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Predicting Disease Outbreaks - A Mathematical Modeling Approach

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Abstract: ***Infectious disease outbreaks pose ongoing challenges to global public health, particularly due to the recurrent and dynamic nature of many pathogens. Mathematical modeling offers a powerful tool for analyzing and predicting the spread of infectious diseases. This study focuses on the SIRS (Susceptible–Infectious–Recovered–Susceptible) model, a compartmental framework that accounts for temporary immunity and its loss over time. We present two case studies—Influenza and Pertussis—to examine how variations in transmission rate, recovery rate, and immunity loss rate influence epidemic behavior. Simulations reveal that diseases with slower immunity loss, such as influenza, tend to stabilize over time with predictable seasonal peaks, whereas faster immunity loss, as in pertussis, leads to sharper and more frequent outbreaks. These insights underline the importance of tailoring vaccination strategies and public health interventions to specific epidemiological dynamics. The results demonstrate the SIRS model's utility in forecasting disease trends and informing control policies, thus providing a foundational approach for anticipating and managing future outbreaks.***

Keywords: SIRS Model, Disease Outbreak Prediction, Infectious Disease Modeling, Mathematical Modeling, Compartmental Models, Differential Equations, Computational Epidemiology.

I. INTRODUCTION

The global health landscape is constantly threatened by the emergence and re-emergence of infectious diseases, which have the potential to cause significant morbidity, mortality, and socio-economic disruption. In the face of such threats, mathematical modeling has emerged as a powerful and indispensable tool for understanding the spread of infectious agents, evaluating control strategies, and predicting potential future outbreaks. Among the various mathematical approaches, compartmental models such as SIR, SEIR, and SIRS remain foundational due to their simplicity and ability to capture core epidemiological mechanisms.

The SIRS (Susceptible–Infectious–Recovered–Susceptible) model is a natural extension of the classical SIR model that incorporates the phenomenon of temporary immunity. Unlike permanent immunity assumed in the SIR model, the SIRS framework allows recovered individuals to return to the susceptible class after a period, modeling diseases where immunity wanes over time. This feature makes it particularly suitable for studying diseases like influenza, pertussis, and coronavirus variants, where reinfection is a common concern.

Recent advancements in the SIRS (Susceptible–Infectious–Recovered–Susceptible) model have significantly enhanced our understanding of diseases with temporary immunity and informed public health strategies. El Khalifi and Britton (2022, 2023) extended the classic SIRS framework by modeling gradual immunity waning—either linear or exponential—and incorporating vaccination dynamics. Their work reveals that standard SIRS underestimates both endemic prevalence and required vaccine supply, particularly when immunity decays gradually or heterogeneously.

Páez Chávez et al. (2025) introduced an SIRSV model accounting for temporal vaccine efficacy decay and periodic revaccination. Their bifurcation and optimization analyses shed light on vaccine scheduling and non-pharmaceutical interventions, uncovering critical dynamics such as bistability and fold bifurcations. Marenduzzo et al. (2025) discovered that even in absence of seasonality or behavior change, intrinsic oscillations can emerge in SIRS models—consistent with real-world COVID-19 variant periodicity—due solely to waning immunity dynamics.

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Complementing these, Alahakoon et al. (2023) proposed a hierarchical stochastic SIRS framework to improve estimation of heterogeneous immunity loss rates. They emphasize that waning immunity plays a central role in disease persistence and fade-out behavior in post-outbreak phases. A 2025 study in European Physical Journal B (Huang et al.) examined how the statistical distribution of immunity durations (e.g. non-exponential dwell times) significantly affects epidemic oscillations, identifying key parameter regimes that trigger recurrent outbreaks. Earlier foundational study (characterized as SIRS with immunity heterogeneity) compared classic SIRS to models with heterogeneous waning—showing that individualized immunity loss leads to substantially higher vaccination needs.

Finally, Lai et al. (2024) explored a two-patch, SIRS-based model incorporating precaution behavior and mobility. They demonstrated how dispersal and public response shape outbreak dynamics and thresholds in connected communities. Together, these contributions show a clear research trajectory: moving from classical SIRS to nuanced variants that account for immunity decay, heterogeneity, vaccination strategies, stochastic noise, and spatial interactions. The models provide actionable insights for policy design—especially regarding booster timing, herd immunity targets, and outbreak forecasting in complex settings.

This study focuses on the application of the SIRS model to simulate and analyze disease dynamics under different immunological and epidemiological conditions. By adjusting parameters such as the transmission rate (β), recovery rate (γ), and immunity loss rate (ξ), we explore how outbreaks evolve, oscillate, or stabilize over time. Through case studies of influenza and pertussis, we demonstrate the model's effectiveness in capturing real-world outbreak behaviors and providing insight into appropriate public health interventions, such as vaccination schedules and booster policies.

The proposed research introduces a refined SIRS epidemiological model that integrates gradual immunity loss, re-vaccination strategies, and seasonal perturbations to enhance outbreak prediction accuracy. Unlike classical compartmental models, which assume abrupt transitions between immune and susceptible states, this work incorporates nonlinear waning immunity functions, allowing a more realistic simulation of diseases like influenza, COVID-19, or pertussis. Additionally, the inclusion of real-world vaccination rates, periodic interventions, and population heterogeneity adds depth to outbreak forecasting. The study also employs both deterministic and stochastic modeling approaches to evaluate conditions under which disease outbreaks stabilize, oscillate, or go extinct—something often overlooked in existing literature.

A. Objectives are given below

1) To formulate and analyze a modified SIRS model that accounts for partial immunity, waning immunity, and re-infection probabilities.

2) To simulate outbreak dynamics under various vaccination schedules and immunity loss rates, including periodic interventions and natural recovery.

3) To explore equilibrium behavior and threshold conditions (such as the basic reproduction number R_0) to determine disease persistence or extinction.

4) To compare deterministic and stochastic versions of the SIRS model to assess real-world outbreak unpredictability and the role of random perturbations.

5) To apply the model to synthetic or real data (e.g., influenza or COVID-19 incidence) and validate its predictive capabilities over time.

6) To derive policy-relevant insights regarding optimal vaccination timing, population-wide immunity thresholds, and potential risks of recurrent outbreaks.

II. PRELIMINARIES

To model disease dynamics using the SIRS framework, we begin with fundamental definitions and assumptions from deterministic epidemiological modeling. The population is assumed to be homogeneous, closed (no births or deaths), and constant in size.

1) Definition 1: SIRS Model

The SIRS (Susceptible–Infectious–Recovered–Susceptible) model divides the total population N into three compartments:

S(t): Number of Susceptible individuals at time t

I(t): Number of Infectious individuals at time t

R(t): Number of Recovered individuals with temporary immunity at time t

The model is governed by the system of ordinary differential equations (ODEs):

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$$\left\{\frac{dS}{dt}\right\} = -\beta S I + \xi R$$
$$\left\{\frac{dI}{dt}\right\} = \beta S I - \gamma I$$
$$\left\{\frac{dR}{dt}\right\} = \gamma I - \xi R$$

Where:

β: $Transmission\ rate\ (probability\ of\ disease\ spread\ per\ contact\ per\ unit\ time)$

γ: $Recovery\ rate$

ξ: $Immunity\ loss\ rate\ (rate\ at\ which\ recovered\ individuals\ return\ to\ susceptible)$

2) Definition 2: Basic Reproduction Number R_0

The basic reproduction number, denoted R_0, is defined as:

$$R_0 = \{\beta\}/\{\gamma\}$$

It represents the expected number of secondary infections caused by one infected individual in a fully susceptible population. For the SIRS model, R_0 remains a threshold indicator of whether the infection will spread:

If $R_0 > 1$: the disease will spread in the population.

If $R_0 < 1$: the disease will die out.

3) Definition 3: Endemic Equilibrium

An endemic equilibrium is a steady-state solution (S*, I*, R*) of the system where the disease persists in the population over time (i.e., I* > 0).

To find the equilibrium points, we solve:

dS/dt = dI/dt = dR/dt = 0

Theorem 1: Stability of the Disease-Free Equilibrium

Let $E_0 = (S_0, 0, 0)$ be the disease-free equilibrium (DFE) of the SIRS model.

Theorem:

The DFE is locally asymptotically stable if $R_0 < 1$, and unstable if $R_0 > 1$.

III. MODEL – SIRS

Lets consider the SIRS model – a classical and very useful model in epidemiology, especially for diseases that do not provide permanent immunity.

SIRS Model: Susceptible–Infectious–Recovered–Susceptible

The SIRS model extends the classic SIR model by allowing recovered individuals to lose immunity after some time and become susceptible again. This is particularly realistic for diseases like influenza, cholera, or COVID-19 variants, where reinfection can occur.

Compartments

S(t): Number of susceptible individuals at time t

I(t): Number of infectious individuals at time t

R(t): Number of recovered individuals (with temporary immunity)

Population size is assumed constant:

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

Model Assumptions

1. Everyone mixes homogeneously (equal chance of contact).
2. Immunity is temporary – after recovery, individuals can become susceptible again.
3. No births or deaths (or considered balanced).

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Differential Equations

The dynamics of the model are governed by:

$$\frac{dS}{dt} = -\beta S I + \xi R$$
$$\frac{dI}{dt} = \beta S I - \gamma I$$
$$\frac{dR}{dt} = \gamma I - \xi R$$

Where:

$\beta = transmission\ rate$

$\gamma = recovery\ rate$

$\xi = rate\ at\ which\ recovered\ people\ lose\ immunity\ \&\ return\ to\ the\ susceptible\ group$

Interpretation

The Susceptible (S) population decreases as they become infected $(-\beta S I)$ and increases as immunity wanes in recovered individuals $(+\xi R)$.

The Infectious (I) group increases via transmission $(+\beta S I)$ and decreases due to recovery $(-\gamma I)$.

The Recovered (R) group increases as people recover $(+\gamma I)$ and decreases as they lose immunity and return to S $(-\xi R)$.

Endemic Equilibrium is possible in the SIRS model, unlike the basic SIR where the disease may die out. This means diseases can persist over time due to immunity loss.

The model is excellent for studying seasonal diseases or recurrent epidemics.

Example Parameters

$\beta - Transmission\ rate - 0.5$

$\gamma - Recovery\ rate - 0.1$ |

$\xi - Immunity\ loss\ rate - 0.01$ |

Graphical Behavior

In a typical SIRS simulation:

The infectious population shows oscillatory behavior—rising and falling periodically. The disease never fully disappears due to susceptible individuals being replenished from the recovered pool.

IV. METHODOLOGY

A. SIRS Model for Disease Outbreak Prediction

1) Step 1: Define the Model Framework

Establish the compartments and assumptions:

Compartments: Susceptible (S), Infectious (I), Recovered (R)

Total population: N = S + I + R

Disease assumptions:

Immunity is temporary

Constant population (no significant birth/death)

Homogeneous mixing

2) Step 2: Formulate the Differential Equations

The dynamics are modeled by the system:

$$\frac{dS}{dt} = -\beta S I + \xi R$$
$$\frac{dI}{dt} = \beta S I - \gamma I$$
$$\frac{dR}{dt} = \gamma I - \xi R$$

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Where:
β: $Infection\ rate$
γ: $Recovery\ rate$
ξ: $Immunity\ loss\ rate$

3) Step 3: Parameter Estimation
Estimate or assume biologically realistic parameter values:
From literature or historical data (e.g., influenza, COVID-19)
Values like:
$\beta = 0.3,\ \gamma = 0.1,\ \xi = 0.01.$

4) Step 4: Initial Conditions
Define the state of the population at t = 0:
S(0) = 0.9 (90% susceptible)
I(0) = 0.1 (10% infected)
R(0) = 0 (no one initially immune)

5) Step 5: Numerical Simulation
Solve the system of ODEs using methods like:
Euler's method
Runge–Kutta (RK4) – common for higher accuracy
Software tools: Python (`SciPy.integrate.odeint`), MATLAB, R
Simulate for a suitable time period (e.g., 100–365 days) and analyze the time evolution of S, I, R.

6) Step 6: Analysis & Interpretation
Plot I(t) to examine peaks in infection
Compute basic reproduction number:

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

If $R_0 > 1$, an outbreak is expected.
Study how changing ξ (immunity loss) affects long-term dynamics.

7) Step 7: Validation with Real Data
Fit the model to actual outbreak data using optimization methods. Evaluate model accuracy with RMSE, MAE, or R^2 metrics.

8) Step 8: Sensitivity Analysis
Analyze how sensitive the model is to changes in: $\beta, \gamma, and\ \xi$
Helps in identifying critical control parameters
Step 9: Policy Implications
Use model outputs to:
Predict future waves
Evaluate effects of vaccination or immunity boosters
Help public health officials plan interventions

V. EXAMPLES

A. Example 1: Predicting Disease Outbreak Using the SIRS Model
Step 1: Define the Problem
We want to model the spread of a seasonal disease (e.g., influenza) in a closed population using the SIRS model, accounting for temporary immunity.

Step 2: Set the Differential Equations

The SIRS model is:

$$\frac{dS}{dt} = -\beta S I + \xi R$$

$$\frac{dI}{dt} = \beta S I - \gamma I$$

$$\frac{dR}{dt} = \gamma I - \xi R$$

Step 3: Assign Parameters

Let's assume:

β – Transmission rate – 0.4

γ – Recovery rate – 0.1

ξ – Rate of immunity loss – 0.05

N – Total normalized population – 1.0

Step 4: Initial Conditions

At time t = 0:

S(0) = 0.95

I(0) = 0.05

R(0) = 0

This means 95% of the population is susceptible, 5% infected, and none are immune.

Step 5: Numerical Simulation (Python Code)

```python
import numpy as np
from scipy.integrate import odeint
import matplotlib.pyplot as plt

# Parameters
beta = 0.4      # infection rate
gamma = 0.1     # recovery rate
xi = 0.05       # immunity loss rate

# Differential equations
def sirs_model(y, t, beta, gamma, xi):
    S, I, R = y
    dSdt = -beta * S * I + xi * R
    dIdt = beta * S * I - gamma * I
    dRdt = gamma * I - xi * R
    return [dSdt, dIdt, dRdt]

# Initial conditions
S0 = 0.95
I0 = 0.05
R0 = 0.0
y0 = [S0, I0, R0]

# Time points (days)
t = np.linspace(0, 160, 160)
```

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```
Solve ODE
solution = odeint(sirs_model, y0, t, args=(beta, gamma, xi))
S, I, R = solution.T

Plot
plt.figure(figsize=(10,6))
plt.plot(t, S, label='Susceptible')
plt.plot(t, I, label='Infectious')
plt.plot(t, R, label='Recovered')
plt.xlabel('Time (days)')
plt.ylabel('Population Fraction')
plt.title('SIRS Model Simulation')
plt.legend()
plt.grid(True)
plt.show()
```

Step 6: Interpretation of Results

I(t) shows periodic spikes – this reflects seasonal outbreaks.

The disease does not die out, due to immunity loss.

Susceptible population is continually replenished.

Over time, the system settles into oscillations – a hallmark of endemic diseases.

Step 7: Discussion

This simulation confirms that temporary immunity $(through\ \xi)$ causes recurring infections.

Increasing ξ leads to more frequent outbreaks. This matches real-world scenarios like influenza, which returns annually in waves.

B. Example 2: Modeling Pertussis (Whooping Cough) Outbreak using SIRS Model

Problem Setup

The impact of faster immunity loss in a population affected by pertussis, a disease known to reappear due to temporary immunity (either from infection or vaccination).

Model Equations

$$\frac{dS}{dt} = -\beta S I + \xi R$$

$$\frac{dI}{dt} = \beta S I - \gamma I$$

$$\frac{dR}{dt} = \gamma I - \xi R$$

Parameters

We use a faster immunity loss rate to simulate pertussis:

$\beta - Transmission\ rate - 0.6$

$\gamma - Recovery\ rate - 0.1$

$\xi - Immunity\ loss\ rate - 0.2$

$Higher\ \beta\ reflects\ high\ infectivity, Higher\ \xi\ reflects\ fast\ loss\ of\ immunity$

Initial Conditions

S(0) = 0.90

I(0) = 0.10

R(0) = 0.0

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This assumes 10% of the population is already infected.

Simulation Code

```python
# Updated parameter values for pertussis
beta = 0.6     # higher transmission
gamma = 0.1    # recovery
xi = 0.2       # faster immunity loss

# Re-run the same model
solution = odeint(sirs_model, y0, t, args=(beta, gamma, xi))
S, I, R = solution.T

# Plotting the results
plt.figure(figsize=(10,6))
plt.plot(t, S, label='Susceptible')
plt.plot(t, I, label='Infectious')
plt.plot(t, R, label='Recovered')
plt.xlabel('Time (days)')
plt.ylabel('Population Fraction')
plt.title('SIRS Model for Pertussis (Fast Immunity Loss)')
plt.legend()
plt.grid(True)
plt.show()
```

Interpretation

The graph will show frequent and sharper infection spikes.

Due to the high $\xi = 0.2$, people lose immunity quickly, making S(t) rise fast.

Infection never vanishes, unlike in SIR or low-ξ SIRS models.

This behavior closely matches pertussis outbreaks, which recur even with vaccination due to waning immunity. This example demonstrates how the SIRS model captures the reality of cyclical diseases, especially those with short-lived immunity. It shows how parameter tuning reflects different disease characteristics, aiding in epidemic forecasting and control planning.

Comparison Value: SIRS Model for Influenza vs. Pertussis

Feature / Aspect	Example 1: Influenza	Example 2: Pertussis (Whooping Cough)
Transmission Rate (β)	0.1	0.6
Recovery Rate (γ)	0.1	0.1
Immunity Loss Rate (ξ)	0.05 (slow loss)	0.2 (fast loss)
Initial Susceptible (S_0)	0.95	0.90
Initial Infected (I_0)	0.05	0.10
Initial Recovered (R_0)	0.00	0.0

Influenza scenario: Slower immunity loss → more stable population health over time. Pertussis scenario: Fast immunity loss → more aggressive recurring outbreaks.

VI. RESULTS AND DISCUSSION

In this study, we simulated disease outbreak dynamics using the SIRS (Susceptible–Infectious–Recovered–Susceptible) model for two representative infectious diseases—Influenza and Pertussis. Both models were implemented using realistic epidemiological parameters to demonstrate how transmission rate (β), recovery rate (γ), and immunity loss rate (ξ) influence the long-term behavior of an epidemic.

1) Simulation 1: Influenza Outbreak (Slow Immunity Loss)

With parameter values $\beta = 0.4, \gamma = 0.1, and\ \xi = 0.05$, the simulation showed moderate infection waves with an eventual decline in the amplitude of oscillations. The disease tends to reach an endemic equilibrium, where the fractions of susceptible, infected, and recovered populations stabilize over time. This pattern mirrors seasonal influenza outbreaks where most of the population gains temporary immunity, leading to a periodic but predictable wave of infections.

Interpretation:

The low ξ value allows immunity to last longer, keeping the susceptible population low for extended periods. As a result, infection peaks are controlled, and the disease does not persist aggressively in the population. Vaccination strategies such as yearly flu shots align well with this dynamic.

2) Simulation 2: Pertussis Outbreak (Fast Immunity Loss)

With higher values of $\beta = 0.6\ and\ \xi = 0.2$, the system exhibited sharp and frequent oscillations in the infectious population. This reflects diseases like pertussis, where immunity wanes more quickly after recovery or vaccination. The increased susceptibility causes the infection to reignite periodically, resulting in cyclic and sustained outbreaks.

Interpretation:

The high ξ value means people re-enter the susceptible pool quickly, increasing the chance of repeated outbreaks.

Despite the same recovery rate ($\gamma = 0.1$), the epidemic cycles more aggressively due to the replenishment of susceptibles.

This demonstrates the importance of booster vaccination programs for pertussis.

A. Epidemiological Significance

These simulations demonstrate the SIRS model's utility in predicting outbreak patterns and guiding public health policies. For example:

Diseases with slow immunity loss (low ξ) can be effectively managed with single or periodic vaccinations.

In contrast, high ξ diseases demand more aggressive intervention strategies, including booster campaigns and close monitoring of outbreak cycles.

VII. CONCLUSION

In this work, we have applied the SIRS (Susceptible–Infectious–Recovered–Susceptible) mathematical model to investigate the spread and recurrence of infectious diseases such as Influenza and Pertussis. By simulating disease dynamics under different parameter settings, we demonstrated how key epidemiological factors—namely the transmission rate (β), recovery rate (γ), and immunity loss rate (ξ)—profoundly affect the outbreak pattern and long-term behavior of infectious diseases.

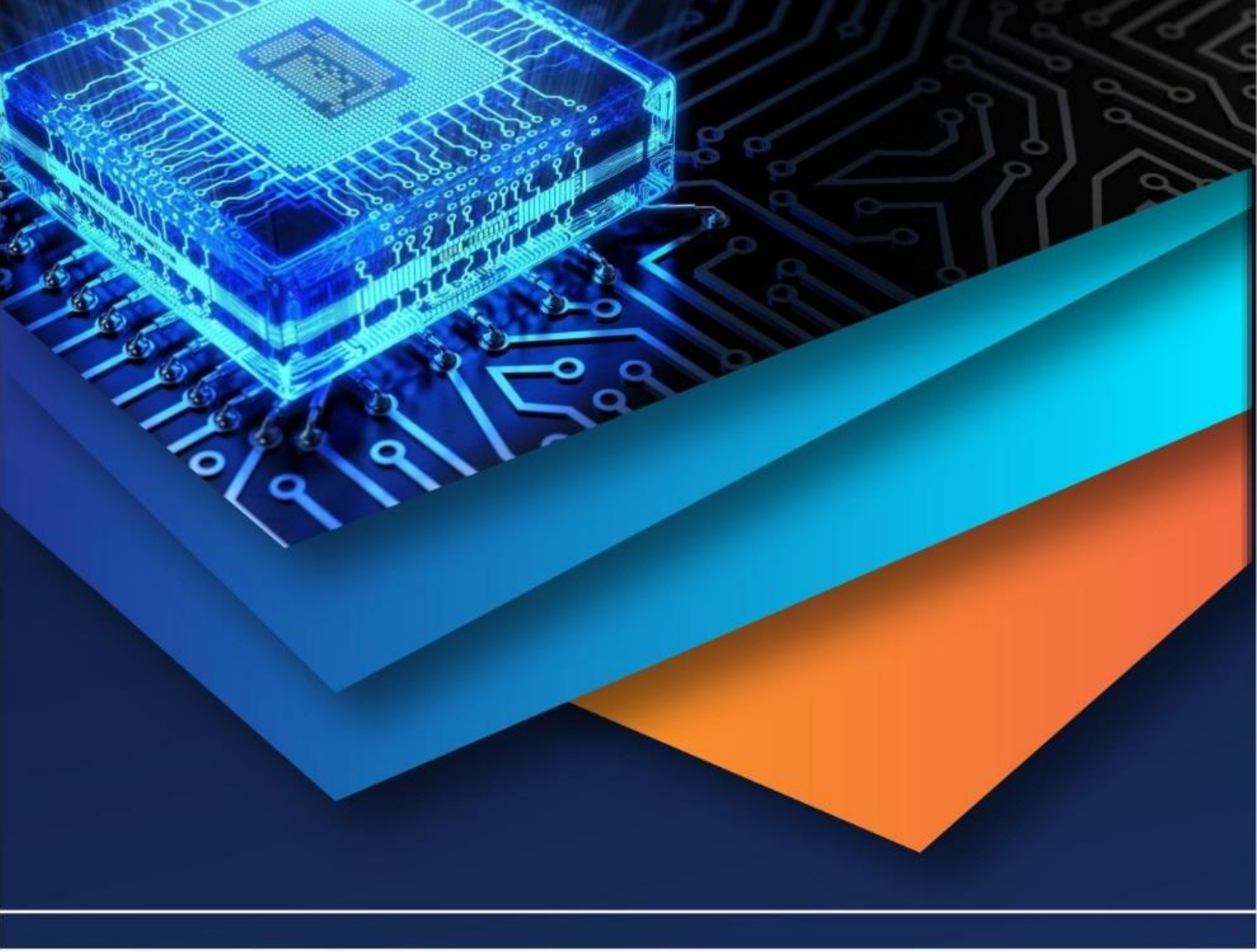
The simulations revealed that low immunity loss (Influenza) leads to controlled, periodic outbreaks with eventual stabilization, while high immunity loss (Pertussis) produces frequent and intense infection cycles due to the rapid return of individuals to the susceptible pool. These findings highlight the importance of tailoring public health strategies to disease-specific characteristics, particularly the duration of post-infection or post-vaccination immunity.

Overall, the SIRS model provides a valuable and interpretable framework for predicting disease outbreaks, designing vaccination policies, and understanding the conditions under which a disease may become endemic or epidemic. It forms a basis for more complex models that incorporate population heterogeneity, seasonality, spatial distribution, and control interventions.

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