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Research Article

# Essential Analysis of New SEVIR Model: A Five Case Epidemic Model

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**Abstract:** In this work, we introduce a new SEVIR epidemic model to analyze the dynamics of infectious diseases by incorporating the key compartments such as susceptible, exposed, vaccinated, infected, and recovered populations. We add the vaccination compartment in the current model and calculate the consequences concerning the disease spreading; it is significantly relevant at the present day considering the general policies of worldwide vaccinations. For a stringent assessment of stability, it should involve derivation of a basic reproduction number, as this parameter characterizes whether an infection will be spread at all. Our results demonstrate that when we have $R_0 < 1$, the disease-free equilibrium is locally asymptotically stable, which implies that an infection will eventually die out. We also identify two distinguished disease-dependent equilibrium points for the first time, giving much deeper insight into long term behavior of the disease. Numerical simulations show the efficacy of vaccination toward reducing the newly infected individuals and suggest the model can predict stabilization for all compartments over time. The exposed and recovered population increases and stabilizes as the susceptible, infected, and vaccinated populations decline. In this regard, these are valuable insights into the progression of epidemics and put emphasis on vaccination programs. Our model provides a perspective on disease control by taking into account the interplay between vaccination and infection dynamics. It can be very useful for predicting future outbreaks and can guide public health policy. Future work will extend the model by adding quarantine measures to further hone our understanding of disease transmission.

***Keywords***: SEVIR model, epidemiology, ordinary differential equation, stability analysis, basic reproduction number, numerical solutions

**MSC:** 74H15, 34A07

## Abbreviation

| | |
|---|---|
| SEVIR model | Susceptible-Exposed-Vaccinated-Infected-Recovered model |
| SIR model | Susceptible-Infected-Recovered model |
| NPIs | Non-Pharmaceutical Interventions |
| HPM | Homotopy Perturbation Method |
| SEIQR model | Susceptible-Exposed-Infectious-Quarantined-Recovered model |


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SVIR model  Susceptible-Vaccinated-Infected-Recovered model

## 1. Introduction

Epidemic modeling has been one of the important tools to understand the dynamics of disease spread and guide the choices of public health interventions. Kermack and McKendrick's seminal work introduced the very basic SIR model that is one of the most widely used models in epidemiology. This model, and its many extensions, including the (SEIR) Susceptible-Exposed-Infected-Recovered model [1], have been critical in determining the basic reproduction number ($R_0$), an important indicator of whether an epidemic will spread or decline. Other early works in epidemic modeling include those of Ross [2–4], who developed probabilistic methods for studying disease transmission, and Kendall [5], who studied both deterministic and stochastic epidemic models.

In the current scenario of global health pandemics, such as the ongoing COVID-19 pandemic, NPIs, like quarantine and lockdowns along with vaccine strategies, have been studied in detail with respect to their effectiveness in controlling endemic infections. According to Baker et al. [6], a long-term consideration of the effects of NPIs on endemic infections shows that these interventions actually change the course of diseases even after the initial waves subside. Similarly, Volpert et al. [7] and Zhou et al. [8] discussed how quarantine and media reporting contribute to the control of COVID-19. Thus, timely public health response can mitigate the spread of infectious diseases.

The integration of vaccination into epidemic models has gained more attention in recent times. For instance, an example of how vaccination strategies can be integrated into epidemic modeling frameworks is the model proposed by Liu et al. [9], known as the (SVIR) Susceptible-Vaccinated-Infected-Recovered model. Such models and others have been very significant in understanding the role that vaccination plays in reducing the susceptible population and slowing down the spread of diseases.

Fuzzy logic and fractional calculus have further been used for modeling diseases with uncertainties as well as complexities. Bharathi et al. introduced fuzzy fractional epidemic models that captured stochastic nature of spreading diseases, introducing delay differential equations and stiff systems to allow for such time delays as exist at the transmission or recovery periods. These models give more realistic representations of the epidemic process, especially for diseases like COVID-19 that involve complex dynamics. Likewise, Windarto et al. used fractional derivatives to model the transmission of dengue, thus demonstrating the power of these models in reflecting the non-integer order behavior of infectious diseases.

HPM has been shown to be a strong tool to be applied in the solution of nonlinear differential equations in the study of epidemic models. Rekha et al. [10, 11] used HPM for the modeling of diseases dengue fever, listeriosis, and anthrax. Thus, through analytical solutions of otherwise challenging-to-solve epidemic models, this work has come forward. Saranya et al. [12] took it further into enzymatic reactions, which showed this method's flexibility in use between biological and epidemiological applications.

Further extensions of the SEIR model are an important part of modern research. Kalpana et al. In [13], they have presented the new simultaneous SEIQR model for providing insights concerning the different compartments' dynamics during the outbreak of disease. In [14], their more recent extension in fractional models was used for analyzing fuzzy as well as crisp solutions applied to the pandemic models of diseases, which could replicate the outbreak scenario of something like COVID-19 diseases in the real world as well.

This study extends the established literature by formulating a new SEVIR model in which vaccination is directly incorporated into the exposed population, filling the gap of models such as the SVIR model [9]. In previous models, vaccination had been considered a direct step from susceptible to recovered or immune. The SEVIR model, however includes a more complex interaction-where vaccinated individuals go through an exposed stage before developing recovery or infection. Highly relevant for diseases such as COVID-19, vaccines may not offer immunity as soon as administered and hence the incubation period prevails.

It presents the disease-dependent existence of two equilibria. As it is seen in Bharathi et al.'s work, the standard models usually emphasize only one equilibrium point. Multiple equilibria make more exciting possibilities for different phases of epidemic transmission to exist with vaccination.

Apart from the new model of SEVIR, this work relies on the early work of Kermack and McKendrick [15–17], Ross [2–4] and Kendall [5], who developed the framework for modern models of epidemiology. Analysis of the basic reproduction number ($R_0$), as well as stability analysis at equilibrium points, remains at the heart of epidemic modeling, and this work extends these techniques by including dynamics for vaccination in the exposed population.

This paper introduces a more detailed SEVIR model with vaccination included in the exposed population and finds several disease-dependent equilibrium points, a question left open by all previous literature. New insights shed light on parts of the long-run dynamics of infectious diseases and feed back into more complex structures meant to capture the influence of vaccination strategies. Future work will proceed along the lines of what quarantine measures and other further public health interventions imply as their role, through such foundational works as those laid out by Baker et al. [6], Kalpana et al. [13], and so forth-to build models that better comport to disease control. Paras. We now detail, in section 2, what prerequisites for, as well as the form that, SEVIR can take, by first derivatively obtaining the governing equations and then laying out foundation assumptions. In section 3, we discuss the equilibrium points and perform a complete stability analysis. In section 4, we carry out numerical simulation to demonstrate the dynamics for the proposed model under varying parameter settings. In this work, we conclude it in section 5, summarizing briefly the key findings and laying out potential directions for the future research work.

## 1.1 *Data assumptions*

The data employed in the numerical simulations of the SEVIR model is assumed rather than actual real-world observations. The assumptions are made in the context of illuminating qualitative behavior of the model as well as demonstrating potential theoretical efficacy in capturing disease dynamics , the Choices for parameters were based on reasonable ranges found in literature related to epidemic modeling [18–23]. It is true that several authors have used the assumed data for the preliminary validation of the models in the area of epidemic modeling, especially when the real-time data is not available. For instance, Pandey et al. [22] and Cai et al. [23] use assumed parameters within their studies to depict the applicability of their models before using real-world data. This practice goes on to present various hypothetical scenarios that will later guide the data gathering process. Validation of Assumed Data and the Model The future work would be directed towards fitting the model to real-world data to produce quantitative accuracy for the dynamical forecast of the disease. Our validation of the model is essentially based on both theoretical analysis and numerical simulation. Here, for this study, we utilized assumed data that would explain the behavior of the SEVIR model under a variety of different parameter settings. This methodology is particularly common in mathematical epidemiology when real data is unavailable or unnecessary to use when conducting theoretical investigations [13, 22]. Section 3.3 provides the stability analysis that confirms the model's ability to capture the dynamics of disease spread, validating the theoretical consistency of the SEVIR model. The qualitative agreement of the model with previously established epidemic models, such as those described in [22, 23], provides further validation. For example, the work by Pandey et al. on the fractional derivative approach in vaccination models reveals the requirement of including vaccination dynamics in the model, and exactly the same thing our SEVIR model embodies. In a similar sense, the global analysis by Cai et al. on vaccination models provides evidence to the structural and functional design of our compartments regarding vaccination. Although this work does not include actual data, future work will be devoted to the estimation of parameters using actual epidemiological data, as described in , to further improve empirical validation of the model. This will allow us to compare the model's predictions with observed dynamics of disease, which will further enhance practical applicability.

## 1.2 *Novelty and contribution of the model*

While epidemic modeling, and specific models using the SEIR type of structure, received enormous attention in the literature, the SEVIR model developed in this contribution holds several key novelties that distinguish it from what has been considered earlier: the vaccination compartment appears naturally as an intrinsic constituent in the dynamics of

disease evolution. This enables a more realistic illustration of epidemic behavior, specially in the context of vaccination strategies modern which have become critical in controlling recent pandemics. For example, it has been found that vaccination models using fractional orders play an important role in determining disease dynamics, as can be visualized from the research made on dengue transmission models in Nepal with fractional derivatives [22]. It identified two different disease-dependent equilibrium points, which, to the best of our knowledge, have not been reported in previous epidemic models. The outcome contributes a new dimension to the stability analysis and allows deeper insights into the possible long-term behavior of diseases in populations with different levels of susceptibility and immunity. This also fits with global analysis of epidemic models with vaccination, undertaken in [23], which provides an additional motivation for carefully including vaccination strategies in determining outcomes of disease, a direction we take here. This model further helps to understand the stabilization of an epidemic in populations under ongoing vaccination. Numerical simulations indicate that the exposed and recovered populations are likely to stabilize over time, and it is the vaccinated population that plays a key role in preventing future outbreaks. The stabilization behavior is not so well captured in traditional SEIR models; hence, our approach is particularly relevant for assessing long-term epidemic control. Lastly, this paper's methodological approach overcomes the existing model shortcomings by using a stability analysis that is much more in-depth and showing the dynamics of disease caused by the different compartments of influence-for example, the vaccination effect. Other possible expansions on this model include putting quarantine measures in place. Indeed, it is these new theory findings, practical applications and novel methods that make a significant contribution in order to extend and considerably amend current knowledge in the model of epidemics.

## 2. Model formulations with preliminaries

To describe the model appropriately, one should indicate what key parameters control the transmission dynamics of disease. Those are, in fact, parameters that describe all biological and social factors that regulate movement of individuals between compartments: transmission rates, recovery rates, and vaccination efficiency. At first, we make several assumptions regarding the state of the population at the observation time. Let's assume we have a population of 1,000, where 100 people fall into the susceptible category, meaning they are open to infection but are not yet infected. Simultaneously, 200 individuals are exposed to the infection, having been exposed to the virus but not yet symptomatic or infectious. We further assume 100 people have been immunized and given some level of immunity against the disease, even though they are not 100.

Another 200 people are actively infected. These are the sections of the population that actually have the infection and would thus be contributing to the spread of the disease. Finally, 400 are counted as recovered. This means that they have recovered from the infection and, at least temporarily, have acquired immunity against further infections. Compartment sizes are dynamic, because through these interactions, such as disease transmission, vaccination, and recovery, the size of the compartments changes with time. Each compartment is linked together by transition rates, quantifying the rates of flow of individuals between different states-for example, how rapidly susceptible individuals become exposed, how well the vaccine stops disease progression, or how rapidly infected individuals recover.

These transition rates depend upon a variety of factors: the transmission potential of the infection, the effectiveness of public health interventions, and the natural course of infection. Table 1 provides detailed parameter descriptions, including the rates of transmission between compartments. It outlines the various assumptions and values that are crucial for simulating the model's behavior and analyzing the spread of the disease under different conditions. Each parameter plays their role to capture the complexity in disease dynamics, and careful calibration is required from these values which ensure the model holds a close resemblance to scenarios in real life.

Taking little modifications in the considered values of the authors Kalpana et al. in their works [13, 14], we are developing our new model for which parameters and values are presented in the following section Figure 1.

**Table 1.** Symbols and descriptions

| Symbols | Descriptions | Values |
|---|---|---|
| $S$ | Susceptible population | - |
| $E$ | Exposed population | - |
| $V$ | Vaccinated population | - |
| $I$ | Infected population | - |
| $R$ | Recovered population | - |
| $S_0$ | Initial number of susceptible individuals | 100 |
| $E_0$ | Initial number of exposed individuals | 200 |
| $V_0$ | Initial number of vaccinated individuals | 100 |
| $I_0$ | Initial number of infected individuals | 200 |
| $R_0$ | Initial number of recovered individuals | 400 |
| $\alpha$ | Rate of transition from susceptible to exposed | 0.07 |
| $\beta$ | Rate at which exposed individuals become infected | 0.08 |
| $\gamma$ | Rate at which exposed individuals are vaccinated | 0.09 |
| $\mu$ | Rate of recovery for infected individuals | 0.3 |
| $\delta$ | Rate at which exposed individuals recover | 0.1 |

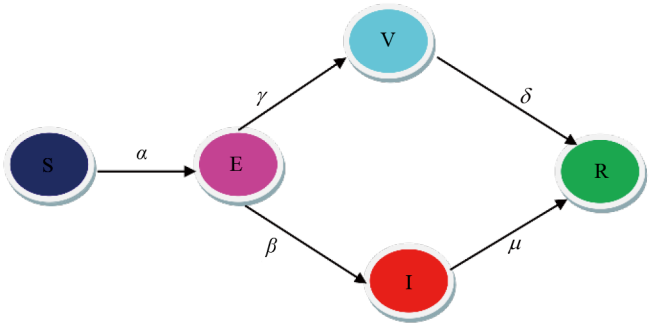


**Figure 1.** SEVIR-model formulation

$$\frac{dS(t)}{dt} = -\alpha S(t)E(t)$$

$$\frac{dE(t)}{dt} = \alpha S(t)E(t) - \gamma E(t)V(t) - \beta E(t)I(t)$$

$$\frac{dV(t)}{dt} = \gamma E(t)V(t) - \delta V(t)R(t)$$

$$\frac{dI(t)}{dt} = \beta E(t)I(t) - \mu I(t)R(t)$$

$$\frac{dR(t)}{dt} = \delta V(t)R(t) + \mu I(t)R(t) \tag{1}$$

The initial conditions are satisfied and therefore the total population of size N becomes constant. $S_0 + E_0 + V_0 + I_0 + R_0 = N$.

# 3. Equilibrium points, reproduction number, and stability examination

## 3.1 *Equilibrium points*

Equilibrium points occur when the system reaches a steady state, meaning no further changes take place, i.e.,

$$S'(t) = 0,\ E'(t) = 0,\ V'(t) = 0,\ I'(t) = 0,\ R'(t) = 0.$$

Through calculations, we identified one disease-free equilibrium point, denoted as $D_f$, and two equilibrium points that depend on the disease, denoted as $D_{d1}$ and $D_{d2}$. The disease-free equilibrium point is:

$$D_f = (S_0,\ 0,\ 0,\ 0,\ 0).$$

The disease-dependent equilibrium points are given by:

$$D_{d1} = \left(0,\ E(0),\ \frac{-\beta}{\gamma}I(0),\ I(0),\ \frac{\gamma}{\delta}E(0)\right),$$

$$D_{d2} = \left(0,\ E(0),\ \frac{-\mu}{\delta}I(0),\ I(0),\ \frac{\beta}{\mu}E(0)\right).$$

Now, with specific parameter values, the equilibrium points become:

$$D_f = (100,\ 0,\ 0,\ 0,\ 0),$$

$$D_{d1} = (0,\ 200,\ -177.778,\ 200,\ 180),$$

$$D_{d2} = (0,\ 200,\ -600,\ 200,\ 53.3333).$$

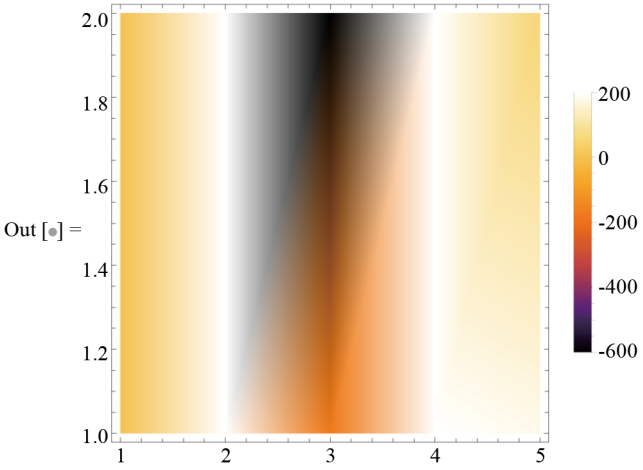


**Figure 2.** Density plot of disease dependence equilibrium points

In a biological context, interpreting equilibrium points mathematically often brings up questions about the feasibility of negative values, particularly in population dynamics. Mathematically, an equilibrium point represents a state where the system undergoes no change-in other words, where the derivatives in each compartment equal zero, reflecting a steady-state condition. However, if an equilibrium point includes negative values, it doesn't necessarily imply a literal negative population. Rather, it represents the underlying trend or transition within the model. A negative equilibrium component indicates that, at this steady state, the respective compartment is not self-sustaining and effectively has a net outflow, transitioning backward in the model structure to a previous compartment. This could imply that in the disease dynamics, individuals are moving out of this compartment more rapidly than they are entering, and so the compartment "declines" at equilibrium. For example, if a vaccinated compartment has a negative value, this might suggest that, in the modeled conditions, individuals are more likely to transition from vaccinated to recovered than to stay in the vaccinated state at equilibrium. In short, a negative equilibrium value in this context does not denote a literal negative population but instead highlights a population flow direction within the model where specific compartments are shrinking rather than sustaining themselves independently at equilibrium.

In order to visualize the density of the region where the disease dependence is large between these two disease dependance equilibrium points we have presented the density plot in the above Figure 2.

## 3.2 *Basic reproduction number-estimation*

Calculation of Basic Reproduction Number: $R_0$.

We are using the notation $R_0$ instead of traditional use of $R_0$ to denote basic reproduction number. It can be seen clearly that secondary susceptible can be formed by the influence of both exposed and infected classes. So by next generation matrix techniques for large domains, we can find the basic reproduction number. Consider,

$$E'(t) = \alpha S(t)E(t) - \gamma E(t)V(t) - \beta E(t)I(t);$$

$$I'(t) = \beta E(t)I(t) - \mu I(t)R(t). \tag{2}$$

The Jacobian matrix of (2) is provided by

$$F = \begin{pmatrix} \alpha S(0) - \gamma V(0) - \beta I(0) & -\beta E(0) \\ \beta I(0) - \mu R(0) & \beta E(0) - \mu R(0) \end{pmatrix}, \tag{3}$$

Now, $F$ can be decomposed as $F = F_1.F_2$ where

$$F_1 = \begin{pmatrix} \alpha S(0) & 0 \\ 0 & 0 \end{pmatrix}, \tag{4}$$

$$F_2 = \begin{pmatrix} -(\gamma V(0) + \beta I(0)) & -\beta E(0) \\ \beta I(0) - \mu R(0) & \beta E(0) - \mu R(0) \end{pmatrix}, \tag{5}$$

Now, let us calculate $X$ from $X = -F_2$ then

$$X = \begin{pmatrix} (\gamma V(0) + \beta I(0)) & \beta E(0) \\ \mu R(0) - \beta I(0) & \mu R(0) - \beta E(0) \end{pmatrix}, \tag{6}$$

Now we have found $Adj.X$, $|X|$ and $X$ as follows

$$Adj.X = \begin{pmatrix} \mu R(0) - \beta E(0) & -\beta E(0) \\ \beta I(0) - \mu R(0) & \gamma V(0) + \beta I(0) \end{pmatrix}, \tag{7}$$

$$|X| = (\mu R(0) - \beta E(0))(\gamma V(0) + \beta I(0)) + (\beta E(0))(\beta I(0) - \mu R(0)) \tag{8}$$

$$X^{-1} = \frac{1}{|X|}(Adj.X)$$

$$F_1 X^{-1} = \frac{\begin{pmatrix} \alpha S(0) & 0 \\ 0 & 0 \end{pmatrix} . \begin{pmatrix} \mu R(0) - \beta E(0) & -\beta E(0) \\ \beta I(0) - \mu R(0) & \gamma V(0) + \beta I(0) \end{pmatrix}}{(\mu R(0) - \beta E(0))(\gamma V(0) + \beta I(0)) + \beta E(0)(\beta I(0) - \mu R(0))} \tag{9}$$

The Basic reproduction number is calculated from $R_0 = \rho F_1.X^{-1}$ where $\rho F_1.Y^{-1}$ is the spectral radius of the matrix $F_1.X^{-1}$ and is given by $\max(|\lambda_{R0}|)$ where $\lambda$ is the eigenvalue of the $F_1.X^{-1}$. The eigenvalues of $\lambda_{R0} = F_1.X^{-1} = (0.7778,\ -0.119)$. $R_0 = \max(|\lambda_{R0}|) = 0.778$ is the basic reproduction number for which the expression is given by

$$R_0 = \frac{\alpha S_0(-\beta E_0 + R_0\mu)}{\beta E_0(\beta V_0 - R_0\mu) + (\gamma V_0 + \beta I_0)(-\beta E_0 + R_0\mu)}.$$

Since the basic reproduction number $R_0 < 1$ the disease will not produce new secondary susceptibles. In detail, when $R_0 < 1$, the disease-free equilibrium (DFE) of the system is locally asymptotically stable. This implies that in the absence

of initial infections, or when the number of infections is very small, the system will naturally return to a disease-free state over time. In other words, when the basic reproduction number $R_0$, which represents the average number of secondary infections caused by a single infected individual in a fully susceptible population, is less than 1, each infection does not lead to enough new infections to sustain an outbreak. Consequently, the infection dies out over time, and the system stabilizes at a disease-free state. This confirms that small perturbations from the DFE decay over time, reinforcing the state where the disease cannot persist in the population.

## 3.3 *Stability analysis*

**Theorem 1** The system with the (1) is locally asymptotically stable when all the eigenvalues of characteristic polynomial obtained by the linearization of (1) are having negative real parts.

**Proof.** The linearized form of (1) can be presented using Jacobian matrix.

$$J = \begin{pmatrix} -\alpha E(0) & -\alpha S(0) & 0 & 0 & 0 \\ \alpha E(0) & \alpha S(0) - \gamma V(0) - \beta I(0) & -\gamma E(0) & -\beta E(0) & 0 \\ 0 & \gamma V(0) & \gamma E(0) - \delta R(0) & 0 & -\delta V(0) \\ 0 & \beta I(0) & 0 & \beta E(0) - \mu R(0) & -\mu I(0) \\ 0 & 0 & \delta R(0) & \mu R(0) & \delta V(0) + \mu I(0) \end{pmatrix}$$

On solving the above matrix by substituting the values of $S(0)$, $E(0)$, $V(0)$, $I(0)$, & $R(0)$ we found the the characteristic polynomial as

$$\lambda^5 + 88\lambda^4 + 3628\lambda^3 + 92688\lambda^2 + 557760\lambda - 3.52088 \times 10^{-9} = 0.$$

The corresponding eigenvalues are found to be

$$(-47.4965,\ -16.1756 + 34.3343i,\ -16.1756 - 34.3343i,\ -8.15219, -6.31254 \times 10^{-15}).$$

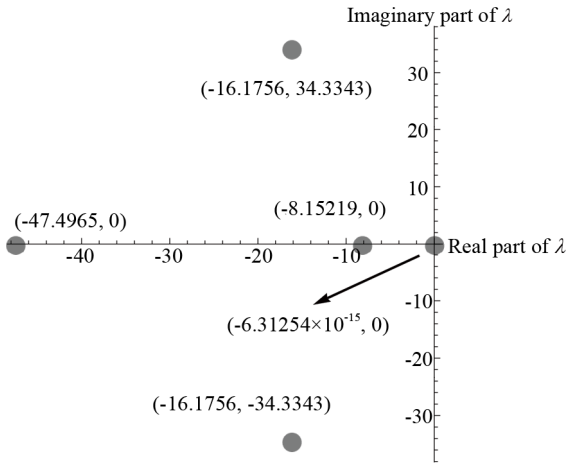


**Figure 3.** Complex plane representation of eigenvalue

Since all the eigenvalues are having the negative real parts the system we considered is locally asymptotically stable which is clearly shown in Figure 3. For our model, we assess stability by examining the eigenvalues of the Jacobian matrix evaluated at the equilibrium points. The local asymptotic stability of the system is confirmed when all eigenvalues possess negative real parts, as this implies that small perturbations in the state variables will decay over time, leading the system back to equilibrium. Specifically, this condition means that the dynamics around the equilibrium points are dominated by forces that restore the system to its stable state, rather than diverging from it. Consequently, since all the eigenvalues in our analysis exhibit negative real parts, we conclude that our system is locally asymptotically stable, meaning any small deviation from equilibrium will gradually diminish, returning to the original equilibrium position.

## 4. Numerical simulations

To visualize the behavior of the model (1), we provide the following graphs, illustrating the time evolution of the different compartments over a unit interval of $t$. Figures 4, 5, 6, 7, and 8 depict the trajectories of the susceptible, exposed, vaccinated, infected, and recovered groups, respectively. Additionally, a comprehensive plot with an enlarged view of all compartments combined is shown in Figure 9 to offer better insight into the dynamics.

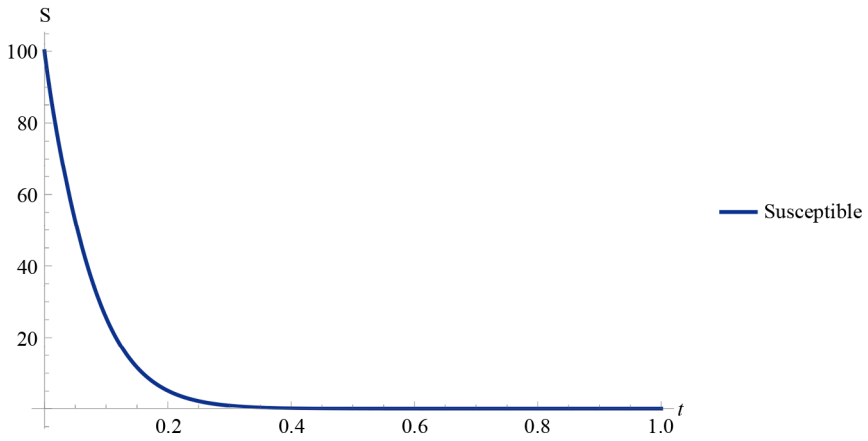


**Figure 4.** Susceptible cases

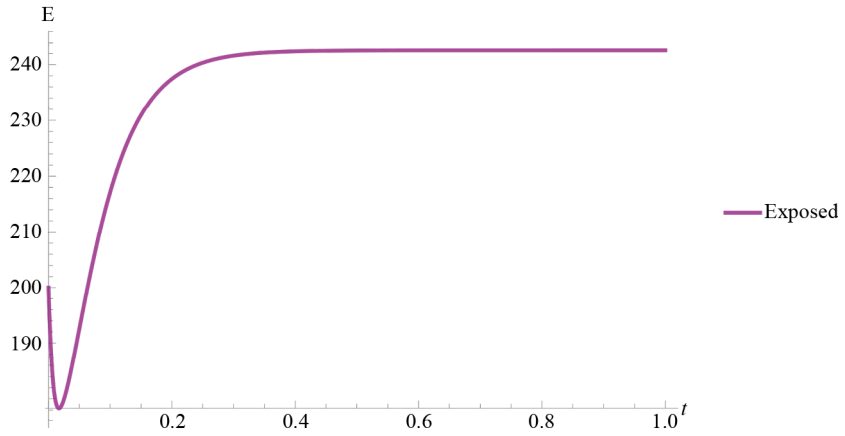


**Figure 5.** Exposed cases

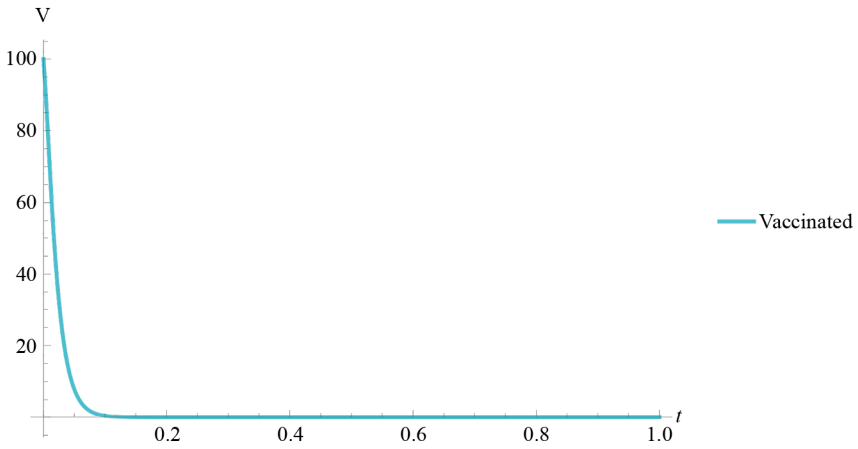


**Figure 6.** Vaccinated cases

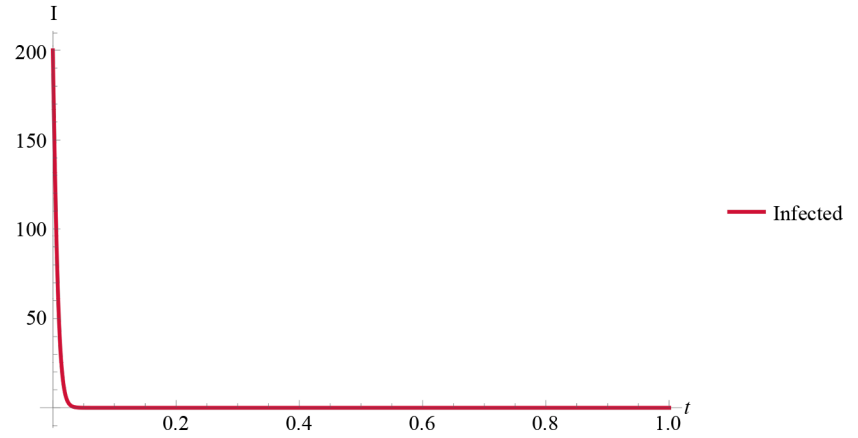


**Figure 7.** Infected cases

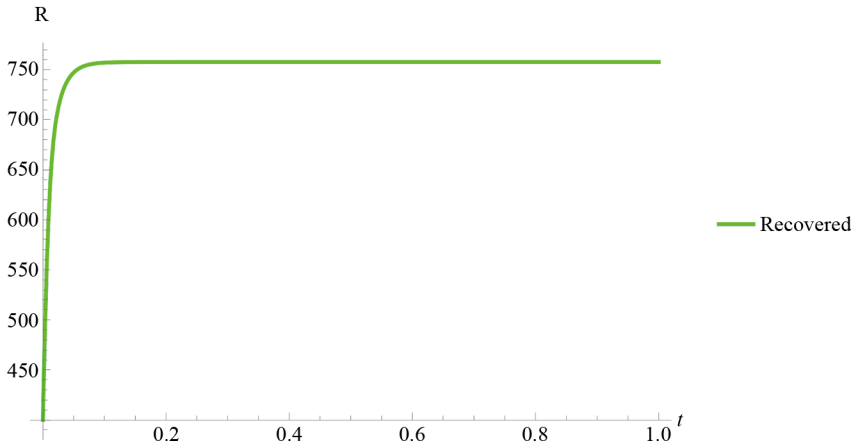


**Figure 8.** Recovered cases

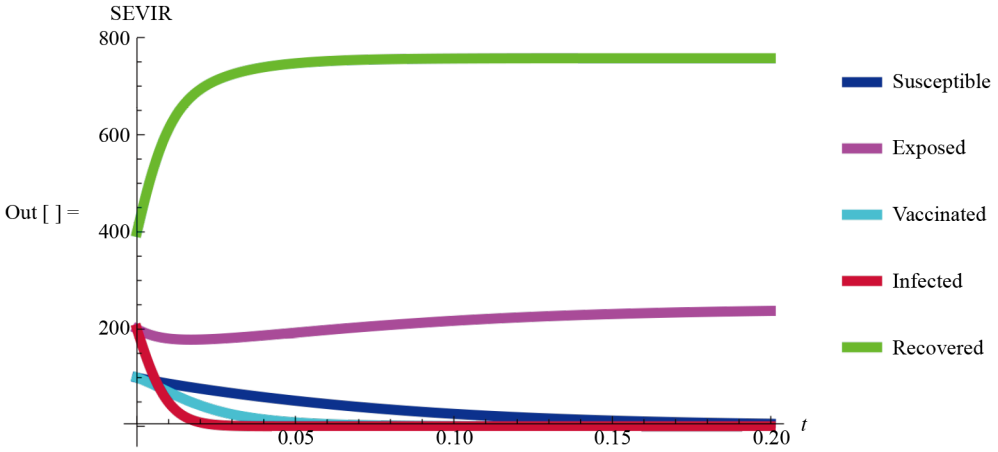


**Figure 9.** SEVIR-cases

### 4.1 *Biological interpretations of the numerical simulations*

Incorporating numerical simulations under various parameter regimes would enable a clearer understanding of the transient behavior of the compartments. Such simulations would allow us to explore how quickly the system converges to equilibrium, or whether transient spikes in infections may occur before stability is achieved. Biologicaly the spread of disease is not happening overnight. It is the transmission happening every second. In order to visualize it very closely the rapid changes that is happening in a day we have presented the Figures 4, 5, 6, 7, 8 and in Figure 9 combining them, we visualize them in how in a few hours (0.20 day) the transistion between the systems solutions is happening. This will make the readers to understand the disease is epidemic spreading quickly and gets dowin in a day due to the implementation of the vaccinaltion.

## 5. Conclusion

This work introduces a new SEVIR epidemic model that emphasizes the dynamics of the disease by considering vaccination strategies and their latent impact on immunity. Vaccination is more realistically modeled compared with the standard model while considering progressive growth in immunity over time. This has involved deep mathematical analysis that eventually yields several epidemiological parameters such as basic reproduction number $R_0$ and multiple equilibrium points that exist in the model. Equilibrium points in the disease are both disease-free and dependent on diseases. This work introduces and analyzes two disease-dependent equilibrium points that are a new contribution to the literature. In fact, most of the existing models hardly consider such equilibria, especially in vaccination delay and interaction between the vaccinated and exposed populations. This provides important information regarding long-term epidemic behavior, constituting a new insight on the persistence or extinction of the diseases in vaccinated populations. The stability analysis showed that the system under given parameters and initial conditions has a tendency to be locally asymptotically stable about the equilibrium points. It thus means that small perturbations from these points would not cause unbounded outbreaks; thus, for similar data, it is unlikely that new infections or a second wave of the epidemic will occur. In addition, the model illustrates that even as exposure to the disease continues to build, a balance is attained whereby the infected population declines and people get healthier. This model has several practical implications for public health planning. For instance, taking into account vaccination delays, it gives much more realistic predictions about when vaccination campaigns are likely to be most effective in terms of timing and impact. In this sense, it will enable public health officials to understand when immunity will become widespread and how long transmission might continue. Such insight is highly relevant to diseases such as COVID-19, for which vaccine rollout has been slow and where immunity

accrues over time. The model also provides a theoretical framework for examining the long-term consequences of vaccine distribution strategies on controlling epidemic waves.

### 5.1 *Future directions*

While this research moves the modeling of vaccination strategies forward within epidemic frameworks, the model itself could be improved upon by incorporating other real-world complexities in follow-up work. The following steps will include quarantine and variation in immunity level based upon vaccine efficacy. Quarantine dynamics would help assess what kinds of isolation policies might influence the diffusion of disease, and differential vaccine efficacy would make the modeling of heterogeneous populations more representative. Explore time-dependent interventions; for example, booster vaccination campaigns would further be able to take this model to the reality of situations. Other future research directions include validating the robustness of the model by testing it with different infectious diseases that are characterized by a different set of transmission properties, for example, influenza or emerging zoonotic diseases. Another type of validation is possible by making comparative studies to ascertain whether the model can operate under different epidemiological conditions. In addition, introducing stochastic elements into the model could help describe the effect of random events or uncertainty in the disease transmission mechanism, thus potentially making the model more predictive. More importantly, we suggest here that elementary model analysis can also be given by stability and numerical methods. We would complement such an approach by introducing a sensitivity analysis which analyzes how parameter changes impact the system, specifically regarding the quantity known as the basic reproduction number $R_0$. By the introduction of sensitivity analysis, better knowledge in the effect of parameter estimation on the model prediction would provide the much needed accuracy toward strategic planning of disease control. This way, the proposed SEVIR model not only extends existing epidemic modeling literature with the introduction of vaccination delays and multiple disease-dependent equilibria but also gives more practical insights, crucial in the molding of future public health interventions. This work therefore gives an important framework to better understand and control epidemic outbreaks within a more nuanced and realistic framework of complexities involving vaccination and disease dynamics.

## Conflict of interest

The authors declare no competing financial interest.

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