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Mathematical Model of COVID-19 Transmission with Education Campaign and Treatment Through Quarantine

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Abstract In this paper, the SIQR-W mathematical model based on a system of ordinary differential equations is formulated to study the dynamics of COVID-19 transmission with health education campaigns and treatment through quarantine as controls against the epidemic. Boundedness and the existence of solutions for this system are shown. The basic reproduction number is computed using the next-generation matrix method. The equilibrium points of the model are determined and their stability is analyzed. Numerical simulation shows that when a health education campaign is efficient, the number of COVID-19-infected individuals decreases faster, implying that a health education campaign is vital in controlling the spread of COVID-19 disease.

1 Introduction

The novel coronavirus pandemic also known as COVID-19 is still posing a stressful time for humans around the globe. This infection first emerged in December of 2019, in Wuhan, a city in Central China [24, 25]. The COVID-19 infection has now spread to more than 99,3 % of countries worldwide. The countries or regions on the globe not yet infected with this infection are still at risk of this novel pandemic. According to the recent WHO measurements, more than 771 million COVID-19 cases are confirmed and over 6.9 million people have died globally due to this infection. Mostly, the severe cases and deaths due to this novel infection are reported in people with older age. Additionally, people with co-morbidities and any chronic respiratory disease that suppresses or compromises the immune system are at high risk from this infection [20]. According to Statista [21], its well known that the mortality rate is about 2%, the infection index is between 1.5 and 3.5, the critical cases is 6.1% and the mortality rate is 15% for persons with an age greater than 80 years.

The main transmission pattern known so far is through direct social contact of one human to an infectious person [5]. A person can also catch the virus via direct contact with contaminated surfaces or objects and during the inhalation of droplets discharge from the nose while spitting, coughing, or sneezing from both either symptomatic or asymptomatically infective humans [5]. Currently, the incubation period of the virus, referring to the time from exposure to the development of symptoms, is estimated to be in the range of 5 to 14 days. Some most common symptoms of COVID-19 are feverish body, tiredness, and dry cough. Whereas diarrhea, headache, pains, or aches in the throat with sore feeling, skin rashes or discoloration developing in legs or fingers, and feeling the loss of smell and taste will come under less common symptoms. Chest pain, high blood pressure, feeling difficulty in breathing, and loss of speech or movement are considered to be serious symptoms [1]. COVID-19 affects many people in various ways. The most infected population will evolve from low to moderate ailment. And those populations will recover without the need for hospital support. However, older individuals with underlying medical conditions

such as diabetes, cancer, cardiovascular disease, and chronic respiratory disease are more prone to developing severe symptoms. To prevent infection and to slow transmission of COVID-19, we want to do the following: Maintain a social distance of at least one distance between you and other people, and avoid unnecessary travel which will help us to stay away from large groups of the population, wash the hands frequently with hand sanitizer or with soap water, refrain from smoking and drinking since these activities may weaken your lungs and stay home if you feel unfit [1].

Various mathematical models have been proposed by researchers to evaluate the dynamical behavior and transmission of Coronavirus, which may aid in the prediction of future events and even the control of the disease, for example, [4, 6, 9, 10, 17, 18, 22]. From this studies, Tang et al. [22] considered, an SEIR-type mathematical model to estimate the transmission risk of COVID-19 and its implication. The study in [18] , formulated a model for novel corona virus disease 2019 (COVID-19) in Lagos, Nigeria and shown the effect of control measures, specifically the common social distancing, use of face mask and case detection on the dynamics of COVID-19.

Isolation and quarantine are two important measures by which asymptomatic or infected individuals could be detached from the population to stop further spread of the disease. Quarantine is generally used for seemingly healthy but possibly infected individuals, while isolation applies to already infected individuals. Quarantine was also applied as one of the effective intervention strategies during the SARS epidemic of 2002–2003 [2]. Several mathematical models have been formulated to study the role of quarantine and other intervention strategies in controlling the spread of infectious diseases like COVID-19. These models typically integrate quarantine as a control measure alongside other public health interventions such as vaccination, social distancing, and isolation of infected individuals. For example, Khan et.al, [13], formulated a fractional mathematical model for the dynamics of COVID-19 with quarantine and isolation. D.K Mamo [15], developed SHEIQRD corona virus pandemic spread model. He Identified that isolation of exposed and infected individuals, reduction of transmission, and stay-at-home return rate can mitigate COVID-19 pandemic. Atanu Bhattacharjee, et al. 2020, predict the trend of the COVID-19 pandemic up to June 2020 with the application of statistics and available data. He also asserts that the implementation of lockdowns and quarantine measures for individuals has played a significant role in reducing the risk of the epidemic's spread [3]. In contrast, another study conducted by Quian Li et al. in 2020 explores the impact of mass influenza vaccination models and public health interventions on COVID-19 epidemics [19]. A model in [16] describes the prediction and control measures with the help of a mathematical model. Also, they computed the equilibria and analyzed the stability control. At last, they conclude with some numerical simulations and present their results in graphs. The optimal control problem in an epidemic model typically involves identifying control measures, like vaccination or quarantine, that minimize a cost function representing the epidemic's overall impact on the population.

To control the outbreak of COVID-19, different governments are actively restricting the movement of people by imposing lockdowns, which may be known as one of the largest quarantines in history. Due to quarantine's economic, social, and psychological repercussions, it was necessary to search for alternative solutions to mitigate and reduce the spread of the epidemic. During the pandemic, governments, health organizations, and media outlets launched widespread efforts to inform the public about how the virus spreads, its symptoms, and the importance of preventive measures like wearing masks, social distancing, and vaccination. These campaigns also focused on debunking misinformation, highlighting the severity of the disease, and promoting health guidelines to reduce transmission. They emphasized the risks for vulnerable populations, like the elderly and those with underlying health conditions, and explained how individuals could protect themselves and others.

The novelty of this paper lies in its comprehensive exploration of a mathematical model based on a system of ordinary differential equations designed to capture the dynamics of COVID-19, with a specific focus on the influence of quarantine measures and the efficiency of health education campaigns as controls against the epidemic. The remaining part of the paper is organized as follows: Section 2 describes the proposed system; Section 3 represents the non-negativity and boundedness; Section 4 discusses the equilibria and basic reproduction number; Section 5 proves the stability of the possible steady; Section 6 demonstrates numerical simulation; and Section 7 concludes the paper.

2 Mathematical model

We formulate a mathematical model with a concentration of SARS-COV-2 in the population $N_W(t)$ which is denoted $W(t)$ and human population $N_H(t)$. This model satisfies the following assumptions:

- Human birth and natural death take place at different rates.
- Quarantined individuals do not shed the COVID-19 virus.
- All identified individuals with COVID-19 infection are quarantined.

$$\begin{cases} \frac{dS}{dt} = \Lambda - (1-\omega)\left(\beta_S I + \beta_W W\right) S - \mu_S S, \\ \frac{dI}{dt} = (1-\omega)\left(\beta_S I + \beta_W W\right) S - (\varepsilon + \delta) I, \\ \frac{dQ}{dt} = \varepsilon I - (\eta + \delta) Q, \\ \frac{dR}{dt} = \eta Q - \mu_R R, \\ \frac{dW}{dt} = (1-\omega)\alpha I - \sigma W, \\ S(0) = S_0 > 0, I(0) = I_0 \geq 0, Q(0) = Q_0 \geq 0, R(0) = R_0 \geq 0, W(0) = W_0 \geq 0. \end{cases} \tag{2.1}$$

Where S, I, Q, and R are the total number of susceptible, infected, quarantined, and recovered populations respectively. W is the concentration of the SARS-COV-2 virus caused by humans, resulting in coughing and sneezing. All parameters are positive and defined in Table 1.

Table 1. Model parameters

Parameters	Epidemiological interpretation
Λ	Birth rate parameter of S population
β_S	Transmission rate from I to S
β_W	Transmission rate from W to S
μ_S	Death rate of people S
η	Recovery rate
δ	Death rate of I population
ε	Rate of quarantine of infected individuals
μ_R	Death rate of R population
α	Shedding coefficients from I to W
$\frac{1}{\sigma}$	Lifetime of the virus in W
$0 < \omega < 1$	A measure of education campaign and treatment efficacy

As the equation of recovered population R depends only on quarantined population Q, it suffices to study the following reduced system:

$$\begin{cases} \frac{dS}{dt} = \Lambda - (1-\omega)\left(\beta_S I + \beta_W W\right) S - \mu_S S, \\ \frac{dI}{dt} = (1-\omega)\left(\beta_S I + \beta_W W\right) S - (\varepsilon + \delta) I, \\ \frac{dQ}{dt} = \varepsilon I - (\eta + \delta) Q, \\ \frac{dW}{dt} = (1-\omega)\alpha I - \sigma W, \\ S(0) = S_0 > 0, I(0) = I_0 \geq 0, Q(0) = Q_0 \geq 0, W(0) = W_0 \geq 0. \end{cases} \tag{2.2}$$

3 Non-negativity and boundedness of solutions

For system (2.2) to be biologically meaningful, it is important to show that all the population variables are nonnegative for all $t \geq 0$; which implies that any trajectory that starts with a positive initial condition will remain positive for $t \geq 0$. It is an important feature of an epidemiological model.

Proposition 3.1. *The set $\mathbb{R}^4_+$ is positively invariant with respect to the system (2.2). Furthermore, all solutions of (2.2) are uniformly bounded in the compact subset.*

$$\Gamma = \{(S, I, Q, W) \in \mathbb{R}^4_+; S + I + Q \leq \frac{\Lambda}{\mu_s}; W \leq \frac{(1-\omega)\Lambda\alpha}{\mu_s\sigma}\}. \tag{3.1}$$

Proof. From the first equation of system (2.2), we have

$$S(t) = S(0) \times exp(\int_0^t -\phi(s)ds) + exp(\int_0^t -\phi(s)ds) \times \int_0^t \Lambda \times exp(\int_0^u \phi(l)dl)du.$$

where $\phi(s) = -(1-\omega)(\beta_S I(s) + \beta_W W(s))$. Thus $S(t) > 0, \forall t > 0$.
To establish that $\forall t > 0$, $I(t) > 0$, $Q(t) > 0$, $W(t) > 0$ whenever $I(0) > 0$, $Q(0) > 0$, $W(0) > 0$, the above arguments can not be easily implemented. We then use an alternative trick. We Consider the following sub-equations related to the time evolution of variables

$$\begin{cases} \frac{dI}{dt} = (1-\omega)(\beta_S I + \beta_W W)S - (\varepsilon + \delta)I, \\ \frac{dQ}{dt} = \varepsilon I - (\eta + \delta)Q, \\ \frac{dW}{dt} = (1-\omega)\alpha I - \sigma W, \\ I(0) > 0, Q(0) > 0, W(0) > 0. \end{cases} \tag{3.2}$$

The system (3.2) can be written as follows:

$$\dot{X}(t) = AX(t),$$

where $X = \begin{pmatrix} I \\ Q \\ W \end{pmatrix}$ and $A = \begin{pmatrix} (1-\omega)\beta_s S - (\gamma + \mu_I) & 0 & (1-\omega)\beta_W S \\ \varepsilon & -(\eta+\delta) & 0 \\ (1-\omega)\alpha & 0 & -\sigma \end{pmatrix}$.
From the expression of A, it's a Metzler matrix and its exponential is positive. Then we deduce the positivity of $I(t), Q(t)$ and $W(t)$ whenever $I(0) > 0, Q(0) > 0$ and $W(0) > 0$. This proves the positively invariant property of $\mathbb{R}^4_+$ with respect to system (2.2).
Let $N(t) = S(t) + I(t) + Q(t)$, then

$$\frac{dN}{dt} = \Lambda - \mu_S S - \gamma I - (\eta + \delta)Q \leq \Lambda - \mu_S N.$$

Hence,

$$\limsup_{t \longrightarrow \infty} N(t) \leq \frac{\Lambda}{\mu_S}.$$

This implies that S, I and Q are uniformly bounded in the region Γ. Furthermore, from the bound of I and the last equation of (2.2), it follows that

$$\limsup_{t \longrightarrow \infty} W(t) \leq \frac{(1-\omega)\Lambda\alpha}{\mu_s\sigma}.$$

This guarantees the boundedness of W. This completes the proof. □

4 Equilibria and basic reproduction number R_0

The equilibrium points of model (2.2) are obtained by solving the algebraic system obtained by canceling all derivatives of $S(t), I(t), Q(t)$ and $W(t)$.
The disease-free equilibrium point denoted by E^0 is the steady-state solution of the model in the absence of disease. Thus: $E^0 = (S^0, 0, 0, 0) = \left(\frac{\Lambda}{\mu_s}, 0, 0, 0\right)$.

R_0 refers to the number of secondary infections generated by a single infective individual in a completely susceptible population. We use next-generation matrix, the approach by [23] to determine R_0. Using this method the basic reproduction number is given by $\rho\left(F_0 V_0^{-1}\right)$ where

F_0 is the Jacobian of f_i at E^0, where f_i is the rate at which new infections appear in compartment i and V_0 is the Jacobian of v_i at E^0, where v_i is the rate of transfer of individuals into and out of compartment i. The infected population is captured in the following system of equations.

$$\begin{aligned}
\frac{dI}{dt} &= (1-\omega)\left(\beta_S I + \beta_W W\right) S - (\varepsilon+\delta)I,\\
\frac{dQ}{dt} &= \varepsilon I - (\eta+\delta)Q,\\
\frac{dW}{dt} &= (1-\omega)\alpha I - \sigma W.
\end{aligned}$$

We have

$$f_i(I,Q,W) = \begin{bmatrix} (1-\omega)\left(\beta_S I + \beta_W W\right) S \\ 0 \\ 0 \end{bmatrix} \text{ and } v_i(I,Q,W) = \begin{bmatrix} (\varepsilon+\delta)I \\ (\eta+\delta)Q - \varepsilon I \\ \sigma W - (1-\omega)\alpha I \end{bmatrix}.$$

It follows that

$$F_0 = \begin{bmatrix} \frac{(1-\omega)\beta_s\Lambda}{\mu_s} & 0 & \frac{(1-\omega)\beta_w\Lambda}{\mu_s} \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{bmatrix}, V_0 = \begin{bmatrix} (\varepsilon+\delta) & 0 & 0 \\ -\varepsilon & (\eta+\delta) & 0 \\ -(1-\omega)\alpha & 0 & \sigma \end{bmatrix}$$

and

$$F_0 V_0^{-1} = \begin{bmatrix} \frac{(1-\omega)\beta_s\Lambda}{\mu_s(\varepsilon+\delta)} + \frac{(1-\omega)^2\beta_w\Lambda}{\mu_s(\varepsilon+\delta)} & 0 & \frac{(1-\omega)\beta_w\Lambda}{\alpha\mu_s} \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{bmatrix}.$$

Thus

$$R_0 = \rho\left(F_0 V_0^{-1}\right) = \frac{(1-\omega)\beta_s\Lambda}{\mu_s(\varepsilon+\delta)} + \frac{(1-\omega)^2\beta_w\Lambda}{\mu_s(\varepsilon+\delta)} = R_h + R_w.$$

where $R_h = \frac{(1-\omega)\beta_s\Lambda}{\mu_s(\varepsilon+\delta)}$ represents the secondary infections caused directly by a single infective while $R_w = \frac{(1-\omega)^2\beta_w\Lambda}{\mu_s(\varepsilon+\delta)}$ through the shedding of virus particles by infectious individuals.
The endemic equilibrium exists if $R_0 > 1$: $E^* = (S^*, I^*, Q^*, W^*)$, with its components given by

$$\begin{aligned}
S^* &= \frac{\Lambda - (\varepsilon+\delta)I^*}{\mu_s},\\
I^* &= \frac{\Lambda}{\varepsilon+\delta} - \frac{\sigma\mu_s}{(1-\omega)\left(\sigma\beta_s + \beta_w\alpha(1-\omega)\right)},\\
Q^* &= \frac{\varepsilon I^*}{\eta+\delta},\\
W^* &= \frac{(1-\omega)\alpha I^*}{\sigma}.
\end{aligned} \tag{4.1}$$

Then, we deduce the following result.

Proposition 4.1. • *If $R_0 \leq 1$, the model (2.2) has only one disease-free equilibrium (DFE), i.e., $E^0 = \left(\frac{\Lambda}{\mu_s}, 0, 0, 0\right)$.*

- *If $R_0 > 1$, in addition to the disease-free equilibrium E^0, the model (2.2) has a unique endemic equilibrium point $E^* = (S^*, I^*, Q^*, W^*)$.*

5 Stability of equilibria

5.1 Local and global stability of E^0

Proposition 5.1. *The disease-free equilibrium E^0 is locally asymptotically stable if $R_0 \leq 1$.*

Proof. Linearizing system (2.2) around an equilibrium point $E = (S, I, Q, W)$, we get the following jacobian matrix:

$$J_{E=(S,I,Q,W)} = \begin{pmatrix} -(1-\omega)(\beta_s I + \beta_w W) - \mu_s & -(1-\omega)\beta_s S & 0 & -(1-\omega)\beta_w S \\ (1-\omega)(\beta_s I + \beta_w W) & (1-\omega)\beta_s S - (\varepsilon+\delta) & 0 & (1-\omega)\beta_w S \\ 0 & \varepsilon & -(\eta+\delta) & 0 \\ 0 & (1-\omega)\alpha & 0 & -\sigma \end{pmatrix}.$$

Replacing E by E^0 and calculating the characteristic equation, we have

$$\det(\lambda I - J_{E^0}) = (\lambda + \mu_s)(\lambda + \eta + \delta)(\lambda^2 + A\lambda + B) = 0. \tag{5.1}$$

Clearly $-\mu_s$ and $-(\eta+\delta)$ are the eigenvalues, the remaining eigenvalues are given by solving the equation

$$\lambda^2 + A\lambda + B = 0, \tag{5.2}$$

where

$$\begin{aligned} A &= \sigma + (\varepsilon+\delta)(1-R_h), \\ B &= (\epsilon+\delta)(\sigma(1-R_h) + \alpha R_w). \end{aligned}$$

Clearly, $A > 0$ and $B > 0$ are satisfied when $R_0 \leq 1$. Hence, a disease-free equilibrium point E^0 is locally asymptotically stable. □

Considering the approach by [7] Castillo-Chavez theorem, the system (2.2) can be expressed as,

$$\begin{aligned} \frac{dX}{dt} &= \mathbf{F}(X, Z), \\ \frac{dZ}{dt} &= \mathbf{G}(X, Z), \quad \mathbf{G}(X, 0) = 0. \end{aligned}$$

Where $X \in \mathbb{R} = (S)$, the number of non-infected individuals and $Z \in \mathbb{R}^3 = (I, Q, W)$, the infected compartments.

The following conditions are for global stability of disease-free equilibrium point $E^0 = (S^0, 0, 0, 0) = \left(\frac{\Lambda}{\mu_s}, 0, 0, 0\right) = (X^0, 0)$, for $X^0 = \frac{\Lambda}{\mu_s}$:

1- $\frac{dX}{dt} = \mathbf{F}(X, 0)$, X^0 is globally asymptotically stable,

2- $\mathbf{G}(X, Z) = MZ - \widehat{\mathbf{G}}(X, Z)$, $\widehat{\mathbf{G}}(X, Z) \geq 0$ for $(X, Z) \in \Gamma$,

where $M = D_Z \mathbf{G}\left(X^0, 0\right)$ is an M-matrix (in that the off-diagonal elements of M are positive) and Γ is the region where the equations of the model make epidemiological sense. If conditions 1 and 2 are satisfied by system (2.2), the following proposition holds.

Proposition 5.2. *Provided that $R_0 \leq 1$ and the conditions 1 and 2 are satisfied, the disease-free equilibrium point $E^0 = \left(X^0, 0\right)$ of the system (2.2) is globally asymptotically stable.*

Proof. Since X = (S) and Z = (I, Q, W),
the condition $\frac{dX}{dt} = \mathbf{F}(X, 0)$ can be written as; $\frac{dS}{dt} = \Lambda - \mu_s S$, which gives:
$\Lambda - \mu_s S(t) = (\Lambda - \mu_s S(0))e^{-\mu_s t}$
$\Rightarrow S(t) = \frac{\Lambda - (\Lambda - \mu_s S(0))e^{-\mu_s t}}{\mu_s}$
$\Rightarrow S(t) \to \frac{\Lambda}{\mu_s}$ as $t \to \infty$
hence E^0 is globally asymptotically stable.
In view of $\mathbf{G}(X, Z) = MZ - \widehat{\mathbf{G}}(X, Z)$, we have

$$\widehat{\mathbf{G}}(X,Z) = MZ - \mathbf{G}(X,Z),$$

$$G(X,Z) = \begin{bmatrix} (1-\omega)\left(\beta_S I + \beta_W W\right) S - (\varepsilon+\delta)I \\ \varepsilon I - (\eta+\delta)Q \\ (1-\omega)\alpha I - \sigma W \end{bmatrix},$$

$$M = D_Z\mathbf{G}\left(X^0,0\right) = \begin{bmatrix} (1-\omega)\beta_s S^0 - (\varepsilon+\delta) & 0 & (1-\omega)\beta_w S^0 \\ \varepsilon & -(\eta+\delta) & 0 \\ (1-\omega)\alpha & 0 & -\sigma \end{bmatrix},$$

$$\mathrm{MZ} = \begin{bmatrix} (1-\omega)\beta_s I S^0 - (\varepsilon+\delta)I + (1-\omega)\beta_w W S^0 \\ \varepsilon I - (\eta+\delta)Q \\ (1-\omega)\alpha I - \sigma w \end{bmatrix}.$$

Therefore

$$\widehat{\mathbf{G}}(X,Z) = \begin{bmatrix} (1-\omega)\beta_s I\left(S^0 - S\right) + (1-\omega)\beta_w W\left(S^0 - S\right) \\ 0 \\ 0 \end{bmatrix}.$$

Since all off diagonal entries of matrix M are positive, it implies that M is an M-matrix.
Also since $0 < \omega < 1$ and $S^0 \geq S$: $\quad \forall (X,Z) \in \Gamma, \widehat{\mathbf{G}}(X,Z) \geq 0$.
Therefore, condition 2 can be expressed as $\frac{dZ}{dt} \leq WZ$.
Since $S^o = \frac{\Lambda}{\mu}$, the characteristic equation of M is given by

$$(\lambda - (\eta+\delta))\left(\lambda^2 + B\lambda + C\right) = 0,$$

or $\lambda = -(\eta+\delta+\mu)$ and

$$\lambda^2 + A\lambda + B = 0, \tag{5.3}$$

where

$$\begin{aligned} A &= \sigma + (\varepsilon+\delta)(1-R_h), \\ B &= (\epsilon+\delta)(\sigma(1-R_h) + \alpha R_w). \end{aligned}$$

Clearly equation (5.3) is the same as equation (5.2). So, $A > 0$ and $B > 0$ are satisfied when $R_0 \leq 1$. Since the conditions 1 and 2 have been met and $R_0 \leq 1$, the proof is complete. □

5.2 Local and global stability of E^*

Proposition 5.3. *If $R_0 > 1$ and condition (H) (5.6) are satisfied, then the endemic equilibrium point E^* of system (2.2) is locally asymptotically.*

Proof. The jacobian matrix J evaluated at the endemic equilibrium point E^* is given by

$$J\left(E^*\right) = \begin{bmatrix} -X-\mu_s & -Y & 0 & -Z \\ X & Y-L & 0 & Z \\ 0 & \varepsilon & -M & 0 \\ 0 & N & 0 & -\sigma \end{bmatrix},$$

where

$$\begin{aligned} X &= (1-\omega)(\beta_s I^* + \beta_w W^*), \\ Y &= (1-\omega)\beta_s S^*, \\ Z &= (1-\omega)\beta_w S^*, \\ L &= (\varepsilon+\delta), \\ M &= (\eta+\delta), \\ N &= (1-\omega)\alpha. \end{aligned}$$

The characteristic equation is given by

$$(\lambda + M)(\lambda^3 + a_2\lambda^2 + a_1\lambda + a_0) = 0.$$

As $\lambda_1 = -M$ is a root of the characteristic equation, the local study of the stability of E^* is reduced to the study of the roots of the equation

$$\lambda^3 + a_2\lambda^2 + a_1\lambda + a_0 = 0, \tag{5.4}$$

where

$$\begin{aligned} a_2 &= L + X - Y + \mu_s + \sigma, \\ a_1 &= L\mu_s + LX + \mu_s\sigma + L\sigma + X\sigma - Y\sigma - Y\mu_s - ZN, \\ a_0 &= XYN - XZN + LX\sigma + L\mu_s\sigma - Y\mu_s\sigma - ZN\mu_s. \end{aligned}$$

Applying Routh Hurwitz criterion [8], all roots of equation (5.4) are negative when $a_2 > 0$, $a_0 > 0$ and $a_2a_1 - a_0 > 0$.

To show that, we substitute equation (4.1) in the second equation of system (2.2) at endemic equilibrium point to get

$$(1-\omega)\left[\beta_s + \frac{\beta_w(1-\omega)\alpha}{\sigma}\right] S^* - (\varepsilon + \delta) = 0,$$

or,

$$\frac{\beta_w(1-\omega)^2\alpha S^*}{\sigma} = L - Y. \tag{5.5}$$

Substituting (4.1) and (5.5) in a_2, a_1, and a_0 appropriately, we obtain

$$\begin{aligned} a_2 &= \frac{\beta_w(1-\omega)^2\alpha S^*}{\sigma} + X + \sigma + \mu_s > 0, \\ a_1 &= LX + \mu_s\sigma + X\sigma + \frac{\beta_w(1-\omega)^2\alpha\mu_s S^*}{\sigma} > 0, \\ a_2a_1 - a_0 &= XYN - XZN + LX\sigma + L\mu_s\sigma - Y\mu_s\sigma - ZN\mu_s. \end{aligned}$$

Since $a_2 > 0$, we consider the following hypotheses:

$$(H) : a_0 > 0 \text{ and } a_2a_1 - a_0 > 0. \tag{5.6}$$

From the hypothesis (H) and the Routh-Hurwitz stability criterion, equation (5.4) has no positive root. Therefore, the endemic equilibrium E^* is locally asymptotically stable. □

Proposition 5.4. *The endemic equilibrium point E^* of the system (2.2) is globally asymptotically stable if $R_0 > 1$.*

Proof. To prove global stability of E^*, we apply LaSalle [14] approach by constructing the following Lyapunov function

$$V(S, I, Q, W) = \left(S - S^* \ln\frac{S}{S^*}\right) + \left(I - I^* \ln\frac{I}{I^*}\right) + \left(Q - Q^* \ln\frac{Q}{Q^*}\right) + \left(W - W^* \ln\frac{W}{W^*}\right).$$

Differentiating V, we get

$$\frac{dV}{dt} = \left(1 - \frac{S^*}{S}\right)\frac{dS}{dt} + \left(1 - \frac{I^*}{I}\right)\frac{dI}{dt} + \left(1 - \frac{Q^*}{Q}\right)\frac{dQ}{dt} + \left(1 - \frac{W^*}{W}\right)\frac{dW}{dt}.$$

Substituting $\frac{dS}{dt}$, $\frac{dI}{dt}$, $\frac{dQ}{dt}$ and $\frac{dW}{dt}$ from system (2.2), we obtain

$$\begin{aligned} \frac{dV}{dt} &= \left(1 - \frac{S^*}{S}\right)\{\Lambda - (1-\omega)(\beta_S I + \beta_W W) S - \mu_S S\} \\ &+ \left(1 - \frac{I^*}{I}\right)\{(1-\omega)(\beta_S I + \beta_W W) S - (\varepsilon + \delta) I\} \\ &+ \left(1 - \frac{Q^*}{Q}\right)\{\varepsilon I - (\eta + \delta) Q\} + \left(1 - \frac{W^*}{W}\right)\{(1-\omega)\alpha I - \sigma W\}. \end{aligned} \tag{5.7}$$

Rearranging system (2.2) at the endemic equilibrium point, we have

$$\begin{aligned} \Lambda &= (1-\omega)\left(\beta_S I^* + \beta_W W^*\right) S^* + \mu_S S^* \\ (\varepsilon+\delta) &= \frac{(1-\omega)}{I^*}\left(\beta_S I^* + \beta_W W^*\right) S^* \\ (\eta+\delta) &= \frac{\varepsilon I^*}{Q^*} \\ \sigma &= \frac{(1-\omega)\alpha I^*}{W^*}. \end{aligned} \tag{5.8}$$

Substituting (5.8) in (5.7), we get

$$\begin{aligned} \frac{dV}{dt} &= \left(1-\frac{S^*}{S}\right)\left[(1-\omega)\left\{\left(\beta_S I^* S^* + \beta_W W^* S^*\right) - \left(\beta_S IS + \beta_W WS\right)\right\} + \mu_S(S^*-S)\right] \\ &+ \left(1-\frac{I^*}{I}\right)(1-\omega)\left[\left(\beta_S IS + \beta_W WS\right) - \frac{I}{I^*}\left(\beta_S I^* S^* + \beta_W W^* S^*\right)\right] \\ &+ \left(1-\frac{Q^*}{Q}\right)\varepsilon\left(I - \frac{QI^*}{Q^*}\right) + \left(1-\frac{W^*}{W}\right)(1-\omega)\alpha\left[I - \frac{WI^*}{W^*}\right]. \end{aligned}$$

When $S = S^*, I = I^*, Q = Q^*$ and $W = W^*$, we obtain $\frac{dV}{dt} = 0$.
Hence by LaSalle's invariance principle [14], every solution of the system (2.2) with initial conditions in $\Gamma = \left\{(S,I,Q,W) \in \mathbb{R}^4_+; S+I+Q \leq \frac{\Lambda}{\mu_S}; W \leq \frac{(1-\omega)\Lambda\alpha}{\mu_S\sigma}\right\}$ tends to the endemic equilibrium point E^*. It follows that E^* is globally asymptotically stable. □

6 Numerical simulation

Analytic studies cannot be complete without numerical verification of the results. In this section, we present some numerical simulations with a hypothetical set of parameters to illustrate our analytical results. We simulated the system (2.2) to investigate the importance of education campaigns and treatment through quarantine. This is achieved using parameter values in Table 2.

Table 2. Parameter values

Parameters	Values
Λ	30-100
β_S	0.06
β_W	0.05
μ_S	0.03
η	0.03
δ	0.03
α	0.07
σ	0.2
ε	0.5
ω	$0 < \omega < 1$

The initial values of the state variables are provided as follows: $S(0) = 100000, I(0) = 100$ and $Q(0) = W(0) = 0$. The results of the simulation are presented in the figures below.

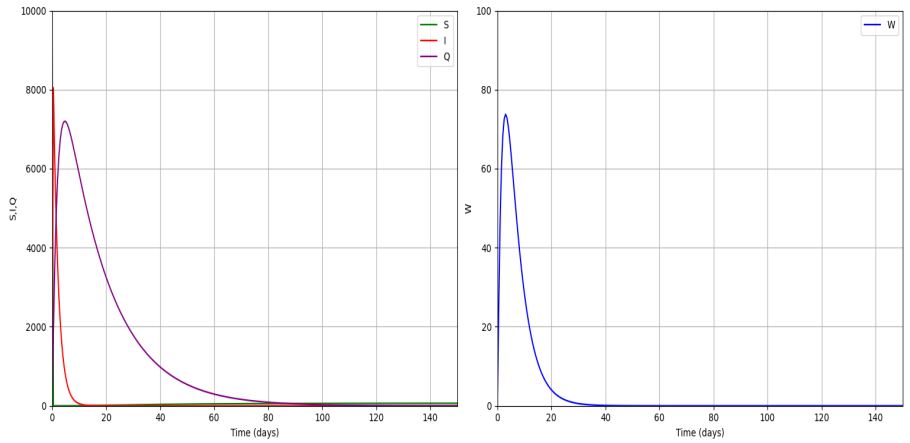

Figure 1. Stability of E^0 and nonexistence of E^* for $R_0 \leq 1$.

For E^0, the reproduction number $R_0 = 0.81 \leq 1$ and $E^0(67, 0, 0, 0)$. Figure 1 depicts the stability of E^0. That means each infected individual does not transmit the disease to enough new individuals to cause further infections, and eventually, the infection dies out. Essentially, the pool of susceptible individuals is large enough that the number of new infections declines over time, and the disease cannot persist in the population.

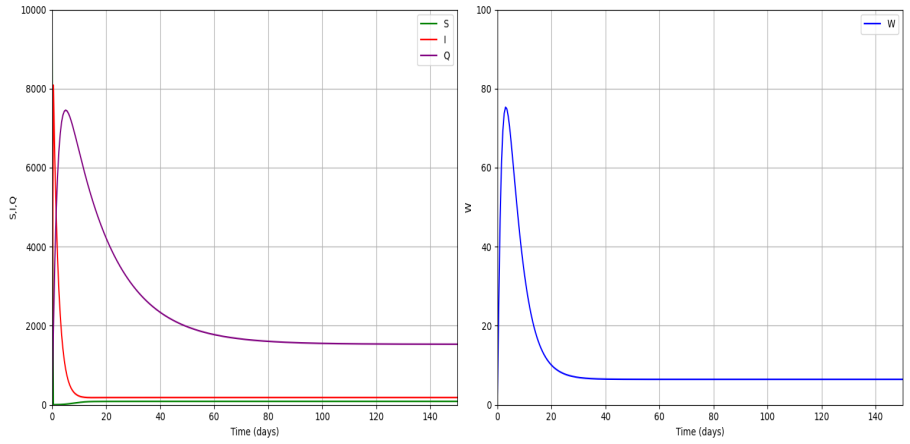

Figure 2. Instability of E^0 and stability of E^* for $R_0 > 1$.

For E^*, the reproduction number $R_0 = 4.52 > 1$ and $E^*(85.82, 183.82, 1531.84, 6.43)$. Figure 2 shows the increase in infected individuals and the concentration of the SARS-COV-2 virus in the early days due to the rapid spread of the disease. The increase in the number of infected people consequently reduces the number of susceptible. Then we notice a decrease in the number of infected people and an increase in the number of quarantined individuals (since we assumed that all individuals who were identified with COVID-19 infection were quarantined). The system then goes to its stable solution E^* as the number of susceptible people becoming infectious decreases to the lowest level and the infected individuals, quarantined individuals, and the concentration of the SARS-COV-2 virus increases to the saturation level.

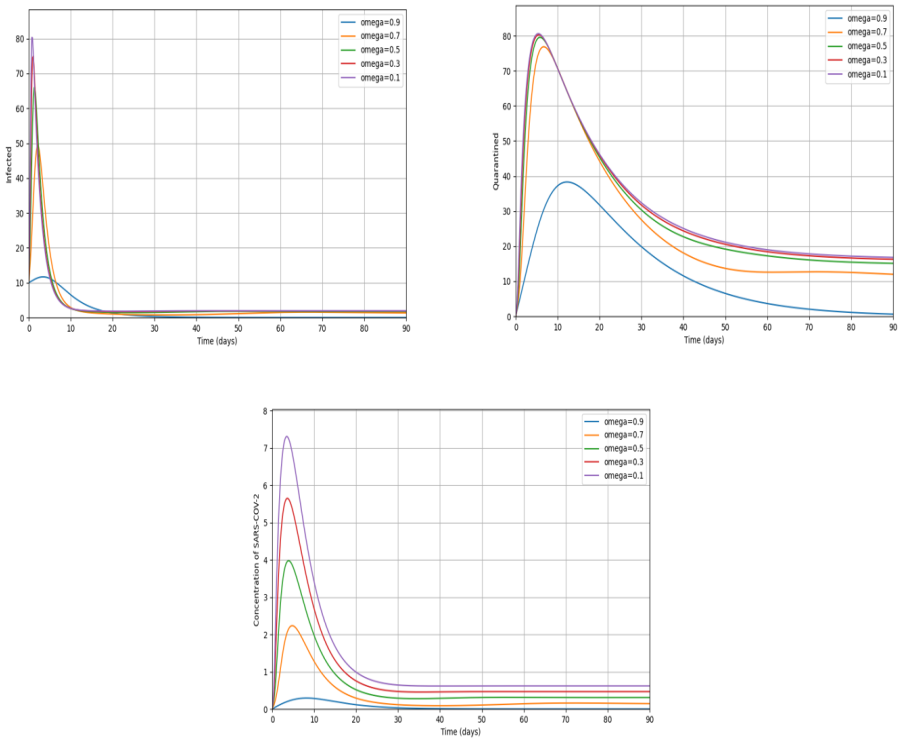

Figure 3. The effect of education campaign and treatment on infected individuals, quarantined individuals, and concentration of SARS-COV-2.

Figure 3 demonstrates the variation of infected individuals, quarantined individuals and concentration of the SARS-COV-2 virus respectively with different values of ω. Particularly, we observe that the value of the infected population declines as the education campaign and treatment efficacy increases. Additionally, if education campaign is 100 percent effective in preventing disease transmission, i.e., $\omega = 1$, the value of R_0 becomes zero, and the disease will not spread from one person to another in that scenario. This means that it is necessary to educate people about COVID-19 infection and how it can be prevented especially those in major and crowded cities and institutions as well as treat the quarantined individuals. Education campaigns should target direct and indirect transmissions. This can be achieved through posters, radio, social media, television, and word-of-mouth communication.

7 Discussion and Conclusion

In this work, we formulated SIQR-W a mathematical model of COVID-19 taking into account the effects of direct and indirect transmissions with education campaigns and treatment through quarantine. Boundedness and the existence of solutions is shown. We studied the stability of the disease-free and endemic equilibrium. The results of the disease-free equilibrium showed that the model is both locally and globally asymptotically stable when $R_0 \leq 1$. This implies that when R_0 is below unity, the spread of COVID-19 disease reduces. Next, we studied the endemic equilibrium which we found to be both locally and globally asymptotically stable when $R_0 > 1$. Numerical simulation indicates that when effective health education campaigns and treatment are in place as control strategies for COVID-19, they lead to a faster reduction of the disease, and eventually the disease decreases to zero.

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