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Mathematical Analysis of 2-Strain Endemic Equilibrium using a SEIR Model with Behavioral Effect

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Abstract. Multi-strain epidemic dynamics have been analyzed using two-strain SEIR models. We have taken into consideration a two-strain SEIR model that incorporates behavioural alterations for strain 2 among the susceptibles, with non-monotone incidence for strain 2 and bilinear incidence for strain 1. We have focused on the co-dynamics of both strains and analytically derived a sufficient condition, $1+\mathcal{R}_k^* < \mathcal{R}_{01} < \mathcal{R}_{02}$, for the existence of the two-strain endemic equilibrium, where $\mathcal{R}_{01}$ and $\mathcal{R}_{02}$ are basic reproduction numbers of respective strains and, $\mathcal{R}_k^* > 0$ is a value derived from endemic state of the model. Under this sufficient condition, we establish the global stability of the two-strain endemic equilibrium using the Lyapunov function method in terms of reproduction numbers and behavioural parameter k. Additionally, the condition, $\mathcal{R}_{02} = \mathcal{R}_{01} > 1$, result in instability of the two-strain endemic equilibrium. Lastly, to support these analytical results regarding the existence and stability of the two-strain endemic equilibrium, we have presented numerical simulations. Interestingly, the study reveals that the principle of competitive exclusion relaxing in this model, as behavioural heterogeneity among susceptibles effectively partitions the population. This partitioning reduces inter-strain competition and enables strain coexistence even when $\mathcal{R}_{02} > \mathcal{R}_{01} > 1$.

Key words and Phrases: multi-strain epidemic dynamics, behavioural heterogeneity, Lyapunov stability, strain coexistence, principle of competitive exclusion.

1. INTRODUCTION

Mathematical modelling of infectious diseases has evolved over more than two centuries, beginning with early formal attempts by Daniel Bernoulli, who analysed smallpox dynamics in 1766 [1, 2], and followed by William Farr's statistical studies of cholera outbreaks in 1852 [3, 2]. Later, Ronald Ross developed a

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threshold-based compartmental framework for malaria transmission between 1908 and 1911 [4, 5], providing a conceptual foundation for modern infectious-disease modelling. This foundation was significantly strengthened in 1927 by Kermack and McKendrick, who introduced the classical SIR model and the epidemic threshold theorem [6, 7, 8]. Subsequent extensions, including SEIR-type formulations incorporating latent periods [9, 10], and the formalization of the modern use of the basic reproduction number ($\mathcal{R}_0$) by Dietz, Anderson, and May [11], shaped contemporary epidemiological theory.

Despite major advances in the 21st century, infectious diseases continue to impose substantial global burdens. The recent COVID-19 pandemic illustrated the difficulty of managing multi-strain diseases driven by emergent variants [12]. Similar mutational mechanisms are observed in diseases such as tuberculosis, dengue, and HIV [13, 14, 15]. Modelling these systems requires accounting for multi-strain structure and incidence mechanisms that incorporate infectiousness, behavioural responses, public awareness, and healthcare availability.

Different strains often vary in both their transmissibility and their perceived severity, leading to behavioural heterogeneity among susceptible individuals. As a result, distinct incidence functions may more realistically capture strain-specific transmission. The literature documents several incidence functions, including bilinear [16], saturated [17, 18], Beddington–DeAngelis [19, 20], Crowley–Martin [21, 22], and non-monotone structures [23, 24, 25, 26].

Researchers have therefore developed multi-strain SEIR models. Some studies [27, 28, 29] assume bilinear incidence rate for all strains, while others [30, 31] incorporate non-monotone incidence functions. A recent study [32] considers general incidence forms for all strains and derives necessary conditions for equilibria and global stability under certain structural assumptions. However, the epidemiological interpretation of these assumptions is not clearly articulated, and sufficient conditions for the existence of equilibria are not explicitly established. Moreover, all of these studies effectively impose homogeneous behavioural responses across strains, which limits their ability to capture realistic behaviour-driven differences in transmission. Despite these advances, there remains no general framework that provides behaviourally interpretable, mathematically rigorous conditions for the existence and stability of two-strain endemic equilibria under heterogeneous incidence structures.

Behavioural heterogeneity plays an important role in multi-strain outbreaks. Populations often react more cautiously to newly emerged or mutated strains because of uncertainty and perceived risk, whereas older, better-understood strains tend to elicit weaker precautionary responses. This motivates modelling the original strain using bilinear incidence and the mutated strain using non-monotone incidence. Such a formulation captures realistic, behaviour-driven differences in contact patterns. A model of this type is analysed in [33]. For such multi-strain diseases, understanding strain coexistence requires analysing the co-dynamics before either strain is eradicated. Thus, the study of the two-strain endemic equilibrium is a foundational step.

The authors in [33] show that a two-strain (co-existence) endemic equilibrium exists and is globally stable if $\mathcal{R}_{01} = \mathcal{R}_{02} > 1$. However, because the strains differ in transmission mechanisms and behavioural responses, and because the condition does not involve any behavioural parameter, this symmetry requirement is rarely satisfied in realistic settings. Motivated by this, we re-examine the co-dynamics of a two-strain SEIR model incorporating behavioural heterogeneity and derive general, epidemiologically interpretable conditions for the existence and stability of a two-strain endemic equilibrium.

In this article, we study a two-strain SEIR model in which one strain follows bilinear incidence and the other a non-monotone incidence rate reflecting behavioural effect. The key contributions of this manuscript are: (i) the development of mathematically rigorous, necessary and sufficient conditions for the existence of the two-strain endemic equilibrium; (ii) the formulation of global stability criteria expressed in terms of the basic reproduction numbers and the behavioural parameter; and (iii) the epidemiological interpretation of these analytical conditions. Finally, all analytical results are supported by numerical simulations to verify consistency between theory and model behaviour.

2. Model Description

We consider a two-strain SEIR model, as illustrated in Figure (1), with a bilinear incidence rate for strain 1 and a non-monotone incidence rate for strain 2 to reflect behavioural changes among susceptible individuals. The model consists of six compartments: susceptible (S), exposed to strain-1 and strain-2 (E_1, E_2), infectious with strain-1 and strain-2 (I_1, I_2), and recovered (R), described in Table (1); following the structure in [33] with a key modification.
To capture behavioural responses to strain–2, we adopt the non-monotone incidence function

$$\frac{\alpha_2 S I_2}{1 + k I_2^2},$$

with $k > 0$. Since behavioural responses to infection typically intensify in a nonlinear manner as disease signals become more prominent, the quadratic term represents the minimal nonlinear form capable of reflecting this effect. It provides a stronger-than-linear reduction in transmission while avoiding the unnecessary complexity, instability, or steepness introduced by cubic or higher-order exponents. In [33], the parameter k is considered in the range $[0, 1]$, which accounts for mild to moderate behavioural effects. However, empirical studies and simulations (e.g., [34] and [35]) show that stronger behavioural responses may occur in severe outbreaks. To capture this, we extend the model by allowing $k > 0$, including values greater than 1. We exclude the case $k = 0$, which corresponds to a purely monotonic incidence rate, as our focus is on dynamics impacted by behavioural modifications. Transitions between model compartments are represented by various parameters described in Table (1).

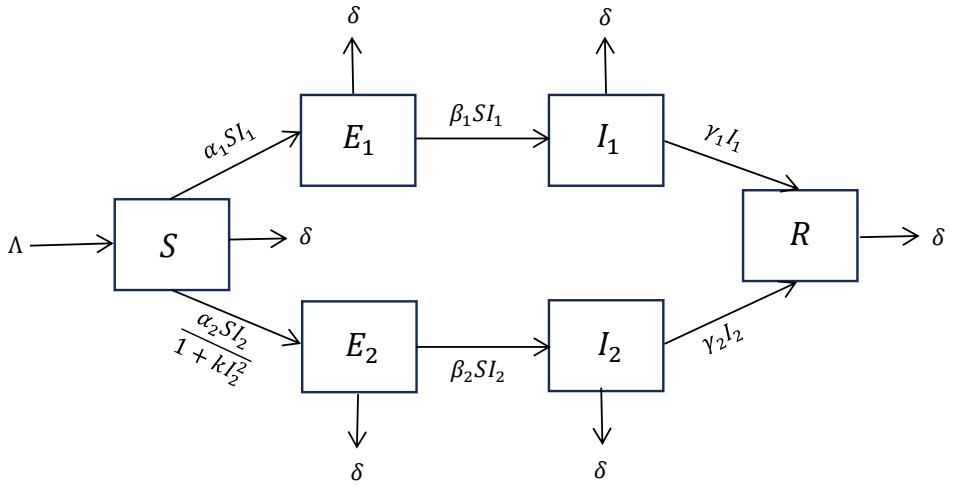


FIGURE 1. Schismatic Diagram

We have chosen this compartmental model over others as it has separate compartments for each strain, which help to understand the dynamics of each strain separately. Also, it is not complicating the dynamics as in models with more compartments. We are considering cross-immunity between strains for simplicity and better understanding of co-existence dynamics [36]. There is no loss of temporary immunity, and so the recovered individuals stay in the recovered compartment (R).

TABLE 1. Description of Parameter for Model (1)

Variable	Description
$S(t)$	Susceptible individuals
$E_1(t)$	Exposed individuals infected with strain-1
$E_2(t)$	Exposed individuals infected with strain-2
$I_1(t)$	Infected individuals with strain-1
$I_2(t)$	Infected individuals with strain-2
$R(t)$	Recovered individual
Parameters	**Description**
Λ	Recruitment rate
$1/\delta$	Average mortality rate
α_1	Infection rate of strain-1
α_2	Infection rate of strain-2
$1/\beta_1$	Average incubation period of strain-1
$1/\beta_2$	Average incubation period of strain-2
γ_1	Recovery rate of strain-1
γ_2	Recovery rate of strain-2
k	Parameter of behavioural response

2.1. **Mathematical Equations of Model.**
Based on the model description given above and (1) along with the model diagram in Figure (1), following equations govern the model:

$$\begin{aligned}
\frac{dS}{dt} &= \Lambda - \alpha_1 S I_1 - \frac{\alpha_2 S I_2}{1 + k I_2^2} - \delta S, \\
\frac{dE_1}{dt} &= \alpha_1 S I_1 - (\beta_1 + \delta) E_1, \\
\frac{dE_2}{dt} &= \frac{\alpha_2 S I_2}{1 + k I_2^2} - (\beta_2 + \delta) E_2, \\
\frac{dI_1}{dt} &= \beta_1 E_1 - (\gamma_1 + \delta) I_1, \\
\frac{dI_2}{dt} &= \beta_2 E_2 - (\gamma_1 + \delta) I_2, \\
\frac{dR}{dt} &= \gamma_1 I_1 + \gamma_2 I_2 - \delta R.
\end{aligned} \tag{1}$$

with $S(0) \geq 0, E_1(0) \geq 0, E_2(0) \geq 0, I_1(0) \geq 0$, $I_2(0) \geq 0$ and, $R(0) \geq 0$.

2.2. **Positivity of the solutions.**

Theorem 2.1. *Any solution of the system* (1) *with non-negative initial values remains non-negative over the time.*

Proof. Let $(S(t), E_1(t), E_2(t), I_1(t), I_2(t), R(t))$ be any solution of (1) with with non-negative initial conditions for each compartment and, $\tau > 0$.
From the equation of susceptible compartment of (1), we have

$$\frac{dS}{dt} = \Lambda - (\alpha_1 I_1 + \alpha_2 I_2 + \delta) S,$$

then

$$\frac{dS}{dt} \geq -(\alpha_1 I_1 + \alpha_2 I_2 + \delta) S.$$

That is

$$\int_0^\tau \frac{1}{S} ds \geq \int_0^\tau -(\alpha_1 I_1 + \alpha_2 I_2 + \delta) dt.$$

This gives

$$S(\tau) \geq S(0) \left(1 / \exp\left(\delta\tau + \int_0^\tau [\alpha_1 I_1(t) + \alpha_2 I_2(t)] dt \right) \right) \geq 0.$$

Similarly, from the equation of all other compartments,

$$\begin{aligned}
E_1(\tau) &\geq E_1(0) \exp(-(\beta_1 + \delta)\tau)) \geq 0, \\
E_2(\tau) &\geq E_2(0) \exp(-(\beta_2 + \delta)\tau)) \geq 0, \\
I_1(\tau) &\geq E_2(0) \exp(-(\gamma_1 + \delta)\tau)) \geq 0,
\end{aligned}$$

$$I_2(\tau) \geq I_2(0)\exp(-(\gamma_2+\delta)\tau)) \geq 0,$$
$$R(\tau) \geq R(0)\exp(-\delta\tau)) \geq 0.$$

Hence the proof. □

Theorem 2.2. *Let $N = S + E_1 + E_2 + I_1 + I_2$, then the set*

$$\mathcal{D} = \{(S, E_1, E_2, I_1, I_2) \in (\mathbb{R}_+ \cup \{0\})^5 : N(t) \leq \frac{\Lambda}{\delta}\}$$

is invariant.

Proof. Let $(S(t), E_1(t), E_2(t), I_1(t), I_2(t))$ be any solution of (1) with

$$(S(0), E_1(0), E_2(0), I_1(0), I_2(0)) \in \mathcal{D}.$$

Then, we have

$$N(0) = S(0) + E_1(0) + E_2(0) + I_1(0) + I_2(0)$$

and so $N(0) \leq \frac{\Lambda}{\delta}$.
Next, by adding all the equations of the system (1), we have

$$\frac{dN}{dt} \leq \Lambda - \delta N.$$

Consequently,

$$N(t) \leq \frac{\Lambda}{\delta} + \left(N(0) - \frac{\Lambda}{\delta}\right)e^{-\delta t} = N(0)e^{-\delta t} + \frac{\Lambda}{\delta}\left((1 - e^{-\delta t}\right)$$

As $N(0) \leq \frac{\Lambda}{\delta}$, we get

$$N(t) \leq \frac{\Lambda}{\delta}, \text{ for all } t \geq 0.$$

This indicates that any solution of (1) with initial values in $\mathcal{D}$ remains in it. That is, $\mathcal{D}$ is invariant. □

2.3. **Disease-Free Equilibrium.**

Since R does not appear in any of the other equations in (1), and we are interested in studying the dynamics of the infectious population, (1) can be minimized to the system as follows:

$$\begin{aligned}
\frac{dS}{dt} &= \Lambda - \alpha_1 S I_1 - \frac{\alpha_2 S I_2}{1 + k I_2^2} - \delta S,\\
\frac{dE_1}{dt} &= \alpha_1 S I_1 - a_1 E_1,\\
\frac{dE_2}{dt} &= \frac{\alpha_2 S I_2}{1 + k I_2^2} - a_2 E_2,\\
\frac{dI_1}{dt} &= \beta_1 E_1 - b_1 I_1\\
\frac{dI_2}{dt} &= \beta_2 E_2 - b_2 I_2,
\end{aligned} \tag{2}$$

with $S(0) \geq 0, E_1(0) \geq 0, E_2(0) \geq 0, I_1(0) \geq 0$ and, $I_2(0) \geq 0$, where

$$a_1 = \beta_1 + \delta, \qquad a_2 = \beta_2 + \delta, \qquad b_1 = \gamma_1 + \delta, \qquad b_2 = \gamma_2 + \delta.$$

The disease-free equilibrium is a steady state solution of model (2) with no infectious individuals in population. Let $\mathcal{E}_0$ denote the disease-free equilibrium, then

$$\mathcal{E}_0 = \left(\frac{\Lambda}{\delta}, 0, 0, 0, 0\right). \tag{3}$$

2.4. **Basic Reproduction Number.**
To find the basic reproduction number, we use the next-generation matrix method given in [37]. Let F contain new infections and V contain all transitions involved in the model (2) be given by

$$\mathcal{F} = \begin{pmatrix} 0 \\ \alpha_1 S I_1 \\ \frac{\alpha_2 S I_2}{1+kI_2^2} \\ 0 \\ 0 \end{pmatrix}, \qquad \mathcal{V} = \begin{pmatrix} -\Lambda + \alpha_1 S I_1 + \frac{\alpha_2 S I_2}{1+kI_2^2} + \delta S \\ a_1 \\ a_2 \\ -\beta_1 E_1 + b_1 \\ -\beta_2 E_2 + b_2 \end{pmatrix}.$$

Then

$$F = D\mathcal{F}(\mathcal{E}_0) = \begin{pmatrix} 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & \alpha_1 S_0^* & 0 \\ 0 & 0 & 0 & 0 & \alpha_2 S_0^* \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \end{pmatrix},$$

$$V = D\mathcal{V}(\mathcal{E}_0) = \begin{pmatrix} \delta & 0 & 0 & 0 & 0 \\ 0 & a_1 & 0 & 0 & 0 \\ 0 & 0 & a_2 & 0 & 0 \\ 0 & -\beta_1 & 0 & b_1 & 0 \\ 0 & 0 & -\beta_2 & 0 & b_2 \end{pmatrix},$$

be the Jacobian matrices of $\mathcal{F}$ and $\mathcal{V}$ respectively. Therefore, the next generation matrix is given by

$$FV^{-1} = \begin{pmatrix} 0 & 0 & 0 & 0 & 0 \\ 0 & \frac{\alpha_1\beta_1 S_0^*}{a_1 b_1} & 0 & \frac{\alpha_1 S_0^*}{b_1} & 0 \\ 0 & 0 & \frac{\alpha_2\beta_2 S_0^*}{a_2 b_2} & 0 & \frac{\alpha_2 S_0^*}{b_2} \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \end{pmatrix}.$$

The basic reproduction number $\mathcal{R}_0$ is given by the spectral radius of the matrix FV^{-1}. So

$$\mathcal{R}_0 = max\left\{\frac{\alpha_1\beta_1 S_0^*}{a_1 b_1}, \frac{\alpha_2\beta_2 S_0^*}{a_2 b_2}\right\}.$$

Say

$$\mathcal{R}_{0,1} = \frac{\alpha_1\beta_1 S_0^*}{a_1 b_1} = \frac{\Lambda\alpha_1\beta_1}{\delta a_1 b_1} \quad \text{and} \quad \mathcal{R}_{0,2} = \frac{\alpha_2\beta_2 S_0^*}{a_2 b_2} = \frac{\Lambda\alpha_2\beta_2}{\delta a_2 b_2}. \tag{4}$$

That is, the basic reproduction number of the model (2) is given by

$$\mathcal{R}_0 = max\{\mathcal{R}_{0,1}, \mathcal{R}_{0,2}\}.$$

It is observed that, $\mathcal{R}_{0,1}$ and $\mathcal{R}_{0,2}$ are reproduction numbers of strain 1 and strain 2 respectively.

2.5. **Existence two-strain Endemic Equilibrium.**
This section discusses findings on the existence and global stability of a two-strain (coexistence) endemic equilibrium, which is a steady-state solution of 2 such that both strains persist in the population.

Let $\mathcal{E} \in \mathcal{D}$ denotes two-strain endemic equilibrium, then

$$\mathcal{E} = (S^*, E_1^*, E_2^*, I_1^*, I_2^*), \tag{5}$$

where

$$\begin{aligned} S^* =& \frac{a_1 b_1}{\beta_1 \alpha_1} = \frac{a_2 b_2}{\alpha_2 \beta_2}(1 + kI_2^{*2}), \\ E_1^* =& \frac{b_1}{\beta_1} I_1^*, \\ E_2^* =& \frac{b_2}{\beta_2} I_2^*, \\ I_1^* =& \frac{\delta}{\alpha_1}(\mathcal{R}_{01} - 1) - \frac{\alpha_2 I_2^*}{\alpha_1(1 + kI_2^{*2})}, \\ I_2^* =& \sqrt{\frac{1}{k}\left(\frac{\mathcal{R}_{02}}{\mathcal{R}_{01}} - 1\right)}. \end{aligned} \tag{6}$$

Notice that

$$I_1^* > 0 \iff \mathcal{R}_{0,1} > 1 + \frac{\alpha_2 I_2^*}{\delta(1 + kI_2^{*2})}, \tag{7}$$

and

$$\begin{aligned} I_2^* > 0 &\iff \frac{\mathcal{R}_{02}}{\mathcal{R}_{01}} - 1 > 0 \\ &\iff \mathcal{R}_{0,2} > \mathcal{R}_{0,1}. \end{aligned} \tag{8}$$

Thus, $\mathcal{E}$ exists and epidemiologically meaningful if and only if $\mathcal{R}_{02} > \mathcal{R}_{01} > 1 + \mathcal{R}_k^*$, where $\mathcal{R}_k^* = \frac{\alpha_2 I_2^*}{\delta(1+kI_2^{*2})}$. Based on the aforementioned discussion, we have the following lemma.

Lemma 2.3. *For endemic equilibria* (5) *of the the model* (2)*, we have*

1. $\mathcal{R}_{01} > 1$ *and* $\mathcal{R}_{02} > 1$ *is necessary condition for existence of two-strain endemic equilibrium* $\mathcal{E}$ *but not sufficient.*(*Figure (2-4)*)
2. $\mathcal{R}_{02} > \mathcal{R}_{01} > 1 + \mathcal{R}_k^*$ *is sufficient condition for existence of two-strain endemic equilibrium* $\mathcal{E}$*.* (*Figure (5-6)*).

Remark 2.4. *$\mathcal{R}_k^*$ is depends on k and reproduction numbers as given below.*

$$\mathcal{R}_k^* = \frac{\alpha_2 I_2^*}{\delta(1+kI_2^{*2})} = \frac{\alpha_2 \mathcal{R}_{01}}{\delta \mathcal{R}_{02}} \sqrt{\frac{1}{k}\left(\frac{\mathcal{R}_{02}}{\mathcal{R}_{01}} - 1\right)} > 0$$

Remark 2.5. *The model* (2) *has strain-1 (respectively, strain-2) endemic equilibrium when $I_2^* = 0$ (respectively, $I_1^* = 0$) and condition for its existence is stated in equation-*(4.2) *of* [33] *(respectively, Remark* 1 *of* [31]*). The stability condition for it is stated in Theorem* 4.1 *of* [33] *(respectively, Theorem 3 of* [31]*).*

3. Global Stability Analysis of two-strain Endemic Equilibrium

This section discusses global stability analysis of the two-strain equilibrium of the model using the Lyapunov direct method on Λ. Throughout this section, we take

$$a_1 = \beta_1 + \delta, \qquad a_2 = \beta_2 + \delta, \qquad b_1 = \gamma_1 + \delta, \qquad b_2 = \gamma_2 + \delta.$$

Proposition 3.1. *Consider system* (2) *and its two-strain endemic equilibrium* (5)*. If $\mathcal{R}_{02} \leq \mathcal{R}_{01}\left(1 + \frac{\delta^2}{k\Lambda^2}\right)$, then $kI_2^* I_2 - 1 \leq 0$.*

Proof. Using (6), we get

$$\frac{\mathcal{R}_{02}}{\mathcal{R}_{01}} \leq 1 + \frac{\delta^2}{k\Lambda^2} \iff kI_2^{*2} \leq \frac{\delta^2}{k\Lambda^2} \iff \frac{k\Lambda}{\delta} I_2^* - 1 \leq 0. \tag{9}$$

As $I_2 \leq \frac{\Lambda}{\delta}$, we get, $kI_2 I_2^* - 1 \leq \frac{k\Lambda}{\delta} I_2^* - 1 \leq 0$. □

Corollary 3.2. *Consider system* (2) *and its two-strain endemic equilibrium* (5)*. If $\frac{k\Lambda^2}{\delta^2} \leq 1$, then $\mathcal{R}_{02} \leq \mathcal{R}_{01}\left(1 + \frac{\delta^2}{k\Lambda^2}\right)$. In particular, $kI_2^* I_2 - 1 \leq 0$.*

Proof. As $I_2^* \leq \frac{\Lambda}{\delta}$, we get

$$\frac{k\Lambda}{\delta} I_2^* - 1 \leq \frac{k\Lambda^2}{\delta^2} - 1$$

Thus, from Equation (9) of Proposition 3.1, $\mathcal{R}_{02} \leq \mathcal{R}_{01}\left(1 + \frac{\delta^2}{k\Lambda^2}\right)$. Again, from Proposition 3.1, $kI_2^* I_2 - 1 \leq 0$. □

Theorem 3.3. *The two-strain endemic equilibrium of* (2) *is globally asymptomatically stable in $\mathcal{D}$ if $1 + \mathcal{R}_k^* < \mathcal{R}_{01} < \mathcal{R}_{02} \leq \mathcal{R}_{01}\left(1 + \frac{\delta^2}{k\Lambda^2}\right)$.*

Proof. Consider Lyapunov function on $\mathcal{D}$ as follows:

$$\begin{aligned}
&\mathcal{L}(S, E_1, E_2, I_1, I_2) \\
=&S - S^*\left(1 + \ln\left(\frac{S}{S^*}\right)\right) + E_1 - E_1^*\left(1 + \ln\left(\frac{E_1}{E_1^*}\right)\right) + E_2 - E_2^*\left(1 + \ln\left(\frac{E_2}{E_2^*}\right)\right) \\
&+ \frac{a_1}{\beta_1}\left(I_1 - I_1^* - I_1^* \ln\left(\frac{I_1}{I_1^*}\right)\right) I_1 + \frac{a_2}{\beta_2}\left(I_2 - I_2^* - I_2^* \ln\left(\frac{I_2}{I_2^*}\right)\right).
\end{aligned} \tag{10}$$

Note that, $\mathcal{L}(\mathcal{E}) = 0$ and $\mathcal{L} > 0$, over $\mathcal{D}$ except $\mathcal{E} \in \mathcal{D}$. Also, $\mathcal{L}$ is radically unbounded.
By taking time derivative of $\mathcal{L}$, we have

$$\begin{aligned}
\dot{\mathcal{L}}&(S, E_1,E_2, I_1, I_2)\\
&= \left(1 - \frac{S^*}{S}\right)\left(\Lambda - \alpha_1 S I_1 - \frac{\alpha_2 S I_2}{1 + kI_2^2} - \delta S\right) + \left(1 - \frac{E_1^*}{E_1}\right)(\alpha_1 S I_1 - a_1 E_1)\\
&\quad + \left(1 - \frac{E_2^*}{E_2}\right)\left(\frac{\alpha_2 S I_2}{1 + kI_2^2} - a_2 E_2\right) + \frac{a_1}{\beta_1}\left(1 - \frac{I_1^*}{I_1}\right)(\beta_1 E_1 - b_1 I_1)\\
&\quad + \frac{a_2}{\beta_2}\left(1 - \frac{I_2^*}{I_2}\right)(\beta_2 E_2 - b_2 I_2)\\
&= \Lambda - \delta S - \frac{S^*}{S}(\Lambda) + \alpha_1 S^* I_1 + \frac{\alpha_2 S^* I_2}{1 + kI_2^2} + \delta S^* - \frac{\alpha_1 E_1^* S I_1}{E_1} + a_1 E_1^* - \frac{\alpha_2 E_2^* S I_2}{E_2(1 + kI_2^2)}\\
&\quad + a_2 E_{2,2}^* - \frac{a_1 b_1}{\beta_1} I_1 - \frac{a_1 I_1^* E_1}{I_1} + \frac{a_1 b_1}{\beta_1} I_1^* - \frac{a_2 b_2}{\beta_2} I_2 - \frac{a_2 I_2^* E_2}{I_2} + \frac{a_2 b_2}{\beta_2} I_2^*.
\end{aligned} \tag{11}$$

Using the endemic equilibrium state

$$\begin{aligned}
\Lambda &= \alpha_1 S^* I_1^* + \alpha_2 S^* I_{2,2}^* + \delta S^*,\\
\alpha_1 S^* I_1^* &= a_1 E_1^* = \frac{a_1 b_1}{\beta_1} I_1^*,\\
\frac{\alpha_2 S^* I_2^*}{1 + kI_2^{*2}} &= a_2 E_2^* = \frac{a_2 b_2}{\beta_2} I_2^*,
\end{aligned} \tag{12}$$

and, by collecting appropriate terms, we get

$$\begin{aligned}
\dot{\mathcal{L}} =&\delta S^*\left(1 - \frac{S^*}{S} - \frac{S}{S_2^*}\right) + \alpha_1 S^* I_1^*\left(3 - \frac{S^*}{S} - \frac{E_1^* S I_1}{E_1 S^* I_1^*} - \frac{E_1 I_1^*}{E_1^* I_1}\right)\\
&+ \frac{a_2 S^* I_2^*}{1 + kI_2^{*2}}\left(4 - \frac{S^*}{S} - \frac{E_2^* S I_2(1 + kI_2^{*2})}{E_2 S^* I_2^*(1 + kI_2^2)} - \frac{E_2 I_2^*}{E_2^* I_2} - \frac{1 + kI_2^{*2}}{(1 + kI_2^2)}\right)\\
&+ \left(\alpha_1 S^* - \frac{a_1 b_1}{\beta_1}\right) I_1 + \frac{a_2 b_2}{\beta_2}\left(\frac{1 + kI_2^{*2}}{1 + kI_2^2} - 1\right) I_2 + \frac{a_2 b_2}{\beta_2} I_2^*\left(\frac{1 + kI_2^2}{1 + kI_2^{*2}} - 1\right).
\end{aligned} \tag{13}$$

From (6), we have $S^* = \frac{a_1 b_1}{\alpha_1 \beta_1}$, then

$$\begin{aligned}
\dot{\mathcal{L}} =&\delta S^*\left(1 - \frac{S^*}{S} - \frac{S}{S_2^*}\right) + \alpha_1 S^* I_1^*\left(3 - \frac{S^*}{S} - \frac{E_1^* S I_1}{E_1 S^* I_1^*} - \frac{E_1 I_1^*}{E_1^* I_1}\right)\\
&+ \frac{a_2 S^* I_2^*}{1 + kI_2^{*2}}\left(4 - \frac{S^*}{S} - \frac{E_2^* S I_2(1 + kI_2^{*2})}{E_2 S^* I_2^*(1 + kI_2^2)} - \frac{E_2 I_2^*}{E_2^* I_2} - \frac{1 + kI_2^{*2}}{(1 + kI_2^2)}\right)\\
&+ \frac{k a_2 b_2 (I_2 + I_2^*)(I_2 - I_2^*)^2}{\beta_2(1 + kI_2^2)(1 + kI_2^{*2})}\left(kI_2^* I_2 - 1\right).
\end{aligned} \tag{14}$$

Using the connection between geometric and arithmetic mean, we have

$$\left(1-\frac{S^*}{S}-\frac{S}{S_2^*}\right)\leq 0$$
$$\left(3-\frac{S^*}{S}-\frac{E_1^*SI_1}{E_1S^*I_1^*}-\frac{E_1I_1^*}{E_1^*I_1}\right)\leq 0$$
$$\left(4-\frac{S^*}{S}-\frac{E_2^*SI_2(1+kI_2^{*2})}{E_2S^*I_2^*(1+kI_2^2)}-\frac{E_2I_2^*}{E_2^*I_2}-\frac{1+kI_2^{*2}}{(1+kI_2^2)}\right)\leq 0, \tag{15}$$

and by Proposition 3.1, $kI_2^*I_2-1\leq 0$. Thus, $\dot{\mathcal{L}}\leq 0$. Moreover, $\{X\in\mathcal{D}:\dot{\mathcal{L}}(X)=0\}=\{\mathcal{E}\}$. Hence, from Lassale's principle of invariance [38], $\mathcal{E}$ is globally asymptotically stable.

□

Corollary 3.4. *The two-strain endemic equilibrium of* (2) *is globally asymptomatically stable in* $\mathcal{D}$ *if* $1+\mathcal{R}_k^*<\mathcal{R}_{01}<\mathcal{R}_{02}$ *and* $\frac{k\Lambda^2}{\delta^2}\leq 1$.

The proof of this corollary follows from Theorem 3.3 and Corollary 3.2.

4. Numerical Simulations and Discussion

In this section, we numerically illustrate and validate the analytical results obtained in the previous sections. Simulations are performed using the assumed parameter values listed in Table 2, and the ODE system is solved using a standard fourth-order Runge–Kutta scheme with biologically plausible initial conditions. The objective is to confirm the existence and global stability of the two-strain endemic equilibrium under the conditions derived in Lemma 2.3 and Theorem 3.3.

Throughout, we focus on verifying the inequalities

$$\mathcal{R}_{01}\left(1+\frac{\delta^2}{k\Lambda^2}\right)\geq\mathcal{R}_{02}>\mathcal{R}_{01}>1+\mathcal{R}_k^*, \tag{16}$$

which characterize the parameter region where coexistence is possible and globally stable.

Table 2. Parameter Table

Parameter	Figure 2	Figure 3	Figure 4	Figure 5	Figure 6
Λ	1	1	1	1	1
k	0.1	0.1	0.5	0.1	0.2
δ	0.1	0.1	0.1	0.1	0.1
α_1	0.131	0.16	0.16	0.16	0.16
α_2	0.2	0.16	0.16	0.165	0.165
β_1	0.16	0.2	0.2	0.17	0.17
β_2	0.2	0.2	0.2	0.2	0.2
γ_1	0.1	0.1	0.1	0.1	0.1
γ_2	0.11	0.1	0.1	0.11	0.11

4.1. **Validation of Analytical Results.**

(1) Failure of coexistence under classical conditions. Figures 2–4 show that the usual requirement

$$\mathcal{R}_{01} > 1, \qquad \mathcal{R}_{02} > 1$$

does not guarantee coexistence. One of the strains always goes extinct, confirming that classical threshold conditions are insufficient when behavioural modification is present. Moreover, Figure 3 demonstrates that the conventional condition

$$\mathcal{R}_{01} = \mathcal{R}_{02} > 1$$

(discussed in [33]) also fails to ensure coexistence as behavioural avoidance suppresses strain 2, leading to $I_2^* = 0$.

(2) Role of the coexistence condition $\mathcal{R}_{01} > 1+\mathcal{R}_k^*$. Figure 2 confirms the prediction of Lemma 2.3: if

$$\mathcal{R}_{01} \leq 1 + \mathcal{R}_k^*,$$

the coexistence equilibrium loses stability and strain 1 dies out. Figures 3–4 further illustrate that increasing the behavioural parameter k intensifies suppression of strain 2, causing its extinction unless its transmission potential exceeds that of strain 1 by a sufficient margin. These observations align with the analytical condition

$$\mathcal{R}_{02} > \mathcal{R}_{01} > 1 + \mathcal{R}_k^*.$$

The inequality means that strain 2 has the higher reproduction number, and strain-1 is sufficiently transmissible (above $1 + R_k^*$) to co-persist but not strong enough to outcompete strain 2. Thus, strain 2 requires a higher reproduction number to offset the behavioural suppression acting against it and thereby maintain coexistence with strain 1.

(3) Stability of the two-strain endemic equilibrium. Figures 5–6 confirm Theorem 3.3: whenever

$$\mathcal{R}_{01}\left(1 + \frac{\delta^2}{k\Lambda^2}\right) \geq \mathcal{R}_{02} > \mathcal{R}_{01} > 1 + \mathcal{R}_k^*,$$

the coexistence equilibrium is globally asymptotically stable. In this regime, both strains persist with positive equilibrium values. The upper bound $\mathcal{R}_{02} \leq \mathcal{R}_{01}\left(1 + \frac{\delta^2}{k\Lambda^2}\right)$ indicates that behavioural avoidance due to k prevents strain 2 from becoming excessively dominant and ensures co-existence. For large k, this condition approaches $\mathcal{R}_{02} \leq \mathcal{R}_{01}$, implying that strong behavioural modification may eliminate strain 2.

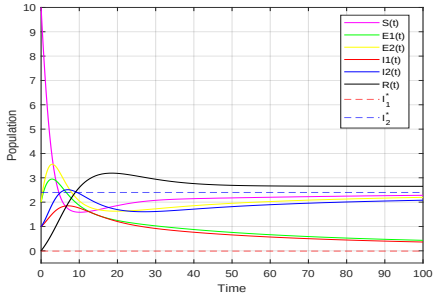


FIGURE 2. $\mathcal{R}_{02} = 6.3492 > \mathcal{R}_{01} = 4.0308 \not> 1 + \mathcal{R}_k^* = 4.0451$

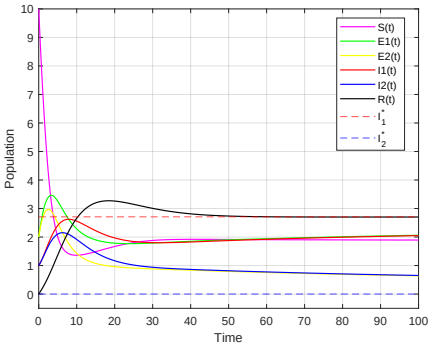


FIGURE 3

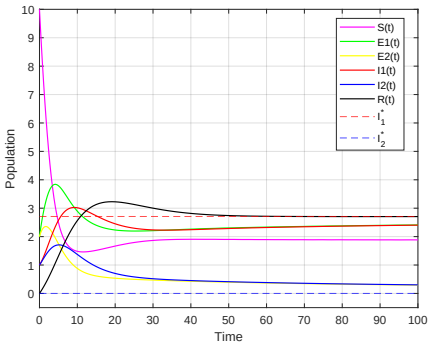


FIGURE 4

$\mathcal{R}_{01} = \mathcal{R}_{02} = 5.3333 > 1$ with $k = 0.1$(left) and $k = 0.5$ (right)

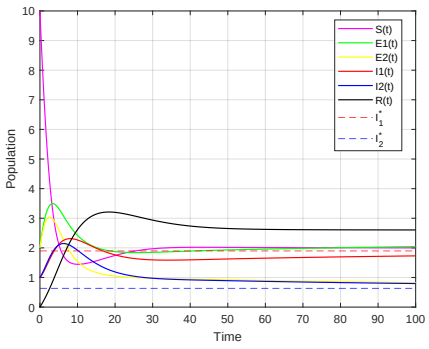


FIGURE 5

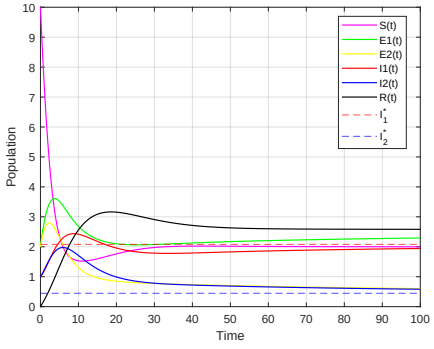
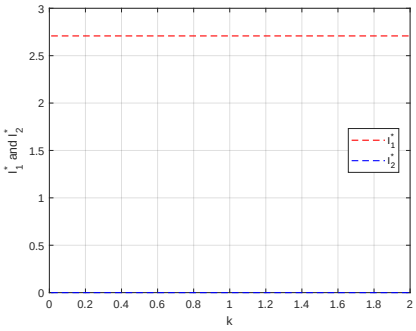


FIGURE 6

$\mathcal{R}_{01}\left(1 + \frac{\delta^2}{k\Lambda^2}\right) = 5.5407 \geq \mathcal{R}_{02} = 5.2381 > \mathcal{R}_{01} = 5.0370 > 1 + \mathcal{R}_k^* = 2.0024 > 1$ with $k = 0.1$ (left) and $k = 0.2$ (right)



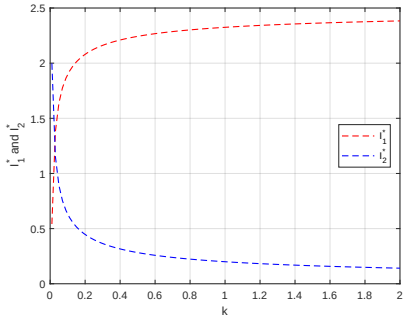


FIGURE 7 FIGURE 8

Effect of k on I_1^* and I_2^*

$\mathcal{R}_{02} = \mathcal{R}_{01} > 1$ (left), $\mathcal{R}_{02} > \mathcal{R}_{01} > 1 + \mathcal{R}_k^*$ (right)

4.2. **Interpretation of Behavioural Effects.**
The numerical results highlight the crucial role of the behavioural parameter k. A stronger behavioural response (large k) significantly reduces effective contact between susceptible individuals and those infected with strain 2. This behavioural avoidance:

- allows strain 1 to dominate even when $\mathcal{R}_{02} > \mathcal{R}_{01}$: Figure 3,
- creates a range where coexistence occurs despite asymmetric transmission mechanisms: Equation (16) and Figures 5–6,
- suppress I_2^*: compare Figure 3 with Figure 4, and Figure 5 with Figure 6.
- let I_2^* decreases with k while I_1^* increasing; illustrating the competitive advantage gained by strain 1 due to behavioural avoidance directed at strain 2: Figure 8.
- has no impact on I_1^* once I_2 eliminated: Figure 7.

Hence, the simulations reinforce the analytical conclusion that behavioural heterogeneity fundamentally alters competitive outcomes in multi-strain systems.

Although our model represents only a modest extension of [33], the present qualitative analysis refines and improves their conclusions. The authors of [33] state that a two-strain endemic equilibrium exists and is globally stable whenever $\mathcal{R}_{01} = \mathcal{R}_{02} > 1$. Our analysis shows that this criterion is incomplete because it does not account for the influence of the behavioural parameter k and does not guarantee the existence of a two-strain endemic equilibrium. In contrast, our work incorporates the behavioural response explicitly and establishes the correct coexistence conditions, which depend jointly on k and the reproduction numbers. Additionally, it provides a clear interpretation of how the parameter k influences the coexistence of the two strains.

5. Conclusion

This study investigated a two-strain SEIR model incorporating behavioural heterogeneity, motivated by the observation that mutated or newly emergent strains often elicit stronger behavioural responses due to unfamiliarity and perceived risk. To capture this mechanism, strain 1 was modelled with a bilinear incidence function, whereas strain 2 was modelled using a non-monotone incidence function modulated by a behavioural parameter k.

We established mathematically rigorous, necessary and sufficient conditions for the existence and global stability of a two-strain endemic equilibrium. These conditions explain that, because behavioural avoidance suppresses strain 2, it must possess a sufficiently higher reproduction number to overcome this behavioural suppression and maintain coexistence. The stability condition further shows that behavioural avoidance modulated by k prevents strain 2 from becoming excessively dominant, thereby supporting long-term coexistence. Moreover, sufficiently strong behavioural modification may even eliminate strain 2 altogether.

Numerical simulations corroborated all analytical results, confirming the predicted coexistence range and demonstrating how behavioural responses reshape the long-term competition between strains. Behavioural heterogeneity effectively partitions the susceptible population into groups with different transmission pathways, thereby relaxing the classical competitive exclusion principle and allowing coexistence even when one strain is intrinsically more transmissible.

Overall, this work provides mathematically grounded and epidemiologically meaningful insights into how behavioural responses influence multi-strain epidemic dynamics. Future investigations may incorporate adaptive behavioural changes, vaccination, treatment interventions, or stochastic effects to further enhance understanding of behaviour-driven epidemiological processes.

Data Availability Statement. The data used to support the findings of this study are available from the corresponding author upon request.

Declarations. The authors declare no conflict of interest.

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