T.C. PÎRÎ REİS UNIVERSITY
INSTITUE FOR GRADUATE STUDIES

CHAOTIC SYSTEM ANALYSIS WITH GENETIC ALGORITHM APPROACH

MSc. THESIS

MUSTAFA ŞENTÜRK

MUSTAFA, ŞENTÜRK
MSc. Thesis
PRU 2023

MAY 2023

T.C. PÎRÎ REİS UNIVERSITY
INSTITUTE FOR GRADUATE STUDIES

CHAOTIC SYSTEM ANALYSIS WITH GENETIC ALGORITHM APPROACH

MSc. THESIS

by
MUSTAFA ŞENTÜRK
B. S., Naval Architecture and Marine Engineering, İstanbul Technical University, 2014

Compuational Science and Engineering Program

Thesis Advisor: Dr. Orhan Özgür AYBAR

MAY 2023

APPROVAL of the INSTITUTE for GRADUATE STUDIES

PRU, Graduate Studies Institute's Master student "20310201001" and "Mustafa Şentürk"
prepared his thesis titled
"CHAOTIC SYSTEM ANALYSIS WITH GENETIC ALGORITHM APPROACH"
by fulfilling all the necessary conditions determined by the relevant regulations and
successfully presented it to the jury whose signatures are below.

Approval of the Institute for Graduate Studies

Prof. Dr. M. Ziya Söğüt
Director

Dr. Orhan Özgür AYBAR
Head of Department

Dr. Orhan Özgür AYBAR
Advisor

Examining Committee Members

Dr. Öğr. Üyesi Orhan Özgür AYBAR ____________________

Dr. Öğr. Üyesi Duygu NİZAMOĞULLARI ____________________

Dr. Öğr. Üyesi Emre Çakmak ____________________

Date of Approval: 14.06.2023

ABSTRACT

CHAOTIC SYSTEM ANALYSIS WITH GENETIC ALGORITHM APPROACH

Dynamical analysis of epidemiological models gained significant attention from applied mathematicians after the Covid-19 pandemic caused a global catastrophe. Although studies involving the decision of the suitable model to describe the epidemiologic data and the predictive analysis of the disease spread have been carried out in-depth during this phase, studies that will expose the unrecognized dynamics through predictive analysis by investigating the algebraic properties of the models such as the invariant spaces, the center manifolds, and the Lyapunov functions in the general parameter space, still have the potential to contribute vastly to comprehending the spread dynamics. This work implements the triangular probabilistic distribution to an epidemic delay differential equation as a distributed continuous delay to determine the recovery rate based on characteristic model parameters. In this way, we propose a way to reduce the model parameters that need to be obtained during model fitting by defining generic coefficients derivable from field research and population characteristics.

Mustafa Şentürk

COMPUTATIONAL SCIENCE AND ENGINEERING MASTER'S PROGRAM

Thesis Supervisor: Dr. Orhan Özgür Aybar

May, 2023, 40 Pages

Keywords: Chaos Theory, Dynamical Analysis, Genetic Algorithm, Epidemic Model, Probability Distribution

ÖZET

GENETİK ALGORİTMA YAKLAŞIMIYLA KAOTİK SİSTEMLERİN ANALİZLERİ

Covid-19 pandemisinin küresel bir felakete yol açması, epidemiyolojik modellerin dinamik analizi konusunda uygulamalı matematikçiler arasında büyük bir ilgi uyandırmıştır. Hastalığın yayılma sürecini anlamak için epidemiyolojik verileri tanımlayacak uygun bir modelin belirlenmesi ve öngörüsel analizleri yapılmasının yanı sıra, modellerin invaryant uzayları, merkez manifoltları ve genel parametre uzayında Lyapunov fonksiyonları gibi cebirsel özelliklerini araştırarak, bilinmeyen dinamikleri ortaya çıkaracak çalışmalar da büyük önem taşımaktadır. Bu çalışma, karakteristik model parametrelerine dayalı iyileşme oranını belirlemek için üçgen olasılık dağılımını, bir gecikmeli diferansiyel denklem sistemine, dağıtılmış bir sürekli gecikme olarak uygulamaktadır. Bu şekilde, saha araştırması ve nüfus özelliklerinden türetilebilen genel katsayıları tanımlayarak model uydurma sırasında elde edilmesi gereken model parametrelerini azaltmanın bir yolunu öneriyoruz.

Mustafa Şentürk

HESAPLAMALI BİLİM VE MÜHENDİSLİK MASTER PROGRAMI

Tez Danışmanı: Dr. Orhan Özgür Aybar

Mayıs, 2023, 40 Sayfa

Anahtar Kelimeler: Kaos Teorisi, Dinamik Analiz, Genetik Algoritma, Epidemik model, Olasılık Dağılımı

DEDICATION

To My Family, for their patience and great support.

ACKNOWLEDGMENTS

SEIR models are preferred as the case of the study, because the heuristic methods are only applicable for pattern recognition in a system or optimizations. As the results of the researches indicates the machine learning based models are ideal for past data and works identically as curve fitting to an existing data, however they are not so useful in forecasting. These methods does not offer causal relations between actors of the system. The SEIR models are ideal for simulating the actors and causes of the epidemic growth.

The nature of epidemic growth is deeply based on random interactions in a populace. As we know that randomness has a pattern when applied to a large amount of events, such as Gaussian distribution. And the transitions between compartments are, in fact, stochastic processes. So we studied on a novel approach for simulating recovery transitions, as a probabilistic distribution implemented as multi delays.

In the development of this novel approached, he believed in my scientific intinctions and encouraged me to follow my enthusiasm under any circumstances in studying an implementation of a novel approach by inspiring and shared his valuable experience and knowledge in the field with great patience. I would like to express my gratitude to Dr. Orhan Özgür AYBAR, who duly fulfilled his valuable and advisory status.

I would like to express my gratitude to my university professors for their sincere endeavour and contributions to my improvement. I would like to thank my wife, Konul Karimova, who has always supported this study by tolerating and bearing the heavy burdens I left upon her during the study.

Mustafa ŞENTÜRK

TABLE OF CONTENTS

TABLE OF FIGURES

LIST OF SYMBOLS / ABBREVIATIONS

t	Time
t_{inc}	Incubation period
t_h	Hospitalization period
T_e	Transition to Exposed group
T_i	Transition to Infected group
κ_{old}	Survival percentage of old people
κ_{young}	Survival percentage of young people
ϕ	Average number of infected people from same infective person
R_s	Number of re-susceptible people
ξ	rate of resusceptibility
β	Average contact number per unit time of an infective person
σ	Coefficient representing the effect measurements
S	Group of susceptible people
E	Group of people exposed to disease
I	Group of infected people
R	Group of recovered people
D	Group of people lost their lives
μ	Natural death rate
Δ	Death rate due to disease

1. INTRODUCTION TO EPIDEMIC MODELS

1.1. Introduction

Epidemic models have been developed to be used for the analysis and predictions of the outcomes of epidemics. The Covid-19 outbreak in 2019 brought these models to the fore and motivated scientists to search more accurate and useful models.

Various differential models have been used to simulate epidemics. Among the most commonly used models are the Susceptible-Infected-Removed (SIR) and Susceptible-Exposed-Infected-Removed (SEIR) models. These models divide the population into the groups and each individual belongs only to one or another group. These models consist of ordinary differential equations (ODE)[1] or delay differential equations (DDE)[2, 3, 4]. There are also different approaches than ODE or DDE such as Markov stochastic epidemic models that are used to represent the stochastic interactions and examine the stochastic nature of the epidemic systems[5].

The SIR or SEIR models assume that the rates of transition between the groups are constant proportions of the group. In other words, a proportion of each group moves to the next group each time step. This approach is only useful if it is used over a period of time where the behavior of the system can be described by constants. When the studies were carried out over long periods, including epidemic waves, the need for time-dependent coefficients arose[5, 6, 7]. As a result, some of the studies focused on modified models using time-dependent coefficients and used heuristic methods to find the best fit to epidemic records[8]. One of the most influential motivations for these studies was to be able to simulate the effect of measures taken by governments to prevent the epidemic. So far, the time-dependent coefficients with heuristic optimizations are little more than a complex curve-fitting problem with limited capability to provide insight into the behavior of the system.

There are some studies dedicated to only one transition of an epidemic system such as recovery. El Haimar et.al. and Paul et.al. present a recovery model of influenza as a stochastic model, and offer triangular distribution as the probabilistic distribution of the recovery[6, 9]. Studies are carried out to examine the periods of the recoveries from the SARS-COV-2 disease. It has been revealed that most of the individuals with symptoms get recovered in three weeks and others may take more. This model more accurately represents the transition from infected to recovered compartments. A single time delay cannot generate, predict, reconstruct, and relate the daily recovery curve from the daily new cases as the triangular distribution laid out by[9]. The reason is that when a group of people become infected, they get recovered from the infection in various time periods, which forms a distribution instead of a delay.

In the Figure 1, the delay between daily new cases and daily recovery graphs is obvious. However, a single time delay cannot produce/predict/reconstruct/relate the daily recovery graph from the daily new cases. The reason is that when a group of people get infected, they get recovered from the infection in various time periods. Studies are held to examine the periods of the recoveries from the sars-cov-2 disease[19][20] have revealed that majority of the infected people get recovered in a month (20% in 10 days and 80% in a month according to the study of Liu at al.) and others need more then a month to get recovered especially when a co-morbidity is seen, even several months are needed for some serious cases.

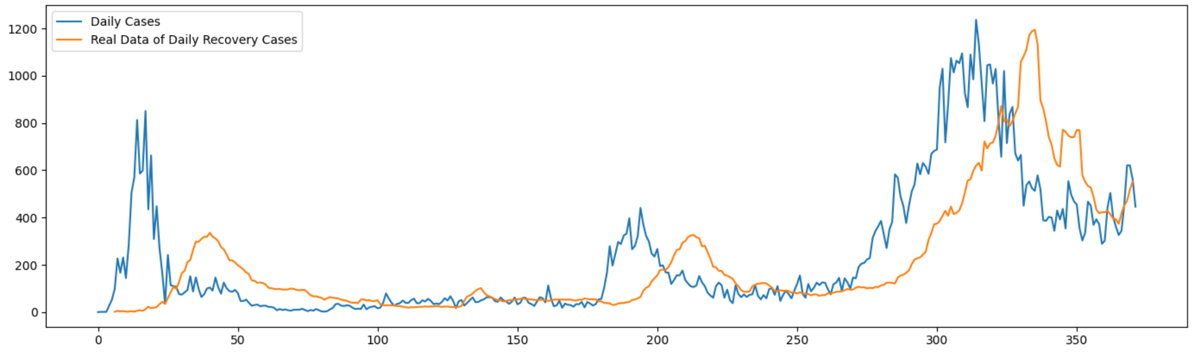


Figure 1.1 Chart of Daily Cases and Daily Recoveries reported in South Korea.

We believe that an epidemic model will be useful for projection of an epidemic development only when the model exhibits meaningful relationships between the actors of the system, and allows meaningful predictions to be derived by studying various scenarios[7, 10]. So we propose a modified SEIR model that incorporates a triangular probabilistic distribution for the recovery transition and time delay for the incubation. In this way, some epidemic characteristics specific to certain societies will be revealed and defined to be used in further studies.

1.2. SIR and SEIR Models

Epidemic models have been developed to be used for the analysis and predictions of the outcomes of epidemics. The Covid-19 outbreak in 2019 brought these models to the fore and motivated scientists to search more accurate and useful models.

Various differential models have been used to simulate epidemics. Among the most commonly used models are the Susceptible-Infected-Removed (SIR) and Susceptible-Exposed-Infected-Removed (SEIR) models. These models divide the population into the groups and each individual belongs only to one or another group. These models consist of ordinary differential equations (ODE)[1] or delay differential equations (DDE)[2, 3, 4]. There are also different approaches than ODE or DDE such as Markov stochastic epidemic models that are used to represent the stochastic interactions and examine the stochastic nature of the epidemic systems[5].

The SIR or SEIR models assume that the rates of transition between the groups are constant proportions of the group. In other words, a proportion of each group moves to the next group each time step. This approach is only useful if it is used over a period of time where the behavior of the system can be described by constants. When the studies were carried out over long periods, including epidemic waves, the need for time-dependent coefficients arose[5, 6, 7].

As a result, some of the studies focused on modified models using time-dependent coefficients and used heuristic methods to find the best fit to epidemic records[8]. One of the most influential motivations for these studies was to be able to simulate the effect of measures taken by governments to prevent the epidemic. So far, the time-dependent coefficients with heuristic optimizations are little more than a complex curve-fitting problem with limited capability to provide insight into the behavior of the system.

Numerous studies have been dedicated to exploring individual transitions within epidemic systems, with a specific focus on the recovery phase. El Haimar et al. and Paul et al. have contributed to this field by presenting a stochastic model for influenza recovery, employing the triangular distribution as a probabilistic representation [6, 9]. Similarly, research has been conducted to investigate recovery periods in individuals affected by SARS-COV-2. These investigations have revealed that most symptomatic individuals recover within three weeks, while others may require longer durations. The adoption of this recovery model, which accurately represents the transition from the infected to the recovered compartments, proves essential. Traditional single time delay models fail to adequately capture and predict the daily recovery curve based on daily new cases, as indicated by the triangular distribution proposed by [9]. This deficiency stems from the fact that infected individuals recover over varying time periods, resulting in a distribution rather than a singular delay.

1.3. History of Epidemic Analysis

The study of infectious disease data dates back centuries. John Graunt's book, "Natural and Political Observations made upon the Bills of Mortality" (1662), initiated this field by analyzing weekly records of deaths in London parishes. Graunt's work identified causes of death and estimated comparative risks for diseases, contributing to the understanding of competing risks.

Daniel Bernoulli is recognized as a pioneer of mathematical epidemiology. In the 18th century, he examined the benefits and risks of variolation for smallpox, calculating potential increases in life expectancy if smallpox were eliminated as a cause of death. Although his work gained more recognition in actuarial literature, it has since been further developed.

These historical milestones paved the way for understanding disease transmission. John Snow's study of the 1855 cholera epidemic highlighted contaminated water as a source of infection, while William Budd's research advanced knowledge of typhoid transmission. These contributions shaped our current understanding of epidemiology[21].

Maybe the first example of compartmental models is proposed by Hammer[22]. He divided the population into susceptibles and infectives. Kermack and McKendrick followed him with their paper[23] and continued to contribute to the development of mathematical theory of epidemics.

1.4. Research on Recovery Rates

xtensive scientific research has been conducted to investigate the recovery rates of diseases, providing valuable insights into disease behavior. A notable study by Liu et al. compares the recovery periods of Covid-19 and influenza, revealing that Covid-19 has a longer recovery period. Examining a cohort of 2904 cases, the study reported that 20% were recovered at the end of day 10, 60% at the end of day 20, 80% at the end of day 30, 91% at the end of day 60, 93% at the end of day 90 and 96% at the end of day 120 days. The median of the distribution of recovery periods is 16 days[19].

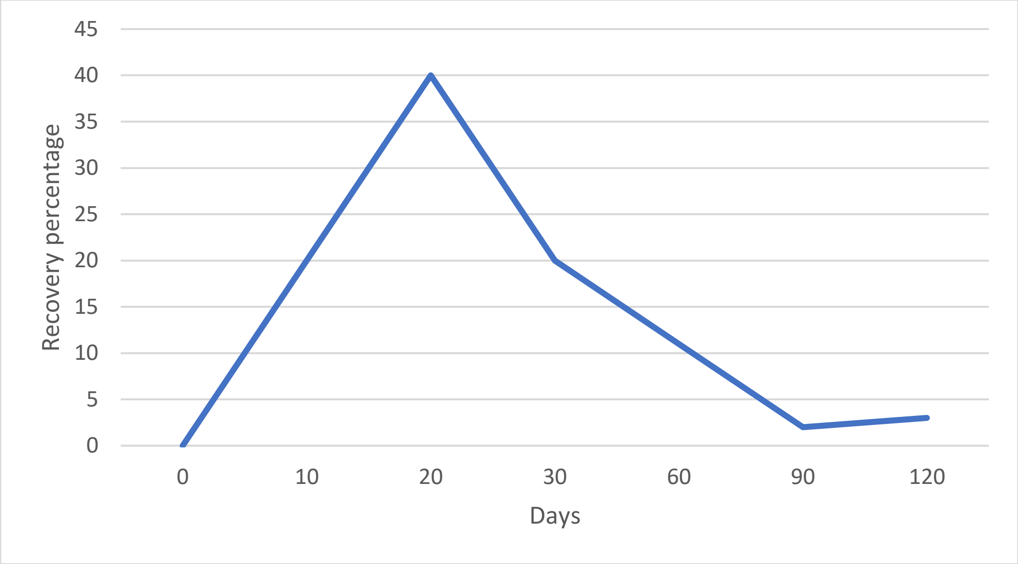


Figure 1.2 Recovery distribution of the results from Liu at al.

Another research belongs to Abrahim at al. studies the recovery rates of Covid-19 by investigating adult cases at Eka Kotebe General Hospital, Ethiopia[20] The study indicates that the median of the distribution of the recovery periods is 19 days within the range from 2 days to 71 days.

Paul, S. and Loring, E. presents more detailed research on the obtain the distributions of the recovered and decease periods based on Canada region (Figure 1.3). Despite these specific and advanced approaches in modeling recovery distributions, we believe that more simple yet effective approach is needed. Under the light of these research we believe triangular probabilistic distribution results in a good approximation to reality.

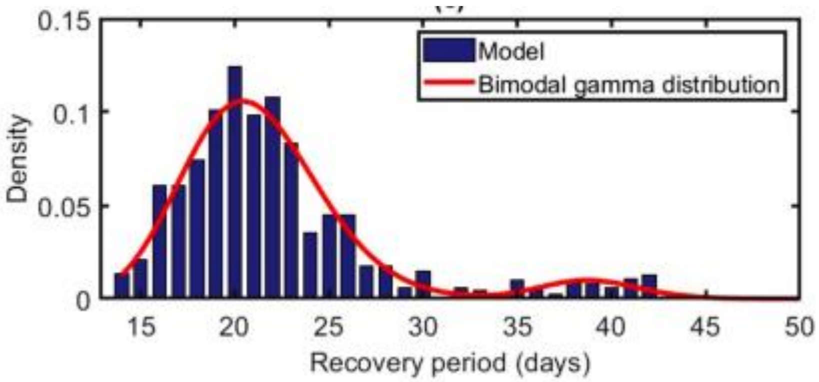


Figure 1.3 Probability density function of the bimodal gamma distribution[24]

2. Model Development

We modified the model proposed by Kok Yew Ng et al. (see [1]) by implementing a transition equation H(t) that defines the number of recovered individuals. The suggested SEIR model can be expressed using the following compartments

N(t): total stock population defined as N(t) = S (t) + E(t) + I(t) + R(t) + D(t),
S(t): individuals who are susceptible to disease,
E(t): number of individuals who have been exposed to virus and are in the incubation period of the virus,
I(t): infected individuals which show symptoms and are capable of infecting others,
R(t): individuals recovered from the disease,
D(t): individuals who have died due to illness.

The SEIR model contains time delays for transitions between the following groups,
t_{inc} : average incubation time,
t_{rec} : average time spent for the treatment before recovery,
t_h : average time of infection from the beginning of symptoms to the end of the treatment due to death or recovery.

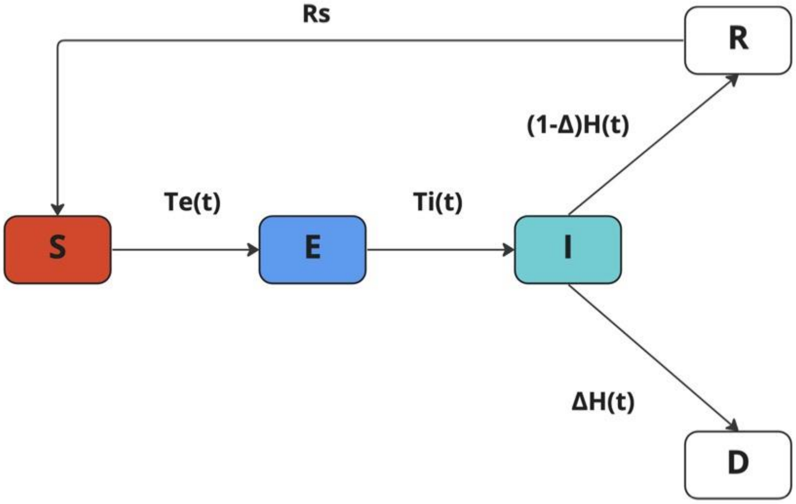


Figure 2.1 Graph of the SEIR Model.

Our proposed SEIR model is explicitly given by following set of equations:

$$
\begin{aligned}
\frac{ds}{dI} &= -\mu S(t) - \frac{\beta(1-\sigma)S(t)I(t)}{N(t)} + \xi R(t),\\
\frac{dE}{dt} &= \frac{\beta(1-\sigma)S(t)I(t)}{N(t)} - \frac{\beta(1-\sigma)S(t-t_{inc})I(t-t_{inc})}{N(t-t_{inc})} - \mu E(t),\\
\frac{dI}{dt} &= \frac{\beta(1-\sigma)S(t-t_{inc})I(t-t_{inc})}{N(t-t_{inc})} - H(t) - \mu I(t),\\
\frac{dR}{dt} &= (1-\Delta)H(t) - (\xi+\mu)R(t),\\
\frac{dD}{dt} &= \Delta H(t)
\end{aligned}
\tag{2.1}
$$

where μ is natural population change, ξ is the rate of resusceptibility, $R_s = \xi R(t)$ is the number of re-susceptible individuals, Δ is the ratio between death to total recovery, and $(1-\Delta)$H(t) is the number of people recovered from infection with $\Delta = \delta\left((1-\kappa_{old})N_{old} + (1-\kappa_{yound})(1-N_{old})\right)$. H(t) becomes the number of recovered individuals if Δ equals to 0. The time spent hospitalized or infected in fatal cases can be assumed as by $t_h = \frac{1}{\delta}$[1] and $\frac{dN}{dt} = -\Delta H(t) - \mu N(t)$. β is the rate for average contact number of the infected and determined by the equation $\frac{\phi}{t_{rec}}$ where ϕ is the average number of individuals who are infected by the same infectious individual. σ is the efficiency of the measures and $\frac{S(t)}{N(t)}$) represents the possibility of the susceptible contacts per infected individual. The block diagram of proposed model is given in figure 1.

The transition functions Ti(t) and Te(t) are defined as the daily number of new infected people

$$Ti(t) = Te(t - t_{inc}) \tag{2.2}$$

and the daily number of individuals who are exposed to the virus

$$Te(t) = \frac{\beta(1-\sigma)S(t)I(t)}{N(t)} \tag{2.3}$$

respectively.

2.1. Boundedness of the Model

Lemma 1 The overall population N(t) is finitely bounded for any non-negative initial condition.
Proof 1 Since E(t) appears only by using $\lim_{t->\infty} S(t) = \lim_{t->\infty} S(t - t_{inc}) = S^*$ and $\lim_{t->\infty} I(t) = \lim_{t->\infty} I(t - t_{inc}) = I^*$, $\lim_{t->\infty} \frac{dE}{dt} = -\mu E(t)$. Under these assumptions, the second equation $\frac{dE^*}{dt}$ is equal to $-\mu E^*$(t). The overall population N^*+D^* should be constant when the natural population change μ is zero where $N^*(0)$ = N0 is initial population. It is suitable to consider the steady state equations of individuals depending on N^*+D^* as

$$\frac{dN}{dt} + \frac{dD}{dt} = \frac{dS}{dt} + \frac{dE}{dt} + \frac{dI}{dt} + \frac{dR}{dt} + \frac{dD}{dt} \leq -\mu(N^*) \tag{2.4}$$

By following the equation 2.4, the population of each compartment in the proposed model is non-negative for all t ≥ 0 for any finite nonnegative initial conditions.

When we assume $N^* >> D^*$, the equation $\frac{dN^*}{dt} \cong -\mu(N^* - D^*)$ becomes $\frac{dN^*}{dt} \cong -\mu N^*(t)$. The integral solution gives $0 < N^*(t) \leq N^0 e^{-\mu t}$ for all t where $N^*(0) = N^0$.

2.2. Types of Endemic Cases

In this subsection, we study the difference between bilinear incidence rate and standard incidence rate on stability.

2.2.1. Effect of Bilinear Incidence Rate on System

There are two equilibrium points of the system 2.1 under the assumption $\beta s = \beta(1 - \sigma)$ with the incidence rate $\frac{\beta_s S(t) I(t)}{N(t)}$. The equation $\frac{dD}{dt} = \Delta H(t)$ is removed for the stability analysis since the other equations in system 2.1 do not depend on it directly. The disease free equilibrium point of system (2.1) is $E_0 = (\frac{R_x}{\mu}, 0,0, -\frac{R_x}{\mu})$ where the system is free from infected and individuals exposed to virus and the entire population is susceptible and recovered from the disease. The Jacobian matrix of system 2.1 is evaluated at E0, one gets the following matrix:

$$J_{E_e} = \begin{bmatrix} -\mu & 0 & -\frac{\beta_s R_s}{\mu N} & 0 \\ 0 & -\mu & 0 & 0 \\ 0 & 0 & -\mu - \frac{\beta_s R_s}{\mu}(1 - \frac{1}{N}) & 0 \\ 0 & 0 & \frac{\beta_s R_s}{\mu}(1 - \Delta) & -\mu \end{bmatrix}$$

with the eigenvalues $\lambda_{1,2,3} = \{-\mu\}$ and $\lambda_4 = \left\{-\mu + \frac{\beta_s R_s}{\mu}(-1 + \frac{1}{N})\right\}$.

The endemic equilibrium point of system (2.1) is given by

$$E_e = \left(\frac{\mu N}{\beta_s(1-N)}, 0, -\frac{\mu N}{\beta_s} + \frac{R_s(1-N)}{\mu}, \frac{(1-\Delta)\mu N^2}{\beta_s(N-1)} + \frac{R_s\big(-1 + N(1-\Delta)\big)}{\mu}\right) \qquad (2.5)$$

The Jacobian matrix of system 2.1 is evaluated at E0, one gets the following matrix:

$$J_{E_e} = \begin{bmatrix} \frac{\beta_s R_s(-1+N)}{\mu N} & 0 & \frac{\mu}{-1+N} & 0 \\ 0 & -\mu & 0 & 0 \\ \mu(-1+\mathrm{N}) + \frac{\beta_s R_s(-1+N)^2}{\mu N} & 0 & 0 & 0 \\ \frac{(-1+\Delta)(\mu^2 N + \beta_s R_s(-1+N))}{\mu} & 0 & \frac{\mu N(-1+\Delta)}{-1+N} & -\mu \end{bmatrix}$$

with the following eigenvalues $\lambda 1,2,3 = \{-\mu\}$ and $\lambda_4 = \left\{\frac{\beta_s R_s}{\mu}(1-\frac{1}{N})\right\}$. We first study stability of the equilibrium point E0, we consider the characteristic polynomial $(\lambda+\mu)^3(\lambda+\mu+\frac{\beta_s R_s(-1+N)}{\mu N})$. According to the Routh-Hurwitz criteria, the disease free equilibrium point is asymptotically stable when $\mathrm{Rs} > 0, \mu > 0, \beta\mathrm{s} > 0, 0 < \mathrm{N} < \frac{18\beta_s R_s}{1+24\mu^2+\sqrt{1+12\mu^2}+18\beta_s R_s}$.

The characteristic polynomial at endemic equilibrium point E_e is given by $\frac{(\lambda+\mu)^3(\lambda\mu N+\beta_s R_s-N(\mu^2+\beta_s R_s))}{\mu N}$. Applying the Routh-Hurwitz criteria, E_e is stable if the following conditions are satisfied $\mathrm{N} > 1\ 2\mathrm{W}1 \wedge \mathrm{R} > 0 \wedge R_s > 0 \wedge \mathrm{S} > 0 \wedge (\mathrm{W}2 \vee (\beta_s > 0 \wedge 2\mu > 1))$, where $W_1 = R_s\sqrt{\frac{\beta_s^2(12\mu^2+1)}{(\mu^2(12\beta_s R_s-1)+\beta_s R_s(9\beta_s R_s-1)+4\mu^4)^2}} + \frac{\beta_s R_s(18\beta_s R_s+12\mu^2-1)}{\mu^2(12\beta_s R_s-1)+\beta_s R_s(9\beta_s R_s-1)+4\mu^4}$ and $W_2 = R_s\left(-18\beta_s R_s - 12\mu^2 + \sqrt{12\mu^2+1}+1\right) < 0 \wedge 0 < \mu \leq \frac{1}{2}$. In the following remarks, we give some results for possible endemic cases depending on parameter values.

Remark 1 When $R_s = \xi R(t) = 0$, E_0 and E_e are not stable.
Remark 2 When $\beta_s = 0$, there is only one unstable equilibrium namely E_0.

2.2.2. Effect of Standard Incidence Rate on System 3.1

There is only one equilibrium point (disease free) $E_0 = (\frac{R_s}{\mu}, 0,0, -\frac{R_s}{\mu})$ under the assumption $\beta_s = \frac{\beta(1-\sigma)}{N(t)}$. To do stability analysis when he jacobian one gets the following matrix:

$$J_{E_e} = \begin{bmatrix} -\mu & 0 & -\frac{\beta_s R_s}{\mu} & 0 \\ 0 & -\mu & 0 & 0 \\ 0 & 0 & -\mu & 0 \\ 0 & 0 & \frac{\beta_s R_s}{\mu}(1-\Delta) & -\mu \end{bmatrix}$$

with the eigenvalues λ1,2,3 = {−μ}.

Remark 3 Standard incidence rate gives unstable disease free equilibrium point E_0.

2.3. Boundedness of Population

The population N(t) shall be finitely upperbounded as initial conditions are always nonnegative. To prove this proposition we can derive state equations on a time $t_f > t_0$ from the equations (2.1) if we assume that

$$\beta_s = \frac{\beta(1-\sigma)}{N(t)} \tag{2.6}$$

and

$$H(t) = \beta_s S(t-t_h) I(t-t_h) \tag{2.7}$$

Then the state equations of the system become

$$\begin{aligned}
S(t_f) &= S_0 - \int_{t_0}^{t_f} \beta_s S(t) I(t) + \int_{t_0}^{t_f} \xi R(t) - \int_{t_0}^{t_f} \mu S(t), \\
E(t_f) &= E_0 + \int_{t_0}^{t_f} \beta_s S(t) I(t) - \int_{t_0}^{t_f} \beta_s S(t-t_{inc}) I(t-t_{inc}) - \int_{t_0}^{t_f} \mu E(t), \\
I(t_f) &= I_0 + \int_{t_0}^{t_f} \beta_s S(t-t_{inc}) I(t-t_{inc}) - \int_{t_0}^{t_f} \beta_s S(t-t_h) I(t-t_h) - \int_{t_0}^{t_f} \mu I(t), \\
R(t_f) &= R_0 + (1-\Delta) \int_{t_0}^{t_f} \beta_s S(t-t_h) I(t-t_h) - \int_{t_0}^{t_f} (\xi+\mu) R(t), \\
D(t_f) &= D_0 + \Delta \int_{t_0}^{t_f} \beta_s S(t-t_h) I(t-t_h)
\end{aligned} \tag{2.8}$$

where S 0, E0, I0, R0, D0 are the initial values. Now as the initial population N0 is constant and $N(t) = \mathrm{S(t)} + \mathrm{E(t)} + \mathrm{I(t)} + \mathrm{R(t)}$. As the equation of N(t) is

$$N(t) = N_0 + D_0 - D(t) - \int_{t_0}^{t_f} \mu N(t), \tag{2.9}$$

And

$$N_0 = S_0 + E_0 + I_0 + R_0 \tag{2.10}$$

Then

$$\begin{aligned} &\mathrm{S(t)} + \mathrm{E(t)} + \mathrm{I(t)} + \mathrm{R(t)} + D(t) \\ &\qquad = N_0 + D_0 - \int_{t_0}^{t_f} \mu(\mathrm{S(t)} + \mathrm{E(t)} + \mathrm{I(t)} + \mathrm{R(t)}) \end{aligned} \tag{2.11}$$

When we sum the state equations we see that

$$\begin{aligned} &\mathrm{S(t)} + \mathrm{E(t)} + \mathrm{I(t)} + \mathrm{R(t)} + D(t) \\ &\qquad = S_0 + E_0 + I_0 + R_0 + D_0 - \int_{t_0}^{t_f} \mu(S(t) + E(t) + I(t) + R(t)) \end{aligned} \tag{2.12}$$

This proves the equation (2.9).

2.4. Nonnegative Population

The population shall always be nonnegative. From the equation (2.9) we have derived N(t) equation. The condition can be defined as

$\frac{dN}{dt} < N(t)$ should be true for any t

and

$$N_0 + D_0 - D(t) - \int_{t_0}^{t_f} \mu N(t) \geq 0 \tag{2.13}$$

$$N_0 > D(t) - D_0 + \mu \int_{t_0}^{t_f} N(t) \tag{2.14}$$

$$\frac{dN}{dt} = -\frac{\Delta\beta(1-\sigma)S(t-t_h)I(t-t_h)}{N(t)} - \mu N(t) \tag{2.15}$$

$-\frac{dN}{dt} < N(t)$ should be true for any t

We know that $N(t) \gg D(t), D(t) > 0, \frac{S(t-t_h)I(t-t_h)}{N(t)} < N(t), \mu \ll 1, \Delta \ll 1,$

$$\begin{gathered}
\frac{\Delta\beta(1-\sigma)S(t-t_h)I(t-t_h)}{N(t)} + \mu N(t) < \mathrm{N(t)} \\
\frac{\Delta\beta(1-\sigma)S(t-t_h)I(t-t_h)}{N(t)} < (1-\mu)\mathrm{N(t)} \\
\Delta\beta(1-\sigma) < \frac{\mathrm{N(t)}^2}{S(t-t_h)I(t-t_h)}
\end{gathered} \tag{2.16}$$

The condition is satisfied as long as

$$\beta < \frac{\mathrm{N(t)}^2}{\Delta(1-\sigma)S(t-t_h)I(t-t_h)} \tag{2.17}$$

3. Probabilistic Triangular Distribution

The nature of epidemic growth relies heavily on random interactions in a population. One of the most important mechanisms of an epidemic model is the recovery mechanism. Individuals of the infected group of the model recover after a period of time that varies from individual to individual. We believe that this phenomenon can be modeled more accurately by defining the time lags as probabilistic distribution. Therefore, we propose a novel approach to simulate recovery transitions using probabilistic distribution in delay differential equations with a continuous time delay window.

El-Haimar et.al. shows that probabilistic distribution of recoveries of the infected populations can be modeled as a triangular probabilistic distribution[6]. So, a new model is developed by implementing triangular probabilistic distribution into the delay differential equation system of SEIR model on the basis of the model developed by Kok et. al.[1].

We now define the next theorem about the transition function for recovery by triangular probabilistic distribution depending on Lemma 1.

3.1. Theorem of Triangular Distribution as Multi-Delay

Triangular distribution is proposed and represented by the function H(t) which represents the recoveries per time where the casualties are ignored, in other words the number of recovered people at time t. As it was mentioned before, the function will be derived from triangular probabilistic distribution. Continuous Triangular Probabilistic Distribution is given by

$$p(x) = \begin{cases} \dfrac{2x}{Lc} & if\ 0 \leq x \leq c, \\ \dfrac{2(L-x)}{L(L-c)} & if\ c < x \leq L, \end{cases} \tag{3.1}$$

where ta is time delay for the first recovery occurrence, c is the mid point of the triangle, (an infected person is most probably will be recovered after a time of t_a + c), and L is length of the distribution (a person, for certain, will be recovered after a time of $t_a + L$). Ti(t) defines the distribution of transform from E to I over R as

$$Ti(x) = \beta(1-\sigma)S(t-t_{inc})\frac{1}{N(t-t_{inc})} \tag{3.2}$$

Ti(t) transition function is also delay function of Te(t) from E to I where Te(t) is the transition function from S to E, Ti(t) = Te(t − tinc). Thus transition function H(t) becomes as follows

$$H(t) = \int_{x=t-(L+t_a)}^{x=t-t_a} P\big((t-t_a)-x\big)Ti(x)dx, \tag{3.3}$$

where P(x) is the probability density as given by equation 3.1.

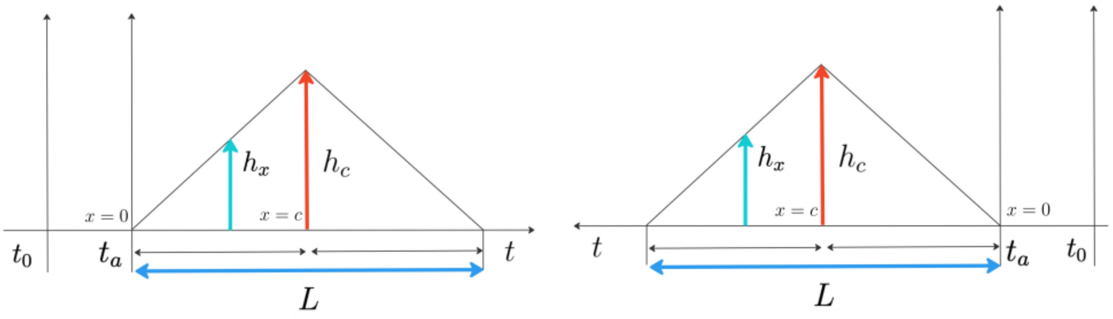


Figure 3.1 Triangle of Probabilistic Distribution (left) and (b) Inverse triangle of Probabilistic Distribution (right).

3.2. Proof of the Theorem

We express the recovery time distribution of the people which have become infectious at t0 with a probabilistic distribution. Each individual will at infectious at least for a duration of ta for certain. After a duration of $t_a + L$ all the infectious people will be recovered (or dead) from the infection. After a time period of ta, the infectious people (who had become infectious at t_0) gets recovered from the infection according to a P(x) distribution. We defined x as time variable and h for the probability in order to distinguish it from the general time indices of t and population.

Boundary condition of the triangle is that the total area of the distribution should be equal to 1 and at x = 0, h = 0 and at x = L, h = 0,

$$A_{total} = \frac{h_c L}{2} = 1 \tag{3.5}$$

hence hc = 2/L and the functions p(x) are obtained as follows

$$p(x) = \begin{cases} dx\,\frac{2x}{Lc} & if\ 0 \le x \le c, \\ dx\,\frac{2(L-x)}{L(L-c)} & if\ c < x \le L, \end{cases} \tag{3.6}$$

$$p_1(x) = \frac{dxh_x}{A_{total}} = \frac{dx(L-x)\frac{h_c}{l_1}}{1} = \frac{2x}{Lc}dx, \tag{3.7}$$

$$p_2(x) = \frac{dxh_x}{A_{total}} = \frac{dx(x)\frac{h_c}{l_2}}{1} = \frac{2(L-x)}{L(L-c)}dx, \tag{3.8}$$

where $l_1 + l_2 = L$. However, this function p(x) shows the distribution of the number of patients who got better at an instant t_0. So now we shall convert this function into the number of recovered patients at time t_0 as shown in figure 2a. For this we use the inverse of the probabilistic distribution as given in figure 2b, since we know that those who get better at an instant t_0 are among those who had become infectious in a time interval in the past and they recovered from the disease according to the probabilistic distribution.

Function Q(t) gives the probability of recovery at t_0 for an individual which had become infectious at any t

$$Q(t) = P\big((t_0 - t) - t_a\big) \tag{3.9}$$

where (t0 − (ta + L)) < t < t0 − ta. Using the equations we have obtained so far, we define our function H(t). H(t) is removing rate function or transition from I to R and D, it gives the number of recovery of infectious individuals from disease at time t where the casualties are ignored. For any t > 0,

$$H(t) = \int_{x=t-(L+t_{a)}}^{x=t-t_a} Q(x)Ti(x)dx, \quad (3.10)$$

and from equation 4.7, we obtain the following result:

$$H(t) = \int_{x=t-(L+t_{a)}}^{x=t-t_a} P\big(t-(t_a+x)\big)Ti(x)dx, \quad (3.11)$$

The function H(t) has been obtained from equation 2.15. As the H(t) is the integration of the multiplication of P and Ti , then numeric integration can be used to obtain the H(t) function from the discrete values of the multiplication. Trapezoidal Rule is used in our model for the numeric integration in the form:

$$H(t) = [P(L)Ti(tL) + P(L-1)Ti(tL+1) + P(L-2)Ti(tL+2) + \ldots + P(0)Ti(tL+L)]$$

$$= \sum_{i=0}^{L} P(L-i)Ti(t_L+i) \quad (3.12)$$

3.3. Implementation of Triangular Distribution

The model shall be implemented into numeric solvers to be useful. AS the system depends on the delays we focused on the DDE23 algorithm. We implemented the Delay Differential

Equations into DDE23 algorithm with multi delays. However, we decided that we can simplify the equations without harming the correctness and main attributes of this study.

3.3.1. Simplified Model

Assuming that the population is in equilibrium, and assuming that for a large population, natural death rates do not significantly affect the population in a short period of time, the natural death rate μ = 0, so we simplify the model as follows

$$
\begin{aligned}
&\frac{ds}{dI} = -\frac{\beta(1-\sigma)S(t)I(t)}{N(t)} + \xi R(t),\\
&\frac{dE}{dt} = \frac{\beta(1-\sigma)S(t)I(t)}{N(t)} - \frac{\beta(1-\sigma)S(t-t_{inc})I(t-t_{inc})}{N(t-t_{inc})},\\
&\frac{dI}{dt} = \frac{\beta(1-\sigma)S(t-t_{inc})I(t-t_{inc})}{N(t-t_{inc})} - H(t),\\
&\frac{dR}{dt} = (1-\Delta)H(t) - \xi R(t),\\
&\frac{dD}{dt} = \Delta H(t)
\end{aligned}
\tag{3.13}
$$

These equations will be used as the natural death is assumed insignificant for the time period of the data that is going to be analysed.

3.3.2. *DDE23 Algorithm*

In order to implement the H(t) function into DDE23, a span of time-delays can be used as an input given in the code block 1. Hence, H(t) will be obtained from the history of Ti (or $Te(t - t_{inc})$) for each time step and used in the equations.

```
lags = [t_inc, (1 : L) + (t_inc + t_0)]; % lag from instant exposed (Te)
sol = dde23(@ddefun, lags, @history, t_span);
```

Listing 1: MATLAB DDE23 code block

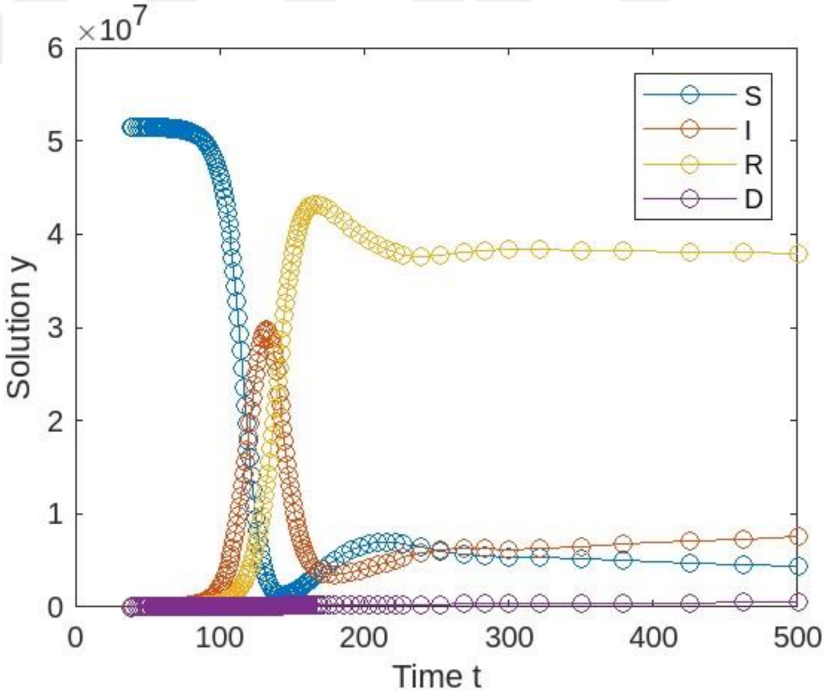
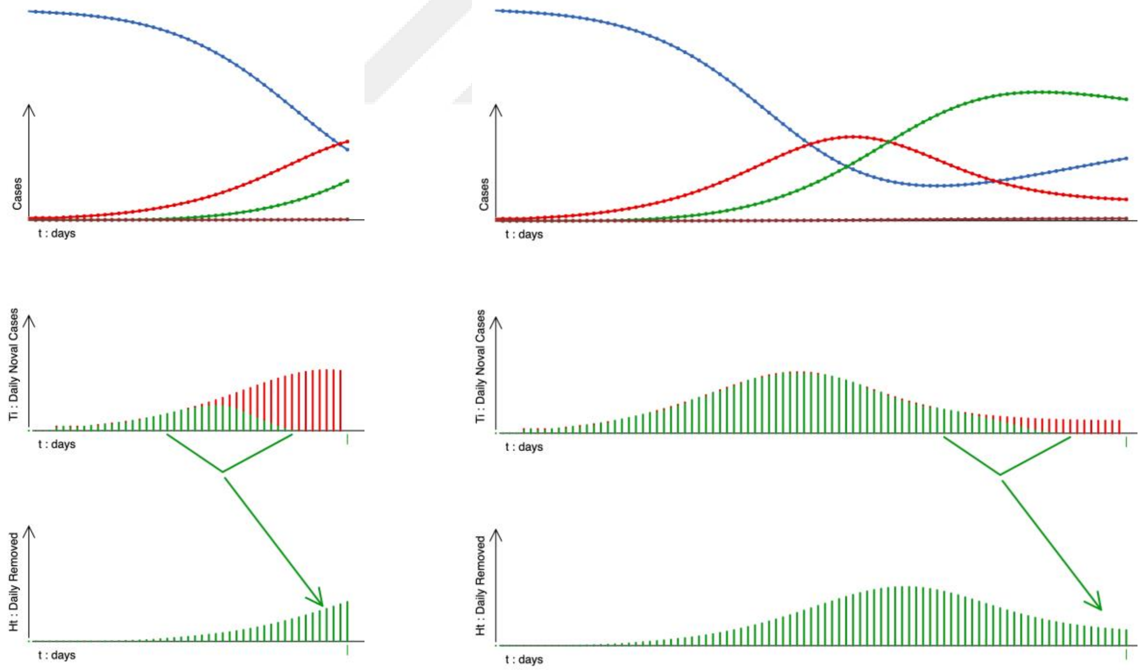


Figure 3.2 Example of the DDE23 run over S.Korea data with random coefficients

3.3.3. Discrete Form

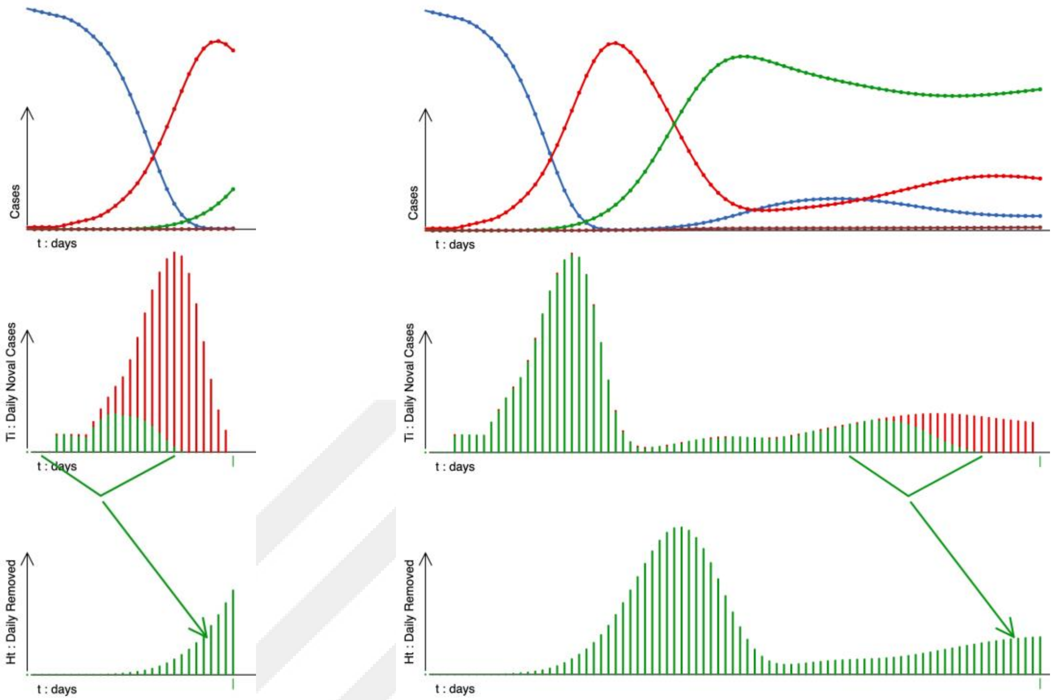
For the interactive simulation of the system, we developed a discrete form of the equations and implemented in a web application [http://kzlsahin.online/TriangularDistribution_ver3/].

Example 1 By using the theorem 1, we simulate the suggested novel model given in (2.1) with the following conditions (S_0, E_0, I_0, R_0) = (1000, 0, 10, 0) and parameter values $\Delta = 0.01$, $\xi = 0.02$, $t_{inc} = 4$, $x_0 = 4$, $c = 10$, $L = 18$, and time period is 180 unit. In figure 4a and 4b where $\beta_s = 0.2$, recovered individuals are covering the infected population then system becomes stable after a long time period with triangular distribution algorithm. When $\beta_s = 0.8$ is chosen, the infected population are exponentially increasing and the recoveries try to fill the red area as given in 4c.



(a) Short time behavior of system 2.1 for βs = 0.2

(b) Long time behavior of system 2.1 for βs = 0.2



(c) Short time behavior of system 2.1 for βs = 0.8

(d) Longtime behavior of system 2.1 for βs = 0.8

Figure 3.3 Solution of triangular distribution implemented system 2.1 where blue, red, green, brown colors represent S, I, R and D respectively

4. Optimization Using Non-Binary Genetic Algorithm

Genetic Algorithm is a search method for almost optimum solutions derived from the natural selection phenomenon of the evolution[11]. Genetic Algorithm consists of individuals with genotype g and phenotype x(g), which also represents a solution in a space of solutions for a given domain, and random operators which implements mutation and crossover of nature into the model. Results are compared according to a fitness function $\phi(g)$[12]. The triangle of the probabilistic distribution has been defined by three parameters and the solution space $T = \{x0, c, L \mid x0 \in Z^+, c \in Z^+, L \in Z^+\}$ as follows

x0 : time delay or the starting point (ta) of the triangular distribution
c : mode of the triangular distribution,
L : period of the triangular distribution.

Problem of finding the best values of the parameters of the triangular distribution which fit best to the real data, can be defined as a Set Covering Problem. A genetic algorithm with a non-binary representation is convenient for this kind of problems. The problem becomes a set covering problem by defining x0, c, and L as $T = \{x0, c, L \mid x0 \in [0, m], c \in [0, n], L \in [0, 1] \ \& \ x0 \in Z^+, c \in Z^+, L \in Z^+\}$ where T defines the genotype. When the data set of daily removed cases Ht is derived from daily infected cases of the epidemic using this genotype, the derived data set is the phenotype of corresponding to this genotype. Eremeev et al[12] offers Non-Binary Genetic Algorithm to solve this kind of problems.

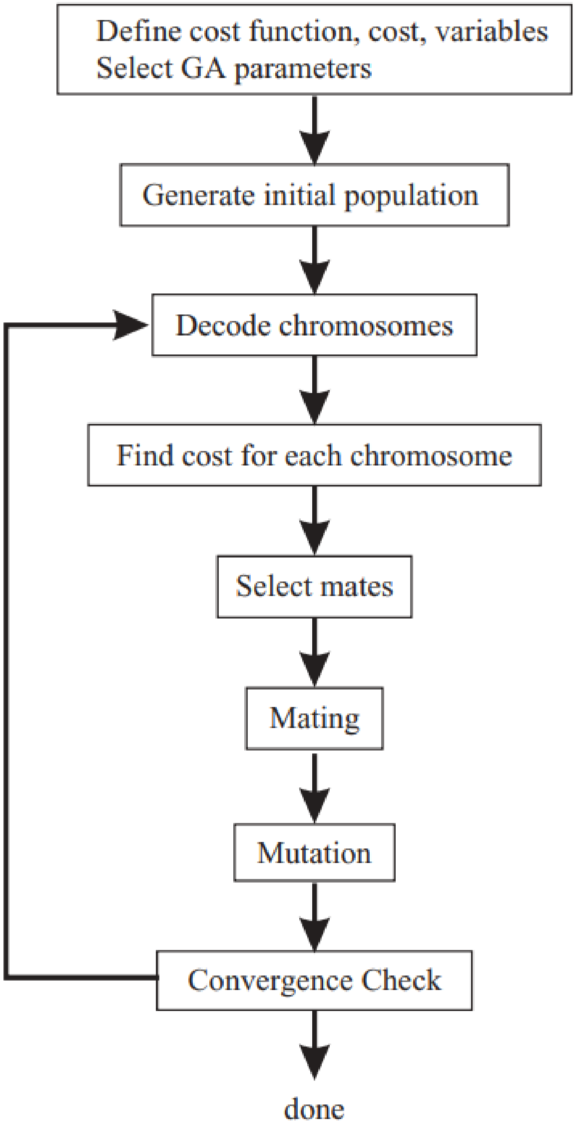


Figure 4.1 Flowchart of a genetic algorithm[25]

One of the important components of the Genetic Algorithm is the fitness function which evaluates the fitness of the genotype to the environment in nature. But here, the fitness function is used to evaluate the fitness of the derived dataset to a reference dataset. The problem reminds the curve fitting to a dataset using regression which is used to find least error, whereas Genetic Algorithm is used to find best fitness. So the fitness function should give higher values when the derived dataset is closer to the reference dataset. The fitness function is defined as $\phi(T) = 1\ 1+MS\ E$ where MS E defines the mean squared error of the dataset which will always be positive and $\phi(T) \in [0, 1]$.

For each iteration, the phenotype, Ht dataset is created for each individual and the fitness of the genotype is calculated. Then the genotypes are sorted by the fitness values. Elitist Selection method is preferred for the simplicity of the algorithm and top n genotypes are selected to survive in the next generation to preserve the best genotypes where the number of population is $n\ 2$. Therefore, as the other genotypes are replaced with the $n(n - 1)$ offsprings of the n survived genotypes preserves the population amount as $n\ 2$. Offsprings are produced by receiving each gen from one or another ancestor randomly. The mutation occurs with the probability of 0.05 during each gen transfer and the gen that the mutation occurs is replaced by a random value inside the related boundary. The initial generations are being created with random genotype according to defined boundaries $x0 \in (1, 15)$, $c \in (1, 10)$, and $L \in (10, 41)$. We show the simulation results depending on determination of parameters for triangular distribution by genetic algorithm in figures 4, 5, 6, and 7. The simulations are operated on short term optimized triangular distribution (423 days), short term optimized triangular distribution (1077 days), long term optimized triangular distribution (1077 days), and optimized on reference parameters, respectively.

4.1. Pseudo Code

The genetic algorithm is written in python. The algorithmic representation of the genetic algorithm code base is represented below.

Non-Binary Genetic Algorithm Pseudocode

```
Input:  daily_new_cases,
            daily_recovered,
            death_rate,
            max_iteration,
            gen_boundaries = {x0: (1,15), c: (1, 10), L: (10, 41)},
            mutationRate = 0.05,

Output:     Solution (x0,  c, L ),
```

```
Let n = 15        // number of survivals
Let N = n ^2      // number of population
Let Pop = [[individual]*N]
Let ref_ $H_t$ = daily_recovered * death_rate
Let Results = [[result]*N]

for i in range(N) do
   Pop.Add(Random Solution)
End
For i in range ( Max_iteration ) do
   For each individual in Pop do
      Let  distArr = GetTriangularDistributionOf ( individual )
      Let  MSE = Sqrt ( Mean ( ref_ $H_t$  - distArr ) )
      Let fitness = $\frac{1}{1+MSE}$
      Add ( individual, fitness ) to Results
   End
   Sort Pop;
   Let S  = [[individual]*n] // survivals
   Add Pop.Best(n) to S
   For each individual in S as parentA do
      For each individual in S as parentB do
         If Not parentA == parent do
            Let offspring = new individual
            Let rnd = random(0 or 1)
            Offspring.x0 = if (rnd = 1) parentA.x0 else parentB.x0
            Offspring.c = if (rnd = 1) parentA.c else parentB.c
            Offspring.L = if (rnd = 1) parentA.L else parentB.L
            Add individual to Pop
         End If
      End
   End
// now apply mutation
```

```
  For each individual in Pop other than S do
    For each gen in individual.Genotype do
      Let rnd = Random [0 to 1]
      If rnd < mutationRate do
        gen = random Gen
      End If
    End
  End
  Print Results
End
Return Best Solution of Pop;
```

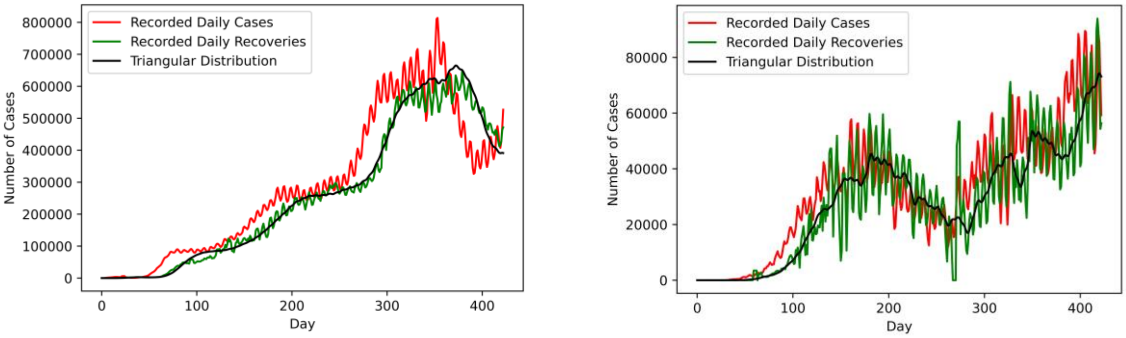
4.2. Optimization Results

The triangular distribution as mentioned in the previous section is based on three parameters: a, c, L. Since these parameters vary according to the health system and physiological resilience (as well as the methods and disciplines of keeping statistics) of the society being studied, these parameters need to be determined according to each society's own data. For this purpose, we believe that it is possible to determine these parameters using a genetic algorithm based on the number of cases per day and the number of recoveries per day. These parameters are calculated by using the genetic algorithm[25] based on data from six countries as given in table 1. These results show how the parameters vary for sample countries. Results of a reference solution of T = {x0, c, L | x0 = 0, c = 15, L = 30} over data of each country are also added to the table to be used as a comparison of the results.

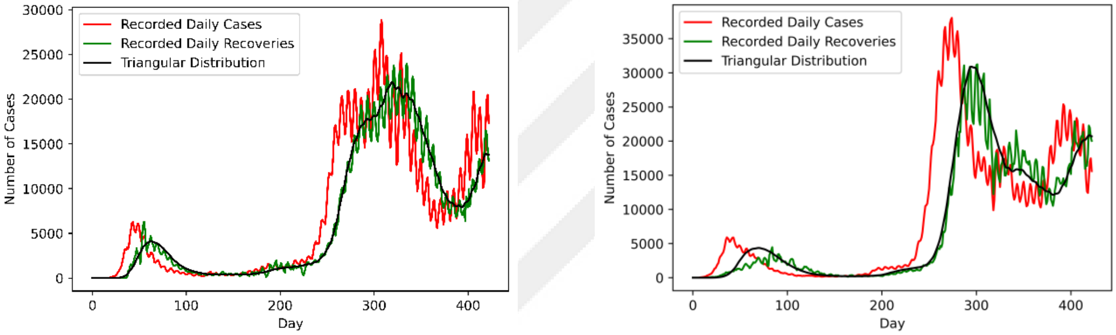
Table 1 Results of the triangular distributions on the six data sets

Countries	Optimization Period	X0	C	L	Fitness (x10^4)
Global	Long optimized (1077 days)	4	3	40	1,43
	Short optimized (423 days)	10	1	41	0,43
	Ref. Dist.	0	15	30	1,11
Brazil	Long optimized (1077 days)	10	10	15	8,97
	Short optimized (423 days)	10	10	15	8,97
	Ref. Dist.	0	15	30	8,05
Germany	Long optimized (1077 days)	7	10	34	8,63
	Short optimized (423 days)	3	1	41	7,82
	Ref. Dist.	0	15	30	7,01
Italy	Long optimized (1077 days)	4	3	28	16,53
	Short optimized (423 days)	10	1	41	8,30
	Ref. Dist.	0	15	30	15,92
Japan	Long optimized (1077 days)	5	10	11	3,60
	Short optimized (423 days)	5	6	11	3,33
	Ref. Dist.	0	15	30	3,29
S.Korea	Long optimized (1077 days)	10	8	15	14,26
	Short optimized (423 days)	5	10	31	11,27
	Ref. Dist.	0	15	30	9,55

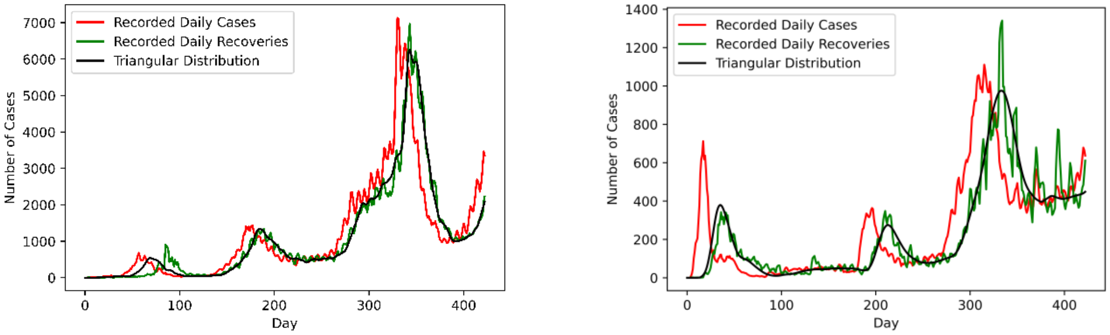
The significance of the triangular distribution is more obvious in the short term compared to the long term, as the outcomes of the distribution tend to approximate the results of a single time delay around the most probable point of occurrence in the probabilistic distribution. The results of the optimization in the table 1 show significant improvement in the MSE values comparing to the results of reference solution. However, it can be claimed that, a reference triangle without an optimization process would also be acceptable for preliminary analysis of a system, as the data of some countries, such as Japan, indicates how close can be results of the reference solution to the results of an optimized solution.



(a) Global Data (b) Brazil

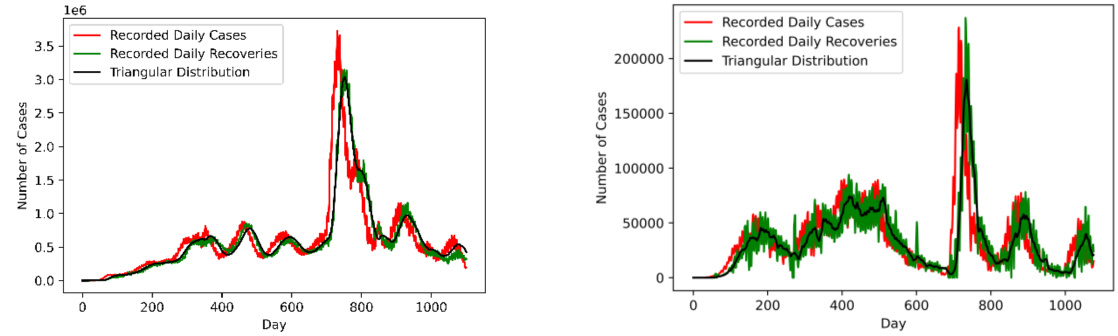


(c) Germany (d) Italy

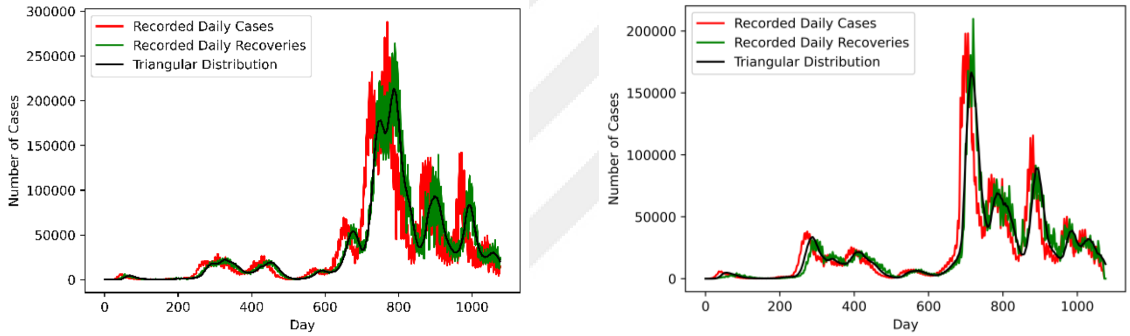


(e) Japan (f) South Korea

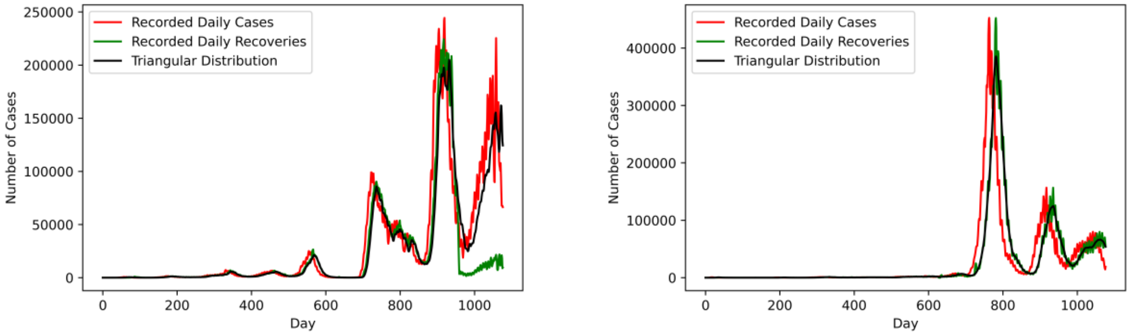
Figure 4.2 Results of the short term optimized triangular distribution charts (423 days)



(a) Global Data (b) Brazil

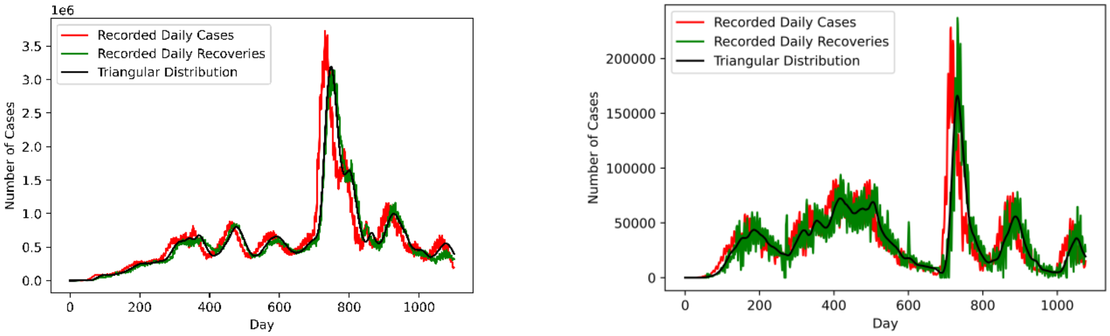


(c) Germany (d) Italy

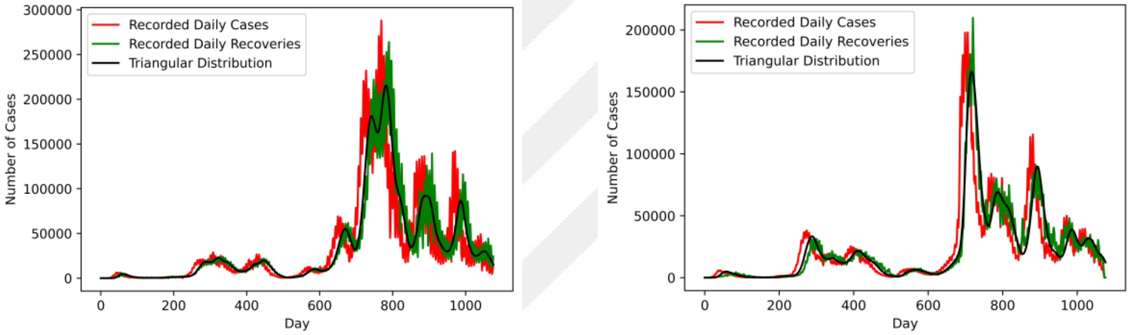


(e) Japan (f) South Korea

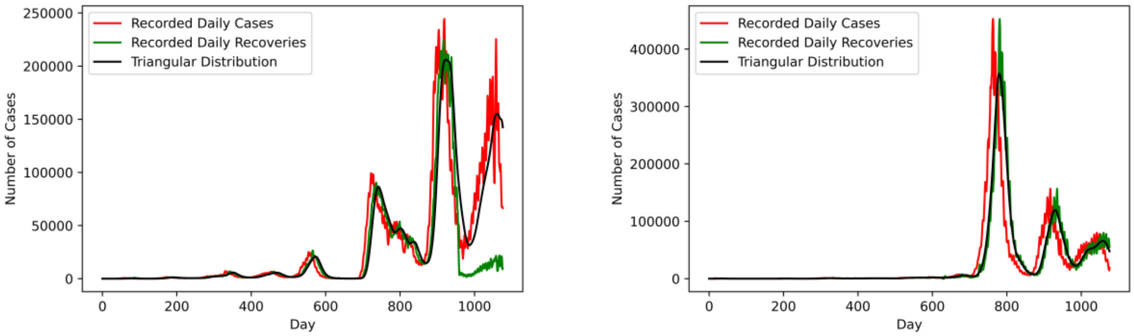
Figure 4.3 Results of the long term optimized triangular distribution



(a) Global Data (b) Brazil



(c) Germany (d) Italy



(e) Japan (f) South Korea

Figure 4.4 Results of the triangular distribution depending on reference parameters (1077 days)

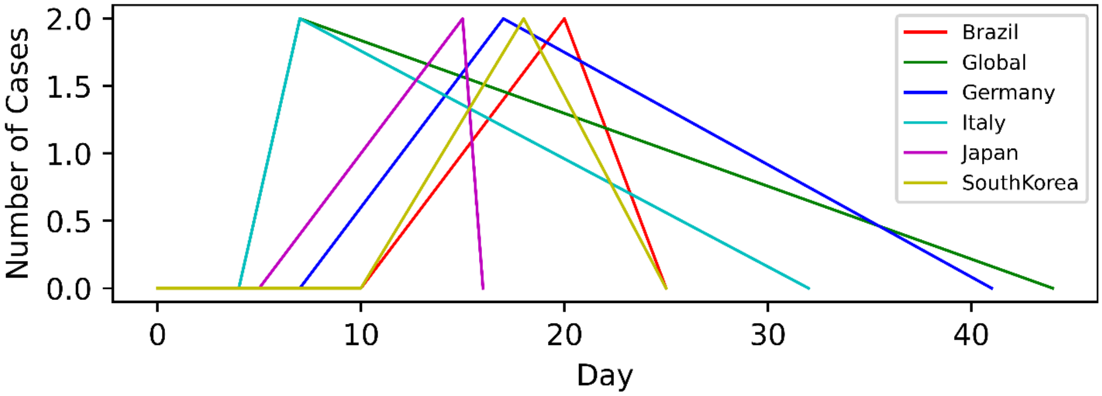


Figure 4.5 Triangulars of the optimization for the long period..

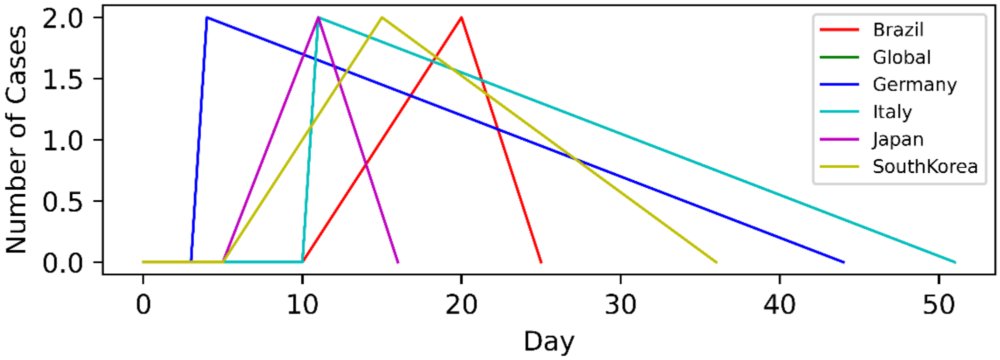


Figure 4.6 Triangulars of the optimization fort he short period

4.3. Spectral Entropy Analysis

Spectral Entropy analysis is recently used to study the complex dynamics of data set. In this section, we investigate the time series vs. optimized data set by using spectral entropy analysis to give a final remark on our method. In this section, we first give related definition 1

Definition 1

[See [17]] We take a time series of N data points

$$\gamma(m) = \{\gamma(0), \dots, \gamma(M-1)\}$$

and then remove the mean of data samples where

$$\bar{\gamma} = \frac{1}{M} \sum_{m=0}^{M-1} \gamma(m) \tag{5.1}$$

to free the information form data points as follow:

$$\gamma(m) = \gamma(m) - \bar{\gamma} \tag{5.2}$$

where $k = 0,1, \dots, N-1$.

For entropy evaluation, the relative power spectral density of κ(n) is calculated as

$$S(k) = \frac{|\beta(k)|^2}{\sum_{m=0}^{\frac{M}{2}-1} |\beta(k)|^2} \tag{5.3}$$

From discrete Fourier transform of γ(n)

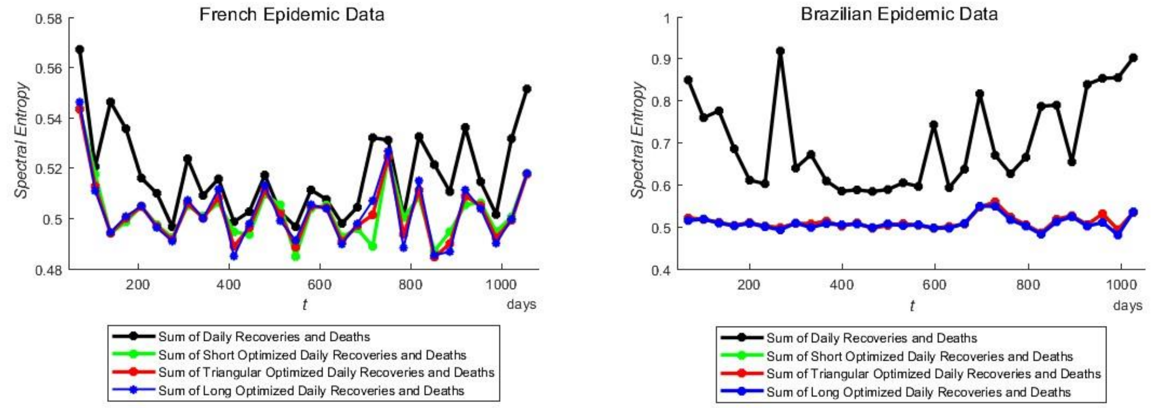
$$\beta(k) = \sum_{m=0}^{\frac{M}{2}-1} \gamma(n) e^{-j\frac{2\pi mk}{M}} \tag{5.4}$$

Where $\sum_{m=0}^{\frac{M}{2}-1} S(k) = 1$. The spectral entropy of the data points is obtained by normalizing the Shannon entropy by using the following equation

$$SE(k) = -\frac{1}{\ln(\frac{M}{2})}\sum_{m=0}^{\frac{M}{2}-1} S(k)\ln(S(k)) \tag{5.5}$$

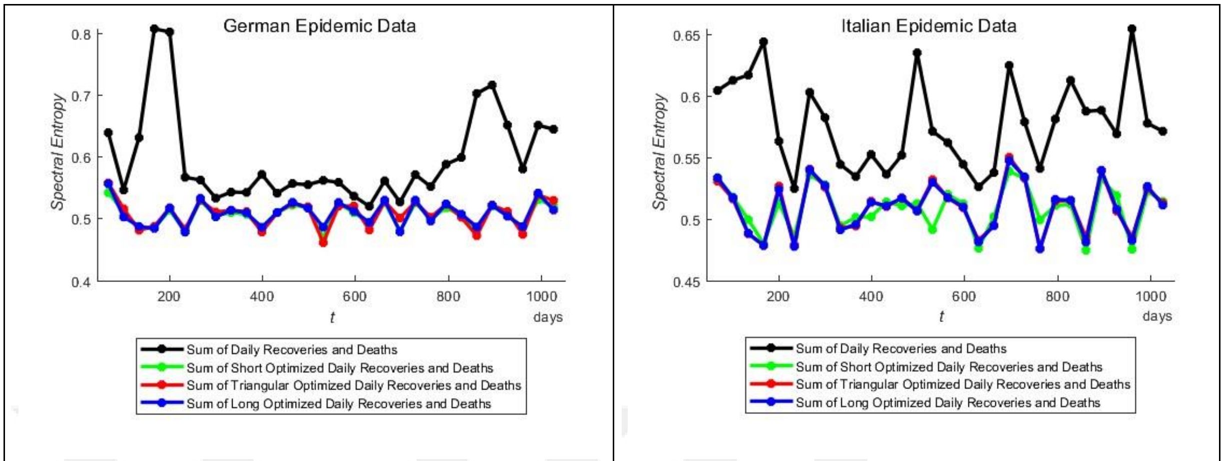
5. Results of Spectral Entropy

Since the spectral entropy is based on normalized form of Shannon entropy, we can interpret the complexity regimes in SEIR data of the countries by using the subplots in figure 8. We observe that long and short term data sets coincide and behave similar to triangular optimized daily recoveries and deaths in figure 8 (a) and (d). On the other hand Sum of daily recoveries and deaths is above them and obtains higher complexity when compared. In figure 8 (b), all sets are similar from the beginning to the end of the time period. In figures 8 (c), (e), and (f), behavior of the spectral entropies for each data set are very close in the given time interval. We also observe the similar behavior for each countries in figures 5, 6, and 7.



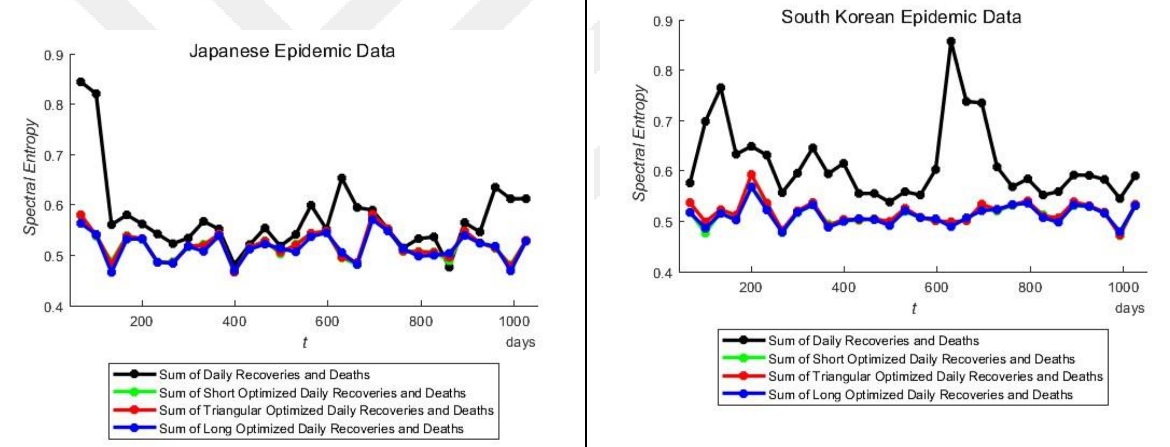
(a) Global Data

(b) Brazil



(c) Germany

(d) Italy



(f) South Korea

(e) Japan

Figure 5.1 Results of Spectral Etropy Analysis

6. DİSCUSSION

The global data in this context is highly controversial due to the fact that the global data is aggregate of divergent data sets of countries. This study ignores the distances and international effects of the disease and focuses on the applicability of the methodology. The conclusions that can be derived from the results of the optimizations may be misleading.

The results still dictates a remarkable distinction among the triangular probabilistic distributions of different countries. Which supports our claims that suggest different societies shall have different characteristics which should be represented by the models which will be used by forecasting studies. Furthermore, the difference between short-term and long-term distributions of the same countries suggests that the characteristics of the disease change over time, maybe due to the measurements taken by the governments or mutations of the virus.

The application of the probabilistic distribution into the differential system is only significant for a short period of time of the simulations. Because, the probabilistic distribution as multiple delay of the Delay Differential equations approximates to a single delay as the time approximates to infinity. Despite the approximation, the precision of the system at the beginning of the time span is crucial as the system is chaotic and sensitive to the initial data.

The genetic algorithm is used by defining the problem as set-covering problem to make the whole process as concise as possible and easily applicable to the system while maintaining the boundary conditions and stability of the system. Also as the optimization of probabilistic distribution is known to have only one optimization state and the boundaries of the solution span is narrowed down according to the field researches, the sensitivity of the optimization to initial condition is low and due to the fitness value of close relatives of the generations are very close, the results are almost identical as much as the triangles have closer shapes.

7. CONCLUSION

In this study, we proposed a new approach using a probabilistic distribution to be used in epidemic simulations. Recovery period for each individual is assumed to be distributed according to a triangular probabilistic distribution. Non-Binary Genetic Algorithm is used to find the optimized solution for the shape of the triangular distribution. The results of the triangular distribution represents the recovery phenomenon more accurately than ordinary transition functions. To support the new approach, we also used the spectral entropy analysis for each data set and obtained the consistent results.

The SEIR model with probabilistic distribution implemented is solved by using DDE23 algorithm. The results show that probabilistic distributions can be applied to the delay differential equations to model the epidemic data and main characteristcs of the model can be derived directly from the data sets. This allows the optimization of the whole epidemic model focus on other characteristics of the model such as control coefficient, σ. This improves the efficiency of the future studies focusing on the impact of policies, health care systems or even genetic diversities over epidemics.

In future works aiming to demonstrate cultural divergence and randomness of recovery rates, we propose using triangular distributions as a representation. Additionally, we investigated the applicability of the triangular probabilistic distribution to model multiple delays on a DDE, effectively reducing computation costs. By deriving the probabilistic distribution from field research, the number of coefficients required in the model can be reduced, eliminating the need for heuristic methods or optimization techniques. Researchers can build their models based on time delays obtained from clinic research, allowing them to focus on exploring the effects of measurements represented by σ in our model, defined as a function F(t,I,D) dependent on the system's state and time. In this context, our model displays the desired behavior even without a time-dependent, variable σ, making it more natural than the ordinary SIR model.

REFERENCES

[1] Kok Yew Ng, M. M. Gui, COVID-19: Development of a Robust Mathematical Model and Simulation Package with Consideration for Ageing Population and Time Delay for Control Action and Re-susceptibility, *Physica D: Nonlinear Phenomena*. 411 (2020) 132599.

[2] A. R. Fathalla, M. Naim Anwar, Qualitative Analysis of Delayed SIR Epidemic Model with a Saturated Incidence Rate, *International Journal of Differential Equations* (2012) Article ID 408637.

[3] M. Bergonzi, E. Kofman , E. Pecker-Marcosig, R. Castro, Discrete-Time Modeling of COVID-19 Propagation in Argentina with Explicit Delays, *Computing in Science & Engineering.* 23 (2021) 35-45.

[4] W. Ma, M. Song, Y. Takeuchi, Global Stability of an SIR Epidemic Model with Time Delay, *Applied Mathematics Letters*, 17 (2004) 1141-1145.

[5] X. Tian and Rui Xu, Stability of a Delayed SIR Epidemic Model with Stage Structure and Nonlinear Incidence, *Discrete Dynamics in Nature and Society*, 979217 (2009).

[6] N. T. J. Bailey, The Mathematical Theory of Epidemics, London: Charles Griffin & Co., Ltd., 194 (1957).

[7] A. E. Haimar, J.R. Santos, A Stochastic Recovery Model of Influenza Pandemic Effects on Interdependent Workforce Systems, *Nat Hazards*, 77 (2015) 987–1011.

[8] Paul, S., Lorin, E. Distribution of Incubation Periods of COVID-19 in the Canadian context. *Sci Rep*, 11 (2021) 12569.

[9] S. Annas, M. I. Pratama, M. Rifandi, W. Sanusi, S. Side, Stability Analysis and Numerical Simulation of SEIR Model for Pandemic COVID-19 Spread in Indonesia, *Chaos, Solitons & Fractals*, 139 (2020) 110072.

[10] J. Wang , J.Z. Zhu Zhang, Z. Jin, Analysis of an SIR Model with Bilinear Incidence Rate, *Nonlinear Analysis: Real World Applications*, 11(4) (2010) 2390-2402.

[11] J.L. Aron, I.B. Schwartz, Seasonality and Period-Doubling Bifurcations in an Epidemic model, *Journal of Theorertical Biology*, 110(4) (1984) 665-679.

[12] M.Y. Li, J.S. Muldowney, Global Stability for the SEIR model in Epidemiology, *Math Biosci*., 125(2) (1995) 155-164.

[13] L. Yang, G, Contagion in Stochastic Models for Epidemics, *the Annals of Mathematical Statistics*, 39(6) (1968) 1863–1889.

[14] W. Edward Back, Walter W. Boles, and Gary T. Fry, Defining Triangular Probability Distributions from Historical Cost Data, *J. Constr. Eng. Manage*. 126 (2000) 29-37.

[15] S. Y. Sahai, S. Gurukar, W. R. KhudaBukhsh, S. Parthasarathy, G. A. Rempala, A machine learning model for nowcasting epidemic incidence, *Mathematical Biosciences*, 343 (2022) 108677.

[16] A.V. Eremeev, A Genetic Algorithm with a Non-Binary Representation for the Set Covering Problem, *Operations Research Proceedings* (1998).

[17] Shannon, C.E., Weaver, W., The Mathematical Theory of Communication, *Univ of Illinois Press*. ISBN 0-252-72548-4, 1949.

[18] MATLAB. (2022b). Natick, Massachusetts: The MathWorks Inc

[19] Liu, Bette, Jayasundara, Duleepa, Pye, Victoria, Dobbins, Timothy, Dore, Gregory, Matthews, Gail, Kaldor, John, Spokes, Paula. (2021). Whole of Population-Based Cohort Study of Recovery Time from COVID-19 in New South Wales Australia. *The Lancet Regional Health - Western Pacific*. 12. 100193. 10.1016/j.lanwpc.2021.100193.

[20] Abrahim S.A., Tessema M., Defar A., Hussen A., Ejeta E., Demoz G, et al. (2020) Time to Recovery and its Predictors Among Adults Hospitalized with COVID-19: A Prospective Cohort Study in Ethiopia. PLoS ONE 15(12): e0244269. https://doi.org/10.1371/journal.pone.0244269

[21] Brauer F. Mathematical Epidemiology: Past, Present, and Future. Infect Dis Model. 2017 Feb 4;2(2):113-127. doi: 10.1016/j.idm.2017.02.001. PMID: 29928732; PMCID: PMC6001967.

[22] Hamer W.H. (1906) Epidemic Disease in England - The Evidence of Variability and Of Persistence, *The Lancet*. 1906;167:733–738.

[23] Kermack W.O., McKendrick A.G. A Contribution to the Mathematical Theory of Epidemics, *Proceedings of the Royal Society of London A: Mathematical, Physical and Engineering Sciences*. 1927;115:700–721.

[24] Paul, S., Lorin, E. Estimation of COVID-19 Recovery and Decease Periods in Canada Using Delay Model. *Sci Rep* **11**, 23763 (2021). https://doi.org/10.1038/s41598-021-02982-w

[25] M. Senturk, “Implementation of triangular probabilistic distribution onto delay differential equations on the basis of seir model,” https://github.com/kzlsahin/TriangularProbabilisticDistributionWithSEIR (2023).

CURRICULUM VITAE

Mustafa Şentürk was graduated from Naval architecture and Marine Engineering program At Istanbul Technical University. He began his career as a naval architect and design engineer. Having seen the problems facing the industry, he joined the civil service as a maritime specialist to help improve the shipbuilding and maritime industry and to gain a broader perspective of the industry. During his time in the Civil Service, he was overwhelmed by the problems that could be solved through the design and development of computational solutions, digital transformation and the advancement of the rules-as-code concept. He was also interested in the philosophy of science and decision disciplines, and studied machine learning and computational engineering. He started MSc program in Computational Science and Engineering at Piri Reis University in 2020. He is now working as a software developer and R&D engineer.