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# Mathematical modeling of covid-19 with emphasis on infected migrant asymptomatic infected and interacting peoples

Akindele Michael Okedoye <sup>1, 2, \*</sup>, Oluseye Shafau Seriki <sup>2</sup> and Savage Adeolu Nelson <sup>3</sup>

<sup>1</sup> Department of Mathematics, Covenant University, Ota, Ogun State, Nigeria.

<sup>2</sup> Department of Mathematics, Federal University of Petroleum Resources Effurun, Nigeria.

<sup>3</sup> Animal Production and Health Research Institute, 58 Sam Ewang Estate, Abeokuta, Ota, Ogun State, Nigeria.

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# Abstract

An S-A-I-Q-R epidemic model is investigated for Covid-19 as a class of infectious diseases that can be transmitted through carriers, not only infected individuals who are contagious but do not show any disease symptoms but through air which contained the virus droplets. Mathematical analysis is carried out that determines the global dynamics of the Modified Compartmental Epidemiological Model describing the transmission of the SARS-CoV-2 virus. The impacts of disease carriers on the transmission dynamics are analyze with emphasis on infected migrant, asymptomatic infected and social interacting people. We presented our discussion through the basic reproduction number as well as numerical simulations. We derived the condition for boundedness was obtained. Results: It was discovered that when  $\pi$ ,  $\delta$ ,  $\lambda$  and  $\rho$  are increased withing the first week of consideration, the reproduction number asymptotically approaches zero while a sudden increase in  $\tau$  was observed to result into a sudden increase in reproduction number within the first week of the outbreak and thereafter decreases with further increment in  $\tau$ . The result also showed that, population under quarantine are rarely infected with the virus except if already infected before being quarantined.

**Keywords:** Mathematical Modeling; Covid-19; Infected Migrant; Asymptomatic; Interacting Peoples; Epidemics; transmission; numerical simulations

# 1. Introduction

Communicable diseases are illnesses due to specific infectious agents or its toxic products, which arise through transmission of that agent, or its toxic products from an infected person, animal or inanimate reservoir to a susceptible host, either directly or indirectly, through an intermediate plant or animal host, vector or inanimate environment Mulugeta [1]. People sometimes refer to communicable diseases as "infectious" or "transmissible" diseases. Pathogens such as bacteria, viruses, fungi, and protists, are causes of communicable diseases [2]. A person may develop a communicable disease after becoming infected by the pathogen. A detailed mathematical model for assessing the community-wide impact of Non-Pharmaceutical Interventions (NPIs) on combating and mitigating the burden of COVID-19 was developed by Ngonghala et al. [3]. Their study showed that, while the early relaxation or lifting of social-distancing and community lockdown measures (and facemask usage in the public) is likely to lead to second wave, extended the duration of the social-distancing and lockdown measures (and face mask usage in public) can significantly reduce the COVID-induced mortality in the US in general, and the state of New York in particular. The potential for a COVID-19 outbreak aboard the Diamond Princess cruise (which experienced a major COVID-19 outbreak during the months of January and February of 2020) was modeled by [4]. Their study showed a high estimate of the reproduction of the model (making major outbreak inevitable), and that the reproduction number substantially decreases with increasing effectiveness of the quarantine and isolation measures implemented on the ship. Adegboye et al. [5] used a

<sup>\*</sup> Corresponding author: Akindele Michael Okedoye

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Figure 1 Schematic Diagram for the system

log-linear Poisson regression model to estimate the early transmissibility of the novel coronavirus in Nigeria. Their 45day estimates

Nigeria has been remarkably lower than expected, more testing needs to be done to stop local transmission. Musa et al [6] estimated the growth rate and basic reproduction number of the novel coronavirus to show the potential of the virus to spread and to reveal the importance of sustaining stringent health measures to control the disease in Africa. Using a stochastic model, Hellewell et al [7] showed that (for most instances) the spread of COVID-19 can be effectively contained in 3 months if contact-tracing and isolation are highly effective. Furthermore, using another stochastic model to study the COVID-19 trajectory in the Wuhan city of China from January to February, 2020, Kucharski et al [8] showed that a reduction in COVID-19 transmission can be achieve when travel restrictions are implemented. Using a model for assessing the effect of mass influenza vaccination on the spread of COVID-19 and other influenza-like pathogens cocirculating during an influenza season, Li et al [9] showed that increasing influenza vaccine uptake (or enhancing the public health interventions) would facilitate the management of outbreaks of respiratory pathogens circulating during the peak flu season.

Crokidakis [10] work is to implement social isolation in a simple way in a compartmental epidemic model, and study it impacts on the evolution of cases in Rio de Janeiro state. In this case, they considered a Susceptible-Infectious-Quarantined-Recovered (SIQR) model. Data were collected from the Rio de Janeiro state Department of Health [11], it was observed that the social distancing policies led about 7 days to effectively decrease the rate of growth of confirmed cases in Rio de Janeiro. It was deduced that there was a sub-exponential growth of cases, and that the non-exponential behavior is reproduced by their model. In the work of Perra et al. [12], mathematical model was used to study the epidemic dynamics and reproductive number,  $R_0$  estimate for COVID-19 in an entirely susceptible Ghanaian population. An estimate of the initial basic Reproductive number and the time-dependent Reproductive number at any time during the outbreak using four different estimation procedures was provided.

# 2. Mathematical formulations

To fully understand the transmission dynamics of COVID-19, we revisit the work of Perra. et al. (2011) and Crokidakis (2021), and based on the available information and the basic understanding of the biological mechanism through which infection spreads in the population, we proposed a model that could generally describes the dynamics of Covid-19 outbreak. To achieve the above, we partitioned the population into five distinct classes (susceptible S, Asymptomatic A, Infectious I, Self-isolation/Quarantined Q, and Recovery R).

We defined susceptible populations as all uninfected individuals at time t but there is the likelihood they may be infected at timet, where t is the incremental continuous-time point. We included newborn babies from all compartments into the susceptible class since there is not enough evidence on vertical transmission of the virus from the pregnant woman to the unborn baby. In order to ensure full control of the disease, we assume that all the population is

Susceptible from which some may show the symptom and some may not. We further classified those who have shown the symptom as Infected class and those who do not show any symptom are likely not to be infected, Asymptomatic

while those who go on self (or forced)-quarantined due to interaction with people are classified as Quarantine. Those who have been confirmed infected are quarantined, treated, recovered or die due to the diseases and those who are confirmed to be infected while in quarantine are moved to infection class. It is also considered that the induced death rate is slightly different from the birth rate of each class while the new birth is considered to be susceptible due to unrestricted interactions within the system.

Further, we assume that

- The confirmed infected individual comes from the asymptomatic infected, quarantined people, infected migrant and contaminated susceptible person by an asymptomatic infected individual.
- Confirmed individual are isolated (quarantine). The quarantine individual is treated and either recovered fully or die.
- Recovered person if exposed can be infected again.
- Latent infections can be confirmed infected if tested, treated, recovered or die, or natural immunity neutralizes the virus or died unconfirmed.
- Susceptible population can only increase through either immigration of infected person or the time scale is short enough so that natural births and deaths are neglected.

Implementing the above assumptions, the modification to Crokidakis [10] and Perra. et al. [12] are represented by the system of equations governing the system under consideration and is written as:

$$\frac{dS}{dt} = \omega - (\beta A + \gamma I)S - \alpha Q + \eta R,$$

$$\frac{dA}{dt} = \beta AS + \rho Q - (\delta + \lambda + \pi + \tau)A,$$

$$\frac{dI}{dt} = \gamma SI + \delta A + \theta Q - (\nu + \mu + \tau)I,$$

$$\frac{dQ}{dt} = \alpha Q + \nu I + \pi A - (\rho + \theta + \xi + \tau)Q,$$

$$\frac{dR}{dt} = \xi Q + \mu I + \lambda A - \eta R.$$
(1)

The current model is more general than the carrier model by incorporate disease-caused death. We also assumed that during the outbreak, new births are recorded and some of the carriers asymptomatic within the disease outbreak period.

## 2.1. Feasible Region for Equilibrium points

#### 2.1.1. Total Population

First of all, it is natural to ask whether this dynamical system provides a non-negative trajectories or not.

The total population *N* is obtained by adding the number of susceptibles (*S*), Asymptomatic (A), Infected carriers (*I*), Quarantined (Q) and those recovered (*R*) from the disease. So then we have

$$N = S(t) + I(t) + A(t) + Q(t) + R(t)$$
(2)

Thus the total accumulative population is given as

$$\sum \frac{d\psi}{dt}$$
, where  $\psi = S(t) + I(t) + A(t) + Q(t) + R(t)$ 

Thus

$$\frac{d\psi}{dt} = \omega - (\beta A + \gamma I)S - \alpha Q + \eta R + \beta AS + (\rho)Q - (\pi + \delta + \lambda + \tau)A + \gamma SI + \delta A + \theta Q$$
$$-(\nu + \mu + \tau)I + \alpha Q + \nu I + \pi A - (\rho + \theta + \xi + \tau)Q + \xi Q + \mu I + \lambda A - \eta R$$

That is

$$N' = \omega - \tau (A + I + Q)$$
(3)  

$$N' = \omega - \tau (S + A + I + Q + R) + \tau S + \tau R$$
  

$$N' = \omega + \tau S + \tau R - \tau N$$
  

$$N' = \omega - \tau N + \tau S + \tau R$$
  

$$N' - \tau (S + R) = \omega - \tau N$$

Which means

 $N' \leq \omega - \tau N$ 

That is

$$N' + \tau N \le \omega \tag{4}$$

Multiply (4) by  $e^{t\tau}$ , we have

 $e^{t\tau}N' + \tau e^{t\tau}N \leq \omega e^{t\tau}$ 

Thus

$$\frac{d}{dt}(e^{t\tau}N) \le \omega e^{t\tau} \tag{5}$$

Integrating (5) we have

 $(e^{t\tau}N) \leq \frac{\omega}{\tau}e^{t\tau}$ 

That is

$$N \le \frac{\omega}{\tau} \tag{6}$$

## 3. Method of Solution and Discussion of Results

In this section, we shall study the dynamic properties of the Covid-19 model (1) feasible region for equilibrium points under the following headings:

## 3.1. Equilibrium Points

The equilibrium point of the system denotes a time when the rate of change of the population is zero.

## 3.1.1. The Disease Free Equilibrium Point (DFEP)

Disease-free equilibrium (DFE) points are steady-state solutions where there is no corona virus infection and the equilibrium points are obtained by setting the right-hand sides of the model equations to zero according to Molalegn and Purnachandra [13]. Disease free equilibrium points are steady state solutions of the system of ODE in the absence of disease in the population that is when I = Q = 0 and then solve the resulting system of non-linear equations. Solving the reduced system of equations, we have

$$(S, A, I, Q, R) = \left(\frac{(\pi + \delta + \lambda + \tau)}{\beta}, \frac{\omega}{(\pi + \delta + \tau)}, 0, 0, \frac{\lambda\omega}{\eta(\pi + \delta + \tau)}\right)$$

#### 3.1.2. The Endemic Equilibrium Point (FEP)

By setting the system of equations to zero and evaluating the state variables, the endemic equilibrium points would be in the form:  $(EE) = \{S^*, A^*, I^*, Q^*, R^*\}$ 

The necessary and sufficient condition for an endemic equilibrium  $P^* = \{S^*, A^*, Q^*, I^*, R^*\}$  to exist in the feasible region  $\Omega$  is that

$$(S^*, A^*, I^*, Q^*, R^*) = \left( \left(\frac{c}{\beta} + \frac{\rho}{a_1 \beta}\right), \left(\frac{\upsilon \omega}{m\pi} + \frac{a_2}{\pi}\right), \left(\frac{n\Delta}{m} - \frac{\omega}{m}\right), \Delta, \frac{\omega(m-e)}{m} - u\Delta \right)$$
(7)

Where

$$\begin{aligned} \alpha_1 > \frac{n(\pi+\upsilon)}{m}, \frac{\upsilon}{m}(\omega+n) - \alpha_1, \Delta &= \frac{-(\upsilon+\pi)\omega - \sqrt{(\upsilon+\pi)^2\omega^2 - \frac{4x\omega n\pi}{\tau}}}{2r}, a_1 = \left(\frac{1}{\pi}\left(\frac{\upsilon}{m}(\omega+n) - \alpha_1\right)\right), a_2 \\ &= \left(\frac{\upsilon n}{m} - \alpha_1\right)\Delta, c = (\delta + \lambda + \pi + \tau) \end{aligned}$$

## 3.2. The Basic Reproduction Number (BRN)

The carriers in the given system can have great effect on the basic reproduction number  $R_0$ . The parameters (S, A, I, Q, R) are all related to the carrier class and they also appear in the BRN.

The basic reproduction number is given by the relation

$$0 < S^* < S \Longrightarrow 0 < 1 \le \frac{S}{S^*}$$

Thus

$$R_0 = \frac{S}{S^*} \tag{8}$$

But

$$S = \left(\frac{\pi + \delta + \lambda + \tau}{\beta}\right), S^* = \left(\frac{c}{\beta} + \frac{\rho}{\alpha_1 \beta}\right)$$
$$R_0 = \frac{S}{S^*} = \frac{\left(\frac{\pi + \delta + \lambda + \tau}{\beta}\right)}{\left(\frac{c}{\beta} + \frac{\rho}{\alpha_1 \beta}\right)} = \frac{c}{\left(c + \frac{\rho}{\alpha_1}\right)} = \frac{\alpha_1 c}{\alpha_1 c + \rho} = \frac{1}{1 + \frac{\rho}{\alpha_1 c}}$$
(9)

This implies

 $R_0 \leq 1$ 

Thus equality holds when ho=0 and strictly less than zero holds whenever ho>0

Consider the effect of each one after the other

$$(\pi + \delta + \lambda + \tau)(\omega + \rho - \alpha + \theta + \xi)$$

#### 4.3 Stability Analysis of the DFE

Here we analyze the stability of the (DFE) and the Endemic Equilibrium Point (EE) using a Lyapunov function V(t) define as

$$V = \frac{1}{2}(S(t)^2) + (A(t)^2) + (I(t)^2) + (Q(t)^2) + (R(t)^2)$$
(10)

Differentiating (10) with respect to t,

$$\Rightarrow \frac{dV}{dt} = S(t)\left(\frac{d}{dt}S(t)\right) + A(t)\left(\frac{d}{dt}A(t)\right) + I(t)\left(\frac{d}{dt}I(t)\right) + Q(t)\left(\frac{d}{dt}Q(t)\right) + R(t)\left(\frac{d}{dt}R(t)\right)$$
(11)

Substituting  $\frac{d}{dt}(*)$  into equation (16) and simplifying,

$$\frac{dV(t)}{dt} = -S(t)^{2} \Big( A(t)\beta + \gamma I(t) \Big) - (\rho + \theta + \xi + \tau - \alpha)Q(t)^{2} - R(t)^{2}\eta - (\upsilon + \mu + \tau)I(t)^{2} 
- (\delta + \lambda + \pi + \tau)A(t)^{2} + (\gamma I(t)^{2} + \beta A(t)^{2})S(t) + ((\pi + \rho)A(t) + (\theta + \upsilon)I(t) - \alpha S(t) + R(t)\xi)Q(t) 
+ (\delta I(t) + \lambda R(t))A(t) + R(t)\mu I(t) + \eta R(t)S(t) + \omega S(t)$$
(12)

By the boundedness on the variables and parameters,

$$\frac{dV(t)}{dt} \le 0 \text{ iff}$$

$$\begin{aligned} (S(t)^{2}.(A(t).\beta + \gamma.I(t)) + (\rho + \theta + \xi + \tau - \alpha)Q(t)^{2} + R(t)^{2}\eta + (\upsilon + \mu + \tau)I(t)^{2} + (\delta + \lambda + \pi + \tau)A(t)^{2}) \\ &\geq ((\pi + \rho)A(t) + (\theta + \upsilon)I(t) - \alpha S(t) + R(t)\xi)Q(t) + (\delta I(t) + \lambda R(t))A(t) + R(t)\mu I(t) + \eta R(t).S(t) \\ &+ \omega S(t) + (\gamma.I(t)^{2} + \beta A(t)^{2}).S(t) \end{aligned}$$

$$\Rightarrow \left( (\pi + \rho)A(t) + (\theta + \upsilon)I(t) - \alpha S(t) + R(t)\xi \right)Q(t) + \left(\delta I(t) + \lambda R(t)\right)A(t) + R(t)\mu I(t) + \eta R(t)S(t) + \omega S(t) + (\gamma I(t)^2 + \beta A(t)^2)S(t) \leq S(t)^2 (A(t)\beta + \gamma I(t)) + (\rho + \theta + \xi + \tau - \alpha)Q(t)^2 + R(t)^2\eta + (\upsilon + \mu + \tau)I(t)^2 + (\delta + \lambda + \pi + \tau)A(t)^2$$

$$(13)$$

Thus

$$\frac{d}{dt}V(t) \le 0$$

Which is negative definite

Hence the system is stable.

For DFE, A = I = Q = 0, system (10) becomes

.

$$\frac{d}{dt}S(t) = \omega - S(t) \tag{14}$$

Defining

$$V = \frac{1}{2}S(t)^{2}$$

$$\Rightarrow \frac{dV}{dt} = S(t)\left(\frac{d}{dt}S(t)\right)$$
That is  $\frac{dV}{dt} = S(t)(\omega - S(t))$ 
(15)

This implies

$$\frac{dV}{dt} = \omega S(t) - S(t)^2 \le 0 \text{ if } f, \omega \le S(t)$$
(16)

That is DFE is stable.

Hence both DFE and EE are stable.

## 3.3. Numerical Simulations of the Model

In this section, we present numerical simulations for the model. A numerical Simulation of the model will therefore be conducted in order to find out the dynamics (behavior) of the disease in the human population. The numerical simulations presented here will be were conducted using the rkf45 solvers coded in Maple programming Language. The results will be shown graphically in order to investigate the dynamics of the disease and then proffer solution based on the numerical simulations as to which method will be more effective in eradicating the disease. Should we increase the vaccination rate  $\theta$  or rather use diagnosis in order to make the those infected in the population aware of their condition and reduce their rate of contact with people, and so prevent the spread of the disease.

Given the following system of differential equation with initial conditions (in terms of proportion), Here we make use of

$$S(0) = 10; A(0) = 3; I(0) = 0; Q(0) = 0 \text{ and } R(0) = 0.0$$

Baseline values of the parameters for model (1) were chosen as

$$\omega = 0.5, \beta = 0.1, \delta = 0.1, \lambda = 0.2, \alpha = 0.001, \pi = 0.15, \theta = 0.0002, \gamma = 0.0001, \mu = 0.102, \tau = 0.4, \xi = 0.0088, \eta = \frac{1}{14}, \nu = 0.015, \rho = 0.001$$

#### 4. Results

Here, we get deep insights into the complex behavior of the model. Under various computational simulations of the proposed model for state variables of interest, the transmission dynamics of an infectious disease can be adequately understood. The Susceptible, Asymptomatic, Infected, Quarantined and Recovered populations are investigated with different values of the parameters.

As shown in Figure 2, an increase in the susceptible humans sub-class S (t) triggers or boost the dynamics of Covid-19 while a decrease in the rate of Infectious humans I(t) declines it. It is worthy of note that an appreciation in the rate of asymptomatic infection humans A(t) depreciates the dynamics of Covid-19 and an increase in the rate of quarantined humans Q(t) causes a decline of the Covid-19 dynamics. For a fact, an enhancement of the rate of symptomatic infectious humans is unsupported by the Covid-19 dynamics. Figure 4.2 represent the different values of  $\beta$  and how it affects the susceptible human sub-class. An increase in the value of  $\beta$  diminishes the rate of susceptible human's profile.

In Figure 2, the dynamics of the infection is displayed as a function of time in weeks. The figure depicts the interactions between the classes of in the dynamics. From the figure, we observed that the population of susceptible class decreases while the infected quarantine and recovery class increases. The Asymptomatic class was seen to remain steady in the first few days after which it also declined as a result of precautions put in place. It is worthy of note that an appreciation in the rate of asymptomatic infectious humans depreciates the dynamics of Covid-19 and an increase in the rate of quarantined humans causes a decline of the Covid-19 dynamics. Figure 3 depict the dynamics of susceptible class against the recruitment into asymptomatic class. We discovered that as the rate of recruitment increases, the susceptible population decreases. In Figure 4, the effect of immigration rate on susceptible is depicted. This figure shows that increase in immigration rate trigger the susceptible population. If the immigration rate is allowed to continue unabated, the implication from the profile is that the population become unstable as the infection rate will be oscillating. This is what is being experienced in some part of Europe and Asia recently with more people being exposed to the virus resulted in pandemic. Figures 5 and 6 indicate the effect of rate of quarantined asymptomatic class and release rate from asymptomatic to symptomatic on susceptible class. It could be seen that if rate of quarantined asymptomatic class is increased, the susceptible population decreases as more of the infected persons would be under quarantine thereby reducing the exposure population while the rate of release from asymptomatic to symptomatic initially dcreases but later after about few days raises the exposure population, and as release rate from asymptomatic to symptomatic the population of susceptible class increases.

Figures 7 – 10 depict the effect of recruitment into asymptomatic class rate, disease induced death rate, release from asymptomatic to symptomatic rate respectively and rate of people recovering from asymptomatic class. From the figures, recruitment into asymptomatic class rate enhances the asymptomatic population while both disease induced death rate, release from asymptomatic to symptomatic rate decline the asymptomatic population. And as expected, rate of people recovering from asymptomatic sub-population decline the decline the asymptomatic population. The peak in the profile indicates that, at lower value of recovery, the asymptomatic population suddenly increases before later declining due to preventive and treatment put in place.

The effect of rate of recovering from infected class, rate of quarantined asymptomatic class, rate of recovering from asymptomatic people, rate of quarantined becoming infected and rate of infection were displayed in Figures 11 – 15. We discovered from the figures that rate of recovering from infected class, rate of quarantined asymptomatic class and rate of recovering from asymptomatic people decreases the infected population whereas, rate of quarantined becoming infected and rate of infection increases the infected population. The Quarantine population is seen to be enhanced by increase in immigration rate because of the enforcement of government policy as shown in Figure 16, while Figures 17 – 20 indicated that increase in rate of people recovering from asymptomatic class, rate of quarantined becoming infected, disease induced death rate and of undiagnosed quarantine infection all brings about reduction in the quarantine sub population.



Figure 2 Covid-19 dynamics with reference to subclasses characteristics



Figure 4 The different values of  $\varphi$  for the susceptible human sub-class



**Figure 3** The different values of  $\beta$  for the susceptible human sub-class



**Figure 5** The different values of  $\pi$  for the susceptible human sub-class

The recovery dynamics is shown to be affected by rate of recruitment into asymptomatic class, release rate from asymptomatic to symptomatic and disease induced death rate as displayed in Figures 21 – 23. From these figures, we discovered that as the rate of recruitment into Asymptomatic class increases, the revery rate also increases as depicted

in Figure 21 while, release rate from asymptomatic to symptomatic and disease induced death rate were seen to decrease the recovery sub population.



**Figure 6** The different values of  $\delta$  for the susceptible human sub-class



Figure 8 The different values of  $\tau$  for the asymptomatic human sub-class



**Figure 10** The different values of  $\lambda$  for the asymptomatic human sub-class



**Figure 7** The different values of  $\beta$  for the asymptomatic human sub-class



Figure 9 The different values of  $\delta$  for the asymptomatic human sub-class



Figure 11 The different values of  $\mu$  for the infected human sub-class



**Figure 12** The different values of  $\pi$  for the infected human sub-class



**Figure 14** The different values of  $\theta$  for the infected human sub-class



Figure 16 The different values of  $\varphi$  for the quarantine human sub-class



Figure 13 The different values of  $\lambda$  for the infected human sub-class



Figure 15 The different values of  $\gamma$  for the infected human sub-class



Figure 17 The different values of  $\lambda$  for the quarantine human sub-class



Figure 18 The different values of  $\theta$  for the quarantine human sub-class



Figure 20 The different values of  $\rho$  for the quarantine human sub-class



Figure 22 The different values of  $\delta$  for the recovery human sub-class



Figure 19 The different values of  $\tau$  for the quarantine human sub-class



Figure 21 The different values of  $\beta$  for the recovery human sub-class



Figure 23 The different values of  $\tau$  for the recovery human sub-class

Parameter Description			
π	Rate of quarantined asymptomatic class	γ	Rate of infection
β	Rate of recruitment into Asymptomatic class rate	ω	Immigration rate
α	Rate of quarantined susceptible peoples	η	Rate of recovery from the virus
λ	Rate of recovering from asymptomatic people	ν	Rate of infection quarantine
θ	Rate of quarantined becoming infected	μ	Rate of recovering from infected
δ	Release rate from asymptomatic to symptomatic	ξ	Rate of recovery from quarantine
ρ	of undiagnosed quarantine infection	τ	Disease induced death rate

# 5. Discussion

In our model, we study the epidemic patterns of Covid-19, from a mathematical modeling perspective. The present model is developed making some reasonable modifications in the corresponding epidemic SAIQR model by considering susceptible and asymptomatic infective immigrants as well as self-isolation measures. Our numerical results indicate that the corona virus infection would remain pandemic, unless the responsible body takes Self isolation measure and intervention programs to reduce the transmission of the disease from asymptomatic and infected individual to the susceptible individual. Among the model parameters the exposed and infected self-isolation rate and exposed (probably asymptomatic infected) immigration rate are very sensitive parameters for the spread of the virus. Disease free equilibrium point is found and endemic equilibrium state is identified. It is shown that the disease free equilibrium point and the endemic equilibrium point is carried out using *MAPLE 2019*.

# 6. Conclusion

In the present study, It is found that the main reasons of successfully controlling and eliminating disease improving of self-protecting ability of susceptible, isolating all the close contacts, where asymptomatic cases or exposed cases are all belong to close contacts. And it tells us that quickly finding and self-isolation of exposed (asymptomatic cases) can effectively control the spread of disease. We have defined the basic reproductive number which provides the expected number of new infections from one infectious individual over the duration of the infectious period given that all other members of the population are susceptible. We also showed the existence of equilibrium points. Numerical simulation of the model shows the dynamic properties of human compartments versus time and the stabilities of the equilibrium points. We can observe from the numerical simulations that human mobility increases the dynamics and spread of covid-19 pandemic.

# Compliance with ethical standards

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# Author Contribution

All authors contributed equally.

# Disclosure of conflict of interest

The authors declare that there is no conflict of interest.

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