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Research Article

Mathematical Model Formulation and Analysis for COVID-19 Transmission with Virus Transfer Media and Quarantine on Arrival

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An outbreak of severe acute respiratory syndrome (COVID-19) killed 287,355 with 4, 257,578 cases worldwide as of May 12, 2020. In this paper, we propose an $SEQI_sI_aRM$ deterministic mathematical model which contains compartments for both human-to-human transmission and transmission through contaminated surfaces. Without intervention, the role of symptomatic and asymptomatic cases in humans is found to be very high in the transmission of the virus. Sensitive parameters which are associated with increased transmission of the COVID-19 virus were identified. According to the sensitivity results, the most sensitive parameters were disease-induced death rates of symptomatic and asymptomatic infectious people (σ), the rate of removal of virus from surfaces and environment (ν), and the rate of infection by asymptomatic infectious people (λ_2) and symptomatic infectious people (λ_1). The numerical results of our model confirm the sensitivity results that there are more new incidences of asymptomatic cases than symptomatic cases, which escalates the transmission of the virus in the community. Combined interventions like increasing both the rate of removal of viruses from surfaces and environment and decreasing the rate of infection in asymptomatic cases can play a significant role in reducing the average number of secondary infection (R_0) to less than unity, causing COVID-19 to die out.

1. Introduction

In December 2019, China reported a cluster of cases of pneumonia which now known to be caused by a novel coronavirus (2019-nCov) [1, 2]. SARS-CoV-2 is a positive single strain RNA virus later named COVID-19 by the WHO on February 11, 2020. COVID-19 originated from the Wuhan, China, in Hubei province. COVID-19 spread rapidly around the globe and present in more than 188 countries and territories around the world as of May 20, 2020 [3–5]. The total number of confirmed cases of COVID-19 rose from 534 to 4.77 million from January 22, 2020, to May 20, 2020, with the daily number of confirmed cases ranging from 142 to 94,557, respectively [6]. From January 22, 2020, to May 20, 2020, the total number of death increased from 17 to 322,483.

The World Health Organization (WHO) declared a global health emergency on January 30, 2020 [7]. At this time, the fatality rate was calculated to be 2.2% with a total of 7734 confirmed cases in China and 90 cases from 25 other countries [8, 9]. The daily number of laboratory-confirmed cases of COVID-19 from January 21, 2020 to February 11, 2020, was growing at about the same speed in China and outside of China [10]. In the USA, the first case of human-to-human transmission of COVID-19 was confirmed on January 30, 2020. This led to the description, identification,

diagnosis, clinical course, and management of this case, with documentation of the patient's initial mild symptoms at presentation and progression to pneumonia on days 9 to 14 of illness [9].

On March 13, 2020, the World Health Organization (WHO) declared Europe as the epicenter of the disease [8, 11]. According to an Our World in Data (OWD) [2] report on the 4th of April, 2020, Italy was labeled as the country with the highest death rate with more than 15,000 deaths recorded and many others infected, while the United States of America (USA) is the hardest hit with 91,921 deaths recorded as of 8th of May, 2020 [12]. Brazil, the UK, Germany, and France followed the USA with more than 100,000 confirmed cases.

The speed of transmission of COVID-19 more closely resembles influenza during the 1918 outbreak in England than severe acute respiratory syndrome (SARS) caused by a coronavirus [12, 13]. Even though COVID-19 has a lower fatality rate than SARS and Middle East respiratory syndrome (MERS), it has killed more people than SARS and MERS combined [14]. COVID-19 does not have an effective vaccine or an exact and effective treatment scheme. It shows a destructive effect especially in individuals with weak immune systems. Intensive care and mechanical ventilation may be required in the treatment process [15]. The mortality rate of COVID-19 is more than 3.7%, which is high compared with the mortality rate of influenza which is less than 1%. There is an urgent need for effective treatment [1].

The symptoms of COVID-19 infection appear after an incubation period of approximately 5.2 days, but it ranges from 1 to 14 days [9, 16]. Fever was the most common symptom, followed by cough and fatigue, while other symptoms include sputum production, headache, hemoptysis, diarrhea, dyspnoea, and lymphopenia [2, 9]. The period from the onset of COVID-19 symptoms to death ranged from 6 to 41 days with a median of 14 days, and this period is dependent on the age and immune system of an individual. Most likely individuals aged >70 have a higher probability of death than individuals with aged <70 [9].

Studies are underway to understand the nature of the virus to inform effective treatment and prevention measures [8]. Even though there is still considerable genetic variation between SARS-CoV-2 (COVID-19) and SARS-CoV, SARS-CoV-2 has closer similarity with SARS-CoV than MERS-CoV with 76% S-protein, 90.6% N-protein, 90.1% M protein, and 94.7% with E-protein [8, 17]. The push for an effective vaccine against SARS-CoV-2 has remained the work of scientists and researchers. Effective vaccination is actually the only solution to eliminate the virus from the human population [16, 18]. However, much is not known about the virus and specific immune responses against SARS-CoV-2, its biological properties, and epidemiology. A clinical trial of such a vaccine has already started recruiting volunteers in Seattle [4].

As vaccine development continues, other protective measures are recommended by the WHO. Governments and policy makers are taking and designing protective measures in all countries in which COVID-19 has appeared including mandated travel restrictions, lockdowns, canceling or postponing event, and massive closures of schools and universities. Medical professionals are also recommending individuals to take protective measures like social distancing, washing hands properly or sanitizing, wearing mask, staying home, and cleaning surfaces with disinfectants [16]. Given the WHO predicts the COVID-19 virus may be with us for a long time, it is especially important to understand the disease transmission and dynamics.

COVID-19 spread by human-to-human transmission via droplets or direct contact. Infection has been estimates to have mean incubation period of 6.4 days, and the early estimation of basic reproduction number (R_0) ranges from 2.24 to 3.58 [2]. Around the first outbreak in Wuhan, the reproduction number estimate increased from 1.6 to 2.6 between January 1, 2020, and January 23, 2020. After the introduction of travel restriction, R_0 declined from 2.35 to 1.05 [9, 17].

Initially in the COVID-19 pandemic, there was a misconception about the transmission of the virus in relation with climate condition. Looking the case of West Africa and Europe, it was confirmed that COVID-19 cases do not support the hypothesis that the virus will spread more slowly in countries with warmer climates [8]. Based on origin destination and air travel flows from China to Africa, some African countries were estimated to have high to moderate risk of importation of COVID-19. According to this, countries with the highest risk (i.e., Egypt, Algeria, and South Africa) have moderate to high capacity to respond to the outbreak. Countries with moderate risk (i.e., Nigeria, Ethiopia, Sudan, Angola, Tanzania, Ghana, and Kenya) have variable ability to respond and high vulnerability [7]. African countries are at high risk of COVID-19 due to dense population and dense traffic between China and Africa. The WHO also identified 13 top priority countries which includes most East African Countries [17].

Some countries in Europe like Italy, Spain, and France seemed to pass the peak in the last few weeks. It is believed that in the second phase, COVID-19 will rise in developing countries [7]. In developing countries with poor health systems, and fewer facilities, the burden of COVID-19 could cause loss of millions of lives [18, 19]. In spite of poor health systems and facilities, majority of the people in these countries seem to be reluctant and dependent on the daily report of new cases to take protective measures. It is known that in developing countries, the capacity of testing for COVID-19 is very limited, and the number of daily cases are the result of the numbers of test. The only way for developing countries to protect against COVID-19 is take early preventive measures [20, 21].

Therefore, it is very important to investigate the dynamics of COVID-19 in relation to the appropriate protective measures that should be taken using mathematical models. In addition, it is important to predict the near future status of the virus in terms of current new cases and exposures. In this study, we have tried to examine the dynamics of COVID-19 transmission in the context of poor uptake of protective measures and a poor understanding of the transmission of the virus.

The proportion of individuals infected by COVID-19 who remain asymptomatic throughout the course of infectious period has not yet been definitely assessed. This makes the transmission and status of the virus to be complicated [17]. Mathematical models have played an important role in determining how infectious diseases are transmitted and are predicting the future dynamics. Currently, such models are being used to predict the likely health burden of the COVID-19 pandemic in many parts of the world [18]. In the situation where there is no medical cure, models play an important role in suggesting effective protective measures based on scientific evidence.

A simple SIR model was developed by [16] for COVID-19 in China, and using the close similarity between statistical data and numerical simulation of the model, transmission of COVID-19 was predicted for effective measure. An age-structured stochastic mathematical model of COVID-19 was developed by [18] for selected cities from Africa and South Asia. In the study, Karachi, Delhi, Nairobi, Addis Ababa, and Johannesburg were included, and predictions were made based on the model. In these cities, there is severe lack of intensive care beds, health-care workers, and financial resources to meet demand during an unmitigated COVID-19 pandemic. The peak of cases in each city found to be between 103 and 119 days after the date of the first introduced case. A modified susceptible, exposed, infected, recovered (SEIR) model was developed by [22], and the time of diagnosing susceptible individuals was found to be a sensitive parameter for the transmission of the disease. [23] developed a mathematical model for Diamond Princess Ship to study the trajectory of the outbreak among passengers and crew members using the data from early February to February 18, 2020. Their estimation of the mean reproduction number in the confined setting was 11, which was higher than the mean estimates reported from community-level transmission dynamics in China and Singapore (which was ranging from 1.1 to 7). Their study confirmed that the reproduction number of COVID-19 is very high in the early stage compared to the time after which some strategies are applied. Other community-level studies found the value of the reproduction number largely below the epidemic threshold. On April 27, 2020, [24] developed a compartmental mathematical model with special focus on the transmissibility of super-spreader individuals and found the secondary infection below the epidemic threshold. Due to the human-to-human transmission, the rate at which an exposed individual become infected and disease induced death rate was found to be the most sensitive parameters. The study recommended the inclusion of important compartments and dynamics to control the spread of the virus. Therefore, in this study, we incorporated the asymptomatic individuals, infected surface class, and other important compartments hypothesized highly influence the transmission of COVID-19.

2. Model Formulation

In this section, we formulate a SEQIRM (susceptible-exposed-quarantined-infected-recovered-media) model of COVID-19 for human-to-human transmission and transmission through surfaces. The media (M) includes all surfaces that were exposed to the virus of COVID-19 through contact with infected individuals or the air. We categorize the human population into six classes as follows: susceptible (S) groups with no disease, but able to be infected in case of contact with exposed, symptomatic, or asymptomatic infectious individuals or surfaces contaminated with COVID-19 virus. Exposed (E) individuals are those who contracted the virus through direct contact with infected people or surfaces. This class also includes people who might have exposed themselves through either lack of awareness or failure to follow one or more protective measures. Quarantined (Q) individuals are those who stay in confined places until they can confirm if they have contracted the virus or not through testing or observation for two or more weeks. Infected individuals are subdivided in to two subclasses which are symptomatic infectious (I_s) people who have been infected with COVID-19 virus and show clinical signs and symptoms and asymptomatic infectious (I_a) people who have been infected with COVID-19 virus and do not show any clinical signs and have no symptoms but are infectious. Recovered (R) individuals are those who recovered through the required treatment and returned back to the susceptible class.

2.1. Model Assumption. In our model, the susceptible class is infected by three other classes: symptomatic infectious , asymptomatic infectious and contaminated surfaces. Newly arrived and all suspected people who have exposed themselves for one or more risks of COVID-19 are quarantined for two or more weeks to observe their health status related to COVID-19. Once the defined number of days are completed, individuals are transferred to susceptible (S), or symptomatic infectious (I_s) or asymptomatic infectious (I_a) classes depending on their status with rates of κ and ϕ in proportion of Φ, respectively. Proportion of exposed individuals move to quarantined, symptomatic infectious, or asymptomatic infectious classes with rates η, ρ_1, and ρ_2, respectively. Both symptomatic and asymptomatic individuals can recover through natural treatment with rates π_1 and π_2, respectively. The contaminated surfaces class can be infected by symptomatic or asymptomatic infectious classes. Individuals who have recovered from COVID-19 have probability of returning back to the susceptible class due to lose of immunity.

Further, our model is developed based on the following assumptions.

Susceptible individuals are recruited at rate of ϑ.

(i) Exposed and asymptomatic infected individuals have more probability of movement from one place to another internally or as an immigrant because they have no symptoms of disease

(ii) Symptomatic infected individuals have less probability of movement from place to place due to experiencing symptoms of disease. They stay at a

confined place either in government prepared quarantined places or in their homes

(iii) All individuals who immigrate from other places join the quarantine class on arrival

(iv) Individuals in each group have equal natural death rate

(v) Populations are homogeneous; that is, each individual has equal probability of being infected by COVID-19

(vi) All surfaces and environments which can transfer the virus are include in one class called media (M)

2.2. Model Parameter and Flowchart. Table 1 shows the parameters used in the model and their descriptions.

Using the above assumptions and definition of variables and parameters, the model flow diagram which depicts the dynamics of COVID-19 transmission is shown in Figure 1.

2.3. Model Equations. By considering [24] and incorporating quarantine (Q), asymptomatic infectious (I_a), symptomatic infectious human (I_s), and the media(M) classes which includes surfaces and contaminated environment, we propose the following system of differential equation.

$$\frac{dS}{dt} = \vartheta + kQ + \omega R - (\lambda_1 I_s + \lambda_2 I_a + \lambda_3 M + \mu)S, \tag{1a}$$

$$\frac{dE}{dt} = (\lambda_1 I_s + \lambda_2 I_a + \lambda_3 M)S - (\rho_1 + \rho_2 + \eta + \mu)E, \tag{1b}$$

$$\frac{dQ}{dt} = \xi + \eta E - (k + \psi + \mu)Q, \tag{1c}$$

$$\frac{dI_s}{dt} = \rho_1 E + \Phi\psi Q - (\pi_1 + \mu + \sigma)I_s, \tag{1d}$$

$$\frac{dI_a}{dt} = \rho_2 E + (1 - \Phi)\psi Q - (\pi_2 + \mu + \sigma)I_a, \tag{1e}$$

$$\frac{dR}{dt} = \pi_1 I_s + \pi_2 I_a - (\omega + \mu)R, \tag{1f}$$

$$\frac{dM}{dt} = q_1 \frac{I_s}{N} + q_2 \frac{I_a}{N} - \nu M. \tag{1g}$$

3. Basic Properties of the Model

3.1. Invariant Region. Since COVID-19 affects people, then, in the modeling process, we assume that all state variables and parameters of the model are nonnegative $\forall t \geq 0$. We analyze the model system in a suitable feasible region where all state variables are positive. This region will be obtained under the following theorem.

Theorem 1. *All forward solutions in $\mathbb{R}_+^7$ of the system are feasible $\forall t \geq 0$ if they enter the invariant region Λ for $\Lambda = \Omega_H \times \Omega_M$.*

where

$$\Omega_H = (S, E, Q, I_a, I_s, R) \in \mathbb{R}_+^6 : S + E + Q + I_a + I_s + R \leq N,$$
$$\Omega_M = M \in \mathbb{R}_+^1, \tag{2}$$

and Λ is the positive invariant region of COVID-19 system.

Proof. We prove the theorem. □

3.1.1. For Human Population. We need to prove that the solution of the system (1) are feasible $\forall t > 0$ as they enter invariant region Ω_H.

We now let $\Omega_H = (S, E, Q, I_a, I_s, R) \in \mathbb{R}^6$ be solution space of the system with nonnegative initial conditions.

The total human population is

$$N = S + E + Q + I_s + I_a + R. \tag{3}$$

Then,

$$\frac{dN}{dt} = \frac{dS}{dt} + \frac{dE}{dt} + \frac{dQ}{dt} + \frac{dI_s}{dt} + \frac{dI_a}{dt} + \frac{dR}{dt}. \tag{4}$$

Adding up the system, we get

$$\frac{dN}{dt} = \vartheta + \xi - \mu_1 N - \sigma I_a - \sigma I_s. \tag{5}$$

We will then have

$$\frac{dN}{dt} \leq \vartheta + \xi - \mu N. \tag{6}$$

We then get

$$\frac{dN}{dt} + \mu N \leq \vartheta + \xi. \tag{7}$$

Finding the integrating factor IF $= e^{\mu t}$ and multiplying it through out, we get

$$e^{\mu t}\frac{dN}{dt} + e^{\mu t} N\mu \leq (\vartheta + \xi)e^{\mu t}, \tag{8}$$

which gives

$$\frac{d(Ne^{\mu t})}{dt} \leq (\vartheta + \xi)e^{\mu t}. \tag{9}$$

Integrating on both sides yields

$$Ne^{\mu_1 t} \leq \frac{(\vartheta + \xi)}{\mu} e^{\mu t} + C. \tag{10}$$

Table 1: Parameters and their description.

Parameter	Description
ϑ	The rate of recruitment of human population
$\lambda_1, \lambda_2, \lambda_3$	The rate of infection of susceptible humans (S) due to symptomatic infectious human (I_s), asymptomatic infectious human (I_a), and virus contaminated media (M)
k	The rate of quarantined people becoming free from COVID-19 virus
μ	The natural death rate of human population
σ	Disease induced death rate of symptomatic human population
ξ	The rate of quarantined individuals on arrival in a given country or city
η	The rate of COVID-19 suspected individuals to be quarantined
ρ_1	The rate of exposed human to become asymptomatic infectious I_a
ρ_2	The rate of exposed human to become symptomatic infectious I_s
ψ	The rate of quarantined human (Q) to be tested positive of COVID-19 virus
$\pi_{(1,2)}$	The rate of infectious individuals to recover from the virus
ω	The rate of recovered individuals to be susceptible again
q_1, q_2	The rate of M through I_s and I_a respectively
Φ	Proportion that Q become either I_a or I_s
ν	Disinfection rate of M

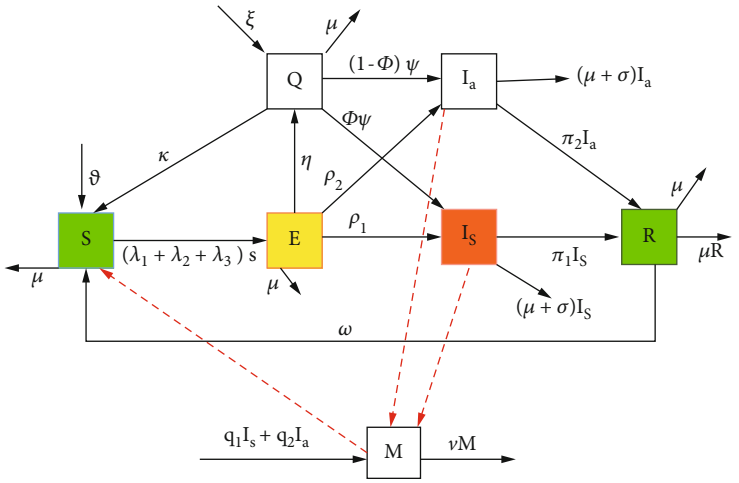

Figure 1: Model flowchart.

Multiplying the equation by $e^{-\mu_1 t}$, we get

$$N \le \frac{(\vartheta + \xi)}{\mu} + Ce^{-\mu t}. \tag{11}$$

Using the initial condition $t = 0, N(t = 0) = N_0$, then, we will get

$$N_0 - \frac{(\vartheta + \xi)}{\mu} \le C. \tag{12}$$

Substituting for the constant C, we get

$$N \le \frac{(\vartheta + \xi)}{\mu} + \left(N_0 - \frac{(\vartheta + \xi)}{\mu}\right)e^{-\mu t}. \tag{13}$$

When $N_0 > ((\vartheta + \xi)/\mu)$, the population decreases asymptotically to $(\vartheta + \xi)/\mu$, and when $N_0 < ((\vartheta + \xi)/\mu)$, the human population increases asymptotically to $(\vartheta + \xi)/\mu$.

Hence, all the feasible solutions of the system enter the region

$$\Omega_H = \left\{(S, E, Q, I_s, I_a, R): N \le \text{Max}\left\{N_0, \frac{(\vartheta + \xi)}{\mu}\right\}\right\}. \tag{14}$$

3.1.2. For Media. We need to prove that the solutions of the system are feasible $\forall t > 0$ as they enter invariant region Ω_Q. We now let $\Omega_Q = Q \in R_+^1$ be any solution of the system with nonnegative initial conditions.

From the media (M) equation

$$\frac{\mathrm{dM}}{\mathrm{dt}} = q_1 \frac{I_s}{N} + q_2 \frac{I_a}{N} - \nu M. \tag{15}$$

But

$$I_s \le N, I_a \le N. \tag{16}$$

Then, this implies that

$$\frac{I_s}{N} \le 1, \frac{I_a}{N} \le 1. \tag{17}$$

Equation (15) becomes

$$\frac{\mathrm{dM}}{\mathrm{dt}} \le q_1 + q_2 - \nu M. \tag{18}$$

Then, we will have

$$\frac{\mathrm{dM}}{\mathrm{dt}} + \nu M \le q_1 + q_2. \tag{19}$$

Finding the integrating factor IF $= e^{\nu t}$ and multiplying it through out, we get

$$e^{\nu t} \frac{\mathrm{dM}}{\mathrm{dt}} + e^{\nu t} \nu M \le e^{\nu t}(q_1 + q_2), \tag{20}$$

which gives

$$\frac{d(Me^{\nu t})}{\mathrm{dt}} \le (q_1 + q_2)e^{\nu t}. \tag{21}$$

Integrating on both sides yields

$$Me^{\nu t} \le \frac{qa_1 + q_2}{\nu} e^{\nu t} + B. \tag{22}$$

Multiplying the equation by $e^{-\nu t}$, we get

$$M(t) \le \frac{q_1 + q_2}{\nu} + Be^{-\nu t}. \tag{23}$$

Using the initial condition $t = 0, M(t = 0) = M_0$, then, we will get

$$M_0 - \frac{q_1 + q_2}{\nu} \le B. \tag{24}$$

Substituting the constant, we get

$$M(t) \le \frac{(q_1 + q_2)}{\nu} + \left(M_0 - \frac{q_1 + q_2}{\nu}\right) e^{-\nu t}. \tag{25}$$

When $M_0 > ((q_1 + q_2)/\nu)$, viruses through the media (M) decrease asymptotically to $M_0 > ((q_1 + q_2)/\nu)$, and when $M < q_1 + q_2/\nu$, viruses through the media increase asymptotically to $q_1 + q_2/\nu$.

Hence, the feasible solutions of the system enter the region

$$\Omega_M = \left\{ M : M \le \mathrm{Max}\left\{ M_0, \frac{q_1 + q_2}{\nu} \right\} \right\}. \tag{26}$$

3.2. Positivity of the Solution. All variables and parameters of the model must be nonnegative $\forall t \ge 0$. We now solve the equations for testing the positivity.

Theorem 2. *Let the initial values of the system (1a)–(1g) be* $(S(0) > 0 \quad and \quad (E(0), I_s(0), I_a(0), Q(0), R(0), M(0)) \ge 0$. *Then, the solution set* $S(t), E(t), Q(t), I_s(t), I_a(t)$, $R(t)$, *and* $M(t)$ *are positive* $\forall t \ge 0$.

Proof. We will prove each equation from our COVID-19 system 2.3.

Using the first equation in the system, we have

$$\begin{aligned} &\frac{\mathrm{dS}}{\mathrm{dt}} = \vartheta + kQ + \omega R - (\lambda_1 I_s + \lambda_2 I_a + \lambda_3 M + \mu)S \ge -(\lambda_1 I_s + \lambda_2 I_a + \lambda_3 M + \mu)S, \\ &\frac{\mathrm{dS}}{\mathrm{dt}} \ge -(\lambda_1 I_s + \lambda_2 I_a + \lambda_3 M + \mu)S. \end{aligned} \tag{27}$$

Integration yields

$$S \ge S_0 e^{-\int_0^t (\lambda_1 I_s + \lambda_2 I_a + \lambda_3 M + \mu) d\tau} > 0, \tag{28}$$

since

$$e^{-\int_0^t (\lambda_1 I_s + \lambda_2 I_a + \lambda_3 M + \mu) d\tau} > 0. \tag{29}$$

From the second equation, we have

$$\frac{\mathrm{dE}}{\mathrm{dt}} = (\lambda_1 I_s + \lambda_2 I_a + \lambda_3 M)S - (\rho_1 + \rho_2 + \eta + \mu)E. \tag{30}$$

Thus

$$\frac{\mathrm{dE}}{\mathrm{dt}} \ge -(\rho_1 + \rho_2 + \eta + \mu)E. \tag{31}$$

Integration yields

$$E \ge E_0 e^{-(\rho_1 + \rho_2 + \eta + \mu)t} > 0, \tag{32}$$

since

$$e^{-(\rho_1 + \rho_2 + \eta + \mu)} > 0. \tag{33}$$

From the third equation of system (1a)–(1g), we have

$$\frac{dQ}{dt} = \xi + \eta E - (k + \psi + \mu)Q. \tag{34}$$

Thus

$$\frac{dQ}{dt} \geq -(k + \psi + \mu)Q. \tag{35}$$

Integrating, we get

$$Q \geq Qe^{-(k+\psi+\mu)t} > 0, \tag{36}$$

since

$$e^{-(k+\psi+\mu)t} > 0. \tag{37}$$

Fourth equation of the system, we will have

$$\frac{dI_s}{dt} = \rho_1 E + \Phi\psi Q - (\pi_1 + \mu + \sigma)I_s. \tag{38}$$

Thus

$$\frac{dI_s}{dt} \geq -(\pi_1 + \mu + \sigma)I_s. \tag{39}$$

Integrating, we get

$$I_s \geq I_{s0}e^{-(\pi_1+\mu+\sigma)t} > 0, \tag{40}$$

since

$$e^{-(\pi_1+\mu+\sigma)} > 0. \tag{41}$$

Fifth equation of the system, we will have

$$\frac{dI_a}{dt} = \rho_2 E + (1 - \Phi)\psi Q - (\pi_2 + \mu + \sigma)I_a. \tag{42}$$

Thus

$$\frac{dI_a}{dt} \geq -(\pi_2 + \mu + \sigma)I_a. \tag{43}$$

Integrating, we get

$$I_a \geq I_{s0}e^{-(\pi_2+\mu+\sigma)t} > 0, \tag{44}$$

since

$$e^{-(\pi_2+\mu+\sigma)} > 0. \tag{45}$$

Six equation of the system (1a)–(1g), we have

$$\frac{dR}{dt} = \pi_1 I_s + \pi_2 I_a - (\omega + \mu)R. \tag{46}$$

Thus

$$\frac{dR}{dt} \geq -(\omega + \mu)R. \tag{47}$$

Integrating, we get

$$R \geq R_0 e^{-(\omega+\mu)t} > 0, \tag{48}$$

since

$$e^{-(\omega+\mu)} > 0. \tag{49}$$

□

3.2.1. For Media. Media is presented by the last equation of the system (1a)–(1g); we will have

$$\frac{dM}{dt} = q_1 \frac{I_s}{N} + q_2 \frac{I_a}{N} - \nu M. \tag{50}$$

Then, we will have

$$\frac{dM}{dt} \geq -\nu M. \tag{51}$$

Integrating, we get

$$M \geq M_0 e^{-\nu t} > 0, \tag{52}$$

since

$$e^{-\nu t} > 0. \tag{53}$$

4. Model Analysis

In this section, we examine the existence of equilibrium states, reproduction number, and stability of the equilibrium states.

4.1. Disease Free Equilibrium. The model has a disease-free equilibrium which is obtained by setting $E = Q = I_a = I_s = R = 0$ and $M = 0$. We substitute the above into the system (1a)–(1g) which are the systems for COVID-19 dynamics in human beings and transmission media and/or environment. Then, we have the disease free-equilibrium point of the entire system as given in

$$X_0\left(S^0, E^0, Q^0, I_a^0, I_s^0, R^0, M^0\right) = \left(\frac{\vartheta}{\mu}, 0, 0, 0, 0, 0, 0\right). \tag{54}$$

4.2. Basic Reproduction Number R_0. Basic reproduction number is the expected number of secondary cases produced by a single infectious individual during the entire infectious period of that particular individual in a completely susceptible population. The epidemiological criterion of R_0 is that if $R_0 < 1$, then the single infected individual in entirely susceptible population infects less than one individual. Hence, the disease may be eradicated from the population, and the disease-free equilibrium point is asymptotically stable. That is, the disease cannot invade the population. If $R_0 > 1$, it

means that a single infected individual in entirely susceptible population infects more than one individual. Hence, the disease may persist in the population, and the disease-free equilibrium point is unstable. In this case, the disease can invade the population and persist for a long time. If $R0 = 1$, it means that a single infected individual in entirely susceptible population infects one new individuals. Hence, the disease will stay alive in the population without an epidemic [25].

We use next-generation method as described by [26] to find the basic reproductive number. Consider a heterogeneous population whose individuals are distinguishable by stage of the disease and hence identifiable and put into epidemiological compartments S, E, Q, I_a, I_s, R and M. By first rearranging the system to have the infection classes come first, we sort the compartments so that the first m compartments correspond to infected individuals.

We now let $F_i(x)$ be the rate of appearance of new infections in compartment i, $V_i^+(x)$ be the rate of transfer of individuals into compartment i by all other means except the epidemic, and $V_i^-(x)$ be the rate of transfer of individuals out of compartment i.

The disease transmission model consists of the system of equations

$$x_i' = F_i(x) - V_i(x), \tag{55}$$

where $V_i(x) = V_i^-(x) - V_i^+(x)$.

Since we already have the disease free equilibrium x_0, we then compute matrices F and V which are $m \times m$ matrices defined by

$$F = \left(\frac{\partial F_i}{\partial x_j}(x_0)\right), V = \left(\frac{\partial V_i}{\partial x_j}(x_0)\right), \tag{56}$$

with $1 \le i, j \le m$.

Since F is nonnegative and V is a nonsingular matrix, then, V^{-1} is nonnegative, and also FV^{-1} is nonnegative. Matrix FV^{-1} is defined as the next-generation matrix [27]. Therefore, the basic reproductive number is defined as

$$R_0 = \rho\left(FV^{-1}\right), \tag{57}$$

where $\rho(FV^{-1})$ is the maximum modulus of the eigenvalues of the nonnegative matrix FV^{-1}.

We first rearrange the system to have the infection classes come first:

$$\frac{dE}{dt} = (\lambda_1 I_s + \lambda_2 I_a + \lambda_3 M)S - (\rho_1 + \rho_2 + \eta + \mu)E, \tag{58a}$$

$$\frac{dI_s}{dt} = \rho_1 E + \Phi\psi Q - (\pi_1 + \mu + \sigma)I_s, \tag{58b}$$

$$\frac{dI_a}{dt} = \rho_2 E + (1 - \Phi)\psi Q - (\pi_2 + \mu + \sigma)I_a, \tag{58c}$$

$$\frac{dM}{dt} = q_1 \frac{I_s}{N} + q_2 \frac{I_a}{N} - \nu M, \tag{58d}$$

$$\frac{dQ}{dt} = \xi + \eta E - (k + \psi + \mu)Q, \tag{58e}$$

$$\frac{dR}{dt} = \pi_1 I_s + \pi_2 I_a - (\omega + \mu)R, \tag{58f}$$

$$\frac{dS}{dt} = \vartheta + kQ + \omega R - (\lambda_1 I_s + \lambda_2 I_a + \lambda_3 M + \mu)S. \tag{58g}$$

Now from the system (58a)–(58g), the infectious classes are (58a) to (58e) with compartment E, Q, I_s, I_a and M, and this will now yield

$$\mathbf{F_i} = \begin{pmatrix} (\lambda_1 I_s + \lambda_2 I_a + \lambda_3 M)S \\ \xi \\ 0 \\ 0 \\ 0 \end{pmatrix},$$
$$\mathbf{V_i} = \begin{pmatrix} (\rho_1 + \rho_2 + \eta + \mu)E \\ (k + \psi + \mu)Q - \eta E \\ (\pi_1 + \mu + \sigma)I_s - \rho_1 E - \Phi\psi Q \\ (\pi_2 + \mu + \sigma)I_a - \rho_2 E - (1 - \Phi)\psi Q \\ \nu M - q_1 \frac{I_s}{N} - q_2 \frac{I_a}{N} \end{pmatrix}. \tag{59}$$

We compute Jacobian matrices of F and V at x_0. For F, we will have

$$\frac{\partial F_i}{\partial x_j} = \begin{pmatrix} \frac{\partial F_1}{\partial E} & \frac{\partial F_1}{\partial Q} & \frac{\partial F_1}{\partial I_s} & \frac{\partial F_1}{\partial I_a} & \frac{\partial F_1}{\partial M} \\ \frac{\partial F_2}{\partial E} & \frac{\partial F_2}{\partial Q} & \frac{\partial F_2}{\partial I_s} & \frac{\partial F_2}{\partial I_a} & \frac{\partial F_2}{\partial M} \\ \frac{\partial F_3}{\partial E} & \frac{\partial F_3}{\partial Q} & \frac{\partial F_3}{\partial I_s} & \frac{\partial F_3}{\partial I_a} & \frac{\partial F_3}{\partial M} \\ \frac{\partial F_4}{\partial E} & \frac{\partial F_4}{\partial Q} & \frac{\partial F_4}{\partial I_s} & \frac{\partial F_4}{\partial I_a} & \frac{\partial F_4}{\partial M} \\ \frac{\partial F_5}{\partial E} & \frac{\partial F_5}{\partial Q} & \frac{\partial F_5}{\partial I_s} & \frac{\partial F_5}{\partial I_a} & \frac{\partial F_5}{\partial M} \end{pmatrix} = \begin{pmatrix} 0 & 0 & \lambda_1 S & \lambda_2 S & \lambda_3 S \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \end{pmatrix}. \tag{60}$$

Now, at x_0, we will have

$$\mathbf{F} = \begin{pmatrix} 0 & 0 & \lambda_1 \frac{\vartheta}{\mu} & \lambda_2 \frac{\vartheta}{\mu} & \lambda_3 \frac{\vartheta}{\mu} \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \end{pmatrix}, \tag{61}$$

and for V, we will have

$$V = \frac{\partial V_i}{\partial x_j}(x_0) = \begin{pmatrix} \frac{\partial V_1}{\partial E} & \frac{\partial V_1}{\partial Q} & \frac{\partial V_1}{\partial I_s} & \frac{\partial V_1}{\partial I_a} & \frac{\partial V_1}{\partial M} \\ \frac{\partial V_2}{\partial E} & \frac{\partial V_2}{\partial Q} & \frac{\partial V_2}{\partial I_s} & \frac{\partial V_2}{\partial I_a} & \frac{\partial V_2}{\partial M} \\ \frac{\partial V_3}{\partial E} & \frac{\partial V_3}{\partial Q} & \frac{\partial V_3}{\partial I_s} & \frac{\partial V_3}{\partial I_a} & \frac{\partial V_3}{\partial M} \\ \frac{\partial V_4}{\partial E} & \frac{\partial V_4}{\partial Q} & \frac{\partial V_4}{\partial I_s} & \frac{\partial V_4}{\partial I_a} & \frac{\partial V_4}{\partial M} \\ \frac{\partial V_5}{\partial E} & \frac{\partial V_5}{\partial Q} & \frac{\partial V_5}{\partial I_s} & \frac{\partial V_5}{\partial I_a} & \frac{\partial V_5}{\partial M} \end{pmatrix},$$

$$\mathbf{V} = \begin{pmatrix} \rho_1 + \rho_2 + \eta + \mu & 0 & 0 & 0 & 0 \\ -\eta & (k + \psi + \mu) & 0 & 0 & 0 \\ -\rho_1 & -\Phi\psi & (\pi_1 + \mu + \sigma) & 0 & 0 \\ -\rho_2 & -(1-\Phi)\psi & 0 & (\pi_2 + \mu + \sigma) & 0 \\ 0 & 0 & \frac{q_1\mu}{\vartheta} & \frac{q_2\mu}{\vartheta} & \nu \end{pmatrix}. \tag{62}$$

We can obtain V^{-1} and FV^{-1} using maple, and then, the spectral radius which is also the basic reproduction number is shown as in

$$R_0 = \frac{\Phi\eta\psi\nu\lambda_1}{\mu a_2 a_1 a_3} + \frac{\nu\lambda_1\rho_1}{\mu a_1 a_3} + \frac{\eta\psi\nu(1-\Phi)\lambda_2}{\mu a_2 a_1 a_4} + \frac{\nu\lambda_2\rho_2}{\mu a_1 a_4} + \frac{\Phi\eta\psi q_1\lambda_3}{a_2 a_1 a_3 \nu} + \frac{\eta\psi q_2(1-\Phi)\lambda_3}{a_2 a_1 a_4 \nu} + \frac{q_2\lambda_3\rho_2}{a_1 a_4 \nu} + \frac{q_1\lambda_3\rho_1}{a_1 a_3 \nu}, \tag{63}$$

where

$$a_1 = \rho_1 + \rho_2 + \eta + \mu, \tag{64a}$$

$$a_2 = k + \psi + \mu, \tag{64b}$$

$$a_3 = \pi_1 + \mu + \sigma, \tag{64c}$$

$$a_4 = \pi_2 + \mu + \sigma. \tag{64d}$$

5. Existence and Stability of the Critical Points

In this section, we assess the existence of equilibrium states and stability of the equilibrium states of the system (1a)–(1g). The model has a disease-free equilibrium in which we set all infectious compartment and the derivatives equal to zero. Then, we have the disease free-equilibrium point as given in

$$X_0\left(S^0, E^0, Q^0, I_a^0, I_s^0, R^0, M^0\right) = \left(\frac{\vartheta}{\mu}, 0, 0, 0, 0, 0, 0\right). \tag{65}$$

5.1. Local Stability of the Disease-Free Equilibrium Point. In this section, we examine the local stability analysis of the disease-free equilibrium point of the COVID-19 disease system. We analyze the local stability of the disease-free equilibrium point using the Jacobian method in which all equations in system (1a)–(1g) are considered and analyzed at the disease free equilibrium X_0. In this method, we compute and examine the eigenvalues of Jacobian matrix of the COVID-19 system to prove that the DFE is locally and asymptotically stable. We are required to show that all real parts of the eigenvalues at X_0 are negative. Now, in order to attest that the eigenvalues are negative, we need to prove the general condition that the determinant and the trace of the Jacobian matrix are positive and negative, respectively [28].

Now, the Jacobian matrix of the system (1a)–(1g) at X_0 is given by

$$\mathbf{J}(\mathbf{X_0}) = \begin{pmatrix} -\mu & 0 & k & -\frac{\lambda_1\vartheta}{\mu} & -\frac{\lambda_2\vartheta}{\mu} & \omega & \frac{\lambda_3\vartheta}{\mu} \\ 0 & -a_1 & 0 & \frac{\lambda_1\vartheta}{\mu} & \frac{\lambda_2\vartheta}{\mu} & 0 & \frac{\lambda_3\vartheta}{\mu} \\ 0 & \eta & -a_2 & 0 & 0 & 0 & 0 \\ 0 & \rho_1 & \Phi\psi & -a_3 & 0 & 0 & 0 \\ 0 & \rho_1 & (1-\Phi)\psi & 0 & -a_4 & 0 & 0 \\ 0 & 0 & 0 & \pi_1 & \pi_2 & -(\omega+\mu) & 0 \\ 0 & 0 & 0 & \frac{q_1\mu}{\vartheta} & \frac{q_2\mu}{\vartheta} & 0 & -\nu \end{pmatrix}, \tag{66}$$

where a_1, a_2, a_3, and a_4 are as given in (64a)–(64d)

We now use trace and determinant method to check the stability of the disease-free equilibrium point X_0 in which we need to prove that the trace and the determinant of matrix (66) are negative and positive, respectively.

Then, using maple software, we prove that trace of the matrix (66) is given by

$$\mathbf{Trace} = -\mu - a_1 - a_2 - a_3 - a_4 - (\omega + \mu) - \nu. \tag{67}$$

It is clear that the trace of the matrix (66) is negative. Then, again using maple software, we are able to prove that the determinant of the matrix (66) is positive provided:

$$\frac{\Phi\eta\psi\nu\lambda_1}{\mu a_2 a_1 a_3} + \frac{\nu\lambda_1\rho_1}{\mu a_1 a_3} + \frac{\eta\psi\nu(1-\Phi)\lambda_2}{\mu a_2 a_1 a_4} + \frac{\nu\lambda_2\rho_2}{\mu a_1 a_4} + \frac{\Phi\eta\psi q_1\lambda_3}{a_2 a_1 a_3 \nu} + \frac{\eta\psi q_2(1-\Phi)\lambda_3}{a_2 a_1 a_4 \nu} + \frac{q_2\lambda_3\rho_2}{a_1 a_4 \nu} + \frac{q_1\lambda_3\rho_1}{a_1 a_3 \nu} < 1, \tag{68}$$

where

$$\frac{\Phi\eta\psi\nu\lambda_1}{\mu a_2 a_1 a_3} + \frac{\nu\lambda_1\rho_1}{\mu a_1 a_3} + \frac{\eta\psi\nu(1-\Phi)\lambda_2}{\mu a_2 a_1 a_4} + \frac{\nu\lambda_2\rho_2}{\mu a_1 a_4} + \frac{\Phi\eta\psi q_1\lambda_3}{a_2 a_1 a_3 \nu} + \frac{\eta\psi q_2(1-\Phi)\lambda_3}{a_2 a_1 a_4 \nu} + \frac{q_2\lambda_3\rho_2}{a_1 a_4 \nu} + \frac{q_1\lambda_3\rho_1}{a_1 a_3 \nu}, \tag{69}$$

is the basic reproduction number, R_0.

R_0 measures the average number of secondary infections produced by a typical infectious individual in an entirely

susceptible population. In the case where there are multiple transmission roots like in COVID-19, the basic reproductive number defines the geometric mean of the number of infections per generation [29].

Thus disease-free equilibrium point X_0 is therefore locally asymptotically stable and leads to the following theorem:

Theorem 3. *The disease-free equilibrium X_0 of COVID-19 disease is locally asymptotically stable if $R_0 < 1$ and unstable if $R_0 > 1$.*

5.2. Global Stability of the Disease-Free Equilibrium Point. In this section, we analyze the global stability of the disease-free equilibrium point using Metzler matrix method as stated by [30]. To do this, we first subdivide the general system (1a)–(1g) of COVID-19 disease into transmitting and nontransmitting components.

Now, let $\mathbf{Y_n}$ be the vector for nontransmitting compartment, $\mathbf{Y_i}$ be the vector for transmitting compartment, and $\mathbf{Y_{X_0,n}}$ be the vector of disease free point. Then

$$\begin{aligned}\frac{d\mathbf{Y_n}}{\mathrm{dt}} &= A_1\left(\mathbf{Y_n} - \mathbf{Y_{X_0,n}}\right) + A_2\mathbf{Y_i},\\ \frac{d\mathbf{Y_i}}{\mathrm{dt}} &= A_3\mathbf{Y_i}.\end{aligned} \tag{70}$$

We then have

$$\mathbf{Y_n} = (S, R)^T,\ \mathbf{Y_i} = (E, Q, I_s, I_a, M),\ \mathbf{Y_{X_0,n}} = \left(\frac{\vartheta}{\mu}, 0\right),$$
$$\mathbf{Y_n} - \mathbf{Y_{X_0,n}} = \begin{pmatrix} S_H - \dfrac{\vartheta}{\mu} \\ R_H \end{pmatrix}. \tag{71}$$

In order to prove that the disease-free equilibrium point of the COVID-19 disease is globally stable, we need to show that matrix A_1 has real negative eigenvalues and A_3 is a Metzler matrix in which all off diagonal element must be nonnegative. Referring to (70), we write the general model as

$$\begin{pmatrix} \vartheta + kQ + \omega R - (\lambda_1 I_s + \lambda_2 I_a + \lambda_3 M + \mu)S, \\ \pi_1 I_s + \pi_2 I_a - (\omega + \mu)R. \end{pmatrix} = A_1 \begin{pmatrix} S - \dfrac{\vartheta}{\mu} \\ R \end{pmatrix} + A_2 \begin{pmatrix} E \\ Q \\ I_s \\ I_a \\ M \end{pmatrix},$$

$$\begin{pmatrix} (\lambda_1 I_s + \lambda_2 I_a + \lambda_3 M)S - (\rho_1 + \rho_2 + \eta + \mu)E, \\ \xi + \eta E - (k + \psi + \mu)Q, \\ \rho_1 E + \Phi\psi Q - (\pi_1 + \mu + \sigma)I_s \\ \rho_2 E + (1 - \Phi)\psi Q - (\pi_2 + \mu + \sigma)I_a \\ q_1\dfrac{I_s}{N} + q_2\dfrac{I_a}{N} - \nu M \end{pmatrix} = A_3 \begin{pmatrix} E \\ Q \\ I_s \\ I_a \\ M \end{pmatrix}. \tag{72}$$

We then use the transmitting and nontransmitting element from the general COVID-19 model to get matrices (73):

$$\mathbf{A_1} = \begin{pmatrix} -\mu_1 & \omega \\ 0 & -(\omega + \mu) \end{pmatrix}, \tag{73}$$

$$\mathbf{A_2} = \begin{pmatrix} 0 & k & -\dfrac{\lambda_1\vartheta}{\mu} & -\dfrac{\lambda_2\vartheta}{\mu} & -\dfrac{\lambda_3\vartheta}{\mu} \\ 0 & 0 & \pi_1 & \pi_2 & 0 \end{pmatrix}, \tag{74}$$

$$\mathbf{A_3} = \begin{pmatrix} -a_1 & 0 & \dfrac{\lambda_1\vartheta}{\mu} & \dfrac{\lambda_2\vartheta}{\mu} & \dfrac{\lambda_3\vartheta}{\mu} \\ 0 & -a_2 & 0 & 0 & \\ \rho_1 & \Phi\psi & -a_3 & 0 & 0 \\ \rho_2 & (1 - \Phi)\psi & 0 & -a_4 & 0 \\ 0 & 0 & \dfrac{q_1\mu}{\vartheta} & \dfrac{q_2\mu}{\vartheta} & -\nu \end{pmatrix}. \tag{75}$$

Considering matrix A_1, it is clear through computation that the eigenvalues are real and negative, which now confirms that the system

$$\frac{d\mathbf{Y_n}}{\mathrm{dt}} = A_1\left(\mathbf{Y_n} - \mathbf{Y_{X_0,n}}\right) + A_2\mathbf{Y_i}, \tag{76}$$

is globally and asymptotically stable at $\mathbf{Y_{X_0}}$.

Considering matrix A_3, it is clear that all its off-diagonal elements are nonnegative, and thus, A_3 is a Metzler stable matrix. Therefore, disease-free equilibrium point for COVID-19 system is globally asymptotically stable, and as a result, we have the following theorem:

Theorem 4. *The disease-free equilibrium point is globally asymptotically stable in X_0 if $R_0 < 1$ and unstable if $R_0 > 1$.*

5.3. Existence of Endemic Equilibrium. In this section, we investigate conditions for the existence of the endemic equilibrium point of the system (1a)–(1g).

The endemic equilibrium point $E^*(S^*, E^*, Q^*, I_s^*, I_a^*, R^*, M^*)$ is obtained by solving the equations obtained by setting the derivatives of (1a)–(1g) equal to zero. We then have system (77a)–(77g) which exist for $R_O > 1$.

$$\vartheta + kQ + \omega R - (\lambda_1 I_s + \lambda_2 I_a + \lambda_3 M + \mu)S = 0, \tag{77a}$$

$$(\lambda_1 I_s + \lambda_2 I_a + \lambda_3 M)S - (\rho_1 + \rho_2 + \eta + \mu)E = 0, \tag{77b}$$

$$\xi + \eta E - (k + \psi + \mu)Q = 0, \tag{77c}$$

$$\rho_1 E + \Phi\psi Q - (\pi_1 + \mu + \sigma)I_s = 0, \tag{77d}$$

$$\rho_2 E + (1 - \Phi)\psi Q - (\pi_2 + \mu + \sigma)I_a = 0, \tag{77e}$$

$$\pi_1 I_s + \pi_2 I_a - (\omega + \mu)R = 0, \tag{77f}$$

$$q_1\frac{I_s}{N} + q_2\frac{I_a}{N} - \nu M = 0. \tag{77g}$$

The existence of the endemic equilibrium points of the model was done using the method proposed by [31, 32]. For the endemic equilibrium to exist, it must satisfy the condition $E \neq 0$ or $Q \neq 0$ or $I_s \neq 0$ or $I_a \neq 0$ or $M \neq 0$; that is, $S > 0$ or $E > 0$ or $Q > 0$ or $I_s > 0$ or $I_a > 0$ or $R > 0$ or $M > 0$ must be satisfied. Now adding system (77a)–(77g), we have

$$\vartheta + \xi - \mu(S + E + I_s + I_a + R) - \sigma(I_s + I_a) + q_1\frac{I_s}{N} + q_2\frac{I_a}{N} - \nu M = 0. \tag{78}$$

But from Equation (77g), we have $q_1(I_s/N) + q_2(I_a/N) - \nu M = 0$,

and $S + E + I_s + I_a + R = N$. It follows that

$$\vartheta + \xi = \mu N + \sigma(I_s + I_a). \tag{79}$$

Now since $\vartheta + \xi > 0$, $\mu > 0$, and $\sigma > 0$, we can discern that $\mu N > 0$ and $\sigma(I_s + I_a) > 0$ implying that $S > 0$, $E > 0$, $Q > 0$, $I_s > 0$, $I_a > 0$, $R > 0$, and $M > 0$.

This prove that the endemic equilibrium point of the COVID-19 disease exists.

5.4. Global Stability of Endemic Equilibrium Point. In this section, we determine the conditions under which the endemic equilibrium points are stable or unstable. We investigate whether the solution remain close to the equilibrium points whenever the initial condition starting arbitrarily sufficiently close to the equilibrium points $t \longrightarrow \infty$.

As postulated in the study by [26], we assert that the local stability of the disease-free equilibrium advocates for local stability of the endemic equilibrium for the reverse condition. We thus find the global stability of endemic equilibrium using a Korobeinikov approach as described by [26, 33, 34].

We formulate a suitable Lyapunov function for COVID-19 model as given in the form

$$V = \sum a_i(y_i - y_i^* \ln y_i), \tag{80}$$

where a_i is defined as a properly selected positive constant, y_i defines the population of the i^{th} compartment, and y_i^* is the equilibrium point.

We will then have

$$\begin{aligned} V = {} & W_1(S - S^* \ln S) + W_2(E - E^* \ln E) + W_3(Q - Q^* \ln Q) \\ & + W_4(I_s - I_s^* \ln I_s) + W_5(I_a - I_a^* \ln I_a) + W_6(R - R^* \ln R) \\ & + W_7(M - M^* \ln M). \end{aligned} \tag{81}$$

The constants W_i are nonnegative in Λ such that $W_i > 0$ for $i = 1, 2, 3, \cdots, 7$. The Lyapunov function V together with its constants $W_1, W_2, \cdots, W_7$ chosen in such a way that V is continuous and differentiable in a space.

We then compute the time derivative of V from which we get

$$\begin{aligned} \frac{dV}{dt} = {} & W_1\left(1 - \frac{S^*}{S}\right)\frac{dS}{dt} + W_2\left(1 - \frac{E^*}{E}\right)\frac{dE}{dt} + W_3\left(1 - \frac{Q^*}{Q}\right)\frac{dQ}{dt} \\ & + W_4\left(1 - \frac{I_s^*}{I_s}\right)\frac{dI_s}{dt} + W_5\left(1 - \frac{I_a^*}{I_a}\right)\frac{dI_a}{dt} + W_6\left(1 - \frac{R^*}{R}\right)\frac{dR}{dt} \\ & + W_7\left(1 - \frac{M^*}{M}\right)\frac{dM}{dt}. \end{aligned} \tag{82}$$

Now using the COVID-19 system (1a)–(1g), we will have

$$\begin{aligned} \frac{dV}{dt} = {} & W_1\left(1 - \frac{S^*}{S}\right)[\vartheta + kQ + \omega R - (\lambda_1 I_s + \lambda_2 I_a + \lambda_3 M + \mu)S] \\ & + W_2\left(1 - \frac{E^*}{E}\right)[(\lambda_1 I_s + \lambda_2 I_a + \lambda_3 M)S - (\rho_1 + \rho_2 + \eta + \mu)E] \\ & + W_3\left(1 - \frac{Q^*}{Q}\right)[\xi + \eta E - (k + \psi + \mu)Q] \\ & + W_4\left(1 - \frac{I_s^*}{I_s}\right)[\rho_1 E + \Phi\psi Q - (\pi_1 + \mu + \sigma)I_s] \\ & + W_5\left(1 - \frac{I_a^*}{I_a}\right)[\rho_2 E + (1 - \Phi)\psi Q - (\pi_2 + \mu + \sigma)I_a] \\ & + W_6\left(1 - \frac{R^*}{R}\right)[\pi_1 I_s + \pi_2 I_a - (\omega + \mu)R] \\ & + W_7\left(1 - \frac{M^*}{M}\right)\left[q_1\frac{I_s}{N} + q_2\frac{I_a}{N} - \nu M\right]. \end{aligned} \tag{83}$$

At endemic equilibrium point after the substitution and simplification into time derivative of V, we get

$$\begin{aligned} \frac{dV}{dt} = {} & -W_1\left(1 - \frac{S^*}{S}\right)^2 - W_2\left(1 - \frac{E^*}{E}\right)^2 - W_3\left(1 - \frac{Q^*}{Q}\right)^2 \\ & - W_4\left(1 - \frac{I_s^*}{I - s}\right)^2 - W_5\left(1 - \frac{I_a^*}{I_a}\right)^2 - W_6\left(1 - \frac{R^*}{R}\right)^2 \\ & - W_7\left(1 - \frac{M^*}{M}\right)^2 + F(S, E, Q, I_s, I_a, R, M), \end{aligned} \tag{84}$$

where the function $F(S, E, Q, I_s, I_a, R, M)$ is nonpositive. Now following the procedures by [35, 36], we have $F(S, E, Q, I_s, I_a, R, M) \leq 0$ for all S, E, Q, I_s, I_a, R, M. Then, $dV/dt \leq 0$ for all S, E, Q, I_s, I_a, R, M, and it is zero when $S = S^*, E = E^*, Q = Q^*, I_s = I_s^*, I_a = I_a^*, R = R^*, M = M^*$. Hence, the largest compact invariant set in S, E, Q, I_s, I_a, R, M such that $dV/dt = 0$ is the singleton E^* which is endemic equilibrium point of the model system (1a)–(1g).

Using LaSalles's invariant principle postulated by [37], we assert that E^* is globally asymptotically stable in the interior of the region of S, E, Q, I_s, I_a, R, M and thus leads to the Theorem 5.

Theorem 5. *If $R_0 > 1$, then, the COVID-19 disease model system (1a)–(1g) has a unique endemic equilibrium point E^* which is globally asymptotically stable in S, E, Q, I_s, I_a, R, M.*

6. Sensitivity Analysis

Sensitivity analysis is commonly used to discover parameters that have a high impact on R_0 and should be targeted by intervention strategies. The normalized forward sensitivity index is the ratio of relative change of a variable to the relative change in parameter [38]. If the variable is a differentiable function of the parameter, then, the sensitivity index is defined as follows:

Definition 6. The normalized forward sensitivity index of variable g that depends on parameter b is defined as

$$\Gamma_b^g = \frac{\partial g}{\partial b} \times \frac{b}{g}. \tag{85}$$

By using the same procedure, the sensitivity indices of R_0 are computed with respect to all parameters embedded to R_0 . Table 2 presents the parameter values and the sensitivity result.

As can be seen from the table the parameters σ, ν, μ, λ_2, and λ_1 are the most sensitive parameter. Increasing the value of σ will decrease the value of R_0, and decreasing the value of ν will decrease the value of R_0.

Table 2: Parameter values and sensitivity result.

Parameter	Value	Sensitivity indices
σ	0.089	-0.9652
ν	0.0048	0.7004
μ	0.00021	-0.8544
λ_2	0.0025	0.5636
λ_1	0.0025	0.2866
ψ	0.04	0.1755
κ	0.44	-0.1747

7. Numerical Results and Discussion

This section presents the results of the analysis for the dynamics of COVID-19 model. We depict the transmission of the disease in human population using numerical simulations. The fourth-order Runge–Kutta numerical scheme coded in MatLab programming language is used. The parameter values that we have used for numerical simulations are presented in Table 3. In simulation of the model, most of the parameter values were obtained from literature that are related to the same study and others were assumed. Unless otherwise stated, the parameter values appeared in Table 3 are used during the simulation process.

If no interventions are considered on the dynamics disease, the transmission and spread of the disease would increase significantly as the force of infection will increase. Without intervention, the model suggests that the dynamics of the COVID-19 virus depend on the force of infection, natural immunity, and the lifestyle of the considered population towards the spread of COVID-19 disease. Figure 2 shows the increase of infection in both symptomatic and asymptomatic in early weeks due to the increased force of infection and the rapid spread of the disease. The increase in the number of infectious people consequently reduces the number of susceptible, exposed, and quarantined populations as most of the people in these compartment will become infectious after the adequate contact with the fellow infectious individual and/or media. The system then goes to its stable solution as the number of susceptible people becoming infectious decreases to the lowest level and the infectious population increases to the saturation level.

The transmission of COVID-19 in symptomatic and asymptomatic population can be reduced by applying different protective measures. Preventing the exponential spread of COVID-19 highly depends on the use of effective protective measures. The protective measures include social distancing, hand hygiene, wearing mask, and using gloves whenever it is necessary. Reducing the transmission rate of newly infected population by applying protective measures will reduce the number of infected people in both the symptomatic and asymptomatic populations. As the transmission from symptomatic infectious (λ_1) and asymptomatic(λ_2) are sensitive parameters, reducing them sufficiently will reduce the transmission of the virus as shown in Figure 3. The protective measures are expected to be taken by all individuals using the guidelines prepared by WHO [42]. The measures can be achieved through a combination of public health measures. For treating infected people, rapid identification and management of the cases are very important. In addition, follow up of exposed people, infection prevention and control in health-care settings, implementation of health measures for travelers, awareness-raising in the population, and risk communication play important role to reduce both symptomatic and asymptomatic infections.

The challenge of COVID-19 transmission through contaminated surfaces and environments suggests opportunities for cleaning and disinfecting and changes in human behavior [43]. Figure 4 shows the exponential decrease of the viruses in media to its saturation level. The rapid decrease of the viruses suggests the presence of factors that are unfavorable for virus. As the COVID-19 infection is a pandemic, surface-to-surface communicable disease is very fast and has short existence. The effectiveness of preventive measures completely relies on the strength of surface disinfectants within the given period of time. Inanimate surfaces are the most prone site for the transmission of COVID-19 infection. Depending upon the nature of the surface, temperature, and relative humidity of the surrounding, virus persistence time varies from 1 to 9 days [44]. The persistence time in different inanimate subjects is different. The high-risk surface areas need to be cleaned or disinfected frequently with a suitable disinfectant.

The fact that COVID-19 may be transmitted through airborne transmission makes it a very dangerous disease that

Table 3: Parameters values used in simulation.

Parameter	Value	Source
ϑ	0.99	Assumed
λ_1	0.0025	[39]
λ_2	0.0025	[39]
λ_3	0.0045	[39]
k	0.44	Assumed
μ	0.00021	Assumed
σ	0.089	[40]
ξ	0.904	Assumed
η	0.07	[40]
ρ_1	0.05	[40]
ρ_2	0.07	[40]
ψ	0.04	Assumed
π_1	0.003	Assumed
π_2	0.003	Assumed
ω	0.035	Assumed
q_1	0.99	Assumed
q_2	0.99	Assumed
Φ	0.05	[41]
ν	0.0048	Assumed

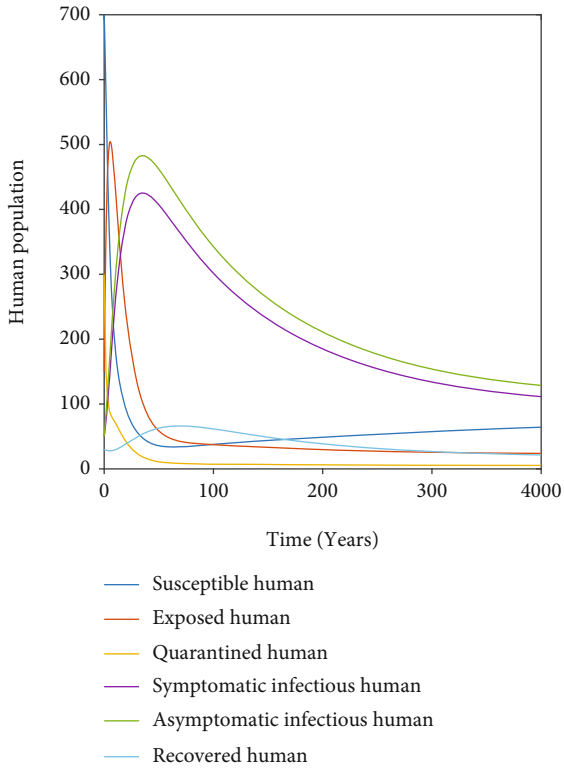

Figure 2: Dynamics of COVID-19 in human population.

needs an effective control plan. Figure 5 shows the role played by the viruses in a media in fostering the transmission and spread of COVID-19. The results show that even though the transmission rate has decreased since the force of infection is only through virus in a media while other roots of infection are ignored, the role of virus in a media in the disease transmission is still significant. If we assume an environment where the virus in a media can survive, and capable of transmitting the disease, the transmission and spread of COVID-19 disease would be extreme to the extent that may lead to extinction of the whole considered human population.

The results show how quick the spread may rise in the early weeks which makes this disease one of global concern. Virus transfer through contaminated surfaces makes COVID-19 a global problem as it increases its transmission capacity to the people in the considered community. Human natural movement and/or migration behavior is another factor that increased the spread of the disease to the outer communities and as a result it globalizes the disease.

This study has shown the important role of virus transmission through contaminates' surfaces. It increases the transmission capacity of the disease even if the human-to-human transmission is controlled. This factor justifies the subtle increase of the COVID-19 cases in areas where the human-to-human transmission is controlled. The results also justify the importance of following the health precaution and procedures to reduce the spread and transmission of COVID-19 in families, communities, and the world at large.

8. Discussion and Conclusion

Currently, COVID-19 is said to have affected all the continents, and it has spread rapidly around the globe to more than 188 countries and territories around the world. The rate at which the virus spread is alarming, and it has become a global public health burden. After the first case of the virus reported from China, on December 2020, the incidence has been increasing from time to time around the globe.

African countries are at high risk of COVID-19 due to dense population and dense traffic between China and Africa. The WHO also identified 13 top priority countries including most of East African Countries. Some countries in Europe like Italy, Spain, and France seem to pass the peak in the last few weeks. It is believed that the second phase of COVID-19 has large impact on countries in Africa.

Our simulation results show the increase of infection in both symptomatic and asymptomatic cases in early weeks due to the increased force of infection and the rapid spread of the disease. As African countries have fewer and less advanced health facilities, applying protective measures is very important. Human natural movement and/or migration behavior is another factor that increased the spread of the disease to the outer communities, and as a result of this, the disease could be globalized. The issue of quarantining people on arrival is very important. From the sensitivity result, the rate of identifying whether an immigrant is infected with COVID-19 or not affect the transmission of

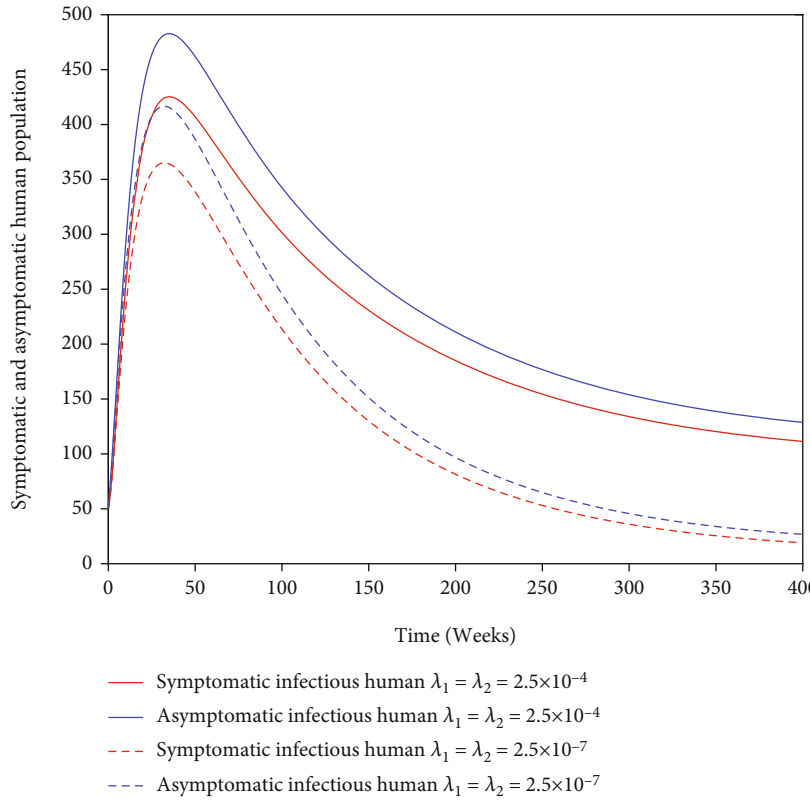

Figure 3: The effect of symptomatic and asymptomatic transmission.

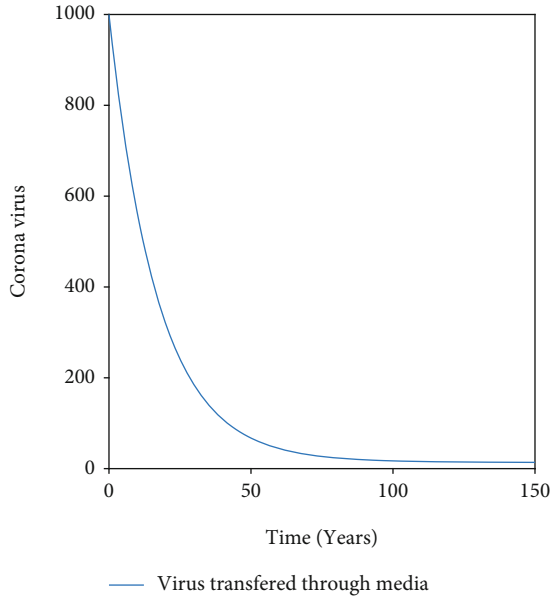

Figure 4: Dynamics of COVID-19 viruses.

the virus very high. As the transmission of the virus escalates through the flow of people across the continent, the rate of quarantine is one of the measures that should be applied on arrival.

The corona viruses in media face a lot of removal challenges that are caused by human behavior and the environment. According to our result of sensitivity analysis, the rate of removal of COVID-19 virus from surfaces is the most sensitive parameter which highly affects the basic reproduction number R_0. This can be done by applying disinfectants and sanitizing surfaces which are exposed to the virus.

The numerical simulation of our model shows how quick the spread of the virus rise in the early weeks which makes the protection process the future global challenge. The media (infected surfaces and air) highly facilitates the transmission of COVID-19 in the community. This study has shown the important role played by viruses through the media in the transmission and spread of corona virus in human population. It increases the transmission capacity of the disease even if the human-to-human transmission may be controlled. This factor justifies the subtle increase of the COVID-19 cases in areas where the human-to-human transmission is controlled. The results also justify the importance of following the health precaution and procedures to reduce the spread and transmission of COVID-19 in families, communities, and the world at large.

We recommend that proper quarantine of people on arrival and the rate of virus removal from surfaces which could be exposed to the virus is very important. Immigrants must stay in a place which is prepared to carefully observe the health condition of individuals who arrived from infected areas. Inconsistent measures towards applying

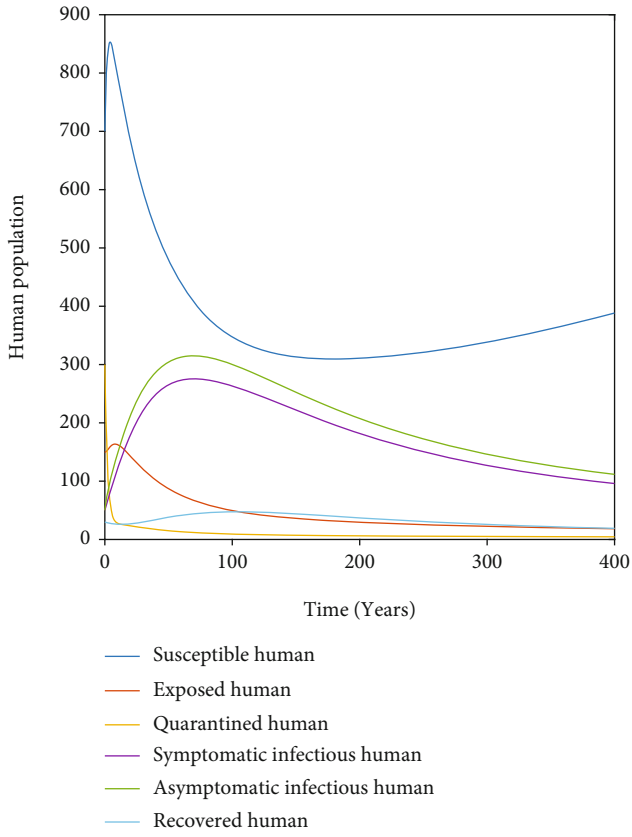

Figure 5: Role of virus in the media in the dynamics of COVID-19.

protective measures will cost the life of individuals and the people around them. COVID-19 virus is a highly transmitted virus; therefore, all individuals are responsible to one another. Stakeholders are advised to motivate people to adhere to COVID-19 protocols.

Data Availability

The authors can provide available data on request.

Conflicts of Interest

The authors declare that they have no conflicts of interest.

Authors' Contributions

Tesfaye Tadesse Ega and Rigobert Charles Ngeleja contributed equally to this work.

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