Global Analysis of an SEIRS Model for COVID-19 Capturing Saturated Incidence With Treatment Response

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Abstract

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Global Analysis of an SEIRS Model for COVID-19 Capturing Saturated Incidence with Treatment Response

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Abstract

Sequel to [10], who studied the dynamics of COVID-19 using an SEIRUS model. We consider an SEIRS model capturing saturated incidence with treatment response. In this theoretical model, we assumed that the treatment response is proportional to the number of infected as long as the incidence cases are within the capacity of the healthcare system, after which the value becomes constant, when the number of confirmed cases exceed the carrying capacity of the available medical facilities. Thus, we obtain the reproduction number stating that when $R_0<1$, the disease free equilibrium is globally asymptotically stable. Also, we studied the existence of the local and global stability of the disease free and endemic equilibria and found that the kind of treatment response and inhibitory measures deployed in tackling the COVID-19 pandemic determines whether the disease will die out or become endemic.

Keywords: *COVID-19, SARS-CoV-2, Saturated Incidence, Treatment Function, Next Generation Matrix, Disease Free and Endemic States, Local and Global Stability, Routh-Hurwitz Criterion, Lozinski¡ measure, Lyapunov Function.*

1.0 Introduction

The novel coronavirus disease (COVID-19) was first confirmed in the Chinese city of Wuhan, late December, 2019. The rapidity of its spread in many countries around the globe made the WHO declare it as a global pandemic and public health emergency, raising concerns that if countries with robust healthcare systems to detect and control disease outbreak are having challenges managing the disease, countries with weak healthcare system need to put adequate measures in place to contain the spread [1]. The coronavirus disease (COVID-19) caused by the Severe Acute Respiratory Syndrome Coronavirus-2 (SARS-CoV-2) presents clinical features which are similar to the diseases caused by other coronaviruses, Severe Acute Respiratory Syndrome (SARS) and Middle East Respiratory Syndrome such as lower respiratory illness with fever, dry cough, myalgia, shortness of breath etc. The Coronavirus disease is *"novel"* in the sense that, it is a new strain of zoonotic origin which has not been previously discovered to affect humans. Historically, the COVID-19 pandemic is a major human coronavirus epidemic in the last two decades aside SARS [2] and MERS [3, 4] respectively. The incubation period of COVID-19 is between 2 – 14 days with symptoms averagely between 5-7 days. Its basic reproduction number is averaged 2.2 [5] and even more ranging from 1.4 – 6.5 in [6]. Globally as at May 5th, 2020, there are 3, 646,304 confirmed cases, 1,200, 296 recovered and 252,425 deaths.

The *SARS-CoV-2* is an enveloped positive-sense stranded RNA virus (*ssRNA*) (Subgenus*: Sarbecovirus, Orthocoronavirinae* subfamily), consisting of 29,903 nucleotides and two untranslated sequences of 254 and 229 nucleotides at the 5' and 3'-ends respectively (GenBank No. MN 908947) [7]. The coronaviruses *(CoV)* are subdivided into 4 genera $\alpha-,\beta-,\gamma-,\delta-¿$ coronaviruses. The $\alpha-¿$ and $\beta-¿$ CoV infects mammals while the $\gamma-¿$ and $\delta-CoV$ infect birds. Previously, 6-*CoVs* have been identified as human susceptible virus, which are $\alpha-CoVs$, $HCoV-229E$, $HCoV-NL63$, $\beta-CoVs$, $HCoV-HKUI$ and $HCoV-OC43$. Specifically, SARS-CoV-2 is of the $\beta-¿$ type, it enters the human epithelial via the binding of the viral spike protein ($S-Protein$) to the human Angiotensin Converting Enzyme 2 (ACE-2) which is a functional receptor involved in arterial blood

pressure control and fibrotic response to damage, using it as its binding site. [8, 9]. Basically, there are 3 main transmission routes for the disease which are: *Droplets transmissions* which occurs when respiratory droplets produced by infected persons when they coughs or sneezes in close proximity with other people are inhaled or ingested; *Contact transmission* which occur when people touches infected surfaces and subsequently touch their mouth, nose or eyes; *Aerosol transmission* which occur when respiratory droplets mix with the air, forming aerosols are inhaled in high doses which is potent enough to cause infection [11].

1.1 Models for Incidence and Treatment Parameters

According to [20], *Incidence* is the rate at which the susceptible become infectious. Thus, if the unit of time is days, then the incidence is the number of new infections per day. The classical Kermack-McKendrick model in [21] proposed a *Bilinear Incidence* rate for simple mass action denoted as $h(I)=\beta SI$, where S and I denote the susceptible and Infected respectively and β denote the probability of transmission per contact. Next, we have *Standard Incidence* which is denoted as $\frac{\beta SI}{N}$, where N is the total population size, β is the average number of effective contacts per unit time of an infective with the susceptible (daily contact rate), since $\frac{S}{N}$ is the susceptible fraction of the population, $\frac{\beta SI}{N}$ is the average number of infection transmissions by all infectives. According to literatures the bilinear and standard incidences has been extensively studied by various authors in [33 - 36] and others. Another kind of incidence that is of interest to our work is the *Saturation incidence*. In 1973, sequel to the study of cholera epidemic which occurred in Bari, Capasso and Serio [22] introduced the saturated incidence denoted as $h(I)S$ into epidemic models, where $h(I)$ tends to a saturation level when I becomes large i.e.

$$h(I)S=\frac{\beta I}{1+\alpha I}S \to \frac{\beta}{\alpha}S \text{ as } I \to \infty \quad \ldots. (1)$$

where βI denotes the infection force of the disease and $\frac{I}{1+\alpha I}$ measures the inhibitory effect from the behavioural change of the susceptible when their number of incidence increases or from the crowding effect of the infective due to unrestrained contact using suitable parameters. Esteva and Matias [20] studied a model for disease transmitted by vector with saturating incidence such that the model assumes a saturating effect in the incidence due to the response of the vector to change in the susceptible and infected densities. The Saturation incidence seems more realistic than the bilinear incidence due to the inclusion of behavioural change and crowding effect of the infective. In the face of the current realities from COVID-19, it is evident that we have high saturation incidence in which useful strategies need to be deployed to contain the spread through various interventions such as good hygiene, physical/social distancing, partial/total lockdown, travel/public gathering ban, good treatments, contact tracing, pool testing etc can help to reduce the high rate of secondary infections as stipulated by the WHO guidelines.

It is a general assumption in classical epidemic models that treatment rate of infection is assumed to be proportional to the number of the infective individuals and the recovery rate depends on the medical resources available such as test kits, drugs, isolation centres, ventilators, availability of trained medical personnel, efficiency of treatment. WHO situation reports from many nations have shown how stretched the healthcare systems of countries have been with its attendant high morbidity. Therefore, it is important for countries with increased cases to adopt suitable treatment function.

Wang and Ruan [23] introduced a constant treatment in SIR models as follows:

$$T(I)=\begin{cases} r, I>0 \\ 0, I=0 \end{cases} \ldots\ldots(2)$$

Where r is the positive constant and I is the number of infected individuals.

Recently, Wang [24] considered a piecewise linear treatment function defined as:

$$T(I)=\begin{cases} rI, 0\le I\le I_0 \\ k, I>I_0 \end{cases} \ldots\ldots..(3)$$

where $k=rI_0$, $r\wedge I_0$ are positive constant.

The first conditions in (3) explains the proportionality of the treatment response to the number of the infectious people when the number of infectious is less than or equal to a fixed value I_0, the second typifies an endemic situation, $(I>I_0)$, where the number of the infectious has increased to a saturation point where the available medical facilities are stretched beyond capacity and death toll rises in an unprecedented manner. Therefore in many disease outbreak there are different kinds of delays when they spread, such as latent period delay before symptoms surfaces and immunity period delay after recovery. Zhou [25] studied an SIR epidemic model with treatment function $h^{i}=\frac{\alpha I}{\omega+I}$. Zhang et'al [26] modified the model in [25], with saturated incidence rate, $\frac{\beta S}{1+\alpha S}$ using the same treatment function. In [27] Agrawal et'al modified the work of [26] considering an SEIRS epidemic model with saturated incidence and treatment rate. Badole et'al in [28] taking some cue from [27] studied the global dynamics of an SEIR model with saturated incidence under treatment. Various authors have considered saturated incidence and treatment to study the stability and bifurcation of different dynamic systems in [24, 34, 37 - 39].

1.2 Existing Compartmental Models for COVID-19

Since the outbreak, many mathematical have appeared in an attempt to assess the dynamics of the COVID-19 epidemic. The first models were dynamic mechanistic models aimed at estimating of the basic reproduction number R_0 [12, 13, 14, 15], also simple exponential growth models [16, 17]. Other compartmental epidemiological models such as SIRD, SEIR, SEIRD and SEIRUS [21, 19, 18, 10,] has been proposed to estimate other epidemiological parameters such as the transmission rate, local and global stability of the disease-free and endemic equilibria to provide insights for forecasting purposes.

Recently, [10] considered an SEIRUS (Susceptible-Exposed-Infected-Recovered-Undetectable-Susceptible) model for COVID-19, where it was predicted that with strict adherence to the guidelines of the WHO on observatory and treatment procedures, the pandemic will soon die out. Based on the motivation from [10, 24, 27, 28 and 29], we present an SEIRS (Susceptible-Exposed-Infected-Recovered-Susceptible) model with saturated incidence and treatment functions which prescribes inbihitory measures such as personal hygiene, wearing of face mask, travel/public gathering ban, partial or total lockdown etc and rapid responses such as public enlightenment, pool testing, increased medical facilities and trained medical personnel etc., as . potent means of slowing down the spread of COVID-19.

2.0 Model Description

The model can be described as follows:

$$S'(t) = A - \mu S - \frac{\beta SI}{1+\alpha I} + (\rho + \varepsilon)$$

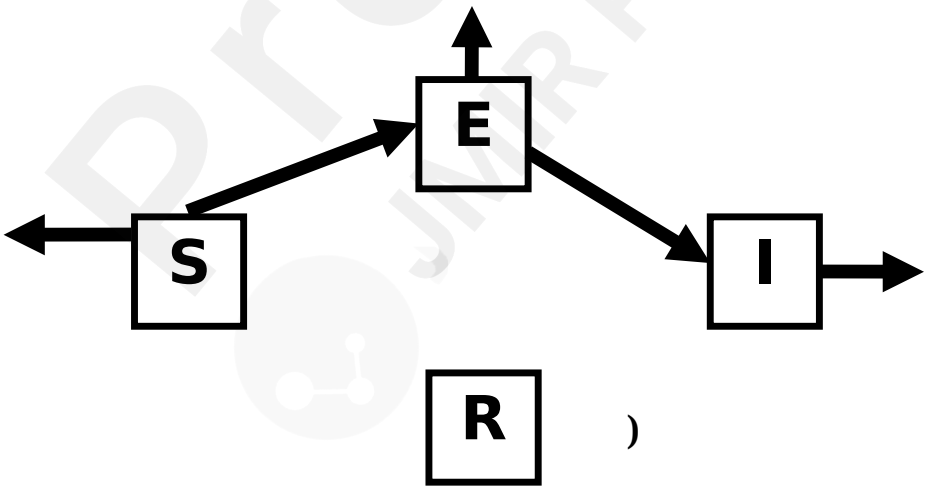
$$E'(t) = \frac{\beta SI}{1+\alpha I} - (\gamma + \mu) E \ldots\ldots\ldots (4)$$

$$I'(t) = \gamma E - (\sigma + \mu + \varphi) - T(I)$$

$$R'(t) = \sigma I - (\rho + \varepsilon + \mu + \varpi) R + T(I)$$

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

Where $S(t)$ is the number of susceptible per unit time, $E(t)$ is the number of the exposed per unit time, $I(t)$ is the number of the Infectious per unit time, $R(t)$ is the number of the recovered per unit time, A is the recruitment rate of the population, μ is the natural death rate of the population per time, ρ is the recovery rate, α is the saturation parameter that measures the inhibitory effect, β is the rate of transmission ε is the proportion of the removed population that is been observed and will subsequently moved to the susceptible, γ is the rate of developing infection/incidence rate. φ is the disease induced death rate of the infected population not quarantined ϖ is the fraction of the removed population under observation (the undetected) before moving to the susceptible class, $h(I)S = \frac{\beta SI}{1+\alpha I}$ is the saturation incidence parameter, $\frac{1}{1+\alpha I}$ is the inhibitory parameter and $T(I)$ is the treatment response as defined in (3).



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It follows from (4) that

$$(S+E+I+R)' = A - \mu(S+E+I+R) - \varphi I - \varpi R$$
$$\leq A - \mu(S+E+I+R)$$

The $\lim_{n \to \infty}$ $_{(S+E+I+R \leq \frac{A}{\mu}}$. The feasible region for system (4) is

$$\Omega = \{(S+E+I+R): \lim_{n \to \infty}{}^{(S+E+I+R \leq \frac{A}{\mu}}, S \geq 0, E \geq 0, I \geq 0, R \geq 0\}$$

Thus is naturally follows that the region Ω is positively invariant with respect to system (4). Hence the system is mathematically and epidemiological well posed in Ω.

2.1 Disease Free State Equilibrium

Case I – when $T(I)=rI$, if $0\le I\le I_0$ we have from (4)

$$S'(t)=A-\mu S-\frac{\beta SI}{1+\alpha I}+(\rho+\varepsilon)$$

$$E'(t)=\frac{\beta SI}{1+\alpha I}-(\gamma+\mu)E \ldots\ldots\ldots(5)$$

$$I'(t)=\gamma E-(\sigma+\mu+\varphi)I-rI$$

$$R'(t)=\sigma I-(\rho+\varepsilon+\mu+\varpi)R+rI$$

By simple calculation from the system (5) we obtain the equilibrium state where $S'(t)=E'(t)=I'(t)=R'(t)=0$ (i.e. the LHS vanish). Thus the steady state of system (5) satisfy the following algebraic system of equation:

$$S^{\iota}=\frac{(A+(\rho+\varepsilon)R)(1+\alpha I)}{\mu(1+\alpha I)+\beta I},\quad E^{\iota}=\frac{BI(A+(\rho+\varepsilon)R)(1+\alpha I)}{(\gamma+\mu)(\mu(1+\alpha I)+\beta I)},$$

$$I^{\iota}=\frac{\gamma\beta I(A+(\rho+\varepsilon)R-r)}{(\gamma+\sigma+\varphi+r+2\mu)(\mu(1+\alpha I)+\beta I)},\quad R^{\iota}=\frac{(\sigma+r)I}{(\rho+\varepsilon+\mu+\varpi)} \quad \ldots\ldots (6)$$

At the disease free equilibrium, when no disease outbreak occurs, no one is in the exposed or infected class and as such no one is in the recovered class. Therefore, $E=I=0$. On substitution the algebraic in (6) reduces to $S=\frac{A}{\mu}$.

Thus the system (5) has a disease free equilibrium $E_0=(\frac{A}{\mu},0,0,0)$.

2.2 Reproduction Number

The reproduction number R_0, measures the probability of an infectious disease spreading through a population or becoming extinct after sometime. This threshold characteristic of R_0 help epidemiologist to make the following assumptions: (a) If $R_0<1$, the infection will die out with time and (b) If $R_0>1$, the disease will be endemic in the population.

Next we find the reproduction number, R_0 of the system (5) by obtaining the Jacobian of the system and using the Next Generation Matrix due to Driessche and Watmough [32].

$$J(S^{¿},E^{¿},I^{¿},R^{¿})=\begin{pmatrix} -\left(\mu+\frac{\beta I}{1+\alpha I}\right) & 0 & \frac{\beta S}{(1+\alpha I)^2} & (\rho+\varepsilon) \\ \frac{\beta I}{1+\alpha I} & -(\gamma+\mu) & \frac{\beta S}{(1+\alpha I)^2} & 0 \\ 0 & \gamma-(\sigma+\mu+\varphi+r) & 0 & \\ 0 & 0(\sigma+r) & -(\rho+\varepsilon+\mu+\varpi) & \end{pmatrix} \quad \text{.....(7)}$$

Applying the disease free state condition $E_0=(\frac{A}{\mu},0,0,0)$ in the Jacobian matrix, we have

$$J(\frac{A}{\mu},0,0,0)=\begin{bmatrix} -\mu & 0 & \frac{\beta A}{\mu} & (\rho+\varepsilon) \\ \frac{\beta I}{1+\alpha I} & -(\gamma+\mu) & \frac{\beta A}{\mu} & 0 \\ 0 & \gamma-(\sigma+\mu+\varphi+r) & 0 & \\ 0 & 0 & (\sigma+r) & -(\rho+\varepsilon+\mu+\varpi) \end{bmatrix} \quad \text{..... (8)}$$

Using the next generation matrices it is clear that the reproduction number R_0 is the spectral radius of the next generation matrix derived from the exposed and infected class i.e.

$$R_0=\rho(K) \quad \text{..... (9)}$$

Where $\rho(K)$ *is the spectral radius* and $K=FV^{-1}$, is the next generation matrix. F is derived from the exposed and infected class and V are the remaining terms after F is taken.

Thus

$$F=\begin{bmatrix} 0 & \frac{\beta A}{\mu} \\ 0 & 0 \end{bmatrix}$$

$$V=\begin{bmatrix} -(\gamma+\mu) & 0 \\ \gamma & -(\sigma+\mu+\varphi+r) \end{bmatrix}$$

$$V^{-1}=\begin{bmatrix} (\gamma+\mu) & 0 \\ -\gamma & (\sigma+\mu+\varphi+r) \end{bmatrix}$$

$$K=FV^{-1}=\begin{bmatrix} 0 & \frac{\beta A}{\mu} \\ 0 & 0 \end{bmatrix}\begin{bmatrix} (\gamma+\mu) & 0 \\ -\gamma & (\sigma+\mu+\varphi+r) \end{bmatrix}$$

$$¿\begin{bmatrix} \frac{\gamma\beta A}{\mu(\mu+\gamma)(\sigma+\mu+\varphi+r)} & \frac{\beta A}{\mu(\mu+\gamma)(\sigma+\mu+\varphi+r)} \\ 0 & 0 \end{bmatrix}$$

K is the next generation matrix of the system (5).
The spectral radius is

$$\rho(K)=\frac{\gamma\beta A}{\mu(\mu+\gamma)(\sigma+\mu+\varphi+r)}$$

Hence the reproduction number, R_0 of system (5) is

$$R_0=\frac{\gamma\beta A}{\mu(\mu+\gamma)(\sigma+\mu+\varphi+r)}\ldots\ldots\ldots(10)$$

Lemma 2.1: The system (5) has a disease free equilibrium point. If $N=\frac{A}{\mu}$

Proof: Consider $N(t)=S(t)+E(t)+I(t)+R(t)$. Then

$\frac{dN}{dt}=A-\mu N(t)$

Integrating both sides

$N=A-\mu N(t)$

Simplify and thus

$\lim_{t\to\infty} N=\frac{A}{\mu}$ ∎

2.3 Local Stability Analysis of the Disease Free Equilibrium

We examined the local stability of the equilibrium by the analysis of the eigenvalues of the Jacobian matrices of (5) at the equilibrium using the *Routh Hurwitz Criterion*.

Theorem 2.2: The disease free equilibrium (E^0) is

(a) Locally asymptotically stable if $R_0<1$

(b) Unstable if $R_0>1$.

Proof: The Jacobian matrix of the system (5) at the disease free equilibrium

$$E^0=J\left(\frac{A}{\mu},0,0,0\right)=\begin{bmatrix} -\mu & 0 & \frac{\beta A}{\mu} & (\rho+\varepsilon) \\ \frac{\beta I}{1+\alpha I} & -(\gamma+\mu) & \frac{\beta A}{\mu} & 0 \\ 0 & \gamma-(\sigma+\mu+\varphi+r) & & 0 \\ 0 & 0 & (\sigma+r) & -(\rho+\varepsilon+\mu+\varpi) \end{bmatrix}$$

The characteristic equation of the system (5) at E^0 is

$$(\mu+\lambda)(\sigma+\mu+\varphi+r+\lambda)\left[\lambda^2+(\gamma+\sigma+\mu+\varphi+r+2\mu)\lambda+(\sigma+\mu+\varphi+r)(\gamma+\mu)-\frac{\gamma\beta A}{\mu}\right]=0 \ldots (11)$$

By (11) it is clear that $\lambda_1=-\mu$, $\lambda_2=-(\sigma+\mu+\varphi+r)$ are the two roots of (11). The other roots of (11) are determined by the equation.

$$\lambda^2+(\gamma+\sigma+\mu+\varphi+r+2\mu)\lambda+(\sigma+\mu+\varphi+r)(\gamma+\mu)-\frac{\gamma\beta A}{\mu}=0$$

Which has negative roots if and only of $(\sigma+\mu+\varphi+r)(\gamma+\mu)-\frac{\gamma\beta A}{\mu}>0$, which implies that the reproduction number, $R_0<1$. This implies that the disease free equilibrium E^0 is locally asymptotically stable when $R_0<1$ and unstable when $R_0>1$ ∎

Theorem 2.3: If $R_0<1$, then the endemic equilibrium $E^*(S^*,E^*,I^*,R^*)$ is locally asymptotically stable.

Proof: We recall the Jacobian of system (5) at the endemic state E^* in (7),

$$J(S^*,E^*,I^*,R^*)=\begin{pmatrix} -\left(\mu+\frac{\beta I^*}{1+\alpha I^*}\right) & 0 & \frac{\beta S^*}{(1+\alpha I^*)^2} & (\rho+\varepsilon) \\ \frac{\beta I^*}{1+\alpha I^*} & -(\gamma+\mu)\frac{\beta S^*}{(1+\alpha I^*)^2} & 0 & \\ 0 & \gamma-(\sigma+\mu+\varphi+r) & 0 & \\ 0 & 0 & (\sigma+r) & -(\rho+\varepsilon+\mu+\varpi) \end{pmatrix}$$

From which we obtain the characteristic equation

$$\lambda^4+a_1\lambda^3+a_2\lambda^2+a_3\lambda+a_4=0 \quad \ldots\ldots (12)$$

where a_1, a_2, a_3, and a_4 are as defined below

$$a_1=(\gamma+\sigma+\varphi+r+\rho+\varepsilon+\varpi+4\mu)+\frac{\beta I^*}{1+\alpha I^*}$$

$$a_2=(\rho+\varepsilon+\mu+\varpi)\left(\mu+\frac{\beta I^*}{1+\alpha I^*}\right)+(\gamma+\mu)\left(\rho+\varepsilon+\varpi+2\mu+\frac{\beta I^*}{1+\alpha I^*}\right)+(\sigma+\mu+\varphi+r)\left(\gamma+\rho+\varepsilon+\varpi+3\mu+\frac{\beta I^*}{1+\alpha I^*}\right)-\frac{\gamma\beta S^*}{(1+\alpha I^*)^2}$$

$$a_3=\left(\mu+\frac{\beta I^*}{1+\alpha I^*}\right)[(\rho+\varepsilon+\mu+\varpi)(\gamma+\sigma+\varphi+r+2\mu)]+(\gamma+\mu)\left(\rho+\varepsilon+\varpi+2\mu+\frac{\beta S^*}{(1+\alpha I^*)^2}\right)(\sigma+\mu+\varphi+r)-\frac{\gamma\beta S^*}{(1+\alpha I^*)^2}(\rho+\varepsilon+\varpi$$

$$a_4=\left(\mu+\frac{\beta I^*}{1+\alpha I^*}\right)(\gamma+\mu)(\rho+\varepsilon+\mu+\varpi)(\sigma+\mu+\varphi+r)+\frac{\gamma\beta}{1+\alpha I^*}\left[\frac{(\rho+\varepsilon+\mu+\varpi)S^*I^*}{(1+\alpha I^*)^2}-\left\{\frac{(\rho+\varepsilon+\mu+\varpi)S^*}{1+\alpha I^*}\left(\mu+\frac{\beta I^*}{1+\alpha I^*}\right)+\rho(\gamma+\right.\right.$$

Thus by direct computation, we have that

$$a_1>0, a_3>if -\frac{\gamma\beta S^*}{(1+\alpha I^*)^2}(\rho+\varepsilon+\varpi+2\mu)<0,$$

$$a_4>0 \text{ if } \frac{(\rho+\varepsilon+\mu+\varpi)S^*}{1+\alpha I^*}\left(\mu+\frac{\beta I^*}{1+\alpha I^*}\right)+\rho(\gamma+\mu)I^*<0$$

and $a_1 a_2 a_3 > a_3^2 + a_1^2 a_4$. Therefore, by Routh-Hurwitz Criterion, it follows that the endemic equilibrium $E^{¿}(S^{¿}, E^{¿}, I^{¿}, R^{¿})$ of (5) is locally asymptotically stable ∎

In this section, we analyze the global stability of the disease-free equilibrium.

Theorem 2.4: If $R_0^{¿} < 1$, then the disease-free state equilibrium E^0 is globally asymptotically stable.

Proof: If $R_0^{¿} < 1$, then $R_0 < 1$, from the system of equation in (5), we have $\frac{dS}{dt} \leq A - \mu S$. A solution of the equation $\frac{dy}{dt} \leq A - \mu y$ is a maximal solution of $S(t)$. We recall that $y \to \frac{A}{\mu}$ as $t \to \infty$. By applying the comparison theorem, we obtain $S(t) \leq \frac{A}{\mu}$, also from the set

$$\Omega = \{(S+E+I+R) : S+E+I+R \leq \frac{A}{\mu}, S \geq 0, E \geq 0, I \geq 0, R \geq 0\}$$

We have $I(t) \leq \frac{A}{\mu}$.

Define the Lyapunov function

$$L = \gamma E - (\gamma + \mu) I \quad \ldots\ldots (13)$$

From $R_0^{¿} < 1$, we have $\beta\gamma\left(\frac{A}{\mu}\right) - (\gamma+\mu)(\sigma+\mu+\varphi+r) < 0$. Thus

$$L' = \gamma E' - (\gamma+\mu) I'$$

$$L' = \gamma\left[\frac{\beta S I}{1+\alpha I} - (\gamma+\mu)E\right] + (\gamma+\mu)[\gamma E - (\sigma+\mu+\varphi+r) I]$$

$$¿\left[\frac{\gamma\beta S}{1+\alpha I} - (\gamma+\mu)(\sigma+\mu+\varphi+r)\right] I$$

$$\leq [\gamma\beta S - (\gamma+\mu)(\sigma+\mu+\varphi+r)] I$$

recall that $S = \frac{A}{\mu}$. Therefore

$$\leq \left[\gamma\beta\left(\frac{A}{\mu}\right) - (\gamma+\mu)(\sigma+\mu+\varphi+r)\right] I \leq 0 \quad \ldots\ldots (14)$$

And $L' = 0$, if and only if $I = 0$. Thus the largest compact invariant set in $\{(S, E, I, R) \in \Omega, L' = 0\}$ is the singleton E^0. Therefore, by Lasalle-Lyapunov theorem, every solution that starts in Ω approaches E^0 as $t \to \infty$ and the proof is complete ∎

3.0 Global Stability of the Disease Free Equilibrium

In this section we analyze the global stability of the disease-free equilibrium and to do this we reduce the system of equation in (5) using $R(t) = \frac{A}{\mu} - S(t) - E(t) - I(t)$ to eliminate the $R(t)$ component from the first equation of system (5) to obtain a three-dimensional given below

$$S'(t)=\frac{A}{\mu}-\mu E-\frac{\beta SI}{1+\alpha I}+(\sigma+\mu+\varphi-r)I$$

$$E'(t)=\frac{\beta SI}{1+\alpha I}-(\gamma+\mu)E \quad \ldots\ldots(15)$$

$$I'(t)=\gamma E-(\sigma+\mu+\varphi+r)I$$

with initial conditions $S\geq 0, E\geq 0, I\geq 0$

We consider the geometric approach due to Li and Muldowney **[**31**]**, to obtain the global stability of the endemic equilibrium and find that the sufficient conditions for which the endemic equilibrium is globally asymptotically stable. We describe the geometric approach method as follows. We consider

$$x'=f(x) \quad \ldots\ldots (16)$$

Where $f:D\to R^n, D\subset R^n$ is an open set and is simply connected and $f\in C^1(D)$.

Let x^* be the solution of the equation (15) i.e. $f(x^*)=0$. Assume that the following hypotheses hold.

$(Y1)$: There exists a compact absorbing set $K\subset D$.
$(Y2)$: Equation (16) has a unique equilibrium $x^*\in D$.

The basic idea in this method is that if the equilibrium x^* is locally stable, then the stability will hold provided the conditions in $(Y1)$ and $(Y2)$ is satisfied and no non constant periodic solution of (16) exists. Thus, the sufficient conditions on f is capable of precluding the existence of such solutions solutions must be found.

Suppose that the conditions $(Y1)$ and $(Y2)$ is satisfied. Assume that (16) satisifies a Bendixson criteria that is robust under C^1 local perturbations of f at all non-equilibrium non-wandering points for (16). The x^* is globally stable in D provided it is stable. Let $P(x)$ be a $\binom{n}{2}\times\binom{n}{2}$ matrix valued function that is C^1, on D and consider

$$B=P_f P^{-1}+P\frac{\partial F^{[2]}}{\partial x}P^{-1} \quad \ldots\ldots (17)$$

Where the matrix P_f is

$$\frac{\partial P_{ij}^*}{\partial x}f=\frac{dP_{ij}}{dt}^{(10)} \quad \ldots\ldots.. (18)$$

and the matrix $J^{[2]}$ is the second additive compound matrix of the Jacobian matrix J, that is, $J(x)=Df(x)$. Generally speaking for an $n\times n$ matrix $J=(J_{ij})$, $J^{[2]}$ is an $\binom{n}{2}\times\binom{n}{2}$ matrix and in the special case $n=3$ one has

$$J^{[2]}=\begin{pmatrix} J_{11}+J_{22} & J_{23} & -J_{13} \\ J_{32} & J_{11}+J_{33} & J_{12} \\ -J_{31} & J_{21} & J_{22}+J_{33} \end{pmatrix}\ldots..(19)$$

Consider the Lozinski$\check{i}$ measure μ of B with respect of a vector norm $\|.\|$ in R^N, $N=\binom{n}{2}$, defined by

$$\mu(B)=\lim_{h\to 0^{+}}\frac{\|I+hB\|-1}{h}\ldots\ldots\ldots(20)$$

It is proved in [31] that if $(K1)$ and $(K2)$ hold, condition

$$q=\lim_{t\to\infty}\sup_{x_0\in K}\frac{1}{t}\int_0^t \mu(B(x(s,x_0)))ds<0\ldots\ldots(21)$$

It is shown in [31] that, if D is simply connected, the condition $q>0$ rules out the presence of any orbit that gives rise to a simple closed rectifiable curve that is invariant for (16), such as periodic orbits, homoclinic orbits and heteroclinic cycles. Moreover, it is robust under C^1 local perturbations of f near any non equilibrium point that is non-wandering. In particular, the following global stability result is proved in [31]**.**

Lemma 3. 1: Assume that D is simply connected and that the assumptions $(Y1)$ and $(Y2)$ is satisified. Then the unique equilibrium x^* of (16) is globally stable in D if $q<0$ ∎

Having established the above, we study the global stability of the endemic equilibrium P^* and obtain.

Theorem 3.2 If $R_0>1$ then the endemic equilibrium P^* of the system (15) is globally stable.

Proof: The Jacobian matrix of system (15) is given as

$$J=\begin{pmatrix}\frac{-\beta SI}{1+\alpha I} & -\mu & \frac{-\beta S}{(1+\alpha I)^2}+(\sigma+\varphi+r-\mu)I\\ \frac{\beta SI}{1+\alpha I} & -(\gamma+\mu) & \frac{\beta S}{(1+\alpha I)^2}\\ 0 & \gamma & -(\sigma+\mu+\varphi+r)\end{pmatrix}\ldots\ldots(22)$$

And its second additive matrix is

$$J^{[2]}=\begin{pmatrix}-(\gamma+\mu+\frac{\beta SI}{1+\alpha I}) & \frac{\beta S}{(1+\alpha I)^2} & \frac{\beta S}{(1+\alpha I)^2}-(\sigma+\varphi+r-\mu)\\ \gamma & -(\sigma+\mu+\varphi+r+\frac{\beta SI}{1+\alpha I}) & -\mu\\ 0 & \frac{\beta SI}{1+\alpha I} & -(\gamma+\sigma+\varphi+r+2\mu)\end{pmatrix}\ldots..(23)$$

Choose the function $P=P(S,E,I)=diag\left(1,\frac{E}{I},\frac{E}{I}\right)$; then $P^{-1}=diag\left(1,\frac{I}{E},\frac{I}{E}\right)$ and

$$P_f=diag\left(0,\frac{E^{'}I-I^{'}E}{I^2},\frac{E^{'}I-I^{'}E}{I^2}\right)\ldots\ldots\ldots(24)$$

Then we have

$$P_f P^{-1}=diag\left(0,\frac{E^{'}}{E}-\frac{I^{'}}{I},\frac{E^{'}}{E}-\frac{I^{'}}{I}\right)$$

$$PJ^{[2]}P^{-1}=\begin{pmatrix} -(\gamma+\mu)-\frac{\beta SI}{1+\alpha I} & \frac{\beta SI}{(1+\alpha I)^2 E} & \frac{\beta SI}{(1+\alpha I)^2 E}-(\sigma+\varphi+r-\mu)\frac{I}{E} \\ \frac{\gamma E}{I} & -(\sigma+\mu+\varphi+r)-\frac{\beta SI}{1+\alpha I} & -\mu \\ 0 & \frac{\beta SI}{1+\alpha I} & -(\gamma+\sigma+\varphi+r+2\mu) \end{pmatrix} \ldots(24)$$

The matrix $B=P_f P^{-1}+PJ^{[2]}P^{-1}$ can be written in matrix form as

$$B=\begin{pmatrix} B_{11} & B_{12} \\ B_{21} & B_{22} \end{pmatrix} \ldots\ldots(25)$$

Where

$$B_{11}=\begin{pmatrix} -(\gamma+\mu)-\frac{\beta SI}{1+\alpha I} \end{pmatrix}, \qquad B_{12}=\begin{pmatrix} \frac{\beta SI}{(1+\alpha I)^2 E}, \frac{\beta SI}{(1+\alpha I)^2 E}-(\sigma+\varphi+r-\mu)\frac{I}{E} \end{pmatrix}$$

$$B_{21}=\begin{pmatrix} \frac{\gamma E}{I}, 0 \end{pmatrix}^T,$$

$$B_{22}=\begin{pmatrix} -(\sigma+\mu+\varphi+r)-\frac{\beta SI}{1+\alpha I}+\frac{E'}{E}-\frac{I'}{I} & -\mu \\ \frac{\beta SI}{1+\alpha I} & -(\gamma+\sigma+\varphi+r+2\mu)+\frac{E'}{E}-\frac{I'}{I} \end{pmatrix}$$

Let (u,v,w) be a vector in R^3, its norm $\|\cdot\|$ is defined as

$$\|(u,v,w)\|=max\{|u|,|v|+|w|\}\ldots..(26)$$

Let $\mu(B)$ be the Lozinskiĭ measure with respect to this norm. Thus we choose

$\mu(B)\leq\{g_1,g_2\}$

where $g_1=\mu_1(B_{11})+|B_{12}|$ and $g_2=\mu_2(B_{22})+|B_{21}|$

$|B_{12}|$, $|B_{21}|$ are matrix norm with respect to l_1 vector norm and μ_1 denotes the Lozinskiĭ measure with respect to l_1 vector norm then,

$$\mu_1(B_{11})=-(\gamma+\mu)-\frac{\beta SI}{1+\alpha I} \ldots.. (26)$$

$$|B_{12}|=max\left(\frac{\beta SI}{(1+\alpha I)^2 E}, \frac{\beta SI}{(1+\alpha I)^2 E}-(\sigma+\varphi+r-\mu)\frac{I}{E}\right)=\frac{\beta SI}{(1+\alpha I)^2 E}$$

$$|B_{21}|=\frac{\gamma E}{I}$$

Now calculating $\mu_2(B_{22})$, taking the non-diagonal elements of each columns of B_{22} in absolute value, and then adding to the corresponding columns of the diagonal elements, we have

$$B_{22}^{'}=\begin{pmatrix} -(\sigma+\mu+\varphi+r)-\frac{\beta SI}{1+\alpha I}+\frac{E^{'}}{E}-\frac{I^{'}}{I} & -\mu \\ \frac{\beta SI}{1+\alpha I} & -(\gamma+\sigma+\varphi+r+2\mu)+\frac{E^{'}}{E}-\frac{I^{'}}{I} \end{pmatrix} \ldots (27)$$

Take the maximum of the two diagonal elements of $B_{22}^{'}$, we have

$$\mu_2(B_{22})=max\{-(\sigma+\mu+\varphi+r)+\frac{E^{'}}{E}-\frac{I^{'}}{I},-(\gamma+\sigma+\varphi+r+3\mu)+\frac{E^{'}}{E}-\frac{I^{'}}{I}\}$$

¿$-(\sigma+\mu+\varphi+r)+\frac{E^{'}}{E}-\frac{I^{'}}{I}$ ….. (28)

Therefore, we have

$$\left.\begin{aligned} g_1=\mu_1(B_{11})+|B_{12}|=¿ \quad \mu_1(B_{11})=-(\gamma+\mu)-\frac{\beta SI}{1+\alpha I}+\frac{\beta SI}{(1+\alpha I)^2 E} \\ g_2=\mu_2(B_{22})+|B_{21}|=-(\sigma+\mu+\varphi+r)+\frac{E^{'}}{E}-\frac{I^{'}}{I}+\frac{\gamma E}{I} \end{aligned}\right\} \quad \ldots\ldots (29)$$

From the system of equation in (5) we have

$$\frac{E^{'}}{E}=\frac{\beta SI}{(1+\alpha I)E}-(\gamma+\mu) \text{ and } \frac{I^{'}}{I}=\frac{\gamma E}{I}-(\sigma+\mu+\varphi+r) \ldots\ldots\ldots\ldots (30)$$

We have,

$$g_1=¿ \quad \mu_1(B_{11})=-(\gamma+\mu)-\frac{\beta SI}{1+\alpha I}+\frac{\beta SI}{(1+\alpha I)^2 E}\leq\frac{\beta SI}{(1+\alpha I)E}-(\gamma+\mu)$$

$$g_2=-(\sigma+\mu+\varphi+r)+\frac{E^{'}}{E}-\frac{I^{'}}{I}+\frac{\gamma E}{I}=\frac{\gamma E}{I}-(\sigma+\mu+\varphi+r)$$

From which we obtain

$$g_1\leq\frac{E^{'}}{E}-\mu \text{ and } g_2=\frac{E^{'}}{E}-\mu\leq\frac{E^{'}}{E}-(\gamma-\mu) \ldots\ldots\ldots (31)$$

Also, we have

$$\mu(B)\leq\{g_1,g_2\}$$

$$\leq\left\{\frac{E^{'}}{E}-\mu,\frac{E^{'}}{E}-(\gamma-\mu)\right\}$$

$$\leq \frac{E^{'}}{E} - (\gamma - \mu) \ldots . (32)$$

Integrating both sides simultaneously, we have

$$\frac{1}{t}\int_0^t \mu(B)\,ds \leq \frac{1}{t}\ln\frac{E(t)}{E(0)} - (\gamma - \mu)$$

$$\lim_{t \to \infty} ¿¿ \frac{1}{t}\int_0^t \mu(B)\,ds < -(\gamma - \mu) < 0 \ldots\ldots\ldots (33)$$

Thus, by (17) we have that $P^{¿}$ is globally asymptotically stable ∎

4. 0 Equilibria and Stability of the Endemic Steady State

In this section we discuss the local and global stability of the second case of the treatment function. The system of equation in (5) reduces to

Case II: When $T(I) = k$, if $I > I_0$. Thus, we have the model

$$S^{'}(t) = A - \mu S - \frac{\beta SI}{1+\alpha I} + (\rho + \varepsilon) R$$

$$E^{'}(t) = \frac{\beta SI}{1+\alpha I} - (\gamma + \mu) E \ldots\ldots\ldots (34)$$

$$I^{'}(t) = \gamma E - (\sigma + \mu + \varphi) I - k$$

$$R^{'}(t) = \sigma I - (\rho + \varepsilon + \mu + \varpi) R + k$$

It follows from the system of equation in (34)

$$(S+E+I+R)^{'} = A - \mu(S+E+I+R) - \varphi I - \varpi R$$
$$\leq A - \mu(S+E+I+R)$$

Then,

$$\lim_{n \to \infty^{(S+E+I+R) \leq \frac{A}{\mu}}} \ldots\ldots\ldots\ldots (35)$$

The feasible region for system (4) is $\Omega = \{(S+E+I+R): \lim_{n \to \infty^{(S+E+I+R) \leq \frac{A}{\mu}}}, S \geq 0, E \geq 0, I \geq 0, R \geq 0\}$. Therefore the system of equation (34) is epidemiologically well-posed.

4.1 Local Stability Analysis of the Endemic Equilibrium

We consider the local stability of the endemic equilibrium point $E^{¿}(S^{¿}, E^{¿}, I^{¿}, R^{¿})$ by analyzing the eigenvalues of the Jacobian matrices of equation (34) at the endemic equilibrium point using the

Routh-Hurwitz Criterion.

Theorem 4.1: If $R_0>1$ then the endemic equilibrium $E^{\iota}(S^{\iota},E^{\iota},I^{\iota},R^{\iota})$ is locally asymptotically stable.

Proof: The Jacobian matrix of the system (34) at E^{ι} is

$$J=\begin{pmatrix} -\left(\mu+\frac{\beta I^{\iota}}{1+\alpha I^{\iota}}\right) & 0 & \frac{\beta S^{\iota}}{(1+\alpha I^{\iota})^2} & (\rho+\varepsilon) \\ \frac{\beta I^{\iota}}{1+\alpha I^{\iota}} & -(\gamma+\mu)-\frac{\beta S^{\iota}}{(1+\alpha I^{\iota})^2} & 0 & \\ 0 & \gamma-(\sigma+\mu+\varphi) & 0 & \\ 0 & 0 & \sigma & -(\rho+\varepsilon+\mu+\varpi) \end{pmatrix} \quad \ldots..(36)$$

From which we obtain the characteristic equation

$$\lambda^4+b_1\lambda^3+b_2\lambda^2+b_3\lambda+b_4=0 \quad \ldots\ldots (37)$$

where b_1, b_2, b_3, and b_4 are as defined below

$$b_1=(\gamma+\sigma+\varphi+\rho+\varepsilon+\varpi+4\mu)+\frac{\beta I^{\iota}}{1+\alpha I^{\iota}}$$

$$b_2=(\rho+\varepsilon+\mu+\varpi)\left(\mu+\frac{\beta I^{\iota}}{1+\alpha I^{\iota}}\right)+(\gamma+\mu)\left(\rho+\varepsilon+\varpi+2\mu+\frac{\beta I^{\iota}}{1+\alpha I^{\iota}}\right)+(\sigma+\mu+\varphi)\left(\gamma+\rho+\varepsilon+\varpi+3\mu+\frac{\beta I^{\iota}}{1+\alpha I^{\iota}}\right)-\frac{\gamma\beta S^{\iota}}{(1+\alpha I^{\iota})^2}$$

$$b_3=\left(\mu+\frac{\beta I^{\iota}}{1+\alpha I^{\iota}}\right)\left[(\rho+\varepsilon+\mu+\varpi)(\gamma+\sigma+\varphi+2\mu)\right]+(\gamma+\mu)\left(\rho+\varepsilon+\varpi+2\mu+\frac{\beta S^{\iota}}{(1+\alpha I^{\iota})^2}\right)(\sigma+\mu+\varphi)-\frac{\gamma\beta S^{\iota}}{(1+\alpha I^{\iota})^2}(\rho+\varepsilon+\varpi+2\mu)$$

$$b_4=\left(\mu+\frac{\beta I^{\iota}}{1+\alpha I^{\iota}}\right)(\gamma+\mu)(\rho+\varepsilon+\mu+\varpi)(\sigma+\mu+\varphi)+\frac{\gamma\beta}{1+\alpha I^{\iota}}\left[\frac{(\rho+\varepsilon+\mu+\varpi)S^{\iota}I^{\iota}}{(1+\alpha I^{\iota})^2}-\left\{\frac{(\rho+\varepsilon+\mu+\varpi)S^{\iota}}{1+\alpha I^{\iota}}\left(\mu+\frac{\beta I^{\iota}}{1+\alpha I^{\iota}}\right)+\rho(\gamma+\mu)\right.\right.$$

Thus by direct computation, we have that

$$b_1>0, b_2>0 \; if \; -\frac{\gamma\beta S^{\iota}}{(1+\alpha I^{\iota})^2}<0,$$

$$b_3>0 \; if \; -\frac{\gamma\beta S^{\iota}}{(1+\alpha I^{\iota})^2}(\rho+\varepsilon+\varpi+2\mu)<0,$$

$$b_4>0 \text{ if } \frac{(\rho+\varepsilon+\mu+\varpi)S^{\iota}}{1+\alpha I^{\iota}}\left(\mu+\frac{\beta I^{\iota}}{1+\alpha I^{\iota}}\right)+\rho(\gamma+\mu)I^{\iota}<0$$

and $b_1b_2b_3>b_3^2+b_1^2b_4$. Therefore, by Routh-Hurwitz Criterion, it follows that the endemic

equilibrium E^{*} of system (34) is locally asymptotically stable ∎

4.2 Global Stability of the Endemic Equilibrium

In this section we analyze the global stability of the endemic steady states. After reducing the system of equation in (34) using $R(t)=\frac{A}{\mu}-S(t)-E(t)-I(t)$ to eliminate the $R(t)$ component from the first equation of system (34) to obtain a three-dimensional model.

Theorem 4.2: If $R_0^{*}>1$ then the endemic equilibrium Q^{*} of the system (34) is globally stable.

Proof: The Jacobian matrix of system (34) is given as

$$J(E_2^{*})=\begin{pmatrix} -\mu-\frac{\beta SI}{1+\alpha I} & 0 & \frac{-\beta S}{(1+\alpha I)^2} \\ \frac{\beta SI}{1+\alpha I} & -(\gamma+\mu) & \frac{\beta S}{(1+\alpha I)^2} \\ 0 & \gamma & -(\sigma+\mu+\varphi) \end{pmatrix} \ldots\ldots (38)$$

And its second additive matrix is

$$J^{[2]}=\begin{pmatrix} -(\gamma+2\mu+\frac{\beta SI}{1+\alpha I}) & \frac{\beta S}{(1+\alpha I)^2} & \frac{\beta S}{(1+\alpha I)^2} \\ \gamma & -(\sigma+\varphi+2\mu+\frac{\beta SI}{1+\alpha I}) & 0 \\ 0 & \frac{\beta SI}{1+\alpha I} & -(\gamma+\sigma+\varphi+2\mu) \end{pmatrix} \ldots..(39)$$

Choose the function $Q=Q(S,E,I)=diag\left(1,\frac{E}{I},\frac{E}{I}\right)$; then $Q^{-1}=diag\left(1,\frac{I}{E},\frac{I}{E}\right)$ and

$$Q_f=diag\left(0,\frac{E^{'}I-I^{'}E}{I^2},\frac{E^{'}I-I^{'}E}{I^2}\right).....(40)$$

Then we have

$$Q_f Q^{-1}=diag\left(0,\frac{E^{'}}{E}-\frac{I^{'}}{I},\frac{E^{'}}{E}-\frac{I^{'}}{I}\right)$$

$$QJ^{[2]}Q^{-1}=\begin{pmatrix} -(\gamma+2\mu)-\frac{\beta SI}{1+\alpha I} & \frac{\beta SI}{(1+\alpha I)^2 E} & \frac{\beta SI}{(1+\alpha I)^2 E} \\ \frac{\gamma E}{I} & -(\sigma+\varphi+2\mu)-\frac{\beta SI}{1+\alpha I} & 0 \\ 0 & \frac{\beta SI}{1+\alpha I} & -(\gamma+\sigma+\varphi+2\mu) \end{pmatrix} \ldots(41)$$

The matrix $C=Q_f Q^{-1}+QJ^{[2]}Q^{-1}$ can be written in matrix form as

$$C=\begin{pmatrix} C_{11} & C_{12} \\ C_{21} & C_{22} \end{pmatrix} \ldots\ldots(42)$$

Where

$C_{11}=$¿ $-(\gamma+2\mu)-\frac{\beta SI}{1+\alpha I}$,

$C_{12}=\left(\frac{\beta SI}{(1+\alpha I)^2 E},\frac{\beta SI}{(1+\alpha I)^2 E}\right)$

$C_{21}=\left(\frac{\gamma E}{I},0\right)^T$,

$C_{22}=\begin{pmatrix} -(\sigma+\varphi+2\mu)-\frac{\beta SI}{1+\alpha I}+\frac{E'}{E}-\frac{I'}{I} & 0 \\ \frac{\beta SI}{1+\alpha I} & -(\gamma+\sigma+\varphi+2\mu)+\frac{E'}{E}-\frac{I'}{I} \end{pmatrix}$

Let (u,v,w) be a vector in R^3, its norm $\|\cdot\|$ is defined as

$$\|(u,v,w)\|=max\{|u|,|v|+|w|\}\ldots..(43)$$

Let $\mu(C)$ be the Lozinskiĭ measure with respect to this norm. Thus we choose

$\mu(C)\leq\{f_1,f_2\}$

where $f_1=\mu_1(C_{11})+|C_{12}|$ and $f_2=\mu_2(C_{22})+|C_{21}|$

$|C_{12}|$, $|C_{21}|$ are matrix norm with respect to l_1 vector norm and μ_1 denotes the Lozinskiĭ measure with respect to l_1 vector norm then,

$$\mu_1(C_{11})=-(\gamma+\mu)-\frac{\beta SI}{1+\alpha I}\ldots..(44)$$

$$|C_{12}|=max\left(\frac{\beta SI}{(1+\alpha I)^2 E},\frac{\beta SI}{(1+\alpha I)^2 E}\right)=\frac{\beta SI}{(1+\alpha I)^2 E}$$

$$|C_{21}|=\frac{\gamma E}{I}$$

Clearly, to obtain $\mu_2(C_{22})$, taking the non-diagonal elements of each columns of C_{22} in absolute value, and then add it to the corresponding columns of the diagonal elements, we have

$$C_{22}^{'}=\begin{pmatrix} -(\sigma+\varphi+2\mu)-\frac{\beta SI}{1+\alpha I}+\frac{E^{'}}{E}-\frac{I^{'}}{I} & -\mu \\ \frac{\beta SI}{1+\alpha I} & -(\gamma+\sigma+\varphi+2\mu)+\frac{E^{'}}{E}-\frac{I^{'}}{I} \end{pmatrix} \ldots(45)$$

Take the maximum of the two diagonal elements of $C_{22}^{'}$, we have

$$\mu_2(C_{22})=max\{-(\sigma+\varphi+2\mu)+\frac{E^{'}}{E}-\frac{I^{'}}{I},-(\gamma+\sigma+\varphi+3\mu)+\frac{E^{'}}{E}-\frac{I^{'}}{I}\}$$

¿$-(\sigma+\varphi+2\mu)+\frac{E^{'}}{E}-\frac{I^{'}}{I}$ ….. (46)

We obtain

$$\left.\begin{aligned} f_1=\mu_1(C_{11})+|C_{12}|=¿\quad \mu_1(C_{11})=-(\gamma+\mu)-\frac{\beta SI}{1+\alpha I}+\frac{\beta SI}{(1+\alpha I)^2E} \\ f_2=\mu_2(C_{22})+|C_{21}|=-(\sigma+\mu+\varphi+r)+\frac{E^{'}}{E}-\frac{I^{'}}{I}+\frac{\gamma E}{I} \end{aligned}\right\} \ldots\ldots(47)$$

From the system of equation in (34) we have

$$\frac{E^{'}}{E}=\frac{\beta SI}{(1+\alpha I)E}-(\gamma+\mu) \text{ and } \frac{I^{'}}{I}=\frac{\gamma E}{I}-(\sigma+\mu+\varphi+r)\ldots\ldots\ldots\ldots(48)$$

Then we have

$$f_1=¿\quad -(\gamma+2\mu)-\frac{\beta SI}{1+\alpha I}+\frac{\beta SI}{(1+\alpha I)^2E}\leq\frac{\beta SI}{(1+\alpha I)E}-(\gamma+\mu)$$

$$f_2=-(\sigma+\varphi++2\mu)+\frac{E^{'}}{E}-\frac{I^{'}}{I}+\frac{\gamma E}{I}=\frac{\gamma E}{I}-(\sigma+\mu+\varphi)-\mu+\frac{E^{'}}{E}-\frac{I^{'}}{I}$$

From which we obtain

$$f_1\leq\frac{E^{'}}{E}-\mu \text{ and } f_2=\frac{E^{'}}{E}-\mu+\frac{k}{I}\leq\frac{E^{'}}{E}-(\sigma+\mu)\ldots\ldots\ldots(49)$$

Also, we have

$$\mu(C)\leq\{f_{1,}f_2\}$$

$$\leq\left\{\frac{E^{'}}{E}-\mu,\frac{E^{'}}{E}-(\sigma+\mu)\right\}$$

$$\leq\frac{E^{'}}{E}-(\sigma+\mu)\ldots.(50)$$

Integrating both sides simultaneously, we have

$$\frac{1}{t}\int_0^t \mu(B)\,ds \leq \frac{1}{t}\ln\frac{E(t)}{E(0)} - (\sigma+\mu)$$

$$\lim_{t\to\infty} ¿¿\frac{1}{t}\int_0^t \mu(B)\,ds < -(\sigma+\mu) < 0 \ldots\ldots\ldots (50)$$

Thus, by (17) we have that $Q^{¿}$ is globally asymptotically stable ∎

5.0. Conclusion

In this paper, we studied the global analysis of an SEIRS epidemic model capturing saturated incidence with treatment response. In the theoretical study of this model, we obtain the reproduction number which explains the dynamic behavior of the model showing that when $R_0<1$, there is no positive equilibrium and the disease free equilibrium is globally asymptotically stable, that is the disease dies out and when $R_0>1,$ it becomes endemic. We also determine the existence of the local and global stability of the disease free and endemic equilibria and found that the efficiency of the treatment response such as contact tracing, quarantine, case search, pool testing, advanced medical technologies, increased trained personnels, funding medical research e.t.c and strict adherence to inhibitory measures such as personal hygiene, social distancing, stay at home orders etc deployed in tackling the COVID-19 pandemic determines whether the disease will die out or become endemic. So how long COVID-19 pandemic stays with us depends on how much we are willing to take responsibility as individuals and government.

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