I. J. Mathematical Sciences and Computing, **2021, 4, 43-59**
Published Online December 2021 in MECS (http://www.mecs-press.org/)
DOI: 10.5815/ijmsc.2021.04.05

Mathematical Modeling for COVID-19 Transmission Dynamics and the Impact of Prevention Strategies: A Case of Ethiopia

Akalu Abriham[1]*, Demsis Dejene[1], Tadele Abera[2], Abayneh Elias[3]
[1]Dire Dawa University, Department of Mathematics, Dire Dawa, Ethiopia
[2]Dire Dawa University, DDIT, Dire Dawa, Ethiopia
[3]Dire Dawa University, Department of Medicine, Dire Dawa, Ethiopia
Email: ake.abrish@gmail.com

Received: 16 May 2021; Revised: 02 June 2021; Accepted: 27 June 2021; Published: 08 December 2021

Abstract: At the end of 2019 the novel coronavirus disease (COVID-19) was declared as a major health hazard by the world health organization (WHO) and the only available way of stopping this threat was via non-pharmaceutical approach. Most authors have studied COVID-19 transmission dynamics using mathematical modeling by involving the basic (major) compartments. In this study we have formulated a mathematical model for the transmission dynamics of COVID-19 which incorporates almost all possible scenarios at present. We have also analyzed the impact of prevention and control strategies. The model has satisfied all the basic properties that infectious disease model should fulfill; Boundedness, positivity of its solutions, stability analysis, epidemic equilibrium point, basic reproduction number and local stability of the disease free equilibrium. We introduced a self-protection parameter, *m* to analyze the impact of physical distancing, staying at home, using masks, washing hands and so on. The impact of isolation and quarantine has been analyzed and their effects on the number of Exposed, infected and dead people were clearly discussed. In addition to these, the effects of symptomatic and asymptomatic individuals on the value of basic reproduction number have been examined. The numerical simulations of this study indicate that the government should increase isolation, quarantine and self-protection rates. Additionally to minimize the contact rate between susceptible and asymptotic individuals, self-protection at all cost and everywhere has to be done, so that both symptomatic and importantly asymptomatic individuals stop transmitting the virus.

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

1. Introduction

The novel coronavirus disease (COVID-19), pandemic is a disease that causes a respiratory illness and spreads from human to human through respiratory droplets and it has been first identified in December 2019 at Wuhan city, Hubei Province, China [1]. On Jan 30, 2020, World Health Organization (WHO) declared that COVID-19 as a Public Health Emergency of International Concern (PHEIC) [2]. The new coronavirus has typically associated with the common cold, pneumonia, and severe acute respiratory syndrome (SARS) in humans [3]. Since December 2019, COVID-19 rapidly spread throughout China and to all over the world. As of February 9, 2020, it transmitted to 34 provinces, regions and municipal cities across China and a total of 40,235 confirmed cases with 908 deaths (2.3%) and 6,484 (16.1%) critically ill cases, and 23,589 suspected cases [4]. Recently, as of April 25, 2020 a total of 2,686,785 cases confirmed with 184,681 deaths in 213 countries including Ethiopia with 112 confirmed cases and 3 deaths [5]. The most affected countries are the United States of America, Italy, Spain, China, Germany, Brazil and France [6]

In the past two decades, there have been two coronavirus epidemics that lead global health anxiety, SARS in 2003 and MERS in 2012. It is striking and devastating that COVID-19 already has killed more people than the combined number of people died by both SARS and MERS [7, 8]. In the meantime, scientists have always had an essential role to play in the study of the transmission dynamics of infectious diseases, particularly in mathematical modeling. The study of epidemiological mathematical models leads to deep understanding of the dynamics of epidemics, being an important tool to assess the potential effects of preventive and controlled measures, especially when their characteristics are still unclear [9,10].

Since Jan, 2020, scientists have been racing to develop vaccine for COVID-19 and a few of them passed through animal trial so that first round human injections are underway. However, the time to get approved and efficient vaccine will take long time, according to most scientists at least 12-to-18 months [11]. Thus, to protect the ongoing damage

until the existence of WHO approved vaccine, it is recommended to accelerate innovative researches that curtail the spread of the epidemic. Moreover, world scientists in coordination with WHO indorsed scientific community to support research priorities that contribute to global research platforms in hopes of learning from the current pandemic response to better prepare for the next unforeseen epidemic [12].

It is for these reasons, the objective of this study was to introduce a new model involving all current prevention and control mechanisms of COVID-19 and analyze their impact and effectiveness on controlling the effects of being infected and make possible prediction. Moreover, this study is intended to:

- Develop a mathematical model for the transmission dynamics of COVID-19 from human-to-human route.
- Investigate which prevention strategies are more effective to minimize infection and death.
- Analyze the effect of symptomatic and asymptomatic individuals on the basic reproductive number separately.
- Provide possible prevention and control measures to minimize rate of infection.

For some diseases, there are management methods, which may involve prevention like vaccination or treatment of symptomatic patients but for diseases with no known treatment, like COVID-19, it is possible to attempt control by isolation of diagnosed patients and quarantine of suspected victims to decrease transmission [13]. However, it is impossible to do experiments to compare possible management strategies; the only way to attempt to compare the effectiveness of different non-pharmaceutical approaches may be to formulate mathematical model and use it to make prediction [14].

Mathematical modeling provides another research tool which commensurate with a new powerful laboratory technique [15]. A mathematical model is a mathematical description of the situation based on the hypotheses and the solution of the model gives conclusions which may be compared with experimental results. This comparison usually requires numerical simulations to give predictions which may be compared with observed data [16]. To have mathematical model on spread of infectious disease it is vital to collect clear and acceptable assumptions about the disease [17].

In order to have a clear understanding of the spread of COVID-19 in host populations, many studies have been published using mathematical modeling since late 2019; by considering early transmission dynamics. These models are presented using several approaches to describe the dynamics of the epidemic and future numbers. The most common approaches are the Susceptible-Infectious-Recovered/Death (SIRD) model and the Susceptible-Exposed-Infectious-Recovered (SEIR) model; both derived from the Susceptible-Infectious-Recovered (SIR) pioneer model described by Kermack and McKendrick in 1927 [18].

As the behavior of the new novel coronavirus and its spread dynamics is under study yet, recently available studies doesn't considered all the possible scenarios published currently. Thus, as an emerging infectious disease, understanding its recent epidemiological and clinical characteristics and incorporating them into the model will provide to take quick prevention and control measures [19].

Thus, in this study we proposed a deterministic mathematical model with compartmental modified SEIR approach; to investigate the transmission dynamics of on-going COVID-19 disease in Ethiopia by including the current understanding and disease progression and the most up-to-date assumptions. In addition to susceptible, exposed, infected and recovered compartments, we included quarantine and hospitalization compartments considering only human-to-human transmission route.

Clinical evidence shows that the incubation period of this disease ranges from 2 to 14 days [3]. During this period of time, infected individuals may not develop any symptoms and may not be aware of their infection, yet they are capable of transmitting the disease to other people [20]. Therefore, in this study the impacts of symptomatic and asymptomatic individuals have been analyzed disjointedly. We also attempted to include control by isolation of diagnosed patients and quarantine of suspected victims to decrease transmission which is effectively and practically under work in Ethiopia.

Furthermore, we comprised control parameter which represents the implementation level of self-protection, where self-protection refers to the level of implementation of prevention strategies like; using face mask, staying home, frequently washing hands, physical distancing, and using sanitizers and so on. In general, we have attempted to incorporate almost all the control measures and prevention strategies that the Ethiopian health system has been using so far to control the transmission of COVID-19.

Applying mathematical modeling as a tool by incorporating consistent assumptions of COVID-19 and simulating the model provided us better understanding on the impact of prevention and control strategies. Recently many authors have studied mathematical modeling on the dynamic transmission of infectious disease in general and COVID-19 in particular. Among these studies, some published related studies are reviewed as follows;

The authors [21] developed a mathematical model for simulating the phase-based transmissibility of a novel coronavirus. In this study authors developed a Bats-Hosts-Reservoir-people transmission network model to simulate the potential transmission from the infectious source (assumed to be bats) to human infection. The reproductive number, R_0 has been calculated using the next generation matrix and used to assess the level of transmission of COVID-19. In [22]

authors represented modeling the transmission of Middle East Respirator Syndrome (MERS) Corona Virus in Republic of Korea based on human-to-human infection and the authors used Susceptible-Exposed-Symptomatic-Asymptomatic-Hospitalized-Removed model. In this study isolation and quarantine compartments are not considered as a control measure which plays a great role on controlling the rate of infections as well as death. In [23] developed a new mathematical modeling and epidemic prediction of COVID-19 and analyzed regarding its significance to epidemic prevention and control measures. In this study authors introduced a model containing six compartments; Susceptible (who may be infected) - Exposed-Infected-Quarantined- Potential victims- Removed (cured after infection). In this case infected individuals are not separated as symptomatic and asymptomatic so that it is impossible to analyze the effect/impact of infected individual who are symptomatic and asymptomatic separately.

Authors in [24] intended to estimate the basic and time-varying transmission dynamics of novel coronavirus pneumonia (NCP) across China, and compared them with SARS and authors used data on NCP cases by February 7, 2020 were collected from epidemiological investigations or official websites and the study concluded that NCP may have a higher transmissibility than SARS, and the efforts of containing the outbreak are effective. However, the efforts are needed to persist in for reducing time-varying reproduction number below one. In [25] authors estimated effectiveness of symptom and risk screening to prevent the spread of COVID-19. The finding of this study shows that most cases missed by screening are fundamentally undetectable; because they have not yet developed symptoms and are unaware they were exposed. This work underscores the need for measures to limit transmission by individuals who become ill after being missed by a screening program. This provides a good indication to include and analyze asymptomatic and symptomatic individuals separately.

Authors in [26] presented mathematical modeling of the spread of the coronavirus disease (COVID-19) considering its particular characteristics in the case of China. In this study authors incorporated various assumptions used compartmental modeling. In [13] authors established a data-driven model to describe and forecast the dynamics of COVID-19 transmission. In this study SEIAHRD model has been used to forecast the evolution of the epidemic and seven compartments are used, but individuals in quarantine compartment are not included. In [27] two mathematical models containing five compartments are developed; using ordinary differential equation and delay differential equation. In this model authors incorporated implementation of major government public restrictions designed to mitigate the severity of the epidemic, both reported and unreported cases and asymptomatic infectious cases in the disease transmission.

Anirudh in [28] described the outcomes and the challenge of several mathematical models and the transmission dynamics in predicting spread, peak and reduction Covid-19 cases. This study concluded that the success of a model depends on the assumed process of experts in the field which in turn depends on understanding the disease and the inputs given. The result of this study suggests that incorporating additional compartments that plays a great role on the transmission dynamics will provide better prediction and more accurate result. In [29] authors proposed a mathematical model to investigate COVID-19 and the model describes the multiple transmission pathways in the infection dynamics, and emphasizes the role of the environmental reservoir in the transmission and spread of this disease. In this study, SEIRV model is used where the interpretation of the E and I compartments is that they contain asymptomatic infected and symptomatic infected individuals, respectively where, V is the concentration of the coronavirus in the environmental reservoir. Both exposed and symptomatic infected individuals were merged in one compartment which precludes separate analysis of exposed and symptomatic infected individuals; identified as one of the limitation of this study.

In [30] authors analyzed the optimal control design of impulsive SQEIAR epidemic models with application to COVID-19. In this study authors presented two actions to eradicate the infection; the quarantine and the treatment of infected people. To reach this purpose, optimal control theory is presented to control the epidemic model over free terminal optimal time control with an optimal cost. In [31] authors examined the stability and numerical simulation of SEIR model for COVID-19 spread in Indonesia and the aim of this research was to construct the SEIR model for COVID-19, stability analysis and numerical simulation of the SEIR model on the spread of the pandemic. The method used to construct the model is the SEIR model by considering vaccination and isolation factors as model parameters.

In [32] authors formulated a mathematical model for the transmission of COVID-19 disease and analyzed. In this study authors incorporated six classes; S- susceptible, Se - susceptible but educated to prevent from the disease, C-infected but asymptomatic, I-infected and symptomatic, R-recovered and E- pathogen concentration on contaminated surfaces. In [33] authors revised estimation of the risk of transmission of the novel coronavirus. Due to significant variations in the control strategies, which have been changing over time, and thanks to the introduction of detection technologies that have been rapidly improved, enabling to shorten the time from infection/symptoms onset to diagnosis, leading to faster confirmation of the new coronavirus cases, the previous estimations on the transmission risk of the COVID-19 need to be revised. Thus, authors re-estimated the effective daily reproduction ratio that better quantifies the evolution of the interventions.

In [34] authors projected the total and severe infections under different lockdown scenarios in east Africa. In this study authors used SECINISRD model where S = Susceptible individuals, E = Exposed, C = Contagious and asymptomatic or mildly symptomatic, IN= Infected with moderate symptoms, IS = Infected with severe symptoms, R = Recovered, D = Dead. The projection of this study was based on four scenarios; baseline, moderate lockdown, hard

lockdown and hard lockdown and continued physical distancing/isolating cases. Accordingly the projection result of Ethiopia with and without lockdown is presented. In [35] authors examined the transmission dynamics of COVID-19 and predict the growth of the infection in Bangladesh using the publicly available data though mathematical modeling. In this article the susceptible-exposed-infectious-recovered-dead (SEIRD) model is employed to describe the transmission dynamics. In [36] authors formulated a deterministic model which measures the effects of different rabies control methods (mass-culling and vaccination of dogs) for urban areas near wildlife. In this study mathematical model has been used to establish the impact of culling and vaccination of dogs. The model is similar with the one formulated in [43], but with a different environment; which demonstrates the importance of pre-existing mathematical models to analyze the impact of prevention and control mechanisms in a different environments.

The authors in [37] proposed the generalized SEIR epidemic model to investigate the dynamics of COVID-19. In this study, infectious individuals are investigated by dividing into two classes; infectious with intervention and infectious without intervention, besides the proposed model has considered quarantine and treatment. The parameters are estimated using Particle Swarm Optimization (PSO) algorithm. This study also explained that the parameters are changing since the control from the government is changing with time. Authors in [38] proposed model based study on the dynamics of COVID-19 prediction and control. In this study researches formulated a mathematical model introducing a quarantine class and government intervention measures to mitigate disease dynamics. In addition to this, authors analyzed the how these measures and control strategies affect the disease dynamics. In [39] authors developed a deterministic mathematical model of COVID-19 transmission in Ethiopia. This study proposed SEIR model with ordinary differential equation. Stability analysis and equilibrium point and sensitive analysis were carefully analyzed. However, the basic prevention and control strategies (quarantine, isolation and self-protection) are not analyzed and also the impacts of asymptomatic individuals are not considered separately.

2. Materials and Methods

A. Model Formulation

In this study, we have developed a mathematical model considering transmission route (human-to-human infection) and based on infection status and using deterministic compartmental approach by dividing the total population into seven mutually exclusive classes. In addition, this study incorporated separate analysis and prediction of the impact of symptomatic and asymptomatic individuals. Still now the findings and current approach is not available in the literature to the best of our knowledge. The main novelty of this work is that the number of classes, which is seven, inclusion of general self-protection parameter and separate analysis and prediction of the impact of symptomatic and asymptomatic individuals. Furthermore stability and equilibrium analysis of the model have been done clearly.

The total population N is divided into seven compartments; S-susceptible, Q-quarantined, E-exposed, I_S-infected and symptomatic, I_N- Infected and non- symptomatic, H-hospitalization (Isolation) and R-recovered individuals. In addition, non-pharmaceutical countermeasures known as self-protection parameter, were used which stands for staying at home, keeping physical distance, frequent hand-washing with soap or sanitizers and wearing masks. The proposed model mainly based on the following assumptions with time dependent state variables notations:

S(t) : Denotes the number of people in the susceptible compartment where the people are capable of being infected but not infected by the disease pathogen yet.

E(t) :Individuals who are in the incubation phase; they have been exposed to the pathogen but are not yet showing symptoms and are not infectious.

Q(t) :Denotes the number of people in the quarantined compartment. Some people who have been infected need to go through a certain incubation period before suspected symptoms can be detected. Quarantined individuals who do not develop symptoms are returned to the susceptible compartment after a maximum incubation time or after testing negative. Otherwise the individual will move to infectious, $I_S(t)$ compartment.

$\boldsymbol{I_S(t)}$:Denotes the number of individuals that are infected and having symptoms or show clinical signs.

$\boldsymbol{I_N(t)}$:Denotes the number of individuals that are infected and have no any symptoms or infected without showing any clinical signs.

H(t) :The number of individuals who hospitalized (isolated) due to illness.

R(t) :The number of individuals in the recover compartment where the person was previously detected as infectious survived the disease and has developed a natural immunity to the virus. The transmission dynamics considered in study is human-to-human root.

The total population, ***N*** is considered as constant. That is, N is not altered throughout the simulation of the model. (Since we assumed that the disease is pandemic). Zoonotic transmission is not considered in the proposed model (based on previous studies and reported cases). Dead individuals came from either class H (Hospitalized) or I (Infected), but not from class I_N (asymptomatic) individuals (based on the data).

In addition to the above state variables, we have transmission variables considered as the time independent parameters:

Table 1. Model parameters and their corresponding description.

Parameter	Description
B	Natural birth rate
$\boldsymbol{\beta_S}$	transmission probability $\mathbf{I_S}$ compartment
$\boldsymbol{\beta_N}$	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
μ	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
τ	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.

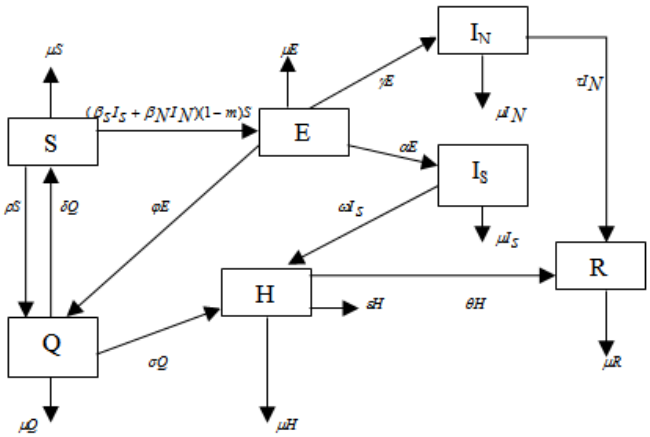

Fig.1. Block diagram representation of the $SEQI_SI_NHR$ dynamical model (1), with seven compartments representation.

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

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

The total population size is denoted by; $N(t)=S(t)+E(t)+Q(t)+I_S(t)+I_N(t)+H(t)+R(t)$ and satisfies $\frac{dN}{dt}=0$.

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;

$$\frac{dS}{dt}=B+\delta Q-(\beta_S I_S+\beta_N I_N)(-m)S-(\rho+\mu)S$$

$$\frac{dS}{dt}\geq -(\beta_S I_S+\beta_N I_N)(1-m)S-(\rho+\mu)S$$

$$\frac{dS}{S}\geq -\left((\beta_S I_S+\beta_N I_N)(1-m)+(\rho+\mu)\right)dt$$

$$\ln S\geq -\left((\beta_S I_S+\beta_N I_N)(1-m)+(\rho+\mu)\right)t+c$$

$$S(t)\geq A_1 e^{-\left((\beta_S I_S+\beta_N I_N)(1-m)+(\rho+\mu)\right)t} \tag{2}$$

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);

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

$$\frac{dE}{dt}\geq -(\alpha+\gamma+\varphi+\mu)E$$

$$\frac{dE}{E}\geq -(\alpha+\gamma+\varphi+\mu)dt$$

$$ln(E)\geq -(\alpha+\gamma+\varphi+\mu)t+C_1$$

$$E(t)\geq B_1 e^{-(\alpha+\gamma+\varphi+\mu)t}\text{ ,}$$

where

$$B_1=e^{C_1} \tag{4}$$

From inequality (4), at initial time, $t=0$ $E(0)\geq B_1$. Thus, inequality (4) becomes;

$$E(t)\geq E(0)e^{-(\alpha+\gamma+\varphi+\mu)t}\geq 0,\forall t\geq 0 \tag{5}$$

Repeating the same process for the remaining state variables, Q,I_S,I_N,H *and* R in the system (1), the following results are obtained:

$$(Q)\geq Q(0)e^{-(\delta+\sigma+\mu)t}\geq 0,\forall t\geq 0 \tag{6}$$

$$(I_S) \geq I_S(0)e^{-(\omega+\mu)t} \geq 0, \forall t \geq 0 \tag{7}$$

$$(I_N) \geq I_N(0)e^{-(\tau+\mu)t} \geq 0, \forall t \geq 0 \tag{8}$$

$$(H) \geq H(0)e^{-(\theta+\varepsilon+\mu)t} \geq 0, \forall t \geq 0 \tag{9}$$

$$(R) \geq R(0)e^{-\mu t} \geq 0, \forall t \geq 0 \tag{10}$$

Since $e^p \geq 0$ for all real values of *p* (where *p* is the exponents in all the above equations 6-10) then it is sufficient to conclude that the solutions for each state variable $S, E, Q, I_S, I_N, H and R$ of the model are positive for all $t \geq 0$. Having satisfied the basic features of the epidemiological model, the model system (1) were suitable to study the transmission dynamics COVID-19 disease and to analyze the impact of control and prevention strategies.

Invariant Region

The invariant region establishes the domain in which the solutions of the model are both biologically and mathematically meaningful. Hence, we shall show that the region Ω where the model is sensible remains positively invariant and attracting for all $t \geq 0$. That is, all the solutions in Ω remains in Ω for all $t \geq 0$. In other words the system (1) is well posed.

Theorem2:-The closed set $\Omega = \left\{(S, E, Q, I_S, I_N, H, R) \in \Re_+^7 : S + E + Q + I_S + I_N + H + R \leq \frac{B}{\mu}\right\}$ *is positivity invariant.*

***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;

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

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.

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

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 $\frac{dS}{dt}=0$, $\frac{dE}{dt}=$, $\frac{dI_S}{dt}=$, $\frac{dI_N}{dt}=0$, $\frac{dQ}{dt}=0$, $\frac{dH}{dt}=0$, $\frac{dR}{dt}=0$ 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);

$$\in_0=\left(S^0,E^0,Q^0,{I_S}^0,{I_N}^0,H^0,R^0\right)=\left(\frac{B}{\mu},0,0,0,0,0,0\right) \tag{16}$$

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 $x=(E,Q,I_S,I_N,H)$.

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

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

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

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

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

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

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

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

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

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;

$$|J_{\varepsilon_0}-I\lambda|=(\mu+\lambda)(a_6+\lambda)\left[\left((a_3+\lambda)(a_1+\lambda)-\delta\rho\right)\begin{vmatrix} -(a_2+\lambda) & a_7 & a_8 \\ \alpha & -(a_4+\lambda) & 0 \\ \gamma & 0 & -(a_5+\lambda) \end{vmatrix}\right]=0 \tag{15}$$

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;

$$\lambda^3+(a_2+a_4+a_5)\lambda^2+\left(a_2a_5+a_2a_4+a_4a_5-(\alpha a_7+\gamma a_8)\right)\lambda+a_2a_4a_5\left[1-\left[\frac{(\gamma a_8a_4+\alpha a_7a_5)}{a_2a_4a_5}\right]\right]=0$$

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

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

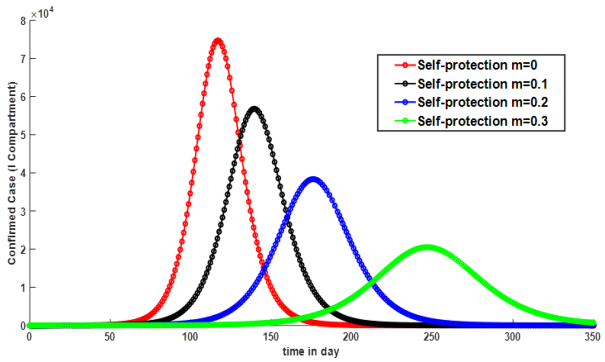

Fig.2.Variation of Confirmed cases for different values of m (Self-protection) (Effect of self-protection on confirmed cases)

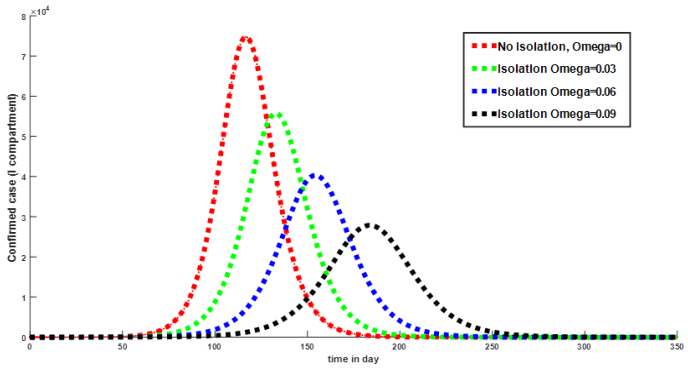

Fig.3.Variation of confirmed cases for different values of ω (Isolation Rate) (Effect of isolation rate on confirmed cases)

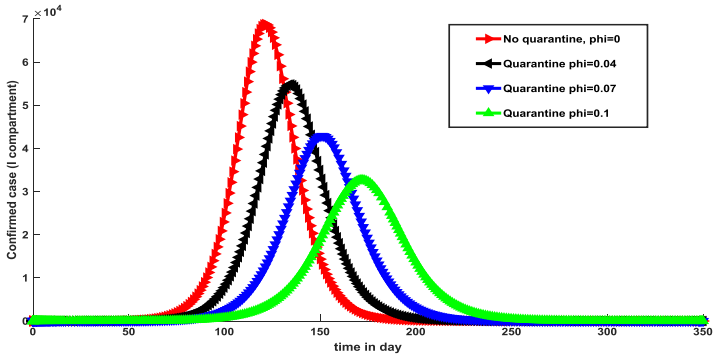

Fig.4.Variation of confirmed cases for different values of φ (Rate of Quarantine)

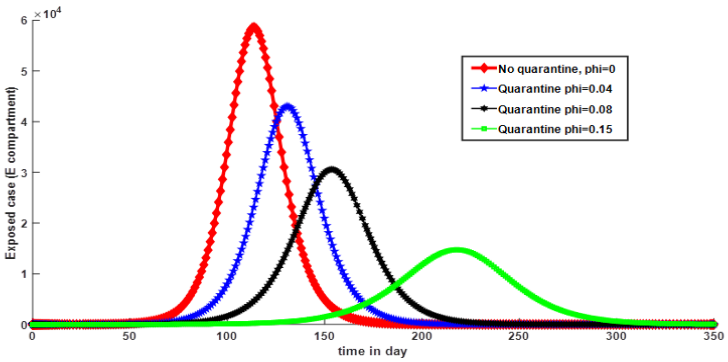

Fig.5.Variation of Exposed cases for different values of φ (Rate of quarantine) (Effect of rate of quarantine on exposed cases).

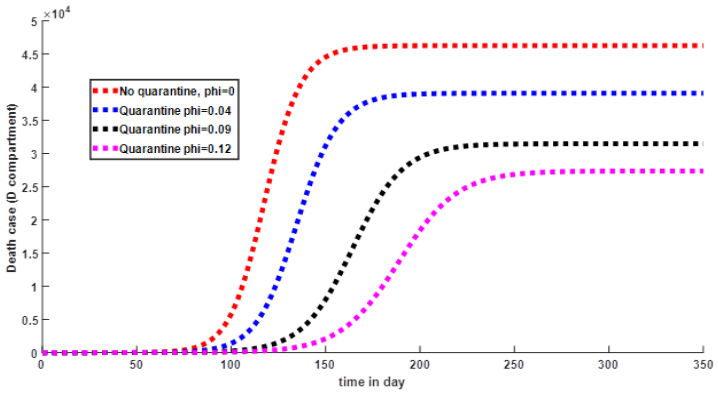

Fig.6.Variation of Death for different values of φ (Rate of quarantine) (Effect of quarantine on commutative death cases)

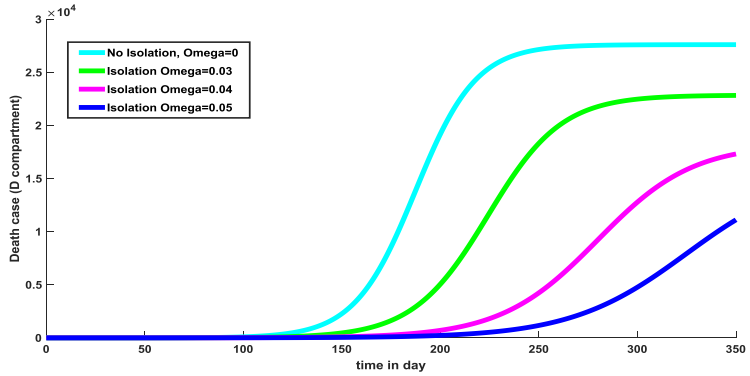

Fig.7.Variation of Death cases for different values of ω (Rate of isolation) (Effect of rate of isolation on death number)

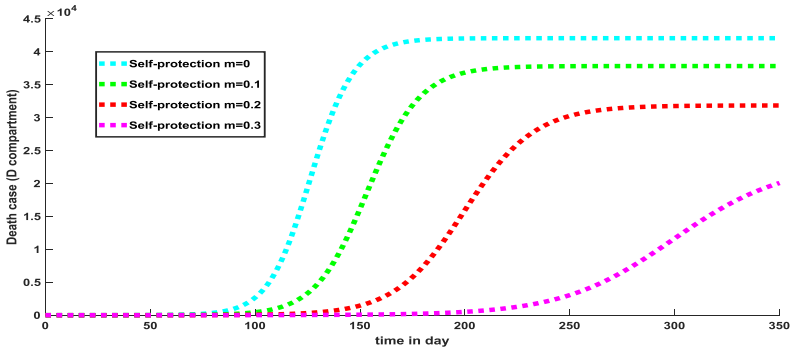

Fig.8.Variation of death cases for different values of m (Self-protection).(Effect of self-protection on death number)

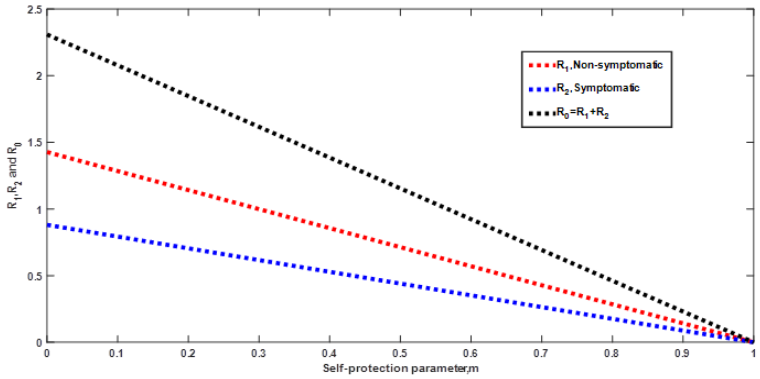

Fig.9.Graphs of R_1 , R_2 and R_0 verses m self-protection

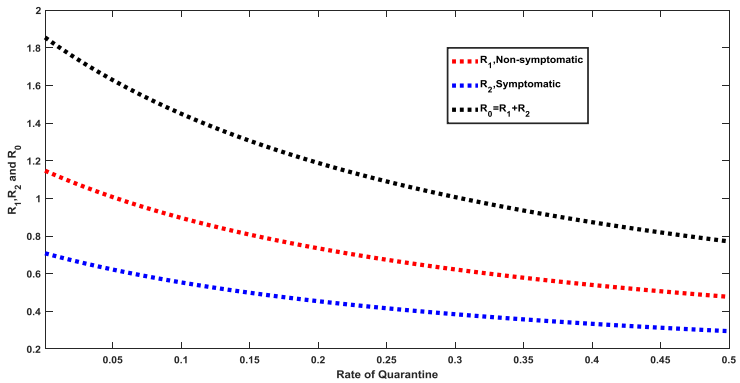

Fig.10.The graphs of R_1 , R_2 and R_0 verses (rate of quarantine).

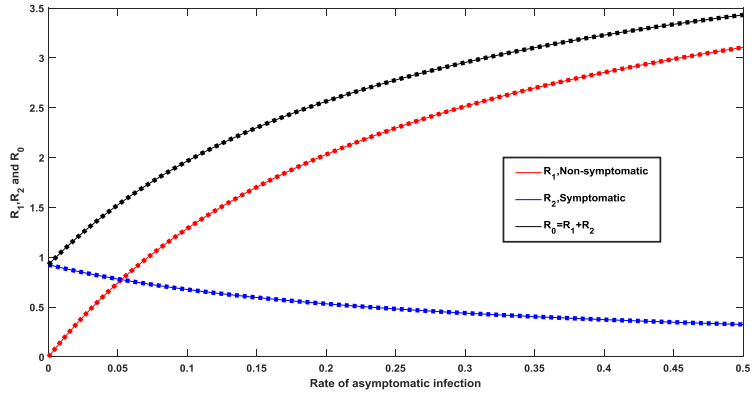

Fig.11.The graphs of R_1 , R_2 and R_0 verses the rate of non-symptomatic infection.

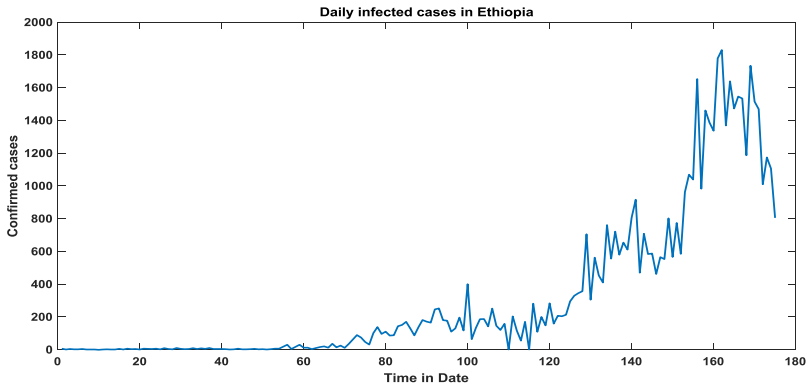

Fig.12. Daily confirmed cases real data

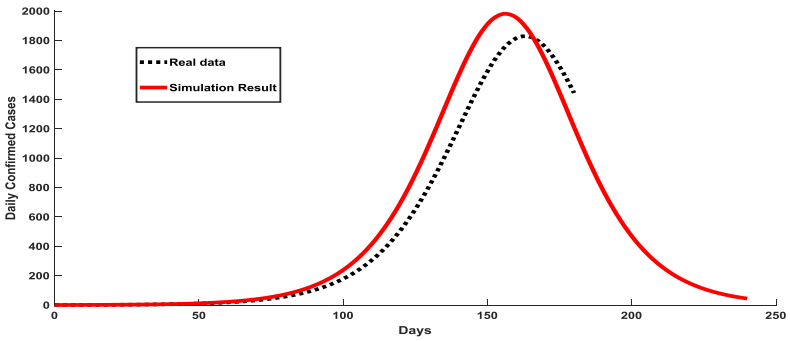

Fig.13. Fitting real daily infected case of COVID-19 in Ethiopia verses model simulation result of infected compartment.

5. Discussion

In this study we proposed $SEQI_SI_NHR$ model and analyzed the impact of prevention and control measures. The boundedness property of the system (1) and positivity of its solutions were examined. The basic reproductive number (R0) obtained using the standard next generation matrix and the effect of asymptomatic and symptomatic individuals on contributing for the value of R_0 were observed separately. Fig.1 shows R_1 (reproductive number as a result of asymptomatic individuals) had greater contribution to R_0 than R_2 (reproductive number as a result of symptomatic individuals), since 85% of infected individuals are asymptomatic [9, 13, 21, 23]. The disease free equilibrium of system (1) is locally asymptotically stable if R_0 <1 and unstable otherwise.

To study the impact of individuals control measures to prevent the extensive transmission of COVID-19, we have introduced a control parameter called self-protection, m. In fig.2 and 8 the impact of self-protection parameter on confirmed and death cases have been presented respectively. In the absence of self-protection (m=0) confirmed cases increases faster and reaches peak value. But, if the rate of self-protection parameter increases (10%, 20% and 30% of the susceptible population), the confirmed and death cases increases slowly and have less peak value depending on the

rate, m. Further, to reduce infection as well as to analyze the impact of government control measures, we incorporated quarantine of suspected individuals and isolation of infected persons. In fig. 3 and 7 the effect of rate of isolation on confirmed and death cases have been observed. In the absence of rate isolation ($\omega = 0$) both confirmed and death cases increases quickly as well as higher peak value. However, increasing the rate of isolation ($\omega = 0.03, 0.04 \& 0.09$) or decreasing isolation time in days (30days, 25days to 10days) reduces both confirmed and death cases.

In fig. 5, 6 and 7 the effect of rate of quarantine on confirmed, death and recovery cases have been presented. In the absence of rate of quarantine ($\phi = 0$) exposed, confirmed and death cases increases rapidly. Conversely, increasing the rate of quarantine ($\phi = 0.04, 0.08 \& 0.12$) or decreasing quarantine time in day (25days, 12days and 6days) automatically minimizes the infection and death as well as exposed cases. In fig.13 model validation has been done by using 180 days (March 13, 2020 to August 13, 2020) data. In fig.9 and 10 the value of R_0 decreased as a result of increasing self-protection parameter, ***m*** and rate of quarantine respectively. In fig 11, the graph of rate of asymptomatic infectious, γ verses R_0, R_1 and R_2 presented. As the value of γ increases, the corresponding reproductive rate, R_1, increase however, the R_2 is decreasing. This indicates that a series emphasis should be given to minimize contact rate between susceptible and asymptomatic individuals.

According to data verses simulation plot in fig.13, the model and the real /confirmed data of COVID-19 in Ethiopia are comparable which allowed us to analyze the control and prevention strategies of COVID-19 in Ethiopia. In the absence of isolation, quarantine and self-protection ($\omega = 0, \phi = 0 \& m = 0$) , the value of R_0=1.93 which is without considering social, cultural interaction and other scenarios.

6. Conclusion

According to reports from WHO and Ministry of Health (MoH) in Ethiopia, the sole opportunity to minimize COVID-19 infection is to implement the non-pharmaceutical approach via permitting to be in its standard. To apply these preventive and control measures, it is essential to; develop a dynamic model and analyze the effectiveness and impact of the proposed model as well as the associated parameters. In addition to this, the number of asymptomatic individual is larger than symptomatic ones. Thus, a serious emphasis should be given to reduce contact rate between susceptible and asymptomatic population.

In this study we proposed a dynamic $SEQI_SI_NHR$ model and analyzed the impact of prevention and control strategies. Further, the impacts asymptomatic individuals were separately analyzed. In order to curtail infection, increasing rate of isolation through quick and effective handling of confirmed individuals is essential and increasing rate of quarantine via various tracing mechanisms should come to function so that the number of days after being exposed to the virus will be reduced. In addition to these, the flow of individuals from susceptible to exposed compartment can be controlled by increasing m; hence increasing m minimizes risk being infected. In order to increase self-protection parameters, government intervention on implementation of powerful policies and guidelines is mandatory. On the other hand, considering asymptomatic infectious class separately indicates that there will be high risk of infection if there is no control measures to minimize the contact rate and rate of transmission from E to , .The higher transmissibility in symptomatic individuals was to be expected, whereas the higher infection rate of asymptomatic individuals may be recognized to them not taking the same safety measures as symptomatic people to avoid spreading the disease.

Based on the simulation result, we have come to the conclusion that the three sensitive parameters (prevention and control parameters), and (the rate of non-symptomatic infectious) are key factors to control the transmission of COVID-19 in Ethiopia. Thus, we conclude that the best procedure to control the disease is increasing isolation and quarantine rate. Besides, reducing contact rate at all cost so that both symptomatic but also importantly asymptomatic individuals stop transmitting of COVID-19.

Acknowledgment

The authors would to thank Dire Dawa University since this work has been supported by the grant from Dire Dawa University. Authors also like to acknowledge and appreciate Ms. Dursitu Abdella for supporting by organizing and feeding collected data.

References

[1] Chan JF, Yuan S, Kok KH, et al. (2020). A familial cluster of pneumonia associated with the 2019 novel coronavirus indicating person-to-person transmission: a study of a family cluster. Lancet 2020; 395:514.

[2] WHO Emergency Committee. Statement on the second meeting of the International Health Regulations (2005) Emergency Committee regarding the outbreak of novel coronavirus (COVID-19). Geneva: WHO, 2020. https://www.who.int/news-room/detail/30-01-2020

[3] Chayu Yang and Jin Wang. A mathematical model for the novel coronavirus epidemic in Wuhan, China, Mathematical Biosciences and Engineering. (2020) MBE, 17(3): 2708–2724

[4] National Health Commission of the People's Republic of China. 2020.
[5] WHO novel coronavirus (2019-nCoV) situation reports. Available from: https://www.who.int/emergencies/diseases/novel-coronavirus-2019/situation-reports.
[6] World Health Organization. Coronavirus disease 2019 (COVID-19): situation report, 82. 2020. Available from: https://www.who.int/docs/default-source/coronaviruse/situation-reports/20200411
[7] Guarner J. Three Emerging Coronaviruses in Two Decades: The Story of SARS, MERS, and Now COVID-19. American Journal of Clinical Pathology. 2020; 153(4): 420-421.https://doi.org/10.1093/ ajcp/aqaa029 10.
[8] Mahase E. Coronavirus covid-19 has killed more people than SARS and MERS combined, despite lower case fatality rate. BMJ (Clinical Research Ed.). 2020 Feb; 368:m641.
[9] Chatterjee K, Chatterjee K, Kumar A, Shankar S. Healthcare impact of COVID-19 epidemic in India: A stochastic mathematical model. Medical Journal Armed Forces India. 2020. Available from: https://doi. org/10.1016/j.mjafi.2020.03.022. 12.
[10] Khan T, Ullah Z, Ali N, Zaman G. Modeling and control of the hepatitis B virus spreading using an epidemic model. Chaos, Solitons and Fractals. 2019; 124: 1–9. https://doi.org/10.1016/j.chaos.2019.04. 033
[11] Herbert W. Hethcote (2000), "The Mathematics of Infectious Diseases", Society for Industrial and Applied Mathematics, Vol. 42, No. 4, pp. 599–653.
[12] Michael Hohle, Erik Jorgensen, Philip D. O'Neill (2005), "Inference in disease transmission experiments by using stochastic epidemic models", Appl. Statist. 54, Part 2, pp. 349–366.
[13] Paiva HM, Afonso RJM, de Oliveira IL, Garcia GF (2020) A data-driven model to describe and forecast the dynamics of COVID-19 transmission. PLoS ONE 15(7): e0236386. https:// doi.org/10.1371/journal.pone.0236386
[14] Kayla Henneman, Dan Van Peursem, Victor C. Huber (2013), "Mathematical modeling of influenza and a secondary bacterial infection", Wseas Transactions on Biology and Biomedicine, Issue 1, Volume 10.
[15] Frank Ball (1990), "Poission Approximation for Some Epidemic Models", J. Applied Probabilty, 27, 479-490.
[16] Michael Y. Li. An Introduction to Mathematical Modeling of Infectious Diseases, University of Alberta Edmonton, AB Canada. Springer International Publishing AG 2018. https://doi.org/10.1007/978-3-319-72122-4
[17] M. Choisy, J.-F. Guégan, and P. Rohani. Encyclopedia of Infectious Diseases:Modern Methodologies, Copyright © 2007 John Wiley & Sons,Inc. page 379-404
[18] Liliana Perez, suzana Dragicevic. An agent-based approach for modeling dynamics of contagious disease spread. International Journal of Health Geographics. (2009, 8:50 doc.10.1186/1476-072X-8-50
[19] C. Rothe, M. Schunk, P. Sothmann, G. Bretzel, G. Froeschl, C. Wallrauch, et al., Transmission of 2019-nCoV Infection from an asymptomatic contact in Germany, N. Engl. J. Med., 2020.
[20] Sheng Bin, Gengxin Sun and Chih-Cheng Chen. Spread of Infectious Disease Modeling and Analysis of Diff erent Factors on Spread of Infectious Disease Based on Cellular Automata. International journal of environmental research and public health. (2019)
[21] Chen et al. A mathematical model for simulating the phase-based transmissibility of a novel coronavirus. (2020) https://doi.org/10.1186/s40249-020-00640-3
[22] Juan Zhang. Modeling the Transmission of Middle East Respirator Syndrome Corona Virus in the Republic of Korea (2015) Article in PLoS ONE DOI: 10.1371/journal.pone. 0144778
[23] Yichi Li, Bowen Wang. Mathematical Modeling and Epidemic Prediction of COVID-19 and Its Significance to Epidemic Prevention and Control Measures. (2020) Annals of Infectious Disease and Epidemiology. Volume 5 | Issue 1 | Article 1052
[24] Frank G. Ball (1991), "Dynamic Population Epidemic Model", Mathematical Biosciences 107:299-324.
[25] Estimated effectiveness of symptom and risk screening to prevent the spread of COVID-19. Katelyn Gostic, Ana CR Gomez, Riley O Mummah, Adam J Kucharski, James O Lloyd-Smith. Epidemology and global health. https://elifesciences.org/articles/55570.
[26] B. Ivorraa, M.R. Ferrándezb, M. Vela-Pérezc, A.M. Ramosd . Mathematical modeling of the spread of the coronavirus disease 2019 (COVID-19) considering its particular characteristics. The case of China. Institute de mathematical interdisciplinary.
[27] Z. Liu , P. Magal , O. Seydi , G. Webb. A COVID-19 epidemic model with latency period, Infectious Disease Modelling 5 (2020) 323-337. https://doi.org/10.1016/j.idm.2020.03.003
[28] A. Anirudh, Mathematical modeling and the transmission dynamics in predicting the Covid-19 - What next in combating the pandemic. Infectious Disease Modeling 5 (2020). 366-374.
[29] S. M.MANOU-ABI, J. BALICCHI. Analysis of the covid-19 epidemic in French overseas department Mayotte based on a modified deterministic and stochastic SEIR model. doi: https://doi.org/10.1101/2020.04.15.20062752
[30] Z. Abbasi, I. Zamani and A.H.A. Mehra et al. Optimal Control Design of Impulsive SQEIAR Epidemic Models with Application to COVID-19. Chaos, Solitons and Fractals 139 (2020) 110054. https://doi.org/10.1016/j.chaos.2020.110054
[31] S. Annas, Muh. Isbar Pratama and Muh. Rifandi et al. Stability analysis and numerical simulation of SEIR model for pandemic COVID-19 spread in Indonesia. Chaos, Solitons and Fractals 139 (2020) 110072. https://doi.org/10.1016/j.chaos.2020.110072.
[32] S. Contreras, H.A. Villavicencio and D. Medina-Ortiz et al. A multi-group SEIRA model for the spread of COVID-19 among heterogeneous populations. Chaos, Solitons and Fractals 136 (2020) 109925. https://doi.org/10.1016/j.chaos.2020.109925
[33] S.M. Kassa, J.B.H. Njagarah and Y.A. Terefe . Analysis of the mitigation strategies for COVID-19: From mathematical modelling perspective. Chaos, Solitons and Fractals 138 (2020) 109968. https://doi.org/10.1016/j.chaos.2020.109968
[34] Isabel Frost.et al. COVID-19 in East Africa: National Projections of Total and Severe Infections under Different Lockdown Scenarios. Center for Disease Dynamics, Economics & Policy. 2020.
[35] Abul Mukid Md. Mukaddes and Mridul Sannyal. Transmission Dynamics of COVID-19 in Bangladesh- A Compartmental Modeling Approach. Shahjalal University of Science and Technology, Bangladesh.
[36] Edwiga Kishinda Renald, Katharina Kreppel, Dmitry Kuznetsov, " Desirable DogRabies Control Methods in an Urban setting in Africa - a Mathematical Model ", International Journal of Mathematical Sciences and Computing(IJMSC), Vol.6, No.1, pp.49-67, 2020. DOI: 10.5815/ijmsc.2020.01.05

[37] Shaobo He, Yuexi Peng and Kehui Sun. SEIR Modeling of the COVID-19 and its Dynamics. Nonlinear Dyn, Springer Nature B.V. 2020. https://doi.org/10.1007/s11071-020-05743-y
[38] Alberto Godio , Francesca Pace and Andrea Vergnano. SEIR Modeling of the Italian Epidemic of SARS-CoV-2 Using Computational Swarm Intelligence. International journal of environmental research and public health. 2020, 17, 3535; https://doi:10.3390/ijerph17103535
[39] Haileyesus Tessema Alemneh and Getachew Teshome Tilahun. Mathematical Modeling and Optimal Control Analysis of COVID-19 in Ethiopia. https://doi.org/10.1101/2020.07.23.20160473
[40] G. Birkhoff, G.C Rota, Ordinary Differential Equations. Boston: Ginn; 1982.
[41] Ega, T. T., Luboobi, L. S., and Kuznetsov, D. (2015). Modeling the dynamics of rabies transmission with vaccination and stability analysis. Applied and Computational Mathematics, 4(6):409–419.
[42] B.Tang et al. An updated estimation of the risk of transmission of the novel coronavirus (2019-nCov). Infectious Disease Modeling. 5(2020) p248-255. https://doi.org/10.1016/j.idm.2020.02.001

Authors' Profiles

Akalu Abriham has received BSC degree from Jigjiga University, Ethiopia in Applied Mathematics on July 2010 and MSC degree in Numerical Analysis from Jimma University, Ethiopia on June 2014. He has been serving as a lecturer and researcher at Dire Dawa University since September, 2010. He is currently serving as Assistant Professor of Numerical Analysis at Department of mathematics in Dire Dawa University. He has published papers in the area of Numerical Analysis. His current research interests include but not limited to Numerical Methods, Mathematical Modeling, Simulation Analysis, Computer Technology and Mathematics of Data Science**.**

Demis Dejene has received BSC degree in Mathematics from Dilla University, Ethiopia on September, 2010 and MSC degree in Computational Mathematics from Hawassa University, Ethiopia on July 2016. He has been serving as a lecturer and researcher in Dire Dawa University since 2016. Currently he is a PhD candidate in the field of Mathematical Modeling at Wollega University, Ethiopia. His main research areas are include but not limited to Mathematical modeling and Numerical Analysis and he has published papers on Mathematical Modeling.

Tadele Abera has received BSC degrees from Hawassa University, Ethiopia by Electrical Engineering in 2011 and MSC degrees from Addis Ababa University, Ethiopia by Electrical Engineering (Communication Engineering Specialization) in 2014. He was test and commissioning Engineer in different electric power upgrading projects in Ethiopia. He is currently lecturer in Dire Dawa University, Ethiopia, under School of Electrical and Computer Engineering. He has publications on ICAST 2019. His research interests are cognitive radio, massive MIMO, millimetre wave communication, backhaul networks and interference management techniques. His research interest extends to power systems and control including power reliability, power quality, PID controllers and MPC.

Abayneh Elias graduated in Medical Biochemistry (MSc. in Medical Biochemistry) in November, 2015 from Medical College Addis Ababa University, Ethiopia. He has conducted his thesis on the effect of traditional medicinal plants on the treatment of non-communicable diseases, specially dyslipidemia and hypertension in ex-vivo and in-vivo experiments in Swiss albino mice and guinea pigs and he found excellent results. Currently he is serving as lecturer in Dire Dawa University; he has been lecturing students of different departments of Health Sciences and Medicine since September, 2010. He is also actively participating in conducting researches and published papers.

How to cite this paper: Akalu Abriham, Demsis Dejene, Tadele Abera, Abayneh Elias," Mathematical Modeling for COVID-19 Transmission Dynamics and the Impact of Prevention Strategies: A Case of Ethiopia ", International Journal of Mathematical Sciences and Computing(IJMSC), Vol.7, No.4, pp. 43-59, 2021. DOI: 10.5815/ijmsc.2021.04.05