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APPLICATIONS OF SIR MODEL AND DYNAMICAL ANALYSIS OF SIR MODEL OF CHICKENPOX

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ABSTRACT

Chickenpox, caused by the varicella-zoster virus remains a significant infectious disease affecting populations worldwide. The SIR (Susceptible-Infectious-Recovered) model serves as a foundational tool in epidemiology that facilitates the analysis of disease transmission dynamics and the evaluation of control measures. This article presents the applications of the SIR model in understanding chickenpox epidemiology, considering its role in predicting disease spread, optimizing vaccination strategies, and informing public health policies. Sensitivity analysis revealed critical parameters such as contact rate and recovery rate, illustrating their impact on disease transmission. Visual representations from simulation results revealed the effectiveness of interventions in reducing susceptibility and lowering infection rate over time, thereby supporting the feasibility of chickenpox eradication through comprehensive control measure and public interventions.

Keywords: Chickenpox, SIR model, Sensitivity analysis, Global stability, Endemic equilibrium, Numerical simulations

INTRODUCTION

Chickenpox is a virus disease caused by the *varicella-zoster virus (VZV)*. It is a highly contagious disease primarily affecting children and unvaccinated adults (CDC, 2020. WHO, 2021). The disease is characterized by a distinctive rash of itchy, fluid-filled blisters that typically begins on the face and spreads to other parts of the body (CDC, 2020). The varicella-zoster virus is transmitted through respiratory droplets or direct contact with the fluid from the blisters of an infected person, making it highly contagious (Edmunds & Brisson, 2023). Before the widespread introduction of the varicella vaccine, chickenpox was a common childhood illness worldwide (Agbata et al, 2019). The vaccine has significantly reduced the incidence of chickenpox and its associated complications, such as bacterial infections and pneumonia (WHO, 2021). Vaccination is now a routine part of childhood immunizations in many countries, administered in two doses to provide long-term protection (CDC, 2020).

While most cases of chickenpox resolve without complications, certain populations are at higher risk for severe illness, including adults, infants, pregnant women, and individuals with weakened immune systems (CDC, 2020). Complications of chickenpox can include bacterial skin infections, pneumonia, and encephalitis.

Management of chickenpox focuses on alleviating symptoms and preventing complications. Over-the-counter medications such as acetaminophen can reduce fever, while antihistamines may help relieve itching (WHO, 2021). It is crucial to avoid scratching the rash to prevent bacterial infections and scarring. In severe cases or in individuals at high risk of complications, antiviral medications may be prescribed to shorten the duration and reduce the severity of the illness.

Edmunds & Brisson (2023) studied mathematical modeling of *varicella-zoster virus (VZV)* transmission. They emphasized the evolution of models to incorporate vaccination strategies and demographic changes. Their findings revealed the significant impact of varicella vaccination on reducing disease incidence and mortality, stressing the importance of high coverage and booster doses to maintain immunity levels.Leung & Lopez (2022). Modeling the impact of varicella vaccination on chickenpox outbreaks in different age groups. Leung and Lopez develop a compartmental model to assess varicella vaccination's impact across different age groups. Their study shows that high vaccination coverage in children effectively reduces incidence across all age groups due to herd immunity, highlighting the importance of maintaining immunity in adolescents and adults to prevent outbreaks.

Jansen, & O'Neill (2021). Studied mathematical modeling of varicella-zoster virus reactivation: implications for vaccination and public health strategies. Jansen and O'Neill developed a stochastic model to explore *varicella-zoster virus* reactivation dynamics. Their findings suggest that while varicella vaccination reduces primary cases, it may increase herpes zoster incidence in older adults due to reduced natural boosting of immunity. Gupta&Orenstein (2020), modeled the impact of varicella vaccination on the epidemiology of chickenpox in the United States. Gupta and Orenstein developed a dynamic transmission model to predict varicella vaccination's long-term impact in the US. Their model predicts substantial reductions in varicella incidence and hospitalizations post-vaccination, emphasizing the need for sustained high coverage and potential booster doses. Mossong, & Hens (2019). modeled the impact of different varicella vaccination strategies on disease incidence in Belgium. Mossong and Hens used a dynamic transmission model to compare varicella vaccination strategies in Belgium. Their study finds that routine childhood vaccination combined with catch-up campaigns is most effective in reducing varicella incidence and associated costs. Other relevant works include (Agbata et al, 2022. Helena,2016. Agbata et al, 2021. Achejeneje et al, 2024).

The aim of the study is to explore the applications of the SIR (Susceptible-Infectious-Recovered) model and develop a specific SIR model for chickenpox. The objectives include creating a tailored SIR model to capture the transmission dynamics of chickenpox, estimating key epidemiological parameters such as the basic reproduction number (R0), infectious period, and transmission rate, and conducting sensitivity analyses to assess the model's reliability. Additionally, the study seeks to simulate chickenpox outbreaks under different conditions and population sizes, and to explore broader applications of the SIR model.

MATERIALS AND METHODS

Model Formulation

In this segment *A* mathematical model describing transmission dynamics of chickenpox is formulate. The total population

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$N(t)$ is divided into three epidemiological compartments.

The dynamics of these compartments are governed by differential equations that describe how individuals move from being susceptible to infectious, and then from infectious to recovered or removed, capturing the spread and control of infectious diseases within populations.

- The susceptible (S): Individuals who are susceptible to the disease and can become infected if exposed to the infectious agent
- The infectious (I): Individuals who are currently infected and can transmit the disease to susceptible individuals
- The recovered (R): Individuals who have recovered from the disease and are assumed to have acquired immunity, or who have been removed from the population (e.g., through death).

Model Description

The model considers three epidemiological compartments of $S(t), I(t), R(t)$ where

$N(t) = S(t) + I(t) + R(t)$ it is assumed that every individual is recruited at a constant rate Λ, the susceptible population decreases as a result of natural death rate μ and contact rate of infection β. It further reduces to rate of recovery class at rate of ϕ. The population of infected humans increases as a result of progression of susceptible humans to infected class at the rate β. The infected humans also reduce due to effective treatment leading to recovery rate of μ. The compartment also reduces due to disease induced death rate σ the recovered population increases as a result of influx of susceptible individuals into recovered class at rate ϕ. This compartment is further decreases due to natural death rate μ.

The total per capital removal rate is defined by ε which consists of natural death rate μ chickenpox death rate ω and recovery rate α, Hence

$$\varepsilon = \alpha + \sigma + \mu$$

Assumptions of the Model

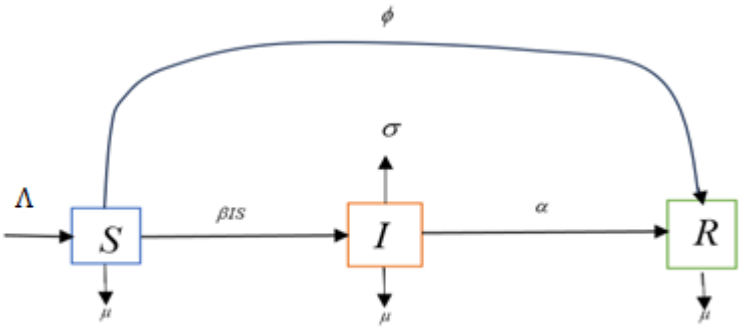
The model is based on the following assumptions:

1. The population mixture is homogeneous
2. A recovered human can also be susceptible to the disease.
3. Sex, age, race and social status do not prevent one from being infected
4. There is no inherited immunity
5. Homogeneous infectivity and recovery rate:

Table 1. variables and parameters used in the model and their interpretations

Variables	Interpretation
$S(t)$	Susceptible individuals at time t
$I(t)$	Infected individuals at times t
$R(t)$	Recovered individuals
Λ	Constant recruitment level
β	Contact rate
σ	Disease induced death rate
μ	Natural death rate
α	Recovery rate
ϕ	Immunity gain rate

Considering the above we have the schematic diagram as.



Mathematical Formulation of the Model Equations

$$\frac{dS}{dt} = \Lambda - \beta IS - \phi S - \mu S$$

$$\frac{dI}{dt} = \beta IS - \sigma I - \alpha I - \mu I \qquad (2.1)$$

$$\frac{dR}{dt} = \alpha I + \phi S - \mu R$$

Analysis of the Model

Suppose the total population $N(t) = S(t) + I(t) + R(t)$ summing the system all the differential equations of model(2.1) and taking the time derivative of $N(t)$. we have

$$\frac{dN}{dt} = \Lambda - \mu N - \sigma I \qquad (2.2)$$

The equation (2.2)is known as population dynamics

Invariant Region

An invariant region in epidemiological models such as the SIR model for diseases like chickenpox, denotes a subset of the state space where solutions remain confined once they enter (Diekmann,1990) . This concept is crucial for understanding the stability and long-term behavior of dynamical systems. It ensures that simulated trajectories of disease spread adhere to realistic boundaries of susceptible, infectious, and recovered individuals within a population.

Theorem 1

Every solution of the model in the equation (2.1) with initial conditions R_+^3 remains in the invariant region Ω as $t \to \infty$

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$$\Omega = \left\{ (\mathrm{S}, \mathrm{I}, \mathrm{R}) \in R_+^3 : N(\mathrm{t}) \le \frac{\Lambda}{\mu} \right\}$$

From equation (2.2)

$$\frac{dN}{dt} = \Lambda - \mu N - \phi I$$

Where there is no disease in the system, we have

$$\frac{dN}{dt} = \Lambda - \mu N(\mathrm{t}) \tag{2.3}$$

So that $\frac{dN}{dt} \le \Lambda - \mu N(\mathrm{t})$ (2.4)

Since $\frac{dN}{dt} \le 0$ if $N(\mathrm{t}) \ge \frac{\Lambda}{\mu}$

We obtain the solution of (2.4) as follows

$$\frac{dN}{dt} + \mu N(\mathrm{t}) \le \Lambda$$

Multiplying through by the integrating factor e^{ut}

$$\frac{dN}{dt}(\mathrm{e}^{ut}) + \mu\, \mathrm{N(t)}(\mathrm{e}^{ut}) \le \Lambda(\mathrm{e}^{ut})$$

$$\frac{d}{dt}\left[e^{ut} N(\mathrm{t}) \right] \le \Lambda e^{ut}$$

$$d\left[e^{ut} N(\mathrm{t}) \right] \le \Lambda e^{ut} dt$$

Integrating both sides

$$\int d\left[e^{ut} N(\mathrm{t}) \right] \le \int \Lambda e^{ut} dt$$

$$N(\mathrm{t})\, \mathrm{e}^{ut} \le \frac{\Lambda e^{ut}}{\mu} + K_O$$

$$N(\mathrm{t}) \le \frac{\Lambda}{\mu} + \frac{K_o}{e^{ut}}$$

$$N(\mathrm{t}) \le \frac{\Lambda}{\mu} + K_o e^{ut} \tag{2.5}$$

Apply the initial condition $t(0) = \mathrm{N}(0)$

$$N(0) - \frac{\Lambda}{\mu} \le K_0 \tag{2.6}$$

So that equation (2.5) becomes

$$N(\mathrm{t}) \le \frac{\Lambda}{\mu} + \left[N(0) - \frac{\Lambda}{\mu} \right] e^{-\mu t} \tag{2.7}$$

Taking limit as $t \to \infty$

$$N(\mathrm{t}) \le \frac{\Lambda}{\mu} \tag{2.8}$$

Which means that $0 \le N \le \frac{\Lambda}{\mu}$

Therefore, the model (2.1) is bounded in the region Ω

$$\Omega = \left\{ (\mathrm{S}, \mathrm{I}, \mathrm{R}) \in \mathrm{R}_{+}^{3} : N(\mathrm{t}) \le \frac{\Lambda}{\mu} \right\}$$

Positivity of Solution

Positivity of solutions in mathematical modeling, particularly in contexts such as epidemiology and dynamical systems, ensures that variables representing real-world quantities, like state variables, remain non-negative throughout simulations or analyses. This principle is crucial for maintaining the physical relevance and realism of model outputs (Diekmann,1990). It requires that initial conditions, boundary conditions, and mathematical formulations respect the constraint that quantities cannot become negative, as negative populations or fractions are not feasible in practical scenarios.

Theorem 2

Let the initial data of the system (2.1) be $\{S(0), \mathrm{I}(0), \mathrm{R}(0) \ge 0\} \in \Omega$

Then solution get $\{S(\mathrm{t}), \mathrm{I}(\mathrm{t}), \mathrm{R}(\mathrm{t})\}$ of the system of differential equation (2.1) is positive for all $t > 0$.

Proof:

From the first equation of model (2.1),we assume that

$$\frac{dS}{dt} = \Lambda - \beta SI - (\phi + \mu)\mathrm{S} \ge -(\phi + \mu)\mathrm{S}, \text{ For } \beta \in [0,1] \text{ and } \beta = \frac{\Lambda}{SI}$$

$$\frac{dS}{dt} \ge -(\phi + \mu)\mathrm{S} \tag{2.9}$$

$$\frac{dS}{S} \ge -(\phi + \mu)\,\mathrm{dt}$$

Integrating both sides

$$\ln S \ge -(\phi + \mu) + \mathrm{C}_1$$

$$S(\mathrm{t}) \ge e^{-(\phi+\mu)\mathrm{t}+\mathrm{C}_1}$$

$$S(\mathrm{t}) \ge K_1 e^{-(\phi+\mu)\mathrm{t}} \tag{2.10}$$

Applying the initial conditions $t = 0, \quad \mathrm{S}(0) \ge K_1$

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Equation (2.10)becomes

$$S(\mathrm{t}) \geq \mathrm{S}(0)\,\mathrm{e}^{-(\phi+\mu)\mathrm{t}} \tag{2.11}$$

From second equation of model (2.1)

$$\frac{dI}{dt} = \beta SI - (\alpha+\sigma+\mu)\,\mathrm{I} \geq -(\alpha+\sigma+\mu)\,\mathrm{I} \tag{2.12}$$

So that

$$\frac{dI}{dt} \geq -(\alpha+\sigma+\mu)\,\mathrm{I}$$

Solving (2.12) and applying the initial conditions

$t = 0,\ I(0) \geq \mathrm{K}_2$

We have

$$I(\mathrm{t}) \geq \mathrm{I}(0)\,\mathrm{e}^{-(\alpha+\sigma+\mu)\mathrm{t}>0} \quad (\alpha+\sigma+\mu) > 0 \tag{2.13}$$

From the third equation model (2.1)

$$\frac{dR}{dt} = \alpha I + \phi S - \mu R \geq -\mu R \tag{2.14}$$

$$\frac{dR}{dt} \geq -\mu R$$

$$\frac{dR}{R} \geq -\mu dt$$

$$\ln R \geq e^{-\mu t + C_2}$$

$$R(\mathrm{t}) \geq \mathrm{e}^{-ut+C_2}$$

$$R(\mathrm{t}) \geq K_3\,\mathrm{e}^{-ut} \tag{2.15}$$

Where $K_3 = e^{C_2}$

Applying the initial conditions at $t = 0$ $R(0) \geq K_3$

Equation (2.15)becomes

$$R(\mathrm{t}) \geq R(0)\,\mathrm{e}^{-ut} > 0 \tag{2.16}$$

Therefore, since $\mu > 0$, the solution set $S(\mathrm{t}), \mathrm{I}(\mathrm{t}), \mathrm{R}(\mathrm{t})$ are positive for all $t > 0$.

and the from the inequalities (2.11), (2.13) and(2.16)above

Stability Analysis of the Model

Equilibrium Point

At equilibrium point

$$\frac{dS}{dt} = \frac{dI}{dt} = \frac{dR}{dt} = 0$$

So that

$$\Lambda - \beta IS - (\phi + \mu)\mathrm{S} = \mathrm{O}$$

$$\beta SI - (\alpha + \sigma + \mu)\mathrm{I} = 0 \qquad (3.1)$$

$$\alpha I + \phi S - \mu R = O$$

Disease Free Equilibrium (DFE)

The disease-free equilibrium (DFE) in epidemiological modeling signifies a stable state within a population where the infectious disease is absent. At this equilibrium point, there are no active cases of the disease, meaning the number of infected individuals is zero (Diekmann,1990). This state is achieved when the rate of new infections balances with the rate of recoveries or removals from the infectious state, resulting in a cessation of disease transmission.

At disease free equilibrium $I = 0, R = 0$

So that equation (3.1) reduces to

$$\Lambda - \beta SI - (\phi + \mu)\mathrm{S} \qquad (3.2)$$

Since $I = R = 0$

$$\Lambda - \mu\mathrm{S} = 0$$

$$S = \frac{\Lambda}{\mu}$$

The disease-free equilibrium of the model (2.1) is

$$\varepsilon_o = (\mathrm{S}^*, \mathrm{I}^*, \mathrm{R}^*) = \left(\frac{\Lambda}{\mu}, 0, 0\right) \qquad (3.3)$$

Local Stability of Disease-Free Equilibrium

Local stability in dynamical systems, refers to the tendency of a system's behavior to return to an equilibrium point after experiencing small perturbations or deviations (Diekmann,1990). Specifically, it assesses whether minor changes in variables like the numbers of susceptible, infectious, and recovered individuals will cause the system to remain near the equilibrium (stable) or move away from it (unstable). .

Let $A = \Lambda - \beta IS - \phi S - \mu S$

$$B = \beta IS - \sigma I - \alpha I - \mu I \qquad (3.4)$$

$$C = \alpha I + \phi S - \mu R$$

The Jacobian matrix J is given by

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$$J = \begin{bmatrix} \frac{dA}{dS} & \frac{dA}{dI} & \frac{dA}{dR} \\ \frac{dB}{dS} & \frac{dB}{dI} & \frac{dB}{dR} \\ \frac{dC}{dS} & \frac{dC}{dS} & \frac{dC}{dS} \end{bmatrix} \tag{3.5}$$

$$J = \begin{bmatrix} (\beta \mathrm{I}^* + \phi + \mu) & -\beta S^* & 0 \\ \beta \mathrm{I}^* & \beta S^* - (\sigma + \alpha + \mu) & 0 \\ \phi & \alpha & -\mu \end{bmatrix} \tag{3.6}$$

At disease free equation $S^* = \frac{\Lambda}{\mu}$, $I^* = 0$

Equation (3.6) becomes

$$J(\varepsilon_0) = \begin{bmatrix} -(\phi + \mu) & \frac{-\beta\Lambda}{\mu} & 0 \\ 0 & \frac{\beta\Lambda}{\mu} - (\sigma + \alpha + \mu) & 0 \\ \phi & \alpha & -\mu \end{bmatrix} \tag{3.7}$$

The characteristics equation is given by

$$\left| J^*(\varepsilon_O) - \lambda \mathrm{I} \right| = 0$$

$$\left| J^*(\varepsilon_O) - \lambda \mathrm{I} \right| = \begin{bmatrix} -(\phi + \mu) - \lambda & \frac{-\beta\Lambda}{\mu} & 0 \\ 0 & \left\{ \frac{\beta\Lambda}{\mu} - (\sigma + \alpha + \mu) \right\} - \lambda & 0 \\ 0 & \alpha & -\mu - \lambda \end{bmatrix} \tag{3.8}$$

From equation (3.8) ,we have

$$\lambda_1 = -(\phi + \mu) = (0.06 + 0.03) = -0.0900$$
$$\lambda_2 = \frac{\beta\Lambda - (\sigma + \alpha + \mu)\mu}{\mu} = \frac{0.02 * 0.05 - (0.40 + 0.167 + 0.03) * 0.03}{0.03} = 0.7367,\ \lambda_3 = -\mu = -0.0300 \tag{3.9}$$

Basic Reproduction Number

The basic reproduction number (R_0) is a key epidemiological metric used to gauge the transmission potential of an infectious disease within a population (Agbata et al, 2021). It represents the average number of secondary infections that one infectious individual would generate in a completely

susceptible population (Diekmann,1990). R_0 serves as a crucial indicator of the contagiousness and potential for epidemic spread of a disease. This metric informs public health strategies by indicating the effectiveness of interventions such as vaccination, quarantine, and social distancing in reducing transmission rates and controlling outbreaks (Vanden Driessche and Watmough 2002).

Considering $\lambda_1 = -(\phi + \mu)$ $\lambda_2 = \dfrac{\beta\Lambda - (\sigma + \alpha + \mu)\mu}{\mu}$ $\lambda_3 = -\mu$

R_0 is obtain from the largest eigen value which λ_2.

Let $\lambda_2 < 0$

$$\frac{\beta\Lambda - (\sigma + \alpha + \mu)\mu}{\mu} < 0 \tag{3.10}$$

It implies

$$\beta\Lambda - (\sigma + \alpha + \mu)\mu < 0$$

$$\frac{\beta\Lambda}{(\sigma + \alpha + \mu)\mu} - 1 < 0$$

$$\frac{\beta\Lambda}{(\sigma + \alpha + \mu)\mu} < 1$$

Hence

$$R_0(S, I, R) = \frac{\beta\Lambda}{(\sigma + \alpha + \mu)\mu} \tag{3.11}$$

For all $\beta, \Lambda, \alpha, \omega, \mu, \phi > 0$ and $(\sigma + \alpha + \mu)\mu \neq 0$

The basic reproduction number can also be obtained as follows

$$\frac{dI}{dt} = \beta IS - (\sigma + \alpha + \mu) I$$

$$= \beta IS - (\sigma + \alpha + \mu) I$$

$$(\sigma + \alpha + \mu) I \left[\frac{\beta S^*}{(\sigma + \alpha + \mu)} - 1 \right]$$

$$R_0 = \frac{\beta S^*}{(\sigma + \alpha + \mu)} = \frac{\beta\Lambda}{(\sigma + \alpha + \mu)\mu}$$

Remark:

Epidemiologically

i. If $R_0 < 1$

When the basic reproduction number (R_0) in epidemiology is less than 1, it indicates that each infected individual is likely to transmit the disease to fewer than one other person on average. This

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situation suggests that the disease is not self-sustaining in the population and will eventually decline (Diekmann,1990). Key implications include the ability to control and potentially eliminate the disease through public health measures such as vaccination, quarantine, and hygiene promotion (Vanden Driessche and Watmough 2002).

ii. If $R_0 = 1$

An $R_0 = 1$ signifies a critical threshold where each infected individual, on average, transmits the disease to exactly one other individual during their infectious period . This condition leads to an endemic equilibrium within the population, where the disease persists at a stable prevalence without causing large-scale outbreaks (Diekmann,1990). $R_0 = 1$ indicates a delicate balance between transmission and control measures. When the basic reproduction number (R_0) of a mathematical model equals 1, it indicates a specific mathematical and epidemiological scenario that requires careful consideration in modeling infectious diseases. It means more research is to be done and careful monitoring, and effective public health interventions are essential to manage disease transmission and prevent outbreaks within the population (Vanden Driessche and Watmough 2002).

iii. If $R_0 > 1$

When the basic reproduction number (R_0) exceeds 1 in epidemiology, it signifies that each infected individual, on average, will transmit the disease to more than one other person. This condition indicates the potential for exponential growth of the epidemic, as each new infection can lead to multiple subsequent infections, causing the number of cases to escalate rapidly over time (Diekmann,1990). Effective public health measures such as vaccination, quarantine, and social distancing are crucial in reducing the effective contact rate between susceptible and infectious individuals, thereby lowering R_0 and preventing sustained transmission

Theorem 3

The disease-free equilibrium ε_0 of the model (2.1) is locally asymptotically

stable in Ω if $R_0 < 1$ and unstable if $R_0 > 1$

$$\left| J^*(\varepsilon_O) - \lambda \mathrm{I} \right| = \begin{bmatrix} -(\phi+\mu)-\lambda & \dfrac{-\beta\Lambda}{\mu} & 0 \\ 0 & \left\{\dfrac{\beta\Lambda}{\mu} - (\sigma+\alpha+\mu)\right\} - \lambda & 0 \\ 0 & \alpha & -\mu-\lambda \end{bmatrix} = 0$$

$$\lambda^3 - \frac{\left(\beta\Lambda - \alpha\mu - \alpha\phi - 3\mu^2 - \sigma\mu - 4\mu\phi - \sigma\phi - \phi^2\right)\lambda^2}{\mu}$$

$$- \frac{\begin{pmatrix} 2\beta\Lambda\mu + \beta\Lambda\phi - 2\alpha\mu^2 - 3\alpha\mu\phi - \alpha\phi^2 - 3\mu^3 \\ -2\sigma\mu^2 - 5\mu^2\phi - 3\sigma\mu\phi - 2\mu\phi^2 - \sigma\phi^2 \end{pmatrix}\lambda}{\mu} -$$

$$\mu\left(\beta\Lambda - \alpha\mu - \alpha\phi - \mu^2 - \sigma\mu - \mu\phi - \sigma\phi\right) = 0$$

$$\lambda^3 - \frac{\left(\beta\Lambda - \alpha\mu - \alpha\phi - 3\mu^2 - \sigma\mu - 4\mu\phi - \sigma\phi - \phi^2\right)\lambda^2}{\phi + \mu}$$

$$- \frac{\begin{pmatrix} 2\beta\Lambda\mu + \beta\Lambda\phi - 2\alpha\mu^2 - 3\alpha\mu\phi - \alpha\phi^2 - 3\mu^3 \\ -2\sigma\mu^2 - 5\mu^2\phi - 3\sigma\mu\phi - 2\mu\phi^2 - \sigma\phi^2 \end{pmatrix}\lambda}{\mu} +$$

$$\mu\left(\left(\phi + \mu\right)\left(\mu + \alpha + \sigma\right) - \beta\Lambda\right) = 0$$

$$\lambda^3 - \frac{\left(\beta\Lambda - \alpha\mu - \alpha\phi - 3\mu^2 - \sigma\mu - 4\mu\phi - \sigma\phi - \phi^2\right)\lambda^2}{\phi + \mu}$$

$$- \frac{\begin{pmatrix} 2\beta\Lambda\mu + \beta\Lambda\phi - 2\alpha\mu^2 - 3\alpha\mu\phi - \alpha\phi^2 - 3\mu^3 \\ -2\sigma\mu^2 - 5\mu^2\phi - 3\sigma\mu\phi - 2\mu\phi^2 - \sigma\phi^2 \end{pmatrix}\lambda}{\mu} +$$

$$\mu\left(\phi + \mu\right)\left(\mu + \alpha + \sigma\right)\left(1 - \frac{\beta\Lambda}{\left(\phi + \mu\right)\left(\mu + \alpha + \sigma\right)}\right) = 0$$

$$\lambda^3 - \frac{\left(\beta\Lambda - \alpha\mu - \alpha\phi - 3\mu^2 - \omega\mu - 4\mu\phi - \sigma\phi - \phi^2\right)\lambda^2}{\mu}$$

$$- \frac{\begin{pmatrix} 2\beta\Lambda\mu + \beta\Lambda\phi - 2\alpha\mu^2 - 3\alpha\mu\phi - \alpha\phi^2 - 3\mu^3 \\ -2\sigma\mu^2 - 5\mu^2\phi - 3\sigma\mu\phi - 2\mu\phi^2 - \sigma\phi^2 \end{pmatrix}\lambda}{\mu} +$$

$$\mu\left(\phi + \mu\right)\left(\mu + \alpha + \sigma\right)\left(1 - R_0\right) = 0$$

Applying Routh-Hurwitz criterion to the Characteristics polynomial, we have that

$$\left(1 - R_0\right) > 0$$

$$\Rightarrow R_0 < 1$$

Thus the DFE point of the model is locally asymptotically stable.

Global Stability of Disease-Free Equilibrium

Global stability refers to the property of a dynamical system where all trajectories (or solutions) converge to a particular equilibrium point, and once they reach it, they remain there indefinitely (Diekmann,1990). This equilibrium point is typically stable, meaning small perturbations from this

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point result in trajectories that return to or stay close to the equilibrium over time. Global stability ensures the reliability and predictive power of mathematical models in epidemiology.

To investigate the global stability of the disease free equilibrium, we apply the method implemented by

Lemma 1

Castillo-Chavez and song

we write the equation in the uninfected class as

$$\frac{dX}{dt} = F\left(X, Z\right)$$

And we re-write the equation in the infected class as

$$\frac{dz}{dt} = G(X, Z)$$

Where $X = \left(S, R\right) \in R^2_+$ represents the uninfected compartment and

$Z = \left(I\right) \in R^1_+$ represents the infected compartment

$\varepsilon_0 = (X^*, 0)$ denotes the disease free equilibrium of the system, and it globally asymptotically stable if it satisfies the following conditions:

$H_1 : \frac{dX}{dt} = F\left(X^*, 0\right), X^*$ is globally asymptotically stable

$$H_2 : \frac{dZ}{dt} = D_Z G\left(X^*, 0\right) Z - \hat{G}\left(X, Z\right)$$

$\hat{G}\left(X, Z\right) \geq 0$ for all $\left(X, Z\right) \in D$ and where $D_Z G\left(X^*, 0\right)$ is an M- matrix (i.e the diagonal elements are no-negative and it is also the Jacobian of $\hat{G}\left(X, Z\right) \geq 0$ evaluated at $(X^*, 0)$.

If the system satisfies the above condition, then the theorem below holds (Diekmann,1990).

Theorem 2

The equilibrium point $\varepsilon_0 = (X^*, 0)$. *of I the model is globally asymptotically stable if* $R_0 \leq 1$ *and* H_1, H_2 are satisfied.

$$F(X, Z) = \begin{bmatrix} \Lambda - \beta IS - \phi S - \mu S \\ \alpha I + \phi S - \mu R \end{bmatrix}$$

$$G(X, Z) = \left[\beta IS - \sigma I - \alpha I - \mu I\right]$$

At disease free equilibrium,

H_1 :

$$\frac{dS}{dt} = \Lambda - \mu S$$

$$\frac{dR}{dt} = 0$$

H_2 :

$$D_Z G\left(X^*, 0\right) Z = \left[\beta IS - \sigma I - \alpha I - \mu I\right]$$

$$\hat{G}\left(X, Z\right) = D_Z G\left(X^*, 0\right) Z - G\left(X, Z\right)$$

$$\hat{G}\left(X, Z\right) = \left[\beta I\left(1 - \frac{S}{N}\right)\right]$$

Clearly, $1 \geq S$ this means that $\hat{G}\left(X, Z\right) = 0$.

Hence, the disease free equilibrium of the given model is globally asymptotically stable [8].

Endemic Equilibrium

Endemic equilibrium in epidemiological modeling denotes a stable state where a disease persists at a constant prevalence within a population over time. It occurs when the rate of new infections matches the rate of recoveries or removals from the infectious state, resulting in a balanced disease prevalence (Agbata et al, 2021). Mathematically, endemic equilibrium is characterized by equilibrium conditions in models such as the SIR (Susceptible-Infectious-Recovered) model, where the numbers of susceptible, infectious, and recovered individuals stabilize.

Let endemic equilibrium of the model (2.1) be ε_1 so that

$$\varepsilon_1 = (\mathrm{S}^{**}, \mathrm{I}^{**}, \mathrm{R}^{**})$$

Recall that

$$\Lambda - (\beta \mathrm{I} + \phi + \mu)\mathrm{S}$$

$$\beta IS - (\alpha + \omega - \mu) = 0 \qquad (3.12)$$

$$\alpha I + \phi S - \mu R$$

From $\beta IS - (\alpha + \sigma + \mu)\mathrm{I} = 0$

$$I\left[\beta S - (\alpha + \sigma + \mu)\right] = 0$$

$$I \neq 0 \ \beta S - (\alpha + \sigma + \mu) = 0$$

$$S^{**} = \frac{(\alpha + \sigma + \mu)}{\beta} \qquad (3.13)$$

Substitute (3.13)into

$$\Lambda - (\beta \mathrm{I} + \phi + \mu)\mathrm{S} = 0$$

Solving for I, we have

$$I^{**} = \frac{\Lambda\beta - (\phi + \mu)(\alpha + \sigma + \mu)}{\beta(\alpha + \sigma + \mu)} \qquad (3.14)$$

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Substitute (3.13) and (3.14)into the equations below.

$\alpha I + \phi S - \mu R$

We have

$$R^{**} = \frac{\alpha\beta\Lambda - (\alpha+\sigma+\mu)(\alpha\mu - \phi\mu - \sigma\phi)}{\beta\mu(\mu+\sigma+\mu)} \tag{3.15}$$

Equation(3.13),(3.14)and(3.15) are the values of $(S^{**}, I^{**}, R^{**}) \neq (0,0,0)$ in vector form, we write

$$(S^{**}, I^{**}, R^{**}) = \left(\frac{(\alpha+\sigma+\mu)}{\beta}, \frac{\Lambda\beta - (\phi+\mu)(\alpha+\sigma+\mu)}{\beta(\alpha+\sigma+\mu)}, \frac{\alpha\beta\Lambda - (\alpha+\sigma+\mu)(\alpha\mu-\phi\mu-\sigma\phi)}{\beta\mu(\mu+\sigma+\mu)} \right)$$

Sensitivity Analysis of the Model

Sensitivity analysis involves systematically varying input parameters within a model to assess their impact on the model's outputs or predictions (Diekmann,1990). It aims to quantify how changes in these parameters influence key outcomes such as disease prevalence, epidemic dynamics, or the effectiveness of interventions. Sensitivity analysis is carried out to determine the parameters that enhance the spread of measles as well as control of the infection in a population.

The sensitivity index of the reproduction number of the model with respect to any parameter say x is given by:

$$\Im_x^{R_0} = \frac{\partial R_0}{\partial x} \times \frac{x}{R_0}$$

Given that

$$R_0(S,I,R) = \frac{\beta\Lambda}{(\alpha+\sigma+\mu)\mu}$$

$$\Im_{R_0}^{\beta} = \frac{\partial R}{\partial \beta} \times \frac{\beta}{R} = 1.0000$$

$$\Im_{R_0}^{\Lambda} = \frac{\partial R}{\partial \Lambda} \times \frac{\Lambda}{R} = 1.0000$$

$$\Im_{R_0}^{\beta} = \frac{\partial R}{\partial \mu} \times \frac{\mu}{R} = -\frac{(\phi + 2\mu + \alpha + \omega)\mu}{(\alpha+\sigma+\mu)(\phi+\mu)} = -0.0218$$

$$\Im_{R_0}^{\alpha} = \frac{\partial R}{\partial \alpha} \times \frac{\alpha}{R} = -\frac{\alpha}{\alpha+\sigma+\mu} = -0.6651$$

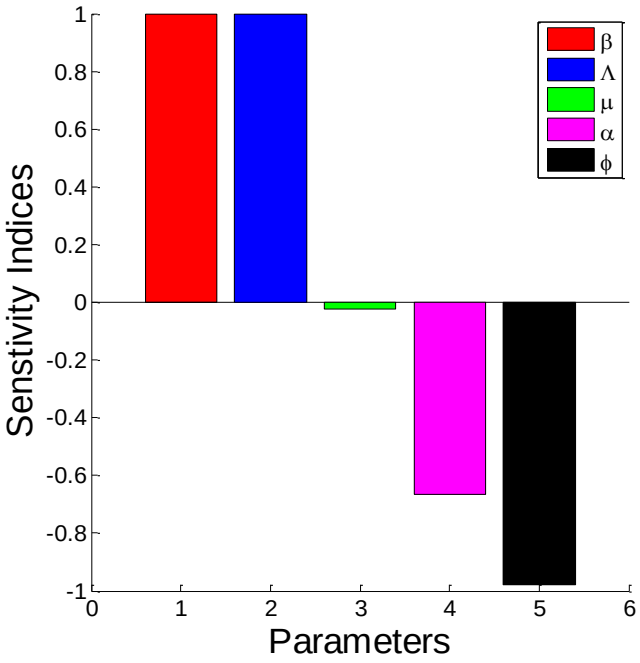

Figure 2. Sensitivity bar chart.

NUMERICAL SIMULATIONS OF THE MODEL

Numerical simulations provide modelers with the means to validate mathematical models against empirical data and adjust model parameters to enhance accuracy. This iterative process ensures that the model accurately captures observed patterns of measles transmission and vaccination outcomes. Introducing double-dose vaccination adds complexity by considering variables such as the interval between doses and the effectiveness of immune boosting (Agbata et al, 2021). Numerical simulations allow for the exploration of various vaccination scenarios, including different levels of vaccine coverage and efficacy, to evaluate their impact on disease control. These simulations contribute to understanding disease dynamics over time, identifying crucial parameters, and optimizing control strategies.

T**able 2. Parameter table of values**

Parameter	**Value**	**Source**
Λ	0.80	Assumed
μ	0.03	(Agbata et al, 2019)
σ	0.167	Assumed
β	0.05	(Agbata et al, 2019)
α	0.40	Assumed
ϕ	0.06	Assumed

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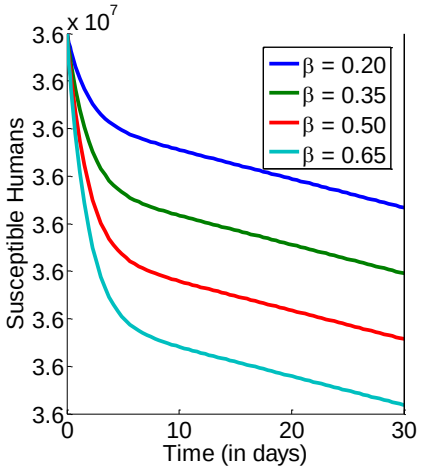

Figure 3a. Graph of susceptible population.

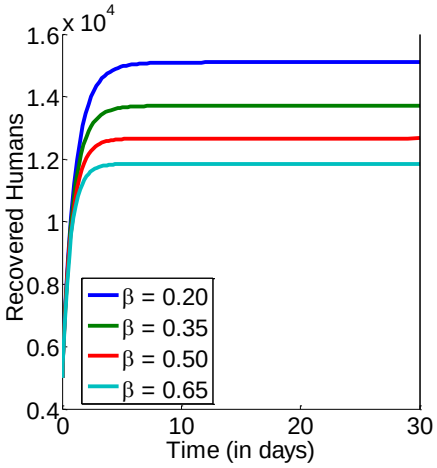

Figure 3b. Graph of recovered population

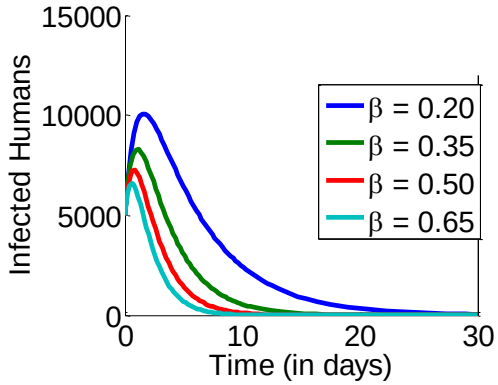

Figure 3c. Graph of infected human against time.

DISCUSSION

The sensitivity analysis and the accompanying bar chart in figure 2 clearly indicate that certain parameters play pivotal roles in the transmission of chickenpox within the human population. Parameters with positive sensitivity indices, such as the contact rate, β significantly contribute to the spread of the disease. This suggests that reducing interactions between infected and susceptible individuals could effectively curb the transmission of chickenpox. On the other hand, parameters like the recovery rate α_1 ,exhibit negative sensitivity index, implying that increasing recovery rates through robust vaccination campaigns and effective control measures would help mitigate the spread of the disease.

Figure 3a portrays a graph depicting the number of susceptible individuals over time. It reveals a declining trend, indicating successful disease control as fewer individuals remain susceptible as time progresses. Figure 3b complements this by demonstrating a notable increase in the recovery rate, corresponding with the decrease in the number of infected individuals depicted in Figure 3c. Collectively, these figures emphasized the potential for eradicating chickenpox from the population through targeted interventions.

The sensitivity analysis highlights critical factors influencing chickenpox transmission, emphasizing the significance of reducing contact rate and enhancing recovery rate through comprehensive vaccination programs and effective disease control strategies. Figures 3a and 3b provide visual evidence of the effectiveness of these measures in reducing

susceptibility and lowering infection rates over time, ultimately supporting the feasibility of eradicating chickenpox from the population.

Applications of SIR Model

- **Health:** The SIR model is important in epidemiology for understanding the transmission dynamics of infectious diseases within populations. This model categorizes individuals into susceptible (S), infectious (I), and recovered or removed (R) compartments. By simulating how individuals move between these compartments based on transmission rate and recovery rate, epidemiologists can predict the spread of diseases such as influenza, measles, and COVID-19 (Helena, 2016). For instance, estimating the basic reproduction number (R0) using the SIR model helps assess the contagiousness of a disease and guide public health interventions. This predictive capability is essential for planning vaccination campaigns, implementing quarantine measures, and recommending social distancing guidelines to mitigate disease spread. The SIR model's ability to simulate different scenarios and evaluate the impact of interventions supports evidence-based decision-making in managing disease outbreaks and safeguarding public health (Pastor-Satorras & Vespignani, 2001).
- **Network:**In network science, the SIR model serves as a powerful tool for studying the spread of information, behaviors, and innovations across interconnected networks. Researchers apply the model to analyze how phenomena propagate through social media platforms, communication networks, and peer-to-peer networks. By simulating the adoption dynamics of new technologies, the diffusion of rumors, or the spread of viral content, the SIR model helps understand network dynamics and social influence processes. For example, it enables the identification of influential individuals or nodes within networks and informs strategies to promote positive behaviors, manage information cascades, and combat misinformation (Pastor-Satorras & Vespignani, 2001). This application underscores the model's relevance in studying complex network phenomena and its potential to improve network resilience and communication strategies.
- **Economics and Finance:**Economists use the SIR model to study "economic epidemics" such as financial contagion, market crashes, and the diffusion of financial innovations. By modeling the spread of economic disturbances through interconnected markets, the SIR model helps predict the propagation of financial shocks and assess the systemic risks within financial networks (Pastor-Satorras & Vespignani, 2001). This capability is crucial for understanding the dynamics of market volatility, evaluating the effectiveness of regulatory policies, and designing interventions to enhance financial stability. For instance, simulating the impact of policy interventions on market behaviors using the SIR model supports proactive measures to mitigate economic crises and improve overall market resilience (Helena, 2016). This application demonstrates the model's utility in analyzing complex economic systems and informing policy decisions aimed at promoting sustainable economic growth.
- **Computer Virus:**In cybersecurity, the SIR model is employed to analyze the spread of computer viruses, malware, and other cyber threats through interconnected computer networks. Security analysts use the model to simulate how viruses propagate among networked devices, assess the vulnerability of computer systems, and evaluate the effectiveness of cybersecurity defenses and response strategies (Helena, 2016). By modeling the dynamics of cyber threats using the SIR framework, analysts can prioritize security measures, enhance incident response

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capabilities, and mitigate the impact of cyberattacks on organizations and individuals (Helena, 2016. Keeling & Rohani, 2008). This application underscores the model's role in safeguarding digital infrastructures and improving cybersecurity resilience in an increasingly interconnected world.

- **Ecology:** Ecologists utilize the SIR model to study the dynamics of diseases affecting wildlife populations, agricultural pests, and endangered species. By modeling disease transmission within ecosystems, the SIR model helps predict disease outbreaks, assess the impact on biodiversity, and inform conservation strategies (Macy & Willer, 2002). For example, researchers apply the model to understand how diseases spread among wildlife populations and evaluate the effectiveness of disease management interventions. This ecological application of the SIR model supports efforts to protect biodiversity, maintain ecosystem health, and promote sustainable agricultural practices by mitigating the impact of diseases on plant and animal populations.
- **Social Sciences:** Social scientists apply the SIR model to analyze the spread of behaviors, social norms, and cultural practices within populations. By simulating behavioral dynamics using the SIR framework, researchers gain insights into how ideas, opinions, and innovations propagate through social networks and communities. For instance, the model helps study the adoption of health behaviors, political ideologies, and social movements, informing strategies to promote positive social change and address societal challenges (Centola, 2010). This application underscores the SIR model's role in understanding human interactions, social influence processes, and the diffusion of innovations across diverse social contexts.
- **Agriculture:** In agriculture, agronomists and plant pathologists use the SIR model to predict and manage the spread of diseases among crops and agricultural systems. By modeling disease transmission dynamics, the SIR model helps optimize disease control strategies, enhance crop yield, and promote sustainable farming practices. For example, researchers apply the model to forecast disease outbreaks, evaluate the efficacy of pest management techniques, and develop resilient agricultural systems that minimize the impact of diseases on crop production and food security (Gilligan et al, 2007). This agricultural application highlights the SIR model's contribution to improving agricultural productivity, sustainability, and resilience in the face of disease threats.
- **Education:** Educators and researchers utilize the SIR model to analyze educational dynamics, including the spread of knowledge, behaviors, and innovations within educational institutions and communities (Gilligan et al, 2007). By simulating the adoption of educational technologies, teaching methods, and policies using the SIR framework, researchers gain insights into factors influencing learning outcomes and educational practices. For example, the model helps study how educational innovations spread among students and educators, informing strategies to enhance teaching effectiveness, improve student engagement, and promote equitable access to quality education (Helena, 2016). This educational application underscores the SIR model's relevance in understanding educational dynamics and supporting evidence-based educational reforms and interventions.
- **War:** The SIR model has been adapted to study the dynamics of conflicts and wars, focusing on the spread of violence, strategies, and the impact of interventions (Macy & Willer, 2002). By modeling the transmission of military strategies, the SIR framework helps analyze the escalation and de-escalation of conflicts, assess the effectiveness of peacekeeping

missions, and predict the outcomes of military interventions. This application supports efforts to promote conflict resolution, peacebuilding, and global security by understanding the factors influencing conflict dynamics and guiding strategic decision-making (Helena, 2016). The SIR model's application in the context of war underscores its role in analyzing complex geopolitical interactions, humanitarian crises, and the management of global peace and security challenges.

CONCLUSION

The SIR (Susceptible-Infectious-Recovered) model has proven to be an indispensable tool in understanding the transmission dynamics of chickenpox, caused by the *varicella-zoster virus*, and in evaluating strategies for disease control and eradication. This article considered several key applications of the SIR model in epidemiology, highlighting its utility in predicting disease spread, assessing the impact of vaccination programs, and informing public health policies. Through sensitivity analysis, critical parameters influencing chickenpox transmission dynamics, such as contact rates and recovery rates, have been identified. This analysis emphasized the importance of high vaccination coverage and effective disease control measures in reducing disease incidence and achieving population immunity. The visual evidence provided by simulation results, particularly figures 3a and 3b, further supports the efficacy of these interventions over time, demonstrating a reduction in susceptibility and infection rates as the control measure increases.

The findings from this study showed that the eradication of chickenpox is within reach through sustained efforts in vaccination, treatment and public health interventions. The SIR model's ability to simulate various scenarios and predict outcomes plays a crucial role in guiding policy decisions aimed at controlling infectious diseases. By quantitatively assessing the impact of interventions on disease dynamics, the model facilitates evidence-based strategies that can minimize disease burden and enhance population health. The SIR model serves as a cornerstone in infectious disease epidemiology, offering insights into disease transmission mechanisms and supporting proactive measures for disease prevention and control. By considering its predictive capabilities and integrating comprehensive data-driven approaches, public health practitioners and policymakers can work towards achieving sustained reductions in chickenpox incidence and ultimately, the eradication of this infectious disease.

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