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Modeling the Impact of Vaccination on Epidemic Disease Variants with Hospitalization: A Case Study for the COVID-19 Pandemic in Turkey

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Highlights:

- Nonlinear Dynamical Systems
- Global and Local Stability Conditions at Equilibrium Points
- Numerical Simulations BAsed on COVID-19 Variants Data

ABSTRACT:

The stability analysis of an epidemic model that takes into account the impact of vaccination and hospitalization is investigated in this study. Disease-free and endemic equilibrium points are obtained for the stability analysis. The necessary conditions for analyzing local stability at equilibrium points as well as global stability at the disease-free equilibrium point are also defined. Using data from three different periods corresponding to the emergence of three different variants of the COVID-19 outbreak in Turkey, the numerical simulation with graph fitting for the model is also taken into account. The analysis considers the efficacy of vaccination in restricting the virus's spread.

Keywords:

- Epidemic mathematical model
- Equilibrium points
- Stability analysis
- Numerical simulation
- Dynamical systems

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INTRODUCTION

The novel coronavirus disease (COVID-19) was first identified on January 13, 2020. The World Health Organization's Technical Report (https://www.who.int/emergencies/diseases/new-coronavirus-2019) states that the novel coronavirus's initial symptoms included fever, coughing, and shortness of breath, and that the respiratory system was typically impacted. While the onset of symptoms is 5–6 days from the first infection, the incubation period can vary between 2–14 days. COVID-19 cases, which started to be seen outside of China as of January 2020, were seen for the first time in Turkey on March 10–11, 2020. More than 15 million cases and almost 100,000 fatalities have been reported as a result of COVID-19 in Turkey, according to data published by the Turkish Ministry of Health (https://covid19.saglik.gov.tr/TR-66935/genel-koronavirus-tablosu.html). The objective of this work is to mathemaically analyze the coronavirus pandemic in Turkey and the spread, persistence, and prevention mechanisms of epidemic diseases via COVID-19-adapted mathematical models with hospitalized variables.

A disease caused by a virus, bacteria, protozoan, or toxin that can pass from one host to another is considered contagious. This transmission can occur through direct physical contact, airborne droplets, water, food, or from mother to newborn. The infected individual may not show symptoms at the early stage of the infection and may develop clinical symptoms later on; this elapsed time is called the incubation period. When infected individuals are included in a susceptible population, the disease spreads throughout the population by means of transmission, and if the number of cases rises above the average in a short period of time, this disease becomes an epidemic. Infected individuals recover from the infection, either by treatment or by the action of the immune system, and acquire varying degrees of immunity. When the number of susceptible individuals decreases, the epidemic slows down or stops—that is, the infection ends. If new susceptible individuals are added to the population through birth or migration, re-infection may occur spread, the epidemic may continue, and the disease may remain in the population for a long time. In this case, the disease is said to be endemic to this population. A pandemic will occur if the disease spreads to many countries and continents (Meltzer et al., 2001; Halloran et al., 2002; Keeling & Eames, 2005).

Despite the rapid spread and lethality of COVID-19, the world is combating this disease with all the means at its disposal. One of the most effective ways to identify and plan ways to fight this virus is through mathematical models. Mathematical models play an important role in predicting the future of the disease and its effects on society. Compartmental models are a very general modeling technique, often applied to the mathematical modeling of infectious diseases. The population is divided into compartments. For example, these compartments can be S (susceptible), I (infected), or R (recovered) for the basic model SIR (Anderson & May, 1991). Individuals in the population can shift between compartments, and labels can be sorted according to the flow order between the compartments. Another epidemic model, SEIR, also has an exposed (E) compartment in addition to the compartments in the SIR model. There are many other epidemic models in the literature that can be taken as a basis, and all these models have been used in the investigation of the transmission of diseases such as tuberculosis, HIV, SARS, and MERS coronaviruses, etc. (Newman, & Girvan 2004; Liu & Zhang, 2011; Sorensen et al., 2012; Rahman et al., 2016; Kim et al., 2021). In 2016, Al-Asouad et al. used the stability analysis of dynamical systems in the analytical examination of mathematical models in the MERS-CoV epidemic and concluded that endemic stability would be ensured by the isolation method to prevent the spread of MERS-CoV. Budhwar and Daniel (2017) used a combination of SEIR and SI

models for humans and mosquitoes, respectively, to investigate the stability of the model generated for malaria. With the COVID-19 epidemic, interest in epidemic mathematical models has increased, and many researchers have considered using epidemic mathematical models to predict the future of the epidemic and its effects on society. The original version of the SEIR model, which is one of the classical models, or the revised version of the model, in which new parameters such as quarantine, hospitalization, and vaccination were added, were used in many COVID-19 studies (He et al., 2020; Ivorra et al., 2020; Ahmed et al., 2021a). In these studies, local or global stability analyses of the models were performed. Bugalia et al. (2020) have developed a new epidemic model for COVID-19, including quarantine and hospitalization. This model consists of compartments that are susceptible individuals, quarantined for isolation, asymptomatic self-quarantined individuals, and asymptomatic individuals. In 2020, Ndaïrou et al. added the super-spreaders class to mathematical modeling of transmission dynamics in the Chinese province of Wuhan (Ndaïrou, et al., 2020). Zeb et al. (2020) developed a new model including an isolation class. Moreover, mathematical models incorporating asymptomatic and symptomatic infected compartments were formulated (Biswas et al., 2020; Ahmed et al., 2021b). Ahmad et al. (2021) suggested a new model, consisting of a susceptible class $S(t)$, the healthy (resistive) class $H(t)$, the infected class $I(t)$, and the quarantine class $Q(t)$. In the study of Yavuz et al. (2021), the effect of vaccination on the spread of COVID-19 was examined. In addition to the studies mentioned above, various mathematical models have been developed to understand the dynamics of the spread of COVID-19 disease. Recently, researchers have extensively investigated COVID-19 from different aspects through these new mathematical models. These studies focused on stability theory, numerical simulation, and global local dynamics (Halloran et al., 2002; Iboi et al., 2020; Ivorra et al., 2020; Samui et al., 2020; Singh et al., 2021). This study examines the COVID-19 disease's spread in Turkey using a new extended SEIR-type dynamical model. The model separates the population into compartments for those who are susceptible, exposed, infected, hospitalized, and recovered.

Mahata et al. (2022) developed numerical solution methods for a model system with vaccination strategies using the Adam-Bashforth-Moulton approach. In addition to numerical solutions of vaccination scenarios, the stability of model at equilibrium points with the time delay parameter was also investigated. Ottaviano et al. (2022) examined the stability of a SAIRS-type epidemic model, specifically considering the impact of symptomatic and asymptomatic infected individuals. Fractional-order SEIR-type models and discrete-time epidemic models were used to characterise the dynamics of COVID-19 outbreaks (Paul et al., 2022; Khalaf et al., 2023; Li et al., 2023;). In addition, stability analyses of fractional-order epidemic models were performed for different virus transmissions such as Nipah and Dengue (Baleanu et al., 2023; Gu et al., 2023).

An extended SEIR-type dynamic model is used in this work to look into the COVID-19 disease's spread throughout Turkey. It is critical to investigate more thorough models that take into account many aspects affecting disease transmission in response to a pandemic like COVID-19. Therefore, we provide an epidemiological model that also includes those who must be hospitalized due to a major infection, in addition to the traditional compartments of susceptible, exposed, infected, and recovered individuals. It also allows us to have a better understanding of the potential cost to the healthcare system and the impact of hospitalization rates on outbreak control.

Additionally, we consider data from Turkey that cover three different time periods, each of which is associated with the predominance of a certain COVID-19 variant. We seek to provide a clearer picture of the dynamics of the outbreak and its response to vaccination attempts by including real-world data into our model. We assess the efficacy of vaccination efforts at various pandemic

stages through numerical simulations and sensitivity studies, and we highlight the crucial variables that affect the outbreak's trajectory.

The findings of this study help to clarify the function of vaccination in combating the COVID-19 pandemic and new epidemic diseases, especially in light of newly developing variations and probable modifications in healthcare requirements. This study emphasizes the value of timely and focused vaccination programs to lessen the virus's effects and offers the framework for a well-informed public health strategy to battle COVID-19 and upcoming pandemics.

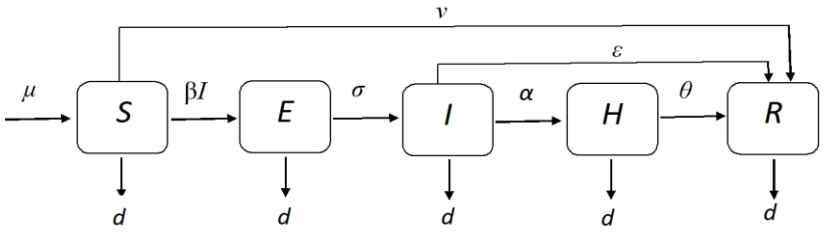

Figure 1: SEIHR model's flow diagram represents the susceptible (*S*), exposed (*E*), infected (*I*), hospitalized (*H*) and recovered (*R*) individuals)

MATERIALS AND METHODS

This study is based on the SEIR model to provide a new perspective on the spread of epidemic diseases, by adding a new variable, hospitalisations and a new parameter vaccination. The SEIR model is very important for the dynamic representation of changes over time and the spread of infection, as it also takes into account exposed individuals, which is one of the main components of epidemics. The inclusion of an exposed compartment in the SEIR model allows a more realistic modelling of disease contagion and transmission. This component describes the latent or incubation period that an infected person goes through before transmitting the disease. This means that the person is infected and has the capacity to spread the disease, even if they are not currently symptomatic. This improves the representation of the spread of real world epidemics and helps to develop more effective disease control strategies.

In this section, we describe the methodology and equipment we employed to examine the dynamics of an epidemic model that takes vaccination and hospitalization into account. We outline the information sources, mathematical constructions, and numerical approaches used to examine how vaccination affects the transmission of disease.

Formulation of the model

$N(t)$, representing the total population at a given time, is classified into five groups: susceptible (S), exposed (E), infected (I), hospitalized (H), and recovered (R). It is assumed that the total population $N = S(t) + E(t) + I(t) + H(t) + R(t)$ is constant. By including the hospitalized compartment in the SEIR model, a new model is created that takes into consideration a parameter associated with the vaccination impact. The following system of ordinary differential equations illustrates a SEIR-type deterministic model of COVID-19 transmission in the community:

$$\frac{d\overline{S}(t)}{dt} = \mu N - \beta \overline{I}(t)\frac{\overline{S}(t)}{N} - \nu\,\overline{S}(t) - d\,\overline{S}(t)$$
$$\frac{d\overline{E}(t)}{dt} = \beta \overline{I}(t)\frac{\overline{S}(t)}{N} - \sigma \overline{E}(t) - d\,\overline{E}(t)$$
$$\frac{d\overline{I}(t)}{dt} = \sigma \overline{E}(t) - \alpha \overline{I}(t) - \epsilon \overline{I}(t) - d\,\overline{I}(t) \quad (1)$$

$$\frac{d\overline{H}(t)}{dt} = \alpha\overline{I}(t) - \theta\overline{H}(t) - d\,\overline{H}(t)$$
$$\frac{d\overline{R}(t)}{dt} = \theta\overline{H}(t) + \epsilon\overline{I}(t) + \nu\,\overline{S}(t) - d\,\overline{R}(t)$$

New variables can be defined using fractions such as $S = \frac{\overline{S}}{N}$, $E = \frac{\overline{E}}{N}$, $I = \frac{\overline{I}}{N}$, $H = \frac{\overline{H}}{N}$, and $R = \frac{\overline{R}}{N}$ in order to normalize the model. The following equations system, with $S + E + I + H + R = 1$, is obtained if we rescale the system (1).

$$\frac{dS}{dt} = \mu - \beta IS - \nu\,S - d\,S$$
$$\frac{dE}{dt} = \beta I\,S - \sigma E - d\,E$$
$$\frac{dI}{dt} = \sigma E - \alpha I - \epsilon I - d\,I \qquad (2)$$
$$\frac{dH}{dt} = \alpha I - \theta H - d\,H$$
$$\frac{dR}{dt} = \theta H + \epsilon I + \nu\,S - d\,R$$

where μ and d denote natural human birth and death rates, respectively. The contact rate between susceptible and exposed individuals is represented by β; ν is the vaccination rate of the susceptible population; σ is the transmission coefficient of exposed to infected populations; α is the rate of transfer of infected individuals to hospitalized individuals; ϵ is the transmission coefficient of infected to the recovered class; θ is the recovery rate of hospitalized individuals. The proposed model's flowchart is presented in Fig. 1. Moreover, system (2) is completed with initial conditions $S(0) = S_0 \geq 0, \quad E(0) = E_0 \geq 0, \quad I(0) = I_0 \geq 0, \quad H(0) = H_0 \geq 0, \quad R(0) = R_0 \geq 0.$

Equilibrium points and stability analysis

The disease-free and endemic equilibrium points need to be determined for stability analysis. The equilibrium points of the system can be found by solving the following system:

$$\frac{dS}{dt} = \mu - \beta IS - \nu\,S - d\,S = 0$$
$$\frac{dE}{dt} = \beta I\,S - \sigma E - d\,E = 0$$
$$\frac{dI}{dt} = \sigma E - \alpha I - \epsilon I - d\,I = 0 \qquad (3)$$
$$\frac{dH}{dt} = \alpha I - \theta H - d\,H = 0$$
$$\frac{dR}{dt} = \theta H + \epsilon I + \nu\,S - d\,R = 0$$

Disease-free equilibrium point

When there is no disease spread and the infected class I is equal to zero, disease-free equilibrium occurs. If system (3) is solved using the $I = 0$ argument, the disease-free equilibrium point E_0 is obtained as follows:

$$E_0(S,E,I,H,R) = (S^0,0,0,0,R^0) = \left(\frac{\mu}{d+\nu},0,0,0,\frac{\nu\mu}{d(d+\nu)}\right) \qquad (4)$$

The expected number of secondary cases produced by a typical infected individual in an entire susceptible population per unit of time is referred to as the basic reproduction number $\mathcal{R}_0$. It is

determined using the matrices with the next-generation method (Diekmann et al., 1990). Based on the system (2), an equation is constructed containing the exposed and infected population classes. According to the definition of $\mathcal{R}_0$, the matrices $\mathcal{F}$ and $\mathcal{V}$, which are the production of the new disease and the disease transition, respectively, for the system (2), are expressed by

$$\mathcal{F} = \begin{bmatrix} \beta IS \\ 0 \\ 0 \end{bmatrix}, \quad \mathcal{V} = \begin{bmatrix} (\sigma + d)E \\ -\sigma E + (\alpha + \epsilon + d)I \\ -\alpha I + (\theta + d)H \end{bmatrix}. \tag{5}$$

The Jacobian matrices of $\mathcal{F}$ and $\mathcal{V}$ are computed at disease-free equilibrium (DFE) point and found as

$$F = \begin{bmatrix} 0 & \beta S^0 & 0 \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{bmatrix}, \quad \mathrm{V} = \begin{bmatrix} \sigma + d & 0 & 0 \\ -\sigma & \alpha + \epsilon + d & 0 \\ 0 & -\alpha & \theta + d \end{bmatrix}.$$

It is also necessary to calculate V^{-1} for basic reproduction number.

$$V^{-1} = \begin{bmatrix} \frac{1}{(\sigma+d)} & 0 & 0 \\ \frac{\sigma}{(\alpha+\epsilon+d)(\sigma+d)} & \frac{1}{(\alpha+\epsilon+d)} & 0 \\ \frac{\sigma\alpha}{(\sigma+d)(\alpha+\epsilon+d)(\theta+d)} & \frac{\alpha}{(\theta+d)(\alpha+\epsilon+d)} & \frac{1}{(\theta+d)} \end{bmatrix}$$

From the definition of repruduction number in next-generation method, it is necessary to compute $D = FV^{-1}$.

$$D = \begin{bmatrix} \frac{\beta\sigma S^0}{(\sigma + d)(\alpha + \epsilon + d)} & \frac{\beta S^0}{(\alpha + \epsilon + d)} & 0 \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{bmatrix}$$

The matrix $D = FV^{-1}$, which is called the next-generation matrix, is a kind of measure of how each class changes multiplicatively to the next generation. This means that the entry *(i, k)* of the matrix *D* is the expected number of new infections in compartment *i* produced by the infected individual initially introduced into compartment *k*. Consider an eigenvector, ***Y*,** which is also a vector of infected classes. The eigenvector equation $D\boldsymbol{Y} = \lambda\boldsymbol{Y}$ is used to determine the changes in the quantity of ***Y***, which is composed of infected classes, at the equilibrium point, where λ indicates eigenvalues. Since ***Y*** represents the infected classes, if $|\lambda| < 1$, ***Y*** will decrease, indicating that the infection will disappear. However, if $|\lambda| > 1$, the disease is spreading. If there is an eigenvalue with an absolute value greater than 1, it can be used to determine whether the disease is spreading. This means that we only need to analyse the dominant eigenvalue, or the eigenvalue with the largest absolute value. Thus, the dominant eigenvalue of $\rho(FV^{-1})$ provides the characteristics we need in the basic reproduction number (Castillo-Garsow et al., 2020).

To find the largest eigenvalues of FV^{-1}, the corresponding matrix is considered.

$$\begin{bmatrix} \frac{\beta\sigma S^0}{(\sigma + d)(\alpha + \epsilon + d)} - \lambda & \frac{\beta S^0}{(\alpha + \epsilon + d)} & 0 \\ 0 & -\lambda & 0 \\ 0 & 0 & -\lambda \end{bmatrix}$$

The spectral radius of the next generation matrix $\rho(FV^{-1})$ is the basic reproduction number, seen as below.

$$\mathcal{R}_0 = \frac{\sigma\beta S^0}{(\sigma + d)(\alpha + \epsilon + d)} \tag{6}$$

The local and global stability of system (2) at DFE point E_0 are demonstrated by the following theorems.

Theorem 1. If $\mathcal{R}_0 < 1$, the system (2) is locally asymptotically stable at the DFE point E_0 .

PROOF. To find the stability of the model, the Jacobian matrix must be evaluated. The Jacobian matrix at DFE point $(S^0, 0,0,0, R^0)$ is presented as follows.

$$J(E_0) = \begin{bmatrix} -\mathrm{d} & 0 & -\beta S^0 & 0 & \gamma \\ 0 & -\sigma - \mathrm{d} & \beta S^0 & 0 & 0 \\ 0 & \sigma & -\alpha - \mathrm{d} & 0 & 0 \\ 0 & 0 & \alpha & -\theta - \mathrm{d} & 0 \\ 0 & 0 & 0 & \theta & -\gamma - \mathrm{d} \end{bmatrix} \tag{7}$$

Characteristic equation of corresponding Jacobian matrix $J(E_0)$ is

$$(-d - \lambda)(-\theta - d - \lambda)(-d - \nu - \lambda)\left[(-\sigma - d - \lambda)(-\alpha - \epsilon - d - \lambda) - \frac{\sigma\beta\mu}{d+\nu}\right]. \tag{8}$$

While the three eigenvalues are obtained directly from the above equation $\lambda_1 = -d$, $\lambda_2 = -(\theta + d), \lambda_3 = -(d + \nu)$, the Routh–Hurwitz criterion for the sign of the real parts of the other two eigenvalues λ_4 and λ_5 is used (Marghitu, 2001). The following characteristic equation, representing the remaining part of the $\det(J(E_0) - \lambda I)$ in Eq. (8) obtained from the Jacobian matrix, is considered as Routh–Hurwitz criterion for second order polynomials: $\lambda^2 + a\lambda + b = 0$ where $a = \alpha + \epsilon + \sigma + 2d > 0$ and $b = (\sigma + d)(\alpha + \epsilon + d)(1 - \mathcal{R}_0)$.

If $\mathcal{R}_0 < 1$, then it is seen that $b > 0$. Therefore, the Routh–Hurwitz criterion, $a > 0$ and $b > 0$, can be verified and it is concluded that other two eigenvalues have negative real part (Marghitu, 2001).

Hence, if $\mathcal{R}_0 < 1$, the system (2) is locally asymptotically stable at the DFE point E_0.

Theorem 2. If $\mathcal{R}_0 < 1$, the system (2) is globally asymptotically stable at DFE point E_0.

PROOF. To prove the global stability of the system (2) at DFE point E_0, the Lyapunov function given in (9) is considered.

$$L = S - S^0 - S^0 \ln\frac{S}{S^0} + \frac{\sigma}{(\sigma + d)(\alpha + \epsilon + d)}E + \frac{1}{(\alpha + \epsilon + d)}I \tag{9}$$

It is obvious that $L(E_0) = 0$. The derivative of L with respect to time is found by

$$L' = S' - S^0\frac{S'}{S} + \frac{\sigma}{(\sigma+d)(\alpha+\epsilon+d)}E' + \frac{1}{(\alpha+\epsilon+d)}I'. \tag{10}$$

If the derivatives S', E' and I' are substituted, we get

$$L' = \left(1 - \frac{S^0}{S}\right)(\mu - \beta IS - \nu S - dS) + \frac{\sigma}{(\sigma+d)(\alpha+\epsilon+d)}(\beta IS - \sigma E - dE) + \frac{1}{(\alpha+\epsilon+d)}(\sigma E - \alpha I - \epsilon I - dI).$$

After rearranging the above equation, we have

$$L' = -S(\nu + d)\left(1 - \frac{S^0}{S}\right)\left(1 - \frac{S^0}{S} + \frac{\beta I}{\nu + d}\right) + I(\mathcal{R}_0 - 1) - \frac{\sigma E}{\alpha + \epsilon + d} + \frac{\sigma E}{\alpha + \epsilon + d} \tag{11}$$

$$\leq -S(\nu + d)\left(1 - \frac{S^0}{S}\right)^2 + I(\mathcal{R}_0 - 1)\,. \tag{12}$$

We obtain from the Eq. (12) that if $\mathcal{R}_0 < 1$, this means that $L' < 0$ and the system is globally asymptotically stable at the DFE point.

Endemic equilibrium point

The system (3) is solved without the assumption of $I = 0$ for the endemic equilibrium point denoted by $E_* = (S^*, E^*, I^*, H^*, R^*)$.

$$\begin{aligned}
&\mu - \beta I^* S^* - \nu S^* - d\, S^* = 0\\
&\beta I^* S^* - \sigma E^* - d\, E^* = 0\\
&\sigma E^* - \alpha I^* - \epsilon I^* - d\, I^* = 0\\
&\alpha I^* - \theta H^* - d\, H^* = 0\\
&\theta H^* + \epsilon I^* + \nu S^* - d\, R^* = 0
\end{aligned} \tag{13}$$

From the third and fourth equation of (13), we obtain

$$E^* = \frac{\alpha + \epsilon + d}{\sigma} I^*, \quad H^* = \frac{\alpha}{\theta + d} I^* \tag{14}$$

Inserting E^* in the second equation of (13), we get

$$S^* = \frac{(\sigma + d)(\alpha + \epsilon + d)}{\beta\sigma}. \tag{15}$$

Substituting H^*, E^* and S^* in the last equation of (13), yields

$$R^* = \left(\frac{\theta\alpha}{d(\theta + d)} + \frac{\epsilon}{d}\right) I^* + \frac{\nu(\sigma + d)(\alpha + \epsilon + d)}{d\beta\sigma}. \tag{16}$$

Using S^* in the first equation of the system (13), we get I^* as

$$I^* = \frac{d+\nu}{\beta}\left(\frac{\beta\mu\sigma}{(d+\nu)(\sigma+d)(\alpha+\epsilon+d)} - 1\right) = \frac{d+\nu}{\beta}(\mathcal{R}_0 - 1). \tag{17}$$

Thus, we conclude with the following theorem.

Theorem 3. The endemic equilibrium point E_* of the system (2) is locally asymptotically stable if $\mathcal{R}_0 \geq 1$.

PROOF. Stability analysis is performed by finding the eigenvalues at the endemic equilibrium point E_* using the Jacobian matrix. It is obvious that if $\mathcal{R}_0 \geq 1$, then E_* exists and is positive.

The characteristic polynomial of the Jacobian matrix at E_* is given by $\det(J(E_*) - \lambda I)$ where λ is the eigenvalue and I is the identity matrix

$$\begin{vmatrix}
-\beta I^* - d - \nu - \lambda & 0 & -\beta S^* & 0 & 0\\
\beta I^* & -\sigma - d - \lambda & \beta S^* & 0 & 0\\
0 & \sigma & -\alpha - \epsilon - d - \lambda & 0 & 0\\
0 & 0 & \alpha * & -\theta - d - \lambda & 0\\
\nu & 0 & \epsilon & 0 & -d - \lambda
\end{vmatrix}.$$

Substituting the I^* and S^* and solving for λ, we obtain

$$\det(J(E_*) - \lambda I) = (-d - \lambda)(-\theta - d - \lambda)[\lambda^3 + a\lambda^2 + b\lambda + c] \tag{18}$$

where

$$\begin{aligned}
a &= (d + \nu)\mathcal{R}_0 + \sigma + \alpha + \epsilon + 2d\\
b &= (d + \nu)(\sigma + \alpha + \epsilon + 2d)\mathcal{R}_0\\
c &= (d + \nu)(\sigma + d)(\alpha + \epsilon + d)(\mathcal{R}_0 - 1)
\end{aligned}$$

It is seen from the equation (18) that the first two eigenvalues are $\lambda_1 = -d$ and $\lambda_2 = -(\theta + d)$. The other eigenvalues are the roots of the characteristic equation given by P in Eq. (19).

$$P = \lambda^3 + a\lambda^2 + b\lambda + c = 0 \tag{19}$$

The Routh-Hurwitz criterion for the characteristic polynomial is used to interpret the signs of the real parts of the roots. The roots of the characteristic equation have a negative real part if and only if $a > 0$, $b > 0$, and $0 < c < ab$. It is clear that $a, b > 0$. Moreover, $c > 0$ when $\mathcal{R}_0 > 1$. It is necessary to show $ab - c > 0$. Calculating and simplifying $ab - c$, we have

$$ab - c = (d+\nu)^2\mathcal{R}_0^2(\alpha+\sigma+\epsilon+2d) + (d+\nu)\mathcal{R}_0(\alpha+\sigma+\epsilon+2d)^2 - \sigma\beta\mu + (d+\nu)(\sigma+d)(\alpha+\epsilon+d). \quad (20)$$

From the $\mathcal{R}_0 > 1$ condition and expression of $\mathcal{R}_0$, we have $\sigma\beta\mu > (d+\nu)(\sigma+d)(\alpha+\epsilon+d)$. Hence, as seen from Eq. (20), if $\mathcal{R}_0 > 1$, then $ab - c > 0$, which means that all roots of the characteristic polynomial (19) have negative real parts. Therefore, system (2) is locally asymptotically stable if $\mathcal{R}_0 > 1$.

Numerical simulation

In addition to mathematical modeling, determining whether this model works with actual data is essential to the validity of the model. Data can be compared with numerical solution results using a curve fitting approach. The parameters of the system are adjusted to fit the data collected within a certain time period, within a margin of relatively small error. To determine the dispersion of data points in regression analysis, the sum of squares is a statistical method that is generally regarded as reliable. The function that best fits the data is determined mathematically using the sum of squares.

In this section, we use the nonlinear curve fitting strategy to obtain the unknown parameters of the proposed model listed in Table 1. The objective of the graph fitting procedure is to define the residual sum of squares (RSS) as

$$RSS = \sum_{i=1}^{n} (f(x_i) - y_i)^2$$

where y_i represents ith value of the given data and $f(x_i)$ is the predicted value from the solution of the model. The error rate was computed as the sum of squares of the differences between the model solutions $f(x_i)$ and the data y_i. The MATLAB programming language was used to insert the dynamic system solution curve into the data with a minimum of error as the selected parameter values changed.

Data was collected between March 24 and July 1, 2020, which coincides with the beginning of the COVID-19 pandemic; between July 22 and October 29, 2021, when the Delta variant predominated; and between February 7 and March 29, 2022, when the Omicron form predominated. The number of cases and the model's numerical results were compared to the data, which was acquired from the Turkey Ministry of Health's website. For the SEIHR model, a graph was inserted into the data with the parameters σ, α, ν and d. The fitted values of these parameters for three cases are given in Table 2. The graphs drawn with fitted values are seen in Figures 2, 3, and 4. The black solid line in these figures represents the model's best-fit curve, whereas the solid red circles show the real COVID-19 cases.

Table 1. Estimated and best fitted values of the parameters used in the proposed COVID-19 model

Parameters	Numerical value
μ	0.2
β	2.5
ϵ	0.7
θ	0.1
σ	fitted
α	fitted
ν	fitted
d	fitted

Table 2. Best fitted values of the parameters used in the proposed COVID-19 model after the graph fitting procedure

Parameters	24.03-01.07 2020	22.07-29.10 2021	07.02-29.03 2022
σ	0.278	0.06	0.076
α	1.22	1.66	0.112
ν	0.275	0.04	1.94
d	0.008	0.06	0.015

RESULTS AND DISCUSSION

The purpose of this study was to construct a mathematical model describing the dynamics of COVID-19. While developing this model, the frequently used mathematical epidemiology literature was taken into account. Moreover, by including the hospitalized variable $H(t)$ and the vaccination parameter ν, the variation in the number of hospitalizations and the effectiveness of vaccination were reviewed.

A stability analysis was performed on the model. In the case of $\mathcal{R}_0 < 1$, the global and local asymptotical stability analyses are confirmed for the disease-free equilibrium point E_0. Furthermore, the endemic equilibrium point E_* is locally asymptotically stable for $\mathcal{R}_0 > 1$.

Since COVID-19 has been around for four years, the virus has undergone mutations, and variants have evolved over time. Some variants of COVID-19, such as Delta and Omicron, are more likely to spread among humans, and even though the Omicron variant is less severe than Delta, it is still dangerous, especially for those who have not received the COVID-19 vaccine. In addition to that, they also have much higher rates of reproduction at baseline. In this study, these two variants are discussed as well as the original COVID-19 virus. The values of α, σ, ν, and d variables were estimated using the graphical fit method during the periods when these two variants were dominant. The values obtained are given in Table 2. Vaccination appears to have had a positive impact on survival during the periods where Delta and Omicron variants were dominant. It is also seen from this table that as the vaccination rate increased for these highly contagious variants, the rate of hospitalization decreased.

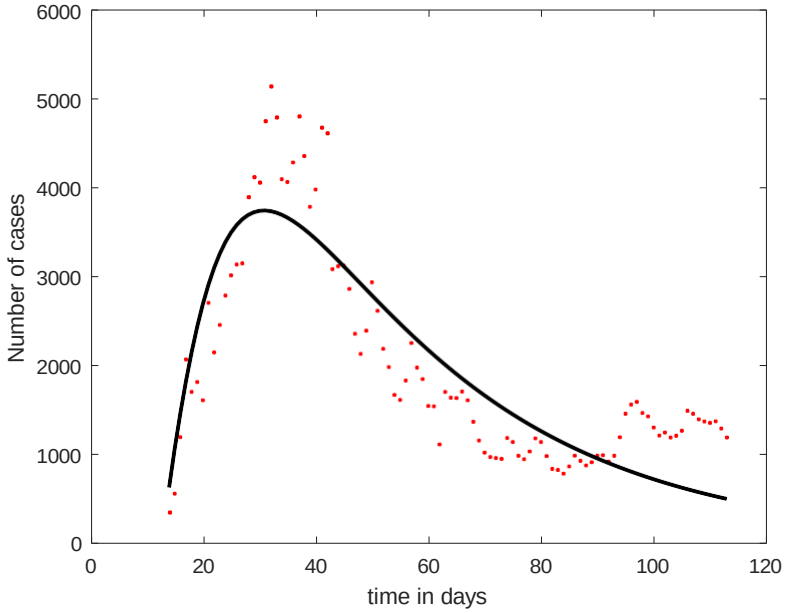

Figure 2: Comparison of our SEIHR model (black solid curve) after performing curve fitting method to the Daily COVID-19 cases (red filled circles) time series in Turkey from March 24 to July 1, 2020, when the orginal COVID-19 predomintes

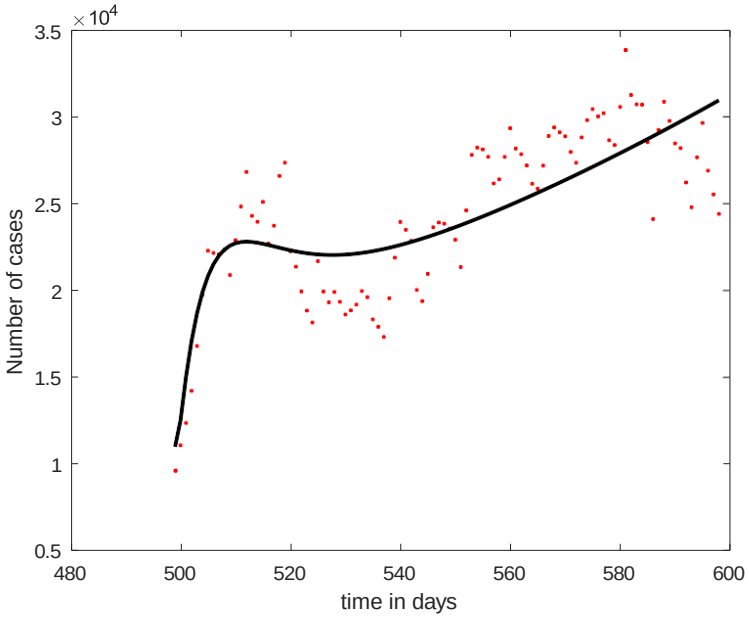

Figure 3: Comparison of our SEIHR model (black solid curve) after performing curve fitting method to the daily COVID-19 cases (red filled circles) time series in Turkey from 22 July to October 29, 2021, when the delta variant predomintes

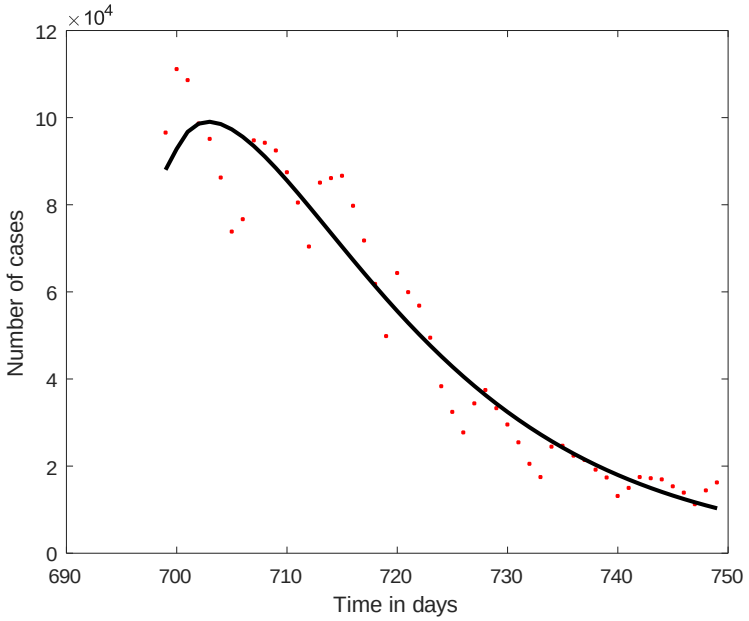

Figure 4: Comparison of our SEIHR model (black solid curve) after performing curve fitting method to the daily COVID-19 cases (red filled circles) time series in Turkey from February 07 to March 29, 2022, when the omicron variant predomintes

CONCLUSION

In this study, we conducted a comprehensive stability analysis of an epidemic model that incorporates vaccination and hospitalization dynamics. We obtained both disease-free and endemic equilibrium points, which serve as key reference points for our stability analysis.

We rigorously developed local stability conditions for these equilibrium points and looked into the overall stability of the disease-free equilibrium point.

We used data from Turkey, representing three unique times corresponding to the emergence of different forms of the COVID-19 virus, to further validate our model and evaluate its applicability in the real world. We included this data into our model through numerical simulations and graph fitting to assess the model's effectiveness in actual-world circumstances.

Our results support the effectiveness of vaccination strategies and provide important information for the control of an epidemic outbreak. Future studies will focus on investigating the global stability of the present model at the endemic equilibrium point and confirming its reliability under different initial conditions and parameter values. In addition, we plan to consider the effects of time delays in the model structure, namely those corresponding to the incubation period of the virus. By taking this into account, we can better understand pandemic dynamics and predict and stop the spread of infectious diseases such as COVID-19.

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Conflict of Interest

The article authors declare that there is no conflict of interest between them

Author's Contributions

The authors declare that they have contributed equally to the article.

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