# Mathematical Analysis of SEITR Model for Influenza Dynamics

K. Arun Kumar [1] and A.Venkatesh[2*]

[1,2]Department of Mathematics,
AVVM Sri Pushpam College (Affiliated to Bharathidasan University),
Poondi, Thanjavur(Dt), Tamilnadu, India-613 503.

December 28, 2022

**Abstract**

In order to give timely hospitalisation for infections that are dangerously ill, our primary goal is to reduce the interaction between susceptibles and infections. For this we add treatment T as a fifth compartment to the SEIR model, converting it from SEIR to SEITR. The stabilities of endemic equilibrium and disease-free equilibrium were tested. The next generation matrix method was used to calculate the SEITR model's basic reproduction number. Numerical simulations were also presented to validate our analytic findings. A graphic depicted the impact of parameters on infected populations. It was perceived that, anytime the treatment rate increased, the infected population, exposed population, and treated population all declined but the susceptible population increased.

**Keywords:** SEITR model, basic reproduction number, stability and numerical simulation.
**AMS Subject Classification:** 34D20

## 1 Introduction

Kermack and McKendrick [21] introduced the first mathematical model, SIR (Susceptible-Infectious-Recovered), early in the 20th century. Later Anderson and May[1] were proposed the SEIR model by adding Exposed (E) as fourth compartment to SIR model to define the spread of epidemic. Many authors introduced a numerous extended SEIR models to define the infectious diseases spread and their preventions [7]. ZhilanFeng (2007) [31] developed a SEIR model which has been used to evaluate the electiveness of different control strategies for the size of endemic with separation and isolation. Rafiqul Islam et al [16] was proposed an SEIR model to analysis the influenza in Bangladesh. Vinod kumar bais and Deepak kumar [29] was introduced a model SITR emphasized the condition of the dynamical classic to the transmission populace of H1N1 virus. By combining these two SEIR and SITR models we developed an new SEITR model by including treatment T as a fifth compartment to investigate the dynamics of the influenza epidemic's transmission. Hethcote and Yorke [14] were charity models to analyze the gonorrhea controller techniques, such as showing, outling infectors, post treatment and vaccination.

*Corresponding author: avenkateshmaths@gmail.com

Chinviriyasit (2007) was introduced a dynamic SIRC model [6] to study the Numerical exhibiting modeling of the spread dynamics of influenza. Samuel Abubakar (2013) was proposed a model [25] to investigation the spread of infectious disease and stability of disease in population. Various researchers such as Andreasen et al. (1997)[2], Hethcote (2000), Earn et al. (2002)[15], Casagrandi et al. (2006)[10], Murray et al. (2008) [23] have been studied the dynamics of influenza and they recommended mathematical models to revision the spread of H1N1 and control the influenza epidemic. Over the past several decades, the field of FDEs has made considerable advancements. To examine the dynamical behaviour of a fish farm in relation to an arbitrary order Atangana-Baleanu derivative, Jagdev et al. [19] suggested a fraction fish farm model. By Jagdev Singh [18], a fractional guava fruit model with memory outcome was introduced. To analyse the COVID-19 trend, Supriya, Yadav et al [28] created the FDE model. A fractional model was created by Jagdev Singh and Arpita Gupta[17] to analyse the results of nonlinear partial modified. To study malaria transmission, Rehman, Attiq ul, et al [**?**] proposed a 9 compartment FDE model. A simple influenza(H1N1) model by means of optimal control studied by Srivastav. A. K et al. (2016) [27], Also Mishra et al. (2013) [22], consume suggested a mathematical model to analyze the spread and control of influenza between two economic groups. Christian Quirouette et al[24] developed to unfolding the localization and spread of influenza virus inside the human breathing area. The Mathematical model [3], plays a crucial role to learning the spread dynamics of the Contagious Disease Influenza, and control the virus through isolation, treatment and vaccination of infected population. Environmental contaminations, global warming, ecosystems, roving etc. are main reasons to spread the contagious diseases. So that certain assumptions and parameters are considered to formulate the model. Influenza is a breathing contagious disease instigated by influenza virus[18], which is also known as flu and it has three kinds A, B and C. This virus spreads easily in the population very fast through the air from coughing, sneezing and through contact by the hands touching our eyes, nose or mouth etc. Communal symptoms of H1N1 are high fever, pain, sore gorge, muscle pain, coughing and weariness [11]. The symptoms were appeared after two days and it has been at most one week [12] but cough may last more than two weeks. Each year individuals are infected by this virus an outbreak particularly in the winter session. The formulation and analysis of the SEITR model were briefly detailed in this article. The analyses of the model, together with the findings on local and global stability, as well as the presence of endemic equilibrium, were investigated. Numerical evidence was used to establish an analytical conclusion. It was seen that if the rate of treatment increased, the susceptible population rose while the infected, exposed, and treated populations all decreased. The limitations of the SEITR model is that it oversimplifies complicated disease processes while still being easily calculable. The SEITR model does take this parameter into account, however additional model extensions would be required.

## 2 Model Formation

In this study we proposed a new model SEITR by adding treatment T as fifth compartment to SEIR model to analyze the spread dynamics of epidemic Influenza in India. The total populace $N(t)$ at time $t$ is separated into five different populaces , namely, Susceptible populace $S(t)$ at time $t$, Exposed populace $E(t)$ at time $t$, Infected inhabitants $I(t)$ at time $t$, Treatment populace $T(t)$ at time $t$, and Recovered populace $R(t)$ at time $t$. The susceptible $(S(t))$ populace are those who are at possibility to become infected by virus. The exposed $(E(t))$ populace are those who are infested by virus but not yet infectious that is not able to infect others. The infected populaces are those who

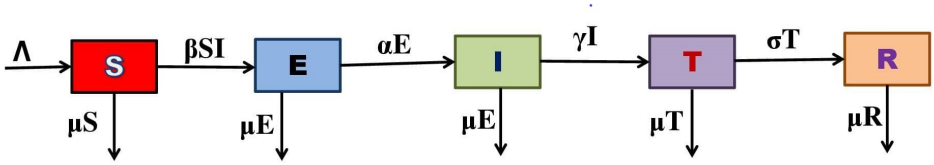


Figure 1: Schematic diagram of SEITR model

are diseased and able to infect others. The treatment populations are those who are infected and taking treatment in hospitals. The recovered populations are those who are recovered after treatment.
The flow diagram of influenza model was presented in fig1.

The susceptible human populace is created by the inflow rate of humans into the populace (at the rate $\wedge$) and the natural death rate $\mu$. Therefore the incidence rate $\beta SI$ incorporate the transmission frequency at which susceptible individuals becomes exposed and entered exposed populace without being infectious. Thus the rate of change of susceptible human populace is given by

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

The exposed human populace at the rate $\alpha$ be the exposed rate which exposed individuals becomes infected but not infectious and entered into infected populace and the natural death rate $\mu$. Thus the rate of variation of exposed human populace is specified by

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

The infected human populace at the rate $\gamma$ be the people are joined in hospital for treatment populace and the natural death rate $\mu$. Thus the rate of variation of infected human populace is specified by

$$\frac{dI}{dt} = \alpha E - (\gamma + \mu)\, I$$

The treatment human populace at the rate $\sigma$ be a rate at which the treatment individuals recovered and entered into recovered populace. Hence the rate of variation of treatment human populace is specified by

$$\frac{dT}{dt} = \gamma I - (\sigma + \mu)\, T$$

Finally, the rate of variation of recovered human populace is specified by

$$\frac{dR}{dt} = \sigma T - \mu R$$

By using all above assumptions, a nonlinear structure of five differential equations for

Table 1: Complete Description of relative parameters of the SEITR model

| Parameter | Depiction |
|---|---|
| $\wedge$ | inflow rate of susceptible individuals |
| $\mu$ | Normal death rate |
| $\beta$ | Rate at which susceptible populace becomes exposed |
| $\alpha$ | Rate at which exposed populace becomes infected |
| $\gamma$ | Rate at which infected populace getting treatment |
| $\sigma$ | Rate at which treatment populace getting recovered |

SEITR model is formed as follows

$$\begin{cases} \frac{dS}{dt} &= \wedge - \beta SI - \mu S \\ \frac{dE}{dt} &= \beta SI - (\alpha + \mu)E \\ \frac{dI}{dt} &= \alpha E - (\gamma + \mu)I \\ \frac{dT}{dt} &= \gamma I - (\sigma + \mu)T \\ \frac{dR}{dt} &= \sigma T - \mu R \end{cases} \tag{1}$$

Where the primary conditions $S(0) \geq 0$, $E(0) \geq 0$, $I(0) \geq 0$, $T(0) \geq 0$ and $R(0) \geq 0$. The total population $N(t) = S(t) + E(t) + I(t) + T(t) + R(t)$ will be assumed as constant.

# 3 Analysis of the SEITR model

In the segment, the elementary belongings of SEITR model 1 such as positivity and boundedness of the solution, basic reproduction number and stability analysis were discorsed.

## 3.1 Positivity and boundedness

**Theorem 1.** *All the solutions ($S(t)$, $E(t)$, $I(t)$, $T(t)$, $R(t)$) $\in R_+^5$ of the sturcture 1 with primary condition $S(t) \geq 0$, $E(t) \geq 0$, $I(t) \geq 0$, $T(t) \geq 0$, and $R(t) \geq 0$ are nonnegative and uniformly bounded for all $t \geq 0$.*

**Proof 1.** *Assume that ($S(t)$, $E(t)$, $I(t)$, $T(t)$, $R(t)$) $\in R_+^5$ is a solution of 1 for $t \in [0, t_0)$, where $t_0 > 0$.*
*Through $1^{st}$ equation of system 1, we get*

$$\frac{dS}{dt} = \wedge - \beta^* S^* I - \mu^* S \geq \wedge - \phi(t)^* S.$$

*where $\phi(t) = \beta^* I + \mu$*
*After integration, we get*

$$S(t) = S_0 \exp\left(-\int_0^t \phi(s)ds\right) + \wedge \exp\left(-\int_0^t \phi(s)ds\right) \int_0^t e^{\int_0^s \phi(u)du} ds \geq 0 \geq 0.$$

$\Rightarrow S(t) \geq 0$.
*From the $2^{nd}$ equation of system 1, we develop*

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

*Which leads*

$$E(t) = E_0 \exp\left(-\int_0^t (\alpha+\mu)\, ds\right) \geq 0$$

$\Rightarrow E(t) \geq 0$
*From the* $3^{rd}$ *equation of system 1, we acquire*

$$\frac{dI}{dt} = \alpha E - (\gamma+\mu) \geq -(\gamma+\mu) I$$

*Which leads*

$$I(t) = I_0 \exp\left(-\int_0^t (\gamma+\mu)\, ds\right) \geq 0.$$

$\Rightarrow I(t) \geq 0$
*Similarly* $4^{th}$ *and* $5^{th}$ *equation of system 1*

$$\frac{dT}{dt} = \gamma I - (\sigma+\mu) T \geq -(\sigma+\mu) T$$

*Which leads to*

$$T(t) = T_0 \exp\left(-\int_0^t (\sigma+\mu)\right) ds \geq 0$$

$\Rightarrow T(t) \geq 0$

$$\frac{dR}{dt} = \sigma T – \mu R \geq -\mu R$$

*which leads to*

$$R(t) = R_0 \exp\left(-\int_0^t \mu ds\right) \geq 0$$

$\Rightarrow R(t) \geq 0$
*Hence, the results (S, E, I, T, R) of 1 sustaining the primary conditions* $S(t) \geq 0$, $E(t) \geq 0$, $I(t) \geq 0$, $T(t) \geq 0$, *and* $R(t) \geq 0$ *for all* $t \in [0, t_0)$ *are nonnegative in the section* $[0, t_0)$.
*Now, we demonstrate that the boundedness of clarifications of system 1.*
*The positivity of the solutions indicates that*
$\frac{dS}{dt} \leq \wedge$ - $\mu S$
*From the beyond equation, we can write that* $\lim_{t\to\infty} \sup S \leq \frac{\wedge}{\mu}$ *and* $S \leq \frac{\wedge}{\mu}$.
*Consider the total populations* $N = S + E + I + T + R$.
*On differentiation gives* $\frac{dN}{dt} \leq \wedge$ -$\mu N$ *which leads to* $\lim_{t\to\infty} \sup N \leq \frac{(\wedge)}{(\mu)}$.
*Then, we get* $N \leq \frac{\wedge}{\mu}$
$\Rightarrow S + E + I + T + R \leq \frac{\wedge}{\mu}$
*Therefore all the solution curves* $(S, E, I, T, R)$ *sustaining by the primary conditions are consistently bounded in* $R_+^5$ *and in the section*
$\Omega = \left\{(S, E, I, T, R) \in R_+^5 : 0 \leq (S, E, I, T, R) \leq \frac{\wedge}{\mu}\right\}$.

## 3.2 Basic Reproduction Number

A crucial factor for communicable disease is the Basic Reproduction Number ($R_0$) which is distinct as the middling number of subordinate cases obtained by distinct primary case during the infectious dated in a susceptible populace. With $R_0$, the epidemic growth rate can be estimated and Stability of model will be analyzed [8]. $R_0$ Value can be determined through approach of Next Generation Matrix method [4], [13].

$R_0 = FV^{-1}$
Where

$$F = \begin{pmatrix} \beta + \mu \\ 0 \\ 0 \end{pmatrix}$$

and

$$V = \begin{pmatrix} (\alpha + \mu) E \\ \alpha E - (\gamma + \mu) I \\ \gamma I - (\sigma + \mu) T \end{pmatrix}$$

The Jacobian of $F$ and $V$ are dual matrices $F$ and $V$ which determined at an disinfection state $E = 0$, $I = 0$ and $T = 0$, we have

$$F = \begin{pmatrix} 0 & \beta & \mu \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{pmatrix}$$

and

$$V = \begin{pmatrix} (\alpha + \mu) & 0 & \\ \alpha & (\gamma + \mu) & 0 \\ 0 & -\gamma & (\sigma + \mu) \end{pmatrix}$$

$FV^{-1}$ is $\frac{\beta\alpha}{(\alpha+\mu)(\gamma+\mu)} + \frac{\alpha\sigma\mu}{(\alpha+\mu)(\gamma+\mu)(\sigma+\mu)}$
Hence $R_0 = \frac{\beta\alpha}{(\alpha+\mu)(\gamma+\mu)} + \frac{\alpha\sigma\mu}{(\alpha+\mu)(\gamma+\mu)(\sigma+\mu)}$

### 3.3 Local Stability of Disease Free Equilibrium

**Theorem 2.** *For $R_0 < 1$, the Disease-Free Equilibrium point $E_0 = (\frac{\wedge}{\mu}, 0, 0, 0, 0)$ was locally asymptotically stable and for $R_0 > 1$, it was unstable [17].*

**Proof 2.** *The Jacobian matrix corresponding to the structure 1 at disease free equilibrium $E_0$ is*

$$J(E_0) = \begin{pmatrix} -\mu & 0 & -\beta & 0 & 0 \\ 0 & -(\mu + \alpha) & \beta & 0 & 0 \\ 0 & \alpha & -(\gamma + \mu) & 0 & 0 \\ 0 & 0 & \gamma & -(\sigma + \mu) & 0 \\ 0 & 0 & 0 & \sigma & -\mu \end{pmatrix}$$

*The characteristic equation is*

$$(\lambda + \mu)^2 (\lambda + (\sigma + \mu)) \left(\lambda^2 + a_1\lambda + a_2\right) = 0$$

*Where $a_1 = 2\mu + \alpha + \gamma$ and $a_2 = (\mu + \alpha)(\gamma + \mu) - \alpha\beta$.*
*There are 5 Eigen values for the Jacobian matrix $J(E_0)$ of which first three are -$\mu$, -$\mu$, -$(\sigma + \mu)$, and the remaining two Eigen values are roots of quadratic equation $(\lambda_2 + a_1\lambda + a_2) = 0$, which are negative.*
*Through Routh-Hurwitz criterion [20], all the roots of charateristics equation have destructive real part which revenues steady equilibrium if $a_1 > 0$ and $a_2 > 0$.*
*Since $\mu > 0$, $\alpha > 0$ and $\gamma > 0$ , we have $2\mu + \alpha + \gamma > 0$ that is $a_1 > 0$.*
*Since $(\mu + \alpha)(\gamma + \mu) - \alpha\beta > 0 > 0$ that is $a_2 > 0$.*
*If $R_0 < 1$, then*

$$\frac{\beta\alpha}{(\alpha + \mu)(\gamma + \mu)} + \frac{\alpha\sigma\mu}{(\alpha + \mu)(\gamma + \mu)(\sigma + \mu)(\alpha + \mu)} < 1$$

$$\Rightarrow \frac{\beta\alpha}{(\alpha+\mu)} < \frac{\beta\alpha}{(\alpha+\mu)(\gamma+\mu)} + \frac{\alpha\sigma\mu}{(\alpha+\mu)(\gamma+\mu)(\sigma+\mu)(\alpha+\mu)} < 1$$

$$\Rightarrow \frac{\beta\alpha}{(\alpha+\mu)(\gamma+\mu)} < 1 \Rightarrow \beta\alpha < (\alpha+\mu)(\gamma+\mu)$$

$$\Rightarrow (\mu+\alpha)(\gamma+\mu) - \alpha\beta > 0 that is a_2 > 0.$$

*Therefore, $a_2 > 0$ if $R_0 < 1$*
*Hence by Routh–Hurwitz Criteria, the disease free equilibrium point $E_0$ is locally asymptotically stable if $R_0 < 1$.*

### 3.4 Global Stability of Disease Free Equilibrium

**Theorem 3.** *The disease-free equilibrium point $E_0 = (\frac{\wedge}{\mu}, 0, 0, 0, 0)$ of structure 1 was globally asymptotic stable if $R_0 < 1$ [19].*

**Proof 3.** *It can be detected that from the structure (1), the disease-free sections are S, R and the infected sections are E, I, T. The system of equations (1) will be arranged as*

$$\frac{dU}{dt} = P(U,V), \frac{dV}{dt} = G(U,V), \text{ and } G(U,0) = 0 \tag{2}$$

*where $U = (S,R) \in R^2_+$, $V = (A,I,Q,J) \in R^3_+$.*
*By using the technique introduced by Castillo-Chavez [5], we derived global stability of the disease-free equilibrium point $E_0$ = ($\frac{\wedge}{\mu}$,0,0,0,0). For the worldwide asymptotic stability of $E_0$ the succeeding two conditions should be satisfied.*
*1. $\frac{dU}{dt} = P(U,0)$ Where $X^*$ is world wide asymptotically steady.*
*2. $G(U,V) = KV$ - $\hat{G}(U,V)$, $\hat{G}(U,V) \geq 0$, where $K = D_V G(U^*,0)$ is the Metzler Matrix and $(X,Y) \in \omega$.*
*If the given system of equations 1 satisfies 2 then the equilibrium point $E_0$ is a global asymptotically stable for $R_0 < 1$.*
*Hence, the system 1 can be rewritten as*

$$P(U,0) = \begin{pmatrix} \wedge - \mu S \\ 0 \end{pmatrix}, K = \begin{pmatrix} (\alpha+\mu) & 0 & 0 \\ \alpha & (\gamma+\mu) & 0 \\ 0 & \gamma & (\sigma+\mu) \end{pmatrix} and$$

$$\hat{G}(U,V) = \begin{pmatrix} \beta I (S_0 - S) \\ 0 \\ 0 \end{pmatrix}$$

*Since $S_0 > S$, by observation, $\hat{G}((U,V)) \geq 0$ $(U,V) \in \Omega$.*
*We can say that the matrix $K$ is $M$ matrix by the definition of $M$ and also we able to find that $X^* = (\frac{\wedge}{\mu}, 0)$ is globally asymptotic stable steady state of the limiting structure $\frac{dU}{dt} = P(U,0)$.*
*Since the two conditions are fulfilled, disease-free steady state $E_0 = (\frac{\wedge}{\mu}, 0, 0, 0, 0)$ of structure of equations 1 is globally asymptotic stable if $R_0 < 1$.*

### 3.5 Local Stability of Endemic Equilibrium point

We conclude the endemic steady state $X^* = (S^*E^*, I^*, T^*, R^*)$ with their possibility conditions are

$$\begin{aligned} S^* &= \frac{\wedge}{\beta I^* + \mu}, \\ E^* &= \frac{\beta S^* I^*}{(\alpha+\mu)}, \\ T^* &= \frac{\gamma I^*}{(\alpha+\mu)}, \\ R^* &= \frac{\sigma T^*}{\mu}, \\ I^* &= \frac{(\wedge\alpha\beta - \mu(\gamma+\mu))}{(\beta(\gamma+\mu(\sigma+\mu)))} = \frac{(\wedge(R_0-1) - \alpha\sigma\mu)}{(\beta(\gamma+\mu)(\sigma+\mu))} \end{aligned}$$

**Theorem 4.** *When $R_0 > 1$,then Endemic Equilibrium point $X^*$ is locally asymptotically steady and unstable if $R_0 < 1$.*

**Proof 4.** *The Jacobian matrix corresponding to the system 1 at endemic equilibrium point $X^*$ is*

$$J(X^*) = \begin{pmatrix} (-\beta I^* + \mu) & 0 & -\beta S^* & 0 & 0 \\ \beta I^* & -(\mu+\alpha) & \beta S^* & 0 & 0 \\ 0 & \alpha & -(\gamma+\mu) & 0 & 0 \\ 0 & 0 & \gamma & -(\sigma+\mu) & 0 \\ 0 & 0 & 0 & \sigma & -\mu \end{pmatrix}$$

*The characteristic equation is*

$$(\gamma+\mu)(\gamma+(\sigma+\mu))\left(\lambda^3 + b_1\lambda^2 + b_2\lambda + b_3\right) = 0$$

*Where* $b_1 = \beta I^* + 3\mu + \alpha + \gamma$,
$b_2 = (\alpha+\mu)(\gamma+\mu) - \alpha\beta S^* + (\gamma+\mu)(\beta I^* + \mu)$ *and*
$b_3 = (\beta I^* + \mu)((\alpha+\mu)(\gamma+\mu) - \alpha\beta S^*) - \beta^2 S^* I^*$
*Hence the first two Eigen values are* $-\mu$,$-(\sigma+\mu)$ *and remaining three Eigen values are the roots of the* $\left(\lambda^3 + b_1\lambda^2 + b_2\lambda + b_3\right) = 0$.
*Yet over again if the constants of specific equation* $a_1 > 0$, $a_2 > 0$, $a_3 > 0$ *and* $a_1 a_2 > a_3$ *are true, formerly by Routh-Hurwitz criterion, altogether the roots of the specific equation have negative real portions and hence a stable equilibrium. Therefore Endemic equilibrium at* $X^*$ *is locally asymptotically stable if* $R_0 > 1$

# 4 Numerical Simulation

Numerical simulation was performed in order to establish analytical result. We assumed some parameter values and initial conditions of proposed $SEITR$ model and it can be shown table 2.

## 4.1 Analysis of results

The basic reproduction number for this set of limitation is $R_0 = 2.806$. The dynamical performance of the system will be observed in 2 with the help of MATLAB programming.
From Fig. 2, we observed that the dynamics behavior of susceptible, exposed, Infected, treatment and recovered classes. This graph demonstrated that when the treatment rate rose, the infected population decreased and joined either the treatment population or the recovered population.

## 4.2 Discussion of results

From Fig.3 it was observed that the infected population(Fig.3b), exposed population(Fig.3c) and treatment population(Fig.3d) were decreased while the susceptible population(Fig.3a) was increased whenever the treatment rate increases.

Table 2: Influenza parameters values of the SEITR model

| Parameter | Values | Source |
|---|---|---|
| $\beta$ | 1.2 | [16],[30] |
| $\alpha$ | 0.2 | [9],[30] |
| $\gamma$ | 0.4 | [16],[30] |
| $\sigma$ | 0.1 | [16],[30] |
| $\mu$ | 0.01 | [26],[29] |
| $S(0)$ | 1 | Assumed |
| $E(0)$ | 0.2 | Assumed |
| $I(0)$ | 0.01 | Assumed |
| $T(0)$ | 0.4 | Assumed |
| $R(0)$ | 0.3 | Assumed |

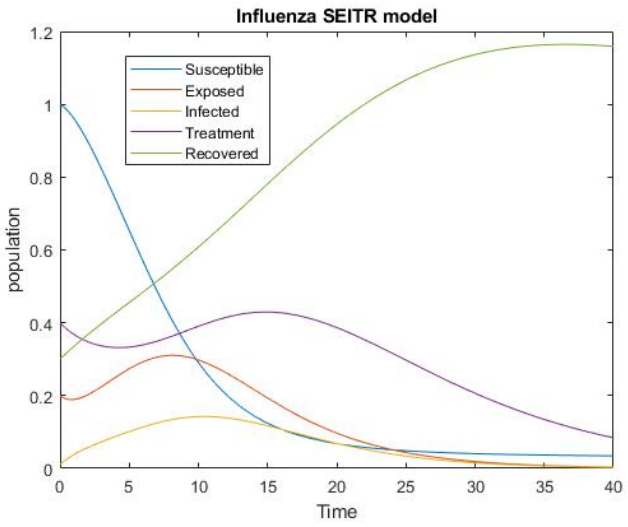


Figure 2: Dynamic behavior various compartments of SEITR model

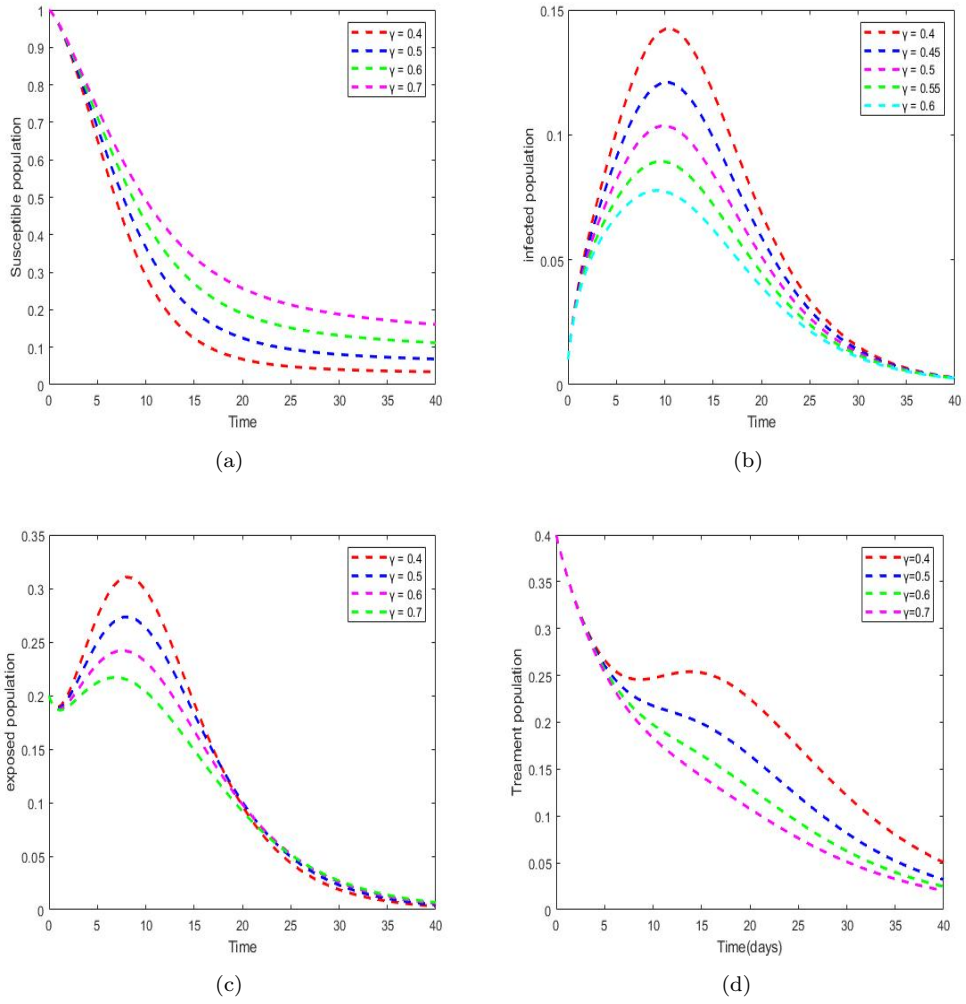


Figure 3: Effect of treatment rate $\gamma$ on susceptible, exposed, Infected and treatment population

# 5 CONCLUSION

The epidemiological models are enabled us a noble knowledge to understanding the spread dynamics of infectious disease in better way. In this article, a five compartment epidemiological model SEITR was proposed and the basic properties were discussed. The basic reproduction number $R_0$ value was determined. The positivity and uniform boundedness were performed. The existence of disease free equilibrium point $E_0$ was discussed and showed that it is locally also globally asymptotically stable for $R_0 < 1$ . Similarly the endemic equilibrium point $X^*$ be real and local asymptotically stable for $R_0 > 1$. The transmission dynamics of influenza has been observed. The result of treatment rate on the susceptible, exposed, infected and treatment populaces has been examined and it has a positive effect on the infected population. The reproduction number $R_0 = 2.806 > 1$ indicates that the outbreak has gotten out of hand and that there are currently more sick people than ever before. Therefore, the only method to reduce the rate of illness spread is to enhance the rate of treatment, which includes the quick hospitalisation of infections that are dangerously ill. The outcome of the SEITR model on the disease program mechanism can be investigated in next studies. Additionally, future research can be done to ascertain the most effective management strategies for the sickness spread model and the belongings of medications and immunizations on the SEITR model.

# References

[1] Anderson. R. M. and May R. M., Infectious Diseases of Humans: Dynamics and Control, Oxford University Press, Oxford, 1991.

[2] Andreasen. V, Lin. J and Levin. S.A, The dynamics of co circulating influenza strains conferring partial cross-immunity. J. Math. Biol., 35, 825-842, 1997.

[3] Attiq ul Rehman, Ram singh, Jagdev Singh, Mathematical analysis of multi-compartmental malaria transmission model with reinfection Chaos, Solitons and Fractals, 163, 2022.

[4] Baojun S. Compute using next generation operator. mtbi, Department of Mathematical Sciences Montclair State University, 12-14, 2013.

[5] Casagrandi. R, Bolzoni. L, Levin. S. A and Andreasen. V, The sirc model and influenza . Math. Biosci. (200) 152-169, 2006.

[6] Chinviriyasit. W, Numerical modelling of the transmission dynamics of influenza, ORSC & APORC, 52-59, 2007.

[7] Coburn. B. J, Wagner B. G. and Blower S., Modeling influenza epidemics and pandemics: insights into the future of swine flu (H1N1), BMC Med, 7(30), 2009.

[8] Dickmann O. and Heeserbeek J. A. P., Mathematical Epidemiology of Infectious Disease: Model Building Analysis and Interpretation, John Wiley, New York, 2000.

[9] Douglas. R, Influenza in man, in: E. Kilbourne (Ed.), The Influenza Viruses and Influenza, Academic Press, New York, USA, 395, 1975.

[10] Earn. D.J.D., Dushoff J. and Levin S.A, Ecology and evolution of the flu, Trends Ecol. Evol., (17) 334-340, 2002.

[11] Eccles. R., Understanding the symptoms of the common cold and influenza, The lancet infectious diseases Vol.5, No.11, 718-25, November 2005.

[12] Gu. Y., Komiya N., Kamiya H., Yasui Y., Taniguchi K., Okabe N., Pandemic A(H1N1) transmission during pre-symptomatic phase, Japan, Emerging infectious diseases, 17(9), September 2011.

[13] Heffernan J.M., Smith R.J. and Wahl L.M, Perspectives on the basic reproductive ratio, Journal of the Royal Society Interface, 2: 281-298, 2005.

[14] Herbert. W. Hethcote, Three basic epidemiological models, Springer - Verlag, NY, 1989.

[15] Hethcote. H.W, The mathematics of infectious diseases, siam rev., 42 599-653, 2000.

[16] Islam, R., Biswas, M. H. A., & Jamali, A. J. U. Mathematical Analysis of Epidemiological Model of Influenza a (H1N1) Virus Transmission Dynamics in Perspective of Bangladesh, GANIT: Journal of Bangladesh Mathematical Society, 37, 39-50, 2018. https://doi.org/10.3329/ganit.v37i0.35724.

[17] Jagdev Singh and Arpita Gupta, Computational analysis of fractional modified Degasperis - Procesi equation with Caputo-Katugampola derivative, AIMS Mathematics, 8(1):194-212, 2022.

[18] Jagdev Singh, Behzad Ganbari, Devendra Kumar, Dumitru Baleanu, Analysis of fractional model of guava for biological pest control with memory effect, Journal of Advanced Research, 32:99-108, 2021.

[19] Jagdev Singh, Devendra Kumar, Dumitru Baleanu, A new analysis of fractional fish farm model associated with Mittag-Leffler type kernel, International Journal of Biomathematics 13(2), 2020.

[20] Jongeun Choi, Lecture on Routh-Hurwitz Stability Criterion, Michigan State University.

[21] Kermack. W. O .and McKendrick A. G, A contribution to the mathematical theory of epidemics, Proc. Roy SocLond, 1927.

[22] Mishra O P, Mishra D K, Spread and control of influenza in two groups: a model, Applied Mathematics and Computation, April,219 (15):7982-7996, 2013.

[23] Murray. E., Alexander M.E., Seyed M., Moghadas S.M., Gergely R. and Jianhong W, A delay differential model for pandemic influenza with antiviral treatment, bull."Math. Biol. 70, 382-396, 2008.

[24] Quirouette C, Younis NP, Reddy MB, Beauchemin CAA (2020) Correction: A mathematical model describing the localization and spread of influenza A virus infection within the human respiratory tract. PLOS Computational Biology 16(11): e1008424. https://doi.org/10.1371/journal.pcbi.1008424

[25] Samuel Abubakar, Bifurcation analysis on the mathematical model of measles disease dynamics, UJAM, 212-216, 2013.

[26] Sanchez Y.G., Sabir Z., Guirao J.L.G. Design of a nonlinear SITR fractal model based on the dynamics of a novel coronavirus (covid -19) Fractals,28(8), 2020.

[27] Srivastav A K, Ghosh M, Analysis of a simple influenza (H1N1) model with optimal control, World Journal of Modelling and Simulation 12(4):307-319, 2016.

[28] Supriya yadav, Devendra Kumar, Jagdev Singh, Dumitru Baleanu, Analysis and Dynamics of Fractional Order Covid-19 Model with Memory Effect, Results in Physics, 24, 2021.

[29] Umar. M, Z. Sabir, M.A.Z. Raja, M. Shoaib, M. Gupta, Y.G. Sánchez, A stochastic intelligent computing with neuro-evolution heuristics for nonlinear SITR system of novel Covid-19, dynamics, Symmetry, 12(10),2020.

[30] Vinod Kumar Bais, and Deepak Kumar, SITR Dynamical Model for Influenza, International Journal of Engineering Technology Science and Research IJETSR,2,2015.

[31] ZhilanFeng, Final and peak epidemic sizes for SEIR models with quarantine and isolation, Mathematical Biosciences and Engineering, 4(4), 2007.