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A Two Strain Mutation Model with Temporary and Permanent Recovery

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Abstract

Mutation occurs when there is a change in the sequence of the genetic code of an organism. Such changes pose some challenges especially in the development of drugs. A good understating of the dynamics of the strains will help to understand the variant of concern. In this research work, we examined a two strain mutation model in which the first strain has a SIS dynamics and the second strain a SIR dynamics. The disease-free equilibrium, endemic equilibrium and the basic reproduction number of each strain are derived. Suitable Lyapunov functions are used to determine the global stability of the disease free equilibrium of each strain. The equilibrium point and the basic reproduction number of the joint model are determined. The possibility for the coexistence of the endemic equilibrium is also determined. It was found out that both strains have direct variation between them. However, it will be difficult to eradicate the disease in the population if the basic reproduction number of strain 1 is greater than strain 2. The effect of degree of transmissibility of the two strains on the population is carried out through numerical simulation.

Keywords: Equilibrium; Mutation; Lyapunov Function, Basic Reproduction Number.
MSC2010: 92B05.

1 INTRODUCTION

Mutation is a big challenge in the medical world as it makes the eradication of a disease difficult and complicated. The severity of mutation can vary from little to great consequences. Some strains tend to displace others making them the dominant strain. Mutation may thus reduce the efficacy of treatments and vaccines.

The causative agents of diseases can occur in multiple strains. The presence of multiple strain complicates disease dynamics and management. One of the major cause of multiple strains is mutation. Mutation occurs when there is a change in the sequence of the DNA or RNA of a microorganism [1]. For example, the novel coronavirus 2019 since its outbreak in 2019 has over a thousand variants [2], some of great concern and some of less concern depending on the degree of virulence, transmissibility, infectivity and mutability.

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Competition and cooperation between strains can affect the pathogen and infection dynamics thereby causing an evolution. Understanding the dynamics of the possible strains will help in control approaches [3]. Mathematical models on multi strain models include [4–8].
Naji and Hussien [9] presented an infectious disease model with vertical transmission comprising of two types of infection. The local and global stability analysis of all possible equilibrium points were determined. Bifurcation analysis was also examined and numerical simulation to confirm obtained results.
Bentaleb and Amine [10] studied a SEIR infectious model with various strains having both non-monotonic and bilinear incidence. The basic reproduction number of each strain was determined. Lyapunov function was constructed to determine the global stability of the disease free equilibrium and numerical simulation was carried out to illustrate important concepts. Rashkov and Kooi [11] considered a two strain dengue model based on the SIR dynamics with a possibility of secondary infection and temporary cross immunity for the host. The condition for dominance and coexistence of endemic equilibrium was determined with the impact of numerical bifurcation analysis.
Most research work on mutation has been on multiple strains with identical flow patterns. The aim of this research work is to examine the behaviour of two mutating strains with different flow patterns. We examine a two strain model in which the first strain follows the SIS model dynamics while the other follows the SIR model dynamics. Our model also captures the effect of early treatment on mutation. Hence, the strain with SIS model dynamics can mutate into the other strain if no treatment is applied within a certain period. Only the the strain with SIR dynamics has permanent recovery. Further insight is given in the next section.

2 MODEL FORMULATION

The entire population, represented by $N(t)$, is classified into susceptible individuals $S(t)$, individuals infected with the first strain $I_1(t)$, individuals infected with the second strain $I_2(t)$ and recovered class $R(t)$. The strain 1 dynamics behaves like a SIS model while strain 2 behaves like a SIR model. Individuals infected with strain 1 experience a temporary recovery if treatment is applied within a certain period otherwise the strain mutates to the second one. Individuals infected with strain 2 can have permanent recovery. It is also assumed that all population are born susceptible. The susceptible population is increased through recruitment at rate Λ and effective treatment from the infectious class with strain 1 at rate σ. It is reduced by natural death at rate μ and interaction with individuals infected with strain 1 and 2 with transmission rate β_1 and β_2 respectively. Thus,

$$\frac{dS}{dt} = \Lambda - \beta_1 S I_1 - \beta_2 S I_2 - \mu S + \sigma I_1.$$

There is an addition to the infectious class with strain 1 due to interaction with the susceptible class at rate β_1 and decrease by mutation to strain 2, natural death and effective treatment with temporary immunity at rate α, μ and σ respectively. Thus,

$$\frac{dI_1}{dt} = \beta_1 S I_1 - (\alpha + \mu + \sigma) I_1.$$

There is an addition to the infectious class with strain 2 due to interaction with the susceptible class at rate β_2 and mutation from strain 1. There is a reduction by natural death, death due to the disease and effective treatment with permanent recovery at rate μ, δ and γ respectively. Thus,

$$\frac{dI_2}{dt} = \beta_2 S I_2 + \alpha I_1 - (\mu + \delta + \gamma) I_2.$$

There is an addition to the recovered class due to permanent recovery from strain 2 at rate γ and reduction by natural death at rate μ. Thus,

$$\frac{dR}{dt} = \gamma I_2 - \mu R.$$

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The system of differential equations below represents the dynamics presented above.

$$\frac{dS}{dt} = \Lambda - \beta_1 S I_1 - \beta_2 S I_2 - \mu S + \sigma I_1. \tag{2.1}$$

$$\frac{dI_1}{dt} = \beta_1 S I_1 - (\alpha + \mu + \sigma) I_1. \tag{2.2}$$

$$\frac{dI_2}{dt} = \beta_2 S I_2 + \alpha I_1 - (\mu + \delta + \gamma) I_2. \tag{2.3}$$

$$\frac{dR}{dt} = \gamma I_2 - \mu R. \tag{2.4}$$

Table 1: Summary of parameters of model (2.1) – (2.4)

Definition	Symbol
Recruitment term of the susceptible population	Λ
Transmission rate of strain 1	β_1
Transmission rate of strain 2	β_2
Natural death rate	μ
Treatment rate of strain 1	σ
Treatment rate of strain 2	γ
Mutation rate from strain 1 to strain 2	α
Disease induced death rate of strain 2	δ

3 MODEL ANALYSIS

The qualitative analysis of the model is explored in this section

3.1 Invariant region

Theorem 3.1. *(Invariant region). The solution of* (2.1)–(2.4) *exist in* $\mathcal{R}$ *defined by* $\{S(t), I_1(t), I_2(t), R(t) \in \mathbb{R}^4_+ : N(0) \leq N(t) \leq \frac{\Lambda}{\mu}\}$ *with initial conditions:* $S(0) \geq 0, I_1(0) \geq 0, I_2(0) \geq 0, R(0) \geq 0$.

Proof: We obtain the invariant region by following the procedure below:

$$N(t) = S(t) + I_1(t) + I_2(t) + R(t). \tag{3.1}$$

$$\frac{dN}{dt} = \Lambda - \mu N - \delta I_2. \tag{3.2}$$

Since $\delta \geqslant 0$, (3.1) can be expressed as:

$$\frac{dN}{dt} \leq \Lambda - \mu N. \tag{3.3}$$

Solving equation (3.1) together with the initial conditions gives:

$$N(0) \leq N(t) \leq \frac{\Lambda}{\mu}. \tag{3.4}$$

Thus, $\mathcal{R}$ is a positive invariant set under the model described by (2.1) – (2.4).

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3.2 Strain 1 only sub model

Strain 1 only model is obtained by setting $I_2 = 0$ in $(2.1) - (2.4)$ which gives:

$$\frac{dS}{dt} = \Lambda - \beta_1 S I_1 - \mu S + \sigma I_1. \tag{3.5}$$

$$\frac{dI_1}{dt} = \beta_1 S I_1 - (\alpha + \mu + \sigma) I_1. \tag{3.6}$$

3.2.1 Disease free equilibrium

We obtain the disease free equilibrium point of strain 1 only model by setting the system $(3.5)-(3.6)$ to zero with $I_1 = 0$.

$$\pi_{01} = (\frac{\Lambda}{\mu}, 0).$$

3.2.2 Basic reproduction number

The next generation matrix approached by Van den Driessche and Watmough [12] is employed to compute the basic reproduction number. We obtain:

$$R_{01} = \frac{\beta_1 \Lambda}{\mu(\alpha + \mu + \sigma)}.$$

3.2.3 Local Stability of Disease-free equilibrium

Theorem 3.2. *(Local stability of disease free equilibrium). The disease-free equilibrium for the system* $(3.5) - (3.6)$ *is locally asymptotically stable if* $R_{01} < 1$ *and unstable if* $R_{01} > 1$.

Proof: Let $J(\pi_{01})$ represents the Jacobian matrix at the disease free equilibrium.

$$J(\pi_{01}) = \begin{pmatrix} -\mu & \sigma - \frac{\beta_1 \Lambda}{\mu} \\ 0 & \frac{\beta_1 \Lambda}{\mu} - (\alpha + \mu + \sigma) \end{pmatrix}$$

The characteristic equation has roots $-\mu$ and $\frac{\beta_1 \Lambda}{\mu} - (\alpha + \mu + \sigma)$. Therefore, $\frac{\beta_1 \Lambda}{\mu} - (\alpha + \mu + \sigma)$ must be negative for the local asymptotic stability of the disease free equilibrium.
Since $\frac{\beta_1 \Lambda}{\mu} - (\alpha + \mu + \sigma) = (\alpha + \mu + \sigma)(R_0 - 1)$, $R_{01} < 1$ is necessary to have a negative root.

3.2.4 Global Stability Analysis

Theorem 3.3. *(Global stability of disease free equilibrium). The disease free equilibrium of model* $(3.5) - (3.6)$ *is globally asymptotically stable if* $R_{01} < 1$.

Proof: The Lyapunov function below is used to test for the global stability.

$$V = \frac{1}{(\alpha + \mu + \sigma)} I_1.$$

$$\dot{V} = \frac{1}{(\alpha + \mu + \sigma)} \dot{I}_1.$$

Substituting and simplifying the expressions for $\dot{I}_1$ gives:

$$\dot{V} = \frac{1}{(\alpha + \mu + \sigma)} [\beta_1 S I_1 - (\alpha + \mu + \sigma) I_1].$$

$$\dot{V} = \left(\frac{\beta_1 S I_1}{(\alpha + \mu + \sigma)} - (\alpha + \mu + \sigma) \right) I_1.$$

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$$\dot{V} = (\alpha + \mu + \sigma)I_1[R_{01} - 1].$$

Thus $\dot{V} \leq 0$ with equality when $I_1 = 0$. Hence by LaSlle's extension to Lyapunov principle, the limit set for each solution is contained in the largest invariant set for which $I_1 = 0$.

3.2.5 Endemic equilibrium point

This is the region where the disease persist in the population.

Theorem 3.4. *(Existence of endemic equilibrium). The system* $(3.5) - (3.6)$ *produces an endemic equilibrium when* $R_{01} > 1$.

Proof: Let $E_1^* = (S^*, I_1^*)$ be a non trivial equilibrium of the system $(3.5) - (3.6)$. At fixed point, the system becomes:

$$\begin{aligned} S^* &= \frac{(\alpha + \mu + \sigma)}{\beta_1}. \\ I_1^* &= \frac{\mu(\alpha + \mu + \sigma)}{\beta_1(\mu + \alpha)}[R_{01} - 1]. \end{aligned}$$

3.2.6 Local Stability of Endemic Equilibrium

Theorem 3.5. *(Local stability of endemic equilibrium). The endemic equilibrium for the system* $(3.5) - (3.6)$ *is locally asymptotically stable if* $R_{01} > 1$ *and unstable otherwise.*

Proof: The Jacobian matrix of the system $(3.5) - (3.6)$ computed at the endemic equilibrium gives:

$$J(E_1^*) = \begin{pmatrix} -\mu\left(\frac{(\alpha+\mu+\sigma)[R_{01}-1]}{(\alpha+\mu)} + 1\right) & -(\alpha + \mu) \\ \frac{\mu(\alpha+\mu+\sigma)[R_{01}-1]}{(\alpha+\mu)} & 0 \end{pmatrix}$$

$R_{01} > 1$ is necessary for the determinant of $J(E_1^*)$ to be greater than zero and the trace of $J(E_1^*)$ to be less than zero. Therefore, the endemic equilibrium is locally asymptotically stable provided that $R_{01} > 1$.

3.3 Strain 2 only sub model

Strain 2 only model is obtained by setting $I_1 = 0$ in $(2.1) - (2.4)$ which gives:

$$\frac{dS}{dt} = \Lambda - \beta_2 S I_2 - \mu S. \tag{3.7}$$

$$\frac{dI_2}{dt} = \beta_2 S I_2 - (\mu + \delta + \gamma)I_2. \tag{3.8}$$

$$\frac{dR}{dt} = \gamma I_2 - \mu R. \tag{3.9}$$

3.3.1 Disease free equilibrium

We obtain the disease-free equilibrium of the strain 2 only model by equating the system $(3.7) - (3.9)$ to zero with $I_2 = 0$.

$$\pi_{02} = (\frac{\Lambda}{\mu}, 0, 0).$$

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3.3.2 Basic reproduction number

The Next-Generation Matrix approached by Van den Driessche and Watmough [12] is used to compute the basic reproduction number. We obtain:

$$R_{02} = \frac{\beta_2 \Lambda}{\mu(\mu + \delta + \gamma)}.$$

3.3.3 Local Stability of Disease free equilibrium

Theorem 3.6. *(Local stability of disease free equilibrium). The disease-free equilibrium for the system* $(3.7) - (3.9)$ *is locally asymptotically stable if* $R_{02} < 1$ *and unstable if* $R_{02} > 1$.

Proof: The Jacobian matrix of the system evaluated at the disease free equilibrium is represented below:

$$J(\pi_{02}) = \begin{pmatrix} -\mu & -\frac{\beta_2 \Lambda}{\mu} & 0 \\ 0 & \frac{\beta_2 \Lambda}{\mu} - (\mu + \delta + \gamma) & 0 \\ 0 & \gamma & -\mu \end{pmatrix}$$

The characteristic equation has roots $-\mu$, $-\mu$ and $\frac{\beta_2 \Lambda}{\mu} - (\mu + \delta + \gamma)$. Therefore, $\frac{\beta_2 \Lambda}{\mu} - (\mu + \delta + \gamma)$ must be negative to establish the local asymptotic stability of the disease free equilibrium of the model.
Since $\frac{\beta_2 \Lambda}{\mu} - (\mu + \delta + \gamma) = (\mu + \delta + \gamma)(R_{02} - 1)$, $R_{02} < 1$ is necessary to confirm its local asymptotic stability.

3.3.4 Global Stability Analysis

Theorem 3.7. *(Global stability of disease free equilibrium). The disease free equilibrium of model* $(3.7) - (3.9)$ *is globally asymptotically stable if* $R_{02} < 1$.

Proof: The test for the global stability is done using the Lyapunov function below:

$$L = \frac{1}{(\mu + \delta + \gamma)} I_2.$$

$$\dot{L} = \frac{1}{(\mu + \delta + \gamma)} \dot{I}_2.$$

Substituting and simplifying the expressions for $\dot{I}_2$ gives:

$$\dot{L} = \frac{1}{(\mu + \delta + \gamma)} [\beta_2 S I_2 - (\mu + \delta + \gamma) I_2].$$

$$\dot{L} = \left(\frac{\beta_2 S I_2}{(\mu + \delta + \gamma)} - (\mu + \delta + \gamma) \right) I_2.$$

$$\dot{V} = (\mu + \delta + \gamma) I_2 [R_{02} - 1].$$

Thus $\dot{L} \leq 0$ with equality when $I_2 = 0$. Hence by LaSlle's extension to Lyapunov principle, the limit set for each solution is contained in the largest invariant set for which $I_2 = 0$.

3.3.5 Endemic equilibrium point

This is the point where the disease persists in the population.

Theorem 3.8. *(Existence of endemic equilibrium). The model* $(3.7) - (3.9)$ *has an endemic equilibrium when* $R_{02} > 1$.

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Proof: We represent the non trivial equilibrium of the model (3.7)−(3.9) with $E_2^* = (S^*, I_2^*, R^*)$. Then the model at steady state becomes

$$\begin{aligned} S^* &= \frac{(\mu+\delta+\gamma)}{\beta_2}. \\ I_1^* &= \frac{\mu}{\beta_2}[R_{02}-1]. \\ R^* &= \frac{\gamma I_2^*}{\mu}. \end{aligned}$$

3.3.6 Local stability of Endemic equilibrium

Theorem 3.9. *(Local stability of disease free equilibrium). The disease-free equilibrium for the system* (3.7) − (3.9) *is locally asymptotically stable if* $R_{02} > 1$ *and unstable otherwise.*

Proof: The Jacobian of the (3.7) − (3.9) computed at the disease free equilibrium is represented with $J(E_2^*)$.

$$J(E_2^*) = \begin{pmatrix} -\mu R_{02} & -(\mu+\delta+\gamma) & 0 \\ \mu(R_{02}-1) & 0 & 0 \\ 0 & \gamma & -\mu \end{pmatrix}$$

one of the roots of the characteristic equation is $-\mu$. The remaining eigenvalues are obtained using the matrix below:

$$\begin{pmatrix} -\mu R_{02} & -(\mu+\delta+\gamma) \\ \mu(R_{02}-1) & 0 \end{pmatrix}$$

$R_{02} > 1$ is necessary for the determinant of the matrix above to be positive and the trace to be negative. Hence, $R_{02} > 1$ affirms the local asymptotic stability.

3.4 Strain 1 and 2 joint model

3.4.1 Disease free equilibrium

We set the system (2.1) − (2.4) to zero to obtain the disease free equilibrium with the diseased points also equal to zero. We obtain:

$$\pi_0 = (\frac{\Lambda}{\mu}, 0, 0, 0).$$

3.4.2 Basic reproduction number

The basic reproductive number is the mean number of secondary infections caused by an infectious individual in an entirely susceptible population. The next generation matrix approach by Driessche and Watmough [12] is applied.
From the infectious stages I_1 and I_2, a vector $\mathcal{F}$ can be created that represent new infections and $\mathcal{V}$ that represents outflows from the model equations [13]. Hence,

$$\mathcal{F} = \begin{pmatrix} \beta_1 S I_1 \\ \beta_2 S I_2 \end{pmatrix}$$

$$\mathcal{V} = \begin{pmatrix} (\alpha+\mu+\sigma) I_1 \\ (\mu+\delta+\gamma) I_2 - \alpha I_1 \end{pmatrix}$$

Next, we compute the Jacobian F from $\mathcal{F}$ and V from $\mathcal{V}$ at the disease free equilibrium point. They are described below:

$$\mathbf{F} = \begin{pmatrix} \frac{\beta_1 \Lambda}{\mu} & 0 \\ 0 & \frac{\beta_2 \Lambda}{\mu} \end{pmatrix}$$

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$$\mathbf{V} = \begin{pmatrix} (\alpha+\mu+\sigma) & 0 \\ -\alpha & (\mu+\delta+\gamma) \end{pmatrix}$$

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

$$R_0 = max\{R_{01}, R_{02}\}.$$

3.4.3 Local stability of Disease free equilibrium

Theorem 3.10. *(Local stability of disease free equilibrium). The disease-free equilibrium for the system* $(2.1) - (2.4)$ *is locally asymptotically stable if* $R_0 < 1$ *and unstable if* $R_0 > 1$.

Proof: The Jacobian matrix evaluated at the disease-free gives:

$$J(\pi_0) = \begin{pmatrix} -\mu & \sigma - \frac{\beta_1\Lambda}{\mu} & -\frac{\beta_2\Lambda}{\mu} & 0 \\ 0 & \frac{\beta_1\Lambda}{\mu} - (\alpha+\mu+\sigma) & 0 & 0 \\ 0 & \alpha & \frac{\beta_2\Lambda}{\mu} - (\mu+\delta+\gamma) & 0 \\ 0 & 0 & \gamma & -\mu \end{pmatrix}$$

There are four roots corresponding to the Jacobian above. Two of the roots are $-\mu$ (twice). The other two are negative provided that $R_0 < 1$ and unstable if $R_0 > 1$.

3.4.4 Endemic equilibrium point

This is the point where the disease persists in the system.

Theorem 3.11. *(Existence of endemic equilibrium). The model has an endemic equilibrium when* $R_{01} > R_{02} > 1$ *and* $\left(\frac{(\alpha+\mu)R_{01}}{\mu R_{02}}\right) > 1$.

Proof: The endemic equilibrium point of the system is represented by $E^{**} = (S^{**}, I_1^{**}, I_2^{**}, R^{**})$. At steady states, the system has endemic equilibrium:

$$\begin{aligned} S^{**} &= \frac{(\mu+\delta+\gamma)}{\beta_2}. \\ R^{**} &= \frac{\gamma I_2^{**}}{\mu}. \\ I_1^{**} &= \frac{\Lambda\beta_2}{\alpha\mu R_{02}R_{02}}[R_{01} - R_{02}]I_2^{**}. \\ I_2^{**} &= \frac{\left(\frac{\alpha}{\beta_2}\right)(R_{01}-1)}{\left(\frac{(\alpha+\mu)R_{01}}{\mu R_{02}}\right) - 1}. \end{aligned}$$

3.4.5 Impact of strain 1 on strain 2 and viceversa

The impact between the strains is obtained when the basic reproduction number of strain 1 is expressed with respect to strain 2 and viceversa.
R_{01} can be expressed in terms of R_{02} and it is given by:

$$R_{01} = \frac{(\mu+\delta+\gamma)R_{02}\beta_1}{(\alpha+\mu+\sigma)\beta_2}.$$

Therefore, evaluating $\frac{\partial R_{01}}{\partial R_{02}}$ and simplifying gives

$$\frac{\partial R_{01}}{\partial R_{02}} = \frac{R_{01}}{R_{02}}.$$

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The value obtained is always positive. Hence, an increase in strain 2 would produce an increase in strain 1.
Similarly,

$$\frac{\partial R_{02}}{\partial R_{01}} = \frac{R_{02}}{R_{01}}.$$

The value obtained is always positive. Hence, an increase in strain 1 would produce an increase in strain 2.

4 Numerical Simulation

The following parameters are used to carry out the numerical simulation of the joint model. $\Lambda = 3, \beta = 0.3, \mu = 0.025, \sigma = 0.1429, \alpha = 0.1, \delta = 0.0286, \gamma = 0.05$ with $S(0) = 1000, I_1(0) = 100, I_2(0) = 100, R(0) = 0$. We considered two cases in which $\beta_1 < \beta_2 (\beta_1 = 0.005, \beta_2 = 0.01)$ and $\beta_1 > \beta_2 (\beta_1 = 0.01, \beta_2 = 0.005)$. Two cases were considered in which the first strain is less transmissible than the second strain and viceversa. The result shows that if the first strain is more transmissible, more people will get infected with the first strain than the second strain but with minute difference. However, if the second strain is more transmissible, more people will get infected with the second strain than the first strain but with significant difference as shown in figure 1 and 2. However, in both cases, the second strain becomes dominant while the recovered population is not affected by the degree of transmissibility.

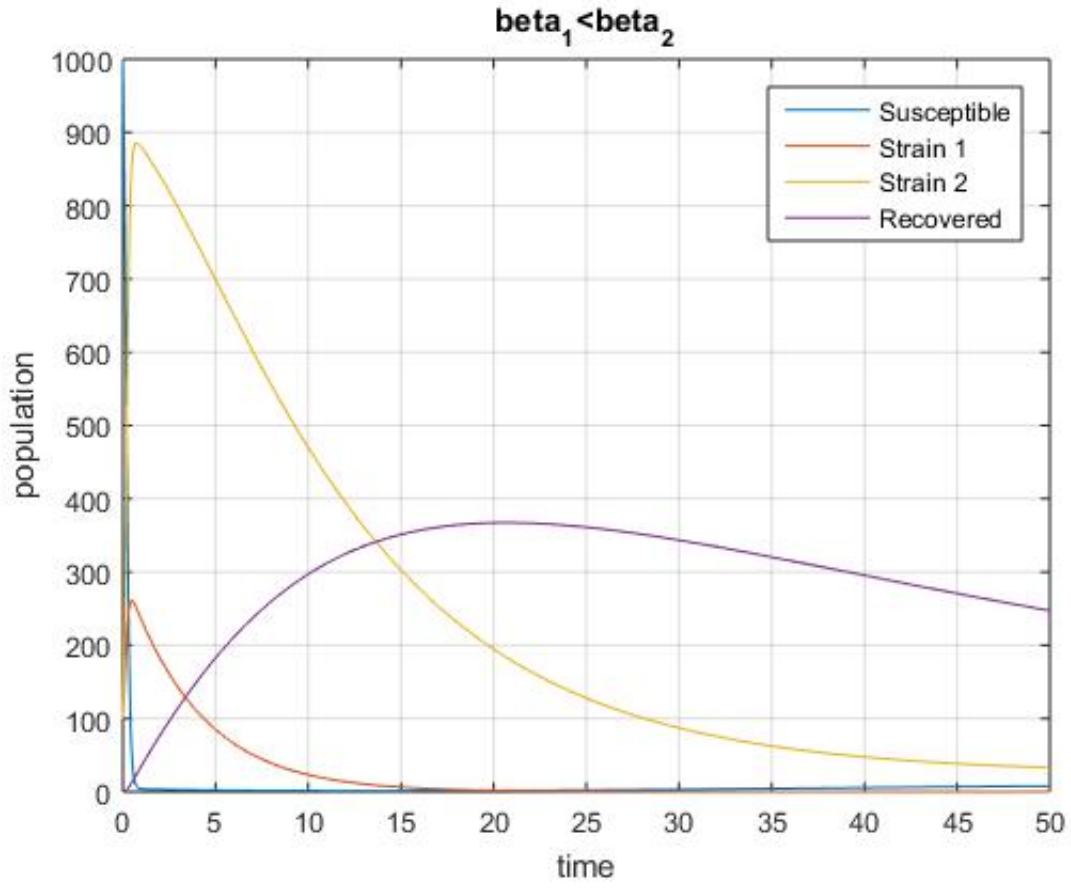

Figure 1: Numerical simulation

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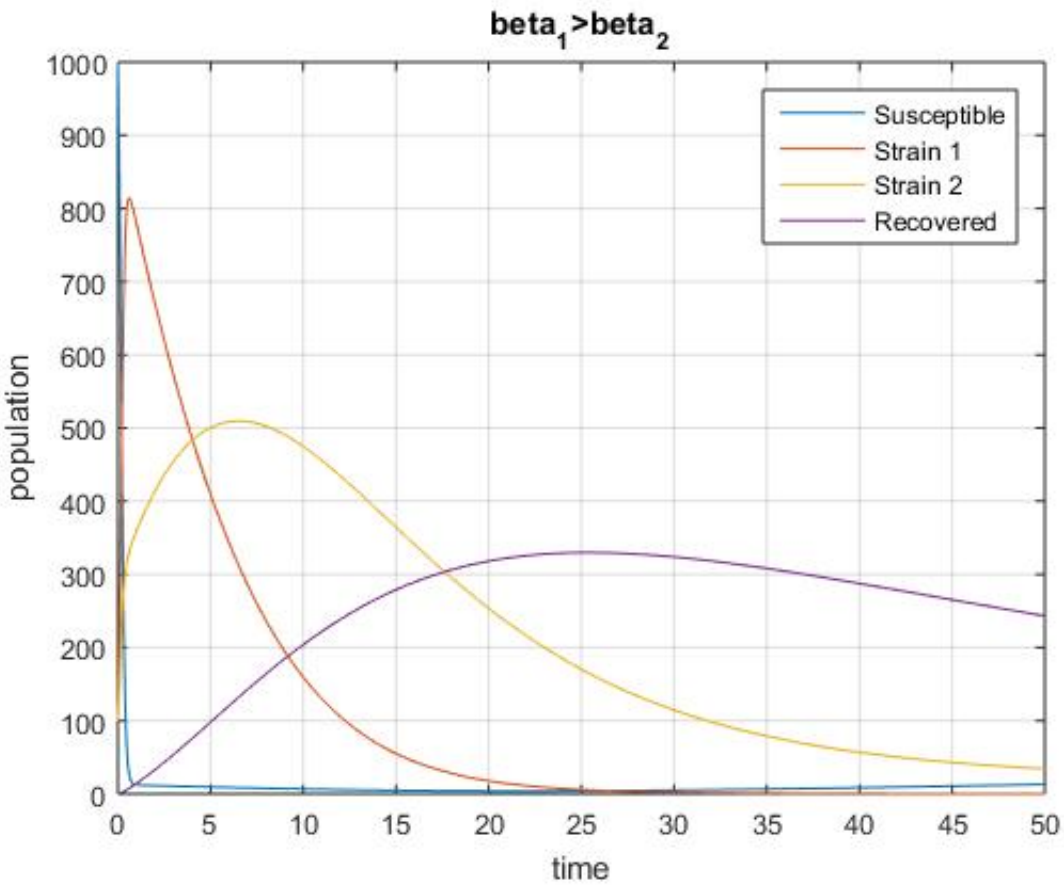

Figure 2: Numerical simulation

5 Conclusion

We present a two mutation strain model that incorporates both SIS and SIR dynamics with temporary and permanent immunity. Each strain is analyzed separately and the basic epidemiological properties are established. The disease-free equilibrium point of each strain is found to be globally asymptotically stable.
The basic reproduction number of the joint model is determined and the conditions for the existence of the disease free equilibrium and endemic equilibrium are also established. It is also established that both strains have a direct variation between them in terms of propagation. However, the basic reproduction number of the first strain must be less than the second strain to prevent endemicity. Numerical simulation revealed that irrespective of the degree of transmissibility of the two strains, the second strain will be dominant.
For future work, vaccination can be incorporated into the model and the number of strains can also be increased. Other compartmental flow patterns can also be considered.

6 Declaration of interest

The author declares no competing interest of any form.

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