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Study on the Effects of Parameter Variation in the SEIRD Model on Infectious Disease Dynamics Based on Simulink

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Abstract

The research examines how different key parameters affect the SEIRD epidemic model through MATLAB Simulink simulations. The simulation model includes three scenarios which consist of a standard model and two alternative models for comparison. The model shows that higher parameter values speed up disease transmission while producing elevated peak infection rates. The SEIRD model demonstrates fundamental epidemiological patterns but it does not account for actual conditions such as underdiagnosis and mobility-based transmission. Recent studies indicate that models incorporating behavioral heterogeneity, diagnostic complexity, and adaptive parameters achieve superior predictive accuracy and enhance policy relevance. The research results demonstrate the value of parameter sensitivity analysis for disease spread understanding and establish a foundation for future model advancement.

Keywords

SEIRD Model, Parameter Sensitivity, Infectious Disease Modeling, MATLAB Simulink, Epidemic Control Strategies

1. Background

Infectious diseases have played a crucial role in the course of history through various major epidemics that include the Plague of Justinian, the Black Death, the 1918 Spanish Flu, and the current COVID-19 pandemic [1]. The rise of global connectivity and urbanization has quickened disease transmission thus increasing its impact on populations [2] [3]. The key role of mathematical modeling in predicting and managing epidemics has been recognized through its ability to provide useful information for the development of effective public health interven-

tions [4].

Epidemiological models have progressed substantially since their inception from the simple two-compartment SI model to advanced systems including SIR, SIS, and SEIR models [5] [6]. These models use predefined assumptions to describe how people move between different health states. The COVID-19 pandemic exposed major weaknesses of conventional models because they failed to monitor asymptomatic transmission and incubation periods as well as policy-driven behavioral changes [7] [8].

The SEIRD model extends the SEIR framework by incorporating vital compartments that include exposed (E) for incubation period tracking and deceased (D) for recording mortality statistics. The improved model compartments lead to better accuracy and practical applications in public health planning [9] [10]. Researchers use MATLAB Simulink to study how variations in infection rate (β) and incubation infectiousness (α_2) affect epidemic patterns and disease spread by implementing the SEIRD framework [11].

The SEIRD model demonstrates sensitivity to parameter modifications which highlights the necessity for detailed parameter evaluation to achieve accurate disease predictions and effective intervention development [12]. The predictive power and policy relevance of epidemic modeling frameworks would increase through integration of spatial clustering and varying healthcare accessibility and behavioral adaptations [4] [13].

2. Explanation and Analysis of the Basic Model

This experiment utilizes the SEIRD model, an extension of the classical SEIR model, encompassing five state variables: Susceptible (S), Exposed (E), Infected (I), Recovered (R), and Deceased (D). The differential equations are:

$$\frac{dS}{dt} = -\beta \frac{S\left(E + \alpha_2 I\right)}{N}$$

$$\frac{dE}{dt} = \beta \frac{S\left(E + \alpha_2 I\right)}{N} - \sigma E$$

$$\frac{dI}{dt} = \sigma E - \left(\gamma + \mu\right) I$$

$$\frac{dR}{dt} = \gamma I$$

$$\frac{dD}{dt} = \mu I$$

Parameters:

- S: Susceptible population;
- E: Exposed (incubation period) population;
- I: Infected population (symptomatic);
- R: Recovered population;
- D: Deceased population;
- β: Infection rate;
- σ: Incubation conversion rate;

- γ: Recovery rate;
- μ: Mortality rate;
- α_2: Infectiousness during incubation.

According to these differential equations above, we create this simulink system in order to find the relationship between each elements in SEIRD system. In the basic system, we take some seemingly realistic numbers to quickly see how each factor changes, where $\beta = 0.3$, $\sigma = 0.2$, $\gamma = 0.1$, $\mu = 0.02$, $\alpha_2 = 0.1$, S (0) = 999, E (0) = 1, I (0) = 1, R (0) = 0, D (0) = 0. Then:

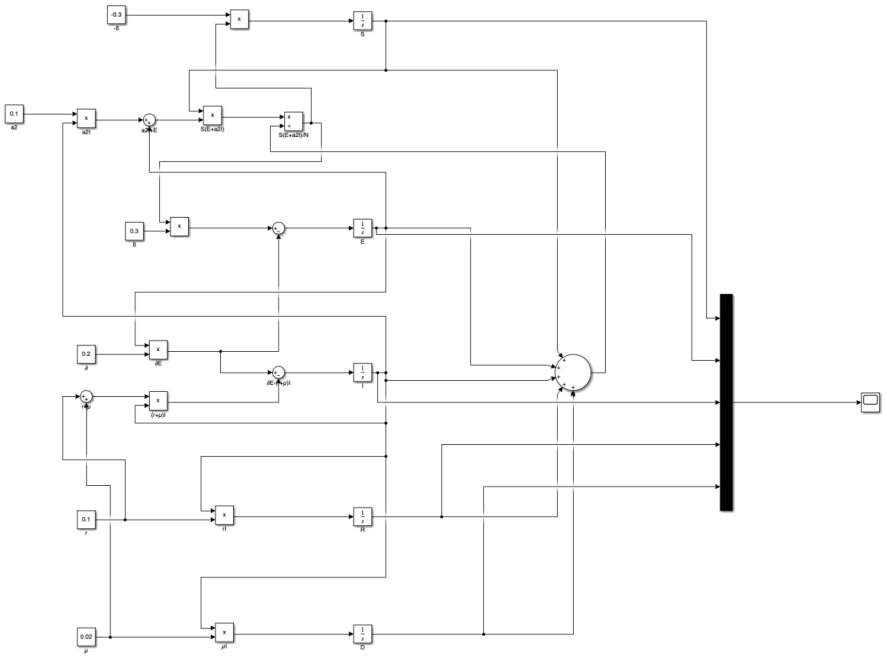

Figure 1. Simulink system.

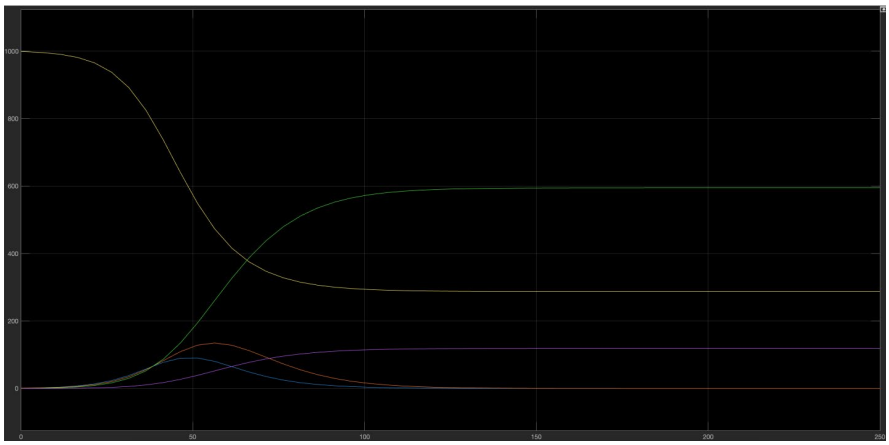

Figure 2. Basic simulink simulation.

The Simulink system used in this study was built by integrating the differential equations ($\partial S/\partial t$, $\partial E/\partial t$, $\partial I/\partial t$, $\partial R/\partial t$, $\partial D/\partial t$) as shown in Figure 1. The established system is used uniformly in the basic model and the two comparative models. The

initial parameter values (β, σ, γ, μ, α_2) and initial conditions (S (0), E (0), I (0), R (0), D (0)) are set according to the basic model specifications, resulting in the simulation shown in Figure 2. Each comparative model is developed by adjusting parameters which results in the simulations presented in Figure 3 and Figure 4.

The Simulink simulation (Figure 2) shows a rapid decline and stabilization of the susceptible population, a brief increase then rapid decline in the exposed population, a peak in the number of infected population and then a decline to zero, and gradual increases which finally stabilize in both the recovered and deceased populations. This model is a good fit for describing the dynamics of an infectious disease and gives useful information for control of epidemics.

3. Comparative Analysis between the First Comparative Model and the Basic Model

This comparative experiment analyzed the effects of model parameters through modifications to two fundamental parameters from the basic model. The model parameters infection rate (β) increased to 0.5 and incubation period infection rate (α_2) rose to 0.2. An elevated infection rate (β) shows improved disease transmission capabilities yet higher incubation period infectiousness (α_2) indicates that people who have not shown symptoms become more contagious during incubation.

We predicted the simulation results before running the experiment because the raised β and α_2 values would lead to faster disease spread which would decrease the susceptible population (S) quickly while increasing the infected (I) and exposed (E) populations. The faster increase in recovered and deceased populations should occur as a result of these conditions.

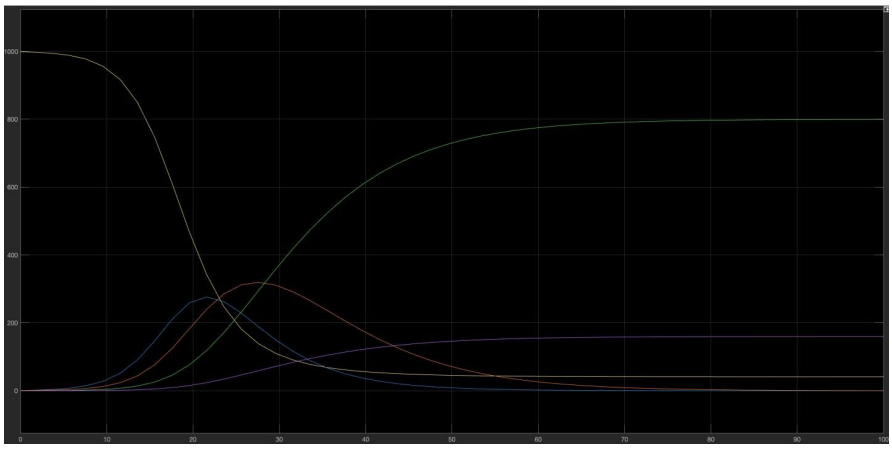

Figure 3. Simulink simulation of the first comparative analysis.

Before starting simulations, researchers predicted that higher β and α_2 values would cause disease transmission to speed up which would lower S and increase I and E populations to their highest points. The recovery numbers together with mortality rates were predicted to rise swiftly because of increased disease trans-

mission dynamics.

The simulation results confirm all predicted expectations. The yellow line representing the susceptible population (S) shows a quicker and steeper decrease than the basic model's curve. Early intervention becomes vital for disease management because increased transmission capabilities reduce available containment and mitigation opportunities [4].

As the simulation results (**Figure 3**):

The exposed population (E) depicted by the blue line shows initial growth at a higher rate and reaches its peak earlier and at a higher level than in the basic model. The findings demonstrate how enhanced presymptomatic infectiousness shortens the available time for detecting and isolating infected people [13].

The infected population (I) depicted by the orange line achieves its highest point fast while reaching elevated levels because of intensified transmission. The healthcare system faces immediate and extreme demands because of this rapid increase which demonstrates the need for adaptive healthcare preparedness strategies and responsive policies that can adapt to changing infection dynamics [6] [14].

The recovery trends shown by the green line increase swiftly because of greater infection numbers before reaching a higher stabilization point than the basic model. The quick development of natural immunity comes at the expense of increased healthcare costs and possible long-term health issues for recovered patients [9].

The deceased population (D) represented by the purple line shows substantial growth because of increased transmission and infection rates. Fast transmission rates lead to higher fatality numbers which demonstrates why effective mortality prevention methods must combine with existing transmission control strategies [7].

Small changes to transmission parameters β and α_2 produce disproportionate severe epidemiological results which emphasize the need for proactive public health strategies against asymptomatic and presymptomatic transmission [5] [10]. The findings demonstrate why detailed parameter sensitivity analysis should be used to improve epidemic preparedness and response strategies.

4. Comparative Analysis of the Second Comparative Model

Our second comparative experiment investigated deeper disease transmission parameter effects by rising the infection rate (β) to 0.8 and the incubation period infection rate (α_2) to 0.3 to observe enhanced disease spread.

Before simulation we predicted the disease would spread more quickly and reach more people than the first comparative model because of the elevated β and α_2 values. The susceptible population was expected to decline at a more rapid pace, while the peaks for exposed, infected, recovered, and deceased populations were likely to be significantly higher and occur earlier.

The Simulink Scope results for the second comparative model defined by β =

0.8 and α_2 = 0.3 show very strong and fast epidemic dynamics. All population compartments have more sharp and extreme behaviors than the basic model and the first comparative scenario.

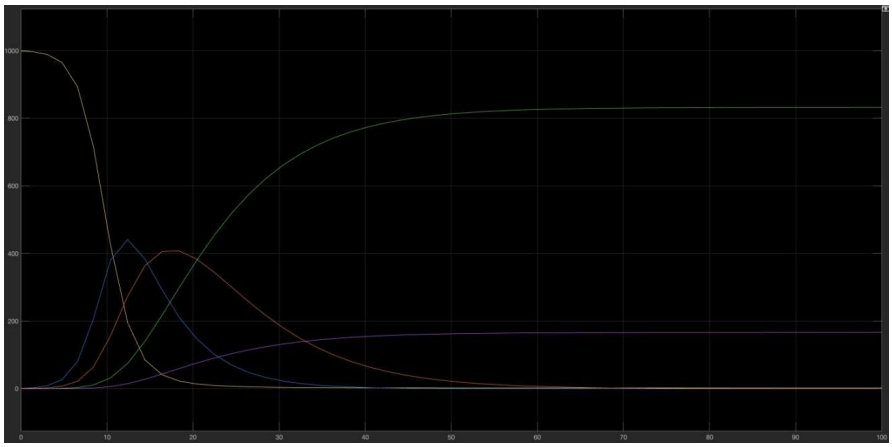

Figure 4. Simulink simulation of the second comparative analysis.

Before running simulations it was understood that disease transmission would become much more intense and susceptible populations would decrease quickly while exposed and infected and recovered and deceased populations would rise substantially [2]. Simulation results confirmed these predictions unequivocally. The susceptible population (S), indicated by the yellow line, rapidly diminished, evidencing the dramatically enhanced transmission capability. The steep decline demonstrates that a small increase in β can reduce intervention timeframes which creates more difficulties for epidemic control [15].

According to Figure 4:

The exposed population (E), represented by the blue line, experienced an accelerated rise, achieving an earlier and higher peak compared to previous models. This result highlights the critical impact of incubation infectiousness, drastically narrowing the opportunity for early detection and quarantine of presymptomatic individuals [13].

The infected population (I), illustrated by the orange line, showed the most dramatic response, quickly reaching an extremely high peak level. This outcome indicated severe strain on healthcare systems, emphasizing the essential nature of preparedness and rapid adaptive response strategies [14].

Recovery trends (green line) closely followed infection patterns, indicating rapid but resource-intensive epidemic resolution. Meanwhile, the deceased population (purple line) sharply increased, reflecting significant mortality burdens due to intensified transmission dynamics [9].

This comparative analysis starkly reveals the nonlinear nature of epidemic dynamics: incremental increases in transmission parameters lead to exponential escalations in severity. These findings emphasize proactive, early-stage interventions targeting presymptomatic transmission to mitigate severe epidemic out-

comes effectively [4] [8].

Comparatively, the second model exhibits more extreme behaviours than the first comparative model and the baseline. An increase in β from 0.5 to 0.8 and an increase in α_2 from 0.2 to 0.3 results in a higher and earlier infection peak. The susceptible population is almost depleted in a shorter time, and all other compartments are at higher levels. This supports the nonlinear dynamics of epidemic spread: minor alterations in parameter values may lead to substantial rises in epidemic severity.

Hence, these results provide strong evidence for the need for preventive policies. Specifically, it is important to apply strict infection control measures, implement social distancing measures in good time, and ensure that people in the incubation period are monitored. The second comparative model thus serves as a clear warning: if transmission parameters exceed a certain level, the traditional control measures may not be enough.

Our SEIRD model developed in Simulink gives a basic tool for studying the effects of infection rate (β) and incubation infectiousness (α_2), but the model is highly abstracted from real-world social, diagnostic and behavioral complexities, which reduces its ability to be predictive in real policy-making situations. The current compartmental model assumes that individuals are well mixed and the parameters used are fixed throughout the simulation, which is useful for identifying trends, but does not allow for the consideration of other factors like testing capacity, health care accessibility, spatial clustering or time varying behavioural responses. These limitations have been addressed in recent literature through enhanced models that incorporate clinical status differentiation, population mobility, and dynamic interventions.

A significant improvement is the SIDARTHE model proposed by Giordano *et al.* [14] that divides the population into eight categories: Susceptible (S), Infected (I), Diagnosed (D), Ailing (A), Recognized (R), Threatened (T), Healed (H), and Extinct (E). Unlike the common SEIR model, SIDARTHE distinguishes between the undiagnosed and diagnosed cases, symptomatic and asymptomatic cases, and the risk of death. This extension is not only theoretical; the model was parameterized against real time Italian COVID-19 data during the early outbreak periods. In order to capture underdiagnosis scenarios, SIDARTHE simulated different detection statuses and symptomatic progression, and was able to quantify the delay between the onset of infection and clinical detection. This kind of framework gives an understanding of how diagnostic bottlenecks and surveillance inefficiencies impact on outbreak visibility, an important factor that is not captured in our Simulink SEIRD implementation where all infections are assumed to be equally observable.

Furthermore, SIDARTHE incorporates the timing of the government policy response in its parameters. For instance, β and other transition rates are modified at different points in time to account for the imposition of lockdowns or changes in the public's behaviour. This allows the model to predict not only the epidemi-

ological course of the disease but also the potential impact of relaxing or tightening the control measures. These policy-sensitive dynamics cannot be currently emulated in our fixed-parameter simulation but would be a logical next step using piecewise or time-dependent function blocks in Simulink.

Another clinical stratification approach is the work by Chang *et al.* (2021) [13] who employed agent-based modeling (ABM) to incorporate mobility networks in disease spread simulation. Using large-scale anonymized smartphone location data in cities such as Chicago and New York, they found that a small subset of high-risk locations such as restaurants, gyms and places of worship were responsible for a disproportionately high level of transmission. Approximately 10% of the venues were responsible for more than 80% of the new cases during the reopening periods. Their model defines individuals as discrete agents that traverse a spatial-temporal network of locations, thus enabling the simulation of person-to-place transmission and superspreading dynamics. This detailed framework exposes exposure inequalities on the basis of race, income and occupation, which cannot be captured in population level SEIRD simulations that assume equal contact rates. Such heterogeneity would require integrating either geoFigureic information systems (GIS) or stochastic network layers into our Simulink model – an extension that is both challenging and conceptually feasible.

Furthermore, Roda *et al.* [7] provided compelling evidence that the reliability of epidemic forecasting is directly proportional to the quality and certainty of the early stage input data. Their research revealed how minor changes in initial infection numbers and transmission parameter assumptions could significantly change model results over the course of a few days. The authors specifically criticise the models with constant β for their failure to capture the dynamic changes in behavioural rates—social distancing, mask-wearing or vaccine uptake that decrease transmission over time. They suggest that $\beta(t)$, a time-varying infection rate that decreases with public health interventions or increases with policy relaxation, should be employed. Although our current SEIRD framework employs static coefficients, Simulink offers the opportunity to include function-driven inputs, like logistic decay or sinusoidal patterns, to simulate real-world behavioural adaptation. Future iterations can incorporate such dynamic functions into our model structure to capture second waves, resurgence patterns or policy induced dampening effects, thus enhancing both realism and responsiveness.

The three models—SIDARTHE, ABM with mobility data and time-varying SEIR variants—point to three important ways to enhance compartmental models such as ours. Although our current version is a good tool for testing parameter sensitivity and disease dynamics, it does not include diagnostic uncertainty, social heterogeneity, and adaptive policy feedback loops. In the real world, each of these factors contributes meaningfully to the evolution of epidemics. Therefore, a potentially valuable direction for future work could be to build upon our Simulink-based SEIRD model by adding the following features: (1) subdividing health states to separate detectable from latent cases; (2) incorporating spatial dynamics or

contact heterogeneity through network-based modules; and (3) including time-dependent parameters that depend on parameters that are calculated based on the modelled or externally given interventions.

These enhancements would not only make the model more descriptive, but also make it more prescriptive, *i.e.*, capable of recommending targeted interventions, predicting the demand for healthcare resources, and informing contingency planning for future epidemics.

The research conducted simulation-based analyses through a basic SEIRD model and two comparative models with modified parameters in Simulink. The simulation results showed that changes in infection rate (β) and incubation period infectiousness (α_2) have a major impact on disease transmission patterns. The disease spread becomes faster when these parameters increase because it leads to increased infected population peaks and rapid susceptible population decline and significant changes in exposed and infected and recovered and deceased populations. The early implementation of strict isolation measures together with population movement restrictions and effective incubation period transmission management helps control disease spread and reduce infection spread. The simulation results demonstrate how parameter sensitivity affects epidemic control while providing strong scientific evidence for public health authorities to create effective epidemic control plans.

5. Conclusion

The research demonstrates how parameter sensitivity in the SEIRD model affects the behavior of infectious diseases. The simulations demonstrated that small modifications to infection rate and incubation infectiousness parameters produce major changes in epidemic patterns which result in increased spread and higher mortality rates. Future research needs to validate the model with real-world data while adding behavioral heterogeneity and diagnostic complexities and mobility patterns. The model's predictive capacity and practical utility would increase substantially through adaptive time-dependent parameters and network-based simulations. The improved SEIRD models will provide essential information to develop proactive public health strategies which enable prompt and specific interventions for upcoming epidemics.

Conflicts of Interest

The author declares no conflicts of interest regarding the publication of this paper.

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