Chapter 13
Compartmental Modelling Approach for Accessing the Role of Non-Pharmaceutical Measures in the Spread of COVID-19

Yashika Bahri, Sumit Kaur Bhatia, Sudipa Chauhan, and Mandeep Mittal

Abstract Epidemic diseases are well known to be fatal and cause great loss worldwide—economically, socially and mentally. Even after around nine months, since the Coronavirus Disease 2019 (COVID-19) began to spread, people are getting infected all over the world. This is one of the areas where human medical advancements fail because by the time the disease is identified and its treatment is figured out, most of the population is already exposed to it. In such cases, it becomes easier to take steps if the dynamics of the disease and its sensitivity to various factors is known. This chapter deals with developing a mathematical model for the spread of Coronavirus disease, by employing a number of parameters that affect its spread. A compartmental modelling approach using ordinary differential equation has been used to formulate the set of equations that describe the model. We have used the next generation matrix method to find the basic reproduction number of the system and proved that the system is locally asymptotically stable at the disease-free equilibrium for $R_0 < 1$. Stability and existence of endemic equilibrium have been discussed, followed by sensitivity of infective classes to parameters like proportion of vaccinated individuals and precautionary measures like social distancing. It is expected that after the vaccine is developed and is available to use, as the proportion of vaccinated individuals will increase, the infection will decrease in the population which can gradually lead to herd immunity. Since, the vaccine is still under development, non-intervention measures play a major role in coping with the disease. The disease generally transmits when the water droplets from an infected individuals' mouth or nose are inhaled by a healthy individual. The best measures that should be adopted are social distancing, washing one's hands frequently, and covering one's mouth with mask, quarantine and lockdowns. Thus, as more and more precautionary measures are taken, it would gradually reduce the infection which has also been proved numerically by the sensitivity analysis of 'w' in our dynamical analysis.

Keywords Compartmental modelling · Reproduction number · Non-pharmaceutical measures · Equilibrium points · Sensitivity analysis

Y. Bahri · S. K. Bhatia (✉) · S. Chauhan · M. Mittal
Amity Institute of Applied Sciences, Amity University, Uttar Pradesh, India
e-mail: skbhatia@amity.edu

N. H. Shah and M. Mittal (eds.), *Mathematical Analysis for Transmission of COVID-19*,
Mathematical Engineering, https://doi.org/10.1007/978-981-33-6264-2_13

Mathematics Subject Classification 34A34 · 34B09 · 34D05 · 34D20

Introduction

Epidemic diseases have always been a major concern worldwide. In addition to many people losing their lives, outbreaks also impose financial, social, and mental strains on individuals and nations as a whole. History has many examples of such brutal outbreaks like Russian plague, Flu pandemic, Spanish Flu, Asian Flu, H1N1 Swine Flu pandemic, West African Ebola epidemic, Zika virus epidemic and many more. Quite a chunk of these have been able to be suppressed by vaccinations, ofcourse only after causing the mass destructions, while some diseases still have no vaccinations. A current such threat is COVID-19 epidemic, which started spreading from the Wuhan city of China in December 2019 and is now brutally taking lives worldwide. Since, people were unaware of this new virus initially, they kept on travelling internationally and the virus soon spread out from China to all across the world. One of the worst affected countries were Italy and Spain. These were the first to suffer at the hands of the virus and had massive death tolls. Most other countries had a buffer period and did not experience an outbreak immediately, so steps like passenger screening were adopted at airports, assuming that the virus could only affect an individual if the person had a travel history to affected countries like China or Italy. Soon cases started appearing wherein the patients did not had a travel history but came in contact with someone who had and it did not take long for the community spread to begin leading to outbreak all over the world. With no vaccination at hand and no prior information about the virus or it's treatment, it had been difficult to handle the disease initially and even months after the spread the number of cases are still increasing. Various researches are being carried out by several countries to develop a vaccination for the disease and some have already come up with one, like Russia.

But a major fact about epidemic diseases is that they mostly cause an outbreak and are unable to control initially because of lack of prior knowledge. Even though the human race has aced in providing themselves health stability over the years, by getting deeper into medical research, it takes time and effort to deal with epidemics. That is where epidemiology comes into play. In order to deal with epidemics, epidemiologists bring together the real life prospects of the disease into a mathematical model and using real life data one can make estimates regarding length and extent of the transmission and also various control measures that can be adopted to control the disease in long and short run. In the presence of a vaccine, one can even make estimates regarding how much of the population has to be vaccinated before achieving herd immunity. For instance, Ochoche and Gweryina in their study on measles using mathematical modelling concluded that atleast 94 % of the population must be vaccinated in order to achieve herd immunity [1]. Similarly, Kassa et al. in their research on COVID-19 have discussed various mitigation strategies to cope with the disease [7]. Different possible scenarios are being considered by researchers in order to be as accurate as

possible [5, 8–10, 14–17]. Ngonghala et al. came up with a complex mathematical model for COVID-19 to study the impact of non-pharmaceutical interventions in [4]. Chang et al. have discussed the impact of media coverage on the spread of the disease in their research [6]. Researchers like Paul et al. have done prediction analysis for various South Asian countries focussing on the impact of various precautionary measures in [3].

In the sections that follow, we have attempted to study the spread of an epidemic with the help of mathematical modelling, bringing together the various stages involved in the spread of an epidemic and analysing various factors affecting this spread. We have first dealt with a general model for an epidemic, incorporating a variety of parameters that affects it's spread. Scenarios like vaccination have been added that can be used based on whether vaccination is available for a particular disease or not. Later on we have used the model to analyse the spread of COVID-19 and the intensity of the affect certain parameters have on it. Section Model Formulation deals with model formulation. In Sect. Stability Analysis, we have discussed about the stability and existence of the two equilibrium points - the disease-free and the endemic equilibrium based on the basic reproduction number. Section Sensitivity Analysis consists of sensitivity analysis of COVID-19 model parameters. Lastly, we have concluded the results in Sect. Conclusion discussing a few mitigation strategies and summarising the importance of epidemiology.

Model Formulation

Description

The model is based on the *S-E-I-R-S* deterministic compartmental modelling approach. The total population(N) is divided amongst seven compartments listed in Table 13.1.

Table 13.1 The seven compartments in the model (Source: own)

Compartment	Description
S	Susceptible individuals (those at a risk of being infected)
E	Exposed individuals (those exposed to the infection)
I	Symptomatic individuals (infected individuals who show symptoms)
I_a	Asymptomatic individuals (infected individuals who do not show any symptoms)
I_q	Quarantined individuals (infected individuals who only show mild symptoms)
H	Hospitalized individuals
R	Recovered individuals

In the beginning the model assumes that the entire population is susceptible. The susceptible individuals are exposed to the disease after coming in contact with the infectious population. The force of infection is defined as $\beta SI_a + \frac{\beta SI}{1+\alpha I}$. Keeping in mind the fact that people tend to avoid direct contact with symptomatic individuals and hence the infection due to symptomatic individuals is less than that of asymptomatic individuals, the inhibitory parameter α has been incorporated in the expression. Once the disease starts spreading people tend to take precautionary measures like washing their hands frequently, using face masks and social distancing which is being taken care by the parameter ω.

The exposed individuals move to the infectious compartments (I, I_a, I_q) at a rate of σ depending on the intensity of infection, in the proportions ρ_1, ρ_2 and ρ_3, respectively. Individuals showing very high symptoms move to the 'Symptomatic' compartment, those showing no symptoms at all move to 'Asymptomatic' compartment and individuals showing mild symptoms move to 'Quarantined' compartment.

The infectious individuals move to 'Hospitalized' and 'Recovered' compartments depending upon their health status. Further at any stage individuals might die a natural death or a disease induced death depending upon their compartmental position. The model also incorporates new births at a rate b.

To provide flexibility to the modelling process parameters p and $\triangle$ have been incorporated in the model that denote the proportion of vaccinated individuals and the rate at which the recovered individuals become susceptible again, respectively. Both of these scenarios are not always sure to occur and hence can be set to 0 whenever the model does not allow for vaccination or when recovery ensures immunity. Figure 13.1 shows the flow diagram of the model, depicting the various stages involved in it.

Based on the above description the model can be represented by the following system of differential equations:

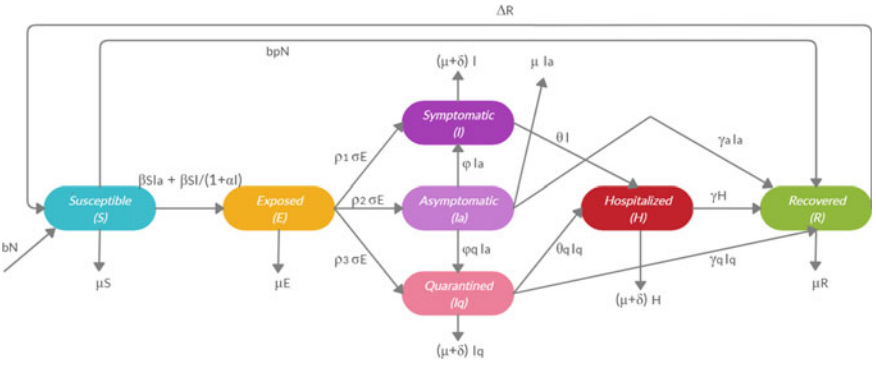


Fig. 13.1 Flow diagram for model (*Source* own)

$$
\begin{aligned}
\frac{dS}{dt} &= bN(1-p) - \beta SI_a - \frac{\beta SI}{1+\alpha I} - \mu S + \triangle R \\
\frac{dE}{dt} &= \beta SI_a(1-\omega) + \frac{\beta SI}{1+\alpha I}(1-\omega) - (\sigma+\mu)E \\
\frac{dI}{dt} &= \rho_1\sigma E + \phi I_a - (\theta+\mu+\delta)I \\
\frac{dI_a}{dt} &= \rho_2\sigma E - (\phi+\phi_q+\gamma_a+\mu)I_a \\
\frac{dI_q}{dt} &= \rho_3\sigma E + \phi_q I_a - (\theta_q+\gamma_q+\mu+\delta)I_q \\
\frac{dH}{dt} &= \theta I + \theta_q I_q - (\gamma+\mu+\delta)H \\
\frac{dR}{dt} &= bpN + \gamma_a I_a + \gamma_q I_q + \gamma H - (\triangle+\mu)R
\end{aligned}
\tag{13.1}
$$

Table 13.2 lists all the parameters used in the model and their description.

Table 13.2 Description of model parameters (Source: own)

Parameter	Description
b	Birth rate
μ	Natural death rate
δ	Disease-induced death rate
p	Proportion of vaccinated individuals
ρ_1	Proportion of exposed individuals moving to Symptomatic class
ρ_2	Proportion of exposed individuals moving to Asymptomatic class
ρ_3	Proportion of exposed individuals moving to Quarantined class
α	Parameter measuring inhibitory effect
β	Transmission rate
σ	Transition rate to infected classes from Exposed class
θ	Transition rate to Hospitalized class from Symptomatic class
θ_q	Transition rate to Hospitalized class from Quarantined class
γ	Transition rate to Recovered class from Symptomatic class
γ_a	Transition rate to Recovered class from Asymptomatic class
γ_q	Transition rate to Recovered class from Quarantined class
ϕ	Transition rate to Symptomatic class from Asymptomatic class
ϕ_q	Transition rate to Quarantined class from Asymptomatic class
$\triangle$	Transition rate to Susceptible class from Recovered class
ω	Parameter capturing the effect of precautionary measures like washing hands frequently, using masks and social distancing

Well Orderedness

The feasible region for the system (13.1) is:

$$\tau = \{(S, E, I, I_a, I_q, H, R) : S + E + I + I_A + I_q + H + R \leqslant \frac{bN}{\mu}, \\ S > 0, E \geqslant 0, I \geqslant 0, I_a \geqslant 0, I_q \geqslant 0, H \geqslant 0, R \geqslant 0\} \quad (13.2)$$

Stability Analysis

Disease-Free Equilibrium (E_0)

Disease-free equilibrium (DFE) is defined as the point at which the disease is completely eradicated from the system. Hence, all the infectious classes become constant at zero and the non-infectious classes attain a constant non-zero level.
In order to compute DFE of system in (13.1), we first set the sum of equations equal to zero:

$$0 = \frac{dS}{dt} + \frac{dE}{dt} + \frac{dI}{dt} + \frac{dI_a}{dt} + \frac{dI_q}{dt} + \frac{dH}{dt} + \frac{dR}{dt}$$

$$0 = bN - (S + E + I + I_a + I_q + H + R)\mu - (I + I_q + H)\delta - \beta S I_a \omega - \frac{\beta S I \omega}{1 + \alpha I}$$

Next we substitute, $E = I = I_a = I_q = H = R = 0$:

$$0 = bN - S\mu$$

$$S = \frac{bN}{\mu}$$

Thus we have the DFE,

$$E_0 = (S^*, E^*, I^*, I_a^*, I_q^*, H^*, R^*) = \left(\frac{bN}{\mu}, 0, 0, 0, 0, 0, 0\right) \quad (13.3)$$

Basic Reproduction Number (R_0)

Basic reproduction number is defined as the average number of new infectious individuals produced when a single infectious individual is exposed to the susceptible popultaion. R_0 plays a major role in stability analysis of epidemic diseases. It is one of the first parameters to be calculated while modelling any epidemic because

it determines important factors like whether the system would be stable or not, the disease will persist or will be eradicated, and what perecentage of population should be vaccinated in order to achieve herd immunity.
Hence, if R_0 is known we can make the following conclusions:

1. $R_0 < 1$ implies that on an average each new infectious will give rise to less than one new infectious upon contact and hence the DFE is stable which implies that the disease will be eradicated.
2. $R_0 > 1$ implies that on an average each new infectious will give rise to more than one infectious upon contact and hence the DFE is unstable which implies that the disease will not be eradicated and will lead to an outbreak.

In order to compute R_0 we will bring into use the next generation matrix method [1]. R_0 is equal to the spectral radius of the next generation matrix.

Step 1: We first express system (13.1) as:

$$\mathcal{H} = (E, I, I_a, I_q, H, R, S)$$

which can be rewritten as:

$$\begin{aligned} \mathcal{H}' &= \mathscr{F}(x) - \mathscr{V}(x) \\ &= \mathscr{F}(x) - [\mathscr{V}^-(x) - \mathscr{V}^+(x)] \end{aligned}$$

where, $\mathscr{F}(x) = \begin{bmatrix} \beta S(1-\omega)(I_a + \frac{I}{1+\alpha I}) \\ 0 \\ 0 \\ 0 \\ 0 \\ 0 \\ 0 \end{bmatrix}$ and $\mathscr{V}(x) = \begin{bmatrix} (\sigma+\mu)E \\ (\theta+\mu+\delta)I - \rho_1\sigma E - \phi I_a \\ (\phi+\phi_q+\gamma_a+\mu)I_a - \rho_2\sigma E \\ (\theta_q+\gamma_q+\mu+\delta)I_q - \rho_3\sigma E - \phi_q I_a \\ (\gamma+\mu+\delta)H - \theta I - \theta_q I_q \\ (\mu+\triangle)R - bpN - \gamma_a I_a - \gamma_q I_q - \gamma H \\ bN(1-p) + \beta S(I_a + \frac{I}{1+\alpha I}) + \mu S - \triangle R \end{bmatrix}$

$\mathscr{F}(x)$ is a column vector whose each entry is the collection of terms which result in new infectious in each compartment and $\mathscr{V}(x)$ is a column vector consisting of remaining terms.

Step 2: Next we find the Jacobian matrix of $\mathscr{F}(x)$:

$$D\mathscr{F} = \begin{bmatrix} 0 & \frac{\beta S(1-\omega)}{(1+\alpha I)^2} & \beta S(1-\omega) & 0\,0\,0 & \beta(1-\omega)(I_a + \frac{I}{1+\alpha I}) \\ 0 & 0 & 0 & 0\,0\,0 & 0 \\ 0 & 0 & 0 & 0\,0\,0 & 0 \\ 0 & 0 & 0 & 0\,0\,0 & 0 \\ 0 & 0 & 0 & 0\,0\,0 & 0 \\ 0 & 0 & 0 & 0\,0\,0 & 0 \\ 0 & 0 & 0 & 0\,0\,0 & 0 \end{bmatrix}$$

The Jacobian matrix of $\mathscr{F}(x)$ at DFE is:

$$D\mathscr{F}(E_0) = \begin{bmatrix} 0 & \frac{b\beta N(1-\omega)}{\mu} & \frac{b\beta N(1-\omega)}{\mu} & 0\,0\,0\,0 \\ 0 & 0 & 0 & 0\,0\,0\,0 \\ 0 & 0 & 0 & 0\,0\,0\,0 \\ 0 & 0 & 0 & 0\,0\,0\,0 \\ 0 & 0 & 0 & 0\,0\,0\,0 \\ 0 & 0 & 0 & 0\,0\,0\,0 \\ 0 & 0 & 0 & 0\,0\,0\,0 \end{bmatrix} = \begin{bmatrix} F & 0 \\ 0 & 0 \end{bmatrix}$$

where,

$$F = \begin{bmatrix} 0 & \frac{b\beta N(1-\omega)}{\mu} & \frac{b\beta N(1-\omega)}{\mu} & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{bmatrix}$$

Step 3: Next we find the Jacobian matrix of $\mathscr{V}(x)$:

$$D\mathscr{V} = \begin{bmatrix} (\sigma+\mu) & 0 & 0 & 0 & 0 & 0 & 0 \\ -\rho_1\sigma & (\theta+\mu+\delta) & -\phi & 0 & 0 & 0 & 0 \\ -\rho_2\sigma & 0 & (\phi+\phi_q+\gamma_a+\mu) & 0 & 0 & 0 & 0 \\ -\rho_3\sigma & 0 & -\phi_q & (\theta_q+\gamma_q+\mu+\delta) & 0 & 0 & 0 \\ 0 & -\theta & 0 & -\theta_q & (\gamma+\mu+\delta) & 0 & 0 \\ 0 & 0 & -\gamma_a & -\gamma_q & -\gamma & (\triangle+\mu) & 0 \\ 0 & \frac{\beta S}{(1+\alpha I)^2} & \beta S & 0 & 0 & -\triangle & \mu+\beta(I_a+\frac{I}{1+\alpha I}) \end{bmatrix}$$

The Jacobian matrix of $\mathscr{V}(x)$ at DFE is:

$$D\mathscr{V}(E_0) = \begin{bmatrix} (\sigma+\mu) & 0 & 0 & 0 & 0 & 0 & 0 \\ -\rho_1\sigma & (\theta+\mu+\delta) & -\phi & 0 & 0 & 0 & 0 \\ -\rho_2\sigma & 0 & (\phi+\phi_q+\gamma_a+\mu) & 0 & 0 & 0 & 0 \\ -\rho_3\sigma & 0 & -\phi_q & (\theta_q+\gamma_q+\mu+\delta) & 0 & 0 & 0 \\ 0 & -\theta & 0 & -\theta_q & (\gamma+\mu+\delta) & 0 & 0 \\ 0 & 0 & -\gamma_a & -\gamma_q & -\gamma & (\triangle+\mu) & 0 \\ 0 & \frac{\beta bN}{\mu} & \frac{\beta bN}{\mu} & 0 & 0 & -\triangle & \mu \end{bmatrix} = \begin{bmatrix} V & 0 \\ J_1 & J_2 \end{bmatrix}$$

where,

$$V = \begin{bmatrix} (\sigma+\mu) & 0 & 0 & 0 \\ -\rho_1\sigma & (\theta+\mu+\delta) & -\phi & 0 \\ -\rho_2\sigma & 0 & (\phi+\phi_q+\gamma_a+\mu) & 0 \\ -\rho_3\sigma & 0 & -\phi_q & (\theta_q+\gamma_q+\mu+\delta) \end{bmatrix}$$

Step 4: The last thing we need to generate the next generation matrix is V^{-1}, which after calculations is as below:

$$V^{-1} = \begin{bmatrix} \frac{1}{(\sigma+\mu)} & 0 & 0 & 0 \\ (\frac{\sigma}{(\sigma+\mu)(\theta+\mu+\delta)})(\rho_1 + \frac{\rho_2\phi}{(\phi+\phi_q+\gamma_a+\mu)}) & \frac{1}{(\theta+\mu+\delta)} & \frac{\phi}{(\theta+\mu+\delta)(\phi+\phi_q+\gamma_a+\mu)} & 0 \\ \frac{\rho_2\sigma}{(\sigma+\mu)(\phi+\phi_q+\gamma_a+\mu)} & 0 & \frac{1}{(\phi+\phi_q+\gamma_a+\mu)} & 0 \\ (\frac{\sigma}{(\sigma+\mu)(\theta_q+\gamma_q+\mu+\delta)})(\frac{\rho_2\phi}{(\phi+\phi_q+\gamma_a+\mu)} + \rho_3) & 0 & \frac{\phi_q}{(\theta_q+\gamma_q+\mu+\delta)(\phi+\phi_q+\gamma_a+\mu)} & \frac{1}{(\theta_q+\gamma_q+\mu+\delta)} \end{bmatrix}$$

Step 5: Now, we shall compute next generation matrix:

$$FV^{-1} = \left[f_{ij}\right]_{7X7}$$

where,

$$f_{11} = \frac{\beta bN\sigma(1-\omega)\sigma}{\mu(\sigma+\mu)}\left(\frac{\rho_1}{(\theta+\mu+\delta)} + \frac{\rho_2\phi}{(\theta+\mu+\delta)(\phi+\phi_q+\gamma_a+\mu)} + \frac{\rho_2}{(\phi+\phi_q+\gamma_a+\mu)}\right);$$

$$f_{12} = \frac{\beta bN(1-\omega)}{\mu(\theta+\mu+\delta)};$$

$$f_{13} = \frac{\beta bN(1-\omega)}{\mu(\phi+\phi_q+\gamma_a+\mu)};$$

and all other entries are zero.

Spectrum of FV^{-1} is the set of eigen values of the matrix:
$\{\frac{\beta bN\sigma(1-\omega)}{\mu(\sigma+\mu)}(\frac{\rho_1}{(\theta+\mu+\delta)} + \frac{\rho_2\phi}{(\theta+\mu+\delta)(\phi+\phi_q+\gamma_a+\mu)} + \frac{\rho_2}{(\phi+\phi_q+\gamma_a+\mu)}); 0; 0; 0\}$

Spectral radius of FV^{-1} is the maximum eigen value of the matrix:
$\frac{\beta bN\sigma(1-\omega)}{\mu(\sigma+\mu)}\left(\frac{\rho_1}{(\theta+\mu+\delta)} + \frac{\rho_2\phi}{(\theta+\mu+\delta)(\phi+\phi_q+\gamma_a+\mu)} + \frac{\rho_2}{(\phi+\phi_q+\gamma_a+\mu)}\right)$

Hence, $R_0 = \frac{\beta bN\sigma(1-\omega)}{\mu(\sigma+\mu)}\left(\frac{\rho_1}{(\theta+\mu+\delta)} + \frac{\rho_2\phi}{(\theta+\mu+\delta)(\phi+\phi_q+\gamma_a+\mu)} + \frac{\rho_2}{(\phi+\phi_q+\gamma_a+\mu)}\right)$

or

$$R_0 = \frac{\beta bN\sigma(1-\omega)(\rho_1(\phi+\phi_q+\gamma_a+\mu) + \rho_2(\phi+\theta+\mu+\delta))}{\mu(\sigma+\mu)(\theta+\mu+\delta)(\phi+\phi_q+\gamma_a+\mu)} \tag{13.4}$$

Local Stability of Disease-Free Equilibrium

The stability of DFE ensures that the disease can be removed from the system over a finite period of time. We will now derive conditions for local stability of system (13.1). The Jacobian matrix for the system in (13.1) is given by:

$$J = \begin{bmatrix} -\mu-\beta_* & 0 & -\frac{\beta S}{(1+\alpha I)^2} & -\beta S & 0 & 0 & \triangle \\ \beta_*(1-\omega) & -(\sigma+\mu) & \frac{\beta S(1-\omega)}{(1+\alpha I)^2} & \beta S(1-\omega) & 0 & 0 & 0 \\ 0 & \rho_1\sigma & -(\theta+\mu+\delta) & \phi & 0 & 0 & 0 \\ 0 & \rho_2\sigma & 0 & -(\phi+\phi_q+\gamma_a+\mu) & 0 & 0 & 0 \\ 0 & \rho_3\sigma & 0 & \phi_q & -(\theta_q+\gamma_q+\mu+\delta) & 0 & 0 \\ 0 & 0 & \theta & 0 & \theta_q & -(\gamma+\mu+\delta) & 0 \\ 0 & 0 & 0 & \gamma_a & \gamma_q & \gamma & -(\triangle+\mu) \end{bmatrix} \tag{3.3}$$

where, $\beta_* = \beta(I_a + \frac{I}{1+\alpha I})$

The Jacobian matrix for the system in (13.1) at DFE is given by:

$$J(E_0) = \begin{bmatrix} -\mu & 0 & -\beta S^* & -\beta S^* & 0 & 0 & \triangle \\ 0 & -(\sigma+\mu) & \beta S^*(1-\omega) & \beta S^*(1-\omega) & 0 & 0 & 0 \\ 0 & \rho_1\sigma & -(\theta+\mu+\delta) & \phi & 0 & 0 & 0 \\ 0 & \rho_2\sigma & 0 & -(\phi+\phi_q+\gamma_a+\mu) & 0 & 0 & 0 \\ 0 & \rho_3\sigma & 0 & \phi_q & -(\theta_q+\gamma_q+\mu+\delta) & 0 & 0 \\ 0 & 0 & \theta & 0 & \theta_q & -(\gamma+\mu+\delta) & 0 \\ 0 & 0 & 0 & \gamma_a & \gamma_q & \gamma & -(\triangle+\mu) \end{bmatrix}$$

where, $S^* = \dfrac{bN}{\mu}$

Now, the system in (13.1) is locally assymptotically stable if the eigen values of the above jacobian matrix are all real and negative, which is true if the following conditions are met:

1. $(\theta+\mu+\delta)+(\sigma+\mu)+(\phi+\phi_q+\gamma_a+\mu) > 0$
2. $-\beta S^*\sigma(1-\omega)(\rho_1+\rho_2)+(\theta+\mu+\delta)[(\sigma+\mu)+(\phi+\phi_q+\gamma_a+\mu)]+(\sigma+\mu)(\phi+\phi_q+\gamma_a+\mu) > 0$
3. $-\beta S^*\sigma(1-\omega)[\rho_1(\phi+\phi_q+\gamma_a+\mu)+\rho_2(\phi+\theta+\mu+\delta)]+(\sigma+\mu)(\theta+\mu+\delta)(\phi+\phi_q+\gamma_a+\mu) > 0$

It can be observed that condition 2 holds true for $R_0 < 1$ and it can be deduced from the third condition that:

$$\frac{\beta S^*\sigma(1-\omega)[\rho_1(\phi+\phi_q+\gamma_a+\mu)+\rho_2(\phi+\theta+\mu+\delta)]}{(\sigma+\mu)(\theta+\mu+\delta)(\phi+\phi_q+\gamma_a+\mu)} < 1$$

$$\frac{\beta bN\sigma(1-\omega)[\rho_1(\phi+\phi_q+\gamma_a+\mu)+\rho_2(\phi+\theta+\mu+\delta)]}{\mu(\sigma+\mu)(\theta+\mu+\delta)(\phi+\phi_q+\gamma_a+\mu)} < 1$$

which implies that:

$R_0 < 1$

Hence, we have the following theorem.

Theorem 1 *The system (13.1) is locally asymptotically stable at the disease-free equilibrium point if* $R_0 < 1$.

Endemic Equilibirum (E_1)

Endemic equilibrium is defined as the point at which the disease is not completely eradicated but it approaches to a constant level in the population. Let the endemic equilibrium for system (13.1) be denoted by:

$$E_1 = (S^{**}, E^{**}, I^{**}, I_a^{**}, I_q^{**}, H^{**}, R^{**})$$

To solve for endemic equilibrium, we assume force of infection to be defined as:

$$\lambda^{**} = \beta I_a^{**} + \frac{\beta I^{**}}{1 + \alpha I^{**}}$$

Each of the equation in system (13.1) is then equated to zero to get the following solution:

$$\begin{aligned} E_1 = (&\frac{M}{\mu + J\lambda^{**}}, A\lambda^{**}S^{**}, \frac{\sigma A\lambda^{**}S^{**}G}{BC}, \frac{\rho_2\sigma A\lambda^{**}S^{**}}{B}, \\ &\frac{\sigma A\lambda^{**}S^{**}L}{BD}, \frac{\sigma A\lambda^{**}S^{**}(\theta DG + \theta_q CL)}{BCDE}, \frac{bpN}{F} + \frac{(1-J)\lambda^{**}S^{**}}{\triangle}) \end{aligned} \tag{13.5}$$

Here, λ^{**} ca the below equation:

$$a_0(\lambda^{**})^2 + b_0\lambda^{**} + c_0 = 0 \tag{13.6}$$

where,

$$\begin{aligned} a_0 &= \frac{J^2}{\mu} + \frac{\alpha\sigma AGM}{\mu BC} \\ b_0 &= 2J + \frac{\alpha\sigma AGM}{BC} - \frac{\alpha\beta\sigma^2\rho_2 A^2 GM^2}{\mu B^2 C} - J(1 - p + \frac{\triangle p}{F})R_0 \\ c_0 &= -\mu[(1 - p + \frac{\triangle p}{F})R_0 - 1] \end{aligned}$$

Further $A, B, C, D, E, F, G, L, M, J$ used in the above expressions can be calculated as:

$$
\begin{aligned}
A &= \frac{1-\omega}{\sigma+\mu} \\
B &= \phi + \phi_q + \gamma_a + \mu \\
C &= \theta + \mu + \delta \\
D &= \theta_q + \gamma_q + \mu + \delta \\
E &= \mu + \gamma + \delta \\
F &= \mu + \triangle \\
G &= B\rho_1 + \phi\rho_2 \\
L &= B\rho_3 + \phi_q\rho_2 \\
M &= bN(1 - p + \frac{\triangle p}{F}) \\
J &= 1 - (\frac{\triangle\sigma A}{FB})(\gamma_a\rho_2 + \frac{\gamma[\theta DG + \theta_q CL]}{CDE} + \frac{\gamma_q L}{D})
\end{aligned}
$$

It can be noted, that corresponding to each value of λ^{**} in equation (13.6) we get a value for endemic equilibrium. Therefore, the number of endemic equilibria of the system (13.1) is equal to the number of positive roots of the equation (13.6). Clearly in equation (13.6), $a_0 > 0$ while $c_0 > 0$ when $R_0 < 1$ and $c_0 < 0$ when $R_0 > 1$. Using Descartes' rule of signs we have the following theorem on the existence of the endemic equilibrium.

Theorem 2 *The system (13.1) has:*

1. *exactly one unique endemic equilibrium, if* $c_0 < 0$ *(i.e.* $R_0 > 1$*).*
2. *exactly one unique endemic equilibrium, if* $b_0 < 0$*, and* $c_0 = 0$ *(i.e.* $R_0 = 1$*) or* ${b_0}^2 - 4a_0c_0 = 0$*.*
3. *exactly two endemic equilibria, if* $c_0 > 0$ *(i.e.* $R_0 < 1$*),* $b_0 < 0$ *and* ${b_0}^2 - 4a_0c_0 > 0$*.*

Remark It can be seen from the above theorem, making $R_0 < 1$ is not sufficient for controlling the disease. Therefore, some extra measures should be taken so that the diease can be controlled.

Local Stability of Endemic Equilibrium

Now we discuss the local stability of endemic equilibrium. Similar to the process followed in subsection (3.3) for the stability of disease-free equilibrium, we start by finding the jacobian matrix of system (13.1) at endemic equilibrium point, which can be obtained by substituing the value of E_1 from equation (13.5) into equation (3.3):

$$J(E_1) = \begin{bmatrix} -\mu - \beta_{**} & 0 & \frac{-\beta S^{**}}{(1+\alpha I^{**})^2} & -\beta S^{**} & 0 & 0 & \triangle \\ \beta_{**}(1-\omega) & -(\sigma+\mu) & \frac{\beta(1-\omega)S^{**}}{(1+\alpha I^{**})^2} & \beta(1-\omega)S^{**} & 0 & 0 & 0 \\ 0 & \rho_1\sigma & -(\theta+\mu+\delta) & \phi & 0 & 0 & 0 \\ 0 & \rho_2\sigma & 0 & -(\phi+\phi_q+\gamma_a+\mu) & 0 & 0 & 0 \\ 0 & \rho_3\sigma & 0 & \phi_q & -(\theta_q+\gamma_q+\mu+\delta) & 0 & 0 \\ 0 & 0 & \theta & 0 & \theta_q & -(\gamma+\mu+\delta) & 0 \\ 0 & 0 & 0 & \gamma_a & \gamma_q & \gamma & -(\triangle+\mu) \end{bmatrix}$$

where, $\beta_{**} = \beta(I_a^{**} + \frac{I^{**}}{1+\alpha I^{**}})$

To find the eigen values for the above jacobian matrix, we put:

$$|J(E_1) - \lambda I| = 0$$

which implies,

$$\begin{vmatrix} -(\mu+\lambda+\beta_{**}) & 0 & \frac{-\beta S^{**}}{(1+\alpha I^{**})^2} & -\beta S^{**} & 0 & 0 & \triangle \\ \beta_{**}(1-\omega) & -(\sigma+\mu+\lambda) & \frac{\beta(1-\omega)S^{**}}{(1+\alpha I^{**})^2} & \beta(1-\omega)S^{**} & 0 & 0 & 0 \\ 0 & \rho_1\sigma & -(\theta+\mu+\delta+\lambda) & \phi & 0 & 0 & 0 \\ 0 & \rho_2\sigma & 0 & -(\phi+\phi_q+\gamma_a+\mu+\lambda) & 0 & 0 & 0 \\ 0 & \rho_3\sigma & 0 & \phi_q & -(\theta_q+\gamma_q+\mu+\delta+\lambda) & 0 & 0 \\ 0 & 0 & \theta & 0 & \theta_q & -(\gamma+\mu+\delta+\lambda) & 0 \\ 0 & 0 & 0 & \gamma_a & \gamma_q & \gamma & -(\triangle+\mu+\lambda) \end{vmatrix} = 0 \quad (13.7)$$

where, $\beta_{**} = \beta(I_a^{**} + \frac{I^{**}}{1+\alpha I^{**}})$

Theorem 3 *The system (13.1) is locally asymtotically stable at the endemic equilibrium point if all the seven eigen values, obtained by solving the determinant in equation (13.7), have negative real parts.*

Sensitivity Analysis

Sensitivity analysis helps us to determine the affect on model results due to change in parameter values and the extent to which this change affects the model results. It plays a crucial role because if the sensitivity to model parameters is known, we can use this in real life to control the spread of the disease. For instance, if we know a certain parameter when decreased leads to the reduction in infectious classes, certain measures can be employed which reduces it's effect in the real life. In this section, we have used our model to analyse the COVID-19 epidemic. We have first calculated the Sensitivity Indices of R_0 with respect to various parameters and then have done the sensitivity analysis using a few parameters.

The first step is to assign the values to model parameters. For simplicity, we have assumed a few parameter values and picked the rest from the existing studies that fit best with our model. The list of parameter estimates and their sources are summarized in Table 13.3 below.

Now, we study the impact on R_0 due to changing paramaters. To do this we obtain the Sensitivity Index of R_0 with respect to different model parameters, which measures the change in R_0 in response to the change in a parameter.

The sensitivity index of R_0 with respect to any parameter Z is calculated as follows (refer to [11]):

Table 13.3 List of parameter values

Parameter	Value	Source	Sensitivity index
b	0.002	Assumed	1.0000
N	60,000	Assumed	1.0000
μ	0.0098	Assumed	−1.0726
δ	0.0175 day^{-1}	[2]	−0.0242
p	0	Assumed	NA
ρ_1	0.5210	[2]	0.0428
ρ_2	0.2740	[2]	0.5011
ρ_3	0.2050	[2]	NA
α	0.25	Assumed	NA
β	0.02	Assumed	1.0000
σ	0.219 day^{-1}	Assumed	0.0428
θ	0.2174 day^{-1}	[2]	−0.5778
θ_q	0.1429 day^{-1}	[2]	NA
γ	0.701 day^{-1}	Assumed	NA
γ_a	0.13978 day^{-1}	[2]	−0.2307
γ_q	0.11624 day^{-1}	[2]	NA
ϕ	0.139 day^{-1}	Assumed	−0.1061
ϕ_q	0.019 day^{-1}	Assumed	−0.0314
$\triangle$	0.001	Assumed	NA
ω	0.2	Assumed	-0.25

Source own

$$\varphi_{R_0}{}^{Z} = \frac{\partial R}{\partial Z}\frac{Z}{R_0}$$

Table 13.3 lists the sensitivity index of R_0 with respect to various parameters appearing in the formula for R_0, while Fig. 13.2 gives a pictorial representation of the same. The positivity or negativity of the index determines the relationship (direct or inverse) of R_0 with the parameter whereas its magnitude determines the strength of dependence of R_0 on the parameter. In Fig. 13.2, for every parameter with index extending towards the right, R_0 increases as the parameter increases while the one with index extending towards the left, R_0 decreases as it increases. Sensitivity Index of R_0 with respect to β is +1.0 (i.e. $\varphi_{R_0}{}^{\beta} = +1.000$), which means that a 1% increase in β results in a 1% increase in R_0. Similarly, $\varphi_{R_0}{}^{\theta} = -0.5778$, which implies that a 1% increase in θ will result in a 0.5778% decrease in R_0.

A pictorial representation of the COVID-19 compartmental model, fitted with parameter values is demonstrated in Fig. 13.3. Since, no vaccination has been brought into use yet we have set the proportion of vaccinated individuals (p) to zero. Also, since there is no concrete evidence that recovery ensures permanent immunity, therefore, we have allowed for the transition from recovered to susceptible state with a very

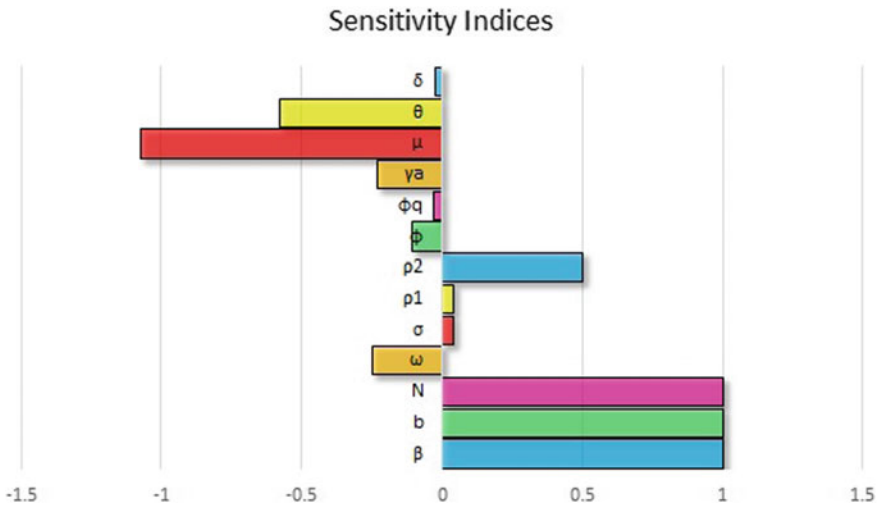


Fig. 13.2 Sensitivity Indices of R_0 corresponding to various parameters (*Source* own)

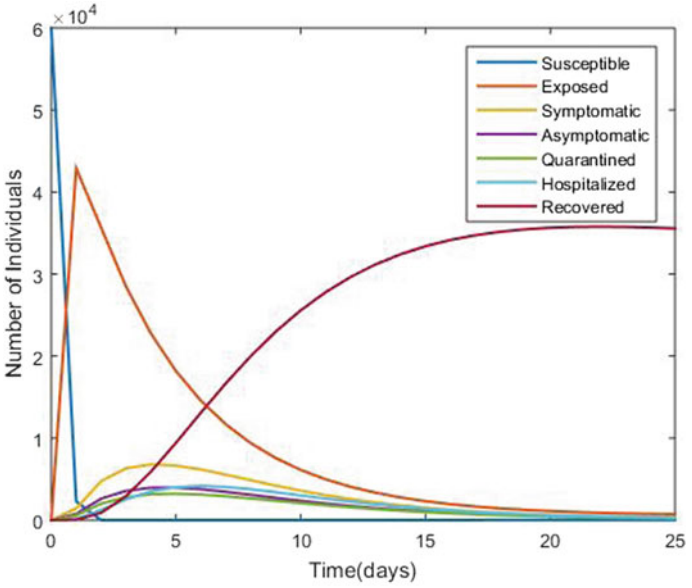
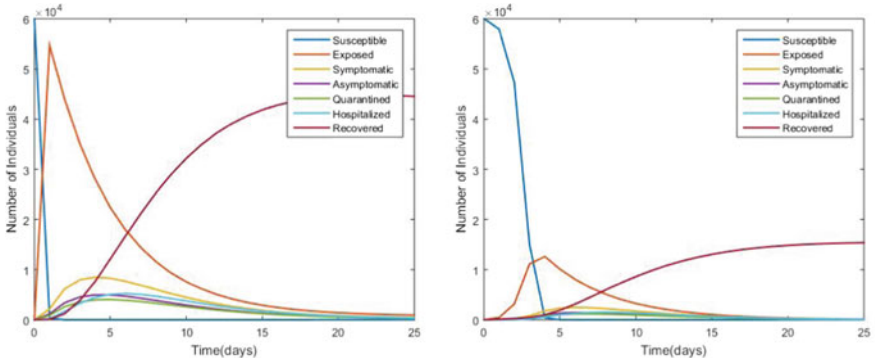


Fig. 13.3 The S-E-I-R-S compartmental model for COVID-19 epidemic (*Source* own)

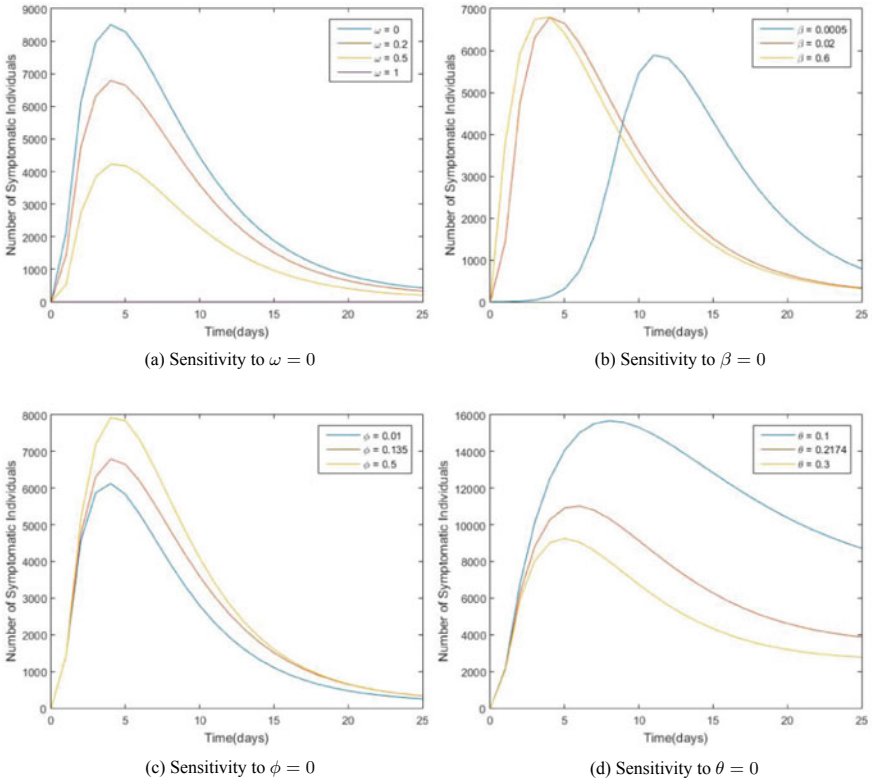


(a) Model without precautionary measures ($\omega = 0$) (b) Model with vaccination ($p = 0.72$)

Fig. 13.4 Effect of precautionary measures and vaccination (*Source* own)

small possibility. We have analysed the sensitivity of the model to the parameters ω and p, which captures the effect of precautionary measures on the spread of the disease and the proportion of vaccinated individuals, respectively. Figure 13.4a, b depicts the behaviour of the model in the absence of precautionary measures ($\omega = 0$) and in the presence of a vaccine ($p = 0.72$), respectively. It can be seen how in the absence of precautionary measures the peaks of infectious classes are higher. Similarly, in the presence of the vaccine the peaks of infectious classes are lower and it can be seen how the spread of the disease is controlled.

Next, we have analysed the impact of various parameters on the spread of the infection. Therefore, we have graphically studied the sensitivity of symptomatic class with respect to a few parameters [18].
Fig. 13.5a depicts how with increasing ω the number of symptomatic individuals decrease in the system, that is, as more and more precautions are taken the infection keeps on reducing. When $\omega = 1$ the curve lies on the x-axis depicting that in case strict precautionary measures are taken, like complete lockdown and social distancing, then there will be no infection in system. Figure 13.5b depicts how with increasing β, the number of symptomatics increase, that is, greater the rate of transmission more the infection. Figure 13.5c depicts how with increasing ϕ the count of symptomatics increase, that is, greater the rate at which asymptomatic individuals show symptoms and move to the symptomatic class more the number of symptomatic individuals. Figure 13.5d depicts how with increasing θ the count of symptomatics decrease, that is greater the rate at which symptomatic individuals are hospitalized lesser the number of susceptible individuals.
This depicts the importance of steps like lockdown and social distancing, along with precautionary measures like using face masks and washing hands frequently, as these steps can help in controlling the spread of the disease by leading to a lesser number of infected individuals in the system. Similarly, better the medical facilities and the treatment, lesser will be the spread of the disease. As more and more infected people



(a) Sensitivity to $\omega = 0$

(b) Sensitivity to $\beta = 0$

(c) Sensitivity to $\phi = 0$

(d) Sensitivity to $\theta = 0$

Fig. 13.5 Sensitivity of Symptomatic class to changing parameters (*Source* own)

will be treated, leading to lower number of infectives in the system, this will result in lower risk of getting exposed and hence control the spread of the disease.

Conclusion

We have defined a general *S*-*E*-*I*-*R*-*S* compartmental model for epidemic diseases and derived important mathematical results like the reproduction number, the diseased-free equilibrium and the endemic equilibrium. While the reproduction number deals with how rapidly the disease will spread, the two equilibriums, in real sense, corresponds to the levels at which the disease would completely be eradicated or atleast will remain at constant levels within the population. We have also derived conditions for the stability of the two equilibrium points, along with the conditions for the existence of the endemic equilibrium and have established that $R_0 < 1$ would imply local asymptotic stability of DFE.

Later, we have fitted the model with a suitable set of parameters to deduce predictions regarding the COVID-19 disease. Firstly, we have calculated the sensitivity indices of R_0 corresponding to various parameters. It can be seen from Fig. 13.2 how R_0 is highly dependent on parameters like β, thus, controlling the values of these parameters can help cope with the disease. Then, we have used the model to do sensitivity analysis for the COVID-19 epidemic disease. We studied the behaviour of symptomatic class in the presence of a few changing parameters. Then we have analyzed the impact of $\omega = 0$ and $p = 0.72$. It is clear that in the presence of a vaccine the spread of the disease can be minimized. We can see from Fig. 13.4b, in the presence of vaccination the infection decreases in the system. But vaccination as a mitigation strategy is not as simple as it sounds and is a time taking process. While many vaccinations are available now there are still a number of diseases for which vaccination has not been developed yet.

The availability of a vaccine alone do not bring down the list of challenges linked with it. One such challenge being the impossibility of vaccinating the entire population of a country. Ofcourse instead of targeting the vaccination of an entire population, epidemics deal with the concept of herd immunity, which can be estimated using the reproduction number (once determined). But even achieving herd immunity could take years in some cases due to the required vaccinated proportion falling almost around the whole of it. For instance, small pox which lasted for around 3000 years required an 80% vaccinated population in each country which was achieved over several years with an effort led by World Health Organization [13]. Similarly, measles lasted for years and required 96% vaccinated population [1].

The next challenge linked to vaccination is that even if a vaccine is available along with all the estimates regarding achieving the herd immunity, not all the countries can afford developing it or some might even face issues in purchasing it. In any case, coming up with a first and safe dose of a vaccine during any epidemic takes time and implementing it would take years.

Until a vaccine is made available, precautionary measures play an important role in eliminating the disease. As reported by WHO, the COVID-19 virus spreads due to the transfer of liquid droplets from an infected individual's mouth or nose while the person sneezes or coughs [12]. It can even stay on non-living bodies for varied amount of times. So, for instance, if a person sneezes and the infected droplets fall on a cereal packet in a super market, the next person touching the packet will be exposed to the disease if he/she touches his/her mouth, nose, or even eyes leading to the virus entering his/her body. Therefore, steps like social distancing, covering one's mouth and nose using a mask and washing one's hands frequently is playing a major role in combating with the disease. It can be noticed from Fig. 13.5a that while ω is set to 1 the infectious classes rest at zero, which means, in case of no contact at all the spread of disease would stop because the virus won't get new living bodies to cling onto and transfer any further. But, obviously the condition of no contact at all cannot be achieved, as even though lockdowns have been imposed all over the world to achieve this as much as possible, people still have to get out of their places for every day essentials and even for providing essential services. So, precautionary measures like lockdown cannot be treated as long term solution as it

drastically affects the economy. But precautionary measures do help in slowing down the process and help in reducing the spread of the disease until long term solutions are explored. Figure 13.4a clearly shows how in the absence of any precautionary measures the infectious trajectories attain high peaks than usual, thus, depicting their importance.

Thus, it can be argued that even though vaccination is a permanent and a long term solution for an epidemic but until a vaccination comes in to play precautionary measures can help a great deal in controlling the disease. Since, all these mitigation strategies and the extent to which they should be allowed for are derived from a close insight into the epidemic model, it can be said the dynamic analysis of an epidemic is the base for it's future elimination. Also, it is to be noted that once sufficient data related to the impact of vaccination is available, from countries like Russia, where vaccination is now available, the model developed in this paper can be used to do future predictions regarding the long term effects of vaccination in the fight against COVID-19.

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