



Research article

Impact of general incidence function on three-strain SEIAR model

Manoj Kumar Singh¹, Anjali .¹, Brajesh K. Singh² and Carlo Cattani^{3,4,*}

¹ Faculty of Mathematics & Computing, Department of Mathematics & Statistics, Banasthali Vidyapith, Rajasthan 304022, India

² Department of Mathematics, Babasaheb Bhimrao Ambedkar University, Lucknow 226025, India

³ Department of Mathematics and Informatics, Azerbaijan University, J. Hajibeyli str., AZ1007, Baku

⁴ Azerbaijan Engineering School, DEIM, University of Tuscia, P.le dell'Università, Viterbo 01100, Italy

* **Correspondence:** Email: cattani@unitus.it, Carlo.Cattani@au.edu.az.

Abstract: We investigate the behavior of a complex three-strain model with a generalized incidence rate. The incidence rate is an essential aspect of the model as it determines the number of new infections emerging. The mathematical model comprises thirteen nonlinear ordinary differential equations with susceptible, exposed, symptomatic, asymptomatic and recovered compartments. The model is well-posed and verified through existence, positivity and boundedness. Eight equilibria comprise a disease-free equilibria and seven endemic equilibrium points following the existence of three strains. The basic reproduction numbers \mathcal{R}_{01} , \mathcal{R}_{02} and \mathcal{R}_{03} represent the dominance of strain 1, strain 2 and strain 3 in the environment for new strain emergence. The model establishes local stability at a disease-free equilibrium point. Numerical simulations endorse the impact of general incidence rates, including bi-linear, saturated, Beddington DeAngelis, non-monotone and Crowley Martin incidence rates.

Keywords: general incidence rate; reproduction number; multi-strain; asymptomatic; Liénard Chipart criterion

1. Introduction

The mathematical model is formulated to protect the environment and ease living beings' lives. Although humans have evolved to fight any circumstance, a state like an outbreak or pandemic still gets critically on our nerves. The biological reasons responsible for the spread of infection must be noted to decrease the hosts' mortality. The wheel of an infection period considers every aspect of the biological, social and physical environment. The genetic core is also a factor to be considered. The SIR (susceptible, infectious and recovered) compartmental model with a homogeneous host population was

first studied by McKendrick and Kermack in 1927 [1]. Further, the McKendrick model was improved by adding an exposed compartment for the individuals during an incubation period. An incubation period is when the carrier does not show the symptoms of the infection. We have focused on the SEIAR model with two infectious compartments, including symptomatic and asymptomatic classes [2–4]. Symptomatic is the class of carriers comprising people showing symptoms within or after the incubation period. On the other hand, the asymptomatic class never shows any symptoms.

Strain is biologically defined as a phenotypically/genotypically distinctive group of isolates that depends on the typing scheme due to the emergence of strains and host immune changes [5,6]. Malaria, tuberculosis, dengue, influenza, Coronavirus and others have different variants. These variants are like plant branches that are somewhat different but have the same base properties. Variants may act differently in terms of resistance, rate of spread and vaccines. Biological data assures that the host's genetic changes and their response to infectious diseases could cause the genome sequencing of the infection. The multi-strain model [7–11] considers more than one variant of the infection, considering strains can coexist or compete with each other for their existence [4, 12]. Here, we are considering three variants of the concerned, alpha, beta and delta, of the Coronavirus for the validation of our mathematical model [13–15].

Inter-agency assemblies like the Centers for Disease Control and Prevention (CDC) and the World Health Organization (WHO) focus on the quick classification of emergent mutations of viruses to observe the possible effect of viruses. To estimate the danger posed by variants of infectious diseases on the earth and recommend appropriate measures, experts estimate and measure the existing data to calculate the severity of the disease and capability of a blowout in hosts. The variants are categorized as variants being monitored (VBM), variant of interest (VOI), variant of concern (VOC) and variant of high consequence (VOHC) [16, 17]. VBM comprise those variants whose statistics are directly connected to a severe infection or have amplified spread but are detected to have no long severity. These variants may have a low spread rate at this time. A VOI is a variant whose mutation has caused significant variation widely in the susceptible class. A VOI grows into a VOC if the spread rate increases and causes a devastating effect of the disease, while a VOHC is a stage when WHO informs other organizations about the infection's spread. A lineage is a cluster of related viruses with a common predecessor [18].

The incidence rate is the frequency of new infections of any disease considering susceptible populations arising in a certain period. The importance of the incidence rate and different kinds of nonlinear incidence functions [19–23] is formulated to explain and discuss the transmission rate of the infection amongst the host [24, 25]. The global dynamics of the mathematical model for the bilinear [26–29], saturated [28–31], Beddington DeAngelis [32, 33], fractional, non-monotonic [27] and Crowley Martin incidence function [34] was studied through SIR and SEIR models. Some mathematical models are studied with homogeneous mixture incidence function, while others consider heterogeneous mixture incidence function. O. Khyar and K. Allali formulated the SEIR model with two strains considering non-monotone and general incidence rates [27, 35–38]. The main objective of the three-strain model with the generalized incidence function is to cover the class of all mentioned incidence rates and study the effect of incidence rate function (The choice of incidence rate plays an important role in the mathematical model) [39, 40].

2. Model formulation

The model includes the vital dynamics where B is the recruitment rate, and $1/\gamma$ is the average life expectancy of the population. The basic model contains five variables, S_i (susceptible), E_i (expected), I_i (infected), A_i (asymptomatic) and R_i (recovered), where $i = 1, 2$ and 3 according to strain 1, strain 2 and strain 3, respectively. α_1, α_2 and α_3 are the ratios of disease transmission rate by asymptomatic class of strain 1, strain 2 and strain 3, respectively. ξ_1, ξ_2 and ξ_3 are the reciprocals of the latent/incubation periods for strain 1, strain 2 and strain 3, respectively. $\delta_{11}, \delta_{21}, \delta_{12}, \delta_{22}, \delta_{13}, \delta_{23}$ are multiplicative factors for δ_{ij} , where i represents the rate of infection of individual R_i in recovery class by strain j . r_1, r_2 and r_3 are the rates at which exposed individuals become symptomatic with strain 1, strain 2 and strain 3, respectively. $\delta_{1I}, \delta_{2I}, \delta_{3I}, \delta_{1A}, \delta_{2A}, \delta_{3A}$ are the recovery rates of infectious and asymptomatic classes in accordance with the strains as mentioned in the subscripts of δ . $f(S, I_1, A_1)$, $g(S, I_2, A_2)$, and $h(S, I_3, A_3)$ are generalized incidence functions which are assumed to be continuously differentiable in the interior of \mathbb{R}_+^3 and satisfy the properties (2.1, 2.2 and 2.3). It is assumed in the mathematical model that at $t = 0$ there was no infection in the host population. After the emergence of an infection, the first virus of concern is the first strain when $t \leq t_1$. At time t_1 , the new virus of concern, the second strain, emerges, for $t_1 < t \leq t_2$. At time t_2 , the third virus of concern is said to emerge. The emerging strain re-infection is not considered. [41]

$$\begin{aligned} f(0, I_1, A_1) &= 0, \\ g(0, I_2, A_2) &= 0, \\ h(0, I_3, A_3) &= 0, \text{ for all } I_j, A_j \geq 0, j = 1, 2, 3. \end{aligned} \quad (2.1)$$

$$\begin{aligned} \frac{\partial f(S, I_1, A_1)}{\partial S} &> 0, \\ \frac{\partial g(S, I_2, A_2)}{\partial S} &> 0, \\ \frac{\partial h(S, I_3, A_3)}{\partial S} &> 0, \text{ for all } S > 0, I_j, A_j \geq 0, j = 1, 2, 3. \end{aligned} \quad (2.2)$$

$$\begin{aligned} \frac{\partial f(S, I_1, A_1)}{\partial I_1} &\leq 0, \\ \frac{\partial g(S, I_2, A_2)}{\partial I_2} &\leq 0, \\ \frac{\partial h(S, I_3, A_3)}{\partial I_3} &\leq 0, \text{ for all } S \geq 0, I_j, A_j \geq 0, j = 1, 2, 3. \end{aligned} \quad (2.3)$$

$$\left. \begin{aligned}
\frac{dS}{dt} &= B - f(S, I_1, A_1)(I_1(t) + \alpha_1 A_1(t)) - g(S, I_2, A_2)(I_2(t) + \alpha_2 A_2(t)) - h(S, I_3, A_3)(I_3(t) + \alpha_3 A_3(t)) - \gamma S(t), \\
\frac{dE_1}{dt} &= f(S, I_1, A_1)(I_1(t) + \alpha_1 A_1(t)) + \delta_{11} f(S, I_1, A_1)(I_1(t) + \alpha_1 A_1(t)) \frac{R_1(t)}{S(t)} \\
&\quad + \alpha_1 A_1(t) \frac{R_1(t)}{S(t)} + \delta_{21} f(S, I_1, A_1)(I_1(t) + \alpha_1 A_1(t)) \frac{R_2(t)}{S(t)} - \xi_1 E_1(t) - \gamma E_1(t), \\
\frac{dI_1}{dt} &= r_1(\xi_1) E_1(t) - \delta_{1I} I_1(t) - \gamma I_1(t), \\
\frac{dA_1}{dt} &= (1 - r_1)(\xi_1) E_1(t) - \delta_{1A} A_1(t) - \gamma A_1(t), \\
\frac{dR_1}{dt} &= \delta_{1I} I_1(t) + \delta_{1A} A_1(t) - \delta_{11} f(S, I_1, A_1)(I_1(t) + \alpha_1 A_1(t)) \frac{R_1(t)}{S(t)} \\
&\quad - \delta_{12} g(S, I_2, A_2)(I_2(t) + \alpha_2 A_2(t)) \frac{R_1(t)}{S(t)} - \delta_{13} h(S, I_3, A_3)(I_3(t) + \alpha_3 A_3(t)) \frac{R_1(t)}{S(t)} - \gamma R_1(t), \\
\frac{dE_2}{dt} &= g(S, I_2, A_2)(I_2(t) + \alpha_2 A_2(t)) + \delta_{12} g(S, I_2, A_2)(I_2(t) + \alpha_2 A_2(t)) \frac{R_1(t)}{S(t)} \\
&\quad + \alpha_2 A_2(t) \frac{R_1(t)}{S(t)} + \delta_{22} g(S, I_2, A_2)(I_2(t) + \alpha_2 A_2(t)) \frac{R_2(t)}{S(t)} - \xi_2 E_2(t) - \gamma E_2(t), \\
\frac{dI_2}{dt} &= r_2(\xi_2) E_2(t) - \delta_{2I} I_2(t) - \gamma I_2(t), \\
\frac{dA_2}{dt} &= (1 - r_2)(\xi_2) E_2(t) - \delta_{2A} A_2(t) - \gamma A_2(t), \\
\frac{dR_2}{dt} &= \delta_{2I} I_2(t) + \delta_{2A} A_2(t) - \delta_{21} f(S, I_1, A_1)(I_1(t) + \alpha_1 A_1(t)) \frac{R_2(t)}{S(t)} \\
&\quad - \delta_{22} g(S, I_2, A_2)(I_2(t) + \alpha_2 A_2(t)) \frac{R_2(t)}{S(t)} - \delta_{23} h(S, I_3, A_3)(I_3(t) + \alpha_3 A_3(t)) \frac{R_2(t)}{S(t)} \\
&\quad + \alpha_3 A_3(t) \frac{R_2(t)}{S(t)} - \gamma R_2(t), \\
\frac{dE_3}{dt} &= h(S, I_3, A_3)(I_3(t) + \alpha_3 A_3(t)) + \delta_{13} h(S, I_3, A_3)(I_3(t) + \alpha_3 A_3(t)) \frac{R_1(t)}{S(t)} \\
&\quad + \alpha_3 A_3(t) \frac{R_1(t)}{S(t)} + \delta_{23} h(S, I_3, A_3)(I_3(t) + \alpha_3 A_3(t)) \frac{R_2(t)}{S(t)} - \xi_3 E_3(t) - \gamma E_3(t), \\
\frac{dI_3}{dt} &= r_3(\xi_3) E_3(t) - \delta_{3I} I_3(t) - \gamma I_3(t), \\
\frac{dA_3}{dt} &= (1 - r_3)(\xi_3) E_3(t) - \delta_{3A} A_3(t) - \gamma A_3(t), \\
\frac{dR_3}{dt} &= \delta_{3I} I_3(t) + \delta_{3A} A_3(t) - \gamma R_3(t).
\end{aligned} \right\} \quad (2.4)$$

3. Model analysis

3.1. Positivity and boundedness of solution

According to the assumptions, the compartmental model (2.4) has all its associated parameters and variables non-negative. The model to fulfill its purpose, must satisfy the well-posedness, for which the existence, positivity and boundedness of the solution with the seed conditions, defined in the Theorem (1), are necessary.

Theorem 1. *Let the seed conditions $S_0 = (S(0), E_1(0), I_1(0), A_1(0), R_1(0), E_2(0), I_2(0), A_2(0), R_2(0), E_3(0), I_3(0), A_3(0), R_3(0))$ be non-negative. Then, the solution $\bar{S} = \{(S(t), E_1(t), I_1(t), A_1(t), R_1(t), E_2(t), I_2(t), A_2(t), R_2(t), E_3(t), I_3(t), A_3(t), R_3(t))\}$ of the system of equations of model (2.4) exists, is non-negative and is bounded.*

That is, $\bar{\Pi} = \left\{ \bar{S} \in \mathbb{R}_+^{13}, 0 \leq N(t) \leq \max \left\{ N(0) + \frac{B}{\gamma} \right\} \right\}$ is the positively invariant feasible region.

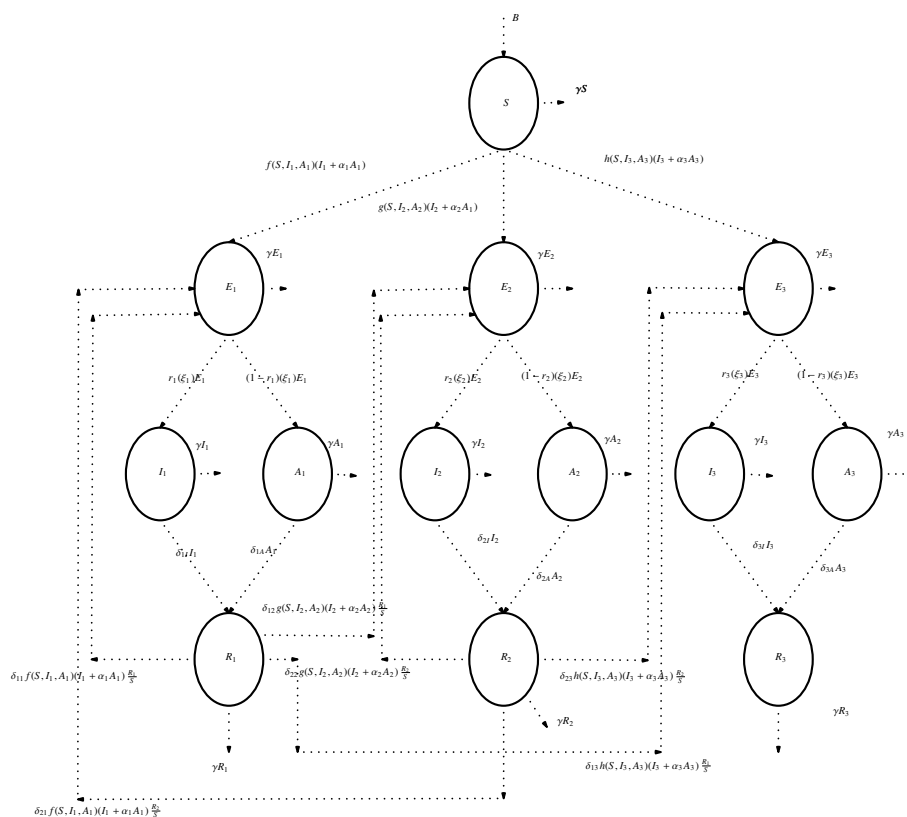


Figure 1. Flow chart of SEIAR model with emerging strains and incidence function (2.4).

Proof. The existence of the unique local solution of the mathematical model (2.4) can be stated by the fundamental theory of ordinary differential equations. To show the non-negativity of the solution, we must prove that any solution initiating from the non-negative region/orthant of $\mathbb{R}_+^{13} = \{\bar{S} \in \mathbb{R}^{13} : S_0 \geq 0\}$ resides in it globally.

Let us define

$$\bar{T} = \sup\{t \geq 0 : S_0 \geq 0\} \in [0, t]. \tag{3.1}$$

Claim that $\bar{T} = +\infty$. Let us assume that \bar{T} is finite, so with the solution's property of being continuous, $S(\bar{T}) = 0$ or $E_1(\bar{T}) = 0$ or $I_1(\bar{T}) = 0$ or $A_1(\bar{T}) = 0$ or $R_1(\bar{T}) = 0$ or $E_2(\bar{T}) = 0$ or $I_2(\bar{T}) = 0$ or $A_2(\bar{T}) = 0$ or $R_2(\bar{T}) = 0$ or $E_3(\bar{T}) = 0$ or $I_3(\bar{T}) = 0$ or $A_3(\bar{T}) = 0$ or $R_3(\bar{T}) = 0$.

Let us assume $S(\bar{T}) = 0$ before any other variables become zero. Hence,

$$\frac{dS(\bar{T})}{dt} = \lim_{t \rightarrow \bar{T}} \frac{S(\bar{T}) - S(t)}{\bar{T} - t} = \lim_{t \rightarrow \bar{T}} \frac{-S(t)}{\bar{T} - t} \leq 0. \tag{3.2}$$

From the leading equation of mathematical model (2.4) and Eq (2.1), we have

$$\begin{aligned}
 \frac{dS(\bar{T})}{dt} &= B - f(S(0), I_1(\bar{T}), A_1(\bar{T}))(I_1(\bar{T}) + \alpha_1 A_1(\bar{T})) \\
 &\quad - g(S(0), I_2(\bar{T}), A_2(\bar{T}))(I_2(\bar{T}) + \alpha_2 A_2(\bar{T})) \\
 &\quad - h(S(0), I_3(\bar{T}), A_3(\bar{T}))(I_3(\bar{T}) - \alpha_3 A_3(\bar{T})) - \gamma S(\bar{T}) \\
 &= B - f(0, I_1(\bar{T}), A_1(\bar{T}))(I_1(\bar{T}) + \alpha_1 A_1(\bar{T})) \\
 &\quad - g(0, I_2(\bar{T}), A_2(\bar{T}))(I_2(\bar{T}) + \alpha_2 A_2(\bar{T})) \\
 &\quad - h(0, I_3(\bar{T}), A_3(\bar{T}))(I_3(\bar{T}) + \alpha_3 A_3(\bar{T})) - \gamma S(0) \\
 &= B > 0.
 \end{aligned} \tag{3.3}$$

This contradicts the above Eq (3.2). Now, assuming $E_1(\bar{T})$ as zero, before any other variables become zero. Hence,

$$\frac{dE_1(\bar{T})}{dt} = \lim_{t \rightarrow \bar{T}^-} \frac{E_1(\bar{T}) - E_1(t)}{\bar{T} - t} = \lim_{t \rightarrow \bar{T}^-} \frac{-E_1(t)}{\bar{T} - t} \leq 0. \tag{3.4}$$

From the second equation of mathematical model (2.4) and Eq (2.2), we have

$$\begin{aligned}
 \frac{dE_1(\bar{T})}{dt} &= f(S, I_1, A_1)(I_1(\bar{T}) + \alpha_1 A_1(\bar{T})) + \delta_{11} f(S, I_1, A_1)(I_1(\bar{T})) \\
 &\quad + \alpha_1 A_1(\bar{T}) \frac{R_1(\bar{T})}{S(\bar{T})} + \delta_{21} f(S, I_1, A_1)(I_1(\bar{T})) \\
 &\quad + \alpha_1 A_1(\bar{T}) \frac{R_2(\bar{T})}{S(\bar{T})} - \xi_1 E_1(\bar{T}) - \gamma E_1(\bar{T}) \\
 &= f(S, I_1, A_1)(I_1(\bar{T}) + \alpha_1 A_1(\bar{T})) + \delta_{11} f(S, I_1, A_1)(I_1(\bar{T})) \\
 &\quad + \alpha_1 A_1(\bar{T}) \frac{R_1(\bar{T})}{S(\bar{T})} + \delta_{21} f(S, I_1, A_1)(I_1(\bar{T})) \\
 &\quad + \alpha_1 A_1(\bar{T}) \frac{R_2(\bar{T})}{S(\bar{T})} > 0.
 \end{aligned} \tag{3.5}$$

This contradicts the above Eq (3.4). Similarly, we can find for $I_1, A_1, R_1, E_2, I_2, A_2, R_2, I_3, A_3, R_3$. Hence, \bar{T} could not have a finite value. Thus, the positivity of the solution of model (2.4) is confirmed.

For boundedness of the solution, consider the total population $N(t)$ that is equal to the sum of all the compartments of the mathematical model. The time derivative of the total population found with the help of system of Eq (2.4) is

$$\frac{dN(t)}{dt} = B - \gamma N(t). \tag{3.6}$$

Therefore,

$$N(t) = \frac{B}{\gamma} + \left(N(0) - \frac{B}{\gamma} \right) e^{(-\gamma t)}. \tag{3.7}$$

For time $0 \leq t < \infty$, we find that $N(t) \leq \frac{B}{\gamma} + N(0)$. This states the boundedness of $N(t)$, which confirms the boundedness of \bar{S} . Hence, the local solution of the mathematical model (2.4) is established to have uniqueness, non-negativity and boundedness property globally. \square

3.2. Steady states and basic reproduction number

This subsection studies the basic reproduction number. The basic reproduction denoted as \mathfrak{R}_0 is the number measuring the secondary infections caused due to one susceptible host in the entire period of infection [42]. It plays a crucial role to decide the risk behind any disease to bloom out. We determine the basic reproduction number by the next generation matrix $\mathfrak{F}\mathfrak{B}^{-1}$ where \mathfrak{F} is the Jacobian matrix of the new infections at time t_2 , with the co-existence of all the three strains together. The matrix \mathfrak{B} is the Jacobian matrix of infection transfer to other classes that is non-singular at time t_2 . The spectral radius of the next generation matrix results in the basic reproduction number. The three-strain epidemic model has three eigenvalues of the next generation matrix \mathfrak{R}_{01} , \mathfrak{R}_{02} and \mathfrak{R}_{03} . The basic reproduction number \mathfrak{R}_0 here is the maximum of the three for the co-existence of all the three strains at time t_2 at an endemic equilibrium point $(\tilde{S}, \tilde{E}_1, \tilde{I}_1, \tilde{A}_1, \tilde{R}_1, \tilde{E}_2, \tilde{I}_2, \tilde{A}_2, \tilde{R}_2, \tilde{E}_3, \tilde{I}_3, \tilde{A}_3, \tilde{R}_3)$.

$$\mathfrak{F} = \begin{pmatrix} 0 & F_1 & F_2 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & G_1 & G_2 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & H_1 & H_2 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \end{pmatrix},$$

$$\mathfrak{B} = \begin{pmatrix} m_1 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ -r_1\xi_1 & m_2 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ \xi_1(r_1 - 1) & 0 & m_3 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & m_4 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & -r_2\xi_2 & m_5 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & \xi_2(r_2 - 1) & 0 & m_6 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & m_7 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & -r_3\xi_3 & m_8 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & \xi_3(r_3 - 1) & 0 & m_9 & 0 \end{pmatrix}.$$

Here,

$$F_1 = \left(f(\tilde{S}, \tilde{I}_1, \tilde{A}_1) + \frac{\partial f(\tilde{S}, \tilde{I}_1, \tilde{A}_1)}{\partial I_1} (\tilde{I}_1 + \alpha_1 \tilde{A}_1) \right) \frac{(\tilde{S} + \delta_{11} \tilde{R}_1 + \delta_{21} \tilde{R}_2)}{\tilde{S}},$$

$$F_2 = \left(\alpha_1 f(\tilde{S}, \tilde{I}_1, \tilde{A}_1) + \frac{\partial f(\tilde{S}, \tilde{I}_1, \tilde{A}_1)}{\partial A_1} (\tilde{I}_1 + \alpha_1 \tilde{A}_1) \right) \frac{(\tilde{S} + \delta_{11} \tilde{R}_1 + \delta_{21} \tilde{R}_2)}{\tilde{S}},$$

$$G_1 = \left(g(\tilde{S}, \tilde{I}_2, \tilde{A}_2) + \frac{\partial g(\tilde{S}, \tilde{I}_2, \tilde{A}_2)}{\partial I_2} (\tilde{I}_2 + \alpha_2 \tilde{A}_2) \right) \frac{(\tilde{S} + \delta_{12} \tilde{R}_1 + \delta_{22} \tilde{R}_2)}{\tilde{S}},$$

$$\begin{aligned}
G_2 &= \left(\alpha_2 g(\tilde{S}, \tilde{I}_2, \tilde{A}_2) + \frac{\partial g(\tilde{S}, \tilde{I}_2, \tilde{A}_2)}{\partial A_2} (\tilde{I}_2 + \alpha_2 \tilde{A}_2) \right) \frac{(\tilde{S} + \delta_{12} \tilde{R}_1 + \delta_{22} \tilde{R}_2)}{\tilde{S}}, \\
H_1 &= \left(h(\tilde{S}, \tilde{I}_3, \tilde{A}_3) + \frac{\partial h(\tilde{S}, \tilde{I}_3, \tilde{A}_3)}{\partial I_3} (\tilde{I}_3 + \alpha_3 \tilde{A}_3) \right) \frac{(\tilde{S} + \delta_{13} R_1 + \delta_{23} R_2)}{\tilde{S}}, \\
H_2 &= \left(\alpha_3 h(\tilde{S}, \tilde{I}_3, \tilde{A}_3) + \frac{\partial h(\tilde{S}, \tilde{I}_3, \tilde{A}_3)}{\partial A_3} (\tilde{I}_3 + \alpha_3 \tilde{A}_3) \right) \frac{(\tilde{S} + \delta_{13} \tilde{R}_1 + \delta_{23} \tilde{R}_2)}{\tilde{S}}, \\
m_1 &= \xi_1 + \gamma, \quad m_2 = \delta_{1I} + \gamma, \quad m_3 = \delta_{1A} + \gamma, \quad m_4 = \xi_2 + \gamma, \quad m_5 = \delta_{2I} + \gamma, \\
m_6 &= \delta_{2A} + \gamma, \quad m_7 = \xi_2 + \gamma, \quad m_8 = \delta_{3I} + \gamma, \quad m_9 = \delta_{3A} + \gamma.
\end{aligned}$$

We know that $\rho(\mathfrak{F}\mathfrak{B}^{-1}) = \mathfrak{R}_0 = \max\{\mathfrak{R}_{01}, \mathfrak{R}_{02}, \mathfrak{R}_{03}\}$, with ρ as spectral radius and $\mathfrak{R}_{01}, \mathfrak{R}_{02}, \mathfrak{R}_{03}$ values as in Eq (3.8). Here, the \mathfrak{R}_0 represents the emergence of the third strain in the environment.

$$\begin{aligned}
\mathfrak{R}_{01} &= \frac{\xi_1 (F_1 m_3 r_1 + F_2 \alpha_1 m_2 (1 - r_1))}{m_1 m_2 m_3}, \\
\mathfrak{R}_{02} &= \frac{\xi_2 (G_1 m_6 r_2 + G_2 \alpha_2 m_5 (1 - r_2))}{m_4 m_5 m_6}, \\
\mathfrak{R}_{03} &= \frac{\xi_3 (H_1 m_9 r_3 + H_2 \alpha_3 m_8 (1 - r_3))}{m_7 m_8 m_9}.
\end{aligned} \tag{3.8}$$

$$\begin{aligned}
\mathfrak{R}_{01} &= \mathfrak{R}_{01}^I + \mathfrak{R}_{01}^A, \quad \mathfrak{R}_{02} = \mathfrak{R}_{02}^I + \mathfrak{R}_{02}^A, \quad \mathfrak{R}_{03} = \mathfrak{R}_{03}^I + \mathfrak{R}_{03}^A, \\
\mathfrak{R}_{01}^I &= \frac{F_1 \xi_1 m_3 r_1}{m_1 m_2 m_3}, \quad \mathfrak{R}_{01}^A = \frac{F_2 \xi_1 m_2 (1 - r_1)}{m_1 m_2 m_3}, \\
\mathfrak{R}_{02}^I &= \frac{G_1 \xi_2 m_6 r_2}{m_4 m_5 m_6}, \quad \mathfrak{R}_{02}^A = \frac{G_2 \xi_2 m_5 (1 - r_2)}{m_4 m_5 m_6}, \\
\mathfrak{R}_{03}^I &= \frac{H_1 \xi_3 m_9 r_3}{m_7 m_8 m_9}, \quad \mathfrak{R}_{03}^A = \frac{H_2 \xi_3 m_8 (1 - r_3)}{m_7 m_8 m_9}.
\end{aligned}$$

The basic reproduction number at the $\mathcal{DFE} = \mathbb{E}_0$ is denoted as $\mathfrak{R}(\mathbb{E}_0) = \max\{\mathfrak{R}_{01}(\mathbb{E}_0), \mathfrak{R}_{02}(\mathbb{E}_0), \mathfrak{R}_{03}(\mathbb{E}_0)\}$. It describes the spread of infections by an individual at the initial stage.

$$\begin{aligned}
\mathfrak{R}_{01}(\mathbb{E}_0) &= \frac{f(\frac{B}{\gamma}, 0, 0) \xi_1 (m_3 r_1 + \alpha_1 m_2 (1 - r_1))}{m_1 m_2 m_3}, \\
\mathfrak{R}_{02}(\mathbb{E}_0) &= \frac{g(\frac{B}{\gamma}, 0, 0) \xi_2 (m_6 r_2 + \alpha_2 m_5 (1 - r_2))}{m_4 m_5 m_6}, \\
\mathfrak{R}_{03}(\mathbb{E}_0) &= \frac{h(\frac{B}{\gamma}, 0, 0) \xi_3 (m_9 r_3 + \alpha_3 m_8 (1 - r_3))}{m_7 m_8 m_9}.
\end{aligned} \tag{3.9}$$

Theorem 2. *The mathematical model (2.4) has disease free equilibrium \mathbb{E}_0 and seven endemic equilibrium points $\epsilon_{s1}, \epsilon_{s2}, \epsilon_{s3}, \epsilon_{s12}, \epsilon_{s23}, \epsilon_{s13}, \epsilon_{s123}$. These endemic equilibrium points exists in accordance with some conditions. [27, 43]*

(i) *The ϵ_{s1} is the first strain equilibrium which exists when $\mathfrak{R}_{01} > 1$.*

$$\begin{aligned}
 I_{s23,2} &= \frac{r_2 \xi_2}{m_5} E_{s23,2}, \quad A_{s23,2} = \frac{(1-r_2)\xi_2}{m_6} E_{s23,2}, \\
 R_{s23,2} &= \frac{(\delta_{21} I_{s23,2} + \delta_{2A} A_{s23,2}) S_{s23}}{\delta_{22} g(S_{s23}, I_{s23,2}, A_{s23,2})(I_{s23,2} + \alpha_2 A_{s23,2}) + \delta_{23} h(S_{s23}, I_{s23,1}, A_{s23,1})(I_{s23,3} + \alpha_3 A_{s23,3}) + \gamma S_{s23}}, \\
 E_{s23,3} &= \frac{h(S_{s23}, I_{s23,3}, A_{s23,3})(S_{s23} + \delta_{23} R_{s23,2})(I_{s23,3} + \alpha_3 A_{s23,3})}{S_{s23} m_7}, \\
 I_{s23,3} &= \frac{r_3 \xi_3}{m_8} E_{s23,3}, \quad A_{s23,1} = \frac{(1-r_3)\xi_3}{m_9} E_{s23,3}, \\
 R_{s23,3} &= \frac{\delta_{31} I_{s23,3} + \delta_{3A} A_{s23,3}}{\gamma}.
 \end{aligned}$$

(vi) The case when there is an infection due to first and second strain surviving in the environment, that is, $I_1 \neq 0, A_1 \neq 0, I_2 = 0, A_2 = 0, I_3 \neq 0$ and $A_3 \neq 0$. Here, the endemic equilibrium is

$$\begin{aligned}
 \epsilon_{s13} &= (S_{s13}, E_{s13,1}, I_{s13,1}, A_{s13,1}, R_{s13,1}, 0, 0, 0, E_{s13,3}, I_{s13,3}, A_{s13,3}, R_{s13,3}). \text{ Here,} \\
 S_{s13} &= \frac{B}{\gamma} - \frac{f(S_{s13}, I_{s13,1}, A_{s13,1})(I_{s13,1} + \alpha_1 A_{s13,1})}{\gamma} - \frac{h(S_{s13}, I_{s13,3}, A_{s13,3})(I_{s13,3} + \alpha_3 A_{s13,3})}{\gamma}, \\
 E_{s13,1} &= \frac{f(S_{s13}, I_{s13,1}, A_{s13,1})(S_{s13} + \delta_{11} R_{s13,1})}{S_{s13} m_1}, \\
 I_{s13,1} &= \frac{r_1 \xi_1}{m_2} E_{s13,1}, \quad A_{s13,1} = \frac{(1-r_1)\xi_1}{m_3} E_{s13,1}, \\
 R_{s13,1} &= \frac{(\delta_{11} I_{s13,1} + \delta_{1A} A_{s13,1}) S_{s13}}{\delta_{11} f(S_{s13}, I_{s13,1}, A_{s13,1})(I_{s13,1} + \alpha_1 A_{s13,1}) + \delta_{13} h(S_{s13}, I_{s13,3}, A_{s13,3})(I_{s13,3} + \alpha_3 A_{s13,3}) + \gamma S_{s13}}, \\
 E_{s13,3} &= \frac{h(S_{s13}, I_{s13,3}, A_{s13,3})(S_{s13} + \delta_{13} R_{s13,1})}{S_{s13} m_7}, \\
 I_{s13,3} &= \frac{r_3 \xi_3}{m_8} E_{s13,3}, \quad A_{s13,1} = \frac{(1-r_3)\xi_3}{m_9} E_{s13,3}, \\
 R_{s13,3} &= \frac{\delta_{31} I_{s13,3} + \delta_{3A} A_{s13,3}}{\gamma}.
 \end{aligned}$$

(vii) The case when there is infection due to first, second and third strain in the environment, that is, $I_1 \neq 0, A_1 \neq 0, I_2 \neq 0, A_2 \neq 0, I_3 \neq 0$ and $A_3 \neq 0$. Here, the endemic equilibrium is

$$\begin{aligned}
 \epsilon_{s123} &= (\tilde{S}, \tilde{E}_1, \tilde{I}_1, \tilde{A}_1, \tilde{R}_1, \tilde{E}_2, \tilde{I}_2, \tilde{A}_2, \tilde{R}_2, \tilde{E}_3, \tilde{I}_3, \tilde{A}_3, \tilde{R}_3). \text{ Here,} \\
 \tilde{S} &= \frac{B}{\gamma} - \frac{f(\tilde{S}, \tilde{I}_1, \tilde{A}_1)(\tilde{I}_1 + \alpha_1 \tilde{A}_1)}{\gamma} - \frac{g(\tilde{S}, \tilde{I}_2, \tilde{A}_2)(\tilde{I}_2 + \alpha_2 \tilde{A}_2)}{\gamma} - \frac{h(\tilde{S}, \tilde{I}_3, \tilde{A}_3)(\tilde{I}_3 + \alpha_3 \tilde{A}_3)}{\gamma}, \\
 \tilde{E}_1 &= \frac{f(\tilde{S}, \tilde{I}_1, \tilde{A}_1)(\tilde{S} + \delta_{11} \tilde{R}_1 + \delta_{21} \tilde{R}_2)(\tilde{I}_1 + \alpha_1 \tilde{A}_1)}{\tilde{S} m_1}, \quad \tilde{I}_1 = \frac{r_1 \xi_1}{m_2} \tilde{E}_1, \quad \tilde{A}_1 = \frac{(1-r_1)\xi_1}{m_3} \tilde{E}_1, \\
 \tilde{R}_1 &= \frac{(\delta_{11} \tilde{I}_1 + \delta_{1A} \tilde{A}_1) \tilde{S}}{\delta_{11} f(\tilde{S}, \tilde{I}_1, \tilde{A}_1)(\tilde{I}_1 + \alpha_1 \tilde{A}_1) + \delta_{12} g(\tilde{S}, \tilde{I}_2, \tilde{A}_2)(\tilde{I}_2 + \alpha_2 \tilde{A}_2) + \delta_{13} h(\tilde{S}, \tilde{I}_3, \tilde{A}_3)(\tilde{I}_3 + \alpha_3 \tilde{A}_3) + \gamma \tilde{S}}, \\
 \tilde{E}_2 &= \frac{g(\tilde{S}, \tilde{I}_2, \tilde{A}_2)(\tilde{S} + \delta_{12} \tilde{R}_1 + \delta_{22} \tilde{R}_2)(\tilde{I}_2 + \alpha_2 \tilde{A}_2)}{\tilde{S} m_4}, \quad \tilde{I}_2 = \frac{r_2 \xi_2}{m_5} \tilde{E}_2, \quad \tilde{A}_2 = \frac{(1-r_2)\xi_2}{m_6} \tilde{E}_2, \\
 \tilde{R}_2 &= \frac{(\delta_{21} \tilde{I}_2 + \delta_{2A} \tilde{A}_2) \tilde{S}}{\delta_{21} f(\tilde{S}, \tilde{I}_1, \tilde{A}_1)(\tilde{I}_1 + \alpha_1 \tilde{A}_1) + \delta_{22} g(\tilde{S}, \tilde{I}_2, \tilde{A}_2)(\tilde{I}_2 + \alpha_2 \tilde{A}_2) + \delta_{23} h(\tilde{S}, \tilde{I}_3, \tilde{A}_3)(\tilde{I}_3 + \alpha_3 \tilde{A}_3) + \gamma \tilde{S}}, \\
 \tilde{E}_3 &= \frac{h(\tilde{S}, \tilde{I}_3, \tilde{A}_3)(\tilde{S} + \delta_{13} \tilde{R}_1 + \delta_{23} \tilde{R}_2)(\tilde{I}_3 + \alpha_3 \tilde{A}_3)}{\tilde{S} m_7}, \quad \tilde{I}_3 = \frac{r_3 \xi_3}{m_8} \tilde{E}_3, \quad \tilde{A}_3 = \frac{(1-r_3)\xi_3}{m_9} \tilde{E}_3, \quad \tilde{R}_3 = \frac{\delta_{31} \tilde{I}_3 + \delta_{3A} \tilde{A}_3}{\gamma}.
 \end{aligned}$$

□

3.3. Local stability at a disease free equilibrium

This subsection discusses the local stability of disease free equilibrium (\mathcal{DFE}). The Jacobian matrix of order thirteen of the system (2.4) is in Eq (3.10).

$$A = (a_{i,j}), \quad \text{where } i \leq 13, j \leq 13. \tag{3.10}$$

The appendix section contains all the non zero terms of the matrix A. It is hard to determine the further dynamics with the matrix A due to the course of constraints.

Theorem 3. *The disease free equilibrium (\mathcal{DFE}) of the mathematical model (2.4) is locally asymptotic stable if the basic reproduction $\mathfrak{R}(\mathbb{E}_0)$ is less than one and unstable for greater than one.*

Proof. The Jacobian $J_{\mathcal{DFE}}$ of the mathematical model (2.4) at the initial stage when $t = 0$, the infection

is not spread (disease free equilibrium) is,

$$\begin{pmatrix} -\gamma & 0 & -F & -\alpha_1 F & 0 & 0 & -G & -\alpha_2 G & 0 & 0 & -H & -\alpha_3 H & 0 \\ 0 & -m_1 & F & \alpha_1 F & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & \xi_1 r_1 & -m_2 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & \xi_1 (1 - r_1) & 0 & -m_3 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & \delta_I & \delta_A & -\gamma & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & -m_4 & G & \alpha_2 G & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & \xi_2 r_2 & -m_5 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & \xi_2 (1 - r_2) & 0 & -m_6 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & \delta_{2I} & \delta_{2A} & -\gamma & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & -m_7 & H & \alpha_3 H & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & \xi_3 r_3 & -m_8 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & \xi_3 (1 - r_3) & 0 & -m_9 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & \delta_{3I} & \delta_{3A} & -\gamma \end{pmatrix}.$$

The above matrix $J_{D\mathcal{F}\mathcal{E}}$ has the characteristic polynomial as,

$$\begin{aligned} & (\gamma + \lambda)^4 \left(\alpha_1 (-F) \xi_1 (r_1 - 1) (\lambda + m_2) - (\lambda + m_3) \left(-F \xi_1 r_1 + \lambda^2 + \lambda m_2 \right. \right. \\ & \quad \left. \left. + m_1 (\lambda + m_2) \right) \right) (\alpha_2 (-G) \xi_2 (r_2 - 1) (\lambda + m_5) \\ & \quad - (\lambda + m_6) \left(-G \xi_2 r_2 + \lambda^2 + \lambda m_5 + m_4 (\lambda + m_5) \right)) (\alpha_3 (-H) \xi_3 (r_3 - 1) \\ & \quad (\lambda + m_8) - (\lambda + m_9) \left(-H \xi_3 r_3 + \lambda^2 + \lambda m_8 + m_7 (\lambda + m_8) \right)) \end{aligned} \quad (3.11)$$

Here, $F = f(\frac{B}{\gamma}, 0, 0)$, $G = g(\frac{B}{\gamma}, 0, 0)$, $H = h(\frac{B}{\gamma}, 0, 0)$. From the characteristic polynomial, four eigenvalues of Eq (3.11) are real and negative, and the other nine are the zeros of Eq (3.12).

$$\begin{aligned} & (A_0 \lambda^3 + A_1 \lambda^2 + A_2 \lambda + A_3)(B_0 \lambda^3 + B_1 \lambda^2 + B_2 \lambda + B_3) \\ & (C_0 \lambda^3 + C_1 \lambda^2 + C_2 \lambda + C_3) = 0 \end{aligned} \quad (3.12)$$

Here, $A_0 = 1$, $A_1 = m_1 + m_2 + m_3$, $A_2 = m_1 m_2 (1 - \mathfrak{R}_{01}^I) + m_1 m_3 (1 - \mathfrak{R}_{01}^A) + m_2 m_3$, $A_3 = m_1 m_2 m_3 (1 - \mathfrak{R}_{01})$, $B_0 = 1$, $B_1 = m_4 + m_5 + m_6$, $B_2 = m_4 m_6 (1 - \mathfrak{R}_{02}^A) + m_4 m_5 (1 - \mathfrak{R}_{02}^I) + m_5 m_6$, $B_3 = m_4 m_5 m_6 (1 - \mathfrak{R}_{02})$, $C_0 = 1$, $C_1 = m_7 + m_8 + m_9$, $C_2 = m_7 m_9 (1 - \mathfrak{R}_{03}^A) + m_7 m_8 (1 - \mathfrak{R}_{03}^I) + m_8 m_9$, and $C_3 = m_7 m_8 m_9 (1 - \mathfrak{R}_{03})$.

By the Liénard Chipart criterion, a polynomial has all its zeros with negative real part if $A_0 > 0$, $A_1 > 0$, $A_2 > 0$, $A_3 > 0$, $B_0 > 0$, $B_1 > 0$, $B_2 > 0$, $B_3 > 0$, $C_0 > 0$, $C_1 > 0$, $C_2 > 0$, $C_3 > 0$, $A_1 A_2 - A_0 A_3 > 0$, $B_1 B_2 - B_0 B_3 > 0$, and $C_1 C_2 - C_0 C_3 > 0$. It is clear that the conditions hold if $\mathfrak{R}_{01} < 1$, $\mathfrak{R}_{02} < 1$ and $\mathfrak{R}_{03} < 1$.

As $A_1 A_2 - A_0 A_3 = (m_1^2 (m_3 (1 - \mathfrak{R}_{01}^A) + m_2 (1 - \mathfrak{R}_{01}^I)) + m_1 (m_3 m_2 (-\mathfrak{R}_{01}^A - \mathfrak{R}_{01}^I + \mathfrak{R}_{01} + 2) + m_3^2 (1 - \mathfrak{R}_{01}^A) + m_2^2 (1 - \mathfrak{R}_{01}^I)) + m_2 m_3 (m_2 + m_3))$, $B_1 B_2 - B_0 B_3 = (m_4^2 (m_6 (1 - \mathfrak{R}_{02}^A) + m_5 (1 - \mathfrak{R}_{02}^I)) + m_4 (m_6 m_5 (-\mathfrak{R}_{02}^A - \mathfrak{R}_{02}^I + \mathfrak{R}_{02} + 2) + m_6^2 (1 - \mathfrak{R}_{02}^A) + m_5^2 (1 - \mathfrak{R}_{02}^I)) + m_5 m_6 (m_5 + m_6))$, and $C_1 C_2 - C_0 C_3 = (m_7^2 (m_9 (1 - \mathfrak{R}_{03}^A) + m_8 (1 - \mathfrak{R}_{03}^I)) + m_7 (m_9 m_8 (-\mathfrak{R}_{03}^A - \mathfrak{R}_{03}^I + \mathfrak{R}_{03} + 2) + m_9^2 (1 - \mathfrak{R}_{03}^A) + m_8^2 (1 - \mathfrak{R}_{03}^I)) + m_8 m_9 (m_8 + m_9))$.

Evidently all the thirteen eigenvalues of the Jacobian at disease free equilibrium have real part negative if and only if $\mathfrak{R}_{01} < 1$, $\mathfrak{R}_{02} < 1$ and $\mathfrak{R}_{03} < 1$. This indicates asymptotic local stability of mathematical model (2.4) with the basic reproduction number less than one and instability otherwise. \square

4. Numerical simulation

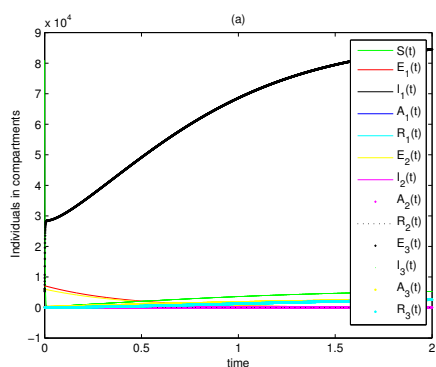
This section studies importance and the effect of each compartment with different incidence rates (transfer of the infection). Considering the three incidence functions $f(S, I_1, A_1)$, $g(S, I_2, A_2)$ and $h(S, I_3, A_3)$ have an impact that cannot be neglected and further clarifies the need to choose the perfect incidence rate in the mathematical model.

Table 1. Cases for different choices of incidence rates ($f(S, I_1, A_1)$, $g(S, I_2, A_2)$ and $h(S, I_3, A_3)$).

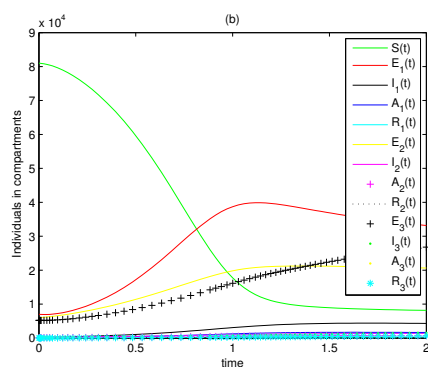
Case	$f(S, I_1, A_1)$	$g(S, I_2, A_2)$	$h(S, I_3, A_3)$
(a)	$\beta_1 S$	$\beta_2 S$	$\beta_3 S$
(b)	$\frac{\beta_1 S}{1 + \zeta_1 S + \zeta_2 I_1 + \zeta_3 A_1}$	$\frac{\beta_2 S}{1 + \zeta_4 S + \zeta_5 I_2 + \zeta_6 A_2}$	$\frac{\beta_3 S}{1 + \zeta_7 S + \zeta_8 I_3 + \zeta_9 A_3}$
(c)	$\frac{\beta_1 S}{1 + \zeta_1 I_1^2}$	$\frac{\beta_2 S}{1 + \zeta_2 I_2^2}$	$\frac{\beta_3 S}{1 + \zeta_3 I_3^2}$
(d)	$\frac{\beta_1 S}{1 + \zeta_1 A_1^2}$	$\frac{\beta_2 S}{1 + \zeta_2 A_2^2}$	$\frac{\beta_3 S}{1 + \zeta_3 A_3^2}$
(e)	$\frac{\beta_1 S}{1 + \zeta_1 S}$	$\frac{\beta_2 S}{1 + \zeta_2 S}$	$\frac{\beta_3 S}{1 + \zeta_3 S}$
(f)	$\frac{\beta_1 S}{1 + \zeta_1 I_1}$	$\frac{\beta_2 S}{1 + \zeta_2 I_2}$	$\frac{\beta_3 S}{1 + \zeta_3 I_3}$
(g)	$\frac{\beta_1 S}{1 + \zeta_1 A_1}$	$\frac{\beta_2 S}{1 + \zeta_2 A_2}$	$\frac{\beta_3 S}{1 + \zeta_3 A_3}$
(h)	$\beta_1 S / (1 + \zeta_1 S + \zeta_2 I_1 + \zeta_3 A_1 + \zeta_1 \zeta_2 S I_1 + \zeta_1 \zeta_3 S A_1 + \zeta_2 \zeta_3 I_1 A_1)$	$\beta_2 S / (1 + \zeta_4 S + \zeta_5 I_2 + \zeta_6 A_2 + \zeta_4 \zeta_5 S I_2 + \zeta_5 \zeta_6 I_2 A_2 + \zeta_4 \zeta_6 S A_2)$	$\beta_3 S / (1 + \zeta_7 S + \zeta_8 I_3 + \zeta_9 A_3 + \zeta_7 \zeta_8 S I_3 + \zeta_8 \zeta_9 I_3 A_3 + \zeta_7 \zeta_9 S A_3)$
(i)	$\beta_1 S / (1 + \zeta_1 S + \zeta_2 I_1 + \zeta_3 A_1 + \zeta_4 S I_1 + \zeta_5 S A_1 + \zeta_6 I_1 A_1)$	$\beta_2 S / (1 + \zeta_7 S + \zeta_8 I_2 + \zeta_9 A_2 + \zeta_{10} S I_2 + \zeta_{11} I_2 A_2 + \zeta_{12} S A_2)$	$\beta_3 S / (1 + \zeta_{13} S + \zeta_{14} I_3 + \zeta_{15} A_3 + \zeta_{16} S I_3 + \zeta_{17} I_3 A_3 + \zeta_{18} S A_3)$
(j)	$\frac{\beta_1 S}{1 + \zeta_1 I_1}$	$\frac{\beta_2 S}{1 + \zeta_2 S + \zeta_3 I_2 + \zeta_4 A_2}$	$\frac{\beta_3 S}{1 + \zeta_5 A_3^2}$

* The parameters in the different cases have different values that are used to represent different scenarios.

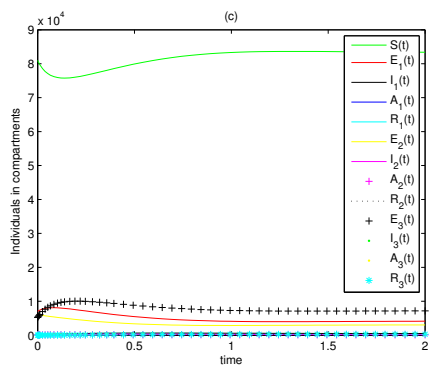
Bilinear incidence rate $\beta * \text{Susceptible}$ is used when the susceptible compartment has direct linear impact on the transmission, and all other compartments play a negligible effect. Beddington DeAngelis function $\frac{\beta * \text{Susceptible}}{1 + \zeta_1 * \text{Susceptible} + \zeta_2 * \text{Infected} + \zeta_3 * \text{Asymptomatic}}$ shows the nonlinear impact of the infected, asymptomatic and susceptible class on the transmission of the disease. Non-monotone function $\frac{\beta * \text{Susceptible}}{1 + \zeta_1 * \text{Infected}^2}$ or $\frac{\beta * \text{Susceptible}}{1 + \zeta_1 * \text{Asymptomatic}^2}$, Crowley Martin function $(\beta * \text{Susceptible}) / (1 + \zeta_1 * \text{Susceptible} + \zeta_2 * \text{Infected} + \zeta_3 * \text{Asymptomatic} + \zeta_1 \zeta_2 * \text{Infected} * \text{Susceptible} + \zeta_1 * \zeta_3 * \text{Susceptible} * \text{Asymptomatic} + \zeta_2 * \zeta_3 * \text{Infected} * \text{Asymptomatic})$, saturated function $\frac{\beta * \text{Susceptible}}{1 + \zeta_1 * \text{Susceptible}}$ or $\frac{\beta * \text{Susceptible}}{1 + \zeta_2 * \text{Infected}}$ or $\frac{\beta * \text{Susceptible}}{1 + \zeta_3 * \text{Asymptomatic}}$ and specific nonlinear function $(\beta * \text{Susceptible}) / (1 + \zeta_1 * \text{Susceptible} + \zeta_2 * \text{Infected} + \zeta_3 * \text{Asymptomatic} + \zeta_4 * \text{Infected} * \text{Susceptible} + \zeta_5 * \text{Susceptible} * \text{Asymptomatic} + \zeta_6 * \text{Infected} * \text{Asymptomatic})$ all describe the non-linear relationship of the susceptible, infected and asymptomatic compartments in one or another way. The definition of the parameter used in these incidence rates also makes them different in one or another way.



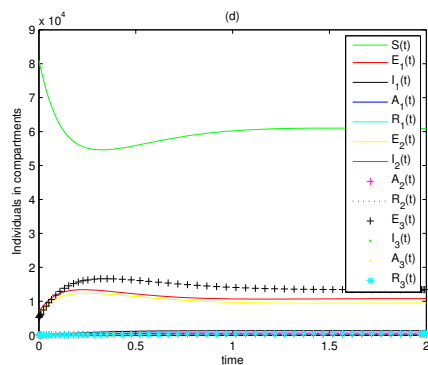
(a) Incidence rate as per Case:(a) in Table 1



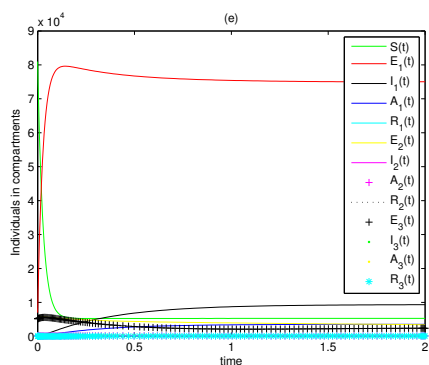
(b) Incidence rate as per Case:(b) in Table 1



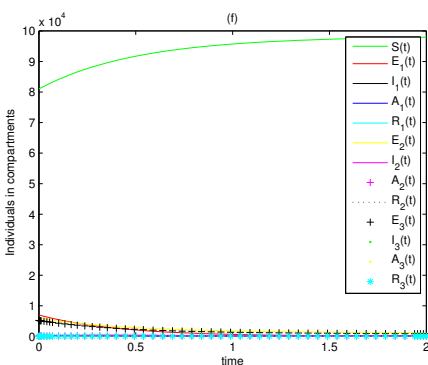
(c) Incidence rate as per Case:(c) in Table 1



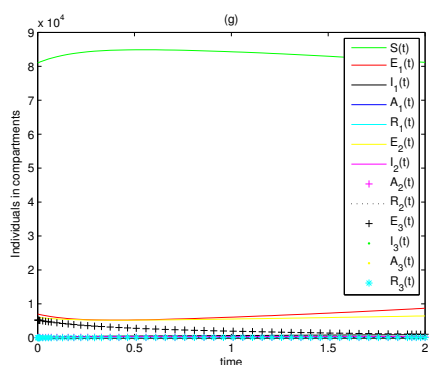
(d) Incidence rate as per Case:(d) in Table 1



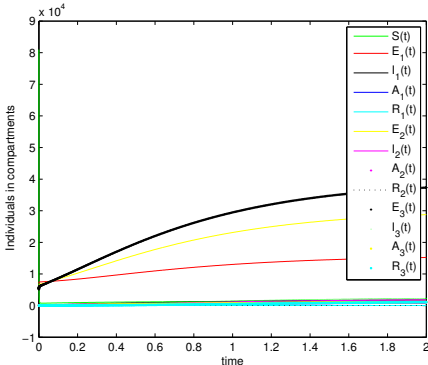
(e) Incidence rate as per Case:(e) in Table 1



(f) Incidence rate as per Case:(f) in Table 1

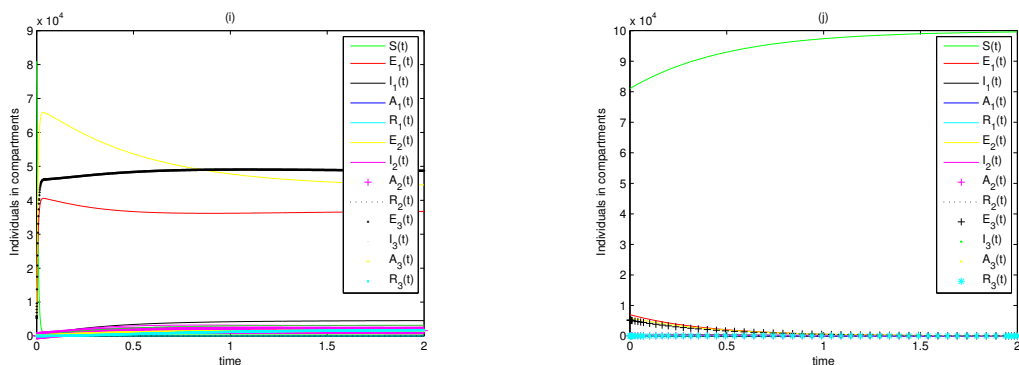


(g) Incidence rate as per Case:(g) in Table 1



(h) Incidence rate as per Case:(h) in Table 1

Figure 2. Impact of the incidence rates according to the cases in Table 1.



(i) Incidence rate as per Case:(i) in Table 1

(j) Incidence rate as per Case:(j) in Table 1

Figure 2. Impact of the incidence rates according to the cases in Table 1.

Figure 2 is the result of the simulation with initial values of the variables as {81000, 7000, 112, 100, 10, 6000, 203, 105, 60, 5200, 100, 100, 10} with hypothetical values of parameters as $\alpha_1 = 0.79$, $\alpha_2 = 0.85$, $\alpha_3 = 0.78$, $\gamma = 2$, $\delta_{11} = 0.57$, $\delta_{12} = 0.39$, $\delta_{22} = 0.3$, $\delta_{13} = 0.4$, $\delta_{21} = 0.59$, $\delta_{23} = 0.459$, $\delta_{1I} = 0.52$, $\delta_{1A} = 0.848$, $\delta_{2I} = 0.94$, $\delta_{2A} = 0.68$, $\delta_{3I} = 0.88$, $\delta_{3A} = 0.548$, $r_1 = 0.7$, $r_2 = 0.67$, $r_3 = 0.7$, $\xi_1 = 0.45$, $\xi_2 = 0.29$, $\xi_3 = 0.27$, $B = 2 * N(0)$, and the parameters included for different choices of incidence rates ($f(S, I_1, A_1)$, $g(S, I_2, A_2)$ and $h(S, I_3, A_3)$) vary.

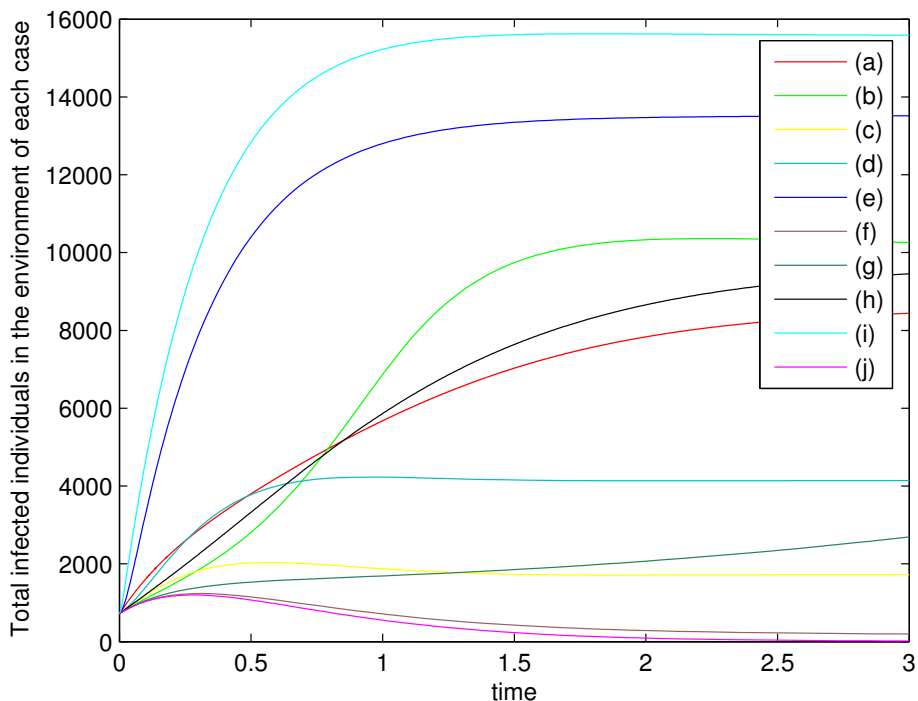


Figure 3. Impact of the incidence rates according to the cases in Table 1 on the total number of infected individuals.

Figure 2(a) has parametric values as $\beta_1 = 0.88$, $\beta_2 = 0.8$ and $\beta_3 = 0.9$ whose graphical plot shows how there is sudden decrease in the susceptible class $S(t)$ and rise in the exposed class $E_3(t)$. The decrease in the exposed class of strain 1 and strain 2 is also observed while the infected class of the third strain rises. Figure 2(b) has the parametric values $\beta_1 = 0.8$, $\beta_2 = 0.76$, $\beta_3 = 0.88$, $\zeta_1 = 0.012$, $\zeta_2 = 0.152$, $\zeta_3 = 0.035$, $\zeta_4 = 0.012$, $\zeta_5 = 0.11$, $\zeta_6 = 0.032$, $\zeta_7 = 0.014$, $\zeta_8 = 0.022$, $\zeta_9 = 0.112$. The decline in the number of individuals in the susceptible class and the wave in number of individuals in the exposed class are easily noticed at the time span of one of all strains. There is a rise in the number of infected and asymptomatic class individuals as well. Figure 2(c) has the parametric values $\beta_1 = 0.87$, $\beta_2 = 0.79$, $\beta_3 = 0.85$, $\zeta_1 = 0.018$, $\zeta_2 = 0.07$, $\zeta_3 = 0.0128$. The line plot shows a sudden decrease in the susceptible class at time 0.1, and at this period exposed classes of all the strain rises, and a little jump in infected class of respected strains are observed. Figure 2(d) has the parametric values $\beta_1 = 0.77$, $\beta_2 = 0.9$, $\beta_3 = 0.75$, $\zeta_1 = 0.012$, $\zeta_2 = 0.02$, $\zeta_3 = 0.01$ with the graphical plot similar to Figure 2(c) but large variation in the number of susceptible and exposed class individuals. Figure 2(e) has the parametric values $\beta_1 = 0.77$, $\beta_2 = 0.9$, $\beta_3 = 0.75$, $\zeta_1 = 0.03$, $\zeta_2 = 0.04$, $\zeta_3 = 0.135$, $\zeta_4 = 0.028$ with the line plot observed to have a rapid decrease in the susceptible class at time 0.1, and at this period exposed classes of strain 1 rises. Also there is a jump in infected class and asymptomatic infected class of strain 1. Figure 2(f) has the parametric values $\beta_1 = 0.77$, $\beta_2 = 0.8$, $\beta_3 = 0.85$, $\zeta_1 = 0.016$, $\zeta_2 = 0.37$, $\zeta_3 = 0.02$, $\zeta_4 = 0.015$, $\zeta_5 = 0.024$, $\zeta_6 = 0.01388$, $\zeta_7 = 0.027$, $\zeta_8 = 0.0262$, $\zeta_9 = 0.0125$, $\zeta_{10} = 0.012$, and the plot has an increase in the susceptible class individuals and a fall in the exposed classes of all the strains for the same, and a negligible increase in the infected individuals is also observed. Figure 2(g) has the parametric values $\beta_1 = 0.787$, $\beta_2 = 0.8$, $\beta_3 = 0.87$, $\zeta_1 = 0.04$, $\zeta_2 = 0.03$, $\zeta_3 = 0.06261$, and it is similar to Figure 2(f). Here, the plot indicates the certain increase in the susceptible class individuals from time 0.2 to 0.6 and decrement in the exposed classes of all the strains for the same. An increase in the infected individuals is also observed. Figure 2(h) has the parametric values $\beta_1 = 0.7$, $\beta_2 = 0.85$, $\beta_3 = 0.75$, $\zeta_1 = 0.033$, $\zeta_2 = 0.025$, $\zeta_3 = 0.016$. The two-dimensional line plot shows a sudden decrease in the susceptible class at time almost near to zero, and at this period exposed classes of all the strain rise and a little jump in the number of individuals in the other classes of respected strains is marked. Figure 2(i) has the parametric values $\beta_1 = 0.88$, $\beta_2 = 0.9$, $\beta_3 = 0.92$, $\zeta_1 = 0.02$, $\zeta_2 = 0.25$, $\zeta_3 = 0.014$ with the line plot observed to have a large decrease in the susceptible class at one third time of 0.2, and at this period exposed classes of all the strains rises. Huge variation in the exposed classes of the strains is noticed in the time span of zero to two. Also there is a jump in infected class and asymptomatic infected class of all strains. Figure 2(j) has the parametric values $\beta_1 = 0.8$, $\beta_2 = 0.9$, $\beta_3 = 0.88$, $\zeta_1 = 0.0327$, $\zeta_2 = 0.014$, $\zeta_3 = 0.035$, $\zeta_4 = 0.018$, $\zeta_5 = 0.016$, $\zeta_6 = 0.014$, $\zeta_7 = 0.01$, $\zeta_8 = 0.0145$, $\zeta_9 = 0.019$, $\zeta_{10} = 0.014$, $\zeta_{11} = 0.0217$, $\zeta_{12} = 0.019$, $\zeta_{13} = 0.0038$, $\zeta_{14} = 0.014$, $\zeta_{15} = 0.0156$, $\zeta_{16} = 0.0245$, $\zeta_{17} = 0.0128$, $\zeta_{18} = 0.017$ and is very largely similar to Figure 2(f), and the same dynamics are observed.

Figure 3 interprets the impact of the incidence rates according to the cases in Table 1 on the total number of infected individuals remarkably. Case (a), case (d), case (i), case (e) and case (h) shows the high rise in the number of infected classes. Meanwhile, case (g) and case (j) have a small rise. Case (c) and case (f) show an increase in the number of total infected individuals at the time of 0.25 and then decrease. Case (b) also increases the total number of infected individuals slowly from a time-span of zero to one. The importance of choosing an incidence rate could lead the mathematical model to interpret different scenarios clearly.

5. Results and discussion

Predictions made with the mathematical models help to control the forthcoming dangerous situations and handle them smartly. An epidemiological model (2.4) has been formulated to understand the transmission behavior of the multi-strain model with respect to the choice of incidence rate function numerically. An evaluation of the following mathematical and epidemiological results of the proposed model has been discussed in the paper:

- (i) The solutions of the system of thirteen differential Eq (2.4) is non-negative and bounded for all times $t > 0$, when initial data is non-negative (Theorem (1)). Thus, the formulated mathematical model (2.4) is mathematically and epidemiologically well-posed in Section 2.
- (ii) The three-strain epidemic model has the basic reproduction number \mathfrak{R}_0 , the maximum of the three \mathfrak{R}_{01} , \mathfrak{R}_{02} and \mathfrak{R}_{03} . \mathfrak{R}_0 in Section 3.2. The reproduction number \mathfrak{R}_0 defines the number of new infections at the time of emerging of a new strain. The three-strain model has a disease-free equilibrium (\mathcal{DFE}) with local-asymptotic stability whenever the associated basic reproduction number \mathfrak{R}_0 (\mathbb{E}_0) is lesser than a unit (Theorem 3). Biologically, if the reproduction number is less than a unit, an infectious host will get exposed to less than a host in the tenure of his infection, leading to contraction in the number of cases of the infection.
- (iii) The proposed model has eight endemic equilibrium according to the strain influence and its impact in Section 3.2. The existence of the strains and their nature of co-existence and competence leads to the scenarios discussed in the Theorem (3).
- (iv) The effect of incidence rate choice leads to different effects on the number of infected persons. Each incidence rate type has equal importance and definition defined in Section 4. Biologically in the transmission of the infection different compartments plays role differently that is described through incidence rate in the differential equation modeling. In both epidemic and virus dynamics models, this general incidence function can represent a wide range of potential incidence functions. The general incidence at which people transition from the class of susceptible individuals to the class of infective individuals has been the focus of such epidemiological models. These common incidences have primarily been modeled using functional responses of the bilinear and Holling types [21, 29]. Such tactics may involve treatments, vaccines, isolation and educational campaigns for epidemic diseases [44, 45]. Mathematical models are now crucial tools for understanding how infectious diseases spread and are managed. Incidence function decides the effect of the variables and parameters on the virus spread; hence, it is important to consider the type of function needed according to the flowchart of the infection.

6. Conclusions

Incidence rates play a crucial role in the epidemiological model formulation. The rapid appearance of any disease can be governed totally by the incidence rate equal to the affected host population in the tenure. Choosing correct incidence rate during the mathematical model formulation could give accurate predictions. Every infectious disease mutates following the host and environment, which shows the importance of multi-strain mathematical models. A variant of an infectious disease can form an epidemic or pandemic during the period.

Use of AI tools declaration

The authors declare they have not used artificial intelligence (AI) tools in the creation of this article.

Conflict of interest

The authors have no words of conflict.

References

1. W. O. Kermack, A. G. McKendrick, A contribution to the mathematical theory of epidemics, *Proc. R. Soc. Lond. A.*, **115** (1927), 700–721. <https://doi.org/10.1098/rspa.1927.0118>
2. J. Murray, *Mathematical Biology*, Springer-Verlag, Heidelberg, **1** (2002).
3. F. Brauer, C. C. Chavez, Z. Feng, *Mathematical Models in Epidemiology*, Springer, **32** (2019). <https://doi.org/10.1007/978-1-4939-9828-9>
4. S. Garmer, R. Lynn, D. Rossi, A. Capaldi, Multistrain infections in metapopulations, *Spora*, **1** (2015), 17–27. <https://doi.org/10.30707/SPORA1.1Garmer>
5. E. Cirulli, D. Goldstein, Uncovering the roles of rare variants in common disease through whole-genome sequencing, *Nat. Rev. Genet.*, **11** (2010), 415–425. <https://doi.org/10.1038/nrg2779>
6. M. Cascella, M. Rajnik, A. Aleem, *Features, Evaluation, and Treatment of Coronavirus (COVID-19)*, StatPearls Publishing, 2022.
7. Z. Yaagoub, K. Allali, Global stability of multi-strain SEIR epidemic model with vaccination strategy, *Math. Comput. Appl.*, **28** (2023), 9. <https://doi.org/10.3390/mca28010009>
8. T. Sardar, I. Ghosh, X. Rodó, J. Chattopadhyay, A realistic two-strain model for MERS-CoV infection uncovers the high risk for epidemic propagation, *PLoS Neglected Trop. Dis.*, **14** (2020), e0008065. <https://doi.org/10.1371/journal.pntd.0008065>
9. M. A. Kuddus, E. S. McBryde, A. I. Adekunle, M. T. Meehan, Analysis and simulation of a two-strain disease model with nonlinear incidence, *Chaos Solitons Fractals*, **155** (2022). <https://doi.org/10.1016/j.chaos.2021.111637>
10. V. P. Bajiya, J. P. Tripathi, V. Kakkar, J. Wang, G. Sun, Global dynamics of a multi-group SEIR epidemic model with infection age, *Chin. Ann. Math. Ser. B*, **42** (2021), 833–860. <https://doi.org/10.1007/s11401-021-0294-1>
11. E. F. Arruda, S. S. Das, C. M. Dias, D. H. Pastore, Modelling and optimal control of multi strain epidemics, with application to COVID-19, *PLoS ONE*, **16** (2021). <https://doi.org/10.1371/journal.pone.0257512>
12. S. Ghosh, M. Banerjee, A multi-strain model for COVID-19, in *Applied Analysis, Applied Analysis, Optimization and Soft Computing*, 2022. https://doi.org/10.1007/978-981-99-0597-3_10

13. S. W. X. Ong, C. J. Chiew, L. W. Ang, T. Mak, L. Cui, M. P. H. S. Toh, et al., Clinical and virological features of severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) variants of concern: A retrospective cohort study comparing B.1.1.7 (Alpha), B.1.351 (Beta), and B.1.617.2 (Delta), *Clin. Infect. Dis.*, **75** (2021), e1128–e1136. <https://doi.org/10.1093/cid/ciab721>
14. M. Massard, R. Eftimie, A. Perasso, B. Saussereau, A multi-strain epidemic model for COVID-19 with infected and asymptomatic cases: application to French data, *J. Theor. Biol.*, **545** (2022), 111117. <https://doi.org/10.1016/j.jtbi.2022.111117>
15. Z. Abreu, G. Cantin, C. J. Silva, Analysis of a COVID-19 compartmental model: a mathematical and computational approach, *Math. Biosci. Eng.*, **18** (2021), 7979–7998. <https://doi.org/10.3934/mbe.2021396>
16. World Health Organization, Coronavirus World Health Organization 19. Available from: <https://www.who.int/health-topics/coronavirus>.
17. Center for Disease Control and prevention (CDC). Available from: <https://www.cdc.gov/coronavirus/2019-ncov/variants/variant-classifications.html>
18. National Center for Biotechnology Information (NCBI). Available from: <https://www.ncbi.nlm.nih.gov/activ>.
19. G. Rohith, K. B. Devika, Dynamics and control of COVID-19 pandemic with nonlinear incidence rates, *Nonlinear Dyn.*, **101** (2020), 2013–2026. <https://doi.org/10.1007/s11071-020-05774-5>
20. S. Ruan, W. Wang, Dynamical behavior of an epidemic model with a nonlinear incidence rate, *J. Diff. Equation*, **188** (2003), 135–163. [https://doi.org/10.1016/S0022-0396\(02\)00089-X](https://doi.org/10.1016/S0022-0396(02)00089-X)
21. H. W. Hethcote, P. van den Driessche, Some epidemiological models with nonlinear incidence, *J. Math. Biol.*, **29** (1991), 271–287. <https://doi.org/10.1007/bf00160539>
22. G. Huang, Y. Takeuchi, W. Ma, D. Wei, Global stability for delay SIR and SEIR epidemic models with nonlinear incidence rate, *Bull. Math. Biol.*, **72** (2010), 1192–1207. <https://doi.org/10.1007/s11538-009-9487-6>
23. W. M. Liu, S. A. Levin, Y. Iwasa, Influence of nonlinear incidence rates upon the behavior of SIRS epidemiological models, *J. Math. Biol.*, **23** (1986), 187–204. <https://doi.org/10.1007/BF00276956>
24. J. Arino, C. C. McCluskey, Effect of a sharp change of the incidence function on the dynamics of a simple disease, *J. Bio. Dyn.*, **4** (2010), 490–505. <https://doi.org/10.1080/17513751003793017>
25. N. Bellomo, R. Bingham, M. A. J. Chaplain, G. Dosi, G. Forni, D. A. Knopoff, et al., A multiscale model of virus pandemic: Heterogeneous interactive entities in a globally connected world, *Math. Models Methods Appl. Sci.*, **30** (2020), 1591–1651. <https://doi.org/10.1142/s0218202520500323>
26. J. J. Wang, J. Z. Zhang, Z. Jin, Analysis of an SIR model with bilinear incidence rate, *Nonlinear Anal. Real World Appl.*, **11** (2010), 2390–2402. <https://doi.org/10.1016/j.nonrwa.2009.07.012>
27. O. Khyar, K. Allali, Global dynamics of a multi-strain SEIR epidemic model with general incidence rates: application to COVID-19 pandemic, *Nonlinear Dyn.*, (2020), 1–21. <https://doi.org/10.1007/s11071-020-05929-4>
28. X. Liu, L. Yang, Stability analysis of an SEIQV epidemic model with saturated incidence rate, *Nonlinear Anal. Real World Appl.*, **13** (2012), 2671–2679. <https://doi.org/10.1016/j.nonrwa.2012.03.010>

29. A. Kaddar, On the dynamics of a delayed SIR epidemic model with a modified saturated incidence rate, *Electron. J. Differ. Equation*, **13** (2009), 1–7.
30. Y. Zhao, D. Jiang, The threshold of a stochastic SIRS epidemic model with saturated incidence, *Appl. Math. Lett.*, **34** (2014), 90–93. <https://doi.org/10.3934/dcdsb.2015.20.1277>
31. X. Q. Liu, S. M. Zhong, B. D. Tian, F. X. Zheng, Asymptotic properties of a stochastic predator-prey model with Crowley–Martin functional response, *J. Appl. Math. Comput.*, **43** (2013), 479–490. <https://doi.org/10.1007/s12190-013-0674-0>
32. J. R. Beddington, Mutual interference between parasites or predators and its effect on searching efficiency, *J. Anim. Ecol.*, **44** (1975), 331–341. <https://doi.org/10.2307/3866>
33. R. S. Cantrell, C. Cosner, On the dynamics of predator-prey models with the Beddington–DeAngelis functional response, *J. Math. Anal. Appl.*, **257** (2001), 206–222. <https://doi.org/10.1006/JMAA.2000.7343>
34. X. Zhou, J. Cui, Global stability of the viral dynamics with Crowley–Martin functional response, *Bull. Korean Math. Soc.*, **48** (2011), 555–574. <https://doi.org/10.1002/mma.3078>
35. K. Hattaf, N. Yousfi, A. Tridane, Mathematical analysis of a virus dynamics model with general incidence rate and cure rate, *Nonlinear Anal. Real World Appl.*, **13** (2012), 1866–1872. <https://doi.org/10.1016/j.nonrwa.2011.12.015>
36. K. Hattaf, M. Mahrouf, J. Adnani, N. Yousfi, Qualitative analysis of a stochastic epidemic model with specific functional response and temporary immunity, *Phys. A.*, **490** (2018), 591–600. <https://doi.org/10.1016/j.physa.2017.08.043>
37. K. Hattaf, N. Yousfi, Global dynamics of a delay reaction–diffusion model for viral infection with specific functional response, *Comput. Appl. Math.*, **34** (2015), 807–818. <https://doi.org/10.1007/s40314-014-0143-x>
38. K. Hattaf, N. Yousfi, A. Tridane, Stability analysis of a virus dynamics model with general incidence rate and two delays, *Appl. Math. Comput.*, **221** (2013), 514–521. <https://doi.org/10.1016/j.amc.2013.07.005>
39. M. K. Singh, Anjali, Mathematical modeling and analysis of seqiahr model: Impact of quarantine and isolation on COVID-19, *App. App. Math.*, **17** (2022), 146–171.
40. S. Sharma, V. Volpert, M. Banerjee, Extended SEIQR type model for covid-19 epidemic and data analysis, *Math. Biosci. Eng.*, (2020), 7562–7604. <https://doi.org/10.3934/mbe.2020386>
41. P. Kim, S. M. Gordon, M. M. Sheehan, M. B. Rothberg, Duration of severe acute respiratory syndrome Coronavirus 2 natural immunity and protection against the Delta variant: A retrospective cohort study, *Clin. Infect. Dis.*, **75** (2022), 185–190. <https://doi.org/10.1093/cid/ciab999>
42. 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](https://doi.org/10.1016/s0025-5564(02)00108-6)
43. D. Bentaleb, S. Amine, Lyapunov function and global stability for a two-strain SEIR model with bilinear and non-monotone, *Int. J. Biomath.*, **12** (2019). <https://doi.org/10.1142/S1793524519500219>

44. F. G. Ball, E. S. Knock, P. D. O'Neil, Control of emerging infectious diseases using responsive imperfect vaccination and isolation, *Math. Biosci.*, **216** (2008), 100–113. <https://doi.org/10.1016/j.mbs.2008.08.008>
45. C. Castillo, Optimal control of an epidemic through educational campaigns, *Electron. J. Differ. Equation*, (2006), 1–11.

Appendix

Entries of the Jacobian matrix of order thirteen associated with the system (2.4) are

$$\begin{aligned}
 a_{1,1} &= -\gamma - \frac{\partial f(S, I_1, A_1)}{\partial S}(I_1 + \alpha_1 A_1) - \frac{\partial g(S, I_2, A_2)}{\partial S}(I_2 + \alpha_2 A_2) - \frac{\partial h(S, I_3, A_3)}{\partial S}(I_3 + \alpha_3 A_3), \\
 a_{1,3} &= -\frac{\partial f(S, I_1, A_1)}{\partial I_1}(I_1 + \alpha_1 A_1) - f(S, I_1, A_1), \\
 a_{1,4} &= -\frac{\partial f(S, I_1, A_1)}{\partial A_1}(I_1 + \alpha_1 A_1) - \alpha_1 f(S, I_1, A_1), \\
 a_{1,7} &= -\frac{\partial g(S, I_2, A_2)}{\partial I_2}(I_2 + \alpha_2 A_2) - g(S, I_2, A_2), \\
 a_{1,8} &= -\frac{\partial g(S, I_2, A_2)}{\partial A_2}(I_2 + \alpha_2 A_2) - \alpha_2 g(S, I_2, A_2), \\
 a_{1,11} &= -\frac{\partial h(S, I_3, A_3)}{\partial I_3}(I_3 + \alpha_3 A_3) - h(S, I_3, A_3), \\
 a_{1,12} &= -\frac{\partial h(S, I_3, A_3)}{\partial A_3}(I_3 + \alpha_3 A_3) - \alpha_3 h(S, I_3, A_3), \\
 a_{2,1} &= \left\{ \frac{\partial f(S, I_1, A_1)}{\partial S} \left(1 + \delta_{11} \frac{R_1}{S} + \delta_{12} \frac{R_2}{S} \right) - f(S, I_1, A_1) \left(\delta_{11} \frac{R_1}{S^2} + \delta_{12} \frac{R_2}{S^2} \right) \right\} (I_1 + \alpha_1 A_1), \\
 a_{2,2} &= -\xi_1 - \gamma, \\
 a_{2,3} &= \frac{\partial f(S, I_1, A_1)}{\partial I_1} \left(1 + \delta_{11} \frac{R_1}{S} + \delta_{21} \frac{R_2}{S} \right) (I_1 + \alpha_1 A_1) + f(S, I_1, A_1) \left(1 + \delta_{11} \frac{R_1}{S} + \delta_{21} \frac{R_2}{S} \right), \\
 a_{2,4} &= \frac{\partial f(S, I_1, A_1)}{\partial A_1} \left(1 + \delta_{11} \frac{R_1}{S} + \delta_{21} \frac{R_2}{S} \right) (I_1 + \alpha_1 A_1) + \alpha_1 f(S, I_1, A_1) \left(1 + \delta_{11} \frac{R_1}{S} + \delta_{21} \frac{R_2}{S} \right), \\
 a_{2,5} &= \delta_{11} f(S, I_1, A_1) \frac{(I_1 + \alpha_1 A_1)}{S}, \\
 a_{3,2} &= r_1 \xi_1, \\
 a_{3,3} &= -\delta_{1I} - \gamma, \\
 a_{4,2} &= (1 - r_1) \xi_1, \\
 a_{4,4} &= -\delta_{1A} - \gamma, \\
 a_{5,1} &= \frac{R_1}{S} \left\{ \delta_{11} f(S, I_1, A_1) (I_1 + \alpha_1 A_1) + \delta_{12} g(S, I_2, A_2) (I_2 + \alpha_2 A_2) + \delta_{13} h(S, I_3, A_3) (I_3 + \alpha_3 A_3) \right. \\
 &\quad \left. - \delta_{11} \frac{\partial f(S, I_1, A_1)}{\partial S} (I_1 + \alpha_1 A_1) - \delta_{12} \frac{\partial g(S, I_2, A_2)}{\partial S} (I_2 + \alpha_2 A_2) - \delta_{13} \frac{\partial h(S, I_3, A_3)}{\partial S} (I_3 + \alpha_3 A_3) \right\},
 \end{aligned}$$

$$\begin{aligned}
a_{5,3} &= -\delta_{11}\delta_{11}\frac{R_1}{S}\left\{\frac{\partial f(S, I_1, A_1)}{\partial I_1}(I_1 + \alpha_1 A_1) + f(S, I_1, A_1)\right\}, \\
a_{5,4} &= -\delta_{11}\delta_{11}\frac{R_1}{S}\left\{\frac{\partial f(S, I_1, A_1)}{\partial A_1}(I_1 + \alpha_1 A_1) + \alpha_1 f(S, I_1, A_1)\right\}, \\
a_{5,5} &= -\{\delta_{11}f(S, I_1, A_1)(I_1 + \alpha_1 A_1) + \delta_{12}g(S, I_2, A_2)(I_2 + \alpha_2 A_2) + \delta_{13}h(S, I_3, A_3)(I_3 \\
&\quad + \alpha_3 A_3)\}/S - \gamma, \\
a_{5,7} &= -\delta_{12}\frac{R_1}{S}\left\{\frac{\partial g(S, I_2, A_2)}{\partial I_2}(I_2 + \alpha_2 A_2) + g(S, I_2, A_2)\right\}, \\
a_{5,8} &= -\delta_{12}\frac{R_1}{S}\left\{\frac{\partial g(S, I_2, A_2)}{\partial A_2}(I_2 + \alpha_2 A_2) + \alpha_2 g(S, I_2, A_2)\right\}, \\
a_{5,11} &= -\delta_{13}\frac{R_1}{S}\left\{\frac{\partial h(S, I_3, A_3)}{\partial I_3}(I_3 + \alpha_3 A_3) + h(S, I_3, A_3)\right\}, \\
a_{5,12} &= -\delta_{13}\frac{R_1}{S}\left\{\frac{\partial h(S, I_3, A_3)}{\partial A_3}(I_3 + \alpha_3 A_3) + \alpha_3 h(S, I_3, A_3)\right\}, \\
a_{6,1} &= \left\{\frac{\partial g(S, I_2, A_2)}{\partial S}(1 + \delta_{21}\frac{R_1}{S} + \delta_{22}\frac{R_2}{S}) - g(S, I_2, A_2)(\delta_{21}\frac{R_1}{S^2} + \delta_{22}\frac{R_2}{S^2})\right\}(I_2 + \alpha_2 A_2), \\
a_{6,5} &= \delta_{12}g(S, I_2, A_2)\frac{(I_2 + \alpha_2 A_2)}{S}, \\
a_{6,6} &= -\xi_2 - \gamma, \\
a_{6,7} &= \frac{\partial g(S, I_2, A_2)}{\partial I_2}(1 + \delta_{12}\frac{R_1}{S} + \delta_{22}\frac{R_2}{S})(I_2 + \alpha_2 A_2) + g(S, I_2, A_2)(1 + \delta_{12}\frac{R_1}{S} + \delta_{22}\frac{R_2}{S}), \\
a_{6,8} &= \frac{\partial g(S, I_2, A_2)}{\partial A_2}(1 + \delta_{12}\frac{R_1}{S} + \delta_{22}\frac{R_2}{S})(I_2 + \alpha_2 A_2) + \alpha_2 g(S, I_2, A_2)(1 + \delta_{12}\frac{R_1}{S} + \delta_{22}\frac{R_2}{S}), \\
a_{6,9} &= \delta_{22}g(S, I_2, A_2)\frac{(I_2 + \alpha_2 A_2)}{S}, \\
a_{7,6} &= r_2\xi_2, \\
a_{7,7} &= -\delta_{2I} - \gamma, \\
a_{8,6} &= (1 - r_2)\xi_2, \\
a_{8,8} &= -\delta_{2A} - \gamma, \\
a_{9,1} &= \frac{R_2}{S}\{\delta_{21}f(S, I_1, A_1)(I_1 + \alpha_1 A_1) + \delta_{22}g(S, I_2, A_2)(I_2 + \alpha_2 A_2) \\
&\quad + \delta_{23}h(S, I_3, A_3)(I_3 + \alpha_3 A_3) - \delta_{21}\frac{\partial f(S, I_1, A_1)}{\partial S}(I_1 + \alpha_1 A_1) - \delta_{22}\frac{\partial g(S, I_2, A_2)}{\partial S}(I_2 \\
&\quad + \alpha_2 A_2) - \delta_{23}\frac{\partial h(S, I_3, A_3)}{\partial S}(I_3 + \alpha_3 A_3)\}, \\
a_{9,3} &= -\delta_{21}\frac{R_2}{S}\left\{\frac{\partial f(S, I_1, A_1)}{\partial I_1}(I_1 + \alpha_1 A_1) + f(S, I_1, A_1)\right\}, \\
a_{9,4} &= -\delta_{21}\frac{R_2}{S}\left\{\frac{\partial f(S, I_1, A_1)}{\partial A_1}(I_1 + \alpha_1 A_1) + \alpha_1 f(S, I_1, A_1)\right\}, \\
a_{9,7} &= -\delta_{21}\delta_{22}\frac{R_2}{S}\left\{\frac{\partial g(S, I_2, A_2)}{\partial I_2}(I_2 + \alpha_2 A_2) + g(S, I_2, A_2)\right\}, \\
a_{9,8} &= -\delta_{2A}\delta_{22}\frac{R_2}{S}\left\{\frac{\partial g(S, I_2, A_2)}{\partial A_2}(I_2 + \alpha_2 A_2) + \alpha_2 g(S, I_2, A_2)\right\},
\end{aligned}$$

$$\begin{aligned}
a_{9,9} &= -\{\delta_{21}f(S, I_1, A_1)(I_1 + \alpha_1 A_1) + \delta_{22}g(S, I_2, A_2)(I_2 + \alpha_2 A_2) + \delta_{23}h(S, I_3, A_3)(I_3 \\
&\quad + \alpha_3 A_3)\}/S - \gamma, \\
a_{9,11} &= -\delta_{23} \frac{R_2}{S} \left\{ \frac{\partial h(S, I_3, A_3)}{\partial I_3} (I_3 + \alpha_3 A_3) + h(S, I_3, A_3) \right\}, \\
a_{9,12} &= -\delta_{23} \frac{R_2}{S} \left\{ \frac{\partial h(S, I_3, A_3)}{\partial A_3} (I_3 + \alpha_3 A_3) + \alpha_3 h(S, I_3, A_3) \right\}, \\
a_{10,1} &= \left(\frac{\partial h(S, I_3, A_3)}{\partial S} (1 + \delta_{13} \frac{R_1}{S} + \delta_{23} \frac{R_2}{S}) - h(S, I_3, A_3) (\delta_{13} \frac{R_1}{S^2} + \delta_{23} \frac{R_2}{S^2}) \right) (I_3 + \alpha_3 A_3), \\
a_{10,5} &= \delta_{13} h(S, I_3, A_3) \frac{(I_3 + \alpha_3 A_3)}{S}, \\
a_{10,9} &= \delta_{23} h(S, I_3, A_3) \frac{(I_3 + \alpha_3 A_3)}{S}, \\
a_{10,10} &= -\xi_3 - \gamma, \\
a_{10,11} &= \frac{\partial h(S, I_3, A_3)}{\partial I_3} (1 + \delta_{13} \frac{R_1}{S} + \delta_{23} \frac{R_2}{S}) (I_3 + \alpha_3 A_3) + h(S, I_3, A_3) (1 + \delta_{13} \frac{R_1}{S^2} + \delta_{23} \frac{R_2}{S^2}), \\
a_{10,12} &= \frac{\partial h(S, I_3, A_3)}{\partial I_3} (1 + \delta_{13} \frac{R_1}{S} + \delta_{23} \frac{R_2}{S}) (I_3 + \alpha_3 A_3) + h(S, I_3, A_3) (1 + \delta_{13} \frac{R_1}{S^2} + \alpha_3 \delta_{23} \frac{R_2}{S^2}), \\
a_{11,10} &= r_3 \xi_3, \\
a_{11,11} &= -\delta_{3I} - \gamma, \\
a_{12,10} &= (1 - r_3) \xi_3, \\
a_{12,12} &= -\delta_{3A} - \gamma, \\
a_{13,11} &= \delta_{3I}, \\
a_{13,12} &= \delta_{3A}, \\
a_{13,13} &= -\gamma.
\end{aligned}$$



AIMS Press

©2023 the Author(s), licensee AIMS Press. This is an open access article distributed under the terms of the Creative Commons Attribution License (<http://creativecommons.org/licenses/by/4.0>)