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Numerical Simulation And Stability Analysis Of A Differential Equation–Based Infectious Disease Transmission Model

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ABSTRACT

In order to understand the dynamics of infectious disease transmission and to promote effective public health interventions, mathematical modelling is essential. For the purpose of describing the spread of disease within a community, this paper develops and analyses an ordinary differential equation based compartmental infectious disease model. There are three sections in the population model: vulnerable, infected, and recovered. Stability analysis is performed utilizing eigenvalue methods and basic reproduction number principles after analytical examination to find the disease-free and endemic equilibrium points. To study the disease's temporal evolution under different parameter values, numerical simulations are run using standard numerical techniques. The effects of the initial population distribution, recovery rate, and transmission rate on illness dynamics are shown by the simulation results. The results demonstrate that numerical simulation is useful for forecasting the actions of outbreaks and evaluating methods of disease control. This study lays the groundwork for future work in mathematics and computational epidemiology that can handle more complicated cases.

Keywords: *Infectious disease modeling, differential equations, stability analysis, numerical simulation, SIR model, epidemiology.*

1. INTRODUCTION

As seen by the frequent outbreaks of influenza, COVID-19, dengue fever, and many other communicable diseases, infectious diseases continue to pose a significant and ongoing threat to international public health systems. In addition to causing high rates of morbidity and mortality, these illnesses place a heavy social and financial cost on civilisations all over the world. The transmission of infectious organisms has been exacerbated by rapid population expansion, increased urbanisation, globalisation, and climate change, making epidemic management and control more difficult. Therefore, it is crucial to comprehend the mechanisms by which infectious diseases spread throughout communities in order to develop efficient prevention, mitigation, and control measures.

Mathematical modelling is an effective and systematic way to study the dynamics of the transmission of infectious diseases. Researchers can utilise models to objectively study the reasons of illness spread by transforming mathematical formulas from biological and epidemiological processes. Because of their versatility, ease of use, and ability to capture essential elements of disease transmission, differential equation-based compartmental models are among the most extensively used modelling tools. The models depict the transitions between various epidemiological compartments, such as susceptible, infected, and recovered individuals, using ordinary

differential equations. Using this model, we can track the effects of key epidemiological variables on disease dynamics, including population vulnerability, transmission rates, and recovery rates.

Numerical simulation is essential to the research of infectious disease models, in addition to offering analytical insights. Numerical approaches are essential for generating approximate solutions because many epidemiological systems are highly nonlinear and analytically difficult. Researchers can assess the possible effects of intervention measures, visualise the temporal evolution of disease transmission, and conduct scenario and sensitivity assessments under various parameter settings using numerical simulations. When it comes to predicting outbreak trends and assisting with data-driven public health decision-making, these simulations are especially useful.

An infectious disease transmission model based on differential equations is the subject of the current work, which aims to build, evaluate, and numerically simulate the model. First, we want to build a model that describes the basic dynamics of infectious disease transmission. Second, we want to analyse the stability properties of the disease-free and endemic equilibrium points using well-established mathematical techniques. Finally, we want to use numerical simulations to look at how the model changes over time. The results of this study should shed light on the efficacy of mathematical and numerical approaches to epidemiological research and our understanding of disease transmission.

2. REVIEW OF LITERATURE

Peddinti and Sabbani (2024) developed a comprehensive mathematical framework for modeling infectious disease spread using systems of differential equations. Their study integrated epidemiological insights with nonlinear dynamical analysis to capture transmission dynamics effectively. The authors analyzed equilibrium points and stability conditions, demonstrating how model parameters influenced disease persistence or eradication.

Costa et al. (2021) reviewed key concepts and applications of mathematical modeling in infectious disease epidemiology. The study discussed classical compartmental models such as SIR and SEIR and explained their relevance in understanding disease transmission mechanisms. The authors highlighted how mathematical models supported public health decision-making by estimating transmission rates and assessing intervention strategies. Their work demonstrated the importance of integrating theoretical models with empirical data.

Li (2018) provided a foundational introduction to the mathematical modeling of infectious diseases, focusing on model construction, analysis, and interpretation. The book explained core epidemiological concepts and established rigorous mathematical approaches for analyzing disease dynamics using differential equations. Li presented numerous examples and case studies that illustrated the practical usefulness of mathematical models in predicting disease spread and evaluating control measures. This work served as a fundamental reference for subsequent research in infectious disease modelling.

2. MATHEMATICAL MODEL FORMULATION

Mathematical modelling provides a structured framework for characterizing infectious illness transmission and studying their dynamic behaviour across time. Here, we build a compartmental model using ordinary differential equations to show how infectious diseases spread inside a contained population. The model not only reflects the interplay among susceptible, infected, and recovered individuals, but it also incorporates crucial epidemiological

components regulating disease transmission and recovery. Using a set of simplifying assumptions ensures that the analysis is clear and tractable.

2.1. Model Assumptions

- A number of assumptions underpin the development of the mathematical model. First, that there would be no changes in the demographics of the population as a whole as a result of migration, births, or deaths during the study period.
- Diseases spread through direct touch between susceptible and infected people, and it is presumed that the population is homogeneously mixed so that every individual has an equal chance of coming into contact with any other individual in the population.
- Individuals who recover from the disease acquire permanent immunity and do not return to the susceptible class.
- Natural births and deaths are neglected to simplify the model and focus exclusively on disease transmission dynamics.

These assumptions allow the model to concentrate on the fundamental mechanisms of disease spread while maintaining mathematical simplicity.

2.2. Model Compartments

There are three distinct epidemiological subsets that make up the entire population:

- People who are vulnerable, "S(t)" stands for "healthy but susceptible to infection" people.
- Infected people, I(t): People who have the disease and can spread it to others who are vulnerable.
- "Recovered individuals," abbreviated as R(t), are those who have fully recovered from the disease and developed a resilient immune system.

The total population size, Ni, is given by at any given time t:

$$N = S(t) + I(t) + R(t)$$

Because it is presumed that the population is constant, N stays the same across time.

2.3. Governing Equations

The dynamics of disease transmission are controlled by the following system of ordinary differential equations, which are based on the assumptions and the compartmental structure:

$$\frac{dS}{dt} = -\beta SI$$
$$\frac{dI}{dt} = \beta SI - \gamma I$$
$$\frac{dR}{dt} = \gamma I$$

As the susceptible population declines as a result of fewer new infections caused by close contact with infected people, the first equation shows the rate of change in this population. The dynamics of the infected population, including both new infections and recoveries, are described by the second equation. When sick people get better, the third equation predicts that the recovered population will grow.

In this context, the transmission rate (β) stands for the likelihood of disease transfer per encounter between a susceptible and an infected person, while the recovery rate (γ) indicates the pace at which infected people become well and join the recovered group.

As a whole, these equations lay the groundwork for future stability analyses and numerical simulations of infectious disease transmission dynamics, and they are both simple and powerful.

3. EQUILIBRIUM POINTS AND STABILITY ANALYSIS

A key element of mathematical epidemiology is stability analysis, which sheds light on the long-term dynamics of disease transmission models. It is feasible to ascertain if an infectious illness will become extinct or continue to exist in a population by locating equilibrium points and analysing their stability characteristics. The equilibrium states of the suggested differential equation-based model are determined and examined in this section. The endemic and disease-free equilibria are given special consideration, as is the function of the fundamental reproduction number in controlling system stability.

3.1. Disease-Free Equilibrium (DFE)

In a disease-free equilibrium, the disease has no effect on the population at all. This happens when there are no infected people in the population and everyone is vulnerable (and maybe even recovered). The disease-free equilibrium can be expressed mathematically as:

$$E_0 = (S_0, 0,0)$$

where S_0represents the initial susceptible population. This state of balance is useful for assessing the likelihood of disease invasion because it depicts a healthy population that is not currently contagious.

3.2. Basic Reproduction Number

An important threshold parameter in epidemiological modelling is the fundamental reproduction number, R0. It is the average number of secondary infections caused by an infected person brought into a group that is entirely susceptible. A definition of R0 for the model being suggested is:

$$R_0 = \frac{\beta S_0}{\gamma}$$

The possibility of disease transmission is determined by the value of $R0$.

- The illness will eventually go extinct if the average number of secondary infections generated by affected individuals is less than one, as shown in $R0<1$.
- The disease can spread across the population and stay there for a long period if $R0> 1$.

Hence, $R0$ determines whether an outcome is disease-free or endemic.

3.3. Stability of the Disease-Free Equilibrium

The stability of the disease-free equilibrium is examined by linearizing the system of nonlinear differential equations around E0. At the disease-free equilibrium, this is accomplished by computing the system's Jacobian matrix and evaluating its eigenvalues. These eigenvalues' stability characteristics are defined by the signs of their real components.

- This research shows that for R0<1, the disease-free equilibrium is locally asymptotically stable, meaning that tiny changes in the number of affected people will eventually lead to the elimination of the disease.
- With R0>1, the disease-free equilibrium is no longer stable, suggesting that even a small number of sick people can cause the disease to spread over time.

These findings demonstrate how important the fundamental reproduction number is for predicting the success or failure of disease control efforts.

3.4. Endemic Equilibrium

The model allows for an endemic equilibrium where the fundamental reproduction number is greater than one, meaning that the disease remains constant in the population. In this state of balance, there is still some infection in the population since the rates of new infections and recoveries are equal.

The endemic equilibrium is found to be locally asymptotically stable under suitable parameter circumstances, according to stability analysis. This consistency indicates that the disease will continue to affect the population for a long time despite efforts to control it. The need for intervention techniques to decrease transmission or increase recovery rates to bring R0 below unity is highlighted by the presence of a stable endemic equilibrium.

4. NUMERICAL SIMULATION

For the analysis of infectious disease models, numerical simulation is a crucial tool, especially when system nonlinearity makes it difficult or impossible to find closed-form analytical solutions. The time evolution of disease transmission under different initial conditions and parameter settings can be studied numerically. This section describes the parameter settings utilised in the simulations, describes the numerical method used to solve the suggested system of differential equations, and talks about the disease dynamics that resulted.

4.1. Numerical Method

Using the fourth-order Runge-Kutta (RK4) method, the infectious illness model's governing system of nonlinear ordinary differential equations is numerically solved. When dealing with initial value problems, this approach is popular since it is accurate, stable, and computationally efficient. By iteratively updating the values of the state variables based on weighted averages of intermediate slopes, the RK4 system delivers trustworthy approximations over time.

The chosen starting conditions are based on a plausible epidemiological scenario where a sizable segment of the population is vulnerable but only a tiny percentage is infected at the outset. In this configuration, we may study how diseases spread through populations and how they behave throughout their early stages of growth.

4.2. Simulation Parameters

To investigate the behavior of the model, simulations are performed using representative parameter values commonly adopted in epidemiological studies. The transmission rate is set to $\beta = 0.3$, reflecting moderate infectiousness, while the recovery rate is chosen as $\gamma = 0.1$, corresponding to an average infectious period of ten-time units. The initial population distribution assumes that 95% of individuals are susceptible and 5% are infected, with no recovered individuals at the initial time.

These parameter values are used to analyze baseline disease dynamics and to study the impact of parameter variation on infection spread and control.

4.3. Simulation Results

The outcomes of the numerical simulation offer important new information on how the illness has changed over time. Because there are many susceptible individuals, the infected population has an initial expansion phase. This is followed by a peak infection level and a subsequent decline as susceptibility and recovery both increase. The typical breakout pattern seen in many infectious diseases is reflected in this behaviour.

Additional investigation reveals that greater transmission rates lead to larger epidemic peaks and faster disease dissemination, both of which indicate a more severe outbreak. Higher recovery rates, on the other hand, show how well isolation and treatment tactics work by drastically lowering the length and severity of infection. As more people contract the infection, the susceptible population declines monotonically, whereas the recovered population grows gradually and eventually reaches saturation.

Graphical representations of $S(t)$, $I(t)$, and $R(t)$over time clearly illustrate these dynamics, offering an intuitive visualization of disease progression and recovery trends. These results reinforce the analytical findings and emphasize the importance of numerical simulation in understanding and predicting infectious disease behavior.

5. DISCUSSION

There is good agreement between the theoretical stability findings and the numerical simulations. The quick decrease of infections confirms the stability of the disease-free equilibrium when R0<1. When R0>1, on the other hand, endemic behaviour is induced via persistent transmission. The model emphasises the significance of prompt interventions like immunisation, quarantine, and treatment by demonstrating how disease dynamics are sensitive to characteristics pertaining to transmission and recovery.

Although the model is simplified, it provides valuable qualitative insights into disease spread. Extensions of the model could include demographic effects, latency periods, vaccination strategies, and stochastic influences to better represent real-world scenarios.

6. CONCLUSION

In order to investigate the dynamic behaviour of disease dissemination within a community, this study creates and evaluates a differential equation-based infectious disease transmission model. The conditions under which the disease either dies out or continues are clearly shown through thorough stability analysis, underscoring the crucial role that important epidemiological characteristics play in controlling transmission dynamics. By offering a thorough, time-dependent visualisation of the evolution of susceptible, infected, and recovered populations under various parameter settings, numerical simulations support the analytical conclusions. The model's interpretability

and practical usefulness are improved by the deeper insights into the mechanisms behind illness spread and control that are provided by the combination of stability analysis and numerical simulation. All things considered, the suggested framework is a strong starting point that can easily expanded to include more biological realism, intervention techniques, and intricate structures, making it appropriate for the examination of both current and developing infectious diseases.

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