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Global Dynamics of a Discrete SEIR Epidemic Model with Treatment

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ABSTRACT: The global dynamics of a discrete SEIR epidemic model with treatment have been considered. A unique positive solution for the proposed model with the positive initial conditions is obtained. The stability analysis of the disease-free equilibrium and endemic equilibrium have been investigated. It has been proved that the DFE is globally asymptotically stable when the basic reproduction number $\mathcal{R}_0 \leq 1$. The proposed model has a unique endemic equilibrium that is globally asymptotically stable whenever $\tilde{\mathcal{R}}_0 > 1$. A numerical simulation illustrates the theoretical results.

Key Words: Discrete model, treatment, backward difference, equilibria, global stability.

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1. Introduction

Every year, people suffer from infectious diseases. After the susceptible individuals are infected, the disease incubates in the susceptibles for a time period; then, it becomes exposed and hence infectious. To control the spreading of the emerging and re-emerging disease such as Hepatitis, Cholera, HIV, SARS, Ebola, Yellow fever, Smallpox, and MERS, treatment has been imposed [1,3,4,5,6,7,8,10,13,15,16,18,19, 20,21,22,23,24,25,26]. DarAssi et al. [5] have studied a delayed SEIR epidemic model with pulse vaccination and treatment. They proved that the infection-free periodic solution is globally attractive when the reproduction number $\Re^* < 1$ and the disease is permanent when $\Re_* > 1$.

Two types of dynamical epidemic models have been studied: the continuous-time models described by differential equations and the discrete-time models described by difference equations. Recently, more attention has been paid to the epidemic discrete-time models. The reasons are that most of the collected disease data come in the form of a discrete point which is more convenient and accurate to describe the disease than the continuous models. Moreover, discrete models have more dynamical behaviors (see [27] and the references therein).

Numerous studies have been made to study the discrete epidemic models. X. Fan et al. [12] have considered a discrete SEIRS epidemic model with general nonlinear incidence. The authors investigated the positivity and boundedness of the solutions with positive initial conditions. They obtained that when

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the reproduction number $\mathcal{R}_0 \leq 1$, the disease is eradicated, and the model has a unique globally attractive endemic equilibrium when $\mathcal{R}_0 > 1$. M. Safi et al. [25] have studied the global dynamics of a discrete quarantine/isolation model. L. Wang et al. [27] have considered a class of discrete SIRS epidemic models with disease courses. The authors computed the basic reproduction number \mathcal{R}_0 and proved that the disease-free equilibrium is globally attractive when $\mathcal{R}_0 < 1$ and the disease is permanent when $\mathcal{R}_0 > 1$. Y. Wang et al. [28] have introduced Lyapunov functions for a class of discrete SIRS epidemic models with nonlinear incidence rates and varying population sizes. The authors established the sufficient and necessary conditions for the global asymptotic stability of the disease-free equilibrium and endemic equilibrium with general nonlinear incidence rate $\beta S g(I)$ and different death rates.

The continuous SEIR epidemic model with treatment [5] is given by the following differential equations:

$$\begin{cases} \dot{S}(t) &= \Pi - \lambda(t) \, S(t) - \nu \, S(t), \\ \dot{E}(t) &= \lambda(t) \, S(t) - (\kappa + \nu) \, E(t), \\ \dot{I}(t) &= \kappa \, E(t) - (\nu + \sigma + \delta_1 + \gamma_1) \, I(t), \\ \dot{Y}(t) &= \sigma \, I(t) - (\nu + \psi + \delta_2 + \gamma_2) \, Y(t), \\ \dot{W}(t) &= \psi \, Y(t) - (\nu + \delta_3 + \gamma_3) \, W(t), \\ \dot{R}(t) &= \gamma_1 \, I(t) + \gamma_2 \, Y(t) + \gamma_3 \, W(t) - \nu \, R(t), \end{cases}$$
(1.1)

where $\lambda(t) = \frac{\beta I(t)}{N(t)}$, Π is the recruitment rate of susceptible corresponding to births and immigration, ν is the natural death rate, β is the contact rate, κ is the progression rate from exposed to infectious class, σ is the treatment rate of infectious individuals, ψ is the treatment failure rate, γ_i (i = 1, 2, 3) is the recovery rate for untreated infectious, treated and fail treated individuals, respectively. δ_i (i = 1, 2, 3) is the disease-induced death rate of infectious, treated and fail treated individuals, respectively. The total population size is given by the equation N(t) = S(t) + E(t) + I(t) + Y(t) + W(t) + R(t).

In this work, we considered the discrete version of the model (1.1). This paper is organized as follows: A discrete SEIR epidemic model with treatment is considered in section (2). In section (3), we presented the fundamental properties of the discrete model. The stability analysis of the disease-free equilibrium is carried out in section (4). The existence and stability analysis of the endemic equilibrium point is conducted in section (5). In section (6), numerical simulations are provided to illustrate the obtained results and conclude the results.

2. Model Formulation

A discrete epidemic SEIR model with treatment is considered. The backward difference scheme is used to discretize the continuous model (1.1). This model is called the SEIYWR model. The population N(t) is divided into the following compartments: S(t) is the susceptible individuals, E(t) is the exposed individuals, I(t) is the infectious individuals but not treated, Y(t) is the treated individuals, W(t) is the treated individuals who failed treatment, and R(t) is the recovered individuals. Hence, N(t) =S(t) + E(t) + I(t) + Y(t) + W(t) + R(t). Therefore, the SEIYWR model is governed by the following difference equations:

$$\begin{cases} S(t+1) - S(t) &= \Pi - \lambda(t+1) S(t+1) - \nu S(t+1), \\ E(t+1) - E(t) &= \lambda(t+1) S(t+1) - k_1 E(t+1), \\ I(t+1) - I(t) &= \kappa E(t+1) - k_2 I(t+1), \\ Y(t+1) - Y(t) &= \sigma I(t+1) - k_3 Y(t+1), \\ W(t+1) - W(t) &= \psi Y(t+1) - k_4 W(t+1), \\ R(t+1) - R(t) &= \gamma_1 I(t+1) + \gamma_2 Y(t+1) + \gamma_3 W(t+1) - \nu R(t+1), \end{cases}$$
(2.1)

where $\lambda(t+1) = \frac{\beta I(t+1)}{N(t+1)}$, $k_1 = \kappa + \nu$, $k_2 = \nu + \sigma + \delta_1 + \gamma_1$, $k_3 = \nu + \psi + \delta_2 + \gamma_2$, $k_4 = \nu + \delta_3 + \gamma_3$, Π is the recruitment rate of susceptible corresponding to births and immigration, ν is the natural death rate,

 β is the contact rate, κ is the progression rate from exposed to infectious class σ is the treatment rate of infectious individuals, ψ is the treatment failure rate, γ_i (i = 1, 2, 3) is the recovery rate for untreated infectious, treated and fail treated individuals, respectively. δ_i (i = 1, 2, 3) is the disease-induced death rate of infectious, treated and fail treated individuals, respectively. Subject to the following initial conditions:

$$S(0) > 0, E(0) > 0, I(0) > 0, Y(0) > 0, W(0) > 0, R(0) > 0.$$
(2.2)

3. Fundamental properties

Lemma 3.1. The model (2.1) with initial conditions (2.2) has a unique positive solution $(S(t), E(t), I(t), Y(t), W(t), R(t)), \forall n = 1, 2, 3, \cdots$

We will apply the mathematical induction to prove the lemma 3.1 as follows:

Proof. Rewrite the model (2.1) as follows:

$$\begin{cases} I(t+1) &= a_1 E(t+1) + b_1, \\ Y(t+1) &= a_2 E(t+1) + b_2, \\ W(t+1) &= a_3 E(t+1) + b_3, \\ R(t+1) &= a_4 E(t+1) + b_4, \\ S(t+1) &= a_5 E(t+1) + b_5, \end{cases}$$
(3.1)

where

$$a_1 = \frac{\kappa}{1+k_2}, \quad b_1 = \frac{I(t)}{1+k_2},$$

$$a_2 = \frac{\sigma\kappa}{(1+k_2)(1+k_3)}, \quad b_2 = \frac{\sigma I(t) + (1+k_2) Y(t)}{(1+k_2)(1+k_3)},$$

$$\begin{aligned} a_3 &= \frac{\psi \sigma \kappa}{(1+k_2)(1+k_3)(1+k_4)}, \quad b_3 &= \frac{\psi \sigma I(t)}{(1+k_2)(1+k_3)(1+k_4)} + \frac{\psi Y(t)}{(1+k_3)(1+k_4)} + \frac{W(t)}{1+k_4}, \\ a_4 &= \frac{\gamma a_1 + \gamma_2 a_2 + \gamma_3 a_3}{1+\nu}, \quad b_4 &= \frac{\gamma_1 b_1 + \gamma_2 b_2 + \gamma_3 b_3 + R(t)}{1+\nu}, \\ a_5 &= -\frac{1+k_1}{1+\nu}, \quad b_5 &= \frac{\Pi + S(t) + E(t)}{1+\nu}, \end{aligned}$$

Substituting the expressions (3.1) in the second equation of model (2.1) yields

$$E(t+1) = \frac{E(t)}{1+k_1} + \frac{\beta \left[a_5 E(t+1) + b_5\right] \left[a_1 E(t+1) + b_1\right]}{\left(1+k_1\right) \left(\sum_{i=1}^5 \left[a_i E(t+1) + b_i\right] + E(t+1)\right)}$$
(3.2)

Let y = E(t+1) and define

$$f(y) = y - \frac{E(t)}{1+k_1} - \frac{\beta [a_5 y + b_5] [a_1 y + b_1]}{(1+k_1) \left(\sum_{i=1}^5 [a_i y + b_i] + y\right)} = 0$$

Since f(y) is an increasing function and $f(0) = -\frac{\beta b_5 b_1}{(1+k_1)\sum_{i=1}^5 b_i} - \frac{E(t)}{1+k_1} < 0$, then f(y) has a unique positive solution. Hence, equation (3.2) has a unique solution E(t+1) > 0 which implies that the system

(3.1) has a unique solution I(t+1) > 0, Y(t+1) > 0, W(t+1) > 0, and R(t+1) > 0.

Let x = S(t+1), then using the first equation in model (2.1) we define

$$g(x) = (1+\nu)x + \frac{\beta [a_1 E(t+1) + b_1] x}{x+L} - \Pi$$

where L = E(t+1) + I(t+1) + Y(t+1) + W(t+1) + R(t+1). Since g'(x) > 0, then g(x) is an increasing function with $g(0) = -\Pi < 0$. Thus, g(x) has a unique positive solution and therefore, there exists a unique S(t+1) > 0 which is completed the proof.

Lemma 3.2. Any solution (S(t), E(t), I(t), Y(t), W(t), R(t)) of model (2.1) with initial conditions (2.2) satisfies $\limsup_{t \to \infty} N(t) \leq \frac{\Pi}{\nu}$.

Proof. Since,

$$\begin{split} N(t+1) &= S(t+1) + I(t+1) + E(t+1) + Y(t+1) + W(t+1) + R(t+1) \\ &= \Pi - \nu \left(S(t+1) + E(t+1) + I(t+1) + Y(t+1) + W(t+1) + R(t+1) \right) \\ &- \delta_1 I(t+1) - \delta_2 Y(t+1) - \delta_3 W(t+1) + N(t) \\ &= \frac{\Pi + N(t) - \delta_1 I(t+1) - \delta_2 Y(t+1) - \delta_3 W(t+1)}{1 + \nu} \\ &\leq \frac{\Pi + N(t)}{1 + \nu}, \quad t = 0, 1, 2, \cdots \\ &= \frac{\Pi}{1 + \nu} + \frac{N(t)}{1 + \nu}. \quad \text{Upon using the iteration method we get} \\ N(t+1) &\leq \frac{\Pi}{1 + \nu} + \frac{\Pi}{(1 + \nu)^2} + \frac{\Pi}{(1 + \nu)^3} + \cdots + \frac{\Pi}{(1 + \nu)^{(t+1)}} + \frac{N(0)}{(1 + \nu)^{(t+1)}} \\ &= \frac{\Pi}{\nu} \left[1 - \frac{1}{(1 + \nu)^{(t+1)}} \right] + \frac{N(0)}{(1 + \nu)^{(t+1)}}. \end{split}$$

Hence,

$$\limsup_{t \to \infty} N(t+1) \le \limsup_{t \to \infty} N(t) \le \frac{\Pi}{\nu}$$

Therefore, the region

$$\mathcal{D} = \left\{ (S(t), E(t), I(t), Y(t), W(t), R(t)) \in \mathcal{R}^6_+ | N(t) \le \frac{\Pi}{\nu} \right\}$$

is positively invariant.

4. Disease-Free Equilibrium (DFE)

4.1. Local stability of DFE

The unique DFE of the system (2.1) is given by

$$\mathcal{E}_0 = \left(\frac{\Pi}{\nu}, 0, 0, 0, 0, 0\right)$$

To compute the basic reproduction number of model (2.1), we will apply the next generation operator method [2,9,11,14,17]. The matrix of the new infection terms, F, and the matrix of the transition terms, V, that are associated with the model (2.1) are given by:

Following [11] the basic reproduction number is denoted by $\mathcal{R}_0 = \rho(FV^{-1})$ and is given by

$$\mathcal{R}_0 = \frac{\beta \, \kappa}{k_1 \, k_2}$$

The following lemma can be proved using theorem (2) in [11].

Lemma 4.1. The DFE point $\mathcal{E}_0 = \left(\frac{\Pi}{\nu}, 0, 0, 0, 0\right)$ of model (2.1) is locally asymptotically stable (LAS) when $\mathcal{R}_0 < 1$ and unstable when $\mathcal{R}_0 > 1$.

4.2. Global stability of DFE

In this section, the global attractivity of the disease-free equilibrium of model (2.1) is investigated, and the following result is concluded.

Theorem 4.2. The DFE of the model (2.1) is globally-asymptotically stable (GAS) in \mathcal{D} whenever $\mathcal{R}_0 \leq 1$.

Proof. Consider the following Lyapunov function

$$F_1(t) = \frac{\kappa}{k_1} E(t) + I(t)$$

The backward difference of F_1 is denoted by ΔF_1 and is given by

$$\begin{split} \Delta F_1 &= F_1(t+1) - F_1(t) = \frac{\kappa}{k_1} E(t+1) + I(t+1) - \frac{\kappa}{k_1} E(t) - I(t) \\ &= \frac{\kappa}{k_1} \left[E(t+1) - E(t) \right] + I(t+1) - I(t) \\ &= \frac{\kappa}{k_1} \left[\lambda(t+1) S(t+1) - k_1 E(t+1) \right] + \kappa E(t+1) - k_2 I(t+1) \\ \text{Since} \quad \frac{S(t+1)}{N(t+1)} \leq 1 \quad \text{in} \quad \mathcal{D}, \quad \text{then} \\ F_1(t+1) - F_1(t) \leq \frac{\kappa}{k_1} \left[\beta I(t+1) - k_1 E(t+1) \right] + \kappa E(t+1) - k_2 I(t+1) \\ &= \left[\frac{\kappa \beta}{k_1} - k_2 \right] I(t+1) \\ &= k_2 \left[\frac{\kappa \beta}{k_1 k_2} - 1 \right] I(t+1) \\ &= k_2 [\mathcal{R}_0 - 1] I(t+1) \end{split}$$

Which implies that $\Delta F_1 = F_1(t+1) - F_1(t) \leq 0$ whenever $\mathcal{R}_0 \leq 1$ and $\Delta F_1 = 0$ if and only if E(t+1) = I(t+1) = Y(t+1) = W(t+1) = 0. Hence, $(E, I, Y, W) \to (0, 0, 0, 0)$ as $n \to \infty$. Upon setting E = I = Y = W = 0 in the first and last equations in model (2.1) we get $S \to \frac{\Pi}{\nu}$ and $R \to 0$ as $n \to \infty$. Thus, the maximum invariable set in $\{(S, E, I, Y, W, R) : F_1(t) = 0\}$ is a disease-free equilibrium \mathcal{E}_0 . Following the theorems of stability of difference equations (theorem 6.3 in [17]) every solution of the equations in model (2.1) with the initial conditions in \mathcal{D} approaches \mathcal{E}_0 as $n \to \infty$. Thus, the disease-free equilibrium \mathcal{E}_0 of model (2.1) is globally attractive. Hence the proof is completed. \Box

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5. Endemic Equilibria

5.1. Existence of the endemic equilibrium point EEP

Let $\mathcal{E}_1 = (S_1, E_1, I_1, Y_1, W_1, R_1)$ be an endemic equilibrium point for the model (2.1). Therefore the following lemma can be established.

Lemma 5.1. The model (2.1) has a unique endemic equilibrium point \mathcal{E}_1 , whenever $\mathcal{R}_0 > 1$.

Proof. Solving the equations of the model (2.1) at steady-state, yields

$$S_{1} = \frac{\Pi}{\lambda_{1} + \nu}, \quad E_{1} = \frac{\lambda_{1} S_{1}}{k_{1}}, \quad I_{1} = \frac{\kappa \lambda_{1} S_{1}}{k_{1} k_{2}}$$

$$Y_{1} = \frac{\sigma \kappa \lambda_{1} S_{1}}{k_{1} k_{2} k_{3}}, \quad W_{1} = \frac{\psi \sigma \kappa \lambda_{1} S_{1}}{k_{1} k_{2} k_{3} k_{4}}$$

$$R_{1} = \frac{\kappa \lambda_{1} S_{1}}{\nu k_{1} k_{2} k_{3} k_{4}} [\gamma_{1} k_{3} k_{4} + \gamma_{2} \sigma k_{4} + \gamma_{3} \sigma \psi]$$
(5.1)

where

$$\lambda_1 = \frac{\beta I_1}{N_1}$$
 and $N_1 = S_1 + E_1 + I_1 + Y_1 + W_1 + R_2$

Substituting equations (5.1) in the expression of λ_1 to get

$$\mathcal{R}_0 = 1 + \Lambda \,\lambda_1$$

where

$$\Lambda = \frac{\nu \, k_2 \, k_3 \, k_4 + \nu \kappa \, k_3 \, k_4 + \nu \kappa \sigma \, k_4 + \nu \kappa \sigma \psi + \kappa (\gamma_1 \, k_3 \, k_4 + \gamma_2 \sigma \, k_4 + \gamma_3 \sigma \psi)}{\nu \, k_1 \, k_2 \, k_3 \, k_4}$$

Hence, $\frac{\mathcal{R}_0 - 1}{\Lambda} > 0$ if and only if $\mathcal{R}_0 > 1$. This implies that $S_1, E_1, I_1, Y_1, W_1, R_1 > 0$ if and only if $\mathcal{R}_0 > 1$. \Box

5.2. Stability of the Endemic Equilibrium

In this section, we assumed that the associated disease-induced mortality is negligible *i.e* ($\delta_1 = \delta_2 = \delta_3 = 0$). To investigate the global stability of the unique endemic equilibrium point for this case, we define

$$\mathcal{D}_0 = \{ (S, E, I, Y, W, R) \in \mathcal{D} | E = I = Y = W = R = 0 \}$$

The following result is established.

Theorem 5.2. The unique endemic equilibrium point (EEP) of the model (2.1) with $\delta_1 = \delta_2 = \delta_3 = 0$ is globally asymptotically stable (GAS) in $\mathcal{D}/\mathcal{D}_0$ if

$$\left. \widetilde{\mathcal{R}}_0 = \mathcal{R}_0 \right|_{\delta_1 = \delta_2 = \delta_3 = 0} = \frac{\beta \kappa}{(\nu + \kappa)(\nu + \sigma + \gamma_1)} > 1$$

Proof. Upon setting $\delta_1 = \delta_2 = \delta_3 = 0$ in model (2.1). Define the following Lyapunov function

$$F_{2}(t) = \frac{1}{2} \left[(S(t) - S_{1}) + (E(t) - E_{1}) + (I(t) - I_{1}) + (Y(t) - Y_{1}) + (W(t) - W_{1}) + (R(t) - R_{1}) \right]^{2}$$

= $\frac{1}{2} \left[N(t) - N_{1} \right]^{2}$

The backward difference of F_2 is given by

$$\begin{split} \Delta F_2 &= F_2(t+1) - F_2(t) \\ &= \frac{1}{2} \left[N(t+1) - N_1 \right]^2 - \frac{1}{2} \left[N(t) - N_1 \right]^2 \\ &= \frac{1}{2} \left[N(t+1) - N(t) \right] \left[N(t+1) + N(t) - 2 N_1 \right] \\ &= -\frac{1}{2} \left[N(t+1) - N(t) \right]^2 + \left[N(t+1) - N_1 \right] \left[N(t+1) - N(t) \right] \\ &\leq \left[N(t+1) - N_1 \right] \left[N(t+1) - N(t) \right]. \end{split}$$

But, setting $\delta_1 = \delta_2 = \delta_3 = 0$ and adding the equations of model (2.1) yields $N(t+1) - N(t) = \Pi - \nu N(t+1)$ and $N_1 = \frac{\Pi}{\nu}$. Thus,

$$\Delta F_2 \leq [N(t+1) - N_1] [\Pi - \nu N(t+1)].$$

= [N(t+1) - N_1] [\nu N_1 - \nu N(t+1)].
= -\nu [N(t+1) - N_1]^2
\le 0

Therefore, F_2 is a Lyapunov function on $\mathcal{D}/\mathcal{D}_0$ and hence, by applying the theorem of stability of difference equations (theorem 6.3 in [17]), we obtain that every solution of the equations in model (2.1) with $\delta_1 = \delta_2 = \delta_3 = 0$ approaches the unique endemic equilibrium point as $t \to \infty$ whenever $\widetilde{\mathcal{R}}_0 > 1.e \square$

6. Discussion and Conclusion

In this section, we will investigate the numerical simulation of the proposed model (2.1). The values of the model (2.1) parameters are listed in Table 1 below.

Parameter	$\mathcal{R}_0 \leq 1$	$\Re_0 > 1$	Parameter	$\mathcal{R}_0 \leq 1$	$\mathcal{R}_0 > 1$
П	136	136	γ_1	0.00337	0.00337
ν	0.000034	0.000034	γ_2	0.00386	0.00386
κ	0.1	0.1	γ_3	0.00335	0.00335
β	0.15	0.25	δ_1	0.1	0.1
σ	0.1	0.1	δ_2	0.1	0.1
ψ	0.15	0.15	δ_3	0.1	0.1

Table 1: The values of the parameters of the model (2.1) when $\mathcal{R}_0 \leq 1$ and when $\mathcal{R}_0 > 1$.

Figure 1 (Left) shows that the disease dies out, which means that the disease-free equilibrium point of model (2.1) is globally attractive, and figure 1 (Right) shows that the disease is permanent.

Figure 2 displays that the number of cumulative cases of infection with treatment is more significant than cumulative cases without treatment.

Figure 3 depicts the relation between the basic reproduction number \mathcal{R}_0 and the birth-death rate ν for several values of the contact rate β . It shows that \mathcal{R}_0 decrease as ν increases and \mathcal{R}_0 increases as β increases.



Figure 1: The infected compartments as functions of time when $\Re_0 = 0.7372 < 1$ (Left). The infected compartments as functions of time when $\Re_0 = 1.2287 > 1$ (Right).



Figure 2: The plot of the cumulative cases of infection verses time in days with treatment (dotted line) and without treatment (solid line) when $\mathcal{R}_0 = 0.7372 < 1$ (Left). The plot of the cumulative cases of infection verses time in days with treatment (dotted line) and without treatment (solid line) when $\mathcal{R}_0 = 1.2287 > 1$ (Right).



Figure 3: The plot of the basic reproduction number as a function of the birth-death rate ν for several values of the contact rate. $\beta = 0.1$ (solid blue), $\beta = 0.15$ (solid red), $\beta = 0.2$ (dashed) and $\beta = 0.25$ (dotted).

In this paper, the global dynamics of a discrete SEIR model with treatment have been proposed and analyzed. This model is obtained from the continuous model in [5] by backward difference scheme. The

threshold conditions for the global attractivity of the DFE and the endemic equilibrium are established. It has proven that the DFE is globally asymptotically stable when $\mathcal{R}_0 \leq 1$. However, the endemic equilibrium is GAS whenever $\widetilde{\mathcal{R}}_0 > 1$. Since $\mathcal{R}_0 = \frac{\beta \kappa}{(\kappa + \nu)(\nu + \sigma + \delta_1 + \gamma_1)}$ is independent of γ_2 and γ_3 , which means that both the recovery rate of treated individuals and the recovery rate of failed treated individuals are not affecting the disease of being permanent or dying out.

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References

- 1. H. Alrabaiah, M.A. Safi, M.H. DarAssi, B. Al-Hdaibat, S. Ullah, M.A.Khan and S.A. Ali Shah (2020). Optimal control analysis of hepatitis B virus with treatment and vaccination. *Results in Physics* **19** 103599.
- R.M. Anderson and R.M. May (1982). Population Biology of Infectious Diseases. Springer-Verlag, Berlin, Heidelrberg, New York.
- 3. CT Bauch, JO Lloyd-Smith, MP Coffee (2005). Dynamically modeling SARS and other newly emerging respiratory illnesses: past, present, and future. *Epidemiology* 16: 791-801.
- 4. Y Bechah, C Capo, JL Mege, D Raoult (2008). Epidemic typhus. The Lancet infectious diseases 8: 417-426.
- M.H DarAssi, M.A. Safi and B. Al-Hdaibat (2018). A delayed SEIR epidemic model with pulse vaccination and treatment. Nonlinear Studies 25 (3): 1-16.
- M.H DarAssi, M.A. Safi and M. Ahmad (2021). Global Dynamics of a Discrete-Time MERS-Cov Model. Mathematics 9 (5): 563.
- M.H DarAssi, M.A. Safi (2021). Analysis of an SIRS epidemic model for a disease geographic spread. Nonlinear Dynamics and Systems Theory 21 (1): 56-67.
- 8. P Daszak, AA Cunningham, AD Hyat (2000). Emerging infectious diseases of wildlife–threats to biodiversity and human health. *Science* 287.
- 9. O. Diekmann, J. A. P. Heesterbeek and J. A. J. Metz (1990). On the definition and computation of the basic reproduction ratio R0 in models for infectious disease in heterogeneous population. J. Math. Biol. 28: 365-382.
- 10. O. Diekmann, JAP Heesterbeek (2000). Mathematical epidemiology of infectious diseases. Chisteter: John Wiley & Son.
- 11. P. van den Driessche and J. Watmough (2002). Reproduction numbers and subthreshold endemic equilibria for compartmental models of disease transmission. *Math. Biosci.* **180**: 29-48.
- X. Fan, L. Wang and Z. Teng (2016). Global dynamics for a class of discrete SEIRS epidemic models with general nonlinear incidence. Advances in Difference Equations 2016:123.
- 13. S Funk, M Salathé, VAA Jansen (2010). Modelling the influence of human behaviour on the spread of infectious diseases: a review. *Journal of the Royal Society* **50**: 1247- 1256.
- 14. H. W. Hethcote (2000). The mathematics of infectious diseases. SIAM Rev. 42: 599-653.

- 15. M.J. Keeling, P. Rohani (2008). Modeling infectious diseases in humans and animals. (Princeton Univ. Press.
- M.A. Khan, K. Khan, M.A. Safi and M.H. DarAssi (2020). A discrete model of TB dynamics in Khyber Pakhtunkhwa-Pakistan. CMES - Computer Modeling in Engineering and Sciences 123 (2): 777-795.
- 17. J. P. LaSalle (1976). The Stability of Dynamical Systems. CBMS-NSF Regional Conf. Ser. in Appl. Math., SIAM, Philadelphia.
- X. P. Li, Y. wang, M.A. Khan, M.Y. Alshahrani and T. Muhammad (2021). A dynamical study of SARS-COV-2: A study of third wave. *Results in Physics* 29 104705.
- X. P. Li, N. Gul, M.A. Khan, R.Bilal, A. Ali, M.Y. Alshahrani, T. Muhammad and S. Islam (2021). A new Hepatitis B model in light of asymptomatic carriers and vaccination study through Atangana–Baleanu derivative. *Results in Physics* 29 104603.
- 20. J.D. Murray (1989). Mathematical Biology. Berlin: Springer-Verlag.
- H. Sato, H Nakada, R Yamaguchi, M kami (2010). When should we intervene to control the 2009 influenza A(H1N1) pandemic, European communicable disease bulletin15
- M.A. Safi, A.B. Gumel, E.H. Elbasha (2013). Qualitative analysis of an age-structured SEIR epidemic model with treatment. Applied Mathematics and Computation 219: 10627-10642.
- M.A. Safi and M.H. DarAssi (2018). Mathematical analysis of a model for ectoparasite-borne diseases. Journal of Computational Methods in Sciences and Engineering 41 (17): 8248-8257.
- M.A. Safi and M.H. DarAssi (2019). Mathematical analysis of an age-structured HSV-2 model. Journal of Computational Methods in Sciences and Engineering 19 (3) 841-856.
- M.A. Safi, B. Al-Hdaibat, M.H. DarAssi and M.A. Khan. Global dynamics for a discrete quarantine/isolation model (2021). Results in Physics 21 103788.
- 26. N. Trebi (2017). Emerging and Neglected Infectious Diseases: Insights, Advances, and Challenges. *BioMed Research International.*
- L. Wang, Q. Cui and Z. Teng (2013). Global dynamics in a class of discrete-time epidemic models with disease courses. Advances in Difference Equations 2013: 57.
- Y. Wang, Z. Teng and M. Rehim (2014). Lyapunov functions for a class of discrete SIRS models with nonlinear incidence rate and varying population sizes. Discrete Dynamics in Nature and Society 2014:1-10

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Variable	Description
S(t)	Population of susceptible individuals
E(t)	Population of exposed individuals
I(t)	Population of infectious individuals but not treated
Y(t)	Population of treated individuals
W(t)	Population of treated individuals who failed treatment
R(t)	Population of recovered individuals

Parameter	Description
П	Recruitment rate
u	Natural death rate
κ	Progression rate from exposed to infectious
β	Contact rate
σ	Treatment rate for infectious individuals
ψ	Treatment failure rate
γ_1	Recovery rate of infectious individuals
γ_2	Recovery rate of treated individuals
γ_3	Recovery rate of failed treated individuals
δ_1	Disease-induced death rate of infectious individuals
δ_2	Disease-induced death rate of treated individuals
δ_3	Disease-induced death rate of failed treated individuals

Table 2: Description of variables and parameters of the model (2.1).