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MATHEMATICAL MODEL ON COVID-19 TRANSMISSION WITH ABSENCE AND PRESENCE OF SECOND DOSE OF VACCINATION IN TURKEY

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Abstract. In this paper, we have explored COVID-19 transmission dynamics with vaccination drive in Turkey. The present study elaborates the impact of vaccination drives to prevent infection transmission among Turkish people. The proposed model is equipped with seven compartments which is mainly focused on the impacts of vaccination drives. The two years real cumulative data of infected Turkish individuals, from 1 January, 2022 to 31 December, 2023, is taken for best data fit with the model which emphasizes the accuracy of our model. The numerical solution provides the better insights which is in the favour of our model compartments. We have estimated the parameters involved in model formulation. The impact of effective transmission rate, both first and second dose of vaccination rates on individuals have been analysed. A comparative study of presence and absence of double dose of vaccination drive emphasizes the importance of vaccination drive to cure the individuals. The observed results demonstrate that the model is suitable for prediction of COVID-19 infection, and emphasize how the second dose of vaccination can be an unavoidable tool to reduce the infection among the individuals. Further, residual, sensitivity analysis, disease free equilibrium, local stability, basic reproduction number, positivity, and boundedness of the model are analysed. These pivotal information leads us to unravel the complexity of the disease. Our model indicates that both vaccination drives are essential to reduce the disease transmission among the individuals and provide a great support to immune system in the combat of current infection. The reduction in observed basic reproduction number dictates the removal of infection in near future.

Keywords: Mathematical model, COVID-19, Basic reproduction number, Stability, Sensitivity

AMS Subject Classification: 34A34, 49K40, 93A30, 92B05.

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1. Introduction

Vaccines are essential for halting the virus's transmission against COVID-19. They have the ability to decrease the adverse effects of infection on public health, and eventually putting an end to the pandemic. Although, vaccination drives have been widely accepted across the globe but do not provide complete protection against the virus due to its mutation. As the virus continues to evolve and produce new variants, more contagious strains are very dangerous and awful. It is very hard to trace the infection and for timely treatment of the individuals to reduce the casualties. The newer mutated variants, include beta, delta, and omicron, are extremely contagious. Currently, sub-variants like BA.2, stealth omicron, BA.2.75, BA.2.86 and newly reported JN.1 are emerged worldwide and have the ability to survive among the individuals. To ensure the best possible defense against the virus, the immune system must be primed and can be boosted with booster doses of the COVID-19 vaccine. It improves effectiveness, offers more resilient immunity, and protects more effectively against newer variants of the virus.

Certain models documented in scientific literature depict real-world occurrences and forecast the severity of infectious diseases by employing mathematical principles [1–3]. Some simple models have been modified which reflect the spread of infection among the individuals, and vaccination has been introduced to control the infection [1, 4–14]. The existing COVID-19 models have been modified by including vaccination techniques, allowing for accurate projections of infection spread and the effectiveness of immunization efforts. These enhanced models offer important new information on how well immunization campaigns contribute to controlling the epidemic and improving public health efforts [15–30].

An epidemiological SEIHR model, with and without instinctive vaccination drive is reported in the literature and the obtained results are in the support of reduction of infection [15]. For Indian territory, an extended mathematical model with ten compartments is developed and put some light on spreading the infection. To maintain the economic burden on government and people, some study have enlightened the science community by opting cost-effectiveness and the optimal intervention strategies [16, 17, 28]. Further, A mathematical model is focused on first wave of infection in Senegal from March 29, 2020, to April 29, 2020, where the data fitting with the model has enhanced its credibility [18]. Several studies have focused on vaccine supply, vaccination impact on infection and vaccine optimal control strategy to mitigate the infection among the individuals [19–21, 25, 26].

The influence of non-pharmaceutical interventions (NPIs) with vaccination drives on infected individuals are reported in the literature [22, 29]. Some computational models with vaccine efficacy are developed to understand the situation of COVID-19 infection in England, Africa, and New York. These models emphasize the vaccination efforts to reduce the burden of COVID-19 broadly [23, 24, 27].

Further, in view of existing models, a modified measles compartmental model with double dose of vaccination is developed. The vaccination drive provide the fruitful result in Bangladesh as mild and critical cases of infection have been decreased after vaccination [8, 12]. It has been observed that adopting double doses of vaccination programme reduces infection among the population globally. Additionally, implementing additional preventive measures and control tactics can be crucial in mitigating the present epidemic [31–43].

The second dose of vaccination drives is one of the pivotal tactics and it is adopted across the globe [31–37]. The implication of double dose has ample outcomes and can be seen in the form of reduction in infection among the individuals. The increase in vaccination drives leads to optimizes the disease burden. The incorporation of other interventions like

quarantine, media campaign etc along with double dose of vaccination are an indispensable part to mitigate the infection [38, 39].

A comparative study between adopting vaccination and no vaccination drives is reported in the literature. The developed model is fitted using Ethiopian data on vaccinated individuals and cumulative daily infected cases [40]. In view of reduction in infection, several studies have focused on optimal control strategies and cost-effectiveness analysis [41, 42]. Further, a case study to rollout of double dose of vaccination drives emphasize the vaccination strategy against COVID-19 imposed in Italy [43]. However, the idea of a booster dose is frequently adopted globally in order to achieve herd immunity. It is revealed that the booster dose helps to improve vaccination efficacy, and slow down the infection rate in individuals [44–52, 54**?** –56].

As of March 17, 2024, the total number of COVID-19 cases reported to the WHO is 17 million and received reports of 101,400 COVID-19 deaths. Further, 69% of the total population has received at least one dose of the COVID-19 vaccine while 63% and 33% of the total population have received the whole primary series and at least one booster dose of the COVID-19 vaccination on 26 November 2023 for Turkey, respectively [57].

In this study, We separated the paper into six sections. The section (2)is bifurcated into five subsections as model formulation, non-negativity, boundedness, disease free equilibrium points and basic reproduction number (R_0), and local stability, respectively. The seven compartmental model $SEIQRV_1V_2$ is formulated where the impact of vaccination drives is focused mainly. For all positive solutions, the non-negativity of the model emphasises that all of the model's variables and parameters are positive for any $t > 0$. The boundedness of the model depicts that the solutions are bounded with respect to non-negative. The DFE points indicates the removal of infection among the individuals while the future of the disease can be predicted with the help of BRN. If the value of R0 is lesser or greater than one then the infection will disappear or remain among the individuals, respectively. Further, in section (3), parameter estimation using MATLAB ode45 package and model fitting by least square curve fitting method are discussed. The sensitivity analysis of each parameter of the model with respect to R_0 elaborates in section (4). The section (5) covers numerical simulations of the proposed model and provide better insights. Finally, section (6) includes discussion and conclusion in the form of outcomes of our proposed study.

2. Mathematical Analysis of the COVID-19 Model

In this section, we have discussed the model with schematic diagram, model formulation, boundedness, non-negativity, epidemic equilibrium, and basic reproduction number of the COVID-19 model (1).

2.1. **Model Formulation.**

The existing several studies are revealed that the infection spread due to contact of human-to-human and their droplets after sneezing [58]. Therefore, it is evident from the literature that the interventions like social distancing, quarantined, vaccination drive etc., can help to reduce the risk of current pandemic and mitigate COVID-19 infection in near future [59]. The vaccination drives is considered the most effective intervention to reduce the infection. To understand the invincible importance of vaccine, we have separated the entire population into seven compartments: susceptible (S), exposed (E), infected (I), isolated or quarantined (Q), recovered (R), the number of first dose vaccinated individuals (V_1), and the number of double dose vaccinated individuals (V_2). $N(t)$ represents the entire population at time t and can be demonstrated as $N(t) = S(t)+E(t)+I(t)+Q(t)+R(t)+V_1(t)+V_2(t)$. In this study, the parameters $\Lambda, \delta, d, \mu, \alpha, \gamma, \beta_I, \beta_Q, \eta_1, \eta_2, \zeta_1, \zeta_2, \tau$ and λ have

been taken into account, and nomenclature is manifested in the Table (1). Further, the Figure (1) demonstrates the dynamics of the model.

2.1.1. *Biological Assumptions of the Model.*

The present study is analysed after having attention towards some biological assumptions. These assumptions have the liberty to asses the model and relevant to the symptoms of spreading the virus infection as follows:

(1) We assume that a susceptible person can go to an exposed class even after taking double doses of vaccination.
(2) It is evident from the literature that the vaccination do not provide total protection. In this situation, we suppose that the vaccinated persons get infected when exposed to the virus.

The model provides a transition from all individuals with birth rate Λ enter into susceptible class, firstly. After receiving the first dose of the COVID-19 vaccine at a rate of η_1, susceptible individuals migrate to the vaccinated class. Because the first dose of the vaccine is insufficient to protect against COVID-19, the vaccinated population goes to the susceptible compartment at a rates of τ. The remaining population moves to the second dose of the vaccinated class at a rate of η_2. It is assumed that individuals in the population who received the second dose of the vaccination shift to the recovered class at a rate of λ. The force of infection among individuals is $\frac{\delta(\beta_I I+\beta_Q Q)}{N}$, where δ is the effective transmission rate, β_I is the reduction rate in disease transmission for infected individuals, and β_Q is the reduction rate in disease transmission for quarantined individuals. First dose and double dose (both first and second) of vaccinated population move to exposed class with the force of infection $\frac{\zeta_1(\beta_I I+\beta_Q Q)}{N}$ and $\frac{\zeta_2(\beta_I I+\beta_Q Q)}{N}$, respectively. The change in compartments from exposed to infected, the individuals are moving with the disease rate μ. The exit rate from infected to quarantine class and quarantine to recovered class are α and γ, respectively. d is considered as the natural death rate in all compartments.

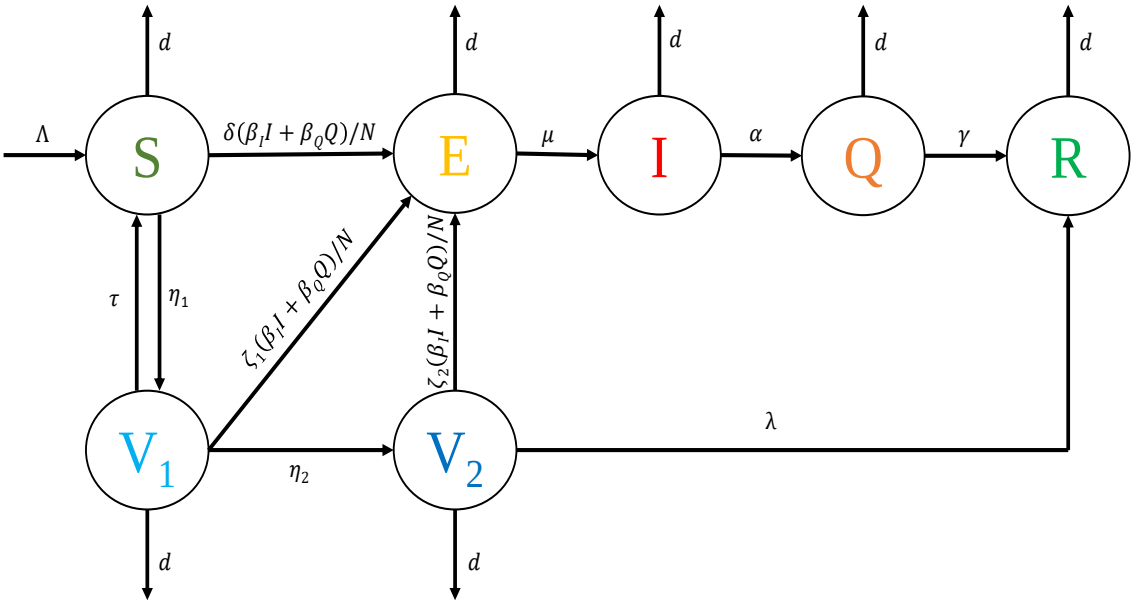

FIGURE 1. Graphical representation of the model system (1).

Thus, the following equations demonstrate the model dynamics as:

$$\begin{aligned}
\frac{dS}{dt} &= \Lambda - \frac{\delta S(\beta_I I + \beta_Q Q)}{N} - (\eta_1 + d)S + \tau V_1, \\
\frac{dE}{dt} &= \frac{(\delta S + \zeta_1 V_1 + \zeta_2 V_2)(\beta_I I + \beta_Q Q)}{N} - (\mu + d)E, \\
\frac{dI}{dt} &= \mu E - (d + \alpha)I, \\
\frac{dQ}{dt} &= \alpha I - (\gamma + d)Q, \\
\frac{dR}{dt} &= \gamma Q - dR + \lambda V_2, \\
\frac{dV_1}{dt} &= \eta_1 S - \frac{\zeta_1 V_1(\beta_I I + \beta_Q Q)}{N} - (\eta_2 + d + \tau)V_1, \\
\frac{dV_2}{dt} &= \eta_2 V_1 - \frac{\zeta_2 V_2(\beta_I I + \beta_Q Q)}{N} - dV_2 - \lambda V_2.
\end{aligned} \tag{1}$$

with the initial conditions $S(0) > 0, E(0) \geq 0, I(0) \geq 0, Q \geq 0, R \geq 0, V_1(0) \geq 0$ and $V_2(0) \geq 0$.

2.2. **Non-Negativity of the Model.**

The developed model system (1) emphasizes the population of living entities i.e, humans. The epidemiological feasibility of our proposed model can be held if the model has all non-negative solutions or has all positive solutions. So, it can be expected that all of the model's variables and parameters are positive for any $t > 0$. Thus, the following theorem demonstrates the non negative solutions with non negative initial condition.

Theorem 2.1. *The dynamic model represented in (1) with initial conditions given by* $S(0) > 0, E(0) \geq 0, I(0) \geq 0, Q \geq 0, R \geq 0, V_1(0) \geq 0$ *and* $V_2(0) \geq 0$ *has positive solutions* $\big(S(t), E(t), I(t), Q(t), R(t), V_1(t), V_2(t)\big)$, $\forall t > 0$.

Proof. In particular, when $S(0) > 0$, an attempt is taken to demonstrate the non-negativity of the susceptible class $S(t)$, $\forall t > 0$.

From first equation of the model system (1), we have,

$$\frac{dS}{dt} = \Lambda - \frac{\delta S(\beta_I I + \beta_Q Q)}{N} - (\eta_1 + d)S + \tau V_1, \tag{2}$$

$$\frac{dS}{dt} \geq -dS. \tag{3}$$

On integrating equation (3) with respect to t, we get

$$S(t) \geq c_1 e^{-dt}. \tag{4}$$

Using initial condition $S(0) = S_0$ in above equation (4), we get,

$$S(t) \geq S_0 e^{-dt} \geq 0, \tag{5}$$

where, $S_0 = e^{c_1}$.

Thus, the positivity of other components can be established similarly. This completes the proof. □

2.3. **Boundedness of the Model.**
The boundedness of the model is one of the key aspect to predict the epidemic accurately. So, in order to accurate prediction of the epidemic, it is necessary to show that the solutions of the proposed model is bounded. The following theorem dictates the boundedness of the model efficiently.

Theorem 2.2. *All solutions of the proposed model with non-negative initial conditions are bounded and* $N(t) \leq \frac{\Lambda}{d}$, $\forall t > 0$.

Proof. From the model system (1), we have

$$\frac{dN}{dt} = \frac{dS}{dt} + \frac{dE}{dt} + \frac{dI}{dt} + \frac{dQ}{dt} + \frac{dR}{dt} + \frac{dV_1}{dt} + \frac{dV_2}{dt}. \tag{6}$$

From the above equation (6), we have

$$\frac{dN}{dt} = \Lambda - d(S + E + I + Q + R + V_1 + V_2), \tag{7}$$

which gives us,

$$\frac{dN}{dt} = \Lambda - dN. \tag{8}$$

Integrating equation (8), we obtain

$$N(t) = \frac{\Lambda}{d} + \left(N_0 - \frac{\Lambda}{d}\right)e^{-dt}. \tag{9}$$

Thus, when $t \to \infty$, the above equation can be written as

$$N(t) \leq \frac{\Lambda}{d}. \tag{10}$$

Thus, it can be seen that $S(t)$, $E(t)$, $I(t)$, $Q(t)$, $R(t)$, $V_1(t)$, and $V_2(t)$ are bounded and hence the Theorem is proved. □

2.4. **Disease Free Equilibrium (DFE) Point and Basic Reproduction Number (R_0) of the Model.**
Equating the right-hand side of equation (1) to zero in order to compute the disease free equilibrium (DFE). Let, the disease state variables $E = I = Q = 0$, and disease free state variables S, R, V_1 and V_2 are non zero. Therefore, the system (1) indicates the disease free equilibrium point which can be demonstrated as $DFE = (S^0, E^0, I^0, Q^0, R^0, V_1^0, V_2^0)$, where
$E^0, I^0, Q^0 = 0$, and

$$\begin{aligned}
S^0 &= \left(\frac{\Lambda\ (d + \eta_2 + \tau)}{d\,\eta_1 + d\,\eta_2 + \eta_1\,\eta_2 + d\,\tau + d^2}\right., \\
R^0 &= \frac{\Lambda\,\eta_1\,\eta_2\,\lambda}{d\ (d + \lambda)\ (d\,\eta_1 + d\,\eta_2 + \eta_1\,\eta_2 + d\,\tau + d^2)}, \\
V_1^0 &= \frac{\Lambda\,\eta_1}{d\,\eta_1 + d\,\eta_2 + \eta_1\,\eta_2 + d\,\tau + d^2}, \\
V_2^0 &= \frac{\Lambda\,\eta_1\,\eta_2}{(d + \lambda)\ (d\,\eta_1 + d\,\eta_2 + \eta_1\,\eta_2 + d\,\tau + d^2)}.
\end{aligned} \tag{11}$$

Further, to understand the spreading of COVID-19 disease among individuals, basic reproduction number (R_0) is pivotal threshold parameter which predicts the future of the disease. If the value of R_0 is less than one then the infection will disappear in near future

while if the value of R_0 is greater than one then the infection will remain among the individuals and disease will not be ended. Based on this, the infection peak and size of epidemic can be demonstrated. More precisely, it can be defined as the expected average number of secondary cases of infection that will occur when a single infectious individual is introduced into a completely susceptible population. For further simulation, we have opted the next generation matrix method to find the basic reproduction number (R_0) [60–65]. Let us consider the following matrices:

$$\mathscr{F} = \begin{pmatrix} \frac{\left(I\,\beta_I + Q\,\beta_Q\right)\left(S\,\delta + V_1\,\zeta_1 + V_2\,\zeta_2\right)}{N} \\ 0 \\ 0 \end{pmatrix},$$

$$\mathscr{V} = \begin{pmatrix} E\,d + E\,\mu \\ I\,(\alpha + d) - E\,\mu \\ Q\,(d + \gamma) - I\,\alpha \end{pmatrix}.$$

The Jacobian matrix of $\mathscr{F}$ and $\mathscr{V}$ at DFE, denoted by F and V are given as follows:

$$F = \begin{pmatrix} 0 & \frac{\beta_I\left(S^0\,\delta + V_1^0\,\zeta_1 + V_2^0\,\zeta_2\right)}{N^0} & \frac{\beta_Q\left(S^0\,\delta + V_1^0\,\zeta_1 + V_2^0\,\zeta_2\right)}{N^0} \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{pmatrix},$$

$$V = \begin{pmatrix} d + \mu & 0 & 0 \\ -\mu & \alpha + d & 0 \\ 0 & -\alpha & d + \gamma \end{pmatrix}.$$

The dominant eigenvalue of FV^{-1} represents R_0, which is

$$R_0 = R_I + R_Q, \tag{12}$$

where,

$$\begin{aligned} R_I &= \frac{\beta_I\,\mu\,\left(S^0\,\delta + V_1^0\,\zeta_1 + V_2^0\,\zeta_2\right)}{N^0\,(\alpha + d)\,(d + \mu)}, \\ R_Q &= \frac{\alpha\,\beta_Q\,\mu\,\left(S^0\,\delta + V_1^0\,\zeta_1 + V_2^0\,\zeta_2\right)}{N^0\,(\alpha + d)\,(d + \gamma)\,(d + \mu)}. \end{aligned}$$

2.5. **Local stability analysis of the disease free equilibrium.**
For local stability, Theorem (2.3) demonstrates wonderful result which is in support of our proposed model. It can be seen that the disease will wipe out in near future if R_0 is smaller than unity. The observed result provides us a straightforward understanding and epidemiological consequence as the entry of small influx of newly infected individuals in the system will not affect the society until the basic reproduction number $R_0 < 1$ holds accurately. In other words, the illness will quickly disappear if the starting size of the infected people is inside the DFE basin of attraction and the basic reproduction number $R_0 < 1$ [31, 66–70].

Theorem 2.3. *The disease-free equilibrium DFE is locally stable if* $R_0 < 1$.

Proof.

$$J = \begin{pmatrix} \frac{\partial f_1}{\partial S} & \frac{\partial f_1}{\partial E} & \frac{\partial f_1}{\partial I} & \frac{\partial f_1}{\partial Q} & \frac{\partial f_1}{\partial R} & \frac{\partial f_1}{\partial V_1} & \frac{\partial f_1}{\partial V_2} \\ \frac{\partial f_2}{\partial S} & \frac{\partial f_2}{\partial E} & \frac{\partial f_2}{\partial I} & \frac{\partial f_2}{\partial Q} & \frac{\partial f_2}{\partial R} & \frac{\partial f_2}{\partial V_1} & \frac{\partial f_2}{\partial V_2} \\ \frac{\partial f_3}{\partial S} & \frac{\partial f_3}{\partial E} & \frac{\partial f_3}{\partial I} & \frac{\partial f_3}{\partial Q} & \frac{\partial f_3}{\partial R} & \frac{\partial f_3}{\partial V_1} & \frac{\partial f_3}{\partial V_2} \\ \frac{\partial f_4}{\partial S} & \frac{\partial f_4}{\partial E} & \frac{\partial f_4}{\partial I} & \frac{\partial f_4}{\partial Q} & \frac{\partial f_4}{\partial R} & \frac{\partial f_4}{\partial V_1} & \frac{\partial f_4}{\partial V_2} \\ \frac{\partial f_5}{\partial S} & \frac{\partial f_5}{\partial E} & \frac{\partial f_5}{\partial I} & \frac{\partial f_5}{\partial Q} & \frac{\partial f_5}{\partial R} & \frac{\partial f_5}{\partial V_1} & \frac{\partial f_5}{\partial V_2} \\ \frac{\partial f_6}{\partial S} & \frac{\partial f_6}{\partial E} & \frac{\partial f_6}{\partial I} & \frac{\partial f_6}{\partial Q} & \frac{\partial f_6}{\partial R} & \frac{\partial f_6}{\partial V_1} & \frac{\partial f_6}{\partial V_2} \\ \frac{\partial f_7}{\partial S} & \frac{\partial f_7}{\partial E} & \frac{\partial f_7}{\partial I} & \frac{\partial f_7}{\partial Q} & \frac{\partial f_7}{\partial R} & \frac{\partial f_7}{\partial V_1} & \frac{\partial f_7}{\partial V_2} \end{pmatrix},$$

where,

$$\begin{aligned} f_1 &= \Lambda - \frac{\delta S(\beta_I I + \beta_Q Q)}{N} - (\eta_1 + d)S + \tau V_1, \\ f_2 &= \frac{(\delta S + \zeta_1 V_1 + \zeta_2 V_2)(\beta_I I + \beta_Q Q)}{N} - (\mu + d)E, \\ f_3 &= \mu E - (d + \alpha)I, \\ f_4 &= \alpha I - (\gamma + d)Q, \\ f_5 &= \gamma Q - dR + \lambda V_2, \\ f_6 &= \eta_1 S - \frac{\zeta_1 V_1(\beta_I I + \beta_Q Q)}{N} - (\eta_2 + d + \tau)V_1, \\ f_7 &= \eta_2 V_1 - \frac{\zeta_2 V_2(\beta_I I + \beta_Q Q)}{N} - dV_2 - \lambda V_2. \end{aligned}$$

Thus, it can be written as

$$J = \begin{pmatrix} -d - \eta_1 - M_1 & 0 & -\frac{S\,\beta_I\,\delta}{N} & -\frac{S\,\beta_Q\,\delta}{N} & 0 & \tau & 0 \\ M_1 & -d - \mu & \beta_I\,M_2 & \beta_Q\,M_2 & 0 & M_3 & M_4 \\ 0 & \mu & -\alpha - d & 0 & 0 & 0 & 0 \\ 0 & 0 & \alpha & -d - \gamma & 0 & 0 & 0 \\ 0 & 0 & 0 & \gamma & -d & 0 & \lambda \\ \eta_1 & 0 & -\frac{V_1\,\beta_I\,\zeta_1}{N} & -\frac{V_1\,\beta_Q\,\zeta_1}{N} & 0 & -M_5 - M_3 & 0 \\ 0 & 0 & -\frac{V_2\,\beta_I\,\zeta_2}{N} & -\frac{V_2\,\beta_Q\,\zeta_2}{N} & 0 & \eta_2 & -d - \lambda - M_4 \end{pmatrix},$$

where,

$$\begin{aligned} M_1 &= \frac{\delta\ (I\,\beta_I + Q\,\beta_Q)}{N}, \\ M_2 &= \frac{(S\,\delta + V_1\,\zeta_1 + V_2\,\zeta_2)}{N}, \\ M_3 &= \frac{\zeta_1\ (I\,\beta_I + Q\,\beta_Q)}{N}, \\ M_4 &= \frac{\zeta_2\ (I\,\beta_I + Q\,\beta_Q)}{N}, \\ M_5 &= d + \eta_2 + \tau. \end{aligned}$$

The above mentioned Jacobian matrix at disease-free equilibrium (DFE) is

$$J(DFE) = \begin{pmatrix} -d-\eta_1 & 0 & -\frac{S^0\,\beta_I\,\delta}{N^0} & -\frac{S^0\,\beta_Q\,\delta}{N^0} & 0 & \tau & 0 \\ 0 & -d-\mu & M_{11} & M_{22} & 0 & 0 & 0 \\ 0 & \mu & -\alpha-d & 0 & 0 & 0 & 0 \\ 0 & 0 & \alpha & -d-\gamma & 0 & 0 & 0 \\ 0 & 0 & 0 & \gamma & -d & 0 & \lambda \\ \eta_1 & 0 & -\frac{V_1^0\,\beta_I\,\zeta_1}{N^0} & -\frac{V_1^0\,\beta_Q\,\zeta_1}{N^0} & 0 & -M_5 & 0 \\ 0 & 0 & -\frac{V_2^0\,\beta_I\,\zeta_2}{N^0} & -\frac{V_2^0\,\beta_Q\,\zeta_2}{N^0} & 0 & \eta_2 & -d-\lambda \end{pmatrix}, \quad (13)$$

where,

$$M_{11} = \frac{\beta_I\,\left(S^0\,\delta + V_1^0\,\zeta_1 + V_2^0\,\zeta_2\right)}{N^0},$$
$$M_{22} = \frac{\beta_Q\,\left(S^0\,\delta + V_1^0\,\zeta_1 + V_2^0\,\zeta_2\right)}{N^0}.$$

Thus, for steady states, it can be seen in (11), S^0 and N^0 are steady states, where $N^0 = S^0+R^0+V_1^0+V_2^0$. In view of disease-free equilibrium of the above Jacobi matrix (13), the following eigenvalues are observed -0.0006096, -0.007531, -9.724×10^{-5}, -0.1192, -0.07789, -0.06749 and -0.03781, which are negative. Therefore, we conclude that DFE of model system (1) is locally stable if $R_0 < 1$, and unstable otherwise. □

3. Parameter Estimation and Model Fitting

Here, we have estimated the parameters using MATLAB ode45 package to solve model system (1). The daily reported cases of COVID-19 infection in Turkey is used for parameters estimation which is given in Table (1). Particularly, the model is configured to examine the impact of vaccination control on disease burden, specifically as measured by the total number of daily reported cases in Turkey between January 01, 2022, and December 31, 2023.

With the values in Table (1), the disease-free equilibrium value is $DFE = (S^0, E^0, I^0, Q^0, R^0, V_1^0, V_2^0) = (986088.27, 0, 0, 0, 114806992.45, 800431.26, 10111815.95)$, and $R_0 = 0.84 < 1$. This shows that the infection is under control and it may be vanish in near future. The model's solution curves with varying initial conditions are shown in Figure (2), where they tend to the stability of the disease-free equilibrium point. The results of the model fitting in Figure (3) and residual in Figure (4) using the reported cumulative number of daily cases are shown below:

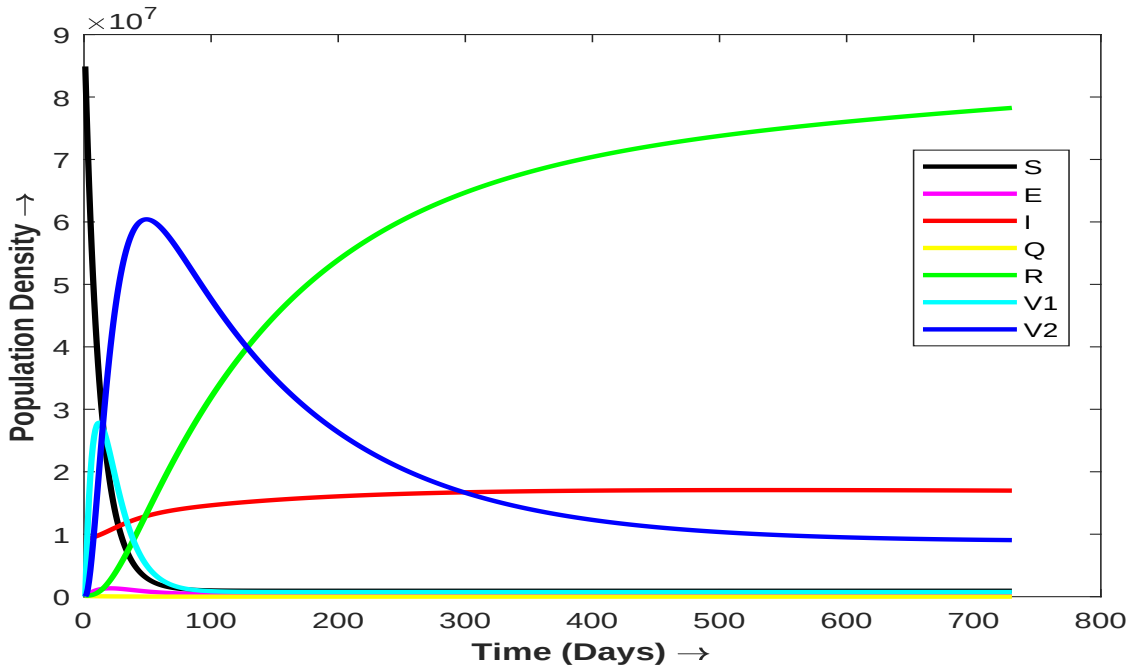

FIGURE 2. Solution curve of COVID-19 transmission dynamics for Turkey from January 1, 2022 to December 31, 2023.

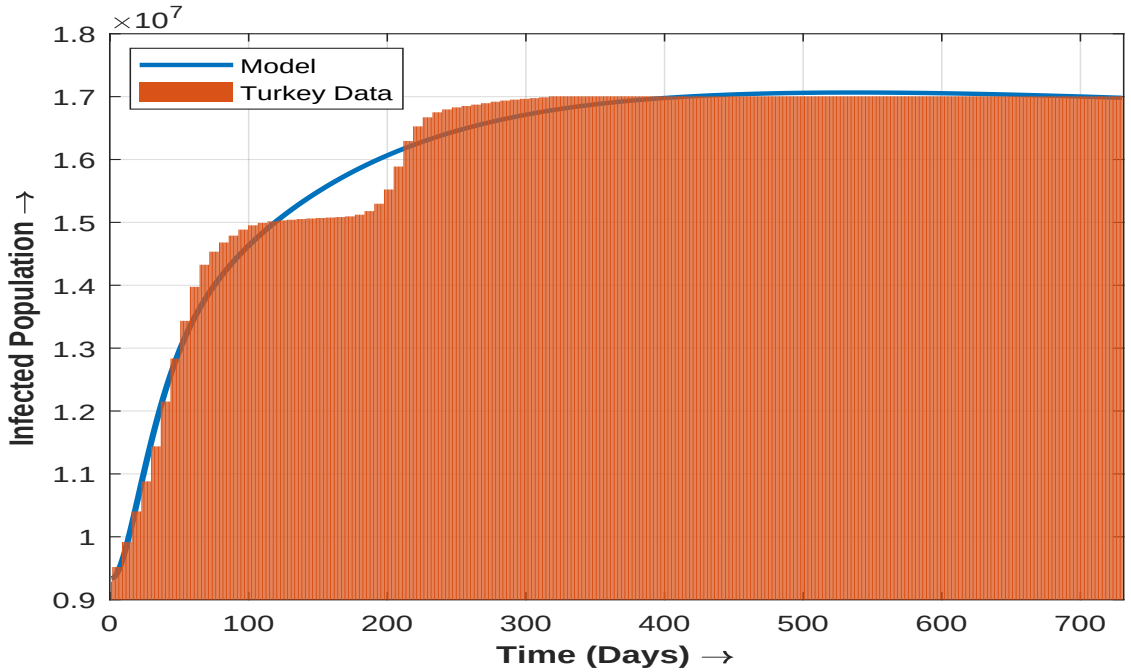

FIGURE 3. Data fitting of total COVID-19 reported cases with model.

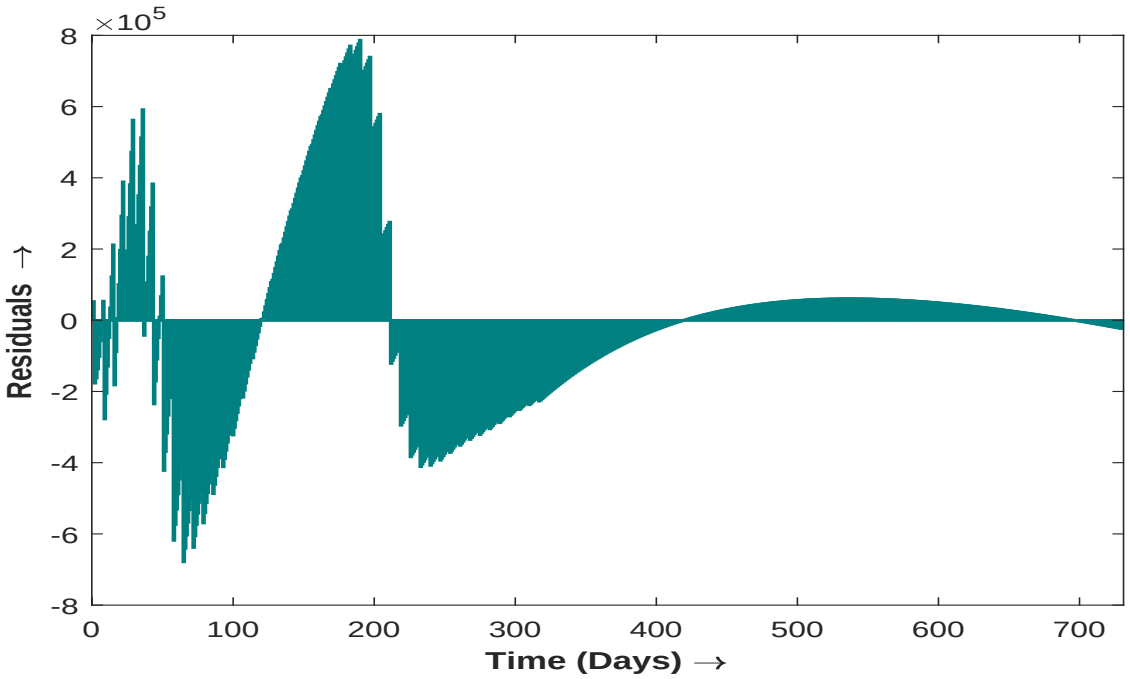

FIGURE 4. Residual of model system (1)

TABLE 1. Nomenclature of parameters used in the model system (1).

Parameters	Meaning	Value	Reference
$S(0)$	Initial Susceptible class	84893759.25	Estimated
$E(0)$	Initial Exposed class	20208.25	Estimated
$I(0)$	Initial Infected class	9340043.75	Estimated
$Q(0)$	Initial Quarantined class	95294.67	Estimated
$R(0)$	Initial Recovered class	160854.12	Estimated
$V_1(0)$	Initial first dose of Vaccinated class	33929.85	Estimated
$V_2(0)$	Initial second dose of Vaccinated class	47456.26	Estimated
Λ	Recruitment rate	77241.98	Estimated
μ	Incubation period	0.08	Estimated
α	Infection time	9.81×10^{-6}	Estimated
d	Natural death rate	6.10×10^{-4}	Estimated
γ	Recovery time	0.04	Estimated
β_I	Reduction rate in disease transmission for infected individuals	0.12	Estimated
β_Q	Reduction rate in disease transmission for quarantined individuals	0.08	Estimated
η_1	First dose of vaccine rate	0.08	Estimated
η_2	Second dose of vaccine rate	0.10	Estimated
ζ_1	Vaccine inefficacy (first dose)	0.16	Estimated
ζ_2	Vaccine inefficacy (second dose)	0.02	Estimated
δ	Effective transmission rate	0.17	Estimated
τ	Progression rate from V_1 to S	0.01	Estimated
λ	Recovery rate due to second dose of vaccination	0.01	Estimated

4. Sensitivity Analysis

The most sensitive parameters for the basic reproduction number R_0 can be measured with the use of the sensitivity index technique. The value of the parameters have been taken from the Table (1) for analysis. The normalized sensitivity index for the basic reproduction number is given by $\mathcal{S}_{\mathcal{P}}^{R_0} = \frac{\partial R_0}{\partial \mathcal{P}} \times \frac{\mathcal{P}}{R_0}$, where $\mathcal{P} = \Lambda, \delta, d, \mu, \alpha, \gamma, \beta_I, \beta_Q, \eta_1, \eta_2, \zeta_1, \zeta_2, \tau$ and λ. We obtain,

$$\begin{aligned} \mathcal{S}_{\mu}^{R_0} &= \frac{\partial R_0}{\partial \mu} \times \frac{\mu}{R_0} \\ &= \frac{d}{d+\mu} \\ &= 0.0079 \end{aligned}$$

In similar manner, the sensitivity index of each parameter for R_0 is assessed and mentioned in Table (2). It has been observed that seven out of fourteen sensitivity indices are positive, the other remaining six indices are negative while one index is non-negative. The positive and negative sensitivity indices indicate that R_0 increases and decreases, respectively, if the corresponding parameters with R_0 are in increasing manner (see Figure (5)). Therefore, the goal of intervention strategies should be to decrease and increase in parameter's value with positive and negative indices, respectively.

Table 2. The sensitivity index of R_0 with respect to parameters $\mathcal{P}$ used in the model.

Parameters	**Meaning**	**Sensitivity Index**
Λ	Recruitment rate	0.0
μ	Incubation period	+0.0079
α	Infection time	-0.0157
d	Natural death rate	-0.0420
γ	Recovery time	-0.0002
β_I	Reduction rate in disease transmission for infected individuals	+0.9998
β_Q	Reduction rate in disease transmission for quarantined individuals	+0.0002
η_1	First dose of vaccine rate	-0.2970
η_2	Second dose of vaccine rate	-0.2467
ζ_1	Vaccine inefficacy (first dose)	+0.2311
ζ_2	Vaccine inefficacy (second dose)	+0.4641
δ	Effective transmission rate	+0.3048
τ	Progression rate from V_1 to S	+0.0202
λ	Recovery rate due to second dose of vaccination	-0.4265

The Figure (5) demonstrates the plot of R_0 corresponding to different parameters manifested in Table (1). We have varied the parameters one by one for each plot that are plotted against R_0.

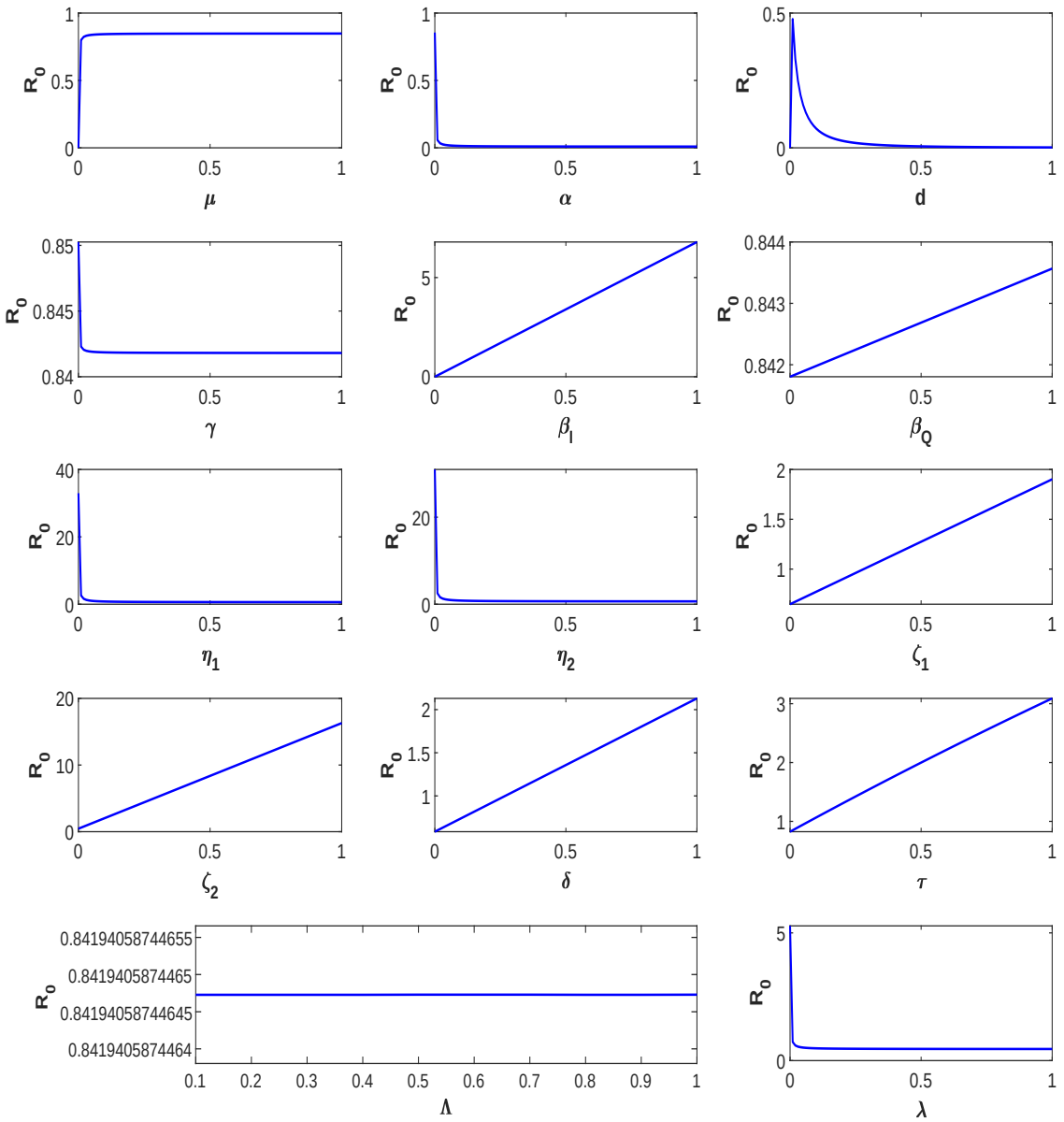

FIGURE 5. Sensitivity analysis of R_0 with respect to parameters for model system (1).

5. Numerical Simulations

5.1. The Impact of Effective Transmission Rate (δ) on Individuals.

Figure (6) displays the impact of transmission rate δ for each of the population compartments specified in model system (1). Here, the other parameters have been fixed and varied the effective transmission rate as $\delta = 0.15, 0.17, 0.19$. It can be seen that E and I population compartments have significant changes while the remaining compartments S, Q, R, V_1 and V_2 have minor changes. This means that in order to prevent the spread of the disease, we must follow some interventions strictly as wearing masks, washing our hands, and avoiding close contact with others.

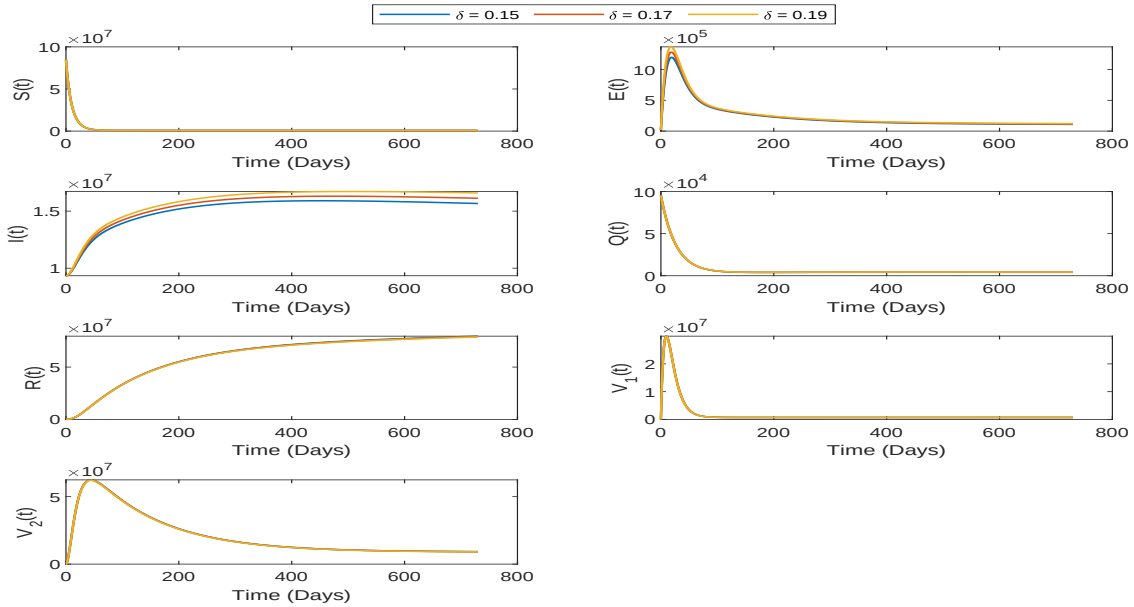

FIGURE 6. Time series of model system (1) with $0.15 \leq \delta \leq 0.19$.

5.2. **The Impact of First Dose of Vaccination Rate** (η_1) **on Individuals.** Figure (7) demonstrates the impact of first dose of vaccination η_1 on the population compartments. It can be seen that the population compartments S, E, I, V_1 and V_2 have major impact while the remaining compartments Q and R are less influenced. Further, if we increase the rate η_1 then the population decrease in S, E, I, and Q compartments while there is an increment in R, V_1 and V_2 population compartments. This indicates that the first vaccination dose significantly reduce the overall number of infected population.

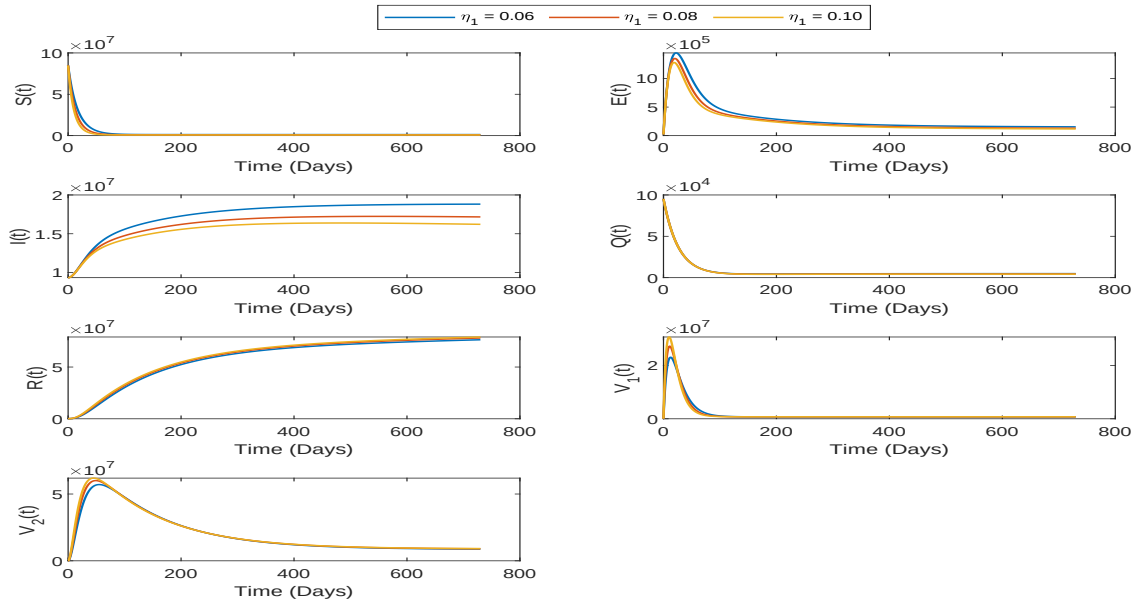

FIGURE 7. Time series of model system (1) with $0.06 \leq \eta_1 \leq 0.10$.

5.3. **The Impact of Second Dose of Vaccination Rate (η_2) on Individuals.** Figure (8) demonstrates that the effect of η_2 for each population compartments in interval $0.08 \leq \eta_2 \leq 0.12$. It is observed that on increasing in second dose of vaccination rate (η_2), there is a decrement in population compartments S, E, I, Q and V_1, while increment in the remaining population compartments R and V_2. According to a biological phenomena, we can mitigate the transmission of COVID-19 and the endemic condition when the rate of second dose vaccination rises.

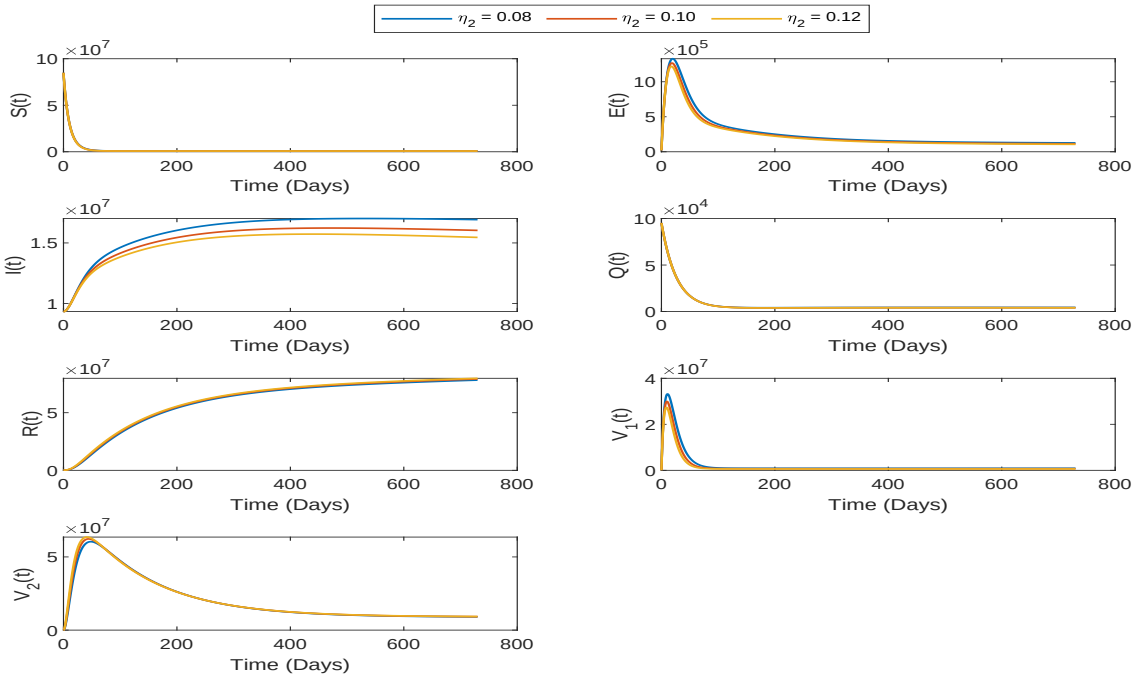

FIGURE 8. Time series of model system (1) with $0.08 \leq \eta_2 \leq 0.12$.

5.4. **The Impact of the Absence of Second Dose of Vaccination Rate ($\eta_2 = 0$) on Individuals.**
Here, Figure (9) demonstrates the effect of the absence of η_2 on population compartments. It has been observed significant impact of η_2. The presence and absence of η_2 provide the better insights to hold down the infection. The presence of second dose of vaccination emphasizes the increment and decrement in recovered individuals and infected individuals, respectively. Thus, infection can be disappeared in near future among the individuals.

6. Discussion and Conclusion

In this study, we have explored the effect of vaccination drive on population of Turkey. The proposed study demonstrates the model formulation, solution, parameters estimation, real cumulative infected data fit with model, residuals, sensitivity analysis, and numerical simulations. We have estimated all the parameters for simulation, mentioned in Table (1). The optimal solution provides the vital information of each compartment and is manifested in Figure (2). For best data fit with the model, we have taken the data for two years confirmed infected cases from January 01, 2022 to December 31, 2023 which is represented successfully in Figure (3). The residual of the model is also carried out and given in Figure (4) which emphasizes the effectiveness of the proposed model using real

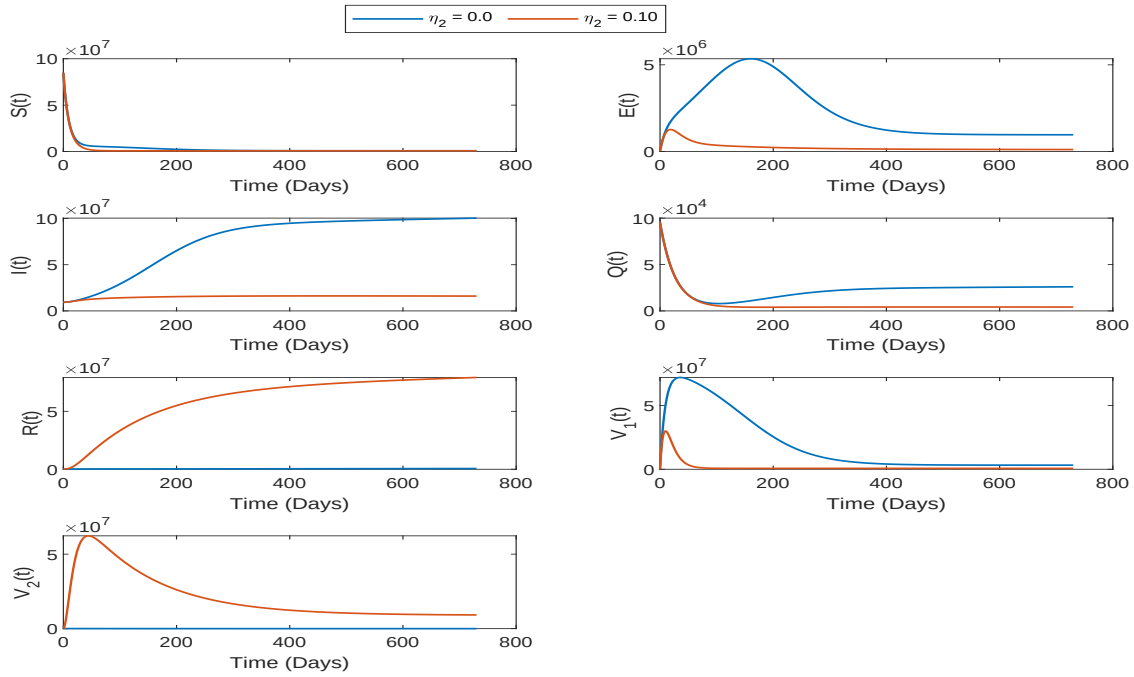

FIGURE 9. The impact of presence and absence of η_2 on model system (1).

data. The sensitivity analysis demonstrates the influence of the parameters on the model. It has been observed that seven out of fourteen sensitivity indices are positive, the other remaining six indices are negative while one index is non-negative which is represented in Figure (5). Thus, it can be seen that the parameters in Table (2) have the most influence on the spread or hold down of the current disease.

Further, we have analysed the impact of effective transmission rate, both first and second dose of vaccination rates on individuals successfully, and the corresponding Figures (6), (7) and (8) are in support of our observations. Thus, in order to prevent the spread of the disease, we must adopt the vaccination drive and interventions like wearing masks, washing our hands, and avoiding close contact with others, strictly. A comparative study of presence and absence of double dose of vaccination drive is portrayed in Figure (9) which emphasizes the importance of vaccination drive that impact the dynamics of COVID-19 transmission among the individuals. We have found the non-negativity, boundedness, disease free equilibrium, basic reproduction number, local stability of the model, successfully. The value of basic reproduction number, $(R_0) < 1$, indicates the disease free environment in near future. Overall, the present study provides the better insights for adopting the interventions to hold down the infection.

The findings that can be drawn from this study are;

(1) The seven compartmental non-linear mathematical model focuses on the vaccination drives in Turkey and assess the COVID-19 infection among the individuals broadly.
(2) The model is non negative or has positive solutions. Further, all solutions of the model system (1) are bounded with non-negative initial conditions. Theorems (2.1) and (2.2) are in support of these findings.
(3) The DFE is pointed out and is locally stable with $(R_0) < 1$ which is elaborated in Theorem (2.3), successfully.

(4) The involved parameters are estimated and the data from January 01, 2022 to December 31, 2023 has been taken for best data fit with model. Figure (3) revealed the data fitting of total COVID-19 reported cases with the proposed model.
(5) The sensitivity analysis, demonstrated in Figure (5), emphasize the influence of the most sensitive parameters on the model.
(6) Further, the impact of effective transmission rate, both first and second vaccination dose, presence and absence of second vaccination dose are elaborated which provide us better insights of the model.

The proposed model can be extended with delay differential equation to understand the current position of the infection waging among the individuals. Some more methods like Statistical methods, different fractional operators, machine learning technique can be adopted to develop this model. The inclusion of some compartments like hospitalized, booster dose of vaccination, symptomatic and asymptomatic infection spreader may be the keen interest of future research and more complex model can be generated. The complex model will generate more information definitely which can be useful for biomedical and science community for further research.

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