



SCHOOL of
GRADUATE STUDIES
EAST TENNESSEE STATE UNIVERSITY

East Tennessee State University
**Digital Commons @ East
Tennessee State University**

Electronic Theses and Dissertations

12-2010

The SIR Model When $S(t)$ is a Multi-Exponential Function.

Teshome Mogessie Balkew
East Tennessee State University

Follow this and additional works at: <http://dc.etsu.edu/etd>

Recommended Citation

Balkew, Teshome Mogessie, "The SIR Model When $S(t)$ is a Multi-Exponential Function." (2010). *Electronic Theses and Dissertations*. Paper 1747. <http://dc.etsu.edu/etd/1747>

This Thesis - Open Access is brought to you for free and open access by Digital Commons @ East Tennessee State University. It has been accepted for inclusion in Electronic Theses and Dissertations by an authorized administrator of Digital Commons @ East Tennessee State University. For more information, please contact dcadmin@etsu.edu.

The SIR Model When $S(t)$ is a Multi-Exponential Function

A thesis presented to
the faculty of the Department of Mathematics
East Tennessee State University

In partial fulfillment
of the requirements for the degree
Master of Science in Mathematical Sciences

by

Teshome Mogessie Balkew

December 2010

Jeff Knisley, Ph.D., Chair

Ariel Cintron-Arias, Ph.D.

Robert Gardner, Ph.D.

Fredrick Norwood, Ph.D.

Keywords: SIR model, Relative Removal Rate, Phase Plane Analysis, R_0 .

ABSTRACT

The SIR Model When $S(t)$ is a Multi-Exponential Function

by

Teshome Mogessie Balkew

The SIR can be expressed either as a system of nonlinear ordinary differential equations or as a nonlinear Volterra integral equation. In general, neither of these can be solved in closed form. In this thesis, it is shown that if we assume $S(t)$ is a finite multi-exponential, i.e. function of the form $S(t) = a + \sum_{k=1}^n r_k e^{-\sigma_k t}$ or a logistic function which is an infinite-multi-exponential, i.e. function of the form $S(t) = c + \frac{a}{b + e^{wt}}$, then we can have closed form solution. Also we will formulate a method to determine R_0 the basic reproductive rate of an infection.

Copyright by Teshome Mogessie Balkew 2010

ACKNOWLEDGMENTS

First and foremost I would like to extend my deepest gratitude to the gracious, merciful Heavenly Father Almighty God, Who helped me in every way in my stay here in ETSU. My special thanks goes to Dr. Robert B. Gardner. I feel that words may not be enough to express how deeply grateful I am for all help he rendered to me. Thank you Dr. Bob!! I also acknowledge the support and encouragement accorded to me by Dr. Fredrick Norwood, specially in the first two or so months of my stay here. My great appreciation and thanks go to my thesis advisor Dr. Jeff Knisley. He has been instrumental at all stages of my thesis development. He gave me all necessary advising for my thesis to go in the right direction. From his excellent coaching I learned a lot. I like to say "thank you" to all my teachers: Dr. Debra Knisley, Dr. Robert M. Price and Dr. Ariel Cintron-Arias who inspired me one or the other way in my stay in ETSU. I acknowledge the help I got from Dr. Yared Nigussie on several occasions. I am so grateful to my friend Azamed Gezahagne and his wife Lidya Hailesilassie. Finally I would like to say "thank you" to all my friends and classmates here in ETSU who influenced me one or the other way. Thank you and May God bless you all.

CONTENTS

ABSTRACT	2
ACKNOWLEDGMENTS	4
LIST OF FIGURES	7
1 INTRODUCTION	8
2 SIR EPIDEMIOLOGICAL MODEL	11
2.1 Two Formulations of the SIR Model	12
2.1.1 The Classical Differential Equation Form of the SIR Model	12
2.1.2 The Integral Equation Form of the SIR Model	13
2.1.3 Equivalence of the Differential and Integral Form of the SIR Model	14
2.2 Phase Plane Analysis	15
2.2.1 Phase Plane in SI Plane	15
2.2.2 Phase Plane Analysis in RS Plane	17
2.2.3 The Basic Reproductive Rate of Infection R_0	19
3 THE SIR MODEL WHEN $S(t)$ IS A MULTI-EXPONENTIAL FUNC- TION	25
3.1 Finite Multi-Exponential Form of $S(t)$	26
3.1.1 Susceptibles as a Single Exponential	28
3.1.2 Susceptibles as a sum of two Exponentials	30
3.2 Susceptible as a Logistic Function	32
3.2.1 The Basic Reproductive Rate of Infection R_0	35

4 CONCLUSION 38
BIBLIOGRAPHY 40
APPENDICES 42
VITA 46

LIST OF FIGURES

1	One Phase Curve in SI Plane	16
2	Phase Curves in SI Plane	18
3	Phase Curves in RS Plane	19
4	Phase Curves in SI Plane	24
5	Phase Curves in RS Plane	24
6	Data Fit Curve for “Susceptible” for One Exponential	29
7	SIR Curves for One Exponential $S(t)$	30
8	Data Fit Curve for the “Susceptible” for Two Exponential	31
9	SIR Curves for Two Exponential $S(t)$	32
10	Best Fit Curve for Logistic Function	34
11	SIR Model Curves for Logistic Function	35
12	Data Fit Curve for “Recovery” Data	36
13	The Graph of β as a Function of Time	37

1 INTRODUCTION

From pre-history to the present day, there have been many waves of epidemics causing death and suffering of human beings. Most of these epidemics seemingly came from nowhere, suddenly changed the normal demography of the affected population, and ultimately disappeared before affecting the entirety of a population. This was a puzzle for human beings for centuries, and there were many attempts to understand these frequent epidemiological occurrences and to create a mechanism to control them [2, 18].

- The first known result in mathematical epidemiology is due to Daniel Bernoulli. In 1760 he formulated and solved a model for a smallpox epidemic [18].
- In 1906 William Hamer formulated a discrete time model for measles epidemics [18].
- A significant contributor to compartmental-based epidemiological models was the public physician R.A. Ross. In 1911 he proposed a differential equation model for malaria as a host-reactor disease and won the second Nobel Prize in Medicine [18].
- The classical *SIR* model as we know it today was first introduced by A.G. Mckendrick and W.O. Kermack in 1927. These two public health physicians extended the work of Ross and developed the concept of an epidemic threshold [18].

Most epidemiological models start from the same basic premise that the population can be subdivided into a set of distinct classes (compartments) depending upon their experience with respect to the disease [18]. One line of investigation is to divide the population under study into the three compartments of [18] “Susceptibles”, “Infecteds” and “Recoveredds.” The number of individuals in each compartment is denoted by $S(t)$, $I(t)$ and $R(t)$ respectively, where

- $S(t)$ denotes the number of individuals who are “Susceptible” to the disease at time t . That is, $S(t)$ is the number of people in the population who are not “infected” at the given time t but who are vulnerable to the disease.
- $I(t)$ signifies the number of “infected” individuals who are infectious and able to spread the disease to a susceptible.
- $R(t)$ counts those in the population who are “recovered” from the disease and are no longer “susceptible”. Thus they do not contribute to the spread of the disease.

Such a model is called an *SIR* model.

A simple mathematical model of a natural phenomenon is meant to highlight the existing qualitative behaviors of the phenomena, so that we omit most details. As natural phenomena are influenced by a variety of internal and external factors, taking all aspects of it and try to model as it is makes the model too complicated, bulky, and is generally unwise, as it is common to end up with equation(s) which are difficult or impossible to solve. So it is common to take certain assumptions in modeling.

The *SIR* model is formulated under the following assumptions [2, 4, 18]:

- (I) The community size is constant and is sufficiently large over the duration of the epidemic. We denote the number of population by N , where

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

- (II) The susceptibles become infected at a percentage rate proportional to the number of “infecteds”.
- (III) It is also assumed that the “infecteds” are uniformly distributed among the “susceptibles”.

Remark

- Assumption (I) says that since the demography of a population does not change significantly within a short period of time we ignore birth and immigration.
- We clearly know that the rate of leaving compartment at the beginning, middle and end of the epidemic varies greatly. What assumption (II) says is that we take the average (mean) number leaving compartments as a constant. This assumption is reasonable if the population size is large, but it is not good if the size of the population is small, since the random contact rate plays a significant role for spreading the disease in small size population [1].
- In assumption (III) we ignored such factors as age, sex, social background, seasons, geographical location, etcetera, which may affect the epidemic significantly.

2 SIR EPIDEMIOLOGICAL MODEL

In this chapter we will study two equivalent ways of formulating the SIR model – the ordinary differential equation form and the integral equation form. The integral equation form of the SIR model is more general than the ordinary differential equation form for the following two reasons [1]:

1. We can deduce the classical ordinary differential equation form of the SIR model by assuming $P(t)$ is exponentially distributed.
2. Different functional forms of the probability density function $P(t)$ gives us alternative types of the SIR model.

Even though the more realistic probability density function is the gamma distribution, which presupposes the probability of leaving the class is a function of the time spent within the class, the classical SIR model is based on the premise that the probability of infection is exponentially distributed, which implies that the rate of transfer from one compartment to the other is independent of the time spent within the class [1]. With this consideration, we recover the classical differential equation form of the SIR model from the integral form. We will explore the relation between two variables with the absence of the third one. What we do here is examine the relation between “S” and “I” in the SI coordinate plane and the relation between “R” and “S” in the RS coordinate plane. Such a study of the relation between two variables with the absence of the third one is called Phase Plane Analysis for the model [18].

2.1 Two Formulations of the SIR Model

We will see two formulations of the SIR model. First we consider the differential equation formulation and then we will see the integral equation formulation of the SIR model.

2.1.1 The Classical Differential Equation Form of the SIR Model

In addition to the assumptions we made in Chapter 1, the SIR model also assumes the following (see [2, 3, 4, 18] for additional discussion of these assumptions):

- $f(I) = \beta I$, where $f(I)$ is an increasing function of “I” called the force of infection and β is a constant called the infection rate. The infection rate tells us the rate at which the “susceptibles” transfer into the “infecteds” group.
- Alternatively, $f(I) = \beta (I/N)$, where I/N is the “infecteds” density in a population of size N .

The relation between the compartments is assumed to be as follows:

- As the epidemic spreads those in “susceptibles” compartment transfer to “infecteds” compartment at a rate of β , i.e. $S' = -f(I)S$.
- The “infecteds” compartment receives the outflow from the first, and it also loses “infecteds” to the “recovereds” one at a rate of β , i.e. $I' = f(I)S - \alpha I$
 - The parameter α is called the removal rate of infection. It is the rate by which the “infected” transfer into the “recovery”. The probability of

remaining “infectious” through time t is given by $P(t)$. $1/\alpha = \int_0^t P(t)dt$ gives us the average infectious period.

- The third one implies $R' = \alpha I$.

From the above premises we have

$$\begin{aligned} S' + I' + R' &= -\frac{\beta}{N}SI + \frac{\beta}{N}SI - \alpha I + \alpha I \\ S' + I' + R' &= 0 \end{aligned}$$

which implies that $S(t) + I(t) + R(t)$ is constant. In fact, from our assumptions we know that at any given time “ t ”, $S(t) + I(t) + R(t) = N$. Then the classical differential equation form of the SIR model can be summarized as

$$\begin{aligned} \frac{dI}{dt} &= \frac{\beta}{N}S(t)I(t) - \alpha I(t) \\ \frac{dS}{dt} &= -\frac{\beta}{N}S(t)I(t) \\ \frac{dR}{dt} &= \alpha I(t) \end{aligned} \tag{1}$$

2.1.2 The Integral Equation Form of the SIR Model

In this section we explore the Integral equation form of the SIR model (1). To begin with, we derive the classic SIR model using an alternative approach. Specifically, we begin with a modeling paradigm of the form

$$\begin{array}{l} \text{The number of “Infecteds”} \\ \text{at time } t \end{array} = \begin{array}{l} \text{Initial number of “Infecteds”} \\ \text{still “Infecteds” at } t \end{array} + \begin{array}{l} \text{Those infected in } [0, t] \\ \text{who remain infectious} \end{array}$$

If we take $P(t)$ to be the probability that an “infected” at time $t_0 = 0$ remains

infectious at time t , then the idea above is given mathematically by

$$I(t) = I(0)P(t) + \int_0^t \beta S(\tau) \frac{I(\tau)}{N} P(t - \tau) d\tau \quad (2)$$

Equation (2) is a *non-linear Volterra Integral equation* [7].

2.1.3 Equivalence of the Differential and Integral Form of the SIR Model

We said that selecting different forms of $P(t)$ gives us alternative types of the SIR model. Let us revisit the result in (1) by showing that if we assume $P(t)$ as exponentially distributed that is, $P(t) = e^{-\alpha t}$, then we recover the classical exponentially distributed *SIR* model. Substituting $P(t) = e^{-\alpha t}$ into (2) we have

$$I(t) = I(0)e^{-\alpha t} + \frac{\beta}{N} \int_0^t S(\tau) I(\tau) e^{-\alpha(t-\tau)} d\tau \quad (3)$$

Differentiating (3) with respect to t , we have

$$\frac{dI}{dt} = \frac{\beta}{N} SI - \alpha \left[I(0)e^{-\alpha t} + \frac{\beta}{N} \int_0^t S(\tau) I(\tau) e^{-\alpha(t-\tau)} d\tau \right]$$

which implies that

$$\frac{dI}{dt} = \frac{\beta}{N} SI - \alpha I.$$

The probability of being recovered at time t is given by $Q(t) = 1 - P(t)$. Differentiating it we have

$$Q'(t) = \alpha P(t)$$

which implies

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

Finally, since $S + I + R$ is constant, we know that $S' + I' + R' = 0$, which in turn implies that $S' = -I' - R'$. This results in

$$\frac{dS}{dt} = -\frac{\beta}{N}SI$$

which completes the equivalence of the two forms of the SIR model formulation.

2.2 Phase Plane Analysis

In this section, we will study the relationship between our variables, S and I in the SI plane, and S and R in the RS plane. This is called phase plane analysis. Moreover, we will also study one of the very important elements of epidemiology called the reproductive rate of infection, commonly denoted by R_0 .

2.2.1 Phase Plane in SI Plane

The first two equations in (1) involves only $S(t)$ and $I(t)$. We examine them to study what the solution looks like in the SI plane. Right at the beginning of the epidemic; there is no “recovery”, i.e. $R(0) = 0$, and let the number of “susceptibles” be given by S_0 and the number of “infecteds” be given by I_0 . Also let S_∞ be the number of people who will not be “infected” throughout the epidemic. Thus $S_0 + I_0 = N$.

Dividing the first equation by the second in (1) we have

$$\begin{aligned} \frac{dI}{dS} &= \frac{\beta SI - \alpha I}{-\beta SI} \\ &= \frac{\alpha}{\beta S} - 1 \\ \frac{dI}{dS} &= \frac{\rho}{S} - 1 \end{aligned} \tag{4}$$

where $\rho = \frac{\alpha}{\beta}$ is the relative removal rate. Equation (4) is called the Phase portrait for the epidemic, and solutions to the phase portrait are called Phase curves [18] and can be obtained as follows

$$\begin{aligned}
 I &= \int \left(\frac{\rho}{S} - 1 \right) dS \\
 &= \rho \ln(S) - S + C_1 \\
 I &= \rho \ln(S) - S + N - \rho \ln(S_0)
 \end{aligned} \tag{5}$$

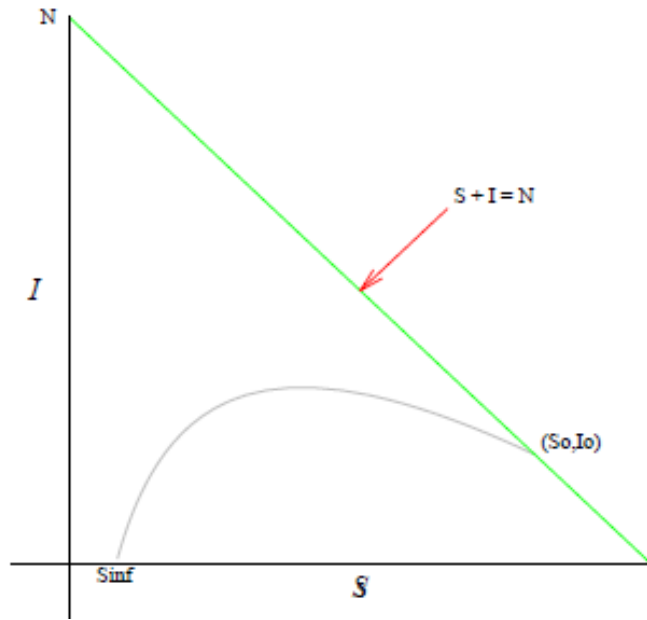


Figure 1: One Phase Curve in SI Plane

From Fig (1), we observe that

- the curve starts at (S_0, I_0) , rises to a maximum, and proceeds to S_∞ . That means the epidemic stops because of the lack of “infecteds”, not because everyone will be sick.

- the epidemic obtains a maximum somewhere between S_0 and S_∞ .

We can determine the maximum point as follows

$$\frac{dI}{dS} = \frac{\rho}{S} - 1$$

Then, the critical value will be the solution of the equation

$$\frac{dI}{dS} = 0$$

which implies

$$\rho = S$$

To show that we have maximum value at ρ , we use the second derivative test. The second derivative of I with respect to S is

$$\frac{d^2I}{dS^2} = -\frac{\rho}{S^2} < 0$$

which is negative. Hence, a maximum occurs at $S = \rho$ as shown in Fig (2).

The maximum value is

$$I_{max} = \rho \ln \left(\frac{\rho}{S_0} \right) - \rho + N \quad (6)$$

2.2.2 Phase Plane Analysis in RS Plane

If we divide the second equation by the third one in (1), then we have

$$\frac{dS}{dR} = -\frac{\beta SI}{\alpha I} = -\rho S$$

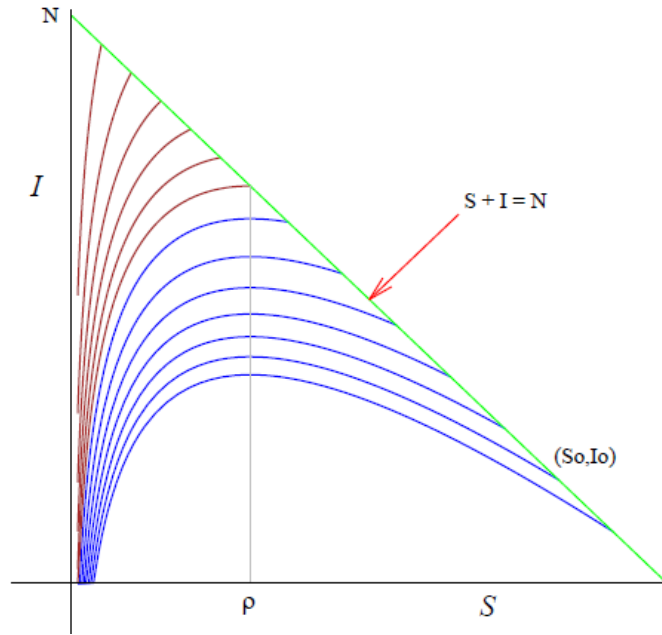


Figure 2: Phase Curves in SI Plane

which leads to

$$S(R) = S_0 e^{-\frac{R}{\rho}} \tag{7}$$

Equation (7) implies that the RS phase curve exponentially decays, which means that the number of “susceptibles” decays exponentially as a function of the number of “recovereds” as shown in Fig (3) . To show mathematically our claim before that in any epidemic there is always a part of the population which will not be “infected” throughout the epidemic we use equation (7) as follows

$$S(R) = S_0 e^{-\frac{R}{\rho}} \leq S_0 e^{-\frac{N}{\rho}} > 0$$

which give us

$$0 < S_\infty \leq N$$

We know that if $t \rightarrow \infty$, then $R \rightarrow R_\infty$ and $I_\infty = 0$ implies $R_\infty = N - S_\infty$. From

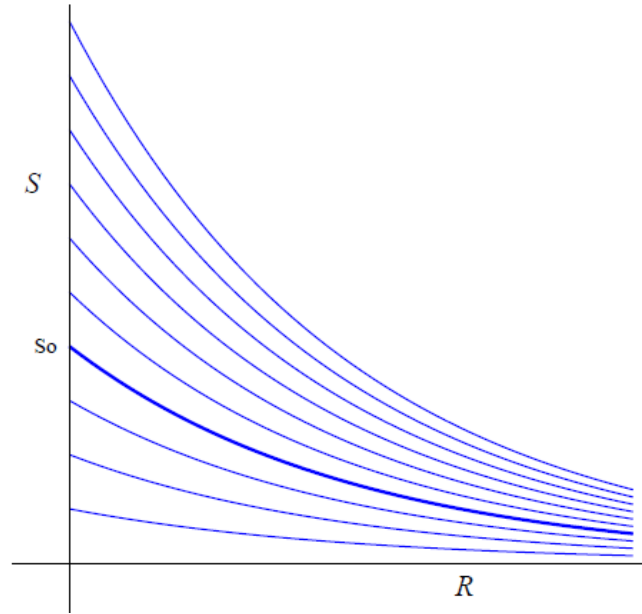


Figure 3: Phase Curves in RS Plane

this it follows that

$$\begin{aligned} S_\infty &= S_0 e^{-\frac{R_\infty}{\rho}} \\ S_\infty &= S_0 e^{-\frac{N-S_\infty}{\rho}} \end{aligned} \tag{8}$$

We can use this relation to calculate the value of S_∞ and/or ρ if we know either of the two.

2.2.3 The Basic Reproductive Rate of Infection R_0

The basic reproductive rate, commonly denoted by R_0 , is also called the basic reproductive number or the basic reproductive ratio [2, 18]. It is basically the expected (average) number of secondary cases produced by a typical primary case in an entirely

“susceptible” population. The reproductive rate of infection R_0 is mathematically expressed as

$$R_0 = \frac{S_0}{\rho}$$

which results in

$$R_0 = \frac{\beta S_0}{\alpha}$$

The reproductive rate R_0 is important for prediction of whether there will be epidemic or not [2, 18].

Theorem 2.1 (*Threshold theorem of Epidemiology*)

If $S_0 \leq \frac{\alpha}{\beta}$, then $I(t)$ is monotonically decreasing. If $S_0 > \frac{\alpha}{\beta}$, then an epidemic occurs in that the number of “infected” will increase to a maximum before dropping to 0 over time.

The threshold theorem can be interpreted in the following way

- if $S_0 \leq \rho$ which gives us $R_0 \leq 1$, then there is no epidemic.
- if $S_0 > \rho$ which implies $R_0 > 1$, then there will be epidemic.

Thus, stopping an epidemic is usually related to reducing R_0 to be below 1.

Consider equation (5) with $I_\infty = 0$ and $S(\infty) = S_\infty$. We have

$$I = \rho \ln(S) - S + N - \rho \ln(S_0) \tag{9}$$

$$I + S - \rho \ln(S) = N - \rho \ln(S_0) \tag{10}$$

If we take the *limit* as t goes to ∞ , then we have

$$S_\infty - \rho \ln(S_\infty) = N - \rho \ln(S_0)$$

Which results in

$$\rho = \frac{N - S_\infty}{\ln\left(\frac{S_0}{S_\infty}\right)}$$

Alternatively, suppose that the only type of data available is from hospital reports of relevant admissions and releases. This type of data commonly reports the number of recovered. We can also determine the average rate of “recovery” from the report. So it is reasonable to find R_0 using the hospital data.

Using $S(R) = S_0 e^{-\frac{R}{\rho}}$, $\frac{dR}{dt} = \alpha I$ and $I + S + R = N$;

$$\begin{aligned} \frac{dR}{dt} &= \alpha(N - R - S) \\ &= \alpha(N - R - S_0 e^{-\frac{R}{\rho}}) \end{aligned}$$

from which we obtain

$$\begin{aligned} R' - \alpha N + \alpha R &= -\alpha S_0 e^{-\frac{R}{\rho}} \\ e^{-\frac{R}{\rho}} &= \frac{\alpha N - \alpha R - R'}{\alpha S_0} \end{aligned}$$

Taking the logarithm on both sides and simplifying, we have

$$\rho = \frac{-R}{\ln\left(\frac{\alpha N - \alpha R - R'}{\alpha S_0}\right)} \quad (11)$$

If we take the *limit* as t goes to infinity, then (10) will be equal to (9) since R' approaches 0.

Let us consider the following example to illustrate the validity of our discussion. We are going to use this example several times in the future discussion too [18].

EXAMPLE

A certain flu outbreak in English boarding school lasted for 14 days. The school has a total of 763 students, of which 512 contracted the flu during that period. One boy is known to have been the initial infected person thus giving us a given data set for $I(t)$.

We can calculate the value of $S(t)$ numerically using the data. Thus, we have the following values:

Table 1: The Number Of “Infected” And “Susceptible” Students

Days	Infected	Susceptible
0	1	762
1	3	758.99
2	7	749.55
3	25	721.20
4	72	645.4
5	222	493.48
6	282	306.89
7	256	171.12
8	233	99.21
9	189	64.18
10	123	46.51
11	70	36.99
12	25	31.54
13	11	28.27
14	4	26.23

Taking the following values for β , α and ρ as

$$\beta = 0.0021, \alpha = 0.441, \quad \text{and} \quad \rho = \frac{\alpha}{\beta} = 210$$

we obtain a reproductive rate of infection

$$R_0 = \frac{S_0}{\rho} = 3.63 > 1,$$

matching the fact that an epidemic did occur. Using (9),

$$\rho = \frac{763 - 25}{\ln \frac{762}{25}} = 215.9745.$$

The phase curves in SI and RS are as follows:

1. The Phase curve in the SI plane is given by substituting, $N = 763$ and $S_0 = 762$ in $I = \rho \ln(S) - S + N - \rho \ln(S_0)$.

Then, we have, $I = 210 \ln(S) - S + 763 - 210 \ln(762)$ whose graphs is shown in Fig (4).

2. Phase curves in the RS plane are obtained by taking the second and the third equation in (1), and assuming $S(0) = S_0 = 762$ and $R(0) = 0$.

we have the following:

$$S(R) = S_0 e^{-R/\rho} = 762 e^{-R/210}$$

Which gives us Figure (5).

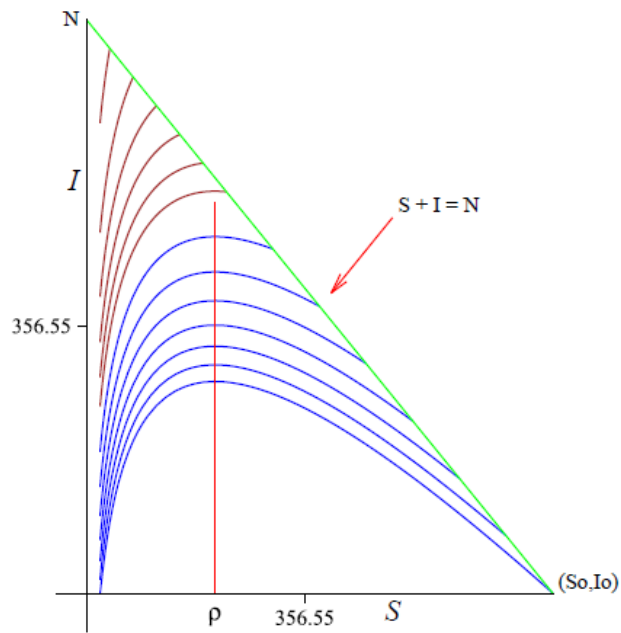


Figure 4: Phase Curves in SI Plane

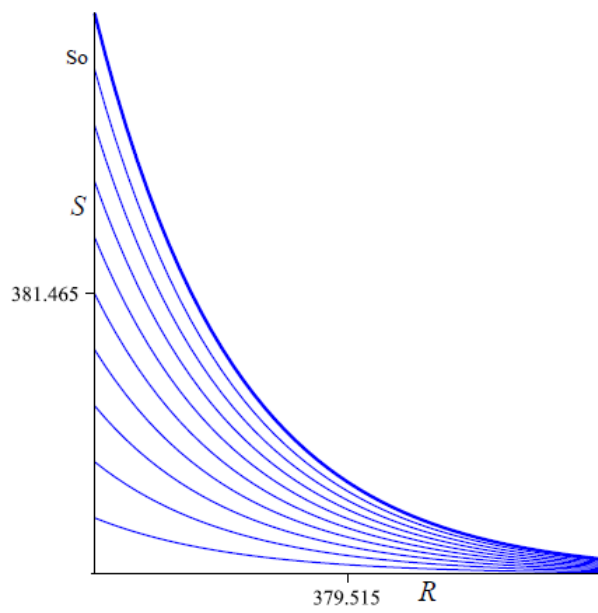


Figure 5: Phase Curves in RS Plane

3 THE SIR MODEL WHEN $S(t)$ IS A MULTI-EXPONENTIAL FUNCTION

As stated earlier, the non-linear Volterra integral equation (2) has no solution in closed form. With appropriate assumptions on $P(t)$, we have shown that the integral equation is equivalent to a system of non-linear ordinary differential equations in (1). Unfortunately, the system of non-linear ODE also has no solution in closed form. The main reason that the equations in (1) and (2) cannot be solved in closed form is because of the nonlinearity of $S(t)I(t)$.

So, in some way, if we could somehow change the product $S(t)I(t)$ to some form which makes the solution simple, then we can solve (2) and as a result we can also solve (1) in closed form. Because of the following two reasons we assume $S(t)$ is a multi-exponential function [12, 18]

- we know that the kernel of (2) is an exponential function. If we take $S(t)$ to be a multi-exponential function, then the expression under the integral will be linear. Thus we can solve the integral equation easily.
- from experience we know that the “susceptible’s” data fits better with $S(t)$ if we take $S(t)$ a multi-exponential function.

After having selected $S(t)$ we solve (2) by changing it to an ODE. This assumption of $S(t)$ will make the ODE a linear ODE, which is simple to solve.

Let us first assume $S(t) = a + \sum_{k=1}^n r_k e^{-\sigma_k t}$ and solve (2). Then, we use one real example to illustrate two cases of $S(t)$ – i.e., $S(t) = a + b e^{-\sigma_k t}$ and $S(t) = a + \sum_{k=1}^2 r_k e^{-\sigma_k t}$. Finally, we take the assumption that $S(t)$ is a function of the form $S(t) = \frac{a}{b + e^{wt}}$ and solve (2).

3.1 Finite Multi-Exponential Form of $S(t)$

Let us consider $S(t)$ to be a multi-exponential function of the form $S(t) = a + \sum_{k=1}^n r_k e^{-\sigma_k t}$. With this $S(t)$, equation (2) becomes

$$I(t) = I(0)e^{-\alpha t} + \frac{\beta}{N} \int_0^t \left(a + \sum_{k=1}^n r_k e^{-\sigma_k \tau} \right) I(\tau) e^{-(t-\tau)} d\tau$$

Thus, the non-linear Volterra integral equation is changed to a linear one. To solve it, we change it to an ODE of the form

$$\begin{aligned} \frac{dI}{dt} &= \left(\frac{\beta}{N} \left(a + \sum_{k=1}^n r_k e^{-\sigma_k t} \right) - \alpha \right) I(t) \\ \frac{dI}{I} &= \left(\frac{\beta}{N} \left(a + \sum_{k=1}^n r_k e^{-\sigma_k t} \right) - \alpha \right) dt \end{aligned}$$

Integrating both sides leads to

$$\begin{aligned} \int \frac{dI}{I} &= \int \left(\frac{\beta}{N} \left(a + \sum_{k=1}^n r_k e^{-\sigma_k t} \right) - \alpha \right) dt + C_2 \\ \ln |I(t)| &= \frac{\beta}{N} \left(at - \sum_{k=1}^n \frac{r_k}{\sigma_k} e^{-\sigma_k t} \right) - \alpha t + C_2 \end{aligned}$$

To solve for $I(t)$, we change both sides of the above expression to exponential function, and then we have the following

$$I(t) = e^{\frac{\beta}{N} \left(at - \sum_{k=1}^n \frac{r_k}{\sigma_k} e^{-\sigma_k t} \right) - \alpha t + C_2}$$

To determine the constant C_2 , we use the initial condition $I(0) = I_0$.

$$I(0) = e^{\frac{-\beta}{N} \left(\sum_{k=1}^n \frac{r_k}{\sigma_k} \right) + C_2}$$

Thus we have

$$e^{C_2} = I_0 e^{\frac{\beta}{N} \left(\sum_{k=1}^n \frac{r_k}{\sigma_k} \right)}$$

Hence we have the following for $I(t)$

$$I(t) = I_0 e^{\frac{\beta}{N} \left(at - \sum_{k=1}^n \frac{r_k}{\sigma_k} e^{-\sigma_k t} \right) - \alpha t + \frac{\beta}{N} \left(\sum_{k=1}^n \frac{r_k}{\sigma_k} \right)}$$

Our next step is to find the function $R(t)$. We know that

$$\frac{dR(t)}{dt} = \alpha I(t).$$

But $I(t) = N - R(t) - S(t)$, so that

$$R'(t) = \alpha (N - R(t) - S(t))$$

$$R'(t) + \alpha R(t) = \alpha (N - S(t))$$

Thus, it is a non-homogeneous linear ODE which can be solved first by determining an integrating factor $\mu(t)$ as

$$\mu(t) = e^{\int \alpha dt} = e^{\alpha t}$$

Using $\mu(t) = e^{\alpha t}$, $R'(t) + \alpha R(t) = \alpha (N - S(t))$ becomes

$$(e^{\alpha t} R(t))' = \alpha e^{\alpha t} \left(N - \left(a + \sum_{k=1}^n r_k e^{-\sigma_k t} \right) \right)$$

Integrating both sides of the above equation, we have

$$e^{\alpha t} R(t) = N e^{\alpha t} - a e^{\alpha t} - \alpha \sum_{k=1}^n \frac{r_k}{\alpha - \sigma_k} e^{(\alpha - \sigma_k)t} + C_3$$

Dividing both sides by $e^{\alpha t}$ we have

$$R(t) = N - a - \alpha \sum_{k=1}^n \frac{r_k}{\alpha - \sigma_k} e^{-\sigma_k t} + C_3 e^{-\alpha t}$$

Now it remains to find C_3 . Using initial condition $R(0) = R_0$, we have

$$R(0) = N - a - \alpha \sum_{k=1}^n \frac{r_k}{\alpha - \sigma_k} + C_3$$

which implies that

$$C_3 = R_0 - N + a + \alpha \sum_{k=1}^n \frac{r_k}{\alpha - \sigma_k}.$$

This completes our solution.

Let us revisit our example taking $S(t)$ with one exponential function and sum of two exponential functions.

3.1.1 Susceptibles as a Single Exponential

Let us assume $S(t)$ as a function of the form $S(t) = a + be^{-\sigma t}$. Using the Fit command in Maple software we fit the data with a curve of $S(t)$ as shown in Fig (6). That gives us the values of a , b , and σ . These values, as discussed and formulated in section 3.1, are used to determine the functions $S(t)$, $I(t)$ and $R(t)$. To have a better curve which best fits our data, we divided the data into two and fit each of them with $S(t)$ type functions. That gives us two functions for each of $S(t)$, $I(t)$ and $R(t)$. These functions approximate the number of ‘‘Susceptibles’’, ‘‘Infecteds’’ and ‘‘Recoveredds.’’ respectively.

By what we formulated in Section 3.1, we have the following functions which give

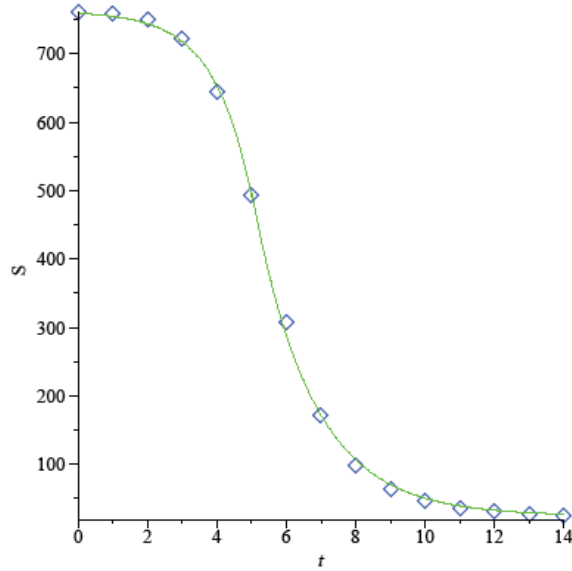


Figure 6: Data Fit Curve for “Susceptible” for One Exponential

us Fig (7)

$$S_1(t) = 762 - 2.948e^{0.904t}$$

$$S_2(t) = 25 + 8820.803e^{-0.584t}$$

$$I_1(t) = e^{0.973t+0.007e^{0.904t}+0.005}$$

$$I_2(t) = e^{-0.389t-31.704e^{-0.584t}+8.807}$$

$$R_1(t) = 1 + 0.967e^{0.904t} - 1.967e^{-0.441t}$$

$$R_2(t) = 763 + 27152.695e^{-0.584t} - 18864.797e^{-0.441t}$$

In addition, the Fit command and other statistics routines in Maple provide measures of variance that can be used to validate the fits. Because the curves are piecewise-defined, we did not measure confidence intervals for these fits. However, correlations between the actual data and the predicted curves are each in excess of

0.98, and chi-square goodness of fit tests similarly confirm our results.

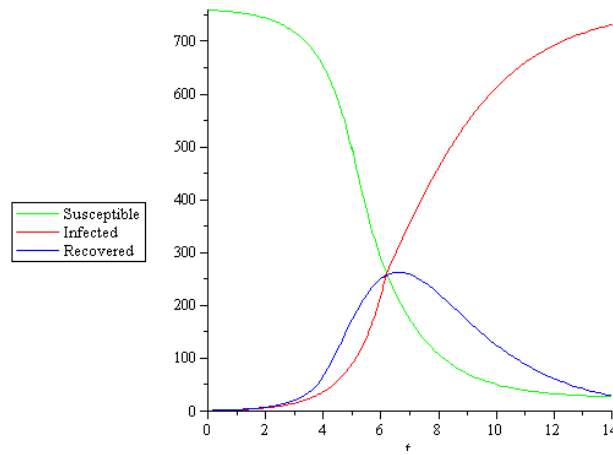


Figure 7: SIR Curves for One Exponential $S(t)$

3.1.2 Susceptibles as a sum of two Exponentials

Let us assume that $S(t)$ is a function of the form

$$S(t) = a + \sum_{k=1}^2 r_k e^{-\sigma_k t}.$$

Using the Fit command in Maple software we fit the “Susceptibles” data with curves of $S(t)$. The fit gives us the corresponding values of a , r_1 , r_2 , σ_1 , σ_2 . Using the discussion and formulation of section 3.1 we can determine functions $S(t)$, $I(t)$ and $R(t)$. To have a better curve which best fits our data we divided the data into two and fit each of them with two different $S(t)$. This gives us two functions for each of $S(t)$, $I(t)$ and $R(t)$ which approximates the number of “susceptibles”, “infecteds” and “recovereds”, respectively. As a result we have Fig (8).

The following functions are the result of what we have discussed in Section 3.1

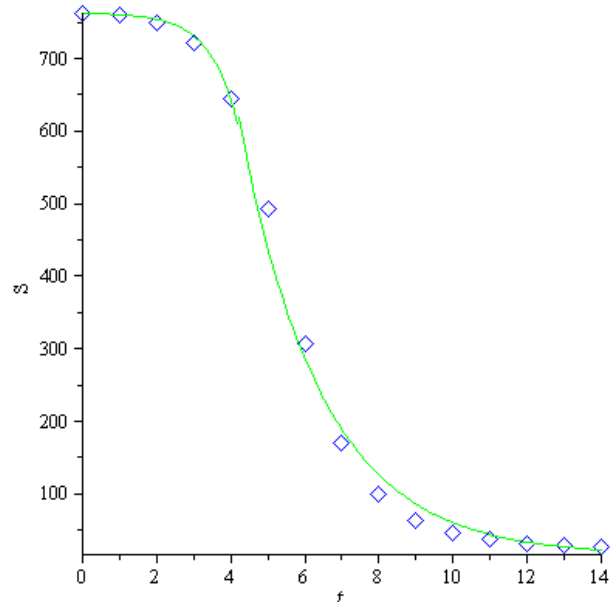


Figure 8: Data Fit Curve for the “Susceptible” for Two Exponential

which results in Fig (9).

$$S_1 = 762.5 - 0.596e^{1.323t} + 2.230 \times 10^{-7}e^{8.701t}$$

$$S_2 = 15 - 1000.752e^{-1 \times 10^5 t} + 3931.869e^{-0.446t}$$

$$I_1 = e^{0.914t + 0.005e^{1.323t} + 1.455 \times 10^{-20}e^{8.701t} - 0.454}$$

$$I_2 = e^{-0.409t + 0.0002e^{-1 \times 10^5 t} - 18.545e^{-0.446t} + 9.352}$$

$$R_1 = 674.679 - 0.0001e^{-8 \times 10^5 t} + 2.429e^{-13.370t} - 11844.678e^{-0.569t}$$

$$R_2 = 13.893 + 0.115e^{1.323t} - 2.478 \times 10^{-19}e^{7.048t} - 19.612e^{-0.445t}$$

Once again, the goodness of fit test shows that the fit is reasonable. Also, the correlation was again in excess of 0.97.

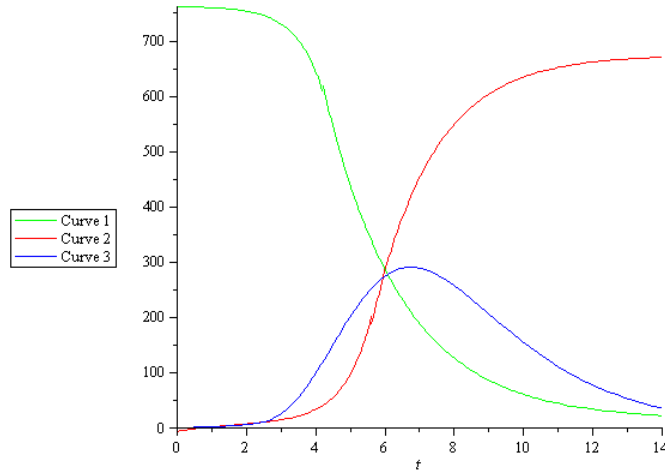


Figure 9: SIR Curves for Two Exponential $S(t)$

3.2 Susceptible as a Logistic Function

Assuming $S(t) = c + \frac{a}{b+e^{wt}}$ (i.e logistic function), we can solve the same non-linear Volterra integral equation we have been solving in section 3.1.1 and section 3.1.2.

For this function

$$S(0) = c + \frac{a}{b+1}, \quad a = (S_0 - c)(b+1)$$

Let us consider (2) with $S(t) = c + \frac{a}{b+e^{wt}}$

$$I(t) = I(0)e^{-\alpha t} + \frac{\beta}{N} \int_0^t \left(c + \frac{a}{b+e^{w\tau}} \right) I(\tau) e^{-(t-\tau)} d\tau$$

As we did before, we will change it to ODE and solve it. Thus,

$$\begin{aligned} \frac{dI}{dt} &= \frac{\beta}{N} S(t) I(t) - \alpha I(t) \\ \frac{dI}{I} &= \left(\frac{\beta}{N} \left(c + \frac{a}{b+e^{wt}} \right) - \alpha \right) dt \end{aligned}$$

This gives us

$$\ln |I| = \frac{\beta}{N} \left(ct + \int \frac{a}{b+e^{wt}} dt \right) - \alpha t + C_4$$

If we later say $u = b + e^{wt}$, then $\frac{du}{w(u-b)} = dt$ and we have the following integral:

$$\ln |I| = \frac{\beta}{N} \left(ct + \frac{a}{w} \int \frac{du}{u(u-b)} \right) - \alpha t + C_4$$

With this replacement, we have

$$\frac{1}{u(u-b)} = \frac{-\frac{1}{b}}{u} + \frac{\frac{1}{b}}{u-b}$$

from which we get

$$\ln |I| = \frac{\beta}{N} \left(ct + \frac{a}{w} \left(\frac{-1}{b} \int \frac{du}{u} + \frac{1}{b} \int \frac{du}{u-b} \right) \right) - \alpha t + C_4$$

From this follows

$$\ln |I| = \frac{\beta}{N} \left(ct + \frac{a}{bw} (\ln |u-b| - \ln |u|) \right) - \alpha t + C_4$$

Changing the above to exponential function and replacing the value of u back produces

$$I(t) = e^{\frac{\beta}{N} \left(ct + \frac{a}{bw} \ln \left(\frac{e^{wt}}{b+e^{wt}} \right) \right) - \alpha t + C_4}$$

To determine the constant C_4 we use the initial condition $I(0) = I_0$

$$e^{C_4} = I_0 e^{-\frac{\beta}{N} \frac{a}{bw} \ln \left(\frac{1}{b+1} \right)}$$

Since we know $I(t)$ and $S(t)$ we can determine $R(t)$ by

$$R(t) = N - I(t) - S(t)$$

which completes our solution.

Now we will consider the same example we have seen in section 3.1.1 and section 3.1.2. With $c = S_\infty = 25$, and using command `Fit` in Maple we will have a function

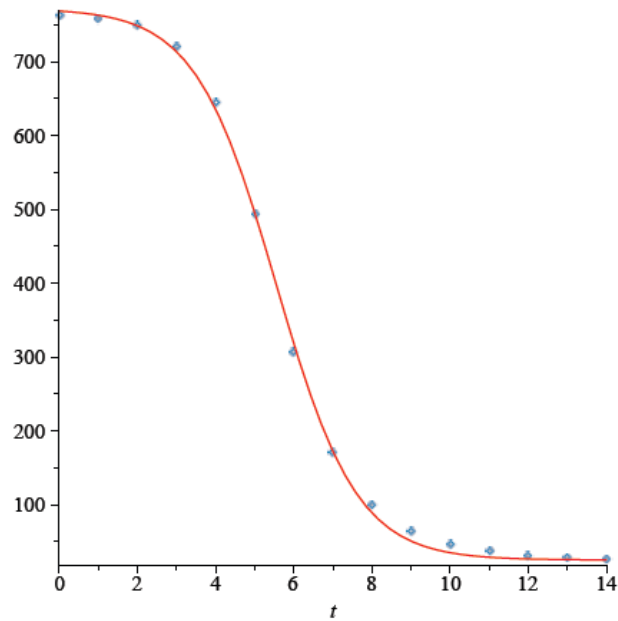


Figure 10: Best Fit Curve for Logistic Function

which best approximates our data with functions of our form as shown in Fig (10).

Then, we have the following functions which gives us Fig (11):

$$\begin{aligned}
 S(t) &= 25 + \frac{1.601 \times 10^5}{214.304 + e^{0.967t}} \\
 I(t) &= e^{-0.389t \ln\left(\frac{1.19e^{0.967t}}{214.217 + e^{0.967t}}\right) + 0.005e^{0.967t}} \\
 R(t) &= 745.122 - \frac{1.601 \times 10^5}{214.304 + e^{0.967t}} - e^{-0.389t \ln\left(\frac{1.19e^{0.967t}}{214.217 + e^{0.967t}}\right) + 0.005e^{0.967t}}
 \end{aligned}$$

Maple again not only reported the fit, but it also reported various measurements of the goodness of the fit. The correlation is more than 0.98 and the chi-square goodness of fit test confirmed our results. Also, we were able to obtain confidence intervals for each of the parameters in the curve fits. Specifically, in $S(t)$, we found that the 95% confidence interval for w is $[0.954, 0.989]$. The 95% confidence interval for a and b are nearly the same as the parameter values themselves. This shows that

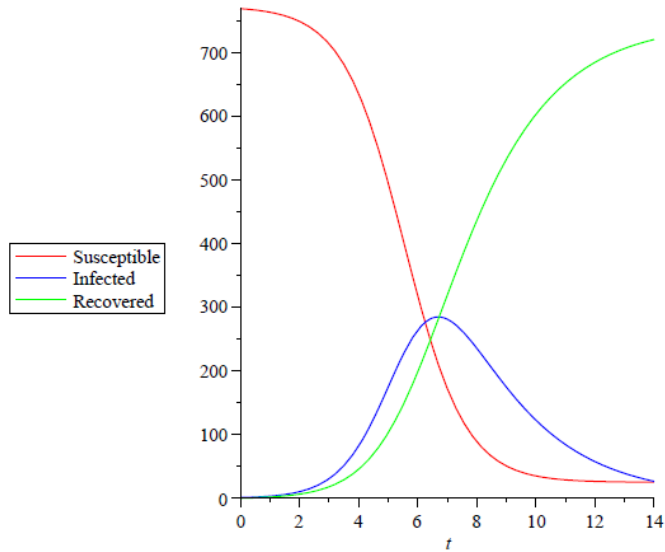


Figure 11: SIR Model Curves for Logistic Function

the logistic function form for $S(t)$ gives good results for the SIR model.

3.2.1 The Basic Reproductive Rate of Infection R_0

The data we usually have for research purposes are data we get from hospitals reports. This type of data commonly reports the number of “recovered”. We can also determine the average rate of recovery from the report. Using the following equations from our previous discussion;

$$\begin{aligned} \frac{dS}{dt} &= -\beta SI \\ S + I + R &= N \\ R' &= \alpha I \end{aligned}$$

Substituting $I = \frac{R'}{\alpha}$ in $S = N - I - R$, we have

$$\beta = \frac{\frac{R''(t)}{\alpha} + R'(t)}{N - \frac{R'(t)}{\alpha} - R(t)}$$

We can use our example to show how we can apply the above equation. Fitting the data with a function of the form

$$R(t) = \frac{a}{b + ce^{-\sigma t}} - d.$$

we have the fit curve Fig (12)

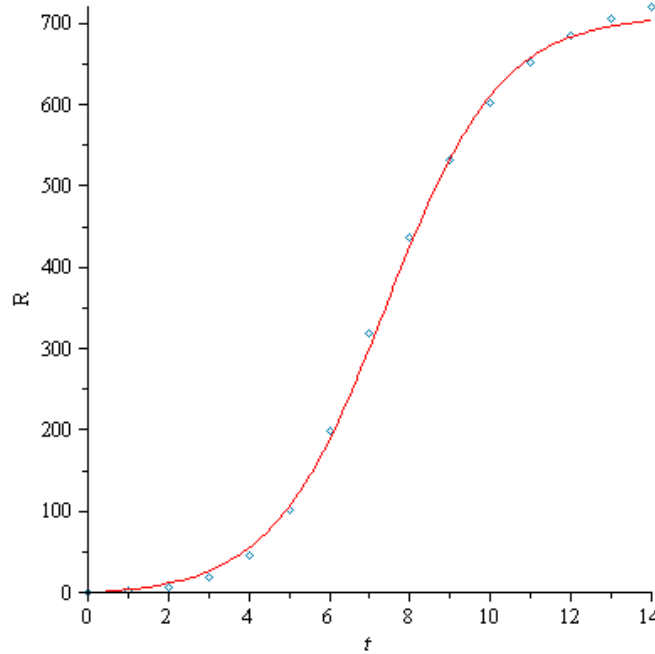


Figure 12: Data Fit Curve for “Recovery” Data

From Fig (12) we have $a = 99175.54$, $b = 138.99$, $c = 26540.56$, $\sigma = -0.709$ and $d = -3.717$. Thus,

$$R(t) = \frac{99175.54}{138.99 + 26540.56e^{-0.709t}} - 3.717$$

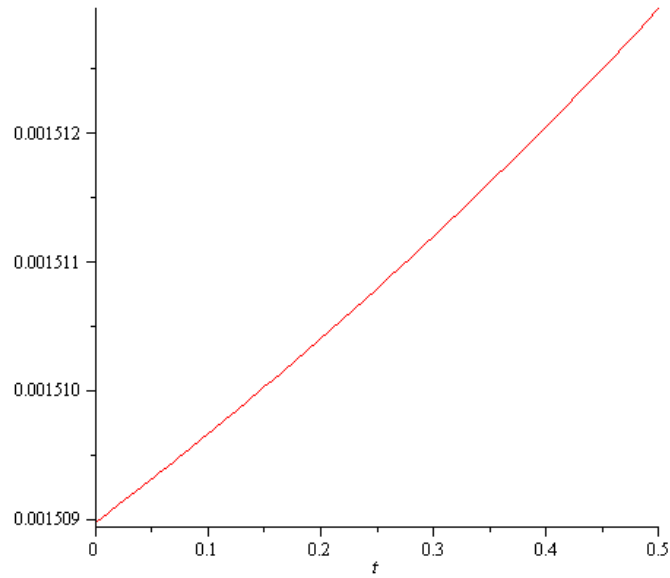


Figure 13: The Graph of β as a Function of Time

Using,

$$\beta = \frac{\frac{R''(t)}{\alpha} + R'(t)}{N - \frac{R'(t)}{\alpha} - R(t)}$$

We have $\beta = 0.001509$. The graph of β is shown in Fig (13). This leads to the approximation $\rho = 220.5$. Thus $R_0 = 3.45578$.

4 CONCLUSION

The classical SIR epidemiological model is one of the earliest and most important results of mathematical biology. It contains the most important features of epidemiology namely “Susceptible”, “Infected” and “Recovered”. The basic SIR model has its own limitation, but it is robust and can easily be extended. Depending on the type of the disease under study, one can modify it to include some more aspects of the disease. As a result we do have many variations of the model: SIS, SEIR, MSIR, SIRS, SQIR.

Experiments with infectious disease in human populations is impossible and unethical. Data are some times available from the natural occurrence of the epidemics. Usually these data are incomplete and under reported. From experience, we know that the data with all its weakness fits better with the results of this thesis’s functions ; $S(t)$, $I(t)$ and $R(t)$. This gives us a way to closely study and interpret the data so that we can make decisions to control a similar epidemic.

- As observed before, to find the appropriate multi-exponential $S(t) = a + \sum_{k=1}^n r_k e^{-\sigma_k t}$ to fit our data better, we may need to partition the data to two or more than two parts and fit each to a function and then we combine them.
- The other result discussed here is that of assuming $S(t) = \frac{a}{b + e^{wt}}$ which is commonly called the logistic function. In this case, to find the function which best fits the data, one should give initial values for variables a and b when using the Fit command with Maple software.

- After selecting $S(t)$, one can use all the results of this thesis to study the important features of the disease. As a result, it helps us to design the analysis of epidemiological surveys, suggest crucial data that should be collected, identify trends, and forecast and estimate uncertainty.

While we are working with this thesis we have observed that the data collected from an epidemic fits nicely with the function of a difference of two logistic functions for the “infectious” data. In case somebody has “infected” population data and wants to know whether there was epidemic or not, he/she can start from that and do what has been done in this thesis. In reality, most data reported from sources indicates the “recovered” population number. One can also start from “recovered” populations data and study the SIR model.

BIBLIOGRAPHY

- [1] Helen J.Wearing, Pejman Rohani and Mat J.Keeling, *Appropriate Model For The Management of Infectious Disease*, PLoS Med 2, No.7, e174, 2005.
- [2] Teri Johnson, *Mathematical Modeling of Disease. Susceptible-Infected-Recovered (SIR) model*, Math 409 Senior Seminar, University of Minnesota , 2009.
- [3] Vrushali A Boki, *Lecture Note, Introduction to the Mathematics of Infectious Disease*, Oregon State University, Corvallis, Oregon, August 1, 2007
- [4] David Smith and Lang Moore, The SIR model for spread of Disease, www.math.duke.edu/education/ccp/materials/diffcalc/sir/index.html, Duke University, Last Accessed July 30, 2010.
- [5] Modification of the SIR model, www.math.lsa.umich.edu/~tjacks/sirlecs.ppt., Last Accessed July 6, 2010.
- [6] Abdul-Majid Wazwaz, *A First Course in Integral Equation*, World Scientific Publishing, 15-20, 1997
- [7] F. G. Tricomi, *Integral Equation*, Dover Publication Inc., 1-42, 1985
- [8] Rainer Kress, *Linear Integral Equation*, Second Edition, Springer-Verlag Inc., 1-13, 1999
- [9] Ram P.Kanwal, *Linear Integral Equations, Theory and Techniques*, Second Edition, The Random house Birkhauser Mathematics Series, 25-36, 1997

- [10] Ida Del Prete, *Efficient Numerical Methods For Volterra Integral Equation of Hammer Stein type*, Universita Di Napoli Federico II, 1-16, 2006
- [11] Herbert W.Hethcote, *Quantitative Analysis of Communicable Disease Model*, Mathematical Biosciences 28, 335-356 , 1976
- [12] Christopher D. Green, *Integral Equation Methods*, Barnes and Noble Inc., 81-90, 1969
- [13] M.Krasnov, A.Kieslev and G.Mkarenko, *Problems and Exercises in Integral Equations*, Mir Publisher, 15-62, 1971
- [14] Gustav Doetsch, *Guide to the Application of the Laplace Transform and Z Transform*, Second Edition, Van Nostrand Reinhold Company, 118-121, 1971.
- [15] Joel L.Schiff, *The Laplace Transform Theory and Application*, Springer-Verlag Inc., 1-34, 1999.
- [16] S.Patrak, A.Maiti, G.P. Samanata, *Rich Dynamics of an SIR epidemic model*, Nonlinear Analysis, Modeling and Control 15, 71-81, 2010
- [17] Michael Holile, *Population Dynamics I, The SIR model*, Department of Animal Science and Health, Royal Veterinary and Agriculture University, 2002
- [18] Fred Brauer and Carlos Castillo-Chavez, *Mathematical Models in Population Biology and Epidemiology*, Springer-Verlag Inc., 273-332, 2001

APPENDICES

Appendix MAPLE COMMANDS

1. TO PRODUCE FIGURE 6

```
dataplot := ScatterPlot(A, B, symbol = diamond, symbolsize = 20, color = blue);
S0 := Fit(762+C[1]*exp(-aa[1]*t), A[1 .. 6], B[1 .. 6], t, initialvalues = [C[1] = -
1.52181363299841910, aa[1] = 1.0852]);
S1 := Fit(25+C[1]*exp(-aa[1]*t), A[6 .. 13], B[6 .. 13], t, initialvalues = [C[1] = 100,
aa[1] = 10-5]);
l0 := plot(S0, t = 0 .. 5, color = green);
l1 := plot(S1, t = 5 .. 14, color = green);
d1 := display(l0, l1);
display(dataplot, l0, l1)
```

2. TO PRODUCE FIGURE 7

```
a1 := 762; b1 := -2.94757082275819516; sigma1 := -.903717448872219053;
a2 := 25; b2 := 8820.8029307937304; sigma2 := .584262909390753205;
I1 := exp(beta*a1*t/(1.132)+beta*b1*exp(-sigma1*t)/sigma1-alpha*t+beta*b1/(1.35*sigma1));
I2 := exp(beta*(a2*t-b2*exp(-sigma2*t)/sigma2)-alpha*t+beta*b2/(3.6*sigma2));
P1 := plot(I1, t = 0 .. 3.81, color = blue);
P2 := plot(I2, t = 3.81 .. 14, color = blue);
```

```

R2 := N-alpha*b2*exp(-sigma2*t)/(alpha-sigma2)+(-N+alpha*b2/(3/2*(alpha-sigma2)))*exp(-
alpha*t);
R12 := plot(R2, t = 6.1 .. 14);
R11 := plot(R1, t = 0 .. 6.2);
d := ScatterPlot(A, R, symbol = diamond, symbolsize = 20, color = blue);
display(d1, d2, d3, linestyle = solid, color = [red, blue, green]);

```

3. TO PRODUCE FIGURE 8

```

S011 := Fit(762.5+C11*exp(-a11*t)+C21*exp(-a21*t), A[1 .. 6], B[1 .. 6], t,
initialvalues = [C11 = -1.52181363299841910, a11 = 10.0852, C21 = -2.1, a21 = -10]);
S11 := Fit(15+C12*exp(-a11*t)+C22*exp(-a21*t), A[5 .. 14], B[5 .. 14], t,
initialvalues = [C12 = -1000.75181363299841910, a11 = 100000.0732, C22 = 3000,
a21 = -.9]);
dtplot := ScatterPlot(A, B, symbol = diamond, symbolsize = 20, color = blue);
l01 := plot(S011, t = 0 .. 4.2, color = green); l11 := plot(S11, t = 4.2 .. 14,
color = green); se := display(l01, l11);
g := 15; r11 := -1000.75181363299998; r21 := 3931.86878378646998;
sigma 11 := 1.00000073199999999*105; sigma 12 := .445677297008923612;
f := 762.5; r1 := -.595872937335464426; sigma 21 := -1.32299589086598157;
r2 := 2.23037891409369903*10-17; sigma 22 := -8.70076794567668976;
I21:=exp(.901*beta*(f*t-2*r1*exp(-sigma 21*t)/sigma 12-3*r2*exp(-sigma 22*t)/sigma 22)
-1.2*alpha*t+beta*(r1/sigma 21+r2/sigma 22))-45399;

```

```

I22 := exp(1.001*beta*(g*t-r11*exp(-sigma 11*t))/sigma 11-r21*exp(-sigma 12*t)/sigma 12)
-alpha*t+5.87*beta*(r11/sigma 21+1/sigma 12));
u1 := plot(I22, t = 2.5 .. 14, color = blue);
u2 := plot(I21, t = 0 .. 2.5, color = blue);
Ie := display(u1, u2);
display(dtplot, l01, l11);

```

4. TO PRODUCE FIGURE 9

```

R77 := 1.0022*N-6*g-29*alpha*r11*exp(-8*sigma 11*t)/(2*N*(alpha-sigma 11))-alpha*r21*exp(-30*sigma
12*t)/(200*N*(alpha-sigma 12))+(-(19/2)*N+(35/2)*g+(6*9)*alpha
r11/(2*N*(alpha-sigma 11))+10*alpha
r21/(N*(alpha-sigma 12))*exp(-1.29*alpha*t);
R21 := 1.011*N-f-alpha*r1*exp(-.999987*sigma 21*t)/(1.3*(alpha-sigma 21))-alpha*
r2*exp(-.81*sigma 22*t)/(22.5*(alpha-sigma 21))+(-1.025*N+f+alpha*r1/(4*(alpha-sigma 21))
+alpha*r2/(alpha-sigma 22))*exp(-1.01*alpha*t)+5;
R9 := plot(R21, t = 0 .. 5.56, color = red);
R99 := plot(R77, t = 5.6 .. 14, color = red);
Ree := display(R9, R99);
display(Ie, Ree, se, linestyle = solid);

```

5. TO PRODUCE FIGURE 10

```

S4 := Fit(25+a/(b+exp(w*t)), A, B, t, initialvalues = [a = 200000, b = 1, w = .3]);
ScatterPlot(A, B, plot(S4, t = 0 .. 14));

```

6. TO PRODUCE FIGURE 11

```
a1 := 1.60081881732383365*105; b1 := 214.217265807576496; w1 := 0.967296187971025124;  
I4 := exp( $\beta$ *(25*t+a1*ln(1.19*exp(w1*t)/(b1+exp(w1*t))))/b1)-alpha*t- $\beta$ *a1*ln(1/(b1+1))/b1);  
ScatterPlot(A, Dat, plot(I4, t = 0 .. 14));  
R4 := N-S4-I4+7.122246889;  
plot([S4, I4, R4], t = 0 .. 14, linestyle = [solid, solid, solid], color = [red, blue, green]);
```

VITA

TESHOME MOGESSIE BALKEW

- Education: B.S. Mathematics, Addis Ababa University in Ethiopia,
 Addis Ababa, Ethiopia, 1989
 M.S. Mathematics, Addis Ababa University
 Addis Ababa, Ethiopia, 1998
 M.S. Mathematical Sciences, East Tennessee State University
 Johnson City, Tennessee, 2010
- Professional Experience: High School Teacher, Wachemo Cop.Sec.School In Ethiopia
 Hossana, Ethiopia, 1989–1995
 Lecturer, Alemaya University
 Alemaya, Ethiopia, 1998-2002
 Lecturer, Addis Ababa University
 Addis Ababa, Ethiopia, 2002-2008
 Graduate Assistant, East Tennessee State University
 Johnson City, Tennessee, 2008-2010