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Modeling the transmission dynamics of middle eastern respiratory syndrome coronavirus with the impact of media coverage

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ABSTRACT

Middle East respiratory syndrome coronavirus has been persistent in the Middle East region since 2012. In this paper, we propose a deterministic mathematical model to investigate the effect of media coverage on the transmission and control of Middle Eastern respiratory syndrome coronavirus disease. In order to do this we develop model formulation. Basic reproduction number \( R_0 \) will be calculated from the model to assess the transmissibility of the (MERS-CoV). We discuss the existence of backward bifurcation for some range of parameters. We also show stability of the model to figure out the stability condition and impact of media coverage. We show a special case of the model for which the endemic equilibrium is globally asymptotically stable. Finally all the theoretical results will be verified with the help of numerical simulation for easy understanding.

Introduction

Infectious diseases are responsible for a quarter of all death in the world annually, such as SARS, MERS, and now COVID-19, that exhibit some distinct features such as rapid spread and visible symptoms [1–3]. One of the initiatives is to inform individuals through media and education as quickly as possible the right preventive understanding of the disease. We understand from experience that the more preventive the inhabitants understand, the better they can stop the disease from spreading.

In recent years, there have been global fears over viruses. Outbreaks of MERS, SARS, or influenza A are deadly and spread fast. It is one area where the media can play a life-saving role. Timely, accurate information about the nature of the illness, and about where the outbreaks were occurring, led to a widespread panic. The research focused on media coverage of the 2015 Middle East respiratory syndrome (MERS) crisis in South Korea.

In particular by the country’s three major terrestrial television stations: KBS and MBC, both public broadcasters, and SBS, a commercial channel. Coverage of the MERS outbreak by these three television stations between May and July 2015 was examined. The author also interviewed eleven journalists and editors, via email and the messaging app ‘Kakao talk’ voice function, between 7th April to 7th June 2017. Other interviews were conducted in the UK, via events at the Refuters Institute for the Study of Journalism in Oxford. Mathematical modeling and analysis are used for the dynamics of infectious diseases, see for instance [12–19]. There have been mathematical modeling studies to analyze the impact of media coverage on the spread and control of infectious disease in a given population. In mathematical epidemiology patient suffering symptoms of a common cold, but could kill within two weeks. The virus had first spread in Saudi Arabia, where 40 percent of those who contracted it died, [9–11]. In South Korea 186 people were infected and 38 of those died within two months of falling ill. A lack of information about the nature of the illness, and about where the outbreaks were occurring, led to a widespread panic. The research focused on media coverage of the 2015 Middle East respiratory syndrome (MERS) crisis in South Korea.

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the role of media communication in alerting the outcome of infectious disease outbreak, continuously having a place. The paper aimed to analyse the flow of information during an epidemic and to understand the impact of media coverage on the transmission of infectious and hospitalized individuals.

In [20], the authors extend the classical SEI model by considering a new incidence functional which reflects the impact of the media coverage to the spreading and control of the disease. The incidence function has been considered to play a key role in ensuring that the model indeed give reasonable qualitative description of the transmission dynamics of the diseases. We consider the model of [20], by taking the hospitalize class.

The paper is arrange as follow: In Section “A SIHRS model of MERS-CoV with media coverage”, we discuss formulation of the model, disease free equilibrium and reproductive number. In Section “Endemic equilibria and backward bifurcation”, we discuss endemic equilibria and the existence of backward bifurcation for some range of parameter. In Section “Sensitivity analysis”, we discuss sensitivity analysis of the model. In Section “Stability analysis”, we find local and global stability analysis. In Section “A Special case of model (1) with $\gamma = 0$”, we discuss a special case of the model and obtained global asymptotic stability of the proposed model. In Section “Numerical simulation”, we discuss numerical simulation of the proposed model. In Section “Discussion”, we give discussion on the obtained results.

A SIHRS model of MERS-CoV with media coverage

We examine the transmission of MERS CoV in a specified region. We distribute the population into the following compartments. The susceptible $S(t)$.

- All new born will goes to the susceptible class only,
- The infected $I(t)$ who are infectious,
- The hospitalize $H(t)$,
- The recovered population $R(t)$.

To incorporate the effect of the behavioral changes of the susceptible individuals, we used a non-linear incidence rate. The mathematical model based on SIHR model with the incident of disease.

$$ \frac{dS}{dt} = \pi - (\beta_1 - \beta_3) \frac{I}{m+I} S \pi - (\beta_1 - \beta_3) \frac{H}{m+H} S \pi + \gamma R - \mu_S S,$$

$$ \frac{dI}{dt} = (\beta_1 - \beta_3) \frac{I}{m+I} S \pi + (\beta_3) \frac{H}{m+H} S \pi + (q + \kappa + \mu_I) I,$$

$$ \frac{dH}{dt} = qI - (\alpha + \mu_H) H,$$

$$ \frac{dR}{dt} = kI + \alpha H - (\mu_R + \gamma) R,$$

with

$$ S(0) > 0, I(0) > 0, H(0) > 0, R(0) > 0. $$

In model (1).

- $\pi$ is the recruitment rate of susceptible population.
- $\mu_S$ represent natural death rate and $\kappa$ is death rate occur due to disease.
- $\beta_1, \beta_3$ are the contact rate before media alert.
- $\beta_1 - \beta_3 \frac{I}{m+I}$ and $\beta_3 - \beta_3 \frac{H}{m+H}$ are the contact rate after media alert.

Disease free equilibrium and basic reproductive number

The model (1) have a disease free equilibrium denoted by $E^0$ and given by $E^0 = (S^0, 0, 0, 0)$, where the components are define as; $S^0 = \frac{\pi}{\mu_S}$.

Basic reproductive number a threshold representing how many secondary infections results from the introduction of one infected individuals in a susceptible population. For the basic reproductive number $R_0$, we use the method of Driessche and Watmough [21].

$$ F = \begin{bmatrix} \beta_1 \pi \mu_S & \beta_1 \pi \mu_S & 0 \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{bmatrix}, \quad V = \begin{bmatrix} (q + \kappa + \mu_I) & 0 \\ -q \epsilon & (\alpha + \mu_H) \end{bmatrix}. $$

$$ FV^{-1} = \begin{bmatrix} \beta_1 \pi \mu_S \mu_I(q + \kappa + \mu_I) + \beta_1 \pi \mu_S \mu_I(\alpha + \mu_H)(q + \kappa + \mu_I) & \beta_1 \pi \mu_I(\alpha + \mu_H) \\ 0 & 0 \end{bmatrix}. $$

then $R_0$ is the spectral radius of $FV^{-1}$, that is

$$ R_0 = \frac{\beta_1 \pi \mu_I(q + \kappa + \mu_I) + \beta_1 \pi \mu_I(\alpha + \mu_H)(q + \kappa + \mu_I)}{\mu_I(\alpha + \mu_H)}. $$

The basic reproduction number $R_0$ of this model consists of two parts, representing the two different transmission routes; i.e., from the infected individuals, from hospitalized individuals before media alert. Where $\beta_1$, $\beta_3$ show contact rate before media alert.

Theorem 1. The solution of the model (1) is bounded.

Proof. The total population is represented by $N(t)$ that is $N(t) = S(t) + I(t) + H(t) + R(t)$. Differentiation $N(t)$ with time and setting the expression for $\frac{dS}{dt}, \frac{dI}{dt}, \frac{dH}{dt}, \frac{dR}{dt}$, we get

$$ \frac{dN(t)}{dt} = \pi - \mu_S S - \mu_I I - \mu_H H - \mu_R R. $$

$$ \Omega_0 = \left\{ \left( S(t), I(t), H(t), R(t) \right)^T, 0 < S(t) + I(t) + H(t) + R(t) \leq \frac{\pi}{\mu_S} \right\} $$

Endemic equilibria and backward bifurcation

Suppose the left hand side of each differential Eq. (1) be zero, the endemic $(S, I, H, R)$ satisfies $S > 0, I > 0, H > 0, R > 0$ and

![Bifurcation diagram of model (1) showing backward bifurcation.](image-url)
\[ S^* = \frac{q + \kappa + \mu_0}{\beta_1 - \beta_2 m + 1} + \frac{\beta_1 (\alpha + \mu_0) - \beta_4 (\kappa \varepsilon)^2}{\alpha + \mu_0 (m (\alpha + \mu_0) + \kappa \varepsilon)} \]

\[ H^* = \frac{q \varepsilon}{\alpha + \mu_0} \]

\[ R^* = \frac{\kappa (\alpha + \mu_0) + q \alpha \varepsilon}{(\mu_0 + \gamma) (\alpha + \mu_0)} \]

putting the above expression into the first equation of model (1) and after simplification, we have

\[ a B^2 + b B + c. \] (7)

\[ a = (\beta_1 - \beta_2) \frac{\gamma (\kappa + \mu_0) + q (q + \kappa + \mu_0) (R_0 - 1)}{(\mu_0 + \gamma)} + (\beta_2 - \beta_4) \frac{\gamma (\kappa + \mu_0)}{\mu_0 + \gamma} \]

\[ b = -\gamma \beta_1 m - \beta_3 m (q + \kappa + \mu_0) - \frac{\gamma \beta_2 m}{\mu_0 + \gamma} - (1 - R_0) \]

\[ c = \mu_0 m (q + \kappa + \mu_0) (R_0 - 1). \]

If \( R_0 > 1 \), then \( c > 0, a > 0 \), it follow that the model (1) get a unique endemic equilibrium \( E' (S^*, I^*, H^*, R^*) \). If \( R_0 < 1 \), we obtain \( a < 0, b < 0, c < 0 \) which does not have any endemic equilibrium.

The significance of backward bifurcation in the epidemiological model is that of the classical requirement of the basic reproduction number \( R_0 \) to be less than one [22,23], while necessary for the elimination of the MERS CoV virus from population. The presence of backward bifurcation in the proposed model suggests that the feasibility of MERS virus elimination, when the basic reproduction number is less than one, depends on the initial size of the sub population of the model.

For \( R_0 = 1 \), the following result holds (Fig. 1).

**Lemma 1.** If \( R_0 = 1 \), the model (1) posses the phenomena of backward bifurcation if \( c < 0 \).

**Sensitivity analysis**

We bring out sensitivity analysis of parameters using in the proposed model. This analysis will make it easy to know the parameters that have a essentially effect on reproductive number. We apply the technic given in [24,25] and given by,

\[ \Delta R_0^h = \frac{\partial R_0}{\partial h} h R_0 \]

where \( h \) is parameter.

![Fig. 2. The variation of different parameters and its effect on the basic reproductive number.](image-url)
\[ \Delta R_0 = \frac{\partial R_0}{\partial \pi} = - \frac{\beta_1 \pi}{\mu_0} \approx 0.99999999 > 0, \]
\[ \Delta R_0 = \frac{\partial R_0}{\partial \mu_0} = - \frac{\beta_1 \pi}{\mu_0} \approx 0.000999899 > 0, \]
\[ \Delta R_0 = \frac{\partial R_0}{\partial \mu_0} = \beta_1 \pi (\alpha + \mu_0) + \beta_1 \pi (q + k + \mu_0) \approx -0.64516294 < 0, \]
\[ \Delta R_0 = \frac{\partial R_0}{\partial \pi} = \beta_1 \pi (\alpha + \mu_0) + \beta_1 \pi (q + k + \mu_0) \approx -0.0321580 < 0, \]
\[ \Delta R_0 = \frac{\partial R_0}{\partial \mu_0} = -1.3226639 < 0, \]
\[ \Delta R_0 = \frac{\partial R_0}{\partial \pi} = -0.00099999 < 0, \]
\[ \Delta R_0 = \frac{\partial R_0}{\partial \mu_0} = 0.99999999 > 0. \]

**Stability analysis**

To examine the local and global stability analysis of the model (1) about \( E^0 = (S^0, I^0, H^0, R^0) \), we use the following results. Fig. 2.

| Parameter | Sensitivity indices | Parameter | Sensitivity indices |
|-----------|--------------------|-----------|--------------------|
| \( \rho_1 \) | + | \( \epsilon \) | + |
| \( \kappa \) | - | \( q \) | - |
| \( \alpha \) | + | \( \mu_0 \) | - |
| \( \beta_3 \) | + | \( \pi \) | + |

**Theorem 2.** We take the model (1) with all positive parameters. For \( R_0 > 1 \) the model (1) possesses a unique endemic equilibrium \( E^* = (S^*, I^*, H^*, R^*) \) and is locally asymptotically stable. For \( R_0 < 1 \) the model (1) get a unique disease free equilibrium \( E_0 = (S_0, I_0, H_0, R_0) \) \& is globally asymptotically stable.

**Proof.** The Jacobian matrix of the suggested model (1) about the DFE point \( E^0 \) is

\[
J = \begin{pmatrix}
\frac{\partial \mu_0}{\partial \mu_0} & \frac{\partial \beta_1 \pi}{\partial \mu_0} & \frac{\partial \beta_2 \pi}{\partial \mu_0} \\
\frac{\partial \beta_1 \pi}{\partial \mu_0} & \frac{\partial \beta_2 \pi}{\partial \mu_0} & \gamma \\
\frac{\partial \beta_2 \pi}{\partial \mu_0} & \gamma & 0 \\
\end{pmatrix}
\]

The first two eigenvalues have already negative real part \( \lambda_1 = -\mu_0 < 0, \lambda_2 = (\alpha + \gamma) < 0 \) for the rest of eigenvalue we take \( 2 \times 2 \) matrix, by Routh-Hurwitz criteria \[26\], we have to prove that trace of \( A \) is negative and determinant of \( A \) is positive, if \( R_0 < 1 \),

\[ J_0 = \begin{pmatrix}
0 & \frac{\beta_1 \pi}{\mu_0} (1 - R_0) & \frac{\beta_2 \pi}{\mu_0} \\
\frac{\beta_1 \pi}{\mu_0} (q + k + \mu_0) & \gamma & 0 \\
\frac{\beta_2 \pi}{\mu_0} (q + k + \mu_0) & 0 & (\alpha + \mu_0) \\
\end{pmatrix}, \]

**Trace(A) = \[\begin{pmatrix}
0 & \frac{\beta_1 \pi}{\mu_0} (1 - R_0) & \frac{\beta_2 \pi}{\mu_0} \\
\frac{\beta_1 \pi}{\mu_0} (q + k + \mu_0) & \gamma & 0 \\
\frac{\beta_2 \pi}{\mu_0} (q + k + \mu_0) & 0 & (\alpha + \mu_0) \\
\end{pmatrix} \approx (q + k + \mu_0)(1 - R_0) + (\alpha + \mu_0), \]

thus **Trace(A) < 0** if \( R_0 < 1 \).

Hence \( \det(A) > 0 \) if \( R_0 < 1 \) and \( \beta_3 \leq \beta_1 \), which implies that \( \det(A) \) is positive, if \( R_0 < 1 \). Therefore, **Trace(A) < 0** and \( \det(A) > 0 \) if and only if \( R_0 < 1 \). Thus the disease free equilibrium is locally asymptotically stable at \( E_0 \).

Let us consider Lyapunov function \( V = I \). Differentiating \( V \) with the solution of model (1), we obtain

\[
V = I = (\beta_1 - \beta_2 \frac{I^*}{m + I})SI + (\beta_1 - \beta_2 \frac{H}{m + H})SH - (q + k + \mu_0)I \\
\leq \beta_1 SI - (q + k + \mu_0)I \in [q + k + \mu_0](R_0 - 1)I < 0.
\]

Hence \( E_0 \) is globally stable at disease free equilibrium point.

For \( R_0 > 1 \), the Jacobian matrix at the equilibrium \( E^* \) is

\[
F(S^* , I^* , H^* , R^*) = \begin{pmatrix}
-A & -B & -C & \gamma \\
D & -E & 0 & 0 \\
0 & -q & -(\alpha + \mu_0) & 0 \\
0 & \kappa & \alpha & -(\mu_0 + \gamma) \\
\end{pmatrix}, \tag{10}
\]

where

\[
A = \mu_0 - (\beta_1 - \beta_2 \frac{I^*}{m + I})I - (\beta_1 - \beta_2 \frac{H^*}{m + H})H, \\
B = \beta_1 mI, \\
C = \beta_1 mH, \\
D = (\beta_1 - \beta_2 \frac{I^*}{m + I})H + (\beta_1 - \beta_2 \frac{I^*}{m + I})H, \\
G = \beta_2 mI(q + \mu_0), \\
E = \beta_2 mI(q + \mu_0), \\
\]

The characteristic equation of the Jacobian matrix is

\[ \xi^4 + a_1 \xi^3 + a_2 \xi^2 + a_3 \xi + a_4 = 0, \tag{11} \]

where

\[
a_1 = (A + 2\mu + q + \gamma), \\
2 = (\alpha + \mu_0)B + (A + \mu_0) + DM, \\
a_3 = (\alpha + \mu_0)A + M + (\alpha + \mu_0)C, \\
a_4 = NG(\alpha + \mu_0)\beta_2 m + (R_0 - 1) + (q + \kappa), \\
a_1 > 0, a_2 > 0, a_3 > 0, a_4 > 0, \text{ Also } a_0 a_2 a_3 > a_1^2 + a_2^2 a_4. \]

It follows from Routh Hurwitz criteria all the eigenvalues (11) have negative real part if \( R_0 > 1 \), which means that \( E^* \) is locally asymptotically stable. \( \square \)

**A special case of model (1) with \( \gamma = 0 \)**

Suppose \( \gamma = 0 \) in model (1) we have the following SIHR model

\[
\frac{dS}{dt} = \pi - (\beta_1 - \beta_2 \frac{I}{m + I})SI - (\beta_3 - \beta_2 \frac{H}{m + H})SH - \mu_0 S, \\
\frac{dI}{dt} = (\beta_1 - \beta_2 \frac{I}{m + I})SI + (\beta_1 - \beta_2 \frac{H}{m + H})SH - (q + k + \mu_0)I, \\
\frac{dH}{dt} = qE - (\alpha + \mu_0)H, \tag{12}
\]

\[ \frac{dR}{dt} = qE - (\alpha + \mu_0)R. \]

The first three equations are independent of the fourth equation in
the model (12). We consider the reduced model as:
\[
\frac{dS}{dt} = \beta (S - m + 1) \frac{I}{(\mu + m + 1)} \frac{H}{m + H} - (\mu + \mu_0) S, \\
\frac{dI}{dt} = (\beta - \beta_1) \frac{I}{(m + 1)} \frac{H}{m + H} - (\mu + \mu_0 + q) I, \\
\frac{dH}{dt} = q I - (\alpha + \mu_0) H.
\]

The model (13) bear disease free equilibrium at \(E_0 = (\frac{\mu}{\mu_0}, 0, 0, 0)\). For endemic equilibrium point we put right hand side of (13) zero, the characteristic equation of the above jacobian matrix is
\[
\text{det}(\lambda I - \mathbf{J}) = 0,
\]
where
\[
\mathbf{J} = \begin{pmatrix}
-\mu_0 & \frac{\beta_1}{\mu_0} & \frac{\beta_2}{\mu_0} \\
0 & (q + \mu_0) (R_0 - 1) & \frac{\beta_1}{\mu_0} \\
0 & \frac{\beta_1}{\mu_0} & -\mu_0
\end{pmatrix}
\]
and is globally asymptotically stable. For \(R_0 > 1\) the model (13) get is locally asymptotically stable.

Theorem 3. We take the model (13) with all positive parameters. For \(R_0 > 1\) the model (13) possess a unique endemic equilibrium \(E' (S', I', H', R')\) and is locally asymptotically stable. For \(R_0 < 1\) the model (13) get is disease free equilibrium of model \((S, I, H)\) is globally asymptotically stable.

Proof. Jacobian matrix of the suggested model (13) about the point \(E_0\) is
\[
J\left(\frac{\mu}{\mu_0}, 0, 0, 0\right) = \begin{pmatrix}
-\mu_0 & \frac{\beta_1}{\mu_0} & \frac{\beta_2}{\mu_0} \\
0 & (q + \mu_0) (R_0 - 1) & \frac{\beta_1}{\mu_0} \\
0 & \frac{\beta_1}{\mu_0} & -\mu_0
\end{pmatrix}
\]
When \(R_0 < 1\), then \((q + \mu_0) (R_0 - 1) < 0\), complete the proof.

Global stability of disease free equilibrium

For global stability at DFE, we use Lyapunov function theory [27].

Theorem 4. For \(R_0 < 1\) the disease free equilibrium of model (13) is stable globally, if \(S = S_0\), other wise unstable if \(R_0 > 1\).

Proof. We define the following Lyapunov function is given by
\[
F(t) = \frac{1}{2} \left( (S - S_0) + I + H + (R - R_0) \right)^2 + w_1 (S - S_0) + w_2 I(t) + w_3 H(t) + w_4 (R - R_0).
\]

where’s \(w_1 = 1, 2, 3, 4\) are positive constant taking time derivative of (17), we have
\[
\frac{dF}{dt} = \frac{1}{2} \left( (S - S_0) + I + H + (R - R_0) \right)^2 \left( (S - S_0) + I + H + (R - R_0) \right) + w_1 \frac{dS}{dt} + w_2 \frac{dI}{dt} + w_3 \frac{dH}{dt} + w_4 \frac{dR}{dt}
\]

By using \(w_1 = w_2 = w_3 = w_4 = \epsilon \delta\)
\[
\frac{dF}{dt} = \frac{1}{2} \left( (S - S_0) + I + H + (R - R_0) \right)^2 \left( (S - S_0) + I + H + (R - R_0) \right) - \mu_0 \left( (S - S_0) + I + H + (R - R_0) \right) - \mu_0 \left( (S - S_0) + I + H + (R - R_0) \right)
\]
\[
\frac{dF}{dt} = \text{negative if } S > S_0 \text{ and } R_0 < 1 \text{ and } \frac{dF}{dt} = 0 \text{ if only if } S = S_0.
\]

Global stability at endemic equilibrium we used the geometrical approach [30,31].

Proof. The linearized model and second additive compound matrix is denoted by \(J\) and \(J^2\) model (13), which becomes
\[
J = \begin{pmatrix}
-a_{11} & a_{12} & a_{13} \\
a_{21} & a_{22} & a_{23} \\
0 & a_{32} & -a_{33}
\end{pmatrix}, \\
J^2 = \begin{pmatrix}
-a_{11} & a_{12} & a_{13} \\
a_{21} & a_{22} & a_{23} \\
-a_{31} & a_{32} & a_{33}
\end{pmatrix}
\]
Consider the function \(G(\chi) = G(S, I, H) = \text{diag} \left( \frac{\partial G}{\partial \chi} \right)\), then, \(G^{-1}(\chi) = \text{diag} \left( \frac{\partial G}{\partial \chi} \right)\) taking derivative of, \(G(\chi)\), we get
\[
G(\chi) = \text{diag} \left( \frac{\partial G}{\partial \chi} \right), \\
G(\chi)^{-1} = \text{diag} \left( \frac{\partial G}{\partial \chi} \right)^{-1}, \\
G(\chi)_G^{-1} = \frac{\partial G}{\partial \chi} \frac{\partial G}{\partial \chi}^{-1}
\]
and take \(N = G(\chi) G^{-1} + G(\chi)_G^{-1}\), which can be written as
\[
N = \begin{pmatrix}
N_{11} & N_{12} \\
N_{21} & N_{22}
\end{pmatrix}
\]
Let \((n_1, n_2, n_3)\) be a vector in \(\mathbb{R}^3\) its norm \(\|\cdot\|\) defined as
\[
\|n_1, n_2, n_3\| = \max\{\|n_1\|, \|n_2\| + \|n_3\|\}.
\] (21)

Now by Martin et al. [31],
\[
\ell(N) \leq \sup \{g_1, g_2\} = \sup \{\ell(N_{11}) + \|N_{12}\|, \ell(N_{22}) + \|N_{21}\|\},
\] where \(g_i = \ell(N_i) + \|N_{ij}\|\) for \(i = 1, 2\) and \(i \neq j\), which implies that
\[
g_1 = \ell(N_{11}) + \|N_{12}\|, \quad g_2 = \ell(N_{22}) + \|N_{21}\|.
\] (22)

where \(\ell(N_{11}) = \frac{\dot{S}}{S} - \left(\beta_1 - \beta_2 \frac{I}{m + I}\right)I - \left(\beta_3 - \beta_4 \frac{H}{m + H}\right)H - \mu_0\),

\(N_{12} = \left[\begin{array}{c}
\frac{\dot{S} I - (\beta_1 - \beta_2 \frac{I}{m + I})I - (\beta_3 - \beta_4 \frac{H}{m + H})H - \mu_0}{S} \\
\end{array}\right]
\]
\[
\dot{S} = -\beta_1 S - \left(2\frac{mH}{N} + \frac{N}{t} \right) q + \mu, \\
\dot{R} = \frac{mH}{N} q + \mu - \frac{\beta_1 S}{N} \dot{I} - \frac{\beta_2}{(m+1)N} \left( q + \kappa + \mu_0 \right) \dot{I}, \\
\dot{I} = \frac{\beta_1 S}{N} \dot{I} + \frac{\beta_2}{(m+1)N} \left( q + \kappa + \mu_0 \right) \dot{I} - \mu - (\beta_3 + \mu) I.
\]

\[
\dot{S} = \frac{1}{N} \left( -2\mu_0 - (q + \kappa) \min(\beta_1, \beta_0) \right) - \frac{1}{N} \min(\beta_1, \beta_0) I + \frac{1}{N} \beta_3 m H.
\]

So finally, we can write
\[
\limsup_{t \to \infty} \int_0^t \epsilon(N) dt < 0.
\]

Thus the system (1) around \((S_0, I_0, H_0)\) is globally asymptotically stable.

### Numerical simulation

In this section, we solved the proposed deterministic model by using Runge–Kutta method of order 4th, see for detail [32]. To further understand the dynamical behavior of the proposed model we used numerical simulation to verify our analytical findings. In order to do this, we assumed some value of parameters, and some are taken from published data given in Table 1. The choice of numerical values of the parameters are taken in such a way that would be more biologically feasible. We also assume the time interval is 10 days with initial population for susceptible \(S\), infected \(I\), recovered \(R\). Moreover, the biological interpretation of these results states that if the value of basic reproductive number is less than one, then the susceptible population decreases, while then become stable and shows that there will be always stable susceptible population, see Fig. 3. The dynamics of \(I(t), H(t), R(t)\) reveals that the number of these populations will be decreases and reaches to zero as shown in Fig. 3a-d, which ensure the stability of the proposed model. One of the important factors is to find the relative impact of the basic reproductive number to epidemic parameters as shown in Fig. 3.

### Discussion

We developed a mathematical model to analyze the impact of media coverage to the spread of infectious diseases in a given region. We get the following results from SIHRS, and SIHR model.

We calculate basic reproductive number \(R_0\) by the method of next generation method. When \(\beta_2, \beta_3 = 0\), the media coverage does not affect the reproductive number \(R_0\). We discuss the stability of the proposed model. Stability analysis show that the disease free equilibrium is locally asymptotically stable if \(R_0 < 1\). If \(R_0 > 1\), it is shown that a unique endemic equilibrium appears and bifurcation can occur which cause oscillatory phenomena. We discuss the role of media coverage on the spreading of MERS-CoV. Though the media coverage itself is not a determined fact, to eradicate the infection of the diseases, the analysis of the model indicates that, to certain extent, the more media coverage in a given population, the less number of individuals will be infected. Our analytical results show that the susceptible \(S(t)\), infected \(I(t)\), hospitalized \(H(t)\), recovered \(R(t)\) converge to equilibrium point which ensures the stability of the proposed model. In future, we are planning to develop an optimal mechanism on the basis of local dynamics and sensitivity analysis. This control strategy will help that how to eradicate the infection from the community. Work on such issue is in progress and will be reported soon in the form of a new article.

### Declaration of Competing Interest

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

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