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COVID-19 created chaos across the globe: Three novel quarantine epidemic models

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\textbf{A B S T R A C T}

The latest version of human coronavirus said to be COVID-19 came out as a sudden pandemic disease within human population and in the absence of vaccination and proper treatment till date, it daunting threats heavily to human lives, infecting more than 12, 11, 214 people and death more than 67, 666 people in 208 countries across the globe as on April 06, 2020, which is highly alarming. When no treatment or vaccine is available till date and to avoid COVID-19 to be transmitted in the community, social distancing is the only way to prevent the disease, which is well taken into account in our novel epidemic models as a special compartment, that is, home isolation. Based on the transmitting behavior of COVID-19 in the human population, we develop three quarantine models of this pandemic taking into account the compartments: susceptible population, immigrant population, home isolation population, infectious population, hospital quarantine population, and recovered population. Local and global asymptotic stability is proved for all the three models. Extensive numerical simulations are performed to establish the analytical results with suitable examples. Our research reveals that home isolation and quarantine to hospitals are the two pivot force-control policies under the present situation when no treatment is available for this pandemic.

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1. Introduction

Pandemic diseases right from Antonine plague (165 A. D.) to the present coronavirus disease 2019 (COVID-19) always have created severe disaster to human kind. As per the report of World Health organisation (WHO) over 208 countries across the globe with total confirmed infected cases of 12, 11, 214 with 67, 666 cases of death due to coronavirus as on April 06, 2020, not only are people widely calling COVID-19 both an epidemic and pandemic, but they are also calling it an outbreak. An outbreak is a “sudden breaking out or occurrence” or “eruption.” When referring to an infectious disease, an outbreak is specifically a sudden rise in cases, especially when it is only or so far affecting a relatively localized area. A pandemic disease is an epidemic that has spread over a large area, that is, it’s “prevalent throughout an entire country, continent, or the whole world.” A pandemic is defined as “an epidemic occurring worldwide, or over a very wide area, crossing international boundaries and usually affecting a large number of people” [1]. Most people infected with the COVID-19 virus experiences mild to moderate respiratory illness and recover without requiring special treatment. Older people, and those with underlying medical problems like cardiovascular disease, diabetes, chronic respiratory disease, and cancer are more likely to develop serious illness. The COVID-19 virus spreads primarily through droplets of saliva or dis-
charge from the nose when an infected person coughs or sneezes [2].

1.1. History of pandemic diseases

The first ancient pandemic well back around 165 A. D. known as the Antonine Plague generally referred as the Plague of Galen, was a pandemic that affected Asia Minor, Egypt, Greece, and Italy and is thought to have been either Smallpox or Measles, though the true cause is still unknown and have killed over approximately 5 million people [3]. Then arose the Plague of Justinian around 541–542 A. D. which is assumed to have killed perhaps half the population of Europe, was an outbreak of the bubonic plague that afflicted the Byzantine Empire and Mediterranean port cities, killing up to 25 million people in its year long reign of terror [4]. From 1346 to 1353 A. D. an outbreak of the Plague called as the Black Death ravaged Europe, Africa, and Asia, with an estimated death toll between 75 and 200 million people. This is assumed to have originated in Asia, the Plague traversed to continents via the fleas living on the rats that so frequently lived aboard merchant ships [5]. Like the first and second pandemics, the Third Cholera Pandemic originated in India, spreading from the Ganges River Delta before making disaster through Asia, Europe, North America and Africa and ending the lives of over a million people. Originally the “Asiatic Flu” or “Russian Flu” (scientifically said to be FLU PANDEMIC (1889–1890)) as it was called, this strain was thought to be an outbreak of the Influenza A virus subtype H2N2, though recent discoveries have instead found the cause to be the Influenza A virus subtype H3N8 [6]. The first cases were observed in May 1889 in three separate and distant locations, Bukhara in Central Asia (Turkestan), Athabasca in north-western Canada, and Greenland. Rapid population growth of the 19th century, specifically in urban areas, only helped the flu spread, and before long the outbreak had spread across the globe. The Sixth Cholera Pandemic originated in India where it killed over 800,000, before spreading to the Middle East, North Africa, Eastern Europe and Russia. The Sixth Cholera Pandemic was also the source of the last American outbreak of Cholera (1910–1911) [7–14]. Between 1918 and 1920 a disturbingly deadly outbreak of influenza (Flu pandemic) tore across the globe, infecting over a third of the world’s population and ending the lives of 20 – 50 million people. Of the 500 million people infected in the 1918 pandemic, the mortality rate was estimated at 10% to 20%, with up to 25 million deaths in the first 25 weeks alone [13]. Asian Flu was a pandemic outbreak of Influenza A of the H2N2 subtype, that originated in China in 1956 and lasted until 1958 [13]. In its two-year spree, Asian Flu travelled from the Chinese province of Guizhou to Singapore, Hong Kong, and the United States and the death toll was approximately 2 millions. A category 2 Flu pandemic sometimes referred to as “the Hong Kong Flu,” the 1968 flu pandemic was caused by the H3N2 strain of the Influenza A virus, a genetic offshoot of the H2N2 subtype. From the first reported case on July 13, 1968 in Hong Kong, it took only 17 days before outbreaks of the virus were reported in Singapore and Vietnam, and within three months had spread to The Philippines, India, Australia, Europe, and the United States. First identified in Democratic Republic of the Congo in 1976, HIV/AIDS has truly proven itself as a global pandemic, killing more than 36 million people since 1981. Currently there are between 31 and 35 million people living with HIV, the vast majority of those are in Sub-Saharan Africa, where 5% of the population is infected, roughly 21 million people. As awareness has grown, new treatments have been developed that make HIV far more manageable, and many of those infected go on to lead productive lives. Between 2005 and 2012 the annual global deaths from HIV/AIDS dropped from 2.2 million to 1.6 million [3–14].

1.2. History of human coronavirus: structure and spreading behavior of COVID- 19

The history of human coronaviruses began in 1965 when Tyrrell and Bynoe found that they could passage a virus named B814. It was found in human embryonic tracheal organ cultures obtained from the respiratory tract of an adult with a common cold [15]. Human coronaviruses, first characterized in the 1960s, are responsible for a substantial proportion of upper respiratory tract infections in children. Since 2003, at least 5 new human coronaviruses have been identified, including the severe acute respiratory syndrome coronavirus, which caused significant morbidity and mortality. NL63, representing a group of newly identified group 1 coronaviruses that includes NL and the New Haven coronavirus, has been identified worldwide. These viruses are associated with both upper and lower respiratory tract disease and are likely common human pathogens [16]. The cause of a very new, severe acute respiratory syndrome, called SARS, emerged in 2002–2003 as a coronavirus from southern China and spread throughout the world with quantifiable speed. The SARS epidemic put the animal coronaviruses in the spotlight. In the last twenty years, several viral epidemics such as the severe acute respiratory syndrome coronavirus (SARS-CoV) in 2002 to 2003, and H1N1 influenza in 2009, have been recorded. Most recently, the Middle East respiratory syndrome coronavirus (MERS-CoV) was first identified in Saudi Arabia in 2012. SARS-CoV provoked a large-scale epidemic beginning in China and involving two dozen countries with approximately 8000 cases and 800 deaths, and the MERS-CoV that began in Saudi Arabia and has approximately 2500 cases and 800 deaths and still causes sporadic cases [17–19]. On February 11, 2020, the WHO Director-General announced that the disease caused by this new CoV was a “COVID-19,” which is the acronym of “coronavirus disease 2019” [20]. Coronavirus is the name given to group of viruses that infect mammals and birds. The name Corona is derived from latin word meaning Crown that refers to its characteristic appearance where its surface is covered in the form of club shaped protein spikes [21]. The host and reservoir of coronavirus is depicted in Fig. 1.

By 2020, coronavirus had gained popularity globally with respect to its nomenclature. This family of viruses seems to expand continuously according to Scientists and SARS-CoV-2 alternatively known as COVID-19 also belongs to this family of viruses. There is a probability that some of these viruses might have been missed by the scientists and hence there is still a lot to explore about COVID-19 and the response mechanism of the human immune system. By now 40 coronaviruses have got their names approved as per the International Committee for the Taxonomy of Viruses. The
numbers of identified coronaviruses have reached to seven including the current COVID-19. Most of these viruses seem to affect the animals. Four among these seven viruses are acquired by community and transmit through population continually for long period of time. On the other hand, SARS-CoV, MERS-CoV and SARS-CoV-2 are recent outbreaks and are associated with very high mortality rates. Shereen et al. [22] states in addition to the above three, H5N1 influenza A and H1N1 2009 cause acute lung injury (ALI) and acute respiratory distress syndrome (ARDS) all leading to pulmonary failure and ultimate fatality. All corona viruses start in animals and pass on to humans through mutation, recombination and adaptation. Shereen et al. [22] supports that the originating source of COVID-19 is bats but the intermediate source before transmission to humans is not yet identified.

Many animal coronaviruses infect animals during a particular season. The reason why these animal coronaviruses do not cause symptoms in their host reservoir is because they continuously co-evolve and adapt over a long period of time with their reservoir host. The symptoms are mild even if they are shown. Symptoms in species vary like in birds it causes upper respiratory tract infections while in cows or pigs it results in Diarrhea. In humans it causes respiratory tract infections that could lead to common cold. The high infection rate, recombination rate and mutation rate of these animal coronaviruses increase the probability of mutant’s ability to transmit into another host. It has been speculated that the severity of disease is significantly very high between the new host and the coronavirus at the new round of adaption. It is not yet proved but speculated that the new host will be able to fight the virus effectively only after a long period of adaption and co-evolution hence leading to milder symptoms. The seven human coronaviruses are transitioned into humans through other animals after originating in animals like bats and rodents. The four human coronaviruses including hCoV-OC43 and hCoV-229E cause mild common cold like symptoms. Although they cause infections across all age groups throughout the year but the rates are higher in winters and early spring. They cause multiple re-infections throughout human lifespan. These viruses are transmitted to human beings when they come in close in contact with the host. These viruses during their incubation period in human beings develop or even mutate for further transmission to other human beings. Earlier form of Coronavirus includes SARS and MERS that were lethal but were not able to Transmit in Exponential manner like COVID 19.

The structure of 2019-nCoV S in the prefusion conformation is depicted in Fig. 2. COVID 19 is associated with the fever, severe respiratory illness, and pneumonia. It is found to be related with SARS-CoV and several bat coronaviruses and is characterized as new member of the betacoronavirus genus. Shereen et al. [22] states that the subgroups of coronaviruses family include alpha (α), beta (β), gamma (γ) and delta (δ) coronavirus and COVID 19 belongs to the β group of coronaviruses. Its infection rate is significantly higher than the severe acute respiratory syndrome coronavirus (SARS-CoV) hence raising the concern of international Public Health Emergency. It enters into the host cells through a densely glycosylated spike (S) protein. This protein undergoes substantial structural change in order to form fusion between the membranes of the virus and the host cell. The protein subunit S1 binds with the host cell receptor. Prefusion trimer is destabilized by the receptor binder resulting in transition to subunit S2. The down (receptor inaccessible) conformation state is more stable than the up (receptor accessible) conformation state. S protein pays an indispensable function and the characterization the Prefusion structure provides vital information which is used in the design and development of the vaccine.
COVID-19 overall structure S resembles that of SARS-CoV S resulting in a root mean square deviation (RMSD) of 3.8 Å over 959 Cα atoms. The position of the Receptor Binding Domains (RBD) in their respective down conformations is one of the larger differences between these two structures. The SARS-CoV RBD packs tightly against the N-terminal domain of the neighboring protomer, while COVID 19 RBD is angled closer to the central cavity of the trimer. COVID 19 S and SARS-CoV S, reflect the high structural homology between the two proteins despite the difference in the alignment. Angiotensin-converting enzyme 2 (ACE2) is the same functional host cell receptor shared between COVID 19 S and SARS-CoV S. The apparent ease with which COVID 19 spreads from human to human is mainly because of its very high affinity for human ACE2. Shereen et al. [22] supports that the transmission rate of SARS-CoV 2 is higher than the SARS-CoV owing to the events of genetic recombination in the RBD domain. Shereen et al. [22] states that a clinically approved antiviruses is not yet available to fight against the COVID 19. The rapid spreading of COVID 19 indicates the urgent need for coronavirus vaccines. The information available on COVID 19 atomic-level structure will facilitate the additional protein-engineering efforts which could result in the improvement of antigenicity and protein expression for the development of vaccine. The structural data will also provide information on occurrence of the mutations when virus undergoes genetic drift; define whether residues map to known antibody epitopes sites for other coronavirus spike proteins. The structure also assures on whether the protein produced is homogeneous and in the Prefusion conformation.

The Structure of Respiratory Syndrome which causes coronavirus in Humans is depicted in Fig. 3. The designing and screening of small molecules with fusion-inhibiting potential are also facilitated by the atomic-level detail. The information is indispensable in supporting the precision design of the vaccine and discovering the antiviral measures [23]. Further, Shereen et al. [22] also describes the comparative analysis of the critical nature of the three outbreaks including COVID 19, SARS CoV and MERS CoV while emulating the approaches that are recommended in developing effective vaccines.

1.3. Symptoms

The COVID-19 virus affects different people in different ways. COVID-19 is a respiratory disease and most infected people develop mild to moderate symptoms and recover without requiring special treatment. People who have underlying medical conditions and those over 60 years old have a higher risk of developing severe disease and death. Common symptoms include fever, tiredness, and dry cough. Other symptoms include: shortness of breath, aches and pains, sore throat, and very few people report diarrhea, nausea or a runny nose [24].

1.4. Transmitting behavior of COVID-19

Pneumonia of unknown cause detected in Wuhan, China was first reported to the WHO Country Office in China on 31 December 2019 [20]. Because the first cases of the COVID-19 disease were linked to direct exposure to the Huanan Seafood Wholesale Market of Wuhan, Republic of China, the animal-to-human transmission was presumed as the main mechanism. Nevertheless, subsequent cases were not associated with this exposure mechanism. Therefore, it was concluded that the virus could also be transmitted from human-to-human, and symptomatic people are the most frequent source of COVID-19 spread. The possibility of transmission before symptoms develop seems to be infrequent, although it cannot be excluded. Moreover, there are suggestions that individuals who remain asymptomatic could transmit the virus. This data suggests that the use of isolation is the best way to contain this epidemic [25]. Based on data from the first cases in Wuhan and investigations conducted by the Chinese Centre for Disease Control and Prevention (CDC) and local CDCs, the incubation time could be generally within 3 to 7 days and up to 2 weeks as the longest time from infection to symptoms was 12.5 days (95% confidence interval, 9.2 to 18) [26]. This data also showed that this novel epidemic doubled about every seven days, whereas the basic reproduction number (R0) is 2.2. In other words, on average, each patient transmits the infection to an additional 2.2 individuals. Of note, estimations of the R0 of the SARS-CoV epidemic in 2002-2003 were approximately 3 [27]. Study suggests that people may acquire the coronavirus through the air and after touching contaminated objects. Scientists discovered the virus is detectable for up to three hours in aerosols, up to four hours on copper, up to 24 hours on cardboard and up to two to three days on plastic and stainless steel [28]. An analysis of publicly available data on infections from the new coronavirus, SARS-CoV-2, that causes the respiratory illness, COVID-19 yielded an estimate of 5.1 days for the median disease incubation period, according to a new study led by researchers at Johns Hopkins Bloomberg School of Public Health [29]. This median time from exposure to onset of symptoms suggests that the 14-day quarantine period used by the U.S. Centers for Disease Control and Prevention for individuals with likely exposure to the coronavirus is reasonable. The analysis suggests that about 97.5 percent of people who develop symptoms of SARS-CoV-
Table 1
World top 10 countries with reported laboratory-confirmed COVID-19 cases and deaths; data as of April 05, 2020 [30].

| Country                  | Total confirmed cases | Total confirmed new cases | Total deaths | Total new deaths |
|--------------------------|-----------------------|---------------------------|--------------|------------------|
| United States of America | 273,808               | 32,105                    | 7020         | 1166             |
| Spain                    | 124,736               | 7026                      | 11,744       | 809              |
| Italy                    | 124,612               | 4805                      | 13,362       | 681              |
| Germany                  | 91,714                | 5936                      | 1342         | 184              |
| China                    | 82,930                | 55                        | 3338         | 3                |
| France                   | 67,757                | 4221                      | 7546         | 1053             |
| Iran                     | 55,743                | 2560                      | 3452         | 158              |
| The United Kingdom       | 41,907                | 3735                      | 4313         | 708              |
| Turkey                   | 23,934                | 3013                      | 501          | 76               |
| Switzerland              | 20,489                | 783                       | 666          | 59               |

Table 2
Nomenclature for the models of stage 1, 2, and 3.

| Symbol | Description                                                                 |
|--------|-----------------------------------------------------------------------------|
| S      | Susceptible population                                                      |
| E      | Immigrant population                                                       |
| I      | Infectious population                                                      |
| Q1     | Home isolation population                                                  |
| Q2     | Quarantine population in hospital                                           |
| R      | Recovered population                                                       |
| A      | The influx rate that expresses the arrival of people to the immigrant population |
| β      | Per infectious contact rate                                                |
| μ1     | Natural death rate, i.e. death other than COVID-19                          |
| μ2     | Death rate due to COVID-19                                                  |
| α1     | Rate of transfer of susceptible population to home isolation population     |
| α2     | Rate of transfer of immigrant population to home isolation population       |
| δ      | Rate of transfer of home isolation population to infectious population      |
| ε      | Rate of transfer of home isolation population to susceptible population     |
| η      | Rate of transfer of infectious population to quarantine population in hospital |
| γ      | Rate of recovery from hospital quarantine population                        |

2 infection will do so within 11.5 days of exposure. Lauer et al. estimated that for every 10,000 individuals quarantined for 14 days, only about 101 would develop symptoms after being released from quarantine [29].

1.5. Stages of COVID-19

Stage-1: Imported cases where those who travelled to other countries have contracted the infection.
Stage-2: Local transmission in which people coming in immediate contact of an infected person report infection.
Stage-3: ‘Community transmission’ when a person who has no travel history has contracted through domestic sources.
Stage-4: When a disease is declared an epidemic.

Based on the report of WHO, the transmission of COVID-19 in the top 10 countries across the globe is mentioned in Table 1 [30].

The subsequent materials of this paper are structured as follows: Section 2 deals with basic terminologies and basic reproduction number of severe pandemic diseases. Three epidemic models of three different stages of COVID-19 and its stability are discussed in Section 3. Section 4 discusses on simulations and its analysis with examples. Finally the paper is concluded in Section 5 followed by references.

Nomenclature used in three epidemic models of the pandemic disease COVID-19 is given in Table 2.

2. Some basic terminologies

Susceptible (S(t)): In epidemiology a susceptible individual (sometimes known simply as a susceptible) is a member of a population who is at risk of becoming infected by a disease.

Immigrant population (E(t)): It is the international movement of people to a destination country of which they are not natives or where they do not possess citizenship in order to settle or reside there, especially as permanent residents or naturalized citizens, or to take up employment as a migrant worker or temporarily as a foreign worker.

Infectious population (I(t)): It refers to the population who are infected by a contagious disease (COVID-19 in our case), may be through immigrant population transmission or by local individual transmission or community transmission.

Home Isolation (Q1(t)): It refers to the restriction of movement or separation of well persons who are susceptible or possibly exposed to a contagious disease (COVID-19 in our case), before it is known whether they will become ill. Isolation usually takes place in the home and may be applied at the individual level or to a group or community of susceptible or exposed population.

Quarantine (Q2(t)): It refers to the separation and restricted movement of ill persons who have a contagious disease (COVID-19 in our case) in order to prevent its transmission to others. It typically occurs in a hospital setting, but under very special cases may be done at home under a special facility. Usually individuals are quarantined, but the practice may be applied in larger groups.

Recovered population (R(t)): It refers to the population who are recovered from COVID-19.

Basic Reproduction number (R0): The basic reproduction number (denoted by R0 [31]) of an infection can be thought of as the expected number of cases directly generated by one case in a population where all individuals are susceptible to infection [32]. Basic Reproduction number of some highly infectious disease is given in Table 3.
3. Epidemic models

In this section we develop three epidemic models on three different stages of infection of COVID-19.

3.1. Hypotheses

In this paper, our mathematical models for stage 1, 2 and 3 are based on the following hypothesis:

(H1) We develop the dynamics of a pandemic model with vital dynamics having unequal birth ($B > 0$) and natural death rates ($\mu_1 > 0$).

(H2) The arrival of people to the immigrant population is at a constant influx rate $A > 0$.

(H3) Immigrant population becomes susceptible at constant rate $\eta > 0$.

(H4) Immigrant population are subjected to home isolation at a constant rate $\varepsilon > 0$.

(H5) Home isolation population becomes susceptible population at time $\frac{\eta}{2} > 0$.

(H6) Home isolation population becomes infectious population at constant rate $\frac{\eta}{2} > 0$.

(H7) Each susceptible individual is infected by an infectious individual at constant rate $\beta > 0$.

(H8) Susceptible population are subjected to home isolation at constant rate $\lambda > 0$.

(H9) Infectious population are subjected to hospital quarantine at a constant rate $\delta > 0$.

(H10) Hospital quarantine population gets recovered at a constant rate $\gamma > 0$.

(H11) Infectious population or hospital quarantine population die due to COVID-19 at constant rate $\mu_2 > 0$.

Most of these hypotheses are common for all the three mathematical models.

3.1.1. Stage 1: Imported cases where those who travelled to other countries have contracted the infection

Here, a mathematical model is developed to express the first stage of COVID-19. This model has six compartments: susceptible population - immigrant population - home isolation population - infectious population - hospital quarantine population - recovered population. Any country or state is in stage 1, if person(s) are found COVID-19 positive that has recent international travel history. This model helps us to understand how this foreign return population spreads COVID-19 to other citizens of that country, if not handled properly. In the absence of vaccination and proper treatment, two force-control policies namely home isolation and hospital quarantine, are adopted by many countries to control this COVID-19 epidemic. The same is framed and analysed here with the help of our model as depicted in Fig. 4.

3.1.2. Model formulation

Based on our hypothesis and flow of transmission of the disease COVID-19 in human population as depicted in Fig. 4, we have the following system of equations:

$$\frac{dN}{dt} = B - \beta SI - \mu_1 S + \varepsilon Q_1 + \alpha_1 E$$

$$\frac{dE}{dt} = A - \alpha_1 E - \alpha_2 E - (1 - \alpha_1 - \alpha_2)E - \mu_1 E$$

$$\frac{dI}{dt} = \beta SI + \delta Q_1 - (\mu_1 + \mu_2 + \varepsilon)I$$

$$\frac{dQ_1}{dt} = \alpha_2 E - (\mu_1 + \delta + \varepsilon)Q_1$$

$$\frac{dQ_2}{dt} = \eta I + (1 - \alpha_1 - \alpha_2)E - (\mu_1 + \mu_2 + \gamma)Q_2$$

$$\frac{dR}{dt} = \gamma Q_2 - \mu_1 R$$

where, the total population $N = S + E + I + Q_1 + Q_2 + R$.

Then,

$$\frac{dN}{dt} = B + A - \mu_1 N$$

This shows that population size $N$ tends to carrying capacity $\frac{B + A}{\mu_1}$ as $t \to \infty$.

It shows that the solution of (1) exists in the region defined by

$$\Omega = \left\{ (S, E, I, Q_1, Q_2, R) \in R_+^6 : S + E + I + Q_1 + Q_2 + R \leq \frac{B + A}{\mu_1} \right\}$$

where, $S > 0, E \geq 0, I \geq 0, Q_1 \geq 0, Q_2 \geq 0, R \geq 0$. 

---

**Table 3**

Values of $R_0$ of well-known infectious diseases [33].

| S. No. | Disease                  | Transmission  | $R_0$     |
|--------|--------------------------|---------------|-----------|
| 1      | Measles                  | Airborne      | 12–18 [34]|
| 2      | Chickenpox (varicella)   | Airborne      | 10–12 [35]|
| 3      | Rubella                  | Airborne droplet | 5–7      |
| 4      | Mumps                    | Airborne droplet | 4–7      |
| 5      | Pertussis                | Airborne droplet | 5.5[36] |
| 6      | Smallpox                 | Airborne droplet | 3.5–6[37]|
| 7      | HIV/AIDS                 | Body fluids   | 2–5      |
| 8      | SARS                     | Airborne droplet | 2–5[38] |
| 9      | Diphtheria               | Saliva        | 1.7–4.3[39]|
| 10     | COVID-19                 | Airborne droplet | 1.4–3.9[40–44] |
| 11     | Influenza (1918 pandemic strain) |            | 1.4–2.8 [45] |
| 12     | Ebola (2014 Ebola outbreak) | Body fluids   | 1.5–2.5 [46] |
| 13     | Influenza (2009 pandemic strain) | Airborne droplet | 1.4–1.6 [47] |
| 14     | Influenza (seasonal strains) | Airborne droplet | 0.9–2.1 [47] |
| 15     | MERS                     | Airborne droplet | 0.3–0.8 [48] |
Since all the solution remains bounded in the positively invariant region \( \Omega \) in the maximal interval \([0, \infty)\). Thus the initial value problem is well posed.

3.1.3. Basic reproduction number

For the sake of simplicity, and also satisfying the necessary constraints, we take the five classes for calculation of basic reproduction number.

\[
\frac{d\xi}{dt} = A - \alpha_1 \xi - \alpha_2 \xi - (1 - \alpha_1 - \alpha_2) E - \mu_1 \xi \\
\frac{d\eta}{dt} = \beta SI + \delta Q_1 - (\mu_1 + \mu_2 + \eta) I \\
\frac{dQ_1}{dt} = \alpha_2 \xi - (\mu_1 + \delta + \epsilon) Q_1 \\
\frac{dQ_2}{dt} = \eta I + (1 - \alpha_1 - \alpha_2) E - (\mu_1 + \mu_2 + \gamma) Q_2 \\
\frac{dR}{dt} = \gamma Q_2 - \mu_1 R
\]

On linearizing the model, we have,

\[
\begin{pmatrix}
\frac{dE}{dt} \\
\frac{d\xi}{dt} \\
\frac{dQ_1}{dt} \\
\frac{dQ_2}{dt} \\
\frac{dR}{dt}
\end{pmatrix} = (F - V) \begin{pmatrix} E \\ I \\ Q_1 \\ Q_2 \\ R \end{pmatrix}
\]

where \( F \) is the rate of infection and \( V \) is the rate of infection by compartment to compartment.

Here

\[
F = \begin{pmatrix}
0 & \beta & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0
\end{pmatrix}, \quad V = \begin{pmatrix}
1 + \mu_1 & \eta + \mu_1 + \mu_2 & 0 & 0 & 0 \\
0 & \mu_1 + \delta + \epsilon & 0 & 0 & 0 \\
-\alpha_2 & 0 & \mu_1 + \delta + \epsilon & 0 & 0 \\
\alpha_1 + \alpha_2 - 1 & -\eta & 0 & \gamma + \mu_1 + \mu_2 & 0 \\
0 & 0 & 0 & 0 & -\gamma \mu_1
\end{pmatrix}
\]

On calculation, the spectral radius of \( \rho(FV^{-1}) = \frac{\beta \delta \alpha_2}{(\mu_1 + \mu_2 + \eta)(\mu_1 + \delta + \epsilon)} \)

Thus the basic reproduction number is obtained by \( R_0 = \frac{(\mu_1 + \mu_2 + \eta)(\mu_1 + \delta + \epsilon)}{(\mu_1 + \mu_2 + \eta)(\mu_1 + \delta + \epsilon)} \)

3.1.4. Equilibrium points

For the equilibrium points in the steady state of the system (1)

\[
B - \beta SI - \mu_1 S + \epsilon Q_1 + \alpha_1 E = 0 \\
A - \alpha_1 E - \alpha_2 E - (1 - \alpha_1 - \alpha_2) E - \mu_1 S = 0 \\
\beta SI + \delta Q_1 - (\mu_1 + \mu_2 + \eta) I = 0 \\
\alpha_2 E - (\mu_1 + \delta + \epsilon) Q_1 = 0 \\
\eta I + (1 - \alpha_1 - \alpha_2) E - (\mu_1 + \mu_2 + \gamma) Q_2 = 0 \\
\gamma Q_2 - \mu_1 R = 0
\]

(2)

3.1.5. Stability for disease free equilibrium

In this section we discuss the local stability of endemic equilibrium and disease free equilibrium of the system (1) (after dropping the last equation of (1)) & by analyzing the corresponding characteristic equations, respectively.

Theorem-1: If \( R_0 < 1 \), the disease free equilibrium of the system (1) is locally asymptotically stable in the region \( \Omega \). If \( R_0 > 1 \), it is unstable in the given region \( \Omega \).

Proof: The characteristic matrix at the diseases free equilibrium is

\[
\Omega_0(1, 0, 0, 0, 0) = J_{DFE} = \begin{pmatrix}
-\mu_1 & \alpha_1 & -\beta & \epsilon & 0 \\
0 & -(1 + \mu_1) & 0 & 0 & 0 \\
-\alpha_2 & 0 & -\delta & 0 & 0 \\
0 & (1 - \alpha_1 - \alpha_2) & \eta & -(\mu_1 + \delta + \epsilon) & 0 \\
0 & 0 & 0 & -(\mu_1 + \mu_2 + \gamma) & 0
\end{pmatrix}
\]

\( \lambda_1 = -\mu_1 \\
\lambda_2 = -(1 + \mu_1) \\
\lambda_3 = -(\mu_1 + \delta + \epsilon) \\
\lambda_4 = -(\mu_1 + \mu_2 + \gamma) \)
Clearly, the system (2) has four negative real roots and the root \( \lambda_5 = \beta - (\mu_1 + \mu_2 + \eta) < 0 \). If \( \beta < (\mu_1 + \mu_2 + \eta) \) clearly all roots are negative real roots. So system (1) is disease free equilibrium and is locally asymptotically stable too.

3.1.6. Endemic equilibrium
The endemic equilibrium \( \Omega^*(S^*, E^*, I^*, Q_1^*, Q_2^*, R^*) \), which is interior of \( \Omega \), can be obtained by taking all the equations of the system (2) equal to zero. Thus,
\[
\begin{align*}
S^* &= \frac{B(1 + \mu_1)(\mu_1 + \delta + \varepsilon) + \alpha_2 \mu_1 \alpha_1 \varepsilon A}{(\beta \mu_1)(\mu_1 + \delta + \varepsilon) + \alpha_1 \mu_1 A(\mu_1 + \delta + \varepsilon)} \\
E^* &= \frac{\alpha_2 \mu_1 \alpha_1 \varepsilon A}{(\beta \mu_1)(\mu_1 + \delta + \varepsilon)} \\
I^* &= \frac{\alpha_2 \mu_1 \alpha_1 \varepsilon A}{(\beta \mu_1)(\mu_1 + \delta + \varepsilon)} \\
Q_1^* &= \frac{\eta I^* (1 + \mu_1)}{(1 + \mu_1)(1 + \mu_1 + \delta + \eta)} \\
Q_2^* &= \frac{\eta I^* (1 + \mu_1)}{(1 + \mu_1)(1 + \mu_1 + \delta + \eta)} \\
R^* &= \frac{\gamma I^* (1 + \mu_1)}{(1 + \mu_1)(1 + \mu_1 + \delta + \eta)}
\end{align*}
\]

Theorem-2. : The endemic equilibrium \( \Omega^*(S^*, E^*, I^*, Q_1^*, Q_2^*, R^*) \) is locally asymptotically stable when \( R_0 > 1 \)
Proof: On linearizing the system (2), we have,
\[
J_{EE} = \begin{bmatrix}
-\beta I^* - \mu_1 & \alpha_1 & -\beta S^* & \epsilon & 0 & 0 \\
0 & -\beta I^* - \mu_1 & 0 & 0 & 0 & 0 \\
0 & 0 & \beta S^* - (\mu_1 + \mu_2 + \eta) & 0 & 0 & 0 \\
0 & 0 & \alpha_2 & 0 & -\mu_1 - \delta & 0 \\
0 & 0 & \alpha_2 & 0 & 0 & -\mu_1 - \delta \\
0 & 0 & 0 & 0 & 0 & \gamma
\end{bmatrix}
\]

Here the four eigen values are
\[
\begin{align*}
\lambda_1 &= -\mu_1 \\
\lambda_2 &= -(\mu_1 + \mu_2 + \gamma) \\
\lambda_3 &= -(\mu_1 + \delta + \varepsilon) \\
\lambda_4 &= -(\mu_1 + \mu_2 + \gamma)
\end{align*}
\]
which have strictly negative real parts and other two eigen values are given by quadratic equations
\[
\lambda^2 + a\lambda + b = 0
\]
where
\[
\begin{align*}
a &= \beta I^* + 2\mu_1 + \mu_2 + \eta - \beta S^* > 0 \\
b &= \beta I^* (\mu_1 + \mu_2 + \eta) + \mu_1^2 + \mu_1 \mu_2 + \mu_1 \eta - \mu_1 \beta S^* > 0 \\
a.b &> 0
\end{align*}
\]
Since \( a.b > 0 \), so by Routh-Hurwitz criteria system (1) is stable.
Since all the eigen values have negative real parts so the system (1) is locally asymptotically stable at the endemic equilibrium \( \Omega^* \), if \( R_0 > 1 \).

3.1.7. Global stability for endemic equilibrium
In this section, we prove the global stability for endemic equilibrium. We adopt the geometrical approach for the mapping \( f: D \subset \mathbb{R}^6 \rightarrow \mathbb{R}^6 \), where \( D \) is an open set, if its differential equations \( x' = f(x) \) be such that its every solution \( x(t) \) can be uniquely determined by its initial condition \( x(0) = x_0 \), then an equilibrium points \( \bar{x} \in D \) and satisfies the conditions
\[
\begin{align*}
D \text{ is simply connected} \\
\text{There exists a compact absorbing sub set K of D} \\
\text{x the only equilibrium point in D is globally stable, if it satisfies the additional Bendixson condition given by} \\
\text{q = lim sup}_{t \rightarrow \infty} \sup_{x \in K} \frac{1}{t} \int_0^t f(M(x(s), x_0)))ds \\
\text{where q =} \frac{t}{0} \int f(M(x(s), x_0)))ds \\
\text{Also M = P_j P^{j-1} + P \delta P^{-j} P^{-1} and P is a matrix valued function satisfying} \\
\text{satisfying} \psi(P_j P^{j-1} + P \delta P^{-j} P^{-1}) < 0 \text{on K. Further} P^{j} \text{is the second compound additive matrix of order four. Again} \psi \text{denote the Lozinskii measure defined as} \\
\text{lim inf} S(t) \geq C, \text{lim inf}\ E(t) > C, \text{lim inf}\ I(t) > C, \text{lim inf}\ Q_1(t) > C, \text{lim inf}\ Q_2(t) > C, \text{lim inf}\ R(t) > C \text{for some C} > 0. \text{Based on this procedure used by system (1) and then by (2), is used to prove for Bendixson condition} \text{q < 0}. \\
\end{align*}
\]

Theorem-3. : If \( R_0 > 1 \), then \( \Omega \) is globally asymptotically stable for the system (1)
Proof: The Jacobian matrix of the reduced system (1), leaving E and R compartments we have

\[
J = \begin{pmatrix}
\beta I - \mu_1 & -\beta S & \varepsilon \\
\beta I - (\mu_1 + \mu_2 + \eta) & \delta & 0 \\
0 & 0 & -\mu_1 - \mu_2 + \gamma \\
\eta & 0 & 0 \\
0 & \eta & 0 \\
0 & 0 & \eta \\
\end{pmatrix}
\]

The second compound additive Jacobian matrix is given by

\[
J^{[2]} = \begin{pmatrix}
\beta S - \beta I - (2\mu_1 + \mu_2 + \eta) & \delta & 0 & -\varepsilon & 0 & 0 \\
0 & -\beta I + \delta + \varepsilon & 0 & 0 & -\beta S & 0 \\
\eta & 0 & -\beta (2\mu_1 + \mu_2 + \gamma) & 0 & -\beta S & \varepsilon \\
0 & \beta I & 0 & \beta S - (2\mu_1 + \mu_2 + \gamma + \eta) & 0 & 0 \\
0 & 0 & \beta I & 0 & -\eta & 0 \\
0 & 0 & 0 & 0 & -\eta & 0 \\
\end{pmatrix}
\]

To obtain matrix M in the Bendixson condition, we define a matrix

\[
P = \text{diag} \left\{ \frac{1}{Q_1}, \frac{1}{Q_2}, \frac{1}{Q_3}, \frac{1}{Q_4}, \frac{1}{Q_5} \right\}
\]

then

\[
P J^{[2]} P^{-1} = \begin{pmatrix}
M_{11} & M_{12} \\
M_{21} & M_{22} \\
\end{pmatrix}
\]

where,

\[
M_{11} = \beta S - \beta I - (2\mu_1 + \mu_2 + \eta) \\
M_{12} = \left\{ \begin{array}{cc}
\delta \frac{Q_1}{Q_2} & 0 \\
0 & (\mu_1 + \delta + \varepsilon) \frac{Q_1}{Q_2} \\
\end{array} \right. \\
M_{21} = \left\{ \begin{array}{cc}
\eta \frac{Q_1}{Q_2} & 0 \\
0 & 0 \\
\end{array} \right. \\
M_{22} = \left\{ \begin{array}{cc}
0 & -\beta S \\
0 & 0 \\
\beta I & 0 \\
0 & \beta S - (2\mu_1 + \mu_2 + \gamma + \eta) + \frac{\varepsilon}{T} - \frac{Q_1}{Q_2} \\
0 & \beta I \\
0 & 0 \\
\eta & 0 \\
0 & 0 \\
\end{array} \right.
\]

The Lozinskii measure of the matrix M can be estimated as \( \psi(M) \leq \sup\{g_1, g_2\} \), where \( g_1 \) and \( g_2 \) is defined as:

\[
g_1 = \psi(M_{11}) + |M_{12}| = \beta S - \beta I - (2\mu_1 + \mu_2 + \eta) + (\mu_1 + \delta + \varepsilon) \frac{Q_1}{Q_2} \\
g_2 = M_{21} + \psi(M_{22}) = \eta \frac{Q_1}{Q_2} + \beta S - (2\mu_1 + 2\mu_2 + \gamma + \eta) + \frac{\varepsilon}{T} - \frac{Q_1}{Q_2}
\]

Hence Lozinskii measure reduces to \( \psi(M) \leq \sup\{g_1, g_2\} \leq \frac{\varepsilon}{T} - \gamma \)

So, \( \int_0^t \psi(M) \, dt \leq \log(t) - \gamma t \)

Hence, \( \int_0^t \psi(M) \, dt \leq \frac{\varepsilon}{T} - \gamma t \)

3.2 Stage-2: Local transmission in which people coming in immediate contact of an infected person report infection

Here, a mathematical model is developed to express the second stage of COVID-19, which is now more difficult to control in comparison to stage 1. This model also has the same six compartments: susceptible population - immigrant population - home isolation population - infectious population - hospital quarantine population - recovered population. Any country or state is in stage 2, if only those found COVID-19 positive that has no international travel history but came in direct contact of those person(s) who has recently returned from countries that are affected by COVID-19. This model helps us to understand how this infected population spreads COVID-19 to other citizens of that country, if not handled properly. In the absence of vaccination and proper treatment, two force-control policies namely home isolation and hospital quarantine, are adopted by many countries to control this COVID-19 epidemic. The same is framed and analysed here with the help of this model as shown in Fig. 5.
3.2.1. Model formulation

Based on our assumptions and flow of transmission of COVID-19 of stage 2 in human population as depicted in Fig. 5, we have the following system of equations:

\[
\begin{align*}
\frac{dS}{dt} &= B - \beta IS - (\lambda + \mu_1)S \\
\frac{dE}{dt} &= A - \alpha_2 E - (1 - \alpha_2)E - \mu_1E \\
\frac{dI}{dt} &= \beta IS + \delta Q_1 - (\mu_1 + \mu_2 + \eta)I \\
\frac{dQ_1}{dt} &= \lambda S + \alpha_2 E - (\mu_1 + \delta)Q_1 \\
\frac{dQ_2}{dt} &= \eta I + (1 - \alpha_2)E - (\mu_1 + \mu_2 + \gamma)Q_2 \\
\frac{dR}{dt} &= \gamma Q_2 - \mu_1R
\end{align*}
\]

(3)

Since all the populations are positive so adding all the total population \(N = S + E + I + Q_1 + Q_2 + R\)

\[
\frac{dN}{dt} = \frac{dS}{dt} + \frac{dE}{dt} + \frac{dI}{dt} + \frac{dQ_1}{dt} + \frac{dQ_2}{dt} + \frac{dR}{dt}
\]

\[
\Rightarrow \frac{dN}{dt} = B + A - \mu_1N - \mu_2(I + Q_2)
\]

If the diseases are absent, then \(\frac{dN}{dt} = B + A - \mu_1N\).

This shows that population size \(N\) tends to carrying capacity \(\frac{B + A}{\mu_1}\) as \(t \to \infty\).

It shows that the solution of (1) exists in the region defined by

\[
\Omega = \left\{ (S, E, I, Q_1, Q_2, R) \in R^6_0 : S + E + I + Q_1 + Q_2 + R \leq \frac{B + A}{\mu_1} \right\}
\]

where, \(S > 0, E \geq 0, I \geq 0, Q_1 \geq 0, Q_2 \geq 0, R \geq 0\).

Since all the solution remains bounded in the positively invariant region \(\Omega\), and in the maximal interval [0,\(\infty\)), the initial value problem is well posed.

Fig. 5. Stage-2: Local transmission in which people coming in immediate contact of an infected person report infection.
3.2.2. Basic reproduction number

Here we consider only five classes for calculation of basic reproduction number for our sake of simplicity and also satisfying the constraints.

\[
\begin{align*}
\frac{dE}{dt} &= A - \alpha_2 E - (1 - \alpha_2) E - \mu_1 E \\
\frac{dI}{dt} &= \beta SI + \delta Q_1 - (\mu_1 + \mu_2 + \eta) I \\
\frac{dQ_1}{dt} &= \lambda S + \alpha_2 E - (\mu_1 + \delta) Q_1 \\
\frac{dQ_2}{dt} &= \eta I + (1 - \alpha_2) E - (\mu_1 + \mu_2 + \gamma) Q_2 \\
\frac{dR}{dt} &= \gamma Q_2 - \mu_1 R
\end{align*}
\]

On linearizing the model, we have,

\[
\begin{pmatrix}
\frac{dE}{dt} \\
\frac{dI}{dt} \\
\frac{dQ_1}{dt} \\
\frac{dQ_2}{dt} \\
\frac{dR}{dt}
\end{pmatrix} = (F - V)
\begin{pmatrix}
E \\
I \\
Q_1 \\
Q_2 \\
R
\end{pmatrix}
\]

where \(F\) is the rate of infection and \(V\) is the rate of infection from compartment to compartment.

\[
F = \begin{pmatrix}
0 & \beta & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0
\end{pmatrix}, \quad V = \begin{pmatrix}
1 + \mu_1 & 0 & 0 & 0 & 0 \\
0 & \eta + \mu_1 + \mu_2 & -\delta & 0 & 0 \\
-\alpha_2 & 0 & \mu_1 + \delta & 0 & 0 \\
\alpha_2 - 1 & -\eta & 0 & \gamma + \mu_1 + \mu_2 & 0 \\
0 & 0 & 0 & -\gamma & \mu_1
\end{pmatrix}
\]

By calculating the spectral radius of \(R_0 = \rho(FV^{-1}) = \frac{\beta \delta \alpha_2}{(\mu_1 + \mu_2 + \eta)(1 + \mu_1)(\mu_1 + \delta)}\cdot\frac{\beta \delta \alpha_2}{(\mu_1 + \mu_2 + \eta)(1 + \mu_1)(\mu_1 + \delta)}\)

Thus the basic reproduction number is given by \(R_0 = \rho(FV^{-1}) = \frac{\beta \delta \alpha_2}{(\mu_1 + \mu_2 + \eta)(1 + \mu_1)(\mu_1 + \delta)}\).

3.2.3. Equilibrium points

For equilibrium points in the steady state of the system (3), we have,

\[
\begin{align*}
A - \alpha_2 E - (1 - \alpha_2) E - \mu_1 E &= 0 \\
\beta SI + \delta Q_1 - (\mu_1 + \mu_2 + \eta) I &= 0 \\
\lambda S + \alpha_2 E - (\mu_1 + \delta) Q_1 &= 0 \\
\eta I + (1 - \alpha_2) E - (\mu_1 + \mu_2 + \gamma) Q_2 &= 0 \\
\gamma Q_2 - \mu_1 R &= 0
\end{align*}
\]

(4)

3.2.4. Stability for disease free equilibrium

In this section, we discuss the local stability of endemic equilibrium and disease free equilibrium of the system (3) (after dropping the last equation of (3)), and by analyzing the corresponding characteristic equations.

**Theorem-4** : If \(R_0 < 1\), the disease free equilibrium of the system (3) is locally asymptotically stable in the region \(\Omega\). If \(R_0 > 1\), it is unstable in the given region \(\Omega\).

**Proof:** The characteristic matrix at the disease free equilibrium is

\[
\Omega_0(1,0,0,0,0) = \begin{pmatrix}
-\lambda - \mu_1 & \alpha_1 & -\beta & 0 & 0 \\
0 & -1(1 + \mu_1) & 0 & 0 & 0 \\
0 & 0 & \beta - (\mu_1 + \mu_2 + \eta) & \delta & 0 \\
\lambda & \alpha_2 & 0 & -(\mu_1 + \delta) & 0 \\
0 & (1 - \alpha_2) & \eta & 0 & -(\mu_1 + \mu_2 + \gamma)
\end{pmatrix}
\]

\[
z_1 = -(1 + \mu_1) \\
z_2 = -(\mu_1 + \mu_2 + \gamma)
\]
Clearly, the system (3) has two negative real roots or Eigen values and other three roots or Eigen values can be solving the cubic equations
\[ Z^3 + A_1Z^2 + A_2Z + A_3 = 0, \]
where
\[ A_1 = 3\mu_1 + \mu_2 + \eta + \lambda + \delta - \beta \]
\[ A_2 = 3\mu_1^2 + 2\lambda\mu_1 + \lambda\mu_2 + \lambda\eta + \lambda\delta + 2\mu_1\mu_2 + 2\mu_1\delta + 2\mu_1\eta + \eta\delta + \mu_2\delta - \beta(\lambda + \delta + 2\mu_1) \]
\[ A_3 = \mu_1^3 + \mu_1\lambda\delta + \mu_2\lambda\delta + \eta\lambda\delta + \lambda\mu_1\mu_2 + \lambda\mu_1\eta + \mu_1^2\mu_2 + \mu_2\lambda\delta + \delta\mu_1\mu_2 + \mu_1^2\mu_2 + \mu_1^2\eta - 2\beta\lambda\delta - \beta\lambda\mu_1 - \beta\delta\mu_1 - \beta\mu_1^2 \]
\[ \therefore A_1A_2 - A_3 > 0 \]
\[ \Rightarrow A_1A_2 > A_3 \]
So by Routh–Hurwitz criteria, system (3) is in disease free equilibrium state and is locally asymptotically stable.

3.2.5. Endemic equilibrium
The endemic equilibrium \( \Omega^*(S^*, E^*, I^*, Q_1^*, Q_2^*, R^*) \), which is in interior of \( \Omega \), is obtained by taking all the equations of the system (3) equal to zero and simultaneously solving it. We have,
\[ S^* = \frac{B}{(B\mu + \lambda + \mu_1)} \]
\[ E^* = \frac{A}{(1 + \mu_1)} \]
\[ I^* = \frac{\alpha_2\delta A(\beta\mu + \lambda + \mu_1) + \delta A(1 + \mu_1)}{(B\mu + \mu_1 + \lambda)(\mu_1 + \mu_2 + \eta) - \beta B} \]
\[ Q_1^* = \frac{\alpha_2 A(\beta\mu + \mu_1 + \lambda) + \lambda B(1 + \mu_1)}{(B\mu + \mu_1 + \lambda)(1 + \mu_1)(\mu_1 + \delta)} \]
\[ Q_2^* = \frac{\eta(1 + \mu_1) + (1 - \alpha_2)A}{(1 + \mu_1)(\mu_1 + \mu_2 + \gamma)} \]
\[ R^* = \frac{\gamma\eta(1 + \mu_1) + (1 - \alpha_2)A}{(1 + \mu_1)(\mu_1 + \mu_2 + \gamma)} \]

**Theorem 5.** The endemic equilibrium \( \Omega^*(S^*, E^*, I^*, Q_1^*, Q_2^*, R^*) \) is locally asymptotically stable when \( R_0 > 1 \).

**Proof:** On linearizing the system (3), we have,
\[ J_{EE} = \begin{pmatrix} -\beta I^* - \lambda - \mu_1 & 0 & -\beta S^* & 0 & 0 & 0 \\ 0 & -(1 + \mu_1) & 0 & 0 & 0 & 0 \\ \beta I^* & 0 & -\beta S^* - (\mu_1 + \mu_2 + \eta) \delta & 0 & 0 & 0 \\ \lambda & \alpha_2 & 0 & -(\mu_1 + \delta) & 0 & 0 \\ 0 & (1 - \alpha_2) & \eta & 0 & -(\mu_1 + \mu_2 + \gamma) & 0 \\ 0 & 0 & 0 & 0 & \gamma & -\mu_1 \end{pmatrix} \]

The eigen values are
\[ z_1 = -\mu_1 \]
\[ z_2 = -(\mu_1 + \mu_2 + \gamma), \]
\[ z_3 = -(1 + \mu_1) \]
And other three can be obtained by solving the cubic equations; we have,
\[ Z^3 + B_1Z^2 + B_2Z + B_3 = 0 \]
where
\[ B_1 = 3\mu_1 + \mu_2 + \eta + \lambda + 1 + \beta I^* - \beta S^* \]
\[ B_2 = 3\mu_1^2 + 2\mu_1\mu_2 + 2\mu_1 + 2\mu_1\eta + \mu_2 + \eta + 2\mu_1\beta I^* + \beta I^* + \beta I^* \mu_2 + \beta I^* \eta - (2\beta S^* \mu_1 + 2\beta^2 S^* I^* + \beta S^*) \]
\[ B_3 = \beta I^* + \mu_1 \delta + \beta I^* \mu_2 \delta + \beta I^* \delta + \beta I^* \mu_1 \mu_2 + \beta I^* \mu_1 \eta + \beta I^* \mu_1^2 + \mu_1^2 \delta + \mu_1 \eta \delta + \mu_1^2 \lambda \]
\[ + \mu_1^2 \mu_2 \mu_2 + \mu_1^2 \eta - (\delta \lambda \beta S^* + \mu_1^2 \beta S^* + \beta S^* \delta \mu_1) \]
\[ \therefore B_1B_2 > B_3 \]

Hence by Routh–Hurwitz criteria system is stable.
Thus all the eigen values have negative real parts and hence the system (3) is locally asymptotically stable at the endemic equilibrium \( \Omega^* \) if \( R_0 > 1 \).

3.2.6. Global stability for endemic equilibrium
In order to prove the global stability of the region \( \Omega \) we take \( S \) and \( I \) compartment, leave \( Q_1 \) population and make it free from the \( R \) population. Therefore, in the positive quadrant of the \( S-I \) plane we apply Dulac’s criteria with multiplier \( D = 1/l \)

Consider,
\[ F_1 = B - \beta S I - \lambda S - \mu_1 S \]
\[ F_2 = [\beta S - (\mu_1 + \mu_2 + \eta)]I \]
Then
\[ DF_1 = \frac{d}{dS} - \beta S - \frac{(\mu_1 + \mu_2 + \eta)}{S} \]
\[ DF_2 = [\beta S - (\mu_1 + \mu_2 + \eta)] \]
We have,

\[
\frac{\partial (DF_1)}{\partial S} + \frac{\partial (DF_2)}{\partial I} = \frac{\partial}{\partial S} \left( \frac{B}{I} - \beta S - \frac{(\lambda + \mu_1)S}{I} \right) + \frac{\partial}{\partial I} \left[ \beta S - (\mu_1 + \mu_2 + \eta) \right]
\]

\[
= \frac{\partial (DF_1)}{\partial S} + \frac{\partial (DF_2)}{\partial I} = -\beta - \frac{(\lambda + \mu_1)}{I} < 0
\]

Thus no periodic solution exists in the region \( \Omega \). Hence by Poincare-Bendixson property, all solution starting in positive quadrant of SI-plane with \( I=0 \) and \( S+I \leq \frac{A_1}{\mu_1} \) approaches \((S^*, I^*)\) as \( t \to \infty \). In this case, the limiting form of rest of the system of equations (3) shows that \( E \to E^* \), \( Q_1 \to Q_1^* \), \( Q_2 \to Q_2^* \), \( R \to R^* \). Thus the endemic equilibrium \( \Omega^*(S^*, E^*, I^*, Q_1^*, Q_2^*, R^*) \) is globally stable in the given region \( \Omega \) for the system (3).

3.3. Stage-3: Community transmission, when a person who has no travel history has contracted COVID-19 through domestic sources

Here, a mathematical model is developed to express the third stage of COVID-19, an extremely difficult situation to tackle. If fact it is the last stage where it can be controlled with great efforts, otherwise in next or last stage it converts into a completely uncontrolled epidemic disaster. Stage 3 model has five compartments: susceptible population - home isolation population - infectious population - hospital quarantine population - recovered population. In stage 3, immigrant population is no more present since international travel restrictions have been imposed during complete lockdown. Any country or state is in stage 3, when person(s) found COVID-19 positive that has neither recent international travel history nor came in direct contact with them. Here COVID-19 is a community transmission, where the disease spreads from one person to the other through direct contact. In the absence of vaccination and proper treatment, two force-control policies namely home isolation and hospital quarantine, are adopted by many countries to control this COVID-19 epidemic. The same is framed and analysed here with the help of this model as shown in Fig. 6.

3.3.1. Model formulation

Based on our hypothesis and flow of transmission of COVID-19 of stage 3 in human population as depicted in Fig. 6, we have the following system of equations:

\[
\begin{align*}
\frac{dS}{dt} &= B - \beta SI - \mu_1 S - \lambda S \\
\frac{dI}{dt} &= \beta SI + \delta Q_1 - (\mu_1 + \mu_2 + \eta)I \\
\frac{dQ_1}{dt} &= \lambda S - (\mu_1 + \delta)Q_1 \\
\frac{dQ_2}{dt} &= \eta I - (\mu_1 + \mu_2 + \gamma)Q_2 \\
\frac{dR}{dt} &= \gamma Q_2 - \mu_1 R
\end{align*}
\]

(5)

Since all the populations are positive, we have the total population, \( N = S + I + Q_1 + Q_2 + R \)

\[
\frac{dN}{dt} = \frac{dS}{dt} + \frac{dI}{dt} + \frac{dQ_1}{dt} + \frac{dQ_2}{dt} + \frac{dR}{dt}
\]

\[
\Rightarrow \frac{dN}{dt} = B - \mu_1 N - \mu_2 (I + Q_2)
\]

If the disease is absent in the population, then \( \frac{dN}{dt} = B - \mu_1 N \)

This shows that population size \( N \) tends to carrying capacity \( \frac{B}{\mu_1} \) as \( t \to \infty \)

It shows that the solution of (5) exists in the region defined by

\[
\Omega = \left\{ (S, I, Q_1, Q_2, R) \in \mathbb{R}_+^5 : S + I + Q_1 + Q_2 + R \leq \frac{B}{\mu_1} \right\}
\]

Since all the solution remains bounded in the positively invariant region \( \Omega \), and in the maximal interval \([0, \infty)\), the initial value problem is well posed.
3.3.2. Basic reproduction number
For the basic reproduction number, we take the following system of equations,
\[
\frac{dI}{dt} = \beta SL + \delta Q_1 - (\mu_1 + \mu_2 + \eta)I \\
\frac{dQ_1}{dt} = \lambda S - (\mu_1 + \delta)Q_1 \\
\frac{dQ_2}{dt} = \eta I - (\mu_1 + \mu_2 + \gamma)Q_2 \\
\frac{dR}{dt} = \gamma Q_2 - \mu_1 R
\]

On linearizing the model, we have,
\[
\begin{pmatrix}
\frac{dI}{dt} \\
\frac{dQ_1}{dt} \\
\frac{dQ_2}{dt} \\
\frac{dR}{dt}
\end{pmatrix} = (F - V) \begin{pmatrix}
I \\
Q_1 \\
Q_2 \\
R
\end{pmatrix}
\]

where F is the rate of infection and V is the rate of infection by compartment to compartment.

We have,
\[
F = \begin{pmatrix}
\beta & 0 & 0 & 0 \\
0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0
\end{pmatrix}, \quad V = \begin{pmatrix}
\eta + \mu_1 + \mu_2 & -\delta & 0 & 0 \\
0 & \mu_1 + \delta & 0 & 0 \\
-\eta & 0 & \gamma + \mu_1 + \mu_2 & 0 \\
0 & 0 & -\gamma & \mu
\end{pmatrix}
\]

Calculating the spectral radius, we have, basic reproduction number as
\[
R_0 = \frac{\beta \delta}{(\mu_1 + \mu_2 + \eta)(\mu_1 + \delta)}
\]

3.3.3. Equilibrium points
For the equilibrium points in the steady state of the system \(5\), we have,
\[
B - \beta SI - \mu_1 S - \lambda S = 0 \\
\beta SI + \delta Q_1 - (\mu_1 + \mu_2 + \eta)I = 0 \\
\lambda S - (\mu_1 + \delta)Q_1 = 0 \\
\eta I - (\mu_1 + \mu_2 + \gamma)Q_2 = 0 \\
\gamma Q_2 - \mu_1 R = 0
\]

3.3.4. Stability for disease free equilibrium
In this section, we discuss the local stability of endemic equilibrium and disease free equilibrium of the system \(5\) (after dropping the last equation of \(5\)) and then by analyzing the corresponding characteristic equations.

**Theorem 6.** If \(R_0 < 1\), the disease free equilibrium of the system \(5\) is locally asymptotically stable in the region \(\Omega\). If \(R_0 > 1\), it is unstable in the given region \(\Omega\).

**Proof:** The characteristic matrix at the disease free equilibrium is
\[
\Omega_0(1, 0, 0, 0) = \text{J}_{DFE} = \begin{pmatrix}
(\lambda + \mu_1) & -\beta & 0 & 0 \\
0 & \beta - (\eta + \mu_1 + \mu_2) & 0 & 0 \\
0 & \lambda & 0 & -(\mu_1 + \delta) \\
0 & \eta & 0 & -(\gamma + \mu_1 + \mu_2)
\end{pmatrix}
\]

One of the characteristic root or eigen value is \(z_1 = -(\gamma + \mu_1 + \mu_2)\), and other characteristic roots or eigen values can be solving the cubic equation \(Z^3 + C_1Z^2 + C_2Z + C_3 = 0\)

Where
\[
C_1 = 3\mu_1 + \mu_2 + \eta + \delta + \lambda - \beta \\
C_2 = 3\mu_1^2 + 3\mu_1\mu_2 + 2\mu_1\delta + 2\mu_1\eta + 2\mu_1\lambda + \lambda\mu_2 + \eta\lambda + \lambda\delta + \mu_2\delta + \delta\eta - \beta\lambda - 2\mu_1\beta - \delta\beta \\
C_3 = \beta\lambda\delta + \lambda\mu_1\mu_2 + \lambda\delta\mu_2 + \delta\eta\lambda + \mu_1^2 + \mu_1\mu_2 + \mu_2^2\delta + \delta\mu_1\mu_2 + \eta\mu_1\mu_2 - (\lambda\beta\mu_1 + \beta\delta\lambda + \mu_1^2\beta + \mu_1\delta\beta)
\]

So by Routh-Hurwitz criteria the system \(5\) in disease free equilibrium is locally asymptotically stable.
3.3.5. Endemic equilibrium

The endemic equilibrium \( \Omega^*(S^*, I^*, Q_1^*, Q_2^*, R^*) \), which is interior of \( \Omega \) and can be obtained by taking all the equations of the system (5) equal to zero and solving it simultaneously. We have,

\[
S^* = \frac{B}{(\beta I^* + \mu_1 + \lambda)} \\
I^* = \frac{\lambda B}{(\beta I^* + \lambda + \mu_1 - R_0B\lambda)(\mu_1 + \delta)} \\
Q_1^* = \frac{\lambda B}{(\beta I^* + \mu_1 + \lambda)(\mu_1 + \delta)} \\
Q_2^* = \frac{\eta B}{(\mu_1 + \mu_2 + \gamma)} \\
R^* = \frac{\gamma B}{(\mu_1 + \mu_2 + \gamma)}
\]

Theorem 7. The endemic equilibrium \( \Omega^*(S^*, I^*, Q_1^*, Q_2^*, R^*) \) is locally asymptotically stable when \( R_0 > 1 \)

Proof: On linearizing the system (6), we have,

\[
J_{EE} = \begin{pmatrix}
-\beta I^* - \lambda - \mu_1 & -\beta S^* & 0 & 0 & 0 \\
\beta I^* & \beta S^* - (\mu_1 + \mu_2 + \eta) & \delta & 0 & 0 \\
\lambda & 0 & -(\mu_1 + \delta) & 0 & 0 \\
0 & \eta & 0 & -(\mu_1 + \mu_2 + \gamma) & 0 \\
0 & 0 & 0 & \gamma & -\mu_1
\end{pmatrix}
\]

The two characteristic roots or Eigen values are

\[
\dot{z}_1 = -\mu_1 \\
\dot{z}_2 = -(\mu_1 + \mu_2 + \gamma)
\]

, which are negative real roots.

And other three roots are by solving the cubic equation

\[
Z^3 + D_1Z^2 + D_2Z + D_3 = 0
\]

Where,

\[
D_1 = 3\mu_1 + \mu_2 + \eta + \delta + \lambda + \beta I^* - \beta S^* \\
D_2 = 2\beta I^* + 3\mu_1 I^* + 2\mu_1 \mu_2 + \beta I^* \mu_1 + \beta I^* \mu_2 + \beta I^* \eta + 2\mu_1 \delta + \mu_1 \eta + \mu_1 + \lambda + \mu_1 + \eta \lambda + \lambda \delta + \mu_2 \delta + \delta \eta + \beta \delta I^* - \beta \lambda S^* - 2\mu_1 \beta S^* - \delta \beta S^* \\
D_3 = \beta S^* \lambda \delta + \lambda \mu_1 I^* + \beta I^* \mu_1 + \delta \beta I^* \mu_2 + \beta I^* \delta \mu_2 + \beta I^* \eta \mu_1 + \beta I^* \eta \delta + \mu_1^2 + \mu_1^2 \delta + \eta \mu_2^2 + \lambda \mu_1^2 + \mu_1^2 \mu_2 + \delta \mu_1 + \delta \mu_1 \mu_2 + \lambda \delta I^* + \lambda \beta I^* H - \lambda \beta I^* S^* - \lambda \beta H S^* - \beta S^* \mu_1^2 - \beta S^* \delta \mu_1
\]

\[:: D_1D_2 - D_3 > 0 \]

So by Routh–Hurwitz criteria the system is locally asymptotically stable. Thus all the Eigen values have negative real parts and hence the system (5) is locally asymptotically stable at the endemic equilibrium \( \Omega^* \) if \( R_0 > 1 \).

3.3.6. Global stability for endemic equilibrium

In order to prove the global stability of the region \( \Omega \) we take S and I compartment, leave Q_1 population and make it free from the R population. Therefore, in the positive quadrant of S-I plane, we apply Dulac’s criteria with multiplier \( D = 1/I \)

Consider,

\[
F_1 = B - \beta SL - \lambda S - \mu_1 S \\
F_2 = [\beta S - (\mu_1 + \mu_2 + \eta)] I
\]

Then

\[
DF_1 = \frac{\partial F_1}{\partial S} - \frac{\partial F_1}{\partial I} = \frac{\partial}{\partial S} [B - \beta I - \lambda I - \mu_1 I] + \frac{\partial}{\partial I} [\beta I - (\mu_1 + \mu_2 + \eta)]
\]

We have,

\[
\frac{\partial DF_1}{\partial S} + \frac{\partial DF_2}{\partial I} = \frac{\partial B}{\partial I} - \beta S - (\mu_1 + \mu_2 + \eta) < 0
\]

Thus there exists no periodic solution in the region \( \Omega \). Hence, by Poincare–Bendixon property all solution starting in positive quadrant of SI-plane with \( S > 0 \) and \( I > 0 \) approaches \( (S^*, I^*) \) as \( t \to \infty \). In this case, the limiting form of rest of the system of equations (5) shows that \( Q_1 \to Q_1^* \), \( Q_2 \to Q_2^* \) \( R \to R^* \). Thus the endemic equilibrium \( \Omega^*(S^*, I^*, Q_1^*, Q_2^*, R^*) \) is globally stable in the given region \( \Omega \) for the system (5).
4. Simulation and analysis

In this section, a large number of experiments have been performed to verify the analytical results obtained in Section 3. Simulations have been carried out for each model to depict local and global stability. Some examples are mentioned below for simulating the models either for $R_0 < 1$ or for $R_0 > 1$.

**Example 1:** The local stability of the infection-free equilibrium point has been numerically simulated and depicted in Figs. 7(a), 9(a) and 11(a) for stage 1, 2 and 3 respectively and the corresponding simulated data for this unsuccessful attack are listed in Tables 4, 7 and 10, respectively. The initial point and the parametric values along with basic reproduction number ($R_0$) are shown in Tables 5, 8 and 11 for stage 1, 2 and 3 respectively. The value of $R_0$ for all the three stages is less than one. It is clearly observed that the infection-free equilibrium point turns out to be stable for all the stages.

**Example 2:** The local stability of the endemic equilibrium point has been numerically simulated and depicted in Figs. 8(a), 10(a) and 12(a) for stage 1, 2 and 3 respectively and the corresponding simulated data for this successful attack are listed in Table 6, 9 and 12 respectively. The initial point and the parametric values along with basic reproduction number ($R_0$) are shown in Tables 5, 8 and 11 for stage 1, 2 and 3 respectively. The value of $R_0$ for all the three stages is less than one. It is clearly observed that the endemic equilibrium point turns out to be stable for all the stages.

![Fig. 7. (a). Local stability of infection-free equilibrium for Stage 1 model when $R_0 < 1$. (b). Quarantine population versus recovered population for Stage 1 model when $R_0 < 1$. (c). Home isolation population versus recovered population for Stage 1 model when $R_0 < 1$. (d). Infectious population versus quarantine population for Stage 1 model when $R_0 < 1$. (For interpretation of the references to colour in this figure legend, the reader is referred to the web version of this article.)](image_url)

![Fig. 7. Continued](image_url)
Fig. 7. Continued

Table 4
Time series for Fig. 7(a).

| Time | S   | E   | I   | Q₁  | Q₂  | R    |
|------|-----|-----|-----|-----|-----|------|
| 0    | 0.8800 | 0.0800 | 0.0400 | 0   | 0   | 0    |
| 0.0512 | 0.8807 | 0.0768 | 0.0400 | 0.0023 | 0.0014 | 0    |
| 1.0467 | 0.8953 | 0.0400 | 0.0463 | 0.0250 | 0.0219 | 0.0002 |
| 2.9053 | 0.9155 | 0.0224 | 0.0658 | 0.0255 | 0.0450 | 0.0014 |
| 5.5984 | 0.9273 | 0.0196 | 0.0900 | 0.0203 | 0.0733 | 0.0044 |
| 10.0414 | 0.9130 | 0.0194 | 0.1241 | 0.0186 | 0.1200 | 0.0119 |
| 18.6824 | 0.8493 | 0.0194 | 0.1777 | 0.0184 | 0.2078 | 0.0345 |
| 25.4880 | 0.7823 | 0.0194 | 0.1995 | 0.0184 | 0.2653 | 0.0576 |
| 34.3490 | 0.7196 | 0.0194 | 0.2015 | 0.0184 | 0.3125 | 0.0897 |
| 42.2205 | 0.6940 | 0.0194 | 0.1915 | 0.0185 | 0.3286 | 0.1160 |
| 50    | 0.6882 | 0.0194 | 0.1810 | 0.0185 | 0.3295 | 0.1377 |
### Table 5
Parameter values for the Figs. 7(a), 7(b), 7(c), 7(d) and 8 (a), (b(i)), (b(ii)), (c), (d), (e).

| Parameter | Fig. 7a, b, c, d | Fig. 8a, b(i), b(ii), c, d, e |
|-----------|-----------------|-----------------|
| A         | 0.02            | 0.02            |
| B         | 0.04            | 0.04            |
| μ₁        | 0.03            | 0.02            |
| μ₂        | 0.035           | 0.035           |
| α₁        | 0.1             | 0.1             |
| α₂        | 0.6             | 0.6             |
| β         | 0.2             | 0.85            |
| δ         | 0.4             | 0.4             |
| ε         | 0.2             | 0.2             |
| η         | 0.12            | 0.05            |
| γ         | 0.02            | 0.016           |
| λ         | —               | —               |
| (S₀, S₁, k₀, Q₁, Q₂, R₀) | (0.88, 0.08, 0.04, 0.0, 0.0, 0.0) | (0.88, 0.08, 0.04, 0.0, 0.0, 0.0) |
| R₀        | 0.39984506      | 3.0721966206    |

### Table 6
Time series for Fig. 8(a).

| Time | S     | E     | I     | Q₁    | Q₂    | R     |
|------|-------|-------|-------|-------|-------|-------|
| 0    | 0.8800| 0.0800| 0.0400| 0     | 0     | 0     |
| 0.0513| 0.8800| 0.0769| 0.0414| 0.0024| 0.0013| 0     |
| 1.0567| 0.8658| 0.0402| 0.0875| 0.0253| 0.0203| 0     |
| 2.9388| 0.7040| 0.0226| 0.2955| 0.0260| 0.0486| 0.0012|
| 3.9983| 0.6325| 0.0215| 0.3712| 0.0249| 0.0576| 0.0016|
| 3.8994| 0.5465| 0.0207| 0.4579| 0.0237| 0.0649| 0.0020|
| 4.4175| 0.4516| 0.0203| 0.5401| 0.0226| 0.0829| 0.0027|
| 5.0270| 0.3548| 0.0200| 0.6369| 0.0216| 0.1001| 0.0035|
| 5.6678| 0.2670| 0.0198| 0.7102| 0.0208| 0.1206| 0.0046|
| 9.4212| 0.0818| 0.0196| 0.7792| 0.0192| 0.2407| 0.0149|
| 13.5949| 0.0738| 0.0196| 0.6837| 0.0190| 0.3317| 0.0323|
| 20.5538| 0.0865| 0.0196| 0.5756| 0.0190| 0.4046| 0.0671|
| 31.5592| 0.0994| 0.0196| 0.5099| 0.0190| 0.4332| 0.1208|
| 40.3006| 0.1033| 0.0196| 0.4948| 0.0190| 0.4341| 0.1572|
| 50    | 0.1047| 0.0196| 0.4898| 0.0190| 0.4315| 0.1905|

### Table 7
Time series for Fig. 9(a) (Stage 2).

| Time | S     | E     | I     | Q₁    | Q₂    | R     |
|------|-------|-------|-------|-------|-------|-------|
| 0    | 0.8800| 0.0800| 0.0400| 0     | 0     | 0     |
| 0.0181| 0.8785| 0.0789| 0.0400| 0.0025| 0     | 0     |
| 0.7062| 0.8252| 0.0487| 0.0405| 0.0848| 0.0204| 0     |
| 2.7476| 0.6927| 0.0230| 0.0483| 0.2562| 0.0505| 0.0016|
| 5.4204| 0.5637| 0.0196| 0.0659| 0.4026| 0.0763| 0.0048|
| 9.7192| 0.4279| 0.0194| 0.0968| 0.5398| 0.1167| 0.0120|
| 18.1170| 0.3055| 0.0194| 0.1340| 0.6343| 0.1906| 0.0326|
| 25.5361| 0.2692| 0.0194| 0.1438| 0.6456| 0.2370| 0.0549|
| 34.1787| 0.2561| 0.0194| 0.1445| 0.6387| 0.2675| 0.0811|
| 42.0230| 0.2532| 0.0194| 0.1427| 0.6309| 0.2804| 0.1025|
| 50    | 0.2528| 0.0194| 0.1409| 0.6251| 0.2858| 0.1210|

### Table 8
Parameter values for the Figs. 9a, b, c, d and 10 a, b(i), b(ii), c, d, e (Stage 2).

| Parameter | Fig. 9a, b, c, d | Fig. 10a, b(i), b(ii), c, d, e |
|-----------|-----------------|-----------------|
| A         | 0.02            | 0.02            |
| B         | 0.04            | 0.04            |
| μ₁        | 0.03            | 0.03            |
| μ₂        | 0.035           | 0.035           |
| α₁        | —               | —               |
| α₂        | 0.6             | 0.7             |
| β         | 0.2             | 0.85            |
| δ         | 0.03            | 0.4             |
| ε         | —               | —               |
| η         | 0.12            | 0.12            |
| γ         | 0.02            | 0.02            |
| λ         | 0.1             | 0.4             |
| (S₀, S₁, k₀, Q₁, Q₂, R₀) | (0.88, 0.08, 0.04, 0.0, 0.0, 0.0) | (0.88, 0.08, 0.04, 0.0, 0.0, 0.0) |
| R₀        | 0.3148779848    | 2.9046883867    |
Table 9
Time series for Fig. 10(a) (Stage 2).

| Time | S   | E   | I   | Q₁  | Q₂  | R   |
|------|-----|-----|-----|-----|-----|-----|
| 0    | 0.8800 | 0.0800 | 0.0400 | 0    | 0    | 0   |
| 0.0060 | 0.8778 | 0.0796 | 0.0401 | 0.0025 | 0    | 0   |
| 0.0461 | 0.8632 | 0.0772 | 0.0412 | 0.0184 | 0.0013 | 0   |
| 0.3077 | 0.7732 | 0.0635 | 0.0543 | 0.1094 | 0.0082 | 0   |
| 1.0504 | 0.5550 | 0.0400 | 0.1381 | 0.2680 | 0.0264 | 0   |
| 2.0987 | 0.3206 | 0.0264 | 0.3077 | 0.3317 | 0.0606 | 0.0011 |
| 3.2284 | 0.1658 | 0.0216 | 0.4574 | 0.3007 | 0.1130 | 0.0030 |
| 4.5265 | 0.0853 | 0.0200 | 0.5403 | 0.2321 | 0.3836 | 0.0067 |
| 7.2394 | 0.0484 | 0.0195 | 0.5307 | 0.1306 | 0.3192 | 0.0195 |
| 10.9836 | 0.0490 | 0.0194 | 0.4260 | 0.0870 | 0.4340 | 0.0446 |
| 17.5783 | 0.0564 | 0.0194 | 0.3113 | 0.0826 | 0.4916 | 0.0933 |
| 26.8424 | 0.0604 | 0.0194 | 0.2709 | 0.0872 | 0.4784 | 0.1495 |
| 36.0504 | 0.0609 | 0.0194 | 0.2659 | 0.0882 | 0.4607 | 0.1888 |
| 44.7723 | 0.0610 | 0.0194 | 0.2655 | 0.0883 | 0.4517 | 0.2152 |
| 50       | 0.0610 | 0.0194 | 0.2655 | 0.0884 | 0.4487 | 0.2275 |

Table 10
Time series for Fig. 11(a) (Stage 3).

| Time | S   | Q₁  | I   | Q₂  | R   |
|------|-----|-----|-----|-----|-----|
| 0    | 0.8800 | 0.0800 | 0.0400 | 0    | 0   |
| 0.5128 | 0.8396 | 0.1210 | 0.0413 | 0.0024 | 0   |
| 2.7844 | 0.0899 | 0.2608 | 0.0513 | 0.0134 | 0   |
| 6.6132 | 0.5175 | 0.4153 | 0.0753 | 0.0347 | 0.0021 |
| 11.8482 | 0.3838 | 0.5007 | 0.1027 | 0.0684 | 0.0068 |
| 18.0982 | 0.3091 | 0.5208 | 0.1177 | 0.1056 | 0.0157 |
| 30.5982 | 0.2673 | 0.4917 | 0.1169 | 0.1463 | 0.0381 |
| 36.8482 | 0.2938 | 0.4761 | 0.1128 | 0.1531 | 0.0662 |
| 43.0982 | 0.2633 | 0.4645 | 0.1092 | 0.1543 | 0.0579 |
| 50       | 0.2638 | 0.4559 | 0.1062 | 0.1531 | 0.0662 |

Table 11
Parameter values for the Fig. 11a, b, c, d and 12 a, b, c, d, e (Stage 3).

| Parameter | Fig. 11a, b, c, d | Fig. 12a, b(i), b(ii), c, d, e |
|-----------|------------------|-----------------------------|
| A         | —                | —                           |
| B         | 0.04             | 0.04                        |
| μ₁        | 0.03             | 0.03                        |
| μ₂        | 0.035            | 0.035                       |
| α₁        | —                | —                           |
| α₂        | —                | —                           |
| β         | 0.2              | 0.85                        |
| δ         | 0.03             | 0.4                         |
| ε         | —                | —                           |
| η         | 0.12             | 0.12                        |
| γ         | 0.02             | 0.02                        |
| λ         | 0.4              | 0.4                         |
| (S₀, Qₓ₀, I₀, Qₓ₋₁ Rₓ) | (0.88, 0.08, 0.04, 0.00, 0.00) | (0.88, 0.08, 0.04, 0.00, 0.00) |
| R₀        | 5.405405405      | 3.2692307692                |

Table 12
Time series for Fig. 12(a) (Stage 3).

| Time | S   | Q₁  | I   | Q₂  | R   |
|------|-----|-----|-----|-----|-----|
| 0    | 0.8800 | 0.0800 | 0.0400 | 0    | 0   |
| 0.0419 | 0.8647 | 0.0930 | 0.0424 | 0    | 0   |
| 0.4175 | 0.7362 | 0.1898 | 0.0746 | 0.0027 | 0   |
| 0.7500 | 0.0335 | 0.2492 | 0.1164 | 0.0664 | 0   |
| 1.1564 | 0.5201 | 0.2949 | 0.1789 | 0.0132 | 0   |
| 1.6368 | 0.4035 | 0.3194 | 0.2597 | 0.0251 | 0   |
| 2.1608 | 0.2989 | 0.3201 | 0.3445 | 0.0426 | 0   |
| 2.7286 | 0.2129 | 0.3015 | 0.4218 | 0.0662 | 0.0012 |
| 4.5195 | 0.0941 | 0.2190 | 0.5204 | 0.1404 | 0.0042 |
| 5.5473 | 0.0600 | 0.1530 | 0.5406 | 0.2068 | 0.0086 |
| 8.5339 | 0.0484 | 0.0761 | 0.4560 | 0.3193 | 0.0232 |
| 10.5719 | 0.0507 | 0.0586 | 0.3870 | 0.3628 | 0.0354 |
| 13.4783 | 0.0554 | 0.0523 | 0.3095 | 0.3898 | 0.0335 |
| 21.2985 | 0.0641 | 0.0579 | 0.2163 | 0.3700 | 0.0961 |
| 32.0259 | 0.0669 | 0.0620 | 0.1962 | 0.3184 | 0.1321 |
| 44.5259 | 0.0671 | 0.0624 | 0.1952 | 0.2905 | 0.1536 |
| 50       | 0.0671 | 0.0624 | 0.1952 | 0.2849 | 0.1594 |
Example 3: The behavior of system 1, 3 and 5 are studied by considering quarantine population – recovered population plane and Fig. 7(b), 9(b) and 11(b) for stage 1, 2 and 3 respectively reflects that recovered population is directly proportionate with quarantine population for $R_o < 1$. Even for $R_o > 1$, the same relationship is achieved in Fig. 8(b)(i), 10(b)(i) and 12(b)(i) for stage 1, 2 and 3 respectively and the same with respect to time is shown with 3-dimentional graph in Fig. 8(b)(ii), 10(b)(ii) and 12(b)(ii) for stage 1, 2 and 3 respectively.

Example 4: The behavior of system (1), (3) and (5) are also studied by considering home isolation population – recovered population plane and Figs. 7(c), 9(c) and 11(c) for stage 1, 2 and 3 respectively reflects that recovered population is not much affected or depends on home isolation population for $R_o < 1$. Even for $R_o > 1$, the same relationship is achieved in Figs. 8(c), 10(c) and 12(c) for stage 1, 2 and 3 respectively.

Example 5: The behavior of system (1), (3) and (5) are also studied by considering infectious population – quarantine population plane and Fig. 7(d), 9(d) and 11(d) for stage 1, 2 and 3 respectively reflects that infectious population gradually becomes quarantine population and is under control. Whereas for $R_o > 1$, Figs. 8(d), 10(d) and 12(d) for stage 1, 2 and 3 respectively depicts that a large number of populations are infectious as well as quarantined.

Example 6: The global stability of the endemic equilibrium point for $R_o > 1$ is shown in Figs. 8(e), 10(e) and 12(e) for stage 1, 2 and 3 respectively. It shows the plane formed by the susceptible population and infectious population. It can be clearly seen in all above said figures that the trajectories are seen to be asymptotically stable at endemic equilibrium point which is unique and globally stable.

From Fig. 7(b) it is clear that, the more we quarantine the positive cases of COVID-19, the more is the recovery in the infectious population.

From Fig. 7(c), it is evident that the more we have social distancing of the susceptible population, that is, home isolation, the larger we have the chances to escape from the infectious COVID-19.

The more we quarantine the infectious COVID-19 population in the hospital, the more is the recovery, which is very clearly reflected from Fig. 10(b)(ii). After 11.85 days, the recovery starts, but after 21 days, there is a sharp increase in the recovery population.

Social distancing, that is, the more we home isolate the susceptible population, the transmission of the disease in population is almost negligible which is clearly reflected in Fig. 10(c).

The susceptible population when are in complete lockdown, the transmission of the disease is very less, which is evident from Fig. 11(c). Fig. 12(b)(i) clearly reflects that, the more we quarantine the positive cases of COVID-19, the recovery population is more.

From Fig. 12(b)(ii), it is very clear that, the more we quarantine the infectious population from COVID-19, the more is the recovery, and after 21 days of hospital quarantine there is a sharp increase in the recovery population.

From Fig. 12(c), it is evident that, the more we home isolate the susceptible population, the more is the chances of mitigation of COVID-19 spread.

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**Fig. 8.** (a) Local stability of infection-endemic equilibrium for Stage 1 model when $R_o > 1$, (b)(i). Quarantine population versus recovered population for Stage 1 model when $R_o > 1$, (b)(ii). Quarantine population versus recovered population with respect to time for Stage 1 model when $R_o > 1$, (c). Home isolation population versus recovered population for Stage 1 model when $R_o > 1$, (d). Infectious population versus quarantine population for Stage 1 model when $R_o > 1$. (e). Global stability of Infection-endemic equilibrium shown in S-I phase plane for Stage 1 model when $R_o > 1$. (For interpretation of the references to colour in this figure legend, the reader is referred to the web version of this article.)
Fig. 8. Continued
Fig. 8. Continued

Fig. 8. Continued
Fig. 8. Continued

Fig. 9. (a). Local stability of infection-free equilibrium for Stage 2 model when $R_0 < 1$. (b). Quarantine population versus recovered population for Stage 2 model when $R_0 < 1$. (c). Home isolation population versus recovered population for Stage 2 model when $R_0 < 1$. (d). Infectious population versus quarantine population for Stage 2 model when $R_0 < 1$. [For interpretation of the references to colour in this figure legend, the reader is referred to the web version of this article.]
Fig. 9. Continued
Fig. 9. Continued

(a). Local stability of infection-endemic equilibrium for Stage 2 model when $R_0 > 1$. (b)(i). Quarantine population versus recovered population for Stage 2 model when $R_0 > 1$. (b)(ii). Quarantine population versus recovered population with respect to time for Stage 2 model when $R_0 > 1$. (c). Home isolation population versus recovered population for Stage 2 model when $R_0 > 1$. (d). Infectious population versus quarantine population for Stage 2 model when $R_0 > 1$. (e). Global stability of infection-endemic equilibrium shown in S-I phase plane for Stage 2 model when $R_0 > 1$. (For interpretation of the references to colour in this figure legend, the reader is referred to the web version of this article.)
(i): Quarantine population verses recovered population for Stage 2 model when $R_0 > 1$

(ii): Quarantine population verses recovered population with respect to time for Stage 2 model when $R_0 > 1$

Fig. 10. Continued
Fig. 10. Continued
Fig. 10. Continued

Fig. 11. (a) Local stability of infection-free equilibrium for Stage 3 model when $R_0 < 1$. (b) Quarantine population versus recovered population for Stage 3 model when $R_0 < 1$. (c) Home isolation population versus recovered population for Stage 3 model when $R_0 < 1$. (d) Infectious population versus quarantine population for Stage 3 model when $R_0 < 1$. (For interpretation of the references to colour in this figure legend, the reader is referred to the web version of this article.)
Fig. 11. Continued
Fig. 11. Continued

Fig. 12. (a) Local stability of infection-endemic equilibrium for Stage 3 model when $R_0 > 1$. (b)(i). Quarantine population verses recovered population for Stage 3 model when $R_0 > 1$. (b)(ii). Quarantine population verses recovered population with respect to time for Stage 3 model when $R_0 > 1$. (c). Home isolation population verses recovered population for Stage 3 model when $R_0 > 1$. (d). Infectious population verses quarantine population for Stage 3 model when $R_0 > 1$. (e). Global stability of infection-endemic equilibrium shown in S-I phase plane for Stage 3 model when $R_0 > 1$. (For interpretation of the references to colour in this figure legend, the reader is referred to the web version of this article.)
(i): Quarantine population verses recovered population for Stage 3 model when $R_0 > 1$

(ii): Quarantine population verses recovered population with respect to time for Stage 3 model when $R_0 > 1$

Fig. 12. Continued
Fig. 12. Continued
5. Conclusion

Three quarantine models are developed for three different stages of COVID-19. Reproduction number for all the three stages is obtained and the condition for local and global asymptotic stability is well established. When the reproduction number is greater than one, the systems developed in all the three stages of infection-endemic equilibrium in S-I phase plane is globally asymptotic stable. The two vital pivot parameters of all the three models of COVID-19, home isolation and hospital quarantine is well analysed. From the numerical simulations it is very clear that the more we have home isolation (or complete lockdown or social distancing), the less we have the chances to be infected and the disease to be transmitted in the population, which is very evident from Figs. 7(c), 8(c), 9(c),10(c),11(c), 12(c). The general home isolation for our results is approximately 11.85 days. From the simulation results it is also evident from Figs. 8(b)(i), 9(b), 10(b)(i)&(ii), 11(b), 12(b)(i) & (ii) that COVID-19 positive population when are subjected to hospital quarantine, the transmission of the disease is reduced and the rate of recovery also increases. Till date when no vaccine or treatment is available for COVID-19, then from our developed models, it is well established that social distancing and hospital quarantine (for positive cases) is the only best treatment.

Declaration of Competing Interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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