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The Mathematical Model on the Effect of Saturated Term on the Seir Epidemical Model

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Abstract
This study presents an enhanced SEITR (Susceptible-Exposed-Infectious-Treatment-Recovered) model that builds upon traditional epidemiological frameworks to address complex disease transmission patterns, particularly for infections with significant incubation periods and treatment dynamics. Unlike classic models such as the SIR model, the SEITR model introduces additional compartments to represent the Exposed (E) and Treatment (T) stages, enabling a more comprehensive representation of disease progression. By incorporating saturated incidence rates, the model accounts for behavioral responses and healthcare capacity constraints, providing a realistic depiction of disease spread and intervention effects.Through rigorous mathematical analysis, we confirm the existence, uniqueness, positivity, and boundedness of the model's solutions, ensuring both mathematical and biological feasibility. We also determine the basic reproduction number and examine the local stability of the disease-free equilibrium, providing insights into the conditions necessary for disease eradication. Numerical simulations further illustrate the impact of saturation terms and treatment effects on the dynamics of each compartment, offering visual insights into the model's applicability to real-world epidemic scenarios. The SEITR model's ability to simulate various intervention strategies and predict outcomes under limited healthcare resources makes it a valuable tool for public health planning and epidemic control, emphasizing the importance of timely intervention and resource allocation. This study highlights the model's potential to inform effective decision-making for managing infectious diseases.

Keywords: *SEITR model, Disease transmission, Saturated incidence rate, Epidemic modeling, Basic reproduction number, Stability analysis.*

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I. Introduction

The SEITR (Susceptible-Exposed-Infectious-Treatment-Recovered) model builds upon traditional epidemiological frameworks to provide a more nuanced representation of disease transmission and intervention dynamics, particularly for diseases with complex incubation periods and significant treatment effects (Shokri, 2015). Classical models, such as the SIR model introduced by Kermack and McKendrick in 1927, are foundational for understanding epidemic patterns but lack the ability to capture incubation periods and the impact of treatments. The SEITR model addresses these limitations by introducing "Exposed" and "Treatment" compartments, which enhance its ability to model disease latency and intervention impacts on transmission (Ndanusa and Tafida, 2016). This refined structure enables a more accurate depiction of disease dynamics, making it particularly useful for diseases like COVID-19 and influenza where incubation and timely treatment play a critical role (Kamoh et al., 2019).

The SEITR model's flexibility makes it a powerful tool for public health decision-making. It aids in designing effective intervention strategies, such as quarantine and vaccination, by providing a detailed view of disease progression and intervention efficacy (Anggriani and Beay, 2022; Banan et al., 2022). The model's incorporation of saturation effects, both in transmission and treatment, helps address real-world constraints in resource allocation and capacity. This approach reflects the importance of timely intervention and scalable treatment in managing outbreaks (Kayode and Adegboro, 2018). Mathematical modeling continues to be essential in epidemic response, offering insights that guide disease management strategies and resource allocation. The SEITR model, by improving on traditional models with saturation and treatment adjustments, demonstrates significant advancements in disease modeling and control (Hilker et al., 2017; Nyerere et al., 2014).

By applying a system of ordinary differential equations (ODEs), the SEITR model tracks the rates of transition between these compartments, offering a comprehensive picture of how diseases spread and respond to interventions. This approach allows public health authorities to analyze intervention strategies—such as quarantine, vaccination, and treatment protocols effectively, thus enhancing resource allocation and outbreak response (Kayode and Adegboro, 2018). In addition, the model's incorporation of nonlinear transmission

dynamics, such as those resulting from crowding or behavior changes, makes it adaptable to real-world scenarios where disease spread is not strictly linear (Kolokolnikov and Iron, 2021). Analytical techniques like the Homotopy Perturbation Method (HPM) and Laplace Adomian Decomposition Method (LADM) further refine SEITR-based analyses by providing insights into parameter sensitivity and improving prediction accuracy in dynamic environments, which is essential for managing disease-induced mortality and outbreak control (Kolawole and Adeniyi, 2020, Kolawole et al., 2023).

The SEIR model constructed by Al-Sheikh, (2012) investigated the impact of saturation terms on an SEIR epidemic model, delineating the population into four distinct epidemiological categories: susceptible (S), exposed (E), infectious (I), and recovered (R).

II. The Model Formulation

This study intends to propose a mathematical model incorporating saturated terms into the SEIR epidemic model to enhance the efficiency and capacity of treatment in controlling the spread of disease. We refer to the modified model SEITR categorizing the population into susceptible (S), exposed (E), infectious (I), Treatment (T) and recovered (R) groups.

2.1 Variables and Parameters of the Model

Table 2.1: Variables of the modified Model

Variables	Definition
S(t)	The number of individuals susceptible to the disease at time t.
E(t)	The number of individuals exposed to the disease (infected but not yet infectious) at time t.
I(t)	The number of individuals infectious (and capable of spreading the disease) at time t.
R(t)	The number of individuals who have recovered from the disease and are assumed to have acquired immunity at time t.
T(t)	The number of individuals receiving treatment for the disease at time t.

Table 2.2: Parameters of the Model

Parameters	Definition
s_0	Initial susceptible population
e_0	Initial exposed population
i_0	Initial infected population
r_0	Initial recovered population
t_0	Initial treated population
A	Recruitment rate
ρ	Treatment time (The rate at which individuals in the infectious compartment move to the treatment)
β	The transmission rate or contact rate, representing the rate at which susceptible
κ	Rate of losing immunity
λ	Natural death rate
ω_1	Disease induced death for the infected individual
ω_2	Disease induced death for the treated compartment
ν	Treatment rate
τ	The rate at which individuals in the treatment compartment recover and move to the recovered compartment.
μ	The rate at which individuals in the recovered compartment lose immunity and become susceptible again, if immunity is temporary.
σ_1 and σ_2	Saturation term

2.2 Assumptions for the Model

The following assumptions help to simplify the mathematical formulation of the SEITR model and make it tractable for analysis and simulation.

i. That the recruitment rate is constant.
ii. The population is homogeneously mixed.
iii. The parameters of the compartment are constant.
iv. The individuals in each compartment are randomly mixed.
v. The time spent in each compartment follows an exponential distribution.
vi. The deaths of new individuals are negligible compared to the total population size.

vii. The susceptible population is constant over the short term.
viii. The individuals who recover from the infection gain permanent immunity and cannot be infected again.
ix. The treatment individuals who are infected and receiving treatment not recovered.
x. Individuals can move between compartments.
xi. The initial conditions for each compartment is used to start the simulation.

2.3 Description of the Model

The total population $N(t)$ is divided in five compartments depending on the epidemiological status of the individual in the population. The compartments are susceptible individual $S(t)$, exposed individual $E(t)$, infected individual $I(t)$, treated individual $T(t)$, recovered individual $R(t)$.

Susceptible individual loss at the $\frac{\beta SI}{1+\sigma_1 S+\sigma_2 I}$ for exposed class and by natural death at the rate of λS also, increase A and those who recovered μR.

Thus, $\frac{dS}{dt} = A - \frac{\beta SI}{1+\sigma_1 S+\sigma_2 I} + \mu R - \lambda S$.

Exposed individual loses at the rate κE for infectious class and by the natural death at the rate also increase by $\frac{\beta SI}{1+\sigma_1 S+\sigma_2 I}$

Thus $\frac{dE}{dt} = \frac{\beta SI}{1+\delta_1 S+\delta_2 I} - (\lambda+\kappa)E$.

Infectious individual loses at the ρI for the treatment class by natural death at the ratio $(\lambda+\omega_2)I$ also increase by κE

Thus $\frac{dI}{dt} = \kappa E - (\rho+\lambda+\omega_2)I$.

Treatment individual loses at the τT for recovery class by natural death at the $(\lambda+\omega_2)I$ and also increase by ρI

Thus $\frac{dT}{dt} = \rho I - (\tau+\lambda+\omega_2)T$

Recovered individual loses at the μR for susceptible class by natural death at the λR also increase by τT .

Thus $\frac{dR}{dt} = \tau T - (\mu+\lambda)R$

2.4 Schematic Diagram of the Modified Model

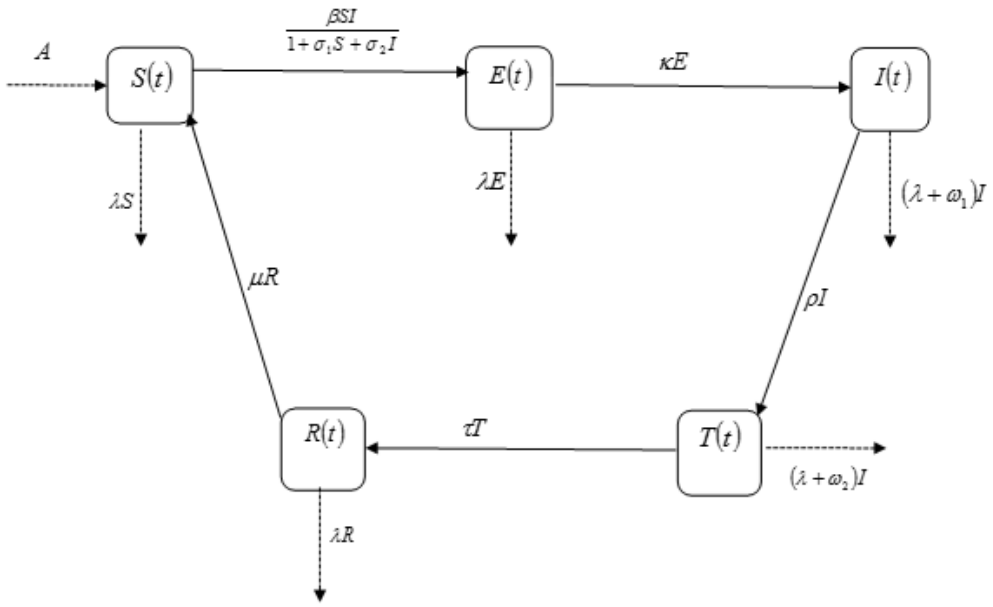

Figure 2.1: Schematic Diagram of Modified Model

2.5 Model Equation

The modified model equation is derived from the assumptions and flow diagram depicted in Figure 3.2, and it is expressed as equation (3.1).

$$\left.\begin{aligned}
\frac{dS}{dt} &= A - \frac{\beta SI}{1+\sigma_1 S+\sigma_2 I} + \mu R - \lambda S \\
\frac{dE}{dt} &= \frac{\beta SI}{1+\delta_1 S+\delta_2 I} - (\lambda+\kappa)E \\
\frac{dI}{dt} &= \kappa E - (\rho+\lambda+\omega_2)I \\
\frac{dT}{dt} &= \rho I - (\tau+\lambda+\omega_2)T \\
\frac{dR}{dt} &= \tau T - (\mu+\lambda)R
\end{aligned}\right\} \tag{2.1}$$

III. Model Analysis

In this section, we will analyzed the basic properties of the modified model. These properties are equilibrium point of the model (Disease free and endemic equilibrium), Basic reproductive number by next generation matrix, local stability of disease free equilibrium point etc.

3.1 Disease Free Equilibrium (DEF) of the Model

The disease-free equilibrium (DFE) of the SEITR (Susceptible-Exposed-Infectious-Treatment-Recovered) model represents a state where there are no infectious individuals in the population, and the disease is not actively spreading,mathematically.

Theorem 3.1

According to Andest *et al.,* (2023), a disease-free equilibrium state of the model (2.1) exist at the point. When there exist no disease or any intervention.

Thus, the Disease-Free Equilibrium (DFE) point for the SEITR model is:

$$\left(S^*, E^*, I^*, T^*, R^*, \right) = \left[\frac{A}{\lambda}, 0, 0, 0, 0\right] \tag{3.1}$$

Referring to theorem 3.1, the disease-free equilibrium (DFE) point of SEITR model was proved as follows:

Proof

The disease free equilibrium are steady state points are steady state solutions of that depict the absence of the disease in the population. This implies that at the SEITR free equilibrium point by setting the right hand side of equation (2.1) to zero. i.e. $\frac{dS}{dt}=\frac{dE}{dt}=\frac{dI}{dt}=\frac{dT}{dt}=\frac{dR}{dt}=0$.

To find the disease-free equilibrium (DFE) of the SEITR model given by the system of differential equations, we set the infectious components *E, I,* and *T* to zero and solve for the remaining variables *S* and *R*. using the system (2.1) for the disease-free equilibrium, we set $E=0, I=0, T=0$ (Andest *et al.,* 2023).

The equations simplify as follows:

$\frac{dS}{dt}=A+\mu R-\lambda S=0$

In the absence of the disease and intervention, we have

$A-\lambda S=0$

$\Rightarrow A=\lambda S$

Solving for S

$S=\frac{A}{\lambda}$

Thus, the disease-free equilibrium (DFE) point for this SEITR model is:

$\left(S^*, E^*, I^*, T^*, R^*, \right)=\left(\frac{A}{\lambda}, 0, 0, 0, 0\right)$.

3.2 Existence and Uniqueness

For the purpose of showing the uniqueness of model solution, the following representations were made:

Let $y_1(t)=S(t), y_2(t)=E(t), y_3(t)=I(t), y_4(t)=T(t), y_5(t)=R(t)$, so that the model equation given by equation (2.1) can be re-written in a complex form as

$$\frac{dy}{dt}=f\left(t, y_1, y_2, y_3, y_4, y_5\right), y_1(t_0)=y_{10}, y_2(t_0)=y_{20}, y_3(t_0)=y_{30}, y_4(t_0)=y_{40}, y_5(t_0)=y_{50} \quad (3.2)$$

Theorem 3.1

Suppose that the function $f\left(t, y_1, y_2, y_3, y_4, y_5\right)$ in the model equation given by system (3.2) satisfies Lipchitz condition in the region $D=\left\{(t, y): 0\le|t-t_0|\le\|y-y_0\|\le b\right\}$ for some $a>0, b>0, a, b\in D$, then, there exist a natural constant number $\delta_1>0$, such that a unique continuous vector solution $y(t)$ of the model equation given by equation (3.1) exists in the interval $|t-t_0|<\delta_1$.

Lemma 3.1: if $f(t, y)$ has continuous partial derivative $\frac{\partial f_i}{\partial y_i}$ for $i=1, 2, \cdots, n$ on a bounded convex domain R, then it satisfies a Lipchitz condition in $R\ \|f(t, y)-f(t, y_{n-1})\|\le k\|y_n-y_{n-1}\|, i=1, 2, 3, \cdots$

According to Lemma 3.1, for the functions given by the equation (2.1) to satisfy Lipchitz condition.

Proof

From the model equation given by system (2.2) let

$$\left.\begin{aligned}
f_1(t, y_1)&=\frac{dS}{dt}=A-\frac{\beta SI}{1+\sigma_1 S+\sigma_2 I}+\mu R-\lambda S\\
f_2(t, y_2)&=\frac{dE}{dt}=\frac{\beta SI}{1+\sigma_1 S+\sigma_2 I}-(\lambda+\kappa)E\\
f_3(t, y_3)&=\frac{dI}{dt}=\kappa E-(\rho+\lambda+\omega_2)I\\
f_4(t, y_4)&=\frac{dT}{dt}=\rho I-(\tau+\lambda+\omega_2)T\\
f_5(t, y_5)&=\frac{dR}{dt}=\tau T-(\mu+\lambda)R
\end{aligned}\right\} \quad (3.3)$$

To show that $\frac{\partial f_i}{\partial y_j}, i, j=1, 2, 3, \cdots, 5$ are continuous and bounded in the region *D*. We consider the partial derivatives of equation (3.3) are:

$$\frac{dS}{dt} = f_1(t, y_1) = A - \frac{\beta SI}{1+\sigma_1 S + \sigma_2 I} + \mu R - \lambda S$$

$$\left|\frac{\partial f_1}{\partial S}\right| = -\frac{\beta I}{\sigma_1} - \lambda < \infty, \left|\frac{\partial f_1}{\partial E}\right| = 0 < \infty, \left|\frac{\partial f_1}{\partial I}\right| = -\frac{\beta S}{\sigma_2} < \infty, \left|\frac{\partial f_1}{\partial T}\right| = 0 < \infty, \left|\frac{\partial f_1}{\partial R}\right| = \mu < \infty \quad (3.4)$$

Also, taking the partial derivatives of the second component of equation (3.3) we obtained

$$\frac{dE}{dt} = f_2(t, y_2) = \frac{\beta SI}{1+\delta_1 S + \delta_2 I} - (\lambda + \kappa)E$$

$$\left|\frac{\partial f_2}{\partial S}\right| = \frac{\beta I}{\sigma_2} - (\lambda + k) < \infty, \left|\frac{\partial f_2}{\partial E}\right| = -(\lambda + \kappa) < \infty, \left|\frac{\partial f_2}{\partial I}\right| = \frac{\beta S}{\sigma_2} < \infty, \left|\frac{\partial f_2}{\partial T}\right| = 0 < \infty, \left|\frac{\partial f_2}{\partial R}\right| = 0 < \infty \quad (3.5)$$

Consider the partial derivatives of the third component of equation (3.3) we get

$$\frac{dI}{dt} = f_3(t, y_3) = \kappa E - (\rho + \lambda + \omega_2)I$$

$$\left|\frac{\partial f_3}{\partial S}\right| = 0 < \infty, \left|\frac{\partial f_3}{\partial E}\right| = \kappa < \infty, \left|\frac{\partial f_3}{\partial I}\right| = -(\rho + \lambda + \omega_2) < \infty, \left|\frac{\partial f_3}{\partial T}\right| = 0 < \infty, \left|\frac{\partial f_3}{\partial R}\right| = 0 < \infty \quad (3.6)$$

The partial derivatives of the fourth component of equation (3.3) are as follows

$$\frac{dT}{dt} = f_4(t, y_4) = \rho I - (\tau + \lambda + \omega_2)T$$

$$\left|\frac{\partial f_4}{\partial S}\right| = 0 < \infty, \left|\frac{\partial f_4}{\partial E}\right| = 0 < \infty, \left|\frac{\partial f_4}{\partial I}\right| = \rho < \infty, \left|\frac{\partial f_4}{\partial T}\right| = \left|-(\tau + \lambda + \omega_2)\right| = (\tau + \lambda + \omega_2) < \infty, \left|\frac{\partial f_4}{\partial R}\right| = 0 < \infty \quad (3.7)$$

The partial derivatives of the fifth component of equation (3.3), we obtained

$$\frac{dR}{dt} = f_5(t, y_5) = \tau T - (\mu + \lambda)R$$

$$\left|\frac{\partial f_5}{\partial S}\right| = 0 < \infty, \left|\frac{\partial f_5}{\partial E}\right| = 0 < \infty, \left|\frac{\partial f_5}{\partial I}\right| = 0 < \infty, \left|\frac{\partial f_5}{\partial T}\right| = -(\mu - \tau + \lambda) < \infty, \left|\frac{\partial f_5}{\partial R}\right| = 0 < \infty \quad (3.8)$$

It can be observed from equations (3.4) to (3.8) that all the partial derivatives of the model equation are continues and bounded in the interval, $0 < D < \infty$ by the lemma 3.1. The functions given in by equation (2.1) satisfy Lipchitz condition and hence, there exists a unique solution of model equation (2.1) in the region *D*.

3.3 Positivity of Solution

Theorem 3.3

For a nonnegative initial conditions, the model equation given in equation (2.1) the solution will remain positive for all time (S, E, I, T, R) of the equation are non-negative for all time $t \geq 0$

Proof:

For the model equations (2.1), let t be the maximum time for the epidemics. This implies that $t = \sup\{t > 0 : (S > 0, E > 0, I > 0, T > 0, R > 0)\}$ that is $t^* \geq 0$. From the first equation of (2.1), we have

$$\frac{dS}{dt} = A - \frac{\beta SI}{1+\sigma_1 S + \sigma_2 I} + \mu R - \lambda S$$

$$\geq \frac{\beta SI}{\sigma_1 S} - \lambda S$$

$$\frac{dS}{dt} \geq \frac{\beta SI}{\sigma_1 S} - \lambda S$$

$$\frac{dS}{S} \geq -\lambda\, dt$$

$$Ln\, S \geq -\lambda\, dt \quad (3.9)$$

Taking the exponential of both side (3.9)

$$S(t) = e^{-\lambda t}$$

$$S(0) \geq S(0)e^{-\lambda t} \geq 0$$

Since $\lambda > 0$.

From the second equation of (2.1) we have

$$\frac{dE}{dt} = \frac{\beta SI}{1+\delta_1 S+\delta_2 I} - (\lambda+\kappa)E$$
$$\geq -(\lambda+\kappa)E$$
(3.10)

Integrating both side (4.10)

$$\int \frac{dE}{E} \geq -\int (\lambda+\kappa)dt$$
$$\ln E \geq -\int (\lambda+\kappa)dt$$
$$E(t) = e^{-\int(\mu_0+\psi)t}$$
$$E(0) \geq E(0)e^{-\int(\mu_0+\psi)dt} \geq 0$$

Since $(\lambda+\kappa)t > 0$.

Similarly, from the third equation of (2.1) we have

$$\frac{dI}{dt} = \kappa E - (\rho+\lambda+\omega_2)I$$
$$\geq -(\rho+\lambda+\omega_2)I$$
(3.11)

Integrating both side (3.11)

$$\int \frac{dI}{I} \geq -\int (\rho+\lambda+\omega_2)dt$$
$$\ln I \geq -\int (\rho+\lambda+\omega_2)dt$$
$$I(t) = e^{-\int(\rho+\lambda+\omega_2)dt}$$
$$I(0) \geq I(0)e^{-\int(\rho+\lambda+\omega_2)dt} \geq 0$$

Since $(\rho+\lambda+\omega_2)t > 0$.

From the fourth equation of (2.1) we have

$$\frac{dT}{dt} = \rho I - (\tau+\lambda+\omega_2)T$$
$$\geq -(\tau+\lambda+\omega_2)T$$
(3.12)

Integrating both side (3.12)

$$\int \frac{dT}{T} \geq -\int (\tau+\lambda+\omega_2)dt$$
$$\ln T \geq -\int (\tau+\lambda+\omega_2)dt$$
$$T(t) = e^{-\int(\tau+\lambda+\omega_2)t}$$
$$T(0) \geq T(0)e^{-\int(\tau+\lambda+\omega_2)dt} \geq 0$$

Since $(\tau+\lambda+\omega_2)t > 0$.

And from the fifth equation of (2.1) we have

$$\frac{dR}{dt} = \tau T - (\mu+\lambda)R$$
$$\geq -(\mu+\lambda)R$$
(3.13)

Integrating both side (3.13)

$$\int \frac{dR}{R} \geq -\int (-(\mu+\lambda))dt$$
$$\ln R \geq -\int (-(\mu+\lambda))dt$$
$$R(t) = e^{-\int(-(\mu+\lambda))t}$$
$$R(0) \geq R(0)e^{-\int(-(\mu+\lambda))dt} \geq 0$$

Since $(\mu+\lambda)>0$.

Theorem 3.3 establishes that for nonnegative initial conditions, the solution of the model equation given in equation (2.1) will remain positive for all time. The proof begins by considering the maximum time for the epidemic, denoted as t and analyzing each equation within the system.

3.4 Feasible Region (Invariant Region)

The invariant region in which the solution model equations given by system (2.1) will be bounded and make biological sense can be established in the following theorem:

Theorem 3.4

The solution of the model given by equation (2.1) is bounded in the closed set

$$\Omega=\left\{(S,E,I,T,R)\in\Re_+^5\le\frac{A}{\lambda}\right\} \tag{3.14}$$

Furthermore, the set Ω is positively invariant and attracting with respect to model equation given by system (2.1).

Proof

To find the feasible region (also known as the invariant region) for the SEITR model, we need to identify the region in which the total population remains bounded and positive over time.

The total population at any time *t* can be represented as:

$$N(t)=S(t)+E(t)+I(t)+T(t)+R(t) \tag{3.15}$$

Differentiating equation (3.15) with respect to time *t* we have

$$\frac{dN}{dt}=\frac{dS}{dt}+\frac{dE}{dt}+\frac{dI}{dt}+\frac{dT}{dt}+\frac{dR}{dt} \tag{3.16}$$

We sum all these equations to get the equation for the total population $N(t)$ using (3.16) we have

$$\begin{aligned}\frac{dN}{dt}&=\frac{dS}{dt}+\frac{dE}{dt}+\frac{dI}{dt}+\frac{dT}{dt}+\frac{dR}{dt}\\&=A-\frac{\beta SI}{1+\sigma_1 S+\sigma_2 I}+\mu R-\lambda S+\frac{\beta SI}{1+\delta_1 S+\delta_2 I}-(\lambda+\kappa)E+\kappa E-(\rho+\lambda+\omega_2)I\\&+\rho I-(\tau+\lambda+\omega_2)T+\tau T-(\mu+\lambda)R\end{aligned} \tag{3.17}$$

Simplifying (3.17), terms involving β,κ,ρ and τ cancel out, leaving us with:

$$\begin{aligned}\frac{dN}{dt}&=A-\lambda S-\lambda E-\lambda I-\lambda T-\lambda E=A-\lambda(S+E+I+T+R)\\&=A-\lambda N\end{aligned} \tag{3.18}$$

This is a linear differential equation in N :

$$\frac{dN}{dt}+\lambda N=A \tag{3.19}$$

To solve for equation (3.19), we use the integrating factor method. The integrating factor is $e^{\lambda t}$

$$e^{\lambda t}\frac{dN}{dt}+\lambda e^{\lambda t}N=Ae^{\lambda t} \tag{3.20}$$

Integrating both sides with respect to *t*:

$$\int\frac{d}{dt}\left(N\lambda e^{\lambda t}\right)dt=\int Ae^{\lambda t}dt$$

$$N\lambda e^{\lambda t}=\frac{A}{\lambda}e^{\lambda t}+C \tag{3.21}$$

Where C is the constant of integration. Solving for N :

$$N = \frac{A}{\lambda} + Ce^{\lambda t} \tag{3.22}$$

As $t \to \infty$, the exponential term $Ce^{\lambda t}$ approaches 0, leaving:

$$N(t) = \frac{A}{\lambda}.$$

Thus, the total population $N(t)$ is bounded by $\frac{A}{\lambda}$. This implies that the feasible region, or the invariant region, for the system is:

$$0 \le N = S + E + I + T + R \le \frac{A}{\lambda}.$$

Therefore, the invariant region for the SEITR model is:

$$\left\{(S, E, I, T, R) \in \Re_+^5 \middle| S + E + I + T + R \le \frac{A}{\lambda}\right\}.$$

This region ensures that the total population remains non-negative and bounded above by $\frac{A}{\lambda}$, guaranteeing the feasibility and sustainability of the population dynamics modeled by the SEITR equations.

3.5 Basic Reproduction Number (R_0)

The basic reproduction number is defined as the average number of the secondary cases infections generated by a typical infected person in an otherwise disease free population. The basic reproduction number (R_0) of the system (2.1) is computed the next generation matrix method. Here the next generation matrix F_i denotes the rate of appearance of new infections and V_i represents the transfer of infection into and out of any compartment respectively.

To find the basic reproduction number, (R_0), for the SEITR model, we use the next-generation matrix approach. This involves identifying the new infections and the transitions between compartments.

Using the SEITR model equations (2.1), we first focus on the infected compartments: E, I and T.

$$F_i = \begin{bmatrix} \frac{\beta SI}{(1+\sigma_1 S + I\sigma_2)^2} \\ \kappa E \\ \rho I \end{bmatrix}$$

$$\frac{\partial f_i(x_0)}{\partial x_j} = \begin{bmatrix} \frac{\partial f_1(x_0)}{\partial E} & \frac{\partial f_1(x_0)}{\partial I} & \frac{\partial f_1(x_0)}{\partial T} \\ \frac{\partial f_2(x_0)}{\partial E} & \frac{\partial f_2(x_0)}{\partial I} & \frac{\partial f_2(x_0)}{\partial T} \\ \frac{\partial f_3(x_0)}{\partial E} & \frac{\partial f_3(x_0)}{\partial I} & \frac{\partial f_3(x_0)}{\partial T} \end{bmatrix} = \begin{bmatrix} 0 & \frac{\beta\Lambda}{\sigma_2\lambda} & 0 \\ \kappa & 0 & 0 \\ 0 & \rho & 0 \end{bmatrix}$$

$$V = \begin{bmatrix} (\lambda + K)E \\ -(\rho + \lambda + \omega_2)I \\ -(\tau + \lambda + \omega_2)E \end{bmatrix}$$

$$V_i = \frac{\partial v_i(x_0)}{\partial x_j} = \begin{bmatrix} \frac{\partial v_1(x_0)}{\partial E} & \frac{\partial v_1(x_0)}{\partial I} & \frac{\partial v_1(x_0)}{\partial T} \\ \frac{\partial v_2(x_0)}{\partial E} & \frac{\partial v_2(x_0)}{\partial I} & \frac{\partial v_2(x_0)}{\partial T} \\ \frac{\partial v_3(x_0)}{\partial E} & \frac{\partial v_3(x_0)}{\partial I} & \frac{\partial v_3(x_0)}{\partial T} \end{bmatrix} = \begin{bmatrix} -(\lambda + K) & 0 & 0 \\ 0 & -(\rho + \lambda + \omega_2) & 0 \\ -(\tau + \lambda + \omega_2) & 0 & 0 \end{bmatrix}$$

The next-generation matrix F (new infection terms) and V (transition terms) are:

$$F=\begin{bmatrix} 0 & \frac{\beta\Lambda}{\sigma_2\lambda} & 0 \\ \kappa & 0 & 0 \\ 0 & \rho & 0 \end{bmatrix}, V=\begin{bmatrix} -(\lambda+K) & 0 & 0 \\ 0 & -(\rho+\lambda+\omega_2) & 0 \\ -(\tau+\lambda+\omega_2) & 0 & 0 \end{bmatrix}$$

where $S=\frac{A}{\lambda}$ and $E=0$

We linearize the system around the disease-free equilibrium (DFE),

$$V^{-1}=\begin{bmatrix} -\frac{1}{(\lambda+K)} & 0 & 0 \\ 0 & -\frac{1}{(\rho+\lambda+\omega_2)} & 0 \\ -\frac{1}{(\tau+\lambda+\omega_2)} & 0 & 0 \end{bmatrix}$$

To find R_0, we compute the eigenvalues of the matrix FV^{-1}:

$$FV^{-1}=\begin{bmatrix} 0 & \frac{\beta\Lambda}{\sigma_2\lambda} & 0 \\ \kappa & 0 & 0 \\ 0 & \rho & 0 \end{bmatrix}\begin{bmatrix} -\frac{1}{(\lambda+K)} & 0 & 0 \\ 0 & -\frac{1}{(\rho+\lambda+\omega_2)} & 0 \\ -\frac{1}{(\tau+\lambda+\omega_2)} & 0 & 0 \end{bmatrix}=\begin{bmatrix} 0 & \frac{\beta\Lambda}{-(\lambda+K)\sigma_2\lambda} & 0 \\ \frac{\kappa}{-(\tau+\lambda+\omega_2)} & 0 & 0 \\ 0 & \frac{\beta\Lambda}{-(\rho+\lambda+\omega_2)\sigma_2\lambda} & 0 \end{bmatrix}$$

$$\left|FV^{-1}-zI\right|=0$$

$$\begin{bmatrix} -z & \frac{\beta\Lambda}{-(\lambda+K)\sigma_2\lambda} & 0 \\ \frac{\kappa}{-(\tau+\lambda+\omega_2)} & -z & 0 \\ 0 & \frac{\beta\Lambda}{-(\rho+\lambda+\omega_2)\sigma_2\lambda} & -z \end{bmatrix}=0$$

The characteristics polynomial is given by

$$\frac{z\left(z^2\alpha_2\lambda^2 p+z^2\alpha_2\lambda p\kappa+z^2\alpha_2\lambda^3+z^2\alpha_2\lambda^2 k+z^2\alpha_2\lambda^2\omega_2+z^2\alpha_2\lambda\omega_2 k-k\beta\Lambda\right)}{(\rho+\lambda+\omega_2)(\lambda+\kappa)\sigma_2\lambda}=0$$

Simplifying the characteristics polynomial we obtain

The eigenvalues of the characteristics polynomial are as follows:

$$z_1=0,\ z_2=\frac{\sqrt{\alpha_2\lambda Ak\beta\Lambda}}{\alpha_2\lambda A},\ z_3=-\frac{\sqrt{\alpha_2\lambda Ak\beta\Lambda}}{\alpha_2\lambda A}$$

Where $A=\rho\lambda+\rho\kappa+\lambda^2+\lambda\kappa+\omega_2\lambda+\omega_2 k$

The largest or dominant eigenvalues is z_2 therefore, the basic reproduction number R_0

$$R_0=\frac{\sqrt{\alpha_2\lambda Ak\beta\Lambda}}{\alpha_2\lambda A}.$$

Thus, the basic reproduction number R_0 for the SEITR model is:

$$R_0=\frac{\sqrt{\alpha_2\lambda Ak\beta\Lambda}}{\alpha_2\lambda A}$$

From here it is clear that the reproduction number with treatment is greater than that without treatment. Therefore, treatment plays a vital role in control of disease.

3.6 Local Stability of the DFE$(\mathbf{x_0})$

Theorem: 3.5

The disease free equilibrium point of the model system (2.1) is

i. Locally asymptotically stable if $R_0 < 1$

ii. Unstable if $R_0 > 1$

If and only if Jacobian $J(x_0)$ has a negative trace and positive determinant (Andest et al. 2023).

Proof

Here we take the partial differentiation of (2.1) with respect to *S, E, I, T, R* at the disease-free equilibrium which gives us:

Thus, we compute the Jacobian matrix *J* of the system at the DFE. The Jacobian matrix *J* is the matrix of first-order partial derivatives of the right-hand side of the system with respect to the variables in equation (2.1).

$$|J(x_0)| = \begin{bmatrix} \frac{\partial f_1}{dS} & \frac{\partial f_1}{dE} & \frac{\partial f_1}{dI} & \frac{\partial f_1}{dT} & \frac{\partial f_1}{dR} \\ \frac{\partial f_2}{dS} & \frac{\partial f_2}{dE} & \frac{\partial f_2}{dI} & \frac{\partial f_2}{dT} & \frac{\partial f_2}{dR} \\ \frac{\partial f_3}{dS} & \frac{\partial f_3}{dE} & \frac{\partial f_3}{dI} & \frac{\partial f_1}{dT} & \frac{\partial f_3}{dR} \\ \frac{\partial f_4}{dS} & \frac{\partial f_4}{dE} & \frac{\partial f_4}{dI} & \frac{\partial f_4}{dT} & \frac{\partial f_4}{dR} \\ \frac{\partial f_5}{dS} & \frac{\partial f_5}{dE} & \frac{\partial f_5}{dI} & \frac{\partial f_5}{dT} & \frac{\partial f_5}{dR} \end{bmatrix}$$

where f_1, f_2, f_3, f_4, f_5 are the right-hand sides of the differential equations (2.1).

Evaluating the partial derivatives at the DFE $(S, E, I, T, R,) = \left(\frac{A}{\lambda}, 0, 0, 0, 0\right)$

$$f_1 = A - \frac{\beta SI}{1 + \sigma_1 S + \sigma_2 I} + \mu R - \lambda S$$

$$f_2 = \frac{\beta SI}{1 + \delta_1 S + \delta_2 I} - (\lambda + \kappa)E$$

$$f_3 = \kappa E - (\rho + \lambda + \omega_2)I$$

$$f_4 = \rho I - (\tau + \lambda + \omega_2)T$$

$$f_5 = \tau T - (\mu + \lambda)R$$

Evaluating at the DFE $(S, E, I, T, R,) = \left(\frac{A}{\lambda}, 0, 0, 0, 0\right)$

$$\frac{df_1}{dS} = 0, \frac{df_2}{dE} = -(\lambda + \kappa), \frac{df_2}{dI} = \frac{df_2}{dT} = \frac{df_2}{dR} = 0$$

$$\frac{df_3}{dS} = 0, \frac{df_3}{dE} = k, \frac{df_3}{dI} = -(\rho + \lambda + \omega), \frac{df_3}{dT} = 0, \frac{df_3}{dR} = 0$$

$$\frac{df_4}{dS} = 0, \frac{df_4}{dE} = 0, \frac{df_4}{dI} = \rho, \frac{df_4}{dT} = -(\tau + \lambda + \omega_2), \frac{df_4}{dR} = 0$$

$$\frac{df_5}{dS} = 0, \frac{df_5}{dE} = 0, \frac{df_5}{dI} = 0, \frac{df_5}{dT} = \tau, \frac{df_5}{dR} = -(\mu + \lambda)$$

The Jacobian matrix at the DFE is:

$$J = \begin{pmatrix} -\lambda & 0 & \frac{\beta \frac{A}{\lambda}}{1 + \sigma_1 \frac{A}{\lambda}} & 0 & \mu \\ 0 & -(\lambda + \kappa) & \frac{\beta \frac{A}{\lambda}}{1 + \delta_1 \frac{A}{\lambda}} & 0 & 0 \\ 0 & \kappa & -(\rho + \lambda + \omega_2) & 0 & 0 \\ 0 & 0 & \rho & -(\tau + \lambda + \omega_2) & 0 \\ 0 & 0 & 0 & \tau & -(\mu + \lambda) \end{pmatrix}$$

$$T_r J(x_0) = -\lambda - (\lambda + \kappa) - (\rho + \lambda + \omega_2) - (\tau + \lambda + \omega_2)$$
$$= -(\lambda + \kappa) < 0$$

This is the trace. While its determinant

$$\lambda(\tau + \lambda + \omega_2)(\mu + \lambda)\begin{pmatrix} \lambda^2\rho + \lambda\rho\sigma_1\beta A + \lambda^3 + \lambda^2\sigma_1\beta A + \lambda^2\omega_2 + \lambda\omega_2\sigma_1\beta A + \kappa\rho\lambda \\ + \kappa\rho\sigma_1\beta A + \kappa\lambda^2 + \kappa\lambda\sigma_1\beta A + \kappa\omega_2\lambda + \kappa\omega_2\sigma_1\beta A - \kappa\beta A \end{pmatrix}$$

3.8 Numerical Simulation

In this section, we give perform/illustration numerical experiment/simulation of our basic model using (MATLAB R2018a).

Table 3.1: Description of variables with their values

Variables	Value	Reference
S(t)	25	Kolawole et al. (2023)
E(t)	18	Kolawole et al. (2023)
I(t)	12	Kolawole et al. (2023)
R(t)	7	Kolawole et al. (2023)
T(t)	20	Biswwas *et al.* (2014)

Table 3.2: Description parameters with their values

Parameters	Value	Reference
s_0	0.25	Kolawole et al. (2023)
e_0	0.5	Biswwas *et al.* (2014)
i_0	0.08	Kolawole et al. (2023)
r_0	0.15	Kolawole et al. (2023)
t_0	0.1	Kolawole et al. (2023)
A	48	Kolawole et al. (2023)
ρ	0.05	Jinhong et. al. (2014)
β	0.3	Kolawole et al. (2023)
κ	1.25	Jinhong et. al. (2014)
λ	0.5	Kolawole et al. (2023)
ω_1	0.2	Jinhong et. al. (2014)
ω_2	0.4	Kolawole et al. (2023)
ν	0.8	Jinhong et. al. (2014)
τ	0.001	Biswwas *et al.* (2014)
μ	0.5	Kolawole et al. (2023)
σ_1 and σ_2	$0 \le \sigma_n < 1\, n = 1, 2$	Kolawole et al. (2023)

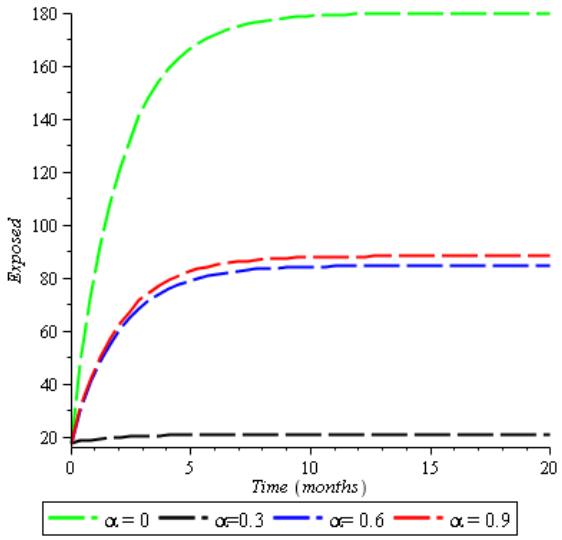

Figure 3.1: Effect of Saturation term on Susceptible class

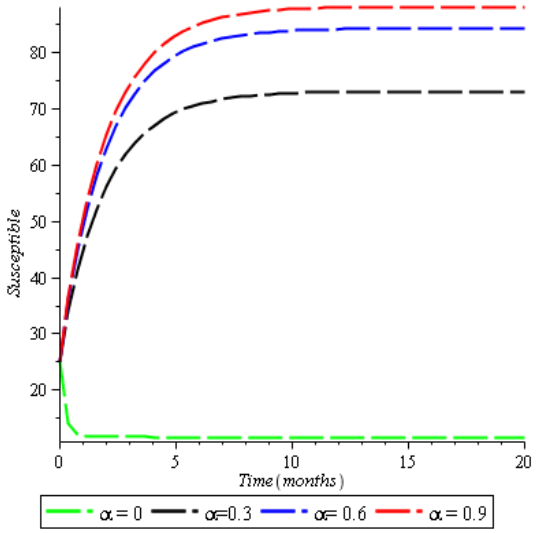

Figure 3.2: Effect of Saturation term on Infected class

IV. Discussion of Results

This study analyzes the SEITR model's core features, focusing on the existence and uniqueness of the solution, positivity, and local stability of the disease-free equilibrium. We first demonstrate that the model meets the conditions for a unique solution by reformulating the SEITR equations and applying the Lipschitz condition, ensuring a continuous and well-defined solution. This approach guarantees the solution's feasibility within a specific interval, affirming the model's theoretical soundness. Ensuring positivity is equally essential since it represents human populations, and we confirm that the model's solutions remain non-negative under nonnegative initial conditions, maintaining biological validity.

We establish the invariant or feasible region by analyzing total population dynamics through the SEITR equations. Summing these equations allows us to derive a bounded region, constrained by the population's birth and death rates, ensuring the population remains biologically meaningful over time. This feasible region acts as a natural limit for the model, ensuring realistic population changes within each compartment. Additionally, we identify the disease-free equilibrium (DFE), representing a condition where no individuals are infected. The DFE is critical as it outlines the state where the disease is eradicated, providing a baseline for studying stability.

The model's basic reproduction number, a pivotal threshold metric, quantifies the average number of secondary infections from a single infected individual in a fully susceptible population. We calculate this using the next-generation matrix method, which helps determine the potential for disease spread and guides control measures. The local stability of the DFE is analyzed by examining the eigenvalues of the Jacobian matrix at the DFE. If all eigenvalues have negative real parts, the DFE is stable, meaning minor disturbances will decay over time, keeping the disease under control. However, if any eigenvalue is positive, the DFE becomes unstable, indicating the disease could invade the population.

The numerical simulations of the basic model was carried out. The simulations demonstrate the impact of various parameters on the population dynamics. Figures 4.1 and 4.2, show the effect of the saturation terms on the susceptible, exposed, and infected classes, respectively. These visualizations help in understanding how different factors influence the spread and control of the disease, providing insights into potential intervention strategies.

The numerical simulations provide a comprehensive understanding of the SEITR model's behavior and validate our analytical findings. They offer a visual and quantitative representation of the model dynamics, making the theoretical results more tangible and applicable to real-world scenarios. Through these simulations, we can explore various scenarios and assess the effectiveness of different public health interventions in controlling the spread of the disease.

V. Summary and Conclusion

This study presents a modified SEIR model that incorporates saturated incidence rates to more accurately depict infectious disease transmission dynamics, building on Al-Sheikh's (2012) work by adding a treatment compartment to account for interactions between infected and recovered individuals. Through rigorous mathematical analysis, the study confirms the existence, uniqueness, positivity and boundedness of the model's solutions, ensuring both mathematical and biological validity. Key findings include the identification of

the disease-free equilibrium point and the calculation of the basic reproduction number, which provide crucial insights into disease eradication conditions. The local stability analysis shows that when the basic reproduction number is less than one, the disease-free equilibrium is stable, meaning the disease will die out over time. This model emphasizes the importance of saturation effects in enhancing predictive accuracy and informing effective disease control strategies.

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