bmatrix} \mu & (1-v)g(T)\beta \frac{\pi}{\mu} & 0 \\ 0 & \mu + \gamma & 0 \\ 0 & -\gamma & \mu \end{bmatrix}$$

$$F(E_0)V(E_0)^{-1} = \begin{bmatrix} 0 & 0 & 0 \\ 0 & (1-v)g(T)\beta \frac{\pi}{\mu} & 0 \\ 0 & 0 & 0 \end{bmatrix} \begin{bmatrix} \mu(\mu + \gamma) & 0 & 0 \\ 0 & (1-v)g(T)\beta \frac{\pi}{\mu} & -\mu\gamma \\ 0 & 0 & \mu(\mu + \gamma) \end{bmatrix}$$

$$F(E_0)V(E_0)^{-1} = \begin{bmatrix} 0 & 0 & 0 \\ 0 & \frac{(1-v)g(T)\beta\pi}{\mu(\mu + \gamma)} & 0 \\ 0 & 0 & 0 \end{bmatrix}$$

The R_0 was obtained from the spectral radius of the $F(E_0)V(E_0)^{-1}$ evaluated at the DFE. From system (1) therefore;

$$F(E_0)V(E_0)^{-1} - \lambda I = 0$$

$$\begin{bmatrix} 0 - \lambda & 0 & 0 \\ 0 & \frac{(1-v)g(T)\beta\pi}{\mu(\mu + \gamma)} - \lambda & 0 \\ 0 & 0 & 0 - \lambda \end{bmatrix} = 0$$

has a characteristic polynomial equation at DFE given by;

$$\lambda^2 \left\{ \frac{(1-v)g(T)\beta\pi}{\mu(\mu + \gamma)} - \lambda \right\} = 0 \tag{5}$$

The eigenvalues of equation (5) are;

$$\lambda_1 = \lambda_2 = 0, \lambda_3 = \frac{(1-v)g(T)\beta\pi}{\mu(\mu + \gamma)}$$

This implies that the spectral radius denoted by $\rho(F(E_0)V(E_0)^{-1} - \lambda I) = \frac{(1-v)g(T)\beta\pi}{\mu(\mu + \gamma)}$ representing the

basic reproduction number for the model $R_0 = \frac{(1-v)g(T)\beta\pi}{\mu(\mu + \gamma)}$

Local Stability analysis of Disease Free Equilibrium

Theorem 3.1 If basic reproduction number is defined as R_0 , the disease-free equilibrium (DFE) of the system (1) is therefore locally asymptotically stable when reproduction number is less than one.

Theorem 3.1: Proof

At DFE then the Infected individuals should less than zero, that is $I < 0$, This implies that

$$\Rightarrow \frac{dI}{dt} = (1 - v)g(T)\beta SI - (\mu + \gamma)I < 0,$$

$\Rightarrow I\{(1 - v)g(T)\beta S - (\mu + \gamma)\} < 0$, at DFE we obtain;

$$\Rightarrow I \left\{ (1 - v)g(T) \frac{\beta S}{\mu + \gamma} - 1 \right\} < 0,$$

$$\Rightarrow (1 - v)g(T) \frac{\beta \pi}{\mu(\mu + \gamma)} - 1 < 0, \Rightarrow R_0 - 1 < 0, R_0 < 1. \blacksquare$$

IV. RESULTS AND DISCUSSION

Numerical Results and Simulation

This section presents the numerical simulation of the model. We used his MATLAB solver ode45 to solve the system of differential equations (1) to investigate the impact of v and $g(T)$ on the dynamics of conjunctivitis infection in Kenya. The default parameter values used in the numerical simulations are shown in Table 1 below. Any parameters not found in the literature were estimated to fall within a sensible range.

Table 3: Parameter values used in the numerical simulations

Parameters	Descriptions	Values	References
π	Recruitment rates in susceptible populations	0.000456 day ⁻¹	[5]
β	Rate of contracting Conjunctivitis by any individual	0.004	
μ	Natural mortality rates in human populations	0.0000456 day ⁻¹	
v	Effectiveness of vaccination campaign	0.09	[6]
$g(T)$	Measuring rainfall during the dry season	0.01	[5]
γ	Recovery rates in infected human populations	0.33 day ⁻¹	
N	Total human population	10000	

Discussions

The discussion on the effects of vaccination efforts and rainfall on the transition from Susceptible to Infected Class in Kenyan conjunctivitis is carried out as follows. Increasing vaccine knowledge through media efforts on television, radio, and social media platforms improves the vaccinated population while decreasing the susceptible population. Immunities obtained through vaccinations assist the infected in fighting off conjunctivitis and moving into the Recovered class. This explains why the number of infected increases while the number of susceptible falls. Reducing the Infected class also reduces the Recovered population, because

only infected people can recover. Furthermore, raising awareness through media campaigns on television, radio, and social media platforms reduces the infected population due to adoption of the information on how to control the spread of the disease. The effects of vaccination campaigns on Conjunctivitis dynamics in Kenya are shown in Figures 2 – 4. The rates of vaccination ω are considered in the interval 0.09 to 0.64. As the vaccination increases, there is a decrease in the Conjunctivitis susceptible population, and the maximum susceptible population at any vaccination level occurs between 8th and 10th months ($8 \leq t \leq 10$). Generally, at any vaccination level, the susceptible population decreases with time as indicated in Figure 2. Figure 3 indicates that the number of infectious decrease as the number of infected people increases before the onset of infection. This implies that the population took the information positively and took the conjunctivitis vaccines. At approximately 18 months the infected people behave differently despite the same increase of ω . However, increasing up to mass vaccination enhances a decrease in the infected and recovered populations. This is because less infections mean less recoveries from the disease. As a result, the decrease in the number of infectious individuals leads to an increase in the recovered population as shown in Figure 4.

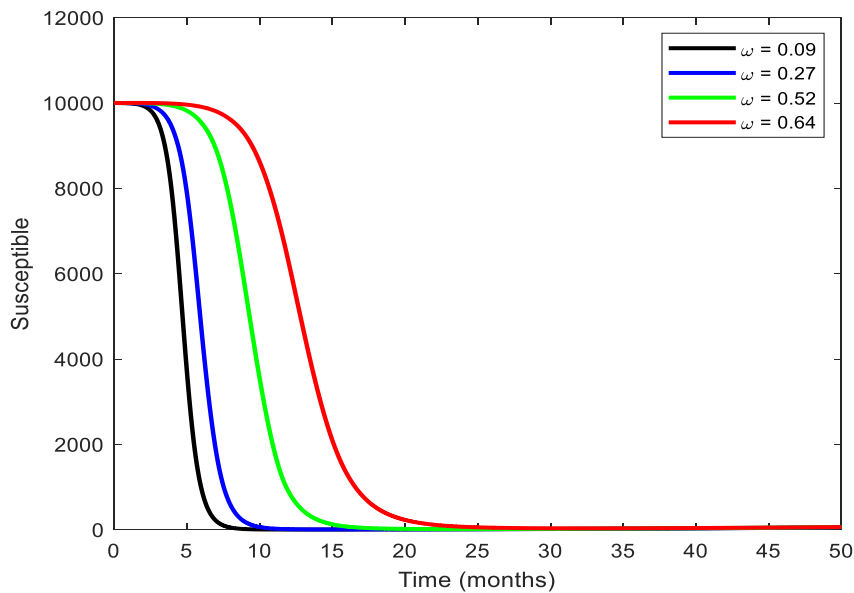


Figure 2: Effect of Vaccination on Conjunctivitis on Susceptible individuals

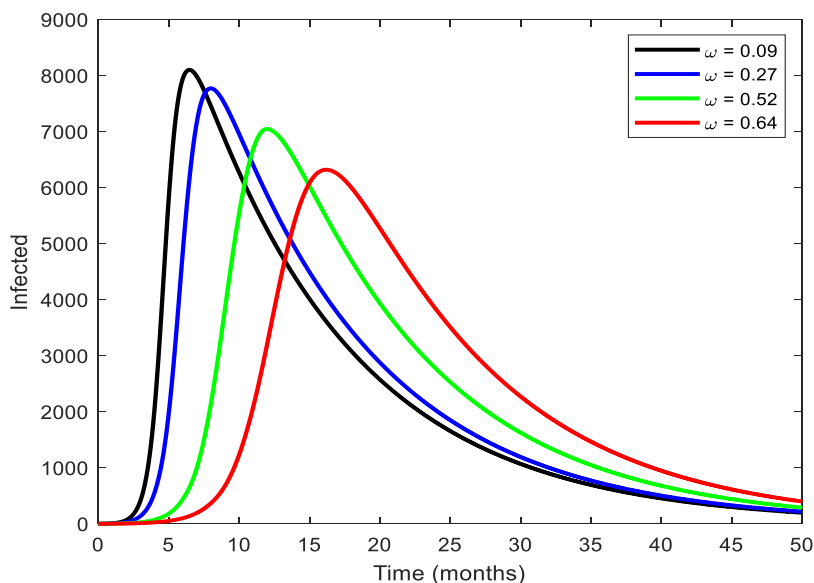


Figure 3: Effect of Vaccination on Conjunctivitis on Infected individuals

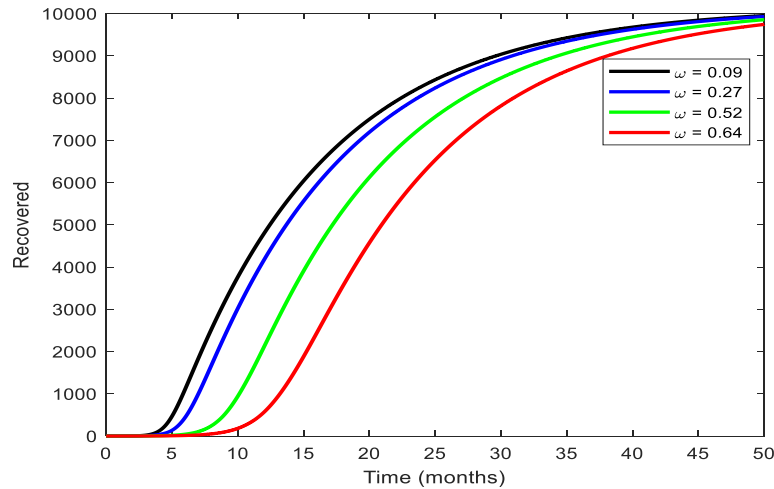


Figure 4: Effect of Vaccination on Conjunctivitis on Recovered individuals

Figures 5-6 show the effects of increase in Rainfall g from the Susceptible class to the Recovered class on the conjunctivitis dynamics in Kenya. The susceptible population decreases with increase in g (figure 5). Figure 6 shows that increase in g increases the number of infected people. This is because this bacterial infection is more likely to spread.

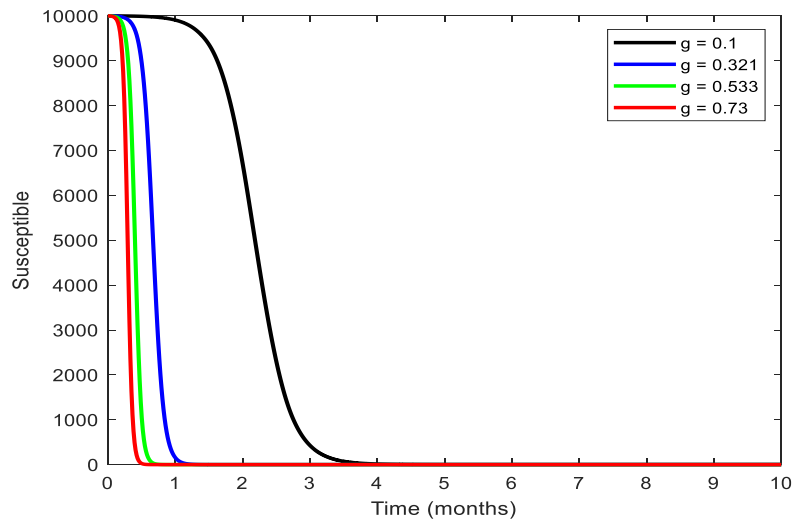


Figure 5: Rainfall Effects on Conjunctivitis on Susceptible individuals

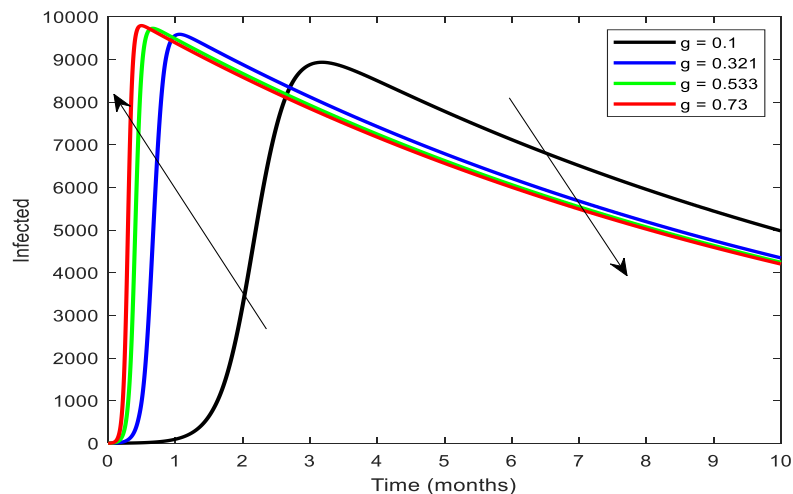


Figure 6: Effect of Rainfall on Conjunctivitis on Infected individuals

V. CONCLUSION

This study modelled the effects of vaccination campaign and rainfall on conjunctivitis dynamics in Kenya. The population studied was divided into three compartments. The system of differential equations representing the resulting model was solved numerically using the MATLAB ode45 solver. Our results should be interpreted within the framework of the study's assumptions and limitations. First, our model vaccinated a large proportion of high-risk individuals. However, achieving this range in a short period of time may be difficult [7].

The results obtained show that communication about vaccine safety can improve acceptance.

The outcomes indicate that increasing vaccination campaigns on televisions, radios and on social media platforms increases the vaccination uptake by susceptible population and reduces the infected population.

Given the limited population immunity to conjunctivitis, vaccination remains an important preventive measure to reduce the disease burden and prevent future outbreaks. Further the increase in rainfall intensity increase the conjunctivitis infections among the susceptible population. In conclusion, creating awareness either on televisions and radios and/or social media platforms on vaccinations and covering themselves well during rainy seasons among the population reduce the conjunctivitis infections. This means that infected people will respond better to vaccination, reducing their bacterial load and therefore reducing the risk of blindness among infected population. Therefore, we recommend the government to increase sensitization among the population by employing both means of awareness and also by setting up billboards with information on conjunctivitis to increase the spread of information among the entire population in Kenya.

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