Optimal COVID-19 epidemic strategy with vaccination control and infection prevention measures in Thailand

Adison Thongtha, Chairat Modnak*

Department of Mathematics, Faculty of Science, Naresuan University, Phitsanulok, 65000, Thailand

**Abstract**

COVID-19 is a severe acute respiratory syndrome caused by the Coronavirus-2 virus (SARS-CoV-2). The virus spreads from one to another through droplets from an infected person, and sometimes these droplets can contaminate surfaces that may be another infection pathway. In this study, we developed a COVID-19 model based on data and observations in Thailand. The country has strictly distributed masks, vaccination, and social distancing measures to control the disease. Hence, we have classified the susceptible individuals into two classes: one who follows the measures and another who does not take the control guidelines seriously. We conduct epidemic and endemic analyses and represent the threshold dynamics characterized by the basic reproduction number. We have examined the parameter values used in our model using the mean general interval (GI). From the calculation, the value is 5.5 days which is the optimal value of the COVID-19 model. Besides, we have formulated an optimal control problem to seek guidelines maintaining the spread of COVID-19. Our simulations suggest that high-risk groups with no precaution to prevent the disease (maybe due to lack of budgets or equipment) are crucial to getting vaccinated to reduce the number of infections. The results also indicate that preventive measures are the keys to controlling the disease.

© 2022 The Authors. Publishing services by Elsevier B.V. on behalf of KeAi Communications Co. Ltd. This is an open access article under the CC BY-NC-ND license (http://creativecommons.org/licenses/by-nc-nd/4.0/).

1. Introduction

Coronavirus disease (COVID-19) is an infectious disease caused by the SARS-CoV-2 virus. The first outbreak occurred in December in Wuhan, China (Cucinotta and Vanelli, 2020). The typical symptoms are fever, dry cough, fatigue, muscle or joint pain, and high temperature (above 38 °C). Viral infection is a respiratory infection that results in pneumonia and, if not treated in time, can lead to death (Centers for Disease Control and Prevention, 2021). COVID-19 is a disease that can spread from person to person through large droplets or smaller aerosols caused by coughing or sneezing of an infected person. Susceptible people can get the virus by breathing or exposing their hands to contaminated surfaces in the environment. Most infected people will have mild to moderate respiratory symptoms and will recover without the need for medical attention. On the other hand, some people will become critically unwell and require medical assistance. COVID-19 can make anyone sick and cause them to get very ill or die at any age (WHO, 2022). COVID-19 has spread around the world, according to the World
Health Organization, causing a catastrophic outbreak. The outbreaks have negatively impacted public health, education, the economy, human life, and people’s health. In September 2022, the World Health Organization reported an estimated 615 million infected cases, with a total number of deaths of approximately 6.5 million (WHO, 2022). A survey in the United States found that wearing a mask in public can prevent infection by up to 72%, avoidance of crowds by 88%, and frequent hand washing or the use of alcohol gel for protection by 50% (COVID-19 Situation Update Worldwide, 2021). The World Health Organization (WHO) suggests that people should follow these precautions to stop the spread of the disease and get vaccinated. WHO has approved the following vaccines: AstraZeneca/Oxford, Johnson & Johnson, Moderna, Pfizer/BioNTech, Sinopharm, and Sinovac against COVID-19 infection. Each vaccine is effective against COVID-19, and each type of vaccine has a different efficacy against COVID-19. Many countries use these vaccines to reduce the number of infections (WHO; CDC, 2022).

COVID-19 in Thailand began in January 2020, with a maximum of 188 daily cases until March. While the epidemic was back in November 2020, the number of infected people increased to 800 each day before declining to almost zero in March 2021 (Department of Disease Control, 2022; Coronavirus Disease 2019 (COVID-19), 2019). In early 2022, Thailand faced another strike of newly COVID-19 variant, Omicron, and the number of infections has been rising. Latest in September 2022, Thailand had 4.67 million infected cases and about 32,554 deaths. The numbers are declining. Thailand has measures for people to protect themselves by wearing masks, avoiding crowds, frequent hand washing, and encouraging the use of alcohol gel to prevent the spread of the virus. With a limited number of vaccines, the public health administration included additional vaccination programs for older and risky people since vaccines are crucial in the fight against COVID-19 infections (A Guide to COVID, 2021). Thailand has distributed the AstraZeneca/Oxford vaccine and the Sinovac vaccine with the last one being the primary vaccination. Other vaccines such as Johnson & Johnson, Moderna, and Pfizer/BioNTech were not widely used due to lack of availability, they are on the table at present. The picture shown in Fig. 1 is the number of infected cases from June to December 2021 that have been confirmed in Thailand (Department of Disease Control, 2022).

COVID-19 will not go away anytime soon, and hence learning how to live with them is necessary. Furthermore, effective management can help lessen the effects of an outbreak. Mathematical models can be tools for exploring preventive measures. Several COVID-19 models have been proposed to analyze and comprehend the dynamics of the COVID-19 epidemics (Yang and Wang, 2020; Hezam et al, Alrasheedi; Choi & Shim, ; Prathumwan and Trachoo, 2020; Riyapan et al., 2021; Senapati et al., DasIChattopadhyay; Dhaiban and Jabbar; Das et al., 2021; Adewole et al., 2021; Diagne et al., 2021; Ahmed et al., Yusuf; Olaniyi et al., 2020; Jankhonkhan and Sawangtong, 2021; Wu et al., 2021; Kaplan and Milstein, 2021; Wintachaia and Prathom; Abioye et al., 2021; Shah et al., 2021; Arruda et al., 2021; Olivares and Staffetti, 2021; Yang et al., 2022; Tang et al., 2019). For example, in 2020, C. Yang and J. Wang (Yang and Wang, 2020) presented a mathematical model for the novel coronavirus epidemic in Wuhan, China. The model describes multiple transmission pathways of the infection dynamics by emphasizing the role of the environmental reservoir in the transmission and spread of this disease. In the same year, Wongyeong Choi and Eunha Shim (Choi & Shim, ) conducted a study of a game-theoretic model for disease transmission and control measures to determine the optimal individual strategies for preventing infectious diseases. In addition, Abhishek Senapati et al. (Senapati et al., DasIChattopadhyay) hypothesized an intervention effect on the spread of COVID-19 in India in 2020. They evaluated the scenario in India during the early stages of the outbreak and investigated the efficacy of preventative interventions in reducing disease burden at various intervention levels. In 2021, M.L. Diagne et al. (Diagne et al., 2021) studied
the epidemic mechanism of COVID-19 with vaccination and treatment using mathematical models. The findings indicated that COVID-19 immunizations and treatments would be particularly effective in reducing the transmission of the virus. In the same year, Adesoye Idowu Abiyo et al. (Abiyo et al., 2021) presented another mathematical model of COVID-19 in Nigeria with optimal control. They studied an optimal control system by using face masks and hand sanitizers, as well as social distancing, COVID-19 patient care, and active screening with testing. An optimal control problem with vaccination and testing policies for COVID-19 was next presented by Alberto Olivares and Ernesto Staffetti (Olivares and Staffetti, 2021). According to the findings, these control techniques would assist those in charge of systems in creating vaccination programs and testing measures to prevent the spread of COVID-19. In 2022, Bo Yang et al. (Yang et al., 2022) developed a mathematical model of the impact of vaccination on the spread of COVID-19. In this research, the author discovered that vaccination, limiting people’s contact rates, and raising the isolation rate of infected persons would dramatically minimize the number of infections and lessen the time it takes for COVID-19 to spread.

In the most recent papers of 2022, several authors have focused on creating and analyzing mathematical models to explore the impact of COVID-19 (Kifle and Obsu; Ssebuliba et al., Mugisha; Paul and Kuddus; Algaarni et al., 2022; Khana and Atangana; Edholm et al., 2022; Aronna et al., 2022; Yong et al., 2022). For instance, Z.S. Ki and A. Thongtha, C. Modnak Infectious Disease Modelling 7 (2022) 835–855

In these studies, we have learned that the protective measures of susceptible individuals influence the spread of COVID-19. Since most of the previous research has not focused on susceptible individuals who comply with preventive measures or do not comply with the measures, we are interested in developing a system of differential equations to describe the behaviors of COVID-19 considering this situation. Besides, we conducted an equilibrium analysis by including the constant controls in the model for simplicity. Then we investigated the model with time-dependent controls to examine the best way to apply intervention measures by evaluating their impacts and costs on the behavior of the COVID-19 epidemic in real-world situations in Thailand.

Meanwhile, to investigate our parameter values, we have considered a general interval or GI value. A generation interval (GI), also referred to as the generation time, is the time lag between infection incidents in an infected—infected pair. The mean generation interval (GI) equals the mean latent period (LP) plus the mean infectious period (IP). Many articles in prestigious publications employed LP or IP in the modeling of COVID-19 (Griffin et al., 1136; Ferretti et al., 1126; Tang et al., 2021). If using LP or IP with the sum (i.e., GI), e.g., >7 days, this discrepancy will lead to an overestimated basic reproductive number and an exaggerated expectation of the infection attack rate. For accurate estimation and prediction, it is crucial to utilize suitable values for an epidemiological parameter. The mean LP, IP, and GI are tools that usually use to estimate the reproductive number of infectious diseases for COVID-19, and the mean GI (mean LP + mean IP) should be between 5 and 6 days, where the mean LP and IP in SEIR models should be around 2–3 days, respectively (Tang et al., 2021).

In the remainder of this paper, we first propose an epidemic model of COVID-19 with prophylactic measures and constant vaccine controls in susceptible people. Section 2 contains the analysis of the model. Sections 3 and 4 represented the optimal control problem and the numerical simulations. Then Section 5 concludes with a summary and discussion.

2. Mathematical model

To suitable for situations and public health intervention programs in Thailand, we first introduce our mathematical model of the COVID-19 pandemic using a population subdivided into twelve compartments: total susceptible (S), high-risk susceptible (S1), low-risk susceptible (S2), high-risk susceptible with no precaution of the preventive measures (S11), high-risk susceptible who follow the preventive measures (S12), low-risk susceptible with no disease prevention (S21), low-risk susceptible with full attention to the disease control (S22), vaccinated population (V), exposed population (E), infectious individuals with mild symptoms (I1), infectious individuals with severe symptoms (I2), and recovered population (R). The total population at any given time is indicated by N(t) = S11(t) + S12(t) + S21(t) + S22(t) + V(t) + E(t) + I1(t) + I2(t) + R(t), with t ∈ [0, T]. Λ is the new recruitment rate. Other parameters are as follows: μ is the proportion of being high-risk susceptible individuals. u is the proportion of high-risk people for NOT seriously taking preventive measures. w is the proportion of low-risk people for NOT follow mandatory preventive guidelines. We have studied some research papers considering mass action transmission and the standard incidence rate. Due to the complexity of the model, if we used the standard incidence rate, our dynamic analyses would be very complex. Assume that susceptible individuals are infected at a rate of χ(t), with infection force defined as χ(t) = β1E(t) + β2I1(t) + β3I2(t), where β1, β2, β3 are the transmission rates from E, I1, and I2, respectively. ψ, θ, ρ, κ are the effective contact rates of S12, S21, S22, and V, respectively. μ is denoted their natural death rate. ν represents the vaccine efficacy of vaccinated individuals. ξ is the exit rate of recovering naturally from E. τ is the rate at which exposed individuals become infectious individuals. α is the proportion of exposed who becomes infected. γ1 and γ2 are the rates of recovery from infectious individuals with mild symptoms (I1) and infectious individuals with severe symptoms (I2), respectively. d1 and d2 are the rates of disease-induced deaths in I1 and I2, respectively. φ1, φ2, φ3 and φ4 are the vaccination rates in S11, S12, S21 and S22, respectively.
We assume that the susceptible is separated into high-risk and low-risk groups. Then each group is divided into two groups depending on their behaviors. The model's diagram is shown in Fig. 2.

The following system of differential equations is obtained to describe the COVID-19 epidemic dynamics:

\[
\begin{align*}
\frac{dS}{dt} &= \Lambda - (1 + \mu)S, \\
\frac{dS_1}{dt} &= mS - (1 + \mu)S_1, \\
\frac{dS_2}{dt} &= (1 - m)S - (1 + \mu)S_2, \\
\frac{dS_{11}}{dt} &= umS_1 - (\chi + \phi_1 + \mu)S_{11}, \\
\frac{dS_{12}}{dt} &= (1 - u)S_1 - \psi\chi S_{12} - (\phi_2 + \mu)S_{12}, \\
\frac{dS_{21}}{dt} &= wS_2 - \theta_1 S_{21} - (\phi_3 + \mu)S_{21}, \\
\frac{dS_{22}}{dt} &= (1 - w)S_2 - \rho\chi S_{22} - (\phi_4 + \mu)S_{22}, \\
\frac{dV}{dt} &= \phi_1 S_{11} + \phi_2 S_{12} + \phi_3 S_{21} + \phi_4 S_{22} - (1 - \nu)\kappa\chi V - (\nu + \mu)V, \\
\frac{dE}{dt} &= \chi(S_{11} + \psi S_{12} + \theta S_{21} + \rho S_{22}) + (1 - \nu)\kappa\chi V - (\tau + \zeta + \mu)E, \\
\frac{dI_1}{dt} &= a\rho E - (d_1 + \gamma_1 + \mu)I_1, \\
\frac{dI_2}{dt} &= (1 - a)\tau E - (d_2 + \gamma_2 + \mu)I_2, \\
\frac{dR}{dt} &= \nu V + \xi E + \gamma_1 I_1 + \gamma_2 I_2 - \mu R.
\end{align*}
\]

Fig. 2. The transmission diagram of COVID-19 dynamics.
2.1. Boundary of solution

**Theorem 1.** The set $\mathbb{R}_{+}^{12}$ is positively invariant with respect to system (1) – (12). Furthermore, all solutions of (1) – (12) are uniformly bounded in the compact subset

$$
\Omega = \left\{ (S,S_{1},S_{2},S_{11},S_{12},S_{21},S_{22},V,E,I_{1},I_{2},R) \in \mathbb{R}_{+}^{12} : S \leq \frac{\Lambda}{1+\mu} \right\}
$$

Let $N(t) = S_{11}(t) + S_{12}(t) + S_{21}(t) + S_{22}(t) + V(t) + E(t) + I_{1}(t) + I_{2}(t) + R(t)$, then

$$
\begin{aligned}
\frac{dN}{dt} &= \frac{dS_{11}}{dt} + \frac{dS_{12}}{dt} + \frac{dS_{21}}{dt} + \frac{dS_{22}}{dt} + \frac{dV}{dt} + \frac{dE}{dt} + \frac{dI_{1}}{dt} + \frac{dI_{2}}{dt} + \frac{dR}{dt}  \\
&= \frac{\Lambda}{1+\mu} - \mu(S_{11} + S_{12} + S_{21} + S_{22} + V + E + I_{1} + I_{2} + R) - d_{1}I_{1} - d_{2}I_{2}  \\
&= \frac{\Lambda}{1+\mu} - \mu N - d_{1}I_{1} - d_{2}I_{2} \leq \Lambda - \mu N
\end{aligned}
$$

Eq. (13) gives that

$$
\lim_{t \to \infty} N(t) \leq \frac{\Lambda}{\mu(1+\mu)}
$$

It follows from the equation of (1) such that $S \leq \frac{\Lambda}{1+\mu}$. This implies that $S, S_{1}, S_{2}, S_{11}, S_{12}, S_{21}, S_{22}, V, E, I_{1}, I_{2}$ and $R$ are uniformly bounded in the region $\Omega$. Thus, we completes the proof. $
\Box$

2.2. Disease-free equilibrium

The disease-free equilibrium (DFE) for the model (1) – (12) is given by

$$
e_0 = (S_{0},S_{1},S_{2},S_{11},S_{12},S_{21},S_{22},V,E,I_{1},I_{2},R_{0}).
$$

where

$$
S_{0}^0 = \frac{\Lambda}{1+r^2}, \quad S_{1}^0 = \frac{m\Lambda}{(1+\mu)^2}, \quad S_{2}^0 = \frac{(1-m)\Lambda}{(1+\mu)^2}, \quad S_{11}^0 = \frac{um\Lambda}{(\phi_{1} + \mu)(1+\mu)^2}, \quad S_{12}^0 = \frac{(1-m)um\Lambda}{(\phi_{2} + \mu)(1+\mu)^2}, \quad S_{21}^0 = \frac{w(1-m)\Lambda}{(\phi_{3} + \mu)(1+\mu)^2}, \quad S_{22}^0 = \frac{(1-w)(1-m)\Lambda}{(\phi_{4} + \mu)(1+\mu)^2},
$$

$$
V_{0} = \frac{\phi_{2}um\Lambda}{(1+\mu)(1+\mu)^2} + \frac{\phi_{3}w(1-m)\Lambda}{(1+\mu)(1+\mu)^2} + \frac{\phi_{4}(1-w)(1-m)\Lambda}{(1+\mu)(1+\mu)^2},
$$

$$
R_{0} = \frac{\phi_{2}um\Lambda}{\mu(1+\mu)(1+\mu)^2} + \frac{\phi_{3}w(1-m)\Lambda}{\mu(1+\mu)(1+\mu)^2} + \frac{\phi_{4}(1-w)(1-m)\Lambda}{\mu(1+\mu)(1+\mu)^2},
$$

Please note that $\chi(t) = \beta_{1}E(t) + \beta_{2}I_{1}(t) + \beta_{3}I_{2}(t)$, hence at the disease-free equilibrium point $\chi = 0$.

2.3. Basic reproduction number

We first compute the basic reproduction number for this model using the method of van den Driessche and Watmough (van Driessche and Watmough, 2002). Here, the associated next-generation matrices are given by

$$
F(e_0) = \begin{bmatrix} \beta_{1}A_{1} & \beta_{2}A_{1} & \beta_{3}A_{1} \\ 0 & 0 & 0 \\ 0 & 0 & 0 \\ 0 & 0 \end{bmatrix}
$$

and

$$
V(e_0) = \begin{bmatrix} \tau + \xi + \mu & 0 & 0 \\ -\alpha\tau & \gamma_{1} + d_{1} + \mu & 0 \\ -\alpha\tau & 0 & \gamma_{2} + d_{2} + \mu \end{bmatrix}.
$$

where $A_{1} = S_{11}^0 + \psi S_{12}^0 + \theta S_{21}^0 + \mu S_{22}^0 + (1-\nu)e V$. The basic reproduction number is then determined as the spectral radius of $FV^{-1}$, which yields
This parameter’s value is changed in accordance with the data.

This parameter’s value is changed in accordance with the data.

Biologically speaking, $R_0 = 1$ is a threshold for disease epidemics: if $R_0 < 1$, the disease will die out; if $R_0 > 1$, the disease will spread.

2.4. Local and global stability of disease-free equilibrium

Mathematically, based on the work in (P van den Driessche and Watmough, 2002), we immediately obtain the result below:

Theorem 2. The disease-free equilibrium of the model (1) – (12) is locally asymptotically stable if $R_0 < 1$, and unstable if $R_0 > 1$.

Next, we examine the global asymptotic stability of the DFE. To that end, we state the following result, introduced by Castillo-Chavez et al. (Castillo-Chavez and Huang, 2002).

Lemma 3. Consider a model system written in the form

$$
\frac{dx_1}{dt} = F(x_1, x_2),
$$

$$
\frac{dx_2}{dt} = G(x_1, x_2),
$$

where $x_1 \in \mathbb{R}^m$ denotes its components the number of uninfected individuals and $x_2 \in \mathbb{R}^n$ denotes its components the number of infected individuals including latent, infectious, etc; $x_0 = (x_1^*, 0)$ denotes the disease-free equilibrium of the system. Also assume the conditions (H1) and (H2) below:

(H1) For $\frac{dx}{dt} = F(x_1, 0)$, $x^*$ is globally asymptotically stable;

(H2) $G(x_1, x_2) = Ax_2 - G(x_1, x_2), G(x_1, x_2) \geq 0$ for $(x_1, x_2) \in X$, where the Jacobian $A = \frac{dx}{dt}(x_1^*, 0)$ is an M-matrix (the off diagonal elements of $A$ are non-negative) and $X$ is the region where the model makes biological sense.

Then the disease-free equilibrium $x_0 = (x_1^*, 0)$ is globally asymptotically stable.

We now apply this lemma to our model. Note that from equation (4), we observe that $\frac{dV}{dt} \leq uS_1 - (\phi_1 + \mu)S_{11}$, which yields $S_{11} \leq \frac{\mu u}{(\phi_1 + \mu)(1 + \mu)}$. Note that from the equation (5), (6), (7), (8), which gives that $S_{12} \leq \frac{1 - (1 - \mu)u}{(\phi_2 + \mu)(1 + \mu)} = S_{21}^0$, and $V \leq \phi_2 \frac{uM_0}{\phi_2 + \mu} + \frac{1 - (1 - \mu)u}{\phi_2 + \mu} \frac{\phi_2}{\phi_2 + \mu} + \frac{1 - (1 - \mu)u}{\phi_2 + \mu} \frac{\phi_2}{\phi_2 + \mu}$

Theorem 4. The disease-free equilibrium of the model (1) – (12) is globally asymptotic stable if $R_0 < 1$. 

### Table 1

| Symbol | Value | Reference |
|--------|-------|-----------|
| $\Lambda$ | 273523621 | Yong et al. (2022) |
| $U$ | 0.2 | Assumed |
| $\beta_1$ | $1.166 \times 10^{-8}$ | Assumed |
| $\beta_3$ | $4.734 \times 10^{-8}$ | Yong et al. (2022) |
| $\theta$ | 0.8 | Assumed |
| $\kappa$ | 0.25 | Assumed |
| $v$ | 0.6 | Yong et al. (2022) |
| $\tau$ | 0.4 | (Tang et al., 2021; Yong et al., 2022) |
| $\gamma_1$ | 0.33 | Tang et al. (2021) |
| $d_1$ | 0.01 | Tang et al. (2019) |
| $\phi_{s_{max}}$ | 0.7 | Assumed |
| $\phi_{\beta_{max}}$ | 0.7 | Assumed |

| Symbol | Value | Reference |
|--------|-------|-----------|
| $m$ | 0.4 | Assumed |
| $\omega$ | 0.2 | Assumed |
| $\beta_2$ | $2.101 \times 10^{-8}$ | Tang et al. (2019) |
| $\psi$ | 0.1 | Assumed |
| $\rho$ | 0.05 | Assumed |
| $\mu$ | 1 | (Choi & Shim.) |
| $\xi$ | 0.11 | Adewole et al. (2021) |
| $\alpha$ | 0.7 | Adewole et al. (2021) |
| $\gamma_2$ | 0.1 | Tang et al. (2021) |
| $d_2$ | 0.033 | Abioye et al. (2021) |

$\text{Table 1 Parameter value and its symbol.}$
Proof. We only need to show that Lemma 3 and verify the conditions (H1) and (H2). In our ordinary differential equation system, we have $X_1 = (S, S_1, S_2, S_{11}, S_{12}, S_{21}, S_{22}, V)^T$, $X_2 = (E, I_1, I_2)^T$, and $X'_1 = \left( S'_0, S'_{11}, S'_{12}, S'_{21}, S'_{22}, V'_0 \right)^T$. Since $\chi(t) = \beta_1 E(t) + \beta_2 I_1(t) + \beta_3 I_2(t)$, hence at the disease-free equilibrium point $\chi = 0$. We note that

$$
\frac{dX_1}{dt} = F(X_1, 0) = \left( \begin{array}{c}
\Lambda - (1 + \mu) S/nS - (1 + \mu) S_1 \\
(1 - m) S - (1 + \mu) S_2 \\
\mu S_1 - (\phi_1 + \mu) S_{11} \\
(1 - u) S_1 - (\phi_2 + \mu) S_{12} \\
w S_2 - (\phi_3 + \mu) S_{21} \\
(1 - w) S_2 - (\phi_4 + \mu) S_{22} \\
\phi_1 S_{11} + \phi_2 S_{12} + \phi_3 S_{21} + \phi_4 S_{22} - (v + \mu) V
\end{array} \right).
$$

(14)

is linear and its solution can be easily found as:

$$
S(t) = S_0 + c_0 e^{-(1 + \mu)t}, S_1(t) = S_{10} + (c_0 n t + c_1) e^{-(1 + \mu)t} \\
S_2(t) = S_{20} + ((1 - m) c_0 t + c_2) e^{-(1 + \mu)t}, \\
S_{11}(t) = S_{10} + [\int (mc_0 s + c_1) e^{-(1 + \mu)t} ds] e^{-(\phi_1 + \mu)t}, \\
S_{12}(t) = S_{120} + [\int (mc_0 s + c_1)(1 - u) e^{-(1 + \mu)t} ds] e^{-(\phi_2 + \mu)t}, \\
S_{21}(t) = S_{210} + [\int ((1 - m) c_0 s + c_2) e^{-(1 + \mu)t} ds] e^{-(\phi_3 + \mu)t}, \\
S_{22}(t) = S_{220} + [\int ((1 - m) c_0 s + c_2)(1 - w) e^{-(1 + \mu)t} ds] e^{-(\phi_4 + \mu)t},
$$

and,

$$
V(t) = V_0 + [\int \int (mc_0 s + c_1) e^{-(1 + \mu)t} ds e^{-(\phi_1 + \mu)t} dr] e^{-(\phi_1 + \mu)t} \\
+ [\int \int ((1 - m) c_0 s + c_2) \phi_2 (1 - u) e^{-(1 + \mu)t} ds e^{-(\phi_2 + \mu)t} dr] e^{-(\phi_2 + \mu)t} \\
+ [\int \int ((1 - m) c_0 s + c_2) \phi_3 w e^{-(1 + \mu)t} ds e^{-(\phi_3 + \mu)t} dr] e^{-(\phi_3 + \mu)t} \\
+ [\int \int ((1 - m) c_0 s + c_2) \phi_4 (1 - w) e^{-(1 + \mu)t} ds e^{-(\phi_4 + \mu)t} dr] e^{-(\phi_4 + \mu)t}.
$$

Clearly, $S(t) \to S_0, S_1(t) \to S_{10}, S_2(t) \to S_{20}, S_{11}(t) \to S_{10}, S_{12}(t) \to S_{120}, S_{21}(t) \to S_{210}, S_{22}(t) \to S_{220},$ and $V(t) \to V_0$, as $t \to \infty$, which $c_0, c_1, c_2$ are constant. Thus $X'_1 = \left( S'_0, S'_{10}, S'_{12}, S'_{21}, S'_{22}, V'_0 \right)$ is globally asymptotically stable for the subsystem (14).

Next, we have

$$
G(X_1, X_2) = \left( \begin{array}{c} (S_{11} + \psi S_{12} + \theta S_{21} + \rho S_{22} + (1 - \nu) \lambda V) (\beta_1 E + \beta_2 I_1 + \beta_3 I_2) - (\tau + \xi + \mu) E \\
\alpha \tau E - (\gamma_1 + d_1 + \mu) I_1 \\
(1 - \alpha) \tau E - (\gamma_2 + d_2 + \mu) I_2 \end{array} \right).
$$

let $A = S_{11}^0 + \psi S_{12}^0 + \theta S_{21}^0 + \rho S_{22}^0 + (1 - \nu) \lambda V$, we can then obtain

$$
A = \left( \begin{array}{ccc} \beta_1 A - (\tau + \xi + \mu) & \beta_2 A & \beta_3 A \\
\alpha \tau & -\gamma_1 + d_1 + \mu & 0 \\
(1 - \alpha) \tau & -\gamma_2 + d_2 + \mu & 0 \end{array} \right)
$$

which is clearly an M-matrix. Meanwhile, we find,

$$
\hat{G}(X_1, X_2) = \left( \begin{array}{c} (S'_{11} - S_{11}) + \psi (S'_{12} - S_{12}) + \theta (S'_{21} - S_{21}) + \rho (S'_{22} - S_{22}) + (1 - \nu) \lambda (V_0 - V) \\
0 \\
0 \end{array} \right).
$$

It is such that $0 \leq S_{11} \leq S_{11}^0, 0 \leq S_{12} \leq S_{12}^0, 0 \leq S_{21} \leq S_{21}^0, 0 \leq S_{22} \leq S_{22}^0,$ and $0 \leq V \leq V_0$, thus $\hat{G}(X_1, X_2) \geq 0$. Therefore the DFE $X_0 = (X'_1, 0)$ is globally asymptotically stable. □
2.5. Endemic equilibrium

The stability at the DFE determines the short-term epidemics of the disease, whereas the stability at the endemic equilibrium is for the long-term dynamics when the disease persists ($R_0 > 1$). In this section, we will analyze the endemic properties of our model.

We first examine the existence of the positive endemic equilibrium. Let

$$f(E^*) = \beta_E E^* + \frac{\beta}{\tau_1 + d_1 + \mu} + \frac{\beta(1 - a)\tau}{\tau_2 + d_2 + \mu}. \quad f_1(E^*) = (f(E^*) + \phi_1 + \mu)(1 + \mu)^2. \quad f_2(E^*) = (\psi f(E^*) + \phi_2 + \mu)(1 + \mu)^2. \quad f_3(E^*) = (\theta f(E^*) + \phi_3 - \mu)(1 + \mu)^2. \quad f_4(E^*) = (\rho f(E^*) + \phi_4 + \mu)(1 + \mu)^2. \quad f_5(E^*) = (1 - v)\phi f(E^*) + v + \mu.$$ 

Denote the endemic equilibrium of the model by

$$E^* = (S^*, S_1^*, S_2^*, I_1^*, I_2^*, V^*, E^*, I_1^*, I_2^*)^T,$$

where

$$S^* = \frac{\Lambda}{1 + \mu}, \quad S_1^* = \frac{m\Lambda}{(1 + \mu)^2}, \quad S_2^* = \frac{(1 - m)\Lambda}{(1 + \mu)^2}, \quad S_{11}^* = \frac{um\Lambda}{f_1(E^*)},$$

$$S_{12}^* = \frac{(1 - u)m\Lambda}{f_2(E^*)}, \quad S_{21}^* = \frac{w(1 - m)\Lambda}{f_3(E^*)}, \quad S_{22}^* = \frac{(1 - w)(1 - m)\Lambda}{f_4(E^*)},$$

$$E^* = \frac{S_{11}^* + \psi S_{12}^* + \theta S_{21}^* + \rho S_{22}^* + (1 - v)eV^*}{\gamma + \frac{\psi}{\gamma_1 + d_1 + \mu}},$$

$$V^* = \frac{f_1(E^*)}{f_2(E^*)} \left( \phi_1 \frac{um\Lambda}{f_1(E^*)} + \frac{\phi_2 (1 - u)m\Lambda}{f_2(E^*)} + \frac{\phi_3 w(1 - m)\Lambda}{f_3(E^*)} + \frac{\phi_4 (1 - w)(1 - m)\Lambda}{f_4(E^*)} \right).$$

Furthermore, we consider $f(E)$,

$$f(E) = \left( \frac{\beta_1 (\gamma_1 + d_1 + \mu) (\gamma_2 + d_2 + \mu) + \beta_2 \sigma (\gamma_2 + d_2 + \mu) + \frac{\beta_3 (1 - a)\tau (\gamma_1 + d_1 + \mu)}{(\gamma_1 + d_1 + \mu)(\gamma_2 + d_2 + \mu)} \right) E.$$

We substitute equation (15) into $E^*$ for $E = E^*$, and we have

$$g_1(E) = g_2(E)$$

where
\[ g_1(E) = \frac{(\tau + \xi + \mu)(\gamma_1 + d_1 + \mu)(\gamma_2 + d_2 + \mu)}{\beta_1(\gamma_1 + d_1 + \mu)(\gamma_2 + d_2 + \mu) + \beta_2\alpha\tau(\gamma_2 + d_2 + \mu) + \beta_3(1 - \alpha)(\gamma_1 + d_1 + \mu)} \]

\[ g_2(E) = \frac{um\Lambda}{f_1(E)} + \frac{\psi(1 - u)m\Lambda}{f_2(E)} + \frac{\phi_1(1 - w)(1 - m)\Lambda}{f_3(E)} + \frac{\phi_2(1 - w)(1 - m)\Lambda}{f_4(E)} \]

Fig. 4. COVID-19 epidemic model simulation results. (a) The total number of individuals with the virus. (b) Separate plots of \(E\), \(I_1\), and \(I_2\).

Fig. 5. The effectiveness of prevention. (a) The effectiveness of preventive measures. (b) Curves of different proportions being high-risk susceptibles.
Clearly, both \( g_1 \) and \( g_2 \) are differentiable functions for \( E \geq 0 \). It is obvious that \( f_1(E) > 0, f_2(E) > 0, f_3(E) > 0, \) and \( f_4(E) > 0 \), as well as \( f_1'(E) > 0, f_2'(E) > 0, f_3'(E) > 0, \) and \( f_4'(E) > 0 \). Taking the derivative of \( g_1 \) and \( g_2 \) yields the following:

\[
g_1'(E) = 0,
\]

\[
g_2'(E) = \frac{-u m (1 - \mu) \Lambda_1}{f_1(E)^2} - \frac{\psi (1 - u) m \Lambda_2}{f_2(E)^2} - \left( \frac{1}{f_3(E)} \left( \frac{\phi_1 u m \Lambda_1}{\phi_2 (1 - u) m \Lambda_2} + \frac{\phi_3 w (1 - m) \Lambda_3}{\phi_4 (1 - w) (1 - m) \Lambda_4} \right) \right)
\]

\[
- \frac{-L(1 - v) k_1 E}{f_3(E)} + \frac{\phi_2 (1 - u) m \Lambda_2}{f_2(E)^2} + \frac{\phi_3 w (1 - m) \Lambda_3}{f_3(E)^2} + \frac{\phi_4 (1 - w) (1 - m) \Lambda_4}{f_4(E)} < 0.
\]

Hence, on \([0, \infty)\), \( g_1(E) \) is a rising straight line and \( g_2(E) \) is a decreasing one. We can compare their vertical intercepts to determine if the two curves intersect and thus, if a positive endemic equilibrium exists. We can easily observe that if \( R_0 > 1 \), then \( g_1(0) < g_2(0) \), it follows that there is a unique endemic equilibrium of \( E = E^* \). However, if \( R_0 \leq 1 \), then \( g_1(0) > g_2(0) \), implying that there is no endemic equilibrium. Therefore, we can establish the following theorem:

**Theorem 5.** The system of differential equations (1)-(12) has a unique endemic equilibrium when \( R_0 > 1 \).

### 2.6. Bifurcation analysis

Let us now investigate the bifurcation of the system of equations (1)-(12) at the bifurcation point \( R_0 = 1 \). We pick \( \beta_1 \) as the bifurcation parameter. Our analysis is based on the center manifold theory, originally stated in (Castillo-Chavez and Baojun, 2004).

Let us consider \( R_0 \) as a function of \( \beta_1 \) such that \( R_0 = \left( \frac{\mu \phi_1 + \mu \phi_2 + \mu \phi_3 + \mu \phi_4 + \mu \phi_5 + \mu \phi_6}{(1 + \mu)^2} \right) \Pi_1 \) where \( l_1 = \gamma_1 + d_1 + \mu, l_2 = \gamma_1 + d_1 + \mu, l_3 = \gamma_2 + d_2 + \mu, l_4 = \phi_1 + \mu, l_5 = \phi_2 + \mu, l_6 = \phi_3 + \mu, l_7 = \phi_4 + \mu, l_8 = \phi_5 + \mu, l_9 = \phi_6 + \mu, l_{10} = (1 + \mu)^2, \Pi_1 = \frac{u m \Lambda (\phi_1 + \phi_2 + \phi_3 + \phi_4 + \phi_5 + \phi_6 + \phi_7 + \phi_8 + \phi_9 + \phi_{10})}{(1 + \mu)^2 (\phi_1 + \phi_2 + \phi_3 + \phi_4 + \phi_5 + \phi_6 + \phi_7 + \phi_8 + \phi_9 + \phi_{10})}, \Pi_2 = \frac{(1 - v) k_1 E}{(1 - v) k_1 E + \phi_2 (1 - u) m \Lambda + \frac{\phi_3 w (1 - m) \Lambda \phi_4 (1 - w) (1 - m) \Lambda}{\phi_3 w (1 - m) \Lambda + \phi_4 (1 - w) (1 - m) \Lambda}}. \) Setting \( R_0 \) equal to 1 and solving for \( \beta_1 = \beta_1^* \), as follows:

\[
\beta_1 = \beta_1^* = \left( \frac{l_2 l_3 l_4 - (\beta_2 \alpha l_3 + \beta_3 (1 - \alpha) \alpha l_3) l_4 l_4}{l_2 l_3 l_4} \right).
\]

We consider the DFE of the system (1)-(12) and with \( R_0 = 1 \). Now, in the term of parameter \( \beta_1^* \), we have the following Jacobian,
\[
\begin{bmatrix}
-(1 + \mu) & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\
(1 - m) & -(1 + \mu) & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\
0 & u & 0 & -(\phi_1 + \mu) & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\
0 & 1 - u & 0 & 0 & -(\phi_2 + \mu) & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & w & 0 & 0 & 0 & - (\phi_3 + \mu) & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 1 - w & 0 & 0 & 0 & 0 & -(\phi_4 + \mu) & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & \phi_1 & \phi_2 & \phi_3 & \phi_4 & -(v + \mu) & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & -(v + \mu) & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & \alpha \tau & -l_2 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & \gamma_1 & \gamma_2 & -\mu & 0 & 0 & 0 \\
\end{bmatrix}
\]
Now, the right eigenvector corresponding to the zero eigenvalue of the system (1)–(12) at \( \beta_1 = \beta_1^* \) is given by \( W = \begin{bmatrix} w_1, w_2, w_3, w_4, w_5, w_6, w_7, w_8, w_9, w_{10}, w_{11}, w_{12} \end{bmatrix}^T \), where

\[
W = \begin{bmatrix}
0, 0, 0, -\left( \frac{\beta_1 l_2 \beta_3 (1 - \alpha) \tau l_2}{l_1 l_2 l_3} \right) \left( \frac{um\mu}{(\phi_1 + \mu)^2(1 + \mu)^2} \right),
- \left( \frac{\beta_1 l_2 \beta_2 \alpha l_3 + \beta_3 (1 - \alpha) \tau l_2}{l_1 l_2 l_3} \right) \left( \frac{\psi (1 - u) m\mu}{(\phi_2 + \mu)^2(1 + \mu)^2} \right),
- \left( \frac{\beta_1 l_2 \beta_2 \alpha l_3 + \beta_3 (1 - \alpha) \tau l_2}{l_1 l_2 l_3} \right) \left( \frac{\phi (1 - w) (1 - m)\mu}{(\phi_4 + \mu)^2(1 + \mu)^2} \right),
- \left( \frac{\beta_1 l_2 \beta_2 \alpha l_3 + \beta_3 (1 - \alpha) \tau l_2}{l_1 l_2 l_3} \right) \left( \frac{\phi_1 u m\mu}{(\phi_1 + \mu)^2(1 + \mu)^2} + \frac{\psi_2 (1 - u) m\mu}{(\phi_2 + \mu)^2(1 + \mu)^2} \right),
+ \frac{\theta \phi_3 w (1 - m)\mu}{(\phi_3 + \mu)^2(1 + \mu)^2} + \phi \phi_4 (1 - w) (1 - m)\mu + (1 - v) \psi \nu \mu \right),
\end{bmatrix}
\]

In similar way, the left eigenvector at \( \beta_1 = \beta_1^* \) is given by \( V = \begin{bmatrix} v_1, v_2, v_3, v_4, v_5, v_6, v_7, v_8, v_9, v_{10}, v_{11}, v_{12} \end{bmatrix} \), where

\[
V = \begin{bmatrix}
0, 0, 0, 0, 0, 0, 0, 0, 1, \frac{\beta_2 \Pi_1}{l_2 \Pi_2}, \frac{\beta_3 \Pi_1}{l_2 \Pi_2}, 0 \end{bmatrix}^T.
\]

Considering only the nonzero derivatives for the terms \( \frac{\partial f_k}{\partial x_k}(\epsilon_0) \) and \( \frac{\partial f_k}{\partial x_k}(\epsilon_0) \), it follows that

\[
a = 2w_9 w_9 \beta_1 (w_4 + \psi w_5 + \theta w_6 + \rho \gamma + (1 - v) x w_8)
+ 2w_9 w_9 \beta_2 (w_4 + \psi w_5 + \theta w_6 + \rho \gamma + (1 - v) x w_8)
+ 2w_9 w_9 \beta_3 (w_4 + \psi w_5 + \theta w_6 + \rho \gamma + (1 - v) x w_8)
\]

and

\[
b = v_9 w_9 (x_4 + \psi x_5 + \theta x_6 + \rho \gamma + (1 - v) x x_8) = v_9 w_9 \Pi_1 \Pi_2 .
\]

With our definitions of \( V \) and \( W \) and some algebra, we obtain \( a < 0 \) and \( b > 0 \). Hence, based on Theorem 4.1 item(iv) in (Castillo-Chavez and Baojun, 2004), the following result holds:

**Theorem 6.**  The system (1)-(12) is locally asymptotically stable around the endemic equilibrium \( \epsilon^* \) for \( R_0 > 1 \). Moreover, the system undergoes transcritical bifurcation at \( R_0 = 1 \).

### 2.7. Global stability of endemic equilibrium

Next, we will follow the geometric approach originally proposed by Lyapunov and La Salle-Lyapunov (Leon) to investigate the global asymptotic stability of the endemic equilibrium. To that end, we first present the following result based on the geometric approach:

**Theorem 7.** (Globally stability at \( \epsilon^* \)) The unique endemic equilibrium \( \epsilon^* \) of model is globally asymptotically stable whenever \( R_0 > 1 \).

**Proof.** We will prove that \( \epsilon^* \) is globally asymptotically stable. First, we consider the following equation:

\[
L(y) = y - y^* + y^* \ln \frac{y}{y^*}
\]

for \( y, y^* > 0 \), \( y \) can be replaced by \( S, S_1, S_2, S_{11}, S_{12}, S_{21}, S_{22}, V, E, I_1 \), and \( I_2 \). Clearly, \( L(y) \geq 0 \) with the equality holds if and only if \( y = y^* \). Let \( \chi = \beta_1 E + \beta_2 F_1 + \beta_3 F_2 \). Differentiating the functions \( L(S), L(S_1), L(S_2), L(S_{11}), L(S_{12}), L(S_{21}), L(S_{22}), L(V), L(E), L(I_1), L(I_2) \) along the solution of system and using the equilibrium equations yields
Fig. 7. Simulations of optimal control vaccination strategy with the maximum rates of $\phi_1 = \phi_2 = \phi_3 = \phi_4 = 70\%$ and without control. (a) The total number of infected individuals with and without control, (b) The optimal control guidelines, (c) Long term COVID-19 infection for with and without control.
\[
\frac{dS}{dt} = \frac{dS_1}{dt} + \frac{dS_2}{dt} = \left(1 - \frac{S'}{S}\right) \frac{dS}{dt} + \left(1 - \frac{S'_1}{S_1}\right) \frac{dS_1}{dt} + \left(1 - \frac{S'_2}{S_2}\right) \frac{dS_2}{dt}
\]
\[
= \left(1 - \frac{S'}{S}\right)((1 + \mu)S' - (1 + \mu)S) + \left(1 - \frac{S'_1}{S_1}\right)(mS - ms'S_1')
\]
\[
+ \left(1 - \frac{S'_2}{S_2}\right)((1 - m)S_2' - (1 - m)s'S_2')
\]
\[
= (1 + \mu)S' \left(1 - \frac{S'}{S}\right) + mS' \left(1 + \frac{S}{S'} - \frac{S_1'}{S_1'}\right) + (1 - m)S_2' \left(1 + \frac{S}{S'} - \frac{S_2'}{S_2'}\right)
\]
\[
\leq (1 + \mu)S' \left(2 - \frac{S}{S'} + \frac{S_1'}{S_1'}\right) + mS' \left(2 - \frac{S_1'}{S_1'}\right)
\]
\[
+ (1 - m)S_2' \left(2 - \frac{S_2'}{S_2'}\right).
\]

The last inequality follows from the assumptions that
\[
\left(\frac{S_1'S'}{S_1'S'} - 1\right) \leq 0, \quad \left(1 - \frac{S}{S'}\right) \geq 0, \quad 0 \leq S \leq S' \quad \text{and} \quad \left(\frac{S_1'S'}{S_1'S'} - 1\right) > 0, \quad \left(1 - \frac{S}{S'}\right) < 0, \quad S > S'.
\]

Hence, it is easy to obtain
\[
\left(\frac{S_1'S'}{S_1'S'} - 1\right) \left(1 - \frac{S}{S'}\right) \leq 0, \quad S > 0.
\]

we consider,
\[
\frac{dS_1'}{dt} = \frac{dS_1'}{dt} = \left(1 - \frac{S_1'}{S_1'}\right) \frac{dS_1'}{dt} = \left(1 - \frac{S_1'}{S_1'}\right) \left(uS_1' - \chi S_1' - uS_1'S_1' + \chi' S_1'\right)
\]
\[
= uS_1' \left(1 + \frac{S_1}{S_1} - \frac{S_1'S_1'}{S_1'S_1'}\right) + \chi' S_1' \left(\frac{S_1'}{S_1'} + \frac{\chi}{\chi'} - 1 - \frac{\chi S_1'}{\chi S_1'}\right)
\]
\[
\frac{dS_2'}{dt} = \frac{dS_2'}{dt} = \left(1 - \frac{S_2'}{S_2'}\right) \frac{dS_2'}{dt} = \left(1 - \frac{S_2'}{S_2'}\right) \left(1 - \theta S_2' - \psi S_2' - (1 - \theta)S_2'S_2'\right)
\]
\[
= (1 - \theta)S_2' \left(1 + \frac{S_2}{S_2} - \frac{S_2'S_2'}{S_2'S_2'}\right) + \psi' S_2' \left(S_2' + \frac{\chi}{\chi'} - 1 - \frac{\chi S_2'}{\chi S_2'}\right)
\]
\[
\frac{dS_1'}{dt} = \frac{dS_1'}{dt} = \left(1 - \frac{S_1'}{S_1'}\right) \frac{dS_1'}{dt} = \left(1 - \frac{S_1'}{S_1'}\right) \left(wS_2' - \theta S_2' - (1 - w)S_2'S_2'\right)
\]
\[
= wS_2' \left(1 + \frac{S_2}{S_2} - \frac{S_2'S_2'}{S_2'S_2'}\right) + \theta' S_2' \left(S_2' + \frac{\chi}{\chi'} - 1 - \frac{\chi S_2'}{\chi S_2'}\right).
\]
\[
\frac{dS_2'}{dt} = \frac{dS_2'}{dt} = \left(1 - \frac{S_2'}{S_2'}\right) \frac{dS_2'}{dt} = \left(1 - \frac{S_2'}{S_2'}\right) \left(1 - \theta S_2' - \psi S_2' - (1 - \theta)S_2'S_2'\right)
\]
\[
= (1 - \theta)S_2' \left(1 + \frac{S_2}{S_2} - \frac{S_2'S_2'}{S_2'S_2'}\right) + \psi' S_2' \left(S_2' + \frac{\chi}{\chi'} - 1 - \frac{\chi S_2'}{\chi S_2'}\right).
\]
\[
\frac{dL(V)}{dt} = \left(1 - \frac{V^c}{V}\right) \frac{dV}{dt} = \left(1 - \frac{V^c}{V}\right) \left(\phi_1 S_{11} + \phi_2 S_{12} + \phi_3 S_{21} + \phi_4 S_{22} - (1 - \nu) c V^c\right)
\]

\[
\frac{dL(E)}{dt} = \left(1 - \frac{E^c}{E}\right) \frac{dE}{dt} = \left(1 - \frac{E^c}{E}\right) \left(\theta S_{11} + \psi_c S_{12} + \rho c S_{21} + \rho_c S_{22} + (1 - \nu) c V^c\right)
\]

From equations (17), (18), (19), (20), (21), (22), we consider

\[
\frac{dL(S_{11})}{dt} + \frac{dL(S_{12})}{dt} + \frac{dL(S_{21})}{dt} + \frac{dL(S_{22})}{dt} + \frac{dL(V)}{dt} + \frac{dL(E)}{dt}
\]

\[
\frac{dL(S_{11})}{dt} = u S_{11}^c \left(1 + \frac{S_{11}}{S_{11}^c} - \frac{S_{11}^c}{S_{11}}\right) + (1 - u) S_{11}^c \left(1 + \frac{S_{11}}{S_{11}^c} - \frac{S_{11}^c}{S_{11}}\right)
\]

\[
\frac{dL(S_{12})}{dt} = \omega S_{12}^c \left(1 + \frac{S_{12}}{S_{12}^c} - \frac{S_{12}^c}{S_{12}}\right) + (1 - \omega) S_{12}^c \left(1 + \frac{S_{12}}{S_{12}^c} - \frac{S_{12}^c}{S_{12}}\right)
\]

\[
\frac{dL(S_{21})}{dt} = \phi_1 S_{21}^c \left(1 + \frac{S_{21}}{S_{21}^c} - \frac{S_{21}^c}{S_{21}}\right) + \phi_2 S_{21}^c \left(1 + \frac{S_{21}}{S_{21}^c} - \frac{S_{21}^c}{S_{21}}\right)
\]

\[
\frac{dL(S_{22})}{dt} = \phi_3 S_{22}^c \left(1 + \frac{S_{22}}{S_{22}^c} - \frac{S_{22}^c}{S_{22}}\right) + \phi_4 S_{22}^c \left(1 + \frac{S_{22}}{S_{22}^c} - \frac{S_{22}^c}{S_{22}}\right)
\]

\[
\frac{dL(V)}{dt} = \left(1 - \frac{V^c}{V}\right) \frac{dV}{dt} = \left(1 - \frac{V^c}{V}\right) \left(\phi_1 S_{11} + \phi_2 S_{12} + \phi_3 S_{21} + \phi_4 S_{22} - (1 - \nu) c V^c\right)
\]

\[
\frac{dL(E)}{dt} = \left(1 - \frac{E^c}{E}\right) \frac{dE}{dt} = \left(1 - \frac{E^c}{E}\right) \left(\theta S_{11} + \psi_c S_{12} + \rho c S_{21} + \rho_c S_{22} + (1 - \nu) c V^c\right)
\]
\[
\begin{align*}
\frac{dL_1}{dt} &= \left(1 - \frac{l_1}{I_1}\right)\frac{dl_1}{dt} = \left(1 - \frac{l_1}{I_1}\right)\left(\alpha E - \alpha E^* l_1\right) \\
&= \alpha E\left(1 + \frac{E}{E^*} - \frac{l_1}{I_1} \frac{E}{E^*}\right) \\
&= \alpha E\left(2 - \frac{E}{E^*} \frac{E^* l_1}{E l_1} + \left(\frac{E}{E^*} l_1 - 1\right) \left(1 - \frac{E}{E^*}\right)\right) \\
&\leq \alpha E\left(2 - \frac{E}{E^*} \frac{E^* l_1}{E l_1}\right),
\end{align*}
\]

\[
\begin{align*}
\frac{dL_2}{dt} &= \left(1 - \frac{l_2}{I_2}\right)\frac{dl_2}{dt} = \left(1 - \frac{l_2}{I_2}\right)\left(\left(1 - \alpha\right) E - \left(1 - \alpha\right) E^* l_2\right) \\
&= \left(1 - \alpha\right) E\left(1 + \frac{E}{E^*} - \frac{l_2}{I_2} \frac{E}{E^*}\right) \\
&= \left(1 - \alpha\right) E\left(2 - \frac{E}{E^*} \frac{E^* l_2}{E l_2} + \left(\frac{E}{E^*} l_2 - 1\right) \left(1 - \frac{E}{E^*}\right)\right) \\
&\leq \left(1 - \alpha\right) E\left(2 - \frac{E}{E^*} \frac{E^* l_2}{E l_2}\right),
\end{align*}
\]

next, we define the Lyapunov function as follows

\[
L = L(S) + L(S_1) + L(S_2) + L(S_{11}) + L(S_{12}) + L(S_{21}) + L(S_{22}) + L(V) + L(E) + L(l_1) + L(l_2) + L(I_1).
\]

The derivative of \( L \) along solutions of the system gives
The bound re

tions listed in Table 1. Fig. 3 shows that for
\[ \text{denote the upper bounds for the effort of vaccines for different susceptible individuals.} \]
\[ \text{Meanwhile, the integrand of the objective functional in (23) is also convex. Hence, standard optimal control theory} \]
\[ \min_{\phi_i \in \Omega} \int_0^T \left[ I_1(t) + I_2(t) + c_{11} \phi_1(t) S_{11}(t) + c_{12} \phi_2(t) S_{12}(t) + c_{22} \phi_4(t) S_{22}(t) + c_{31} \phi_3(t) S_{31}(t) + c_{41} \phi_4(t) S_{41}(t) \right] dt \]
\[ \text{where } i = 1, 2, 3, 4. \]
\[ \text{Meanwhile, the integrand of the objective functional in (23) is also convex. Hence, standard optimal control theory} \]
\[ \min_{\phi_i \in \Omega} \int_0^T \left[ I_1(t) + I_2(t) + c_{11} \phi_1(t) S_{11}(t) + c_{12} \phi_2(t) S_{12}(t) + c_{22} \phi_4(t) S_{22}(t) + c_{31} \phi_3(t) S_{31}(t) + c_{41} \phi_4(t) S_{41}(t) \right] dt \]
\[ \text{where } i = 1, 2, 3, 4. \]
To find the best control solution, we'll employ Pontryagin's Maximum/Minimum Principle (Pontryagin et al., Mishchenko). This method introduces adjoint functions and defines an optimal control in terms of state and adjoint functions, effectively transforming the issue of minimizing the objective function into minimizing the Hamiltonian with regard to the controls. Let us first define the adjoint functions \( \lambda_5, \lambda_5', \lambda_5', \lambda_5', \lambda_5', \lambda_5', \lambda_5', \) and \( \lambda_5 \) associated with the state equations for \( S, S_1, S_2, S_1, S_2, S_2, V, E, I, \) and \( I_2 \), respectively. We then form the Hamiltonian, \( H \), by corresponding state equations, and adding each of these products to the integrand of the objective functional. As a result, we obtain

\[
H = l_1(t) + l_2(t) + c_1 \phi_1(t)S_1(t) + c_2 \phi_2(t)S_2(t) + c_3 \phi_3(t)
\]

Thus, we have

\[
\frac{dl_1}{dt} = -\frac{\partial H}{\partial S}, \quad \frac{dl_2}{dt} = -\frac{\partial H}{\partial S_1}, \quad \frac{dl_3}{dt} = -\frac{\partial H}{\partial S_2}, \quad \frac{dl_4}{dt} = -\frac{\partial H}{\partial \psi}, \text{ etc.}
\]

To achieve the optimal control, the adjoint functions must satisfy

\[
\frac{dl_1}{dt} = -\frac{\partial H}{\partial S} = -[-\lambda_5(1 + \mu + \lambda_5(1 + \lambda_5)]
\]

\[
\frac{dl_2}{dt} = -\frac{\partial H}{\partial S_1} = -[-\lambda_5(1 + \mu + \lambda_5(1 + \lambda_5)]
\]

\[
\frac{dl_3}{dt} = -\frac{\partial H}{\partial S_2} = -[-\lambda_5(1 + \mu + \lambda_5(1 + \lambda_5)]
\]

\[
\frac{dl_4}{dt} = -\frac{\partial H}{\partial \psi} = -[-\lambda_5(1 + \mu + \lambda_5(1 + \lambda_5)]
\]

With transversality conditions (or final time conditions):

\[
\lambda_5(T) = 0, \quad \lambda_5'(T) = 0, \quad \lambda_5(T) = 0, \quad \lambda_5(T) = 0, \quad \lambda_5(T) = 0, \quad \lambda_5(T) = 0, \quad \lambda_5(T) = 0, \quad \lambda_5(T) = 0, \quad \lambda_5(T) = 0, \quad \lambda_5(T) = 0.
\]

The characterizations of the optimal controls \( \phi_1(t), \phi_2(t), \phi_3(t) \) and \( \psi(t) \) are then based on the conditions

\[
\frac{\partial H}{\partial \phi_1} = 0, \quad \frac{\partial H}{\partial \phi_2} = 0, \quad \frac{\partial H}{\partial \phi_3} = 0 \quad \text{and} \quad \frac{\partial H}{\partial \phi_4} = 0
\]

and for minimization, we have

852
\[
\frac{\partial^2 H}{\partial \phi_1^2} \geq 0, \quad \frac{\partial^2 H}{\partial \phi_2^2} \geq 0, \quad \frac{\partial^2 H}{\partial \phi_3^2} \geq 0 \quad \text{and} \quad \frac{\partial^2 H}{\partial \phi_4^2} \geq 0
\]

subject to the constraints \(0 \leq \phi_1 \leq \phi_{1 \text{ max}}\), \(0 \leq \phi_2 \leq \phi_{2 \text{ max}}\), \(0 \leq \phi_3 \leq \phi_{3 \text{ max}}\) and \(0 \leq \phi_4 \leq \phi_{4 \text{ max}}\). Specifically, we have

\[
\phi_1(t) = \max(0, \min(\phi_1(t), \phi_{1 \text{ max}})), \quad \phi_2(t) = \max(0, \min(\phi_2(t), \phi_{2 \text{ max}})),
\]

\[
\phi_3(t) = \max(0, \min(\phi_3(t), \phi_{3 \text{ max}})), \quad \phi_4(t) = \max(0, \min(\phi_4(t), \phi_{4 \text{ max}})),
\]

where

\[
\phi_1(t) = \frac{(\lambda_{S_1} - \lambda_V - c_{11})S_{11}}{2c_{12}}, \quad \phi_2(t) = \frac{(\lambda_{S_2} - \lambda_V - c_{12})S_{12}}{2c_{22}},
\]

\[
\phi_3(t) = \frac{(\lambda_{S_3} - \lambda_V - c_{13})S_{21}}{2c_{32}}, \quad \phi_4(t) = \frac{(\lambda_{S_4} - \lambda_V - c_{14})S_{22}}{2c_{42}},
\]

and \(\frac{\partial^2 H}{\partial \phi_i^2} \geq 0\) for \(i = 1, 2, 3\) and \(4\).

4. Numerical simulation

We have conducted numerical simulations using various choices of cost parameters and time intervals and have observed a unique solution in each case. The model parameters, including sources, and their values employed in our numerical simulation are listed in Table 1. With the data observed in Thailand, we set the initial infection number to \(S(0) = 4 \times 10^3\), \(S_1(0) = S_2(0) = 0, S_1(0) = S_2(0) = 0, S_2(0) = 0, V(0) = 0, E(0) = 6 \times 10^6, I_1(0) = 2.6 \times 10^4, I_2(0) = 4 \times 10^3, R(0) = 0\), and the entire period of time \(T = 200\) days.

In our mathematical model, the mean GI of an infectious disease equals the sum of the mean LP and the mean IP which in the research of He et al. (He et al., 2010) is defined as \(LP = \frac{1}{\tau - \delta}\) and \(IP = \frac{1}{\gamma_1 - \delta}\), where \(\delta\) designates the time discretization step. The result of the calculation is the sum of the mean LP and mean IP, which is estimated about 5.5 days with 2.5 days of LP and 3 days of IP. Hence our parameter values are as follows: \(\tau^{-1} = 2.5\) days, \(\gamma_1^{-1} = 3\) days, \(\gamma_2^{-1} = 10\) days, and \(\xi^{-1} = 9\) days, and they are suitable in a biologically reasonable sense (see Table 1).

Our simulation shows that the trend of infections as shown in Fig. 4 (a) is similar to the number of infected people in Thailand shown in Fig. 1. Fig. 5 (a) shows the infection levels of the high and low-risk people who follow the intervention programs. The smaller numbers of \(u\) and \(w\) mean fewer people follow the public health interventions. The results of the study show that the effectiveness of cooperation in the implementation of self-protection measures can assist in minimizing the number of people infected with COVID-19. In Thailand’s case, \(u\) and \(w\) rates would be about 0.3 which the result is about the red line. As naturally expected, if more people encounter risky situations, they are considered high-risk groups, and the number of infected will rise as seen in Fig. 5 (b). The situation that occurred in Thailand during the time would be about \(m = 0.4\) since most people were taking the mandatory measures seriously.

Next, we investigate the vaccine efficacy as shown in Fig. 6. The results show that if vaccines distributed in Thailand had high efficacy, the number of infections would be fewer as shown in Fig. 6 (a). We further investigate what if Thailand had not given vaccines as a control measure at all (shown in Fig. 6 (b)), the infection number would be tremendous (blue line) compared to distributing a vaccine program with efficacy only 50 percent of protection (dashed - line).

In this simulation, we proposed optimal vaccination guidelines and interpreted what if Thailand followed these strategies. Fig. 7 (a) shows that if Thailand followed the vaccination guidelines (dashed - lines), it could dramatically lower the number of low-infected humans. In this simulation, we set the cost parameters \(c_{11} = 1, c_{12} = 3, c_{21} = 1, c_{22} = 3, c_{31} = 1, c_{32} = 3, c_{41} = 1, c_{42} = 3\), where the cost “1” represents the cost of vaccine per individual and the cost “3” is the cost of accessing to get vaccinated of individuals. Using Thailand data, the blue line is the approximate infections close to real data. The vaccination strategy guidelines for the different susceptible groups are shown in Fig. 7 (b) resulting very low number of infections shown in Fig. 7 (a). After an outbreak, all susceptibles need to get vaccines, and the number of vaccinated individuals should be at 70 percent of each group. The guidelines suggest as follows: high-risk individuals with no precaution of disease prevention require 20 days, 17 days for low-risk susceptibles with no disease prevention, 5 days for high-risk individuals who follow the preventive measure, and no vaccine needed for low-risk susceptibles with full attention to the disease control. Fig. 7 (c) shows the dynamic of COVID-19 for a much longer time. Without any control, the disease may return sometime. In contrast, with an optimal vaccination strategy, the infection is quickly reduced to a level very close to zero and does not return to spread.

5. Conclusions

In this research, we have developed a system of differential equations to describe the behavior of COVID-19. The model classified the population into susceptible individuals who follow preventative measures and who do not. We then performed an analysis of the disease-free and endemic dynamics of the model. In particular, we established the local and global stability based on the basic reproductive number \((R_0)\). If \(R_0\) is less than one, then the disease dies out, and the disease-free
equilibrium is stable. If $R_0$ is more than one, then the disease persists, and the disease-free equilibrium is unstable. We have also conducted a numerical simulation to confirm our global stability analysis. Besides, we have investigated a bifurcation analysis.

Our model simulations indicated that the infection pattern, as shown in Fig. 4 (a), showed a similar trend to real data of Thailand as represented in Fig. 1. The data is the number of infected people from the epidemic situation in Thailand from June 2021 to December 2021. To perform numerical simulations, we investigated suitable parameter values using the mean general interval (GI), and with the listed values in Table 1 we had an average GI of 5.5 days, with 2.5 days of LP and 3 days of IP, which is reasonable for COVID-19 (Tang et al., 2021). Meanwhile, our study shows that personal protection is an effective factor in reducing the number of infections, as shown in Fig. 5. As for the efficacy of the vaccine, Fig. 6 (a) shows that vaccine efficacy is another factor that can reduce the number of infections. Vaccination is one of the essential factors, as seen in Fig. 6 (b). Even if the vaccine is only 50% effective, it is clear that vaccination can lower the number of infections. Moreover, our investigations recommend that risk people with no precaution to prevent the disease should be first in line to get vaccinated with full strength since this group may be the key to stopping the disease from spreading. Interestingly, the optimal control guidelines point out that high-risk and low-risk people with no precaution for disease prevention should get vaccinated at a high level of implementation. However, low-risk people with full attention to protecting themselves do not need to get vaccinated at all. The simulation results may help manage vaccine distribution programs when having limited resources. However, even vaccinated people can get the disease, so other measures are still necessary. Our model used the mass action transmission rate due to the complexity of the model. However, we have seen many works using the standard one, and that may be our future work for model modification.

Declaration of competing interest

We declare that the authors have no conflict of interest.

Acknowledgments

The author thanks Dr.Chairat Modnak for counseling and guidance of research in this regard. Thank you to the Department of Mathematics, Faculty of Science, Naresuan University, Thailand, for supporting required services to complete this research.

References

A. I. Abioye, O. J. Peter, H. A. Ogunseye, F. A. Oguntolu, K. Oshinubi, A. A. Ibrahim, I. Khan, Mathematical model of covid-19 in nigeria with optimal control, Results in Physics 8. https://www.sciencedirect.com/science/article/pii/S2546140520302771.

Adewole, M. O., Onifade, A. A., Abdullah, F. A., Kasali, F., & Ismaili, A. I. M. (2021). Modeling the dynamics of covid-19 in Nigeria. International Journal of Algorithms, Computing and Mathematics, 7, 67.

I. Ahmed, G. U. Modu, A. Yusuf, P. Kumam, I. Yusuf, A mathematical model of coronavirus disease (covid-19) containing asymptomatic and symptomatic classess, Results in Physicin 21.

Algarni, A. D., Hamed, A. B., Hamdi, M., Elmennai, H., & Meshoul, S. (2022). Mathematical covid-19 model with vaccination: A case study in Saudi Arabia. PeerJ Computer Science, 17. https://doi.org/10.7717/peerj-cs.959.

Aronna, M., Guglielmi, R., & Moschen, L. (2022). Estimate of the rate of unreported covid-19 cases during the first outbreak in rio de janeiro. Infectious Disease Modelling, 7, 317–332. https://doi.org/10.1016/j.idm.2022.06.001.

Arnuda, E. F., Das, S. S., Dias, C. M., & Pastore, D. H. (2021). Modelling and optimal control of multi strain epidemics, with application to covid-19. PLoS One, 16(9), 1–18.

Asano, E., Gross, J. J., Lenthart, S., & Real, L. A. (2008). Optimal control of vaccine distribution in a rabies metapopulation model. Mathematical Biosciences and Engineering, 5, 219–238.

Buonomo, B. (2011). A simple analysis of vaccination strategies for rubella. Mathematical Biosciences and Engineering, 8, 677–687.

Castillo-Chavez, C., & Baqjun, S. (2004). On computation of $R_0$ and its role on global stability, in mathematical approaches for emerging and reemerging infectious diseases: An introduction. Dynamical models of tuberculosis and their applications, I(2), 361–404.

Castillo-Chavez, C., & Huang, W. F. W. (2002). On computation of $R_0$ and its role on global stability. In Mathematical approaches for emerging and reemerging infectious diseases: An introduction. IMA Volumes in Mathematics and its Applications. Springer–Verlag.

CDC. Vaccines for COVID-19, centers for disease control and prevention. https://www.cdc.gov/coronavirus/2019-ncov/vaccines/index.html. (Accessed 21 January 2022).

Centers for Disease Control and Prevention. Symptoms of COVID-19. https://www.cdc.gov/coronavirus/2019-ncov/symptoms-testing/symptoms.html. (Accessed 22 February 2021).

Coronavirus Disease 2019 (COVID-19). WHO Thailand situation report 197-19 August 2021 [EN/TH]-Thailand. https://reliefweb.int/report/thailand/coronavirus-disease-2019-who-thailand-situation-report-197-19-august-2021. (Accessed 19 August 2021).

COVID-19 situation update Worldwide, as of week 21. Updated 10 June 2020. https://www.ecdc.europa.eu/en/geographical-distribution-2019-ncov-cases. (Accessed 13 June 2021).

Cucinotta, D., & Vanelli, M. (2020). Who declares covid-19 a pandemic. Acta BioMedica, 91, 157–160.

Das, P., Upadhyay, R. K., Misra, A. K., Rihan, F. A., Das, P., & Ghosh, D. (2021). Mathematical model of covid-19 with comorbidity and controlling using non-pharmaceutical interventions and vaccination. Nonlinear Dynamics, 106, 1213–1227.

Department of Disease Control. Corona virus disease (COVID-19). https://ddc.moph.go.th/viralpneumonia/eng/index.php. (Accessed 1 January 2022).

A. K. Dhaiban, B. K. Jabbar, An optimal control model of covid-19 pandemic: A comparative study of five countries, OPSEARCH.

Diagne, M. L., Rweazura, H., Thcoumi, S. X., & Tchuente, J. M. (2021). A mathematical model of covid-19 with vaccination and treatment. Computational and Mathematical Methods in Medicine.

den Driessche, P. V., & Watmough, J. (2002). Reproduction numbers and sub-threshold endemic equilibria for compartmental models of disease transmission. Mathematical Biosciences, 180, 29–48.
Edholm, C. J., Levy, B., Spence, L., Agusto, F. B., Chirovecchio, F., Chukwu, C. W., Goldsiman, D., Kosimore, M., Maposa, I. White, K., & Lenhart, S. (2022). A vaccine model for COVID-19 in Gauteng, South Africa. *Infectious Disease Modelling*, 7(3), 333–345. https://doi.org/10.1016/j.idm.2022.06.002

L. Ferretti, C. Wyman, M. Kendall, L. Zhao, A. Nurtay, Abeler-Dorner, M. Parker, D. Bonsall, C. Fraser. Quantifying sars-cov-2 transmission suggests epidemic control with digital contact tracing. *Science* 368. https://doi.org/10.1126/science.abb6936.

Lenhart, S. & Workman, J. (2007). *Optimal control applied to biological models*. Chapman Hall/CRC.

Z. S. Khan, A. Atangana, Mathematical modeling and analysis of COVID-19: A study of new variant omicron, *Physica A: Statistical Mechanics and Its Applications* 599.

J. Ssebuliba, J. N. Nakakawa, A. Ssematimba, J. Y. T. Mugisha, Mathematical modelling of COVID-19 transmission dynamics in a partially comorbid community, *Journal of Theoretical Biology*. https://doi.org/10.1016/j.jtbi.2022.111235.

WHO. COVID-19 vaccines/COVID-19 Vaccines Advice, COVID-19 advice for the public: Getting vaccinated. https://www.who.int/emergencies/diseases/novel-coronavirus-2019/covid-19-vaccines/advice.

Yong, B., Hoseana, J., & Owen, L. (2022). From pandemic to a new normal: Strategies to optimise governmental interventions in Indonesia based on an sveir-type mathematical model. *Infectious Disease Modelling*, 7, 346–363. https://doi.org/10.1016/j.idm.2022.06.004