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AN SEIR EPIDEMIC MODEL WITH TWO INFECTIOUS PATHWAYS

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Abstract. In this research work, we present the mathematical framework of a SEIR epidemic model with two infectious pathways. The model is formulated by extending the classical SEIR mathematical model to involve two different but connected infectious states. The well posedness of the solutions of the system are shown. The basic reproduction number denoted by R_0 is computed through the next generation matrix method. The disease free equilibrium is asymptotically stable locally if $R_0 < 1$ and unstable otherwise while there exists a unique endemic equilibrium provided that $R_0 > 1$. The stability analysis for the disease free and endemic equilibrium is investigated by a suitable Lyapunov function globally. The effect of the parameters of the model on the basic reproduction number is measured by sensitivity analysis. Optimal control characterization analysis is also discussed. Some theoretical results obtained are also augmented through numerical simulation.

Keywords: Lyapunov function; equilibrium; basic reproduction number; stability; optimal control.

2020 AMS Subject Classification: 92B05.

1. INTRODUCTION

The SEIR epidemic model consists of four segments namely, susceptible segment (S), exposed segment (E), Infectious segment (I) and Recovered segment (R). There have been many extensions to the classical SEIR model. The global analysis of a variable population dynamics

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with SEIR model was investigated by Sun and Hsieh [1]. Likewise, Wachira et al.[2] extended the SEIR dymanics with vaccination to model COVID-19 with major focus on sensitivity and optimal control analysis.

Infectivity can vary over time especially for diseases that have a long progression rate. TB for example can have latent stage, active stage and drug resistance stage. Guo et al.[3] proposed a framework of multistage mathematical dynamics which permit possible amelioration side by side with deterioration among the infected segments. An important features of the model is the possibility of forward and backward progression in the infected class with certain transfer rates. The key result showed that the model dynamics is completely determined by the basic reproduction number. Korobeinikov [4] established the global properties of SIR and SEIR epidemic models comprising of parallel multiple infectious segments. He assumed the scenario where a percentage of the individuals infected are treated while the remaining undetected and untreated remains. Melesse and Gumel [5] established the global asymptotic properties of an SEIRS epidemic model with various infectious segments using a standard incidence to analyze the behaviour of an infectious disease by a suitable Lyapunov function. The dynamics was found to possess a disease free equilibrium that is asymptotically stable globally if the basic reproduction number is below one and when the basic reproduction number is above one, the endemic equilibrium is asymptotically stable globally. Otunuga and Ogunsolu[6] studied a stochastic STEIR model with various infection segments and treatment segments to investigate how recovery, treatment and transmission rates are affected by external fluctuation and treatments. Sangotola and Sangoyinka [7] investigated the dynamics of a combined SIS and SIR dynamics with two infection stages. Rifanti [8] worked on the behaviour of the spread of an infection using SIS transmission dynamics with two infectious stages.

The mathematical framework of an extended SEIR model is discussed in this research work to include two infectious pathways with connectivity and varying infectivity between the stages.

2. Model Formulation

The system is partitioned into five segments: S represents the individuals that are susceptible; E represents exposed individuals; I_1 represents the first infectious state, I_2 represents the second infectious state and R represents the recovered state. It is assumed that the second infectious

state is more advanced than the first infectious state. The susceptible population is recruited at rate Λ. It is due to interaction with I_1 and I_2 classes at rate β which leads to a corresponding increase in the exposed class. The parameter $k \leq 1$ is a modification factor which is responsible for the reduced chance of the individuals in the first infectious state to infect the susceptible population when compared to the individuals in the second infectious state. There is a reduction in E class due to migration to I_1 class at rate α. The I_1 class is reduced by effective treatment at rate γ_1, progression to I_2 which can be due to drug resistance, mutation or any other causes at rate r and death caused by the infection at rate δ_1. The I_2 class is also reduced by extensive treatment at rate γ_2 and disease induced death at rate δ_2. Every compartment is reduced at rate μ by natural death. The differential equations below is used to describe the dynamical system described.

$$\frac{dS}{dt} = \Lambda - \beta S(kI_1 + I_2) - \mu S. \tag{1}$$

$$\frac{dE}{dt} = \beta S(kI_1 + I_2) - (\alpha + \mu)E. \tag{2}$$

$$\frac{dI_1}{dt} = \alpha E - (r + \gamma_1 + \delta_1 + \mu)I_1. \tag{3}$$

$$\frac{dI_2}{dt} = rI_1 - (\gamma_2 + \delta_2 + \mu)I_2. \tag{4}$$

$$\frac{dR}{dt} = \gamma_1 I_1 + \gamma_2 I_2 - \mu R. \tag{5}$$

with initial conditions: $S(0) \geq 0, E(0) \geq 0, I_1(0) \geq 0, I_2(0) \geq 0, R(0) \geq 0$.

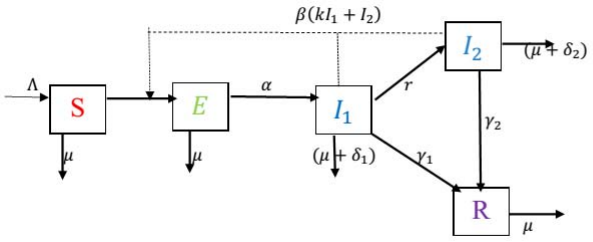

FIGURE 1. Flow diagram of the model

TABLE 1. Summary of parameters used in model $(1)-(5)$

Definition	Parameters
Rate of Recruitment into the susceptible section	Λ
Rate of transmission	β
Rate of death from other natural causes	μ
Rate of progression from E to I_1 stage	α
Rate of progression from I_1 to I_2 stage	r
Modification factor	k
Effective treatment rate from I_1 class	γ_1
Effective treatment rate from I_2 class	γ_2
Infection death rate arising from the I_1 class	δ_1
Infection death rate arising from the I_2 class	δ_2

3. MAIN RESULTS

3.1. Invariant Region. This is done by obtaining the region Ω where the trajectory of the model is bounded and for any time $t \geq 0$, any trajectory that starts in Ω remains in Ω.

Theorem 3.1: (Invariant region). All feasible trajectory of the dynamics $(1)-(5)$ enters the region Ω defined by $\{S(t),E(t),I_1(t),I_2(t),R(t) \in \mathbb{R}^5_+ : N(0) \leq N(t) \leq \frac{\Lambda}{\mu}\}$ with initial values: $S(0) \geq 0, E(0) \geq 0, I_1(0) \geq 0, I_2(0) \geq 0, R(0) \geq 0$ and are bounded.

Proof: The total individuals in the system $S(t)+E(t)+I_1(t)+I_2(t)+R(t) = N(t)$. Hence,

$$\frac{dN}{dt} = \Lambda - \mu N - \delta_1 I_1 - \delta_2 I_2. \tag{6}$$

equation (6) thus becomes:

$$\frac{dN}{dt} \leq \Lambda - \mu N. \tag{7}$$

Solving equation (7) gives:

$$0 \leq N(t) \leq N(0)e^{-\mu t} + \frac{\Lambda}{\mu}(1-e^{-\mu t})$$

If $N(0) \leq \frac{\Lambda}{\mu}$ then $N(t) \leq \frac{\Lambda}{\mu}$ as $t \to \infty$. Hence,

$$N(0) \leq N(t) \leq \frac{\Lambda}{\mu} \tag{8}$$

Hence, the feasible solution set of the system is trapped within the region Ω. Thus, the model under consideration is epidemiologically and mathematically well posed. The behaviour of the system can therefore be rigorously examined within the positive invariant set Ω.

3.2. Positivity of Solution. The initial condition of $(1)-(5)$ is nonnegative since population cannot be negative and the positivity of solution of the system shall be established.

Theorem 3.2: (Positivity of Solution). Given the region Ω defined by $\{S(t),E(t),I_1(t),I_2(t),R(t) \in \mathbb{R}^5_+\}$ with initial conditions: $S(0) \geq 0, E(0) \geq 0, I_1(0) \geq 0, I_2(0) \geq 0, R(0) \geq 0$; the solutions $S(t),E(t),I_1(t),I_2(t),R(t)$ of $(1)-(5)$ are positive for $t \geq 0$.

Proof: Equation (1) can be expressed as

$$\frac{dS}{dt} = \Lambda - \beta S(kI_1 + I_2) - \mu S \geq -\beta S(kI_1 + I_2) - \mu S$$

$$\frac{dS}{dt} \geq -[\beta(kI_1 + I_2) - \mu]S$$

$$\frac{dS}{S} \geq -[\beta(kI_1 + I_2) - \mu]dt$$

$$\int \frac{dS}{S} \geq -\int [\beta(kI_1 + I_2) - \mu]dt$$

Applying separation of variables method gives

$$S(t) \geq S(0)e^{-[\int_0^t P d\tau + \mu t]} \geq 0$$

where $P = \beta(kI_1 + I_2)$.

Applying similar procedure helps to establish that $E(t) \geq 0, I_1(t) \geq 0, I_2(t) \geq 0, R(t) \geq 0$, Hence, positivity of the model solution is established.

3.3. Disease-free equilibrium. The disease-free equilibrium of the system (1)-(5) is obtained by setting equations (1)-(5) to zero with the condition that $I_1 = I_2 = 0$. It is given by

$$\pi_0 = (S^0, E^0, I_1^0, I_2^0, R^0) = (\frac{\Lambda}{\mu}, 0, 0, 0, 0). \tag{9}$$

3.3.1. *Basic Reproduction number.* It is computed by using the Next-Generation Matrix approach by [9]. The notation $\mathscr{F}$ and $\mathscr{V}$ are used to represent the new infection and outflow terms respectively.

$$\mathscr{F} = \begin{pmatrix} \beta S(kI_1 + I_2) \\ 0 \\ 0 \end{pmatrix}$$

$$\mathscr{V} = \begin{pmatrix} (\alpha+\mu)E \\ -\alpha E + (r+\gamma_1+\delta_1+\mu)I_1 \\ -rI_1 + (\gamma_2+\delta_2+\mu)I_2 \end{pmatrix}$$

F and V below are linearized matrices computed at the disease-free equilibrium from above.

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

while

$$V = \begin{pmatrix} (\alpha+\mu) & 0 & 0 \\ -\alpha & (r+\gamma_1+\delta_1+\mu) & 0 \\ 0 & -r & (\gamma_2+\delta_2+\mu) \end{pmatrix}$$

The basic reproduction number R_0 is defined by $\rho(FV^{-1})$ where ρ represents the spectral radius. Thus,

$$R_0 = \frac{\alpha\beta\Lambda[r+k(\gamma_2+\delta_2+\mu)]}{\mu(\alpha+\mu)(r+\gamma_1+\delta_1+\mu)(\gamma_2+\delta_2+\mu)}. \tag{10}$$

3.3.2. *Local stability of disease-free equilibrium.* The local stability of the disease-free equilibrium is examined in this section.

Theorem 3.3: (Local stability of disease-free equilibrium). The disease-free equilibrium for the system $(1)-(5)$ is locally asymptotically stable if $R_0 < 1$ and unstable otherwise.

Proof: We evaluate the Jacobian matrix of $(1)-(5)$ at the disease-free equilibrium π_0 as given

below:

$$J(\pi_0) = \begin{pmatrix} -\mu & 0 & \frac{-\beta k\Lambda}{\mu} & \frac{-\beta\Lambda}{\mu} & 0 \\ 0 & -(\alpha+\mu) & \frac{\beta k\Lambda}{\mu} & \frac{\beta\Lambda}{\mu} & 0 \\ 0 & \alpha & -(r+\gamma_1+\delta_1+\mu) & 0 & 0 \\ 0 & 0 & r & -(\gamma_2+\delta_2+\mu) & 0 \\ 0 & 0 & \gamma_1 & \gamma_2 & -\mu \end{pmatrix}$$

$-\mu$ and $-\mu$ are obviously part of the eigenvalues of the matrix above. The other eigenvalues can be derived from the matrix given below.

$$J(\pi_0) = \begin{pmatrix} -(\alpha+\mu) & \frac{\beta k\Lambda}{\mu} & \frac{\beta\Lambda}{\mu} \\ \alpha & -(r+\gamma_1+\delta_1+\mu) & 0 \\ 0 & r & -(\gamma_2+\delta_2+\mu) \end{pmatrix}$$

The other eigenvalues can be obtained from the sub matrix and its characteristic equation can be expressed as:

$$p(\lambda) = \lambda^3 + c_1\lambda^2 + c_2\lambda + c_3 = 0$$

where

$c_1 = (\alpha+\mu)+(r+\gamma_1+\delta_1+\mu)+(\gamma_2+\delta_2+\mu)$

$c_2 = (\alpha+\mu)(r+\gamma_1+\delta_1+\mu)+(\alpha+\mu)(\gamma_2+\delta_2+\mu)+(r+\gamma_1+\delta_1+\mu)(\gamma_2+\delta_2+\mu)-\frac{\alpha k\beta\Lambda}{\mu}$

$c_3 = (\alpha+\mu)(r+\gamma_1+\delta_1+\mu)(\gamma_2+\delta_2+\mu)(1-R_0)$

Applying Routh Hurwitz criterion gives $c_1 > 0, c_2 > 0$, and $c_1c_2 > c_3$ provided that $R_0 < 1$. Hence, the zeros of the characteristic equation have a negative real part which confirms the local asymptotical stability of the disease-free equilibrium point if $R_0 < 1$.

3.4. Endemic equilibrium. It occurs when the disease cannot be eradicated in the population. It is obtained by setting $(1)-(5)$ to zero.

Theorem 3.4: (Existence of endemic equilibrium). The model $(1)-(5)$ has an endemic equilibrium when $R_0 > 1$.

Proof: Let $E_e^* = (S^*, E^*, I_1^*, I_2^*, R^*)$ be a non-trivial equilibrium of the model $(1)-(5)$. The

model $(1)-(5)$ thus have an endemic equilibrium and it is given by

$$\begin{aligned}
S^* &= \frac{\Lambda}{\mu R_0}. \\
E^* &= \frac{\Lambda(R_0-1)}{R_0(\alpha+\mu)}. \\
I_1^* &= \frac{\alpha\Lambda(R_0-1)}{R_0(\alpha+\mu)(r+\gamma_1+\delta_1+\mu)}. \\
I_2^* &= \frac{r\alpha\Lambda(R_0-1)}{R_0(\alpha+\mu)(\gamma_2+\delta_2+\mu)(r+\gamma_1+\delta_1+\mu)}. \\
R^* &= \frac{1}{\mu}\left(\frac{\alpha\Lambda[\gamma_1(\gamma_2+\delta_2+\mu)+r\gamma_2](R_0-1)}{R_0(\alpha+\mu)(\gamma_2+\delta_2+\mu)(r+\gamma_1+\delta_1+\mu)}\right).
\end{aligned}$$

3.5. Global Stability. Here, we investigate the asymptotic stability property of the disease-free equilibrium globally for the epidemic model.

Theorem 3.5: (Global stability of disease-free equilibrium). The disease free equilibrium of the model $(1)-(5)$ is globally asymptotically stable if $R_0 \leq 1$.

Proof: A suitable Lyapunov function V is used to prove this theorem.

$$V = \frac{\alpha[r+k(\gamma_2+\delta_2+\mu)]}{(\alpha+\mu)(r+\gamma_1+\delta_1+\mu)}E + \frac{[r+k(\gamma_2+\delta_2+\mu)]}{(r+\gamma_1+\delta_1+\mu)}I_1 + I_2 \tag{11}$$

Differentiating both sides gives

$$\begin{aligned}
\dot{V} &= \frac{\alpha[r+k(\gamma_2+\delta_2+\mu)]}{(\alpha+\mu)(r+\gamma_1+\delta_1+\mu)}(\beta S(kI_1+I_2)-(\alpha+\mu)E) \\
&+ \frac{[r+k(\gamma_2+\delta_2+\mu)]}{(r+\gamma_1+\delta_1+\mu)}(\alpha E-(r+\gamma_1+\delta_1+\mu)I_1)+rI_1-(\gamma_2+\delta_2+\mu)I_2
\end{aligned}$$

Simplifying gives

$$\dot{V} = \frac{\alpha[r+k(\gamma_2+\delta_2+\mu)]}{(\alpha+\mu)(r+\gamma_1+\delta_1+\mu)}[\beta S(kI_1+I_2)]-(\gamma_2+\delta_2+\mu)(kI_1+I_2)$$

At $S = S_0 = \frac{\Lambda}{\mu}$

$\dot{V} \leq (\gamma_2+\delta_2+\mu)(kI_1+I_2)[R_0-1]$

Thus, $\dot{V} \leq 0$ if $R_0 \leq 1$ with equality provided that $I_1 = I_2 = 0$. Every solution with initial conditions in Ω as $t \to \infty$ approaches π_0 according to LaSalle Invariance Principle [10].

Theorem 3.6: (Global stability of endemic equilibrium). The endemic equilibrium of model $(1)-(5)$ is globally asymptotically stable if $R_0 > 1$.

Proof: Consider the following nonlinear Lyapunov function defined by:

$$\begin{aligned} V &= S - S^{**} - S^{**}\ln\left(\frac{S}{S^{**}}\right) + E - E^{**} - E^{**}\ln\left(\frac{E}{E^{**}}\right) + \frac{(\alpha+\mu)}{\alpha}\left[I_1 - I_1^{**} - I_1^{**}\ln\left(\frac{I_1}{I_1^{**}}\right)\right] \\ &\quad + \frac{(\alpha+\mu)(r+\gamma_1+\delta_1+\mu)}{r\alpha}\left[I_2 - I_2^{**} - I_2^{**}\ln\left(\frac{I_2}{I_2^{**}}\right)\right] \end{aligned}$$

$$\dot{V} = \dot{S} - \frac{S^{**}}{S}\dot{S} + \dot{E} - \frac{E^{**}}{E}\dot{E} + \frac{(\alpha+\mu)}{\alpha}\left[\dot{I}_1 - \frac{I_1^{**}}{I_1}\dot{I}_1\right] + \frac{(\alpha+\mu)(r+\gamma_1+\delta_1+\mu)}{r\alpha}\left[\dot{I}_2 - \frac{I_2^{**}}{I_2}\dot{I}_2\right]$$

$$\begin{aligned} \dot{V} &= \Lambda - \mu S - \frac{S^{**}}{S}[\Lambda - \beta S(kI_1 + I_2) - \mu S] + \frac{E^{**}}{E}[\beta S(kI_1 + I_2) - (\alpha+\mu)E] \\ &\quad - \frac{(\alpha+\mu)I_1^{**}}{\alpha I_1}[\alpha E - (r+\gamma_1+\delta_1+\mu)I_1] + \frac{(\alpha+\mu)(r+\gamma_1+\delta_1+\mu)}{r\alpha}[-(\gamma_2+\delta_2+\mu)I_2 \\ &\quad - \frac{I_2^{**}}{I_2}(rI_1 - (\alpha_2+\delta_2+\mu)I_2)] \end{aligned}$$

$$\begin{aligned} \dot{V} &= \Lambda\left(1 - \frac{S^{**}}{S}\right) - \mu S\left(1 - \frac{S^{**}}{S}\right) + \beta S^{**}(kI_1 + I_2) - \frac{E^{**}}{E}\beta S(kI_1 + I_2) + (\alpha+\mu)E^{**} \\ &\quad - \frac{(\alpha+\mu)EI_1^{**}}{I_1} + \frac{(\alpha+\mu)(r+\gamma_1+\delta_1+\mu)I_1^{**}}{\alpha} - \frac{(\alpha+\mu)(r+\gamma_1+\delta_1+\mu)(\gamma_2+\delta_2+\mu)I_2}{r\alpha} \\ &\quad - \frac{(\alpha+\mu)(r+\gamma_1+\delta_1+\mu)I_1I_2^{**}}{\alpha I_2} - \frac{(\alpha+\mu)(r+\gamma_1+\delta_1+\mu)(\gamma_2+\delta_2+\mu)I_2^{**}}{r\alpha} \end{aligned}$$

At the endemic steady state, the following relations obtained from model $(1)-(5)$ hold:

$$\Lambda = \beta S^{**}(kI_1^{**} + I_2^{**}) + \mu S^{**}$$

$$(\alpha+\mu) = \frac{\beta(kI_1^{**} + I_2^{**})}{E^{**}}$$

$$(r+\gamma_1+\delta_1+\mu) = \frac{\alpha E^{**}}{I_1^{**}}$$

$$(\gamma_2+\delta_2+\mu) = \frac{rI_1^{**}}{I_2^{**}}$$

Substituting the above relations into $\dot{V}$ gives

$$\begin{aligned} \dot{V} &= [\beta S^{**}(kI_1^{**} + I_2^{**} + \mu S^{**}]\left(1 - \frac{S^{**}}{S}\right) - \mu S\left(1 - \frac{S^{**}}{S}\right) + \beta S^{**}(kI_1 + I_2) \\ &\quad + \frac{\beta S(kI_1 + I_2)E^{**}}{E} + \beta S^{**}(kI_1^{**} + I_2^{**}) - \frac{\beta S^{**}(kI_1^{**} + I_2^{**})EI_1^{**}}{E^{**}I_1} + \beta S^{**}(kI_1^{**} + I_2^{**}) \end{aligned}$$

$$-\frac{\beta S^{**}(kI_1^{**}+I_2^{**})I_2}{I_2^{**}}-\frac{\beta S^{**}(kI_1^{**}+I_2^{**})I_1I_2^{**}}{I_1^{**}I_2}+\beta S^{**}(kI_1^{**}+I_2^{**})$$

Adding and Subtracting $\beta S^{**}(kI_1^{**}+I_2^{**}), \frac{\beta S(kI_1^{**}+I_2^{**})E^{**}}{E}$ and simplifying gives

$$\begin{aligned}\dot{V} &= \mu S^{**}\left(2-\frac{S^{**}}{S}-\frac{S}{S^{**}}\right)+\beta S^{**}(kI_1^{**}+I_2^{**})\left(5-\frac{S^{**}}{S}-\frac{I_1I_2^{**}}{I_1^{**}I_2}-\frac{I_2}{I_2^{**}}-\frac{EI_1^{**}}{E^{**}I_1}-\frac{SE^{**}}{S^{**}E}\right)\\ &\quad -\beta S^{**}(kI_1^{**}+I_2^{**})\left(1-\frac{SE^{**}}{S^{**}E}\right)\left(1-\frac{kI_1+I_2}{kI_1^{**}+I_2^{**}}\right)\end{aligned}$$

Since arithmetic mean is less than or equal to geometric mean, we have

$$\left(2-\frac{S^{**}}{S}-\frac{S}{S^{**}}\right)\leq 0$$

$$\left(3-\frac{S^{**}}{S}-\frac{SI_1^{**}I_2}{S^{**}I_1I_2^{**}}\right)\leq 0.$$

Also,
$\left(1-\frac{SE^{**}}{S^{**}E}\right)\left(1-\frac{kI_1+I_2}{kI_1^{**}+I_2^{**}}\right)\geq 0$ provided that $\left(1-\frac{SE^{**}}{S^{**}E}\right)\geq 0$ and $\left(1-\frac{kI_1+I_2}{kI_1^{**}+I_2^{**}}\right)\geq 0$ or $\left(1-\frac{SE^{**}}{S^{**}E}\right)\leq 0$ and $\left(1-\frac{kI_1+I_2}{kI_1^{**}+I_2^{**}}\right)\leq 0$.
Thus, $\dot{V}\leq 0$. Equality holds provided that $S=S^{**},E=E^{**},I_1=I_1^{**},I_2=I_2^{**}$. Hence, the largest compact invariant subset of the set where $\dot{V}=0$ is the singleton $(S,E,I_1,I_2)=(S^{**},E^{**},I_1^{**},I_2^{**})$. It follows from LaSalle Invariance Principle [10], that all trajectory of the system $(1)-(5)$ approaches the associated unique endemic equilibria of the model for $R_0>1$.

3.6. Sensitivity Analysis. Sensitivity analysis on basic parameters is examined in order to identify how the parameters contribute to the basic reproduction number. If a parameter p depends on X, the normalised forward sensitivity index is defined as follows:

$$\Upsilon_p^X=\frac{\partial X}{\partial p}\times\frac{p}{X}$$

$\Upsilon_\beta^{R_0}=1$ means that there will be a 10% increase in the basic reproduction number if the transmission rate is increased by 10%. The sign of the normalized sensitivity analysis indicated if there will be an increase or decrease in the basic reproduction number as the parameter gets varied; whereas the magnitude indicates the importance of the parameter relatively. The negative sign shows an indirect relation while positive sign shows a direct relation. Below are the sensitivity indices with respect to some parameters in the model and the basic reproduction number.

TABLE 2. Sensitivity index

parameter	$\Upsilon^{R_0}_{parameter}$
β	1
Λ	1
α	$\frac{\mu}{(\alpha+\mu)}$
r	$\frac{r[(\gamma_1+\delta_1+\mu)-k(\gamma_2+\delta_2+\mu)]}{(r+\gamma_1+\delta_1+\mu)[r+k(\gamma_2+\delta_2+\mu)]}$
γ_2	$\frac{-r\gamma_2}{(\gamma_2+\delta_2+\mu)[r+k(\gamma_2+\delta_2+\mu)]}$
δ_2	$\frac{-r\delta_2}{(\gamma_2+\delta_2+\mu)[r+k(\gamma_2+\delta_2+\mu)]}$
γ_1	$\frac{-\gamma_1}{(r+\gamma_1+\delta_1+\mu)}$
δ_1	$\frac{-\delta_1}{(r+\gamma_1+\delta_1+\mu)}$
k	$\frac{k(\gamma_2+\delta_2+\mu)}{r+k(\gamma_2+\delta_2+\mu)}$

4. OPTIMAL CONTROL MODEL ANALYSIS

The analysis of optimal control is done by introducing two functions $u_1(t)$ and $u_2(t)$ which serves as control into system $(1)-(5)$. The aim of the first control is a preventive control to reduce the contact among the susceptible and two infectious classes while the second control is a treatment control on infectious individuals. Thus the system $(1)-(5)$ becomes

$$\frac{dS}{dt} = \Lambda - \beta S(1-u_1)(kI_1+I_2) - \mu S. \tag{12}$$

$$\frac{dE}{dt} = \beta S(1-u_1)(kI_1+I_1) - (\alpha+\mu)E. \tag{13}$$

$$\frac{dI_1}{dt} = \alpha E - (r+u_2\gamma_1+\delta_1+\mu)I_1. \tag{14}$$

$$\frac{dI_2}{dt} = rI_1 - (u_2\gamma_2+\delta_2+\mu)I_2. \tag{15}$$

$$\frac{dR}{dt} = u_2\gamma_1 I_1 + u_2\gamma_2 I_2 - \mu R. \tag{16}$$

The relevant optimization problem which involves obtaining the best strategy at minimum cost that minimizes the population within the exposed and infectious classes. This minimization problem can be solved by execution of controls $u_1(t)$ and $u_2(t)$ within the time horizon [0, T]. The objective functional is described as

$$J(u_1,u_2) = \int_0^T (lE + mI_1 + nI_2 + n_1u_1^2 + n_2u_2^2)dt \tag{17}$$

T denotes the final time and parameters l,m,n,n_1,n_2 are positive weights to balance the factors. We consider the state system $(12)-(16)$ with the set of admissible control functions

$$\mathscr{U} = \left\{u_1,u_2 \in L^1(0,T) \mid 0 \le u_1(t),u_2(t) \le 1 \forall t \in [0,T]\right\} \tag{18}$$

Thus, an optimal control u_1^*,u_2^* is obtained such that

$$J((u_1^*,u_2^*) = min\left\{J(u_1,u_2) : (u_1,u_2) \in \mathscr{U}\right\} \tag{19}$$

Theorem 4.1: Problems $(12)-(19)$ with initial values
$S(0),E(0),I_1(0),I_2(0),R(0)$ and final fixed time T admits a unique optimal solution $(S^*(t),E^*(t),I_1^*(t),I_2^*(t),R^*(t))$ with an associated optimal pair (u_1^*,u_2^*) on $[0,T]$.

Proof: we define our Lagrangian as follows.

$$\begin{aligned} H &= lE + mI_1 + nI_2 + n_1u_1^2 + n_2u_2^2 + \lambda_1\left[\Lambda - \beta S(1-u_1)(kI_1+I_2) - \mu S\right] \\ &\quad + \lambda_2\left[\beta S(1-u_1)(kI_1+I_2) - (\alpha+\mu)E\right] + \lambda_3\left[\alpha E - (r+u_2\gamma_1+\delta_1+\mu)I_1\right] \\ &\quad + \lambda_4\left[rI_1 - (u_2\gamma_2+\delta_2+\mu)I_2\right] + \lambda_5\left[u_2\gamma_1 I_1 + u_2\gamma_2 I_2 - \mu R\right] \end{aligned}$$

where $\lambda_1, \lambda_2, \lambda_3, \lambda_4, \lambda_5$ describes the associated adjoint functions with the respective states. There exists adjoint functions satisfying

$$\begin{aligned}
\frac{d\lambda_1}{dt} &= \beta(1-u_1)(kI_1+I_2)(\lambda_1-\lambda_2)+\lambda_1\mu \\
\frac{d\lambda_2}{dt} &= -l+(\alpha+\mu)\lambda_2-\alpha\lambda_3 \\
\frac{d\lambda_3}{dt} &= -m+\beta kS(1-u_1)(\lambda_1-\lambda_2)+(r+u_2\gamma_1+\delta_1+\mu)\lambda_3 \\
&\quad -r\lambda_4-u_2\gamma_1\lambda_5 \\
\frac{d\lambda_4}{dt} &= -n+\beta S(1-u_1)(\lambda_1-\lambda_2)+(u_2\gamma_2+\delta_2+\mu)\lambda_4-u_2\gamma_2\lambda_5 \\
\frac{d\lambda_5}{dt} &= \mu\lambda_5
\end{aligned}$$

with the terminal conditions

$$\lambda_1(T)=0, \lambda_2(T)=0, \lambda_3(T)=0, \lambda_4(T)=0, \lambda_5(T)=0 \tag{20}$$

Furthermore, u_1^*, u_2^* are represented by

$$\begin{aligned}
u_1^* &= min\left(u_{1max}, max\left(0, \frac{\beta S(kI_1+I_2)(\lambda_2-\lambda_1)}{2n_1}\right)\right) \\
u_2^* &= min\left(u_{2max}, max\left(0, \frac{\gamma_1 I_1\lambda_3+\gamma_2 I_2\lambda_4-(\gamma_1 I_1+\gamma_2 I_2)\lambda_5}{2n_2}\right)\right)
\end{aligned}$$

5. NUMERICAL SIMULATION

The behaviour of model $(1)-(5)$ is observed for some set of feasible set of hypothetical parameters values. The objectives are to determine what happens by varying some parameters on the solution of the model relative to the basic reproduction number.

Figure 2 describes the solution of the model such that $S(0)=300, E(0)=150, I_1(0)=100, I_2(0)=50, R(0)=0$. The following hypothetical parameter values are used: $\Lambda=0.1; \beta=0.3; \mu=0.025; \alpha=0.08; \gamma_1=0.019; \gamma_2=0.057; \delta_1=0.05; \delta_2=0.2; k=0.5; r=0.38$ and its basic reproduction number is $R_0=4.3965>1$.

Figure 3 describes the solution of the model such that $S(0)=300, E(0)=150, I_1(0)=100, I_2(0)=50, R(0)=0$. The following parameter values are used: $\Lambda=0.05; \beta=0.15; \mu=$

$0.025; \alpha = 0.08; \gamma_1 = 0.09; \gamma_2 = 0.27; \delta_1 = 0.05; \delta_2 = 0.2; k = 0.5; r = 0.38$ and its basic reproduction number is $R_0 = 0.6497 < 1$.

Two simulation cases were performed to augment the theoretical results by keeping some parameters constant and varying others, and it is observed to be in good agreement. Furthermore, the simulation result shows that a decrease in contact rate and transmission rate and increase in recovery rate reduces the basic reproduction number which agrees with the sensitivity analysis. Similarly, the simulation analysis also reveals the possibility of a low basic reproduction number leading to a higher recovery rate.

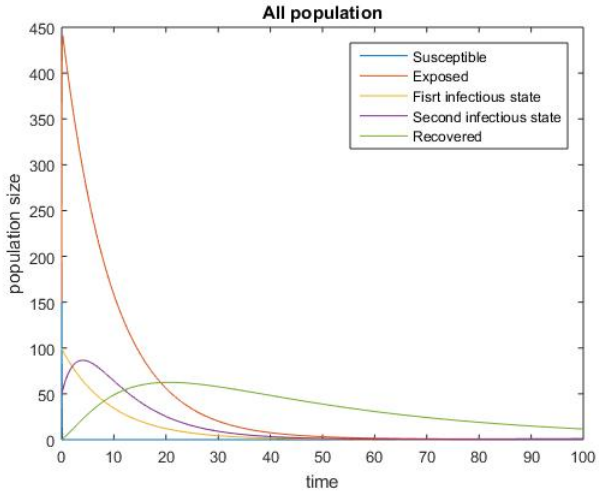

FIGURE 2. Numerical simulation

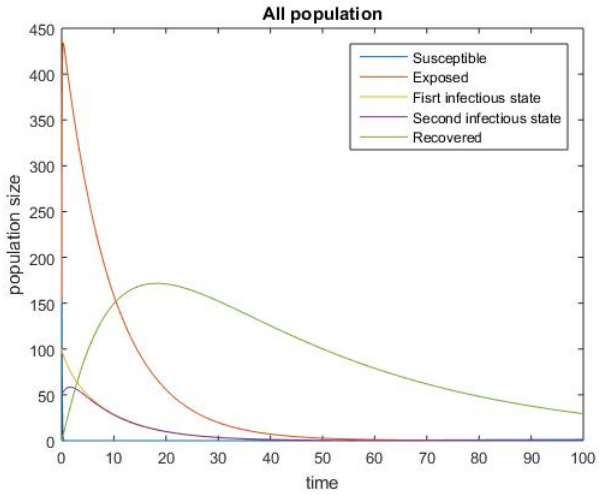

FIGURE 3. Numerical simulation

6. CONCLUSION

The mathematical framework of a two infectious pathways SEIR model in a population is formulated. The disease free, endemic equilibrium and the basic reproduction number are derived and analysed. The disease-free equilibrium point of the system is found to be locally asymptotically stable if $R_0 < 1$ and unstable if $R_0 > 1$. A unique endemic equilibrium of the system also exists provided that $R_0 > 1$. Furthermore, the disease-free equilibrium of the model is found to be asymptotically stable globally when $R_0 \leq 1$ and under certain conditions, the endemic equilibrium is asymptotically stable globally when $R_0 > 1$.

The parameters which contribute to the basic reproduction number are identified through sensitivity analysis. Recruitment term, transmission rate, progression rate and modification factor have a positive influence on the basic reproduction number while death due to infection and treatment rate have a negative influence on the basic reproduction number. Progression rate from first infectious state to the second infectious state can both have positive and negative impact under certain conditions.

The Pontryagin maximum principle is also applied to investigate and analyze the optimal control with preventive and treatment control efforts. Some theoretical results were also validated through numerical simulations.

CONFLICT OF INTERESTS

The authors declare that there is no conflict of interests.

REFERENCES

[1] C. Sun, Y.H. Hsieh, Global analysis of an SEIR model with varying population size and vaccination, Appl. Math. Model. 34 (2010), 2685–2697. https://doi.org/10.1016/j.apm.2009.12.005.

[2] C.M. Wachira, G.O. Lawi, L.O. Omondi, Sensitivity and optimal control analysis of an extended SEIR COVID-19 mathematical model, J. Math. 2022 (2022), 1476607. https://doi.org/10.1155/2022/1476607.

[3] H. Guo, M.Y. Li, Z. Shuai, Global dynamics of a general class of multistage models for infectious diseases, SIAM J. Appl. Math. 72 (2012), 261–279. https://doi.org/10.1137/110827028.

[4] A. Korobeinikov, Global properties of SIR and SEIR epidemic models with multiple parallel infectious stages, Bull. Math. Biol. 71 (2008), 75–83. https://doi.org/10.1007/s11538-008-9352-z.

[5] D.Y. Melesse, A.B. Gumel, Global asymptotic properties of an SEIRS model with multiple infectious stages, J. Math. Anal. Appl. 366 (2010), 202–217. https://doi.org/10.1016/j.jmaa.2009.12.041.

[6] O.M. Otunuga, M.O. Ogunsolu, Qualitative analysis of a stochastic SEITR epidemic model with multiple stages of infection and treatment, Infect. Dis. Model. 5 (2020), 61–90. https://doi.org/10.1016/j.idm.2019.12.003.

[7] A.O. Sangotola, L.J. Sangoyinka, A two infection stage model with SIS and SIR dynamics, Int. J. Math. Anal. Model. 5 (2022), 335–343.

[8] U.M. Rifanti, Dynamic model of disease spread with two infection stages, J. Phys.: Conf. Ser. 1567 (2020), 022075. https://doi.org/10.1088/1742-6596/1567/2/022075.

[9] P. van den Driessche, J. Watmough, Reproduction numbers and sub-threshold endemic equilibria for compartmental models of disease transmission, Math. Biosci. 180 (2002), 29–48. https://doi.org/10.1016/s0025-5564(02)00108-6.

[10] J.P. La Salle, The stability of dynamical systems, Regional Conference Series in Applied Mathematics, SIAM, Philadelphia, USA, (1976).

[11] P. van den Driessche, Reproduction numbers of infectious disease models, Infect. Dis. Model. 2 (2017), 288–303. https://doi.org/10.1016/j.idm.2017.06.002.