EPIDEMIOLOGY OF CHICKENPOX IN AGONA WEST MUNICIPALITY, GHANA

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DECLARATION

I hereby declare that this submission is my own work towards theM. Phil and that, to the best of my knowledge, it contains no material previously published by another person nor material which has been accepted for the award of any other degree of the University, exceptwhere dueacknowledgement has been made in the text

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DEDICATION

This work is dedicated to almighty Allah whose grace and mercies has brought me far to this level. I also dedicate this work to my wife, brothers and sisters whose prayers and encouragement has sustained me all this while.

ABSTRACT

The study shown that chickenpox cases in recent time has increased and is persistent (endemic) in the Agona West Municipality with $R_0 = 2$, impling that on average 2 people will contract chickenpox in a year. We estimated the reproductive number chickenpox with vaccination to be $R_o = 0.3 < 1$which shows that after vaccination the disease will die out. 46.2% corresponding to 53,241 people needed to be vaccinated in order to control the chickenpox. The stability analysis of disease free and endemic equilibrium point of chickenpox transmission without vaccination was estimated to be a centre. We also estimated the stability analysis of disease free and endemic equilibrium point with vaccination to be unstable and asymptotically stable.

Sensitivity analysis of the SEIR model without vaccination showed that the latency rates (κ) is more sensitive to the model than the transmission rate and the recovery rate while Sensitivity analysis of SEIR model with vaccination also indicated that the vaccination rate coefficient (θ) being is more sensitive to the model than the transmission rate (β), latency rates (κ) and the recovering rate (γ).

TABLE OF CONTENT

LIST OF TABLE

LIST OF FIGURES

CHAPTER 1

INTRODUCTION

1.1 Background to the Study

Chickenpox (Varicella) is a contagious viral disease characterized by vesicular skin lesions, fever, mild fatigue and general malaise. The primary infection with Varicella-Zoster Virus (VZV) causes Varicella. The varicella-zoster virus (VZV) is one of eight herpes viruses that cause infections in humans. These viruses rapidly proliferate, invade and destroy infected cells like other herpes viruses and it is a double-stranded DNA virus that is closely related to herpes simplex virus types 1and 2. The Varicella - Zoster Virus establish a latent infection in nerve ganglions and later reactivate as shingles or Zoster and Herpes Zoster (HZ) is characterized by a localized vesicular rash and often associated with pain. Elderly suffer more pain from Herpes Zoster and can persist for more than 3 months. The virus has a shorter survival time frame in the environment and the incubation period is normally between 14 - 16 days with exposure range of 10 - 21 days (NCIRS, 2009).

Infection in adolescents and adults is more severe than infection in children and less in healthy young children. The mean number of skin vesicles is normally from 250 to 500 but greater lesions of 500 ~~vesieles~~ may occur in severe cases.

Complications of chickenpox infection occur in around 1% of all cases, with the most being secondary bacterial infection of the skin lesions. Pneumonia, encephalitis (inflammation of the brain) and cerebellar ataxia, thrombocytopenia and hepatitis are other complications associated with chickenpox (NCIRS, 2009).

1.2 Mode of Transmission

Varicella-Zoster Virus (VZV) is transmitted through the respiratory tract with viral particles present in respiratory droplets from 24 to 48 hours before the appearance of the rash and from the fluid of the skin lesions of an infected person and can also be transmitted indirectly by contact with particles of clothing's and other items exposed to fresh drainage from the open sores or aerosols from vesicle fluid of skin of acute varicella (NCIRS, 2009).

The secondary attack rate from a case with primary varicella to susceptible children has been estimated to be between 61% and 100%, whereas secondary attack rates with shingles are lower at approximately 15% (NCIRS, 2009).

1.3 Signs and Symptoms

Chickenpox starts with rashes with adults experiencing 1 to 2 days fever and malaise. In children, rash is often the first sign of the disease. The rash is associated with itching and pain that rapidly progresses from macules to papules to vesicles lesion that are 1 to 4mm in diameter. The rash normally appears on the head, followed by the trunk (centripetal distribution). Lesions can occur on the mucous membrane of the respiratory tract, oropharynx, conjunctiva and the cornea. The rashes develop into blisters called vesicles and the vesicles are superficial and delicate with clear fluid on the erythematous base. Vesicles rupture or become purulent before they dry and crust. Successive crops appear over several days with lesions present in several days of development. Healthy, susceptible children usually have 200 to 500 lesions in 2 to 4 successive crops.

Clinical course in healthy children is generally mild, with malaise, itching and fever up to 102°F for 2 to 3 days but no respiratory or gastrointestinal symptoms. Adult may

experience more severe illness than children. Complications occur most frequently in immune compromised persons, pregnant women and adults (Harpaz et al, 2008).

1.4 Complications of Chickenpox

Complications are not common among healthy children but occur more frequently with those who are above 15 years of age and infants below one year of age. Staphylococcus or Streptococcus, dehydration, pneumonia and central nervous system involvement are most common complications that are caused by secondary bacterial infection of skin lesions. The overall case-fatality rate in the United States is 2/100,000 but rises to 30/100,000 in adults. Mothers who develop the disease between 5 days before or within 2 days after delivery are at increased risk of developing complicated varicella with a fatality rate of up to 30% and Neonates developed varicella between the ages of 5 to 10 days. Immunocompromised persons have a high risk of disseminated disease (Kansas Disease Investigation Guidelines, 2012).

1.5 Chickenpox in Pregnancy

Primary infection with Varicella-Zoster Virus during pregnancy may result in viral transmission to the fetus or the newborn. Congenital varicella syndrome, neonatal varicella or herpes zoster is the result of intrauterine transmission of Varicella-Zoster Virus during infancy. Congenital varicella syndrome is most commonly associated with primary Varicella-Zoster Virus maternal infection during the first trimester (three months) of pregnancy and is associated with low birth weight, limb deformities and ocular problems in the newborn. Severe, fatal and perinatal chickenpox in the newborn infant is the causes of maternal infection with Varicella which occurs from 5 days before

delivery to 2 days after delivery (Federal Bureau of Prisons Clinical Practice Guideline, 2011).

1.6 Treatment of Chickenpox

Antiviral drugs are normally used to treat chickenpox and herpes zoster infections. Acyclovir, Valacyclovir or famcyclovir are considered for treatment of varicella for the first day of the rash but recommended for immunocompromised patients and treatment for mild cases of varicella consists of isolation. In the case of resistance, Foscarnet is considered the second line drug. The fingers are cut short to avoid scratching and warm water bath is recommended for bath to reduce itching (Federal Bureau of Prisons Clinical Practice Guideline, 2011).

1.7 Chickenpox Vaccination

Varicella vaccine is a live attenuated viral vaccine derived from the Oka strain of VZV. The vaccine virus was isolated by Takahashi in the early 1970s from vesicular fluid from a healthy child with varicella disease. Varicella vaccine was licensed for general use in Japan and Korea in 1988 and later licensed in the United States in 1995 for persons 12 months of age and older. The virus was attenuated by sequential passage in human embryonic lung cell culture, embryonic guinea pig fibroblasts and in WI-38 human diploid cells. The Oka (Merck) vaccine has undergone further passage through MRC-5 human diploid cell cultures for a total of 31 passages.

The composition of the vaccine contains small amounts of sucrose, processed porcine gelatin, sodium chloride, monosodium L-glutamate, sodium diphosphate, potassium phosphate and potassium chloride and trace quantities of residual components of MRC-5

cells, EDTA, neomycin and fetal bovine serum. The vaccine contains no preservative and reconstituted with sterile water (MMWR, 2006).

1.8 Statement of the Problem

A good understanding of some recent reported cases of chickenpox incidence in Ghana would be of interest in the SEIR epidemiology of chickenpox in Agona West Municipality, Ghana. World Health Organization (WHO) observed that chickenpox is one of the endemic diseases in the sub-Saharan country (PAO, 2012).

Ghana Health Service (2010) observed that 77,790, 45,512, 19,614, 35,667 cases of chickenpox were recorded in 2008, 2004, 2003 and 2002 respectively.

Ghanaian Times (2008) reported an outbreak of chickenpox in Accra Metropolis. The Times survey conducted in six medical centers revealed that about 600 chickenpox cases has been reported in first quarter of 2008. This includes three Hospitals and three Polyclinics. In 2007, 356 cases were reported during the first quarter at hospitals and clinics located in three sub-metros of Ayawaso, Kpeshie and Okaikoi within the Accra metropolitan area. La recorded 194 cases at its General Hospital, Maamobi 189 at the Sulemana Memorial Hospital, a private hospital and Nima 137 in the government clinic according to the Times survey. The survey revealed that the new outbreak is more prevalent in densely populated areas. Chickenpox cases in recent time have increased in Agona West Municipality and Mathematical models for the dynamics of the chickenpox with special emphasis in the municipality are unknown. It is against this background that the study is focused using mathematical model to estimate the endemicity of chickenpox in the Municipality.

1.9 Objectives of the Thesis

The objectives of the study are

- ❖ to use SEIR model (Anderson and May, 1991) to estimate the reproductive number of chickenpox.
- ❖ to use SEIR model with vaccination to estimate the reproductive number of chickenpox in Agona West Municipality, Ghana.
- ❖ to study the stability analysis of the equilibria of the systems.
- ❖ to study the effects of the model parameters in the solution (sensitivity analysis).

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1.10 Methodology

The Population used for this study is Agona West Municipal and the data was obtained from Agona West Municipal Health Directorate. We use Anderson and May (1991) SEIR model to model the spread of chickenpox followed by modeling the chickenpox with vaccination. Differential equations were used to formulate the model equations. Stability and Sensitivity analysis are then performed on the model equations and Matlab software was used for the graph simulation.

1.11 Justification of the Thesis

Mathematical modeling of diseases in the host of population is of great practical value in predicting and controlling the spread of diseases. The outcomes of this study will help policy makers and stakeholders in the country to design appropriate programs for the control and the prevention of chickenpox diseases.

It will provide more information on some of the dangers of chickenpox disease and the need to vaccinate against it. Moreover, the findings would add to the existing body of knowledge in the epidemiology of chickenpox.

1.12 Organization of the Thesis

This thesis is organized into five chapters. Chapter 1 is the introduction comprising background to the study, statement of the problem, objectives, methodology, justification and the organization of the thesis. Chapter 2 focuses on the previous research work related to the thesis. Chapter 3 examines the methodology used and the Chapter 4 discusses the result obtained from the research with the model equations. Finally Chapter 5 gives the recommendations and the conclusions drawn from the studies

CHAPTER 2

REVIEW OF RELATED WORKS

Anderson and May (1991) proposed an unforced SEIR model on small world networks produces sustained oscillations with periods and amplitudes compatible with the pertussis data both before and after mass vaccination for realistic values of life expectancy, latency and recovery time. Analysis of the homogeneously mixed limit shows that the infectiousness parameter is also within the reported range of pertussis.

Alonso and Ibeas (2011) incorporated the design of a vaccination strategy for a SEIR model with incomplete knowledge about the populations. Their design is oriented towards the measurement and use of the infectious population in the design of the vaccination rule with the eventual incorporation of an observer to deal with uncertain model state knowledge.

Deguen et al. (2000) applied SEIR model to unveil the seasonal pattern of varicella epidemics with a regular contact rate function from1991 – 1996 in France. The contact rate was assumed to be either a continuous, or a piecewise constant periodic function. The estimate for the incubation and infectious periods were consistent with values from clinical and serological grounds. Moreover, that seasonal fluctuation of the contact rate function reflected on the impact of school holidays on chickenpox diffusion among school children.

De la Sen et al. (2011) used SEIR model with vaccination for propagation disease model. The work unveiled total population amounts as a refrain for the illness transmission since its increase makes it more difficult contacts among the susceptibles and infectives. The control objective is the asymptotically tracking of the removed by immunity population to the total population while achieving simultaneously the remaining population (susceptible, infected, and infectious) that asymptotically converge to zero. They use a state observer to estimate the true various partial populations of the susceptible, infected, infectious, and immune which are assumed to be unknown. Their model parameters assumed to be unknown and the parameters are replaced by available estimates to implement the vaccination action.

Sun et al. (2012) made used of an SEIRS model with saturating contact and shown that when the reproduction ratio is less than 1, the disease-free equilibrium is globally asymptotically stable. The Krasnoselku sub-linearity method was used to determine the stability of the equilibria. The nonlinear lyapunov function was also used to show that the endemic equilibrium is globally stable.

Huang and Takeuchi (2011) applied SIR, SIS, SEIR and SEI models of epidemiological dynamics with time delays and a general incidence rate. The work obtained new Lyapunov function which was used to shown the global asymptotic stability of the equilibria.

Rost and Wu (2008) proposed new SEIR model with distributed infinite delay when the infectivity depends on the age of infection. The work revealed that the disease-free equilibrium is globally asymptotically stable and the endemic equilibrium point in a small neighbourhood is stable. They apply a permanence theorem for infinite dimensional systems and obtained that the disease is always present when $R_o > 1$.

Al-Showaikh and Twizell (2006) extended SEIR model to second order derivative for the transmission dynamics of measles to enable the geographic spread of the disease in a population which has not been vaccinated. The resulting system of three reaction diffusion equations was solved by a convergent finite-difference technique which is second order accurate in space and time. They studied a parallel implementation procedure and tested their method using two initial distributions.

Black and Markane (2010) studied a systematic method of analyzing models of the spread of childhood diseases in order to consistently separate the effects of demographic stochasticity, external forcing and modeling choices. Such a technique is provided by the Van Kampen system-size expansion and was used to provide analytical expressions for quantities of interest. The method was applied to the susceptible–exposed–infected and recovered (SEIR) model with distributed exposed and infectious periods and calculated the form that stochastic oscillations take in terms of the model parameters.

They use suitable approximation and apply the formalism to analyze a model of whooping cough which includes seasonal forcing. This allows them to accurately interpret the results of simulations and made a more quantitative assessment of their predictions of the model. The observed dynamics are as a result of a macroscopic limit

cycle induced by the external forcing and resonant stochastic oscillations about this cycle.

De la Sen et al. (2010) extended time-varying SEIR propagation disease model subject to delays which includes mixed regular and impulsive vaccination rules. The model takes into account the natural population growth and the mortality associated with the disease and the potential presence of disease endemic thresholds for both the infected and infectious population dynamics, as well as the loss of immunity of new-borns. Finite number of time varying distributed delays in the susceptible-infected coupling dynamics influencing the susceptible and infected differential equations was considered.

Li and Chen (2005) applied an age structured SEIR epidemic model with vertical and horizontal disease transmission and established threshold results for the existence of endemic states under certain conditions and obtained expression for its uniqueness. The threshold condition was in terms of demographic and epidemiological parameters of the model differential equations. Their proposed regular vaccination control objective is the tracking of a prescribed suited infectious trajectory for a set of given initial conditions. They suggested that impulsive vaccination can be used to improve discrepancies between the SEIR model and its ~~suitable~~ reference one.

Peter (2012) extended the SEIR structure to include a new class of Conditional Autoregressive (CAR) class of spatial models. This is to account for the Mumps data set they have procured, which contains mismatched lattice structures that cannot be handled by traditional CAR models. Their use of CAR models is desirable here, as these models

are known to produce spatial smoothing on lattices and serve as a natural way of drawing strength spatially in estimating spatial effects.

Lastly, they developed a pair of spatial SEIR models utilizing their CAR structure. They first utilize the exponential assumption, which is very robust and developed a highly flexible spatial SEIR model by embedding the CAR structure into the SEIR structure. This allows for a realistic analysis of epidemic data occurring on a lattice.

Juan et al. (2006) studied SEIR epidemic model that includes constant inflows of new susceptible, exposed, infective, and recovered. The model takes into account a population size dependent contact rate and a disease related death. As the infected fraction cannot be eliminated from the population, the model they considered showed a threshold phenomenon and a sharp threshold under the special case where the new members of immigration are all susceptible. In order to prove the global asymptotical stability of the endemic equilibrium, they introduced the change of variable which reduced the four dimensional system to a three dimensional asymptotical autonomous system with limit equation.

Hethcote (1976) considered an SI model with constant population size and two kinds of susceptible having different infection rates. The exact solution was given in an implicit form and derived an approximation to the solution which permits simple estimates of the infection rates.

Arafa et al. (2012) made use of SIR model that monitors the temporal dynamics of a childhood disease in the presence of preventive vaccine. Homotopy Analysis Method

(HAM) was used to obtain an analytic approximate solution of this model and was compared with the classical - fourth order Runge- Kutta method (RK4) to gauge its effectiveness. The obtained results proved that the disease will persist within the population if the vaccination coverage level is below a certain threshold.

Chen et al. (2006) used control measure modeling approaches to show that models can be derived from an integrated scale analysis generated from three different types of functional relationship; Wells–Riley mathematical model, competing-risks model, and Von Foerster equation both with key epidemiological determinants and functional connections between them.

They examined mathematically, the impact of engineering control measures in containing the spread of indoor airborne infections including influenza, chickenpox, measles and severe acute respiratory syndrome (SARS). The test proved that engineering controls could reduce the basic reproductive number (R_o) below 1.60 for chickenpox and 3 for measles but their simulations shown that a prepared response with public health interventions would have a high probability of containing the indoor airborne infections.

Combinations of engineering control measures and public health interventions could moderately contain influenza strains with R_o of 4. The analysis indicated that effective isolation of symptomatic patients with low efficacy contact tracing is sufficient to control a SARS outbreak. They suggested that a valuable added dimension to public health inventions could be provided by systematically quantifying transmissibility and proportion of asymptomatic infection of indoor airborne infection. High childhood incidence rates of chickenpox diseases are witness in temperate climates and substantial

prove is presented regarding the morbidity associated with primary infection both in children and high risk groups. The increased in adult prevalence of disease in warmer countries is associated with significantly higher rates of complications and death. The differences in age specific incidence of chickenpox maybe in line with decreased of viral transmission in warmer temperatures especially in rural settings and high humidity areas. Moreover, these factors were not always consistent in Australia and South American countries.

Sengupta and Breuer (2009) observed that, universal there has been a great decrease in childhood vaccination incidence, hospitalizations and deaths associated with Varicella-Zoster Virus in the USA. A recommendation of two dose schedule is provided following evidence of increased incidence of break through disease in vaccine recipients over a period of time. Vaccine that prevents Zoster has been recommended for use in the elderly to address the significant burden posed by this illness on health resources in temperate countries.

Mandal et al. (1998) reported higher seropositivity rates of 96.6% in adults aged 25 years and above who were living in a city slum compared to adult's resident in a village (69.9%) seropositive in West Bengal, India. The authors suggested that a low seroprevalence rates was a rural phenomenon and related to the density of the population. Overcrowding in many urban areas can overcome factors that result in low Varicella - Zoster Virus transmission in warmer climates that result in higher rates of disease.

Sinha (1976) observed a very low transmission rates amongst children despite household exposure which coincided with a greater proportion of the people being co infected by other respiratory viruses. Herpes viruses were isolated from 14% of total virus isolations and the authors postulated that this may result in later infection by Varicella-Zoster Virus as characteristic in the population studied.

Tunbridge et al. (2008) released that in UK, 90% of adults above the age of 18 years are seropositive for Varicella-Zoster Virus and the epidemiology is different in many tropical countries with less than 60% of adults being immune. The incidence of chickenpox in adolescents and adults is higher due to population density and climatic effects. Morbidity and mortality is increasing in adults more than children. For every 100,000 individuals who develop chickenpox, four to nine will die of whom 81 to 85% are adults. Chickenpox is five times more likely to be fatal in pregnancy than in the non-pregnant adult

Van Rijckevorsel et al. (2012) studied the seroprevalence of Varicella-Zoster Virus antibodies among various ethnic groups in Amsterdam and identify factors associated with seronegative Varicella-Zoster Virus status. Varicella zoster virus infection is a common childhood illness and regular vaccination against Varicella-Zoster Virus is not done in the Netherlands. In 1995, it was calculated that 98 - 100% of the adult Dutch general population is immune but the calculation represent a very small number of non-Dutch ethnic origin. Subtropical and tropical countries like Morocco, Surinam, and Turkey which has large immigrant communities recorded lower VZV transmission in Amsterdam.

Garnett and Grenfell (1992) presented mathematical models and analyzed the data to examine the epidemiological implications of possible immunologically mediated links between patterns of varicella and herpes-zoster incidence in human communities. No significant difference was shown to exist between the risk of zoster caused by the vaccine and the wild virus. The work revealed the influence of the prevalence of varicella on viral reactivation and the impact of vaccination with attenuated virus, which may be able to recrudesce.

They found out that under some conditions, mass application of such vaccines may have the impact of increasing zoster incidence. The results they presented indicated that before starting any vaccination programme against varicella, its outcome needs to be assessed before given to people.

Ferguson et al. (1996) examined the impact of transmission events from patients with shingles (zoster) on the epidemiology of varicella before and after the introduction of mass immunization by using a stochastic mathematical model of transmission dynamics. Reactivation of the virus is shown to damp stochastic fluctuations and move the dynamics toward simple annual oscillations. They estimated the force of infection due to zoster cases by comparing simulated and observed incidence with time series. They realized that the presence of infectious zoster cases reduces the tendency for mass immunization to increase varicella incidence at older ages when disease severity is typically higher.

Manfredi et al. (1997) studied the frequency and clinical spectrum of chickenpox complications among immunocompetent hospitalized children. Their results shown that two hundred and nineteen out of 991 patients (22.1%) hospitalized for varicella suffered from a complicated disease. 104 cases of Central nervous system (CNS) was recorded, followed by skin/soft tissue infections, lower respiratory tract involvement, and thrombocytopenia. A complicated disease was significantly associated with the male gender and an elevated incidence of varicella-zoster (VZ) virus infection acquired by household contacts.

The involvement of lower respiratory airways and skin/soft tissues seemed to occur at an earlier age, compared with CNS and thrombocytopenia. However, lower respiratory tract and skin/soft tissue infections occurred earlier during disease course than complications interesting the CNS and coagulation system. All patients with complicated chickenpox showed a favourable outcome within 5 - 40 days, except two patients developing a lethal cardiomyopathy and Reye syndrome.

Gani (1965) considered the differential-difference equations of the SIR model with constant population size and gives the partial differential equation which the associated probability generating function satisfies and outlines a mathematical method for solving it. However, the mathematics involved is so complicated that it limits its success in the SIR model varying the population sizes of at most 3 individuals.

Khoshnood et al. (2006) assessed the age-specific seroprevalence of varicella in the French population and explored age-adjusted differences between gender and geographic region. They said most varicella-zoster virus infections occur during early

childhood and the seroprevalence rates reach almost 50% by 4 years of age and approximately 90% by 8 years. They recommended the best strategy to reduce the prevalence of wild-type varicella-zoster virus in the French population would be to immunize children 12 to18 months of age, as is currently done in the United States.

Elahi et al. (2007) conducted studies to calculate the seroprevalence of immunity to the varicella-zoster virus (VZV) infection and evaluated the positive predictive value (PPV) and the negative predictive value (NPV) of the self-reported history of VZV infection in pregnant women. The studies were conducted in 18 private medical analysis laboratories with information on socio-demographic characteristics and past history of varicella or zoster were collected using a questionnaire. They determined the serological levels of past exposure to VZV using the blood samples they obtained.

In all, 486 pregnant women were recruited and the seroprevalence of VZV antibodies was 98.8%. Six women were seronegative of which four were primiparous. The PPV was high (99.5%) compare to NPV of 10.3%. They concluded that PPV is a reliable marker of prior VZV infection while a negative history does not predict lack of immunity and should be completed by serological analysis which might be introduced to regular antenatal blood tests.

Gidding et al. (2003) performed a serosurvey using opportunistically collected sera submitted to diagnostic laboratories across Australia from 1997 - 1999. They tested a sample by state and sex of 2027 sera from persons aged 1 - 49 years using an enzyme immunoassay method. The average age of infection and age-specific forces of infection (the probability that a susceptible individual acquires infection) were calculated using

published methodologies. The result shown seropositivity increases with age, with 83% of sera positive by ages 10 - 14 years and the highest force of infection between 5 - 9 years age group (0.195 per susceptible year) followed by the 0 - 4 years age group (0.139 per susceptible year) and the mean age of infection was 8.15 years. Their results provided valuable baseline information to measure the impact of vaccination and indicated that vaccination should be aimed at children below 5 years of age.

Socan et al. (2010) conducted studies on a cross-sectional, age stratified to determine varicella-zoster seroprevalence and force of infection in Slovenia. Out of the sample collected, 3689 serum were tested for VZV IgG antibodies with an enzyme immunoassay. Semi parametric and parametric modeling was used to estimate the force of infection and concluded that regardless of the age group used, the highest transmission occurred in children in their first years of school.

Pollock and Golding (1993) studied a quantitative description of factors independently predictive of reported chickenpox infections in two national cohorts of British children. Their studies shown that people aged 10 years with chickenpox was more common in the children of well to do families (higher social class, higher parental education levels) with a higher prevalence in those parts of the United Kingdom normally associated with affluence, such as the South East and South West of England and reported lower rates in Wales and Scotland. Chickenpox by 10 years was as a result of overcrowded in the home.

They observed that a similar but less marked pattern occurred for chickenpox by the age of 11 years in the 1958 NCDS cohort. This social distribution obviously reflected in the

population rather than age-specific susceptibility. They indicated that both social and climatological factors may be important in defining groups at risk and recommended further research if a vaccination service is to be implemented in the country.

Schuette and Hethcote (1999) developed an age structured epidemiologic demographic model with vaccination for varicella and zoster and estimated parameters from the epidemiological data. They realized two possible dangers of an extensive varicella. Vaccination program are more varicella (chickenpox) cases in adults, when the complication rates are greater, there's an increase in zoster (shingles) cases. They used mathematical and computer simulation model to evaluate the effects of varicella vaccination programs, and the age distribution of varicella cases shown a shift in the simulation. Their results confirm that many of the adult cases occur after vaccine-induced immunity fanes and there were mild varicella cases with fewer complications. Their simulation shows that, zoster incidence increases in the first three decades after starting of a vaccination program, because people who had varicella in childhood age did not boost it. The simulations validated the second danger of greater zoster cases.

Keeling and Rohani (2008) studied an introduction to the modeling of infectious diseases and used simplest mathematical models to show how appropriate elements of biological complexity leads to improved understanding of disease dynamics and control. The work emphasized on the development of models and their uses as a predictive tool and a means of understanding epidemiological process. Both computer simulation and analytic results were obtained which revered the behaviour of the disease.

Muir et al. (2002) used heteroduplex mobility assay to identify variants of varicella-zoster virus circulating in the United Kingdom and elsewhere. 58 segregating sites were found out of the 23,266 (0.25%) examined and nucleotide diversity was estimated to be 0.00063 within the United Kingdom. These were an order of magnitude smaller than comparable estimates from herpes simplex virus type 1.

Sixteen substitutions were nonsynonymous, the majority of which were clustered within surface expressed proteins. Extensive genetic correlation between widely spaced sites indicated that recombination has been rare. Phylogenetic analysis of varicella-zoster viruses from four continents distinguished at least three major genetic clades. Most geographical regions contained only one of these three strains, apart from the United Kingdom and Brazil, where two or more strains were found. There was minimal genetic differentiation between the samples collected from Africa (Guinea Bissau, Zambia) and the Indian subcontinent (Bangladesh, South India) suggesting recent rapid spread of low mutation rates.

They argued that geographic pattern of strain distribution would favour a major influence of the former and genetic uniformity of most virus populations makes recombination difficult to detect. They found among the samples at least one probable recombinant between two of the major strains originating from Brazil, where mixtures of genotypes co-occur.

CHAPTER 3

METHODOLOGY

This chapter presents mathematical models that mimic the prominent aspects of epidemiology of Chickenpox in Agona West Municipal, Ghana. These models will help in predicting the spread of Chickenpox in the Municipality. Two variations of the Susceptible-Exposed-Infected and Removed (SEIR) epidemiological model and SEIR with vaccination are used to study and analyse the disease.

3.1 Description of the SEIR Model of Chickenpox without Vital Dynamics and Vaccination

In the SEIR model, the total population is divided into four separate compartments: Susceptible (S), Exposed (E), Infective (I) and Recovered (R). Susceptible people can contract the disease if they come into contact with the chickenpox, while those in the exposed compartment are individuals infected but not yet infectious with the chickenpox and are not able to pass the chickenpox to others. Infectives are the individuals that are infectious and capable of transmitting the infection to any susceptible that they come in contact and those in the recovered compartment are individuals previously infected but now neither infected nor susceptible. They have an infection-acquired immunity (permanent immunity). The proportion of individuals in each compartment S,E,I and R at time t is given as S(t), E(t), I(t) and R(t).The figure below is the flow chart of the SEIR model without vaccination.

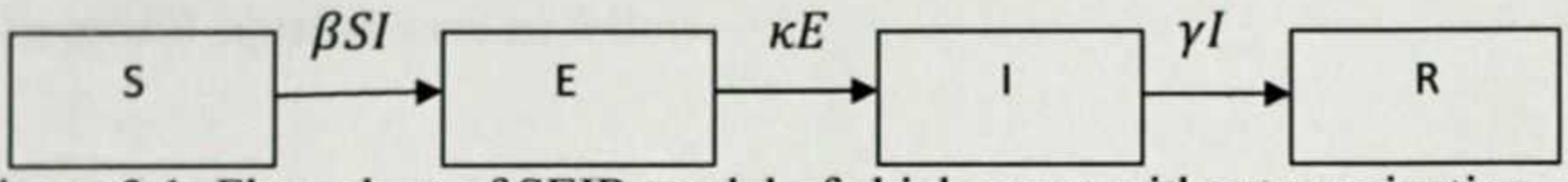

Figure 3.1: Flow chart of SEIR model of chickenpox without vaccination

3.1.1 The Model Assumptions

1. The members in the population mix homogeneously. (the degree of interaction is the same)

2. The disease spreads in a closed environment; there is no birth and death, emigration or immigration and so the total population N remains constant for all time. Thus

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

3. The number of susceptible who are infected by chickenpox (infective individual) per unit time is proportional to the total number of Susceptible; β is proportional coefficient (Infection rate). So the total number of those infected by chickenpox (new infective) per unit time is given as $-\beta S(t)I(t)$, the negative sign indicate a decrease in the number of susceptible.

4. The rate at which individual leave the exposed compartment (E) into the infective (I) compartment at the time t is given by $\kappa E(t)$, where κ is the latency rate of individual exposed to the chickenpox.

5. The number of individuals from the infective compartment to the recovered (R) at the time t is given as $\gamma I(t)$, where γ is the recovery rate coefficient of chickenpox and those who recovered from chickenpox gain permanent immunity.

6. Age, sex, color, social status and race do not affect the probability of being infected.

The model equations are as follows

$$\frac{ds}{dt} = -\beta SI \tag{3.1}$$

$$\frac{dE}{dt} = \beta SI - kE \tag{3.2}$$

$$\frac{dI}{dt} = kE - \gamma I \tag{3.3}$$

$$\frac{dR}{dt} = \gamma I \tag{3.4}$$

We let $s(t) = \frac{S(t)}{N}, e(t) = \frac{E(t)}{N}, i(t) = \frac{I(t)}{N}$ and $r = \frac{R(t)}{N}$ then $s(t) + e(t) + i(t) + r(t) = 1$, where $s(t), e(t), i(t)$ and $r(t)$ represent Susceptible, exposed, infected and recovered fraction of the population.

3.2 The Basic Reproductive Number (R_o) of Chickenpox Transmission without Vaccination Using the Next Generation Matrix Method

The reproductive number is defined as the mean number of secondary infections caused by infections by one infected individual during the mean course of infection in a totally susceptible population. The reproductive number (R_o) is a threshold that determines whether a disease will spread in a population or dies out. Whenever R_o is >1, the disease will spread in a population leading to an epidemic. If R_o is < 1, the diseases fail to spread in the population and when $R = 1$, the disease remains in the population (Johnson, 2009).

3.2.1 Description of the Next Generation Matrix

The Next Generation matrix is given by $G = FV^{-1}$. FV^{-1} has (i, k)entry and is defined as the expected number of new infections in i compartment produced by infected individuals originally introduced into compartment k. F has (i, j) entry and is defined as the rate at which infected individuals in j compartment produced by infections in i compartment. V^{-1} has (j, k) entry and is the mean length of time the individual spends in j compartment. The Next Generation matrix (G) has four 2×2 matrix with the top left being F-V, the right bottom sub matrix is zero, the left bottom is T_1 and the right bottom gives us T_2 (Van Den Driessche and Watemough, 2002).

We re- arrange equations (3.1), (3.2), (3.3), (3.4) and linearize the SEIR model without vaccination to obtain the Next Generation matrix of the disease- free equilibrium.

$$\frac{dE}{dt} = \beta SI - kE \tag{3.2}$$

$$\frac{dI}{dt} = kE - \gamma I \tag{3.3}$$

$$\frac{dS}{dt} = -\beta SI \tag{3.1}$$

$$\frac{dR}{dt} = \gamma I \tag{3.4}$$

We let $\frac{dS}{dt} = p$, $\frac{dE}{dt} = x$, $\frac{dI}{dt} = y$, $\frac{dS}{dt} = 0$ and $\frac{dR}{dt} = z$. We will have our Next generation matrix (G) as

$$G = \begin{vmatrix} \frac{dp}{dE} & \frac{dp}{dI} & \frac{dp}{dS} & \frac{dp}{dR} \\ \frac{dx}{dE} & \frac{dx}{dI} & \frac{dx}{dS} & \frac{dx}{dR} \\ \frac{dy}{dE} & \frac{dy}{dI} & \frac{dy}{dS} & \frac{dy}{dR} \\ \frac{dz}{dE} & \frac{dz}{dI} & \frac{dz}{dS} & \frac{dz}{dR} \end{vmatrix}$$

$$G = \begin{vmatrix} -\kappa & \beta & 0 & 0 \\ \kappa & -\gamma & 0 & 0 \\ 0 & -\beta & 0 & 0 \\ 0 & 0 & 0 & 0 \end{vmatrix}$$

We consider the first two linearized differential equations

$$G = \begin{vmatrix} F - V & 0 \\ T_1 & T_2 \end{vmatrix}$$

We consider $F - V$

$$F - V = \begin{vmatrix} -\kappa & \beta \\ \kappa & -\gamma \end{vmatrix}$$

$$F - V = \begin{vmatrix} 0 & \beta \\ 0 & 0 \end{vmatrix} - \begin{vmatrix} \kappa & 0 \\ -\kappa & \gamma \end{vmatrix}$$

$$F = \begin{vmatrix} 0 & \beta \\ 0 & 0 \end{vmatrix} \text{ and } V = \begin{vmatrix} \kappa & 0 \\ -\kappa & \gamma \end{vmatrix}$$

$$\text{Det } V = \kappa\gamma - 0$$

$$\text{Det } V = \kappa\gamma$$

$$V^{-1} = \frac{1}{k\gamma} \begin{vmatrix} \gamma & 0 \\ \kappa & \kappa \end{vmatrix}$$

$$V^{-1} = \begin{vmatrix} \frac{1}{\kappa} & 0 \\ \frac{1}{\gamma} & \frac{1}{\gamma} \end{vmatrix}$$

$$FV^{-1} = \begin{vmatrix} 0 & \beta \\ 0 & 0 \end{vmatrix} \begin{vmatrix} \frac{1}{\kappa} & 0 \\ \frac{1}{\gamma} & \frac{1}{\gamma} \end{vmatrix}$$

$$FV^{-1} = \frac{\beta}{\gamma}$$

But $R_0 = FV^{-1}$

$$R_0 = \frac{\beta}{\gamma} \tag{3.5}$$

3.3 Equilibrium Point of Chickenpox Transmission without Vaccination

At the equilibrium point, we equate $\frac{dS}{dt} = 0$, $\frac{dI}{dt} = 0, \frac{dE}{dt} = 0$ and $\frac{dR}{dt} = 0$ of equations (3.1), (3.2), (3.3) and (3.4)

$$-\beta SI = 0$$

$$\beta SI - kE = 0$$

$$kE - \gamma I = 0$$

$$\gamma I = 0$$

Solving the equations simultaneously, we have $S = 0, E = 0, I = 0$ and $R = 0$.

The equilibrium point of SEIR model without vaccination is $(S^*E^*I^*R^*) = (0, 0, 0, 0)$

3.3.1 Stability Analysis of the Disease Free Equilibrium point of Chickenpox Transmission without Vaccination

We evaluate the equilibrium points of the ordinary differential equations (3.1), (3.2), (3.3) and (3.4).We linearized the disease-free equilibrium at the point where I= 0 to obtain the Jacobian matrix

$$J = \begin{vmatrix} 0 & 0 & -\beta & 0 \\ 0 & -k & \beta & 0 \\ 0 & k & -\gamma & 0 \\ 0 & 0 & \gamma & 0 \end{vmatrix}$$

$$J - \lambda I = \begin{vmatrix} 0 & 0 & -\beta & 0 \\ 0 & -k & \beta & 0 \\ 0 & k & -\gamma & 0 \\ 0 & 0 & \gamma & 0 \end{vmatrix} - \begin{vmatrix} \lambda & 0 & 0 & 0 \\ 0 & \lambda & 0 & 0 \\ 0 & 0 & \lambda & 0 \\ 0 & 0 & 0 & \lambda \end{vmatrix}$$

$$J - \lambda I = \begin{vmatrix} -\lambda & 0 & -\beta & 0 \\ 0 & -k-\lambda & \beta & 0 \\ 0 & k & -\gamma-\lambda & 0 \\ 0 & 0 & \gamma & -\lambda \end{vmatrix} \tag{3.6}$$

$$Det\,(J - \lambda I) = \lambda \begin{vmatrix} \kappa+\lambda & -\beta & 0 \\ -\kappa & \gamma+\lambda & 0 \\ 0 & -\gamma & \lambda \end{vmatrix}$$

$$Det\,(J - \lambda I) = \lambda((\kappa+\lambda)\begin{vmatrix} \gamma+\lambda & 0 \\ -\gamma & \lambda \end{vmatrix} - (-\kappa)\begin{vmatrix} -\beta & 0 \\ \gamma & \lambda \end{vmatrix}$$

$$Det\,(J - \lambda I) = \lambda((\kappa+\lambda)(\lambda\gamma+\lambda^2) - \lambda\kappa\beta)$$

$$Det\,(J - \lambda I) = \lambda(\lambda\kappa\gamma + \lambda^2\kappa + \lambda^2\gamma + \lambda^3 - \lambda\kappa\beta)$$

$$Det\,(J - \lambda I) = \lambda^2\kappa\gamma + \lambda^3\kappa + \lambda^3\gamma + \lambda^4 - \lambda^2\kappa\beta$$

$$Det\,(J - \lambda I) = \lambda^4 + \lambda^3(\kappa+\gamma) + \lambda^2(\kappa\gamma - \kappa\beta)$$

$$Det\ (J - \lambda I) = 0$$

$$\lambda^4 + \lambda^3(\kappa + \gamma) + \lambda^2(\kappa\gamma - \kappa\beta) = 0$$

Let $a = \kappa + \gamma$

$$b = \kappa\gamma - \kappa\beta$$

$$c = 0$$

$$d = 0$$

$$\lambda^4 + 2\lambda^3 a + \lambda^2 b + c + d = 0$$

If the eigenvalues λ_1, λ_2, λ_3 and λ_4 are real and negative then the disease free equilibrium point is asymptotically stable, if one of the eigenvalues is positive then the disease free equilibrium point is unstable and if one of the eigenvalues is zero then the disease free equilibrium point is a centre

3.3.2 Stability Analysis of the Endemic Equilibrium point of Chickenpox Transmission without Vaccination

We linearized equations (3.1), (3.2), (3.3) and (3.4) to obtain the Jacobean matrix for the stability analysis of chickenpox transmission without vaccination.

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

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

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

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

$$J = \begin{vmatrix} -\beta I & 0 & -\beta S & 0 \\ \beta I & -\kappa & \beta S & 0 \\ 0 & k & -\gamma & 0 \\ 0 & 0 & \gamma & 0 \end{vmatrix}$$

$$J - \lambda I = \begin{vmatrix} -\beta I & 0 & -\beta S & 0 \\ \beta I & -\kappa & \beta S & 0 \\ 0 & k & -\gamma & 0 \\ 0 & 0 & \gamma & 0 \end{vmatrix} - \begin{vmatrix} \lambda & 0 & 0 & 0 \\ 0 & \lambda & 0 & 0 \\ 0 & 0 & \lambda & 0 \\ 0 & 0 & 0 & \lambda \end{vmatrix}$$

$$J - \lambda I = \begin{vmatrix} -\beta \mathrm{I} - \lambda & 0 & -\beta S & 0 \\ 0 & -\kappa - \lambda & \beta S & 0 \\ 0 & \kappa & -\gamma - \lambda & 0 \\ 0 & 0 & \gamma & -\lambda \end{vmatrix} \tag{3.7}$$

$$Det(J - \lambda I) = (\beta I + \lambda) \begin{vmatrix} \kappa + \lambda & -\beta S & 0 \\ -\kappa & \gamma + \lambda & 0 \\ 0 & -\gamma & \lambda \end{vmatrix}$$

$$Det(J - \lambda I) = (\beta I + \lambda)((\kappa + \lambda) \begin{vmatrix} \gamma + \lambda & 0 \\ -\gamma & \lambda \end{vmatrix} - (-\kappa) \begin{vmatrix} -\beta S & 0 \\ -\gamma & \lambda \end{vmatrix})$$

$$Det(J - \lambda I) = (\beta I + \lambda)((\kappa + \lambda)(\lambda\gamma + \lambda^2 + 0) + \kappa(-\lambda\beta S + 0))$$

But $\beta I = 0$ and $\beta S = 0$

$$Det(J - \lambda I) = (\lambda)((\lambda\kappa\gamma + \lambda^2\kappa + \lambda^2\gamma + \lambda^3) + \kappa)$$

$$Det(J - \lambda I) = \lambda^2\kappa\gamma + \lambda^3\kappa + \lambda^3\gamma + \lambda^4 + \lambda\kappa$$

But $Det(J - \lambda I) = 0$

$$\lambda^4 + \lambda^3\kappa + \lambda^3\gamma + \lambda^2\kappa\gamma + \lambda\kappa = 0$$

$$\lambda^4 + \lambda^3(\kappa + \gamma) + \lambda^2\kappa\gamma + \lambda\kappa = 0$$

$$p = k + \gamma, q = k\gamma, r = \kappa \; and \; s = 0,$$

$$\lambda^4 + p\lambda^3 + q\lambda^2 + \kappa + s = 0$$

3.4 SEIR Model of Chickenpox Transmission with Vaccination

3.4.1 Model Assumptions

1. We assume here that individuals who are vaccination against chickenpox vaccination gain permanent immunity.

2. We also assume that a portion of the susceptible, θS go to the recovered compartment (R) directly due to permanent immunity gained from the chickenpox vaccination.

The assumptions in 3.2.1 also hold here. The flow chart of SEIR of Chickenpox with vaccination is given below

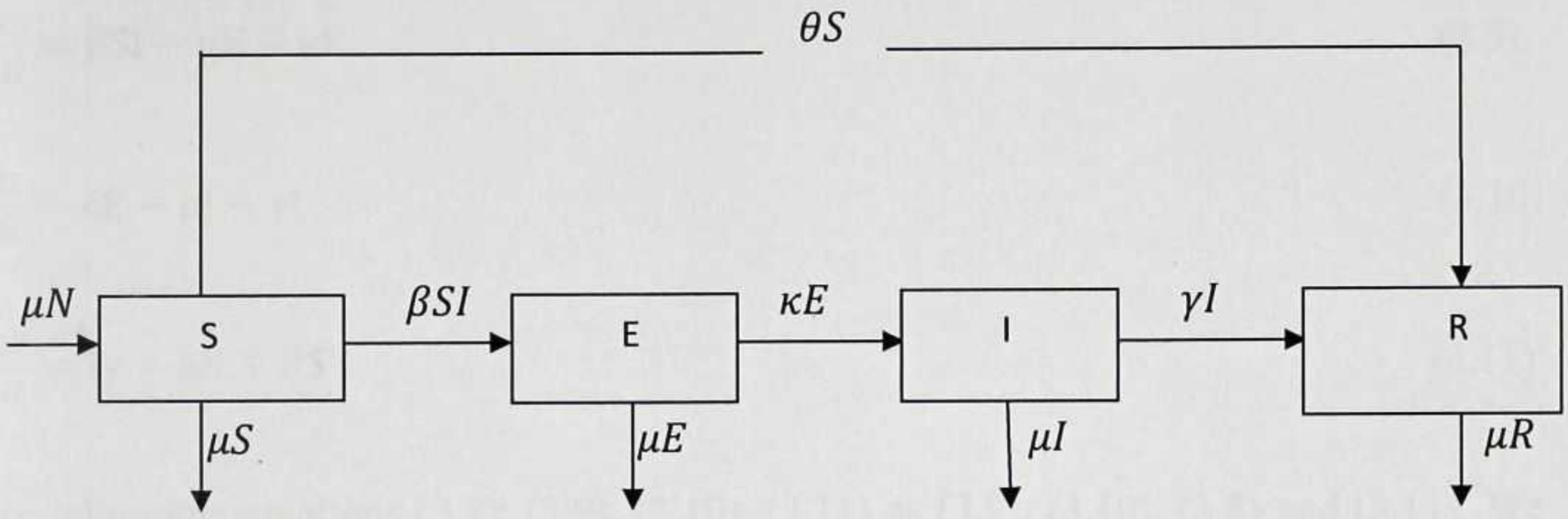

Figure 3.2: Flow chart of SEIR of chickenpox transmission with vaccination

Where

μN = Birth rate of susceptible population

θ = Vaccinated rate coefficient

β = Infection rate

κ = Latency rate

γ = Recovery rate

μ = Natural death rate

3.4.2 The Basic Reproduction Number (R_o) SEIR of Chickenpox with Vaccination Using the Next Generation Matrix

The model equations (Anderson and May (1991) for chickenpox with vaccination are as follows

$$\frac{dS}{dt} = \mu N - \beta SI - \mu S - \theta S \qquad (3.8)$$

$$\frac{dE}{dt} = \beta SI - \mu E - \kappa E \tag{3.9}$$

$$\frac{dI}{dt} = \kappa E - \mu I - \gamma I \tag{3.10}$$

$$\frac{dR}{dt} = I\gamma - \mu R + \theta S \tag{3.11}$$

Re-arranging equations (3.8), (3.9), (3.10), (3.11) as (3.9), (3.10), (3.8) and (3.11). We then linearized to obtain the Next Generation matrix at the disease-free equilibrium. We have

$$\frac{dE}{dt} = \beta SI - (\mu + \kappa)E \tag{3.9}$$

$$\frac{dI}{dt} = \kappa E - (\mu + \gamma)I \tag{3.10}$$

$$\frac{dS}{dt} = \mu N - \beta SI - \mu S - \theta S \tag{3.8}$$

$$\frac{dR}{dt} = I\gamma - \mu R + \theta S \tag{3.11}$$

We let $\frac{dE}{dt} = x, \frac{dI}{dt} = y, \frac{dS}{dt} = z,$ and $\frac{dR}{dp} = p$. Our linearized Next Generation matrix equal to

$$G = \begin{vmatrix} \frac{dx}{dE} & \frac{dx}{dI} & \frac{dx}{dS} & \frac{dx}{dR} \\ \frac{dy}{dE} & \frac{dy}{dI} & \frac{dy}{dS} & \frac{dy}{dR} \\ \frac{dz}{dE} & \frac{dz}{dI} & \frac{dz}{dS} & \frac{dz}{dR} \\ \frac{dp}{dE} & \frac{dp}{dI} & \frac{dp}{dS} & \frac{dp}{dR} \end{vmatrix}$$

$$G = \begin{vmatrix} -(\mu + k) & \beta & 0 & 0 \\ k & -(\mu + \gamma) & 0 & 0 \\ 0 & \beta & -\mu + \theta & 0 \\ 0 & \gamma & \theta & -\mu \end{vmatrix}$$

We consider the first two linearized differential equation

$$G = \begin{vmatrix} F - V & 0 \\ T_1 & T_2 \end{vmatrix}$$

$$F - V = \begin{vmatrix} -(\mu + \kappa) & \beta \\ k & -(\mu + \gamma) \end{vmatrix}$$

$$F - V = \begin{vmatrix} 0 & \beta \\ 0 & 0 \end{vmatrix} - \begin{vmatrix} (\mu + \kappa) & 0 \\ -k & (\mu + \gamma) \end{vmatrix}$$

$$F = \begin{vmatrix} 0 & \beta \\ 0 & 0 \end{vmatrix} \text{ and } V = \begin{vmatrix} (\mu + \kappa) & 0 \\ -\kappa & (\mu + \gamma) \end{vmatrix}$$

$$Det\ V = (\mu + \kappa)(\mu + \gamma)$$

$$Det\ V = (\mu + \kappa)(\mu + \gamma)$$

$$V^{-1} = \frac{1}{(\mu+\kappa)(\mu+\gamma)} \begin{vmatrix} (\mu + \gamma) & 0 \\ \kappa & (\mu + \kappa) \end{vmatrix}$$

$$V^{-1} = \begin{vmatrix} \frac{1}{(\mu+\kappa)} & 0 \\ \frac{k}{(\mu+\kappa)(\mu+\gamma)} & \frac{1}{(\mu+\gamma)} \end{vmatrix}$$

$$FV^{-1} = \begin{vmatrix} 0 & \beta \\ 0 & 0 \end{vmatrix} \begin{vmatrix} \frac{1}{(\mu+\kappa)} & 0 \\ \frac{k}{(\mu+\kappa)(\mu+\gamma)} & \frac{1}{(\mu+\gamma)} \end{vmatrix}$$

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

But $FV^{-1} = R_o$

$$R_o = \frac{\beta\kappa}{(\mu+k)(\mu+\gamma)} \tag{3.12}$$

3.5 Equilibrium Points of Chickenpox Transmission with Vaccination

We evaluate the equilibrium points of the ordinary differential equations by setting the right hand side of equation (3.8), (3.9), (3.10) and (3.11) to zero to obtain the values for S, E, I and R respectively. At the equilibrium $\frac{dS}{dt} = 0, \frac{dE}{dt} = 0, \frac{dI}{dt} = 0$, and $\frac{dR}{dt} = 0$

$$\mu N - \beta SI - \mu S - \theta S = 0 \tag{3.8}$$

$$\beta SI - \mu E - \kappa E = 0 \tag{3.9}$$

$$kE - \mu I - \gamma I = 0 \tag{3.10}$$

$$I\gamma - \mu R + \theta S = 0 \tag{3.11}$$

From (3.6) $\beta SI = (\mu + k)E$

$$S = \frac{(\mu+k)E}{\beta I} \tag{3.13}$$

From (3.7)

$$E = \frac{(\mu+\gamma)I}{k} \tag{3.14}$$

Putting (3.10) into (3.9), we obtained

$$S = \frac{(\mu+k)(\mu+\gamma)I}{\beta Ik}$$

$$S = \frac{(\mu+k)(\mu+\gamma)}{\beta k} \tag{3.15}$$

Adding equation (3.5) and (3.6), we obtain

$$\mu N - (\mu + \theta)S = (\mu + k)E$$

$$E = \frac{\mu N-(\mu+\theta)S}{(\mu+K)}$$

Putting (3.11) into (3.12), we get

$$E = \frac{\mu N}{(\mu+k)} - \frac{(\mu+\theta)(\mu+k)(\mu+\gamma)}{\beta k(\mu+k)}$$

$$E = \frac{\mu N}{(\mu+k)} - \frac{(\mu+\theta)(\mu+\gamma)}{\beta k}$$

$$E = \frac{\mu\beta kN-(\mu+\theta)(\mu+\gamma)+(\mu+k)}{\beta k(\mu+k)} \tag{3.16}$$

Now $I = \frac{kE}{(\mu+\gamma}$, putting (3.13) into I, we will have

$$I = \frac{k}{(\mu+\gamma)}\left(\frac{\mu N}{(\mu+k)} - \frac{(\mu+\theta)(\mu+\gamma)}{\beta k}\right)$$

$$I = \frac{\mu kN}{(\mu+\gamma)(\mu+k)} - \frac{k(\mu+\theta)(\mu+\gamma)}{\beta k(\mu+\gamma)}$$

$$I = \frac{\mu kN}{(\mu+\gamma)(\mu+k)} - \frac{(\mu+\theta)}{\beta k} \tag{3.17}$$

$$I = \frac{\mu\beta kN-(\mu+\theta)(\theta+\gamma)(\mu+k)}{\beta k(\mu+\gamma)(\mu+k)}$$

From (3.8) $R = \frac{\gamma I}{\mu} + \frac{\theta S}{\mu}$, Putting (3.11) and (3.14) into R, we get

$$R = \frac{\gamma}{\mu}\left(\frac{\mu k N}{(\mu+\gamma)(\mu+k)} - \frac{(\mu+\theta)}{\beta}\right) + \frac{\theta}{\mu}\left(\frac{(\mu+k)(\mu+\gamma)}{\beta k}\right)$$

$$R = \frac{\gamma k N}{(\mu+\gamma)(\mu+k)} - \frac{\gamma(\mu+\theta)}{\mu\beta} + \frac{\theta(\mu+k)(\mu+\gamma)}{\mu\beta k}$$

$$R = \frac{\mu\gamma\beta k N - \gamma k(\mu+\theta)(\mu+\gamma)(\mu+k) + \theta((\mu+k)(\mu+\gamma))^2}{\mu\beta k(\mu+\gamma)(\mu+k)} \quad (3.18)$$

The equilibrium point of chickenpox with vaccination is $(S^*, E^*, I^*, R^*) =$

$$\left(\frac{(\mu+\gamma)(\mu+k)}{\beta k}, \frac{\mu\beta k N - (\mu+\theta)(\mu+\gamma)(\mu+k)}{\beta k(\mu+k)}, \frac{\mu\beta k N - (\mu+\theta)(\mu+\gamma)(\mu+k)}{\beta k(\mu+\gamma)(\mu+k)}, \frac{\mu\gamma\beta k N - \gamma k(\mu+\theta)(\mu+\gamma)(\mu+k) + \theta((\mu+k)(\mu+\gamma))^2}{\mu\beta k(\mu+\gamma)(\mu+k)}\right)$$

3.5.1 Stability Analysis of the Diseases-Free Equilibrium point of Chickenpox Transmission with Vaccination

We linearized equations (3.8), (3.9), (3.10) and (3.11) at the point where S = 1and I = 0 to obtain their jacobian matrix

$$\frac{dS}{dt} = \mu N - \beta S I - \mu S - \theta S$$

$$\frac{dE}{dt} = \beta S I - (\mu + k)E$$

$$\frac{dR}{dt} = I\gamma - \mu R + \theta S$$

$$J = \begin{vmatrix} -\beta I - (\mu+\theta) & 0 & -\beta S & 0 \\ 0 & -(\mu+k) & \beta S & 0 \\ 0 & k & -(\mu+\gamma) & 0 \\ \theta & 0 & \gamma & -\mu \end{vmatrix}$$

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

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

Finding the determinant of the 4x4, we get

$$Det(J-\lambda I)=(\mu+\lambda)\begin{vmatrix}(\mu+\theta)+\lambda & 0 & \beta\\ 0 & (\mu+\kappa)+\lambda & -\beta\\ 0 & -\kappa & (\mu+\gamma)+\lambda\end{vmatrix} \tag{3.19}$$

$$Det(J-\lambda I)=(\mu+\lambda)\big[(\mu+\theta)+\lambda\big)\big((\mu+\kappa)+\lambda\big)(\mu+\gamma)+\lambda)-\kappa\beta)\big]-0+0$$

$$=(\mu+\lambda)\big[(\mu+\theta)+\lambda)((\mu+k)(\mu+\gamma)+\lambda(\mu+k)+\lambda(\mu+\gamma)+\lambda^2-\kappa\beta)\big]=(\mu+$$

$$\lambda)[((\mu+\theta)+\lambda)((\lambda^2+\lambda(2\mu+k+\gamma)+(\mu+k)(\mu+\gamma)-k\beta)]$$

$$=(\mu+\lambda)\big[\lambda^2(\mu+\theta)\big]+\lambda(\mu+\theta)(2\mu+k+\gamma)+(\mu+\theta)(\mu+k)(\mu+\gamma)-k\beta(\mu+\theta)+$$

$$\lambda^3+\lambda^2(2\mu+k+\gamma)+\lambda(\mu+k)(\mu+\gamma)-\lambda k\beta]$$

$$=(\mu+\lambda)[(\lambda^3+\lambda^2((\mu+\theta)+\lambda^2(2\mu+k+\gamma)+\lambda(\mu+\theta)(2\mu+k+\gamma)+\lambda(\mu+k)(\mu+$$

$$\gamma)-\lambda k\beta+(\mu+\theta)(\mu+k)(\mu+\gamma)-k\beta(\mu+\theta)$$

$$=\mu\lambda^3+\mu\lambda^2(3\mu+\theta+\kappa+\gamma)+\mu\lambda\big((\mu+\theta)(2\mu+k+\gamma)+(\mu+k)(\mu+\gamma)-k\beta)\big)+$$

$$\mu(\mu+\theta)(\mu+k)(\mu+\gamma)-k\beta(\mu+\theta)+\lambda^4+\lambda^3(3\mu+\theta+\kappa+\gamma)+\lambda^2((\mu+\theta)(2\mu+$$

$$k+\gamma)+(\mu+k)(\mu+\gamma)-k\beta)+\lambda(\mu+\theta)(\mu+k)(\mu+\gamma)-k\beta(\mu+\theta)=\lambda^4+$$

$$\lambda^3(4\mu+\theta+\kappa+\gamma)+\lambda^2((3\mu^2+\mu\theta+\mu\kappa+\mu\gamma)+\big((\mu+\theta)(2\mu+k+\gamma)+$$

$$(\mu+k)(\mu+\gamma)-k\beta)\big)+\lambda((\mu+\theta)(2\mu+k+\gamma)+(\mu+k)(\mu+\gamma)-k\beta)+(\mu+$$

$$\theta)\big((\mu+k)(\mu+\gamma)-k\beta)\big)+(\mu^2+\mu\theta)\big((\mu+k)(\mu+\gamma)-k\beta)\big).$$

We let

$a = (4\mu + \theta + \kappa + \gamma),$

$b = (3\mu^2 + \mu\theta + \mu\kappa + \mu\gamma) + ((\mu + \theta)(2\mu + k + \gamma) + (\mu + k)(\mu + \gamma) - k\beta)$

$c = (\mu + \theta)(2\mu + k + \gamma) + (\mu + k)(\mu + \gamma) - k\beta) + (\mu + \theta)((\mu + k)(\mu + \gamma) - k\beta))$

$d = (\mu^2 + \mu\theta)((\mu + k)(\mu + \gamma) - k\beta)$

$\text{Det}(J - \lambda I) = \lambda^4 + a\lambda^3 + b\lambda^2 + c\lambda + d$

But $\text{Det}(J - \lambda I) = 0$

$\lambda^4 + a\lambda^3 + b\lambda^2 + c\lambda + d = 0$

3.5.2 Stability Analysis of the Endemic Equilibrium Points of Chickenpox Transmission with Vaccination

Whenever R> 1, the system has endemic equilibrium due to introduction of secondary infection into the system. We consider the case where I≠ 0 then we linearize equations (3.6), (3.7) and (3.8) to obtain the jacobian matrix

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

$\frac{dE}{dt} = \beta SI - (\mu + k)E$

$\frac{dI}{dt} = kE - (\mu + \gamma)I$

$$J = \begin{vmatrix} -\beta I - (\mu + \theta) & 0 & -\beta S \\ \beta I & -(\mu + k) & \beta S \\ 0 & k & -(\mu + \gamma) \end{vmatrix}$$

$$J - \lambda I = \begin{vmatrix} -\beta \mathrm{I} - (\mu + \theta) & 0 & \beta S \\ \beta I & -(\mu + k) & \beta S \\ 0 & k & -(\mu + \gamma) \end{vmatrix} - \begin{vmatrix} \lambda & 0 & 0 \\ 0 & \lambda & 0 \\ 0 & 0 & \lambda \end{vmatrix}$$

$$= \begin{vmatrix} -\beta I - (\mu + \theta) - \lambda & 0 & -\beta S \\ \beta I & -(\mu + k) - \lambda & \beta S \\ 0 & k & -(\mu + \gamma - \lambda \end{vmatrix} \tag{3.20}$$

$$Det(J - \lambda I) =$$

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

$$= (\beta I + (\mu + \theta) + \lambda)((\mu + k) + \lambda)(\mu + \gamma) + \lambda) - k\beta S) - k\beta^2 SI$$

$$= (\beta I + (\mu + \theta) + \lambda)\big((\mu + k)(\mu + \gamma) + \lambda(\mu + k) + \lambda(\mu + \gamma) + \lambda^2 - k\beta S\big) - k\beta^2 SI$$

$$= (\beta I + (\mu + \theta) + \lambda)(\lambda^2 + \lambda(2\mu + k + \gamma) + (\mu + k)(\mu + \gamma) - k\beta S) - k\beta^2 SI$$

$$= \lambda^2 \beta \mathrm{I} + \lambda \beta \mathrm{I}(2\mu + \mathrm{k} + \gamma) + \beta \mathrm{I}(\mu + \mathrm{k})(+\gamma) - k\beta^2 SI + +\lambda(\mu + \theta)(2\mu + k + \gamma) +$$

$$(\mu + \theta)(\mu + k)(\mu + \gamma) - k\beta S(\mu + \theta) + \lambda^3 + \lambda^2(2\mu + k + \gamma) + \lambda(\mu + k)(\mu + \gamma) -$$

$$\lambda k\beta S - k\beta^2 SI$$

$$\mathrm{Det}(J - \lambda I) = \lambda^3 + \lambda^2((3\mu + k + \theta + \gamma + \beta I) + (\mu + k)(\mu + \gamma) - k\beta S) +$$

$$\lambda(\beta I(2\mu + k + \gamma) + (\mu + k)(\mu + \gamma) - k\beta S) + (\mu + \theta)((2\mu + k + \gamma) + (\mu + k)(\mu +$$

$$\gamma) - k\beta S)) - 2k\beta^2 SI$$

From equations (3.14), (3.15), (3.16) and (3.17)

$$E = \frac{(\mu+k)I}{k}$$

$$\beta S = \frac{(\mu+k)(\mu+\gamma)}{k}$$

$$I = \frac{(\mu\beta kN-(\mu+k)(\mu+\theta)(\mu+\gamma)}{(\mu+\gamma)(\mu+k)},.$$

$$E = \frac{(\mu\beta kN-(\mu+k)(\mu+\theta)(\mu+\gamma}{k\beta(\mu+k)(\mu+\gamma)}.$$

Putting these equations into the determinant, We get

$$Det(J-\lambda I) = \lambda^3 + \lambda^2\left((3\mu+\kappa+\theta) + \frac{\mu\beta kN-(\mu+k)(\mu+\theta)(\mu+\gamma)}{(\mu+\gamma)(\mu+k)}\right) + (\mu+\gamma)(\mu+k) -$$

$$\frac{k(\mu+k)(\mu+\gamma)}{k}) + \lambda(\frac{(\mu\beta kN-(\mu+k)(\mu+\theta)(\mu+\gamma)}{(\mu+\gamma)(\mu+k)})(2\mu+k+\gamma) + (\mu+\gamma)(\mu+k) -$$

$$\frac{k(\mu+k)(\mu+\gamma)}{k}) + (\mu+\theta)(2\mu+k+\gamma) + (\mu+k)(\mu+\gamma) - \frac{k(\mu+k)(\mu+\gamma)}{k})$$

$$-2\frac{(\mu+\gamma)(\mu+k)(\mu\beta kN-(\mu+k)(\mu+\theta)(\mu+\gamma)}{(\mu+\gamma)(\mu+k)}$$

$$Det(J-\lambda I) = \lambda^3 + \lambda^2 + \lambda\left((2\mu+k+\gamma)\left(\frac{(\mu\beta kN-(\mu+k)(\mu+\theta)(\mu+\gamma)}{(\mu+\gamma)(\mu+k)}\right)\right) + (\mu+\theta)(2\mu+$$

$$k+\gamma) - 2(\mu\beta kN-(\mu+k)(\mu+\theta)(\mu+\gamma)$$

We let

$$x = (3\mu+k+\theta+\gamma) + \frac{(\mu\beta kN-(\mu+k)(\mu+\theta)(\mu+\gamma)}{(\mu+\gamma)(\mu+k)}$$

$$y = (2\mu+k+\gamma)(\frac{(\mu\beta kN-(\mu+k)(\mu+\theta)(\mu+\gamma)}{(\mu+\gamma)(\mu+k)})$$

$$z = (\mu+\theta)(2\mu+k+\gamma) - 2(\mu\beta kN-(\mu+k)(\mu+\theta)(\mu+\gamma)$$

$$Det\ (J - \lambda I) = \lambda^3 + x\lambda^2 + y\lambda + z$$

But $Det\ (J - \lambda I) = 0$

$$\lambda^3 + x\lambda^2 + y\lambda + z = 0$$

3.6 HERD IMMUNITY THRESHOLD (H_I)

Herd immunity refers to the resistance of group of susceptible population by an infectious disease due to the immunity of large proportion of the susceptible population. Herd immunity prevents the occurrence of disease in a population. Herd immunity threshold is the percentage of the susceptible population that needs to be vaccinated to control disease transmission. The herd immunity equation is given by Diakmann and Heesterbeek as

$$H_I = 1 - \frac{1}{R_0} \text{ (Johnson, 2009)} \qquad (3.21)$$

where R_0 is the reproductive number of chickenpox without vaccination

CHAPTER 4

MODEL ANALYSIS AND DISCUSSION OF RESULTS

In this chapter, we analyze the problem discussed in the previous chapter. The initial population of Agona West Municipal according Ghana Statistical Service (GSS, 2010) was 115,358. The data collected from Agona West Municipal Health Directorate at Agona Swedru shows that a total of 60,684 people attended Hospital in 2007 and we consider them as susceptibles and 23 people were infected with chickenpox and they are infectives. From appendix 1, S_o= 60,684, I_o= 23 and S_∞= 31,527 where S_o is the initial number of Susceptible, I_o is the initial number of infective and S_∞ is the final number of Susceptible in the Agona West Municipality.

We measure time in years and the data is from January, 2007 to December, 2011. So we let $t_o = 0$ as January 2007 and $t_{end} = 4$ as December, 2011as shown in appendix 1.

The parameter values for the study of the epidemiology of chickenpox were obtained from appendix 1 and presented in the Table 4:1 below

parameter	Description	value
β	Transmission rate	0.026
κ	Latency rate	0.036
γ	Recovery rate	0.014
μ	Death rate	0.020
θ	Vaccinated rate coefficient	0.00019
N	Population	115,358

Table 4.1: shows the parameter values obtained from Agona West Municipality

4.1 ESTIMATION OF REPRODUCTIVE NUMBER OF CHICKENPOX WITHOUT VACCINATION

Substituting the values of β and γ in Table 4.1 into equation (3.5) yield

$$R_0 = \frac{0.026}{0.014}$$

$$R_0 = 1.8571$$

$$R_0 \simeq 2$$

This implies that on average 2 people contracted chickenpox in Agona West Municipality.

4.2 ANALYSIS OF THE EQUILIBRIA

4.2.1 Stability Analysis of the Disease Free Equilibrium point of Chickenpox Transmission without Vaccination

Substituting the values of κ, γ and β into equation (3.6), we obtain

$$J-\lambda I=\begin{vmatrix} -\lambda & 0 & -0.026 & 0 \\ 0 & -0.036-\lambda & 0.026 & 0 \\ 0 & 0.0.036 & -0.014-\lambda & 0 \\ 0 & 0 & 0.014 & -\lambda \end{vmatrix}$$

$$J-\lambda I=\begin{vmatrix} \lambda & 0 & 0.026 & 0 \\ 0 & 0.036+\lambda & -0.026 & 0 \\ 0 & -0.036 & 0.014+\lambda & 0 \\ 0 & 0 & -0.014 & \lambda \end{vmatrix}$$

$$Det\,(J-\lambda I)=\lambda\begin{vmatrix} 0.0.36+\lambda & -0.026 & 0 \\ -0.036 & 0.014+\lambda & 0 \\ 0 & -0.014 & \lambda \end{vmatrix}$$

$$\lambda((0.036+\lambda)\begin{vmatrix} 0.014+\lambda & 0 \\ -0.014 & \lambda \end{vmatrix}-(0.036)\begin{vmatrix} -0.026 & 0 \\ 0.014 & \lambda \end{vmatrix}+0)$$

$$Det\,(J-\lambda I)=\lambda((0.036+\lambda)(0.014\lambda+\lambda^2)+\lambda(0.036x0.026))$$

$$Det\,(J-\lambda I)=0$$

$$\lambda(5.04\times10^{-4}\lambda+0.036\lambda^2+0.014\lambda^2+\lambda^3+9.36\times10^{-4}\lambda)=0$$

$$\lambda_1=0$$

$$5.04 \times 10^{-4}\lambda + 0.036\lambda^2 + 0.014\lambda^2 + \lambda^3 + 9.36 \times 10^{-4}\lambda = 0$$

$$\lambda(5.04 \times 10^{-4} + 0.036\lambda + 0.014\lambda + \lambda^2 + 9.36 \times 10^{-4} = 0$$

$$\lambda_2 = 0$$

$$\lambda^2 + 0.05\lambda + 1.44 \times 10^{-3} = 0$$

$$\lambda_3 = -0.025 + 0.0285i$$

$$\lambda_4 = -0.025 - 0.0285i$$

Since the real parts of $\lambda_3 = \lambda_4 < 0$ and $\lambda_1 = \lambda_2 = 0$, the disease free equilibrium point is a centre.

4.2.2 Stability Analysis of the Endemic Equilibrium point of Chickenpox Transmission without Vaccination

Substituting the values of κ, γ and β into equation (3.7), we obtain

$$Det(J - \lambda I) = \begin{vmatrix} -\beta \mathrm{I} - \lambda & 0 & -\beta S & 0 \\ 0 & -0.036 - \lambda & \beta S & 0 \\ 0 & 0.036 & -0.014 - \lambda & 0 \\ 0 & 0 & 0.014 & -\lambda \end{vmatrix}$$

$$Det(J - \lambda I) = \begin{vmatrix} \beta \mathrm{I} + \lambda & 0 & \beta S & 0 \\ 0 & 0.036 + \lambda & -\beta S & 0 \\ 0 & -0.036 & 0.014 + \lambda & 0 \\ 0 & 0 & -0.014 & \lambda \end{vmatrix}$$

$$Det(J - \lambda I) = (\beta I + \lambda) \begin{vmatrix} 0.036 + \lambda & -\beta S & 0 \\ -0.036 & 0.014 + \lambda & 0 \\ 0 & -0.014 & \lambda \end{vmatrix}$$

But $\beta I = 0$ and $\beta S = 0$

$$Det(J - \lambda I) = (\lambda)((0.036 + \lambda)\begin{vmatrix} 0.014 + \lambda & 0 \\ -0.0144 & \lambda \end{vmatrix} - (-0.036)\begin{vmatrix} 0 & 0 \\ -0.014 & \lambda \end{vmatrix})$$

$$Det(J - \lambda I) = (\lambda)((0.036 + \lambda)(0.014\lambda + \lambda^2 + 0) + 0.036)$$

But $Det(J - \lambda I) = 0$

$$\lambda(5.04 \times 10^{-3}\lambda + 0.036\lambda^2 + 0.014\lambda^2 + \lambda^3) = 0$$

$$\lambda_1 = 0$$

$$5.04 \times 10^{-3}\lambda + 0.036\lambda^2 + 0.014\lambda^2 + \lambda^3 = 0$$

$$\lambda(5.04 \times 10^{-3} + 0.036\lambda + 0.014\lambda + \lambda^2) = 0$$

$$\lambda_2 = 0$$

$$\lambda^2 + 0.05\lambda + 5.04 \times 10^{-3} = 0$$

$\lambda_3 = -0.025 + 0.6645i$ and $\lambda_4 = -0.025 - 0.6645i$

Since the real parts of $\lambda_3 = \lambda_4 < 0$ and $\lambda_1 = \lambda_2 = 0$, the endemic equilibrium point is a centre.

4.2.3 The Basic Reproductive Number (R_o) of Chickenpox transmission with Vaccination

Substituting the values of $\beta, \kappa, \mu, \gamma$ into equation (3.12), we obtain

$$R_o = \frac{0.014 \text{ x } 0.036}{(0.02+0.036)(0.02+0.014)}$$

$$R_o = \frac{5.04\times10^{-4}}{(0.056)(0.034)}$$

$$R_o = 0.264706$$

$$R_o \simeq 0.3$$

Since $R_o \simeq 0.3 < 1$,

It implies that chickenpox dies out in Agona West Municipality and no person will contract chickenpox in the Municipality.

4.2.4 Stability Analysis of the Diseases-Free Equilibrium point of Chickenpox Transmission with Vaccination

Substituting the values of $\beta, \kappa, \mu, \gamma$ and θ into equation (3.19), we obtain

$$J - \lambda I = \begin{vmatrix} -(0.02+0.00019)-\lambda & 0 & -0.026 & 0 \\ 0 & -(0.02+0.036)-\lambda & 0.026 & 0 \\ 0 & 0.036 & -(0.02+0.014)-\lambda & 0 \\ 0.00019 & 0 & 0.014 & -0.02-\lambda \end{vmatrix}$$

$$J - \lambda I = \begin{vmatrix} (0.02+0.00019)+\lambda & 0 & 0.026 & 0 \\ 0 & (0.02+0.036)+\lambda & -0.026 & 0 \\ 0 & -0.036 & (0.02+0.014)+\lambda & 0 \\ -0.00019 & 0 & -0.014 & 0.02+\lambda \end{vmatrix}$$

$$Det(J-\lambda I) = (0.02+\lambda)\begin{vmatrix} 0.02019+\lambda & 0 & 0.026 \\ 0 & 0.056+\lambda & -0.026 \\ 0 & -0.036 & 0.034+\lambda \end{vmatrix} - 0 + 0$$

$$Det(J-\lambda I) = (0.02+\lambda)[(0.02019+\lambda)((0.056+\lambda)(0.034+\lambda) - 9.36\times10^{-3})]$$

$$Det(J-\lambda I) = (0.02+\lambda)((0.02019+\lambda)(1.904\times10^{-3} + 0.056\lambda + 0.034\lambda + \lambda^2 - 9.36\times10^{-3})$$

$$Det(J-\lambda I) = (0.02+\lambda)((0.02019+\lambda)(1.904\times10^{-3} + 0.09\lambda + \lambda^2)$$

$$Det(J-\lambda I) = (0.02+\lambda)(3.844176\times10^{-5} + 1.8171\times10^{-3}\lambda + 0.02019\lambda^2 + 1.904\times10^{-3}\lambda + 0.09\lambda^2 + \lambda^3)$$

$$Det(J-\lambda I) = (0.02+\lambda)(\lambda^3 + 0.11019\lambda^2 + 3.7211\times10^{-5}\lambda + 3.844176\times10^{-5})$$

$$Det(J-\lambda I) = 0.02\lambda^3 + 2.2038\times10^{-3}\lambda^2 + 7.4422\times10^{-7}\lambda + 7.688352\times10^{-7} + \lambda^4 + 0.11019\lambda^3 + 3.7211\times10^{-5}\lambda^2 + 3.844176\times10^{-5}\lambda)$$

$$Det(J-\lambda I) = \lambda^4 + 0.13019\lambda^3 + 2.241011\times10^{-3}\lambda^2 + 3.918598\times10^{-5}\lambda + +7.688352\times10^{-7}$$

but $Det(J-\lambda I) = 0$

$$= \lambda^4 + 0.13019\lambda^3 + 2.241011\times10^{-3}\lambda^2 + 3.918598\times10^{-5}\lambda + 7.688352\times10^{-7}$$

$\lambda_1 = 0.0200$

$\lambda_1 = 0.0202$

$\lambda_3 = 0.0125$

$\lambda_4 = 0.0775$

Since all the eigenvalues are positive, the disease free equilibrium point is unstable.

4.2.5 Stability Analysis of the Endemic Equilibrium Points of Chickenpox Transmission with Vaccination

Substituting the values of $\beta I, \beta S, \gamma, \theta, \mu$ and κ into equation (3.20), we obtain

$$(J - \lambda I) =$$

$$\begin{vmatrix} -0.021 - (0.02 + 0.00019) - \lambda & 0 & -0.053 \\ 0.021 & -(0.02 + 0.036) - \lambda & 0.053 \\ 0 & 0.036 & -(0.02 + 0.014 - \lambda) \end{vmatrix}$$

$$(J - \lambda I) =$$

$$\begin{vmatrix} 0.021 + (0.02 + 0.00019) + \lambda & 0 & 0.053 \\ -0.021 & (0.02 + 0.036) + \lambda & -0.053 \\ 0 & -0.036 & (0.02 + 0.014 + \lambda) \end{vmatrix}$$

$$(J - \lambda I) = \begin{vmatrix} 0.04199 + \lambda & 0 & 0.053 \\ -0.021 & 0.056 + \lambda & -0.053 \\ 0 & -0.036 & 0.034 + \lambda) \end{vmatrix}$$

$$Det(J - \lambda I) =$$

$$(0.04199 + \lambda) \begin{vmatrix} 0.056 + \lambda & -0.053 \\ -0.036 & 0.034 + \lambda \end{vmatrix} - 0 + (0.053) \begin{vmatrix} -0.021 & 0.053 + \lambda \\ 0 & -0.036 \end{vmatrix}$$

$$Det(J - \lambda I) = (0.04199 + \lambda)((0.056 + \lambda)(0.034 + \lambda) - 1.908x10^{-3}) +$$

$$(0.053)(7.56\,x10^{-3}))$$

$$Det(J-\lambda I) = (0.04199+\lambda)(1.904x10^{-3}+0.056\lambda+0.034\lambda+\lambda^2+4.0068$$

$$x10^{-4}-1.908x10^{-3})$$

$$Det(J-\lambda I) = (0.04199+\lambda)(0.056\lambda+0.034\lambda+\lambda^2+3.9668x10^{-4})$$

$$Det(J-\lambda I) = 2.35144x10^{-3}\lambda+1.42766x10^{-3}\lambda+0.04199\lambda^2+1.665693x10^{-5}+$$

$$0.09\lambda^2+\lambda^3+3.9668x10^{-4}\lambda)$$

$$Det(J-\lambda I) = \lambda^3+0.13199\lambda^2+3.9239x10^{-3}\lambda+1.665693x10^{-5}$$

But $Det(J-\lambda I) = 0$

$$\lambda^3+0.13199\lambda^2+3.9239x10^{-3}\lambda+1.665693x10^{-5}=0$$

$$\lambda_1 = -4.2867\times10^{-3}$$

$$\lambda_2 = -4.45613\times10^{-3}+0.06218i$$

$$\lambda_3 = -4.45613\times10^{-3}-0.06218i$$

Since the real parts of the eigenvalues are negative, the endemic equilibrium point of chickenpox transmission with vaccination is asymptotically stable. Chickenpox is persistent in the Municipality but it is under control as time changes.

4.2.6 HERD IMMUNITY THRESHOLD (H_I)

Herd immunity threshold is the percentage of the susceptible population that needs to be vaccinated to control disease transmission. Substituting R_o into equation (3.21), the percentage of people that needs to be vaccinated against chickenpox in the municipality is given by

$$H_I = 1 - \frac{1}{R_o}$$

$$H_I = \frac{1.8571-1}{1.8571}$$

$$H_I = 0.461526$$

$$H_I \simeq 46.2\%$$

More than 46.2% of the susceptible population in Agona West Municipality are given vaccine against chickenpox; the chickenpox will be under control for a long period of time. The number of people that needs to be vaccinated is $0.461526 \times 115358 = 53{,}240$ in order to control the chickenpox in the Municipality.

4.3 SENSITIVITY ANALYSIS OF SEIR WITHOUT VACCINATION

The parameter values for the sensitivity analysis are given below.

β	γ	κ
0.026	0.014	0.036
0.40	0.014	0.036
0.90	0.014	0.036

Table 4:2: Shows variation in β values keeping κ and γ constant at equilibrium

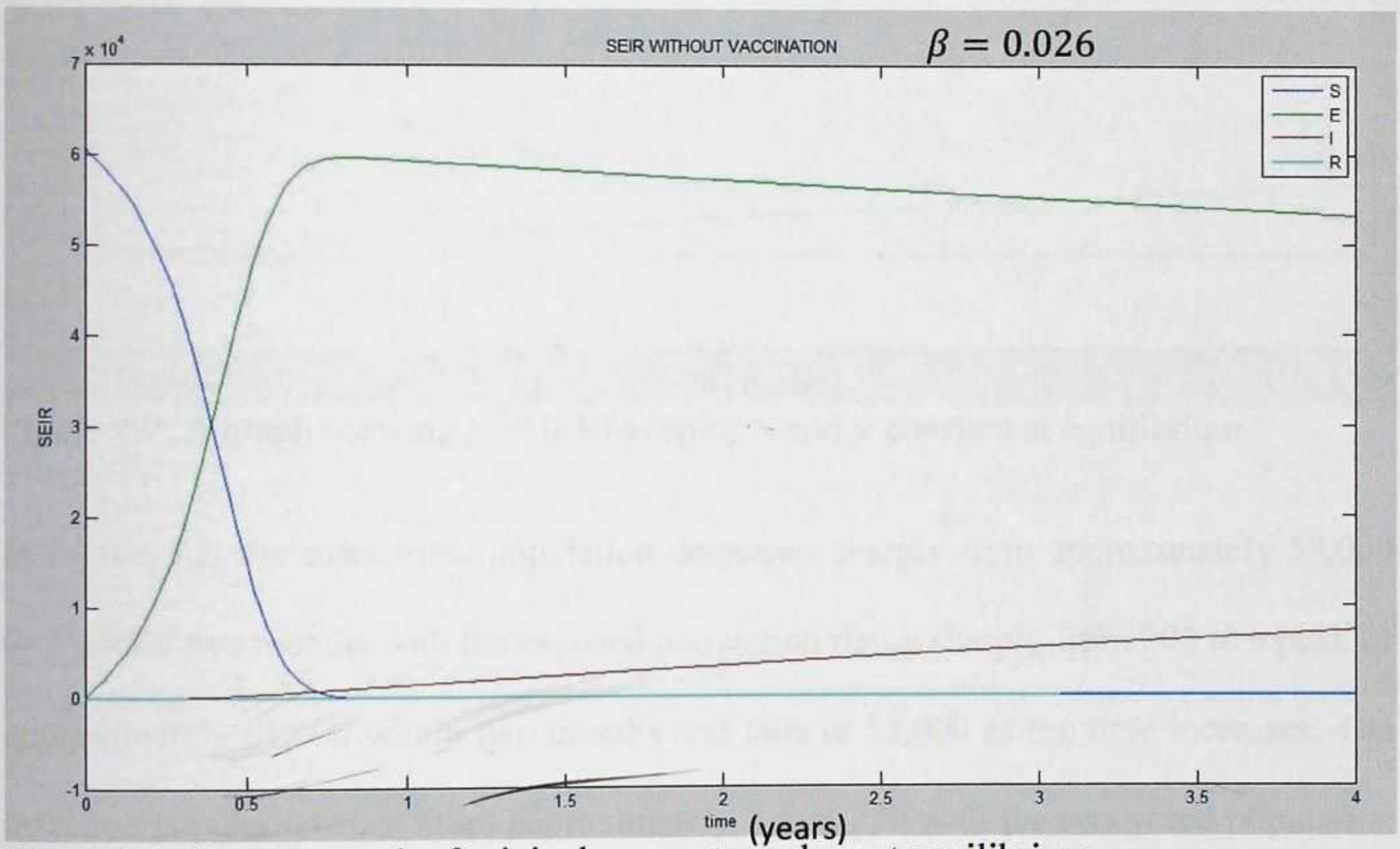

Figure 4.1: Shows a graph of original parameter values at equilibrium

The susceptible population decreases sharply from approximately 60,000 and asymptotically to zero as time increases within three years five and half months and the exposed increases from approximately 2 to a peak of 60,000 and decreases slowly to

53,000 as time increases from three years five months with infective population rising from 2 to 700. The recovered population is asymptotic to the time axis as the time increases from zero to 4 years.

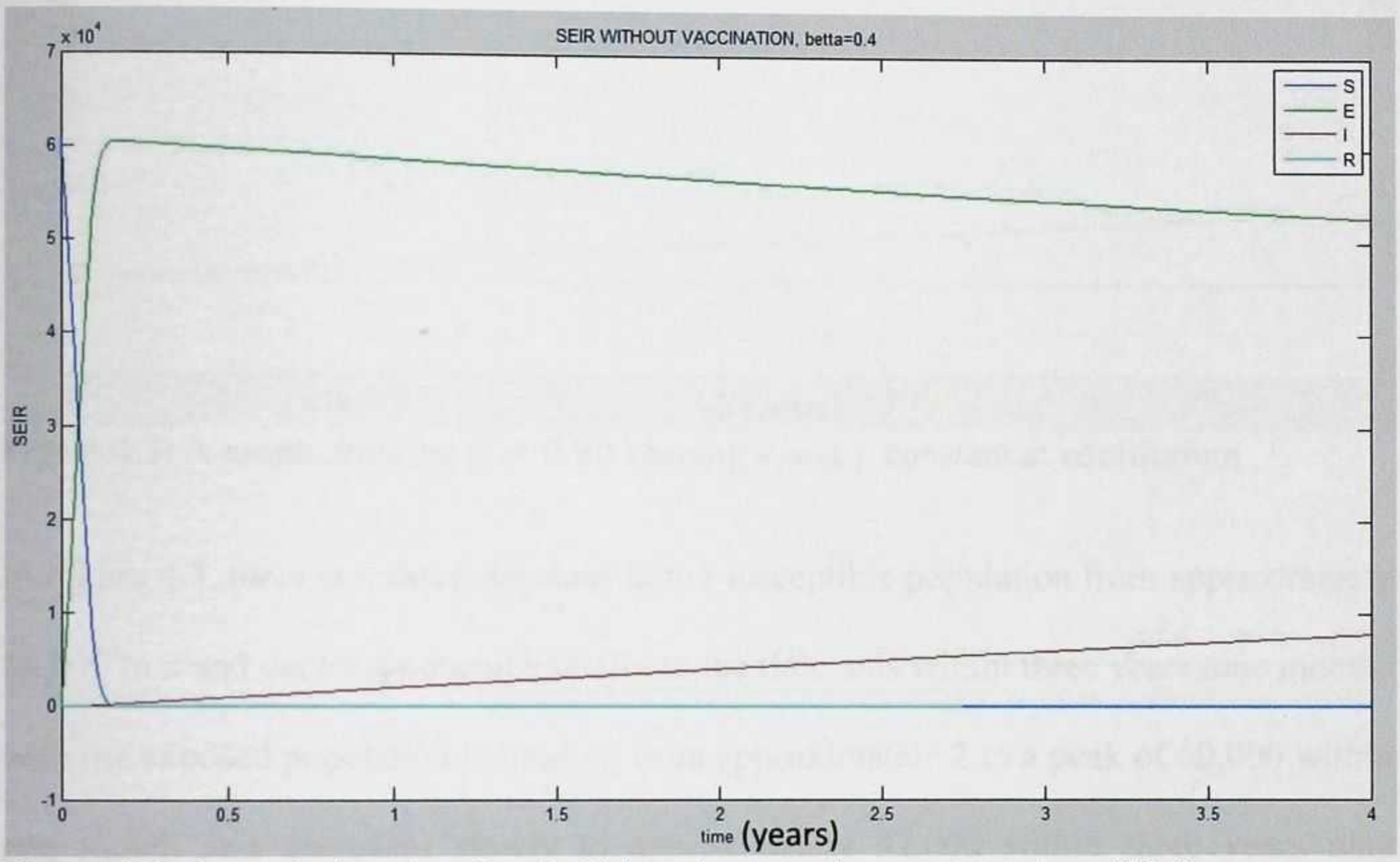

Figure 4.2: A graph showing $\beta = 0.40$ keeping κ and γ constant at equilibrium

In Figure 4.2, the susceptible population decreases sharply from approximately 58,000 to 2 within two months with the exposed population rising sharply from 200 to a peak of approximately 61,000 within two months and falls to 53,000 as the time increases. The infective population rises from approximately 2 to 1,750 with the recovered population being asymptotic to the time axis as time increases to 4 years.

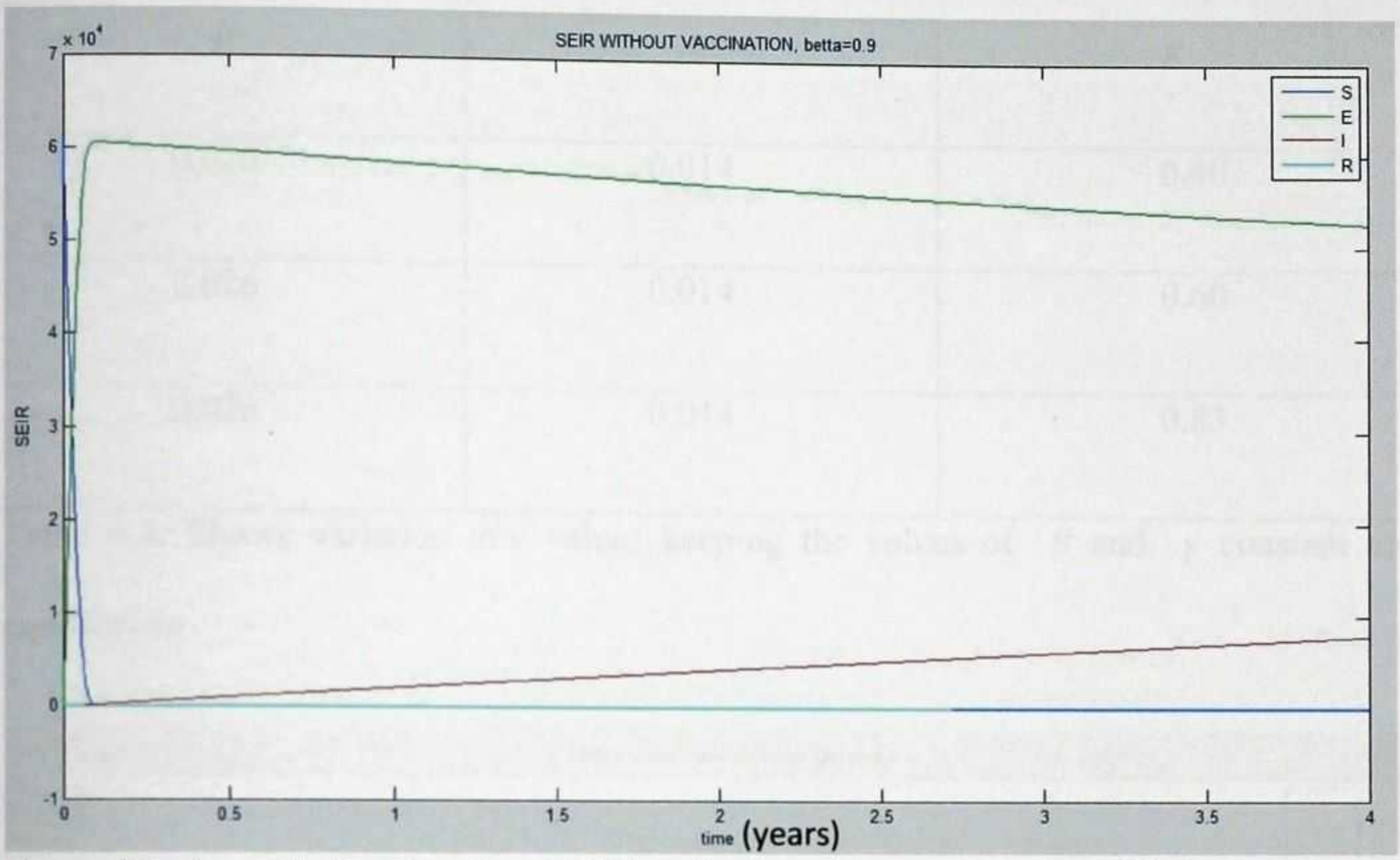

Figure 4.3: A graph showing $\beta = 0.90$ keeping κ and γ constant at equilibrium

In Figure 4.3, there is a sharp decrease in the susceptible population from approximately 56,000 to 2 and decreases asymptotically to the time axis within three years nine months with the exposed population increasing from approximately 2 to a peak of 60,000 within one month and decreases slowly to approximately 52,000 within three years nine months. The infective population increases from approximately 2 to 750 within 4 years and the recovered population increases asymptotically to the time axis.

We observed that the susceptible population decreases sharply with a sharp increase in the exposed with more people contracting the chickenpox with the recovered population being asymptotic to the time axis as β varies from 0.026 to 0.90.

β	γ	κ
0.026	0.014	0.40
0.026	0.014	0.60
0.026	0.014	0.83

Table 4.3: Shows variation in κ values keeping the values of β and γ constant at equilibrium

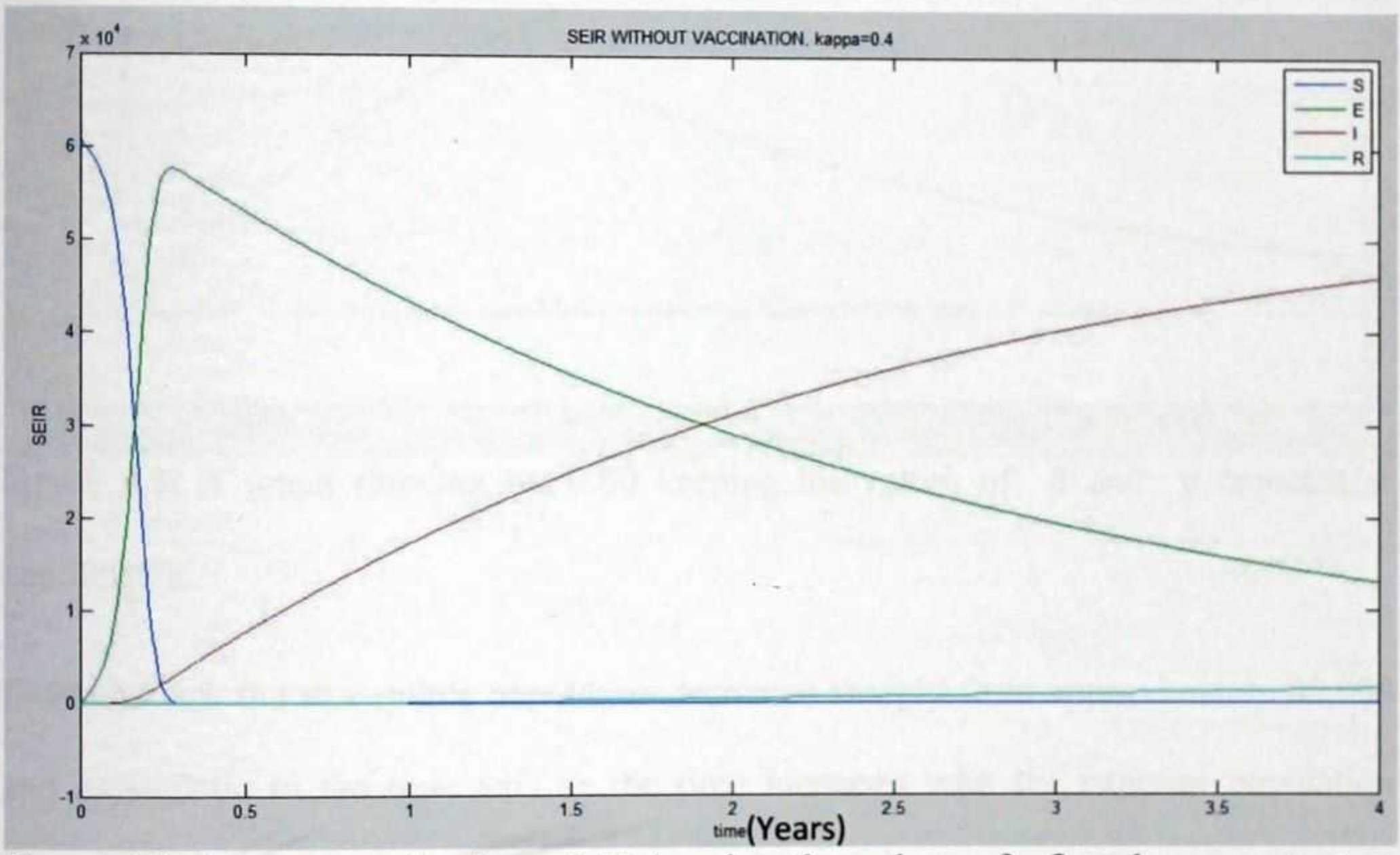

Figure 4.4 shows a graph of $\kappa = 0.40$ keeping the values of β and γ constant at equilibrium

From Figure 4.4, the susceptible population decreases sharply from approximately 60, 000 to 2 within two and half months as time increases while the exposed population rises from approximately 2 to a peak of 58,000 and decreases slowly to approximately 1,300 within three years seven and half months. The infective population increases

sharply from approximately 2 to 46,000 within 4 years with a gradual increase of 150 people recovering from chickenpox within 4 years.

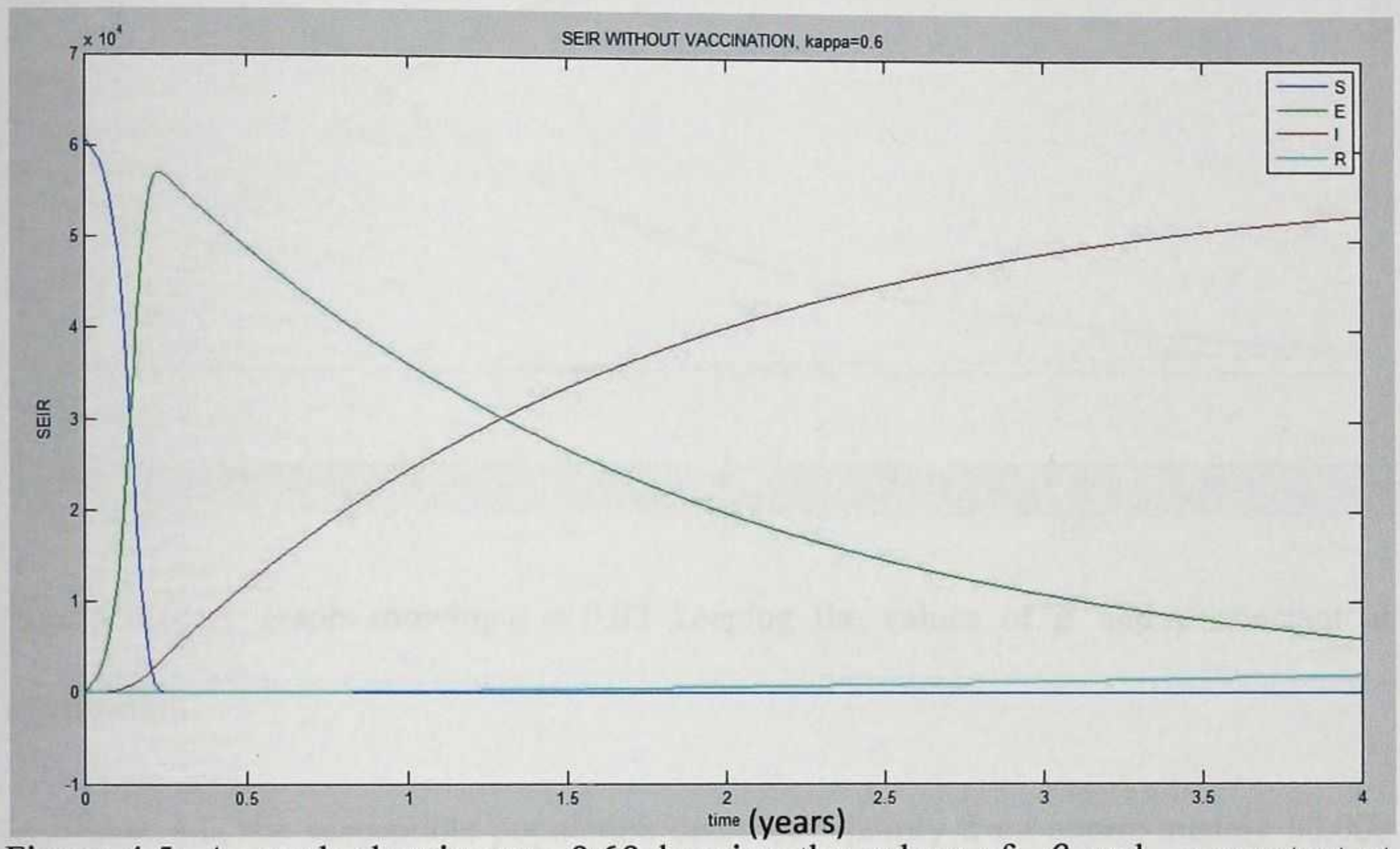

Figure 4.5: A graph showing $\kappa = 0.60$ keeping the values of β and γ constant at equilibrium

In Figure 4.5, the susceptible population decreases sharply from approximately 60,000 and asymptotic to the time axis as the time increases with the exposed population increasing from approximately 2 to a peak of 57,000 within two and half months and decreases slowly to approximately 600 within three years seven and half months with approximately 200 people recovering from chickenpox within 4 years.

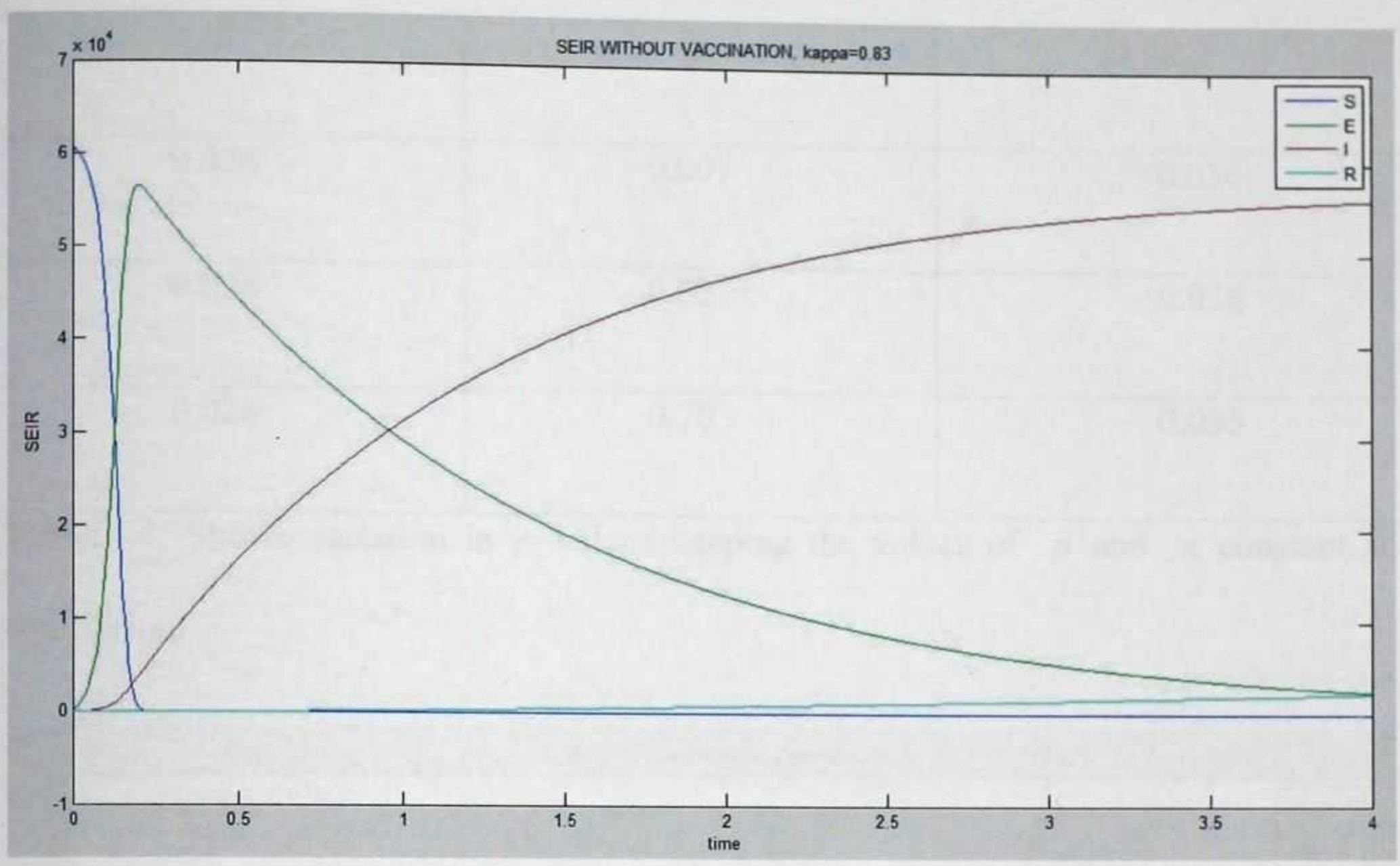

Figure 4.6: A graph showing $\kappa = 0.83$ keeping the values of β and γ constant at equilibrium

In Figure 4.6, the susceptible population decreases sharply from approximately 60,000 to 2 within three months with the exposed population rising from approximately 2 to a peak of 56,000 within three months and decreases sharply to approximately 200 in three years seven months. The infective population increases from approximately 2 to 56,000 within 4 years with the recovered population being asymptotic to the time axis within the 4 years.

We observed that as κ varies from 0.4 to 0.83, more people become exposed to chickenpox as a result chickenpox patient rose from approximately 46,000 to 56,000 with no significant change in the recovered population.

β	γ	κ
0.026	0.007	0.036
0.026	0.02	0.036
0.026	0.70	0.036

Table 4.4: Shows variation in γ values keeping the values of β and κ constant at equilibrium

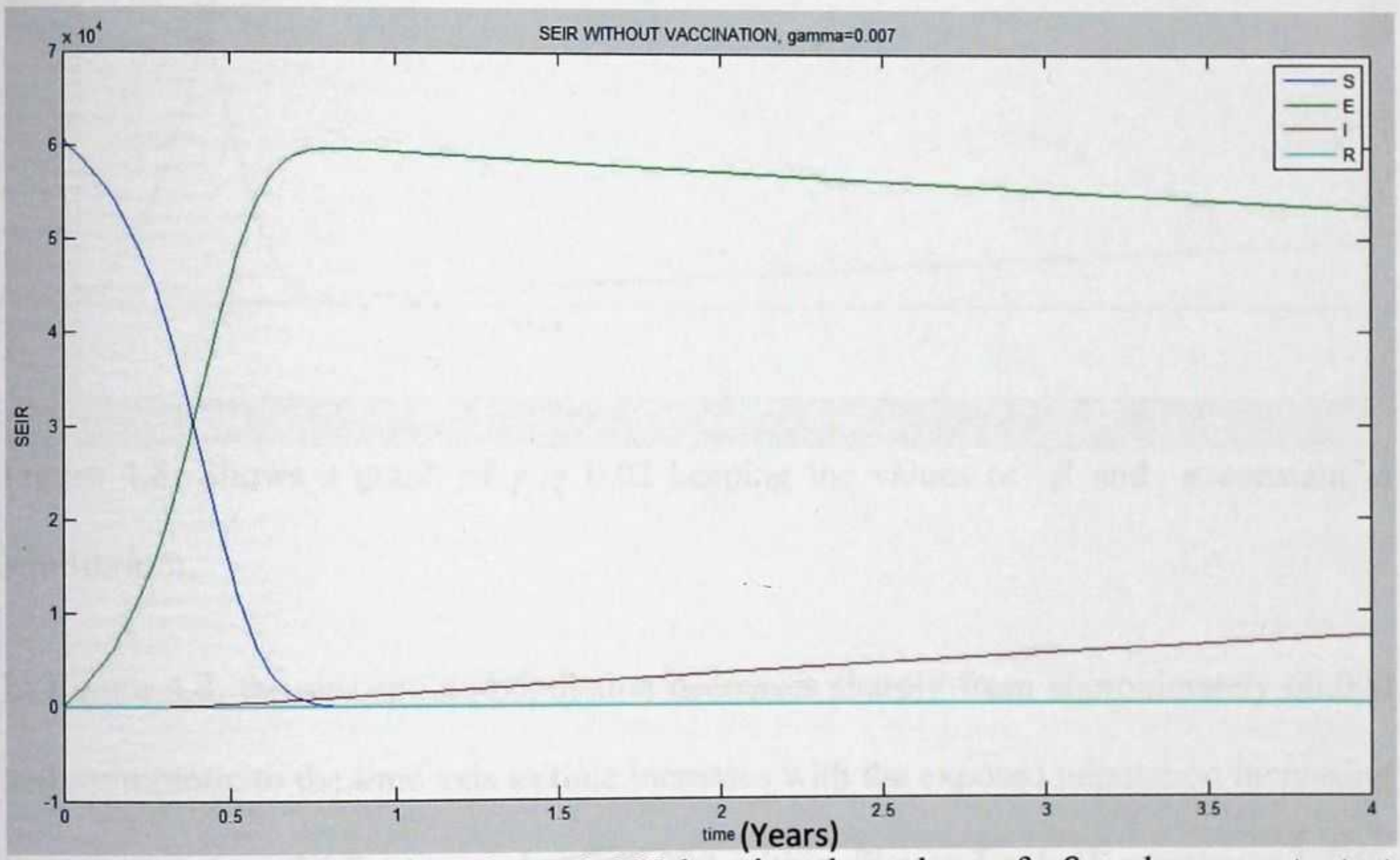

Figure 4.7: Shows a graph of $\gamma = 0.007$ keeping the values of β and κ constant at equilibrium.

In figure 4.7, the susceptible population decreases sharply from approximately 60,000 to 2 within five and half months as there is an increase in the exposed population from approximately 2 to a peak of 60,000 and decreases slowly to approximately 53,000 within three years five months. The infective population increases sharply from

approximately 2 to 700 with the recovered population being asymptotic to the time axis within the 4 years.

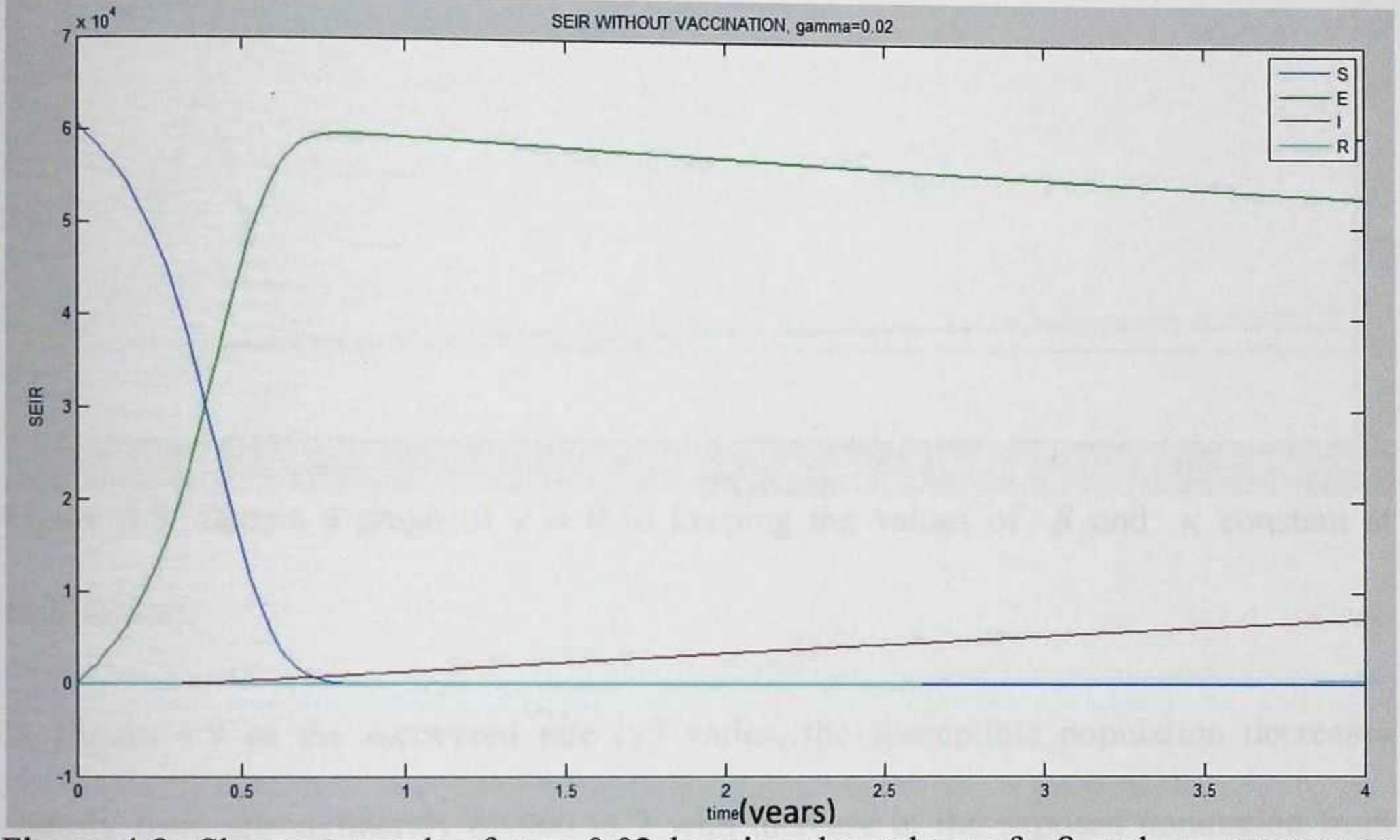

Figure 4.8: Shows a graph of $\gamma = 0.02$ keeping the values of β and κ constant at equilibrium.

In Figure 4.8, the susceptible population decreases sharply from approximately 60,000 and asymptotic to the time axis as time increases with the exposed population increasing from approximately 2 to a peak of 60,000 within five and a half months and falls gradually to approximately 53,000 within three years five month. The infective population increases from approximately 2 to 700 within 4 years the recovered population being asymptotic to the time axis as time increases.

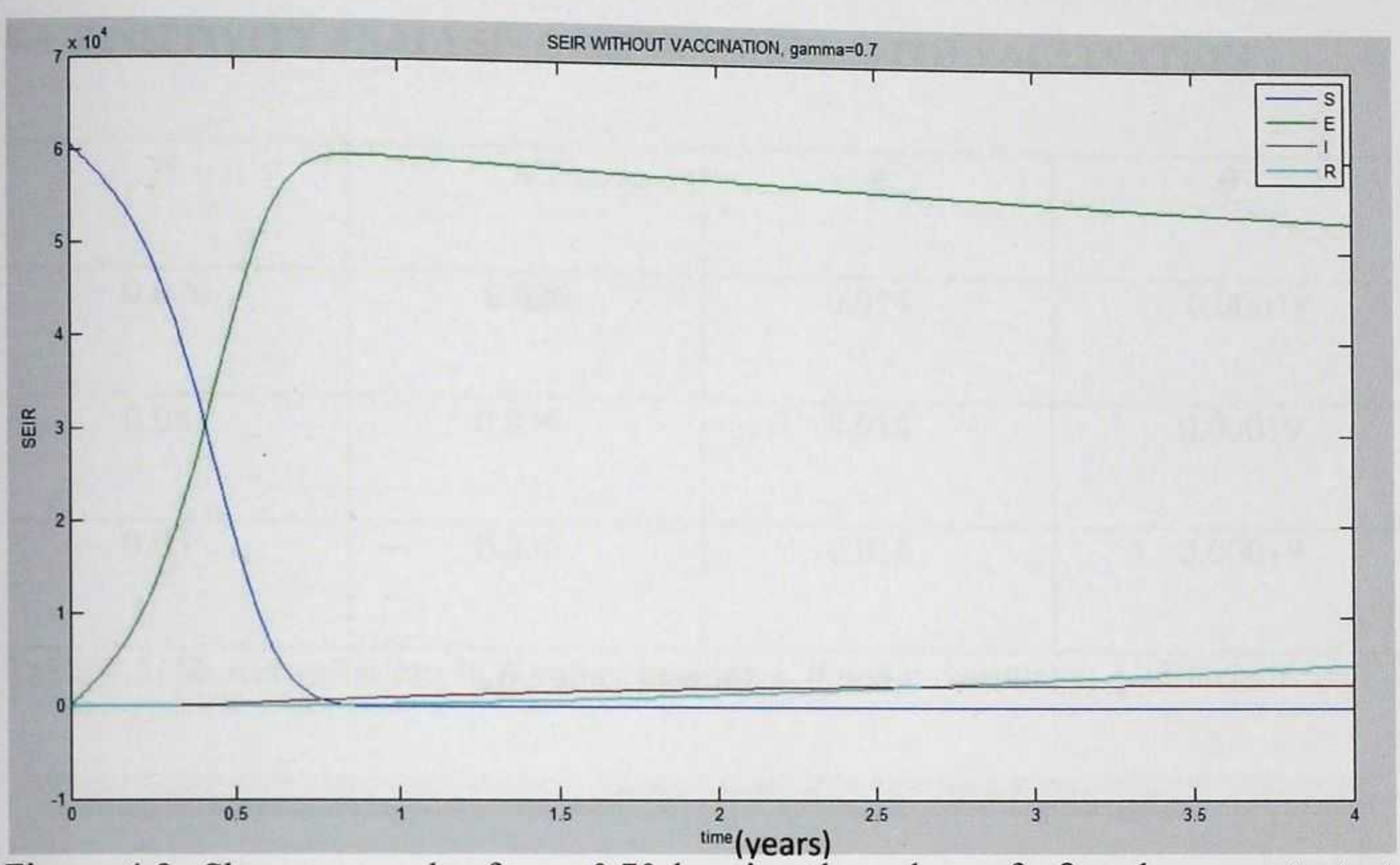

Figure 4.9: Shows a graph of $\gamma = 0.70$ keeping the values of β and κ constant at equilibrium.

In Figure 4.9 as the recovered rate (γ) varies, the susceptible population decreases sharply from approximately 60,000 to 2 with increase in the exposed population from approximately 2 to a peak of 60,000 within five and a half months and falls gradually to approximately 53,000 within three and a half years. The infective population decreases sharply from approximately 700 to 250 with the recovered population increasing sharply from approximately 2 to 450 within the 4 years.

We observed that as the recovered rate (γ) varies from 0.007 to 0.70, people who contracted chickenpox decreases with more people recovering.

4.4 SENSITIVITY ANALYSIS OF SEIR MODEL WITH VACCINATION

β	κ	γ	θ
0.026	0.036	0.014	0.00019
0.08	0.036	0.014	0.00019
0.65	0.036	0.014	0.00019

Table 4.5: Shows variations in β values keeping γ, θ and κ constant at equilibrium.

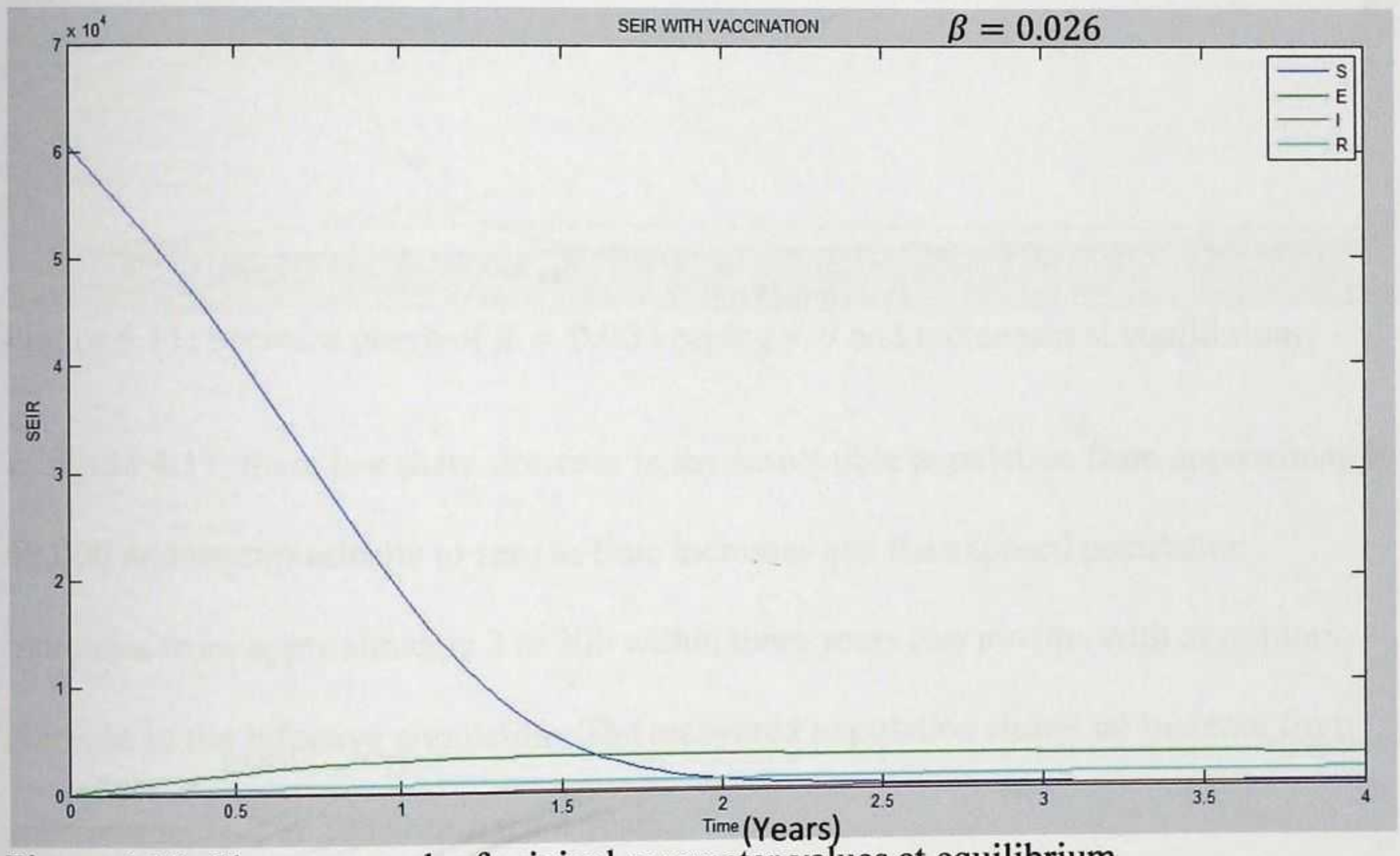

Figure 4.10: Shows a graph of original parameter values at equilibrium.

In figure 4.10, the susceptible population decreases sharply from approximately 60,000 and asymptotically to zero within two years five months with the exposed population increasing from approximately 2 to 300 within the 4 years. The infective population rises asymptotically to zero as time increases with the recovered population rising from

approximately 2 to 200 within the 4 years as the recovered population rises asymptotically to zero as time increases.

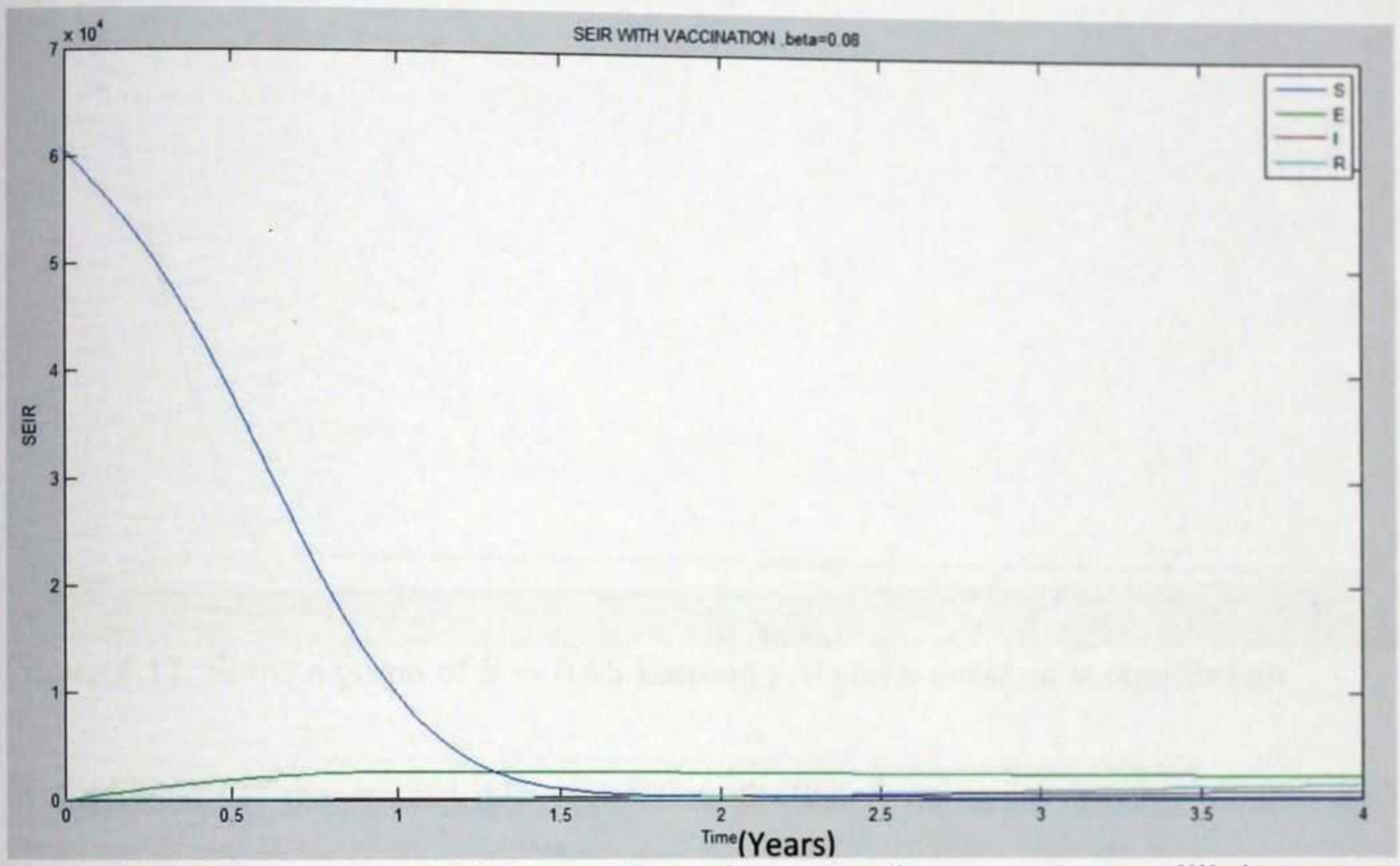

Figure 4.11: Shows a graph of $\beta = 0.08$ keeping γ, θ and κ constant at equilibrium

In figure 4.11, there is a sharp decrease in the susceptible population from approximately 60,000 and asymptotically to zero as time increases and the exposed population increases from approximately 2 to 300 within three years two months with asymptotic increase in the infective population. The recovered population shows no increase from approximately 2 to 200 within the 4 years.

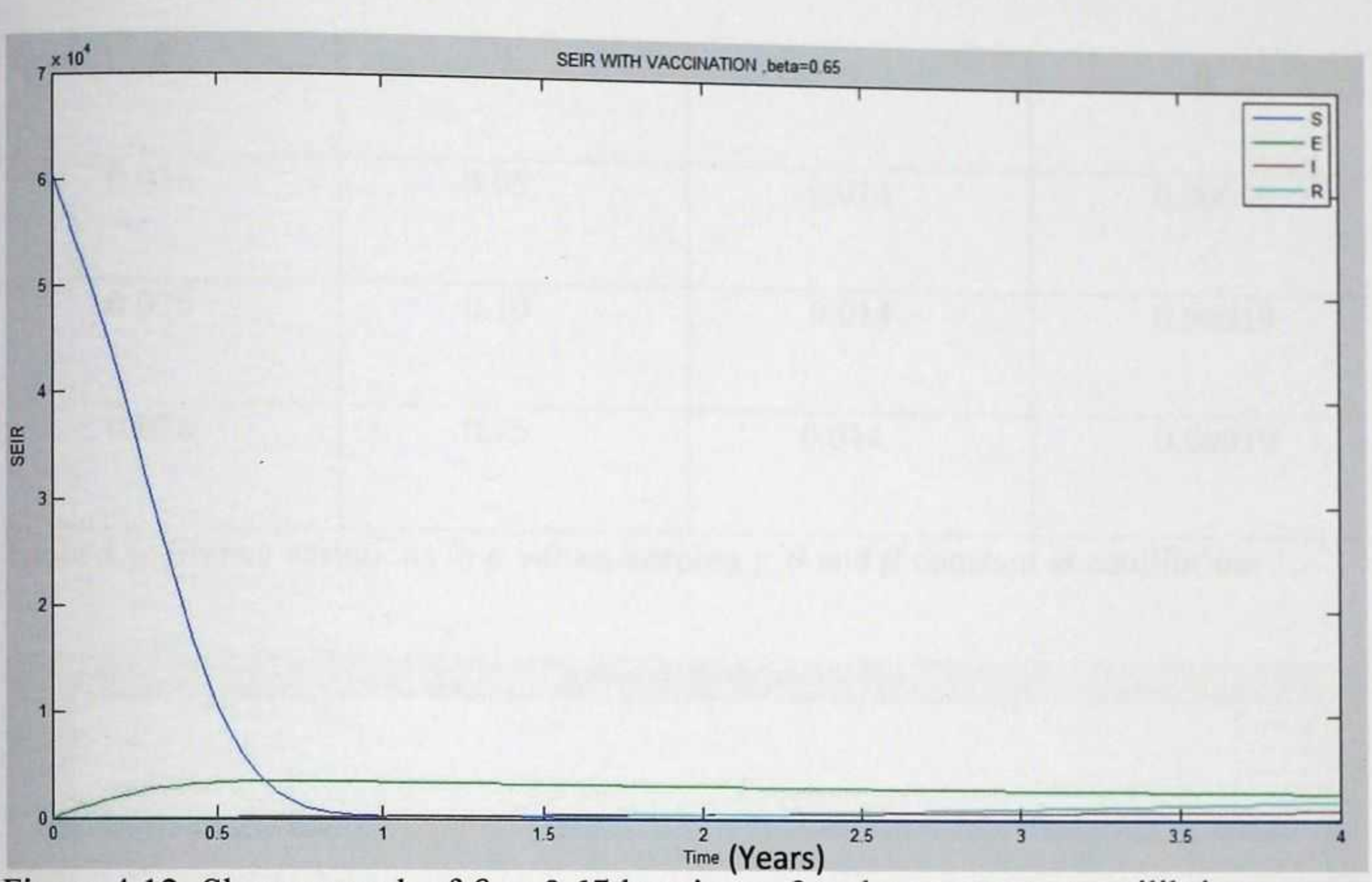

Figure 4.12: Show a graph of $\beta = 0.65$ keeping γ, θ and κ constant at equilibrium

In Figure 4.12, the susceptible population decline from approximately 60,000 and asymptotically to zero within 4 years and the infective population increases asymptotically to zero while the recovered population also increases asymptotically as time increases. The exposed population increases from approximately 2 to 300 as time increases.

We observed that as the value of β varies from 0.08 to 0.65, the susceptible population decreases sharply with time while the infective and recovered population also decreases asymptotically within the 4 years.

β	κ	γ	θ
0.026	0.05	0.014	0.00019
0.026	0.10	0.014	0.00019
0.026	0.75	0.014	0.00019

Table 4.6: Shows variations in κ values keeping γ, θ and β constant at equilibrium

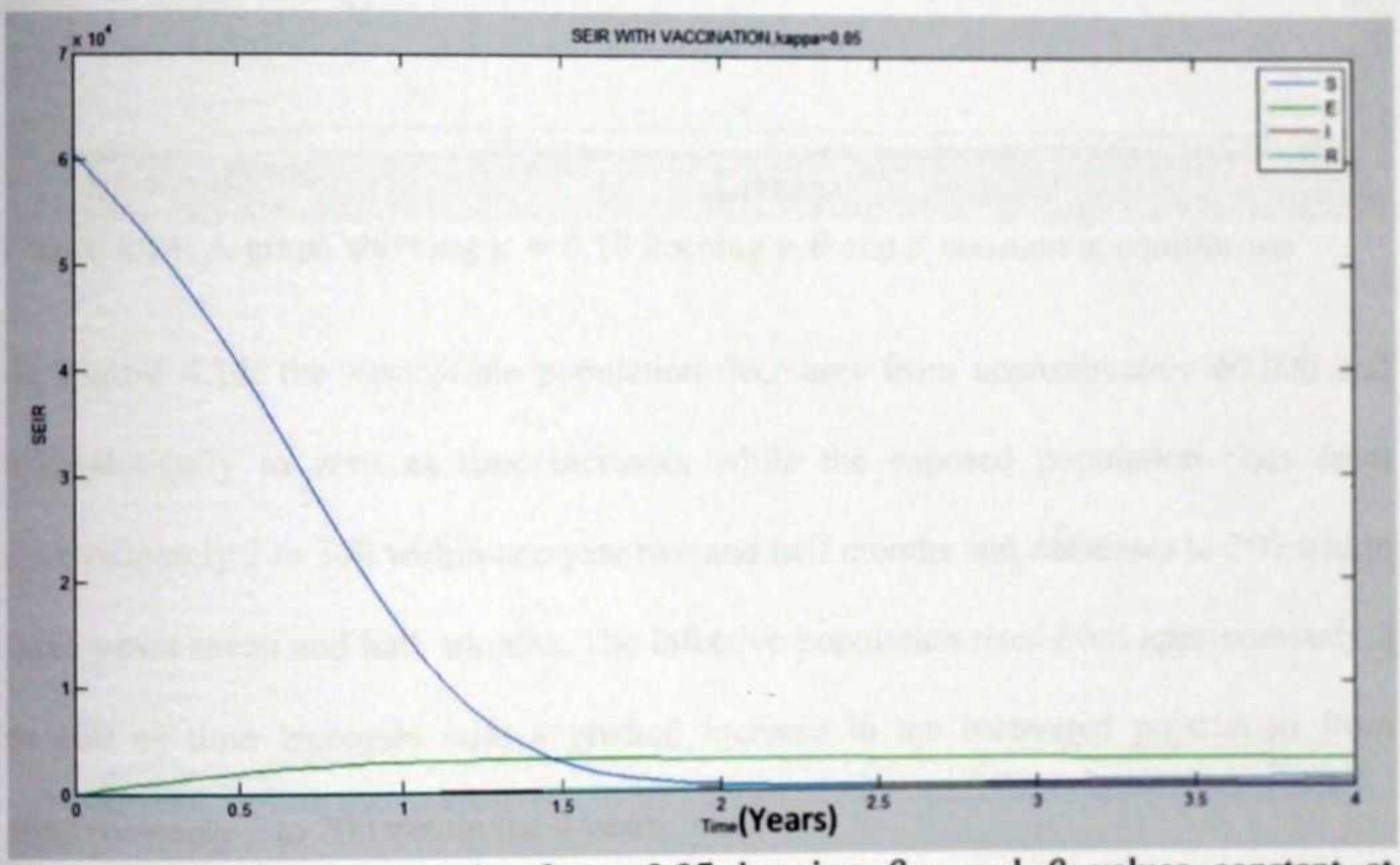

Figure 4.13: Shows a graph of $\kappa = 0.05$ keeping β, γ and θ values constant at equilibrium

In Figure 4.13, the infective population and the recovered population increase asymptotically within the 4 years with the exposed population rising from approximately 2 to 300 as time increases. The susceptible population decreases sharply from approximately 60,000 and asymptotically to zero within the 4 years.

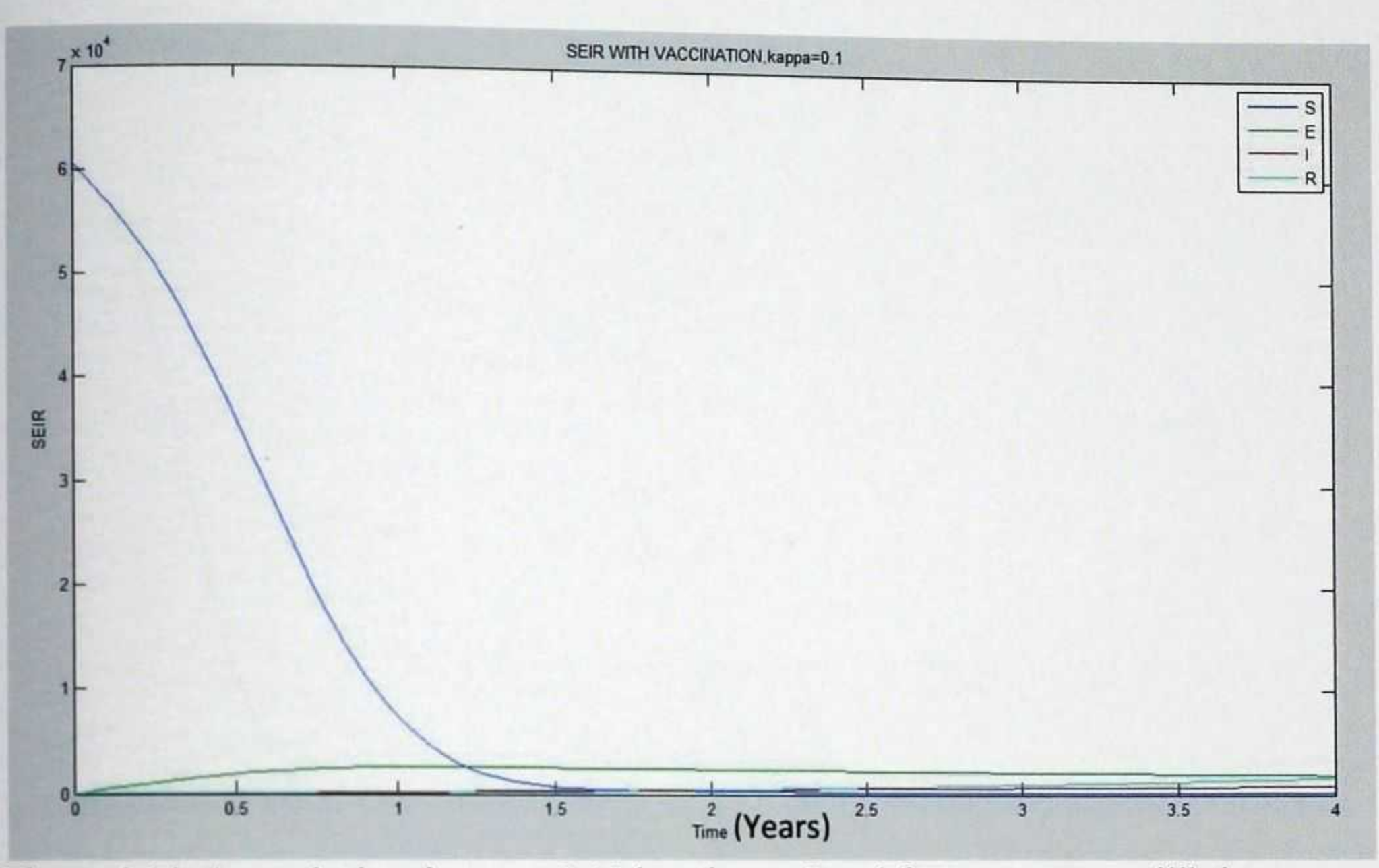

Figure 4.14: A graph showing $\kappa = 0.10$ keeping γ, θ and β constant at equilibrium

In Figure 4.14, the susceptible population decreases from approximately 60,000 and asymptotically to zero as time increases while the exposed population rises from approximately 2 to 300 within one year two and half months and decreases to 200 within three years seven and half months. The infective population rises from approximately 2 to 100 as time increases with a gradual increase in the recovered population from approximately 2 to 200 within the 4 years.

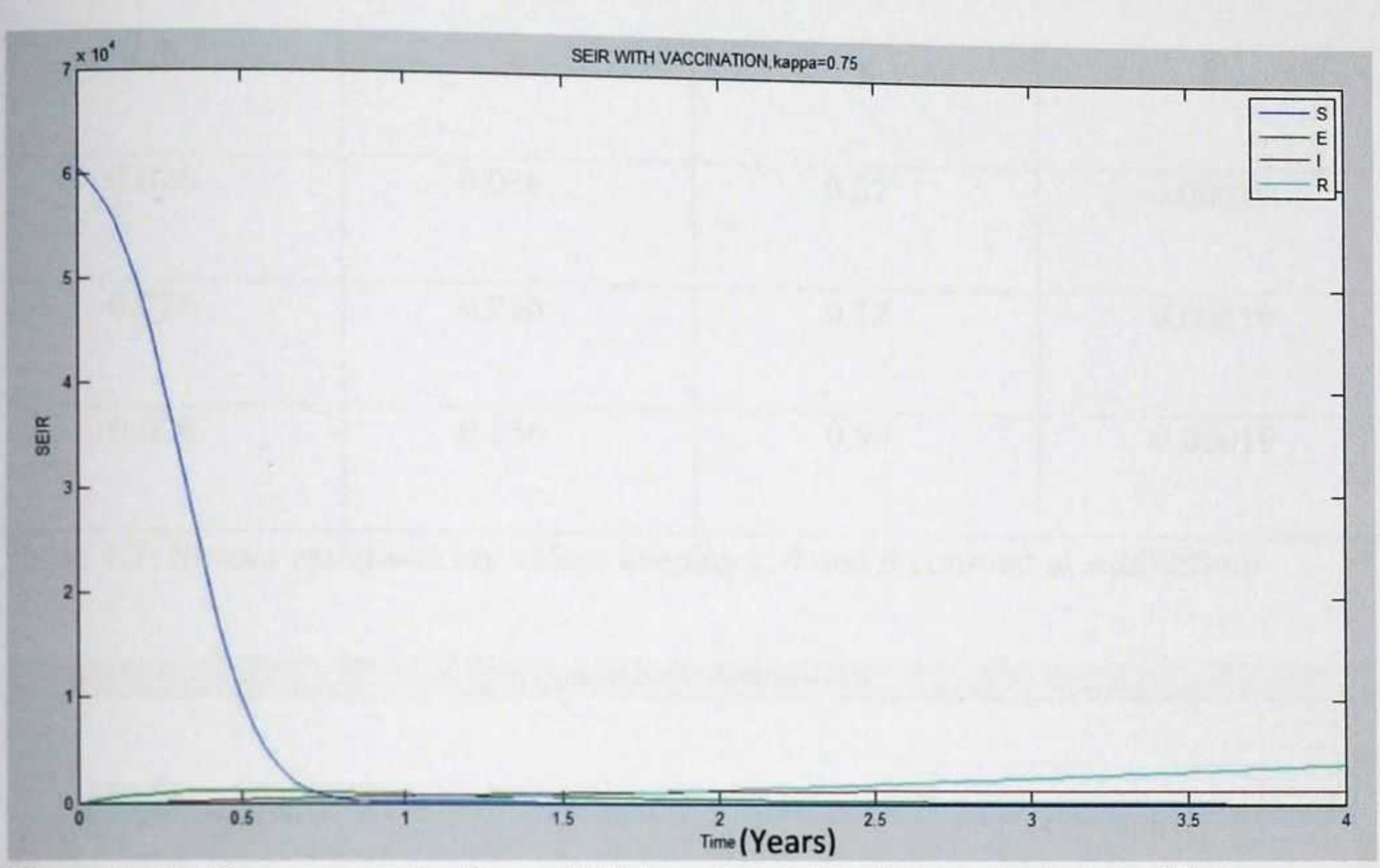

Figure 4.15: Shows a graph of $\kappa = 0.75$ keeping γ, θ and β constant at equilibrium

In Figure 4.12, there is a sharp decrease in the susceptible population from approximately 60,000 to asymptotically zero as time increases with a gradual decrease in the exposed population from approximately 100 to asymptotically zero as time increases. The infective population increases from approximately 2 to150 as time increases with a significant rise in the recovered population from approximately 2 to 400 within the 4 years.

We observed that as the latency rate (κ) increases from 0.05 to 0.75 there is a sharp decrease in the susceptible population from approximately 60,000 to100 within the 4 years with more people recovering from approximately 2 to 400 and a small increase in the infective population from approximately 2 to 150 with a decline in the exposed population from approximately 300 to asymptotically zero as time increases.

β	κ	γ	θ
0.026	0.036	0.07	0.00019
0.026	0.036	0.12	0.00019
0.026	0.036	0.99	0.00019

Table 4.7: Shows variations inγ values keeping κ, θ and β constant at equilibrium

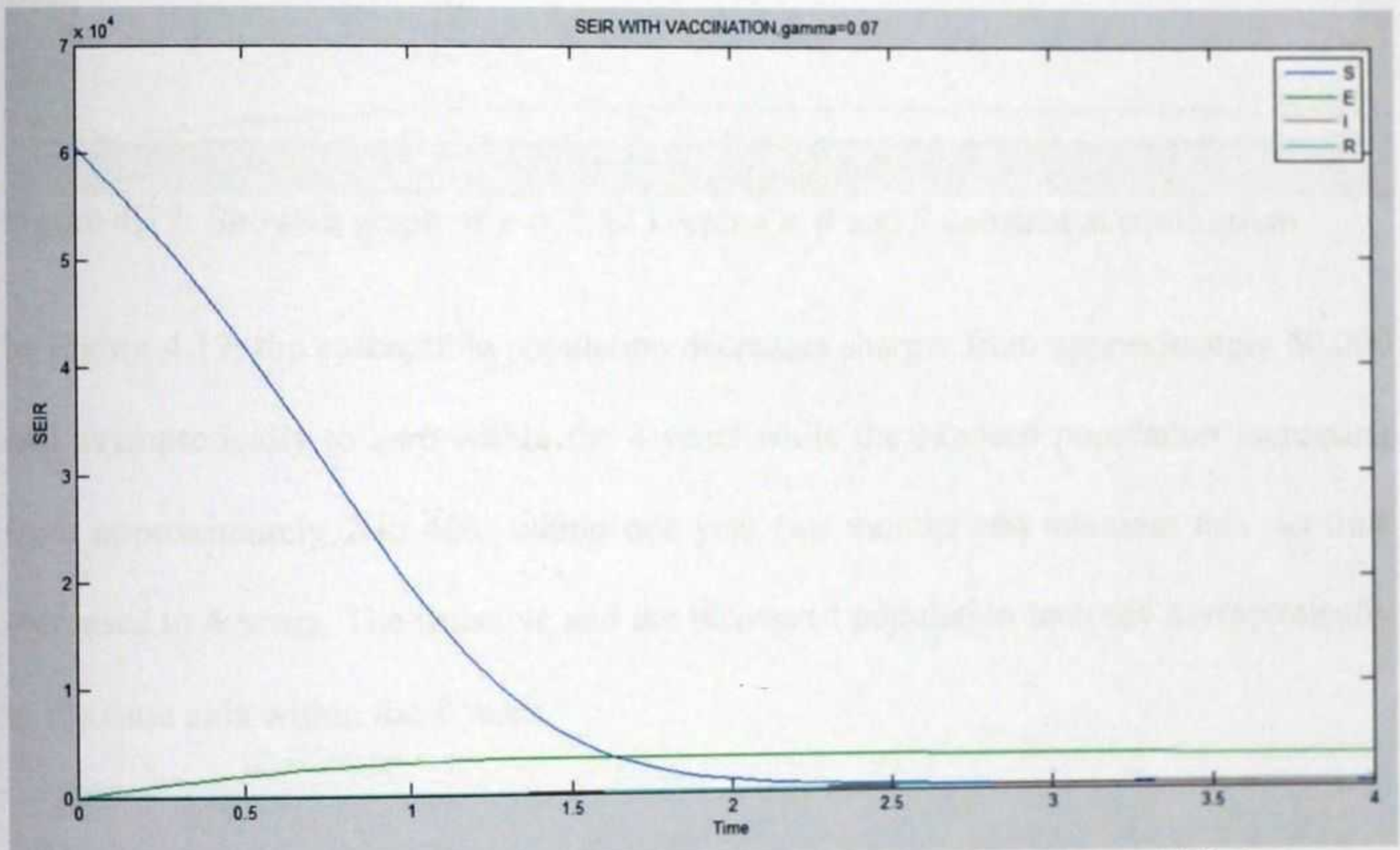

Figure 4.16: Shows a graph of $\gamma = 0.07$ keeping κ, θ and β constant at equilibrium

From Figure 4.16, the susceptible population decreases sharply from approximately 60,000 and asymptotically to zero with the exposed population increasing from approximately 2 to 400 within one year two months and maintain this as time increases to 4 years. The recovered population increase asymptotically as time increases within the 4 years with while the infective increases asymptotically as time increases.

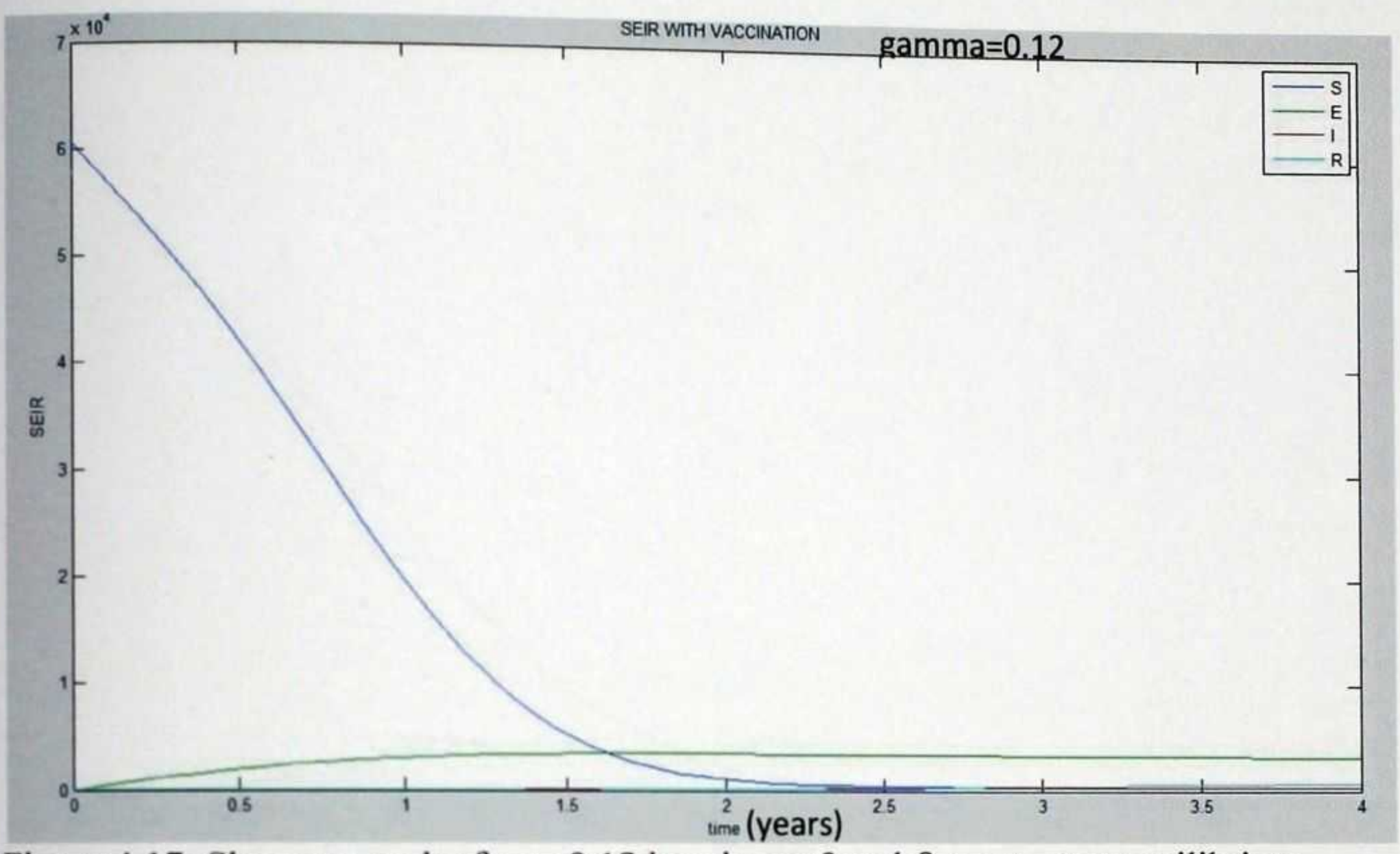

Figure 4.17: Shows a graph of $\gamma = 0.12$ keeping κ, θ and β constant at equilibrium

In Figure 4.17, the susceptible population decreases sharply from approximately 60,000 and asymptotically to zero within the 4 years while the exposed population increasing from approximately 2 to 400 within one year two months and maintain this as time increases to 4 years. The infective and the recovered population increase asymptotically to the time axis within the 4 years.

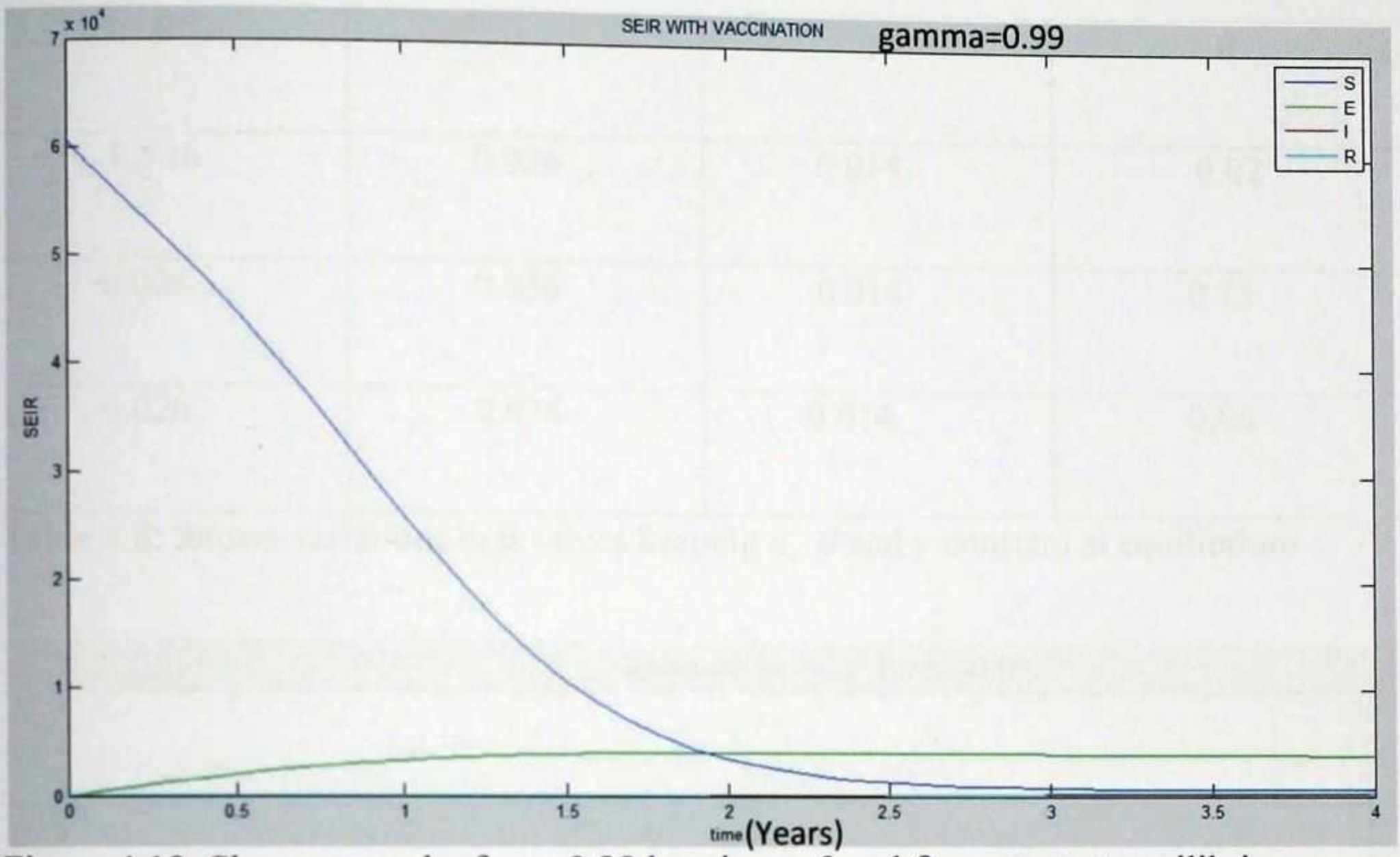

Figure 4.18: Shows a graph of $\gamma = 0.99$ keeping κ, θ and β constant at equilibrium

In Figure 4.18, the susceptible population decreases sharply from approximately 60,000 and asymptotically to zero as time increases to 4 years with the exposed population increasing from approximately 2 to 400 within the 4 years. The infective and the recovered population decreases asymptotically to zero within the 4 years.

We observed that as the recovery rate (γ) varies from 0.07 to 0.99, the susceptible population decreases with no significant increase or decrease in the exposed population. The infective and the recovered population decreases asymptotically to zero within the 4 years.

β	κ	γ	θ
0.026	0.036	0.014	0.02
0.026	0.036	0.014	0.75
0.026	0.036	0.014	0.96

Table 4.8: Shows variations in θ values keeping κ, β and γ constant at equilibrium

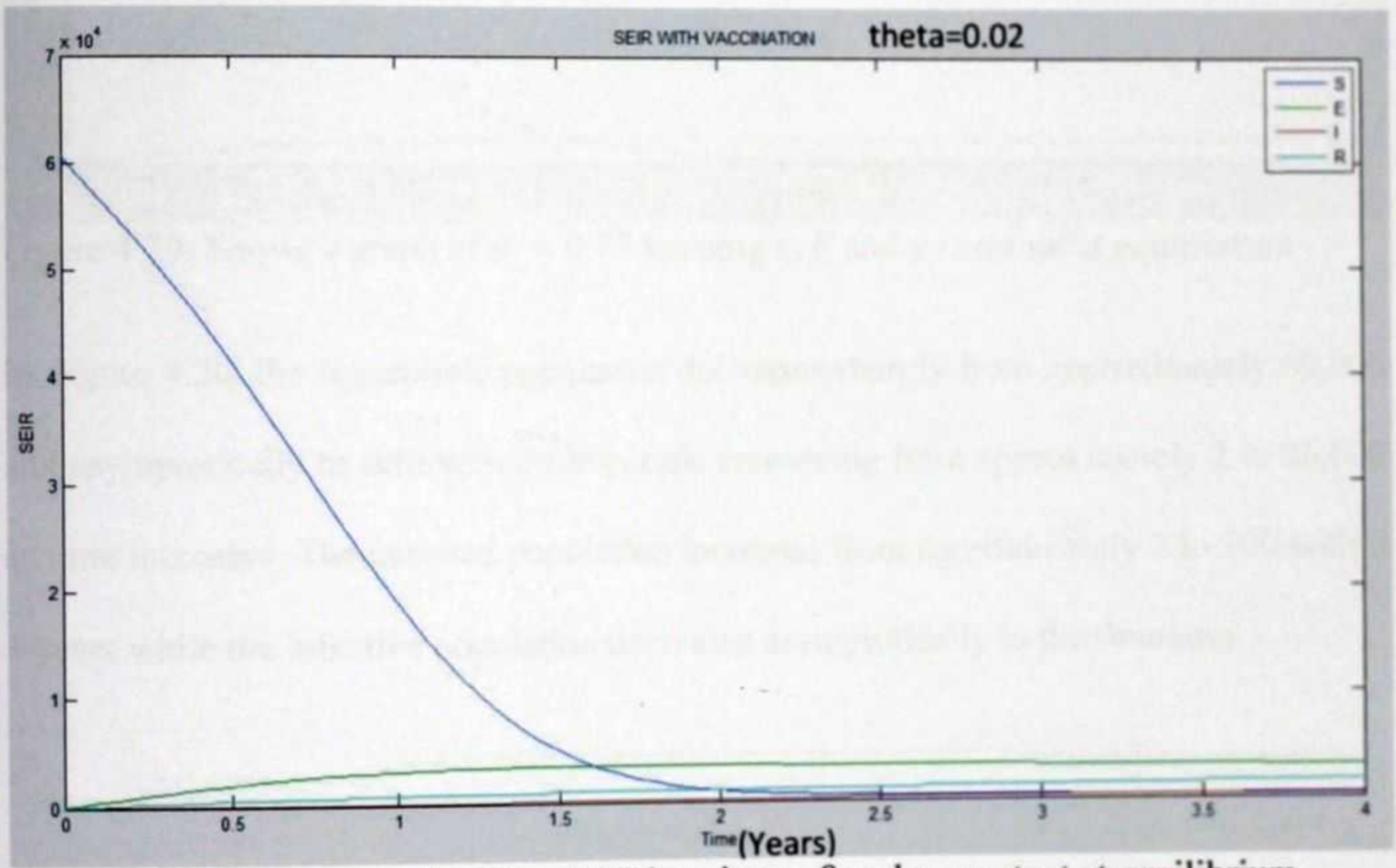

Figure 4.19: Shows a graph of $\theta = 0.02$ keeping κ, β and γ constant at equilibrium

In Figure 4.19, the susceptible population decreases sharply from approximately 60,000 and asymptotically to zero as time increases to 4 years. The exposed population increases from approximately 2 to 400 as time increases with the recovered population rising from approximately from 2 to 200 as time increases with a gradual rise in the infective population.

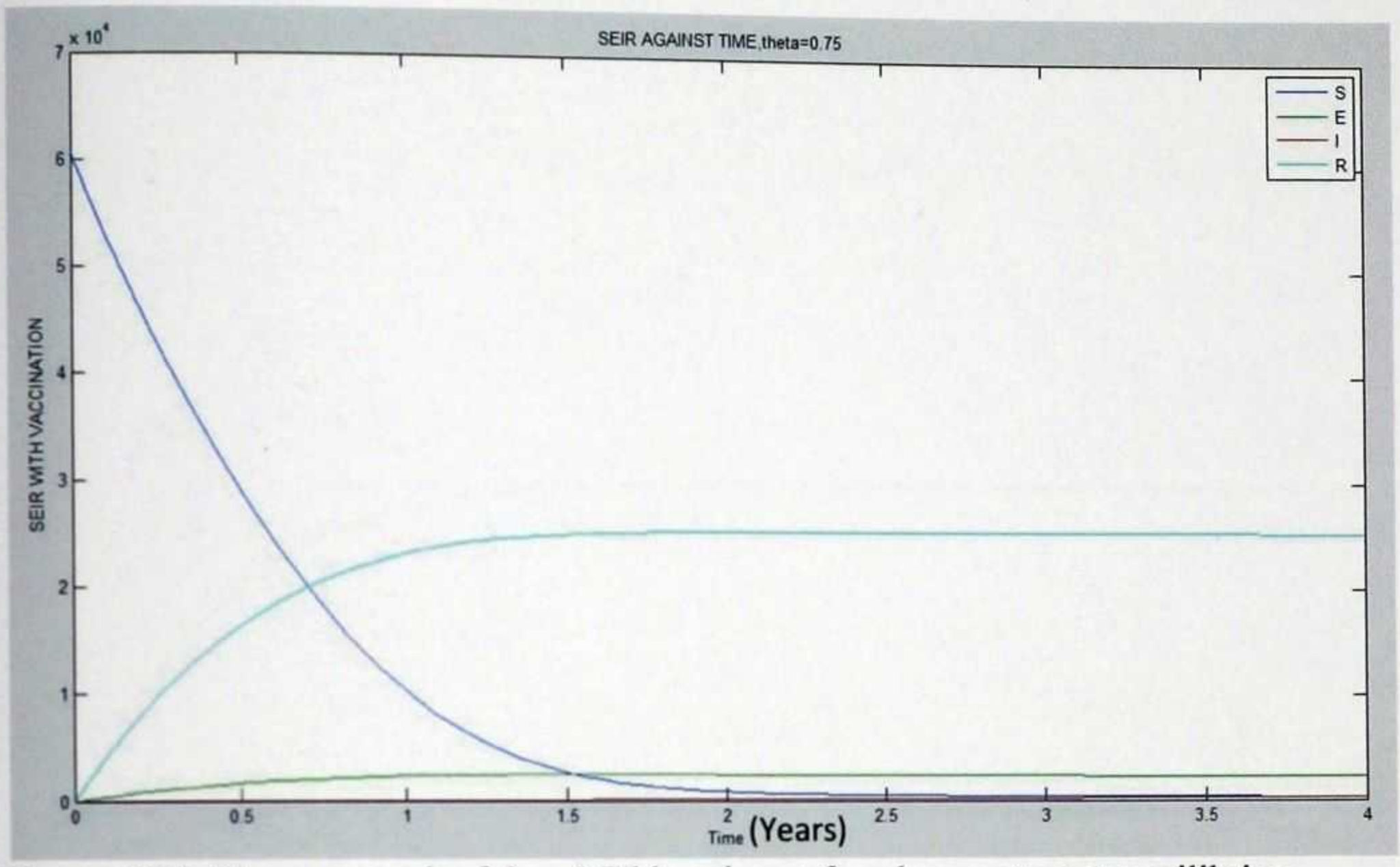

Figure 4.20: Shows a graph of $\theta = 0.75$ keeping κ, β and γ constant at equilibrium

In Figure 4.20, the susceptible population decreases sharply from approximately 60,000 and asymptotically to zero with more people recovering from approximately 2 to 26,000 as time increases. The exposed population increases from approximately 2 to 300 within 4 years while the infective population decreases asymptotically to the time axis.

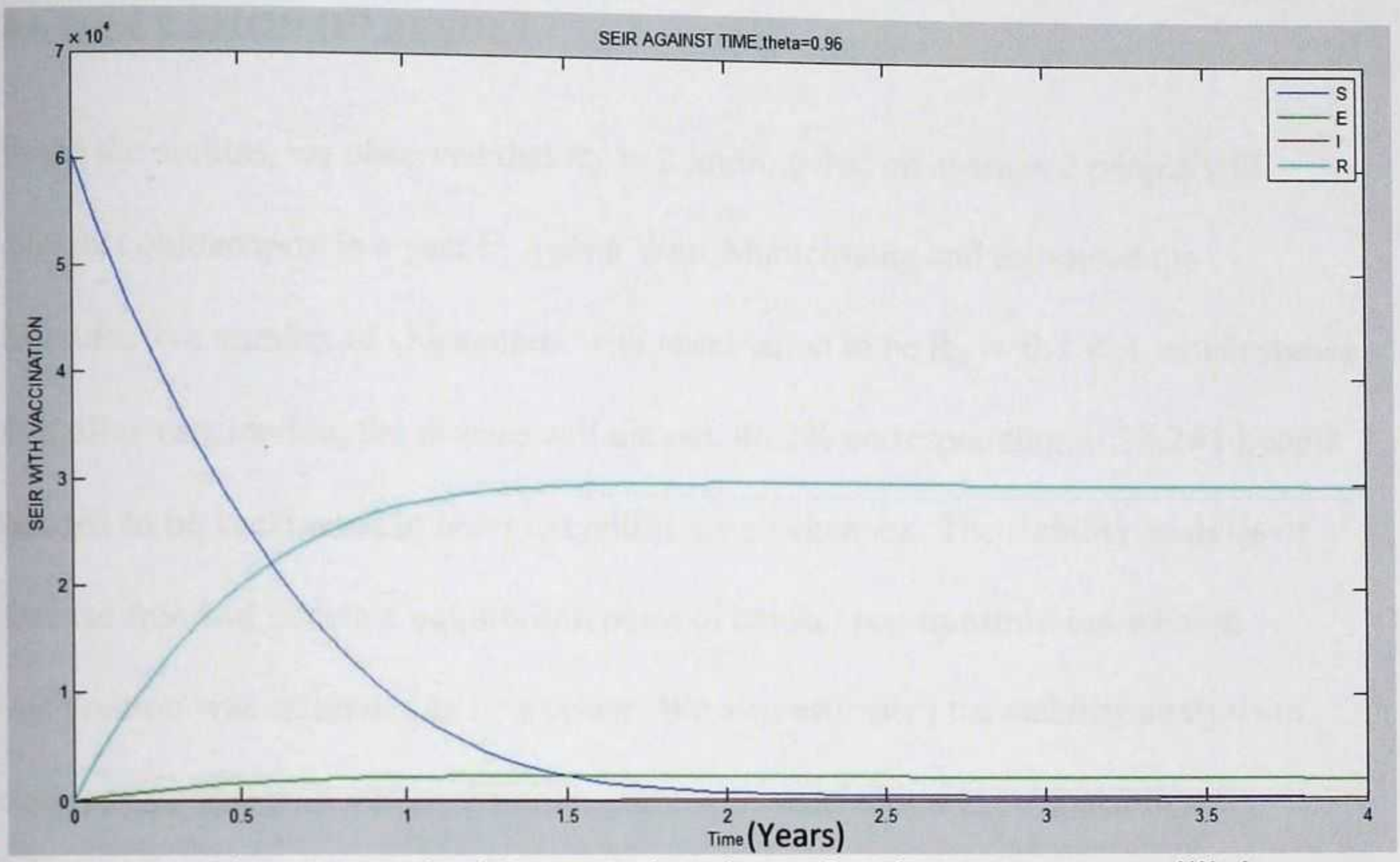

Figure 4.21: Shows a graph of $\theta = 0.96$ keeping κ, β and γ constant at equilibrium

In Figure 4.21, the susceptible population decreases approximately from 60,000 and asymptotically to zero with more people recovering from approximately 2 to 30,000 as time increases. The exposed population increases from approximately 2 to 250 as time rises to 4 years while the infective population decreases asymptotically to the time axis within the 4 years.

We observed that as the vaccination rate coefficient (θ) varies from 0.02 to0.96, more people recovered chickenpox from approximately 200 to 30,000 with a decrease in the exposed population from approximately 300 to 250 with no significant increase or decrease in the infective as time increases.

4.6 DISCUSSION OF RESULTS

From the studies, we observed that $R_0 = 2$ impling that on average 2 people will contract chickenpox in a year in Agona West Municipality and estimated the reproductive number of chickenpox with vaccination to be $R_o = 0.3 < 1$ which shows that after vaccination, the disease will die out. 46.2% corresponding to 53,241 people needed to be vaccinated in order to control the chickenpox. The stability analysis of disease free and endemic equilibrium point of chickenpox transmission without vaccination was estimated to be a centre. We also estimated the stability analysis of disease free and endemic equilibrium point with vaccination to be unstable and asymptotically stable.

By Sensitivity analysis of the SEIR model without vaccination, we observed that as transmission rate (β) varies from 0.08 to 0.90 the susceptible population decreases sharply with a sharp increase in the exposed population with more people contracting chickenpox while the recovered population was asymptotic to the time axis. We observed that as the latency rate (κ) varies from 0.40 to 0.83, more people become exposed to chickenpox and as a result chickenpox patient rose from approximately 46,000 to 56,000 with no significant change in the recovered population

We observed that as the recovered rate (γ) varies from 0.007 to 0.70, people who contracted chickenpox decreases with more people recovering while the latency rates (κ) is more sensitive to the model than the transmission rate (β) and the recovery rate (γ) . Sensitivity analysis of SEIR model with vaccination showed that the susceptible population decreases sharply with time while the infective and recovered population also

decreases asymptotically within the 4 years as the value of transmission rate (β) was varied from 0.08 to 0.97. We observed that as the recovery rate (γ) varies from 0.07 to 0.99, the susceptible population decreases with no significant increase or decrease in the exposed population. While the infective and the recovered population decreases asymptotically to zero within the 4 years.

We observed that as the latency rate (κ) increases from 0.05 to 0.75 there is a sharp decrease in the susceptible population from approximately 60,000 to100 within the 4 years with more chickenpox patients recovering from approximately 2 to 400 and a small increase in the infective population from approximately 2 to 150 with a decline in the exposed population from approximately 300 to asymptotically zero as time increases.

As the vaccination rate coefficient (θ) varies from 0.02 to 0.96, we observed that more chickenpox patients recovered from approximately 200 to 30,000 with a decrease in the exposed population from approximately 300 to 250 with no significant increase or decrease in the infective as time increases. We observed that vaccination rate coefficient (θ) was more sensitive to the transmission rate (β) , latency rate (κ) and the recovering rate (γ) with the model with vaccination.

CHAPTER 5

CONCLUSION AND RECOMMENDATIONS

5.1 Conclusion

The study shown that chickenpox in recent time has increased in Agona West Municipality. Based on the data obtained from the Agona West Municipal Health Directorate, we observe that chickenpox is persistent (endemic) in the municipality with $R_0 = 2$ which implies that on average 2 people will contract chickenpox in a year in Agona West Municipality. We estimated the reproductive number chickenpox with vaccination to be $R_o = 0.3 < 1$ which shows that after vaccination the disease will die out. 46.2% corresponding to 53,241 people needed to be vaccinated in order to control the chickenpox. The stability analysis of disease free and endemic equilibrium point of chickenpox transmission without vaccination was estimated to be a centre. We also estimated the stability analysis of disease free and endemic equilibrium point with vaccination to be unstable and asymptotically stable.

Sensitivity analysis of the SEIR model without vaccination shown that the latency rates (κ) is more sensitive to the model than the transmission rate and the recovery rate while Sensitivity analysis of SEIR model with vaccination also indicated that the vaccination rate coefficient (θ) being is more sensitive to the model than the transmission rate (β), latency rates (κ) and the recovering rate (γ).

5.2 Recommendations

We observed that chickenpox can be prevented if vaccine for chickenpox is not given in Agona West Municipality and the latency rates (κ) is reduced significantly, chickenpox will be under control and if more people are vaccinated against chickenpox (vaccination rate coefficient (θ) is increase), chickenpox will be under control in the Municipality.

We call on all stakeholders in the health sector to come together to ensure that there is a law that enforces all the health sector to keep up to date data on all the diseases reported at the various hospitals, clinics and the health centers due to the difficulties in obtaining data for research.

We recommend further studies on

(1) SEIR model using non constant population and incorporation of age.

(2) Researchers and students can extend the model to non constant population size

(3) Extend to unequal birth/death rate

(4) Different method of analysis such as analytic solution could also be ascertained by researchers and students

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APPENDIX 1

The table 4.1 below shows the summary of the data obtained from the Agona West Municipal Health Directorate

Time(year)	Susceptible	Exposed	Infective	Recovered
$t = 0$	60684	9	23	0
$t = 1$	55950	5	13	15
$t = 2$	58265	7	18	20
$t = 3$	48463	11	28	41
$t = 4$	31527	16	41	43

APPENDIX 2

Matlab code for the graphs

```
function dy=sire(t,y)

m=0.026;t=0.00019;p=115358;b=0.026;k=0.036;g=0.014;

dy=zeros(4,1);

dy(1)=m*p-b*y(1)*y(3)-m*y(1)-t*y(1);

dy(2)=b*y(1)*(3)-(m+k)*y(2);

dy(3)=k*y(2)-(m+g)*y(3);

dy(4)=y(3)-m*y(4)+t*y(1);


[t y]=ode45(@seirw,[0 4],[60684 9 23 0])

plot(t,y(:,1),t,y(:,2),t,y(:,3),t,y(:,4))

legend('S','E','I','R')

title('SEIR WITHOUT VACCINATION, gamma=0.02')

xlabel('time')

ylabel('SEIR')


function dy=seirw(t,y)

k=0.036;g=0.02;b=0.026;

dy=zeros(4,1);

dy(1)=-b*y(1)*y(3);

dy(2)=b*y(1)*y(3)-k*y(2);
```

```
dy(3)=k*y(2)-g*y(3);

dy(4)=g*y(3);


s=[60684 559507 58265 48463 31527]';

e=[9 5 7 11 16]';

i=[23 13 18 28 41]';

r=[0 15 20 41 43]';

t=[0 1 2 3 4];

[t y]=ode45(@sir,[0 4],[60684 9 23 0])

plot(t,y(:,1),t,y(:,2),t,y(:,3),t,y(:,4))

title('SEIR AGAINST TIME,theta=0.02')

xlabel('Time');

ylabel('SEIR');

legend('S','E','I','R')
```