International Journal of Current Science Research and Review
ISSN: 2581-8341
Volume 05 Issue 12 December 2022
DOI: 10.47191/ijcsrr/V5-i12-20, Impact Factor: 5.995
IJCSRR @ 2022
www.ijcsrr.org

Modelling Cyclic Fluctuations of SEIR Epidemic Diseases

Placidie MUKARUGWIRO[1], Emmanuel Havugimana[2], Projecte HABYARIMANA[3], Viateur NIZEYIMANA[4]
[1] Assistant lecturer at IPRC-KIGALI, department of General Courses.
[2,3] Assistant Lecturer at IPRC-KIGALI, Department of General courses.
[4]Assistant Lecture at IPRC-KIGALI, Integrated Polytechnic Regional College (IPRC-Kigali)
KK 492 Street Kicukiro Kigali Rwanda, 6579 Kigali Kicukiro, Kigali, Ville de Kigali

ABSTRACT: Seasonality of infectious disease is an important factor in disease incidence, outbreaks, control and prevention. Many mathematical models that incorporate seasonality in the transmission were formulated and analyzed. In this essay a qualitative analysis is given in terms of the effective reproduction number R_0, the existence and stability of the disease-free equilibrium and endemic equilibrium of both the SEIR model and seasonal SEIR model. We perform numerical simulations to validate the model formulation.

KEYWORDS: Cyclic, Epidemic diseases, SEIR-model, Simulation.

Abbreviations: DFE-disease free equilibrium.

1 HISTORICAL OF THE STUDY

1.1 General Introduction

Infectious diseases are disorders caused by organisms such as bacteria, viruses, parasites or fungi and many of these organisms live in our bodies, but they can become harmful under certain conditions. It is possible to infectious diseases from one person to another person. Some of the diseases are endemics and others epidemics. The spread of infectious diseases through human population has been the subject of scientific research for a several years. Now the endeavor of many researchers onepidemiology diseases shows the threat to human life. Seasonality can be described as a periodic pattern typically occurring every calendar year, it is repeti- tive and generally regular and predictable. Seasonality, as a period of seasonal changes in the diseaserate, affects many infectious diseases. In general, some of the diseases are usually described in termsof rainfall, and humidity during a given period, and this seasonal variation may vary for different areas.
In this project, we probe the seasonal parameters of the SEIR model, give a brief review of seasonality and provide numerical solution.

1.2 Background of the study

Infectious epidemics diseases become an active research area where strategies have been increased based on mathematical models. Although in 1960, Daniel Bernoulli provided the earliest mathe- matical model describing the infectious diseases where he has formulated and solved a model for smallpox, deterministic epidemiology modeling seems to have started in the 20^{th} century Bernoulli (1760).Thus one can come across with books like Keeling and Rohani (2011) which introduces mod-eling of infectious diseases and show that they can have an important application in diseases control. Infectious diseases are part of the leading causes of death for people on the world (contribution of 26% of global mortality 2001 Organization et al. (2002)).
The change in the environment is some of the big problems for human life. One of the causes of seasonal changes in incidence is the pathogen's ability to survive outside the host depending on thehumidity, the temperature and sunlight exposure Grassly and Fraser (2008). Many diseases such as flu LONDON and YORKE (1973), measles, chickenpox, and mumps Earn et al. (2002); AL-AJAM et al. (2006) show seasonal behavior. From 1942 to 1945, Malaria Control in War Areas (MCWA) was established to control Malaria spread around military camps. On one hand, Ross Ross (1911) was interested in malaria incidence and control, and developed differential equations while consideringMalaria as a host-vector disease. On one other hand researchers like Earn et al. (2002); AL-AJAMet al. (2006) (on measles, chickenpox, and mumps), Ross (1911) on malaria have been worked on sea- sonal variation diseases modeling. Their analytic results show that the most transmission parameterof diseases on the population is the seasonal variation based on the behavior of the diseases.
Epidemics are sensitive to seasonal forcing by Grassly and Fraser. (2006). The cause of seasonal pat-terns may vary from natural causes such as true seasonal phenomena that generate periodic tempera- tures, humidity or periodic birth rates Altizer (2006a), to human forced phenomena such as the suc-cession of school terms and holidays. More specifically, childhood diseases such as rubella, measles and whooping cough

International Journal of Current Science Research and Review
ISSN: 2581-8341
Volume 05 Issue 12 December 2022
DOI: 10.47191/ijcsrr/V5-i12-20, Impact Factor: 5.995
IJCSRR @ 2022
www.ijcsrr.org

are some of the highly sensitive diseases to seasonality due to school terms andholidays. One of the studies about seasonality due to school is by London and Yorke (1973); Grasslyand Fraser. (2006), who, studying weekly periods for diseases of measles in England and Wales, where he demonstrated the decline of transmission during the school holidays. London and Yorke (1973); Grassly and Fraser. (2006) also studied this seasonality of schooling.

The epidemic model dynamics, due to its practical and theoretical significance has been studied ex-tensively by Anderson and May (1979); Hethcote (2000). In most of the epidemic, models have constants parameters. This is the case for instance for the contagious diseases spread by mosquitoes, where most of the mosquitoes die out of winter but they reproduce hugely in summer, hence the spread of diseases is seasonal. Thus, under periodic environment, it is more realistic to investigate the corresponding epidemic models with periodic parameters. Mathematical epidemiology seems to have grown exponentially starting in the middle of 20^{th} century. Many of the models have been formulated, mathematically analyzed and have been applied to infectious diseases. Many of the researchers havebeen applied the SEIR to infectious diseases. Shah and Jyoti (2013) have been applied it to borne vector diseases. The method for analyzing compartment model on infectious diseases by Shah and Jyoti (2013). Al-Sheikh (2012) analyzed an SEIR model with limited resources for treatments. Sim- ulations are carried out using python programming and the basic reproduction number is explained within seasonal model have been studied by many investigators, both due to its relevance to the understand- ing of the epidemiology of seasonal infectious diseases Altizer (2006b); Stone et al. (2007); Aron and Schwartz (1984). Of course, many diseases have a seasonal component. The seasonal contact rate that incurs permanent immunity have been computed by Fine and Clarkson (1982). London and Yorke (1973) show that for monthly data, the contact rate appears to be smooth and periodic with a period about one year. Fine and Clarkson (1982) have been take weekly data for measles and have used theextra detail to investigate the factors causing seasonality in the contact rate.

The compartment model with labels such as S, E, I and R are used in epidemiology diseases and SEIR is the abbreviation of Susceptible, Exposed, Infectious and Recovered. In 1995, Michael Y. Li and James S. Mulroney studied the global stability for the SEIR model in epidemiology, where they showed the global stability of period of the endemic equilibrium as well as using the theory of competitive systems of differential equations M. Y. Li (1995). After this study, there have been re- searches about epidemic models with latent periods Turner (2010). Mathematical modeling is beingincreasingly used to understand the transmission of infectious disease and to evaluate the potentialstrategy in reducing the mortality of the population caused by infectious diseases. The model we formulate here is an SEIR model where the population is divided into compartments containing susceptible, exposed, infectious and recovered individuals Feng et al. (2009); Bauch and Earn (2003); Bernoulli (1760). The compartments with labels S,E,I, R are used for epidemiology classes as shownin Fig. 11. The application of the SEIR model includes determining optimal control strategies againstnew infections such as Ebola, Tuberculosis, and Malaria, and predicting the impact of vaccination strategies against common infectious as Rubella measles.

The SEIR model is an extension of SIR (Susceptible, Infectious, Recovered) model which was origi- nally developed by Kermack and McKendrick (1927). A fourth compartment which contains exposed persons which are infected but not yet infectious is added. Mathematical models of infectious dis- eases can help us to understand disease dynamics and transmission. They also allow us to stimulatethe spread of the diseases in different settings and scenarios in order to develop and evaluate different interventions strategies to ameliorate infectious and better allocation resources (choosing the target population, the location and the time for intervention). The SEIR model was used to model the dy- namics of an influenza outbreak in a population using differential equations. Li (2012) presented theapplication of SEIR model on the work done in the field of malaria modeling. Shah and Gupta. (2013)have been designed and analyzed the SEIR model for malaria when it was in the endemic situation.

Some epidemiological models were studied using numerical simulations to investigate the effect onthe behavior of the disease of a seasonally varying contact rate. Dietz (1976); Aron and Schwartz (1984); Rohani and Grenfell. (2002) employ continuous seasonality models. Special interest was taken in the calculation of solutions in these different studies, the period of doubling bifurcations and the description of attraction basins of stable periodic solutions. Several authors such as Smith (1983); B. and H. L. Smith. (1983) conducted theoretical studies of periodic continuous models. TheSEIR seasonal disease model was formulated by all the researchers but they don't often to include the strategic parameter to stop seasonal diseases. For our contribution, we simulate the seasonal mathematical model with a strategic parameter (vaccination) to stop diseases as it is the best strategic way to prevent contagious diseases from spreading.

International Journal of Current Science Research and Review
ISSN: 2581-8341
Volume 05 Issue 12 December 2022
DOI: 10.47191/ijcsrr/V5-i12-20, Impact Factor: 5.995
IJCSRR @ 2022
www.ijcsrr.org

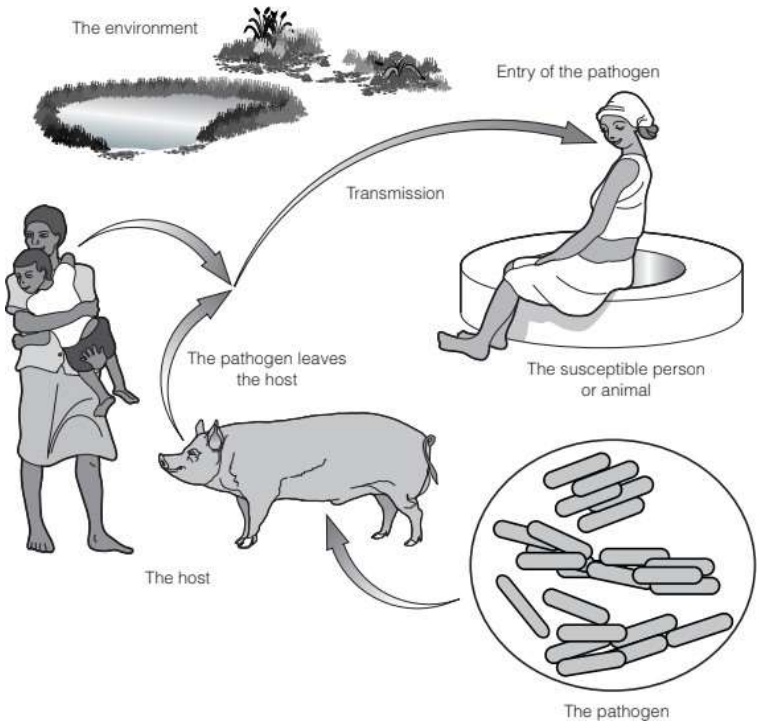


Figure 1: The different elements of the transmission cycle of disease Beard et al. (2003).

2 SEIR MODEL

The SEIR models the flows of people between four states; Susceptible (S), exposed (E), infected (I) and resistant or recovered (R). Each of those variables represents the population in those groups.The vaccination or other strategy moves people from the susceptible to recovered directly, withoutbecoming exposed or infected.

A very large number of epidemiological models involve the separation of individuals from each other.It is also commonly assumed that people make contacts that show no preferences at random. One ofthe basic compartmental models (SIR) is proposed by Kermack and McKendrick (1927), where pop-ulation are classified as susceptible to infection, labeled S, pathogen-infected, labeled I, or recoveredfrom the infection, labeled R.

2.1 Mathematical Modeling

Mathematical modeling is a way of transforming the real-life problem or situation into the mathematical language in order to make it easy to understand, analyze and solve the problem for future predictions and decision making. Here are various mathematical model types, deterministic and stochasticmodels. Deterministic models are mathematical models that use parameter values and initial conditions to accurately determine the predicted outputs. Whereas stochastic are models that have the distribution of possible results. Fig. 2 shows the SEIR model.

2.1.1 Assumptions of model

The model starts with the assumption saying that the total population N is constant at any time,the individuals are assuming to be homogeneous and mix uniformly. The basic as- Sumption is saying that the population N can be subdivided into 4 groups depends on the level of diseases. The susceptible population are people who have never come into contact with the diseasecalled susceptible group S(t) and the exposed population are the one who have been infected by dis-ease but who are not able to spread disease to other people can be called exposed group E(t). The exposed group can stay in the same group up to (1), where μ is the natural mortality rate. When exposed individuals start to spread the disease, they moved into the infectious group I(t). The infected individual can spread the disease to susceptible and can stay in the infectious group for a certain period of time ($\frac{1}{\gamma}$)(γ is capita infection rate per unit time) before moving into the recovered group. Recovered individuals are assumed to be immune for life. Then, the whole population is given as N=

International Journal of Current Science Research and Review
ISSN: 2581-8341
Volume 05 Issue 12 December 2022
DOI: 10.47191/ijcsrr/V5-i12-20, Impact Factor: 5.995
IJCSRR @ 2022
www.ijcsrr.org

S(t)+ E(t)+I(t)+ R(t) and the SEIR compartments model is shown in Fig. 2.

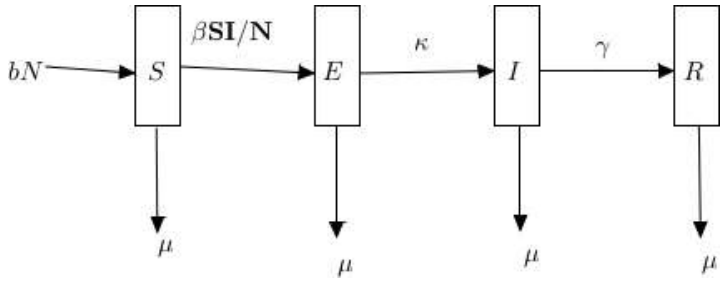


Figure 2: The general transfer diagram for SEIR epidemic model.

2.2 The basic Reproduction number

In epidemiology, the basic reproductive number is the average number of secondary infections pro-duced by a single infected person in a completely susceptible population (Anderson and May, 1979).The reproductive number is a measure of disease spread in the population. R_0 provides a thresholdcondition for the stability of the disease-free equilibrium point. The disease-free equilibrium point issaid to be asymptotically stable when $R_0 < 1$: the disease dies out. The disease-free equilibrium points is said to be unstable when $R_0 > 1$: the diseases remains in the population (Heffernan et al., 2005).An alternative method proposed by (Diekmann et al., 1990) and elaborated (Van den Driessche andWatmough, 2002) gives the a way of determining R_0 for ODE compartments model by using next generation matrix. Here a more details of this methods and the proofs is given and further details canbe found in (Driessche and Watmough, 2008) and (Van den Driessche and Watmough, 2002)

Let $X = (X_1, X_2, ..., X_n)^T$ be the numbers of individuals in each compartment, where the first m<ncompartments contain infected individuals. Assume that the DFE X_0 exists and stable in the ab- since of disease, and that the linearized equations for X_1, ..., X_n at the DFE decouples from the other equations. The assumptions are given in more details in the references cited above. Consider these equations written in the form

Then, we have $\frac{dx_i}{dt} = \mathscr{F}_i(X)X(t) - \mathscr{V}_i(X)$ for i= 1, ..., m, where $\mathscr{F}_i(x)$ is the rate of appearance of new infection in compartment i and $\mathscr{V}_i(X)$ is the rate of other transition between compartment i and other infected compartments.

Now define $\mathscr{F} = \frac{\partial \mathscr{F}(x_0)}{\partial x_i}, \mathscr{V} = \frac{\partial \mathscr{F}(x_0)}{\partial x_j}$ for $1 \leq i$ and $j \leq m$, $\mathscr{F}_i$ and $\mathscr{V}_i$ represent respectively new infection and the transfer of infection from one compartment to another, wherex_0 is the DFE (ie no infection).

The product of the inverse of matrix $\mathscr{V}$ and matrix $\mathscr{F}$ gives the next generation matrix called P=$\mathscr{F}\mathscr{V}^{-1}$.

Then, the R_0 is the absolute value of the largest eigenvalue of the next generation matrix and it is denoted by $R_0 = \rho(\mathscr{F}\mathscr{V}^{-1})$ (Heffernan_et_al., 2005).

Now we can apply the basic reproduction number R_0 using the second-generation matrix to the SEIRmodel. By normalization the system (1) becomes

$$\begin{cases} \frac{ds}{dt} = b - \beta si - \mu s \\ \frac{de}{dt} = \beta si - (\kappa + \mu)e \\ \frac{di}{dt} = \kappa e - (\gamma + \mu)i \\ \frac{dr}{dt} = \gamma i - \mu r \end{cases} \quad (6)$$

International Journal of Current Science Research and Review
ISSN: 2581-8341
Volume 05 Issue 12 December 2022
DOI: 10.47191/ijcsrr/V5-i12-20, Impact Factor: 5.995
IJCSRR @ 2022
www.ijcsrr.org

After computing the DFE, we have to select sub-model that only consider the diseases compartment,which is a subset of the SEIR model. The sub-model which contains only the E and I equations can bewritten as

$$\begin{bmatrix} \frac{E}{dt} \\ \frac{I}{dt} \end{bmatrix} = \mathcal{F}(\bar{x}) - \mathcal{V}(\bar{x}).$$

Let $X = (S,E,I,R)^T$,

then the system Eq. (6) can be written as

$$\frac{d\bar{x}}{dt} = \mathcal{F}(\bar{x}) - \mathcal{V}(\bar{x})$$

where F is the infection matrix and V is nonsingular matrix. where $(\bar{x})$ is a vector of the j disease compartments; in the SEIR model j=2, since the compartments are E and I.

This equation is given as

$$\begin{cases} \frac{de}{dt} = \beta si - \kappa e - \mu e \\ \frac{di}{dt} = \kappa e - \gamma i - \mu i. \end{cases}$$

The right hand sides of Eq.(7) are therefore contained in the vectors $F(\bar{x})$ and $V(\bar{x})$, $F(\bar{x})$ contains any terms that leads the new infections entering the compartments j. The second element of $F(\bar{x})$ is zero since no new infection enter the I compartment, whereas the transition from that E compartment
to I compartment.
The Eq. (7) can be express in matrix form as

$$\mathcal{F}(\bar{x}) - \mathcal{V}(\bar{x}) = \begin{bmatrix} \beta si \\ 0 \end{bmatrix} - \begin{bmatrix} \kappa e + \mu e \\ -\kappa e + \gamma i + \mu i \end{bmatrix}.$$

This is exactly equivalent to our sub-model original Eq. (7).
Next step, we have to linearize around the DFE to obtain the Jacobian, evaluated at the DFE. It isgiven by

$$J(S,E,I,R) = \begin{bmatrix} \frac{\partial \mathcal{F}}{\partial I} & \frac{\partial \mathcal{F}}{\partial I} \\ \frac{\partial \mathcal{V}}{\partial I} & \frac{\partial \mathcal{V}}{\partial I} \end{bmatrix} = \begin{bmatrix} 0 & \beta s \\ 0 & 0 \end{bmatrix} - \begin{bmatrix} \kappa + \mu & 0 \\ -\kappa & \gamma + \mu \end{bmatrix}$$

ie

$$\frac{d\bar{x}}{dt} = \mathcal{F}(\bar{x}) - \mathcal{V}(\bar{x}) = \begin{bmatrix} 0 & \beta s \\ 0 & 0 \end{bmatrix} - \begin{bmatrix} \kappa + \mu & 0 \\ -\kappa & \gamma + \mu \end{bmatrix}$$

At $J(S^*,E^*,I^*,R^*) = \left(\frac{b}{\mu},0,0,0\right)$ one has

$$J\left(\frac{b}{\mu},0,0,0\right) = \begin{bmatrix} 0 & \frac{\beta b}{\mu} \\ 0 & 0 \end{bmatrix} - \begin{bmatrix} \kappa + \mu & 0 \\ -\kappa & \gamma + \mu \end{bmatrix}.$$

which are the rates of new infections and transitions near equilibrium

$$F = \begin{bmatrix} 0 & \frac{\beta b}{\mu} \\ 0 & 0 \end{bmatrix}, \quad V = \begin{bmatrix} \kappa + \mu & 0 \\ -\kappa & \gamma + \mu \end{bmatrix}$$

International Journal of Current Science Research and Review
ISSN: 2581-8341
Volume 05 Issue 12 December 2022
DOI: 10.47191/ijcsrr/V5-i12-20, Impact Factor: 5.995
IJCSRR @ 2022
www.ijcsrr.org

Knowing $\mathcal{F}$ and $\mathcal{V}$, we can compute the following

$$V^{-1} = \begin{bmatrix} \frac{1}{\kappa+\mu} & 0 \\ \frac{1}{\kappa+\mu} & \frac{1}{\kappa+\mu} \end{bmatrix}$$

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

The eigenvalues of FV^{-1} satisfied

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

$$\left(\frac{\beta b}{(\kappa+\mu)(\gamma+\mu)}\right)(\lambda) = 0.$$

This gives $\lambda_1 = 0, \lambda_2 = \frac{\beta b}{\mu(\gamma+\mu)}$. Since the spectral radius (maximum eigenvalues) is the basic reproduction number

$$R = \frac{b\beta}{(\kappa+\mu)(\mu+\gamma)}$$

and its numerical value is $R = 0.0445$ which is less than 1. In other words, there is no diseases in the population.

2.3 Numerical solution

The ordinary differential equation function solves differential equations numerically. we specify thetime point at which we want ode to record the states of the system (here we use different days withtime increments per day by specifying in each code).

Program were written in MATLAB programming to simulate the non-seasonal model given in the system Eq. (6) and result verified using detailed outputs. For numerical procedure, we select parameters values for the parameters used in system Eq. (6). We have the following interpretation for each parameter used: For the sake of numerical illustration, we choose N=1 ie S=0.7, E=0.2, I=0.1 and R=0.

From the Fig. (3), the blue line is rapid decline shows the number of people who have not yet been infected. It indicates that the disease is very contagious, with pretty evidence that every susceptibleperson being infected by day 35. The line for a less infectious disease would slope more gently to the right. The yellow line shows that the number of infected people follows the pi-curve for disease. It also changes rapidly up to a maximum number about 25% of people on day 20, and starts vanishingfrom day 70. This means near everyone has recovered. This shows that almost all the susceptible population has been infected. The point whereby the yellow and the blue lines cross for the first timeis the day when more people are in the infected category than the susceptible one. The red line shows

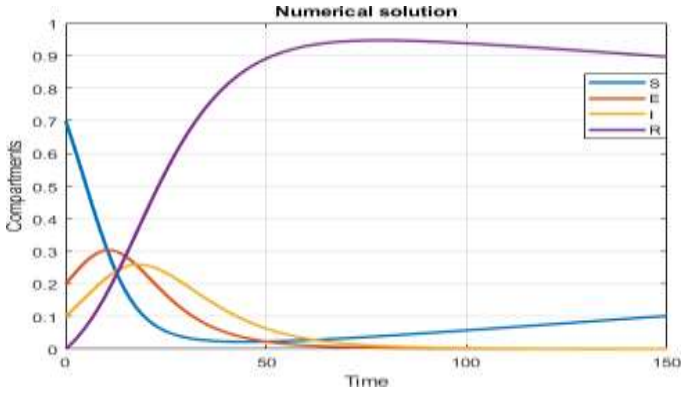


Figure 3: The numerical solution for SEIR epidemic model for $\beta = 0.507$, $\kappa = 0.108$, $\gamma = 0.104$ $S[0] = 0.7, E[0] = 0.2, I[0] = 0.1, R[0] = 0.$

The number of people who have met with the infected people but not yet infected, and the maximumnumber of exposed people is 30%

International Journal of Current Science Research and Review
ISSN: 2581-8341
Volume 05 Issue 12 December 2022
DOI: 10.47191/ijcsrr/V5-i12-20, Impact Factor: 5.995
IJCSRR @ 2022
www.ijcsrr.org

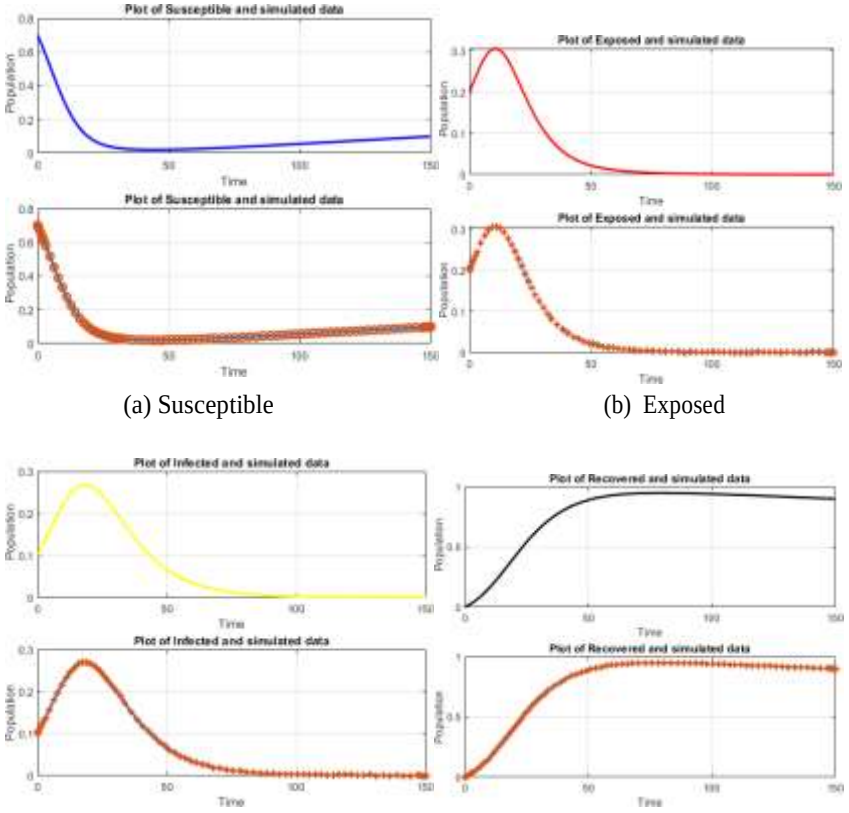
on day 10.

The pink line shows the number of people who recovered from the disease, typically by recovering,steady increase, leveling of near day 30, because there are essentially no more infected people whoneed to recover. The recovered population becomes bigger than the susceptible is where by the blueline and the pink line inter-cross (about day 12). The final effective of the removed people is 95%, which is less than the initial population. The 5% of the people died or remain in susceptible class.

2.4 Fitness of the SEIR model

The fitness of the model has been simulated by using the MATLAB software and the results are narrated in Fig. (4) which shows the fitness of our model on compartments S, E, I and R with respecttime. and we can observe that our model fits the data set exactly and gives us the confidence to continue using this model with $R^2 = 0.978$.

Regarding the relationships among the state: as κ, γ, β increases, the number exposed grows higherfaster in peak up when β increase and the susceptible down because more people are moving to theexposed class, the infected people increase as κ increase and decrease when γ increase and affect also the recovered population in increasing. The infected population increase in peak as there are more contact between the infected population and the susceptible one and the result of more contactis to more population to be infected. At the maximum level of infected graph, the infected populationdecrease since there is more strategical for reducing diseases. Of course, as time progresses, the number of recovered people increases as shown in graph with high peak because as the season of diseases becomes low as more population recovered. It is almost a mirror of the number of susceptible people. At certain values of parameters, it is possible that the susceptible and recovered appear to remain constant when there is no diseases. This indicates that the disease never reaches epidemic proportions and dies out within the population.



(a) Susceptible (b) Exposed

(c) infected (d) Recovered

Figure 4: Numerical solution with simulated data model. $\beta = 0.507$, $\kappa = 0.108$, $\gamma = 0.104$, $S[0] = 0.7$, $E[0] = 0.2$, $I[0] = 0.1$ and $R[0] = 0$.

International Journal of Current Science Research and Review
ISSN: 2581-8341
Volume 05 Issue 12 December 2022
DOI: 10.47191/ijcsrr/V5-i12-20, Impact Factor: 5.995
IJCSRR @ 2022
www.ijcsrr.org

3 SEIR Seasonality model.

A seasonal variation is component of time series which occurs periodically and yearly. The principalfactors that are the responsible for seasonal variation are climate conditions.

Some diseases occur in a cold weather whereas others are prevented in warm or hot weather. The underlying factors of this variation remains a big problem to understand. The identification of the main factors behind this seasonal variability of infectious diseases may offer the possibilities to device preventive measures, and can even help in the development of effective policies and allow a more efficient use of available resources and effective (Fares, 2011).

The nature of seasonality, exploring the consequences of the population that may affect the change for ecological system, because seasonality mechanisms can generate the complex fluctuating population.

Among those pathogen and parasites which appears seasonally, one can point out influenza and res-piratory infection which dominates and Malaria which follow the rain season in warmer region. Formathematical study, one can incorporate the periodic parameter into epidemiological model by un-derstanding how the seasonality impacts can be described mathematically and the consequences on it. The seasonality can further be simulated by susceptible hosts through year variation in hosts death or births and can cause change in underlying immunity to infection. In SEIR, we assume forms of transmission rate to have a non-seasonal SEIR model, when we have a constant of transmission rateand a seasonal SEIR model for a sinusoidal transmission rate. We assume that the transmission rate is

$$\beta(t) = \beta_0(1 + \beta_1 \cos(2\pi t)) \quad (9)$$

where β_0 is the starting level of the transmission, and β_1 is the strength of seasonality and $\beta(t)$ isthen infection rate function. When we have $\beta_0 = 0$, the SEIR model is said to be a non-seasonal model, while for $\beta_1 \in (0,1)$, we have a seasonal model (Greenhalgh and Moneim,2014).

(a) $\beta_1 = 0.2$ (b) $\beta_1 = 0.8$

Figure 5: Seasonality diagram $\beta(t)$

From the simulation of fig (5) with $\beta_1 = 0.2$ there is low periodic oscillation compare to with $\beta_1 = 0.8$. For $\beta_1 = 0.2$ the maximum number of $\beta(t)$) is 650 whereas for $\beta_1 = 0.8$ the maximum number of$\beta(t)$ is 950.

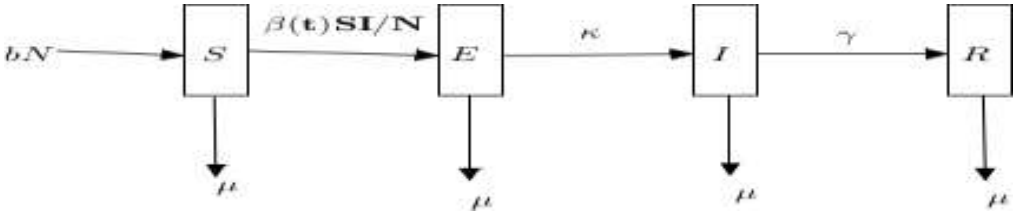


Figure 6: The general transfer diagram for the SEIR model with seasonality.

3.1 Model formulation

The model is come up to seasonal epidemics, by considering a general susceptible-exposed-infectious-recovered compartment model. The model takes the form

$$\begin{cases} \frac{dS}{dt} = bN - \beta(t)S\frac{I}{N} - \mu S \\ \frac{dE}{dt} = \beta(t)S\frac{I}{N} - E(\kappa+\mu) \\ \frac{dI}{dt} = \kappa E - I(\gamma+\mu) \\ \frac{dR}{dt} = \gamma I - \mu R \end{cases}$$

International Journal of Current Science Research and Review
ISSN: 2581-8341
Volume 05 Issue 12 December 2022
DOI: 10.47191/ijcsrr/V5-i12-20, Impact Factor: 5.995
IJCSRR @ 2022
www.ijcsrr.org

$$\begin{cases} \frac{dS}{dt} = bN - \beta(t)S\frac{I}{N} - \mu S \\ \frac{dE}{dt} = \beta(t)S\frac{I}{N} - E(\kappa+\mu) \\ \frac{dI}{dt} = \kappa E - I(\gamma+\mu) \\ \frac{dR}{dt} = \gamma I - \mu R \end{cases}$$

Table 2: The initial parameters and Estimation parameters used to find numerical solution.

Parameters	Symbol	Initial value	Estimated parameters
Birth rate	B	0.02	0.0201
beta1	β_1	2.8	2.0976
beta0	β_0	0.8	0.8138
Natural mortality rate	μ	0.002	0.0022
Recovered rate	Γ	1/7.02	1/7.1880
Infection rate	K	1/6.25	1/6.2345

Together with the following conditions:

$$S(t_0) = S_0, E(t_0) = E_0, I(t_0) = I_0, R(t_0) = R_0, R(t_0) = N - S_0 - E_0 - I_0 \quad (11)$$

And

$$\beta(t) = \beta_0(1 + \beta_1 \cos(2\pi t)) \quad (12)$$

The parameters used in the model is defined as follows: μ, γ, κ and $\beta(t)$ Eq.(12) in used in Eq.(1) stand for capita mortality rate per unit time , the transmission rate per of time, capita recovery rate per unit time, capita infection per unit time and seasonality transmission rate respectively.

4 DISEASE-FREE EQUILIBRIUM

4.1 Model rescaling

To study the analysis of the model we have first to change the model variable because the compart- ments can have a large size which is difficult to analyze, therefore it is important to use the rescaledsubject variables:

Let $\frac{ds}{dt} = \frac{ds,de}{Ndt} = \frac{de,di}{dt} = \frac{di}{Ndt}, \frac{dr}{dt} = \frac{dr}{Ndt}$.

After introducing the new variables and making simplification in Eq. (10), the system becomes,

$$\begin{cases} \frac{ds}{dt} = b - \beta(t)si - \mu s \\ \frac{de}{dt} = \beta(t)si - (\kappa+\mu)e \\ \frac{di}{dt} = \kappa e - (\gamma+\mu)i \\ \frac{dr}{dt} = \gamma i - \mu r \end{cases}$$

International Journal of Current Science Research and Review
ISSN: 2581-8341
Volume 05 Issue 12 December 2022
DOI: 10.47191/ijcsrr/V5-i12-20, Impact Factor: 5.995
IJCSRR @ 2022
www.ijcsrr.org

4.2 Equilibrium Point

An equilibrium point is the point $y \in R$ at which the derivative vanishes the differential equations $\frac{dy}{dt}|_{y=y*} - f(t, y*) = 0.$ In epidemiology model, we consider two types of equilibrium point which are disease free-equilibrium (I= 0) and endemic equilibrium point $I = 0$. The disease free-equilibrium is defined as the state where the disease is absent in the population while endemic equilibrium point is the point at which there is a disease in the population.Then, we have

$$\begin{cases} b - \beta(t)si - \mu s = 0 \\ \beta(t)si - (\kappa + \mu)e = 0 \\ \kappa e - (\gamma + \mu)i = 0 \\ \gamma i - \mu r = 0 \end{cases}$$

For the first case where i=0 . The disease free-equilibrium for SEIR seasonal model E_0 , At the beginning i= 0.

Taking Eq.(14) and replace i=0 , we have

$$s = \frac{b}{\mu} \tag{18}$$

$E_0(S, E, I, R) = (\frac{b}{\mu}, 0, 0, 0)$.

For the second case where $i \neq 0$. The disease endemic-equilibrium for SEIR seasonal model.

Taking Eq.(16), we get

$$i(t) = \frac{\kappa e}{\gamma + \mu} \tag{19}$$

Taking Eq.(15), we get

$$i(t) = \frac{(\gamma + \mu)e}{\beta(t)s} \tag{20}$$

Taking Eq.(19) and (20), we get

$$s(t) = \frac{(\kappa + \mu)(\gamma + \mu)}{\beta(t)\kappa} \tag{21}$$

Substituting the Eq.(21) into Eq.(14), we get

International Journal of Current Science Research and Review
ISSN: 2581-8341
Volume 05 Issue 12 December 2022
DOI: 10.47191/ijcsrr/V5-i12-20, Impact Factor: 5.995
IJCSRR @ 2022
www.ijcsrr.org

$$i(t) = \frac{b\kappa\beta(t) - \mu(\kappa+\mu)(\gamma+\mu)}{\beta(t)(\kappa+\mu)(\gamma+\mu)} \quad (22)$$

Replacing the Eq.(24) in Eq. (16) and Eq.(17), we get

$$e(t) = \frac{b\beta(t)\kappa - \mu(\kappa+\mu)(\gamma+\mu)}{\beta(t)\kappa(\kappa+\mu)} \quad (23)$$

and

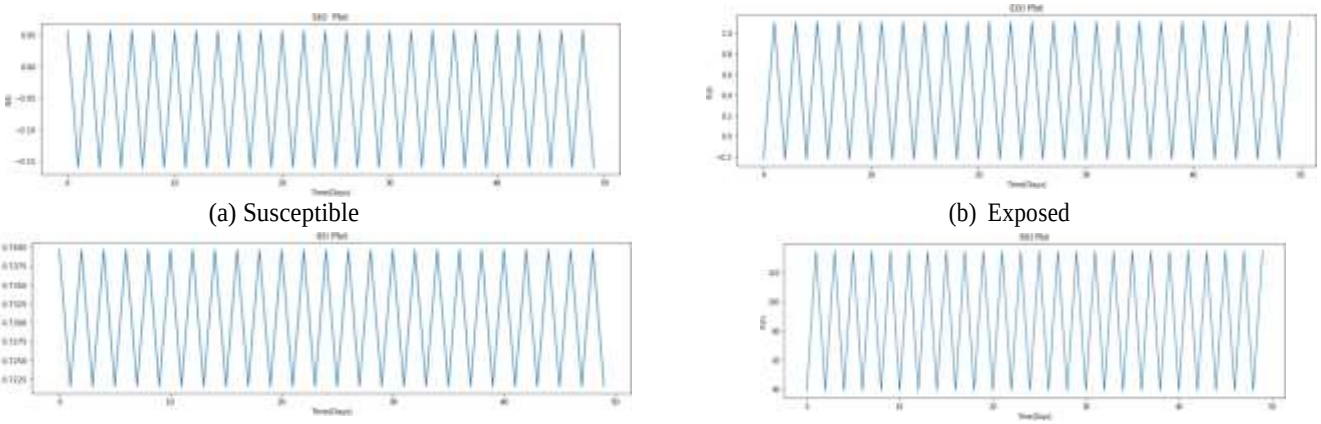
$$r(t) = \frac{(b\kappa\beta(t) - (\kappa+\mu)(\gamma+\mu))\gamma}{\beta(t)\mu(\kappa+\mu)(\gamma+\mu)} \quad (24)$$

Now, the endemic equilibrium point for SEIR model for $(s^*(t), e^*(t), i^*(t), r^*(t))$ becomes

$$\left(\frac{(\kappa+\mu)(\gamma+\mu)}{\beta(t)\kappa}, \frac{b\kappa\beta(t) - (\kappa+\mu)(\gamma+\mu)}{\beta(t)\kappa(\kappa+\mu)}, \frac{b\kappa\beta(t) - \mu(\kappa+\mu)(\gamma+\mu)}{\beta(t)(\kappa+\mu)(\gamma+\mu)}, \frac{(b\kappa\beta(t) - (\kappa+\mu)(\gamma+\mu))\gamma}{\beta(t)\mu(\kappa+\mu)(\gamma+\mu)}\right). \quad (25)$$

and the endemic equilibrium for SEIR model is valid if

$$\frac{b\kappa\beta(t) - (\kappa+\mu)(\gamma+\mu)}{\beta(t)\kappa(\kappa+\mu)} \geq 0.$$



(a) Susceptible (b) Exposed

(c) Infected (d) Recovered

Figure 7: Compartments stability with variable $\beta(t)$.

Therefore $\beta(t) \geq \frac{(k+\mu)(\gamma+\mu)}{\kappa b}$ this ensures that the number of individuals in the infectious compartment remains non negative. So, the endemic equilibrium is stable as long as $\beta(t) \geq \frac{(\kappa+\mu)(\gamma+\mu)}{\kappa b}$. Substituting the values of the parameters from the Table (2) in Eq.(25), we get $E_0^* = (0.052, 0.18, 0.738, 38.64399)$,which is approximate the same for the initial value for Fig.(7).It means that the endemic equilibrium point is stable since $I_t > 0$.

4.3 The stability analysis for disease free-equilibrium

To make a study of the disease-free state, we have to analyses the model around disease-free equilibrium E_0. We compute Jacobian matrix at E_0, then find eigenvalues of the Jacobian matrix which helps to analyze the stability of equilibrium point. If all eigenvalues are real and negative then thedisease-free equilibrium is asymptotically stable. Whereas the disease-free equilibrium is said to beunstable if eigenvalues have distinct signs (one positive, others negative). If there are complex eigen-values with the negative real part then the disease-free equilibrium is table and unstable otherwise.

International Journal of Current Science Research and Review
ISSN: 2581-8341
Volume 05 Issue 12 December 2022
DOI: 10.47191/ijcsrr/V5-i12-20, Impact Factor: 5.995
IJCSRR @ 2022
www.ijcsrr.org

Let $X = (S, E, I, R)^T$, then the system Eq.(6) can be written as

$$\frac{d\bar{x}}{dt} = \mathcal{F}(\bar{x}) - \mathcal{V}(\bar{x})$$

$$J(S,E,I,R) = \begin{bmatrix} \frac{\partial S}{\partial S} & \frac{\partial S}{\partial E} & \frac{\partial S}{\partial I} & \frac{\partial S}{\partial R} \\ \frac{\partial E}{\partial S} & \frac{\partial E}{\partial E} & \frac{\partial E}{\partial I} & \frac{\partial E}{\partial R} \\ \frac{\partial I}{\partial S} & \frac{\partial I}{\partial E} & \frac{\partial I}{\partial I} & \frac{\partial I}{\partial R} \\ \frac{\partial R}{\partial S} & \frac{\partial R}{\partial E} & \frac{\partial R}{\partial I} & \frac{\partial R}{\partial R} \end{bmatrix}$$

$$= \begin{bmatrix} -\beta(t)i - \mu & 0 & -\beta_t s & 0 \\ \beta(t)i & -(\kappa+\mu) & \beta_t s & 0 \\ 0 & \kappa & -(\gamma+\mu) & 0 \\ 0 & 0 & -\gamma & -\mu \end{bmatrix}$$

Hence, we can compute Jacobian matrix around $E_0(S,E,I,R) = \left(\frac{b}{\mu}, 0, 0, 0\right)$

$$J|_{E_0} = \begin{bmatrix} -\mu & 0 & -\beta(t)\frac{b}{\mu} & 0 \\ 0 & -(\kappa+\mu) & \beta(t)\frac{b}{\mu} & 0 \\ 0 & \kappa & -(\gamma+\mu) & 0 \\ 0 & 0 & -\gamma & -\mu \end{bmatrix}$$

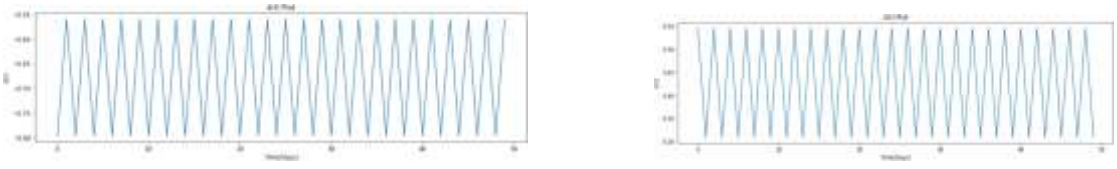
We proceed to compute the eigenvalues of the above Jacobian matrix via the characteristic equation given as

$$J|_{E_0} - \lambda I = \begin{vmatrix} -\mu-\lambda & 0 & -\beta(t)\frac{b}{\mu} & 0 \\ 0 & -(\kappa+\mu)-\lambda & \beta(t)\frac{b}{\mu} & 0 \\ 0 & \kappa & -(\gamma+\mu)-\lambda & 0 \\ 0 & 0 & -\gamma & -\mu-\lambda \end{vmatrix}$$

$$= (\mu+\lambda)^2\left(\kappa+\mu)(\gamma+\mu-\kappa\beta_0\frac{b}{\mu}) + (\gamma+2\mu+\kappa)\lambda+\lambda^2\right) = 0.$$

$$\lambda_1 = \lambda_2 = -\mu, \lambda_{3,4} = \frac{-(\gamma+2\mu+\kappa) \pm \sqrt{(\gamma+2\mu+\kappa)^2 - 4(\gamma+\mu-\kappa\beta_0\frac{b}{\mu})}}{2}. \quad (26)$$

Substituting the values of the parameters from the Table (2) into Eq.(26), we get the eigenvalues of Jacobian matrix at disease free-equilibrium which are all real with distinct roots (one positive andother negative sign) : $\lambda_1 = \lambda_2 = -0.001, \lambda_3 = -0.3889$, $\lambda_4 = 0.1379$.
From Fig.(8), the $\lambda_1(t)$ the oscillations is always in negative side whereas $\lambda_2(t)$ is in positive side. Itmeans that the endemic equilibrium point is saddle point unstable which means that disease will



(a) $\lambda_1(t)$ (b) $\lambda_2(t)$

Figure 8: Seasonality diagram $\lambda(t)$

International Journal of Current Science Research and Review
ISSN: 2581-8341
Volume 05 Issue 12 December 2022
DOI: 10.47191/ijcsrr/V5-i12-20, Impact Factor: 5.995
IJCSRR @ 2022
www.ijcsrr.org

4.4 The stability analysis for disease at endemic equilibrium.

$$J(S,E,I,R) = \begin{bmatrix} \frac{\partial S}{\partial S} & \frac{\partial S}{\partial E} & \frac{\partial S}{\partial I} & \frac{\partial S}{\partial R} \\ \frac{\partial E}{\partial S} & \frac{\partial E}{\partial E} & \frac{\partial E}{\partial I} & \frac{\partial E}{\partial R} \\ \frac{\partial I}{\partial S} & \frac{\partial I}{\partial E} & \frac{\partial I}{\partial I} & \frac{\partial I}{\partial R} \\ \frac{\partial R}{\partial S} & \frac{\partial R}{\partial E} & \frac{\partial R}{\partial I} & \frac{\partial R}{\partial R} \end{bmatrix}$$

$$= \begin{bmatrix} -\beta_t i - \mu & 0 & -\beta(t)s & 0 \\ \beta_t i & -(\kappa+\mu) & \beta(t)s & 0 \\ 0 & \kappa & -(\gamma+\mu) & 0 \\ 0 & 0 & -\gamma & -\mu \end{bmatrix}.$$

Hence, we can compute $E_0^*(S^*,E^*,I^*,R^*) = \left(\frac{(\kappa+\mu(\gamma+\mu)}{\beta(t)\kappa}\right), \frac{b-(\kappa+\mu)(\gamma+\mu)}{\beta(t)\kappa(\kappa+\mu)}, \frac{b-(\kappa+\mu)(\gamma+\mu)}{\beta(t)(\kappa+\mu)(\gamma+\mu)}, \frac{(b-(\kappa+\mu)(\gamma+\mu))\gamma}{\beta(t)\mu(\kappa+\mu)(\gamma+\mu)}\right)$

$$J|_{E_0} = \begin{bmatrix} -\left(\frac{b\kappa\beta(t)-(\kappa+\mu)(\gamma+\mu)}{(\kappa+\mu)(\gamma+\mu)}\right) - \mu & 0 & -\left(\frac{(\kappa+\mu)(\gamma+\mu)}{\kappa}\right) & 0 \\ \frac{b\kappa\beta(t)-(\kappa+\mu)(\gamma+\mu)}{(\kappa+\mu)(\gamma+\mu)} & -(\kappa+\mu) & \frac{(\kappa+\mu)(\gamma+\mu)}{\kappa} & 0 \\ 0 & \kappa & -(\gamma+\mu) & 0 \\ 0 & 0 & -\gamma & -\mu \end{bmatrix}.$$

We proceed to compute the eigenvalues of the above Jacobian matrix via the characteristic equation given as

$$J|_{E_0} - \lambda I = \begin{vmatrix} -\left(\frac{b\kappa\beta(t)-(\kappa+\mu)(\gamma+\mu)}{(\kappa+\mu)(\gamma+\mu)}\right) - \mu - \lambda & 0 & -\left(\frac{(\kappa+\mu(\gamma+\mu)}{\kappa}\right) & 0 \\ \frac{b\kappa\beta(t)-(\kappa+\mu)(\gamma+\mu)}{(\kappa+\mu)(\gamma+\mu)} & -(\kappa+\mu)-\lambda & \left(\frac{(\kappa+\mu)(\gamma+\mu)}{\kappa}\right) & 0 \\ 0 & \kappa & -(\gamma+\mu)-\lambda & 0 \\ 0 & 0 & -\gamma & -\mu-\lambda \end{vmatrix}$$

$$= (\mu+\lambda)\left[\left(\frac{b\kappa\beta(t)-(\kappa+\mu)(\gamma+\mu)}{(\kappa+\mu)(\gamma+\mu)} + \mu + \lambda\right)(\kappa+\mu+\lambda)(\mu+\gamma+\lambda)\left((\mu+\lambda)(\kappa+\mu)(\gamma+\mu)\right)\right]$$

The eigenvalues provided by Jacobian matrix at endemic equilibrium point after solving the equationand replace the initial conditions are all real with distinct signs (one positive and others negative): $\lambda_1 = -0.001, \lambda_2 = 0.6017, \lambda_3 = -0.1855, \lambda_4 = -0.0837$.
It means that the endemic equilibrium point is saddle point unstable which means that disease will invade the population.

4.5 The basic reproduction number

The basic reproduction number indicates the number of people who can be contaminated by one infected person. (Jones, 2007) explains how to determine R_0 using next-generation matrix
$H = FV^{-1}$, R_0 is the absolute value of largest eigenvalue of matrix H.
From the Eq. (13), we have,

The eigenvalues of FV^{-1} is given as

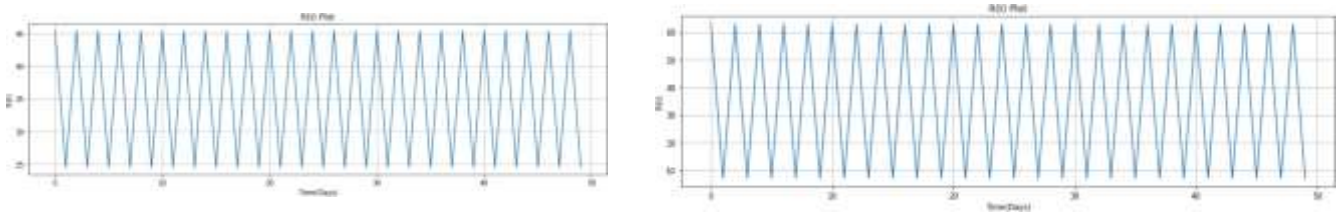
$$\lambda_1 = 0, \lambda_2 = \frac{\beta(t)b}{(\kappa+\mu)(\gamma+\mu)\mu}$$

This gives

$$R_0 = \frac{\beta(0)b}{(\kappa+\mu)(\gamma+\mu)\mu}$$

International Journal of Current Science Research and Review
ISSN: 2581-8341
Volume 05 Issue 12 December 2022
DOI: 10.47191/ijcsrr/V5-i12-20, Impact Factor: 5.995
IJCSRR @ 2022
www.ijcsrr.org

Since the spectral radius (maximum eigenvalues) is the basic reproduction number and its numerical value is $R_0 = 45.2988$.
$R_0 > 1$, In other words, disease spreads in the population. The disease is therefore an epidemic, mean- ing it will invade the population if no measurement taken by government.



(a) $\beta_1 = 0.2$ (b) $\beta_1 = 0.8$

Figure 9: Plot of $R(t)$ for seasonality SEIR model.

From the fig (9), the reproduction number $R(t)$ increase when β_1 increase and cause the high oscillation to the system. The $R(t)$ shows that the population will affect with seasonal diseases for each seasonal and the diseases will remain in the population for long time if no measurement taken by thegovernment. The results shows that the maximum reproduction number for each season with $\beta_1 = 0.2$ and $\beta_1 = 0.8$ is 45.2988, 65.32 respectively.

4.6 Results and Interpretation

After estimating the model parameters, we have found the estimated value of disease-free equilibrium, endemic equilibrium point and basic reproduction number R_0. We were also enabled to declare disease free equilibrium stability as well as the endemic equilibrium point. We find the disease-freeequilibrium by using the estimated values.
By using the estimated values, we find the disease-free equilibrium for SEIR seasonality

$E_0(S,E,I,R) = (\frac{b'}{\mu}0,0,0)$

and the eigenvalues

$$\lambda_1 = \lambda_2 = \mu, \lambda_{3,4} = \frac{(\gamma + 2\mu + \kappa) \pm (\gamma + 2\mu + \kappa)^2 - 4\left(\gamma + 2\mu - \kappa\beta_0 \frac{b}{\mu}\right)}{2}$$

and numerical values is
$(-0.011, -0.001, -0.3971, 0.1453)$

Since all are real with one positive sign and others negative sign, we say that the diseases at free equilibrium point is saddle point unstable which means that the diseases will remain in the populationif there are no measures taken on it. means there is no seasonal diseases in the population.
The endemic equilibrium point was also calculated and the numerical value is

$E_0^* = (0.052, -0.18, 0.738, 38.64399)$.

The eigenvalues provided by Jacobian matrix at endemic equilibrium point are
$\lambda_1 = -0.001, \lambda_2 = 0.6017, \lambda_3 = -0.1855, \lambda_4 = -0.0837$
and are real with distinct signs (one positive and others negative), this shows that the endemic equilibrium point is saddle point unstable and the diseases will remain in the population if no measurement taken eradicate it.
The basic production number is

$RO = \frac{\beta O b}{K(\mu+\gamma)(\mu+\eta)} = 44.29$

The seasonal disease remains in the population if no measurement taken by the government.

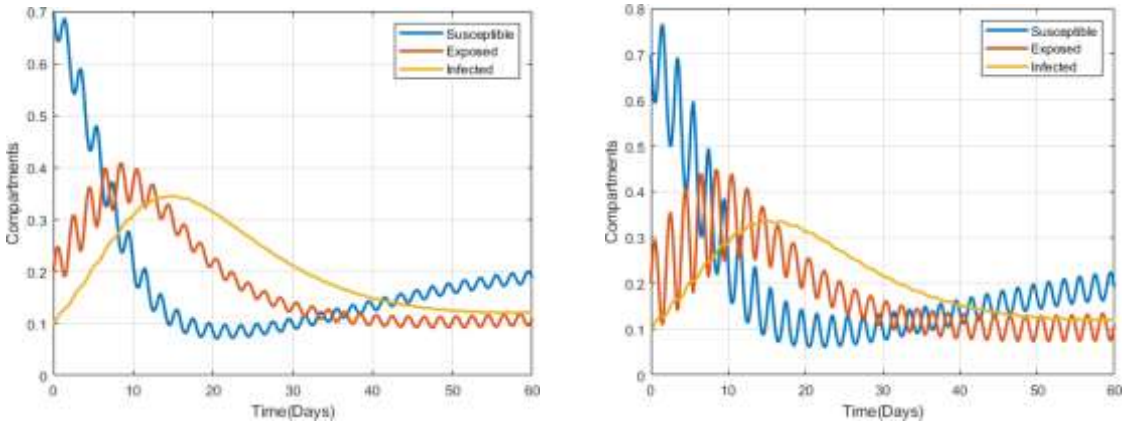
International Journal of Current Science Research and Review
ISSN: 2581-8341
Volume 05 Issue 12 December 2022
DOI: 10.47191/ijcsrr/V5-i12-20, Impact Factor: 5.995
IJCSRR @ 2022
www.ijcsrr.org

4.7 Numerical solution

In this section we present the numerical solutions of our model.

The Fig. (10) represents the behavior of SEIR seasonality model with different incidence peak for each compartment. To compare (a) and (b), we have to look the behavior of each compartment for the two-transmission rate of seasonality used. The blue line oscillation shows that the number of susceptiblepopulations decrease as the oscillation also becomes small due to the fact that more population move to exposed class as the sinusoidal oscillations becomes low because the number of susceptible becomes very low. The red line shows that the exposed population with high rate of sinusoidal oscillation till the maximum of 40% (a), 45% (b) and starts decreasing because the diseases is increasing in the population more population move from exposed class to infected class. For the yellow line where theoscillation starts to be low because there are remaining small number of susceptible populations.

At the maximum level of infected graph, the infected population decrease because the susceptible population is getting low or other strategical for eradication of diseases.



(a) $\beta_1 = 2.0976$ (b) $\beta_1 = 5.0976$

Figure 10: Numerical solution for seasonality SEIR model

4.8 Fitness of the model

To see if the model will be useful in prediction, we have to check if the model fits the collected orsimulated data. In this project, we use simulated data.

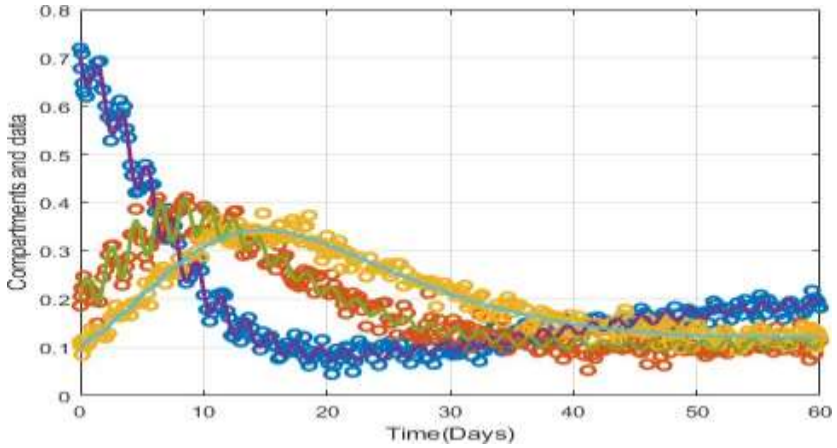


Figure 11: Numerical solution with simulated data model.

Fig (11) indicates how our model fits the simulated data. We can say that the model fits very well the data because there are few outliers (observation points which are distant from others) and we see that the number of susceptible decreases slowly by slowly due to the contagious of diseases whereas the infected and exposed people increase, and also at the maximum level the infected people decreasedue to the people die with other natural diseases or other strategies for eradication the diseases.

International Journal of Current Science Research and Review
ISSN: 2581-8341
Volume 05 Issue 12 December 2022
DOI: 10.47191/ijcsrr/V5-i12-20, Impact Factor: 5.995
IJCSRR @ 2022
www.ijcsrr.org

5 CONCLUSIONS AND RECOMMENDATIONS

5.1 Conclusions

In this work, we have converted the classical SEIR epidemic model with seasonal fluctuations into anapproximation system and disused its dynamical behaviors.

Having started with the analysis of the dynamics features of the epidemics without seasonality, we have used different time interval, considering the evolution of diseases depends on susceptible, infectious, exposed and recovered density. From SEIR model, we have derived the equilibrium point atdisease free equilibrium which is unstable and the basic reproduction number which is greater than1 which shows that the disease will remain in the population if no measurement taken. For SEIR seasonality model, the disease at free equilibrium point is unstable and the basic reproduction number

$R_0 = 44.29$, which means the disease will remain in the population.

We have seen also the seasonal pattern of infectious diseases which is established that rates of trans- mission peak at the start of season and steadily decline reaching through during the end of season months. In our model, the oscillations in the incidence of diseases are frequently observed, even in non-seasonal infections where at least partially protective immunity leads to a decline in the susceptible population during an epidemic. A subsequent period of low incidence flows, while the susceptible population is replenished until an epidemic can occur. The introduction of a seasonal forcing provides the discrete SEIR model with potential to generate more complex oscillatory with sinusoidal behavior. Given the importance of controlling this sinusoidal behavior of diseases, the necessity of havingpredictable densities for the epidemic's population, we have to plan vaccination of newborn infants,drugs and other strategical that can reduce diseases depends on season, which has acted as an effected control strategy.

5.2 Recommendations

The diseases come to different season in each year. The government and pharmaceutical companieshave to explore different potential types of vaccination depends on seasonal diseases which can be used at the same time in immunization of susceptible and curing them with seasonal diseases. For future research, we recommend them the development of seasonal diseases model with different types of vaccination for different diseases according to season (like precipitation, humidity and other climatechange). Public health actions including education of various health care providers and improved communication to patients should be pursued to achieve higher coverage of the vaccine for seasonal diseases in the at-risk population, as well as to enhance interventions.

Finally, as mathematical modeling can be used as a tool for diseases control strategy, we recommendthe next researchers can predict the vaccination for each kind of seasonal diseases.

REFERENCES

1. AL-AJAM, M. R., BIZRI, A. R., MOKHBAT, J., WEEDON, J., and LUTWICK, L. (2006). Mu cormycosis in the eastern Mediterranean: a seasonal disease. Epidemiology and Infection,134(2):34-46.
2. Al-Sheikh, S. A. (2012). Modeling and analysis of a seir epidemic model with a limited resource for treatment. Global Journal of Science Frontier Research: Mathematics and Decision Sciences, 12(14):57–66.
3. Altizer, Sonia, e. a. (2006a). Seasonality and the dynamics of infectious diseases. Ecology letters, 9.4:467–484.
4. Altizer, Sonia, e. a. (2006b). "Seasonality and the dynamics of infectious diseases.". pages 467–484.
5. Anderson, R. M. and May, R. M. (1979). Population biology of infectious diseases: Part i. Nature 280.5721, (7).
6. Aron, J. L. and Schwartz, I. B. (1984). Seasonality and period-doubling bifurcations in an epidemic model. Journal of theoretical biology, 110(4):665–679.
7. B., I. and H. L. Smith., S. (1983). Infinite subharmonic bifurcation in an seir epidemic model. Journal of mathematical biology, 18.3:233–253.
8. Bauch, C. and Earn, D. J. (2003). Interepidemic intervals in forced and unforced seir models. Fields Inst. Commun, 36:33–44.
9. Beard, C. B., Pye, G., Steurer, F. J., Rodriguez, R., Campman, R., Peterson, A. T., Ramsey, J., Wirtz, R. A., and Robinson, L. E. (2003). Chagas disease in a domestic transmission cycle in southernTexas, usa. Emerging infectious diseases, 9(1):103.
10. Bernoulli, D. (1760). Essai d'une nouvelle analyse de la mortalité causée par la petite vérole, et des avantages de l'inoculation pour la prévenir. Histoire de l'Acad., Roy. Sci.(Paris) avec Mem, pages 1–45.
11. Diekmann, O., Heesterbeek, J. A. P., and Metz, J. A. (1990). On the definition and the computation of the basic reproduction

ratio r 0 in models for infectious diseases in heterogeneous populations.Journal of mathematical biology, 28(4):365–382.
12. Dietz, K. (1976). The incidence of infectious diseases under the influence of seasonal fluctuations. Mathematical models in medicine, pages 1–15.
13. Driessche, P. and Watmough, J. (2008). Mathematical epidemiology.
14. Earn, D., Dushoff, J., and Levin, S. (2002). Ecology and evolution of the flu. Trends in Ecology andEvolution, 17(7):334–340.
15. Fares, A. (2011). Seasonality of tuberculosis. Journal of global infectious diseases, 3(1):46.
16. Feng, Z., Xu, D., and Zhao, H. (2009). The uses of epidemiological models in the study of diseasecontrol. In Modeling and Dynamics of Infectious Diseases, pages 150–166. World Scientific.
17. Fine, P. E. and Clarkson, J. (1982). The recurrence of whooping cough: possible implications for assessment of vaccine efficacy. The Lancet, 319(8273):666–669.
18. Grassly, N. C. and Fraser., C. (2006). Seasonal infectious disease epidemiology. Proceedings of the Royal Society B: Biological Sciences, 273.1600:2541–2550.
19. Grassly, N. C. and Fraser, C. (2008). Mathematical models of infectious disease transmission. Nature Reviews Microbiology, 6(6):477.
20. Greenhalgh, D. and Moneim, I. (2014). Periodicity in general seasonally driven epidemic models. Heffernan, J. M., Smith, R. J., and Wahl, L. M. (2005). Perspectives on the basic reproductive ratio. Journal of the Royal Society Interface, 2(4):281–293.
21. Hethcote, H. W. (2000). The mathematics of infectious diseases. SIAM Review, 42(4):599–653. Keeling, M. J. and Rohani, P. (2011). Modeling infectious diseases in humans and animals. Princeton University Press.
22. Kermack, W. O. and McKendrick, A. G. (1927). A contribution to the mathematical theory of epi- demics. Proceedings of the royal society of London. Series A, Containing papers of a mathematicaland physical character, 115(772):700–721.
23. Li, J. (2012). Discrete-time models with mosquitoes carrying genetically-modified bacteria. Mathematical biosciences, 240(1):35–44.
24. London, W. P. and Yorke, J. A. (1973). Recurrent outbreaks of measles, chickenpox and mumps: I. seasonal variation in contact rates. American journal of epidemiology 98, (6):453–468.
25. LONDON, W. P. and YORKE, J. A. (1973). RECURRENT OUTBREAKS OF MEASLES, CHICK- ENPOX AND MUMPS: I. SEASONAL VARIATION IN CONTACT RATES1. American Journal of Epidemiology, 98(6):453–468.
26. Lynch, J. and Smith, G. D. (2005). A life course approach to chronic disease epidemiology. Annu. Rev. Public Health, 26:1–35.
27. M. Y. Li, J. S. M. (1995). Global stability for the seir model in epidemiology. math. biosci. 5:155–164.Organization, W. H. et al. (2002). Scaling up the response to infectious diseases. Accessed February, 3:2010.
28. Rohani, Pejman, M. J. K. and Grenfell., B. T. (2002). The interplay between determinism and stochas-ticity in childhood diseases. American journal of epidemiology 98, pages 469–481.
29. Ross, R. (1911). The prevention of malaria. John Murray; London.
30. Shah, N. and Jyoti, G. (2013). Seir model and simulation for vector borne diseases. Applied Mathe- matics, 4:13 – 17.
31. Shah, N. H. and Gupta, J. (2013). Seir model and simulation for vector borne diseases. AppliedMathematics, 4.8:13.
32. Smith, H. (1983). Multiple stable subharmonics for periodic epidemic model. 17:179âĂŞ190.
33. Stone, L., Olinky, R., and Huppert, A. (2007). Seasonal dynamics of recurrent epidemics. Nature,446(7135):533.
34. Turner, K. (2010). Introduction to infectious disease modelling. Sexually transmitted infections, 87.
35. Van den Driessche, P. and Watmough, J. (2002). Reproduction numbers and sub-threshold endemicequilibria for compartmental models of disease transmission. Mathematical biosciences, 180(1- 2):29–48.