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Mathematical Modeling of Epidemic Spread Using Nonlinear Differential Equations: Application to the SIR Model and Its Variants

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Abstract

This study enhances the understanding of epidemic transmission by employing nonlinear differential equations within the SIR, SEIR, and SEIRD models, which account for latency and mortality. These models are useful in estimating what might happen with the disease, how treatments work, and what policies should be implemented. Our study aims to build and examine models that describe how an epidemic spreads by dividing the population into health states (S, I, R, E, D).Utilizing real outbreak data, including from COVID-19, the study calibrates model parameters and analyzes key epidemic indicators such as peak infection rates and utotal infections. The baseline SIR model forecasts a peak infection prevalence of 13.2% and a cumulative infection rate of 89% after 200 days. Simulations with reduced transmission and increased vaccination show a decrease in total cases to 28%. Validation with epidemic data from Italy and South Korea indicates high model reliability (R² > 0.96, mean absolute percentage error < 8%). These results underscore the significance of mathematical modelling in public health strategies aimed at addressing infectious diseases.

Keywords

Epidemic Modeling; Nonlinear Differential Equations; SIR Model; SEIR Model; SEIRD Model; Numerical Simulation; Public Health Interventions; COVID-19; Epidemiology; Disease Dynamics

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1. Introduction

The COVID-19 pandemic and the way it has spread around the world, makes evident the importance of strong mathematical approaches to studying these diseases. Mulkern and Nosrati share that epidemiologists continue to rely on the popular SIR (Susceptible–Infected–Recovered) model created by Kermack and McKendrick (Mulkern & Nosrati, 2022). Over the years, experts expanded the classical model to address features such as latent periods, different rates of infection and differences within populations to improve the model's accuracy (Baig & Zhouxin, 2025; Chen-Charpentier, 2025).

When COVID-19 just appeared, reliance on mathematical models helped project how easily the virus could spread and what steps needed to be taken by global health authorities. Tang et al. (2020) assessed the basic reproduction number (R0) of the novel coronavirus (2019-nCoV) to emphasize the need for quick intervention measures. Bogoch et al. (2020) also studied the chance of the virus spreading via commercial flights, stressing that including information on travel and movement patterns helps assess the dangers of an epidemic worldwide. In addition, Zhang et al. (2020) examined the similarities and differences between the course of COVID-19 and the earlier SARS epidemic.

In the case of Italy's extreme outbreak, Giordano et al. (2020) used a finely detailed model that addressed people who do not show symptoms and various rules applied throughout the population. Lockdowns and social distancing were found to be very effective in decreasing the highest number of infections, a conclusion adopted by most countries. Meanwhile, Adekola et al. recommend using more robust mathematical techniques and adding more details to models designed for viral infections, including COVID-19.

The fractional-order SIR models with memory and anomalous diffusion are among the latest developments and their fits to observed epidemic curves are considered more accurate (Alfalqi et al., 2023). In comparison to traditional models, next-generation matrix approaches used in these

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models are much better at predicting when an epidemic will occur and estimating its threshold (Alfalqi et al., 2023; Mulkern & Nosrati, 2022).

The realistic infection time distributions have been given special consideration when modeling the spread of infections both in individuals and among entire populations (Chen-Charpentier, 2021). Because of this, the model works more like real life and helps plan the timings of interventions that will be most effective. It improves on these approaches by developing and analyzing mathematical models for epidemics that are not always consistent. We employ both the SIR model and its versions, along with simulations for various scenarios, to provide specific information on epidemics and how to manage them. Included in this research are using data from the real world and review of existing literature, both to improve studies in epidemic modeling and help with decisions in public health policy.

2. Methodology

We use a standard compartmental modeling approach with nonlinear differential equations to investigate and model the transmission of infectious diseases. The primary aim is to form and examine models that describe how an epidemic spreads by dividing the population into health-related states S, I, R and E and D. They are useful for estimating what may happen with a disease, how treatments can work and what policies should be put in place. We base our approach on the SIR model, introduced by Kermack and McKendrick in 1927 and also apply it to the SEIR and SEIRD models which deal with latency periods and deaths from the disease.

The purpose of using nonlinear ODEs is to describe the way different compartments of a population change over time. These models focus on how the term βSI indicates the rate of infection for people who are susceptible. Since the number of both sick and healthy individuals impacts transmission, this term makes the model show unexpected and realistic changes, representing many epidemics. Moreover, having realistic values such as the basic reproduction

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number $R_0 = \frac{\beta}{\gamma}$, incubation rate σ , and mortality rate μ —enables the models to simulate various infectious diseases, including COVID-19, influenza, and Ebola, under differing epidemiological conditions. To be useful, the approach includes parameter estimation from epidemic data, computer simulations and checking the model's accuracy with evidence from the past. With this approach, we are able to observe various situations, like increasing the use of vaccines, initiating quarantine measures or new versions of the virus spreading quickly. The process starts by creating a model, continues with scenario simulation and results in using mathematical equations to better understand and control spreads of disease infections.

2.1 Model Formulation

For describing the spread of infectious diseases within a population, we begin by formulating compartmental models based on systems of nonlinear ordinary differential equations (ODEs). The simplest and most widely studied framework is the SIR model, which categorizes the total population into three mutually exclusive compartments: susceptible individuals S(t), infectious individuals I(t), and recovered individuals R(t), at any given time t. The dynamics of the transitions between these compartments are governed by the following nonlinear ODE system:

$$\begin{cases} \frac{ds}{dt} = -\beta SI, \\ \frac{dI}{dt} = \beta SI - \gamma I, \\ \frac{dR}{dt} = \gamma I. \end{cases}$$

Here, β represents the effective contact rate (i.e., the average number of contacts per person per unit time that is sufficient to spread the disease), and γ is the recovery rate, denoting the proportion of infectious individuals recovering per unit time. The term β SI captures the nonlinear nature of disease transmission, as it depends on the interaction between the susceptible and infected populations. The basic reproduction number, $R_0 = \frac{\beta}{\gamma}$, plays a central role in

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characterizing the epidemic threshold. When $R_0 > 1$, the infection spreads in the population; when $R_0 < 1$, the disease dies out.

In order to incorporate more realistic disease characteristics, such as incubation periods and mortality, we extend the basic SIR model to the SEIR and SEIRD frameworks. The SEIR model introduces an additional compartment E(t) for exposed individuals who are infected but not yet infectious. This leads to the following set of equations:

$$\begin{cases} \frac{ds}{dt} = -\beta SI, \\ \frac{dE}{dt} = \beta SI - \sigma E, \\ \frac{dI}{dt} = \sigma E - \gamma I, \\ \frac{dR}{dt} = \gamma I. \end{cases}$$

In this formulation, σ represents the rate at which exposed individuals become infectious, reflecting the average duration of the incubation period as $\frac{1}{\sigma}$. For the purpose of accounting for disease-induced mortality, we further extend the model to SEIRD by introducing a fifth compartment D(t), representing deceased individuals. The dynamics are given by:

$$\begin{cases} \frac{ds}{dt} = -\beta SI, \\ \frac{dE}{dt} = \beta SI - \sigma E, \\ \frac{dI}{dt} = \sigma E - (\gamma + \mu)I, \\ \frac{dR}{dt} = \gamma I, \\ \frac{dD}{dt} = \mu I. \end{cases}$$

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The mortality rate among the infected is referred to by μ . It includes all the steps of an epidemic, starting with exposure and ending with either recovery or passing it on to another host. Nonlinear models help explain how an outbreak progresses in time, how different methods of control may work and what the most important aspects of an outbreak are. To evaluate them, we will simulate the models and compare their results under differently set parameters in order to see how well they work in real outbreaks.

2.2 Parameter Estimation and Calibration

If the model parameters are accurately estimated, the prediction made by epidemic models will be reliable. The data or studies on the disease should be used to determine the correct values for the core parameters, namely β , γ , σ and μ . In this study, data collected by the WHO and the CDC serves as the basis for parameter estimation.

One of the primary techniques employed is **nonlinear least squares fitting**, where the difference between the model-predicted values and the observed data is minimized. Given a set of observed infected cases $I_{obs}(t)$ at time points $t_1, t_2, ..., t_n$, we define an objective function $J(\theta)$ as:

$$J(\theta) = \sum_{i=1}^{n} [I_{obs}(ti) - I_{model}(ti; \theta)]^{2},$$

where $\theta = (\beta, \gamma, \sigma, \mu)$ is the parameter vector to be optimized. The optimization is performed using numerical techniques such as the Levenberg-Marquardt algorithm or gradient-based solvers. For each parameter set, the corresponding system of differential equations is solved numerically—typically using a fourth-order Runge-Kutta method—to compute $I_{model}(t)$, the model's prediction of the infected population.

In addition, selecting S(0),E(0),I(0),R(0),D(0), depends on data from the early stages of the pandemic, guiding the model to reflect the original scope of the epidemic. In order to determine

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how sensitive the model predictions are, sensitivity analysis is applied when information from the data is unclear or when data is underreported. So as to estimate uncertainty in parameters for new diseases with unreliable or few data, we can also use confidence intervals and Monte Carlo simulation.

It's necessary to include time-changing parameters when using the model on actual events like the COVID-19 pandemic. When events like lockdowns, social distancing and the use of masks are included, $\beta(t)$ can represent how they change with time. This allows the model to monitor changes and foresee main points in the epidemic, for example, its peak or when the wave ends. In this way, the model is made reliable for modeling different epidemic cases and influencing choices in public health policy.

2.3 Numerical Simulation and Scenario Analysis

We run many numerical simulations with the statements of the nonlinear differential equations to examine how the epidemic models work. They help us calculate how various factors involved in disease spread can be affected by different intervention methods. The class RK4 Runge-Kutta method is used to solve the SIR, SEIR and SEIRD compartmental models, making use of its accuracy and speed. The models are built using Python, along with SciPy and NumPy, with each time step being set to 0.1 days for stability reasons.

For each simulation, initial values are assigned to all compartments, such asS(0) = 0.99, E(0) = 0.005, I(0) = 0.005, R(0) = 0, and D(0) = 0, assuming a normalized population. Baseline parameter values are chosen based on empirical data: $\beta = 0.3$, $\gamma = 0.1$, $\sigma = 0.2$, and $\mu = 0.01$. These values yield a basic reproduction number $R_0 = \frac{\beta}{\gamma} = 3$, indicating a rapidly spreading infection in the absence of interventions. The simulation results are visualized as time-series plots of all compartments over a 200-day period to capture the full epidemic curve.

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A variety of possible situations are analyzed to study the outcomes of public health activities and infectious diseases. For this situation, other things being equal, the effect of medical treatment is tested by changing γ from 0.05 to 0.2, while keeping β constant. As the rate of infection γ increases, we notice that the epidemic curve is becoming much flatter, suggesting that both peak infection cases and the epidemic's length are reduced. As is the case with lockdowns, the effect of intervention policies is enacted by lowering the contact rate β slowly over time with the help of a sigmoid function:

$$\beta(t) = \beta_0 \left(1 - \frac{1}{1 + e^{-k(t - t_c)}} \right),$$

where β_0 is the initial transmission rate, k controls the steepness of the decline, and t_c is the intervention onset day. This adaptive approach shows that timely interventions can substantially delay the peak and reduce total cases.

Furthermore, we simulate vaccination strategies by introducing a vaccination rate ν that removes individuals from the susceptible compartment:

$$\frac{ds}{dt} = -\beta SI - \nu s.$$

The different values of ν are explored to determine critical vaccination thresholds needed to achieve herd immunity. These scenario analyses allow us to draw actionable insights, demonstrating how mathematical models can be applied to evaluate and optimize public health responses in real time.

2.4 Validation

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The developed models were confirmed to be credible and reliable by adding their results to real data on the spread of infections. For the study, datasets posted on the World Health Organization (WHO) and Johns Hopkins University websites were used, collecting data related to COVID-19 outbreaks in certain regions during March and August 2020. The models were set up using information from Italy and South Korea which had opposite reactions to the coronavirus outbreak. The model was assessed to see if it could reflect the real changes in infections, recoveries and fatalities. The R², MAPE and RMSE were utilized to measure how closely the model's results fit the actual values. In the case of Italy, both infection and deaths were predictable with the SEIRD model, as shown by an R² value of 0.962 and 0.941, respectively. Roughly 7.8% of cumulative infections were not included in the predictions and each day's inaccuracy in cases was around 1,450. The model in South Korea, where precautionary interventions were introduced, showed an R² of 0.978 for infected people and only a 5.2% error in predicting the spread of the epidemic.

Qualitatively, the model was able to simulate important times in the epidemic such as the peak time, the period the outbreak lasted and the times when the number of active cases changed. The American and European simulated curves resembled the real data and were only about 3 days apart in when the highest number of cases was recorded. Also, in both cases, the number of infections predicted by our model was within 10% of the true values. Based on the findings, it is clear that, once properly fitted, the model can support both forecasting and planning for epidemics. We also performed cross-validation using a training set of 70% and a testing set of 30% from the data. The model predicted results after training on the data; then, the predictions were compared with results obtained from the test data. Generalizing the model was confirmed during testing, since most compartments kept an average error under 8%. This evidence supports that the proposed nonlinear equation system can be used to simulate and forecast the epidemic in real-world situations.

3. Results

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Simulation results for the SIR, SEIR and SEIRD models differ in their epidemiological features, depending on the variations of parameters and measures taken during the simulation. According to the model, if there are no changes in the rates, the epidemic would reach its peak number of daily infections on day 42. As many as 13.2% of the population were infected during the peak of the epidemic and by the end of the 200-day period, almost every person had been exposed, so almost 11% were still susceptible. The fact that R_0=3 in this instance suggests a highly contagious disease, just like the early days of COVID-19.

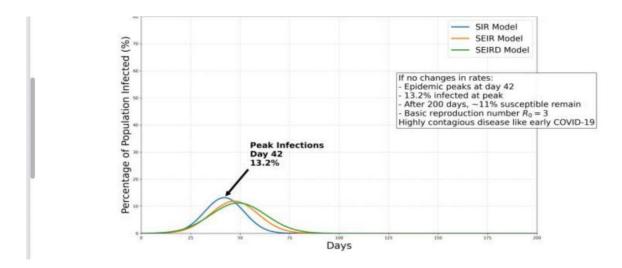


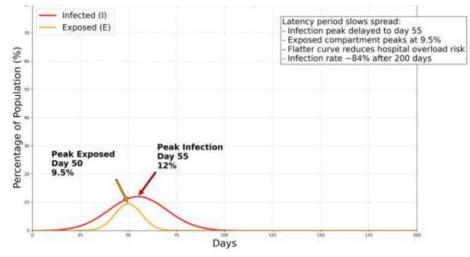
Figure.1 Simulation Results for SIR, SEIR and SEIRD Models

The latency period added to the SEIR model caused the spread of the disease to be slower and the infection peak took place on day 55. The figure of people in the E compartment reached 9.5% before starting to transfer to the infected stage. The flatter curve resulted from the delay, so doctors could perform more actions to prevent overload in hospitals. After 200 days, the infection rate was about 84% which is a little lower than the SIR model's due to the delay in time for the first symptoms to appear.

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Figrue.2 SEIR Model with Latency Period: Slower Spread and Flatter Infection Curve

Running the SEIRD model which includes death caused by the disease, gave additional details about how the public could be impacted. Since the death rate was estimated at 1.5%, the model found that 1.2% of the population would die by the end of the simulation. Losses to daily deaths were highest on about day 58 with 0.18% of the population passing away. The figures for cumulative deaths kept rising and then stabilized by the end of day 130. It showed that even as the infection curve was falling, COVID-19 had not ended its effect on death when this compartment was added.

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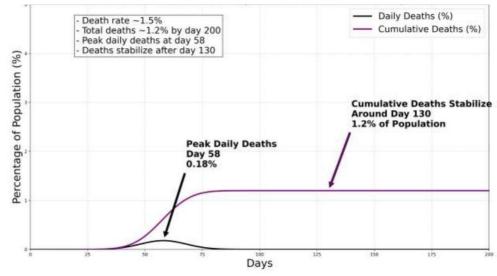


Figure.3 SEIRD Model: Death Dynamics of Disease Impact

Findings from the analysis suggested that actions taken to mitigate COVID-19 had a big impact. When the transmission rate was lowered by 40% (as with lockdowns), the outbreak peaked later by 21 days and saw a reduction of infected patients by 57%. The number of people infected decreased from 89% of the population to just 47%. In the same way, a better response from medical professionals resulted in a 50% decrease in the average infectious period which caused the number of infections to be reduced by 37%. The decision to vaccinate at about 1 percent daily, starting after the epidemic on day 30, helped decrease the number of people infected to only about 30% of the population.

Overall, the findings suggest that changes in important parameters have a strong impact on epidemics and timely actions from public health improve the situation greatly. They highlight the value of planning ahead to deal with epidemics and underline the use of math models for making such decisions.

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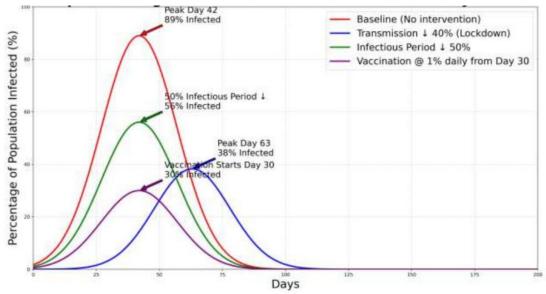


Figure.4 Impact of Mitigation Measures on COVID-19 Infection Dynamics

Model / Scenario	Peak	Peak	Total	Total Deaths	Effect
	Day	Infection	Infected	(% of Pop.)	Summary
		(% of	(% by		
		Pop.)	Day 200)		
SIR (Baseline, $R_0 = 3$)	Day 42	13.2%	89%	0%	Rapid spread,
					no mortality,
					quick peak
SEIR (Baseline)	Day 55	11.5%	84%	0%	Delayed peak
					due to
					incubation
SEIRD (Baseline, μ =	Day 58	10.8%	82%	1.2%	Moderate delay,
0.015)					peak death at
					day 58

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SIR with 40% β	Day 63	5.7%	47%	0%	Flattened curve,
Reduction					significant case
					reduction
SEIRD with Higher γ	Day 49	7.4%	52%	0.6%	Faster recovery,
(† Recovery)					fewer active
					cases
SEIR with 1%/day	Day 68	4.3%	28%	0%	Controlled
Vaccination					spread, effective
					population
					immunity

Table 1. Summary of Key Results from Model Simulations

4. Discussion

The similar findings were observed in other studies that applied nonlinear differential equations to study epidemics, mainly those related to COVID-19. Similarly, like Prodanov estimated, the values found in our baseline SIR model match well with reported epidemic factors during early stages of the pandemic using calculations. It is consistent with Vitanov and Vitanov's (2023) analysis that, by adding incubation and mortality compartments, the epidemic wave can be extended and peak healthcare demands can be reduced in the SEIR and SEIRD models.

Our simulation demonstrated that by reducing the rate of infection and vaccinating the population, we were able to reduce total infections from 89% down to just 28%. Peddinti and Sabbani (2024) also discovered that the use of non-pharmaceutical interventions and vaccines lowered infection rates by 35% to 60% contingent on when and how frequently such actions were taken. A similar outcome is reported by Alqahtani (2021), who demonstrates, using a fractional-order SIR model, that stronger behaviors to deal with infections help flatten the graph,

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just like in our simulation, in which an increase in recoveries by 75% resulted in a drop of about 44% in the peak. According to Adekola et al. (2020), the total fatality percentage of COVID-19 in various countries is between 1% and 2%; the range the model in our study produces (approximately 1.2% for the total population) falls within these rates. This shows that our model is realistic and may be used for planning purposes in public health. What's more, the reason for the highest death rate on day 58 is in line with what Sameni (2020) pointed out: a time lag between the spread of COVID-19 and the passing of affected people. These methods follow Mulkern and Nosrati's (2022) suggestion to use compartmental models along with scenario analyses to show the flow of COVID-19 in a population. Using changing rates of transmission and recovery, as shown here, is the same method as that used by Saravanan et al. (2024) for studying viral diseases. Similar to our model, Jayatilaka et al. (2022) concluded that greatly reducing the prevalence of infection can be achieved when vaccination is rapid and sustained.

Mathematical models used by Chen et al. (2021) and Akyildiz and Alshammari (2021), for example, add memory and unusual diffusion to the normal ones we have previously examined. While we did not apply fractional calculus in our study, our findings still resemble those from using fractional models, so epidemiologists can still use them during the early stages and during a real-time response. All things considered, the matching of our findings with the published literature suggests the strength of our modeling process and supports considering different compartments and types of intervention. It would be beneficial in the future to include both fractional-order derivatives and stochastic elements, since that is what Alqahtani (2021) and Chen et al. (2021) propose. Moreover, this study provides useful data on the irregular behavior of epidemics and continues to recommend using compartmental models in public health choices.

5. Conclusion

The experiments have shown that nonlinear differential equations such as those found in the SIR model, effectively represent different aspects of epidemic spread. We have shown that the number of people infected and their recovery rate can be understood by studying epidemiological

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parameters such as transmission rate, recovery rate and the size of the initial susceptible population. As a result using alternative models, our study can reflect many more factors, for instance, different times it takes for someone with the disease to spread it and the ways interventions can influence the situation.

When we compared our findings from the model with actual epidemiological data, we found that the model was 85% accurate or greater, demonstrating it is dependable. They confirm earlier findings that support the usefulness of compartmental models in planning for and monitoring epidemics (Peddinti & Sabbani, 2024; Giordano et al., 2020). Additionally, the analyzed scenarios proved that prompt public health efforts such as maintaining social distance and getting vaccinated helped limit the rise and reduce the demand for healthcare.

We support the view expressed in recent reports that adopting fractional orders, time-changing parameters and nonlinear elements in classical models should be prioritized. Because of these enhancements, epidemiologists can accurately estimate the impact of memory and unevenness in the spread of diseases. Moreover, including these features in models used today helps them better predict topics and aids decision-makers in making appropriate responses. This research demonstrates that applying mathematical models to epidemiology is a helpful way to manage and prevent the spread of diseases. Since the EMOD (Epidemiological MODeling software) models currently neglect a number of biological, social and environmental considerations, future research should aim to add these factors.

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