MANAL GHANNAM

SVIR MODEL OF VARICELLA VIRUS IN JORDAN

MASTER THESIS

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DEPARTMENT OF MATHEMATICS

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M.Sc. THESIS

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Approval

We certify that we have read the thesis submitted by Manal Ghannam titled "**SVIR MODEL OF VARICELLA VIRUS IN JORDAN**" and that in our combined opinion it is fully adequate, in scope and in quality, as a thesis for the degree of Master of Educational Sciences.

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Declaration

I hereby declare that all information, documents, analysis and results in this thesis have been collected and presented according to the academic rules and ethical guidelines of the Institute of Graduate Studies, Near East University. I also declare that as required by these rules and conduct, I have fully cited and referenced information and data that are not original to this study.

Manal Ghannam

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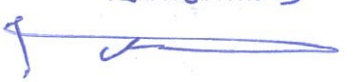

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Manal Ghannam

Abstract

SVIR Model of Varicella Virus in Jordan

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Varicella virus, also referred to as chickenpox, is an extremely infectious disease. Mathematical modelling is a very helpful technique, that used to Study the structure of the infectious diseases transmission and assess possible treatments needed. In this thesis we propose a SVIR model for Varicella Virus in Jordan and analyse the dynamics of transmission using mathematical modelling and simulation.

Firstly, the model is formulated using four compartments, susceptible (S), Vaccinated (V), Infected (I), and Recovery (R), with four differential equations. Then two equilibrium points are found and the local stability of them are existing depending on the basic reproduction number (R_0). If $R_0 > 1$,the endemic equilibrium point is stable and the infectious disease is present between individuals continuously, while when $R_0 < 1$, then the disease-free equilibrium remains stable and the disease will be under control. Sensitivity analysis is conducted in order to identify the parameters that would have the greatest influence on the fundamental reproduction ratio (R_0).

Overall, the results of this research help to clarify the mechanisms of varicella virus transmission and guide policy decisions related to public safety. Governments and medical organizations can develop solid strategies that reduce the impact of varicella virus and secure those most affected with the help of the knowledge gathered from this study.

Key Words: SVIR model, Varicella virus, basic reproduction number, local stability, sensitivity analysis.

ÖZET

SVIR Model of Varicella Virus in Jordan

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Suçiçeği olarak da adlandırılan varisella virüsü son derece bulaşıcı bir hastalıktır. Matematiksel modelleme, bulaşıcı hastalıkların bulaşma yapısını incelemek ve ihtiyaç duyulan olası tedavileri değerlendirmek için kullanılan çok yararlı bir tekniktir. Bu tezde, Ürdün'de Varisella Virüsü için bir SVIR modeli önermekte ve matematiksel modelleme ve simülasyon kullanarak bulaşma dinamiklerini analiz etmekteyiz.

İlk olarak, model, dört diferansiyel denklem ile duyarlı (S), Aşılanmış (V), Enfekte (I) ve İyileşme (R) olmak üzere dört bölme kullanılarak formüle edilmiştir. Daha sonra iki denge noktası bulunmuş ve bunların yerel kararlılığı temel üreme sayısına (R_0) bağlı olarak mevcut olmuştur. $R_0 > 1$ ise, endemik denge noktası kararlıdır ve bulaşıcı hastalık bireyler arasında sürekli olarak mevcuttur, $R_0 < 1$ olduğunda ise hastalıksız denge kararlı kalır ve hastalık kontrol altında olur. Temel üreme oranı (R_0) üzerinde en büyük etkiye sahip olacak parametreleri belirlemek için duyarlılık analizi yapılmıştır.

Genel olarak, bu araştırmanın sonuçları suçiçeği virüsü bulaşma mekanizmalarının açıklığa kavuşturulmasına yardımcı olmakta ve kamu güvenliği ile ilgili politika kararlarına rehberlik etmektedir. Hükümetler ve sağlık kuruluşları, bu çalışmadan elde edilen bilgiler yardımıyla suçiçeği virüsünün etkisini azaltan ve en çok etkilenenleri güvence altına alan sağlam stratejiler geliştirebilir.

Anahtar Kelimeler: SVIR modeli, Varisella virüsü, temel üreme sayısı, yerel kararlılık, duyarlılık analizi.

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List of Abbreviations

SIVR:	Susceptible, Infected, Vaccinated, Recovery
CDC:	Centres for Disease Control
VZV:	Varicella-zoster virus
HZ:	Herpes zoster
MMR:	Measles, Mumps, and Rubella
DFE:	Disease Free Equilibrium
ODE:	Ordinary Differential Equation

CHAPTER I
Introduction

1.1 Statement of the Problem

The varicella Virus, also called chickenpox, is an extremely transmissible viral infectious disease. Although there is a vaccination for varicella, it is still a serious public health issue in Jordan. Children, who are more likely to experience more severe symptoms, are among those who are at risk from the virus's spread. Thus, in order to decrease the effects of this infectious disease in Jordan, it is essential to comprehend the dynamics of varicella transmission to determine efficient management techniques.

The varicella Virus infection dynamics specifically in Jordan have received relatively little academic attention. Current research depends frequently on models created for various populations or regions in the world, that might not correctly reflect the varicella spread in Jordan. In addition, limited study has been done on how well different management methods including vaccination drives and preventive measures work in the Jordanian conditions. This thesis was inspired by worries concerning the serious nature of the Varicella virus infection (chickenpox) in Jordan.

1.2 Purpose of the Study

The aim of this thesis is to create and evaluate a Susceptible-Vaccinated-Infected-Recovered (SVIR) model that is specifically adapted to the varicella virus spread dynamics in Jordan. Utilizing the epidemiological information currently accessible.

1.3 Research Question

How the different parameters of the SIVR model affect the Varicella virus potential spread and how the number of infected people daily contact will affect or even stop the spread of the disease in Jordan.

1.4 Significance of the Study

Studying the dynamics of Varicella virus transmission in Jordan is significant, especially these days, to take immediate actions before the massive increase of infections by examining the SVIR model's essential parameters which aid the government in establishing new governing policies and plans for future occurrences of situations resembling the current one.

In Jordan, 82856 cases of chickenpox were documented between 2008 till 2021 as showed in table 1.1 (Annual Statistical Report of Communicable Diseases 2021).

Table 1.1 Varicella Virus history in Jordan from 2008 to 2021

Year	Chickenpox Cases
2008	11356
2009	6906
2010	9362
2011	6181
2012	6435
2013	6706
2014	7888
2015	4715
2016	4074
2017	6880
2018	6211
2019	3515
2020	999
2021	1628

The number of the total cases clearly increased in 2014 with percentage of 19.24% linked to the immigration of Syrian refugees, while the least number of cases (2.44%) was recorded in 2020 as a result of the COVID-19 as well as quarantine during the pandemic, which was particularly due to the closure of clinics and schools (Alfauri, et al., 2022).

People who got infected with Varicella virus can easily infect those who did not previously experience the virus or received no vaccinations against it. If an individual gets it, up to 90% of their nearby non-immune contacts will catch it as well (CDC, 2021). The probability for Varicella virus to transmit in the susceptible individuals is estimated to be about 0.09 (Steiner, Wallrafen, & Weiss, 2018).

1.5 Limitations

In order to make the study easier to understand, mathematical models frequently introduce a few assumptions. Such presumptions might fail to fully represent the level of detail of actual-life dynamics, which could restrict the accuracy or utility of the model, for example, age and gender's effects are neglected to simplify the model and decrease the parameters.

It is necessary to clarify that increasing the number of the parameters will make the structure of the model more complicated. It is challenging to determine all the factors with objectivity in the absence of epidemiological data (Harb, & Harb, 2020). Large uncertainty in the model's forecast will result from having so many unknown parameters. In order to minimize the uncertainty, this work developed a simplified SVIR model with the fewest available parameters using the limited data available.

Each area or nation has its own distinct qualities, such as population statistics, medical facilities, and traditions of culture. In this SVIR model in Jordan, it was very difficult to take these factors under consideration because of the limit of resources and the lack of data, also an increase in population due to different nations immigration to Jordan could be a relevant factor to consider when studying the spread of infectious diseases, including varicella, and that is neglected in this study, because of that it is considered one of the important limitations.

1.6 Definition of Terms

1.6.1 Infectious disease

A variety of species, including fungus, virus, parasites, and bacteria can cause infectious disorders. Direct contact between people or animals can result in the direct transmission of infectious diseases. Additionally, they can be transferred by indirect contact, which includes touching object like a doorknob. Furthermore, insects and polluted food may transfer diseases (UMAR, 2021).

Infectious diseases happened to be the greatest cause of death globally at the start of the 20th century. In 1900 a new born baby had a nearly 10% risk of passing away before reaching the age of 4, mainly from asthma or diarrhea. However, overall life duration significantly rose as a result of advancements in living conditions, cleanliness, and the reach of healthier water and food. The higher predicted life expectancy is also a result of the development of antibiotics during the middle of the 20th century. In 1969, Surgeon General William H. Stewart stated to the US Congress to "close the book on infectious diseases" due to the incredible advancements achieved in the fight against illness. He had no idea of the rise in newly emerging and resistance to antibiotics infections that occurred over the period of the next 30 years. For example, less than 100 people died from the H5N1(avian flu) in 2007, while 2.1 million people died from AIDS at the same year (Avila, Saïd, & Ojcius, 2008).

In 2011, the Health and Social Act developed an effective statement which was “Good infection prevention and control are essential to ensure that people who use health and social care services receive safe and effective care," (Török, Moran, & Cooke, 2017).

Recently, a very dangerous virus attacked all the populations causing a lot of death, it is Coronavirus Disease also known as CoViD-19, which is a type of infectious disease caused by a virus called SARS-CoV-2 virus. The novel virus is transmitted by a very small drop of liquid (droplets) that escape through the lungs (Wang, et al., 2020). Globally, as of December 2022, there have been 642,924,560

confirmed cases of COVID-19, including 6,625,029 deaths, reported to WHO (WHO, 2022).

Health Ministries alone are unable to protect public health. It takes the participation of several sectors to develop awareness. The world has a comprehensive grasp at this stage in history of how well-prepared most nations are to handle pandemics and what needs to be taken to increase readiness. A well-planned action should be executed at scale as quickly as possible. This will create the safest life (Shahpar, et al., 2019).

1.6.2 Varicella Virus

Varicella-zoster Virus (VZV), only affects humans. VZV is a very contagious disease that affects millions of individuals all over the world each year. Acute varicella, sometimes known as "chickenpox," results from a primary infection, which generally occurs through direct touching with a bump on the infected skin or through the breathing of droplets from the lungs. After initial infection, VZV develops permanent latency in the cranial nerve and dorsal root ganglia and may become active decades or even years later as "shingles" or herpes zoster (HZ) (Pergam, Limaye, & AST Infectious Diseases Community of Practice, 2009). Usually, a person has their first symptoms two to three weeks after being infected. It started by fever, exhaustion, and a lack of desire for food which are followed by a widespread rash later. The rash begins as itching red spots but quickly develops into blisters that appears more common on the arms, legs, face, and head. The blisters remain for three to four days before they heal over and dry out. Kids and adults with good health often recover in ten days. It can be treated by paracetamol to reduce the fever, but usually the majority of individuals don't need medical help. Fluid intake and rest are advised. Maintaining nails short and using anti-itch soaps and creams can help to reduce the risk of infection (Centre for Disease Control, 2023).

In the 1970s and 1980s, a live attenuated VZV (Oka strain) vaccine was created and experimentally evaluated. In 1984 Sweden and Germany started using the vaccine. Today these vaccines can be either varicella vaccine only or (MMR) which is combined with rubella, mumps and measles (WHO, 2014).

In 1995, USA approved the vaccination and advised to routinely provide the live, attenuated varicella vaccine, created by Takahashi in 1974, to healthy infants between the ages of 12 and 18 months and to older kids who have not yet experienced chickenpox. There exists a present additional finding from case-control research that is currently being conducted on the impact of age at vaccination as well as the effectiveness of this vaccine after a certain period of time (Vázquez, et al., 2004). While it is commonly thought of this virus to be a mild childhood disease, it can have serious consequences in select populations, including adults and immunocompromised people.

1.6.3 Herd immunity

Herd immunity is a phrase used in the field of healthcare to explain how a group of people might be immunized from disease by reaching a certain percentage of immunized members. Vaccination is the major technique used to create herd immunity. Herd immunity plays an essential role in preventing the spread of the infectious disease that has only human as host. Herd immunity is considered to be maintained when at least 90% of a community is immune to the infectious disease; however, even when this crucial threshold is reached, still the problem of the annual cases that is increasing specially in developed countries occurs (Wessel, 2016).

1.6.4 Mathematical Modelling

A mathematical modelling is using mathematical equations and concepts to illustrate real-world issues. Many sectors, including the social sciences, agriculture, medicine, and economy frequently employ mathematical models. These models might be stochastic or deterministic, linear or nonlinear. Mathematical models have been used to forecast disease epidemics and understanding how a virus spreads within a population. Currently, there are various mathematical models used to explain disease processes (Ahmed, et al., 2021).

There have been several models created since Kermack and McKendrick first published epidemic models in 1927, including SIR, SIS, SEIR, SVIR, SVEIR, and others. Analysis of stability and existence of equilibria is a common task for writers (Ramadani, & Aldila, 2019).

Kermack Mckendrick's SIR model studied epidemics of measles in United Kingdom a constant population was assumed in the model which is divided into three compartments. Individuals in the susceptibility compartment have no immunity to the infectious disease; any member of the susceptible class could contract it. Individuals in the infectious compartment are those who are currently infected and can spread the virus to others. Individuals in the Recovered compartment have recovered from the infection and have achieved permanent immunity. Their research showed that the infection threshold happens whenever the reproduction rate R_0equals one. If the $R_0 > 1$, the infectious disease will spread. If the $R_0 < 1$, the disease vanished out in the susceptible individuals. This is known as the SIR model (Narsingani, & Bhathawala, 2017).

Li, J., & Zou, X. (2009) wrote Generalization of the Kermack-McKendrick SIR model to a patchy environment for a disease with latency, their research believed that an infectious disease had a defined latent period in a population. A demographic framework for two distinct towns was developed. Their framework uses a system of delay differential equations with a fixed delay to account for latency and non-local components induced by the people' movement throughout the latent time frame. The infection eventually fades out, with just a small percentage of the susceptible individuals unaffected. Their approach was later shown to be incompatible with the Kermack-McKendrick (1927) model.

Basic reproduction statistics are constructed to illustrate the model's degree of endemic regions. One of the most effective mathematical models for illustrating the spread of a pandemic is the susceptible-vaccinate-infectious-recovered (SVIR) model (Abou-Ismail, 2020). Usually, individuals only get chickenpox once. Assuming a SVIR model, where individuals transition from being susceptible to being infected to recovering, or from susceptible to being vaccinated then recovered without being infected to make the model more realistic. Consequently, this model has four sections (Steiner, Wallrafen, & Weiss, 2018).

The analysis of various scenarios and the comprehension of Varicella transmission can be done with the use of mathematical modelling. However, rather than concentrating on which model is true, we should acknowledge that "one model cannot answer it all" and that we need more models that provide answers to related questions in order to put the puzzle together and stop the spread of the disease (Mohamadou, Halidou, & Kapen, 2020).

1.7 Thesis Structure

There are six essential chapters in the thesis. The introduction to the thesis is presented in Chapter 1. Relevant studies and articles are reviewed in chapter two. Chapter three presents the mathematical model's construction. The analysis and findings of the model are presented in Chapter 4. In Chapter 5 the discussion of the findings is written there, and in chapter six conclusion and recommendations for additional research are clarified, at the end of this thesis references and appendices are listed to finalize the research process correctly.

CHAPTER II

Literature Review

2.1 Theoretical Framework

A conceptual platform for comprehending of varicella virus transmission dynamics and the variables and parameters affecting its transmission is provided by the SVIR model theoretical framework of the varicella virus in Jordan. These theoretical frameworks can be summarized the following points:

2.1.1 Concepts related to epidemiology

To measure the spreading ability of the varicella virus, the theoretical framework uses the concepts of the basic reproduction number (R_0), which will be defined later in details in chapter 3. Then the framework clarifies the disease Progress by showing the movement of the individuals between the susceptible, vaccinated, infected, and recovered compartments, by taking into account the life cycle of varicella virus, which includes the contagious duration, and healing time.

2.1.2 Vaccination Regulations

The theoretical framework shows the vaccination coverage percentages in Jordan, taking into account the efficiency of the varicella vaccination, which will be clear while dealing with the parameters of the model.

2.1.3 Control Techniques

Assess how the application of quarantine measures such as rules for susceptible and isolation of infected individuals, in addition to health awareness and education, the effect of all of these factors in decreasing the Varicella virus infection transmission will be proved.

2.2 Literature Review of Infectious Diseases Modelling

This section reviews some of the previously published studies on the transmission of contagious infectious diseases, particularly varicella virus. Mathematical modelling for infectious diseases has been successfully used to investigate the transmission of numerous infectious diseases. These models sometimes couldn't describe the real-life situation or provided a realistic result for the disease.

In particular the research of Varicella virus's epidemiology has not received much attention in Jordan. The spread of this infectious disease can be investigated using mathematical models. Various researchers have modelled the chickenpox disease in a number of ways throughout the years. As an example, Diekmann, et al. (1990) gave the meaning of the basic reproduction ratio R_0 as the anticipated individual of secondary cases that an infected person should cause over the course of the disease's infectiousness in a community that is entirely susceptible. The well-known threshold principle then indicates that if $R_0 > 1$, the infectious disease could attack but if $R_0 < 1$, it couldn't (Diekmann, et al., 1990). Any new infectious disease's risk of an epidemic is calculated using the magnitude of R_0 (Sayan, et al., 2018). This magnitude of R0, is typically computed from a population to anticipate the potential seriousness of epidemics of infectious diseases like SARS, tuberculosis, HIV, and chickenpox (Breban, Vardavas, & Blower, 2007), and recently in covid-19 pandemic, R0 and Rt (the effective reproduction number) used as herd immunity to fight this pandemic in North Cyprus (Hincal, Kaymakamzade, & Gokbulut, 2020). Capaldi, et al. discussed the use of sensitivity analysis and asymptotic statistical theory to determine measures of uncertainty for estimations of model parameters and (R_0) (Capaldi, et al., 2012).

In 2014 a model of the Varicella zoster virus with vaccination was created and stability study was performed by Stephen et al. The model's computer simulations demonstrated that the best strategy for controlling the spread of VZV in the community is to combine vaccination with therapy (Edward, Kuznetsov, & Mirau, 2014). Another study shows that it is possible that the seasonal variation in the predicted contact rates represents how vacations affect the spread of chickenpox

among students using SEIR model (Deguen, Thomas, & Chau 2000). While some researchers studied the potential effects of varicella vaccinations by examining the epidemics of measles virus before and after vaccination (Ferguson, Anderson, & Garnett, 1996).

As Daley and Gani introduced in their book, to make the model fit the prediction, three aims should be satisfied. The first is to research the most effective methods for disease transmission. Second to forecast how the epidemic may develop in the future, and the third to comprehend how quarantine, vaccine, and awareness can be used to control the epidemic's spread (Daley, & Gani, 2001). Steiner model explained that because of vaccination, a specific percentage of the susceptible population transfers each day from the susceptible to the recovered compartment without being infected (Steiner, Wallrafen, & Weiss, 2018). Forde, & Meeker (2010), proposed a model of varicella-zoster reactivation. They analysed the model's implications for vaccine enhancement plans intended to avoid herpes zoster after building the model and showing that it displays the kind of periodic pattern required for ongoing latency period and activation.

Based on the number of chickenpox cases recorded in Assam's Kamrup Metro district in India, where a pandemic of the disease occurred in 2017, SIR model has been developed by Devi (2018), to estimate the number of susceptible, infected, and recovered people. The SIR model reveals that the impact on the R_0 is unity, indicating that the disease is totally curable. Thus, the outcomes of this SIR model explained how chickenpox propagated in that region and aided in the forecasting of disease outbreaks.

Witbooi, Muller, & Van Schalkwyk, (2015) demonstrated almost certain exponential stability of the disease-free equilibrium for the stochastic differential equation-based SVIR model using includes vaccine, when R_0 is less than 1 and in their research, they studied the optimal control and created an effective technique to use vaccine. The results succeed to decrease the transmission of the virus and balance the total cost of vaccine.

For the purpose to describe the impact of vaccination upon a viral infectious disease by the SVIR, Ozdemir, Ucar, & Avcı, (2022) presented a research of a nonlinear fractional order system. The model provided by the ordinary differential equation is redone using the Caputo fractional derivative to observe the impact of memory on the system components. Following a stability analysis and justifications of the disease SVIR model, the system's existence and uniqueness are established. The results produced with the use of MATLAB show that the infection remains controllable by vaccination when $R_0 < 1$.

Mathematical modelling of chickenpox transmission dynamics can provide useful insights into the disease's spread and improve public health methods for controlling and preventing its spread. The prevalence of chickenpox in Jordan has progressively increased in recent years, underscoring the need for a better understanding of its epidemiology. The purpose of this thesis is to create a mathematical model for the transmission of chickenpox in Jordan that incorporates demographic, social, and environmental elements, and to use the model to assess the impact of various management efforts. The findings of this study can assist policymakers in Jordan in making educated decisions about chickenpox prevention and control, as well as serve as a valuable resource for future research in this field.

CHAPTER III

Methodology

3.1 Model Assumptions

In epidemiological research, the SVIR Model is applied to calculate the proportion of susceptible, vaccinated, infected, and recovered individuals in a community. Additionally, it is employed to describe how the number of individuals in need of medical care during the outbreak. SVIR model can be applied appropriately if the recovered individual has a lifetime immunity from the disease (Johnson, & McQuarrie, 2009). The population (N) is made up of four different compartments of people, which are represented by Susceptible (S), Vaccinated (V), Infected (I), and Recovered (R), all of them are functions of time(t) that depends on a set of differential equations. In order to create our SVIR model, we have to assume many assumptions which can be very helpful in our analysis. These assumptions are:

i. Population homogeneity—the model does not take into account the possibility that individuals may differ from one another in ways that are important for the spread of infection.

ii. Susceptible persons who have received vaccinations may still be susceptible because of vaccination failure, or they can transfer to recovery compartment without getting infected if the vaccine offers strong protection.

iii. Permanent immunity is granted after being infected and recovered.

iv. Age and gender do not affect the probability of getting the infection.

v. Every parameter in this model is assumed to be positive.

vi. Exponentially distributed duration of infection—this is a reference to the model's assumption that a person becomes infectious as soon as it is infected.

3.2 Model Formulation

The basic strategy that modelers can use to answer questions is to build more complex models, swapping out some of the unrealistic assumptions with more realistic ones, and then analyse how the behaviour of the models changes when the assumptions are changed. My SVIR model consists of four compartments:

1. Susceptible individuals that may catch the virus.
2. Vaccinated individuals that took the vaccine and have lifelong immunity from the virus.
3. Infected individuals that have the virus and can infect others
4. Recovered individuals that took the virus and recovered with lifelong immunity from the virus.

The suggested model, segmented the total population size, N, into four stages of disease: S, susceptible; V, vaccinated; I, infected; R, recovered, and can be written by

$$N(t) = S(t) + V(t) + I(t) + R(t), \tag{3.1}$$

the interaction between these four stages is illustrated in figure 3.1.

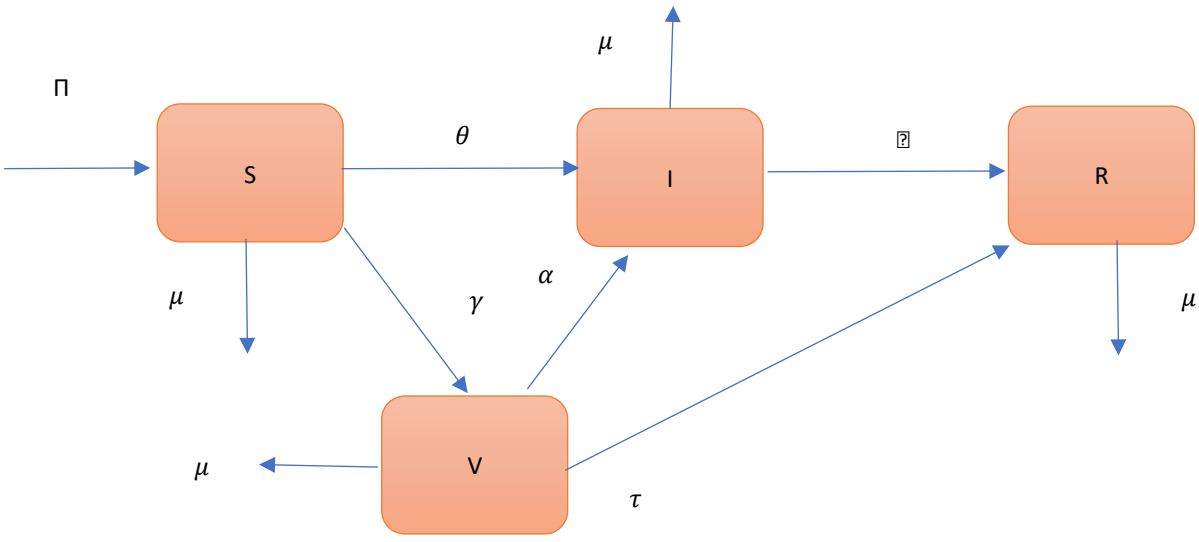

Figure 3.1: Transfer diagram of the model

3.3 Model Equations

Now by using the Mass Action Law of infectious diseases, we get the following system of nonlinear ODEs to explain the Varicella outbreak in Jordan:

$$\frac{dS}{dt} = \Pi N - (\mu + \gamma + \theta I)S, \tag{3.2}$$

$$\frac{dV}{dt} = \gamma S - (\alpha I + \tau + \mu)V, \tag{3.3}$$

$$\frac{dI}{dt} = \theta IS + \alpha IV - (\beta + \mu)I, \tag{3.4}$$

$$\frac{dR}{dt} = \beta I + \tau V - \mu R, \tag{3.5}$$

where,

$\frac{dS}{dt}, \frac{dV}{dt}, \frac{dI}{dt}, and\ \frac{dR}{dt}$ measure the rates of change of the quantities S(t), V(t), I(t), and R(t). All the descriptions and definitions of the parameters and the variables that used in the model are listed in tables 3.1 and 3.2.

Table 3.1 Variables of the model

Variables	Descriptions
S	Individuals who may get infected with Varicella virus
I	Individuals who are infected with Varicella virus
R	Individuals who have recovered from infection and acquired immunity
V	individuals who are vaccinated
N	The total number of population
t	Time

Table 3.2 Parameters of the model

Parameters	Descriptions
Π	The birth rate
θ	The contact rate (transmission rate)
β	The recovery rate
μ	The natural death rate
γ	The rate that susceptible persons become vaccinated
α	The rate that vaccinated persons become infected because of vaccination failure
τ	The effect rate of vaccine in protecting from the infection(get immunity)

3.4 Equilibrium Points of the Model

As our model system is represented by the above four differential equations (3.2 to 3.5) which explained the dynamics of the virus and the outbreak in Jordan. This system of equations considered non-linear differential equations.

Now, as we mentioned in equation 3.1, N(t) represents the total population in Jordan which is about 10,888,834 till the end of 2021 according to Statistical Report of Communicable Diseases in Jordan. We can re-write each compartment as a ratio of the total population to get,

$$P_s(t) = \frac{S(t)}{N}, \tag{3.6}$$

$$P_V(t) = \frac{V(t)}{N}, \tag{3.7}$$

$$P_I(t) = \frac{I(t)}{N}, \tag{3.8}$$

$$P_R(t) = \frac{R(t)}{N}, \tag{3.9}$$

$$P_N(t) = \frac{N(t)}{N} = P_s(t) + P_V(t) + P_I(t) + P_R(t) = 1. \tag{3.10}$$

By substituting the equations (3.6-3.9) in the system of equations (3.2-3.5), we get,

$$\frac{dP_S}{dt} = \Pi - (\mu + \gamma + \theta P_I)P_S, \quad (3.11)$$

$$\frac{dP_V}{dt} = \gamma P_S - (\alpha P_I + \tau + \mu)P_V, \quad (3.12)$$

$$\frac{dP_I}{dt} = \theta P_I P_S + \alpha P_I P_V - (\beta + \mu)P_I, \quad (3.13)$$

$$\frac{dP_R}{dt} = \beta P_I + \tau P_V - \mu P_R, \quad (3.14)$$

$$\frac{dP_S}{dt} + \frac{dP_V}{dt} + \frac{dP_I}{dt} + \frac{dP_R}{dt} = 0. \quad (3.15)$$

Definition 3.1

SVIR model can have an equilibrium point E^*, if $E^* = ({P_S}^*, {P_V}^*, {P_I}^*, {P_R}^*)$, satisfies:

$$\begin{cases} \frac{dP_S}{dt} = 0, \\ \frac{dP_V}{dt} = 0, \\ \frac{dP_I}{dt} = 0. \end{cases}$$

To determine the equilibrium points of the model, first the system of non-linear equations must be linearized, the three equations of the reduced system are 3.11, 3.12 and 3.13, each of them is equal to zero, to get,

$$\frac{dP_S}{dt} = \Pi - (\mu + \gamma + \theta P_I)P_S = 0, \quad (3.16)$$

$$\frac{dP_V}{dt} = \gamma P_S - (\alpha P_I + \tau + \mu)P_V = 0, \quad (3.17)$$

$$\frac{dP_I}{dt} = \theta P_I P_S + \alpha P_I P_V - (\beta + \mu)P_I = 0. \quad (3.18)$$

There are two equilibrium points of this SVIR model system:

1. E_0, the disease-free equilibrium point (DFE) and that when $P_I = 0$.
2. E^*, the endemic equilibrium point when $P_I > 0$.

Now let assume that the infection is not existing and substitute $P_I = 0$, $and\ P_R = 0$ in equation (3.16) and (3.17) to get,

$$P_S = \frac{\Pi}{(\mu+\gamma)}, \tag{3.19}$$

$$P_V = \frac{\Pi\gamma}{(\mu+\gamma)(\tau+\mu)}, \tag{3.20}$$

therefore, the infection free equilibrium or (DFE) point is

$$E_0 = \left(P_{S_0}, P_{V_0}, P_{I_0}, P_{R_0}\right) = \left(\frac{\Pi}{(\mu+\gamma)}, \frac{\Pi\gamma}{(\mu+\gamma)(\tau+\mu)}, 0,0\right). \tag{3.21}$$

To find the second equilibrium point for the system while assuming the existence of the infection, equation (3.16) will be used and solved for P_S to get,

$$P_S = \frac{\Pi}{\mu+\gamma+\theta {P_I}^*}, \tag{3.22}$$

now by solving (3.17) for P_V to get,

$$P_V = \frac{\gamma\Pi}{(\alpha {P_I}^*+\tau+\mu)(\mu+\gamma+\theta {P_I}^*)}, \tag{3.23}$$

by substituting (3.22) and (3.23) in (3.18), we get,

$$\frac{\theta\Pi}{\mu+\gamma+\theta {P_I}^*} + \frac{\Pi\alpha\gamma}{(\mu+\gamma+\theta {P_I}^*)(\alpha {P_I}^*+\tau+\mu)} - \mu - \beta = 0, \tag{3.24}$$

then by rearranging equation 3.24 in a quadratic equation form, we get,

$$C_1 {P_I}^{*2} + C_2 {P_I}^* + C_3(1-k) = 0, \tag{3.25}$$

where,

$$C_1 = \alpha\theta(\mu + \beta) > 0,$$
$$C_2 = (\mu + \beta)[\alpha(\mu + \gamma) + \theta(\tau + \mu)] - \alpha\theta\mu,$$
$$C_3 = (\mu + \beta)(\mu + \gamma)(\tau + \mu) > 0,$$
$$k = \frac{\theta\Pi}{(\mu+\beta)(\mu+\gamma)} + \frac{\Pi\alpha\gamma}{(\mu+\beta)(\mu+\gamma)(\tau+\mu)},$$

now after finding the roots of 3.25, we have,

$$P_I{}^*{}_{1,2} = \frac{-C_2 \pm \sqrt{C_2{}^2 - 4C_1C_3(1-k)}}{2C_1},$$

it is clear that $P_I{}^* > 0$, and $k > 1$.

Thus, the second equilibrium point which is called the Endemic equilibrium point of the system when the infection is existing is,

$$E^* = (P_S{}^*, P_V{}^*, P_I{}^*, P_R{}^*) = \left(\frac{\Pi}{\mu+\gamma+\theta P_I{}^*}, \frac{\gamma\Pi}{(\alpha P_I{}^* + \tau + \mu)(\mu+\gamma+\theta P_I{}^*)}, P_I{}^*, 0\right). \quad (3.26)$$

3.5 Basic Reproduction Rate

The basic reproduction number (R_0) can be explained as the estimated number of individuals of secondary infections caused by a particular individual in a population that is totally susceptible. (R_0) can be a good indicator for the probability that a certain infectious disease will spread through a population causing a pandemic or not (Van den Driessche, & Watmough, 2008).

If $(R_0 > 1)$, it means that the infectious disease could attack causing a pandemic but if $R_0 < 1$, it couldn't (Diekmann, et al., 1990). Awareness of the overall epidemiological studies of infectious diseases and the effects of prevention efforts requires an understanding of the basic reproduction rate, or (R_0) which it should be decreased to be less than unity in order to get rid of an infection (Anderson, & May,1982).

Table 3.3 lists a few approximated basic reproduction rates. Diverse populations of individuals could be related to various values of (R_0) for the same kind of infection because of variations in population size rates, and the effect of the environment, the surroundings, and the contact structure.

Table 3.3 Estimated R_0 for infectious diseases (Rodrigues, 2012).

Infectious disease	**Estimated(R_0)**
Influenza	3-4
Foot and mouth disease	3.5-4.5
Smallpox	3.5-6
Rubella	6-7
Dengue	1.3-11.6
Chickenpox	10-12
Measles	16-18

3.5.1 Next Generation Matrix

The method used to calculate the basic reproduction number is the Next Generation matrix (Diekmann, Heesterbeek, & Roberts, 2010). This method described any nonlinear system of ODE as,

$$x_i = f_i = F_i(x) - V_i(x),$$

and V_i can be expressed as

$$V_i = V_i(out) - V_i(in),$$

where F_I is the rate of the new infection occurrence into the i^{th} compartment, $V_i(in)$ is the rate of input individuals transfer to i^{th} compartment, $V_i(out)$ is the rate of the output of i^{th} compartment, and if $F_i(x)$ is set to zero, then all eigenvalues of the derivatives of $f(x_0)$, have negative real parts.

Lemma 3.2 (Driesschea, & Wamough, 2002): If E_0 is a disease-free equilibrium point, then the derivatives of $F(x_0)$, and the derivatives of $V(x_0)$, can be expressed as:

$$F = \left[\frac{\partial f_i}{\partial x_j}(x_0)\right], \text{ and } V = \left[\frac{\partial V_i}{\partial x_j}(x_0)\right], 1 \leq i, j \leq m.$$

Thus, the derivation of basic reproduction number depends on the linearization of the ordinary differential equations of the SVIR model. By applying lemma 3.2 on the system of equations (3.16) - (3.18), we get,

$$F = \begin{bmatrix} \theta P_{S_0} + \alpha P_{V_0} & 0 \\ 0 & 0 \end{bmatrix},$$

$$V = \begin{bmatrix} \beta + \mu & 0 \\ 0 & \alpha P_I + \tau + \mu \end{bmatrix},$$

$$V^{-1} = \begin{bmatrix} \frac{1}{\beta+\mu} & 0 \\ 0 & \frac{1}{\alpha P_I+\tau+\mu} \end{bmatrix},$$

$$FV^{-1} = \begin{bmatrix} \frac{\theta P_{S_0}+\alpha P_{V_0}}{\beta+\mu} & 0 \\ 0 & 0 \end{bmatrix},$$

we get two eigenvalues of the matrix FV^{-1}, which are (0, $\frac{\theta P_{S_0}+\alpha P_{V_0}}{\beta+\mu}$) .

Definition 3.3 (Burden, Faires, & Burden, 2015).

A matrix A has a spectral radius $\rho(A)$ which is determined by the formula $\rho(A) = max|\lambda|$, where λ is the calculated eigenvalue of A.

Thus, using definition 3.3 the basic reproduction number is

$$R_0 = \rho[FV^{-1}] = \frac{\theta P_{S_0}+\alpha P_{V_0}}{\beta+\mu}, \qquad (3.27)$$

now by substituting P_{S_0} and P_{V_0} in equation 3.27 we get,

$$R_0 = \frac{\Pi\theta}{(\mu+\beta)(\mu+\gamma)} + \frac{\Pi\alpha\gamma}{(\mu+\beta)(\mu+\gamma)(\tau+\mu)} = \frac{\Pi\theta(\tau+\mu)+\Pi\alpha\gamma}{(\mu+\beta)(\mu+\gamma)(\tau+\mu)}, \qquad (3.28)$$

which is the same value of k in 3.25, and that makes sense, if Ro$<$ 1, then it is free disease stage and the infectious disease is under control, while if the value of $R_0 > 1$, then the disease is spread and the pandemic is starting.

3.6 Existence and Uniqueness

It is vital to demonstrate the existence and uniqueness of solutions for the SVIR model. Researchers can prove the model's reliability, validate its assumptions, and enable detailed investigation and prediction of infection dynamics among a population by showing the existence and uniqueness of solutions for the SVIR model, and provide a solid mathematical foundation for studying the model. Take the first-order ordinary differential equation in the form:

$$x` = f(t,x), \qquad x(t_0) = x_0, \tag{3.29}$$

for equation (3.29),

i- When the solution of equation (3.29) exists?

ii- When there is a unique solution to equation (3.29)?

Assume the following:

$f_1 = \Pi - (\mu + \gamma + \theta P_I)P_S$,

$f_2 = \gamma P_S - (\alpha P_I + \tau + \mu)P_V$,

$f_3 = \theta P_I P_S + \alpha P_I P_V - (\beta + \mu)P_I$, and

$f_4 = \beta P_I + \tau P_V - \mu P_R$,

now to show the existence and the uniqueness of the SVIR model, we have two theorems (Sowole, et. Al., 2019).

Theorem 3.3 (Uniqueness of the Solution)

Assuming that D represent the domain, and $f(t,x)$ satisfies the Lipschitz condition, then

$$|t - t_0| \leq a\ , \|x - x_0 \leq b\ \|, x = (x_1, x_2, \dots, x_n), x_0 = (x_{10}, x_{20}, \dots, x_{n0}), \tag{3.30}$$

and,

$$\|f(t,x_1) - f(t,x_2)\| \leq k\|x_1 - x_2\ \|, \tag{3.31}$$

If $f(t, x_1)$ and $f(t, x_2)$ are in the domain D, and k is a positive constant, then, there exists a constant $\sigma > 0$, such that a unique vector solution $x(t)$ exists for the system 3.29 in the interval $|t - t_0| \leq \sigma$, where the inequality (3.31) satisfies if,

$$\begin{cases} \frac{\partial f_i}{\partial x_j}, & i, j = 1,2, \dots, n \end{cases}$$

is continuous and bounded in the domain D

Lemma 3.4

If $f(t, x)$ has continuous partial derivative $\frac{\partial f_i}{\partial x_j}$ on a bounded closed convex domain of real number R, then it satisfies a Lipschitz condition in R. Assume the domain is,

$$1 \leq \sigma \leq R, \tag{3.32}$$

then, the proof will be completed if a bounded solution found in a form of,

$$0 < R < \infty.$$

Theorem 3.5 (Existence of the Solution)

Let D denote the domain defined in (3.30) such that (3.31) and (3.32) hold. Then there exist a solution of model system of equations (3.11)-(3.14) which is bounded in the domain D.

Proof:

Let,

$$f_1 = \Pi - (\mu + \gamma + \theta P_I) P_S, \tag{3.33}$$

$$f_2 = \gamma P_S - (\alpha P_I + \tau + \mu) P_V, \tag{3.34}$$

$$f_3 = \theta P_I P_S + \alpha P_I P_V - (\beta + \mu) P_I \text{ , and} \tag{3.35}$$

$$f_4 = \beta P_I + \tau P_V - \mu P_R, \tag{3.36}$$

to prove that $\frac{\partial f_i}{\partial x_j}$ are continuous and bounded, the partial derivatives for every equation should be obtained.

Starting with equation (3.33),

$$\frac{\partial f_1}{\partial P_S} = -(\mu + \gamma + \theta P_I), \qquad \left|\frac{\partial f_1}{\partial P_S}\right| = |-(\mu + \gamma + \theta P_I)| < \infty,$$

$$\frac{\partial f_1}{\partial P_V} = 0, \qquad \left|\frac{\partial f_1}{\partial P_V}\right| = |0| < \infty,$$

$$\frac{\partial f_1}{\partial P_I} = -\theta, \qquad \left|\frac{\partial f_1}{\partial P_V}\right| = |-\theta| < \infty,$$

$$\frac{\partial f_1}{\partial P_R} = 0, \qquad \left|\frac{\partial f_1}{\partial P_R}\right| = |0| < \infty.$$

Similarly, from equation 3.34, we get,

$$\frac{\partial f_2}{\partial P_S} = \gamma, \qquad \left|\frac{\partial f_2}{\partial P_S}\right| = |\gamma| < \infty,$$

$$\frac{\partial f_2}{\partial P_V} = -(\alpha P_I + \tau + \mu), \qquad \left|\frac{\partial f_2}{\partial P_V}\right| = |-(\alpha P_I + \tau + \mu)| < \infty,$$

$$\frac{\partial f_2}{\partial P_I} = -\alpha, \qquad \left|\frac{\partial f_2}{\partial P_I}\right| = |-\alpha| < \infty,$$

$$\frac{\partial f_2}{\partial P_R} = 0, \qquad \left|\frac{\partial f_2}{\partial P_R}\right| = |0| < \infty.$$

Now, from equation 3.35, we get,

$$\frac{\partial f_3}{\partial P_S} = \theta P_I, \qquad \left|\frac{\partial f_3}{\partial P_S}\right| = |\theta P_I| < \infty,$$

$$\frac{\partial f_3}{\partial P_V} = \alpha P_I, \qquad \left|\frac{\partial f_3}{\partial P_V}\right| = |\alpha P_I| < \infty,$$

$$\frac{\partial f_3}{\partial P_I} = \theta P_S + \alpha P_V + -(\beta + \mu)\,, \qquad \left|\frac{\partial f_3}{\partial P_I}\right| = |\theta P_S + \alpha P_V + -(\beta + \mu)\,| < \infty,$$

$$\frac{\partial f_3}{\partial P_R} = 0, \qquad \left|\frac{\partial f_3}{\partial P_R}\right| = |0| < \infty.$$

Last one, from equation 3.36, we get,

$$\frac{\partial f_4}{\partial P_S} = 0, \qquad \left|\frac{\partial f_4}{\partial P_S}\right| = |0| < \infty,$$
$$\frac{\partial f_4}{\partial P_V} = \tau, \qquad \left|\frac{\partial f_4}{\partial P_V}\right| = |\tau| < \infty,$$
$$\frac{\partial f_4}{\partial P_I} = \beta, \qquad \left|\frac{\partial f_4}{\partial P_I}\right| = |\beta| < \infty,$$
$$\frac{\partial f_4}{\partial P_R} = -\mu, \qquad \left|\frac{\partial f_4}{\partial P_R}\right| = |-\mu| < \infty.$$

Thus, by using theorem 3.3, it is clearly established that all these partial derivatives are continuous and bounded, hence, by Theorem (3.3), In the domain D, there exists a unique solution of the model system of equations (3.11) - (3.14).

3.7 The Stability Analysis of the Model

The stability of equilibrium points must be tested since it allows us to understand the disease's long-term behaviour and forecast how it will spread in a community. We can predict whether the infection will die out or continue by studying the stability, as well as how the different compartments (S, V, I, R) change through time.

To study the stability of the two equilibrium points of the SVIR model which has nonlinear differential equations, we have to define the method that we will use. One of the methods is Hartman Grobman method (linearization method).

Definition 3.6 *(Sastry, 2013)*

Assume that $f: R^n \to R^n$ is a map that has m which is a point that
$f(m) = 0,$
where m is a fixed point for the ordinary differential equation
$\frac{dx}{dt} = f\big(x(t)\big),$
While $Df(m)$, is the partial derivatives matrix at m,
For $x \epsilon R^n$

$$Df(m) = \begin{bmatrix} \frac{\partial f_1}{\partial x_1}(m) & \frac{\partial f_1}{\partial x_2}(m) & \dots & \frac{\partial f_1}{\partial x_n}(m) \\ \frac{\partial f_2}{\partial x_1}(m) & \frac{\partial f_2}{\partial x_2}(m) & \dots & \frac{\partial f_2}{\partial x_n}(m) \\ \vdots & \vdots & \vdots & \vdots \\ \vdots & \vdots & \dots & \vdots \\ \frac{\partial f_n}{\partial x_1}(m) & \frac{\partial f_n}{\partial x_2}(m) & & \frac{\partial f_n}{\partial x_n}(m) \end{bmatrix}.$$

Definition 3.7

Hartman Grobman theorem said that if the result of the linearization of the equations has no zero or imaginary eigenvalues, at that point there is a continuous function with a continuous inverse in the neighbourhood of this point into R^n.

Thus, we can write Jacobian matrix for the model (P_S, P_V, P_I) to be

$$J = \begin{bmatrix} -(\mu+\gamma+\theta P_I) & 0 & -\theta P_S \\ \gamma & -(\alpha P_I+\tau+\mu) & -\alpha P_V \\ \theta P_I & \alpha P_I & \theta P_S+\alpha P_V-\beta-\mu \end{bmatrix}. \quad (3.37)$$

3.7.1 Local Stability of DFE

Now apply this matrix to the first equilibrium point which is the disease-free equilibrium point (DFE) from equation 3.21 to have,

$$J = \begin{bmatrix} -(\mu+\gamma) & 0 & -\theta P_S \\ \gamma & -(\tau+\mu) & -\alpha P_V \\ 0 & 0 & \theta P_S+\alpha P_V-\beta-\mu \end{bmatrix}. \quad (3.38)$$

Theorem 3.8

To prove the stability of DFE point we should prove that the eigenvalues of the Jacobian matrix have a negative real part, then we can say that the point is locally asymptotically stable (Hethcote, & van den Driessche,1991).

Using Maple software, we found 3 eigenvalues of DFE, which are,

$$\lambda_1 = -(\tau + \mu),$$
$$\lambda_2 = -(\mu + \gamma),$$
$$\lambda_3 = \theta P_S + \alpha P_V - \beta - \mu,$$

using theorem 3.8, it is clear that λ_1 and λ_2 have negative real values as we used nonnegative parameters. Now we have to check λ_3 and check if it is negative, it means our DFE is locally stable.

$$\theta P_S + \alpha P_V - \beta - \mu < 0,$$

$$\theta P_S + \alpha P_V < \beta + \mu,$$

$$\frac{\theta P_S + \alpha P_V}{\beta + \mu} < 1,$$

which is the same value of Ro, that means,Ro < 1 , which is true in case of disease-free equilibrium point, then we can say that our system is locally stable when $P_I = 0$.While if Ro > 1, then λ_3 is positive and that means E_0 is unatable.

3.7.2 Local Stability of the Endemic Equilibrium Point

Now apply this matrix to the second equilibrium point which is called the endemic equilibrium point of the system from equation 3.25 to have,

$$J = \begin{bmatrix} -(\mu + \gamma + \theta P_I^*) & 0 & -\theta P_S \\ \gamma & -(\alpha P_I^* + \tau + \mu) & -\alpha P_V \\ \theta P_I^* & \alpha P_I^* & \theta P_S + \alpha P_V - \beta - \mu \end{bmatrix}, \quad (3.39)$$

now, if we rearrange the parameters elements in a_{ii} in the matrix (3.39), (Kaymakamzade, Baba, & Hincal, 2016), we will get the following:

$$-(\mu + \gamma + \theta P_I) = \frac{-\mu}{P_S{}^*},$$

$$-\left(\alpha P_I{}^* + \tau + \mu\right) = \frac{-\gamma P_S{}^*}{P_V{}^*},$$

and from equation 3.18, we can conclude that $\theta P_S + \alpha P_V - \beta - \mu = 0$.

Thus, we get,

$$J = \begin{bmatrix} \frac{-\mu}{P_S{}^*} & 0 & -\theta P_S{}^* \\ \gamma & \frac{-\gamma P_S{}^*}{P_V{}^*} & -\alpha P_V{}^* \\ \theta P_I{}^* & \alpha P_I{}^* & 0 \end{bmatrix}. \tag{3.40}$$

Theorem 3.6 (Routh-Hurwitz stability criterion)

From the characteristic polynomial of the matrix,

$q(\lambda) = \lambda^m + a_1\lambda^{m-1} + \cdots + a_m$, where a_i represents all the real coefficients for all $i = 1,2,3,\ldots,m$. The roots found for this polynomial when $q(\lambda) = 0$ all are negative or have negative real part if and only if all the determinants of all Routh-Hurwitz matrices are nonnegative. (Islam, Asaduzzaman, & Mondal, 2014).

Now using theorem 3.6 and following Routh- Hurwitz stability equation we have the following results:

The characteristic equation for (3.40) matrix is,

$$\lambda^3 + b_1\lambda^2 + b_2\lambda + b_3 = 0,$$

where,

$$b_1 = \frac{\mu}{P_S{}^*} + \frac{\gamma P_S{}^*}{P_V{}^*} > 0,$$

$$b_2 = \frac{\gamma P_S{}^*}{P_V{}^*} + \alpha^2 P_V{}^* P_I{}^* + \theta^2 P_S{}^* P_I{}^* > 0,$$

$$b_3 = \gamma\alpha\theta P_S{}^* P_I{}^* + \frac{\gamma\theta^2 P_I{}^* P_S{}^{*2}}{P_V{}^*} + \frac{\mu\alpha^2 P_V{}^* P_I{}^*}{P_S{}^*} > 0,$$

now, if $b_1 b_2 - b_3 > 0$, then the endemic equilibrium point is locally stable as Routh-Hurwitz stability criterion said, to check that we substitute in the last inequality to have,

$$\frac{\gamma\mu^2}{P_S^* P_V^*} + (\mu + \theta P_I^*)\theta^2 P_I^* P_S^* + \frac{\gamma^2 \mu P_S^*}{P_V^{*2}} + \alpha P_I^* P_S^* (\theta - \alpha)^2 + \gamma\alpha\theta P_S^* P_I^* > 0.$$

As a result of applying Routh-Hurwitz stability criterion, it is clear that all the eigenvalues of the matrix are negative and we can conclude that the endemic equilibrium point is locally stable.

3.8 Sensitivity Analysis of R_0

Sensitivity analysis of R_0 in SVIR model is examining the effect of varying the basic reproduction number (R_0) on the dynamics and outcomes of the model. The basic reproduction number's sensitivity is the key to understand the essential parameters employed throughout the model. The calculation of R_0 was shown in previous Section. The calculation of the indices of normalized local sensitivity of R_0 proportional to the parameters that occurred in it's formula. In this model, the parameters that related to R_0 are $\lambda = \{\Pi, \theta, \beta, \gamma, \tau, \mu, \alpha\}$.
We can write R_0 by using the normalized local sensitivity index according to a certain parameter found in λ , where λ can be expressed as $\Omega_\lambda^{R0} = \frac{\partial \boldsymbol{R_0}}{\partial \lambda}\frac{\lambda}{\boldsymbol{R_0}}$. Now by applying this new difinition, we can calculate the indices below using table 3.1 and that will give R_0 according to each parameter occured in. (Bagkur, et. Al., 2022).

$$\Omega_{\theta}^{R_0} = \frac{\partial \boldsymbol{R_0}}{\partial \theta}\frac{\theta}{\boldsymbol{R_0}} = \frac{\Pi}{(\mu+\beta)(\mu+\gamma)}\frac{\theta(\mu+\beta)(\mu+\gamma)(\tau+\mu)}{\theta\prod(\tau+\mu)+\Pi\alpha\gamma} = \frac{\theta(\tau+\mu)}{\theta(\tau+\mu)+\alpha\gamma}, \quad (3.41)$$

$$\Omega_{\beta}^{\boldsymbol{R_0}} = \frac{\partial \boldsymbol{R_0}}{\partial \beta}\frac{\beta}{\boldsymbol{R_0}} = \frac{-[\theta\Pi(\tau+\mu)+\Pi\alpha\gamma](\mu+\gamma)(\tau+\mu)}{[(\mu+\gamma)(\tau+\mu))(\beta+\mu)]^2}\frac{\beta(\mu+\beta)(\mu+\gamma)(\tau+\mu)}{\theta\Pi(\tau+\mu)+\Pi\alpha\gamma} = \frac{-\beta}{(\beta+\mu)}, \quad (3.42)$$

$$\Omega_{\alpha}^{\boldsymbol{R_0}} = \frac{\partial \boldsymbol{R_0}}{\partial \alpha}\frac{\alpha}{\boldsymbol{R_0}} = \frac{(\gamma\Pi)}{(\mu+\beta)(\mu+\gamma)(\tau+\mu)}\frac{\alpha(\mu+\beta)(\mu+\gamma)(\tau+\mu)}{\theta\Pi(\tau+\mu)+\Pi\alpha\gamma} = \frac{\alpha\gamma}{\theta(\tau+\mu)+\alpha\gamma}, \quad (3.43)$$

$$\Omega_{\mu}^{\boldsymbol{R_0}} = \frac{\partial \boldsymbol{R_0}}{\partial \mu}\frac{\mu}{\boldsymbol{R_0}} = \frac{[\theta\tau+2\theta\mu+\alpha\gamma][(\mu+\beta)(\mu+\gamma)(\tau+\mu)]+[-2\tau\mu-\tau\gamma-\beta\tau-3\mu^2-2\mu\gamma-2\mu\beta-\beta\gamma][\theta\mu(\tau+\mu)+\mu\alpha\gamma]}{(\mu+\beta)(\mu+\gamma)(\tau+\mu)[\theta(\tau+\mu)+\gamma\alpha]},$$

(3.44)

$$\Omega_{\tau}^{\boldsymbol{R_0}} = \frac{\partial \boldsymbol{R_0}}{\partial \tau}\frac{\tau}{\boldsymbol{R_0}} = \frac{-\tau\alpha\gamma}{(\tau+\mu)[\theta(\tau+\mu)+\alpha\gamma]}, \quad (3.45)$$

$$\Omega_{\gamma}^{\boldsymbol{R_0}} = \frac{\partial \boldsymbol{R_0}}{\partial \gamma}\frac{\gamma}{\boldsymbol{R_0}} = \frac{\gamma[\alpha(\mu+\gamma)-\theta(\tau+\mu)-\gamma\alpha]}{(\mu+\gamma)[\theta(\tau+\mu)+\alpha\gamma]}, \quad (3.46)$$

$$\Omega_{\Pi}^{R_0} = \frac{\partial \boldsymbol{R_0}}{\partial \theta}\frac{\Pi}{\boldsymbol{R_0}} = \frac{\theta(\tau+\mu)+\alpha\gamma}{(\mu+\beta)(\mu+\gamma)(\tau+\mu)}\frac{\Pi(\mu+\beta)(\mu+\gamma)(\tau+\mu)}{\theta\prod(\tau+\mu)+\Pi\alpha\gamma} = 1. \quad (3.47)$$

CHAPTER IV

Findings and Discussion

Numerical Simulation of the model

In the model simulation we will use table 3.1 which included parameter values for the system of equations of the SVIR model in (3.11) − (3.14) taking under consideration the situation of infection in Jordan, and how the individuals transfer from susciptible, vaccinated, or infected to recovered.

In this model we stareted assuming that the population converts from being free of disease to the situation of endemicity.

Table 4.1 The values of the Parameters of the Model

Parameters	Descriptions	Value
θ	The contact rate (transmission rate)	0.15
β	The recovery rate	1/14
Π	The birth rate	0.024
μ	The natural death rate	0.003916
γ	The rate that susceptible persons become vaccinated	0.05
α	The rate that vaccinated persons become susceptible because of vaccination failure	0.08
τ	The effect rate of vaccine in protecting from the infection(get immunity)	0.92

4.1 R_0 for Varicella Virus in Jordan

The basic reproduction number R_0 is created by using next generation matrix as shown in the previous section, which was our indicator to check the infectious disease potential, as we mentioned if $R_0 < 1$, that means the infectious disease is vanishing, while if $R_0 > 1$, this means that it is spreading. By using the equation 3.27, we can calculate R_0 for the varicella virus in Jordan yearly depending on the number of real cases in table 1.1 to get,

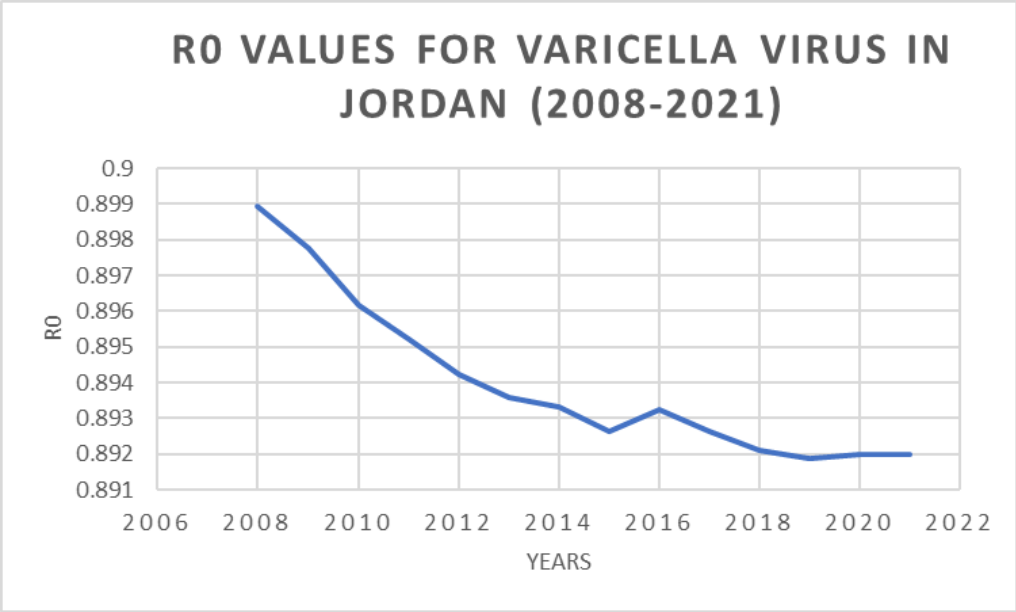

Figure 4.1: R_0 values for Varicella virus in Jordan from 2008 to 2021

In figure 4.1 summarized the situation in Jordan, and it is obvious from this graph that since 2016 there is a big decreasing in R_0 and the cases became under control and that when Jordanian health officials discussed their intended approach for fighting infectious diseases from 2016 to 2020 in public, with a plan to add additional vaccinations like varicella virus vaccination to the Jordan's national immunization program (Abdullat, et al., 2021). The severe viral infection, Varicella Virus, is still very contagious. The number of cases and the statistical data of varicella in Jordan are not clearly available. Even though estimates suggest that varicella infection likely represents a sizable health and financial burden in the country that could be reduced through universal varicella vaccination, unfortunately, the varicella vaccine remains absent in Jordan's vaccination program and it is not mandatory.

4.2 Simulating SVIR model

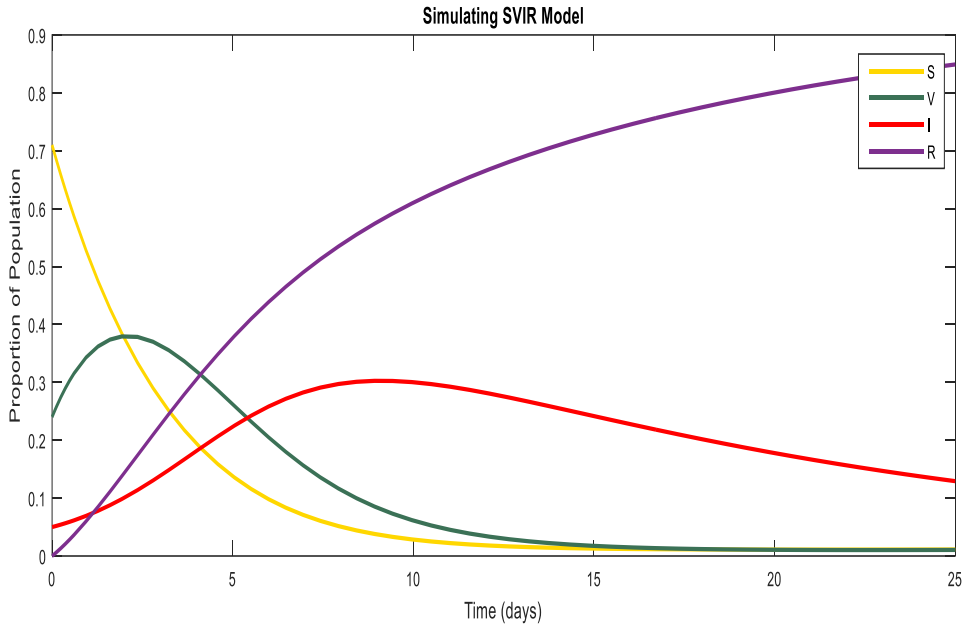

Figure 4.2: The dynamics of the various compartments during the outbreak with $P_{V_0} = 0.24$

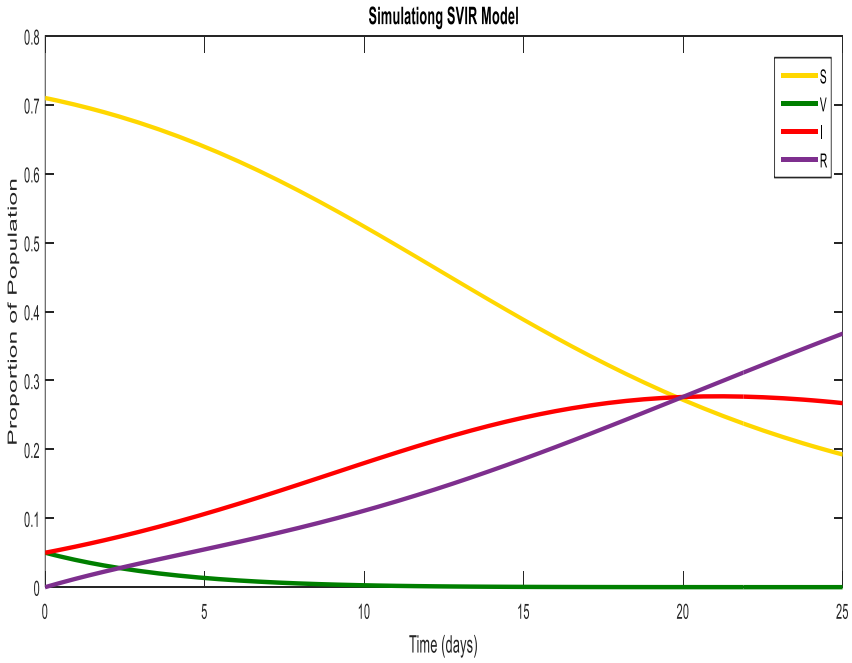

Figure 4.3: The dynamics of the various compartments during the outbreak with $P_{V_0} = 0.05$

Figure 4.2, shows that when the starting percentage of infected individuals is 0.05, and the vaccination percentage is 0.24 It is clear that the susceptible individuals' percentage starts decreasing with time as we take the initial value is 0.71, and the infected ratio started to increase with time and the individuals transfer to recovery compartment with the time. While, in figure 4.3 the vaccination percentage decreases to 0.05, keeping the same percentage of infected individuals. As a result, the infected cases increase rapidly with time comparing to graph 4.2. Individuals keep moving from the infected compartment to the recovered, and that causes the increase in the recovered individuals. As noticed from both graphs, the transmission of the virus can be reduced by vaccination susceptible individuals, lowering the overall illness burden. Vaccination protects people from becoming infected with varicella virus and causing health problems. Varicella vaccination provides lasting immunity, lowering the possibility of outbreaks and promoting a healthier community. By decreasing the total burden of varicella virus infection, medical funding can be better directed to other health challenges.

4.3 Simulating the Sensitivity of R_0

For the purpose of demonstrating how every single parameter affect R_0, we will use the local sensitivity analysis. The sensitivity values for every parameter can be determined by substituting the values from table 3.1 in the equations from 3.41 to 3.47. Table 4.2 displays the computed sensitivity values.

Table 4.2 The sensitivity values for each parameter in R_0

Sensitivity	Sinsitivity Values
$\Omega_{\theta}^{\boldsymbol{R_0}}$	0.972
$\Omega_{\beta}^{\boldsymbol{R_0}}$	-0.948
$\Omega_{\alpha}^{\boldsymbol{R_0}}$	0.0281
$\Omega_{\prod}^{\boldsymbol{R_0}}$	1.000
$\Omega_{\mu}^{\boldsymbol{R_0}}$	-0.021
$\Omega_{\tau}^{\boldsymbol{R_0}}$	-0.028
$\Omega_{\gamma}^{\boldsymbol{R_0}}$	-0.899

From table 4.2 we can decide the effect of each parameter on R_0, starting with the transmission rate θ, the value demonstrate that if it increased by for example 10% that will make R_0 to increase with percentage of 9.72%.

While for the recovery rate β and the rate of vaccination for the susceptible γ the situation is different, here it is noticeable that the increase in β and γ by 10% will make R_0 decrease by 9.48%, and 8.99% respectively and that makes sense.

For the parameter α, which is the rate that vaccinated persons become infected because of vaccination failure, if it increases by 10%, that will make R_0 to increase by 0.281%, while the opposite for τ which is the rate of getting immunity from the virus by vaccination, if it increases by 10%, $\boldsymbol{R_0}$ will decrease by 0.28%.

If $\boldsymbol{\mu}$ which is the death rate increased by 10% that will decrease R_0 by 0.21%, but, if the birth rate increases by 10%, that will increase $\boldsymbol{R_0}$by 10%. The parameters are graphically illustrated in the following figures (4.4) - (4.10).

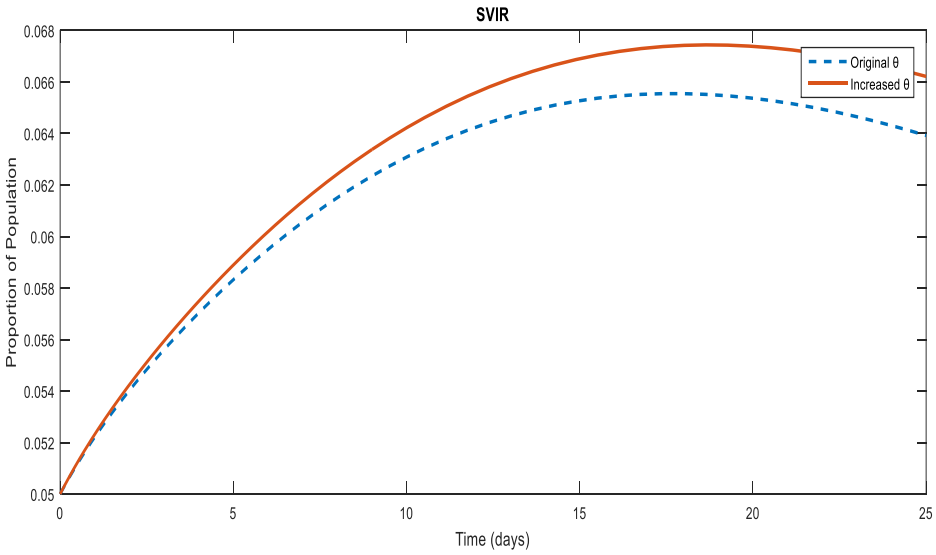

Figure 4.4: Proportion of Population that infected with Varicella Virus estimated by the model with the value of the original θ in Table 4.2 and with increasing of 10% in θ parameter.

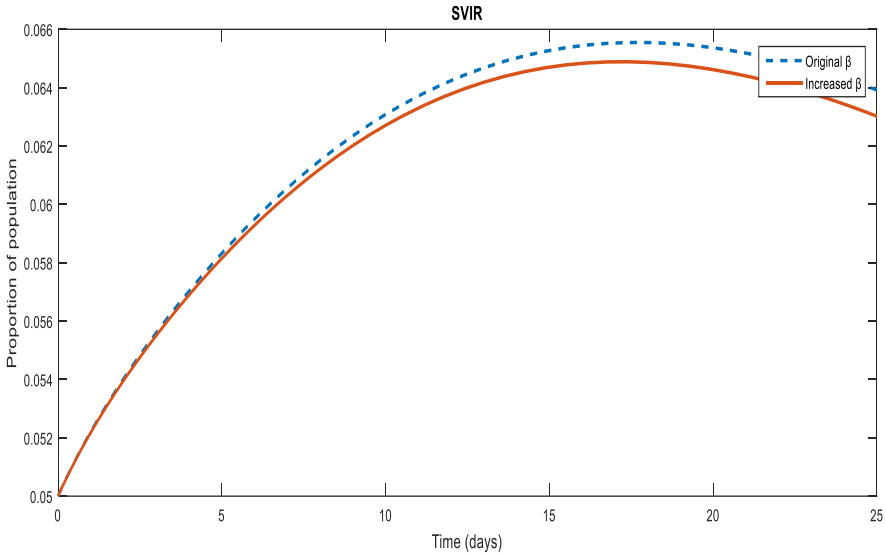

Figure 4.5: Proportion of Population that infected with Varicella Virus estimated by the model with the value of the original β in Table 4.2 and with increasing of 10% in β parameter.

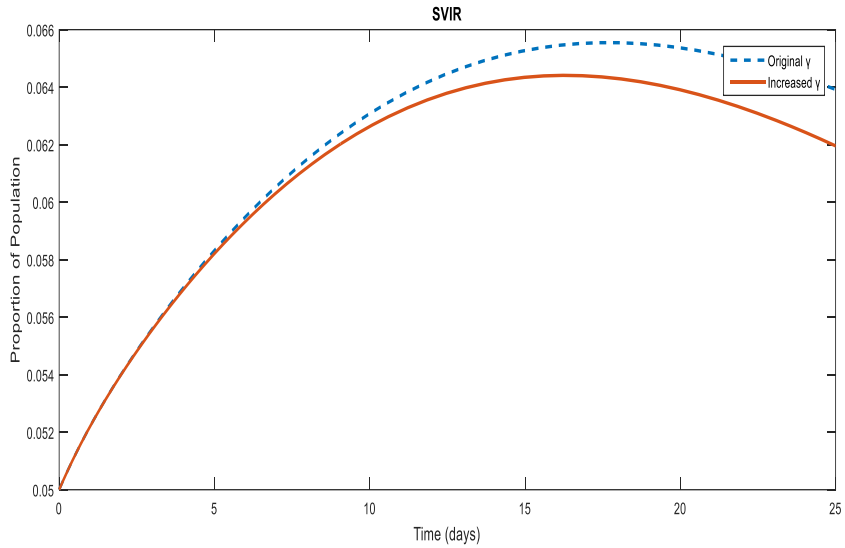

Figure 4.6: Proportion of Population that infected with Varicella Virus estimated by the model with the value of the original γ in Table 4.2 and with increasing of 10% in γ parameter.

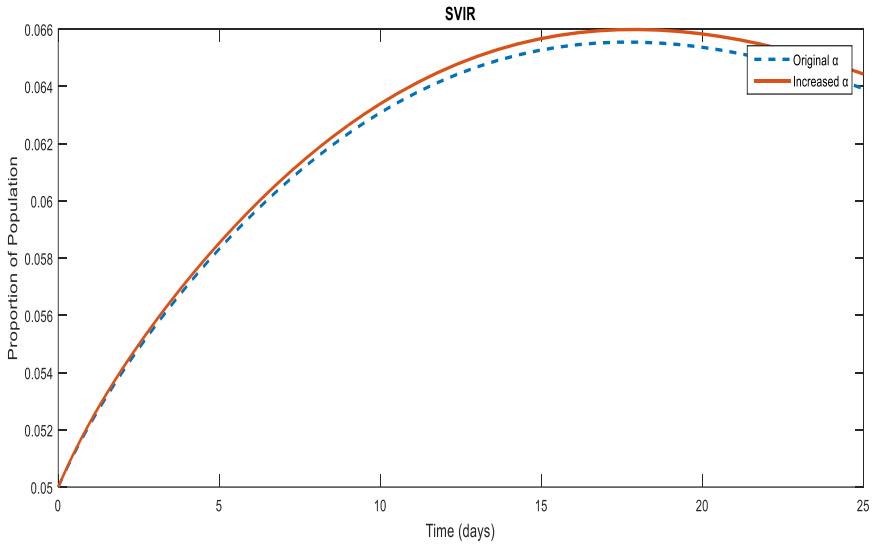

Figure 4.7: Proportion of Population that infected with Varicella Virus estimated by the model with the value of the original α in Table 4.2 and with increasing of 10% in α parameter.

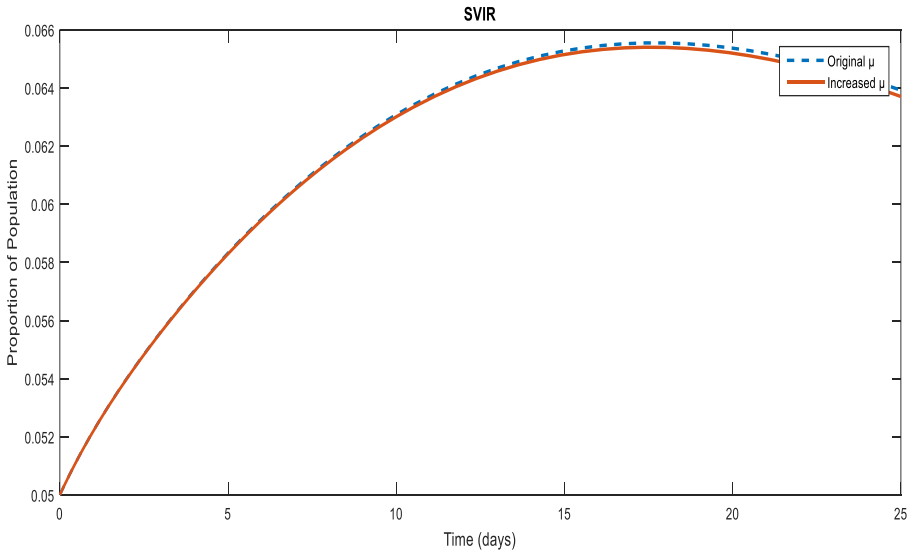

Figure 4.8: Proportion of Population that infected with Varicella Virus estimated by the model with the value of the original μ in Table 4.2 and with increasing of 10% in μ parameter.

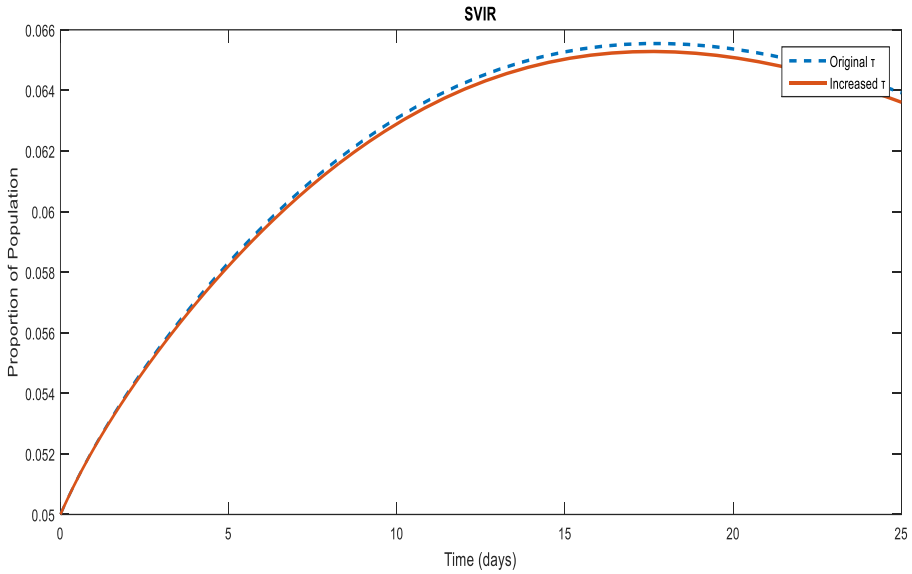

Figure 4.9: Proportion of Population that infected with Varicella Virus estimated by the model with the value of the original τ in Table 4.2 and with increasing of 10% in τ parameter.

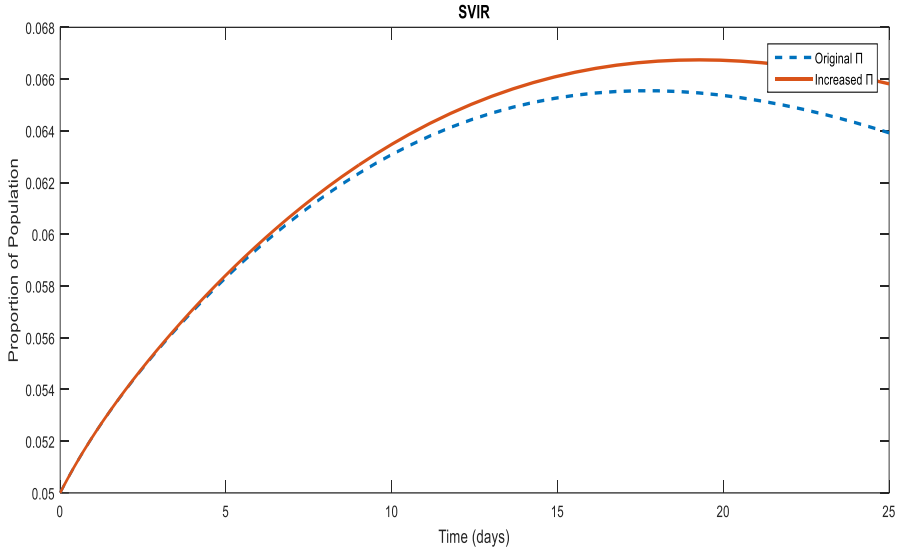

Figure 4.10: Proportion of Population that infected with Varicella Virus estimated by the model with the value of the original Π in Table 4.1 and with increasing of 10% in Π parameter.

As noted from the previous graphs, we studied the effect of each parameter on the infection proportion of population, and the results from the simulation of the system gave the same result as the calculated ones in table 4.2. starting from the effect of increasing the contact rate by 10%, from the graph, we can notice that the infection increases by this change in figure 4.4, also if the failure of vaccination increases that will increase the infection as shown in figure 4.7.

In the other hand if the recovery rate and the vaccination rate increase that will decrease the infection transmission between individuals as shown in figure 4.5 and 4.6. In addition to these two parameters, we have in figure 4.9, the graph that clarifies if the vaccination successes to give immunity and the rate of transfer from vaccination compartment to recovery compartment increases that will decrease the infection between individual.

By evaluating the effects of parameter changes on the model's results and offering opinions on the robustness and reliability of the model, sensitivity analysis performs an important role in this model for the SVIR of varicella virus. One of these roles is locating the parameter which has the greatest impact on the results of the model. Experts can focus their attention on precisely determining or evaluating this essential parameter. Sensitivity analysis is commonly utilized to identify parameters that have a substantial impact on the dynamics of the varicella virus disease inside the framework of the SVIR model. These parameters include vaccination effectiveness, immunity duration, interaction rates, and the vaccination availability.

In addition, Sensitivity analysis aids in achieving optimal performance of treatments by evaluating the impact of parameter changes on the performance of control strategies. Policymakers may determine the most significant factors and create measures that have a major effect on lowering varicella occurrence by examining how modifications in key parameters affect the final results of vaccine schedules, additional doses or specific treatments.

CHAPTER V

Conclusion and Recommendations

This research investigates the SVIR model's dynamics and numerical simulation was provided for the model. The model's equilibrium points were determined, along with the basic reproduction number R_0, existence and uniqueness of the solution of the model, and local stability. Existence and uniqueness Proofs verify that the forecasts made by the model are reasonable and can be utilized to make decisions. We found that when $R_0 > 1$, the endemic equilibrium point is stable and disease are present between individuals continuously. But when $R_0 < 1$, it is proved that the disease-free equilibrium remains stable.

By changing the values of the parameters while leaving all other health-related variables constant, we measure the sensitivity of R_0 and explained it graphically, and it was clear that increasing the contact rate will increase R_0, while when the rate of the vaccination increases R_0 decrases, and here the role of the public health awareness campaigns, educational initiatives, and behavioural interventions will contribute in promoting varicella prevention measures, vaccine acceptance, and adherence to control strategies, in addition to the healthcare resources, such as hospital beds and medical personnel, to evaluate the capacity for managing varicella cases and the potential strain on healthcare systems during outbreaks if needed.

Sensitivity analysis of R_0 plays an essential role in the SVIR model of varicella virus. It aids in the identification of crucial parameters, the assessment of model flexibility, and improving the effectiveness of intervention techniques. By performing sensitivity analysis of R_0 in SVIR model, researchers can obtain a better knowledge of the dynamics of infectious diseases, analyse the possible consequences of various control measures, and inform public health strategies to reduce disease spread.

In conclusion, SVIR model does not account for changes in population size or demographic composition resulting from immigration, it could be considered, including such factor can provide a more realistic representation of disease dynamics and allow for better predictions and policy recommendations. As a varicella virus places a significant load on Jordan's medical system because of that a universal varicella vaccination is necessary, and the Jordanian Ministry of Health should implement vaccination programs to fully control the disease. Varicella vaccination provides lasting immunity, lowering the possibility of outbreaks and promoting a healthier community. By decreasing the total burden of varicella virus infection, medical funding can be better directed to other health challenges.

Finally, more studies are recommended, especially for non-constant and varied populations, also employing fractional-order differential equations instead of ordinary differential equations can assist us in minimizing the mistakes brought on by the omitted parameters while simulating real-world processes, and that what I recommended for next study to improve this model.

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Appendices

Appendix A

```
function fval=svir(t,y)

S=y(1);
V=y(2);
I=y(3);
R=y(4);

theta=0.15;
pi=0.024;
beta=0.0714;
mu=0.003916;
gamma=0.05;
alpha=0.08;
tau=0.92;
%legend('Susceptible', 'Infected', 'vaccinated', 'Recovered');
%xlabel('Time');
%ylabel('Population');
%title('SVIR Model');

fval(1,1)=pi-gamma*S-theta*S*I-mu*S;
fval(2,1)= gamma*S-tau*V-alpha*V*I-mu*V;
fval(3,1)= theta*S*I-beta*I-mu*I+alpha*I*V;
fval(4,1)=beta*I+tau*V-mu*R;



y0=[0.71;0.05;0.24;0];
t=[0 100];
[tsol, ysol]=ode45(@tamara,t,y0);
I1org=ysol(:,3);
[tsolincr, ysol]=ode45(@tamara1,t,y0);
I1incr=ysol(:,3);
```

plot(tsol,I1org,'--',tsolincr,I1incr)

Appendix B

turnitin
Manal Thesis
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