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MATHEMATICAL MODELING AND NUMERICAL ANALYSIS OF THE EFFECTS OF COVID-19 VACCINE ON HEART ATTACKS

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ABSTRACT

The Coronavirus disease is an infectious disease caused by the SARS-CoV-2 virus. The disease spreads from person to person and is known to cause severe acute syndrome and death. A vaccine against the disease was developed at short notice to reduce its fatal impact. The reduction in deaths was achieved with the vaccine. However, in addition to this reduction, the vaccine has also been associated with side effects. One of these is an observed increase in heart attacks in vaccinated people. This study investigates the effect of the COVID-19 vaccine on heart attacks. A mathematical model has been developed, and susceptible individuals have been divided into groups of those who have had a heart attack before vaccination and those who have not. Using the developed mathematical model, the study discusses whether the increase in heart attack cases after COVID-19 has been related to the vaccine. First, a diagram of the system has been obtained, and the basic reproduction number has been calculated. Equilibrium points were determined to assess whether the system has correct dynamics. The stability of the equilibrium points has been analyzed using eigenvalues. Numerical calculations and graphs have also been provided to support the findings. It has been demonstrated that the model can be examined with real-life data, allowing the impact of the COVID-19 vaccine on heart attacks to be analyzed. This study has made a significant contribution to literature in this field.

Keywords: COVID-19 Vaccination, Equilibrium point, Heart attack, Runge-Kutta method.

1 INTRODUCTION

Coronavirus disease (COVID-19) was first identified on 13 January 2020 following investigations into a group of patients in Wuhan Province, China, who presented with symptoms of fever, cough and shortness of breath. COVID-19 belongs to a family of viruses that can cause

disease in animals and humans. Coronaviruses can also lead to more serious diseases such as Severe Acute Respiratory Syndrome (SARS). In a short time, this virus has spread around the world, prompting various efforts to control it. One of these is vaccination. In addition, one of the most effective ways to identify ways to combat this virus and plan these strategies is through mathematical modelling. For this reason, many mathematical modelling studies have been carried out to analyze and understand the effects of the disease.

In 2020, Zeb et al. developed a mathematical model incorporating an isolation class to understand the spread of COVID-19 infection. The model concluded that isolating infected individuals could reduce the risk of future spread and that human contact is the primary cause of epidemics. Furthermore, numerical solutions were obtained to enhance the accuracy of the model using the Nonstandard Finite Difference (NSFD) method and the fourth-order Runge-Kutta method [1].

Adiga et al. (2020) stated that the COVID-19 pandemic has been experienced as a major global health crisis over the past 100 years, with increasing impacts on the economy, society, and health. Mathematical models have played a significant role in informing public health policies and supporting social distancing measures. The use of various models in the fight against the pandemic has been comprehensively addressed [2].

Tuan et al. modeled the spread of COVID-19 using Caputo fractional derivatives, calculating the equilibrium points and reproduction numbers. They proved that the model has a unique solution using fixed point theory and solved the system by obtaining approximate solutions with the Adams-Bashforth-Moulton method. As a result, the basic reproduction number was found, indicating that the epidemic is still ongoing [3].

Peter et al. developed a COVID-19 model using an eight-dimensional differential equation system, considering the first and second doses of vaccination. The model was analyzed to obtain the control reproduction number, and the equilibrium points of the system were examined to assess the stability of the model. In the model calibrated with vaccination data from Malaysia for 2021-2022, it was determined that the transmission rate, vaccination dose rates, and recovery from the second dose had a positive effect. Numerical measurements showed that vaccination significantly reduced the number of infected individuals [4].

Logeswari et al. modeled the spread of the SARS-CoV-2 virus using the Atangana–Baleanu derivative in an equation and examined the existence and singularity of the system using the fixed-point method. They conducted digital simulations to predict the virus spread in

India. The study provides a permanent analysis of the infectious dynamics of COVID-19, enabling the evaluation of epidemics [5].

Magadum et al. investigated the effects of COVID-19 on the body systems. They found that individuals with pre-existing conditions such as cardiovascular diseases, diabetes, hypertension, and obesity experience more severe courses of the disease, with the virus's destructive effects on the heart becoming even more pronounced. COVID-19 leads to various conditions in the cardiovascular system, including myocarditis, acute myocardial injury, stress-induced cardiomyopathy, cardiogenic shock, arrhythmia, and eventually heart failure (HF). It was noted that the virus directly damages heart tissue through ACE2 receptors, and these effects are exacerbated by systemic treatment, hypoxia, and cytokine storms. The development of a comprehensive understanding of the viral effects and the advancement of new treatment methods has facilitated the protection of these complements and minimized long-term damage [6].

Özköse et al. developed a fractional-order pandemic model to explain the spread of the COVID-19 pandemic and its relationship with heart attacks. The model, considering the impact of Omicron's strength, analyzed the relationship between heart attacks and quarantine. The data for the model were determined with the help of real data. Numerical simulations showed an increase in heart attack cases during the period of rising Omicron cases, but it was expected that the risk of heart attacks would decrease as Omicron cases declined [7].

In their study, Ahmad et al. developed a mathematical model using the fractional fractal operator (FFO) to investigate the effects of the SARS-CoV-2 (SC-2) virus on heart attacks. The model was evaluated using Lyapunov stability analyses to assess the local and global stability of the SC-2 virus. To validate the combined effects of COVID-19 and heart patients, solutions for the fractional-order system were derived with the help of the advanced FFO tool for different fractional values. This model, developed to understand the impact of the SC-2 outbreak on heart attack patients and observe its global behavior, will be useful for future prediction and control strategies [8].

Evirgen et al. developed a new comprehensive model to differentiate between heart attacks and Omicron characteristics. The model includes two adjustable parameters to control the number of individuals who could be infected and carry Omicron, and analyzes the possible locations of these individuals. Digital simulations using real COVID-19 data from Turkey have shown positive results on the proposed control samples and Omicron characteristics [9].

The purpose of this study is to analyze whether COVID-19, in relation to vaccination, affects heart attacks by performing stability analyses of a mathematical model created using first-order differential equations. The key difference of this study from the literature is that the susceptible population group is expressed in two subcategories. This population is divided into individuals who experienced a heart attack before vaccination and those who did not. This allows the mathematical model to predict changes in the number of both vaccinated and unvaccinated individuals, not just those who contracted COVID-19. Thus, a mathematical model has been developed that can work in alignment with real-world data.

This study consists of five sections. The second section provides basic information and definitions related to the developed model. These include the definitions of the basic reproduction number, stability analysis, and the Runge-Kutta method. In the third section, modeling of the effect of the vaccine on heart attacks is carried out using differential equations, and a diagram is created. Continuing in the third section, the equilibrium points for both diseased and healthy states are found, and the stability analysis of these points is performed. In the fourth section, using assumed parameter values, the consistency of the equilibrium points is demonstrated by finding the corresponding eigenvalues. Subsequently, numerical solutions of the system are presented with the help of the Runge-Kutta method, and convergence to the equilibrium points is shown. The fifth section provides information about the suitability of the developed model for its purpose and the results of the obtained data.

2 BASIC INFORMATION AND DEFINITIONS

This section provides the necessary basic information and definitions for finding and analyzing the equilibrium points. Additionally, the formulation of the Runge-Kutta method used for the numerical solution is also included in this section.

The basic reproduction number (R_0) is a measure of the potential for disease spread in a population. R_0 is a threshold for the stability of the disease-free equilibrium point and is related to the peak and final size of an epidemic. Predictions about the course of the epidemic can be made based on R_0 . When $R_0 > 1$, the infection leads to an epidemic [10]-[12].

Definition 2.1: To calculate basic reproduction number, the next generation matrix method can be used. Let's consider the next generation matrix G . The matrix G consists of two parts F and V^{-1}.

$$\mathrm{F} = \left[\frac{\partial \mathrm{F_i}(\mathrm{x_0})}{\partial \mathrm{x_j}}\right], \qquad \mathrm{V} = \left[\frac{\partial \mathrm{V_i}(\mathrm{x_0})}{\partial \mathrm{x_j}}\right] \tag{1}$$

Here, F_i represents new infections, and V_i represents transfers of infections from one compartment to another. x_0 denotes the disease-free equilibrium points. R_0 is the largest eigenvalue of the matrix $\mathrm{G} = \mathrm{F}.\mathrm{V}^{-1}$ [11].

Differential equations may not have an analytical solution, or finding these solutions can be quite difficult. In such cases, equilibrium points and stability analyses can be used to make interpretations about the solutions of the given system of equations.

Equilibrium points and stability analyses used in the solutions of equation systems are not primarily meant to provide quantitative information about the equations but rather to offer qualitative insights. In other words, they help us make interpretations about the solutions of nonlinear equation systems that cannot be solved quantitatively

Definition 2.2: In the equation $\frac{dx}{dt} = f(t,x)$, if $f(t,x^*) = 0$ for some values of t, then $x^* \in R^n$ is called an equilibrium point.

The equilibrium points of the system are analyzed by examining the Jacobian matrices, and eigenvalues are found, which allow for the categorization of the equilibrium points. For the equilibrium point to be physically meaningful, it must meet certain stability criteria to a certain degree [13].

Definition 2.3: Let $p \in R^m$and $f = (f_1, f_2, f_3, \cdots f_m)$ be a function in. The Jacobian matrix of the function f at point p is denoted as $Df(p)$. The Jacobian matrix $Df(p)$ is given by [14]:

$$Df(p = \begin{bmatrix} \frac{\partial f_1}{\partial x_1} & \frac{\partial f_1}{\partial x_2} & \cdots & \frac{\partial f_1}{\partial x_m} \\ \frac{\partial f_2}{\partial x_1} & \frac{\partial f_2}{\partial x_2} & \cdots & \frac{\partial f_2}{\partial x_m} \\ \vdots & \vdots & \ddots & \vdots \\ \frac{\partial f_m}{\partial x_1} & \frac{\partial f_m}{\partial x_1} & \cdots & \frac{\partial f_m}{\partial x_m} \end{bmatrix} \quad (2)$$

The Jacobian matrix is a matrix that provides the linear approximation of a vector function and generally contains the derivatives of multivariable functions. The determinant of a function's Jacobian matrix is used to analyze the local stability of the system at equilibrium points. The characteristic equation is a determinant equation that must be solved to find the eigenvalues of a matrix, and the eigenvalues are the roots of this equation [14]-[20].

Definition 2.4: The behavior at each equilibrium point of the system can be qualitatively determined by finding the eigenvectors associated with each eigenvalue [13]:

- If all eigenvalues have negative real parts, the point is stable,
- If none of the eigenvalues have a real part, the equilibrium point is called hyperbolic,
- If at least one eigenvalue has a positive real part, the point is unstable,
- If at least one eigenvalue has a negative real part and at least one eigenvalue has a positive real part, the equilibrium point is a saddle point and is unstable.

Definition 2.5: The Runge-Kutta method is a numerical technique used to solve differential equations. For an initial value problem in general form, an iterative approach is used as follows:

$$y_{n+1} = y_n + h\sum_{i=1}^{k} w_i\, k_i \tag{3}$$

Here, h is the step size, and w_i are the weights of the Runge-Kutta method. k_i are the intermediate values calculated at each step, and they are usually computed as follows:

$$k_1 = f(t_n,\ y_n),\ k_2 = f(t_n + h/2,\ y_n + h/2\, k_1),\ k_3 = f(t_n + h/2,\ y_n + h/2\, k_2),$$
$$k_4 = f(t_n + h,\ y_n + h\, k_3).$$

This formula is commonly known as the 4th-order Runge-Kutta method (RK4), which provides high accuracy [21]-[22].

The 4th-order Runge-Kutta method utilised in this study is a highly reliable and accurate technique for the numerical solution of differential equations. The advantages of this method include high accuracy, ease of implementation, and effectiveness across a wide range of problems. RK4 improves the solution by using more information at each step, resulting in faster and more accurate results compared to lower-order methods. The accuracy of the method can be further enhanced by adjusting the step size (h), thereby enabling the model to generate more precise outcomes. Furthermore, the Runge-Kutta method exhibits high reliability in terms of sensitivity to initial conditions and the proximity to the solution. Its wide applicability to various types of differential equations, including linear, nonlinear, and stiff systems, renders it a popular choice in mathematical modelling and health sciences. The step-by-step implementation of this method, in conjunction with error analysis and associated processes, serves to enhance the clarity of the solution process and augment the reliability of the results [21]-[22].

3 DEVELOPMENT OF A NEW MATHEMATICAL MODEL AND ITS DIAGRAM

The investigation of the impact of the COVID-19 vaccine on heart attacks is of great importance for public health, as the potential effects of COVID-19 on the cardiovascular system have been shown to trigger critical conditions such as heart attacks. Therefore, mathematical modeling is necessary to assess the long-term effects of this situation. Mathematical models can help identify the most effective strategies by simulating disease spread and the impact of vaccination campaigns on public health. These models play a crucial role in determining the most effective vaccination policies to protect the health of individuals and communities and minimize the risks of complications such as heart attacks. In this section, a mathematical model has been developed to analyze whether there is a direct relationship between the increased number of heart attack cases after COVID-19 and the COVID-19 vaccines. The equilibrium points of the model have been determined, and stability analyses have been conducted to make inferences about the disease's progression.

In the modelling process, the transmission dynamics of the infection, along with its potential impact on the risk of developing heart attacks after infection, were given full consideration, thereby establishing a bidirectional interaction mechanism. In this context, cardiovascular complications that may be triggered by the infection, in particular the risk of heart attacks, were incorporated as a key component of the model. The model parameters were defined with consideration for biological processes, and the probability of experiencing a heart attack after infection was integrated into the model as a dynamic risk factor.

Mathematically, a transition rate was defined, representing the proportion of infected individuals who are at risk of experiencing a heart attack. This risk was assumed to vary depending on the severity of the infection, the individual's pre-existing cardiovascular history, and their vaccination status. The transition parameters were designed in such a way that vaccinated individuals have lower risks of both infection and post-infection heart attacks. The model was structured to simultaneously track the direct effects of the virus and its long-term cardiovascular outcomes. This holistic approach combines epidemiological analyses with clinical cardiology data, enhancing the interpretability of the study from a health sciences perspective.

A Susceptible, Susceptible, Vaccinated, Infected, Recovered (SSVIR) model has been developed to model whether heart attack cases in a population will continue or cease. In the

developed model, the population is divided into five subgroups: $S_1(t)$ Susceptible individuals who have had both a heart attack and COVID-19, $S_2(t)$ Susceptible individuals who have had COVID-19 but not a heart attack, V(t) Vaccinated individuals, I(t) Infected individuals and R(t) Individuals who have acquired immunity or have recovered from the disease.The diagram of the system for the developed model is shown as follows:

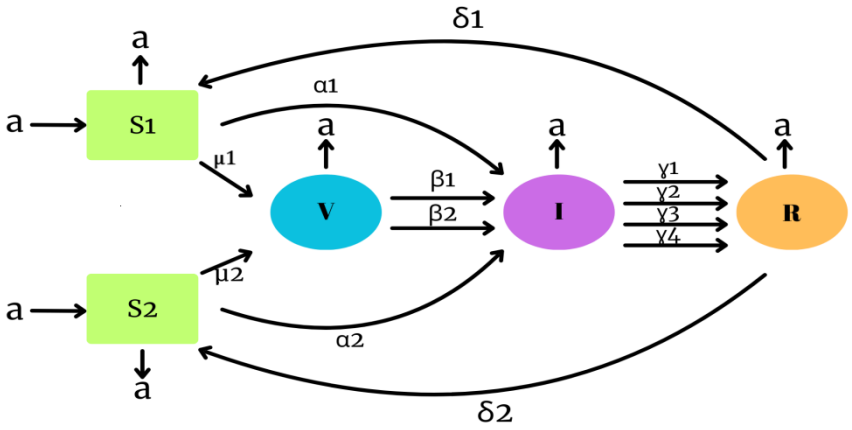

Figure 1. Mathematical Diagram of SSVIR Model.

The mathematical model examining the effect of the COVID-19 vaccine on heart attacks using a diagram has been developed as follows:

$$\frac{dS_1}{dt} = a - aS_1 - \mu_1 S_1 - \alpha_1 S_1 + \delta_1 R$$

$$\frac{dS_2}{dt} = a - aS_2 - \mu_2 S_2 - \alpha_2 S_2 + \delta_2 R$$

$$\frac{dV}{dt} = \mu_1 S_1 + \mu_2 S_2 - \beta VI - aV \qquad (4)$$

$$\frac{dI}{dt} = \alpha(S_1 + S_2)I + \beta VI - \gamma I - aI$$

$$\frac{dR}{dt} = \gamma I - aR - \delta R$$

The parameters used in the model are as follows: a is the renewal and death rate, μ_1 is the vaccination rate of sensitive individuals who have experienced both heart attacks and COVID-19, μ_2 is the vaccination rate of sensitive individuals who have experienced COVID-19 but not a heart attack, α_1 is the rate at which unvaccinated individuals who have experienced heart attacks and COVID-19 experience another heart attack, α_2 is the rate at which unvaccinated individuals who have experienced COVID-19 but not a heart attack experience a

heart attack, β_1 is the rate at which vaccinated individuals who have experienced heart attacks and COVID-19 experience another heart attack, β_2 is the rate at which vaccinated individuals who have experienced COVID-19 but not a heart attack experience their first heart attack, γ_1 is the recovery rate when unvaccinated individuals who have experienced heart attacks and COVID-19 experience another heart attack, γ_2 is the recovery rate when vaccinated individuals who have experienced heart attacks and COVID-19 experience another heart attack, γ_3 is the recovery rate when unvaccinated individuals who have experienced COVID-19 but not a heart attack experience their first heart attack, γ_4 is the recovery rate when vaccinated individuals who have experienced COVID-19 but not a heart attack experience their first heart attack, δ_1 is the rate at which vaccinated individuals who have experienced heart attacks and COVID-19 transition back to the population after recovery, and δ_2 is the rate at which vaccinated individuals who have experienced COVID-19 but not a heart attack transition back to the population after recovery.

While finding the equilibrium points in the SSVIR model, the equations are set to zero according to Definition 2.2.

$$\begin{aligned}
\frac{dS_1}{dt} &= a - aS_1 - \mu_1 S_1 - \alpha_1 S_1 + \delta_1 R = 0 \\
\frac{dS_2}{dt} &= a - aS_2 - \mu_2 S_2 - \alpha_2 S_2 + \delta_2 R = 0 \\
\frac{dV}{dt} &= \mu_1 S_1 + \mu_2 S_2 - \beta V I - aV = 0 \\
\frac{dI}{dt} &= \alpha(S_1 + S_2) I + \beta V I - \gamma I - aI = 0 \\
\frac{dR}{dt} &= \gamma I - aR - \delta R = 0
\end{aligned} \tag{5}$$

Accordingly, it is necessary to solve the expression in Equation (5). At the disease-free equilibrium point, there should be no infected individuals, meaning $\mathrm{I} = 0$ is assumed. Accordingly, the equilibrium point E_0 is obtained as follows:

$$E_0 = \left(\frac{a}{a+\mu_1}, \frac{a}{a+\mu_2}, \frac{a+2\mu_1\mu_2}{a^2 + a(\mu_1+\mu_2) + \mu_1\mu_2}, 0, 0\right) \tag{6}$$

Remark 3.1: For the endemic equilibrium point E_1, the equations are quite complex; therefore, the analysis of this point will be performed in later sections by substituting specific parameter values.

In this part, the basic reproduction number for the model system developed is calculated. For this calculation, the system has been revised in the following form:

$$\begin{aligned}\frac{dS}{dt} &= a - aS - \mu S - \alpha SI + \delta R \\ \frac{dV}{dt} &= \mu S + \beta VI - aV \\ \frac{dI}{dt} &= \alpha SI + \beta VI - \gamma I - aI \\ \frac{dR}{dt} &= \gamma I - aR - \delta R\end{aligned} \tag{7}$$

where $S = S_1 + S_2,\ \alpha = \alpha_1 + \alpha_2, \mu = \mu_1 + \mu_2,\ \beta = \beta_1 + \beta_2,\ \gamma = \gamma_1 + \gamma_2 + \gamma_3 + \gamma_4$, and $\delta = \delta_1 + \delta_2$. According to the revised system with given Equation 7, the Jacobian matrix, and the F and V matrices are obtained as follows:

$$\mathrm{J}(\mathrm{R}_0) = \begin{bmatrix} -a-\mu-\alpha I & 0 & -\alpha S & \delta \\ \mu & -\beta I - a & -\beta V & 0 \\ \alpha I & \beta I & \alpha S + \beta V - \gamma - a & 0 \\ 0 & 0 & \gamma & -a-\gamma \end{bmatrix},$$

$$\mathrm{F} = \begin{bmatrix} 0 & 0 & 0 & 0 \\ 0 & 0 & -\dfrac{\beta\mu}{a+\mu} & 0 \\ 0 & 0 & \dfrac{\beta\mu}{a+\mu} & 0 \\ 0 & 0 & 0 & 0 \end{bmatrix}, \quad V = \begin{bmatrix} a+\mu & 0 & \dfrac{\alpha a}{a+\mu} & -\delta \\ -\mu & a & 0 & 0 \\ 0 & 0 & -\dfrac{\alpha a}{a+\mu} + \gamma + a & 0 \\ 0 & 0 & -\gamma & a+\delta \end{bmatrix}.$$

The basic reproduction number is derived using the equation $\mathrm{R}_0 = \mathrm{F}\,.V^{-1}$ as follows:

$$R_0 = \frac{\beta\mu}{a^2 + \alpha a + \gamma a + a\mu + \gamma\mu}.$$

Theorem 3.2: The disease-free equilibrium point E_0 is asymptotically stable if $R_0 < 1$.

Proof: If the values of the disease-free equilibrium point are substituted into the Jacobian matrix of the SSVIR model:

$$\mathrm{J}(\mathrm{S}_1, \mathrm{S}_2, \mathrm{V}, \mathrm{I}, \mathrm{R}) = \begin{bmatrix} -a-\mu_1-\alpha_1 I & 0 & 0 & -\alpha_1 S_1 & \delta_1 \\ 0 & -a-\mu_2-\alpha_2 I & 0 & -\alpha_2 S_2 & \delta_2 \\ \mu_1 & \mu_2 & -\beta I - a & -\beta V & 0 \\ \alpha I & \alpha I & \beta I & a(S_1+S_2) + \beta I - \gamma - a & 0 \\ 0 & 0 & 0 & \gamma & -a-\delta \end{bmatrix}$$

From here, the eigenvalues are determined using the characteristic function as follows:

$$\lambda_1 = -a, \quad \lambda_2 = -a - \mu, \quad \lambda_3 = -a - \mu_2, \quad \lambda_4 = -a - \delta,$$

$$\lambda_5 = \frac{a^3 - 2a^2\alpha + a^2\gamma + \mu I a^2 + a^2\mu_2 - a\alpha\mu_2 + a\beta\mu_1 - a\beta\mu_2 + a\gamma\mu_1 + a\gamma\mu_2 + a\mu_1\mu_2 - 2\beta\mu_1\mu_2 + \gamma\mu_1\mu_2}{a^2 + a\mu_1 + a\mu_2 + \mu_1\mu_2}$$

As can be seen from the eigenvalues, the first four eigenvalues have a negative real part. To ensure that the fifth eigenvalue has a negative real part, necessary simplifications reveal that $R_0 < 1$ is required.

4 NUMERICAL ANALYSIS FOR SSVIR MODEL

In this section, the stability analysis of the equilibrium points of the newly defined SSVIR mathematical model has been examined. The numerical solution of the system was carried out using the Runge-Kutta method, and graphs were provided to show the convergence to the equilibrium points. The parameters used in the stability analysis and numerical solution are provided in two different groups in Table 1.

Table 1. Parameter values for numerical and stability analysis.

Parameter Values Conditions 1	Parameter Values Conditions 2
a = 0.02	a = 0.002
μ_1=0.05	μ_1= 0.25
μ_2 =0.06	μ_2 = 0.26
μ=0.11	μ= 0.51
α_1=0.13	α_1= 0.13
α_2 =0.15	α_2 = 0.15
α = 0.28	α = 0.28
β = 0.18	β = 0.3
γ = 0.7	γ = 0.27
δ_1 = 0.8	δ_1 = 0.002
δ_2 = 0.13	δ_2 = 0.003

When the parameter values from Condition 1 in Table 1 are substituted, the value of R_0 is found to be 0.2249999999. Using these parameters, the equilibrium points are calculated as follows:

$$E_0 = (0.2857142858, 0.2500000000, 1.464285715, 0, 0),$$

$$E_1 = (0.7112619653, 0.3056088098, \ 2.418201016, 0.01271745789, 0.03870530663)$$

When the obtained disease-free equilibrium point E_0 is substituted into the Jacobian matrix and the necessary operations are performed, the characteristic function of the system is found as follows:

$$P(\lambda) = (0.07 + \lambda)(0.08 + \lambda)(0.02 + \lambda)(0.3064 + \lambda)(0.23 + \lambda)$$

When the roots of the characteristic equation are calculated, the following results are obtained:

$$\lambda_1 = -0.3064, \lambda_2 = -0.23, \lambda_3 = -0.07, \lambda_4 = -0.08, \lambda_5 = -0.02$$

By analyzing all the eigenvalues, it is observed that they all have a negative real part, indicating that the E_0 equilibrium point is asymptotically stable.

If the same operations are performed using the Jacobian matrix for the E_1 equilibrium point, the characteristic equation is:

$$P(\lambda) = -8.457207478\,10^{-6} - 0.0002262074224\,\lambda + 0.0005117418046\,\lambda^2$$

$$+0.05122609544\,\lambda^3 + 0.4058500307\,\lambda^4 + \lambda^5$$

By solving characteristic equation, the eigenvalue is found in the following form:

$$\lambda_1 = 0.06351087630, \lambda_2 = -0.1689745623 + 0.06308289406I,$$
$$\lambda_3 = -0.05073935353, \lambda_4 = -0.08067242892, \lambda_5 = -0.1689745623 - 0.06308289406I$$

Since λ_1 has a positive real part, the E_1 equilibrium point is unstable under these conditions.

To support the stability results, the numerical solution of the system was carried out using the Runge-Kutta method. The Maple software package was used for the solution. As a result of the analysis, the solutions shown in Figure 2 were obtained. As seen in Figure 2, considering the parameter values used in the system of equations, the numerical results indicate that the system converges to the E_0 equilibrium point. In other words, under these conditions, the equation becomes disease-free, and the mortality rate of the individuals in the system will approach zero. As demonstrated in Figure 2, an initial positive correlation is observed between the increase in vaccination and the number of individuals experiencing either a heart attack or not. However, as the vaccination rate rises and the number of infected individuals decreases, this increase stabilises, reaching a balanced state. The findings emphasise the critical importance of effective measures to control the spread of the virus and the implementation of appropriate health policies to improve public health. Furthermore, the observed correlation

between the occurrence of heart attacks and the presence of the virus serves to highlight the significant role of vaccination and preventive measures in public health, as the system reaches a disease-free state.

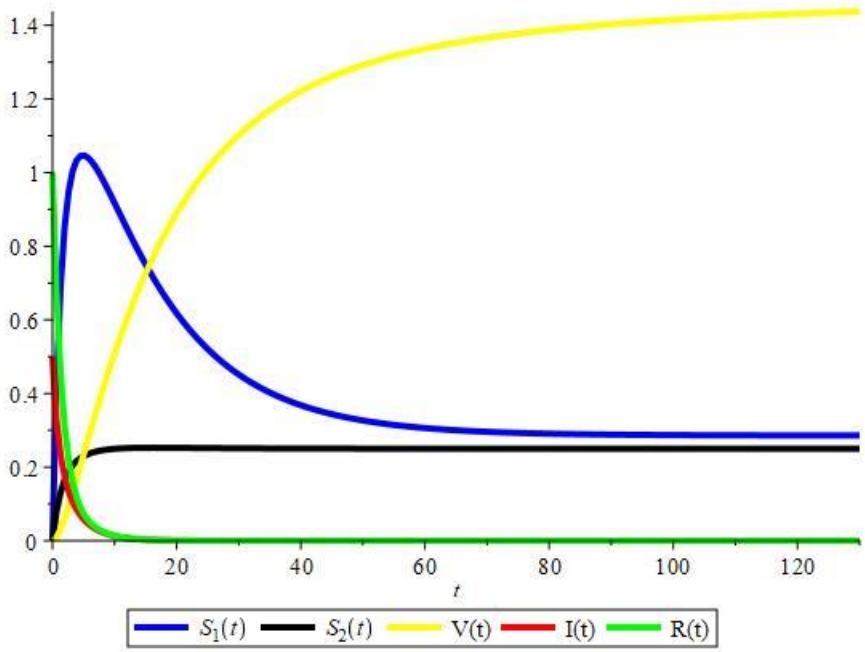

Figure 2. Numerical Simulation of the Equilibrium Point E_0.

If similar operations are performed using the values from Condition 2 in Table 1, R_0=1.103068405 is obtained. According to Theorem 3.2, the disease-free equilibrium point is unstable under these conditions. On the other hand, the endemic equilibrium point must become stable. To conduct this analysis, when the equilibrium points are calculated, they are obtained as follows:

$$E_0 = (0.007936507937, 0.007633587786, 1.984429904, 0, 0),$$

$$E_1 = (0.01670822445, 0.02031273199, 0.8721137740, 0.02948445150, 1.137257415).$$

First, if the characteristic equation is calculated using the Jacobian matrix for E_0, it will be obtained as follows:

$$P(\lambda) = -3.028943679\ 10^{-7} - 0.0001961515191\ \lambda - 0.02255437509\ \lambda^2$$

$$-0.100717136\ \lambda^3 + 0.1953114020\ \lambda^4 + \lambda^5$$

When the eigenvalues of the equation are calculated, the following results are obtained:

$$\lambda_1 = -0.262,\ \lambda_2 = -0.252,\ \lambda_3 = -0.007,\ \lambda_4 = -0.002,\ \lambda_5 = 0.3276885980$$

Since the fifth eigenvalue does not have a negative real part, the equilibrium point is unstable. This result is consistent with Theorem 3.2.

Subsequently, if the characteristic equation for E_1 is found using the Jacobian matrix, it will be obtained as follows:

$$P(\lambda) = 3.003752875\ 10^{-7} + 0.0001686549892\ \lambda + 0.002493453426\ \lambda^2 + 0.07991277695\ \lambda^3 + 0.5401009819\ \lambda^4 + \lambda^5$$

By solving this equation, the eigenvalues are found as follows:

$$\lambda_1 = -0.007994303602 + 0.04844835615\ I,\ \lambda_2 = -0.001827525481,$$
$$\lambda_3 = -0.2558455535,\ \lambda_4 = -0.2664392957,\ \lambda_5 = -0.007994303602 - 0.04844835615\ I$$

It is clear that all the eigenvalues have a negative real part. Therefore, the equilibrium point E_1 is stable.

Numerical simulations for Condition 2 values were performed using the Runge-Kutta method, and the results are presented in Figure 3. In the graphs, the selection of parameter values and stability analysis show convergence towards the equilibrium point E_1, as expected. In this case, where convergence occurs to the endemic equilibrium point, the risk of death from the disease continues. As demonstrated in Figure 3, it is evident that the prolongation of the vaccination process, in conjunction with the high rate of infection, exerts an augmented effect on individuals who have experienced a heart attack, as well as those who have not. The effect manifests as a fluctuating increase in the rate of change until the diseased equilibrium point is attained. Conversely, as the number of recovered individuals increases and the vaccination reaches a stable state, the effect undergoes a decline. Moreover, when two different sets of parameter values are considered, it is observed that the convergence to both disease-free and endemic equilibrium points does not significantly affect the system. This suggests that the dynamic behavior of the system is consistent, and accurate results will be obtained when using real-world data. As a result of the stability analysis and numerical simulations, the effects of the COVID-19 vaccine on heart disease can be further explored by dividing the population into two sensitive groups, allowing for more detailed results to be obtained.

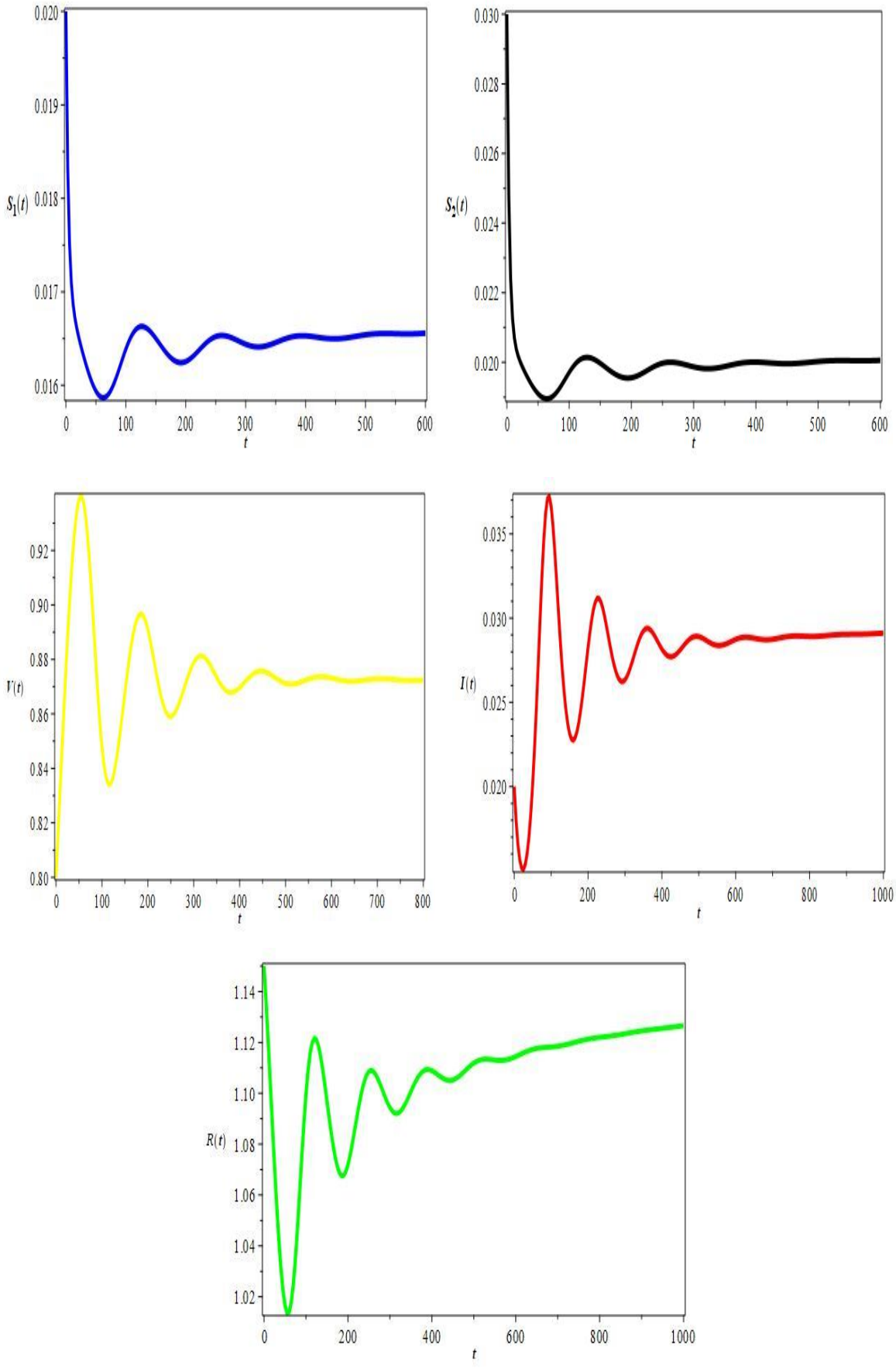

Figure 3. Numerical Simulation of the Equilibrium point E_1 for all Population Groups.

5 CONCLUSION

In this study, one of the side effects of the COVID-19 vaccine has been examined to understand its impact on public health dynamics. The effect of the vaccine on heart attacks has been investigated. For this purpose, a new mathematical model system SSVIR has been developed.

Unlike sources in the literature, susceptible individuals are divided into two groups. The first group consists of individuals who have had COVID-19 and a prior history of heart attacks, while the second group includes those who have had COVID-19 but no prior history of heart attacks. Thus, the model analysis aims to predict individuals who have had COVID-19 and heart attacks with distinct interpretations.

To determine the behaviours of the SSVIR mathematical model system, equilibrium points were first identified. Then, the analyses of these equilibrium points were supported with parameter values. In interpreting the stability of equilibrium points, the basic reproduction number was calculated, and the interpretation was proven based on this number. Using the Runge-Kutta method, numerical solutions were performed, and graphs were drawn under different parameter values. These graphs also supported the stability of the equilibrium points. Accordingly, it has been shown that the system's character works correctly based on real-life data. This study contributes to the literature in this field. In future studies, the work will be further developed using updated data.

Conflict of Interest Statement

There is no conflict of interest between the authors.

Statement of Research and Publication Ethics

The study is complied with research and publication ethics.

Artificial Intelligence (AI) Contribution Statement

This manuscript was entirely written, edited, analyzed, and prepared without the assistance of any artificial intelligence (AI) tools. All content, including text, data analysis, and figures, was solely generated by the authors.

Contributions of the Authors

M. Kocabıyık was responsible for the methodology, data analysis, writing, and supervision. Z. B. Akyol contributed to the research design, editing, and writing.

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