Proceedings of Science and Mathematics
Faculty of Science, Universiti Teknologi Malaysia
Vol. 23, 2024, page 60-72

Application of Susceptible-Exposed-Infected-Removed (SEIR) Mathematical Modelling on the Spread of Chicken Pox Disease

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Abstract
The varicella-zoster virus (VZV), which causes chickenpox, is a highly contagious viral illness that mostly affects children but affects people of all ages. Understanding the dynamics of its transmission is the prime importance for public health interventions. In this study, the transmission of chickenpox within a population is examined using mathematical modelling tools, especially the Susceptible-Infected-Exposed-Removed (SIER) model. To account for the disease's incubation time, the SIER model includes an exposed compartment in the traditional SIR model. By utilizing this model, we aim to gain insights into the temporal evolution of the epidemic, assess the impact of various control measures, and predict future trends in disease transmission. We will examine important variables and how they affect the dynamics of the chickenpox epidemic, including the rate of transmission, length of incubation, and rate of recovery. The study showed that chickenpox cases in recent times have increased in Agona West Municipality with $R_0 = 2$. We estimated the reproductive number of chickenpox with vaccination to be $R_0 = 0.3$ which is less than 1. This showed that after vaccination, the disease will die out. It was estimated that 46.2% corresponding to 53, 241 of the people needs to be vaccinated in order to control the chickenpox. The stability analysis of disease free and endemic equilibrium point of chickenpox transmission with vaccination was estimated to be unstable and asymptotically stable. Sensitivity analysis of the SEIR model with vaccination showed that when vaccination rate coefficient is increased chickenpox will be under control.

Keywords: Chickenpox; equilibrium point; transmission; stability; vaccination; Varicella-Zoster-Virus (VZV); Susceptible-Exposed-Infected-Removed (SEIR).

1. Introduction

Globally, infectious illnesses have shaped health care policies and practices and have remained a chronic threat to public health. The varicella-zoster virus, which causes chickenpox, is one of these diseases that has drawn attention for a number of reasons. It is extremely infectious, has a high attack rate in vulnerable people, and can cause serious consequences in some groups, including newborns, pregnant women, and those with weakened immune systems. In the last decade, emergency and re-emerging pandemics such as AIDS, Chickenpox, and Malaria have caused the death of millions of people each year. According to the UNAIDS report on the global AIDS epidemic, an estimated 34 million people, including 3.4 million children, were living with HIV worldwide at the end of 2010, while the related deaths and near infections were 1.8 and 2.7 million, respectively [1].

Based on Kasabwala(2018), Chickenpox is very itchy and can make children feel miserable, even if they do not have many spots. Chickenpox is usually much worse in adults. It is possible to get chickenpox more than once, although it is unusual [2]. The initiative of chickenpox control has experienced a radical change since the vaccine was developed. The vaccine is highly effective in preventing chickenpox and its complications. Since the introduction of new dynamics to the spread of chickenpox that resulted from the use of vaccines in disease prevention, numerous nations have initiated mass vaccination programs. This makes mathematical modelling an invaluable tool to understand and predict the consequences of these vaccination strategies.

The problem statements involve in this research is to develop an accurate and representative SIER (Susceptible-Infectious-Exposed-Removed) model for chickenpox by calculating the reproduction number, R_0 which presents a formidable challenge due to the intricacies involved in capturing the disease's dynamics, particularly the latent 2 period. The research aims to address the question of how to create a model that effectively encompasses the various facets of the disease's progression.

The scope of this research is to use differential equations to analyse the global chickenpox outbreak. Based on the compartmental dynamics of infectious illnesses, the Susceptible-Exposed-Infected-Recovered (SEIR) model is the fundamental mathematical technique that will be used in this study.

Next, it is essential to calculate the basic reproduction number, R_0. The basic reproduction number, R_0, is employed to assess a disease's capacity for transmission. It is the average quantity of secondary infections that a normal infection case in a community where all individuals are susceptible produces. When $R_0 < 1$, that means the disease is no longer able to enter the population. But if $R_0 > 1$, it shows that the disease can enter the population. This model might be used to study the effects of the parameters on the population. Model analysis and simulation are performed to determine how the parameters function to stop the virus from spreading and reducing the chances of becoming infected with this infectious disease.

2. Literature Review

2.1. Varicella-Zoster Virus (VZV) Disease

Chickenpox, caused by the varicella-zoster virus (VZV), is highly infectious and easily transmitted through intimate contact with infected individuals. Based on Pereira.L (2018), up to 90 percent of those in close proximity to the infected individual will get it if they have it. The primary way that the virus spreads is through intimate contact with a chickenpox victim. Chickenpox results in a skin rash that forms small, itchy blisters, which scabs over. It typically starts on the chest, back, and face then spreads. It is accompanied by fever, fatigue, pharyngitis, and headaches which usually last five to seven days[3]. Complications include pneumonia, brain inflammation, and bacterial skin infections. According to Hickling,J(1987), Shingles are also caused by the varicella-zoster virus After having chickenpox, VZV can reactivate in a person's body and cause shingles. Individuals who have never had chickenpox or who have not gotten the vaccination can get VZV from persons who have shingles. This can occur when viral particles from the blisters are inhaled or when fluid from shingles rash blisters comes into direct contact with the skin. If they contract the infection, they will have chickenpox rather than shingles[4].

2.1.1. Origin of Varicella Zoster Virus

Based on Debrus,S(1995), Varicella-Zoster-Virus(VZV) infection of cells in the dorsal root ganglia is probably a consequence of all primary VZV infections. VZV DNAis detected by in situ hybridization and PCR in these tissues obtained at autopsy from individuals who have serologic evidence of prior VZV infection but no signs of VZV disease. VZV may reach dorsal root ganglia from mucocutaneous lesions by an ascending route along neuronal cell axons, or it may be carried to these sites by infected PBMC before cutaneous lesions appear [7]. In contrast to HSV, Hickling,J.K(1987) mentions that latent VZV cannot be reactivated by explanting human ganglia unless the patient has acute herpes zoster at autopsy. Transcription of ORF 63 has been detected in explanted ganglia in the rat model.[4]

2.1.2. Spreading of Varicella Zoster Virus

The ancient civilizations are the source of the oldest accounts of vesicular rashes, which are today known to be caused by herpes simplex and zoster. However, a connection between herpes zoster and chickenpox was not proposed until 1888[10]. One of the biggest obstacles in the history of the varicella zoster virus was establishing this relationship. Since there was no animal host, a large portion of the data had to be gathered through epidemiological and clinical observation. According to Qureshi,S.(2019), the introduction of acyclovir in the 1980s and live attenuated vaccine virus in 1974, respectively, have had a significant influence on treatment and prevention since the connection was established in the 1950s. In 1986, the whole DNA sequence of VZV was determined. The development

of recombinant vaccines and targeted treatments could be made possible by a deeper comprehension of the VZV genome and its gene products.[11] Herpes zoster is very unusual in children younger than 10 years old, with an incidence of 0.74 per 1,000 persons per year. [9]

2.1.3. The Current Therapies of Varicella-Zoster-Virus

The natural history of varicella zoster virus (VZV) infection and the molecular mechanisms of viral pathogenesis are incompletely understood. New insights into primary viraemia and latency are being gained by the development of VZV infection models and the generation of genetically modified VZV recombinants, despite the fact that no animal model can yet replicate every element of VZV infection. Based on Breuer,J(2007), T-cells carry VZV to the skin during viraemia, where cell-free viral replication promotes person-to-person transmission and transmission to the neurons 9 that induce latency. Both host and viral components are involved in the alternative viral processes of lytic infection or latency, which seem to be determined by the kind of cell. Since varicella in children is often mild, antiviral medication is safe and effective for treating varicella, but treatment is normally advised solely for adults and adolescents who have the illness. For everyone with herpes zoster, treatment is advised, although it is especially important for those over 50. [12] the examples of VZV vaccines such as Valaciclovir and famciclovir are becoming the recommended treatments for certain varicella and zoster infections, replacing aciclovir, the original standard. [13]

2.2 Mathematical Modelling Approach in controlling the transmission rate of Chickenpox Disease

There are many outbreaks of Varicella-Zoster virus that have taken place over the years all over the world. Many researches have produced their thesis on the methodologies used in order to prevent the spreading of this chickenpox disease.

2.2.1 Agent Based Susceptible-Exposed-Infectious-Recovered (ABM-SEIR)

As of the Varicella Outbreak that took place in a school in Shenzen, China, Agent BasedSusceptible-Exposed-Infectious-Recovered (ABM-SEIR) Model have beenused in controlling the transmission rate [14].

2.2.2 Maternally-derived immunity, Susceptible, Exposed, Infectious, and Recovered (MSEIR)

Besides, a new epidemiological fractional order mathematical model called MSEIR (Maternally-derived immunity, Susceptible, Exposed, Infectious, and Recovered) using three most widely used operators, namely, the classical Caputo, the Caputo–Fabrizio (CF) and the Atangana–Baleanu–Caputo (ABC) were researched and and applied for the chickenpox outbreak in 2014 among school children of the Shenzhen city of China. Tuse of fixed-point theory helps to prove the existence and the uniqueness for the solutions of each fractional order model under consideration [15].

2.2.3 Various Ordinary Differential Equation Models

In addition, a research have also been carried out based upon four different types of frequently used models of ordinary differential equations related to the chickenpox outbreak among school children of Schenzen city of China in 2013. ITo find the model with the maximum efficiency rate, three novel models with power law type (Caputo), exponentially decaying type (Caputo–Fabrizio), and Mittag Leffler type (Atangana–Baleanu in the Caputo sense) kernels have been suggested and thoroughly examined. By carrying the corresponding fractional-order parameter on each dimensional quantity included in the model, the dimensions of each differential equation for all state variables and parameters have been balanced within the suggested models. The current study's use of fixed point theory produced the evidence for the existence and uniqueness of the fractional-order models under investigation. [16].

2.2.4 Susceptible-Exposed-Infectious-Recovered (SEIR)

SEIR (susceptible-exposed-infected-recovered) dynamic model was established to explore the optimal prevention and control measures according to the epidemiological characteristics about varicella

outbreak in a school in a central city of China. In this study, we can see that the Susceptible-Infected-Exposed-Removed (SIER) model plays a significant role in controlling the transmission rate of chickenpox disease [17].

Methodology

3.1. Model Formulation

The SEIR model for Varicella-Zoster-Virus disease transmission is divided up into four compartments: Susceptible (*S*), Exposed (*E*), Infected (*I*), and Recovered (*R*). A set of differential equations can be used to represent the Chickenpox disease model such as

$$\frac{dS}{dt} = \mu N - \beta SI - \mu - \theta S, \quad (1)$$

$$\frac{dE}{dt} = \beta SI - \mu E - \kappa \mathrm{E}, \quad (2)$$

$$\frac{dI}{dt} = \kappa \mathrm{E} - \mu \mathrm{I} - \gamma \mathrm{I}, \quad (3)$$

,

$$\frac{dR}{dt} = \gamma \mathrm{I} - \mu \mathrm{R} + \theta \mathrm{S} \quad (4)$$

where μN is the total population, μi is the birth rate, θ is the vaccination rate coefficient, β is infection rate, κ is latency rate, γ is recovery rate and μ is natural death rate [38].

3.2 Equilibrium Points

Setting equation (1) to equation (4) to 0 yields the equilibrium points of the system as follows

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$$\frac{dS}{dt} = \mu N - \beta SI - \mu - \theta S = 0 \quad (5)$$

$$\frac{dE}{dt} = \beta SI - \mu E - \kappa \mathrm{E} = 0 \quad (6)$$

$$\frac{dI}{dt} = \kappa \mathrm{E} - \mu \mathrm{I} - \gamma \mathrm{I} = 0 \quad (7)$$

,

$$\frac{dR}{dt} = \gamma \mathrm{I} - \mu \mathrm{R} + \theta \mathrm{S} = 0 \quad (8)$$

As a result, the system's disease-free equilibrium point (DFEP) is determined as

$$(S^*, E^*, I^*, R^*) = \left(\frac{\mu N}{\mu+\theta}, 0, 0, \frac{\theta(\mu N)}{\mu(\mu+\theta)}\right) \quad (9)$$

and the endemic equilibrium point (EEP) is

$$(S^*, E^*, I^*, R^*) = \left(\frac{\mu N}{\beta I^* + \mu + \theta}, 0, \frac{\mathrm{I}^*(\mu+\gamma)}{\kappa}, \frac{\gamma \mathrm{I}^*(\mu+\theta)+\mu\theta(\mu N)}{\mu(\mu+\theta)}\right)$$

(10)

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3.3 Stability Analysis

3.3.1 Disease-free Equilibrium Points

In order to test the stability using the Routh-Hurwitz criterion, the associated characteristic polynomial have be obtained by using the formula to solve the determinant of the Jacobian matrix in order to test. $|J-\lambda I|$ given by

$$\begin{vmatrix} -(\mu+\theta-\lambda) & 0 & -\beta\dfrac{\mu N}{\mu+\theta} & 0 \\ \beta I & -(\mu+\kappa-\lambda) & \beta(\dfrac{\mu N}{\mu+\theta}) & 0 \\ 0 & \kappa & -(\mu+\gamma-\lambda) & 0 \\ \theta & 0 & \gamma & -(\mu-\lambda) \end{vmatrix} = 0$$

Therefore, the characteristics polynomial obtained is

$$(\lambda-\mu)\big(\lambda+(-\mu-\theta)\big)\big(\lambda+(\mu+\kappa)\big)\big(\lambda+(\mu+\gamma)\big)-\beta\kappa(\mu N\mu+\theta)=0$$
$$(\lambda+A)(\lambda+B)((\lambda+C)(\lambda+D)-E)=0 \quad (11)$$

where, $A=-\mu, B=-\mu-\theta, C=\mu+\kappa, D=\mu+\gamma$ and $E=\frac{\mu}{\mu+\theta}$

From Equation (0), the characteristics polynomial equation is as follows :

$\lambda 4+(A+B+C+D)\lambda 3+(AB+(A+B)(C+D)+CD-E)\lambda 2+((A+B)(CD-E)+AB(C+D))\lambda+ABCD-ABE=0$ (12)

The number of possible negative real roots of Equation (4.10) depends on the signs of coefficients. To analyse this, we use the Descartes' rule of signs [34] on the characteristic polynomial below :

$$P(\lambda) = a_1\lambda^4 + a_2\lambda^3 + a_3\lambda^2 + a_4\lambda + a_5$$

Where,

$$a_1 = 1,$$
$$a_2 = (A+B+C+D),$$
$$a_3 = (AB+(A+B)(C+D)+CD-E),$$
$$a_4 = \big((A+B)(CD-E)+AB(C+D)\big),$$
$$a_5 = ABCD - ABE$$

From the Descartes' rule of signs, the number of negative real zeros of $P(\lambda)$ is either equal to the number of changes in sign of $P(-\lambda)$ or less than this by an even number. Therefore the characteristic polynomial (4.10) is multiplied by-1.

$$= a_1(-\lambda)^4 + a_2(-\lambda)^3 + a_3(-\lambda)^2 + a_4(-\lambda) + a_5$$
$$= a_1\lambda^4 + a_2(-\lambda)^3 + a_3\lambda^2 + a_4(-\lambda) + a_5$$

The number of variations in $P(-\lambda)$ is four. Consequently, the characteristic polynomial (4.10) has four negative roots. Thus,

$$= a_1\lambda^4 + a_2(-\lambda)^3 + a_3\lambda^2 + a_4(-\lambda) + a_5$$

has negative roots. The disease-free equilibrium point of the model is locally asymptotically stable whenever $R0 < 1$. From an epidemiological perspective, if a small number of infected individuals are introduced into a completely susceptible population, each infected individual will, on average, infect more than one person during their infectious period. This indicates that when $R0 > 1$, the disease will continue to spread within the population.

3.4. Basic Reproduction Number, R_0

The basic reproduction number influences the duration of the infective phase, the likelihood of infecting a susceptible individual during a single interaction, and the amount of newly susceptible persons contacted per unit of time.

R_0 is calculated using the next-generation matrix method. Only individuals who are capable of spreading infections should be concerned when using the next-generation matrix method. As a result, only E and I compartments are present in the system as follows

$$\frac{dE}{dt} = \beta SI - \mu E - \kappa \mathrm{E},$$

$$\frac{dI}{dt} = \kappa \mathrm{E} - \mu \mathrm{I} - \gamma \mathrm{I},$$

The matrices ***F*** and ***V*** at disease-free equilibrium can be represented as

$$\boldsymbol{F} = \begin{bmatrix} 0 & \beta \\ 0 & 0 \end{bmatrix}$$

$$\boldsymbol{V} = \begin{bmatrix} \mu + \kappa & 0 \\ -\kappa & \mu + \gamma \end{bmatrix}$$

The next generation matrix is given by FV^{-1}, where

$$\boldsymbol{V^{-1}} = \begin{bmatrix} \dfrac{\mathbf{1}}{\mu + \kappa} & \mathbf{0} \\ \dfrac{\kappa}{(\mu + \kappa)(\mu + \gamma)} & \dfrac{\mathbf{1}}{\mu + \gamma} \end{bmatrix}$$

giving

$$\boldsymbol{FV^{-1}} = \begin{bmatrix} 0 & \beta \\ 0 & 0 \end{bmatrix} \begin{bmatrix} \dfrac{\mathbf{1}}{\mu + \kappa} & \mathbf{0} \\ \dfrac{\kappa}{(\mu + \kappa)(\mu + \gamma)} & \dfrac{\mathbf{1}}{\mu + \gamma} \end{bmatrix}$$

$$\boldsymbol{FV^{-1}} = \frac{\beta\kappa}{(\mu + \kappa)(\mu + \gamma)}$$

Hence, since $\boldsymbol{FV^{-1}} = R_0$, the equation for the reproductive number is,

$$R_0 = \frac{\beta\kappa}{(\mu+\kappa)(\mu+\gamma)} \tag{13}$$

3. Results and discussion

We estimated the parameter values from the data obtained from the Agona West Municipal Health Directorate as follows: Transmission rate (β) =0.026, Latency rate (κ) = 0.036 , Recovery rate (γ) = 0.014, Death rate (μ) = 0.02, Vaccinated rate coefficient (θ) = 0.00019 and Population (N) = 115,358. The summary of the data obtained from the Agona West Municipal Health Directorate can be found at Table 1.

Parameter	Definition	Estimated Value
β	Transmission rate	0.026
κ	Latency rate	0.036
γ	Recovery rate	0.014
μ	Death rate	0.02
θ	Vaccinated rate coefficient	0.00019
N	Population	115,358

Table 1: Parameter Values of *SEIR* Model

From the studies, we estimated the reproductive number of chickenpox with vaccination to be R_0=0.03 which is less than 1. This shows that after vaccination, the chickenpox will die out and will not populate. We estimated that 53, 241 out of 115,358 people needs to be vaccinated against chickenpox and this corresponds to 46.2% of the Agona West Municipality needs to be vaccinated in order to control the chickenpox. We estimated the stability analysis of disease free and endemic equilibrium point with vaccination to be unstable and asymptotically stable.

The graphs depicted in Figure 1, shows the visuals of the four classes: S, E, I, and$R$. In the upcoming figures shows how the parameter values evolve and what are the differences that occurs as the values changes. Figure 1 depicts the graph of all four classes of SEIR model with initial parameters of Transmission rate(β) = 0.026, Latency rate (κ) = 0.036, Recovery rate (γ) = 0.014, Vaccination rate, (θ)= 0.00019 and the Death rate(μ) = 0.02.

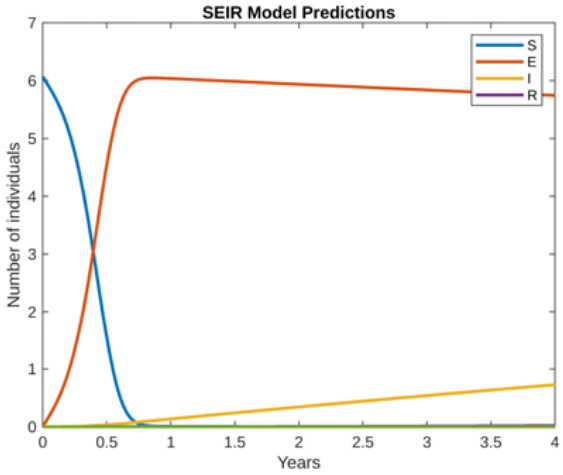
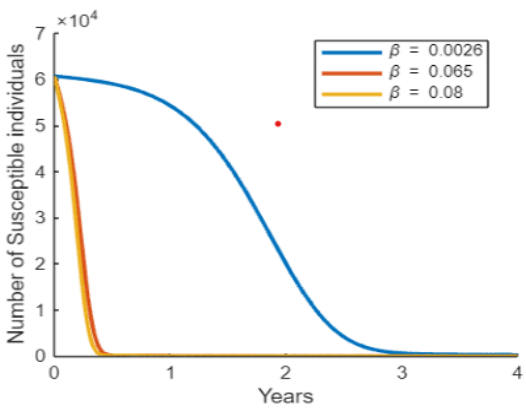


Figure 1 Simulation of the $SEIR$ model for β = 0.026 , κ = 0.036, γ = 0.014 and θ = 0.00019

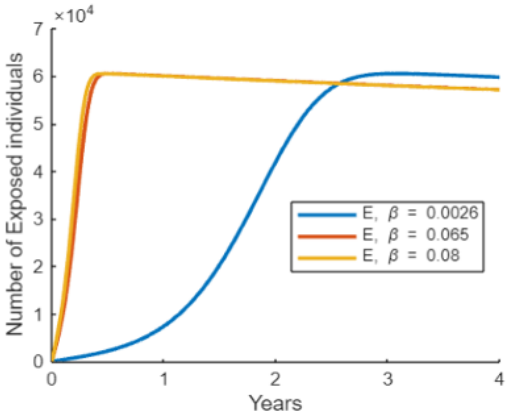
4.2. Changed in Parameter Values

4.2.1. Changes in Transmission rate, β

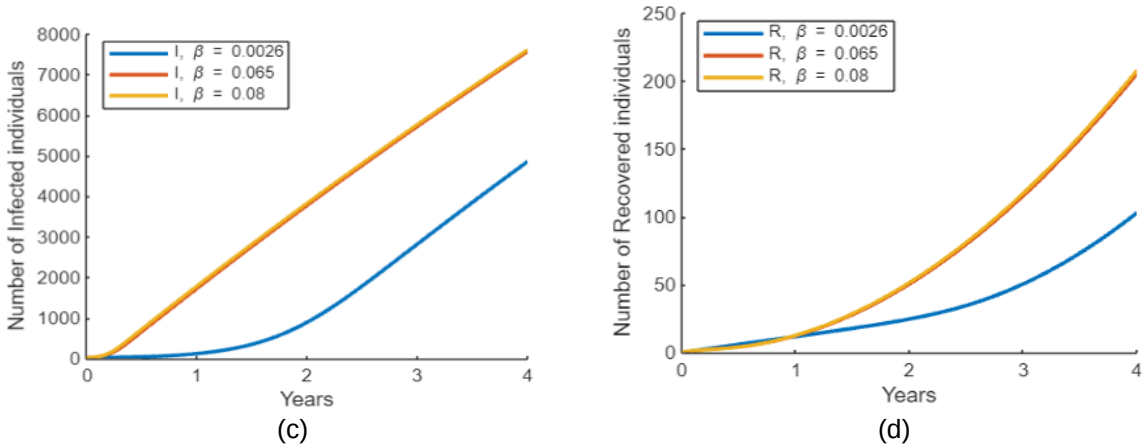
In this segment, the transmission rate,β will be changed to observe the spread of the chickenpox disease in a population. The transmission rate is made to be varied from 0.08 to 0.065 from the initial value which is 0.026. The remaining parameters are the same as for the basis simulation such as Latency rate (κ) = 0.036, Recovery rate (γ) = 0.014, Death rate(μ) = 0.02 and Vaccinated rate coefficient (θ) = 0.00019. The following results are analysed by comparing the curves to the base model.



(a)



(b)



(c) (d)

Figure 2 SEIR model with changes in β value where a) Susceptible, b) Exposed, c) Infected and d) Recovered

In particular, we can see that as the infection rate increases, in (a), the graph flattens out earlier compared to the original value of β = 0.0026. If the value of the transmission rate increases, so the population of susceptible individuals will decrease over time. The Susceptible population's curve flattens in half a year when the value of β = 0.08 and flattens at the third year when the value of β= 0.0026. Hence, we can conclude that the susceptible population decreases sharply with time when the transmission rate,β is at 0.08 and 0.065. As in this case according to (b), the number of the exposed population spikes up must drastically at way at the beginning of the first year and flattens in the middle of the first year and decreases a little when the β value is 0.08 compared to the β= 0.0026 which increases slowly between the first year till the third year. The infected population also portrays the spiking differences of the graph when β= 0.08 compared to the initial transmission rate. It shows that the infected population will be much higher in a small period of time when the transmission rate is at 0.08. The recovered population shows us the much intricate difference in the recovered population which increases much faster than compared to the initial transmission rate, whereby the gradient of the graph tend to be higher compared to the β value being 0.0026.

4.2.2. Changes in Recovery rate, γ

In this section, the recovery rate, γ is varied to observe the changes in dynamics of the population. The recovery rate is made to be varied from 0.07 to 0.99 from its initial value which is 0.014. The remaining parameters are the same as for the basis simulation such as Transmission rate (β) = 0.026, Latency rate (κ) = 0.036, Death rate(μ) = 0.02 and Vaccinated rate coefficient (θ) = 0.00019. Figure 3 display the curves for each class separately. The following results are analysed by comparing the curves to the base model.

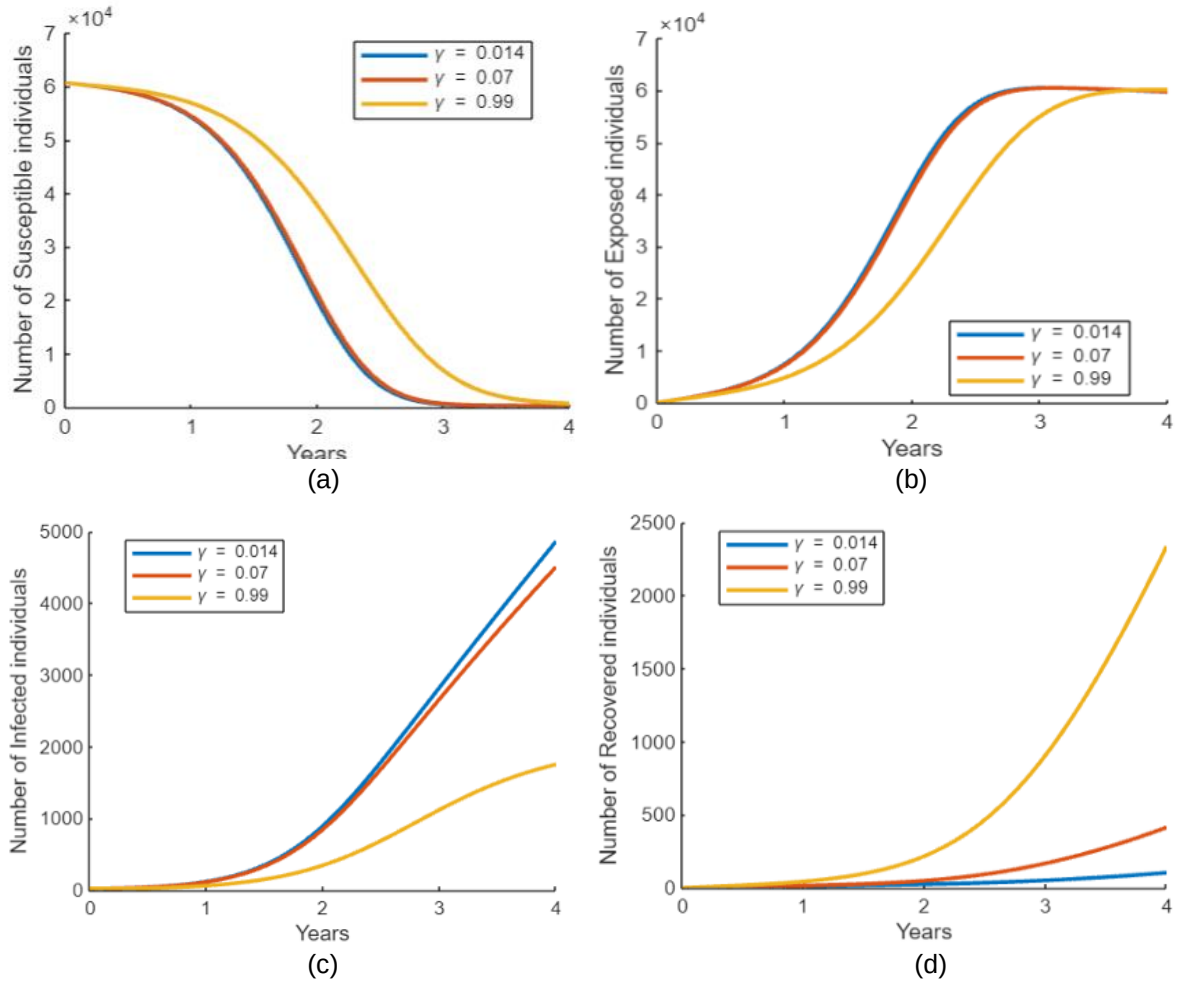


Figure 3 SEIR model with changes in γ value where a) Susceptible, b) Exposed, c) Infected and d) Recovered

In (a), it is observable that the susceptible population decreases when the recovery rate varies from 0.07 to 0.09 as compared to the initial recovery rate which is 0.014. In (b), there is no significant increase or decrease however, we can see the slower rate of increase trend in the exposing population as the higher recovery rate, decreases the speed of the exposure of the disease. The infected and recovered population decreases in (c) and (d) when the recovery rate increases as compared to the initial value of the recovery rate.

4.2.3. Changes in Latency rate, κ

The rate at which individuals transition from the exposed to the infected classes, the latency rate,κ is varied from 0.05 to 0.75 from its initial value which is 0.036. The remaining parameters are the same as for the basis simulation: Transmission rate(β) = 0.026, Recovery rate (γ) = 0.014, Vaccination rate, (θ) = 0.00019 and the Death rate(μ) = 0.02. The result obtained is as in the following Figure 4.

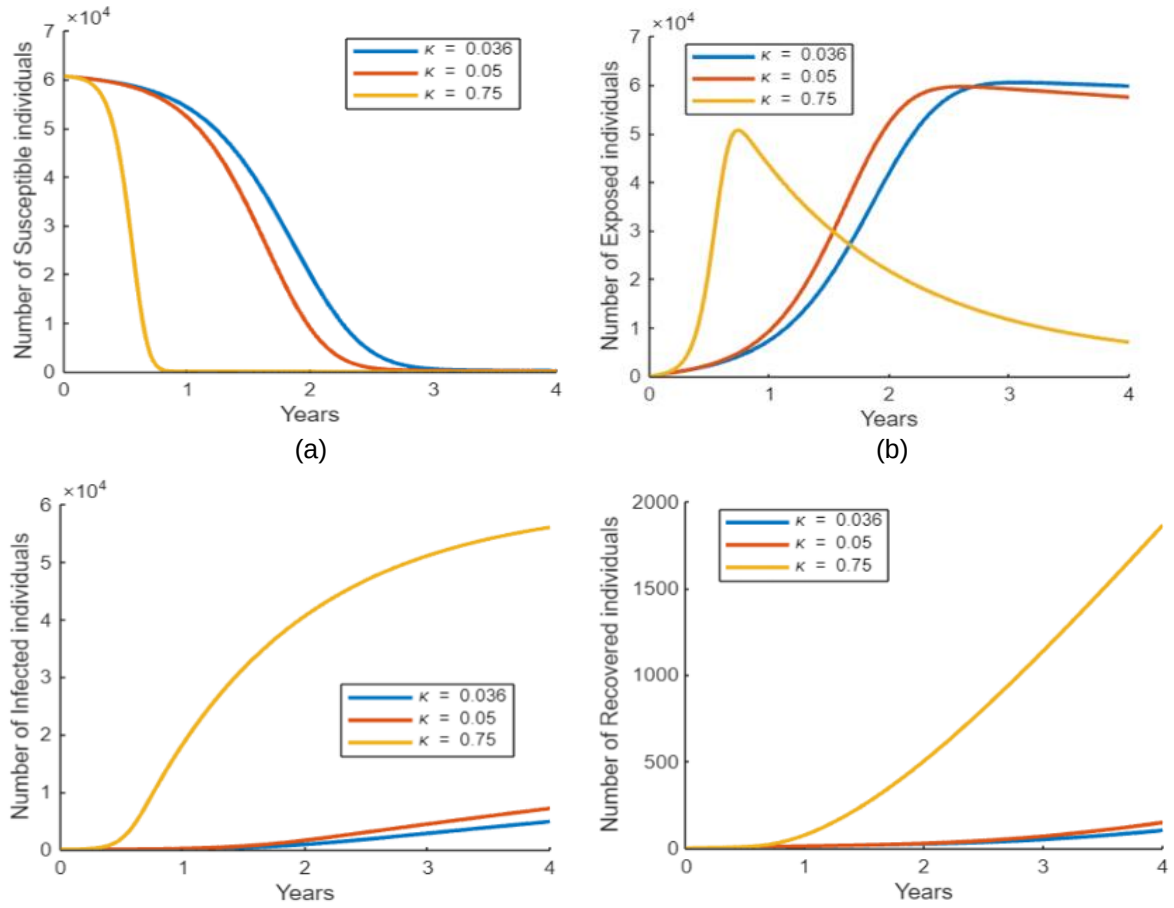


Figure 4 SEIR model with changes in κ value where a) Susceptible, b) Exposed, c) Infected and d) Recovered

The number of the susceptible populations sharply decreases when κ is varied from 0.05 to 0.75. However, when compared to the initial condition which is 0.0036, the curve is much steeper descending and flattens to be zero at the first year of the pandemic. The decrease of population is approximately from 60,000 to 100injust a year when the latency rate, κ is 0.75. The exposed population declines in (b) from approximately 50,000 to asymptotically to the time axis as the time increases when the latency rate, κ is 0.75 compared to the initial value which is 0.036 and the varied value which is 0.05. In (c) it shows that when the latency rate is at 0.75 there in an increase in the infected population as compared to the initial value. In (d) shows that the recovered population increases slowly in the first year and proceeds to increase drastically when κ = 0.75 compared to the initial condition whereby the latency rate is 0.0036.

4.2.4. Changes in Vaccination coefficient rate, θ

In this section, we see the changes in the vaccination rate coefficient (θ) affects the dynamics of the $SEIR$ mode. The vaccination rate varies from 0.02 to 0.96 from its initial value which is 0.00019.

Looking into Figure 5, in (a) the susceptible population gradually decreases asymptotically to the time axis as the time increases when the vaccination rate is at 0.96. However as for the rate of vaccination in 0.02, there is not much difference in the dynamics of the curve as compared to the initial value. Hence, we can conclude that the number of susceptible populations decreases much quicker when the vaccination rate is increased. Besides, as of for the Exposed Population based on (b), with the vaccination rate or 0.96, we can see that there is a decrease in exposed individuals compared to the initial value and 0.02 whereby there is an increased in number of them and then proceeds to be

constant. We can also see that with the vaccination rate at 0.96, the number of individuals exposed is so much lesser approximately from 60,000 to 10,000. In (c), it is obvious that the number of infected individuals is far more lesser when the rate of vaccination is at 0.96 compared to the initial value. In (d), the number of individuals recovered increases drastically.

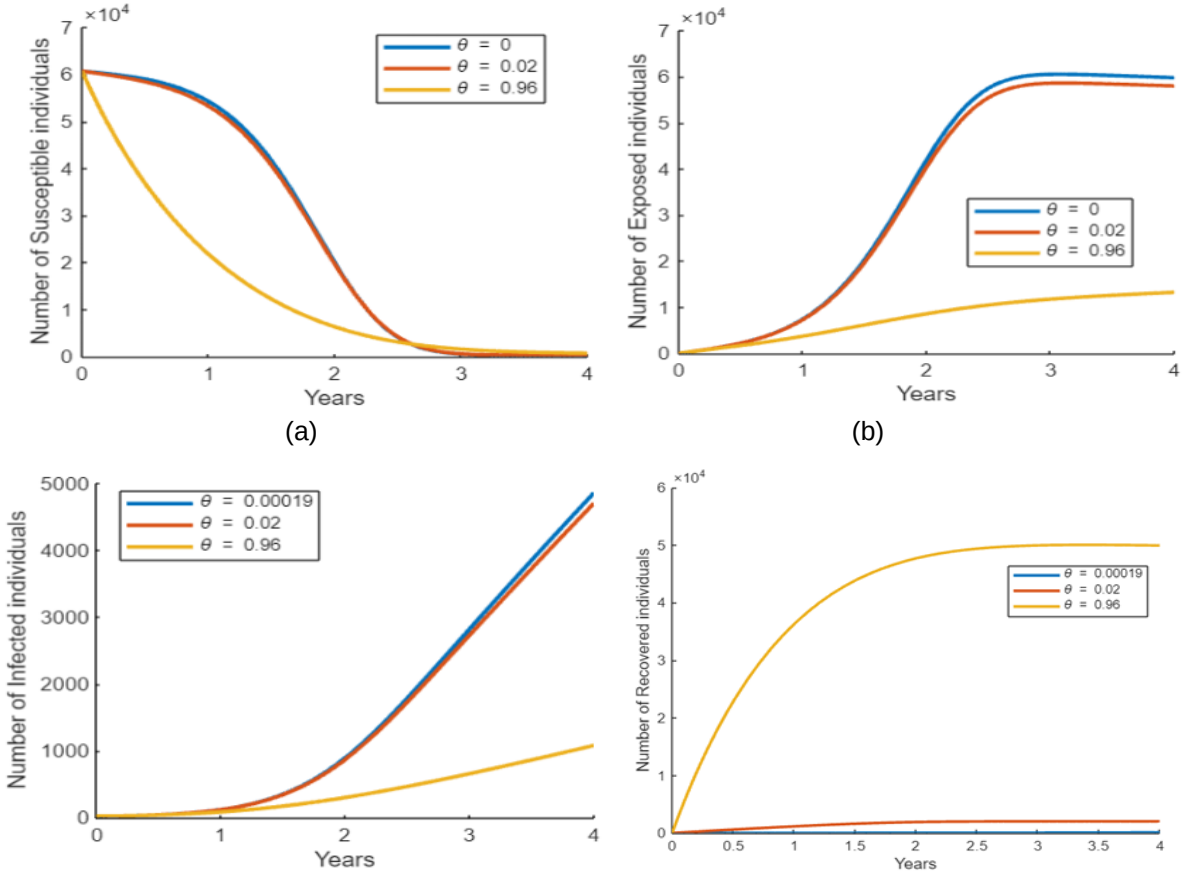


Figure 5 SEIR model with changes in θ value where a) Susceptible, b) Exposed, c) Infected and d) Recovered

Conclusion

As a summary, the importance of the vaccination rate coefficient (θ) on the process to control the spreading of the disease is assured in the case this SEIR model for the chickenpox disease in a West Municipality. A higher vaccination rate (θ) decreases the number of susceptible, (S) population where higher number of infected, (I) individuals are recovered. This model predicts that in order to control varicella in the Municipality, 53,241 of 115,358 individuals should be vaccinated which corresponds to 46.2% vaccination rate. This stresses the significance of vaccination campaigns in limiting disease dissemination.

Acknowledgement

I would like to express my sincere gratitude to my supervisor for their guidance, support, and invaluable feedback throughout the course of my thesis. I am also thankful for the support and encouragement received from my friends and family, which has been instrumental in the completion of this work. Additionally, I extend my appreciation to the academic and administrative staff who have contributed to my educational journey. Their collective assistance has greatly enriched my learning experience.

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