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Examining the impact of the COVID-19 vaccine on pneumonia cases using a SEIR type model

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Abstract

In this study, the effect of vaccination on the evolution of COVID-19 to pneumonia was investigated using a developed SEIR-type compartmental disease model. Equilibrium points were determined for the model, its stability was examined, and the basic reproduction number was calculated. To obtain numerical solutions for the model, two periods of 62 days were used. The data was obtained from the website of the Ministry of Health of the Republic of Turkey. The effectiveness of the vaccine was examined by comparing these two periods. Graphs of the numerical solutions were then created, and their compatibility with the real data was examined.

Keywords: Epidemic, COVID-19, compartmental disease model, impact of vaccination, stability, basic reproduction number.

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1 Introduction

Since the dawn of humanity, humans have faced many challenges in the fight against diseases. Throughout history, the value of human life has become increasingly important, and it has been understood that health sciences should be given priority in order to improve the quality of life. For this reason, epidemiology, which is a branch of medical science that examines the conditions

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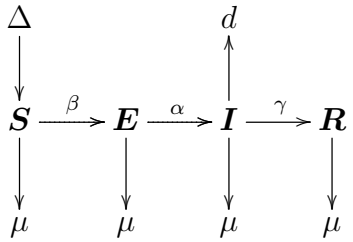
related to public health, their distribution, frequency, and factors affecting them, has gained importance. As can be understood from its definition, epidemiology is a science that works with data.

In the process from the past to the present, epidemic diseases have been an important factor that greatly affects public health. It is of great importance to be able to comment on the emergence and spread of epidemic diseases in a certain population and what the consequences will be in the long and short term. At this point, modeling epidemiological diseases makes a great contribution to having an idea about the course of the epidemic in society.

In the early 20th century, W.H. Hamer [10] argued that the spread of infection would vary depending on the number of susceptible and infected individuals in the population. Thus, the importance of compartmental disease models was understood, and simple compartmental models for infectious diseases were introduced in articles published by Kermack and McKendrick in 1927, 1932, and 1933 [19–21]. Studies on the development of compartmental models have increased over time. Hethcote's [12–16] studies provided the basis for these developments. Some studies on the compartmental disease model are [2–7, 17, 24–26].

An SEIR model is one of the compartmental epidemic disease models. In the SEIR model, society is divided into four classes: S (susceptible) is the class of healthy individuals without disease; E (exposed) is the class of diseased but not showing disease symptoms (incubation class of the disease, class consisting of carrier individuals); I (infected) is the class of diseased individuals; R (recovered) is the class of individuals who have recovered from the disease. In the SEIR model, depending on the time variable t, the number of people in each compartment is expressed by the functions $S(t)$, $E(t)$, $I(t)$, and $R(t)$. Besides, $N(t) = S(t) + E(t) + I(t) + R(t)$ is the total population. The schematic representation of a SEIR model with birth and death is shown below:



The model has the following parameters:

Δ: Number of healthy individuals born per unit time (Crude birth rate)

μ: Natural death rate (Crude death rate)

d: Case mortality rate

β: Disease transmission rate

α: Ratio of those who passed from class E to class I

γ: Recovery rate.

The rate of change of the number of people over time is given by the derivatives:

$$S' = \frac{dS}{dt}, E' = \frac{dE}{dt}, I' = \frac{dI}{dt}, R' = \frac{dR}{dt}.$$

The change in the number of people for each compartment over time is obtained by subtracting the number of people leaving the compartment from the number of people entering the compartment. For example, for compartment S, it would be

$$S' = \text{population growth in } S - \text{population decline in } S$$

When similar operations are applied to all compartments, the following system of differential equations is obtained.

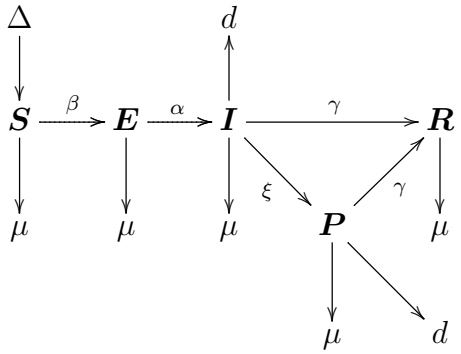
$$\begin{aligned}
\frac{dS}{dt} &= \Delta N - \frac{\beta}{N} SI - \mu S \\
\frac{dE}{dt} &= \frac{\beta}{N} SI - (\alpha + \mu) E \\
\frac{dI}{dt} &= \alpha E - (\gamma + \mu + d) I \\
\frac{dR}{dt} &= \gamma I - \mu R
\end{aligned}$$

The main objective of this study is to investigate the impact of vaccination, regardless of the brand of the vaccine, on the rate of pneumonia in patients affected by COVID-19. This investigation is conducted using a compartmental disease model. The study determines the equilibrium points, stability, and basic reproduction number of the model to ensure its mathematical accuracy. Additionally, numerical solutions are obtained using real data from the Ministry of Health of the Republic of Turkey to provide an example of how the model works. The effectiveness of the vaccine is examined by comparing two periods of 62 days, and graphs of the numerical solutions are created and compared with real data.

2 A SEIR-type compartmental disease model

In this section, we introduce the SEIPR model, which includes people with pneumonia due to COVID-19 disease. We created this model by adding the

class P to the standard SEIR model:



To create the SEIPR model, we added the parameter ξ to the standard SEIR model. The parameter ξ represents the proportion of people passing from class I to class P, which corresponds to the pneumonia rate in patients. Additionally, the total population is changed to

$$N(t) = S(t) + E(t) + I(t) + P(t) + R(t).$$

The corresponding system of differential equations for the SEIPR epidemic model is as follows:

$$\begin{aligned}
\frac{dS}{dt} &= \Delta N - \beta\frac{SI}{N} - \mu S \\
\frac{dE}{dt} &= \beta\frac{SI}{N} - (\alpha+\mu)E \\
\frac{dI}{dt} &= \alpha E - (\xi+\gamma+d+\mu)I \\
\frac{dP}{dt} &= \xi I - (\gamma+d+\mu)P \\
\frac{dR}{dt} &= \gamma(I+P) - \mu R.
\end{aligned} \tag{1}$$

In addition to the model created above, the following assumptions are valid:

a) The population is assumed to have a homogeneous structure, and individuals are assumed to give the same response to infectious diseases. Transmission of the disease is assumed to occur horizontally between individuals through direct contact, and infected individuals are assumed to be contagious without any delay.

b) The number of contacts of individuals in different compartments is assumed to depend on the number of individuals in each compartment. Additionally, since $\frac{I}{N}$ is the ratio of sick individuals in the population to the entire population, the rate of $\beta\frac{I}{N}$ (where β is the rate of transmission of the disease by the individual) gives the rate of transmission in the population. Thus, the number of infections in the entire population is expressed by $\beta\frac{SI}{N}$ [23].

c) Individuals in all compartments are assumed to either move to the next compartment or die by disease or natural means. Additionally, individuals are assumed to not become ill again after recovery, meaning they gain full immunity. Therefore, there is no transition from compartment R back to compartment S.

d) In this study, a new model was created by separating the P compartment from the I compartment to investigate the effect of vaccination on pneumonia. However, since the data from the Ministry of Health of the Republic of Turkey did not provide information about the recovery rates of individuals with pneumonia, it was assumed that the recovery rate of individuals with pneumonia would be the same as the recovery rate (γ) of individuals in compartment I.

e) The basic epidemiological concept that should be used is the case fatality rate. It has been declared that the number of cases announced at the beginning of the epidemic in Turkey is not the number of cases, but the number of patients. Therefore, the case fatality rate has been defined as "the number of deaths/the number of patients". This was done in an attempt to create the most meaningful criterion possible with the available data [1].

The variation of the total population with respect to time is

$$\frac{dN}{dt} = (\Delta - \mu)N - d(I + P),$$

which can be obtained by adding the equations in system (1) side by side. Thus, it can be understood that the total population N is not constant.

The system (1) can be converted to a new system using population ratios instead of the population in system (1). We can obtain $s(t) = \frac{S(t)}{N(t)}$, $e(t) = \frac{E(t)}{N(t)}$, $i(t) = \frac{I(t)}{N(t)}$ $p(t) = \frac{P(t)}{N(t)}$, and $r(t) = \frac{R(t)}{N(t)}$ by considering that $N(t) = S(t) + E(t) + I(t) + P(t) + R(t)$. After changing the variables, the converted system is obtained as:

$$\begin{aligned}
\frac{ds}{dt} &= \Delta(1-s) - \beta si + ds(i+p) \\
\frac{de}{dt} &= \beta si - (\alpha + \Delta)e + de(i+p) \\
\frac{di}{dt} &= \alpha e - (\xi + \gamma + d + \Delta)i + di(i+p) \\
\frac{dp}{dt} &= \xi i - (\gamma + d + \Delta)p + dp(i+p) \\
\frac{dr}{dt} &= (\gamma + dr)(i+p) - \Delta r
\end{aligned} \tag{2}$$

where $s(t) + e(t) + i(t) + p(t) + r(t) = 1$.

3 Equilibrium Points

In this section, we calculated the disease-free and endemic equilibrium points of system (2) using the following system:

$$\begin{aligned}
\frac{ds}{dt} &= \Delta(1-s) - \beta si + ds(i+p) = 0 \\
\frac{de}{dt} &= \beta si - (\alpha + \Delta)e + de(i+p) = 0 \\
\frac{di}{dt} &= \alpha e - (\xi + \gamma + d + \Delta)i + di(i+p) = 0 \\
\frac{dp}{dt} &= \xi i - (\gamma + d + \Delta)p + dp(i+p) = 0 \\
\frac{dr}{dt} &= (\gamma + dr)(i+p) - \Delta r = 0
\end{aligned} \tag{3}$$

3.1 Disease Free Equilibrium Point

In order to obtain the disease-free equilibrium point of system (2), it is necessary to solve the system (3) when $i(t) = p(t) = 0$. Thus, the disease-free equilibrium point of the system (2) is obtained by solving the following system of equations:

$$\begin{aligned}
\frac{ds}{dt} &= \Delta(1-s) = 0 \\
\frac{de}{dt} &= -(\alpha + \Delta)e = 0 \\
\frac{dr}{dt} &= -\Delta r = 0
\end{aligned}$$

The solution is given by:

$$\widehat{E} = (\widehat{s}, \widehat{e}, \widehat{i}, \widehat{p}, \widehat{r}) = (1, 0, 0, 0, 0).$$

3.2 Endemic Equilibrium Point

The endemic equilibrium point of system (2) can be found by solving system (3) in terms of $\tilde{i}$. Thus, the endemic equilibrium point is

$$\tilde{E} = (\tilde{s}, \tilde{e}, \tilde{i}, \tilde{p}, \tilde{r}),$$

where:

$$\begin{aligned}
\tilde{s} &= \frac{\Delta}{(d-\beta)\left(\frac{\gamma+\Delta+d-d\tilde{p}}{d\tilde{p}+\xi}\right)\tilde{p} + d\tilde{p} - \Delta}, \\
\tilde{e} &= \frac{\xi\tilde{p}(\xi+\gamma+d+\Delta)(\gamma+d+\Delta-d\tilde{p})}{\alpha(d\tilde{p}+\xi)^2}, \\
\tilde{i} &= \tilde{p}\frac{\gamma+\Delta+d(1-\tilde{p})}{d\tilde{p}+\xi}, \\
\tilde{p} &= \frac{\gamma+d+\Delta}{\gamma+d+\Delta-2\xi}, \\
\tilde{r} &= \frac{\gamma\tilde{p}(\gamma+d+\Delta+\xi)}{\Delta\xi - d\tilde{p}(\gamma+d+\xi)}.
\end{aligned}$$

4 Local Stability of Disease-Free Equilibrium Point

To ensure the stability of the nonlinear system of equations (2) at the disease-free equilibrium point $\widehat{E} = (\widehat{s}, \widehat{e}, \widehat{i}, \widehat{p}, \widehat{r}) = (1, 0, 0, 0, 0)$, we need to obtain the Jacobian matrix $\mathbf{J}(\widehat{\mathbf{E}})$ of the corresponding system of linearized equations

$$\begin{aligned}
\frac{d\overline{s}}{dt} &= -\Delta\overline{s} + (d-\beta)\overline{i} + d\overline{p} \\
\frac{d\overline{e}}{dt} &= -(\alpha+\Delta)\overline{e} + \beta\overline{i} \\
\frac{d\overline{i}}{dt} &= \alpha\overline{e} - (\xi+\gamma+d+\Delta)\overline{i} \\
\frac{d\overline{p}}{dt} &= \xi\overline{i} - (\gamma+d+\Delta)\overline{p} \\
\frac{d\overline{r}}{dt} &= \gamma(\overline{i}+\overline{p}) - \Delta\overline{r}
\end{aligned} \tag{4}$$

as described in [18]. So, we get:

$$\mathbf{J}(\widehat{\mathbf{E}}) = \begin{pmatrix} -\Delta & 0 & d-\beta & d & 0 \\ 0 & -(\alpha+\Delta) & \beta & 0 & 0 \\ 0 & \alpha & -(\xi+\gamma+d+\Delta) & 0 & 0 \\ 0 & 0 & \xi & -(\gamma+d+\Delta) & 0 \\ 0 & 0 & \gamma & \gamma & -\Delta \end{pmatrix}$$

The eigenvalues of the Jacobian matrix $\mathbf{J}(\widehat{\mathbf{E}})$ are obtained by solving the equation

$$\det[\mathbf{J}(\widehat{\mathbf{E}}) - \lambda\mathbf{I}] = 0.$$

To simplify the notation, we use

$$K^* = \gamma + d + \Delta,\ L^* = \alpha + \Delta,\ M^* = \xi + \gamma + d + \Delta,\ N^* = \alpha\beta. \tag{5}$$

The eigenvalues are then obtained from the equation

$$(\Delta+\lambda)^2\Big\{(K^*+\lambda\Big\}\Big\{\lambda^2 + (L^*+M^*)\lambda + L^*M^* - N^*\Big\} = 0.$$

They are given by

$$\begin{aligned} \lambda_{1,2} &= -\Delta \\ \lambda_3 &= -K^* \\ \lambda_{4,5} &= \frac{-(L^*+M^*) \mp \sqrt{(L^*+M^*)^2 - 4(L^*M^* - N^*)}}{2} \end{aligned}$$

If $(L^*+M^*)^2 > 4(L^*M^* - N^*)$, then all eigenvalues of the Jacobian matrix are real numbers. Since all parameters are positive here, it follows that

$$L^* + M^* > 0, \tag{6}$$

and that $\lambda_1 < 0$, $\lambda_2 < 0, \lambda_3 < 0$. Furthermore, if it is

$$|L^* + M^*| > \sqrt{(L^*+M^*)^2 - 4(L^*M^* - N^*)} \tag{7}$$

then $\lambda_4 < 0$ and $\lambda_5 < 0$. Thus, the condition that all eigenvalues are negative will be satisfied. Under these conditions, the disease-free equilibrium point $\widehat{E} = (1, 0, 0, 0, 0)$ is locally asymptotically stable.

5 Local Stability of Endemic Equilibrium Point

To examine the stability of the $\tilde{E} = (\tilde{s}, \tilde{e}, \tilde{i}, \tilde{p}, \tilde{r})$ endemic equilibrium point of the nonlinear differential equation system (2), we need to calculate the Jacobian matrix at the $\tilde{E}$ endemic equilibrium point of the linearized differential equation system of (2) [18].

To simplify the notation, we use the following definitions:

$$\begin{aligned}
A^* &= -\Delta + (d-\beta)\tilde{i} + d\tilde{p},\\
B^* &= -\Delta + d(\tilde{i} + \tilde{p}),\\
C^* &= -K^* + 2d(\tilde{i} + \tilde{p}),\\
D^* &= -L^* + d(\tilde{i} + \tilde{p}),\\
E^* &= -M^* + d(\tilde{i} + \tilde{p}).
\end{aligned}$$

The Jacobian matrix can be found as follows:

$$\mathbf{J}(\tilde{\mathbf{E}}) = \begin{pmatrix}
A^* & 0 & (d-\beta)\tilde{s} & d\tilde{s} & 0\\
-\beta\tilde{i} & D^* & \beta\tilde{s} + d\tilde{e} & d\tilde{e} & 0\\
0 & \alpha & -M^* + 2d\tilde{i} + d\tilde{p} & d\tilde{i} & 0\\
0 & 0 & \xi + d\tilde{p} & -K^* + d\tilde{i} + 2d\tilde{p} & 0\\
0 & 0 & \gamma + d\tilde{r} & \gamma + d\tilde{r} & B^*
\end{pmatrix}.$$

From here, the eigenvalues are obtained as:

$$(A^*-\lambda)(B^*-\lambda)\Big\{(C^*-\lambda)(D^*-\lambda)(E^*-\lambda) - \alpha d\tilde{e}(E^*-\lambda) - \alpha\beta\tilde{s}(1-\beta\tilde{i})(C^*-\lambda)\Big\} = 0.$$

So, the 5^{th} degree equation is obtained as

$$\begin{aligned}
&\lambda^5 - \lambda^4(A^* + B^* + C^* + D^* + E^*) + \lambda^3\big\{ - C^*D^* - E^*(C^* + D^*)\\
&\quad - (A^* + B^*)(C^* + D^*) + (A^* + B^*)E^* - A^*B^* + d\tilde{e} + \alpha\beta\tilde{s}(1-\beta\tilde{i})\big\}\\
&\quad - \lambda^2\big\{C^*D^*E^* + (A^* + B^*)C^*D^* + (A^* + B^*)(C^* + D^*)E^* + A^*B^*(C^* + D^*)\\
&\quad + A^*B^*E^* - \alpha d\tilde{e}E^* - \alpha\beta\tilde{s}(1-\beta\tilde{i})C^* - (A^* + B^*)(\alpha d\tilde{e}E^* - \alpha\beta\tilde{s}(1-\beta\tilde{i}))\big\}\\
&\quad + \lambda\big\{(A^* + B^*)C^*D^*E^* + (A^* + B^*)(\alpha d\tilde{e}E^* - \alpha\beta\tilde{s}(1-\beta\tilde{i})C^*) - A^*B^*C^*D^*\\
&\quad - A^*B^*(C^* + D^*)E^* + A^*B^*(\alpha d\tilde{e}E^* - \alpha\beta\tilde{s}(1-\beta\tilde{i})\big\}\\
&\quad - A^*B^*C^*D^*E^* + A^*B^*(\alpha d\tilde{e}E^* - \alpha\beta\tilde{s}(1-\beta\tilde{i})C^* = 0. \qquad (8)
\end{aligned}$$

The Routh-Hurwitz criteria are used because it is difficult to calculate the eigenvalues from this equation. If the Routh-Hurwitz criteria for $n = 5$ are

met, the given system is stable [2]. The Routh-Hurwitz criteria for $n = 5$ are; $(a_1a_4 - a_5)(a_1a_2a_3 - a_3^2 + a_1^2a_4) > a_5(a_1a_2 - a_3)^2(a_1a_5^2)$, $a_i > 0$, $(i = 1, 2, \ldots, 5)$, and $a_1a_2a_3 > a_3^2 + a_1^2a_4$.

So, from equation (8) the coefficients are found as:

$$
\begin{aligned}
a_1 =& -(A^* + B^* + C^* + D^* + E^*)\\
a_2 =& C^*D^* - E^*(C^* + D^*) + (A^* + B^*)(C^* + D^*) - (A^* + B^*)E^* + A^*B^*\\
&- d\tilde{e} - \alpha\beta\tilde{s}(1 - \beta\tilde{i})\\
a_3 =& -\{C^*D^*E^* + (A^* + B^*)C^*D^* + (A^* + B^*)(C^* + D^*)E^* + A^*B^*(C^* + D^*)\\
&+ A^*B^*E^* - \alpha d\tilde{e}E^* - \alpha\beta\tilde{s}(1 - \beta\tilde{i})C^* - (A^* + B^*)(\alpha d\tilde{e}E^* - \alpha\beta\tilde{s}(1 - \beta\tilde{i}))\}\\
a_4 =& -\big\{(A^* + B^*)C^*D^*E^* + (A^* + B^*)(\alpha d\tilde{e}E^* - \alpha\beta\tilde{s}(1 - \beta\tilde{i})C^*) - A^*B^*C^*D^*\\
&- A^*B^*(C^* + D^*)E^* + A^*B^*(\alpha d\tilde{e}E^* - \alpha\beta\tilde{s}(1 - \beta\tilde{i})\big\}\\
a_5 =& -A^*B^*C^*D^*E^* + A^*B^*(\alpha d\tilde{e}E^* - \alpha\beta\tilde{s}(1 - \beta\tilde{i})C^*.
\end{aligned}
$$

6 Basic Reproduction Number

The basic reproduction number is important because it provides preliminary information about the course of an epidemic. Epidemiologically, the reproduction number is the number of secondary cases one infectious individual will produce in a population consisting only of susceptible individuals [24].

If $R_0 > 1$, the disease remains in the population. If $R_0 < 1$, the number of infected individuals gradually declines to zero, and the disease disappears from the population [24]. Some studies on the basic reproduction number are [9], [30], and [28].

For the SEIPR epidemic model, the basic reproduction number, R_0, is calculated at the disease-free equilibrium point using the next-generation matrix method [29], [8].

The Jacobian matrix of the following equations:

$$
\begin{aligned}
\frac{de}{dt} &= \beta i - L^*e,\\
\frac{di}{dt} &= \alpha e - M^*i,\\
\frac{dp}{dt} &= \xi i - K^*p,
\end{aligned}
$$

is in the form:

$$
\mathbf{J} = \begin{pmatrix} -L^* & \beta & 0\\ \alpha & -M^* & 0\\ 0 & \xi & -K^* \end{pmatrix}.
$$

Let T be a matrix representing contamination, and V be a matrix representing transport, satisfying the condition $J = T + V$. Thus, we get:

$$\mathbf{T} = \begin{pmatrix} 0 & \beta & 0 \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{pmatrix},$$

and

$$\mathbf{V} = \begin{pmatrix} -L^* & 0 & 0 \\ \alpha & -M^* & 0 \\ 0 & \xi & -K^* \end{pmatrix}.$$

The $P = -TV^{-1}$ matrix is:

$$\mathbf{P} = \begin{pmatrix} \frac{\alpha\beta}{L^*M^*} & \frac{\beta}{M^*} & 0 \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{pmatrix}.$$

If the eigenvalues of the P matrix are calculated, we get:

$$\det[\mathbf{P} - \lambda\mathbf{I}] = \begin{vmatrix} \frac{\alpha\beta}{L^*M^*} - \lambda & \frac{\alpha\beta}{L^*M^*} & 0 \\ 0 & -\lambda & 0 \\ 0 & 0 & -\lambda \end{vmatrix} = 0.$$

So, we get $\lambda^2\left(\frac{\alpha\beta}{L^*M^*} - \lambda\right) = 0 \Rightarrow \lambda_1 = \lambda_2 = 0,\ \lambda_3 = \frac{\alpha\beta}{L^*M^*}$. The largest of the eigenvalues of the P matrix gives us the basic reproduction number R_0 as:

$$R_0 = \frac{\alpha\beta}{L^*M^*} = \frac{\alpha\beta}{(\alpha + \Delta)(\xi + \gamma + d + \Delta)}. \tag{9}$$

7 Incidence Rate and Vaccine Efficacy

In this study, the first period was used as the control group to compare the effect of the vaccine on pneumonia with the second period. To make this comparison, the incidence rate of each period must be calculated first. Incidence is a measure of the probability of healthy people at risk of developing a certain disease within a certain period of time. It is obtained by dividing the number of new cases detected in a given population in a given period by the population of that community at mid-year or by the number of people at risk [27]. The formula to calculate the incidence rate for any disease is as follows:

$$\text{Incidence Rate} = \frac{\text{Number of new cases of disease in a given time period}}{\text{Total person at risk during the study period}} \times 100$$

The incidence rate for the first period is 0.005157, and for the second period it is 0.002234.

Relative risk is calculated by dividing the incidence rate of those exposed to the agent by the incidence rate of those not exposed to the agent [22].

Vaccine efficacy is the percentage of the population immunized by the vaccine administered under routine field conditions [27].

The protection level against a disease of interest from a licensed vaccine whose effectiveness has been proven in a population is measured by "vaccine effectiveness", which is determined through observational epidemiological studies conducted under non-ideal field conditions [11]. Therefore, the vaccine effectiveness of the second vaccine was calculated to determine the protection level of the second vaccine for the population.

The formula to calculate vaccine effectiveness for the second vaccine is as follows:

$$\text{Vaccine Effectiveness} = \frac{1^{st} \text{ period incidence rate} - 2^{nd} \text{ period incidence rate}}{1^{st} \text{ period incidence rate}}$$

8 Numerical Simulation

This study used data from the website of the TR Ministry of Health to investigate the effect of vaccination on pneumonia. Specifically, the first 62-day period between 11 December 2020 and 10 February 2021 and the second 62-day period between 30 April 2021 and 30 June 2021 were analyzed using a SEIPR model. These periods were chosen to be as identical as possible and to cover the desired vaccination status of the population. The contagious period of COVID-19 disease was taken as 20 days based on the Ministry of Health's determination of the duration of disease transmission and the quarantine period of 21 days. The recovery rate was calculated using the formula $\gamma = 1/T_0$ for both periods. The value of d was obtained from the Ministry of Health's website. Values for Δ and μ were calculated based on 2019 Turkish Statistical Institute (TUIK) data, while α and ξ were determined by fitting the data to find the most optimal values. All calculations and graphic drawings were done using the fourth-order Runge-Kutta method and the Maple and Mathematica computer algebra systems.

8.1 First Period

The values of the parameters for the first period are:

$$d = 0.091689,\ \xi = 0.005596,\ \Delta = 0.0143,\ \gamma = 0.05,$$
$$\beta = 0.000066,\ \alpha = 0.010375,\ \mu = 0.0053. \tag{10}$$

To obtain the contamination rate β, we used the following equality [24]

$$\frac{\beta}{\gamma} = \frac{\ln \frac{s(0)}{s(62)}}{s(0) - i(0) - s(62)},$$

where

$$s(0) = 825, i(0) = 0.05203, s(62) = 818.$$

The initial values are taken as

$$e(0) = 0.29136,\ i(0) = 0.05203,\ p(0) = 0.00161,$$
$$r(0) = 0.20191,\ s(0) = 825. \tag{11}$$

8.2 Second Period

The values of the parameters for the first period are:

$$\begin{aligned} \Delta &= 0.0143,\ \gamma = 0.05,\ \mu = 0.0053,\ \beta = 0.000063, \\ d &= 0.116803,\ \xi = 0.008779,\ \alpha = 0.002989. \end{aligned} \tag{12}$$

To obtain the contamination rate β, we used the following equality [24]

$$\frac{\beta}{\gamma} = \frac{\ln \frac{s(0)}{s(62)}}{s(0) - i(0) - s(62)},$$

where

$$s(0) = 795, i(0) = 0.02673, s(62) = 789.$$

The initial values are taken as

$$e(0) = 0.31891,\ i(0) = 0.02673,\ p(0) = 0.00064,$$
$$r(0) = 0.68183,\ s(0) = 795. \tag{13}$$

9 Concluding Remarks and Observations

Based on the given data, the value of $R_0 = \frac{\alpha\beta}{(\alpha+\Delta)(\xi+\gamma+d+\Delta)}$ for system (2) is calculated for the first period as $R_0 = 0.000171098$, and for the second period as $R_0 = 0.0000577343$. Since $R_0 < 1$ for both periods, system (2) is locally asymptotically stable at the disease-free equilibrium point $\widehat{E}$. Also, it is evident from equations (6) and (7) that the disease-free equilibrium point is locally asymptotic for both periods, irrespective of the value of R_0. Therefore, every solution of the (2) system approaches this equilibrium and the disease disappears from the population.

Regarding the stability of the endemic equilibrium point, calculating the values of a_i, $i = 1, 2, \ldots, 5$ by using the parameter values given in (10), we obtain for the first period:

$$a_1 = -0.5466410,\ a_2 = 0.0751164,\ a_3 = 0.0016841,$$
$$a_4 = -0.0015632,\ a_5 = 3.12049 \times 10^{-6},$$

and for the second period:

$$a_1 = -0.6499810,\ a_2 = -0.0234219,\ a_3 = 0.00334928,$$
$$a_4 = -0.0105422,\ a_5 = 8.84953 \times 10^{-6}.$$

As a result, it can be seen that the Routh-Hurwitz criteria are not met for both periods, indicating that the endemic equilibrium point is not stable.

According to the data used in this study, the incidence rate during the first period was 0.005157 and during the second period was 0.002234. The second vaccine's efficacy was 0.57 and the relative risk was 0.43.

This means that the risk of developing pneumonia from the disease was 0.43 times lower for the second vaccine than for the first vaccine. Since 0.43 is less than 1, the second vaccine is protective against the disease turning into pneumonia. In other words, the risk of the disease turning into pneumonia in the second period was %57 lower than in the first period. Hence, the protection of the second vaccine is %57.

For the graphs of the real values and the function p, which obtained from the system (2), for both periods, please check Figure 1. Figures 1a and 1b, created with the data obtained, show that the disease tended to disappear from the population in both periods, and it was stable at the disease-free equilibrium point.

When compared with the data from the TR Ministry of Health, the results obtained were consistent. The number of pneumonia patients in the second period was fewer than the number of pneumonia patients in the first period.

Therefore, the decrease in the number of pneumonia patients continued during the second period with the effect of vaccination.

Furthermore, the decrease in the basic reproduction number during the second period indicated that the epidemic had decreased in society. An increase in the number of individuals who are immune to a disease reduces the incidence of that disease when other factors are constant [27].

Thus, it can be concluded that the second vaccine has positive effects on the fight against pneumonia.

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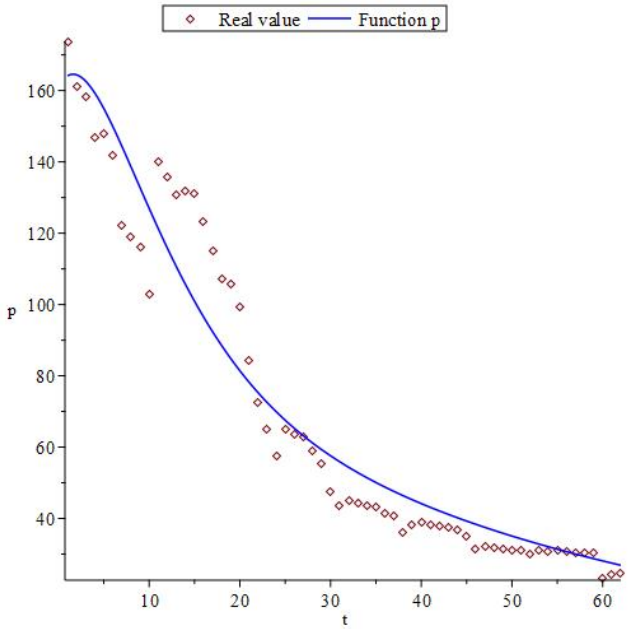
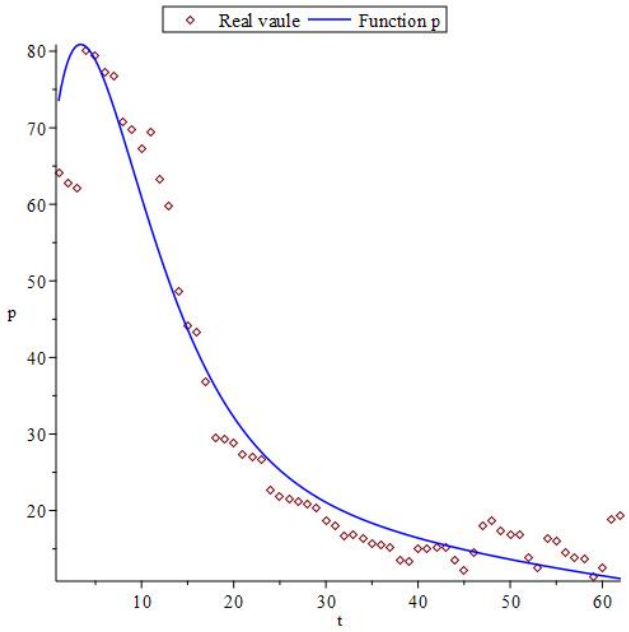


(a) First period



(b) Second period

Figure 1: Graphical comparison of the real values and the p function obtained from system (2) for both periods