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A Compartmental Model for Chickenpox Transmission Dynamics Incorporating Control Interventions

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Abstract:

Chickenpox remains a highly contagious viral disease that poses significant public health challenges, particularly in populations with low vaccination coverage. Understanding its transmission dynamics is essential for designing effective control strategies. This study presents a comprehensive mathematical model to analyze the spread of Chickenpox, incorporating key intervention measures such as vaccination, isolation, and treatment. The model categorizes the human population into six compartments: susceptible, exposed, infected, isolated, treated, and recovered individuals. A system of ordinary differential equations is employed to describe the interactions among these groups. The model is further examined through the determination of disease-free and endemic equilibrium states, and stability analysis is conducted to establish the conditions under which the disease can either be eliminated or persist within the population. Sensitivity analysis is performed to identified the most influential parameters affecting disease transmission, revealing that transmission rates, vaccination coverage, and recovery rates play critical roles. Numerical simulations are also carried out to evaluate the impact of various intervention strategies. The results demonstrate that increased vaccination coverage, timely isolation of infected individuals, and effective treatment significantly reduce the number of new infections and overall disease burden. The findings highlight the importance of integrated public health strategies in controlling Chickenpox transmission. This study provides valuable insights that can inform policy decisions and guide the implementation of effective disease prevention and control measures.

Keywords: *Chickenpox transmission, Mathematical modeling, Stability analysis, Sensitivity analysis, Numerical simulation.*

Introduction

Chickenpox, also known as varicella, is a highly contagious viral infection caused by the varicella-zoster virus (VZV), a member of the herpesvirus family. It primarily affects children but can also occur in adults, where it tends to be more severe. The disease spreads through respiratory droplets or direct contact with infected lesions, making it highly transmissible in crowded environments such as schools and households [1][2]. Despite being generally self-limiting, chickenpox remains a public health concern globally due to its potential complications and continued outbreaks in areas with low vaccination coverage [1]. Clinically, chickenpox is characterized by a prodromal phase of fever, fatigue, and malaise, followed by the appearance of a vesicular rash that progresses through different stages, including macules, papules, vesicles, and crusts. A distinguishing feature is the presence of lesions at various stages simultaneously on the body [1]. While most cases resolve without complications, severe outcomes such as pneumonia, encephalitis, and secondary bacterial infections may occur, particularly among immunocompromised individuals, pregnant women, and adults [2]. Empirical clinical observations have consistently shown that complication rates are significantly higher in these high-risk populations [3].

Recent empirical studies have expanded understanding of the epidemiology of chickenpox. An observational study conducted in England between 2016 and 2022 revealed that chickenpox continues to impose a substantial burden on healthcare systems, particularly in regions without universal vaccination programs [4]. Similarly, a 2024 study assessing the impact of the COVID-19 pandemic on childhood infections found notable changes in the incidence and clinical patterns of chickenpox, suggesting that public health disruptions can influence transmission dynamics [5]. These findings highlight the evolving epidemiological patterns of the disease. Further empirical evidence from molecular and surveillance studies underscores the persistence and complexity of chickenpox outbreaks. A 2026 serological and molecular investigation in India identified ongoing transmission and multiple outbreaks across regions, emphasizing gaps in vaccination coverage and surveillance systems [6]. Additionally, metagenomic research conducted during a 2024 outbreak in Uganda found that 86% of suspected mpox-negative cases were actually caused by VZV, revealing a hidden burden of chickenpox that may be underdiagnosed in outbreak settings [7]. Such studies demonstrate the importance of laboratory confirmation and integrated disease surveillance.

The burden of chickenpox extends beyond the acute phase of infection, as the virus establishes latency in the host and can reactivate later in life as herpes zoster (shingles). This reactivation can lead to significant morbidity, including chronic pain and neurological complications [2]. A recent systematic review and meta-analysis (2023/2024) quantified the global prevalence of varicella-associated complications, confirming that although often mild, the disease can result in diverse and serious health outcomes affecting multiple organ systems [3]. These findings reinforce the need for comprehensive prevention strategies.

Vaccination remains the most effective preventive measure against chickenpox. The introduction of the live attenuated varicella vaccine has led to significant reductions in disease incidence, hospitalizations, and mortality in countries with established immunization programs [1]. Empirical modeling and epidemiological studies further suggest that widespread vaccination can drastically reduce transmission and contribute to herd immunity over time [8]. Therefore, strengthening vaccination programs, improving surveillance systems, and promoting public health awareness are essential steps toward controlling the disease globally [9][10].

Recent studies applied mathematical modeling techniques to better understand the transmission dynamics of chickenpox. El-Mesady and Ali [11] developed a fractional-order compartmental model that incorporated both vaccination and isolation strategies. The study demonstrated that these control measures significantly reduced the spread of infection. It further showed that fractional derivatives improved the model's ability to reflect real-world transmission patterns, making it more reliable for predicting disease trends. Adepoju et al [12] constructed a deterministic mathematical model focusing on childhood transmission of chickenpox. Their findings revealed the existence of disease-free and endemic equilibrium states and indicated that effective vaccination and treatment strategies reduced the basic reproduction number. The study concluded that sustained intervention efforts were essential for controlling the spread of the disease in susceptible populations.

Okafor et al [13] (analyzed chickenpox transmission using the Laplace Adomian Decomposition Method to solve nonlinear differential equations. The study showed that increasing vaccination rates significantly reduced infection prevalence. Through numerical simulations, the researchers confirmed the stability of the disease-free equilibrium under improved immunization coverage, emphasizing the importance of vaccination programs. Anwar et al [14] conducted a comprehensive scoping review of mathematical models of Plasmodium vivax transmission. The study found that models incorporating biological complexities such as relapse due to hypnozoites and host immunity produced more accurate predictions. It highlighted the importance of integrating biological realism into models to enhance their usefulness in guiding malaria control strategies. Anwar et al., [15] evaluated different modeling approaches used to assess intervention strategies. The researchers found that combining vector control, treatment, and immunity within models significantly improved the prediction of malaria transmission patterns. The study emphasized that multi-scale modeling, which integrates both within-host and population-level dynamics, provided more effective insights for policy formulation.

Kumar et al [16] developed a mathematical model based on real outbreak data from the United States. The study applied numerical and fractional modeling techniques and found that the model closely matched observed epidemiological data. It demonstrated that accurate parameter estimation was crucial for predicting outbreak progression and evaluating intervention strategies. Meharry [17] employed a data-driven deterministic model to examine the transmission dynamics of monkeypox. The study showed that incorporating real-time outbreak data improved the model's predictive accuracy. It also revealed that early intervention strategies, such as isolation and contact tracing,

significantly reduced disease spread, highlighting the importance of timely public health responses.

The aim of this study is to assess the effectiveness of treatment and isolation methods in reducing the transmission of chickenpox and to explore its transmission dynamics. By addressing the need for a deeper understanding of the mechanisms through which the disease spreads, this research seeks to provide valuable insights for managing its transmission and to offer potential policy recommendations for public health interventions. The objectives of this study are to: (i) develop a mathematical model that accurately represents the transmission patterns of chickenpox; (ii) determine the basic reproduction number associated with the proposed chickenpox model; (iii) analyze the stability of the model through a comprehensive examination; (iv) perform sensitivity analysis to evaluate the impact of individual parameters on the basic reproduction number; and (v) carry out numerical simulations of the model to validate specific theoretical results obtained in the study.

The novelty of this study lies in the development of a comprehensive compartmental model that captures the complex transmission dynamics of chickenpox by incorporating eight distinct population classes. The total population is stratified into Susceptible, Exposed, Asymptomatic Infectious, Symptomatic Infectious, Isolated Infectious, Treated Infectious, Recovered, and Vaccinated compartments, allowing for a more detailed and realistic representation of disease progression. Unlike many existing models, this study explicitly integrates both isolation and treatment strategies alongside vaccination, thereby providing a more holistic framework for evaluating control measures. Additionally, the inclusion of asymptomatic infectious individuals enhances the model's ability to reflect hidden transmission pathways often overlooked in simpler models. The study further distinguishes itself through the combined use of analytical techniques, including stability and sensitivity analysis, as well as numerical simulations to validate theoretical findings. This integrated approach offers deeper insights into the relative impact of epidemiological parameters and supports more informed public health decision-making aimed at reducing the spread of chickenpox.

Model Formulation

The total population (N) is divided into eight compartments: Susceptible (S), Exposed (E), Asymptomatic Infectious (I_A), Symptomatic Infectious (I_S), Isolated Infectious (I_I), Treated Infectious (T), Recovered (R), and Vaccinated (V). Thus, $N = S + E + I_A + I_S + I_I + T + R + V$. The recruitment rate of the population into the susceptible compartment is denoted as Λ. Susceptible individuals can become exposed through contact with different categories of infectious individuals at a transmission rate λ . Exposed individuals progress to the infectious compartment at a rate σ . Upon progression, individuals may become asymptomatic at a rate $(1-\alpha)\sigma$ or isolated infectious at a rate $\alpha\sigma$. Asymptomatic infectious individuals can progress to symptomatic infectious at a rate γ_A , while symptomatic infectious individuals can be isolated at a rate $(1-\rho)\gamma_S$ or treated at a rate $\rho\gamma_S$.

Each infectious compartment has distinct dynamics: asymptomatic infectious individuals (I_A) can progress to symptomatic state or experience disease-induced mortality at rate δ_A, symptomatic infectious individuals (I_S) can progress to isolated or treated states or experience disease-induced mortality at rate δ_S, isolated (I_I) individuals recover at rate τ_I or experience disease-induced mortality at rate δ_I, and treated (T) individuals recover at rate τ_T or experience disease-induced mortality at rate δ_T. The model incorporates disease-induced mortality rates δ_A, δ_S, δ_I, and δ_T for asymptomatic, symptomatic, isolated, and treated compartments, respectively. Additionally, the model accounts for a vaccination process, where susceptible individuals can be vaccinated at a rate θ, with a vaccine failure rate ε. Natural mortality μ is applied across all living compartments. Recovered individuals remain in the R compartment, and those in the V compartment represent vaccinated individuals. The system captures the complex transmission dynamics of the disease, accounting for multiple infectious states, treatment, isolation, and vaccination strategies.

The formulation of the model is based on several key assumptions to simplify and accurately represent the transmission dynamics of chickenpox. First, the model assumes homogeneous mixing within the population, meaning that all individuals have an equal probability of contact with one another and disease transmission occurs at a uniform rate. Second, individuals who recover from the infection are assumed to acquire permanent immunity, and therefore remain in the recovered compartment with no possibility of returning to the susceptible class. Third, the model incorporates vaccination as an important preventive strategy; however, it acknowledges the possibility of vaccine failure, implying that vaccination does not provide complete protection and that some vaccinated individuals may eventually return to the susceptible compartment.

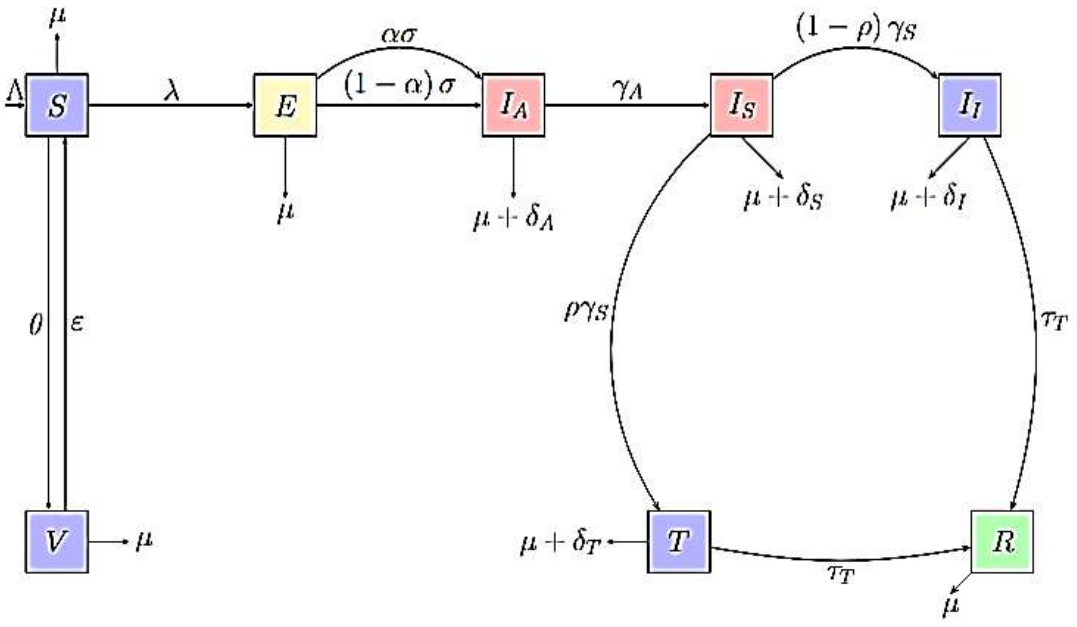


Figure 1: Schematic diagram of the Chickenpox model

Variable and Parameters description

Table 1: Variable Description

S	Susceptible population (individuals who can contract chickenpox)
E	Exposed population (individuals who have been exposed to chickenpox but are not yet infectious)
I_A	Asymptomatic infected population (individuals infected with chickenpox who show no symptoms)
I_S	Symptomatic infected population (individuals showing symptoms of chickenpox)
I_I	Isolated infected population (symptomatic individuals who are isolated to prevent spread)
T	Treatment population (individuals receiving medical care for chickenpox)
R	Recovered population (individuals who have recovered from chickenpox and are immune)
V	Vaccinated population (individuals who have been vaccinated against chickenpox)

Table 2: Parameter Description

Λ	Birth rate or immigration rate (related to new individuals entering the population)
λ	Force of infection (transmission rate of chickenpox)
θ	Vaccination rate (proportion of individuals vaccinated against chickenpox)
ε	Rate of vaccine waning immunity (how quickly immunity from vaccination decreases)
σ	Rate of progression from exposed to infected (time it takes for exposed individuals to show symptoms)
α	Fraction of exposed individuals who become isolated upon infection (to prevent spread)

γ_A	Rate of progression from asymptomatic to symptomatic (time from exposure to symptoms)
γ_S	Rate of progression from symptomatic infection (time to recovery from chickenpox)
ρ	Fraction of symptomatic individuals who receive treatment (medical care for symptomatic cases)
τ_T	Recovery rate from treatment (how quickly individuals recover when treated)
τ_I	Recovery rate from isolation (time taken to recover when isolated)
μ	Natural death rate (general mortality rate in the population)
δ_A	Disease-induced death rate for asymptomatic individuals (risk of death despite no symptoms)
δ_S	Disease-induced death rate for symptomatic individuals (risk of death during symptomatic phase)
δ_I	Disease-induced death rate for isolated individuals (risk of death while in isolation)
δ_T	Disease-induced death rate for individuals in treatment (risk of death despite receiving treatment)

MODEL EQUATIONS

In the light of the description of the model above, the differential equations modeling the transmission dynamics of Chickenpox in the population is given as

$$\frac{dS}{dt} = \Lambda - (\lambda + \theta + \mu) S + \varepsilon V$$

$$\frac{dV}{dt} = \theta S - (\varepsilon + \mu) V$$

$$\frac{dE}{dt} = \lambda S - (\sigma + \mu) E$$

$$\frac{dI_A}{dt} = (1-\alpha)\sigma E - (\gamma_A + \mu + \delta_A) I_A$$

$$\frac{dI_S}{dt} = \gamma_A I_A - (\gamma_S + \mu + \delta_S) I_S$$

$$\frac{dI_I}{dt} = \alpha\sigma E + (1-\rho)\gamma_S I_S - (\tau_I + \mu + \delta_I) I_I$$

$$\frac{dT}{dt} = \rho\gamma_S I_S - (\tau_T + \mu + \delta_T)T$$
$$\frac{dR}{dt} = \tau_T T + \tau_I I_I - \mu R$$

Where $\lambda = \frac{\beta_A I_A + \beta_S I_S + \beta_I I_I}{N}$ is the force of infection.

Let, $K_1 = (\theta + \mu)$, $K_2 = (\varepsilon + \mu)$, $K_3 = (1-\alpha)\sigma$, $K_4 = \alpha\sigma$, $K_5 = (1-\rho)\gamma_S$, $K_6 = \rho\gamma_S$

$P_1 = (\sigma + \mu)$, $P_2 = (\gamma_A + \mu + \delta_A)$, $P_3 = (\gamma_S + \mu + \delta_S)$, $P_4 = (\tau_I + \mu + \delta_I)$,
$P_5 = (\tau_T + \mu + \delta_T)$

Hence with this simplifications, the new differential equations modeling the transmission dynamics of chicken becomes

$$\frac{dS}{dt} = \Lambda - \lambda S - K_1 S + \varepsilon V$$
$$\frac{dV}{dt} = \theta S - K_2 V$$
$$\frac{dE}{dt} = \lambda S - P_1 E$$
$$\frac{dI_A}{dt} = K_3 E - P_2 I_A$$
$$\frac{dI_S}{dt} = \gamma_A I_A - P_3 I_S$$
$$\frac{dI_I}{dt} = K_4 E + K_5 I_S - P_4 I_I$$
$$\frac{dT}{dt} = K_6 I_S - P_5 T$$
$$\frac{dR}{dt} = \tau_T T + \tau_I I_I - \mu R$$

Analysis of the Chickenpox Model

Invariant region of the Chickenpox model

Theorem 1

The solutions of the proposed Chickenpox model are feasible for all $t > 0$, if they enter the invariant region D, which is given by:

$$D = \{(S, E, I_A, I_S, I_I, T, R, V) : S > 0, E > 0, I_A > 0, I_S > 0, I_I > 0, T > 0, R > 0, V > 0, N \le \frac{\Lambda}{\mu}\}.$$

Proof

The total population $N(t)$ of the Chickenpox model is given as:

$$N(t) = S(t) + E(t) + I_A(t) + I_S(t) + I_I(t) + T(t) + R(t) + V(t).$$

Adding all the differential equations of the model, we have:

$$\frac{dN}{dt} = \frac{dS}{dt} + \frac{dE}{dt} + \frac{dI_A}{dt} + \frac{dI_S}{dt} + \frac{dI_I}{dt} + \frac{dT}{dt} + \frac{dR}{dt} + \frac{dV}{dt}.$$

Substituting the equations of the model into the above expression, we get:

$$\frac{dN}{dt} = \Lambda - \mu(S + E + I_A + I_S + I_I + T + R + V) - (\delta_A I_A + \delta_S I_S + \delta_I I_I + \delta_T T).$$

Simplifying further, we obtain:

$$\frac{dN}{dt} \leq \Lambda - \mu N.$$

This is a standard first-order linear differential inequality. Solving it using the integrating factor method:

$$\frac{dN}{dt} + \mu N \leq \Lambda.$$

Multiplying through by the integrating factor $e^{\mu t}$:

$$\frac{d}{dt}(Ne^{\mu t}) \leq \Lambda e^{\mu t}.$$

Integrating both sides with respect to t, we have:

$$N(t)e^{\mu t} \leq \frac{\Lambda}{\mu} e^{\mu t} + C,$$

where C is the constant of integration. Dividing through by $e^{\mu t}$:

$$N(t) \leq \frac{\Lambda}{\mu} + \left(N(0) - \frac{\Lambda}{\mu} \right) e^{-\mu t}.$$

As $t \to \infty$, the exponential term $e^{-\mu t} \to 0$, hence:

$$N(t) \leq \frac{\Lambda}{\mu}.$$

Applying Birkhoff and Rota's theorem on the inequality, we obtain:

$$0 \leq N(t) \leq \frac{\Lambda}{\mu} \quad \text{as} \quad t \to \infty.$$

Thus, D is a positively invariant set under the flow described by the model equations. Therefore, no solution path leaves through the boundary of region D, and the model is epidemiologically and mathematically well-posed in this region.

Positivity of solution of the Chickenpox model

It is necessary to prove that all state variables of the Chickenpox model are nonnegative for all time t, ensuring that the model is epidemiologically and mathematically well-posed in a feasible region C, defined as:

$$C=\left\{(S,E,I_A,I_S,I_I,T,R,V): S>0,E>0,I_A>0,I_S>0,I_I>0,T>0,R>0,V>0,\frac{\Lambda}{\mu}\le N\right\}$$

Lemma 1

Let the initial conditions of the Chickenpox model satisfy

$$\left(S(0),E(0),I_A(0),I_S(0),I_I(0),T(0),R(0),V(0)\right)>0$$

.

Then, the solutions (S,E,I_A,I_S,I_I,T,R,V) of the model are positive for all $t>0$.

Proof

Let

$$t=\sup\left\{t>0: S>0,E>0,I_A>0,I_S>0,I_I>0,T>0,R>0,V>0,\ \text{for } t\in[0,t]\right\}.$$

Thus, $t>0$. From the first equation of the Chickenpox model:

$$\frac{dS}{dt}=\Lambda-\lambda S-\theta S+\varepsilon V-\mu S.$$

We observe that:

$$\frac{dS}{dt}\ge-(\lambda+\theta+\mu)S.$$

This inequality can be written as:

$$\int\frac{dS}{S}\ge-\int(\lambda+\theta+\mu)dt.$$

Integrating, we obtain:

$$\ln S\ge-(\lambda+\theta+\mu)t+C_1,$$

where C_1 is the constant of integration. Therefore:

$$S(t)\ge C_2e^{-(\lambda+\theta+\mu)t},$$

where $C_2=S(0)>0$. This shows that $S(t)\ge0$ for all $t>0$.

Similarly, we analyze the equations for E,I_A,I_S,I_I,T,R, and V:

In each case, following the same approach, we find that

$E(t),I_A(t),I_S(t),I_I(t),T(t),R(t),V(t)>0$ for all $t>0$, provided the initial conditions are positive.

Thus, the solutions $(S, E, I_A, I_S, I_I, T, R, V)$ of the Chickenpox model are positive for all time $t > 0$. This completes the proof.

Asymptotic stability of the disease-free equilibrium of the Chickenpox model

The disease-free equilibrium point (DFE) is the steady state where only the susceptible and vaccinated populations are non-zero, while all infected compartments are zero.

At the DFE, we have: $E^* = I_A^* = I_S^* = I_I^* = T^* = R^* = 0$

Setting $\frac{dS}{dt} = 0$ and $\frac{dV}{dt} = 0$ at equilibrium:

From

$\frac{dS}{dt} = \Lambda - \lambda S - K_1 S + \varepsilon V = 0$:

Since $\lambda = 0$ at DFE (no infected individuals),

we have: $\Lambda - K_1 S^* + \varepsilon V^* = 0$ ... (a)

From $\frac{dV}{dt} = \theta S^* - K_2 V^* = 0$:

$$\theta S^* = K_2 V^* \; V = \frac{\theta S^*}{K_2} \quad \text{... (b)}$$

Substituting equation (b) into equation (a):

$$\Lambda - K_1 S^* + \varepsilon \cdot \frac{\theta S^*}{K_2} = 0$$

$$\Lambda = K_1 S^* - \frac{\varepsilon \theta S^*}{K_2}$$

$$\Lambda = S^* \left(K_1 - \frac{\varepsilon \theta}{K_2} \right)$$

$$S^* = \frac{\Lambda}{K_1 - \frac{\varepsilon \theta}{K_2}} = \frac{\Lambda K_2}{K_1 K_2 - \varepsilon \theta}$$

And from equation (b):

$$V^* = \frac{\theta S^*}{K_2} = \frac{\theta \Lambda}{K_1 K_2 - \varepsilon\theta}$$

Since $K_1 = \theta + \mu$ and $K_2 = \varepsilon + \mu$:

$$K_1 K_2 - \varepsilon\theta = (\theta + \mu)(\varepsilon + \mu) - \varepsilon\theta$$

$$= \theta\varepsilon + \theta\mu + \mu\varepsilon + \mu^2 - \varepsilon\theta$$

$$= \mu(\theta + \varepsilon + \mu)$$

Therefore, the disease-free equilibrium is:

$$E_0 = \left(S^*, E^*, I_A^*, I_S^*, I_I^*, T^*, R^*, V^*\right)$$

Where:

$$S^* = \frac{\Lambda(\varepsilon + \mu)}{\mu(\theta + \varepsilon + \mu)}$$

$$V^* = \frac{\theta\Lambda}{\mu(\theta + \varepsilon + \mu)}$$

$$E^* = I_A^* = I_S^* = I_I^* = T^* = R^* = 0$$

This can be further simplified as:

$$S^* = \frac{\Lambda K_2}{\mu(K_1 + K_2 - \mu)}$$

$$V^* = \frac{\theta\Lambda}{\mu(K_1 + K_2 - \mu)}$$

Basic reproduction Number of the Chickenpox model

The basic reproduction number of Chickenpox infected individuals denoted by R_0 is defined as the average number of secondary infections produced by a single Chickenpox infectious individual introduced in a wholly susceptible population during his or her

entire infectious period. We calculate the basic reproduction number by using the next generation operator method on the dynamical system (1).

To find the basic reproduction number R_0, we use the next-generation matrix method.

We identify the infected compartments as E, I_A, I_S, I_I, and T.

The transmission matrix F (new infections) and transition matrix V (transitions out of infected compartments) are:

$$F = \begin{bmatrix} \dfrac{\beta_A I_A + \beta_S I_S + \beta_I I_I}{N} S^* \\ 0 \\ 0 \\ 0 \\ 0 \end{bmatrix}, \quad V = \begin{bmatrix} P_1 E \\ -K_3 E + P_2 I_A \\ -\gamma_A I_A + P_3 I_S \\ -K_4 E - K_5 I_S + P_4 I_I \\ -K_6 I_S + P_5 T \end{bmatrix}$$

The Jacobian matrices at DFE are:

$$\mathbf{F} = \begin{bmatrix} 0 & \dfrac{\beta_A S^*}{N^*} & \dfrac{\beta_S S^*}{N^*} & \dfrac{\beta_I S^*}{N^*} & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \end{bmatrix}$$

$$\mathbf{V} = \begin{bmatrix} P_1 & 0 & 0 & 0 & 0 \\ -K_3 & P_2 & 0 & 0 & 0 \\ 0 & -\gamma_A & P_3 & 0 & 0 \\ -K_4 & 0 & -K_5 & P_4 & 0 \\ 0 & 0 & -K_6 & 0 & P_5 \end{bmatrix}$$

The inverse of $\mathbf{V}$ is:

$$\mathbf{V}^{-1}=\begin{bmatrix} \frac{1}{P_1} & 0 & 0 & 0 & 0 \\ \frac{K_3}{P_1P_2} & \frac{1}{P_2} & 0 & 0 & 0 \\ \frac{K_3\gamma_A}{P_1P_2P_3} & \frac{\gamma_A}{P_2P_3} & \frac{1}{P_3} & 0 & 0 \\ \frac{K_4}{P_1P_4}+\frac{K_3\gamma_AK_5}{P_1P_2P_3P_4} & \frac{\gamma_AK_5}{P_2P_3P_4} & \frac{K_5}{P_3P_4} & \frac{1}{P_4} & 0 \\ \frac{K_3\gamma_AK_6}{P_1P_2P_3P_5} & \frac{\gamma_AK_6}{P_2P_3P_5} & \frac{K_6}{P_3P_5} & 0 & \frac{1}{P_5} \end{bmatrix}$$

The next-generation matrix is $\mathbf{K}=\mathbf{F}\mathbf{V}^{-1}$, and R_0 is its spectral radius.

Since only the first row of $\mathbf{F}$ is non-zero, the basic reproduction number is:

$$\mathrm{R}_0=\frac{S^*}{N^*}\left[\frac{\beta_AK_3}{P_1P_2}+\frac{\beta_SK_3\gamma_A}{P_1P_2P_3}+\beta_I\left(\frac{K_4}{P_1P_4}+\frac{K_3\gamma_AK_5}{P_1P_2P_3P_4}\right)\right]$$

Local Asymptotic Stability of the DFE of the Chickenpox Model

Theorem 2 *The disease-free equilibrium point of the Chickenpox model is locally asymptotically stable (LAS) if* $\mathrm{R}_0<1$*, and unstable if* $\mathrm{R}_0>1$*.*

Proof

The Jacobian matrix at the disease-free equilibrium point $J(E_0)$ is:

$$J(E_0)=\begin{bmatrix} -K_1 & 0 & -\frac{\beta_AS^*}{N^*} & -\frac{\beta_SS^*}{N^*} & -\frac{\beta_IS^*}{N^*} & 0 & 0 & \varepsilon \\ 0 & -P_1 & \frac{\beta_AS^*}{N^*} & \frac{\beta_SS^*}{N^*} & \frac{\beta_IS^*}{N^*} & 0 & 0 & 0 \\ 0 & K_3 & -P_2 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & \gamma_A & -P_3 & 0 & 0 & 0 & 0 \\ 0 & K_4 & 0 & K_5 & -P_4 & 0 & 0 & 0 \\ 0 & 0 & 0 & K_6 & 0 & -P_5 & 0 & 0 \\ 0 & 0 & 0 & 0 & \tau_I & \tau_T & -\mu & 0 \\ \theta & 0 & 0 & 0 & 0 & 0 & 0 & -K_2 \end{bmatrix}$$

To analyze stability, we examine the characteristic polynomial: $\det(J(E_0)-\lambda I)=0$

The matrix can be partitioned into blocks corresponding to the disease-free subsystem (S, V, R) and the infection subsystem (E, I_A, I_S, I_I, T).

For the disease-free subsystem, the eigenvalues are:

1. $\lambda_1 = -K_1$
2. $\lambda_2 = -K_2$
3. $\lambda_3 = -\mu$

All these eigenvalues are negative since all parameters are positive.

For the infection subsystem, the stability is determined by the dominant eigenvalue of the 5×5 submatrix:

$$J_{infection} = \begin{bmatrix} -P_1 & \frac{\beta_A S^*}{N^*} & \frac{\beta_S S^*}{N^*} & \frac{\beta_I S^*}{N^*} & 0 \\ K_3 & -P_2 & 0 & 0 & 0 \\ 0 & \gamma_A & -P_3 & 0 & 0 \\ K_4 & 0 & K_5 & -P_4 & 0 \\ 0 & 0 & K_6 & 0 & -P_5 \end{bmatrix}$$

The characteristics polynomial of $J_{infection}$ becomes

$$P(\lambda) = \lambda^5 + (P_1 + P_2 + P_3 + P_4 + P_5)\lambda^4 + [P_1P_2 + P_1P_3 + P_1P_4 + P_1P_5 + P_2P_3 + P_2P_4 + P_2P_5 + P_3P_4 + P_3P_5 + P_4P_5]\lambda^3$$

$$+[P_1P_2P_3 + P_1P_2P_4 + P_1P_2P_5 + P_1P_3P_4 + P_1P_3P_5 + P_1P_4P_5 + P_2P_3P_4 + P_2P_3P_5 + P_2P_4P_5 + P_3P_4P_5$$

$$-K_3\frac{\beta_A S^*}{N^*} - K_3\frac{\beta_S S^* \gamma_A}{N^*} - \frac{\beta_I S^* K_4}{N^*} - \frac{\beta_I S^* K_3 \gamma_A K_5}{N^*}]\lambda^2$$

$$+[P_1P_2P_3P_4 + P_1P_2P_3P_5 + P_1P_2P_4P_5 + P_1P_3P_4P_5 + P_2P_3P_4P_5$$

$$-K_3\frac{\beta_A S^*}{N^*}(P_3 + P_4 + P_5) - K_3\frac{\beta_S S^* \gamma_A}{N^*}(P_4 + P_5) - \frac{\beta_I S^* K_4}{N^*}(P_2 + P_3 + P_5)$$

$$-\frac{\beta_I S^* K_3 \gamma_A K_5}{N^*}(P_5)]\lambda + P_1P_2P_3P_4P_5(1 - \mathrm{R}_0)$$

Applying the Routh Hurwitz criterion, which states that to ensure the characteristics polynomial have negative real parts, the co-efficient of λ_i (for $i = 0,1,2,3,4,5$) must exceed zero (or be greater than zero), we have that, $(1 - \mathrm{R}_0) > 0$

And hence $\mathrm{R}_0 < 1$ and the disease-free equilibrium point of the chickenpox model is locally asymptotically stable.

Global Asymptotic Stability of the Disease-free equilibrium point of the Chickenpox Model

Theorem 3 *The disease-free equilibrium of the Chickenpox model is globally asymptotically stable whenever* $\mathrm{R}_0 \leq 1$.

Proof

Consider the following Lyapunov function:

$\mathrm{L} = c_1 E + c_2 I_A + c_3 I_S + c_4 I_I + c_5 T$, where c_1, c_2, c_3, c_4, and c_5 are positive constants to be determined.

Taking the time derivative of L along the solutions of the system, we have:

$$\frac{d\mathrm{L}}{dt} = c_1 \frac{dE}{dt} + c_2 \frac{dI_A}{dt} + c_3 \frac{dI_S}{dt} + c_4 \frac{dI_I}{dt} + c_5 \frac{dT}{dt}.$$

Substituting the model equations:

$$\frac{d\mathrm{L}}{dt} = c_1(\lambda S - P_1 E) + c_2(K_3 E - P_2 I_A) + c_3(\gamma_A I_A - P_3 I_S) + c_4(K_4 E + K_5 I_S - P_4 I_I) + c_5(K_6 I_S - P_5 T)$$

Rearranging:

$$\frac{d\mathrm{L}}{dt} = c_1 \lambda S + E(-c_1 P_1 + c_2 K_3 + c_4 K_4) + I_A(-c_2 P_2 + c_3 \gamma_A) + I_S(-c_3 P_3 + c_4 K_5 + c_5 K_6) + I_I(-c_4 P_4) + T(-c_5 P_5)$$

We choose the coefficients such that:

$c_2 K_3 + c_4 K_4 = c_1 P_1$,

$c_3 \gamma_A = c_2 P_2$,

$c_4 K_5 + c_5 K_6 = c_3 P_3$,

$c_4 P_4 = c_4 P_4$,

$c_5 P_5 = c_5 P_5$,

Let $c_1 = 1$. Then:

$$c_2 = \frac{K_3}{P_2}$$

$$c_3 = \frac{c_2 P_2}{\gamma_A} = \frac{K_3}{\gamma_A}$$

From $c_2 K_3 + c_4 K_4 = P_1$:

$$c_4 = \frac{P_1 - c_2 K_3}{K_4} = \frac{P_1 - \frac{K_3^2}{P_2}}{K_4}$$

From $c_4 K_5 + c_5 K_6 = c_3 P_3$:

$$c_5 = \frac{c_3 P_3 - c_4 K_5}{K_6}$$

With these choices, the derivative simplifies to:

$$\frac{d\mathrm{L}}{dt} = c_1 \lambda S - c_4 P_4 I_I - c_5 P_5 T$$

Since $\lambda = \frac{\beta_A I_A + \beta_S I_S + \beta_I I_I}{N} S$ and at the DFE $S^* \le N^*$,

we can write: $\frac{d\mathrm{L}}{dt} \le c_1 S^* \frac{\beta_A I_A + \beta_S I_S + \beta_I I_I}{N^*} - c_4 P_4 I_I - c_5 P_5 T$

This can be bounded as:

$$\frac{d\mathrm{L}}{dt} \le \left(\frac{c_1 S^*}{N^*} \right) \left[\frac{\beta_A K_3}{P_1 P_2} + \frac{\beta_S K_3 \gamma_A}{P_1 P_2 P_3} + \beta_I \left(\frac{K_4}{P_1 P_4} + \frac{K_3 \gamma_A K_5}{P_1 P_2 P_3 P_4} \right) \right] (E + I_A + I_S + I_I + T) - (\delta_A I_A + \delta_S I_S + \delta_I I_I + \delta_T T)$$

Since the bracketed expression equals R_0, we have:

$$\frac{d\mathrm{L}}{dt} \le K(\mathrm{R}_0 - 1)(E + I_A + I_S + I_I + T)$$

where $K > 0$ is a positive constant.

When $\mathrm{R}_0 \leq 1$:

1. $\frac{d\mathrm{L}}{dt} \leq 0$
2. $\frac{d\mathrm{L}}{dt} = 0$ if and only if $E = I_A = I_S = I_I = T = 0$

By LaSalle's Invariance Principle, since L is a Lyapunov function on the feasible region and the largest invariant set where $\frac{d\mathrm{L}}{dt} = 0$ is the disease-free equilibrium E_0, every solution to the system approaches the disease-free equilibrium point as $t \to \infty$ when $\mathrm{R}_0 \leq 1$. Hence the Chickenpox model is globally asymptotically stable.

Endemic Equilibrium Point of the Chickenpox Model

Theorem 4 *The endemic equilibrium point of the chickenpox model exists and is stable if* $\mathrm{R}_0 > 1$.

Proof

To find the endemic equilibrium of the chickenpox model, we set each derivative to zero:

$$\frac{dS}{dt} = \frac{dV}{dt} = \frac{dE}{dt} = \frac{dI_A}{dt} = \frac{dI_S}{dt} = \frac{dI_I}{dt} = \frac{dT}{dt} = \frac{dR}{dt} = 0$$

Let the endemic equilibrium be represented by $\left(S^*, V^* E^* I_A^* I_S^*, I_I^* T^* R^*\right)$.

From the equilibrium equations:

From $\frac{dV}{dt} = 0$: $V^* = \frac{\theta S^*}{K_2}$

From $\frac{dE}{dt} = 0$: $E^* = \frac{\lambda^* S^*}{P_1}$

From $\frac{dI_A}{dt} = 0$: $I_A^* = \frac{K_3 E^*}{P_2} = \frac{K_3 \lambda^* S^*}{P_1 P_2}$

From $\frac{dI_S}{dt} = 0$: $I_S^* = \frac{\gamma_A I_A^*}{P_3} = \frac{K_3 \gamma_A \lambda^* S^*}{P_1 P_2 P_3}$

From $\frac{dI_I}{dt} = 0$: $I_I^* = \frac{K_4 E^* + K_5 I_S^*}{P_4} = \frac{\lambda^* S^*}{P_1 P_4}(K_4 + \frac{K_3 \gamma_A K_5}{P_2 P_3})$

From $\frac{dT}{dt} = 0$: $T^* = \frac{K_6 I_S^*}{P_5} = \frac{K_3 \gamma_A K_6 \lambda^* S^*}{P_1 P_2 P_3 P_5}$

From $\frac{dR}{dt} = 0$: $R^* = \frac{\tau_T T^* + \tau_I I_I^*}{\mu}$

From $\frac{dS}{dt} = 0$: $\Lambda - \lambda^* S^* - K_1 S^* + \varepsilon V^* = 0$

Substituting $V^* = \frac{\theta S^*}{K_2}$: $\Lambda - \lambda^* S^* - K_1 S^* + \varepsilon \frac{\theta S^*}{K_2} = 0$

$$\Lambda = S^* \left(\lambda^* + K_1 - \frac{\varepsilon\theta}{K_2} \right)$$

The force of infection is: $\lambda^* = \frac{\beta_A I_A^* + \beta_S I_S^* + \beta_I I_I^*}{N^*}$

Substituting the expressions for I_A^*, I_S^*, and I_I^*:

$$\lambda^* = \frac{\lambda^* S^*}{N^*} \left[\frac{\beta_A K_3}{P_1 P_2} + \frac{\beta_S K_3 \gamma_A}{P_1 P_2 P_3} + \beta_I \left(\frac{K_4}{P_1 P_4} + \frac{K_3 \gamma_A K_5}{P_1 P_2 P_3 P_4} \right) \right]$$

Since the bracketed expression equals $N^* \mathrm{R}_0 / S^*$ (from our R_0 calculation), we have:

$$\lambda^* = \frac{\lambda^* S^*}{N^*} \cdot \frac{N^* \mathrm{R}_0}{S^*} = \lambda^* \mathrm{R}_0$$

For a non-trivial solution ($\lambda^* \neq 0$), we need $\mathrm{R}_0 = 1$, which gives the transcritical bifurcation point.

For $\mathrm{R}_0 > 1$, we can solve the system to get: $\lambda^* = \frac{\Lambda(\mathrm{R}_0 - 1)}{S^*(\mathrm{R}_0 - 1) + N^*(K_1 - \frac{\varepsilon\theta}{K_2}) / \mathrm{R}_0}$

This gives positive values for all endemic state variables when $\mathrm{R}_0 > 1$.

Hence, the endemic equilibrium point of the chickenpox model is stable whenever $\mathrm{R}_0 > 1$

Sensitivity Analysis of the Chickenpox model

Definition 1

The sensitivity index of the reproduction number R_0 with respect to any parameter x is given by: $\Im_x^{R_0} = \frac{\partial R_0}{\partial x} \times \frac{x}{R_0}$

The expanded form of the basic reproduction number is given as:

$$\mathrm{R}_0 = \frac{\varepsilon+\mu}{\theta+\varepsilon+\mu}\cdot\frac{\sigma}{\sigma+\mu} \times\left[\frac{\beta_A(1-\alpha)}{\gamma_A+\mu+\delta_A}+\frac{\beta_S(1-\alpha)\gamma_A}{(\gamma_A+\mu+\delta_A)(\gamma_S+\mu+\delta_S)}+\frac{\beta_I\alpha}{\tau_I+\mu+\delta_I}+\frac{\beta_I(1-\alpha)\gamma_A(1-\rho)\gamma_S}{(\gamma_A+\mu+\delta_A)(\gamma_S+\mu+\delta_S)(\tau_I+\mu+\delta_I)}\right]$$

Let us define the components:

1. $A=\dfrac{\varepsilon+\mu}{\theta+\varepsilon+\mu}$
2. $B=\dfrac{\sigma}{\sigma+\mu}$
3. $C_1=\dfrac{\beta_A(1-\alpha)}{\gamma_A+\mu+\delta_A}$
4. $C_2=\dfrac{\beta_S(1-\alpha)\gamma_A}{(\gamma_A+\mu+\delta_A)(\gamma_S+\mu+\delta_S)}$
5. $C_3=\dfrac{\beta_I\alpha}{\tau_I+\mu+\delta_I}$
6. $C_4=\dfrac{\beta_I(1-\alpha)\gamma_A(1-\rho)\gamma_S}{(\gamma_A+\mu+\delta_A)(\gamma_S+\mu+\delta_S)(\tau_I+\mu+\delta_I)}$

So $\mathrm{R}_0 = A\cdot B\cdot(C_1+C_2+C_3+C_4)$

1. Sensitivity with respect to β_A:

$$\Im_{\beta_A}^{\mathrm{R}_0}=\frac{C_1}{C_1+C_2+C_3+C_4} =\frac{\dfrac{\beta_A(1-\alpha)}{\gamma_A+\mu+\delta_A}}{\dfrac{\beta_A(1-\alpha)}{\gamma_A+\mu+\delta_A}+\dfrac{\beta_S(1-\alpha)\gamma_A}{(\gamma_A+\mu+\delta_A)(\gamma_S+\mu+\delta_S)}+\dfrac{\beta_I\alpha}{\tau_I+\mu+\delta_I}+\dfrac{\beta_I(1-\alpha)\gamma_A(1-\rho)\gamma_S}{(\gamma_A+\mu+\delta_A)(\gamma_S+\mu+\delta_S)(\tau_I+\mu+\delta_I)}}$$

2. Sensitivity with respect to β_S:

$$\Im_{\beta_S}^{\mathrm{R}_0}=\frac{C_2}{C_1+C_2+C_3+C_4} =\frac{\dfrac{\beta_S(1-\alpha)\gamma_A}{(\gamma_A+\mu+\delta_A)(\gamma_S+\mu+\delta_S)}}{\dfrac{\beta_A(1-\alpha)}{\gamma_A+\mu+\delta_A}+\dfrac{\beta_S(1-\alpha)\gamma_A}{(\gamma_A+\mu+\delta_A)(\gamma_S+\mu+\delta_S)}+\dfrac{\beta_I\alpha}{\tau_I+\mu+\delta_I}+\dfrac{\beta_I(1-\alpha)\gamma_A(1-\rho)\gamma_S}{(\gamma_A+\mu+\delta_A)(\gamma_S+\mu+\delta_S)(\tau_I+\mu+\delta_I)}}$$

3. Sensitivity with respect to β_I :

$$\Im_{\beta_I}^{R_0} = \frac{C_3 + C_4}{C_1 + C_2 + C_3 + C_4}$$

$$= \frac{\dfrac{\beta_I \alpha}{\tau_I + \mu + \delta_I} + \dfrac{\beta_I (1-\alpha)\gamma_A (1-\rho)\gamma_S}{(\gamma_A + \mu + \delta_A)(\gamma_S + \mu + \delta_S)(\tau_I + \mu + \delta_I)}}{\dfrac{\beta_A (1-\alpha)}{\gamma_A + \mu + \delta_A} + \dfrac{\beta_S (1-\alpha)\gamma_A}{(\gamma_A + \mu + \delta_A)(\gamma_S + \mu + \delta_S)} + \dfrac{\beta_I \alpha}{\tau_I + \mu + \delta_I} + \dfrac{\beta_I (1-\alpha)\gamma_A (1-\rho)\gamma_S}{(\gamma_A + \mu + \delta_A)(\gamma_S + \mu + \delta_S)(\tau_I + \mu + \delta_I)}}$$

4. Sensitivity with respect to γ_A :

$$\Im_{\gamma_A}^{R_0} = -\frac{\gamma_A}{\gamma_A + \mu + \delta_A} \cdot \frac{C_1 + C_2 + C_4}{C_1 + C_2 + C_3 + C_4} + \frac{\gamma_A \cdot (C_2 + C_4)}{(\gamma_A + \mu + \delta_A)(C_1 + C_2 + C_3 + C_4)}$$

5. Sensitivity with respect to γ_S :

$$\Im_{\gamma_S}^{R_0} = -\frac{\gamma_S}{\gamma_S + \mu + \delta_S} \cdot \frac{C_2 + C_4}{C_1 + C_2 + C_3 + C_4} + \frac{(1-\rho)\gamma_S \cdot C_4}{(\gamma_S + \mu + \delta_S)(C_1 + C_2 + C_3 + C_4)}$$

6. Sensitivity with respect to δ_A : $\Im_{\delta_A}^{R_0} = -\dfrac{\delta_A}{\gamma_A + \mu + \delta_A} \cdot \dfrac{C_1 + C_2 + C_4}{C_1 + C_2 + C_3 + C_4}$

7. Sensitivity with respect to δ_S : $\Im_{\delta_S}^{R_0} = -\dfrac{\delta_S}{\gamma_S + \mu + \delta_S} \cdot \dfrac{C_2 + C_4}{C_1 + C_2 + C_3 + C_4}$

8. Sensitivity with respect to δ_I : $\Im_{\delta_I}^{R_0} = -\dfrac{\delta_I}{\tau_I + \mu + \delta_I} \cdot \dfrac{C_3 + C_4}{C_1 + C_2 + C_3 + C_4}$

9. Sensitivity with respect to α (progression to severe infection probability):

$$\Im_{\alpha}^{R_0} = \frac{\alpha}{1-\alpha} \cdot \frac{C_3 - C_1 - C_2 - C_4}{C_1 + C_2 + C_3 + C_4}$$

10. Sensitivity with respect to ρ (progression to treatment probability):

$$\Im_{\rho}^{R_0} = -\frac{\rho}{1-\rho} \cdot \frac{C_4}{C_1 + C_2 + C_3 + C_4}$$

11. Sensitivity with respect to θ (vaccination rate): $\Im_{\theta}^{R_0} = -\dfrac{\theta}{\theta + \varepsilon + \mu}$

12. Sensitivity with respect to ε (vaccine waning rate): $\Im_{\varepsilon}^{\mathrm{R}_0} = \dfrac{\varepsilon}{\theta+\varepsilon+\mu}$

13. Sensitivity with respect to σ (incubation rate): $\Im_{\sigma}^{\mathrm{R}_0} = \dfrac{\mu}{\sigma+\mu}$

14. Sensitivity with respect to μ (natural death rate):

$$\Im_{\mu}^{\mathrm{R}_0} = \frac{\theta}{\theta+\varepsilon+\mu} - \frac{\sigma}{\sigma+\mu} - \frac{\mu}{\gamma_A+\mu+\delta_A}\cdot\frac{C_1+C_2+C_4}{C_1+C_2+C_3+C_4} - \frac{\mu}{\gamma_S+\mu+\delta_S} \times \frac{C_2+C_4}{C_1+C_2+C_3+C_4} - \frac{\mu}{\tau_I+\mu+\delta_I}\cdot\frac{C_3+C_4}{C_1+C_2+C_3+C_4}$$

By substituting the parameter values in table **4**, we obtained the final sensitivity indices of each parameter on the basic reproduction number of chickenpox as follows

Table 3: Table of sensitivity Indices on R_0

Parameter	Value
β_A	0.226565
β_S	0.640672
β_I	0.132762
σ	0.000587
α	-0.049694
γ_A	-0.226345
γ_S	-0.717105
δ_A	-0.000680
δ_S	-0.007379
δ_I	-0.016296
ρ	-0.419420
θ	-0.864636
ò	0.118443
μ	-0.135396

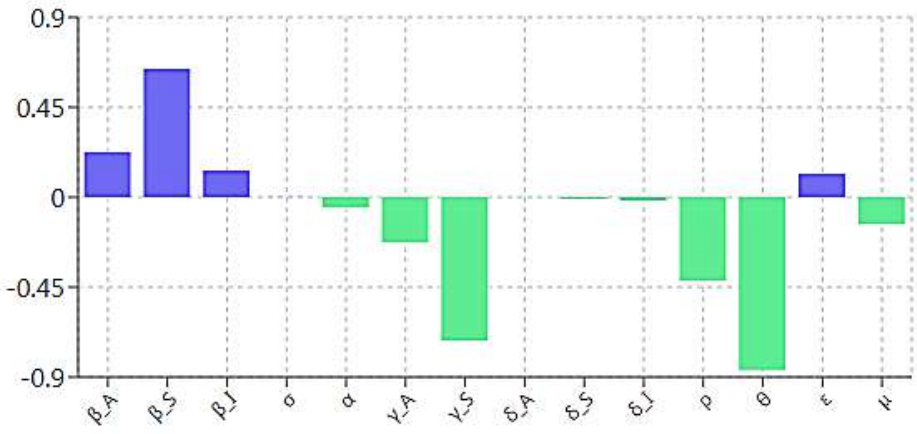


Figure 2: Sensitivity Bar chart of the Chickenpox model

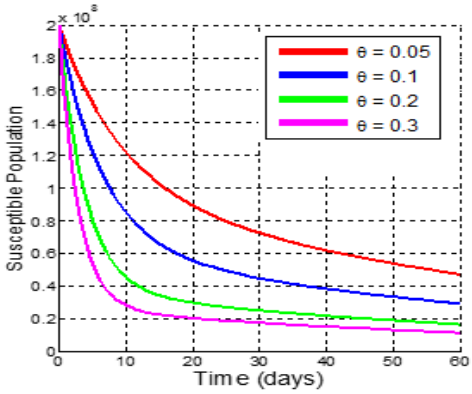
From the sensitivity analysis above, we observed that parameters with positive sensitivity indices which includes β_A, β_S, β_I and ε with positive sensitivity indices will increase the burden of chickenpox in the human population. Whereas, parameters with negative sensitivity indices which includes σ, α, γ_a, δ_A, μ will reduce the prevalence and burden of chickenpox in the human population.

Table 4: Parameter table of values

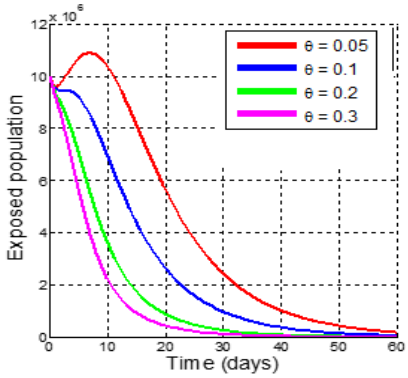
Symbol	Value	Unit	Source
β_A	0.6	per day	[18]
β_S	1.2	per day	[25]
β_I	0.8	per day	[20]
σ	1/15	per day	[1]
α	0.05	dimensionless	[21]
γ_A	1/7	per day	[23]
γ_S	1/10	per day	[29]
τ_I	1/14	per day	[22]
ρ	0.8	dimensionless	[27]
δ_A	0.0001	per day	[19]
δ_S	0.001	per day	[26]
δ_I	0.01	per day	[28]
δ_T	0.005	per day	[33]
θ	0.002	per day	[31]
ε	1/(10×365)	per day	[24,30]
μ	1/(70×365)	per day	[32]

Λ	0.02×N	per day	[31,33]

4.8. Numerical Simulation of Chickenpox

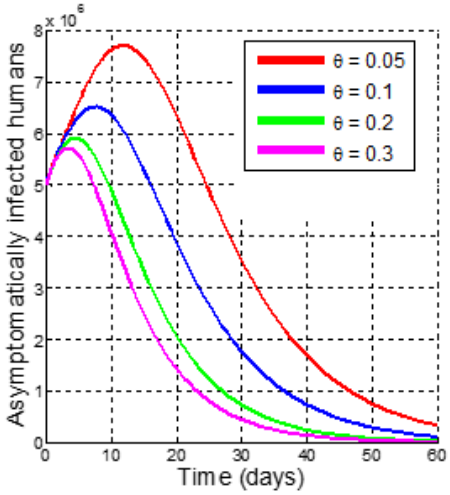


a. Effect of varying β_S on susceptible Humans

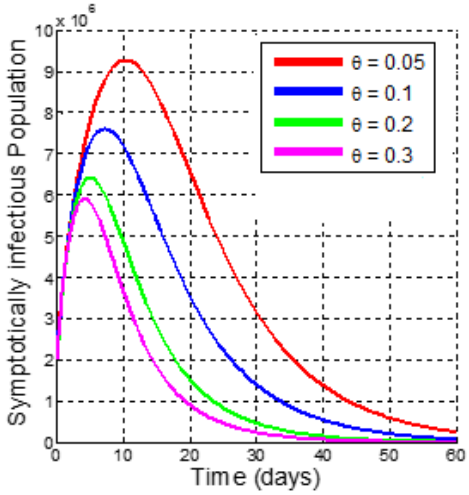


b. Effect of varying β_S on Exposed Humans

Figure 3

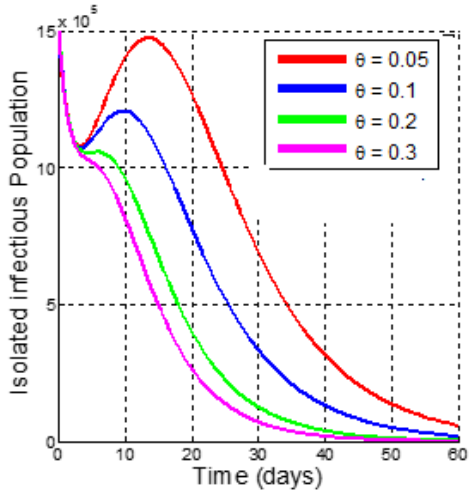


a. Effect of varying β_S on I_A Humans

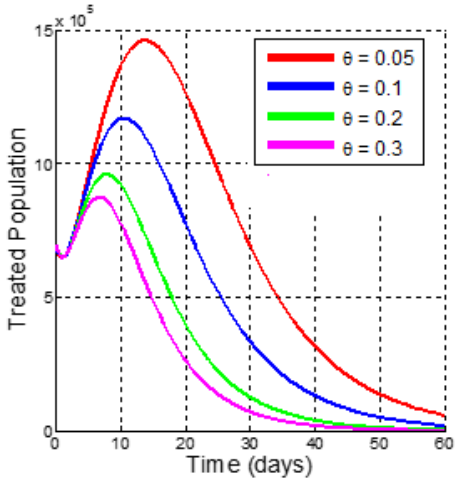


b. Effect of varying β_S on I_S Humans

Figure 4

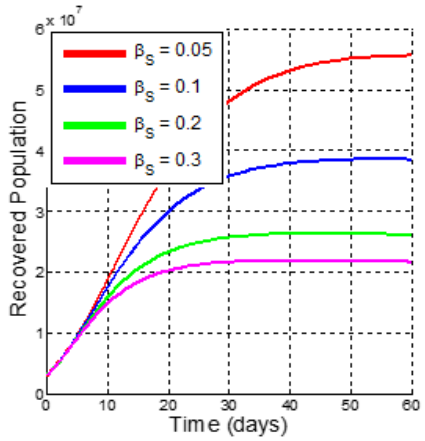


a. Effect of varying β_S on I_I Humans

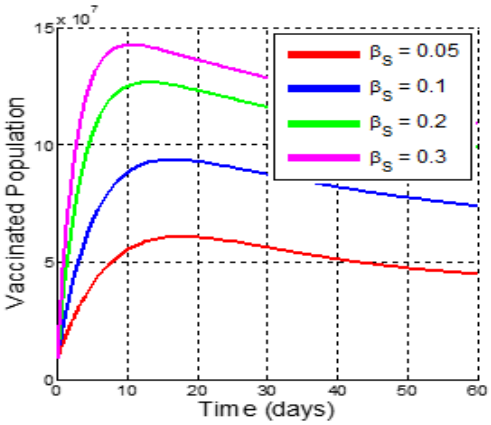


b. Effect of varying β_S on I_T Humans

Figure 5

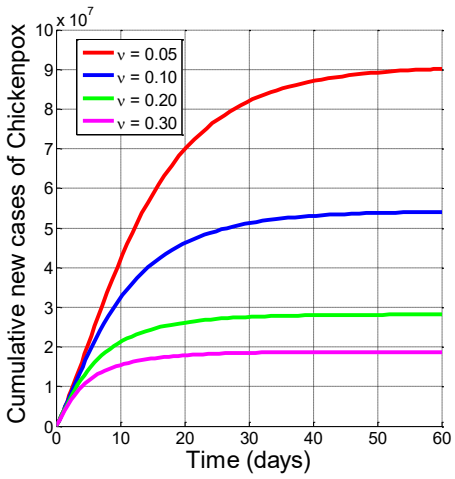


a. Effect of varying β_S on R Humans

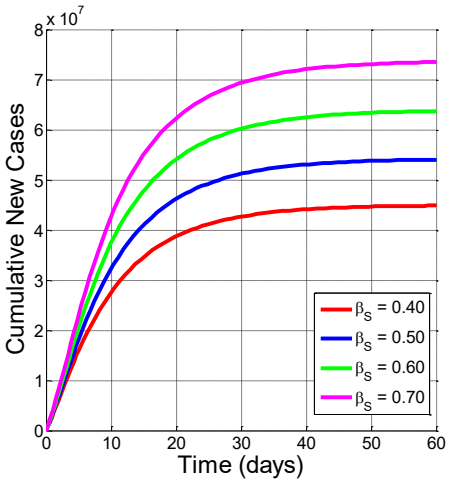


b. Effect of varying β_S on the V class

Figure 6

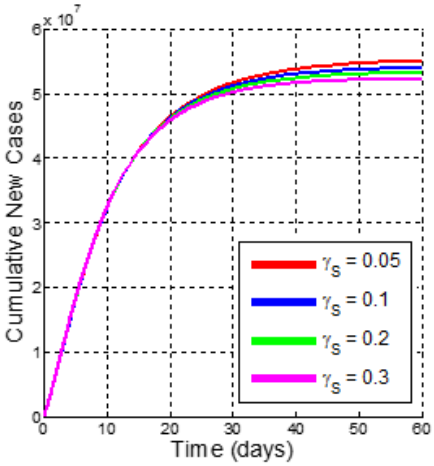


b. Effect of varying V on the cumulative new cases of chickenpox

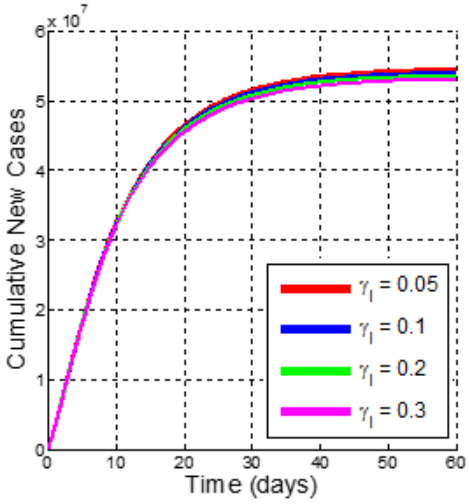


b. Effect of varying β_S on the cumulative new cases of chickenpox

Figure 7



a. Effect of varying γ_S on the cumulative new cases chickenpox



b. Effect of varying γ_I on the cumulative new cases of chickenpox

Figure 8

From Figure **3a**, it is observed that increasing the vaccination rate reduces the population of susceptible humans due to the high efficacy of the Chickenpox vaccine. This indicates that more individuals in the population become less susceptible to the disease through consistent efforts to enhance vaccination strategies. A fraction of susceptible individuals who refuse vaccination progress to the exposed class, as reflected in the gradual increase in the exposed population shown in Figure 3b. However, due to the highly infectious nature of the Chickenpox virus, exposed individuals gradually transition into either

symptomatic or asymptomatic infectious classes, depending on their immunity levels. The subsequent decrease in the exposed population aligns with the observed increases in Figures **4a** and **4b**. To effectively curb the spread of the disease, symptomatic infectious individuals are isolated from the general population and moved into isolation centers, as depicted by the steady rise in Figure **5a**. This measure reduces transmission via human-to-human contact. Isolated individuals are further transferred to treatment facilities for proper medical care, as shown by the increase in Figure **5b**. Effective medical attention in this treatment class facilitates the recovery of infected individuals, as evidenced by the rising population of recovered individuals in Figure **6a**. The vaccinated population also increases, as shown in Figure **6b**, due to the vaccination of susceptible individuals. However, the waning efficacy of the Chickenpox vaccine, explains the decrease observed in the same figure over time.

The cumulative new cases of Chickenpox represent the total number of individuals who have contracted the disease within a specific period, aggregated from the beginning of an epidemic or observation. This metric provides a comprehensive count of all new infections, accounting for every instance when a susceptible individual transitions to an infected state after exposure to the virus. From Figure **7b**, it is evident that an increase in the contact rate between susceptible and symptomatically infected individuals leads to a rise in cumulative new cases. Conversely, Figures **7a**, **8a**, and **8b** show that increasing the vaccination rate of susceptible individuals (V), the isolation rate of infected individuals (γ_{IS}), and the treatment rate of isolated infected individuals significantly decreases the cumulative new cases of the disease.

CONCLUSION

This study provides a comprehensive analysis of the transmission dynamics of chickenpox through the development and application of a mathematical model that incorporates key epidemiological compartments, including susceptible, exposed, asymptomatic infectious, symptomatic infectious, isolated, treated, recovered, and vaccinated individuals. The model successfully captures the complexity of disease spread and highlights the interactions between different population groups and intervention strategies. Through stability analysis and equilibrium characterization, the study establishes a solid theoretical foundation for understanding the conditions under which chickenpox can either be eradicated or persist within a population. These analytical results provide important insights into the threshold conditions required for effective disease control. The sensitivity analysis and numerical simulations reveal that vaccination, isolation, and treatment are critical determinants of the epidemic trajectory. In particular, high vaccination coverage significantly reduces the susceptible population, thereby limiting the potential for outbreaks. Similarly, timely isolation of infectious individuals plays a vital role in interrupting transmission chains, while effective treatment reduces the duration and severity of infection. The results also emphasize that failure in any of these control measures can lead to increased transmission and possible endemicity. The observed trends in the vaccinated and treated compartments underscore the importance of sustained investment in healthcare systems, including vaccination programs and treatment facilities. Overall, this study highlights that an integrated

approach combining vaccination, isolation, and treatment is essential for minimizing the spread and impact of chickenpox and for improving public health outcomes.

Based on the findings of this study, several recommendations are proposed to enhance the control and prevention of chickenpox. There is a need for intensified public health education and awareness campaigns to inform individuals about the importance of vaccination, early detection, isolation, and proper treatment. Such campaigns should emphasize the safety and effectiveness of the chickenpox vaccine and encourage timely immunization, particularly among children and high-risk groups. In addition, governments and health authorities should ensure that vaccination programs are accessible, affordable, and widely implemented, including school-based immunization initiatives and targeted interventions in underserved communities. Addressing vaccine hesitancy through culturally sensitive and evidence-based communication strategies is also crucial. Moreover, strengthening healthcare infrastructure is essential to support efficient diagnosis, isolation, and treatment of infected individuals. This includes training healthcare personnel on best practices in chickenpox management and ensuring the availability of adequate medical resources. Effective isolation protocols should be reinforced to limit person-to-person transmission, while comprehensive treatment strategies should be implemented to reduce complications and speed up recovery. Additionally, robust surveillance systems should be established to monitor disease trends, assess intervention effectiveness, and identify high-risk populations for targeted action. Community engagement and collaboration among stakeholders including healthcare providers, policymakers, and researchers are vital for implementing sustainable and evidence-based interventions. Finally, strong governmental support and policy frameworks are necessary to integrate chickenpox prevention into broader public health strategies, ensuring long-term sustainability and improved preparedness against future outbreaks.

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