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Epidemiological modeling for COVID-19 spread in India with the effect of testing

Anurag Singh *, Md Arquam
Department of Computer Science and Engineering, National Institute of Technology Delhi, New Delhi 110040, India

A R T I C L E   I N F O

Article history:
Received 5 April 2021
Received in revised form 5 September 2021
Available online 24 December 2021

Keywords:
COVID-19
Epidemic modeling
Basic reproduction number
Critical threshold
Complex network

A B S T R A C T

A novel coronavirus has resulted in an outbreak of viral pneumonia in China. Person-to-person transmission has been demonstrated, but, to our knowledge, the spreading of novel coronavirus takes place due to an asymptomatic carrier. Most models are not considering testing and underlying network topology that shows the spreading pattern. By failing to integrate testing into the epidemiological model, models missed a vital opportunity to better understand the role of asymptomatic infection in transmission. In this work, we propose a model considering testing as well as asymptomatic infection considering underlying network topology. We extract the transmission parameters from the data set of COVID-19 of India and apply those parameters in our proposed model. The simulation results support our theoretical derivations, which show the impact of testing and asymptomatic carrier in infection spreading.

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1. Introduction

Pandemics are not an uncommon phenomenon in this modern era. As in the past decade, we have seen many pandemics, such as H1N1, SARS, EBOLA, and because of COVID-19, humanity is currently facing its greatest crisis in 2020. It is possible to grasp the seriousness of these pandemics from the death toll claimed by them. The SARS outbreak of 2002–2004 was a severe acute respiratory syndrome (SARS) epidemic caused by the severe acute respiratory syndrome coronavirus (SARS-CoV or SARS-CoV-1). The outbreak began on November 16, 2002, in Foshan, Guangdong, China. Due to this, more than 8000 peoples are infected from different countries and territories, and approx 774 persons died due to it in the world. It survived up to about eight months. The pandemic H1N1/09 virus has resulted in 18,036 deaths, according to the WHO in 2019. In December 2019, the evolution of a new coronavirus strain, SARS-CoV-2, was discovered in Wuhan, Hubei Province, China, closely related to the one that causes SARS. It is the cause of the global COVID-19 pandemic currently underway. Coronavirus has infected 21,842,782 individuals and claimed 773,279 fatalities worldwide (as of August 17, 2020). Lockdown, social distancing, social hygiene, and masks are actively being used worldwide to counter this outbreak.

There are many lives influenced by the epidemic in a time. It spreads into a specific region from person to person. If epidemics start spreading to an entire country, or into the world, then it becomes Pandemic. There are various models proposed to understand the spreading of the diseases. Specifically, they rely under the category of agent-based modeling [1–3] and compartmental models [4–8]. The agent-based modeling simulates autonomous agents’ actions and interactions solely, e.g., individuals, organizations, or groups [9]. On the other hand, in the compartmental models’
whole population is divided into the various compartments, e.g., suspected (S), infected (I), and recovered (R) \[4\]. In the compartmental model, the rate equations are provided to mention the change in each compartments population over the time. There are other compartment models exist, e.g., SI \[10\], SIS \[11\], SIR \[4\], SIRS \[12\], SIR with mobility \[13,14\], etc. Agent-based model researchers often criticize the compartmental models are constantly criticized by the researchers who use agent-based modeling. They argue that these models struggle to capture the connectivity between different geographical locations, and real-world population properties are different, e.g., worldwide population distribution \[15,16\].

Now, modeling the dynamics of coronavirus disease 2019, COVID-19, remains a big challenge, although there has been a sporadic investigation in the past few months \[17\]. This work aims to develop a more realistic model for modeling the COVID-19 by using the SEIR model in response to the spread of the COVID-19 pandemic based on its specific epidemiological characteristics and dynamic migration.

In India, the first case of COVID-19 was found in Thrissur district of Kerala on January 30, 2020. The severe effect of COVID-19 was predicted in India as a poor rate of COVID-19 testing in the world \[18\]. Initially, the disease was spreading due to the travel from various countries to India, and those persons were traveling among the states and cities across India. The major cause of the disease was social contacts with the infected persons. Hence, India adopted a complete lockdown on March 23, 2020, after the announcement of the “Junta Curfew”, on March 22, 2020, to avoid the contacts to stop this outbreak \[19\]. During the lockdown, most social network contacts were broken to slow down the COVID-19 disease spreading. Some of the precautionary measures were adopted to make the underlying social network too sparse by maintaining social distance; schools, colleges, offices, and places of the mass gathering were closed. Any disease is spreading over the population, one person to another by any means of physical contact or, in the case of COVID-19, by maintaining a distance less than the social distance (approx. 6 ft). It helps to make a very sparse (less robust) network of the human contact network. Hence, it is important to make a mathematical model for COVID-19 spreading by considering the underlying human contact network. Some persons may have higher contacts into the network, like a person at the airport, a doctor or nurse, or a person visiting at higher gathering places. These persons have a higher degree in the human contact network. Therefore, they have a higher chance of getting infected. If they become infected, there will be a very high chance to spread the disease into the population at a very high speed. Therefore, it is very much important to break the links of the networks until the proper vaccination developed to stop it. Hence, various following measures are adopted \[20\].

- Hands should be washed often. Use soap and water or an alcohol-based hand rub to clean your hands.
- Keep a safe distance away from anyone coughing or sneezing.
- If physical separation is not possible, wear a mask.
- Keep your hands away from your eyes, nose, and mouth.
- When you cough or sneeze, cover your nose and mouth with your bent elbow or a tissue.
- When you are sick, stay at home.
- Seek medical help if you have a fever, cough, or trouble breathing.
- Get vaccinated

In the proposed work, a modified epidemiological model using classical SEIR model \[21\] is proposed for COVID 19 spreading by involving the new compartments for COVID 19 related terms Quarantine, lockdown, isolation, testing, symptomatic and asymptomatic infections. In this model, the basic reproduction number is calculated theoretically and compared with the COVID-19 data set for the Indian states. Similarly, the epidemic spreading threshold is derived and calculated. This model mainly highlights the structure of the underlying human social contact network study the spreading. In this work, the human contact network is considered as static. The proposed model also contributes to inform the effect of the testing of the population on the epidemic spreading and effect onto the other compartments.

The rest of the paper is organized as follows. The related researches are discussed for disease spreading models in Section 2, which promoted COVID 19 spreading. Further, the model is explained in the next section by introducing various parameters to model COVID-19 in Section 3. In the same section, the basic reproduction number and the critical epidemic threshold are derived. Section 4 presents the proposed model’s simulation results and its analysis. The same is validated with the real data-set of COVID-19 taken from India. Finally, the work is concluded, and future directions are discussed in Section 5.

2. State of art

In this section, we discuss relevant literature with respect to epidemic modeling, which involves two different lines of works, at the convergence of which our work lies. In the first segment, we explore the mathematical modeling of epidemic spreading and their modifications. In the second segment, we elaborate and discuss the epidemic spreading considering network connectivity, as we know that humans are more connected nowadays than ever before. This helps in understanding the spreading patterns of epidemics.
2.1. Mathematical modeling of epidemic spreading

Mathematical models play an important role in the understanding of epidemic spreading and in designing strategies to control the quick spread of infectious diseases without considering the antiviral or effective vaccine. Epidemic studies have a long and fascinating history [22–24]. It was further improved by Kermack & McKendrick, and Anderson & May [4,25].

The classical SIR model [4] is given as,

\[
\frac{dS(t)}{dt} = -\beta S(t)I(t)
\]
\[
\frac{dI(t)}{dt} = \beta S(t)I(t) - \mu I(t)
\]
\[
\frac{dR(t)}{dt} = \mu I(t)
\]

where \( S(t) \), \( I(t) \), \( R(t) \) denotes the proportion of the population that is susceptible, infected, and recovered at time \( t \) with spreading rate \( \beta \) and recovery rate \( \mu \).

There have been various works that investigated for the understanding of pandemic spreading. For instance, Shi et al. [26] proposed a model considering the propagation vector and examined that the propagation vector decreased the epidemic threshold but lead to the spreading. After that, many researchers work on delayed SIR model analytically without underlying network structure [27,28]. Now, mathematical modeling of infectious diseases is omnipresent, and a significant number of them can precisely portray the spread of epidemics. Various mathematical models have been proposed to study the spreading of the COVID-19. Chen et al. proposed a Bats–Hosts–Reservoir–People network model to investigate the dynamics of spreading of novel coronaviruses [29]. Lin et al. [30] modified the SEIR (susceptible-exposed-infected-removed) model to study the dynamics of COVID-19 by considering the public consciousness of risk and the number of cumulative cases. Khajanchi et al. [31] developed an extended SEIR model to visualize the spreading behavior of COVID-19 and carry out a short-term prophecy based on the data from India. Researchers studied the extended SEIR (susceptible–Exposed–Infectious–Removed) model to describe the spreading dynamics of COVID-19 outbreak in Wuhan, China, and computed the basic reproduction number \( R_0 \) by considering the data of Wuhan, China from December 31, 2019, to January 28, 2020 [32,33]. Vandenberg et al. [34] studied the role of diagnostic tests in spreading of during COVID-19. They described the technical problems that evolved during the implementation of testing of the pandemic. However, the classical epidemic model is not able to explain the spreading pattern due to the lack of the heterogeneity and topology of a underlying human network.

2.2. Epidemic spreading on network structure

The infection needs a physical connection to transmit disease from an infected person to the susceptible population. It may not have a physical contact in case of airborne diseases [14]. This led to the consideration of contact networks in epidemiological modeling, where a node represents an individual, while a link represents the contact between the individuals through which infections spread. There are small-world network [35] and Scale-free network [36] models which are used to study the real-world connectivity. In a small-world network, a node has a short distance in the non-locality of nodes in the network, which helps in the spreading of the disease in the network with high clustering coefficient [35]. In the scale-free networks, degree distribution follow power-law property as \( P(k) \propto k^{-\alpha} \) with degree \( k \), with power law exponent values of \( \alpha \) typically in the range \( 2.0 < \alpha < 3.0 \). Real-world networks are often scale-free [37]. In this work, we consider the heterogeneous contact network. Indeed, in a homogeneous contact network, each host has a similar number of connectivity, while in a realistic scenario the connectivity patterns of the hosts are generally quite different. Most empirical studies reported in the literature on the topology of real-world networks show that there is a small number of nodes that are highly connected, while the vast majority of nodes have few connections. This heterogeneous nature of the distribution of the number of contact is well approximated by a power-law distribution [38–41]. The degree distribution \( P(k) \) of nodes, which quantifies the probability of a randomly selected node having \( k \) connections, is an important property of real-world networks like contact networks. Real-world networks are often scale-free, i.e., they have a power-law degree distribution of the form \( P(k) \propto k^{-\alpha} \), with values ranging from 2.0 to 3.0 [42].

Researchers proposed various models considering underlying network structure to study the spreading of disease in human contact networks [43,44]. Moreno et al. [45] proposed an epidemiological framework considering a heterogeneous network to see the heterogeneity impact on epidemic spreading. Li et al. [46] studied the impact of small-world evolving networks on epidemic spreading by incorporating control strategies. Pastor-Satorras et al. [41] explained the dynamics of epidemic in the complex network by applying a degree-based mean-field approach. Gatto et al. presented a model to describe the COVID-19 spreading in Italy and explained the impact of emergency containment measures. The authors used the spatial network of 107 provinces of Italy, considering the mobility of humans. The network used by the author is based on community which includes local and imported infections due to contacts within the local community or associated with citizens’ mobility by considering the community-dependent force of infection. It is just like the transfer of flux from one location to another location where a location can be a community [47].
The untested population, $J$, is referred as isolation compartment. These individuals are not allowed to interact with other susceptible individuals. This compartment is referred as isolation compartment.

10. $R(k, t)$: the fraction of individuals who have been recovered from infection goes to recovered compartment.

The following are our assumptions about individual transmission from one compartment to another:

1. A healthy (or susceptible) individual after exposing to either symptomatic or asymptomatic infected individual(s) moves from susceptible to the exposed compartment. The spreading rate of the infection due to symptomatic and asymptomatic infected individuals are $\beta_s$ and $\beta_{as}$ respectively.

2. An exposed individual can either develop symptoms and goes to Testing with rate $\sigma_T$. After testing it is moved to infected compartment ($I_t$) with rate $\alpha$ after tested positive, or do not develop any symptoms and moves to quarantine compartment with rate $\sigma_Q$. Let $f$ proportion of population is tested and moved to tested compartment with the rate of $\sigma_T$ and $(1-f)$ proportion of population remains untested which causes further infection. From untested population, $r$ fraction of population is moved to asymptotic and $(1-r)$ population is moved to symptomatic compartment with the rate of $\kappa$.

3. A quarantine may show some symptom and can be tested with rate $\tau$ and move to test compartment.

4. A tested individual can either be tested positive with rate $\alpha$ and move to infected compartment and further to isolation compartment with rate $\kappa$. 

3. Model preliminaries and derivations

The proposed model is explained in this section for understanding the spread of COVID-19 infection. In the proposed model, whole human population is divided into ten compartments: Susceptible ($S$), Exposed ($E$), Quarantine ($Q$), Test ($T$), Untested ($UT$), Infected symptomatic ($I_s(k, t)$), Infected untested symptomatic ($I_{us}(k, t)$), Infected asymptomatic ($I_{as}(k, t)$), Isolation ($J(k, t)$) and Recovered ($R(k, t)$). Individuals may switch between states in the direction indicated by the arrow, as well as the direction of transmission.

Fig. 1. A Block diagram of the proposed Model is created, in which the host population is split into 10 states: Susceptible ($S(k, t)$), Exposed ($E(k, t)$), Quarantine ($Q(k, t)$), Tested ($T(k, t)$), Untested ($UT(k, t)$), Infectious (symptomatic) ($I_s(k, t)$), Infectious (untested asymptomatic) ($I_{us}(k, t)$), Infectious (asymptomatic) ($I_{as}(k, t)$), Isolation ($J(k, t)$) and Recovered ($R(k, t)$). Individuals may switch between states in the direction indicated by the arrow, as well as the direction of transmission.
5. An individual can recover spontaneously at any time with the recovery rates \( \mu_Q, \mu_I, \mu_{as} \) and \( \mu_{as} \) from compartment quarantine, isolation, untested symptomatic infected and asymptomatic infected respectively. The recovery of an individual is independent of any other compartments’ individuals.

6. Once the individual gets recovered, it is assumed that it will become immune to the disease and thus, will not transmit the infection to individuals to the susceptible population.

7. Furthermore, the demography, or the birth or death of individuals, is not taken into account in this model. To put it another way, the population remains constant in the process.

The following mean-field equations is defined for the dynamics of the pandemic, considering the discussed compartments, interactions and parameters,

\[
\begin{align*}
\frac{dS(k, t)}{dt} &= -\left( \beta_s \Theta_s(t) + \beta_{as} \Theta_{as}(t) \right) kS(k, t) \quad (4) \\
\frac{dE(k, t)}{dt} &= \left( \beta_s \Theta_s(t) + \beta_{as} \Theta_{as}(t) \right) kS(k, t) - \sigma_Q E(k, t) - f \sigma_I E(k, t) \\
\frac{dQ(k, t)}{dt} &= \sigma_Q E(k, t) - \mu_Q Q(k, t) - \tau Q(k, t) \quad (5) \\
\frac{dT(k, t)}{dt} &= f \sigma_I E(k, t) + \tau Q(k, t) - \alpha T(k, t) \quad (6) \\
\frac{dUT(k, t)}{dt} &= (1 - f) \sigma_I E(k, t) - (1 - r) \sigma_I UT(k, t) - r \sigma_I UT(k, t) \quad (7) \\
\frac{dl_s(k, t)}{dt} &= \alpha T(k, t) - \kappa l_s(k, t) \quad (8) \\
\frac{dl_{as}(k, t)}{dt} &= (1 - r) \sigma_I UT(k, t) - \mu_{as} l_{as}(k, t) \quad (9) \\
\frac{dl_{as}(k, t)}{dt} &= r \sigma_I UT(k, t) - \mu_{as} l_{as}(k, t) \quad (10) \\
\frac{dj(k, t)}{dt} &= \kappa l_s(k, t) - \mu_j j(k, t) \quad (11) \\
\frac{dR(k, t)}{dt} &= \mu_Q Q(k, t) + \mu_I I(k, t) + \mu_{as} l_{as}(k, t) + \mu_{as} l_{as}(k, t) \quad (13)
\end{align*}
\]

where,

\[
\begin{align*}
\Theta_s(t) &= \sum_{k' = 1}^{k} \Psi(k') P \left( \frac{k'}{k} \right) l_s(k', t) \quad (14) \\
\Theta_{as}(t) &= \sum_{k' = 1}^{k} \Psi(k') P \left( \frac{k'}{k} \right) l_{as}(k', t) \quad (15) \\
P \left( \frac{k'}{k} \right) &= \frac{k' P(k')}{\langle k \rangle} \quad (16)
\end{align*}
\]

Infectivity \( (\Psi(k')) \) may be considered as 1. Please refer Table 1 for notations and their meaning.

3.1. Dynamical behavior of the model

The mean-field Eq. (4)–(13) represents nonlinear dynamical system of pandemic spreading at any time \( t \). The sum of the fraction of all individuals is one,

\[
S(t) + E(t) + Q(t) + I_s(t) + I_{as}(t) + T(t) + J(t) + R(t) = 1 \quad (17)
\]

We can represent \( X(t) = \sum_k X(k, t) \); \( X \in \{S, E, Q, I_s, I_{as}, T, J, R\} \) is the total population of the respective compartment at time \( t \) of any degree, \( k \)

Infection spreading occurs when the rate of infected individuals increases, that is,

\[
\frac{dl(t)}{dt} > 0
\]

Where, \( I(t) = I_s(t) + I_{as}(t) + I_{as}(t) \). Therefore, by using Eqs. (9) and (11),

\[
\frac{dl(t)}{dt} > 0
\]
If we do not consider the quarantine and isolation compartment then parameter of these compartment then it give the

\[
\frac{dl_s(t)}{dt} + \frac{dl_{as}(t)}{dt} + \frac{dl_{is}(t)}{dt} > 0
\]

\[
\alpha T(k, t) - \kappa l_s(k, t) + (1 - r)\sigma_T UT(k, t) - \mu_s I_s(k, t) + r \sigma_T UT(k, t) - \mu_{as} I_{as}(k, t) > 0
\]

\[
\alpha T(k, t) - \kappa l_s(k, t) + \sigma_T UT(k, t) - \mu_s I_s(k, t) - \mu_{as} I_{as}(k, t) > 0
\]

\[
\left(\frac{\beta_s + \beta_{as}}{\sigma_T}\right)^{\frac{k^2}{\kappa}} > 1
\] (19)

The effective reproduction number \( R_c \) is derived considering test, quarantine and isolated compartments. The effective reproduction number \( R_c \) is also a time-dependent which continues to track the expected number of secondary infections caused by each infectious as the epidemic continues with control measures considering test, quarantine and isolation in place. Therefore, \( R_c = R_c * S(t) \) allows time-dependent parameter.

\( R_0 \), or the basic reproductive number, is traditionally defined as the mean number of secondary cases that arise from a primary infection in a completely susceptible population [48,49]. In a deterministic model, if \( R_0 < 1 \) an epidemic cannot develop from a small influx of SARS-infected individuals but an epidemic will develop if \( R_0 > 1 \). By contrast, an epidemic is not guaranteed in a stochastic model if \( R_0 > 1 \), but the probability of an epidemic increases with \( R_0 \).

It is considered that the basic reproduction number, \( R_0 \), is defined in the absence of control measures and introduce the control reproduction number, \( R_c \), to denote the reproduction number when control measures are in place. \( R_c \) is derived in the same way as \( R_0 \) is derived but using the full model with test, quarantined and isolated classes. There is also a time-dependent effective reproduction number \( R_c \) which continues to track the expected number of secondary infections caused by each infectious as the epidemic continues with control measures (test, quarantine and isolation) in place. In practice, control measures are implemented quickly and the number of infected individuals is small relative to the total population size, \( N \). This implies \( S(t) \) is approximately one and \( R_c \) is simply \( R_c \) with possibly time varying parameters. Thus, \( R_0 \) determines whether there will be an outbreak, and \( R_c \) determines whether the control measures introduced when an outbreak is recognized will suffice to turn matters around right away. Left side of Eq. (19) is represented as effective reproduction number \( (R_c) \) at \( t \rightarrow 0 \) \( S(t) = 1 \), hence, \( R_c \) can be defined as

\[
R_c = \frac{(\sigma_T)(\beta_s + \beta_{as})^{\frac{k^2}{\kappa}}}{(\sigma_Q + \sigma_T)(\mu_s + \kappa + \mu_{as})}
\] (20)

where, \( R_c \) is called effective reproduction number which determines the spread of infection with control parameters.
Table 2
COVID-19 Data description of India and 7 different states of India from March 01, 2020 to Feb 01, 2021.

| India & Other States | Confirmed Cases | Active Cases | Recovered |
|----------------------|-----------------|--------------|-----------|
| India                | 1,08,69,628     | 1,39,651     | 1,05,70,032 |
| Andhra Pradesh       | 8,88,555        | 917          | 8,80,478   |
| Delhi                | 6,36,387        | 1,046        | 6,24,457   |
| Gujarat              | 2,64,165        | 1,800        | 2,57,968   |
| Haryana              | 2,68,677        | 824          | 2,64,820   |
| Jammu & Kashmir      | 1,25,052        | 605          | 1,22,502   |
| Uttar Pradesh        | 6,01,562        | 3,306        | 5,89,565   |
| Rajasthan            | 3,18,491        | 1,450        | 3,14,264   |

The basic reproduction number $R_0$ as

$$R_0 = \frac{(\sigma_T)(\beta_s + \beta_{as})}{(\sigma_T)(\mu_s + \mu_{as})}$$

$$R_0 = \frac{(\beta_s + \beta_{as})}{(\mu_s + \mu_{as})}$$  \hspace{1cm} (21)

where, $R_0$ is called basic reproduction number which determines the spread of infection. One can see the derivation of $R_0$, it looks like ratio of spreading rate and recovery rate coupled with network parameter. When $R_0 > 1$, the propagation occurs at a fast rate. When $R_0 = 1$, the propagation happens at a slow rate. When $R_0 < 1$, the propagation finishes.

3.2. Derivation of critical threshold of epidemic spreading

In this work, total population is considered as constant, therefore, \(\sum_{k}\{S(k, t) + E(k, t) + Q(k, t) + I_s(k, t) + I_{as}(k, t) + T(k, t) + J(k, t) + R(k, t)\} = 1\). Infection spread in susceptible population and infected population is recovered, therefore, we can say recovered population are those susceptible population who is infected. So, as the epidemic develops, the number of susceptible declines and the number of recovered increases. Considering Eq. (4) and (13),

$$\frac{dS(k, t)}{dt} = -\frac{(\beta_s \Theta_s(t) + \beta_{as} \Theta_{as}(t))kS(k, t)}{\mu Q(k, t) + \mu_j I_s(k, t) + \mu_{as} I_{as}(k, t) + \mu_{as} I_{as}(k, t)}$$

$$\beta > \frac{\langle k \rangle (\mu_j + \mu_s + \mu_{as})}{\langle k^2 \rangle}$$  \hspace{1cm} (22)

Therefore, the critical spreading rate under heterogeneous network can be defined as,

$$\{ (\beta_s + \beta_{as}) = \beta_c > \frac{\langle k \rangle (\mu_j + \mu_s + \mu_{as})}{\langle k^2 \rangle} \}$$  \hspace{1cm} (23)

If we consider all transmission parameter as constant then critical threshold $\beta_c$ will depend on network parameter as \(\frac{\langle k \rangle}{\langle k^2 \rangle}\), which is similar to [44].

4. Results and analysis

In this section, first plotted the basic reproduction number of India and other Indian states are plotted day wise from March 01, 2020 to February 01, 2021, to infer the spreading rate of COVID-19 spreading. Further, the experimental setup are described and simulations results are demonstrated using the proposed model on a synthetic network generated through a configuration model to make it a real-world network with the power-law distribution.

4.1. Evaluation of $R_t$ value for India and Indian states

In this section, COVID-19 data is taken from https://www.covid19india.org/ [50] from March 01, 2020 to Feb 01, 2021 to find the trend of infection spreading in India and listed in Table 2. We have extracted the confirmed cases of infection, active cases of infection, and recovered cases for India and seven other states till February 01, 2021. Confirmed infection cases are total infection occurred till February 01, 2021, while active cases are infectious and may spread infection. Recovered are those who are recovered from infection.

The basic reproduction number is calculated using a method proposed by Bettencourt et al. [51]. The author used the Bayesian method to estimate the $R_t$. They consider all the new cases reported daily. The number of new cases gives an
Table 3
Network statistics of synthetic network. It is sparse due to the removal of parallel edges and self-loops. The average degree and density is low for the generated network.

| Network Properties | Network Structure 1 | Network Structure 2 | Network Structure 3 | Delhi |
|--------------------|---------------------|---------------------|---------------------|-------|
| Nodes              | 10,000              | 15000               | 20000               |       |
| Edges              | 9,960               | 14950               | 19972               |       |
| Average degree     | 1.992               | 2.99                | 3.994               |       |
| Edge density       | 0.0002              | 0.00013             | 9.98e−05            |       |
| Number of triangles| 390                 | 384                 | 168                 |       |
| Average clustering coefficient | 0.0038          | 0.00199            | 0.00096             |       |
| Number of components| 1117              | 1664                | 2292                |       |
| Power-law exponent | 3                   | 3                   | 3 [57]              |       |

idea about the current value of \( R_t \). Therefore, today’s value of \( R_t \) is related to yesterday’s value of \( R_t \). Based on these assumptions, the authors used the Bayes’ Theorem as follows,

\[
P(R_t | x) = \frac{P(x | R_t) P(R_t)}{P(x)}
\]

Where \( x \) is the new cases, so authors considered that the distribution of \( R_t \) is dependent on the likelihood of coming \( x \) new cases given \( R_t \) times, the prior of the value of \( p(R_t) \) without the data and divided by the probability of seeing new cases. This is considered for any particular day. When it is considered for a brief period of time: For each day authors used previous day prior \( P(R_{t-1}) \) to estimate current day prior \( P(R_t) \). Prior is previous cases. The author considered the distribution of \( R_t \) to be a Gaussian centered around \( R_{t-1} \). Initially, (on the very first day, where no previous day data available), the probability of \( R_t \) will be,

\[
P(R_{t1} | x_1) \propto P(R_{t1}) \cdot L(R_{t1} | x_1)
\]

Further on next day,

\[
P(R_{t2} | x_1, x_2) \propto P(R_{t2}) \cdot L(R_{t2} | x_2) = \sum_{R_{t1}} P(R_{t1} | x_1) \cdot P(R_{t2} | R_{t1}) \cdot L(R_{t2} | x_2)
\]

Where \( L \) is the likely-hood function. A likelihood function describes how likely \( x \) new cases are coming for a given a value of \( R_t \).

The \( R_t \) of India and some other Indian states are estimated and plotted in Fig. 2 by using method of Bettencourt et al. [51]. To plot the \( R_t \), data of infected cases are taken from March 01, 2020 to Feb 01,2021. The \( R_t \) is defined as ratio of spreading rate (\( \beta_s \)) and recovery rate (\( \mu_s \)). An infected person recovers and returns to normal health after seven days of tested positive as per survey of 2020 conducted by Centers for Disease Control and Prevention (CDC) [52,53]. Therefore, the value of \( \mu_s \) will be \( \frac{7}{3} = 0.143 \). By using the value of \( R_t \) and \( \mu_s, \beta_s \) can be calculated. The \( R_t \) of Indian states as well as India is plotted to show the spreading pattern of infection in India and Indian states. Our aim is to show the how infection is spread in India.

4.2. Experimental setup

For the simulation setup and analysis, A synthetic network is created using configuration model to represent real human contact network [54], which follows the power-law distribution (see Fig. 3). The fact that human activity patterns are non-Poisson argues that the observed bursty character represents some fundamental and potentially generic property of human dynamics [55,56]. The random_powerlaw_tree_sequence and configuration_model function of networkx are used here [57]. The configuration model produces a random pseudograph (graph with no parallel edges and self-loops) by randomly assigning edges to fit the given degree sequence. Table 3 provides the statistics of the synthetic network. Based on the configuration model definition and various properties of the synthetic network, this network reflects the real-world contact network [58].

The aim of the simulations is to answer the following questions,

- How the rate \( \beta_s \) affects the \( X(t) \)?
- How the rate \( \sigma_t \) affects the \( X(t) \)?

4.3. Calculation of transmission rates for epidemic process

To calculate the various transmission parameters to move individuals from one compartment to other compartments, ratio is chosen arbitrary.
Fig. 2. $R_t$ of India and Other Indian States are plotted which inform about the secondary infection where, $R_t = \frac{\beta}{\mu_s}$. The value of $R_t$ lie between 3.0 and 0.6 for India (a). For Andhra Pradesh, $R_t$ lie between 3.2 and 0.4 (b). $R_t$ lie between 4.7 and 0.56 for Delhi (c). $R_t$ lie between 3.4 and 0.5 for Gujarat (d). In Haryana $R_t$ lie between 3.06 and 0.59 (e). $R_t$ lie between 3.0 and 0.26 in Jammu & Kashmir (f). $R_t$ lie between 3.0 and 0.4 for Uttar Pradesh (g), $R_t$ lie between 3.2 and 0.46 for Rajasthan (h).

1. **S to E ($\beta_s$, $\beta_{as}$):** Two compartments $I_s$ and $I_{as}$ i.e., Infected symptomatic and Infected asymptomatic respectively are responsible for the transition from S to E. According to WHO report,\(^1\) $I_{as}$ transmit major infection of COVID-19 among population. An infected person recovers and returns to normal health after seven days of tested positive as per survey of 2020 conducted by Centers for Disease Control and Prevention (CDC)\(^{[53]}\). The average value of $R_t = 1.6$ and $\mu_s = 1/7 = 0.143$.

- $S$ to $E$ because of $I_s$: $\beta_s = \frac{1.6}{0.143} = 0.23$, i.e., transmission rate of population from susceptible to symptomatic infection compartment.
- $S$ to $E$ because of $I_{as}$ is unknown, so its impact is analyzed by keeping it the same as $\beta_s$ i.e., 0.23.

\(^{1}\) [Website Link](http://www.emro.who.int/health-topics/corona-virus/transmission-of-covid-19-by-asymptomatic-cases.html)
2. $E$ to $UT$, $T$ and $Q$ ($\sigma_Q$ and $\sigma_T$): When an individual get infected and start to spread it further, then transmission starts from $E$ to others states.

- The value of $\sigma_T$ and $\sigma_Q$ are taken from literature [59–62].
- A per report published by ICMR, 20% of population is tested [61] and 80% of population is untested. Therefore, the value of $f$ is 0.2.
- So transition from $E$ to other states after 3 days. It means that population moves from $E$ to other states with rate of $\frac{1}{3}$.
- Hence, rates are: (1) $E$ to $UT$ and (2) $E$ to $T$: $\sigma_T = 0.215$ [61], and (3) $E$ to $Q$ : $\sigma_Q = 0.33511$ [62].

3. $UT$ to $l_{as}$ and $l_{is}$ ($\sigma_I$): When an individual is undetected get infected and start to spread it further, then transmission starts from $E$ to others states.

- According to WHO report [60], "According to current COVID-19 results, 80% of infections are mild or asymptomatic, 15% are serious infections requiring oxygen, and 5% are critical infections requiring ventilation". Therefore, the value of $r$ is 0.8.
- The value of $\sigma_I$ is taken from the literature [59,60].
- The ratio of asymptomatic infection is taken as 80% and symptomatic infection as 20% symptomatic infection from $UT$ to $l_{as}$ and $l_{is}$.
- Hence, rates are: (1) $UT$ to $l_{as}$ and (2) $UT$ to $l_{is}$: $\sigma_I = 0.333$ [59,60].

4. $T$ to $l_s$ ($\alpha$): An individual goes for a test after developing symptoms. According to [63,64], After 5 days, an infected person begins to show symptoms. As it is covered three days in $E$ earlier, it is required to add two days before the test, i.e., $\frac{1}{2}$. Also, this rate is not available; we are considering this rate also as a variable between 0 to 100%. So, the final rate $\alpha$ will be in range $[0^{\frac{1}{2}}, 1^{\frac{1}{2}}]$. Note: We also limit one test/person.

5. $l_s$ to $J$ ($\kappa$): This transition depends upon test efficiency, which is directly influenced by the time period of test after exposing to the disease. It is assumed that once a person is detected positive, he must be sent for isolation. Therefore, the value of $\kappa$ is taken as 1.

6. $l_{as}$ to $R$ ($\mu_{as}$):

- According to WHO report [60], "According to current COVID-19 results, 80% of infections are mild or asymptomatic, 15% are serious infections requiring oxygen, and 5% are critical infections requiring ventilation".
- Also according to WHO report [65], patients with moderate cases recover in around two weeks, while those with serious or critical disease recover in 3–6 weeks.
- Rate $l_{as}$ to $R$: $\mu_{as} = 0.05^{\frac{1}{39}} + 0.15^{\frac{1}{25}} + 0.8^{\frac{1}{11}} = 0.08$.

7. $J$ to $R$ ($\mu_J$): Isolated population cannot support in spreading of disease, So that the value of $\mu_J$ is considered as 1.

8. $Q$ to $T$ and $R$ ($\tau$, $\mu_Q$):

- As per report of [66], quarantined population will be released after 14 days, but as it is already covered 3 days in $E$ state earlier. Therefore, rate of transfer of population from $Q$ to other states will be $1/11 = 0.091$.

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2. https://www.who.int/docs/default-source/coronaviruse/situation-reports/20200306-sitrep-46-covid-19.pdf
3. https://www.who.int/docs/default-source/coronaviruse/situation-reports/20200306-sitrep-46-covid-19.pdf
4. https://www.who.int/docs/default-source/coronaviruse/who-china-joint-mission-on-covid-19-final-report.pdf
Fig. 4. Epidemic spreading pattern of proposed model considering synthetic contact network as underlying topology. The degree distribution of synthetic contact network follows the power law. The transmission rate values are $\beta_s = \beta_{as} = 0.23$, $\sigma_t = 0.333$, $\sigma_I = 0.215$, $\sigma_Q = 0.33511$, $\mu_{as} = 0.08$, $\mu_{Q} = 0.07272$, $\mu_{I} = 1$, $\tau = 0.018$, $\kappa = 1$, $f = 0.2$, $r = 0.8$ and $\alpha = 0.5$.

- The ratio of compartments are kept as $80\%$ ($R$) and $20\%$ ($T$) [67]
- Therefore rates are: $Q$ to $T$: $\tau = 0.2*1/11 = 0.018$, and $Q$ to $R$: $\mu_{Q} = 0.8*1/11 = 0.07272$

4.4. Results

We start by analyzing the dynamic behavior of the proposed model’s epidemic spreading according to the real data set, considering the real data set’s transmission parameters.

The simulations are performed considering various transmission parameters in order to assess the impact of spreading. Epidemic spreading in the human population increases with the increment in time at a certain point. After that, it starts decreasing and finally vanishes. The susceptible population decreases to 0.1 at 100-time steps and decreases slowly as compared to the initial trend (Fig. 4(a)). The exposed population are those that are in close contact with infectious agents.
The infection spread only to those population that is exposed. As time passes, people start exposing to an infectious agent, and it reaches the maximum as 0.04 at 63-time steps (Fig. 4(b)). The certain exposed population is quarantined till symptom arises. If a symptom arises, it is tested and moved to the tested symptomatic infection compartment, and after that, it is sent to isolation (Fig. 4(c)). In this course, the quarantined population has reached the peak of 0.125 in 72-time steps. In the meantime, the testing process is also started. The testing process is done when the exposed population showing symptom of infection (Fig. 4(d)).

When the testing process is started, some infectious agent is detected while the infection is also increased due to the exposed population which is untested or undetected. Untested Asymptomatic infection and Untested Symptomatic infection plays a major role in the infection spreading as compared to tested symptomatic infection. Due to testing, a number of symptomatic infection is checked and isolated. Therefore, the tested symptomatic infection does not spread the infection to a large extent as compared to untested asymptomatic and symptomatic infection. This is because the untested asymptomatic and symptomatic is directly dependent on the exposed population, while the tested symptomatic infection is checked and isolated (Fig. 4(e), (f), (g) & (h)). The peak of tested symptomatic infection, untested symptomatic infection and untested asymptomatic infection reaches to 0.0017, 0.056 and 0.0089 in 65, 75, 72 time steps. All tested symptomatic infection is sent to isolation, while the isolated population reaches the maximum value of 0.00165 in 66-time steps (Fig. 4(i)). Recovery starts when an infected individual recovers from the infection. The total recovered from infection at 100-time steps are 0.71 and still going on (Fig. 4(j)).

SEPIA model proposed by Gatto et al. [47] is compared with the proposed model. In SEPIA model, they are not considering any test compartment. From exposed compartment, every individual is considered presymptomatic after that with some rate it is moved to symptomatic and asymptomatic. Same set of parameters are used to simulate the SEPIA model to make comparison with the proposed model. Spread of an epidemic in the human population increases with some rate it is moved to symptomatic and asymptomatic. (Fig. 4(a)) shows that the susceptible population decreases to 0 at 30 time steps and decreases quickly, while in proposed model, it decreases to 0.1 at 100-time steps and decreases slowly as compared to SEPIA model. During exposure to an infectious agent, people reach the maximum level as 0.325 in 4-time steps, while it reaches the maximum as 0.04 at 63-time steps in proposed model (Fig. 5(b)).

Presymptomatic infection is converted into symptomatic infection till symptom arises (Fig. 5(c)). Once symptom arises, it is transferred to Infected symptomatic compartment, otherwise in asymptomatic compartment (Fig. 5(d)). Asymptomatic population is reached to peak of 0.225 at 18 time steps while 0.056 at 75 time steps in proposed model. There are two types of symptomatic compartments as tested and untested. Tested Symptomatic population is reached to peak of 0.00165 in 66-time steps and untested symptomatic reaches to the peak of 0.0089 in 72 time steps in proposed model while peak is 0.07 in SEPIA model at 10 time steps (Fig. 5(e)). From symptomatic compartment population is transferred into quarantine, hospital (isolation), recovered and death compartment. Some population of symptomatic infection is transferred into hospital. Isolation compartment reaches to the peak of 0.00123 in 11 time steps, while 0.00165 in 66-time steps in proposed model (Fig. 5(f)). While quarantine population reaches to the peak of 0.013 in 19 time steps in SEPIA model while 0.125 at 72 time steps (Fig. 5(g)). Maximum recovery of population is 0.86 in SEPIA model while in proposed model recovered population is 0.85 and it become constant (Fig. 5(h)).

\( R_t \) of real data of India is plotted and corresponding \( R_t \) of the proposed model in Fig. 6. To plot \( R_t \) of the proposed model, transmission parameter is taken from Table 4. This plot shows the same pattern of the curve as real data. In the proposed model, the value of \( R_t \) starts with 3.7, and after 100-time steps, it reaches to 0.4. In real data of India, it started with 3 in March 2020 and decreases gradually to near about 1 in February 2021.

To see the infection spreading pattern, the daily infection (\( I(t) \)) of real data of India and corresponding infection on each time steps (\( I(t), I_{wu}(t) \) & \( I_{wu}(t) \)) of the proposed model are plotted in Fig. 7. The total population is taken as 10000 in our proposed model. In proposed model, \( I(t), I_{wu}(t) \) & \( I_{wu}(t) \) follow the same trend of infection like real data. \( I_{wu}(t) \) & \( I_{wu}(t) \)

### Table 4

| Parameter | Description                          | Value   | Source |
|-----------|--------------------------------------|---------|--------|
| \( \beta_s \) | Spreading rate of symptomatic infected individuals | 0.23    | [50]   |
| \( \beta_w \) | Spreading rate of symptomatic infected individuals | 0.23    | [50]   |
| \( \sigma_l \) | Rate of exposed individual develop symptoms | 0.333   | [59,60]|
| \( \tau \) | Fraction of infection to be asymptomatic | 0.8     | [60]   |
| \( \sigma_q \) | Rate of individuals being tested | 0.215   | [61]   |
| \( f \) | Fraction of exposed population tested | 0.2     | [61]   |
| \( \sigma_q \) | Rate of individuals get quarantine | 0.33511 | [62]   |
| \( \tau_q \) | Rate of quarantined population being tested after showing symptom | 0.018   | [67]   |
| \( \alpha \) | Rate of tested population transferred to Infection(symptomatic) | 0.5     | [63,64]|
| \( \kappa \) | Rate of transfer of Infected population to Isolation | 1       | Assume |
| \( \mu_{wu} \) | Recovery rate of asymptomatic infected individuals | 0.08    | [60,65]|
| \( \mu_q \) | Recovery rate of quarantine individuals | 0.07272 | [67]   |
| \( \mu_{wu} \) | Recovery rate of symptomatic infected individuals | 0.143   | [52,53]|
| \( \mu_I \) | Recovery rate of isolated individuals | 1       | Assume |
Fig. 5. Epidemic spreading pattern of SEPIA model proposed by Gatto et al. and proposed model considering synthetic contact network as underlying topology. The degree distribution of synthetic contact network follows the power law. The transmission rate values are $\beta_s = 0.23$, $\sigma_I = 0.333$, $\sigma_T = 0.335$, $\sigma_Q = 0.215$, $\sigma_I = 0.08$, $\mu_as = 0.143$, $\mu_T = 0.07272$, $\mu_Q = 1$, $\tau = 0.018$, $k = 1$, $f = 0.2$, $\tau = 0.8$ and $\alpha = 0.5$.

plays a major role in the infection spreading in the population, while $I_s(t)$ is a known and tested infection. That is why $I_s(t)$ & $I_a(t)$ exists for a longer time. The value of $I_s(t)$ reaches to peak of 0.0017 fraction of population at 65-time steps and decreases to 0.0002 fraction of population at 100-time steps, while the value of $I_a(t)$ and $I_u(t)$ reaches to peak of 0.056 and 0.0089 fraction of population at 75 and 72-time steps and decreases to 0.029 and 0.003 at 100-time steps respectively. In real data, the infection reaches to peak of 97,859 on the day of 16 September 2020. Now daily infection is reduced to 8947 on the day of 8 February 2021. The curve of real data and synthetic data follow the same pattern.

The cumulative recovery ($R$) of real data of India and corresponding cumulative recovery on each time step ($R(k, t)$) of the proposed model are plotted in Fig. 8. In the proposed model, cumulative recovery follows the same trend of recovery as real data. In real data, recovery starts after June 2020 and reaches 10,559,604 in February 2021. In synthetic data, recovery starts after 19-time steps and reaches 0.71 fraction of population at 100-time steps.

The infection spreading vs. spreading rate is plotted in Fig. 9. When the spreading rate reaches 0.08, then epidemic spreading starts, and after 0.12, it increases fast. The value of $\beta_c$ is varied from 0 to 0.24 to see the critical threshold’s impact. This critical threshold is responsible for the outbreak of epidemic. Below this threshold value, epidemic will not spread. At $\beta_c = 0.24$, the infection reaches to 0.08 of total population. As per data, Indian population is 1,390,160,589. Total infected population will be $1,390,160,589 \times 0.08 = 111,212,847$, which is near about to 1,077,820,9 real infection on February 1, 2021.
Fig. 6. (a) $R_t$ of COVID-19 of India from March 01, 2020, to Feb 01, 2021. (b) $R_t$ of the proposed model considering synthetic network of three value of number of nodes as underlying topology by applying transmission parameters from Table 4, which shows a similar trend of the curve of COVID-19 of India.

Fig. 7. (a) Infection spreading on daily basis of COVID-19 of India from March 01, 2020 to Feb 01, 2021. (b) Infection spreading on daily basis of proposed model considering synthetic contact network as underlying topology. The degree distribution of synthetic contact network follows the power law. The transmission rate values are $\beta_i = \beta_{as} = 0.23$, $\sigma_i = 0.333$, $\sigma_T = 0.215$, $\sigma_Q = 0.33511$, $\mu_{as} = 0.08$, $\mu_{as} = 0.143$, $\mu_Q = 0.07272$, $\mu_J = 1$, $r = 0.018$, $k = 1$, $f = 0.2$, $\tau = 0.8$ and $x = 0.5$. 
Fig. 8. (a) Total Recovery of COVID-19 of INDIA from March 01, 2020 to Feb 01, 2021 (b) Total Recovery pattern of proposed model considering synthetic contact network as underlying topology. The degree distribution of synthetic contact network follows the power law. The transmission rate values are $\beta_s = 0.23$, $\sigma_I = 0.33$, $\sigma_T = 0.215$, $\sigma_Q = 0.33511$, $\mu_{as} = 0.08$, $\mu_{ac} = 0.143$, $\mu_J = 0.07272$, $\mu_T = 1$, $r = 0.018$, $\kappa = 1$, $f = 0.2$, $r = 0.8$ and $\alpha = 0.5$.

Fig. 9. Plot showing the impact of Critical Threshold of spreading rate $\beta_c$ of the proposed model over the synthetic network. The value of $\beta_c$ is varied from 0 to 0.24 to see the critical threshold’s impact.

4.5. Effect of underlying network topology on epidemic spreading

To see the impact of underlying network structure with different values of population is plotted in Fig. 10. Increasing the number of nodes in network increases the peak of infection as well as less time is taken to reach the peak. Three values of population 10000, 15000 and 20000 is used to create underlying network. It is observed that changing the number of nodes in network does not change the behavior of curve. It only impacts on the duration of disease and peak of infection. Increasing the number of nodes in network increases the number of hub nodes in scale-free network, that why, peak of infection increases in less time. It is observed that the topology of the network has a great influence in the overall behavior of epidemic spreading. The connectivity fluctuations of the network play a major role in infection spreading. Scale Free networks exhibit connectivity fluctuations. Scale free networks are therefore very helpful for the outbreak in face of infections spreading. The heterogeneity of Scale free networks finds signatures also in the peculiar susceptibility to infections starting on the most connected individuals within populations of varying connectivity $k$.

4.6. Effect of asymptomatic transmission rate ($\beta_{as}$) and testing on fraction of population ($f$) on epidemic spreading

To see the combined impact of $\beta_{as}$ and $f$ on infection spreading, contours are plotted in Fig. 11. In this course, those compartment are plotted which is effected by $\beta_{as}$ and $f$. The value of $\beta_{as}$ and $f$ are varied from 0 to 0.6 and see the affect
Fig. 10. Epidemic spreading pattern of proposed model considering synthetic contact network as underlying topology with varying number of nodes as 10000, 15000, and 20000. The degree distribution of synthetic contact network follows the power law. The transmission rate values are \( \beta_s = 0.23, \sigma_I = 0.333, \sigma_T = 0.215, \sigma_Q = 0.33511, \mu_{as} = 0.08, \mu_{us} = 0.143, \mu_Q = 0.07272, \mu_j = 1, \tau = 0.018, \kappa = 1, f = 0.2, r = 0.8 \) and \( \alpha = 0.5 \).

Fig. 11. Contour plot showing the effect of \( \beta_{as} \) and \( f \) on Epidemic spreading of proposed model considering synthetic network as underlying topology. The value of \( \beta_{as} \) and \( f \) are varied from 0 to 0.6. While value of other transmission rate values are \( \beta_s = 0.23, \sigma_I = 0.333, \sigma_T = 0.215, \sigma_Q = 0.33511, \mu_{as} = 0.08, \mu_{us} = 0.143, \mu_Q = 0.07272, \mu_j = 1, \tau = 0.018, \kappa = 1, f = 0.2, r = 0.8 \) and \( \alpha = 0.5 \).

4.7. Effect of asymptomatic transmission rate (\( \beta_{as} \))

One can notice in Fig. 12 that increasing the value of \( \beta_{as} \) raises the peak of exposed (\( E_k \)), tested (\( T_k \)), untested (\( UT_k \)), tested infection (Symptomatic) (\( I_{sk} \)), untested infection (Asymptomatic) (\( I_{usk} \)), untested infection (Symptomatic) (\( I_{usk} \)) quarantined (\( Q_k \)) and isolated (\( J_k \)) the population. However, it affects the disease duration that last around 90, 100, 160 and 200 time steps. Variations of \( \beta_{as} \) is directly affecting the proportion of exposed (\( E_k \)), tested (\( T_k \)), untested (\( UT_k \)), tested infection (Symptomatic) (\( I_{sk} \)), untested infection (Asymptomatic) (\( I_{usk} \)), untested infection (Symptomatic) (\( I_{usk} \)) quarantined (\( Q_k \)) and isolated (\( J_k \)) the population. Values of the maximum fraction of exposed (\( E_k \)), tested (\( T_k \)), untested (\( UT_k \)), tested infection (Symptomatic) (\( I_{sk} \)), untested infection (Asymptomatic) (\( I_{usk} \)), untested infection (Symptomatic) (\( I_{usk} \)) quarantined (\( Q_k \)) and isolated (\( J_k \)) population and associated time \( t \) are reported in Table 5 by varying the \( \beta_{as} \) in the range 0.1 to 0.9.
Fig. 12. Plot showing the effect of $\beta_{\text{as}}$ on epidemic spreading for the various values of $\beta_{\text{as}}$ considering synthetic contact network as underlying topology. The value of $\beta_{\text{as}}$ is varied from 0.1 to 0.9, while values of other transmission parameters keep fixed as $N = 10000$, $\beta_i = 0.23$, $\sigma_t = 0.333$, $\sigma_T = 0.215$, $\mu_Q = 0.33511$, $\mu_{\text{as}} = 0.08$, $\mu_{\text{is}} = 0.145$, $\mu_{\text{Q}} = 0.07272$, $\tau = 0.018$, $\kappa = 1$, $f = 0.2$, $r = 0.8$ and $\alpha = 0.5$.

Table 5
Impact of $\beta_{\text{as}}$ on the maximum proportion of compartmental population at time $t$.

| $\beta_{\text{as}}$ | $E_k$ | $Q_k$ | $I_{sk}$ | $I_{ask}$ | $J_t$ | $R_t$ |
|---------------------|-------|-------|----------|----------|-------|-------|
| 0.1                 | 0.005 | 0.025 | 0.0004   | 0.00013  | 0.00025| 0.2   |
| 0.2                 | 0.046 | 0.14  | 0.004    | 0.002    | 0.0025 | 0.85  |
| 0.3                 | 0.0759| 0.2   | 0.0061   | 0.0031   | 0.00198| 0.875 |
| 0.4                 | 0.096 | 0.226 | 0.008    | 0.0039   | 0.0031 | 0.875 |
| 0.5                 | 0.115 | 0.226 | 0.0092   | 0.0046   | 0.0038 | 0.875 |
| 0.6                 | 0.128 | 0.253 | 0.0105   | 0.0051   | 0.005 | 0.875 |
| 0.7                 | 0.128 | 0.253 | 0.0105   | 0.0051   | 0.005 | 0.875 |
| 0.8                 | 0.128 | 0.253 | 0.0105   | 0.0051   | 0.005 | 0.875 |
| 0.9                 | 0.128 | 0.253 | 0.0105   | 0.0051   | 0.005 | 0.875 |
Table 6
Impact of \( \sigma T \) in combination of fraction of exposed population \( f \) on the maximum proportion of compartmental population at time \( t \).

| \( f \) | 0    | 0.2  | 0.4  | 0.6  | 0.8  | 1    |
|--------|------|------|------|------|------|------|
| \( T_k \) | 0    | 0.0034 | 0.0049 | 0.0038 | 0.00029 | 0.0003 |
| \( t \) | 0    | 62   | 78   | 110  | 300  | 10  |
| \( UT_k \) | 0.032 | 0.021 | 0.011 | 0.004 | 0.000001293 | 0 |
| \( t \) | 60   | 70   | 77   | 113  | 0    | 0   |
| \( J_k \) | 0    | 0.0017 | 0.00241 | 0.00189 | 0.0002 | 0.0002 |
| \( t \) | 0    | 65   | 80   | 112  | 300  | 10  |
| \( I_{sk} \) | 0.082 | 0.056 | 0.03217 | 0.01238 | 0.0000139 | 0 |
| \( t \) | 69   | 75   | 88   | 125  | 0    | 0   |
| \( I_{usk} \) | 0.013054 | 0.008411 | 0.004317 | 0.001923  | 0 |
| \( t \) | 67   | 72   | 86   | 120  | 0    | 0 |

4.8. Effect of testing (\( f \))

In order to investigate the influence of the testing \( \sigma T \) in combination of fraction of exposed population \( f \) on the epidemic dynamics, the value of \( f \) is increased linearly in the range 0 to 1 with a step of 0.2 while keeping other transmission parameters are fixed. The evolution of the tested infection (symptomatic) population is dependent on testing as well as quarantined population. The tested population \( (T_k) \), tested infection (Symptomatic) \( (I_{sk}) \), untested population \( (UT_k) \), untested infection (Symptomatic) \( (I_{usk}) \), untested infection (asymptomatic) \( (I_{sk}) \) and Isolated \( (J_k) \) versus time are plotted for the various values of the \( f \) varying from 0 to 1 in Fig. 13. All the curves of Test \( (T_k) \), Infection (Symptomatic) \( (I_{sk}) \) and Isolated \( (J_k) \) population exhibit the same behavior, while untested population \( (UT_k) \), untested infection (Symptomatic) \( (I_{usk}) \), untested infection (asymptomatic) \( (I_{sk}) \) exhibit the similar curve. The proportion of compartmental population increases up to a maximum value when time increases, and then it decreases until there is no more population left in compartment. However, both the max proportion of compartmental population and the time \( t \) varies. The fractional value \( f \) also affect the epidemic spreading behavior in the population as shown in Table 6.

4.9. Prediction of COVID-19 spreading in future considering proposed model

To predict the future spreading patterns of the proposed model’s epidemic dynamics with a given set of transmission parameters with underlying network topology, when the epidemic will die, how recovery is completed. Further plots are generated. Up to some time, the spread of an epidemic in the human population increases with time as shown in Fig. 14. Further, it begins to fade and gradually decreases. At 300 time stages, the susceptible population drops to 0.04 and remains constant. Recovery starts when an infected individual recovers from the infection. The whole infection is recovered at 200-time steps (Fig. 14(a)).

The exposed population are those that are in close contact with infectious agents. The infection spread only to those populations that are exposed. As time passes, people start exposing and contacting with an infectious agent, and it reaches maximum at 63-time steps. In the meantime, the testing process is also started from the exposed population. The testing process is done up to the 300-time steps (Fig. 14(b)). The certain exposed population is quarantined till symptom arises. If a symptom arises, it is tested and moved to the tested symptomatic infection compartment, and after that, it is sent to isolation. In this course, the quarantined population has reached the peak of 0.125 in 72-time steps and vanishes after 200-time steps (Fig. 14(c)). In the meantime, the testing process is also started. The testing process is done when the exposed population showing symptom of infection (Fig. 14(d)). As exposed population exists, tests are done. Both reaches to its peak at 63-time steps after that start decreasing and vanishes after 175-time steps.

When the testing process is started, some infectious agent is detected while the infection is also increased due to the exposed population which is untested or undetected. Untested Asymptomatic infection and Untested Symptomatic infection plays a major role in the infection spreading as compared to tested symptomatic infection. Due to testing, a number of symptomatic infection is checked and isolated. Therefore, the tested symptomatic infection does not spread the infection to a large extent as compared to untested asymptomatic and symptomatic infection. This is because the untested asymptomatic and symptomatic is directly dependent on the exposed population, while the tested symptomatic infection is checked and isolated (Fig. 14(e), (f), (g) & (h)). The peak of tested symptomatic infection, untested symptomatic infection and untested asymptomatic infection reaches to 0.0017, 0.056 and 0.0089 in 65, 75, 72 time steps after that it starts decreasing and vanishes after 175, 175, 200 and 175-time steps. All tested symptomatic infection is sent to isolation, while the isolated population reaches the maximum value of 0.00165 in 66-time steps (Fig. 14(i)) and vanishes after 200-time steps. Recovery starts when an infected individual recovers from the infection. The total recovered from infection at 100-time steps are 0.71 and become constant after 200 time steps (Fig. 14(j)).
Fig. 13. Plot showing the effect of $\sigma_T$ in combination of fraction of exposed population $f$ on Epidemic spreading for the various values of $f$ considering synthetic network as underlying topology. The value of $f$ is varied from 0 to 1, while value of other transmission parameters keep fixed as $\beta_s = 0.23$, $\sigma_I = 0.333$, $\sigma_T = 0.215$, $\mu_{as} = 0.08$, $\mu_{as} = 0.143$, $\mu_Q = 0.07272$, $\mu_J = 1$, $r = 0.018$, $\kappa = 1$, $r = 0.8$ and $\alpha = 0.5$.

5. Conclusions and future directions

In this work, an epidemiological model is proposed by modifying the SEIR model with additional compartments as Test, Quarantine, Tested Symptomatic Infection, Untested, Untested Asymptomatic Infection and Isolation considering underlying heterogeneous network topology. It is investigated the impact of $\beta_{as}$ and $\sigma_T$ on the epidemiological process in this paper. It is observed that, Untested (symptomatic and asymptomatic) infection plays major role in spreading of infection as it influence the Exposed compartment from where, population is moved to other compartments. Further it is investigated the impact of $f$ by varying the value from 0 to 1. It explains that increasing the value of $f$ increases the Symptomatic Infection and Isolation, while decreasing the untested population. There are three types of reproduction numbers derived in this paper: control reproduction number, $R_c$, time-dependent effective reproduction number $R_t$, and basic reproduction number $R_0$. The basic reproduction rate that tells about the secondary infection for the proposed model. We have plotted the curve of $R_t$ of the proposed model, which shows a similar curve of $R_t$ with real data of COVID-19 that shows the effectiveness of our model. The critical threshold is also derived for the proposed model, which is responsible for the outbreak of infection.

There are various future directions for this work. Tailoring the model by considering spatiotemporal networks as the underlying network is one of the potential directions. This is not a simple problem since even though data is accessible, linking all of the experimental conditions to these data is difficult. Furthermore, by considering the various interactions between hosts, such as modular networks and dynamic networks, a more realistic scenario can be developed.

CRediT authorship contribution statement

Anurag Singh: Conceptualization, Methodology, Software, Formal Analysis, Investigation, Resources, Writing – original draft, Writing – review & editing, Supervision, Project administration, Funding acquisition. Md Arquam: Software, Formal Analysis, Data-Curation, Writing – original draft, Writing – review & editing, Visualization, Validation, Resources.

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.

Acknowledgment

This work is supported by Science and Engineering Research Board (SERB), DST, Government of India under MATRICS Short term special call on COVID-19 project (File.No. MSC/2020/000500).
Fig. 14. Plot showing the prediction curve of Epidemic spreading of proposed model considering synthetic contact network as underlying topology. The degree distribution of synthetic contact network follows the power law. The transmission rate values are $\beta_s = \beta_{aw} = 0.23$, $\sigma_I = 0.333$, $\sigma_T = 0.215$, $\sigma_Q = 0.33511$, $\mu_{aw} = 0.08$, $\mu_Q = 0.143$, $\mu_T = 0.07272$, $\mu_J = 1$, $\tau = 0.018$, $\kappa = 1$, $f = 0.2$, $r = 0.8$ and $\alpha = 0.5$.

Appendix A

Initially, it is considered that total population is fixed. Hence,

\[
\frac{dT(k, t)}{dt} = 0
\]

\[
f \sigma_T E(k, t) + \tau Q(k, t) - \alpha T(k, t) = 0
\]

$Q(k, t)$ does not help to go infection out.

\[
T(k, t) = \frac{f \sigma_T E(k, t)}{\alpha}
\]
Similarly,

\[
\frac{dE(k, t)}{dt} = 0
\]

\[
(\beta_s \Theta_s(t) + \beta_{as} \Theta_{as}(t))kS(k, t) - \sigma_Q E(k, t) - f \sigma_T E(k, t)
\]

\[
(1 - f)\sigma_T E(k, t) = 0
\]

\[
E(k, t) = \frac{(\beta_s \Theta_s(t) + \beta_{as} \Theta_{as}(t))kS(k, t)}{\sigma_Q + \sigma_T}
\]

\[
(1 - f)\sigma_T E(k, t) - (1 - r)\sigma_T UT(k, t) - r \sigma_T UT(k, t) = 0
\]

\[
UT(k, t) = \frac{(1 - f)\sigma_T E(k, t)}{\sigma_T}
\]

Putting the value of (24), (26) and (25) in Eq. (18),

\[
\alpha T(k, t) - \kappa I_0(k, t) + \sigma_T UT(k, t) - \mu_s I_s(k, t) - \mu_{as} I_{as}(k, t) > 0
\]

\[
\alpha \left( \frac{f \sigma_T E(k, t)}{\alpha} + \sigma_T \left( \frac{(1 - f)\sigma_T E(k, t)}{\sigma_T} \right) \right)
\]

\[
- \kappa I_0(k, t) - \mu_s I_s(k, t) - \mu_{as} I_{as}(k, t) > 0
\]

\[
\sigma_T E(k, t) - \kappa I_0(k, t) - \mu_s I_s(k, t) - \mu_{as} I_{as}(k, t) > 0
\]

Putting the value of (27),

\[
\left( \sigma_T \right) \frac{(\beta_s \Theta_s(t) + \beta_{as} \Theta_{as}(t))kS(k, t)}{\sigma_Q + \sigma_T} - (\mu_s + \kappa + \mu_{as}) l(k, t) > 0
\]

\[
\left( \sigma_T \right) \frac{(\beta_s \Theta_s(t) + \beta_{as} \Theta_{as}(t))kS(k, t)}{\sigma_Q + \sigma_T} > (\mu_s + \kappa + \mu_{as}) l(k, t)
\]

\[
\left( \sigma_T \right) \frac{(\beta_s \Theta_s(t) + \beta_{as} \Theta_{as}(t))kS(k, t)}{\sigma_Q + \sigma_T} > (\mu_s + \kappa + \mu_{as}) l(k, t) \]

Appendix B

Q(k, t) does not help to go infection out, while J(k, t) is dependent on I_s(k, t)

\[
\frac{ds(k, t)}{dr(k, t)} = \frac{-\beta_s \Theta_s(t) + \beta_{as} \Theta_{as}(t))kS(k, t)}{\mu_j f(k, t) + \mu_s I_s(k, t) + \mu_{as} I_{as}(k, t)}
\]

Population of J(k, t) is considered, when it is fixed,

\[
\frac{dj(k, t)}{dt} = 0
\]

\[
\kappa I_0(k, t) - \mu_j f(k, t) = 0
\]

\[
J(k, t) = \frac{\kappa I_0(k, t)}{\mu_j}
\]

Putting the value of J(k, t) in Eq. (29)

\[
\frac{ds(k, t)}{dr(k, t)} = \frac{-\beta_s \Theta_s(t) + \beta_{as} \Theta_{as}(t))kS(k, t)}{\kappa I_0(k, t) + \mu_s I_s(k, t) + \mu_{as} I_{as}(k, t)}
\]

\[
\frac{ds(k, t)}{dr(k, t)} = \frac{-\beta_s \Theta_s(t) + \beta_{as} \Theta_{as}(t))kS(k, t)}{\kappa I_0(k, t) + \mu_s I_s(k, t) + \mu_{as} I_{as}(k, t)}
\]

\[
\frac{ds(k, t)}{dr(k, t)} = \frac{-\beta_s \Theta_s(t) + \beta_{as} \Theta_{as}(t))kS(k, t)}{\kappa I_0(k, t) + \mu_s I_s(k, t) + \mu_{as} I_{as}(k, t)}
\]

\[
\frac{ds(k, t)}{dr(k, t)} = \frac{-\beta_s \Theta_s(t) + \beta_{as} \Theta_{as}(t))kS(k, t)}{\kappa I_0(k, t) + \mu_s I_s(k, t) + \mu_{as} I_{as}(k, t)}
\]

Integrating both side

\[
S(k, t) = e^{\frac{-\beta_s \Theta_s(t) + \beta_{as} \Theta_{as}(t))kS(k, t)}{\kappa I_0(k, t) + \mu_s I_s(k, t) + \mu_{as} I_{as}(k, t)}}
\]
Let,
\[ \sum_{k=1}^{k_{\text{max}}} P(k'|k)R(k, t) = \Omega(k, t) \quad (30) \]

At \( t \to \infty \), the epidemic reaches a steady state hence, all intermediate compartment including \( I_h(\infty) = 0 \).
\[ S(k, \infty) = 1 - R(k, \infty) \quad (31) \]
\[ S(k, \infty) = e^{-\frac{\beta k}{Z} \Omega(k, \infty)} \]
\[ R(k, \infty) = 1 - e^{-\frac{\beta k}{Z} \Omega(k, \infty)} \quad (32) \]

Assume, \((\kappa + \mu_s + \mu_{as}) = Z \) and \((\beta_s + \beta_{as}) = \beta \)
Therefore,
\[ R(k, \infty) = 1 - e^{-\frac{\beta}{Z} \Omega(k, \infty)} \quad (33) \]

Negative exponent in Eq. (33) shows that the number of susceptible nodes are decreasing and converted into recovered nodes. Therefore,
\[ \Omega(k, t) = \sum_{k=1}^{k_{\text{max}}} P(k'|k)R(k, t) \]
\[ \Omega(k, \infty) = \sum_{k=1}^{k_{\text{max}}} P(k'|k)R(k, \infty) \]
\[ \Omega(k, \infty) = \sum_{k=1}^{k_{\text{max}}} P(k'|k) \left[ 1 - e^{-\frac{\beta k}{Z} \Omega(k, \infty)} \right] \quad (34) \]
\[ = f(\Omega(k, \infty)) \]

Assume that \( f(\Omega(k, \infty)) \) is strictly increasing as a function of \( \Omega(k, \infty) \). When \( R(k, \infty) \) is set to 0, the total population recovers. It is also known as a disease-free state. As a result, we must find a solution that is between 0 and 1. However, since Eq. (34) is coupled with network parameters, we cannot express it in terms of \( R(k, \infty) \). As a result, we must find a solution (30).

Now deriving the \( f(\Omega(k, \infty)) \) with respect to \( \Omega(k, \infty) \), where,
\[ f(\Omega(k, \infty)) = \sum_{k=1}^{k_{\text{max}}} P(k'|k) \left[ 1 - e^{-\frac{\beta k}{Z} \Omega(k, \infty)} \right] \]

Hence,
\[ \frac{df(\Omega(k, \infty))}{d\Omega(k, \infty)} \bigg|_{\Omega(k, \infty)=0} > 1 \quad (35) \]
\[ \sum_{k=1}^{k_{\text{max}}} P(k'|k) \beta k \frac{1}{Z} > 1 \quad (36) \]

Reporting \( \sum_{k=1}^{k_{\text{max}}} P(k'|k) = \frac{\sum_{k=1}^{k_{\text{max}}} k p(k)}{\langle k \rangle} \) in Eq. (36),
\[ \frac{\sum_{k=1}^{k_{\text{max}}} k p(k)}{\langle k \rangle} \beta k \frac{1}{Z} > 1 \]
\[ \frac{(k^2) \beta}{\langle k \rangle Z} > 1 \]

Therefore, critical threshold can be represented as,
\[ \beta > \frac{(k) Z}{(k^2)} \quad (37) \]

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