Modeling the Dynamics of COVID-19 Transmission and the Effects of Prevention Measures Mathematically

Abriham[1], Dejene Abayneh Elias[2]
Department of Mathematics,

Article Info
Received: 28-02-2025 Revised:06-04-2025 Accepted:17-04-2025 Published:27-04-2025

Abstract: World Health Organization (WHO) officials identified new coronavirus illness (COVID-19) as a serious public health concern at the year's conclusion, and they emphasized the need of finding alternatives to pharmaceuticals in combating the disease. Using mathematical modeling using the fundamental (main) compartments, the majority of writers have explored the dynamics of COVID-19 transmission. Here, we provide a mathematical model of COVID-19 transmission dynamics that accounts for almost every situation that can be considered right now. We have also looked at how control and preventative measures have fared. Boundedness, positivity of solutions, stability analysis, epidemic equilibrium point, basic reproduction number, and local stability of the disease free equilibrium are all fundamental features that an infectious disease model should have. The model has fulfilled all of these criteria. To examine the effects of remaining inside, wearing a mask, washing hands often, etc., we included a self-protection parameter, m. There has been an analysis of the effects of quarantine and isolation on the number of exposed, infected, and deceased individuals. Not only that, but researchers have also looked at how symptomatic and asymptomatic people affect the fundamental reproduction number. According to the study's numerical simulations, the government needs to raise the rates of isolation, quarantine, and self-protection. Also, everyone, symptomatic and especially asymptomatic, has to take extreme precautions to prevent the spread of the virus, which will reduce the amount of time when susceptible and asymptotic people come into touch with one another.

Index Terms: COVID-19 Disease, Mathematical Model, prevention and control, Impact, Ethiopia.

1. Introduction

In December 2019, in Wuhan city, Hubei Province, China, scientists made the first detection of the new coronavirus disease (COVID-19), a pandemic that causes respiratory sickness and transmits from person to person by respiratory droplets [1]. The World Health Organization (WHO) classed the COVID-19 pandemic as a PHEIC on January 30, 2020 [2]. Colds, pneumonia, and SARS are some of the human respiratory illnesses often linked to the novel coronavirus [3]. The fast global spread of COVID-19 since December 2019 began in China. There have been 40,235 confirmed cases as of February 9, 2020, with 908 fatalities (2.3%), 6,484 seriously sick patients (16.1%), and 23,589 suspected cases spanning 34 provinces, regions, and municipal cities in China [4]. Among the 213 nations affected, Ethiopia has 112 confirmed cases and 3 fatalities as of April 25, 2020 [5]. The overall number of confirmed cases was 2,686,785. American, Italian, Spanish, Chinese, German, Brazilian, and French citizens are among the most impacted [6].
In the last twenty years, two coronavirus outbreaks—SARS in 2003 and MERS in 2012—have caused widespread concern about public health throughout the world. More individuals have died from COVID-19 than from SARS and MERS put together, which is shocking and terrible [7, 8]. At the same time, mathematical modeling of infectious disease transmission dynamics has long been an important area of research for scientists. An essential tool for evaluating the possible impacts of controlled and preventative actions, particularly while their features are still unknown, mathematical models in epidemiology provide light on the dynamics of epidemics [9,10].
A number of COVID-19 vaccines have made it through animal testing, and now the first round of injections into humans is under way, after scientists have been rushing to produce more vaccines since January 2020. On the other hand, most experts agree that it will take at least twelve to eighteen months for a vaccine to be licensed and effective [11]. Hence, in order to prevent the continuing harm

It is advised to expedite creative research aimed at reducing the epidemic's spread until a vaccine authorized by the WHO is available. In order to learn from the current pandemic response and be better prepared for the next unexpected outbreak, experts from across the globe have endorsed research goals that contribute to global research platforms, in conjunction with the globe Health Organization (WHO) [12].

Because of this, the researchers set out to develop a new model that incorporates all existing COVID-19 control and preventive strategies, assess their influence and efficacy in mitigating the virus's impacts, and ultimately, to forecast the virus's future spread. In addition, the goal of this research is to Create a mathematical model that describes the kinetics of COVID-19 transfer from humans to humans.

Determine the most effective preventative techniques for reducing the risk of infection and mortality. Examine the impact on the basic reproductive number of persons experiencing symptoms and those without symptoms independently.

To reduce the infection rate, provide potential methods of prevention and control. Diseases for which there is currently no cure, such as COVID-19, can be managed in the same way as others, with the goal of reducing transmission through measures such as quarantining suspected victims and isolating those who have been diagnosed with the virus [13]. It is hard to conduct studies to assess potential management options, thus developing mathematical models and using them to generate predictions may be the sole option for trying to compare the efficacy of alternative non-pharmaceutical treatments [14].

Another research method that can compete with a new strong laboratory approach is mathematical modeling [15]. It is possible to compare the findings of experiments with those of mathematical models, which provide a mathematical description of the situation based on assumptions. Predictions from numerical simulations that may be compared with observed data are often necessary for this kind of comparison [16]. It is essential to gather explicit and accepted assumptions about the illness in order to develop a mathematical model on the transmission of infectious diseases [17].

Several research have released mathematical models including early transmission patterns to get a good knowledge of how COVID-19 spreads in host populations since late 2019. In order to depict the dynamics of the epidemic and future numbers, these models are presented using numerous ways. The pioneering Susceptible-Infectious-Recovered (SIR) model, first published in 1927 by Kermack and McKendrick [18], is the basis for the two most popular methods, the Susceptible-Infectious-Recovered/Death (SIRD) model and the Susceptible-Exposed-Infectious-Recovered (SEIR) model.

New coronavirus behavior and transmission dynamics are still a mystery, therefore existing research hasn't taken into account every potential situation. Consequently, as a newly-emerging infectious illness, it is important to comprehend its current clinical and epidemiological features and include them into the model in order to implement prompt control and preventive strategies [19].

Our goal in this work was to examine the dynamics of the ongoing COVID-19 illness in Ethiopia using a deterministic mathematical model using a compartmental modified SEIR technique. This model would include our current knowledge of the disease's evolution and the most recent assumptions. Considering just the human-to-human transmission method, we included quarantine and hospitalization compartments alongside vulnerable, exposed, infected, and recovered ones. This illness has an incubation period of 2–14 days, according to clinical data [3]. Infected persons may still infect others within this window of opportunity, even if they show no symptoms themselves and have no idea they're sick [20]. Consequently, this research has examined the effects of symptomatic and asymptomatic persons separately. We also tried to include the control measures that are already in place in Ethiopia, such as quarantining potential victims and isolating patients who have been diagnosed with the disease, in order to limit transmission.

Also, we included a control parameter that stands for the degree to which people practice self-protection measures. This includes things like how often people wash their hands, how far away they

stand from others, how often they wear face masks, how often they use hand sanitizer, and so on. In an effort to curb the spread of COVID-19, we have, for the most part, implemented almost all of the preventative and control measures implemented by the Ethiopian health system.
To further understand the effects of control and preventative measures, we used mathematical modeling as a tool, using consistent COVID-19 assumptions, and ran the model simulations.
Numerous writers have recently delved into mathematical modeling of infectious disease dynamics, with a focus on COVID-19. The following is a summary of a few relevant papers that have been published among these studies:
To model the new coronavirus's phase-based transmissibility, the authors [21] created a mathematical model. The authors of this research modeled the possible transmission of an infectious agent from its presumed source—bats—to humans using a Bats-Hosts-Reservoir-people transmission network. An evaluation of the COVID-19 transmission level was conducted using the reproductive number, R0, which was computed using the next generation matrix. At [22]

The authors used the Susceptible-Exposed-Symptomatic-Asymptomatic-Hospitalized-Removed model to assess the transmission of the Middle East Respirator Syndrome (MERS) Corona Virus in the Republic of Korea, which is a human-to-human infection. Isolation and quarantine units, which are crucial for reducing infection and mortality rates, are not regarded as a control strategy in this research.
Created a novel mathematical model for COVID-19 epidemic prediction and analysis of its relevance to control and preventive strategies in [23]. Researchers in this research proposed a six-part model: susceptible (those who may be infected), exposed, infected, quarantined, potential victims, and removed (those who have been cured after infection). Here, it's not feasible to distinguish between infected people who are experiencing symptoms and those who aren't, making it hard to assess the effects of the infection on each group independently.
Novel coronavirus pneumonia (NCP) may have a higher transmissibility than SARS, according to data on NCP cases collected from epidemiological investigations or official websites as of February 7, 2020. The authors of the study compared NCP with SARS with the goal of estimating the basic and time-varying transmission dynamics of NCP across China. The study found that the outbreak is being effectively contained. The time-varying reproduction number should ideally be less than one, however this will need persistent efforts. In order to curb the spread of COVID-19, the authors of [25] calculated the efficacy of screening for symptoms and risk factors. The majority of instances that screening misses are really undetected, according to the study's findings. This is because these individuals have not yet shown any symptoms and are therefore unaware that they were exposed. This investigation highlights the need of implementing strategies to reduce the spread of disease when a screening program misses certain people. This suggests that it would be beneficial to examine symptomatic and asymptomatic patients independently.
Taking China's unique features into account, the authors of [26] provided mathematical models of the COVID-19 coronavirus disease's propagation. The authors of this research used compartmental modeling, which included a number of assumptions. The authors of [13] used a data-driven model to explain and predict how COVID-19 will spread. The researchers in this study employed the SEIAHRD model to predict how the epidemic would develop; however, they omitted the data from the quarantine compartment. Using the ordinary differential equation and the delay differential equation, two mathematical models with five compartments are created in [27]. A number of factors, including the number of reported and unreported cases as well as asymptomatic infectious cases, were considered by the developers of this model when they set out to determine how to reduce the impact of the epidemic.
In [28], Anirudh detailed the results and difficulties of using transmission dynamics and several mathematical models to forecast the spread, peak, and decline of COVID-19 cases. Based on the results of this research, it is clear that a model's performance is highly dependent on the inputs provided and the presumed procedure of industry experts. Better prediction and more accurate results will be provided by include more compartments that significantly impact the transmission dynamics, according to this study's results. The authors of [29] put out a mathematical model to study the dynamics of COVID-19 infections; the model details the many ways the virus might spread and

highlights the importance of environmental reservoirs in this process. The SEIRV model is used in this work; the E and I compartments are understood to include infected persons with no symptoms and symptoms, respectively; and V represents the concentration of coronavirus in the environmental reservoir. One disadvantage of this research is that those who were exposed and those who were symptomatic were both included in the same compartment. This makes it impossible to separate the two groups and analyze them separately.
Using COVID-19 as an example, the authors of [30] examined the best way to regulate impulsive SQEIAR pandemic models. Quarantine and treatment of affected individuals were proposed as two measures to eliminate the virus in this research. This is accomplished by applying optimum control theory to the epidemic model, which allows for free terminal optimal time control at an optimal cost. The purpose of this research was to build the SEIR model for COVID-19, conduct stability analysis, and numerically simulate the model's impact on the pandemic's propagation. The authors of the aforementioned study looked at the stability and numerical simulation of the model in [31]. The model is built using the SEIR approach, which takes vaccination and isolation variables into account as model parameters.
Authors developed and evaluated a mathematical model of COVID-19 disease transmission in [32]. The six categories used in this research are as follows: S-susceptible, Se-susceptible but educated to avoid the illness, C-infected but asymptomatic, I-infected and symptomatic, R-recovered, and E-pathogen concentration on contaminated surfaces. The authors updated their risk assessment for the new coronavirus in [33]. The earlier predictions regarding the COVID-19 transmission risk need to be updated because of the substantial changes in control strategies over time and the introduction of detection technologies that have been greatly improved, allowing for a shorter time from infection/symptom onset to diagnosis and faster confirmation of new coronavirus cases. To more accurately measure the treatments' development, the scientists re-calculated the effective daily reproduction ratio.
The authors of [34] simulated several lockdown situations in East Africa and estimated the total and serious illnesses. The SECINISRD model was used in this investigation. The model goes as follows: S = susceptible persons; E = exposed; C = contagious and asymptomatic or minimally symptomatic; IN = infected with moderate symptoms; IS = infected with severe symptoms; R = recovered; and D = dead. Four scenarios were used to make projections in this study: baseline, mild lockdown, hard seclusion, strict seclusion, and ongoing physical separation or isolation if necessary. As a consequence, we show you the projected outcome for Ethiopia both with and without the lockdown. Using publicly accessible data and mathematical modeling, the authors of [35] investigated the dynamics of COVID-19 transmission and projected the spread of the virus in Bangladesh. Here, the transmission dynamics are described using the susceptible-exposed-infectious-recovered-dead and SIERD models. For urban areas bordering wildlife, the authors of [36] developed a deterministic model to compare the efficacy of two rabies management strategies: mass culling and dog vaccination. To determine the effects of dog vaccination and culling, this research used a mathematical model.

2. Materials and Methods

A. Model Formulation

Using a deterministic compartmental method that divides the whole population into seven mutually exclusive groups, we have constructed a mathematical model that takes into account transmission channel (human-to-human infection), infection state, and other relevant factors. Also, the effects on symptomatic and asymptomatic people were predicted and analyzed independently in this research. As far as we are aware, the results and the present method are still missing from the literature. The seven-class structure, universal self-protection parameter, and symptomatic/asymptomatic individual effect analysis and prediction are the key innovations of this study. The model's stability and equilibrium analyses have also been carried out with clarity.
There are seven subsets of the entire population N: SI (susceptible), Q (quarantined), E (exposed), IS (infected and symptomatic), IN (infected but non-symptomatic), H (hospitalization/isolation), and R (recovered). Additionally, non-pharmaceutical procedures called the "self-protection parameter" were used, which includes things like being inside, maintaining a safe distance, washing hands often with soap or sanitizer, and donning masks. Assumptions with time-

dependent state variable notations form the basis of the proposed model:

The number of individuals in the susceptible compartment, where the disease pathogen has not yet infected them but may in the future, is denoted by S(t).
Those who have been exposed to the pathogen but are not yet exhibiting symptoms and are not contagious are said to be in the incubation period, or E(t).
Q(t): Indicates the total number of individuals housed in the restricted area. There is an incubation phase that some infected individuals must go through before any symptoms may be seen. After the full incubation period or after testing negative, persons in the susceptible compartment are restored to the quarantined section if they do not develop symptoms. In every other case, the person will be sent to the infectious state (IS(t)) compartment.
The number of infected persons exhibiting symptoms or clinical indications is denoted by IS(t). When t is measured, IN(t) represents the number of infected persons who do not exhibit any clinical indications or symptoms.
The number of people who were hospitalized (isolated) because of sickness is represented by H(t). The number of people in the recovery compartment who were previously identified as infectious and have now established a natural immunity to the virus is represented by R(t). This research focuses on the fundamental mechanics of transmission between humans.
The whole population, denoted as N, is held constant. That is, N remains constant while the model is simulated. We also have transmission variables that are thought of as time-independent factors, in addition to the state variables mentioned earlier:

Table 1. Model parameters and their corresponding description.

Parameter	Description
B	Natural birth rate
β_S	transmission probability $\mathbf{I_S}$ compartment
β_N	transmission probability $\mathbf{I_N}$ compartment per contact rate
m	Self-protection rate.
α	Inverse of Incubation period for $\mathbf{I_S}$ compartment
γ	Inverse of Incubation period for $\mathbf{I_N}$ compartment
σ	rate of hospitalization from quarantine
θ	Rate of recover from H compartment R
	Natural mortality rate
ε	Disease induced death rate
ω	Rate of Isolation from infected with symptomatic to hospitalization
φ	Rate of quarantine from Exposed
ρ	Rate of susceptible population to quarantine
δ	Rate of quarantine population returned to the susceptible
	Rate of recover from I_N to R
N	Population in million

The above assumptions and descriptions leads to the compartmental diagram in Figure 1.

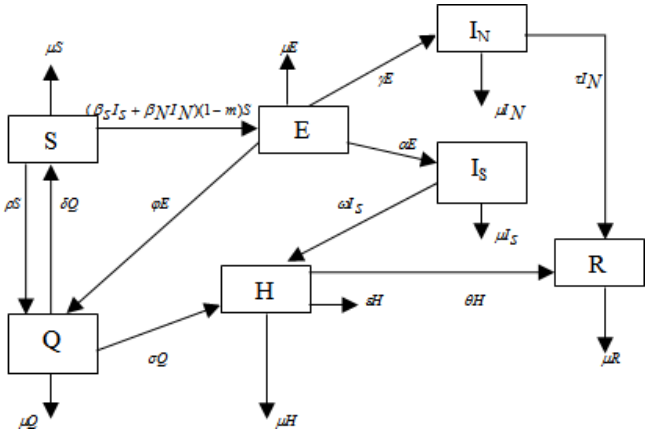


Fig.1. Block diagram representation of the $SEQI_SI_NHR$ dynamical model (1), with seven compartments representation.

Thus, the governing non-linear system of differential equation representing COVID-19 $SEQI_SI_NHR$ model is given

by:

$$\begin{cases} \dfrac{dS}{dt} = B + \delta Q - (\beta_S I_S + \beta_N I_N)(1-m)S - (\rho + \mu)S \\ \dfrac{dE}{dt} = (\beta_S I_S + \beta_N I_N)(1-m)S - (\alpha + \gamma + \phi + \mu)E \\ \dfrac{dQ}{dt} = \rho S + \phi E - (\delta + \sigma + \mu)Q \\ \dfrac{dI_S}{dt} = \alpha E - (\omega + \mu)I_S \\ \dfrac{dI_N}{dt} = \gamma E - (\tau + \mu)I_N \\ \dfrac{dH}{dt} = \sigma Q + \omega I_S - (\theta + \varepsilon + \mu)H \\ \dfrac{dR}{dt} = \theta H + \tau I_N - \mu R \end{cases} \tag{1}$$

$N(t) = S(t) + E(t) + Q(t) + I_S(t) + I_N(t) + H(t) + R(t)$ and satisfies $\frac{dN}{dt} = 0$.

The total population size is denoted by;

Positivity of Solutions

The positivity of solution establishes the non-negativity of solutions of the model under the study. Since the model monitors living population, it is assumed that all the variables and parameters of the model are positive for all $t > 0$ and we expect positive solutions for the model.

Theorem 1:- Suppose the initial values for the state variables are all positive i.e. $S > 0; E \geq 0; Q \geq 0; I_S \geq 0; I_N \geq 0; H \geq 0$ *and* $R \geq 0$ *then the solution* $\{S, E, Q, I_S, I_N, H, R\}$ *of the model system (1) are all positive for all* $t > 0$ *i.e. the model has positive solutions for all the state variables as long as the initial values of the state variables are positive.*

***Proof*:-**The total human populations is given by $N = S(t) + E(t) + Q(t) + I_S(t) + I_N(t) + H(t) + R(t)$ .This implies that for all $t > 0$, $S \leq N, E \leq N, Q \leq N, I_S \leq N, I_N \leq N, H \leq N$ *and* $R \leq N$.: Thus, the above theorem shall be proved and the differential equation for the state variable S from system (1) is;

$$\begin{aligned} \frac{dS}{dt} &= B + ?\delta Q - (\beta_S I_S + \beta_N I_N)(-m)S - (\rho + \mu)S \\ \frac{dS}{dt} &\geq -(\beta_S I_S + \beta_N I_N)(1-m)S - (\rho + \mu)S \\ \frac{dS}{S} &\geq -\left((\beta_S I_S + \beta_N I_N)(1-m) + (\rho + \mu)\right)dt \\ \ln S &\geq -\left((\beta_S I_S + \beta_N I_N)(1-m) + (\rho + \mu)\right)t + c \\ S(t) &\geq A_1 e^{-\left((\beta_S I_S + \beta_N I_N)(1-m) + (\rho + \mu)\right)t} \end{aligned} \tag{2}$$

where $A_1 = e^c$.

At initial time, $t = 0$ and up on substitution into inequality (2), $S(0) \geq A_1$

Thus, inequality (2) becomes;

$$S(0) \geq S(0)\, e^{-\left((\beta_S I_S + \beta_N I_N)(1-m) + (\rho + \mu)\right)t} \geq 0 \text{ , } \forall t \geq 0 \tag{3}$$

Similarly, from the second state variable E in system (1);

$$\frac{dE}{} = (\beta_S I_S + \beta_N I_N)(1-m)S - (\alpha + \gamma + \varphi + \mu)E$$

where

***Proof*: -** The total human population N at any time t is given as; $N(t) = S(t) + E(t) + Q(t) + I_S(t) + I_N(t) + H(t) + R(t)$.

Since the human population can vary with time; $\frac{dN}{dt} = \frac{dS}{dt} + \frac{dE}{dt} + \frac{dQ}{dt} + \frac{dI_S}{dt} + \frac{dI_N}{dt} + \frac{dH}{dt} + \frac{dR}{dt}$, then

The sum of the right side of system (1) gives as

$$\frac{dN}{dt} = B - \mu N(t) - \varepsilon H \tag{11}$$

In the absence of COVD-19 pathogen there is no death ($\varepsilon = 0$). Thus equation (11 became;

$$\frac{dN}{dt} \leq B - \mu N(t) \quad \Rightarrow \quad \frac{dN}{B - \mu N(t)} \leq dt$$

Using Birkhoff and Rota's theorem in [40], as applied in [39]

$$\ln(B - \mu N(t)) \geq t + c_1$$

$$B - \mu N(t) \geq Ce^{-\mu t} \quad \text{where } C = e^{c_1} \tag{12}$$

At initial time, $t = 0$, $N(0) = N_0$ hence $B - \mu N_0 \geq C$

Hence

$$B - \mu N(t) \geq (B - \mu N_0)e^{-\mu t} \tag{13}$$

Rearranging inequality (13) in terms of $N(t)$:

$$N(t) \leq \frac{B}{\mu} + \left(\frac{B - \mu N0}{\mu}\right)e^{-\mu t} \tag{14}$$

As $t \to \infty$ in inequality (14), then the total human population $N(t)$ reduces to $N(t) \leq \frac{B}{\mu}$.

In this regards, all the feasible solutions for the living population in the system (1) exist in the region Ω . That is, the system (1) is bounded.

$$\Omega = \left\{ (S, E, Q, I_S, I_N, H, R) \in R_+^7, \; N(t) \leq \frac{B}{\mu} \right\}$$

Thus, the model has positively invariant set which shows that the model is both biologically and mathematically meaningful in the domain Ω . Hence, every analysis of the dynamics of the flow produced by the model can be considered in Ω .

3. Stability Analysis

Equilibrium Analysis

In epidemiology, models generally have two important equilibriums; the disease-free equilibrium (DFE) and the endemic equilibrium (EE). The DFE requires that there are no infected individuals in the population, or in the case of $SEQI_SI_NHR$ model $E^*, Q^*, I_S^*, I_N^*, H^* = 0$, where the star (*) designates an equilibrium solution. In contrast the endemic equilibrium corresponds to the state in which infected individuals persist indefinitely such that; $E^*, Q^*, I_S^*, I_N^* , H^* > 0$.

Based on equation (1), stability analysis is carried out to determine the disease free equilibrium (DFE) point. To determine this equilibrium point, each equation in system (1), must be equal to zero and the infected compartments are empty. Hence $\frac{dS}{dt} = 0$, $\frac{dE}{dt} =$, $\frac{dIS}{dt} =$, $\frac{dIN}{dt} = 0$, $\frac{dQ}{dt} = 0$, $\frac{dH}{dt} = 0$, $\frac{dR}{dt} = 0$ and $E = Q = I_S = I_N = H = 0$ .Upon simple algebraic manipulation gives us the disease free equilibrium point $\in_0$ of system (1);

$$\in = S^0, E^0, Q^0, I^0, I^0, H^0, R^0 = \left(\frac{\Lambda}{B}, 0, 0, 0, 0, 0, 0\right) \tag{16}$$

Basic Reproduction Number

The basic reproduction number, R_0 is defined as the average number of secondary cases generated by a primary case over his/her infectious period when introduced into a large population of susceptible individuals. The constant R_0 thus measures the initial growth rate of the epidemic.

In order to compute R_0 we use the next generation matrix denoted by FV^{-1} ; where $F = \frac{\partial F_i(\in_0)}{\partial x_j}$ and $V = \frac{\partial V_i(\in_0)}{\partial x_j}$ (Jacobian matrices of F_i and V_i at the equilibrium point) where F_i denotes the new infection terms from the compartments containing the disease and V_i is the remaining transfer terms (both into and out) of the compartments containing the pathogen. In this study there are five compartments involving the pathogen namely; E, Q, I_S, I_N *and* H .Hence $1 \le i \le 5$ $1 \le j \le 5$ and $x = (E, Q, I_S, I_N, H)$.

$$J_F(x) = \frac{\partial F_i}{\partial x_i} = \begin{bmatrix} \frac{\partial}{\partial E}F_1 & \frac{\partial}{\partial Q}F_1 & \frac{\partial}{\partial I_S}F_1 & \frac{\partial}{\partial I_N}F_1 & \frac{\partial}{\partial H}F_1 \\ \frac{\partial}{\partial E}F_2 & \frac{\partial}{\partial Q}F_2 & \frac{\partial}{\partial I_S}F_2 & \frac{\partial}{\partial I_N}F_2 & \frac{\partial}{\partial H}F_2 \\ \frac{\partial}{\partial E}F_3 & \frac{\partial}{\partial Q}F_3 & \frac{\partial}{\partial I_S}F_3 & \frac{\partial}{\partial I_N}F_3 & \frac{\partial}{\partial H}F_3 \\ \frac{\partial}{\partial E}F_4 & \frac{\partial}{\partial Q}F_4 & \frac{\partial}{\partial I_S}F_4 & \frac{\partial}{\partial I_N}F_4 & \frac{\partial}{\partial H}F_4 \\ \frac{\partial}{\partial E}F_5 & \frac{\partial}{\partial Q}F_5 & \frac{\partial}{\partial I_S}F_5 & \frac{\partial}{\partial I_N}F_5 & \frac{\partial}{\partial H}F_5 \end{bmatrix} = \begin{bmatrix} 0 & 0 & \beta_S(1-m)S & \beta_I(1-m)S & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \end{bmatrix}$$

$$J_V(x) = \frac{\partial V_i}{\partial x_i} = \begin{bmatrix} \frac{\partial}{\partial E}V_1 & \frac{\partial}{\partial Q}V_1 & \frac{\partial}{\partial I_S}V_1 & \frac{\partial}{\partial I_N}V_1 & \frac{\partial}{\partial H}V_1 \\ \frac{\partial}{\partial E}V_2 & \frac{\partial}{\partial Q}V_2 & \frac{\partial}{\partial I_S}V_2 & \frac{\partial}{\partial I_N}V_2 & \frac{\partial}{\partial H}V_2 \\ \frac{\partial}{\partial E}V_3 & \frac{\partial}{\partial Q}V_3 & \frac{\partial}{\partial I_S}V_3 & \frac{\partial}{\partial I_N}V_3 & \frac{\partial}{\partial H}V_3 \\ \frac{\partial}{\partial E}V_4 & \frac{\partial}{\partial Q}V_4 & \frac{\partial}{\partial I_S}V_4 & \frac{\partial}{\partial I_N}V_4 & \frac{\partial}{\partial H}V_4 \\ \frac{\partial}{\partial E}V_5 & \frac{\partial}{\partial Q}V_5 & \frac{\partial}{\partial I_S}V_5 & \frac{\partial}{\partial I_N}V_5 & \frac{\partial}{\partial H}V_5 \end{bmatrix}$$

$$J_V(X) = \begin{bmatrix} (\alpha+\gamma+\varphi+\mu) & 0 & 0 & 0 & 0 \\ -\varphi & (\delta+\sigma+\mu) & 0 & 0 & 0 \\ -\alpha & 0 & (\omega+\mu) & 0 & 0 \\ -\gamma & 0 & 0 & (\tau+\mu) & 0 \\ 0 & -\sigma & -\omega & 0 & (\theta+\varepsilon+\mu) \end{bmatrix}$$

The Jacobian of F and V at the disease free equilibrium point $\in_0$ takes the form;

$$J_F(\in_0) = \begin{bmatrix} 0 & 0 & \beta_S(1-m)\frac{B}{\quad} & \beta_I(1-m)\frac{B}{\quad} & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \end{bmatrix}$$

and

$$J_V(\in_0) = \begin{bmatrix} (\alpha+\gamma+\varphi+\mu) & 0 & 0 & 0 & 0 \\ -\varphi & (\delta+\sigma+\mu) & 0 & 0 & 0 \\ -\alpha & 0 & (\omega+?\mu) & & 0 \\ -\gamma & 0 & 0 & (\tau+?\mu) & \\ 0 & -\sigma & -\omega & 0 & (\theta+\varepsilon+?\mu) \end{bmatrix}$$

respectively.

It can be verified that the matrix $J_V(\in_0)$ is nonsingular and its inverse is given by;

$$J_V(\in_0)^{-1} = \begin{bmatrix} \frac{1}{(\alpha+\gamma+\varphi+\mu)} & 0 & 0 & 0 & 0 \\ \frac{\varphi}{(\alpha+\gamma+\varphi+\mu)(\delta+\sigma+\mu)} & \frac{1}{(\delta+\sigma+\mu)} & 0 & 0 & 0 \\ \frac{\alpha}{(\alpha+\gamma+\varphi+\mu)(\omega+\mu)} & 0 & \frac{1}{(\omega+\mu)} & 0 & 0 \\ \frac{\gamma}{(\alpha+\gamma+\varphi+\mu)} & 0 & 0 & \frac{1}{(\tau+\mu)} & 0 \\ \frac{(\alpha\omega+\sigma\varphi)\mu+\omega((\delta+\sigma)\alpha+\sigma\varphi}{(\alpha+\gamma+\varphi+\mu)(\delta+\sigma+\mu)(\omega+\mu)(\theta+\varepsilon+\mu)} & \frac{\sigma}{(\delta+\sigma+\mu)(\theta+\varepsilon+\mu)} & \frac{\omega}{(\omega+\mu)(\theta+\varepsilon+\mu)} & 0 & \frac{1}{(\theta+\varepsilon+\mu)} \end{bmatrix}$$

The eigenvalues of the matrix $\left[J_F(\in_0)\right]\left[J_V(\in_0)\right]^{-1}$

$$\lambda_{1,2,3,4.5} = \begin{bmatrix} 0 \\ 0 \\ 0 \\ 0 \\ \frac{B}{\mu}\frac{\gamma(\omega+\mu)\beta_N(1-m)+\alpha\beta_S(1-m)(\tau+\mu)}{(\omega+\mu)(\alpha+\gamma+\varphi+\mu)(\tau+\mu)} \end{bmatrix}$$

The spectral radius (the largest of the absolute value of the eigenvalues) of the matrix $\left[J_F(\in_0)\right]\left[J_V(\in_0)\right]^{-1}$ is;

$$\rho\left[J_F(\in_0)\right]\left[J_V(\in_0)\right]^{-1} = \frac{B}{\mu}\frac{\gamma(\omega+\mu)\beta_N(1-m)+\alpha\beta_S(1-m)(\tau+\mu)}{(\omega+\mu)(\alpha+\gamma+\varphi+\mu)(\tau+\mu)}$$

The reproductive number

$$\boldsymbol{R}_0 = \rho\left[J_F(\in_0)\right]\left[J_V(\in_0)\right]^{-1}$$

$$\Rightarrow \boldsymbol{R}_0 = \frac{B}{\mu}\frac{\gamma(\omega+\mu)\beta_N(1-m)+\alpha\beta_S(1-m)(\tau+\mu)}{(\omega+\mu)(\alpha+\gamma+\varphi+\mu)(\tau+\mu)}$$

$$R_0 = \frac{\gamma S_0\beta_N(1-m)}{(\alpha+\gamma+\varphi+\mu)(\tau+\mu)} + \frac{\alpha S_0\beta_S(1-m)}{(\omega+\mu)(\alpha+\gamma+\varphi+\mu)} = R_1 + R_2$$

The first term, R_1 measure the contributions from infectious without symptoms-to-susceptible (infection as a result of β_N) and the second term R_2 represents the contribution from infectious with symptoms -to-susceptible individuals (infection as a result of β_S). These two transmission modes collectively shape the overall infection risk of human-to-human transmission of COVID-19 outbreak and we call the spectral radius, R_0 the threshold value or the basic reproductive number.

Locally stability of the disease free equilibrium point

In this section, stability analysis of the system (1) is considered and mainly focuses on DFE point alone. The endemic equilibrium points are not acceptable on the reality grounds since COVID-19 is an epidemic disease and is a short time outbreak. We now analyze the local stability of DFE point of the system (1) by analyzing the characteristic equation. In the absence of the infectious disease, the model has a unique disease free steady state. To find the local stability we use the Jacobian of the model evaluated at $\in_0$. Stability of this steady state is then determined based on the eigenvalues of the corresponding Jacobian, which are functions of the model parameters [9].

Theorem 3: The disease free equilibrium point of system (1) is said to be locally asymptotically stable, if all the eigenvalues of the Jacobian matrix at $\in_0$ are negative or unstable otherwise.

Proof:-The characteristic equation of the Jacobian matrix at the DFE point is given by;

$$\left|J_{\in_0} - \lambda I\right| = \begin{vmatrix} -(a_1+\lambda) & 0 & \delta & -a_7 & -a_8 & 0 & 0 \\ 0 & -(a_2+\lambda) & 0 & a_7 & a_8 & 0 & 0 \\ \rho & \varphi & -(a_3+\lambda) & 0 & 0 & 0 & 0 \\ 0 & \alpha & 0 & -(a_4+\lambda) & 0 & 0 & 0 \\ 0 & \gamma & 0 & 0 & -(a_5+\lambda) & 0 & 0 \\ 0 & 0 & \sigma & \omega & 0 & -(a_6+\lambda) & 0 \\ 0 & 0 & 0 & 0 & \tau & \theta & -(\mu+\lambda) \end{vmatrix}$$

where

$a_1 = (\rho+\mu)$, $a_2 = (\alpha+\gamma+\varphi+\mu)$, $a_3 = (\delta+\sigma+\mu)$, $a_4 = (\omega+\mu)$, $a_5 = (\tau+\mu)$, $a_6 = (\theta+\varepsilon+\mu)$, $a_7 = \beta_S(1-m)B/\mu$ and $a_8 = \beta_N(1-m)B/\mu$.

Expanding using 7th and 1st columns respectively and collecting common factors;

$$|J_{\mathbf{E}_0} - I\lambda| = (\mu+\lambda)(a_6+\lambda)\left[\left((a_3+\lambda)(a_1+\lambda)-\delta\rho\right)\begin{vmatrix} -(a_2+\lambda) & a_7 & a_8 \\ \alpha & -(a_4+\lambda) & 0 \\ \gamma & 0 & -(a_5+\lambda) \end{vmatrix}\right] = 0 \quad (15)$$

In equation (15) we have three factors that should be utilized to determine the eigenvalues:

$$(\mu+\lambda)(a_6+\lambda) = 0$$

$$\left((a_3+\lambda)(a_1+\lambda)-\delta\rho\right) = 0$$

$$\begin{vmatrix} -(a_2+\lambda) & a_7 & a_8 \\ \alpha & -(a_4+\lambda) & 0 \\ \gamma & 0 & -(a_5+\lambda) \end{vmatrix} = 0$$

From (a) $\lambda_1 = -\mu < 0$ or $\lambda_2 = -a_6 = -(\theta+\varepsilon+?\mu) <$, since $a_6 = (\theta+\varepsilon+\mu)$

Using (b) $(\rho+\mu+\lambda)(\delta+\sigma+\mu+\lambda)-\delta\rho = 0$ since $a_1 = (\rho+\mu)$ and $a_3 = (\delta+\sigma+\mu)$

Thus, after simplification;

$$\lambda^2 + (\rho+\delta+\sigma+2\mu)\lambda + \rho(\sigma+\mu) + \mu(\delta+\sigma+\mu) = 0$$

$\lambda_{3,4} < 0$ (Negative), since all the coefficients of λ are positive.

From (c), using 3rd column expansion and simplification yields;

$$\lambda^3 + (a_2+a_4+a_5)\lambda^2 + \left(a_2a_5 + a_2a_4 + a_4a_5 - (\alpha a_7 + \gamma a_8)\right)\lambda + a_2a_4a_5\left[1 - \left[\frac{(\gamma a_8 a_4 + \alpha a_7 a_5)}{a_2 a_4 a_5}\right]\right] = 0$$

$$\lambda^3 + (a_2+a_4+a_5)\lambda^2 + (a_2a_5+a_2a_4+a_4a_5-(\alpha a_7+?\gamma a_8))\lambda + a_2a_4a_5\left[-R_0\right] = 0 \quad (16)$$

From equation (16), if $R_0 < 1$ and $a_2a_5 + a_2a_4 + a_4a_5 > (\alpha a_7 + \gamma a_8)$, all the coefficients and constant terms are positive, then the three eigenvalues $\lambda_{5,6,7} < 0$ (negative).

Therefore, we conclude that the real parts of all the seven eigenvalues are less than zero (negative), then the diseases is asymptotically stable, if $R_0 < 1$.

4. Numerical Simulation and Result

In this section we applied our model to study COVID-19 epidemic transmission dynamics in Ethiopia. In addition to this we analyze the impact of control and prevention strategies used to battle the spread of the pandemic. Data for this study was collected from Ethiopian Public Health Institute (EPHI) and other sources, such as WHO daily published report [5]. The data is presented in table 3 in appendix section, comprising the daily reported new cases, cumulative cases, deaths and recovery.

Model simulations are performed using MATLAB software and the initial values of the state variables and parameters are presented in table 2 together with basic reproduction number obtained based on equation (19). The rate of quarantine and isolation are taken between 3 to 30 days and 12 to 30 days respectively after being infected. Within these ranges the effect of quarantine and isolation on the number of infection, recovery and death were analyzed. On the other hand the rate of self-protection parameter has been considered between 0 and 1.

Numerical simulation to determine the effect of variations in isolation time, quarantine time and self-protection on the dynamics of the number of Infected, Exposed, Death and Recovery were presented. The simulation uses values of parameters in Table 2 and the numerical simulation result of the transmission dynamics.

Parameter	Mean Estimated value	Source
B	0.0000301	[31]
β_S	0.000000052	[30]
β_N	0.0000018922	Estimated
m	(0, 1)	Verified
α	0.2	[32]
γ	0.689	Estimated
σ	0.07143	[32]
θ	0.6667	Estimated
μ	0.0000301	[31]
ε	0.0300	Calculated
ω	0.1922	Verified
φ	0.5	Verified
ρ	0.1971	Assumption
δ	0.1200	Estimated
τ	0.2	Estimated
N (In million)	110	Verified

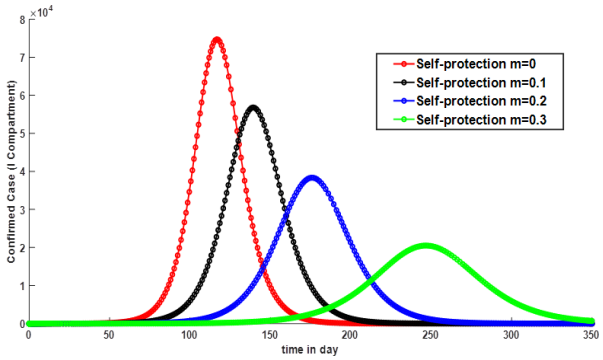


Fig.2.Variation of Confirmed cases for different values of m (Self-protection) (Effect of self-protection on confirmed cases)

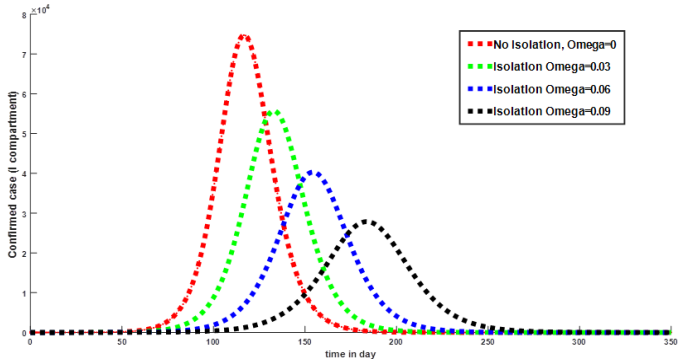


Fig.3.Variation of confirmed cases for different values of ω (Isolation Rate) (Effect of isolation rate on confirmed cases)

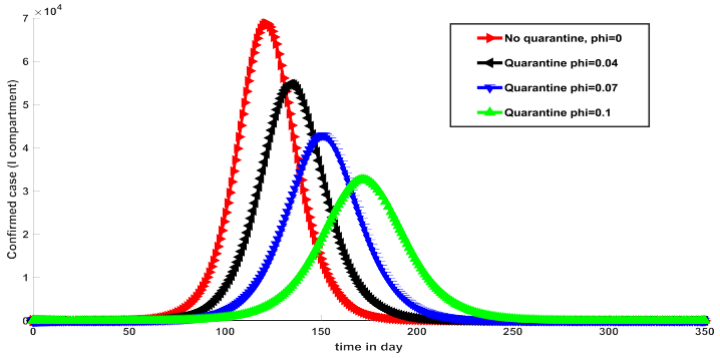


Fig.4.Variation of confirmed cases for different values of φ (Rate of Quarantine)

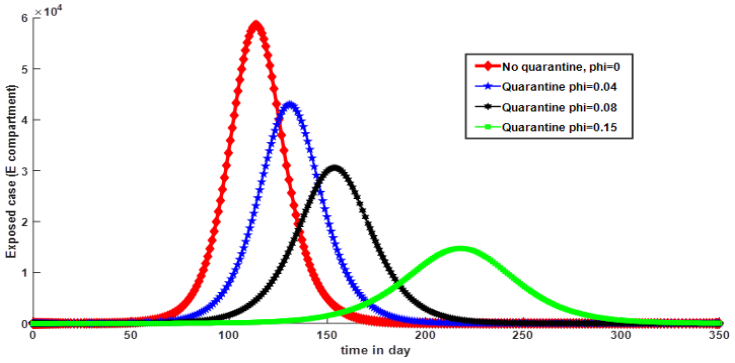


Fig.5.Variation of Exposed cases for different values of φ (Rate of quarantine) (Effect of rate of quarantine on exposed cases).

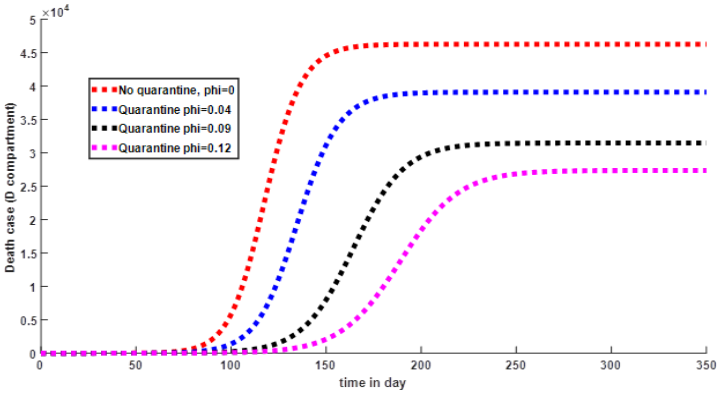


Fig.6.Variation of Death for different values of φ (Rate of quarantine) (Effect of quarantine on commutative death cases)

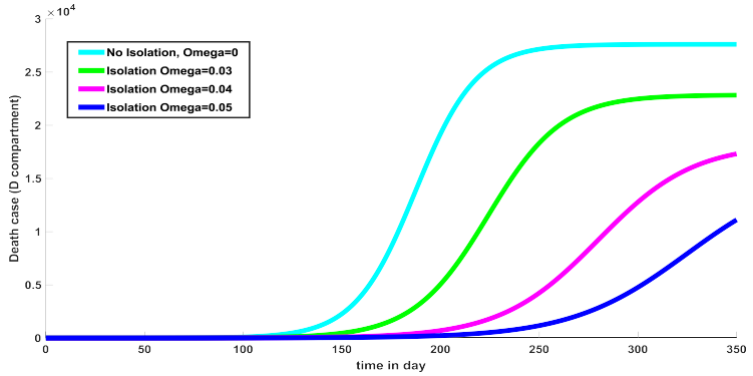


Fig.7.Variation of Death cases for different values of ω (Rate of isolation) (Effect of rate of isolation on death number)

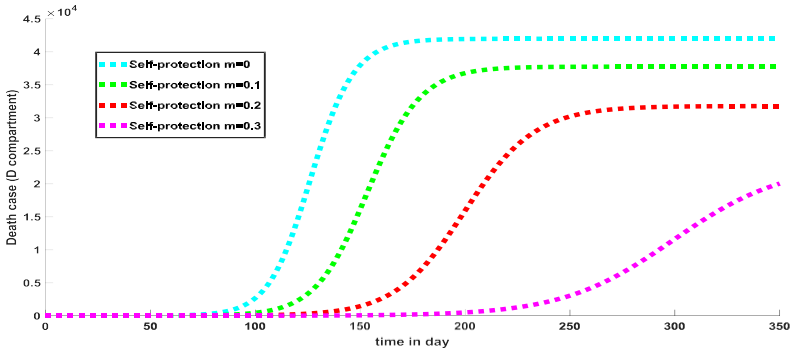


Fig.8.Variation of death cases for different values of m (Self-protection).(Effect of self-protection on death number)

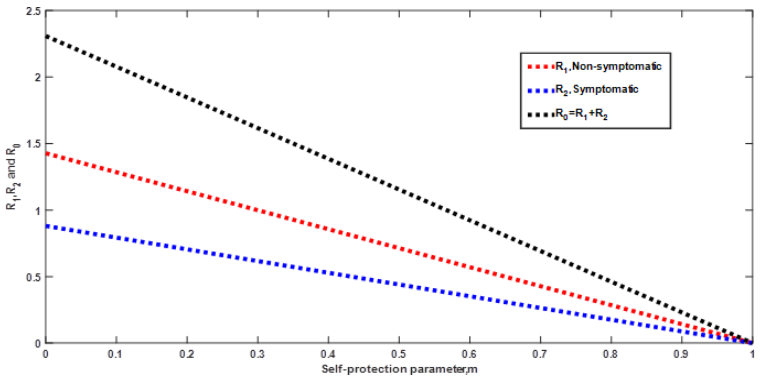


Fig.9.Graphs of R_1 , R_2 and R_0 verses m self-protection

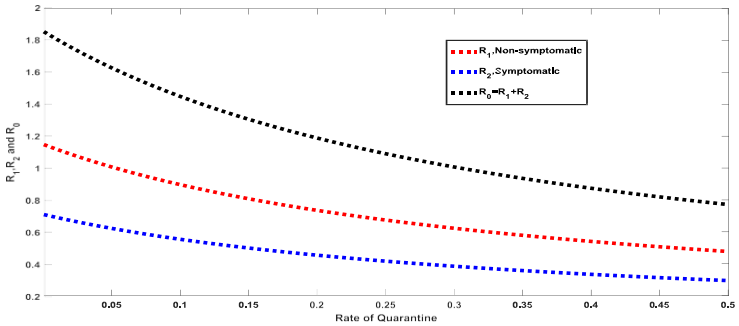


Fig.10.The graphs of R_1 , R_2 and R_0 verses (rate of quarantine).

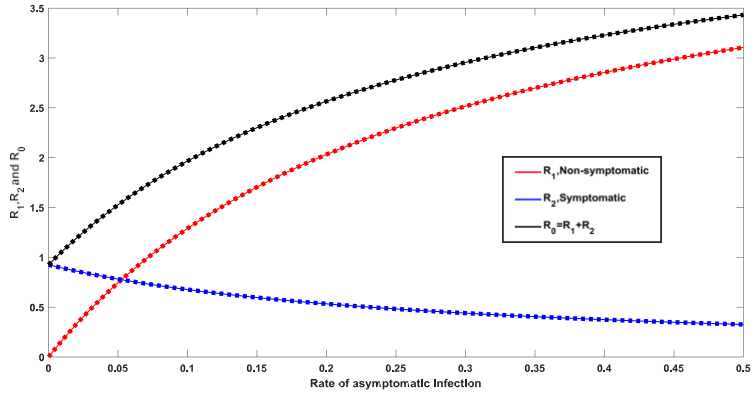


Fig.11.The graphs of R_1 , R_2 and R_0 verses the rate of non-symptomatic infection.

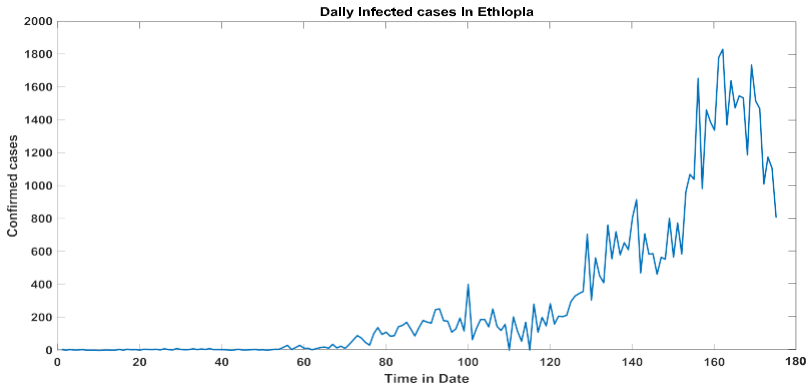


Fig.12. Daily confirmed cases real data

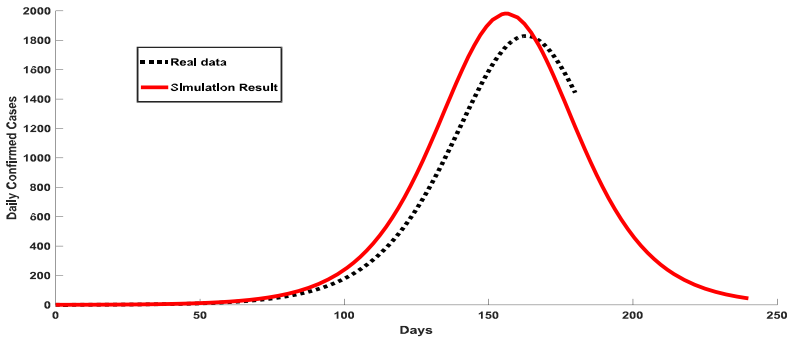


Fig.13. Fitting real daily infected case of COVID-19 in Ethiopia verses model simulation result of infected compartment.

5. Discussion

We examined the effects of control and preventative strategies and presented the SEQISINHR model in this research. We looked at the positivity of the solutions to the system (1) and its boundedness characteristic. Separate analyses were conducted to determine the impact of asymptomatic and symptomatic persons on the value of the basic reproductive number (R0), which was calculated using the standard next generation matrix. Since 85% of infected persons are asymptomatic, R1 (reproductive number as a consequence of asymptomatic individuals) made a larger contribution to R0 than R2 (reproductive number as a result of symptomatic individuals), as seen in Fig.1 [9, 13, 21, 23]. Systems (1) have a disease-free equilibrium that is locally asymptotically stable when R0 < 1 and unstable under other conditions. We established a control parameter dubbed self-protection, m, to explore the influence of people'

control actions to avoid the broad transmission of COVID-19. The effects of the self-protection parameter on confirmed instances are shown in figure 2, while the effects on fatal cases are shown in figure 8. The number of confirmed instances grows rapidly and reaches its maximum when self-defense is not present (m=0). However, the number of confirmed and fatal cases develops slowly and has a lower peak value when the rate of self-protection parameter increases from 10% to 30% of the vulnerable population. magnitude, m. In addition, we included quarantine of suspects and isolation of sick people to help lower infection rates and assess the efficacy of government control efforts. Figures 3 and 7 show the impact of the isolation rate on confirmed and fatal cases. Both confirmed and fatality cases, as well as the peak value, rapidly rise when rate isolation ($\omega = 0$) is not present. Changing the isolation rate from 0.03% to 0.04% or from 25 days to 10 days decreases the number of confirmed and fatal cases. Figures 5, 6, and 7 show the impact of the quarantine rate on confirmed, death, and recovery cases. The number of confirmed cases, exposures, and deaths rises sharply when the quarantine rate is zero. On the other hand, reducing the quarantine length in days (25 days, 12 days, and 6 days) or raising the quarantine rate (0.04, 0.08, and 0.12), immediately reduces the number of infections, deaths, and exposed cases. The model validation was conducted using data spanning 180 days, from March 13, 2020 to August 13, 2020, as shown in figure 13. As the self-protection parameter, m, and the rate of quarantine were increased, the values of R0 in figures 9 and 10 were found to decrease, respectively. The incidence of asymptomatic infectious disease (IVD) vs R0, R1, and R2 is shown in curve 11 of figure 11. While the reproductive rate, R1, rises as γ increases, the reproductive rate, R2, falls. It is crucial to prioritize reducing the incidence of interaction between vulnerable and asymptomatic persons.
The data vs simulation plot in figure 13 shows that the model and the actual/confirmed COVID-19 data in Ethiopia are similar, allowing us to examine COVID-19 control and preventative measures in the country. Assuming no social or cultural contact and no isolation, quarantine, or self-protection (where $\omega < 0$, $\phi < 0$, and $m < 0$), the value of R0 is 1.93.

6. Conclusion

The only way to reduce the spread of COVID-19, say the World Health Organization and the Ethiopian Ministry of Health (MoH), is to use a non-pharmaceutical strategy that allows people to be infected. It is crucial to create a dynamic model and evaluate the impact and efficacy of the suggested model and its parameters in order to implement these control and prevention measures. On top of that, there are more people who are asymptomatic than those who are experiencing symptoms. Therefore, minimizing the incidence of interaction between the vulnerable and asymptomatic populations should be a top priority.
The purpose of this research was to examine the effects of control and preventative measures using a dynamic SEQISINHR model. Separate analyses were also conducted on the effects on those who did not exhibit any symptoms. In order to prevent the spread of infection, it is crucial to increase the rate of isolation by addressing confirmed cases quickly and effectively. Additionally, quarantine should be implemented using different tracing systems to shorten the amount of time that infected persons spend in isolation. On top of that, raising m controls the movement of persons from the susceptible to the exposed compartment, which means that it decreases the danger of infection. It is imperative that the government step in and impose strong regulations and standards in order to raise self-defense boundaries. However, if control measures are not put in place to decrease the contact rate and rate of transmission from E to, there is a substantial risk of infection when the asymptomatic infectious class is considered individually.It was anticipated that symptomatic persons would be more susceptible to transmission, but the greater infection incidence among asymptomatic individuals might be explained by their lack of adherence to the same safety protocols as symptomatic patients in order to prevent the spread of the illness.
We conclude from the simulation results that the rate of non-symptomatic infection, the three sensitive parameters (prevention and control parameters), and the overall effectiveness of

COVID-19 transmission control in Ethiopia are critical. As a result, we think that increasing the rate of isolation and quarantine is the best way to manage the illness. Furthermore, lowering the contact rate should be prioritized in order to prevent the transmission of COVID-19 by both infected and, critically, uninfected persons.

References

[1] For example, in 2020, Chan, Yuan, and Kok published a study. The 2019 new coronavirus and a cluster of pneumonia in one family: evidence of person-to-person transmission. "The Lancet" (2020, 395:514). Committee established by the World Health Organization to address emergencies [2]. In light of the recent COVID-19 epidemic, the International Health Regulations (2005) Emergency Committee held its second meeting and issued a statement. World Health Organization, Geneva, 2020. Chayu Yang and Jin Wang are cited in [3] (https://www.who.int/news-room/detail/30-01-2020). Wuhan, China's new coronavirus pandemic: a mathematical model, Mathematical Biosciences and Engineering. Journal of Biological Engineering, Volume 17, Issue 3, Pages 2708–2724, 2020.

[4] The People's Republic of China's National Health Commission. 2020. 5. Reports on the status of the 2019 new coronavirus (nCoV)–WHO. You may get this information at the World Health Organization's website: https://www.who.int/emergencies/diseases/novel-coronavirus-2019/situation-reports. Situation report on coronavirus disease 2019 (COVID-19), 82. 2020. Gainer J. Three Emerging Coronaviruses in Two Decades: The Story of SARS, MERS, and Now COVID-19. Available from: https://www.who.int/docs/default-source/coronaviruse/situation-reports/20200411. Print publication of the American Journal of Clinical Pathology. publication year 2020; volume 153, issue 4, pages 420-421.The number of fatalities caused by Mahase E. Coronavirus COVID-19 is more than that of SARS and MERS put together, even though the number of cases is fewer. Medical Research Edition of the British Medical Journal. 2020 February; 368:m641.
The effects of the COVID-19 pandemic on India's healthcare system: a probabilistic mathematical model [9] Chatterjee et al. Military Medicine in India. 2020. You may get this article at this link: https://doi. org/10.1016/j.mjafi.2020.03.022. 12. [10] Khan T, Ullah Z, Ali N, Zaman G. Hepatitis B virus epidemic model modeling and control. "The Mathematics of Infectious Diseases" by Herbert W. Hethcote was published in 2000 in the Society for Industrial and Applied Mathematics' journal, Society for Chaos, Solitons and Fractals, volume 42, issue 4, pages 599-653. The article may be found online at https://doi.org/10.1016/j.chaos.2019.04. 033.
Inference in disease transmission studies by employing stochastic epidemic models is discussed in a 2005 paper by Michael Hohle, Erik Jorgensen, and Philip D. O'Neill, which can be found in Appl. Statist. 54, Part 2, pages 349-366. [13] A data-driven model to characterize and anticipate the dynamics of COVID-19 transmission was developed by Paiva HM, Afonso RJM, de Oliveira IL, and Garcia GF in 2020. This article was published in the journal PLoS ONE with the DOI: e0236386. KAyla Henneman, Dan Van Peursem, and Victor C. Huber (2013), "Mathematical modeling of influenza and a secondary bacterial infection," https://doi.org/10.1371/journal.pone.0236386. The tenth volume of the Wseas Transactions on Biomedicine and Biology, issue one.
Frank Ball's 1990 article "Poission Approximation for Some Epidemic Models" in the Journal of Applied Probability, 27, 479-490, is cited as reference 15.
[16] Michael Y. Li wrote an introductory textbook on mathematical modeling of infectious diseases, which was published by the University of Alberta in Edmonton, Alberta, Canada.
Presenting 2018 by Springer International Publishing AG. P. Rohani, J.-F. Guégan, and M. Choisy. [17] https://doi.org/10.1007/978-3-319-72122-4. The information on pages 379–404 comes from the 2007 edition of the Encyclopedia of Infectious Diseases, specifically the section on Modern Methodologies.
Suzanac Dragicevic and Liliana Perez. A model for the dynamics of the transmission of infectious diseases based on the behavior of agents. Transmission of 2019-nCoV Infection from an asymptomatic contact in Germany, N. Engl. J. Med., 2020, by C. Rothe, M. Schunk, P. Sothmann, G. Bretzel, G. Froeschl, and C. Wallrauch, et al., International Journal of Health Geographics, 2009, 8:50 doi.10.1186/1476-072X-8-50.
Gengxin Sun, Sheng Bin, and Chih-Cheng Chen [20] took part. Using cellular automata, we model and analyze many factors that contribute to the spread of infectious diseases. Public health and environmental research journal published on an international level. This year (2019)