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Dynamics of an SEQIHRS epidemic model with media coverage, quarantine and isolation in a community with pre-existing immunity

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\textbf{A R T I C L E  I N F O}

\textbf{Article history:}
Received 9 December 2013
Available online 19 August 2014
Submitted by J.J. Nieto

\textbf{Keywords:}
Epidemic model
Isolation
Quarantine
Media coverage
Pre-existing immunity
Sensitivity analysis

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

An autonomous deterministic non-linear epidemic model SEQIHRS is proposed for the transmission dynamics of an infectious disease with quarantine and isolation control strategies in a community with pre-existing immunity. The model exhibits two equilibria, namely, the disease-free and a unique endemic equilibrium. The existence and local stability of the disease free and endemic equilibria are explored in terms of the effective reproduction number \( R_C \). It is observed that media coverage does not affect the effective reproduction number, but it helps to mitigate disease burden by lowering the number of infectious individuals at the endemic steady state and also lowering the infection peak. A new approach is proposed to estimate the coefficient of media coverage. Using the results of central manifold theory, it is established that as \( R_C \) passes through unity, transcritical bifurcation occurs in the system and the unique endemic equilibrium is asymptotically stable. It is observed that the population level impact of quarantine and isolation depend on the level of transmission by the isolated individuals. Moreover, the higher level of pre-existing immunity in the population decreases the infection peak and causes its early arrival. Theoretical findings are supported by numerical simulation. Sensitivity analysis is performed for \( R_C \) and state variables at endemic steady state with respect to model parameters.

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1. Introduction

Mathematical modeling has become an important tool in analyzing the spread and control of infectious diseases taking into account the main factors governing development of a disease, such as transmission and recovery rates. Mathematical models are being used to predict how the disease will spread over a period of...
time. In recent years, many attempts have been made to develop realistic mathematical models for investigating the transmission dynamics of infectious diseases, and the asymptotic behaviors of these epidemic models are studied [10,11,42,62]. Vaccination and antiviral drugs are the two most effective pharmaceutical interventions used for the control of an infectious disease. But due to strain’s novelty most people may lack innate immunity to the disease, available vaccine may not provide protection against the pathogen and effective antiviral drug may not be available in sufficient amount. For example, Bird flu viruses H5N1 and H7N9 that have sporadically infected humans, could, with a few mutations to a key protein on their surface, become capable of infecting cells along the human upper airway and thereby take a step towards turning into pandemic-causing strains [50,51]. In that scenario, the role of isolation, quarantine and other non-pharmaceutical interventions stimulated by media coverage becomes more significant as disease control strategies.

Despite initial concern that little protective immunity existed in the general population for pandemic H1N1 (2009), subsequent epidemiological data showed that morbidity in the elderly was lower than that in younger individuals, suggesting the existence of pre-existing immunity [16,19,27,58]. The Centers for Disease Control and Prevention (Atlanta, GA, USA) reported that among persons > 60 years old, 33% have pre-existing, cross-reactive neutralizing antibodies against the new virus of pandemic (H1N1) 2009 [58]. Phylogenetic analyses on the HA of the 2009 pandemic H1N1 virus demonstrated its close relationship with the 1918–1919 Spanish H1N1 virus [15,59]. Low virulence of the virus and pre-existing immune status are among the main factors that account for lower death rates in influenza outbreaks [58]. Pre-existing immunity is an important factor countering the pandemic potential of an emerging infectious disease. Thus, studying of pre-existing immunity will advance our understanding of the pathogenesis and disease dynamics of the emerging pathogen.

When an infectious disease breaks out in a population, people’s response to the threat of the disease is dependent on their perception of risk, which is affected by public and private information disseminated widely by the media [28,36,49]. Media coverage about an epidemic gives a sense about the risk level and the relative need for precautions in risk areas and encourage the public to take precautionary measures against the disease such as wearing masks, avoiding public places, avoiding travel when sick, frequent hand washing, etc. Massive news coverage and fast information flow can generate a profound psychological impact on public health [55]. This is extremely important in the early stages of an epidemic, when pharmaceutical interventions are not often possible because treatment or vaccination options have not yet been developed [47]. Media coverage and education may reduce the contact rate of human beings and the use of NPIs may reduce the transmission probability. Many researchers investigated the impact of media awareness using mathematical modeling [2,7,8,14,22,23,26,28,30–32,34,36,43,47–49,52,53,55,57,61]. One method is to form an information set summarized by a new state variable, mostly based on the publicly available information on both present and on the recent past spreading of the disease [2,30–32,43]. The population is broadly classified as educated and non-educated to reflect the awareness through media coverage [30–32,43]. Complex network model is used to model media effect by Wang et al. and Yuan et al. [53,61]. Liu et al. proposed an EIH model where $H$ denotes hospitalized individuals assuming transmission coefficient of the form $\beta_0 = \beta e^{-\alpha_1 I - \alpha_2 H}$ [28]. Possible multiple outbreaks and sustained periodic oscillations of the infection were observed. Cui et al. used similar transmission coefficient and established that multiple positive equilibria are possible when the media effect is sufficiently strong [7]. Reduction function of the negative exponential form to describe the reduction factor either by large number of infectious cases or by significant change in the number of infectious cases is used to model the media coverage [48,57]. Cui et al. [8] proposed a general contact rate, $\beta(I) = c_1 - c_2 f(I)$, to reflect some intrinsic characters of media coverage. Using the same contact rate proposed by Liu and Cui [26], Tchuenche et al. [49] studied the impact of media coverage on the spread and control of an influenza strain. Sun et al. [47] used similar non-linear contact rate as in [8] to study media-induced social distancing in a two patch setting. Li and Cui [23] studied the effect of constant and pulse vaccination on SIS epidemic models incorporating media-induced incidence function similar as in [26,49].
Emerging infectious diseases have devastating impacts on public health and impose great financial burden on the community, which attracts a major concern to public health agency. So it is of great importance to evaluate optimal methods for controlling these diseases [46]. Historically, quarantine (of individuals feared exposed to a communicable disease) is one of the oldest public health control measures for the spread of communicable diseases. These measures have been successfully applied dating back to the plague epidemic of the 13th century, to the influenza epidemics of the 20th century. More recently, this measure was successfully used to combat the spread of some emerging and re-emerging human and animal diseases, such as the severe acute respiratory syndrome (SARS) [18,25,29,33,54,60], foot-and-mouth disease [21] and the 2009 swine influenza pandemic [17]. The SARS outbreaks of 2003 provided an important example of a novel disease that was effectively controlled using quarantine and isolation [18,25,29]. Implementing these measures, however, can inflict significant socio-economic and psychological costs. Public health officials need to be able to present comprehensive, understandable assessments of the options to other government officials in a timely manner [37]. For the purpose of this study, quarantine means the removal of individuals suspected of being infected (yet exhibiting no clinical symptoms) from the general population. Thus, these individuals could be asymptomatically-infected or susceptible. On the other hand, isolation refers to the removal of infected individuals exhibiting clinical symptoms of the disease. Although isolation is probably always a desirable public health measure, quarantine is more controversial. Mass quarantine can inflict significant social, psychological, and economic costs without resulting in the detection of many infected individuals [9]. Isolation is primarily used for controlling the disease when it suddenly emerges or reemerges [5]. A successful example is the isolation of those infected with SARS during 2003–2004. However, the disadvantages of this strategy are the difficulty of detecting infected individuals and the cost of isolation. In general, to achieve perfect isolation is difficult at large scale resulting in a leaky isolation causing nosocomial infections.

Numerous mathematical modeling works have been carried out to assess the impact of quarantine and isolation in controlling the spread of communicable diseases in human and animal populations [9,18,20,25,29, 33,35,38–41,54,56,60]. Further, the emergence of SARS in 2003 led to the formulation of numerous quarantine and isolation models for curtailing its spread [9,18,25,29,54]. Most of the disease modeling studies, published in the literature, provided quantitative evaluation of the control measures (quarantine and isolation) by simulating the models with available epidemiological and demographic data [9,18,25,29,54].

The primary goal of this article is to theoretically study the impact of use of NPIs stimulated by media coverage, quarantine and isolation for an infectious disease in a community with pre-existing immunity. The rest of the paper is organized as follows: In Section 2, the proposed model in formulated. In Section 3, existence and local behavior of disease free (DFE) and endemic equilibria are explored. Global stability of DFE and uniform persistence is established. In Section 4, important thresholds are calculated. Numerical simulation is performed in Section 5. Sensitivity analysis is performed for effective reproduction number and steady states at endemic level with respect to model parameters in Section 6. Finally, results are discussed in Section 7.

2. Model formulation and basic properties

In this section, we will formulate an epidemic model incorporating quarantine, isolation, use of non-pharmaceutical interventions stimulated by media coverage in presence of pre-existing cross-protective immunity. The total population at time $t$, denoted by $N(t)$, is sub-divided into six mutually exclusive compartments of susceptible ($S(t)$), exposed ($E(t)$), quarantined ($Q(t)$), infections ($I(t)$), hospitalized ($H(t)$) and recovered ($R(t)$) individuals, so that $N(t) = S(t) + E(t) + Q(t) + I(t) + H(t) + R(t)$. In the modeling of infectious diseases, the incidence function plays a very important role, it can determine the rise and fall of epidemics [42]. In many epidemic models, the bilinear incidence rate $\beta SI$ and the standard incidence rate $\beta SI/N$ are frequently used, where $\beta$ measures the effect of both the infectiousness of the disease and the contact transmission rates. However, these incidence functions do not consider the impact of media coverage
to the spread and control of infectious diseases. The media induced transmission rate \( \tilde{\beta}(\tilde{I}) = \tilde{\beta}e^{-m\tilde{I}} \), used in [7,28], has two limitations. First, \( \tilde{\beta}e^{-m\tilde{I}} \rightarrow 0 \) as \( \tilde{I} \rightarrow \infty \), independent of the value of \( m \). It is not reasonable since the media coverage is not the intrinsic deterministic factor responsible for the transmission and hence the transmission rate cannot be reduced below a certain level merely through media awareness and alert. Second, even for a fixed \( m \), the minimum transmission rate differs for different population sizes, regardless of the similarity in social structure (i.e., education and awareness level) and climatic condition.

We propose media induced transmission rate as \( \tilde{\beta}(\tilde{I}) = \tilde{\beta}e^{-\frac{m}{\bar{c}}\tilde{I}} \), which is more reasonable than that used in [7,28] overcoming the aforementioned limitations (see Fig. 1).

Keeping in view the above, in our proposed model we choose the media induced transmission rate of the form \( \tilde{\beta}e^{-\frac{m_1\tilde{I}+m_2\tilde{H}}{N}} \). The parameters \( m_1 \) and \( m_2 \) represent the coefficients of media coverage using non-pharmaceutical interventions corresponding to infectious (\( \tilde{I} \)) and isolated (\( \tilde{H} \)) individuals, respectively. Now, the next important question is how to measure the media coefficient \( m \). One innovative way of estimation of the media awareness coefficient \( m \) will be discussed in numerical simulation Section 5. Other modeling assumptions are as follows: The population grows at constant rate \( \Lambda \). Population in all the compartments decreases at rate \( \tilde{\mu} \) due to natural death. Susceptible individuals acquire infection through effective contact with infectious individuals at rate

\[
\tilde{\beta}e^{-\frac{m_1\tilde{I}+m_2\tilde{H}}{N}} \frac{\tilde{S}(\tilde{I} + \eta\tilde{H})}{N}.
\]

The parameter \( \tilde{\beta} \) is the effective contact rate (that is, contact capable of leading to infection), while the modification parameter, \( 0 < \eta < 1 \), accounts for the assumed reduction in disease transmission by isolated individuals in comparison to non-hospitalized infectious individuals in the \( \tilde{I} \) class. Thus, \( \eta \) measures the efficacy of isolation or treatment given to hospitalized individuals (isolation is perfect if \( \eta = 0 \); leaky if \( 0 < \eta < 1 \) and completely ineffective if \( \eta = 1 \)). The schematic flow diagram of the proposed model is shown in Fig. 2. Based on the aforementioned modeling assumptions, the proposed model is governed by the following system of ordinary differential equations:

\[
\frac{d\tilde{S}}{dt} = \Lambda - \tilde{\mu}\tilde{S} - \tilde{\beta}e^{-\frac{m_1\tilde{I}+m_2\tilde{H}}{N}} \frac{\tilde{S}(\tilde{I} + \eta\tilde{H})}{N} + \tilde{\theta}\tilde{R},
\]

\[
\frac{d\tilde{E}}{dt} = \tilde{\beta}e^{-\frac{m_1\tilde{I}+m_2\tilde{H}}{N}} \frac{\tilde{S}(\tilde{I} + \eta\tilde{H})}{N} - (\tilde{\mu} + \tilde{k} + \tilde{\sigma} + \tilde{\xi})\tilde{E},
\]
Fig. 2. Schematic diagram of proposed SEQIHRS epidemic model.

Table 1
Description of parameters for the system (2)–(7).

| Parameter | Description | Unit |
|-----------|-------------|------|
| $\Lambda$ | Recruitment rate | days$^{-1}$ |
| $\tilde{\mu}$ | Natural death rate | days$^{-1}$ |
| $\tilde{\beta}$ | Contact rate (in absence of NPIs through media coverage) | days$^{-1}$ |
| $\eta$ | Modification parameter for reduction in infectiousness of hospitalized individuals | $m_1, m_2$ |
| $\tilde{\sigma}$ | Progression rate from exposed to infectious class | days$^{-1}$ |
| $\tilde{\xi}$ | Recovery rate due to pre-existing cross-protective immunity | days$^{-1}$ |
| $k$ | Quarantine rate for exposed individuals | days$^{-1}$ |
| $\alpha$ | Hospitalization rate for quarantined individuals | days$^{-1}$ |
| $\phi$ | Hospitalization rate for infectious individuals | days$^{-1}$ |
| $\gamma_1$ | Recovery rate for non-hospitalized infectious individuals | days$^{-1}$ |
| $\gamma_2$ | Recovery rate for hospitalized infectious individuals | days$^{-1}$ |
| $1/\theta$ | Average waning period of disease-induced immunity | days |
| $\delta_1$ | Disease-induced death rate for non-hospitalized infectious individuals | days$^{-1}$ |
| $\delta_2$ | Disease-induced death rate for hospitalized individuals | days$^{-1}$ |

\[
\frac{d\tilde{Q}}{dt} = k\tilde{E} - (\tilde{\alpha} + \tilde{\xi} + \tilde{\mu})\tilde{Q}, \quad (4)
\]
\[
\frac{d\tilde{I}}{dt} = \tilde{\sigma}\tilde{E} - (\gamma_1 + \tilde{\phi} + \tilde{\mu} + \tilde{\delta}_1)\tilde{I}, \quad (5)
\]
\[
\frac{d\tilde{H}}{dt} = \tilde{\alpha}\tilde{Q} + \tilde{\phi}\tilde{I} - (\gamma_2 + \tilde{\mu} + \tilde{\delta}_2)\tilde{H}, \quad (6)
\]
\[
\frac{d\tilde{R}}{dt} = \gamma_1\tilde{I} + \tilde{\xi}\tilde{Q} + \gamma_2\tilde{H} + \xi\tilde{E} - (\tilde{\mu} + \tilde{\theta})\tilde{R}; \quad (7)
\]

with initial conditions: $\tilde{S}(0) = \tilde{S}_0 > 0$, $\tilde{E}(0) = \tilde{E}_0 > 0$, $\tilde{Q}(0) = \tilde{Q}_0 > 0$, $\tilde{I}(0) = \tilde{I}_0 > 0$, $\tilde{H}(0) = \tilde{H}_0 > 0$, $\tilde{R}(0) = \tilde{R}_0 > 0$. Descriptions of all the parameters are summarized in Table 1. Note that

\[
\frac{d\tilde{N}}{dt} = \Lambda - \tilde{\mu}\tilde{N} - \tilde{\delta}_1\tilde{I} - \tilde{\delta}_2\tilde{H}.
\]

We consider only solutions with initial conditions inside the biologically feasible region

\[
\Gamma = \left\{ (\tilde{S}, \tilde{E}, \tilde{Q}, \tilde{I}, \tilde{H}, \tilde{R}) \in \mathbb{R}_+^6 : 0 \leq \tilde{S}, \tilde{E}, \tilde{Q}, \tilde{I}, \tilde{H}, \tilde{R}, \tilde{S} + \tilde{E} + \tilde{I} + \tilde{Q} + \tilde{H} + \tilde{R} \leq \frac{\Lambda}{\tilde{\mu}} \right\}
\]

in which the usual existence, uniqueness of solutions and continuation results hold. We study the system (2)–(7) and claim that the region $\Gamma$ is bounded and positively invariant with respect to the proposed system (2)–(7).
Proposition 2.1. All the solution trajectories of system (2)–(7) initiating inside \( \Gamma \) approach enter or stay within the interior of \( \Gamma \).

Proof. Let \( \mathbb{R}^6_+ = \{(\tilde{S}, \tilde{E}, \tilde{Q}, \tilde{I}, \tilde{H}, \tilde{R}) \in \mathbb{R}^6; \ \tilde{S} \geq 0, \ \tilde{E} \geq 0, \ \tilde{Q} \geq 0, \ \tilde{I} \geq 0, \ \tilde{H} \geq 0, \ \tilde{R} \geq 0\} \) denote the non-negative cone in six-dimensional Euclidean space. From the system (2)–(7), we observe that

\[
\frac{d\tilde{S}}{dt}_{|\tilde{S}=0} = A + \tilde{\theta} \tilde{R} > 0, \quad \frac{d\tilde{E}}{dt}_{|\tilde{E}=0} = \tilde{\beta}e^{-\frac{m_1 \tilde{I} + m_2 \tilde{H} \tilde{S}(\tilde{I} + \eta \tilde{H})}{N}} > 0, \quad \frac{d\tilde{Q}}{dt}_{|\tilde{Q}=0} = \tilde{k} \tilde{E} > 0,
\]

\[
\frac{d\tilde{I}}{dt}_{|\tilde{I}=0} = \tilde{\sigma} \tilde{E} > 0, \quad \frac{d\tilde{H}}{dt}_{|\tilde{H}=0} = \tilde{\gamma}_1 \tilde{I} + \tilde{\xi} \tilde{Q} + \tilde{\gamma}_2 \tilde{H} + \tilde{\xi} \tilde{E} > 0
\]

and \( \tilde{S}(\tilde{t}), \tilde{E}(\tilde{t}), \tilde{Q}(\tilde{t}), \tilde{I}(\tilde{t}), \tilde{H}(\tilde{t}), \tilde{R}(\tilde{t}) \) are continuous functions of \( \tilde{t} \). Thus the vector field on each bounding hyperplane of \( \mathbb{R}^6_+ \) is pointing inward direction of \( \mathbb{R}^6_+ \). Hence all the solution trajectories initiating in \( \mathbb{R}^6_+ \) will remain inside \( \mathbb{R}^6_+ \) for all the time. This establishes the fact that \( \mathbb{R}^6_+ \) is positively invariant for the system (2)–(7). Also, the total population \( \tilde{N}(\tilde{t}) \) satisfies \( \frac{d\tilde{N}}{dt} = A - \tilde{\mu} \tilde{N} - \tilde{\delta}_1 \tilde{I} - \tilde{\delta}_2 \tilde{H} \). Then, \( \frac{d\tilde{N}}{dt} < A - \tilde{\mu} \tilde{N} \), applying Birkhoff’s and Rota’s theorems on differential inequality [1,45], as \( \tilde{t} \to \infty \), we have \( 0 \leq \tilde{N}(\tilde{t}) \leq \frac{A}{\mu} = \tilde{N}_0 \). Therefore the solution of system (2)–(7) is bounded and hence any solution of the system originated from \( \Gamma \) remains in \( \Gamma \). \( \quad \Box \)

We reduce the above system into non-dimensional form using

\[
S = \frac{\tilde{S}}{\tilde{N}}, \quad E = \frac{\tilde{E}}{\tilde{N}}, \quad Q = \frac{\tilde{Q}}{\tilde{N}}, \quad I = \frac{\tilde{I}}{\tilde{N}}, \quad H = \frac{\tilde{H}}{\tilde{N}}, \quad R = \frac{\tilde{R}}{\tilde{N}}, \quad N = \frac{\tilde{N}}{\tilde{N}_0}, \quad t = \tilde{\mu} \tilde{t}.
\]

Since \( S = 1 - E - Q - I - H - R \), dropping the equation

\[
\frac{dS}{dt} = \frac{1}{N} - \beta e^{-m_1 I - m_2 H} S(I + \eta H) + \theta R - S - \frac{S}{N} \frac{dN}{dt};
\]

the equivalent non-dimensional system is given by:

\[
\frac{dE}{dt} = \beta e^{-m_1 I - m_2 H} S(I + \eta H) - (1 + k + \sigma + \xi) E - \frac{E}{N} \frac{dN}{dt} := f_1,
\]

\[
\frac{dQ}{dt} = k E - (1 + \alpha + \xi) Q - \frac{Q}{N} \frac{dN}{dt} := f_2,
\]

\[
\frac{dI}{dt} = \sigma E - (1 + \gamma_1 + \delta_1 + \phi) I - \frac{I}{N} \frac{dN}{dt} := f_3,
\]

\[
\frac{dH}{dt} = \alpha Q - (1 + \gamma_2 + \delta_2) H + \phi I - \frac{H}{N} \frac{dN}{dt} := f_4,
\]

\[
\frac{dR}{dt} = \gamma_1 I + \gamma_2 H + \xi E + \xi Q - \theta R - R - \frac{R}{N} \frac{dN}{dt} := f_5,
\]

\[
\frac{dN}{dt} = 1 - (1 + \delta_1 I + \delta_2 H) N := f_6;
\]

where

\[
\beta = \frac{\tilde{\beta}}{\mu}, \quad \delta_1 = \frac{\tilde{\delta}_1}{\mu}, \quad \delta_2 = \frac{\tilde{\delta}_2}{\mu}, \quad k = \frac{\tilde{k}}{\mu}, \quad \sigma = \frac{\tilde{\sigma}}{\mu}, \quad \xi = \frac{\tilde{\xi}}{\mu},
\]

\[
\alpha = \frac{\tilde{\alpha}}{\mu}, \quad \phi = \frac{\tilde{\phi}}{\mu}, \quad \gamma_1 = \frac{\tilde{\gamma}_1}{\mu}, \quad \gamma_2 = \frac{\tilde{\gamma}_2}{\mu}, \quad \theta = \frac{\tilde{\theta}}{\mu}.
\]
and the initial conditions:

\[ \begin{align*}
E(0) &= E_0 > 0, & Q(0) &= Q_0 > 0, & I(0) &= I_0 > 0, \\
H(0) &= H_0 > 0, & R(0) &= R_0 > 0, & N(0) &= N_0 > 0. 
\end{align*} \]  

(14)

In the following sections, we will study the dynamical behavior of the system (8)–(13) with initial condition (14).

3. Dynamical behavior of the system

In this section, we calculate all feasible steady states and the basic reproduction number for the system. Observe that the biologically feasible region for the non-dimensional system is

\[ \Omega = \{ (E, Q, I, H, R, N): 0 \leq E, Q, I, H, R, N \leq 1 \}, \]

which is positively invariant for the system (8)–(13). We consider only solutions with initial conditions inside the region \( \Omega \).

3.1. Local stability of disease-free equilibrium

The system (8)–(13) always has the disease-free equilibrium (DFE) \( (E^0 = (0, 0, 0, 0, 0, 1)) \). The local stability of DFE \( E^0 \) will be explored using the effective reproduction number \( R_C \). The non-negative matrix \( F \), of the new infection terms, and the matrix \( V \), of the remaining terms are given, respectively, by

\[
F = \begin{pmatrix}
0 & 0 & \beta & \beta \eta \\
0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 \\
\end{pmatrix},
\]

and

\[
V = \begin{pmatrix}
E(-1 + \frac{1}{N} - I\delta_1 - H\delta_2) + E(1 + k + \xi + \sigma) \\
-Ek + Q(-1 + \frac{1}{N} - I\delta_1 - H\delta_2) + Q(1 + \alpha + \xi) \\
I(-1 + \frac{1}{N} - I\delta_1 - H\delta_2) - E\sigma + I(1 + \gamma_1 + \delta_1 + \phi) \\
-Q\alpha + H(1 + \gamma_2 + \delta_2) + H(-1 + \frac{1}{N} - I\delta_1 - H\delta_2) - I\phi \\
\end{pmatrix}.
\]

The corresponding linearized matrices evaluated at the DFE \( E^0 \) are

\[
F = \begin{pmatrix}
0 & 0 & \beta & \beta \eta \\
0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 \\
\end{pmatrix},
\]

and

\[
V = \begin{pmatrix}
1 + k + \xi + \sigma & 0 & 0 & 0 \\
-k & 1 + \alpha + \xi & 0 & 0 \\
-\sigma & 0 & 1 + \gamma_1 + \delta_1 + \phi & 0 \\
0 & -\alpha & -\phi & 1 + \gamma_2 + \delta_2 \\
\end{pmatrix}.
\]
respectively. It follows that

\[
FV^{-1} = \begin{pmatrix}
\frac{\beta(1 + b_3\eta)}{b_1(1 + k + \xi + \sigma)} & \frac{\alpha\beta\eta}{(1 + \gamma_2 + \delta_2)(1 + \alpha + \xi)} & \frac{\beta}{1 + \gamma_1 + \delta_1 + \phi} & \frac{\eta\phi}{1 + \gamma_2 + \delta_2} & \frac{\beta\eta}{1 + \gamma_2 + \delta_2} \\
0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0
\end{pmatrix}.
\]

The effective reproduction number \( R_C = \rho(FV^{-1}) \), where \( \rho \) is the spectral radius, is given by

\[
R_C = \frac{\beta(1 + b_3\eta)}{b_1(1 + k + \xi + \sigma)} = \frac{\beta\sigma}{(1 + k + \xi + \sigma)(1 + \gamma_1 + \delta_1 + \phi)} \left(1 + \frac{\eta(\sigma\phi(1 + \alpha + \xi) + k\alpha(1 + \gamma_1 + \delta_1 + \phi))}{(1 + \gamma_2 + \delta_2)(1 + \alpha + \xi)}\right),
\]

where

\[
b_1 = \frac{1 + \gamma_1 + \delta_1 + \phi}{\sigma}, \quad b_2 = \frac{k(1 + \gamma_1 + \delta_1 + \phi)}{(1 + \alpha + \xi)\sigma} \quad \text{and} \quad b_3 = \frac{b_2\alpha + \phi}{1 + \gamma_2 + \delta_2}.
\]

It is worth mentioning that in the absence of a combined quarantine and isolation program \((k = 0, \alpha = 0, \phi = 0, \delta_2 = 0, \gamma_2 = 0)\), the effective reproduction number reduces to the basic reproduction number

\[
R_0 = \frac{\beta\sigma}{(1 + \xi + \sigma)(1 + \gamma_1 + \delta_1)}.
\]

Using Theorem 2 in [12], one can establish the following result.

**Theorem 3.1.** The disease-free equilibrium of the system (8)–(13) is locally-asymptotically stable if \( R_C < 1 \), and unstable if \( R_C > 1 \).

Biologically speaking, \( R_C \) represents the average number of secondary infections produced by a typical infected individual in a community that adopts isolation and quarantine programs. The epidemiological implication of this result is that if \( R_C < 1 \), then the influx of a few infected individuals will not generate large outbreaks (and the disease will die out). Disease outbreak will occur if \( R_C > 1 \).

### 3.2. Existence and local stability of endemic equilibrium

The possible endemic equilibria of proposed model are derived by solving the system of non-linear equations obtained from the system (8)–(13) equating the derivatives to zeroes. The endemic equilibrium \( E = (E^*, Q^*, I^*, H^*, R^*, N^*) \) of the model (8)–(13) is given by

\[
E^* = b_1 I^*, \quad Q^* = b_2 I^*, \quad H^* = b_3 I^*, \quad R^* = b_4 I^*, \quad N^* = \frac{1}{1 + \delta_1 I^* + b_3 \delta_2 I^*},
\]

where

\[
b_4 = \frac{\gamma_1 + b_3 \gamma_2 + (b_1 + b_2)\xi}{1 + \theta}, \quad b_5 = 1 + b_1 + b_2 + b_3 + b_4 \quad \text{and} \quad m = m_1 + m_2 b_4.
\]

The value of \( I^* \) is given by the solution of the equation

\[
1 - b_5 I^* = e^{m I^*} / R_C.
\]
In case, there is no media effect, i.e., \( m = 0 \), we get \( I^* = \frac{1}{b_5}(1 - \frac{1}{R_C}) \). It follows that \( I^* \) exists at positive level (and so as the unique endemic equilibrium \( \bar{E} \)) if and only if \( R_C > 1 \). Otherwise, the value of \( I^* \) is given from Eq. (15). Now we establish the existence of \( I^* \) for \( R_C > 1 \) though graphical approach. In Figs. 3 and 4, we plot the curve \( \frac{mI^*}{R_C} \) and straight line \( 1 - b_5I^* \) against \( I^* \) in the range \([0-1]\). Note that since \( b_5 > 1 \), we have \( 1/b_5 < 1 \). From Fig. 3, it follows that there is no point of intersection when \( R_C \leq 1 \), resulting the non-existence of the endemic equilibrium, but as \( R_C > 1 \), \( I^* \) exists uniquely at positive level and hence the unique endemic exists in this case (see Fig. 4).

From the above discussion we conclude that

**Theorem 3.2.** The system (8)–(13) has no endemic equilibrium for \( R_C \leq 1 \), but has a unique endemic equilibrium \( \bar{E} \) if \( R_C > 1 \).

Now we will state and prove the local stability of the endemic equilibrium in the following theorem:

**Theorem 3.3.** The endemic equilibrium \( \bar{E} \) is locally asymptotically stable for \( R_C > 1 \), but close to 1.
**Proof.** The Jacobian matrix $J_0$ at DFE is given by

$$
egin{pmatrix}
-1 - k - \xi - \sigma & 0 & \beta & \beta \eta & 0 & 0 \\
1 & -1 - \alpha - \xi & 0 & 0 & 0 & 0 \\
\sigma & 0 & -1 - \gamma_1 - \delta_1 - \phi & 0 & 0 & 0 \\
0 & \alpha & \phi & -1 - \gamma_2 - \delta_2 & 0 & 0 \\
\xi & \xi & \gamma_1 & \gamma_2 & -1 - \theta & 0 \\
0 & 0 & -\delta_1 & -\delta_2 & 0 & -1
\end{pmatrix}.
$$

Here, we use the method based on the central manifold theory to establish the local stability of endemic equilibrium taking $\beta$ as bifurcation parameter [4]. A critical value of bifurcation parameter $\beta$ at $R_C = 1$ is given as

$$
\beta_c = \frac{(1 + \gamma_2 + \delta_2)(1 + \alpha + \xi)(1 + k + \xi + \sigma)(1 + \gamma_1 + \delta_1 + \phi)}{k \alpha \xi (1 + \gamma_1 + \delta_1 + \phi) + (1 + \alpha + \xi) \sigma (1 + \gamma_2 + \delta_2 + \eta \phi)}.
$$

It can be easily verified that the Jacobian $J_0$ at $\beta = \beta_c$ has a right eigenvector (corresponding to the zero eigenvalue) given by $W = (w_1, w_2, w_3, w_4, w_5, w_6)^T$, where

$$
w_1 = 1 + \gamma_1 + \delta_1 + \phi, \quad w_2 = \frac{k(1 + \gamma_1 + \delta_1 + \phi)}{1 + \alpha + \xi}, \quad w_3 = \sigma,
$$

$$
w_4 = \frac{(1 + \alpha + \xi) \sigma \phi + k \alpha (1 + \gamma_1 + \delta_1 + \phi)}{(1 + \gamma_2 + \delta_2)(1 + \alpha + \xi)},
$$

$$
w_5 = \frac{k(\alpha \gamma_2 + (1 + \gamma_2 + \delta_2) \xi)(1 + \gamma_1 + \delta_1 + \phi) + (1 + \alpha + \xi)}{(1 + \theta)(1 + \alpha + \xi)(1 + \gamma_2 + \delta_2)} \times (\gamma_1(1 + \gamma_2 + \delta_2) \sigma + \gamma_2 \sigma \phi + (1 + \gamma_2 + \delta_2) \xi(1 + \gamma_1 + \delta_1 + \phi)) \quad \text{and}
$$

$$
w_6 = -\frac{k \alpha \phi (1 + \gamma_1 + \delta_1 + \phi) + (1 + \alpha + \xi) \sigma (\delta_1(1 + \gamma_2 + \delta_2) + \delta_2 \phi)}{(1 + \gamma_2 + \delta_2)(1 + \alpha + \xi)}.
$$

Furthermore, the components of the left eigenvector (corresponding to the zero eigenvalue), $V = (v_1, v_2, v_3, v_4, v_5, v_6)$, must satisfy the equalities $V J_0 = 0$ and $V W = 1$, so that we obtain

$$
v_1 = \frac{v_4(k \alpha \eta (1 + \gamma_1 + \delta_1 + \phi) + (1 + \alpha + \xi) \sigma (1 + \gamma_2 + \delta_2 + \eta \phi))}{\eta (1 + \alpha + \xi)(1 + k + \xi + \sigma)(1 + \gamma_1 + \delta_1 + \phi)},
$$

$$
v_2 = \frac{v_4 \alpha}{1 + \alpha + \xi}, \quad v_3 = \frac{v_4(1 + \gamma_2 + \delta_2 + \eta \phi)}{\eta (1 + \gamma_1 + \delta_1 + \phi)}, \quad v_4 = \frac{1}{w_4 + B_1 + B_2}, \quad v_5 = 0 \quad \text{and} \quad v_6 = 0,
$$

where

$$
B_1 = \frac{k \alpha \phi (1 + \gamma_1 + \delta_1 + \phi) + (1 + \alpha + \xi) \sigma (1 + \gamma_2 + \delta_2 + \eta \phi)}{(1 + \alpha + \xi)^2},
$$

$$
B_2 = \frac{k \alpha \eta (1 + \gamma_1 + \delta_1 + \phi) + (1 + \alpha + \xi) \sigma (1 + \gamma_2 + \delta_2 + \eta \phi)}{\eta (1 + \alpha + \xi)(1 + k + \xi + \sigma)}.
$$

Use the notations $x_1 \equiv E$, $x_2 \equiv Q$, $x_3 \equiv I$, $x_4 \equiv H$, $x_5 \equiv R$, $x_6 \equiv N$. Hence, we have

$$
a = \sum_{k,i,j=1}^6 v_k w_i w_j \frac{\partial^2 f_k(0,0)}{\partial x_i \partial x_j} \quad \text{and}
$$
Substituting the values of all the second order derivatives evaluated at DFE and $\beta = \beta_c$, we get

$$a = v_1 (2w_1 w_6 - 2w_2 w_3 \beta_c - 2w_3 w_5 \beta_c + w_3^2(-2\beta_c - 2m_1 \beta_c) + 2w_1 w_3(-\beta_c + \delta_1) \nonumber$$

$$- 2w_2 w_4 \beta_c \eta - 2w_4 w_5 \beta_c \eta + 2w_1 w_4 (\delta_2 - \beta_c \eta) + w_2^2(-2\beta_c \eta - 2m_2 \beta_c \eta) \nonumber$$

$$+ 2w_3 w_4(-\beta_c - m_2 \beta_c - \beta_c \eta - m_1 \beta_c \eta)) \nonumber$$

$$+ v_2(2w_2 w_6 + 2w_2 w_3 \delta_1 + 2w_2 w_4 \delta_2) \nonumber$$

$$+ v_3(2w_3 w_6 + 2w_3^2 \delta_1 + 2w_3 w_4 \delta_2) \nonumber$$

$$+ v_4(2w_4 w_6 + 2w_3 w_4 \delta_1 + 2w_4^2 \delta_2) \nonumber$$

$$+ v_5(2w_5 w_6 + 2w_3 w_5 \delta_1 + 2w_4 w_5 \delta_2) \nonumber$$

$$+ v_6(-2w_3 w_6 \delta_1 - 2w_4 w_6 \delta_2);$$

and

$$b = v_1 (w_3 + \eta w_4).$$

Finally, substituting the values of $V$ and $W$, we obtain

$$a = \frac{-2v_4 B_3 (1 + k + \xi + \sigma)}{(1 + \gamma_2 + \delta_2)(1 + \theta)(1 + \alpha + \xi)},$$

$$b = \frac{v_4 B_3^2 \eta (1 + k + \xi + \sigma)}{(1 + \gamma_2 + \delta_2)(1 + \alpha + \xi + \delta_1 + \phi)}.$$ 

where

$$B_3 = (k(\alpha(1 + m_2 + \gamma_2 + \theta + m_2 \theta) + (1 + \gamma_2 + \delta_2))(1 + \theta + \xi)) \nonumber$$

$$\times (1 + \gamma_1 + \delta_1 + \phi) + (1 + \alpha + \xi)((1 + \gamma_2 + \delta_2)((1 + \gamma_1 + \delta_1) \nonumber$$

$$\times (1 + \theta + \xi) + (1 + m_1 + \gamma_1 + \theta + m_1 \theta)\sigma) + ((1 + \gamma_2 + \delta_2) \nonumber$$

$$\times (1 + \theta + \xi) + (1 + m_2 + \gamma_2 + \theta + m_2 \theta)\sigma)\phi).$$

Since $a < 0$ and $b > 0$ at $\beta = \beta_c$, therefore using Theorem 4.1 and Remark 1 stated in [4], a transcritical bifurcation occurs at $R_C = 1$ and the unique endemic equilibrium is locally asymptotically stable for $R_C > 1$. \(\square\)

### 3.3. Global stability of DFE and uniform persistence

In this section, we analyze the global stability of the disease-free steady states for a special case. We state the following theorem:

**Theorem 3.4.** Suppose $R_C < 1$ and $\delta_1 = \delta_2 = 0$, then the disease-free equilibrium $E^0$ is globally asymptotically stable.
**Proof.** Here, we prove global stability of DFE applying the method used in [3]. When $\delta_1 = \delta_2 = 0$, we have $\frac{dN}{dt} = 1 - N$. Then $N \to 1$ as $t \to \infty$. Take the $N$ in the limiting case, i.e., $N = 1$, then the system (8)–(13) reduces to

\[
\begin{align*}
\frac{dE}{dt} &= \beta e^{-m_2 H - m_1 I} S(I + \eta H) - (1 + k + \sigma + \xi)E, \\
\frac{dQ}{dt} &= kE - (1 + \alpha + \xi)Q, \\
\frac{dI}{dt} &= \sigma E - (1 + \gamma_1 + \phi)I, \\
\frac{dH}{dt} &= \alpha Q + \phi I - (1 + \gamma_2)H, \\
\frac{dR}{dt} &= \gamma_1 I + \gamma_2 H + \xi E + \xi Q - \theta R - R.
\end{align*}
\]

Let $X = (R)$ and $Z = (E, Q, I, H)$, here $U_0 = (X^0, Z^0)$, where $X^0 = (0)$ and $Z^0 = (0, 0, 0, 0)$. We have

\[
\frac{dX}{dt} = F(X, Z) = \gamma_1 I + \gamma_2 H + \xi E + \xi Q - \theta R - R.
\]

At $Z = Z^0$, $G(X, 0) = 0$. Now $\frac{dX}{dt} = F(X, 0) = -(1 + \theta)X$, as $t \to \infty$, $X \to X^0$. Hence, $X = X^0$ ($= R^0 = 0$) is GAS. Thus, condition (H1) is satisfied. From Eqs. (20)–(23), we get

\[
\frac{dZ}{dt} = G(X, Z) = BZ - \hat{G}(X, Z),
\]

where

\[
B = \begin{pmatrix}
-1 - k - \xi - \sigma & 0 & \beta & \beta \eta \\
k & -1 - \alpha - \xi & 0 & 0 \\
\sigma & 0 & -1 - \gamma_1 - \phi & 0 \\
0 & \alpha & \phi & -1 - \gamma_2
\end{pmatrix}
\]

and

\[
\hat{G}(X, Z) = \begin{pmatrix}
\beta(I + \eta H)(1 - e^{-m_1 I - m_2 H S}) \\
0 \\
0 \\
0
\end{pmatrix}.
\]

Clearly, $B$ is an M-matrix. For $I \geq 0$, $H \geq 0$, we have $0 < e^{-m_1 I - m_2 H} \leq 1$, therefore $\hat{G}(X, Z) \geq 0$ since $0 \leq S \leq 1$. Thus both the conditions (H1) and (H2) are satisfied. Hence, the DFE $E^0$ is globally asymptotically stable if $R_C < 1$. $\Box$

Now, we explore the uniform persistence for the system (8)–(13). Again, the system (8)–(13) is said to be uniformly-persistent if there exists a constant $c$ such that any solution $(E(t), Q(t), I(t), H(t), R(t))$ satisfies

\[
\liminf_{t \to \infty} E(t) \geq c, \quad \liminf_{t \to \infty} Q(t) \geq c, \quad \liminf_{t \to \infty} I(t) \geq c, \\
\liminf_{t \to \infty} H(t) \geq c, \quad \liminf_{t \to \infty} R(t) \geq c, \quad \liminf_{t \to \infty} N(t) \geq c
\]

provided that $(E(0), Q(0), I(0), H(0), R(0), N(0)) \in \Omega$ [13].

Similar as in [41], we can state the following theorem for persistent:
Theorem 3.5. The system (8)–(13) is uniformly-persistent in \( \Omega \) if and only if \( R_C > 1 \).

Proof. From Theorem 3.1, the DFE of the model (8)–(13) is unstable whenever \( R_C > 1 \). Apply the uniform persistence result stated in [13], finally it can be proved in a similar manner as Proposition 3.3 of [24]. \qed

The consequence of this result is that in limiting case, all the infected state variables \( E, Q, I \) and \( H \) of the model will remain above a certain positive threshold and the disease will persist in the population.

4. Threshold analysis

In this section, the effect of quarantine and isolation on the transmission dynamics of the disease is measured qualitatively. A threshold analysis on the parameters associated with the quarantine of exposed individuals \( (k) \) and the isolation of the infected individuals \( (\phi) \) is performed by computing the partial derivatives of the effective reproduction number \( R_C \) with respect to these parameters. We observe that

\[
\frac{\partial R_C}{\partial k} = \frac{\alpha \beta \eta}{(1 + \gamma_2 + \delta_2)(1 + \alpha + \xi)(1 + k + \xi + \sigma)} \left( \beta(\kappa \alpha \eta(1 + \gamma_1 + \delta_1 + \phi) + (1 + \alpha + \xi)\sigma(1 + \gamma_2 + \delta_2 + \eta \phi)) \right)
\]

so that,

\[
\frac{\partial R_C}{\partial k} < 0 \quad (> 0) \quad \text{iff} \quad \eta < \eta_k \quad (\eta > \eta_k),
\]

where

\[
0 < \eta_k = \frac{(1 + \gamma_2 + \delta_2)(1 + \alpha + \xi)\sigma}{\alpha(1 + \gamma_1 + \delta_1)(1 + \xi + \sigma) + (1 + \xi)(\alpha - \sigma)\phi}.
\]

From the above analysis it is clear that if the relative infectiousness of the hospitalized individuals \( (\eta) \) does not exceed the threshold value \( \eta_k \), then quarantining of exposed individuals results in reduction of the effective reproduction number \( R_C \) and therefore, reduction in disease burden (new infections, hospitalization etc.). On the other hand, if \( \eta < \eta_k \), then due to increase in the rate of quarantine, the effective reproduction number \( R_C \) will increase and consequently, the disease burden also increases. Thus, the use of quarantine is detrimental in this case. The result is summarized as follows:

Theorem 4.1. For the model (8)–(13), the use of quarantine of the exposed individuals will have positive (negative) population-level impact if \( \eta < \eta_k \) \((\eta > \eta_k)\).

Similarly, the impact of isolation of infectious individuals is assessed by calculating the partial derivatives of \( R_C \) with respect to the isolation parameter \( \phi \). Thus, we obtain

\[
\frac{\partial R_C}{\partial \phi} = \frac{\beta \sigma(\eta(\gamma_1 + \delta_1 + 1) - (1 + \gamma_2 + \delta_2))}{(\gamma_2 + \delta_2 + 1)(\gamma_1 + \delta_1 + \phi + 1)^2(k + \xi + \sigma + 1)}.
\]

It follows that

\[
\frac{\partial R_C}{\partial \phi} < 0 \quad (> 0) \quad \text{iff} \quad \eta < \eta_\phi \quad (\eta > \eta_\phi),
\]
Table 2

Parameter values used in the simulation for the system (8)–(13).

| Parameter | Nominal values (per day) |
|-----------|--------------------------|
| $\Lambda$ | 136                      |
| $\tilde{\mu}$ | 0.0000351               |
| $m_1$ | 0.2                     |
| $m_2$ | 0.2                     |
| $\eta$ | 0.5                     |
| $\tilde{k}$ | 0.01                   |
| $\tilde{\sigma}$ | 0.1                  |
| $\tilde{\xi}$ | 0.0002                |
| $\delta_1$ | 0.04227            |
| $\delta_2$ | 0.027855            |
| $\tilde{\alpha}$ | 0.056986          |
| $\tilde{\gamma}_1$ | 0.03521            |
| $\tilde{\gamma}_2$ | 0.042553          |
| $\tilde{\phi}$ | 0.050619           |
| $\tilde{\beta}$ | [0.05, 0.4]          |

where

$$\eta_\phi = \frac{1 + \gamma_2 + \delta_2}{1 + \gamma_1 + \delta_1}.$$  

Therefore, the use of isolation of infected individuals will be helpful to control the disease in the community if the relative infectiousness of the hospitalized individuals ($\eta$) does not exceed the threshold $\eta_\phi$. The result is summarized below:

**Theorem 4.2.** *For the model (8)–(13), the use of isolation of infectious individuals will have positive (negative) population-level impact if $\eta < \eta_\phi$ ($\eta > \eta_\phi$).*

Hence, we conclude that the combine use of quarantine of exposed individuals and isolation of individuals with symptoms will have positive population-level impact if and only if

$$\eta < \min\{\eta_k, \eta_\phi\}. \quad (25)$$

The quarantine and isolation strategies will have negative population-level impact if

$$\eta > \max\{\eta_k, \eta_\phi\}. \quad (26)$$

The effective reproduction number $R_C$ is a decreasing (non-decreasing) function of the quarantine and isolation parameters $k$ and $\phi$ if condition (25) [(26)] is satisfied (see Figs. 13 and 14 obtained from simulation of the model in which the results are consistent with the analytical findings discussed earlier).

5. **Numerical simulation**

In this section, we provide numerical simulations to illustrate previously established results with the biological feasible parametric values as shown in Table 2. Most of the values of parameters are taken in the reference from the existing literature ([39] and references therein) and the rest of the parametric values are assumed for numerical computation. The system (8)–(13) is simulated taking initial population size $S_0 = 400$, $E_0 = 250$, $Q_0 = 60$, $I_0 = 100$, $H_0 = 40$, $R_0 = 150$ and hence $\tilde{N} = 1000$. The corresponding initial condition in non-dimensional form is $S_0 = 0.40$, $E_0 = 0.25$, $Q_0 = 0.06$, $I_0 = 0.10$, $H_0 = 0.04$, $R_0 = 0.15$ and $N_0 = 0.0026$. For $\tilde{\beta} = 0.09$, the effective reproduction number $R_C$ is 0.9238 (i.e., $R_C < 1$), in this
The variation of the scaled population in scaled-time, taking $m_1 = m_2 = 0.2$ and $\tilde{\beta} = 0.09$ with $\mathcal{R}_C = 0.9238 < 1$.

Fig. 6. The variation of the scaled population in scaled-time, taking $m_1 = m_2 = 0.2$ and $\tilde{\beta} = 0.4$ with $\mathcal{R}_C = 4.1058 > 1$. The coefficients of media coverage $m_1$ and $m_2$ should depend on the disease under consideration, the social structure (education, awareness, responsiveness, economy, etc.) of the population and the NPIs used in a particular region. Here, we use the formula $m_j = -\log_e (p + q_j - pq_j)$, to quantify the coefficients $m_1$ and $m_2$ of media coverage, where $q_j$ quantifies the response of the population aware to media recommended NPIs with respect to the number of infective and the hospitalized individuals. If people are not responding to media alert, then $q_j = 1$ and if all the people are adopting the recommended NPIs, then $q_j = 0$. It is assumed that the disease transmission rate can be reduced by $p$ fraction when all individuals follow the
media recommended NPIs to protect themselves. In general, the impact of reported number of hospitalized individuals is more as compared to the number of infectives on people’s response to NPIs. For different combinations of values of $p$ and $q$, the estimated values of $m$ are shown in Fig. 7.
It is observed from the analysis that the coefficients of media coverage $m_1$ and $m_2$ do not affect $R_C$ and the qualitative features of the model remain unaltered. From (18), we observe that $a$ is always negative, which precludes the existence of backward bifurcation in the system and hence ensures transcritical (i.e., forward) bifurcation about $R_C = 1$. Hence in this case, the classical requirement of $R_C < 1$ is necessary and sufficient for disease control. Moreover, from (15) we observe that

\[
\frac{\partial I^*}{\partial m} = -\frac{e^{mI^*}}{b_5 R_C + me^{mI^*}} < 0.
\]

One can easily observe that the use of NPIs stimulated by media coverage helps to mitigate the disease burden from the environment by lowering the level of infectious individuals at steady state. The effect of $m_1$ and $m_2$ on the fraction of infectious individuals ($I$) is shown in Figs. 10 and 11 taking $\tilde{\beta} = 0.09$, and $\tilde{\beta} = 0.4$, respectively and the rest of the parametric values are as in Table 2. It is observed that the level of endemic equilibrium is significantly affected by media coefficients $m_1$ and $m_2$.

The pre-existing immunity in the population has significant role in the disease outbreak as it lowers the basic as well as effective reproduction numbers. The effect of pre-existing immunity parameter $\tilde{\xi}$ on the
number of infectious individuals over time is shown in Fig. 12. It is clear from the figure that higher level of pre-existing immunity helps to reduce disease burden.

The effectiveness of quarantine and isolation depends on the size of the modification parameter ($\eta$) for the reduction in infectiousness of hospitalized individuals. For $\bar{\beta} = 0.04$, the threshold value of $\eta$ with respect to quarantine parameter $k$ is $\eta_k = 0.91$ and with respect to isolation parameter $\phi$ is $\eta_\phi = 0.90$. From Fig. 13, it is clear that quarantine $k$ has positive population-level impact ($R_C$ decreases with increase in $k$) for $\eta < 0.91$ and have negative population level impact for $\eta > 0.91$. Similarly for $\eta < 0.90$, isolation has positive level impact, whereas isolation has negative impact if $\eta > 0.90$ (see Fig. 14).

6. Sensitivity analysis

In this section, we perform sensitivity analysis of effective reproduction number $R_C$ and endemic equilibrium taking parametric values given in Table 2. Sensitivity indices allow us to measure the relative change in a state variable/derived parameter when a model parameter changes.
The sensitivity indices, \( \Upsilon_{R_C}^{y_j} = \frac{\partial R_C}{\partial y_j} \times \frac{y_j}{R_C} \), of the effective reproduction number \( R_C \) to the parameters, \( y_j \), for parameter values given in Table 2.

| Parameter \((y_j)\) | Sensitivity index of \( R_C \) w.r.t. \( y_j \) \((\Upsilon_{R_C}^{y_j})\) |
|---------------------|-------------------------------------------------|
| \( \beta \)        | +1.0000                                         |
| \( \eta \)         | +0.3103                                         |
| \( k \)            | −0.0282                                         |
| \( \sigma \)       | +0.0304                                         |
| \( \zeta \)        | −0.0020                                         |
| \( \delta_1 \)     | −0.3093                                         |
| \( \delta_2 \)     | −0.1227                                         |
| \( \alpha \)       | 0.0003                                          |
| \( \gamma_1 \)     | −0.2576                                         |
| \( \gamma_2 \)     | −0.1874                                         |
| \( \phi \)         | −0.1225                                         |
| \( \theta \)       | 0                                               |
| \( m_1 \)          | 0                                               |
| \( m_2 \)          | 0                                               |

**Definition.** (See [6,44].) The normalized forward sensitivity index of a variable, \( u \), that depends on a parameter, \( p \), is defined as:

\[
\Upsilon_{p}^{u} = \frac{\partial u}{\partial p} \times \frac{p}{u}.
\]

Estimation of highly sensitive parameter should be done very carefully, because a small variation in the parameter will lead to relatively large quantitative change. On the other hand, a less sensitive parameter does not require as much effort to estimate, since a small variation in that parameter will not produce large change to the quantity of interest.

The normalized sensitive indices of effective reproduction number \( R_C \) with respect to parameters are shown in Table 3. From Table 3, we observe that \( \beta \), \( \eta \), \( \sigma \), and \( \alpha \) have positive impact on \( R_C \) and the rest of the parameters have negative impact. For example, 10% increase (decrease) in \( \sigma \), resulting in 0.304% increase (decrease) in \( R_C \), on the other hand 10% increase (decrease) in \( \gamma_1 \), will decrease (increase) \( R_C \) by 2.576%. Moreover, parameters \( \beta \), \( \delta_1 \) and \( \gamma_1 \) are most sensitive to \( R_C \), hence we observe significant change in \( R_C \) by small changes in these parameters.
Again, we perform sensitivity analysis of state variables at endemic steady state with respect to model parameters. Sensitivity indices of state variables at endemic equilibrium are shown in Table 4 using parametric values shown in Table 2. From Table 4, we observe that parameters $\beta$, $\eta$, $\delta_2$, $\alpha$, $\theta$ and $\sigma$ have positive impact on $I^*$ and the rest of the parameters have negative impact. Moreover, parameters $\phi$, $\sigma$, $\beta$, $\gamma_1$, $\theta$ and $\delta_1$ are most sensitive parameters to $I^*$, hence we observe significant change in $I^*$ by small changes in these parameters.

7. Result and discussion

An SEQIHRs epidemic model for the transmission dynamics of an infectious disease is proposed and rigorous mathematical analysis is carried out to get insight into the qualitative dynamics in presence of pre-existing immunity and the use of NPIs stimulated by media coverage. The main mathematical and epidemiological findings of the proposed model presented in this article are as follows:

(i) The model (2)–(7) has a locally–asymptotically stable disease-free equilibrium whenever the associated effective reproduction number is less than unity (Theorem 3.1). Moreover, if the disease-induced death rates are neglected then the DFE is globally–asymptotically stable (Theorem 3.4).

(ii) The model has a unique endemic equilibrium whenever the effective reproduction number exceeds unity and then EE is locally asymptotically stable (Theorems 3.2 and 3.3).

(iii) The presence of pre-existing immunity ($\xi$) in the population has significant impact on the transmission dynamics of the disease. Higher level of pre-existing immunity in the population decreases the infection peak and causes its early arrival.

(iv) The coefficients of media awareness $m_1$ and $m_2$ do not affect the effective reproduction number $R_C$. Hence, it does not change the qualitative behavior of the model, but it helps to mitigate disease burden by lowering the level of infection over time.

(v) The use of quarantine and/or isolation could have positive, no or negative population level impact depending on the relative infectiousness of isolated individuals ($\eta$).

(vi) Since disease transmission is directly related to the effective reproduction number, and the disease prevalence is directly related to the endemic equilibrium point $E$, specifically to the magnitude of $I^*$, therefore normalized forward sensitivity indices of the effective reproduction number and $I^*$ will be helpful to determine decisive parameter(s) for containing the infectious disease. Sensitivity indices of the effective reproduction number and $I^*$ are calculated and highly sensitive parameters are discovered.
Acknowledgments

The authors are grateful to the anonymous reviewers for their helpful suggestions and comments that have improved the quality and presentation of the manuscript.

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