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MATHEMATICAL MODELLING OF INFECTIOUS DISEASE SPREAD USING DIFFERENTIAL EQUATIONS: ANALYSIS AND APPLICATIONS OF THE SIR AND SEIR MODELS

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ABSTRACT:
Mathematical epidemiology provides a quantitative framework for studying how infectious diseases move through populations. Differential equations are particularly valuable because infection, recovery, loss of susceptibility, and intervention are processes that evolve continuously in time. This paper develops a focused study of two foundational compartmental models: the Susceptible–Infected–Recovered (SIR) model and the Susceptible–Exposed–Infected–Recovered (SEIR) model. The work explains how epidemiological assumptions are translated into ordinary differential equations, how initial information is incorporated, and how model properties can be interpreted through equilibrium analysis, reproduction numbers, stability, sensitivity analysis, and numerical solution.
The SIR model represents susceptible, infectious, and recovered groups, while the SEIR model adds an exposed compartment to represent a latent period between infection and infectiousness. The paper derives governing equations, conservation relations, threshold conditions, and intervention formulations. Numerical approaches including Euler and fourth-order Runge–Kutta methods are discussed, together with positivity, boundedness, and numerical stability. Three diagrammatic outputs and two analytical tables clarify the model structure, epidemic trajectories, and intervention scenarios.
The principal conclusion is that SIR and SEIR differential equations are powerful explanatory and decision-support tools, but their predictions depend strongly on assumptions, parameter estimation, data quality, population mixing, and the temporal stability of transmission conditions. Mathematical results should therefore be interpreted as scenario-dependent estimates rather than exact forecasts.

KEYWORDS: Differential equations; mathematical epidemiology; SIR model; SEIR model; reproduction number; stability; numerical simulation; vaccination; intervention; sensitivity analysis.

1. INTRODUCTION

Infectious disease outbreaks are dynamic systems. The number of people susceptible to infection changes as transmission occurs; the infectious population changes as new infections arise and infected individuals recover; and the recovered population grows as immunity accumulates. Because these quantities evolve over time, differential equations provide a natural mathematical language for epidemic analysis. The classical SIR framework introduced by Kermack and McKendrick established one of the most influential deterministic compartmental formulations in mathematical epidemiology [1].

The basic modelling idea is to divide a population into epidemiologically meaningful compartments and specify the rate at which individuals move between them. The SIR model contains susceptible, infected, and recovered groups. The SEIR model adds an exposed class to represent individuals who have acquired infection but are not yet infectious. Modern research has expanded these structures to include exact solutions, numerical schemes, stability, parameter estimation, intervention strategies, demographic effects, spatial heterogeneity, unreported infection, and fractional-order formulations [2]–[10].

This paper focuses on the SIR and SEIR models as applications of differential equations. Its objectives are to derive the governing systems; examine conservation, positivity, equilibria, and thresholds; explain numerical solution methods; demonstrate intervention modelling; and discuss limitations and extensions. The emphasis is on how mathematical assumptions become equations and how those equations support interpretation and decision-making.

2. differential-equation foundations

Let $S(t), I(t), E(t)$, and $R(t)$ denote epidemiological compartments. For a closed population, the conservation relation is

$N = S(t) + I(t) + R(t).$ (1)

A general compartmental model can be represented as

$dx/dt = f(x, t, \theta, u), \quad x(0) = x_0.$ (2)

where x is the state vector, θ contains parameters, and u represents an intervention or external input. A sound model should preserve non-negativity and, when appropriate, total population. Homogeneous mixing gives the standard incidence term SI/N. More elaborate models replace this with nonlinear, age-structured, network, or spatial transmission functions. The distinction between model structure and parameter values is important because structural error and parameter uncertainty have different consequences.

3. The classical SIR Model

The SIR model assumes that susceptible individuals become infectious through effective contacts and subsequently recover or are removed.

$dS/dt = -\beta SI/N.$ (3)

$dI/dt = \beta SI/N - \gamma I.$ (4)

$dR/dt = \gamma I.$ (5)

$d(S + I + R)/dt = 0.$ (6)

Here β is the effective transmission rate and γ is the recovery/removal rate. The average infectious period is approximately 1/γ. Dividing the first equation by the third gives

$dS/dR = -\beta S/(\gamma N).$ (7)

$ln(S/S_0) = -(\beta/\gamma N)(R - R_0).$ (8)

The basic reproduction number is

$R_0 = \beta/\gamma.$ (9)

If $R_0 > 1$, infection can initially grow in a fully susceptible population; if $R_0 < 1$, the infectious class tends to decline. The threshold interpretation is one of the central mathematical results of epidemic modelling. Figure 1 illustrates the compartmental structure of the SIR/SEIR model, representing the movement of individuals through different disease stages. It shows the transitions between Susceptible, Exposed, Infected, and Recovered populations, providing a framework for understanding epidemic transmission.

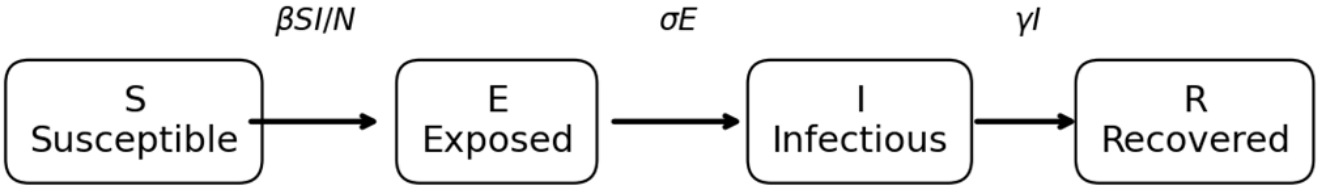

Fig. 1. Compartmental structure of the SIR/SEIR model family.

4. The SEIR Model

The SEIR model introduces an exposed compartment E. Exposed individuals have acquired infection but are not yet infectious under the model assumptions.

$dS/dt = -\beta SI/N,$ (10)

$dE/dt = \beta SI/N - \sigma E,$ (11)

$dI/dt = \sigma E - \gamma I,$ (12)

$dR/dt = \gamma I,$ (13)

$N = S + E + I + R.$ (14)

The parameter σ is the progression rate from exposure to infectiousness; its inverse approximates the latent period. The linearized infected subsystem near the disease-free state can be written

$d/dt\,[E, I]^{\mathrm{T}} = [[-\sigma, \beta], [\sigma, -\gamma]]\,[E, I]^{\mathrm{T}}$ (15)

The eigenvalues of this matrix determine local growth or decay. The SEIR model is useful when a latent period is epidemiologically meaningful, but the added parameter must be identifiable or supported by independent evidence. Global stability and exact-solution results have been established for important classes of SEIR systems [4], [17].

5. Equilibria, thresholds, and stability

For the basic closed SIR model, the disease-free condition has I*= 0 and infection eventually disappears because the susceptible pool is depleted. With demographic turnover, a persistent endemic state can arise. A simple demographic SIR system is

$dS/dt = \Lambda - \beta SI/N - \mu S,$ (16)

$dI/dt = \beta SI/N - (\gamma + \mu)I,$ (17)

$dR/dt = \gamma I - \mu R.$ (18)

$R_0 = \beta/(\gamma + \mu).$ (19)

The disease-free equilibrium is locally stable under standard assumptions when $R_0 < 1$. When $R_0 > 1$, an endemic equilibrium may exist. Stability analysis can be performed with Jacobian matrices, eigenvalues, Lyapunov functions, or global dynamical arguments. Recent studies combine these analyses with numerical simulation [11], [12], [14].

Seasonality can be introduced through a time-dependent transmission rate:

$$\beta(t) = \beta_0[1 + a\cos(2\pi t/T)]. \quad (20)$$

Periodic forcing can produce recurrent epidemics and, in nonlinear systems, complex dynamics [19].

6. Numerical solution of SIR and SEIR equations

Because nonlinear epidemic systems rarely have elementary closed-form solutions, numerical integration is essential. For $y' = f(t, y)$, forward Euler gives

$$y_{n+1} = y_n + h\,f(t_n, y_n). \quad (21)$$

The classical fourth-order Runge–Kutta method uses

$$k_1 = f(t_n, y_n), \quad (22)$$

$$k_2 = f(t_n + h/2, y_n + hk_1/2), \quad (23)$$

$$k_3 = f(t_n + h/2, y_n + hk_2/2), \quad (24)$$

$$k_4 = f(t_n + h, y_n + hk_3), \quad (25)$$

$$y_{n+1} = y_n + (h/6)(k_1 + 2k_2 + 2k_3 + k_4). \quad (26)$$

Numerical epidemic studies emphasize positivity, boundedness, stability, and conservation. A solver should be checked by reducing the step size and verifying that $S + E + I + R$ remains close to N [20]. Figure 2 illustrates the progression of an epidemic over time, showing how the number of cases changes across different stages. It highlights key patterns such as the rise, peak, and decline of infections, helping to understand epidemic dynamics.

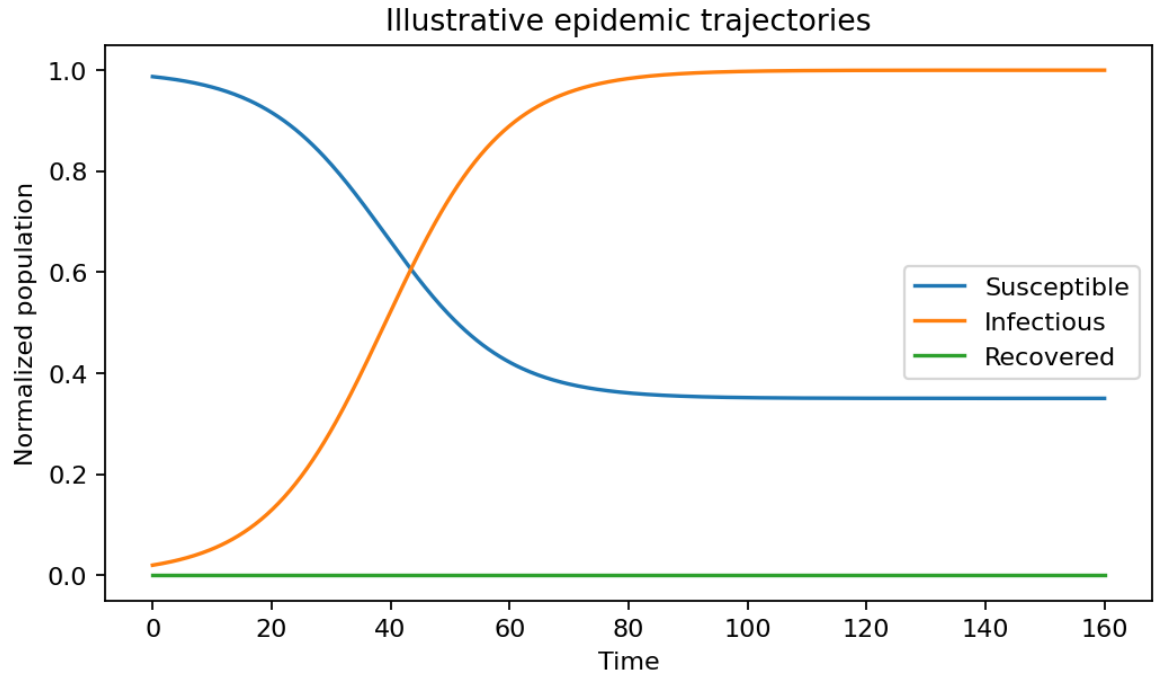

Fig. 2. Illustrative epidemic trajectories

7. Modeling intervention strategies

Vaccination can move susceptible individuals into an immune compartment:

$$dS/dt = -\beta SI/N - \nu S, \quad (27)$$

$$dI/dt = \beta SI/N - \gamma I, \quad (28)$$

$$dR/dt = \gamma I + \nu S. \quad (29)$$

$$R_e(t) = R_0\,S(t)/N. \quad (30)$$

Transmission reduction can be represented by a control $u(t)$:

$$\beta\_eff(t) = \beta[1 - u(t)], \quad 0 \le u(t) \le 1. \quad (31)$$

Thus, reducing β or increasing the effective removal rate can push R_e below one. Intervention models have been used to compare vaccination, social distancing, contact tracing, testing, and hospitalization strategies [7], [13].

Figure 3 illustrates the conceptual effect of reducing the disease transmission rate on the epidemic dynamics. A lower transmission rate results in a slower increase in the number of infected individuals and reduces the height of the epidemic peak. The curves shown are conceptual representations intended to demonstrate the qualitative behaviour of the model and are not based on empirical or real-world epidemiological data.

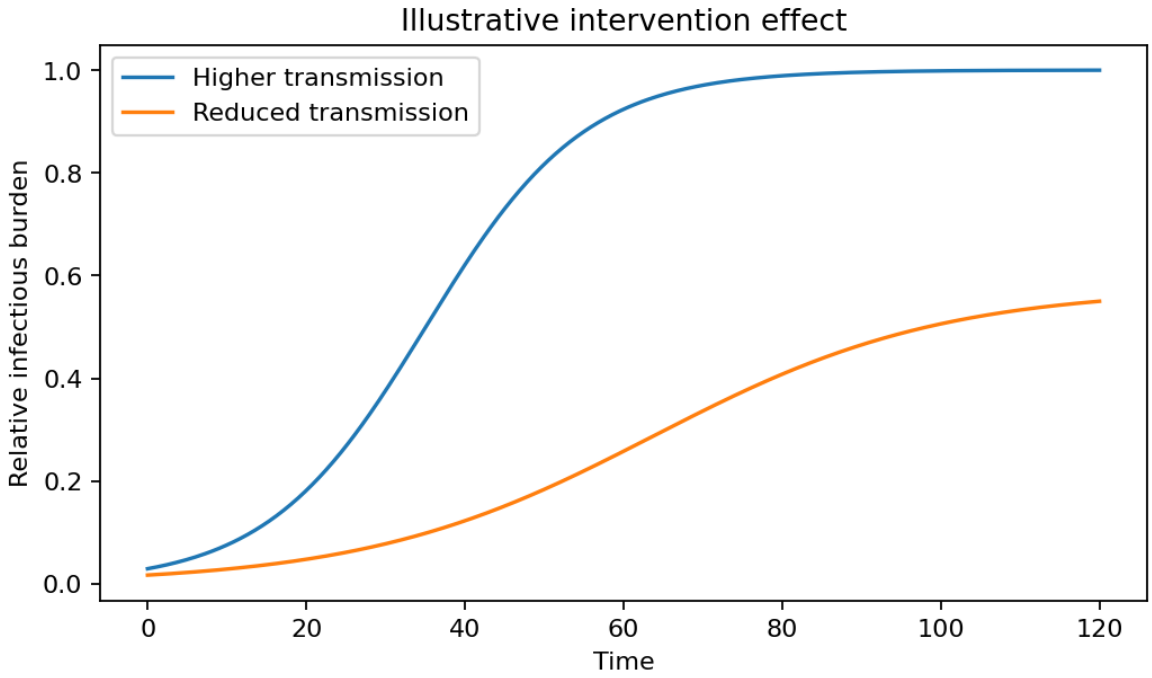

Fig. 3. Illustrative effect of reducing transmission; curves are conceptual rather than empirical.

8. Parameter estimation and sensitivity

Parameters must usually be estimated from observations or informed by prior evidence. A least-squares objective can be written

$$J(\theta) = \Sigma_i\,[y\_obs(t_i) - y\_model(t_i;\theta)]^2. \qquad (32)$$

Sensitivity can be expressed as

$$S_p^y = \left(\frac{p}{y}\right)\left(\frac{\partial y}{\partial p}\right). \qquad (33)$$

Large sensitivity indicates that uncertainty in a parameter can strongly affect model output. Transmission rate, infectious duration, reporting rate, and latent-period parameters may be particularly influential. A good fit does not imply unique parameter identification, especially when only reported cases are observed. Independent validation and multiple data streams can reduce this ambiguity.

9. Comparison of SIR and SEIR

Table 1 provides a comparative overview of the SIR and SEIR differential-equation models based on their compartmental structures and epidemiological assumptions. The SIR model classifies the population into susceptible, infected, and recovered groups, whereas the SEIR model introduces an additional exposed compartment to account for the incubation period. This distinction enables the SEIR model to represent disease transmission dynamics more realistically when individuals may be infected but not yet infectious.

Feature	**SIR**	**SEIR**
Compartments	S, I, R	S, E, I, R
Latent period	Not explicit	Explicit
Parameters	β, γ	β, σ, γ
Advantage	Simple and transparent	Represents delayed infectiousness
Limitation	May omit latency	More parameters
Typical use	Thresholds and scenarios	Latency-sensitive scenarios

Table 1. Comparison of the SIR and SEIR differential-equation models.

Model choice should follow the scientific question. A more complex model is not automatically better; it must provide identifiable and decision-relevant information.

10. MAIN RESULTS

The analysis yields four principal mathematical results. First, the basic systems preserve total population in a closed setting. Second, the reproduction number provides a threshold for initial invasion. Third, the SEIR model introduces a latent-period delay that changes timing even when long-run thresholds are similar. Fourth, intervention effectiveness is strongly linked to the reduction of effective transmission.

For the simplest model,

$$R_0 = \beta/\gamma. \qquad (34)$$

Therefore, proportional changes in β produce proportional changes in R_0. Increasing γ through faster recovery or effective removal also reduces R_0. These relationships provide a direct mathematical explanation for why

prevention and treatment can alter epidemic trajectories. Numerical analysis additionally shows that the timing and height of an epidemic peak are sensitive to parameter values and initial conditions. Consequently, uncertainty should be propagated through simulations rather than represented by one deterministic curve.

11. LIMITATIONS

The homogeneous-mixing assumption is a major limitation. Real populations contain age, household, workplace, geographic, and social-network structure. A single β averages across these patterns. Deterministic ODEs also ignore stochastic variation, which can be important during early outbreaks. Reported infections are imperfect measurements of actual infections because of under-reporting, asymptomatic cases, testing changes, and delays.

Constant parameters may also be unrealistic. Behavior, immunity, seasonality, policy, and pathogen characteristics can change transmission over time. Spatially heterogeneous models may be required where urban and rural transmission differ substantially [21]. Model structure itself is uncertain: SIR, SEIR, and extended compartmental models can all explain limited data. For this reason, mathematical predictions should be interpreted conditionally and validated against independent evidence.

12. Extensions and Future Directions

SIR and SEIR models can be extended with births and deaths, vaccination, waning immunity, asymptomatic infection, hospitalization, age structure, spatial movement, networks, and stochastic processes. A controlled system can be written

$$dx/dt = f(x,\theta) + G(x)u(t). \quad (35)$$

An intervention optimization problem can be represented by

$$min\_u\, J(u) = \int_0^T [A\, I(t) + B\, u(t)^2]dt. \quad (36)$$

Fractional-order epidemic models have also been developed to represent memory effects, while network and spatial models represent heterogeneity that compartmental averages cannot capture [18], [22]. Recent reviews indicate that model choice should depend on scale, available data, and the realism required [9].

13. DISCUSSION

The SIR and SEIR frameworks demonstrate the central role of differential equations in converting qualitative epidemiological mechanisms into quantitative predictions. Terms such as βSI/N, σE, and γI correspond directly to assumptions about transmission, progression, and recovery. This transparency makes compartmental models useful for teaching, hypothesis testing, scenario comparison, and policy analysis.

At the same time, mathematical transparency does not guarantee empirical accuracy. A correct solution of an incorrectly specified model remains a poor representation of reality. Parameter uncertainty, structural uncertainty, changing behavior, and imperfect observations must therefore be considered.

The most defensible use of these models is often comparative: evaluate plausible interventions, identify thresholds, estimate broad timing, and determine which parameters require better measurement. More detailed models should be introduced when additional data and a clear scientific need justify their complexity.

14. CONCLUSION

This paper examined infectious disease spread through the SIR and SEIR systems of differential equations. The models demonstrate how compartment definitions, conservation laws, transmission assumptions, and recovery processes become nonlinear ordinary differential equations. Analysis of reproduction numbers, equilibria, stability, numerical integration, and sensitivity provides a coherent mathematical framework for understanding outbreaks.

The principal conclusion is that SIR and SEIR models are powerful because they expose mechanisms and allow interventions to be represented quantitatively. Their predictions, however, are conditional on assumptions and parameter estimates. Numerical solutions should be checked for stability, positivity, conservation, and convergence, while fitted models should be validated using independent evidence.

Future work should combine mechanistic differential equations with stochastic methods, spatial and network structure, uncertainty quantification, data assimilation, and carefully constrained machine learning. Such integration can retain the interpretability of mathematical epidemiology while improving its ability to represent complex real-world outbreaks.

15. Parameter Summary

The principal parameters employed in the SIR/SEIR differential-equation models are presented in Table 2. These parameters represent key epidemiological factors that govern the transmission, progression, and recovery dynamics of the disease. The values assigned to these parameters play a crucial role in determining the accuracy and reliability of the model predictions.

Symbol	Meaning	Interpretation
B	Transmission rate	Controls infection generation
Γ	Recovery/removal rate	Controls infectious duration
Σ	Progression rate	Controls latent-period duration
N	Vaccination rate	Moves susceptible people toward immunity

R_0	Basic reproduction number	Initial invasion threshold
R_e	Effective reproduction number	Current transmission potential
N	Population size	Total modelled population

Table 2. Principal parameters used in SIR/SEIR differential-equation modelling.

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