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Modeling of COVID-19 spread with self-isolation at home and hospitalized classes

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A R T I C L E   I N F O

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A B S T R A C T

This work examines the impacts of self-isolation and hospitalization on the population dynamics of the Corona-Virus Disease. We developed a new nonlinear deterministic model eight classes compartment, with self-isolation and hospitalized being the most effective tools. There are (Susceptible $S(t)$, Exposed $E(t)$, Asymptomatic infected $I_A(t)$, Symptomatic infected $I_S(t)$, Self-isolation $T_d(t)$, Hospitalized $T_H(t)$, Healed $H(t)$, and Susceptible individuals previously infected $I_S(t)$). The expression of basic reproduction number $R_0$ comes from the next-generation matrix method. With suitably constructed Lyapunov functions, the global asymptotic stability of the non-endemic equilibria $\Sigma_0$ for $R_0 < 1$ and that of endemic equilibria $\Sigma_1$ for $R_0 > 1$ are established. The computed value of $R_0 = 3.120277403$ proves the endemic level of the epidemic. The outbreak will lessen if a policy is enforced like self-isolation and hospitalization. This is related to those policies that can reduce the number of direct contacts between infected and susceptible individuals or waning immunity individuals. Various simulations are presented to appreciate self-isolation at home and hospitalized strategies if applied sensibly. By performing a global sensitivity analysis, we can obtain parameter values that affect the model through a combination of Latin Hypercube Sampling and Partial Rating Correlation Coefficients methods to determine the parameters that affect the number of reproductions and the increase in the number of COVID cases. The results obtained show that the rate of self-isolation at home and the rate of hospitalization have a negative relationship. On the other hand, infections will decrease when the two parameters increase. From the sensitivity of the results, we formulate a control model using optimal control theory by considering two control variables. The result shows that the control strategies minimize the spread of the COVID infection in the population.

Introduction

In a period not reaching half a decade, the world is shocked by the increasing number of human deaths due to Corona Virus Disease (COVID-19). The transmission of COVID-19 through direct contact to human-to-human with contaminated surfaces [1,2] and the droplets from infectious agents [1–6]. In the spreads of COVID-19, the incubation period of those viruses are approximately 2–14 days [2,7–9]. The Varying of the COVID-19 symptoms is from cough, core throat, diarrhea, pneumonia for severe cases, fever or chills, muscle or body aches, runny nose or stuffy nose, loss of taste or smell, difficulty breathing, fatigue, vomiting or nausea, and headache [2,9,10]. Researchers worldwide have given various responses due to pandemic COVID-19 with the severity of the disease [5].

Mathematical epidemiology models play a vital role in studying various physical and biological phenomena and their mechanisms. Mathematical models can help solve problems in the spread of infectious diseases. These include Tuberculosis (TB) [11], Malaria [12,13], Zika [14], Measles [15,16], Influenza [17], Dengue Fever [18,19], Ebola [20,21], Rabies [22], HIV/AIDS [23], HIV–TB [24], Hepatitis B virus [25] as well as COVID-19 [4,26–31]. The study predicts future dynamics develops control and cure procedures to be applied in real cases to reduce the spread of disease. Moreover, mathematically, the complex dynamics of epidemic problems can be studied its behavior by using the compartment model, where there are several sub-populations based on the status of each individual to the disease. The SEIR epidemic model have been studied by many researchers with various approaches, for
example ordinary-differential equations [29,32], discrete [33], stochastics [34], reaction-diffusion [35], fractional-order [8,36,37], and many more [38–40].

Regarding the implications of the COVID-19 outbreak for public health, a self-isolation policy has been implemented to reduce the various consequences of contact between infectious and susceptible individuals [41–43]. Notwithstanding the self-isolation policy, any infected individual with clinical symptoms requires hospitalization [1, 31,44,45]. Therefore, the combination of self-isolation and hospitalized policies is more effective than just one of them. This matter has received attention from several researchers in their works. For instance, in [46], Khan et al. considered the factor of the infection human when visiting the market for purchasing food at the seafood market. The simulation by the authors used the actual data of COVID-19 of mainland China. Next, based on the actual data in India, Biswas et al. in [7] show that increasing the proportion of people keeping a safe distance from each other can reduce the outbreak size and peak prevalence. Finally, a model to predict the COVID-19 outbreaks in India has been considered [47]. Recently, still using the actual data in India, Senapati et al. in [48] investigated the importance of eradicating the disease effectively with increasing the strength of the interventions. A mathematical model approach and the simulation with the actual data from Pakistan involving the data for fatalities, the number of cases, the data for hospitalizations to predict the trend of COVID-19 is worked by Peter et al. in [49]. Next, by different models and the actual prevalence of COVID-19 cases in Pakistan, Memon et al. found that the most effective strategy in fighting a pandemic is to control contact between individuals and extend the quarantine period [50]. Further, the analysis and simulation used the data from Nigeria. Finally, Musa et al. recommend that awareness programs and timely hospitalization of active cases are needed due to effective and efficient control of the COVID-19 pandemic [51].

Several studies of the COVID-19 model with waning immunity have been analyzed; Zargarra et al. Inayaturohm et al. and Diagne et al. concluded that vaccination, isolation, and treatment is an effective strategy to reduce COVID-19 transmission [52–54]. Sasmita et al. used a mathematical model to pattern the progression of COVID-19 in Indonesia. Furthermore [55], Abioye et al. used optimal control of COVID-19 model in Nigeria [56]. Finally, a scenario of reinfection of COVID-19 in Malaysia by Zamir et al. [57].

While this paper aims to consider the waning immunity of humans, it is based on the reality that the reinfection in the case of COVID-19 may cause a more severe impact [58]. Furthermore, the decrease in the individuals immunity may be caused reinfection to occur [59]. As such, self-isolation should be a special consideration in a mathematical model due to the COVID-19 outbreaks. Very recently, Anggriani et al. suggested a new model able to predict the cases of COVID in West Java, Indonesia. The authors considered the aspects of decreasing immunity in humans to emerge a new compartment thus. For detail, see [4]. Their model is different when compared with the previous models. Nevertheless, their model only considers the quarantined or isolation class and does not separate the hospitalized class. In addition, to avoid misperceptions, the term isolation in this paper means self-isolation at home. We extend the model given in [4] by incorporating self-isolation and hospitalized classes to study the dynamics behavior of COVID-19. This paper consists of five sections. The following section studied the model Mathematics, the transmission model of COVID-19. In Section Mathematical Analysis, we discuss fundamental mathematical substances in a biologically feasible region and the basic reproduction number then analyze the stability of the equilibria. Furthermore, we find conditions for the local and global behaviors of the possible equilibria of the proposed model. We also provide numerical simulations to verify and support our analytical findings with sensitivity analysis and Optimal control problems in Section Numerical Simulation. The concluding remarks are presented in the final section.

\[\chi(t) = S_C(t) + E(t) + I_A(t) + A_S(t) + T_M(t) + T_H(t) + H(t) + H_C(t)\]

We assume that the asymptomatic reported infected individuals will undergo self-isolation. Next, reported infected individuals but symptomatic will move to the hospital. Thus, only \(I_A(t)\) or \(A_S(t)\) are the infectious individuals that will spread the diseases. The transmission of COVID-19 is illustrated in Fig. 1, with description of the parameters given in Table 1. The model compose of the following set of nonlinear differential equations:

\[
\begin{align*}
\frac{dS_C}{dt} &= -\eta (S_C(t) + a_1A_S(t) + a_2A_S(t)) - \mu_0S_C(t) \\
\frac{dE}{dt} &= (\alpha_1A_S(t) + \alpha_2A_S(t)) - \beta E(t) - \mu_1E(t) \\
\frac{dI_A}{dt} &= \rho E(t) - \delta_1I_A(t) - \omega_1I_A(t) - \mu_1I_A(t) \\
\frac{dA_S}{dt} &= (1 - \rho)E(t) + \alpha_1A_S(t)H_C + \alpha_2A_S(t)H_C + \phi T_M - \omega_2A_S(t) - \delta_2A_S(t) - \mu_1A_S(t) \\
\frac{dT_M}{dt} &= \omega_1I_A(t) - \sigma_1T_M - \phi T_M - \mu_1T_M \\
\frac{dT_H}{dt} &= \omega_2A_S(t) - \sigma_2T_H - \mu_1T_H \\
\frac{dA_S}{dt} &= \delta_1I_A(t) + \delta_2A_S(t) + \sigma_1T_M + \sigma_2T_H - \chi H(t) - \mu_0H(t) \\
\frac{dH}{dt} &= \delta_1I_A(t) + \phi T_M - \omega_2A_S(t) - \delta_2A_S(t) - \mu_0H(t) \\
\end{align*}
\]

where \(S_C(0) \geq 0, E(0) \geq 0, I_A(0) \geq 0, A_S(0) \geq 0, T_M(0) \geq 0, T_H(0) \geq 0, H(0) \geq 0, H_C(0) \geq 0\) as the initial conditions.

 Mathematical analysis

Positivity and boundedness

Lemma 1. If the initial values \(Y(0) \geq 0\), where \(Y(t) = (S_C(t), E(t), I_A(t), A_S(t), T_M(t), T_H(t), H(t), H_C(t))\). Then, the solutions of system (1) are non-negative for all \(t > 0\). Further,

\[
\lim_{t \to \infty} \chi(t) \leq \frac{\eta}{\chi},
\]

with \(\chi(t) = S_C(t) + E(t) + I_A(t) + A_S(t) + T_M(t) + T_H(t) + H(t) + H_C(t)\).
Similarly, it can be shown that $S$ is bounded for all $t$. If $0 < S_C(0) \leq \chi(t), 0 < E(0) \leq \chi(t), 0 < I_A(0) \leq \chi(t), 0 < A_S(0) \leq \chi(t), 0 < T_S(0) \leq \chi(t), 0 < T_H(0) \leq \chi(t), 0 < H(0) \leq \chi(t), 0 < H_C(0) \leq \chi(t)$. If all the equations in system (1) are added and choose $\mu_0 = \mu_1 = \kappa$, we get

$$\frac{d\chi}{dt} = \eta - \kappa \chi. \quad \text{so} \quad \lim_{t \to \infty} \chi(t) \leq \frac{\eta}{\kappa}. \quad \Box$$

**Lemma 2.** All solutions of the system (1) are bounded for all $t \in [0, t_0]$. 

**Proof.** Since $\chi(t) = S_C(t) + E(t) + I_A(t) + A_S(t) + T_M(t) + T_H(t) + H(t) + H_C(t)$, and moreover we get

$$\frac{d\chi}{dt} + \kappa \chi = \eta - (\mu_0 - \kappa)(S_C + H + H_C) = \eta.$$ 

Choose $\kappa = \min\{\mu_0, \mu_1\}$, so we obtain that $\frac{d\chi}{dt} + \kappa \chi \leq \eta$. By the analytical process, then the result is

$$\chi(t) \leq \frac{\eta}{\kappa} (1 - e^{-\kappa t}) + \chi(0) e^{-\kappa t} \leq \frac{\eta}{\kappa}.$$ 

Consequently, all solutions of system (1) is bounded for all $t \in [0, t_0]$. \quad \Box

**Proof.** Motivated by Zhang et al. [6] and Khan et al. [46], we proved the positivity of solution of system (1). Consider

$$t_1 = \sup \{t > 0 : Y(t) > 0\}. \quad \text{So} \quad t_1 > 0. \quad \text{From the first equation in the system (1), we get}

$$\frac{dS_C}{dt} = \eta - \varpi S_C - \mu_S S_C, \quad \text{where} \quad \varpi = a_1 I_A + a_2 A_S.$$

It can be re-written as: Therefore,

$$\frac{d}{dt} \left( S_C(t) \exp \left( \int_0^t \varpi(r) dr + \mu_S t \right) \right) = \eta \exp \left( \int_0^t \varpi(r) dr + \mu_S t \right). \quad \text{(2)}$$

Hence,

$$S_C(t) \exp \left( \int_0^t \varpi(r) dr + \mu_S t \right) - S_C(0) = \eta \exp \left( \mu_S t + \int_0^u \varpi(v) dv \right) du. \quad \text{(3)}$$

So,

$$S_C(t) = S_C(0) \exp \left( - \int_0^t \varpi(r) dr - \mu_S t \right) + \exp \left( - \int_0^t \varpi(r) dr - \mu_S t \right) \times \int_0^t \eta \exp \left( \mu_S t + \int_0^u \varpi(v) dv \right) du > 0. \quad \text{(4)}$$

Similarly, it can be shown that $E(t), I_A(t), A_S(t), T_M(t), T_H(t), H(t)$, and $H_C(t)$ are positive for all $t > 0$. To show the other claim, note that $0 < S_C(0) \not\leq \chi(t), 0 < E(0) \not\leq \chi(t), 0 < I_A(0) \not\leq \chi(t), 0 < A_S(0) \not\leq \chi(t), 0 < T_S(0) \not\leq \chi(t), 0 < T_H(0) \not\leq \chi(t), 0 < H(0) \not\leq \chi(t), 0 < H_C(0) \leq \chi(t)$. If all the equations in system (1) are added and choose $\mu_0 = \mu_1 = \kappa$, we get

$$\frac{d\chi}{dt} = \eta - \kappa \chi. \quad \text{so} \quad \lim_{t \to \infty} \chi(t) \leq \frac{\eta}{\kappa}. \quad \Box$$

**Table 1** Parameters description.

| Parameters | Description | Units |
|------------|-------------|-------|
| $\eta$     | The recruitment rate of susceptible individuals | people × day$^{-1}$ |
| $a_1$      | The probability of transmission from asymptomatic infected individuals | (people × day)$^{-1}$ |
| $a_2$      | The probability rate of transmission from symptomatic infected individuals | day$^{-1}$ |
| $\mu_0$    | The natural mortality rate | day$^{-1}$ |
| $\mu_1$    | Natural death rate plus COVID-19 death rate | day$^{-1}$ |
| $\beta$    | The probability of exposed individuals become infected | day$^{-1}$ |
| $p$        | The proportion of exposed individuals become infected | day$^{-1}$ |
| $\delta_1$ | The natural recovery rate of infected asymptomatic individuals | day$^{-1}$ |
| $\delta_2$ | The natural recovery rate of infected symptomatic individuals | day$^{-1}$ |
| $\xi$      | The probability of recovered individuals become susceptible (waning immunity) | day$^{-1}$ |
| $\psi$     | The rate of asymptomatic infected individuals which undergo self-isolation at home | day$^{-1}$ |
| $\omega$   | The recruitment rate of susceptible individuals | people × day$^{-1}$ |
| $\alpha$   | The rate of asymptomatic infected individuals which undergo self-isolation at home | day$^{-1}$ |
| $\psi_1$   | The recovered rate of hospitalized | day$^{-1}$ |
| $\psi_2$   | The recovered rate of self-isolation at home | day$^{-1}$ |

**Fig. 2.** Dynamical Population each class: (a) Population of $S_C, H, H_C$ (b) Population of $E, I_A, A_S$. 

**Non-endemic equilibria and basic reproduction number**

The non-endemic equilibria of the COVID-19 disease model is obtained by setting $E = 0, I_A = 0, A_S = 0, T_M = 0$, and $T_H = 0$ then substituting it into system (1) to obtain:

$$\Xi_0 = \left( S_C^0, E^0, I_A^0, A_S^0, T_M^0, T_H^0, H^0, H_C^0 \right) = \left( \frac{\eta}{\mu_0}, 0, 0, 0, 0, 0, 0, 0 \right). \quad \text{(5)}$$
and the Basic Reproduction Number is given by

\[
F = \begin{bmatrix}
\frac{\eta p \rho s_1}{\mu_0(\beta + \mu_1)} & \frac{\eta p (1-p) s_2}{\mu_0(\beta + \mu_1)} & \frac{\eta p s_3}{\mu_0(\beta + \mu_1)} \\
0 & 0 & 0 \\
0 & 0 & 0 \\
0 & 0 & 0 \\
\end{bmatrix} \quad \text{(6)}
\]

\[
V = \begin{bmatrix}
\beta + \mu_1 & 0 & 0 \\
-\rho \beta & \delta_1 + \omega_1 + \mu_1 & 0 \\
-1(1-p)\beta & 0 & \delta_2 + \omega_2 + \mu_1 \\
\end{bmatrix} \quad \text{(7)}
\]

To determine the Basic Reproduction Number \(R_0\) of the system (1), we can analyze the eigenvalues of \(FV^{-1}\). The matrix of \(FV^{-1}\) is as follows.

\[
FV^{-1} = \begin{bmatrix}
\frac{\eta p \rho s_1}{\mu_0(\beta + \mu_1)(\delta_1 + \omega_1 + \mu_1)} & \frac{\eta p (1-p) s_2}{\mu_0(\beta + \mu_1)(\delta_2 + \omega_2 + \mu_1)} & \frac{\eta p s_3}{\mu_0(\beta + \mu_1)(\delta_2 + \omega_2 + \mu_1)} \\
0 & 0 & 0 \\
0 & 0 & 0 \\
0 & 0 & 0 \\
\end{bmatrix} \quad \text{(8)}
\]

Next, by analyzing the eigenvalues of \(FV^{-1}\) in (8), we obtain that the spectral radius is

\[
R_0 = R_{\text{asymptomatic}} + R_{\text{symptomatic}} \quad \text{(9)}
\]

where

\[
R_{\text{asymptomatic}} = \frac{\eta \rho s_1}{\mu_0(\beta + \mu_1)(\delta_1 + \omega_1 + \mu_1)}
\]

\[
R_{\text{symptomatic}} = \frac{\eta p (1-p) s_2}{\mu_0(\beta + \mu_1)(\delta_2 + \omega_2 + \mu_1)}
\]

\(R_0\) is important in epidemiology, defined as the number of secondary infections caused by one primary infection in a population [4]. It can be seen that two components establish \(R_0\) in (9). The first component is \(R_{\text{asymptomatic}}\) which illustrates the infection in \(S_C\) due to contact with asymptomatic infectious humans. Next, the second component is \(R_{\text{symptomatic}}\) which illustrates a contact directly with symptomatic infectious humans led to infection in \(S_C\).

Endemic equilibria

By setting the right hand side equals to zero of system (1), we obtained:

\[
E^* = \frac{a_1 I_1^* S_1^* + a_2 A_2^* S_2^*}{\beta + \mu_1}
\]

\[
I_1^* = \frac{\rho \beta E^*}{\delta_1 + \omega_1 + \mu_1},
\]

\[
A_2^* = \frac{(1-p)\beta E^* + a_1 I_1^* H_2^* + \rho T_2^*}{a_2 H_2^* + (\delta_2 + \omega_2 + \mu_1)}.
\]

The non-endemic equilibria of system (1) is locally asymptotically stable whenever it exists.

\[
J = \begin{bmatrix}
\Phi_1 & 0 & -\chi_1 & 0 & 0 & 0 \\
-\rho & 0 & -\chi_2 & 0 & 0 & 0 \\
0 & -\chi_2 & 0 & 0 & 0 & 0 \\
(1-p) \beta & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 \\
\end{bmatrix} \quad \text{(10)}
\]

where

\[
\Phi_1 = a_1 I_1 + a_2 A_2, \quad \Phi_2 = a_1 I_1 + a_2 A_2 + \mu_0, \quad \text{and} \quad \Phi_3 = a_2 H_2 - (\delta_2 + \omega_2 + \mu_1).
\]

Theorem 3. The non-endemic equilibria of system (1) is locally asymptotically stable whenever it exists.

Proof. Substituting the non-endemic equilibria \(E_0\) from (5) into the Jacobian matrix in (11), we get:

\[
J_{E_0} = \begin{bmatrix}
\rho & 0 & -\chi_2 & 0 & 0 & 0 \\
0 & -\chi_2 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 \\
(1-p) \beta & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 \\
\end{bmatrix} \quad \text{(12)}
\]

The characteristics of the polynomial is

\[
D(\lambda) = (\lambda + \mu_0)(\lambda + (\sigma_2 + \mu_1))(\lambda + (\xi + \mu_0))\lambda \quad \text{(13)}
\]

where \(D(\lambda) = g_0 \lambda^2 + g_1 \lambda + g_3 \lambda^2 + g_2 \lambda + g_4\), where:

\[
g_0 = 1.
\]
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Fig. 4. Simulation of the effects of asymptomatic infected parameter ($\alpha_i$) with respect to time ($t$) for each class (a) $E$, (b) $I_s$, (c) $A_s$, (d) $S_C$, and (e) $H_C$.

$g_1 = (\beta + \mu_1)(\delta_1 + \omega_1 + \mu_1) + (\delta_2 + \omega_2 + \mu_1) + (\sigma_1 + \varphi + \mu_1)$,

$g_2 = (\beta + \mu_1)(\delta_1 + \omega_1 + \mu_1) \left( 1 - R_{\text{asymptotic}} \right)$
$+ (\beta + \mu_1)(\delta_2 + \omega_2 + \mu_1) \left( 1 - R_{\text{symptomatic}} \right)$
$+ (\sigma_1 + \varphi + \mu_1)((\beta + \mu_1) + (\delta_1 + \omega_1 + \mu_1))$
$+ (\delta_2 + \omega_2 + \mu_1)((\delta_1 + \omega_1 + \mu_1) + (\sigma_1 + \varphi + \mu_1)).$

$g_3 = (\beta + \mu_1)(\delta_1 + \omega_1 + \mu_1)((\delta_2 + \omega_2 + \mu_1) + (\sigma_1 + \varphi + \mu_1))$
$\times \left( 1 - R_{\text{asymptotic}} \right)$
$+ (\delta_2 + \omega_2 + \mu_1)((\sigma_1 + \varphi + \mu_1)((\beta + \mu_1) + (\delta_1 + \omega_1 + \mu_1)))$
$\times \left( 1 - R_{\text{symptomatic}} \right),$

$g_4 = (\beta + \mu_1)(\delta_1 + \omega_1 + \mu_1)(\delta_2 + \omega_2 + \mu_1)(\sigma_1 + \varphi + \mu_1)$
$\frac{\delta_1}{\omega_1 \varphi}$
$\times \left[ (1 - R_0) + R_{\text{symptomatic}} \right].$

If and only if $R_0 < 1$ then we get that the values of $g_i, i = 0, 1, 2, 3, 4$ are positive.

The eigenvalues of the variational matrix $J_{\Sigma_0}$ are $-\mu_0 - \mu_1$, $-(\delta_1 + \mu_1)$, $-(\sigma_2 + \mu_1)$, $-\lambda_1$, $-\lambda_2$, $-\lambda_3$, and $-\lambda_4$. Further, $\Re(\lambda_i) < 0, i = 1, \ldots, 4$ so that the non-endemic equilibria is locally asymptotically stable whenever it exists. Numerically, the analytical results can be seen in the Appendix A. Based on these results, consistently, we use the parameter values in Table 2, except $\omega_1 = \omega_2 = 0.9771$, obtained the real parts of eigenvalues are negative. □

Local stability of the endemic equilibria

Theorem 4. The endemic equilibria $\Sigma_*$ of the system (1) is locally asymptotically stable whenever it exists.
Fig. 5. Simulation of the effects of symptomatic infected parameter ($\alpha_2$) with respect to time ($t$) for each class (a) $E$, (b) $I_A$, (c) $A_S$, (d) $S_C$, and (e) $H_C$.

Proof. Substituting the non-endemic equilibria $\Sigma_0$ into the Jacobian matrix in (11), we get:

$$J_{\Sigma_0} = \begin{bmatrix}
-\phi_2 & 0 & -S_0^c \alpha_1 & -S_0^c \alpha_2 & 0 & 0 & 0 & 0 \\
\phi_3 & -(\beta + \mu_1) & S_0^c \alpha_1 & S_0^c \alpha_2 & 0 & 0 & 0 & 0 \\
0 & \beta_2 & -(-\delta_2 + \alpha_1 + \mu_1) & 0 & 0 & 0 & 0 & 0 \\
0 & (1-p)\beta & a_1 H_0^c & \psi & 0 & 0 & \phi_1 & 0 \\
0 & 0 & a_1 & 0 & -(\delta_1 + \phi + \mu_1) & 0 & 0 & 0 \\
0 & 0 & a_1 & 0 & 0 & -(\sigma_2 + \mu_1) & 0 & 0 \\
0 & 0 & \delta_1 & \delta_2 & \sigma_1 & \sigma_2 & -\zeta & -\phi_2 \\
0 & 0 & -a_2 H_0^c & -a_2 H_0^c & 0 & 0 & \zeta & -\phi_2 \\
\end{bmatrix}, \quad (14)$$

where $\phi_1 = a_1 I_A^* + a_2 A_S^*$, $\phi_2 = a_1 I_A^* + a_2 A_S^* + \mu_1$, and $\phi_3 = a_2 H_0^c - (\delta_2 + \omega_2 + \mu_2)$.

From the matrix $J_{\Sigma_0}$ in (14), we get $\Re(\lambda_i) < 0$, $i = 1, 2, \ldots, 8$ so that the endemic equilibria is locally asymptotically stable whenever it exists. Numerically, the analytical results can be seen in the Appendix B. Based on these results, consistently, we use the parameter values in Table 2, obtained the real parts of eigenvalues are negative. □

Global stability of the non-endemic equilibria

Lemma 5. If $\max \left\{ \frac{\alpha_1 S_0^c}{\beta_1}, \frac{\alpha_2 S_0^c}{\beta_1} \right\} < 1$ and $R_0 < 1$, the non-endemic equilibria $\Sigma_0$ of system (1) is globally asymptotically stable.
Using the relation between the geometric means and arithmetic means, \( \delta \frac{dV}{d\tau} = \frac{S_C}{S_C^*} \frac{dE}{d\tau} + \frac{1}{2} \frac{dA_1}{d\tau} + \frac{dA_2}{d\tau} + \frac{dT}{d\tau} + \frac{dH}{d\tau} + \frac{dH_C}{d\tau} \),

Differentiating with respect to time yields

\[
\frac{dV(t)}{dt} = \left( 1 - \frac{S_C}{S_C^*} \right) \frac{dE}{dt} + \frac{1}{2} \frac{dA_1}{dt} + \frac{dA_2}{dt} + \frac{dT}{dt} + \frac{dH}{dt} + \frac{dH_C}{dt}
\]

\[
= \left( 1 - \frac{S_C}{S_C^*} \right) (\eta - (a_1 I_A S_C + a_2 A_2 S_C) - \mu_0 S_C) + S_C(a_1 I_A + a_2 A_2) - \beta + \mu_1) E
\]

\[
+ (\beta + (1 - \rho) + (1 - \mu_1) - \beta + \mu_1) E - \mu_1 I_A + A_1
\]

\[
= S_C(a_1 I_A + a_2 A_2) + \beta - \mu_1 I_A + A_1 \frac{\sigma_1}{\sigma_1 - 1} A_S
\]

\[
+ (\beta + \mu_1) \left( \frac{a_1}{\mu_1 S_C} + \frac{a_2}{\mu_2 S_C} - 1 \right) A_S
\]

\[
+ (\beta + \mu_1) \left( \frac{a_1}{\mu_1 S_C} + \frac{a_2}{\mu_2 S_C} - 1 \right) A_S
\]

\[
+ (\beta + \mu_1) \left( \frac{a_1}{\mu_1 S_C} + \frac{a_2}{\mu_2 S_C} - 1 \right) A_S
\]

\[
+ (\beta + \mu_1) \left( \frac{a_1}{\mu_1 S_C} + \frac{a_2}{\mu_2 S_C} - 1 \right) A_S
\]

\[
+ (\beta + \mu_1) (R_0 - 1) E - \mu_1 (T_M + T_H) - \rho_0 (H + H_C).
\]

\[
(15)
\]

The value of \( \frac{dV}{dt} \) < 0 if (i) \( R_0 < 1 \) and (ii) \( \max \left( \frac{a_1 S_C}{\mu_1}, \frac{a_2 S_C}{\mu_2} \right) < 1 \).

Using the relation between the geometric means and arithmetic means, we confirm that \( \frac{dV}{dt} \leq 0 \) and fulfilled the equality only at \( \Sigma_0 \). Thus, the non-endemic equilibria \( \Sigma_0 \) is globally asymptotically stable if \( R_0 < 1 \).

Global stability of the endemic equilibria

**Lemma 6.** The endemic equilibria \( \Sigma \) of the system (1) exists and globally asymptotically stable if \( R_0 > 1 \).
Fig. 7. Simulation of the effects of self-isolation parameter ($\omega_t$) with respect to time ($t$) for each class (a) $E$, (b) $I_A$, (c) $A_S$, (d) $T_M$, (e) $S_C$, and (f) $H_C$.

\[
\frac{dV_E}{dt} = -\frac{H_S^*}{H_C^*} \left( \delta_1 I_A + \delta_2 A_S + \sigma_1 T_M + \sigma_2 T_H \right) - \frac{H}{H_T} (\delta_1 I_A^* + \delta_2 A_S^* + \sigma_1 T_M^* + \sigma_2 T_H^*).
\]

\[
\frac{dV_{I_A}}{dt} = \left( 1 - \frac{H_S^*}{H_C^*} \right) [\xi H - \alpha_1 I_A H_C - \alpha_2 A_S H_C - \mu_0 H_C]
\]

\[
= \frac{H H_C^*}{H_C} (a_1 I_A^* + a_2 A_S^* + \mu_0) + H_C (a_1 I_A + a_2 A_S + \mu_0)
\]

\[- \frac{H H_C^*}{H^* H_C^*} (a_1 I_A^* + a_2 A_S^* + \mu_0)]
\]

\[
\frac{dV_{A_S}}{dt} = \left( \frac{\delta_1}{\sigma_1} \right) \left( \frac{a_1 I_A^* + a_2 A_S^* + \mu_0}{\sigma_1} \right) + \frac{\sigma_2}{\sigma_1} \left( \frac{a_1 I_A + a_2 A_S + \mu_0}{\sigma_1} \right)
\]

Now

\[
\frac{dV_{E}(t)}{dt} = a_1 I_A^* S_C \left( 2 - \frac{S_C}{S_C} - \frac{E^*}{E^*} \frac{I_A S_C}{E^* T_A S_C} \right)
\]

\[+ a_2 A_S^* S_C \left( 2 - \frac{S_C}{S_C} - \frac{E^*}{E^*} \frac{A_S S_C}{E^* A_S^* S_C} \right)
\]

\[+ \mu_0 S_C \left( 2 - \frac{S_C}{S_C} - \frac{S_C}{S_C} \right) + P_1 - P_2,
\]

where

\[P_1 = S_C^* (a_1 I_A + a_2 A_S) + \rho \beta (E + E^*) + (1 - \rho) \beta (E + E^*)
\]

\[+ \alpha_1 (I_A H_C + I_A^* H_C^*)
\]

\[+ \alpha_2 (A_S H_C + A_S^* H_C^*) + \varphi (T_M + T_M^*) + \omega_1 (I_A + I_A^*)
\]

\[+ \omega_2 (A_S + A_S^*) + \delta_1 (I_A + I_A^*)
\]

\[+ \delta_2 (A_S + A_S^*) + \sigma_1 (T_M + T_M^*) + \sigma_2 (T_H + T_H^*)
\]

\[+ H_C^* (a_1 I_A^* + a_2 A_S^* + \mu_0)]
\]

\[P_2 = \rho \beta \frac{E I_A^*}{T_A} + \rho \beta \frac{E I_A}{T_A} + \frac{A_S}{A_S^*} [(1 - \rho) \beta E^* + H_C^* (a_1 I_A^* + a_2 A_S^*) + \varphi T_M^*]
\]

\[+ \frac{A_S}{A_S} [(1 - \rho) \beta E + H_C (a_1 I_A + a_2 A_S) + \varphi T_M]
\]

\[+ \omega_1 \frac{I_A T_M}{T_M} + \omega_2 \frac{I_A^* T_M}{T_M} + \omega_2 \frac{A_S T_H}{T_H}
\]

\[+ \omega_2 \frac{A_S^* T_H}{T_H} + H^* (\delta_1 I_A + \delta_2 A_S + \sigma_1 T_M + \sigma_2 T_H)
\]
Fig. 8. Simulation of the effects of self-isolation parameter ($\omega$) with respect to time ($t$) for each class (a) $E$, (b) $I$, (c) $A$, (d) $T$, (e) $S$, and (f) $H$.

Fig. 9. The relationship among $\omega_0$, $\omega_1$, and $\omega_2$. Using the relation of geometric means and arithmetic means, we confirm that $\frac{dV}{dt} \leq 0$ and fulfilled the equality only at $\Sigma_\ast$. Thus, the endemic equilibria $\Sigma_\ast$ is globally asymptotically stable.

**Numerical simulation**

In this present section, we perform numerical simulations for the model to support the analysis results in the previous section. From the parameter values in Table 2 and initial conditions in Table 3, we solved model (1) and applied the Fourth-Order Runge–Kutta (RK-4). One of the most popular numerical approaches is the RK-4 Method because of its accuracy, precision, program efficiency.

RK-4 method is one of the most popular numerical approach due to the accuracy, the stability, the efficiency, to program. The RK-4 method has a better performance in terms of computational speed, stability, and provides a more accurate solution [60,61].
susceptible classes. Exposed, Asymptomatic, Symptomatic, previously infected human, and their increases very fast. This affects the dynamics of the populations in the infection in the population led to the numbers of total infected individuals. \( A \) takes effect in the behavior of the population for each class: self-isolation and hospitalized classes, subsequently decreases. In addition, the individuals of self-isolation at home class also increases. \( T \) infected individuals. \( I \) the numbers of healed individuals, \( H \) increase in hospitalized individuals. \( S \) individuals and the asymptomatic infected individuals became the infected individuals \( T \) as the self-isolation at home \( H \), and the hospitalized \( H \) as well as the self-isolation at home \( T \).

Nevertheless, after the 50th day, the exposed \( E \) and asymptomatic infected individuals \( A \) decreased, this happens because the exposed individuals and the asymptomatic infected individuals became the healed individuals and some of them are self-isolating at home. While number of symptomatic individuals \( A \) have declined due to an increase in hospitalized individuals \( T \). Moreover, this process led to the numbers of healed individuals \( H \) and susceptible that previously infected individuals \( H \) have increased.

Fig. 3 illustrates the behavior of self-isolation individuals \( T \) and the hospitalized individuals \( T \). The number of individuals in hospitalized class increases as the asymptomatic infection increases. In addition, the individuals of self-isolation at home class also increases. The peak occurs at 60 days and 70 days respectively, for self-isolation and hospitalized classes, subsequently decreases.

The changes in the parameter value of rate of asymptomatic infection take effect in the behavior of the population for each class: \( E, I, A, H, S \) are presented in Fig. 4. If the value of parameter \( a \) is increased, within the range \([1.272 \times 10^{-7}, 1.272 \times 10^{-6}]\), the number of infected individuals can be increases. Increased the asymptomatic infection in the population led to the numbers of total infected increases very fast. This affects the dynamics of the populations in the Exposed, Asymptomatic, Symptomatic, previously infected human, and susceptible classes.

Next, to analyze the effects of parameter \( a \), related to the symptomatic infection of humans, we used the parameter values and initial conditions, respectively, in Tables 2 and 3, except for \( a \), within the range \([7.478 \times 10^{-10}, 7.478 \times 10^{-7}]\). Significantly, every \( a \) make an impact for the change in the population size of each class: \( E, I, A, H, S \), and \( T \). For the graphic of the behavior of this case, see Fig. 5.

Furthermore, in Fig. 6, we showed the relationship among \( R \), \( a \), and \( a \). As the values of \( a \) and \( a \) increase at the time, the basic reproduction number also increases sharply. This shows that increasing the level of asymptomatic infected human and the rate of symptomatic infected human has a significant effect for increase the numbers of COVID-19 infected.

### The effect of self-isolation

In this subsection, we study the effect of parameter \( a \) due to self-isolation at home policy. We choose \( a = 0.01, 0.05, 0.13266, 0.2 \) and consistently use the values in Tables 2 and 3. Increased the self-isolation at home led to the population size of the \( E, I, A, H, S \) classes be reduced. This change is due to reduced contact between asymptomatic infected individuals \( I \) class) and susceptible individuals (respectively, \( S \) and \( H \) classes) be decreases. So the policy of self-isolation at home plays a role to eliminate the number of infection cases. For detail of this behavior are performed in Fig. 7.

### The effect of hospitalized

To study the implications of implementing hospital care policies, we investigate the effect of parameter \( a \). Selected value of \( a \) within the range \([0.091, 1]\). Increased the value of hospitalized parameter \( a \) causing the peak of the Exposed \( E \), Asymptomatic infected \( A \), and Symptomatic infected \( A \) individuals are decreases. Meanwhile, individuals of Symptomatic infected which did care in hospital caused the population in the hospitalized class \( T \) increase. This show that the policy to increase the intensity of hospitalized may reduce the spread of COVID-19. The changes of the value of hospitalized parameters affect the population of each class: \( E, I, A, H, S \) are presented in Fig. 8.

Furthermore, we showed the relationship among \( R \), \( a \), and \( a \). As the values of \( a \) and \( a \) increase at the time, the basic reproduction number decreases sharply. Hence, the numbers of COVID-19 cases can be reduced due to increase the level of self-isolation at home and the rate of hospitalization. For detailed dynamics, see Fig. 9.

### Partial rank correlation coefficient

In this section, we analyze the global sensitivity analysis using a sample from Latin Hypercube Sampling (LHS). We then use the Partial Rank Correlation Coefficient (PRCC) to determine the most influential and significant model parameters. Finally, all parameters were measured against the increase in infections. The results of the sensitivity analysis are given in Fig. 10.

Fig. 10 shows that the influential parameter for all time is \( a \). This parameter has negative relationship. If the value of \( a \) increase, the number of infections decrease. It means self isolation is an effective solution to decrease infection. The other parameter, \( a \) also has negative relationship, it means hospitalize also decrease the infection.

### Optimal control

In this section, We used optimal control for the Asymptomatic infected individuals \( I \) and the Symptomatic infected individuals \( A \). \( u = \) the treatment to the Asymptomatic infected individuals \( I \) and \( v = \) the treatment to the Symptomatic infected individuals \( A \). These cases have been decided to evaluate whether the combination of treatment control can better affect both infected individuals. The objective functional as follow

\[
J(u, v) = \int_0^T (TI + Lu^2 + Zv^2 + YAv) \, dt
\]
Fig. 11. Dynamical population for each compartments (a) without control, (b) with control.

Fig. 12. Comparison between population with control \( w_c \) and without control \( n_c \) for (a) Asymptomatic infected individuals \( I_A \), (b) Symptomatic infected individuals \( A_S \).

Fig. 13. Comparison between control \( u \) and \( v \).

\[ \frac{dE}{dt} = (\alpha_1 I_A(t)S_C(t) + \alpha_2 A_S(t)S_C(t)) - \beta E(t) - \mu_1 E(t) \]
\[ \frac{dI_A}{dt} = p\beta E(t) - u(t)I_A(t) - \omega_1 I_A(t) - \mu_1 I_A(t) \]
\[ \frac{dA_S}{dt} = (1 - p)\beta E(t) + \alpha_1 I_A(t)H_C(t) + \alpha_2 A_S(t)H_C(t) + \phi T_M(t) - \omega_2 A_S(t) - v(t)I_A(t) - \mu_1 A_S(t) \]
\[ \frac{dT_M}{dt} = \omega_1 I_A(t) - \sigma_1 T_M(t) - \phi T_M(t) - \mu_1 T_M(t) \]
\[ \frac{dT_H}{dt} = \omega_2 A_S(t) - \sigma_2 T_H(t) - \mu_1 T_H(t) \]
\[ \frac{dH}{dt} = (u(t)I_A(t) + v(t)A_S(t) + \sigma_1 T_M(t) + \sigma_2 T_H(t) - \xi H(t) - \mu_0 H(t) \]
\[ \frac{dH_C}{dt} = \xi H(t) - \omega_1 I_A(t)H_C(t) - \omega_2 A_S(t)H_C(t) - \mu_0 H_C(t) \]

where \( S_C(t) \geq 0, E(t) \geq 0, I_A(t) \geq 0, A_S(t) \geq 0, T_M(t) \geq 0, T_H(t) \geq 0, H(t) \geq 0, H_C(t) \geq 0, 0 \leq u^*, v^* \leq 1 \) and \( 0 \leq t \leq t_f \).

Where \( t_f \) is the final time, and the coefficients \( T \) and \( Y \) are the weight. \( L \) and \( Z \) are the cost of treatments. The purpose of these control are to minimize the Asymptomatic infected individuals \( I_A \) and the Symptomatic infected individuals \( A_S \). We need to find \( u^*, v^* \) such that

\[ J(u^*, v^*) = \min(J(u, v)) \]

\[ s.t \]

\[ \frac{dS_C}{dt} = \eta - (\alpha_1 I_A(t)S_C(t) + \alpha_2 A_S(t)S_C(t)) - \mu_0 S_C(t) \]
The Co-State of the controls are given by
\[
\frac{d\lambda_i(t)}{dt} = -\lambda_i(t) \left(-\alpha_i I(t) - \alpha_i A_i(t) - \mu_i\right) - \lambda_i(t) \left(\alpha_i I(t) + \alpha_i A_i(t)\right)
\]
\[
\frac{d\lambda_2(t)}{dt} = -\lambda_2(t) \left(-\beta - \mu_2\right) - \lambda_2(t) \beta - \lambda_2(t)(1 - p)\beta
\]
\[
\frac{d\lambda_3(t)}{dt} = -T - \lambda_3(t)\alpha_i S_i(t) - \lambda_3(t)\alpha_i S_i(t) - \lambda_3(t) \left(u(t)\beta_i - \omega_i - \mu_i\right)
\]
\[
-\lambda_3(t)\alpha_i H_i(t) - \lambda_3(t)\alpha_i H_i(t) - \lambda_3(t)\beta_i - \lambda_3(t)(-\alpha_i H_i(t))
\]
\[
\frac{d\lambda_4(t)}{dt} = -Y - \lambda_4(t)(-\alpha_i S_i(t) - \lambda_4(t)S_i(t)
\]
\[
-\lambda_4(t)\alpha_i H_i(t) - \alpha_2 - \psi(t)\beta_i - \mu_i
\]
\[
-\lambda_4(t)\alpha_i H_i(t) - \lambda_4(t)\alpha_i H_i(t) - \lambda_4(t)(-\alpha_i H_i(t))
\]
\[
\frac{d\lambda_5(t)}{dt} = -\lambda_5(t)\psi - \lambda_5(t) \left(-\sigma_i - \psi - \mu_i\right) - \lambda_5(t)\sigma_i
\]
\[
\frac{d\lambda_6(t)}{dt} = -\lambda_6(t)\beta - \lambda_6(t)\beta - \lambda_6(t)(-\alpha_i H_i(t))
\]
\[
\frac{d\lambda_7(t)}{dt} = -\lambda_7(t)(-\xi - \mu_0) - \lambda_7(t)\xi
\]
\[
\frac{d\lambda_8(t)}{dt} = -\lambda_8(t) \left(\alpha_i I(t) + \alpha_i A_i(t)\right) - \lambda_8(t) \left(-\alpha_i I(t) - \alpha_i A_i(t) - \mu_i\right)
\]

Furthermore, the controls \(u^*\) and \(v^*\) are given by
\[
u^* = \min \left(1, \max \left(0, \frac{\lambda_5(t)\sigma_i I(t) - \lambda_5(t)\beta_i A_i(t)}{2Z}\right)\right)
\]
\[
u^* = \min \left(1, \max \left(0, \frac{\lambda_5(t)\sigma_i A_i(t) - \lambda_5(t)\beta_i A_i(t)}{2L}\right)\right)
\]

From Fig. 11, we can see the dynamics of the population. Exposed individuals (\(E_i\)), Asymptomatic infected individuals (\(I_i\)), Symptomatic infected individuals (\(A_i\)) decreased to 0 as \(t\) goes to positive infinity, that means the disease will be eliminated from the population. Furthermore, from Fig. 12 we can see the effect of treatment as control (\(\nu^*, \nu^*\)) of the model. Numerical simulation proves that control can reduce Asymptomatic infected individuals (\(I_i\)). Symptomatic infected individuals (\(A_i\)) faster than without control. Fig. 13 shows that at the beginning of the times, the control needs to be applied at the maximum level (\(\nu^* = 1\) and \(\nu^* = 1\)). But as time goes, the control can be relaxed to the minimum level depend on the weight of the control.

Discussion and conclusion

We have developed a mathematical model for the outbreaks of COVID-19 with self-isolation and hospitalized. The model is compartment-based in the form of a differential equations system. The population is divided into susceptible (\(S_i\)), Exposed (\(E_i\)), Asymptomatic infected (\(I_i\)), Symptomatic infected (\(A_i\)), Healed (\(H_i\)), Susceptible individuals that have been infected (\(H_i\)), Self-isolation at home (\(T_i\)), Hospitalized (\(T_i\)). By using the next-generation matrix, we obtained the Basic Reproduction Number (\(R_0\)), which crucial controlling the spread of COVID-19. Then the simulation results provide that the factor of self-isolation and hospitalized can affect the COVID-19 transmissions. The period self-isolation and hospitalized can reduce and slow the spreading of COVID-19. The results of this research can be used as a reference to do early prevention of the spread of COVID-19. We used the control in the form of treatment to the model using optimal control theory from the sensitivity results by considering two control variables. The results show that the spread of COVID-19 infection in the community can be minimized more quickly with control than without control.

Thus, model (1) constructed because it was motivated by various researches on the COVID-19 models that had been analyzed previously. Although the parameter values used for the simulation are data from several articles discussing the COVID-19 models, the model (1) can be a reference for the models of other disease as long as the behavior of disease is similar.

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Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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Appendix A. Proof of numerical analysis of non-endemic equilibria

The \(Re(\lambda_i)\) of eigenvalues are :
\[
Re(\lambda_1) = -0.00004214963119
\]
\[
Re(\lambda_2) = -0.00004214963119
\]
\[
Re(\lambda_3) = -0.00004214963000
\]
\[
Re(\lambda_4) = -0.08510000000000
\]
\[
Re(\lambda_5) = -1.32978858251587
\]
\[
Re(\lambda_6) = -0.76724286272831
\]
\[
Re(\lambda_7) = -0.85243436042583
\]
\[
Re(\lambda_8) = -0.00847045762997
\]

In addition to \(R_0 = 0.8151913483 < 1\).

Appendix B. Proof of numerical analysis of endemic equilibria

The \(Re(\lambda_i)\) of eigenvalues are :
\[
Re(\lambda_1) = -0.59902921816303
\]
\[
Re(\lambda_2) = -0.275777250098021
\]
\[
Re(\lambda_3) = -0.275777250098021
\]
\[
Re(\lambda_4) = -0.085183084583903
\]
\[
Re(\lambda_5) = -0.002094273918742
\]
\[
Re(\lambda_6) = -0.002094273918742
\]
\[
Re(\lambda_7) = -0.0016341291761530
\]
\[
Re(\lambda_8) = -0.000300205999203
\]

In addition to \(R_0 = 3.1202777403 > 1\).

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