Modeling the impact of precautionary measures and sanitation practices broadcasted through media on the dynamics of bacterial diseases

Rabindra Kumar Gupta1,2 · Soumitra Pal1 · A. K. Misra1

Received: 22 April 2022 / Accepted: 13 July 2022 / Published online: 28 August 2022 © The Author(s), under exclusive licence to Springer Nature Switzerland AG 2022

Abstract
The media has a significant contribution in spreading awareness by broadcasting various programs about prevalent diseases in the society along with the role of providing information, feeding news and educating a large mass. In this paper, the effect of media programs promoting precautionary measures and sanitation practices to control the bacterial infection in the community is modeled and analyzed considering the number of media programs as a dynamical variable. In the modeling phenomena, human population is partitioned into three classes; susceptible, infected and recovered. The disease is supposed to spread by direct contact of susceptible with infected individuals and indirectly by the ingestion of bacteria present in the environment. The growth in the media programs is considered proportional to the size of infected population and the impact of these programs on the indirect disease transmission rate and bacteria shedding rate by infected individuals is also considered. The feasibility of equilibria and their stability conditions are obtained. Model analysis reveals that broadcasting media programs and increasing its effectiveness shrink the size of infected class and control the spread of disease to a large extent.

Keywords · Bacterial disease · Precautionary measures · Sanitation practices · Media programs · Transcritical bifurcation

Introduction
Infectious diseases are posing a great problem to mankind, as they impose mortality, disability, and create socio-economic hindrance for people all over the world (Lopez et al. 2006; WHO 2012). Infectious diseases are becoming more dangerous due to the speedy mutation of their pathogens (changes in their DNA code) to survive in difficult situations. Bacteria (pathogens) are microscopic, single-cell organisms that cause contagious bacterial infections and can result in many serious or life-threatening complications. Bacterial infection takes place directly during the interaction between susceptible and infected individuals and indirectly through ingestion of bacteria from the environment. The bacterial disease cholera is indirectly transmitted mainly through ingestion of Vibrio cholerae bacteria from contaminated food or water and directly transmitted due to unhygienic contact with cholera patients faeces, barf or corpses. Infectious diseases cause mortality of more than 11 million people in developing countries (Lopez et al. 2006). Almost 1.4 million kids lose their life each year due to Pneumonia (WHO 2012). Also, about 1.5 million kids below the age of five expire each year due to diarrheal disease, 1.4 million mortality occurred due to TB in 2010 and 655,000 with malaria (WHO 2009). It is estimated that about 2.0 billion people still are not facilitated with toilets. Out of which, 673 million people are still defecating in the open area, like in street gutters, on the banks of rivers and ponds and behind bushes (WHO 2019). More than 10% of population around the world is supposed to consume food products irrigated by contaminated water (WHO 2019). Since sanitation refers to clean environment, safe disposal of dungs and human excreta and precautionary measures stand for using appropriate masks in proper way, frequently cleaning hands,
maintaining social distance, avoiding unnecessary contact with infected individuals, practicing better hygiene, and avoiding ingestion of contaminated water or food, etc., protect human beings from infection. Therefore, sanitation and precautionary measures are the rational steps to control the bacterial infection. Almost 10% of the total disease burden is still due to the poor sanitation (Mara et al. 2010). Proper and adequate sanitation with essential precautions have a significant impact on human health and socioeconomic status of any nation, specially of developing countries.

Media being the major source of information, disseminate awareness among people regarding the mode of disease transmission and its preventive measures. Social media along with social networking sites assisted by the mobile technology have revolutionized the speed and way of spread of information. 71% of social network users among 3.77 billion internet users around the world have capacity to communicate instantaneously with hundreds of new people (Kemp et al. 2017). Awareness programs through social media play a very fundamental role to convey information about the mode of disease transmission and protection from disease and also address the issues of healthy sanitation that induce changes in people’s attitude so that they adopt adequate precautionary measures. Media also inform the public about government policies and programs and its usefulness, like “Swachh Bharat Abhiyan”, an initiative taken by the Indian government to propagate awareness among the individuals to change their behavior regarding healthy sanitation practices (Swachh Bharat Abhiyan 2017). Some diseases are eradicated by vaccination and awareness. The production of vaccines takes long time and is expensive too. Also, there is a great challenge to carry out vaccination effectively due to lack of public beliefs in the efficacy as well as safety of vaccines that creates vaccine hesitancy (i.e., delay or disagree to get vaccinated) (Larson et al. 2014). Therefore, utmost importance must be assigned to awareness measures via media programs for the control and possible eradication of bacterial diseases. Thus, media can play a decisive role in restricting the spread of the diseases by altering the behaviors of susceptible individuals.

Mathematical modeling is an efficient mechanism to understand the dynamics of disease transmission and is useful in making appropriate decisions to control disease prevalence. In the past few decades, researchers have exploited this branch to model the effect of media campaigns on the dynamics of diseases like, AIDS (Nyabadza et al. 2010; Khan and Odinsyah 2020), Ebola (Njankou and Nyabadza 2017), Avian Influenza (Khan et al. 2020), Listeriosis (Chukwu and Nyabadza 2021), vector-host disease (Khan et al. 2021) and COVID-19 (Tripathi et al. 2021). Some mathematical studies are also performed to assess the impact of awareness on the control of infectious diseases by taking rate of transmission as decreasing function of media programs (Liu and Cui 2008; Cui et al. 2008; Liu et al. 2007; Tchuenche et al. 2011; Sundar et al. 2018). A few models with media as dynamical variable in which programs that propagate awareness are executed proportional to the infective population (Collinson et al. 2015; Dubey et al. 2016; Huo et al. 2018; Kumar et al. 2017; Basir 2018; Chang et al. 2020; Shanta and Biswas 2020). Yang et al. (2017) have suggested a comparative study of two problems to explore the impact of awareness to mitigate cholera outbreak. The susceptible class of the first model is divided into aware and unaware class in the second model. Their findings have shown that the transmission rate and bacteria shedding rate decline with the increase of awareness programs which help in controlling infection of cholera disease. Mara et al. (2010) have studied the impact of sanitation in controlling infections from diseases. They have shown that enriched sanitation is accountable for better human health and also for socioeconomic development, particularly in developing nations. Misra et al. (2011) put forward a mathematical model to analyze the effect of awareness programs through the media on the dynamics of infectious diseases. In this study, it is assumed that the growth of media is proportional to the number of infectives and the peoples are made aware through awareness programs forming a separate aware class. Their study proclaims that the awareness programs through media is helpful in minimizing the spread of infectious disease but the disease remains in the community due to immigration. Shukla et al. (2020) suggested a model in which they have shown that the bacterial density can be reduced if the rate of sanitation effort is increased that ultimately decreases the infected population. Musa et al. (2021) have developed a model to study the impact of public health education programs on transmission dynamics of the typhoid fever (a bacterial infection). The analysis of this work reveals that the presence of public health education programs control the spread of diseases. The final epidemic size relation is developed taking only human to human transmission route to estimate the size of susceptible class during typhoid epidemic. The model is fitted to the data of typhoid fever cases in Taiwan from 2009 to 2018 and wavelet analysis is done to know the local periodicity of typhoid fever.

As pointed out earlier, sanitation and precautionary measures are effective mechanisms to control the spread of infections. In this regard, Tiwari et al. (2022) have developed a model to investigate the effect of awareness and sanitation programs propagated through social media on disease prevalence and epidemic outbreak, where they have considered aware class as a separate state variable along with susceptible and infected class. The growth rate of programs through media is assumed to be proportional to infected population and decreases with the increase of number of aware people. They have considered that the bacterial density in the environment is reduced due to sanitation practices broadcasted.
through media programs. In this paper, we have suggested a model to study the influence of precautionary measures and sanitation practices broadcasted through media on the prevalence of bacterial disease by controlling bacterial density at its source that is reducing the bacteria shedding in the environment by infected individuals and also limiting the ingestion of bacteria by people from the environment. The details of modeling phenomena are discussed in the following sections.

The mathematical model

This section covers the formulation of a mathematical model with some considerable assumptions for the spread of bacterial disease. In the considered region, let the entire human population \( N(t) \) be partitioned into three subclasses; \( S(t) \), \( I(t) \) and \( R(t) \) defined in Table 1 along with the bacterial density in the environment \( B(t) \) and the total number of media programs \( M(t) \). People are supposed to join the susceptible class at a constant rate \( \Lambda \), attributed to birth or immigration. The population is assumed to be homogeneously mixed and disease propagates through direct contact with infectives, at the rate \( \beta \), following the law of simple mass action and indirectly through ingestion of bacteria from the environment at the rate \( \eta \). Bacterial density is supposed to have saturated impact on susceptibles and is incorporated using the term \( \frac{\omega M}{p+M} \). It is also assumed that the recovered individuals join the susceptible class due to the immunity loss caused by high stress level, frequent infections, lack of good hygiene and some people born with weak immune system. Also, each class experiences natural mortality, whereas infected class suffers extra mortality induced by the disease. The abundance of bacteria in the environment is due to self growth of the bacteria and the release of bacteria from the infected humans. The increment of media programs is considered to vary with the size of infected population and it is assumed that a certain level of media programs \( M_0 \) always broadcasted to create awareness about the risk of disease prevalence. Moreover, broadcasting of some programs are being stopped by the passage of time subject to failure in the long run and their ineffectiveness. Hence, the decay in media programs is incorporated in the model with the term \( r_0(M-M_0) \). With these considerations, the proposed model can mathematically be presented as follows:

\[
\frac{dS}{dt} = \Lambda - \beta SI - \eta \left(1 - \frac{\omega M}{p+M}\right) S \left(\frac{B}{K+B}\right) + \delta R - dS, \tag{1a}
\]

\[
\frac{dI}{dt} = \beta SI + \eta \left(1 - \frac{\omega M}{p+M}\right) S \left(\frac{B}{K+B}\right) - (\nu + \alpha + d)I, \tag{1b}
\]

\[
\frac{dR}{dt} = \nu I - \delta R - dR, \tag{1c}
\]

\[
\frac{dB}{dt} = sB - s_0B + s_1 \left(1 - \frac{\theta M}{m+M}\right) I, \tag{1d}
\]

\[
\frac{dM}{dt} = rI - r_0(M-M_0). \tag{1e}
\]

Schematic flowchart diagram of the above model system is shown in Fig. 1. Biologically, it is obvious that the bacterial density at any instant of time experiences a net decay without any external contribution. So, the natural mortality rate of bacteria exceeds its self-growth rate, i.e., \( s < s_0 \). We define \( s_0 - s = s_2 \), and \( 0 \leq \theta \leq 1, 0 \leq \omega \leq 1 \). We analyze the system (1) with non-negative initial conditions, and the media programs at level \( M_0 \) initially. The parameters used in the system (1) are all taken to be positive constants, and their epidemiological features are interpreted in Table 2. Since, \( N = S + I + R \), system (1) can equivalently be expressed as follows:

\[
\frac{dI}{dt} = \beta I(N - I - R) + \eta \left(1 - \frac{\omega M}{p+M}\right) (N - I - R) \left(\frac{B}{K+B}\right) - (\nu + \alpha + d)I, \tag{2a}
\]

Table 1 Descriptions of variables used in the model system (1)

| Variables | Descriptions |
|-----------|--------------|
| \( S(t) \) | Size of susceptible class |
| \( I(t) \) | Size of infected class |
| \( R(t) \) | Size of recovered class |
| \( B(t) \) | Bacterial density in the environment |
| \( M(t) \) | Cumulative number of programs broadcasted through media |
Fig. 1 Flowchart of model system (1). The red dotted lines represent the interaction between the respective compartments that is contribution of one of connecting compartment on other, whereas the blue solid lines with arrow depict the flow into/out the compartment.

Table 2 Descriptions of parameters involved in the system (1)

| Parameters | Descriptions |
|------------|--------------|
| Λ          | Immigration rate of susceptible population |
| β          | Transmission rate of susceptible into infected class due to their contact with infected individuals |
| η          | Rate of transmission from susceptible to infected class due to ingestion of bacteria from environment |
| δ          | Transfer rate from recovered to susceptible class due to immunity loss |
| d          | Death rate of human population due to natural factors |
| υ          | Recovery rate of infected population |
| α          | Disease induced death rate |
| ω          | Efficacy of media programs focusing precautionary measures |
| θ          | Efficacy of media programs targeting sanitary measures |
| s          | Self growth rate of bacteria |
| s₀         | Rate of decrease of bacteria due to natural factors |
| s₁         | Bacteria shedding rate by each infected person |
| p          | Saturation level of media programs at which the efficiency of precautionary measure related programs becomes half of its maximum value |
| m          | Saturation level of media programs at which the efficiency of sanitation measure related programs becomes half its optimum value |
| K          | Density of bacteria that yields 50% chance to catch the disease |
| r          | Growth rate of media programs |
| r₀         | Diminution rate of media programs |
| M₀         | Base line number of media programs |

\[
\frac{dR}{dt} = υI - (δ + d)R, \quad (2b) \quad \frac{dB}{dt} = sB - s₀B + s₁\left(1 - \frac{θM}{m + M}\right)I, \quad (2d)
\]
\[
\frac{dN}{dt} = Λ - dN - αI, \quad (2c) \quad \frac{dM}{dt} = rI - r₀(M - M₀). \quad (2e)
\]
Systems (1) and (2) are equivalent because \( S \) is just changed to \( N \) using \( N = S + I + R \) and no other changes are made. After knowing the value of \( N, I \) and \( R \) at any instant of time \( t \), the value of \( S \) at the same time can be obtained by using the relation \( S = N - I - R \). Therefore, it is sufficient to study and analyze the dynamics of system (2).

**Mathematical analysis**

**Positivity and boundedness**

**Theorem 1** The biologically feasible region for the solution of system (2) commencing in the positive orthant is as follows:

\[
\Omega = \left\{ (I, R, N, B, M) \in \mathbb{R}^5_+ : 0 \leq I(t) + R(t) \leq N(t) \leq \frac{\Lambda}{d}, 0 \leq B \leq \frac{s_1 \Lambda}{d s_2}, 0 \leq M(t) \leq \frac{r \Lambda}{d r_0} + M_0 \right\}.
\]

The region \( \Omega \) is compact and invariant with respect to system (2) which is closed and bounded in a hyper-cuboid of five dimensions.

The proof of theorem is provided in Appendix A.

**Equilibrium analysis and basic reproduction number**

**Existence of disease-free equilibrium**

The equilibrium equations of model (2) are solved to get the disease-free equilibrium which is \( E_0 = (0, 0, \frac{\Lambda}{d}, 0, M_0) \). \( E_0 \) is always feasible.

**Basic reproduction number**

Health organizations and policymakers all over the world have been using basic reproduction number \( R_0 \) as key estimator to measure the intensity of the epidemic. It is defined as \( R_0 = \rho(T_2^{-1}T_1) \) with \( \rho(T_2^{-1}T_1) = \sup \{ |x| : x \in \sigma(T_2^{-1}T_1) \} \), where \( \sigma \) is the spectrum and \( \rho \) is spectral radius of the matrix \( T_2^{-1}T_1 \). The next-generation matrix technique (Diekmann et al. 2010) is used in calculating \( R_0 \). The transmission and transition terms in the infected subsystem of the system (2) are, respectively,

\[
T_2 = \begin{bmatrix} (\nu + \alpha + d)I \\ s_2B - s_1 \left( 1 - \frac{\theta M}{m + M} \right) I \end{bmatrix}.
\]

The transmission and transition matrices at \( E_0 \) of system (2) are, respectively,

\[
T_1 = \begin{bmatrix} \frac{\beta}{d} & \frac{m}{k} \left( 1 - \frac{\alpha M_0}{p + M_0} \right) \\ 0 & 0 \end{bmatrix} \quad \text{and} \quad T_2 = \begin{bmatrix} (\nu + \alpha + d) & 0 \\ -s_1 \left( 1 - \frac{\theta M}{m + M} \right) & s_2 \end{bmatrix}.
\]

The value of \( R_0 \) for the system (2) is

\[
R_0 = \frac{\Lambda}{d(\nu + \alpha + d)} \left[ \beta + \frac{\eta s_1}{Ks_2} \left( 1 - \frac{\alpha M_0}{p + M_0} \right) \left( 1 - \frac{\theta M_0}{m + M_0} \right) \right].
\]

**Existence of endemic equilibrium**

The components of \( E^* = (I^*, R^*, N^*, B^*, M^*) \) are obtained from the equilibrium equations of the system (2). Here, we claim that the positive value of components of \( E^* \) exist.

From equilibrium Eq. (2b)

\[
R = \frac{\nu I}{\delta + d}, \tag{3}
\]

from the equilibrium Eq. (2c)

\[
N = \frac{\Lambda - \alpha I}{d}, \tag{4}
\]

and from the equilibrium Eq. (2e) we have

\[
M = \frac{r I}{r_0} + M_0 = g(I). \tag{5}
\]

Using (5) in the equilibrium Eq. (2d) we have

\[
B = \frac{s_1}{s_2} \left( 1 - \frac{\theta g(I)}{m + g(I)} \right) I = lh(I). \tag{6}
\]

Now, from the equilibrium Eq. (2a) we get

\[
F(I) = \left[ \beta + \eta \left( 1 - \frac{\alpha g(I)}{p + g(I)} \right) \frac{h(I)}{K + lh(I)} \right] \times \left[ \frac{\Lambda - \alpha I}{d} - I - \frac{\nu I}{\delta + d} \right] - (\nu + \alpha + d), \tag{7}
\]

where
\[ g'(I) = \frac{r}{r_0} > 0, \quad h'(I) = -\frac{\beta \gamma m r}{r_0^2 (m + g(I))^2} < 0. \quad (8) \]

Differentiating Eq. (7) with respect to \( I \), we have
\[
F'(I) = \begin{bmatrix} \frac{-\eta p g(I)}{p + g(I)^2} h(I) \\
\eta \left( 1 - \frac{\gamma g(I)}{p + g(I)} \right) (K h(I) - h^2(I)) \\
\left( \frac{\Lambda - \eta d}{\delta} \right) - I - \frac{\eta d}{\delta + d} \\
\eta \left( 1 - \frac{\gamma g(I)}{p + g(I)} \right) \frac{h(I)}{K + h(I)} \left( \frac{\alpha}{d} + 1 + \frac{v}{\delta + d} \right) \end{bmatrix}
\]

Here we note that at any instant of time \( t \), \( S(t) = N - I - R = \frac{\Lambda}{d} - \left( \frac{\alpha}{d} + \frac{v}{\delta + d} + 1 \right) I \). For \( S(t) \) to be positive, \( I < \frac{\Lambda}{\delta + \frac{\alpha}{d} + 1} = I_c \). (say)

From Eq. (7) it is noted that

(i) \( F(0) = \frac{\Lambda}{d} \left[ \beta + \frac{\eta M_0}{K_0} \right] \left( 1 - \frac{\alpha M_0}{p + M_0} \right) \left( 1 - \frac{\eta M_0}{m + M_0} \right) \]

\[-(v + \alpha + d) = (v + \alpha + d)(R_0 - 1) > 0, \quad \text{provided} \quad R_0 > 1 \]

(ii) \( F(I_c) = \frac{\Lambda}{d} \left( \frac{\alpha}{d} + \frac{v}{\delta + d} + 1 \right) \]

\[-(v + \alpha + d) < 0, \quad \text{where}, \quad 0 < I_c = \frac{\Lambda}{\delta + \frac{\alpha}{d} + 1} < \frac{\Lambda}{d} \]

(iii) \( F'(I) < 0 \) for \( I \in (0, I_c) \).

This implies that the unique root (say \( I = I^* \)) of \( F(I) \) exists in the interval \((0, I_c)\), whenever \( R_0 > 1 \). With this value of \( I = I^* \) and from Eqs. (3), (4), (5) and (6) we can easily obtain the values of \( R^*, N^*, M^* \) and \( B^* \), respectively. Thus, equilibrium \( E^* \) is feasible, when \( R_0 > 1 \), and \( I \in (0, I_c) \).

### Stability analysis

This section covers the local and global stability of equilibria of the system (2). This is achieved either by evaluating sign of the eigenvalues of the Jacobian matrix of the system (2) at the equilibrium points or by Lyapunov method. The local stability analysis of \( E_0 \) stated in the theorem (2) indicates whether the disease disappears gradually or spreads in the community.

### Local stability analysis of disease free equilibrium

**Theorem 2** The disease-free equilibrium \( E_0 \) is always feasible and locally asymptotically stable if \( R_0 < 1 \), whereas it is unstable whenever \( R_0 > 1 \) and a unique stable endemic equilibrium exists.

**Proof** The Jacobian matrix of the system (2) is
\[
J = \begin{bmatrix} J_{11} & J_{12} & J_{13} & J_{14} & J_{15} \\
0 & -(\delta + d) & 0 & 0 & 0 \\
0 & 0 & -r_0 & 0 & 0 \\
0 & 0 & 0 & -s_2 & 0 \\
r & 0 & 0 & 0 & -r_0 \end{bmatrix}
\]

where
\[
J_{11} = \beta(N - 2I - R) - \frac{\eta B}{K + B} \left( 1 - \frac{\alpha M}{p + M} \right) \]

\[-(v + \alpha + d), \]

\[
J_{12} = \beta I + \frac{\eta B}{K + B} \left( 1 - \frac{\alpha M}{p + M} \right), \]

\[
J_{14} = \frac{\eta K}{(K + B)^2} (N - I - R), \]

\[
J_{15} = \frac{\eta B(N - I - R)}{(K + B)(p + M)^2}, \]

\[
J_{41} = \frac{\eta B(N - I - R)}{(K + B)(p + M)^2}, \]

The Jacobian matrix at \( E_0 \) is
\[
J_{E_0} = \begin{bmatrix} \frac{\beta \Lambda}{\delta} - (v + \alpha + d) & 0 & 0 & \frac{\eta B}{\delta} \left( 1 - \frac{\alpha M_0}{p + M_0} \right) & 0 \\
0 & -(\delta + d) & 0 & 0 & 0 \\
0 & -r_0 & 0 & 0 & 0 \\
0 & 0 & -s_2 & 0 & 0 \\
r & 0 & 0 & 0 & -r_0 \end{bmatrix}
\]

Since three characteristic values, \(-r_0, -(\delta + d)\) and \(-d\) of the matrix \( J_{E_0} \) are negative. The rest two characteristic values are the roots of equation
\[
\lambda^2 + c_1 \lambda + c_2 = 0, \quad (9)
\]

where
\[
c_1 = -\left( \frac{\beta \Lambda}{\delta} - (v + \alpha + d) - s_2 \right) \quad \text{and} \]

\[
c_2 = s_2 (v + \alpha + d)(1 - R_0). \]

If \( R_0 < 1 \), then \( c_1 > 0 \) as \( \frac{\beta \Lambda}{\delta} < (v + \alpha + d) + s_2 \) and \( c_2 > 0 \), so both roots of Eq. (9) are real negative or with negative real parts, while at least one positive root whenever \( R_0 > 1 \). That is, \( E_0 \) is locally asymptotically stable if \( R_0 < 1 \) and unstable for \( R_0 > 1 \) either in \( I \) or \( B \) direction.

To visualize the complete characterization of the endemic equilibrium \( E^* \) we obtain the conditions of its local and global stability. We use Lyapunov method to find these
conditions. Lyapunav function is positive definite function which is zero at the equilibrium point. The equilibrium point is stable if the time derivative of Lyapunav function is negative definite. The conditions of local and global stability are presented in the following theorems.

**Local stability analysis of** $E^*$

**Theorem 3** The equilibrium $E^*$, if exists, is locally asymptotically stable in the region of attraction $\Omega$ if

$$b_{14}^2 < \frac{4}{9} s_1^2 b_{11} \min \left\{ \frac{b_{11}}{b_{41}}, \frac{2r_0 b_{15}}{rb_{45}} \right\},$$

(10)

where $b_{ij}$ are defined in the proof.

The proof is provided in Appendix B.

**Global stability analysis of** $E^*$

**Theorem 4** The endemic equilibrium $E^*$, if feasible, is globally asymptotically stable in the region of attraction $\Omega$ if the following conditions hold:

$$\left[ \frac{\eta \Lambda s_1}{\delta s_2 R^*} \right]^2 < \frac{2}{3} \min \left\{ \frac{d}{\alpha}, \frac{\delta + d}{\nu} \right\},$$

(11)

and

$$\frac{N^* - I^* - R^*}{I^*} < \frac{2}{27} \frac{\beta^2 s_1^2}{\nu^2} \min \left\{ \frac{2}{3} \min \left\{ \frac{1}{s_1^2}, \frac{2r_0^2}{9s_2^2} \left( \frac{m + M^*}{\delta I^*} \right)^2 \right\}, \right.$$

$$\left. \times \left( \frac{K + B^*}{1 - \frac{wM^*}{p + M^*}} \right)^2 \left( \frac{Kd_0(p + M^*)}{s_2 \Lambda} \right)^2 \right\}.$$  

(12)

The proof is provided in Appendix C.

**Bifurcation analysis**

The exchange of stability between $E_0$ and $E^*$ occurs at $R_0 = 1$, which indicates the occurrence of transcritical bifurcation in the system. Since $R_0$ is a function of $\beta$, therefore, without any loss of generality, we can choose $\beta$ as a bifurcation parameter. At $R_0 = 1$, we note that

$$\beta = \beta^* = \frac{d(u + a + d)}{\Lambda} - \frac{\eta s_1}{K} \left( 1 - \frac{\omega M_0}{p + M_0} \right)$$

$$\times \left( 1 - \frac{\theta M_0}{m + M_0} \right).$$

The linearized matrix of the system (2) at $\beta = \beta^*$ about $E_0$ is

$$J_{E_0}(\beta^*) = \begin{bmatrix}
  a_{11} & 0 & 0 & g \frac{s_1}{d_0} & 0 \\
  \nu & -(\delta + a) & 0 & 0 & 0 \\
  -\alpha & 0 & -d & 0 & 0 \\
  s_1 \left( 1 - \frac{\theta M_0}{m + M_0} \right) & 0 & 0 & -s_2 & 0 \\
  \frac{r}{r_0} & 0 & 0 & 0 & -r_0 \\
\end{bmatrix},$$

where

$$a_{11} = -\frac{\Lambda \eta s_1}{K} \left( 1 - \frac{\omega M_0}{p + M_0} \right) \left( 1 - \frac{\theta M_0}{m + M_0} \right) < 0.$$}

The eigenvalues are $-(\delta + d), -d, -r_0, (a_{11} - s_2)$ and 0. Thus, all four eigenvalues are negative and one is simple zero. The equilibrium $E_0$ is non-hyperbolic equilibrium point. The right eigenvector of $J_{E_0}(\beta^*)$ corresponding to the 0 eigenvalue is

$$W = (w_1, w_2, w_3, w_4, w_5)$$

$$= \left( \frac{1}{v}, \frac{\nu}{(\delta + d)}, -\alpha \frac{1}{d}, s_1 \left( 1 - \frac{\theta M_0}{m + M_0} \right), \frac{r}{r_0} \right),$$

and the left eigenvector is

$$U = (u_1, u_2, u_3, u_4, u_5) = (1, 0, 0, 0, 0)$$

so that $U^TW = 1$.

We have applied the result of theorem (4.1) of Chavez and Song (2004) for the local dynamics of the system (2). Accordingly, the coefficients are given by

$$a = \sum_{i,j=1}^{5} u_i w_i w_j \frac{\partial^2 f_k(E_0, \beta^*)}{\partial x_i \partial x_j}, \quad b = \sum_{i=1}^{5} u_i w_i \frac{\partial \frac{d}{dx}(E_0, \beta^*)}{\partial \beta}.$$  

Now, the value of $a$ and $b$ for model system (2) is as follows:

$$a = -2\beta^*(1 + w_2 - w_3)$$

$$- \eta \frac{e_1}{K} \left( w_4 + w_2 w_4 - w_3 w_4 + \frac{2 \Lambda w_4^2}{Kd} \right) - \Lambda \eta w_5 e_2 \frac{d}{dK},$$

where

$$e_1 = \left( 1 - \frac{\omega M_0}{p + M_0} \right) > 0, \quad e_2 = \frac{a p w_1}{(p + M_0)^2} > 0,$$

and

$$b = \frac{\Lambda}{d}. $$
Here \( a < 0 \) and \( b > 0 \). We have summarized the above results in the theorem (5) to know the qualitative behavior of the solution of the system (2) when the value of parameter \( R_0 \) changes, that is the value of \( R_0 \) changes from the critical value one.

**Theorem 5** For \( a < 0 \) and \( b > 0 \), the equilibrium \( E_0 \) of the system (2) is locally asymptotically stable, whenever \( R_0 < 1 \) with \( R_0 \approx 1 \) along with the existence of a negative unstable equilibrium \( E^* \). But, the equilibrium \( E_0 \) becomes unstable and a positive locally stable equilibrium \( E^* \) appears if \( R_0 > 1 \) with \( R_0 \approx 1 \). That is, the system (2) undergoes forward bifurcation.

**Proof** The proof follows from Chavez and Song (2004), theorem (4.1) pp. 373 and remark 1 pp. 375. \( \square \)

**Numerical simulations**

This section includes the numerical simulation of model system (2) using MATLAB and MATCONT to substantiate the analytical outcomes and also to manifest the effect of media programs on the disease prevalence. The value of the parameters used in numerical simulation and their corresponding units are given in Table 3.

The value of parameters are kept same as mentioned in Table 3 throughout the simulation unless otherwise stated. The components of endemic equilibrium of system (2) for these parameters are

\[
\begin{align*}
I^* &= 108.14, \\
R^* &= 20597.67, \\
N^* &= 99978.37, \\
B^* &= 2548.18, \\
M^* &= 143.25.
\end{align*}
\]

The eigenvalues of corresponding Jacobian matrix are

\[ -0.343145, -0.02848, -0.00298, -0.19823, \text{ and } -0.00005 \],

which are all negative reals and so the equilibrium is locally asymptotically stable. Also, the global stability conditions are satisfied with the considered set of parameter values. Figure 2 depicts that the trajectories with different initial starts converging to the only feasible endemic equilibrium. The value of \( R_0 \) for these parameter values is 1.98425. The surface plot (Fig. 3) of basic reproduction number \( R_0 = R_D + R_I \), where \( R_D \) and \( R_I \) correspond to direct transmission (human to human) and indirect transmission (environment to human) is plotted by varying both transmission rates \( \beta \) and \( \eta \), simultaneously. From the figure, we can see that, even if the values of \( R_D \) and \( R_I \) are separately

\begin{table}[h]
\centering
\caption{Parameter values used for numerical simulation}
\begin{tabular}{llll}
\hline
Parameters & Values & Units & References \\
\hline
\( \Lambda \) & 5 & person day\(^{-1} \) & Misra et al. (2018) \\
\( \beta \) & 0.000002 & person\(^{-1} \) day\(^{-1} \) & Misra et al. (2018) \\
\( \eta \) & 0.0001 & (cells mm\(^{-3} \)) day\(^{-1} \) & Codeço (2001) \\
\( \omega \) & 0.3 & – & Assumed \\
\( p \) & 60 & progms. & Assumed \\
\( K \) & 1000 & cells mm\(^{-3} \) & Codeço (2001) \\
\( u \) & 0.2 & day\(^{-1} \) & Wang et al. (2015) \\
\( \alpha \) & 0.00001 & day\(^{-1} \) & Misra et al. (2018) \\
\( d \) & 0.00005 & day\(^{-1} \) & Misra et al. (2018) \\
\( \delta \) & 0.001 & day\(^{-1} \) & Wang et al. (2015) \\
\( s \) & 0.07 & day\(^{-1} \) & Codeço (2001) \\
\( s_0 \) & 0.4 & day\(^{-1} \) & Codeço (2001) \\
\( s_1 \) & 10 & cells mm\(^{-3} \) person\(^{-1} \) day\(^{-1} \) & Codeço (2001) \\
\( \theta \) & 0.3 & – & Assumed \\
\( m \) & 50 & progms. & Assumed \\
\( r \) & 0.08 & progms. person\(^{-1} \) day\(^{-1} \) & Assumed \\
\( r_0 \) & 0.2 & day\(^{-1} \) & Assumed \\
\( M_b \) & 100 & progms. & Assumed \\
\hline
\end{tabular}
\end{table}

Fig. 2 Global stability plot of \( E^* \) in \( I - R \) plane
less than unity but their cumulative value which represents the net basic reproduction number $R_0$ may exceed unity which implies that the disease still persists in the population. Thus, it is important to control both the transmissions direct and indirect to limit the spread of disease in the population. Thereafter, we have focused on the effect of media programs which disseminate the information regarding precautionary measures as well as sanitary practices on the dynamics of the disease pervasion. Media programs related to the precautionary measures make people aware, so they avoid ingesting the contaminated water and food and thus their chance of being infected is reduced. Also through proper sanitation practices, the bacterial density in the environment can be reduced, which indirectly implies that the chance of getting infected is also reduced. Consequently, the size of infected class is reduced. Figure 4 represents the contour plots of $R_0$ plotted by varying the efficacy of these media broadcasted programs, i.e., $\omega$ and $\theta$. As the values of $\omega$ and $\theta$ get lower, the value of $R_0$ becomes larger. This suggests that the disease extinct (persists) if the efficacy of the media increases (decreases). In Figs. 5 and 6, we have drawn variation plots with respect to $\theta$ and $\omega$, respectively. These figures show that with the increasing values of either

$$R_D = \frac{\Lambda \beta}{d(\nu + \alpha + d)}, \quad R_I = \frac{\Lambda}{d(\nu + \alpha + d)} \frac{\eta s_1}{K(s_0 - s)} (1 - \frac{\omega M_0}{p + M_0}) (1 - \frac{\theta M_0}{m + M_0})$$
Fig. 7 Effect of $\theta$ and $\omega$ on the size of infected class

$\theta$ or $\omega$, the number of infected individuals decreases. Figure 7 depicts the combined effect of $\theta$ and $\omega$ on the number of infected persons. We can infer that the media programs broadcasting precautionary measures have more impact than that of programs targeting sanitation practices in reducing the infected individuals. Figure 8 delineates the transcritical bifurcation with respect to $R_0$ (obtained by varying $\beta$) in the forward direction. For $R_0 < 1$, the system (2) has only a stable disease-free equilibrium which loses its stability with the generation of unique stable endemic equilibrium, as the value of $R_0$ crosses unity. Therefore, from the figure we can infer that the disease invades in the population if $R_0 > 1$ and is extinct if $R_0 < 1$.

Sensitivity analysis

Sensitivity analysis aims to find the response of variables to changes in the parameters value. It is performed to determine how the model behavior reacts to the changes in the parameters. It helps to study the uncertainty associated with the parameters in the model. It also assists to understand the system’s dynamics and determines the level of accuracy essential for a parameter to shape the model extremely useful and valid. We have performed the sensitivity analysis to identify the relative strength of model parameters to disease transmission and prevalence. The differential sensitivity analysis of system characterizes the relation between parameters of a system and the behavior of model solution. We have used the basic differential analysis approach in order to visualize the sensitivity analysis (Eslami et al. 2013; Chitnis et al. 2008). The semi-relative sensitivity solution of a variable $Y$ with respect to the parameter $\tau$ is given by $\frac{\Delta Y(t,\tau)}{\tau} = \frac{Y(t,\tau)}{Y(t,\tau)}$ and the logarithmic sensitivity is given by $\frac{\delta\log Y(t)}{\delta(\log \tau)} = \frac{\tau Y(t,\tau)}{Y(t,\tau)}$. The semi-relative sensitivity analysis provides information about the amount the state will alter, whereas the logarithmic sensitivity analysis indicates the percentage change in the solution when the parameter is doubled. Study of both type of sensitivity solutions gives better understanding of system’s dynamics. The basic sensitivity analysis of the parameters $\eta$, $s_1$, and $r$ are obtained following (Bortz and Nelson 2004). The differential sensitivity systems corresponding to model system (2) in regard to $\eta$, $s_1$, and $r$ are, respectively, written as

Fig. 8 Transcitical bifurcation in forward direction with respect to $R_0$ (obtained by varying $\beta$), all the parameters are same as of Table 3 except $\eta = 0.00005$

Fig. 9 Semi-relative sensitivity plot of the variables $I(t)$ and $B(t)$ with respect to $\eta$, $s_1$, and $r$
\[
\frac{dI_t}{dt} = \beta [(N_{t_1} - I_{t_1} - R_{t_1})I_t - (N - I - R)I_{t_1}] - \theta (N - I - R)\left( \frac{\omega p M_{t_1}}{(p + M)^2} K + B - \frac{KB_{t_1}}{(K + B)^2}\right) - \omega M \frac{N_{t_1}}{(p + M)} + \eta (N_{t_1} - I_{t_1} - R_{t_1})\left( \frac{B}{K + B} \right) + (1 - \frac{\omega M}{p + M}) (N - I - R)\left( \frac{B}{K + B} \right) - (\delta + d) I_{t_1},
\]

\[
\frac{dR_t}{dt} = v I_t - (\delta + d) R_t,
\]

\[
\frac{dN_t}{dt} = -dN_{t_1} - a I_{t_1},
\]

\[
\frac{dB_t}{dt} = s B_{t_1} - s_0 B_{t_1} + s_1 \left( 1 - \frac{\theta M}{m + M} \right) I_{t_1} - \frac{m \theta M_{t_1}}{(m + M)^2} I_{t_1},
\]

\[
\frac{dM_t}{dt} = r I_{t_1} - r_0 M_{t_1}.
\]

\[
\frac{dI_s}{dt} = \beta [(N_s - I_s - R_s)I_s - (N - I - R)I_s] - \eta (N - I - R)\left( \frac{\omega M_{s_1}}{(p + M)^2} K + B - \frac{KB_{s_1}}{(K + B)^2}\right) - \omega M \frac{N_{s_1}}{(p + M)} + \eta (N_{s_1} - I_{s_1} - R_{s_1})\left( \frac{B}{K + B} \right) + (1 - \frac{\omega M}{p + M}) (N - I - R)\left( \frac{B}{K + B} \right) - (\delta + d) I_{s_1},
\]

\[
\frac{dR_s}{dt} = v I_s - (\delta + d) R_s,
\]

\[
\frac{dN_s}{dt} = -dN_{s_1} - a I_{s_1},
\]

\[
\frac{dB_s}{dt} = s B_{s_1} - s_0 B_{s_1} + \left( 1 - \frac{\theta M}{m + M} \right) I_{s_1} + s_1 \left( 1 - \frac{\theta M}{m + M} \right) I_{s_1} - \frac{s_1 \theta M_{s_1}}{(m + M)^2} I_{s_1},
\]

\[
\frac{dM_s}{dt} = r I_{s_1} - r_0 M_{s_1}.
\]

With the chosen set of parameter values given in Table 3, we have calculated both semi-relative and logarithmic sensitivity solutions to determine the effect of \( \eta, s_1 \), and \( r \) on the number of infected individuals \( I \) and bacterial density \( B \). Figure 9 shows that doubling \( \eta \), there will be an increase of 21 in the number of infectives over 30 days period. If the bacteria shedding rate \( s_1 \) gets doubled, the infectives number will increase by 10, whereas it is reduced by 12 over a period of 30 days, if the growth rate of media programs \( r \) gets doubled. 300 cells/ml\(^3\) and 800 cells/ml\(^3\) adds to bacteria density with doubling \( \eta \) and \( s_1 \), respectively. Doubling \( r \), the bacteria density is reduced by 1900 cells/ml\(^3\), over 30 days period. Figure 10 gives the percentage change in the number of infectives and bacterial density. \( \eta \) and \( s_1 \) having positive impact on number of infected individual as well as bacterial density, whereas \( r \) has negative impact on both.
Conclusion

Media plays a vital role in shaping a society, paves pathway for the development of community as it connects almost every people by some means. In this paper, a nonlinear model is formulated to analyze the effect of precautionary measures and sanitation practices broadcasted through the media programs to control the invasion of the bacterial diseases in human population. Some media programs make people aware about the disease so that they adapt proper precautions to avoid getting infected and some focus on sanitation practices that help to reduce the density of bacteria in the environment. In the modeling phenomenon, total human population is classified into three classes; susceptible, infected and recovered. The bacterial density in the environment and the cumulative number of media programs are also taken as state variables. Due to dissemination of precautionary measures via media people adapt proper precautions that minimizes the ingestion of bacteria from the environment. Therefore, the transmission rate from susceptible to infected class is considered as a decreasing function of media programs. Infected people are also become aware of the sanitation practices due the continuous broadcast of programs regarding the sanitation practices via media. So the infected people also adapt precautions to dump their wastes properly in appropriate area and take care of personal hygiene. Thus they restrict the increment of bacterial density in the environment.

All feasible equilibria (disease free and endemic) are obtained. The linear and nonlinear stability of endemic equilibrium are discussed. Basic reproduction number \( R_0 \) is calculated, and one can see both transmission rates \( \beta \) and \( \eta \) have fair contribution to the cumulative basic reproduction number \( R_0 \). \( R_0 \) can be written as \( R_0 = R_D + R_I \), where direct (human–human) transmission rate \( \beta \) contributes to \( R_D \) and indirect (environment–human) transmission rate \( \eta \) contributes to \( R_I \). So, even if one of \( (R_D) \) or \( (R_I) \) is less than unity, the other part may raise the cumulative number above unity which implies that the disease would persist in the community. The disease dies out if it is possible to keep \( R_0 \) below unity. The system shows transcritical bifurcation in forward direction between disease-free and endemic equilibria, with respect to \( R_0 \) (obtained by varying \( \beta \)). Our obtained results show that efficacy of programs broadcasted through media to disseminate precautionary and sanitation awareness plays significant role in reducing size of infected class. In fact disease gradually dissappears with the increase of the efficacies of the awareness programs. More precisely, we can infer that precautionary measures related programs are more effective in reducing the size of infected class in comparison with the sanitary practices. So, concerned authorities and government should not only just broadcast the programs through media but also have to look after their effectiveness to get the desired results. From the sensitivity analysis, it is concluded that the bacteria shedding rate and transfer rate to infected class from susceptible have positive impact on the number of infectives but the growth rate of media programs has negative impact on both infectives and bacterial density. So, the epidemic outbreak can be controlled with appropriate control over these parameters. Our study highlights that both precautionary measures and sanitation strategies are constructive measures to control the spread of infectious bacterial diseases in the population.

**Fig. 10** Logarithmic sensitivity plot of the variables \( I(t) \) and \( B(t) \) with respect to \( \eta, s_1 \), and \( r \)
Appendix A

Proof System (1) can be written as
\[
d\frac{V}{dt} = AV + D,
\]
with \( V = [S, I, R, B, M]^T \), and
\[
A = \begin{bmatrix}
-A_{11} & 0 & \delta & 0 & 0 \\
A_{21} & -A_{22} & 0 & 0 & 0 \\
0 & 0 & -\delta & 0 & 0 \\
0 & A_{42} & 0 & -s_2 & 0 \\
0 & r & 0 & 0 & -r_0
\end{bmatrix},
\]
where
\[
A_{11} = \beta I + \eta \left( 1 - \frac{\omega M}{p + M} \right) \frac{B}{K + B} + d,
\]
\[
A_{21} = \beta I + \eta \left( 1 - \frac{\omega M}{p + M} \right) \frac{B}{K + B},
\]
\[
A_{22} = \alpha + a + d, \quad A_{42} = s_1 \left( 1 - \frac{m M}{m + M} \right).
\]
and \( D = [\Lambda, 0, 0, 0, r_0 M_0]^T \). Since all entries of the matrix \( A \) except in the diagonal are all non-negative, it is a Metzler matrix in \( V \in \mathbb{R}^5 \). It means that the trajectories of system (1) emerging from the initial point in \( V \in \mathbb{R}^5 \) remain therein for all time \( t \). That is the system (1) is positively invariant with respect to \( V \in \mathbb{R}^5 \). Now, from Eq. (2c) we have
\[
d\frac{N}{dt} + dN = \Lambda - a I \leq \Lambda.
\]
Its solution is obtained by applying standard comparison theorem as follows:
\[
0 \leq N(t) \leq \frac{\Lambda}{d} + \left( N(0) - \frac{\Lambda}{d} \right) e^{-dt}.
\]
This yields \( \lim_{t \to \infty} \sup N(t) \leq \frac{\Lambda}{d} \). Hence, we have
\[
0 \leq N(t) \leq \frac{\Lambda}{d} \forall t > 0.
\]
Now, we have
\[
d\frac{(N - I - R)}{dt} = \Lambda - \left( \beta I + \frac{B}{K + B} + d \right) (N - I - R) + \delta R.
\]
Again, taking Eq. (2d) with \( I \leq \frac{\Lambda}{d} \), we get
\[
\frac{dB}{dt} \leq \frac{s_1 \Lambda}{d} - s_2 B.
\]
By the theory of differential inequality, we get
\[
\lim_{t \to \infty} \sup B(t) \leq \frac{s_1 \Lambda}{ds_2}.
\]
This gives, \( 0 \leq B(t) \leq \frac{s_1 \Lambda}{ds_2} \) for all \( t > 0 \). Furthermore, Eq. (2e) gives,
\[
\frac{dM}{dt} \leq \frac{r \Lambda}{d} - r_0 (M - M_0).
\]
Using the concept of differential inequality
\[
\lim_{t \to \infty} \sup M \leq \frac{r \Lambda}{dr_0} + M_0.
\]
This yields \( 0 \leq M(t) \leq \frac{r \Lambda}{dr_0} + M_0 \) for all \( t > 0 \). Therefore, it is concluded that \( \Omega \) is an attracting set which means that every feasible solution of the system (2) is confined in this region.

Appendix B

Proof Consider the Lyapunav function
\[
\mathcal{L} = \frac{1}{2} \varepsilon_1^2 + \frac{1}{2} k_1 \varepsilon_2^2 + \frac{1}{2} k_2 \varepsilon_3^2 + \frac{1}{2} k_3 \varepsilon_4^2 + \frac{1}{2} k_4 \varepsilon_5^2,
\]
where, \( k_i, i = 1, \ldots, 4 \) are constants which are all positive and are to be chosen relevantly. \( \varepsilon_i, j = 1, \ldots, 5 \) are small changes in \( I, R, N, B \) and \( M \), respectively. The time derivative of \( \mathcal{L} \) with linearized model system (2) is given by
\[
\frac{d\mathcal{L}}{dt} = -b_{11} \varepsilon_1 - k_1 \delta \varepsilon_2^2 - k_2 \varepsilon_3^2 - k_3 \varepsilon_4^2 - k_4 \varepsilon_5^2 \\
+ (k_1 t - b_{12}) \varepsilon_1 \varepsilon_2 + (b_{13} - k_2) \varepsilon_1 \varepsilon_3 + (k_3 b_{41} + b_{14}) \varepsilon_1 \varepsilon_4 \\
+ (k_4 t - b_{15}) \varepsilon_1 \varepsilon_5 + k_3 b_{45} \varepsilon_4 \varepsilon_5.
\]
where,
Choosing, $k_1 = \frac{b_{12}}{a}$, $k_2 = \frac{b_{13}}{a}$ and $k_4 = \frac{b_{15}}{r}$, we have

$$\frac{dL}{dt} = -b_{11}e_1^2 - k_1(\delta + d)e_2^2 - k_2e_3^2 - k_3s_2e_4^2 - k_4r_0e_5^2$$

$$+ (k_3b_{41} + b_{14})e_1e_4 + k_3b_{45}e_4e_5.$$ 

$\frac{dC}{dt} < 0$ if the inequalities stated below are verified,

$$3k_3b_{41}^2 < 4s_2b_{11},$$

$$3b_{14}^2 < 2k_3b_{11}s_2,$$

$$3k_3r_{b_{45}}^2 < 4r_0s_2b_{15}.$$ 

From inequalities (13), (14) and (15), we have

$$\frac{3b_{14}^2}{2b_{11}s_2} < k_3 < \frac{2s_2}{3} \min \left\{ \frac{b_{11}}{b_{41}}, \frac{2r_0b_{15}}{rb_{45}} \right\}.$$ 

The value of $k_3$ is obtained from (16) satisfying the inequality (10). 

**Appendix C**

**Proof** Let us define a positive definite function to establish the global stability of $E^*$ compatible with the reduced system (2) as,

$$G = \left( I - I^* \left( 1 + \frac{ln I}{\beta} \right) \right) + \frac{m_1}{2}(R - R^*)^2 + \frac{m_2}{2}(N - N^*)^2$$

$$+ \frac{m_3}{2}(B - B^*)^2 + \frac{m_4}{2}(M - M^*)^2.$$ 

where $m_i$'s ($i = 1, \ldots, 4$) are constant which are all positive and to be chosen relevantly. The derivative of $G$ with respect to time including the model system (2) with $m_1 = \frac{\beta}{r}$ and $m_2 = \frac{\beta}{a}$ is given by,

$$\frac{dG}{dt} = -\left[ \beta + \frac{\eta(N - R)}{I^*} \left( 1 - \frac{\omega M}{p + M} \right) \frac{B}{K + B} \right] (I - I^*)^2$$

$$- \frac{\eta \tilde{d} d}{\alpha} (R - R^*)^2$$

$$- \frac{\eta \tilde{d} d}{\alpha} (N - N^*)^2 - m_3s_2(B - B^*)^2 - m_4r_0(M - M^*)^2$$

$$+ \frac{\eta}{I^*} \left( 1 - \frac{\omega M}{p + M} \right) \frac{B}{K + B} (I - I^*) (N - N^*)$$

$$- \frac{\eta}{I^*} \left( 1 - \frac{\omega M}{p + M} \right) \frac{B}{K + B} (I - I^*) (R - R^*)$$

$$+ \frac{(N^* - I^* - R^*)}{I^*} \left( 1 - \frac{\omega M}{p + M} \right)$$

$$- \frac{K\eta}{(K + B)(K + B^*)} (B - B^*) (I - I^*)$$

$$- \frac{B}{(K + B)} (M - M^*) (I - I^*)$$

$$+ m_3s_1 \left( 1 - \frac{\delta M}{m + M} \right) (B - B^*) (I - I^*)$$

$$- \frac{m_3\delta M}{(m + M)(m + M^*)} (B - B^*) (M - M^*)$$

$$+ m_4r(I - I^*) (M - M^*).$$ 

$\frac{dG}{dt} < 0$ in the domain of attraction $\Omega$ if the following conditions hold:

$$\left[ \frac{\eta \Lambda s_1}{ds KI^*} \right]^2 < 2\beta^2d < \frac{2\beta^2d}{3\alpha},$$ 

$$\left[ \frac{\eta \Lambda s_1}{ds KI^*} \right]^2 < 2\beta^2(\delta + d) < \frac{2\beta^2(\delta + d)}{3v},$$ 

$$\left[ \frac{N^* - I^* - R^*}{I^*} \left( 1 - \frac{\omega M^*}{p + M^*} \right) \frac{\eta}{K + B^*} \right]^2 < \frac{2m_3\beta s_2}{9},$$ 

$$\left[ \frac{N^* - I^* - R^*}{I^*} \omega \frac{\Lambda \eta s_1}{m + M^* d K s_2} \right]^2 < \frac{2m_4\beta r_0}{3},$$ 

$$m_3s_1^2 < \frac{2\beta s_2}{9},$$

$$m_3 \left[ \frac{\delta I^*}{m + M^*} \right]^2 < \frac{4r_0m_4s_2}{9},$$

$$m_4 < \frac{2\beta r_0}{9r^2}.$$ 

 Springer
From the inequalities (18) and (19), we get the inequality (11). Also from the inequality (24), choosing the value of \( m_4 = \frac{\beta_0}{q_4} \) and then using inequalities (20), (22), and (23) the positive value of \( m_3 \) can be obtained satisfying the inequality (12).

Acknowledgements Rabindra Kumar Gupta is thankful to UGC Nepal for partial financial support in the form of “PhD Fellowship and Research Support” (No. PhD/76-77 S & T-17) and Sounmitra Pal is thankful to Council of Scientific and Industrial Research (CSIR), Government of India for providing financial support in the form of senior research fellowship (File No. 09/013(0915)/2019-EMR-I). The authors are thankful to anonymous reviewers and the editor for their valuable comments and suggestions, which have helped in improving the paper.

Data availability The data that support the findings of this study are available within the article.

Declarations

Conflict of interest The authors have no conflicts to disclose.

References

Basir FA (2018) Dynamics of infectious diseases with media coverage and two time delay. Math Models Comput Simul 10(6):770–783. https://doi.org/10.1134/S2070048219010071

Bortz DM, Nelson PW (2004) Sensitivity analysis of a nonlinear lumped parameter model of HIV infection dynamics. Bull Math Biol 66(5):1009–1026. https://doi.org/10.1016/j.bulm.2003.10.011

Chavez CC, Song B (2004) Dynamical models of tuberculosis and their applications. Math Biosci Eng 1(2):361. https://doi.org/10.3934/mbe.2004.1.361

Chang X, Liu M, Jin Z, Wang J (2020) Studying on the impact of media coverage on the spread of COVID-19 in Hubei Province, China. Math Biosci Eng 17(4):3147–3159. https://doi.org/10.3934/mbe.2020178

Chitnis N, Hyman JM, Cushing JM (2008) Determining important parameters in the spread of malaria through the sensitivity analysis of a mathematical model. Bull Math Biol 70(5):1272–1296. https://doi.org/10.1007/s11538-008-9299-0

Chukwu CW, Nyabadza F (2021) Mathematical Modeling of Listeriosis Incorporating Effects of Awareness Programs. Math Models Comput Simul 13(4):723–741. https://doi.org/10.1134/S2070048220040116

Codoce CT (2001) Epidemic and endemic dynamics of cholora: the role of the aquatic reservoir. BMC Infect Dis 1(1):1–14. https://doi.org/10.1186/1471-2334-1-1

Collinson S, Khan K, Heffernan JM (2015) The effects of media reports on disease spread and important public health measurements. PLoS ONE 10(11):e0141423. https://doi.org/10.1371/journal.pone.0141423

Cui J, Sun Y, Zhu H (2008) The impact of media on the control of infectious diseases. J Dyn Diff Eqn 20(1):31–53. https://doi.org/10.1007/s10884-007-9075-0

Dieckmann O, Heesterbeek JAP, Roberts MG (2010) The construction of next-generation matrices for compartmental epidemic models. J R Soc Interface 7(47):873–885. https://doi.org/10.1098/rsif.2009.0386

Dubey B, Dubey P, Dubey US (2016) Role of media and treatment on an SIR model. Nonlinear Anal: Model and Control 21(2):185–200 https://doi.org/10.15388/NA.2016.2.3

Eslami M (2013) Theory of sensitivity in dynamic systems: an introduction. Springer Sci & Busi Media

Funk S, Gilad E, Watkins C, Jansen VA (2009) The spread of awareness and its impact on epidemic outbreaks. Proc Natl Acad Sci 106(16):6872–6877. https://doi.org/10.1073/pnas.0810762106

Huo HF, Yang P, Xiang H (2018) Stability and bifurcation for an SEIS epidemic model with the impact of media. Phys A: Stat Mech Appl 490:702–720. https://doi.org/10.1016/j.physa.2017.08.139

Kemp S (2017) Digital in 2017 global overview (28.01.18) https://wearesocial.com/special-reports/digital-in-2017-global-overview

Khan MA, Ondisyah HP (2020) Fractional model of HIV transmission with awareness effect. Chaos Solit Fractals 138:109676. https://doi.org/10.1016/j.chaos.2020.109676

Khan MA, Ullah S, Khan Y, Farhan M (2020) Modeling and scientific computing for the transmission dynamics of avian influenza with half-saturated incidence. Int J Model simul Sci Comput 11(04):2050035. https://doi.org/10.1142/S179362302050035X

Khan MF, Alrabaiah H, Ullah S, Khan MA, Farooq M, Mamat MB, Asjad MI (2021) A new fractional model for vector-host disease with saturated treatment function via singular and non-singular operators. Alex Eng J 60(1):629–645. https://doi.org/10.1016/j.alexj.2020.09.057

Kumar A, Srivastava PK, Takeuchi Y (2017) Modeling the role of information and limited optimal treatment on disease prevalence. J Theor Biol 414:103–119. https://doi.org/10.1016/j.jtbi.2016.11.016

Larson HJ, Jarrett C, Eckersberger E, Smith DM, Paterson P (2014) Understanding vaccine hesitancy around vaccines and vaccination from a global perspective: a systematic review of published literature, 2007 2012. Vaccine 32(19):2150–2159. https://doi.org/10.1016/j.vaccine.2014.01.081 (PMID:24598724)

Liu R, Wu J, Zhu H (2007) Media/psychological impact on multiple outbreaks of emerging infectious diseases. Comput Math Methods Med 8(3):153–164

Liu Y, Cui JA (2008) The impact of media coverage on the dynamics of infectious disease. Int J Biomath 1(01):65–74. https://doi.org/10.1142/S1793524508000235

Lopez AD, Mathers CD, Ezzati M, Jamison TD, Murray CJ (2006) Changes in individual behavior could limit the spread of infectious diseases. London University Press, London http://www.dcp2.org/le/6/

Mara D, Lane J, Scott B, Trouba D (2010) Sanitation and health. PLoS Med 7(11):e1000363. https://doi.org/10.1371/journal.pmed.1000363

Misra AK, Sharma A, Shukla JB (2011) Modeling and analysis of an HIV/AIDS model with public-health information and limited optimal treatment on disease prevalence. Math Biosci Eng 15(6):1315. https://doi.org/10.3934/mbe.2018061

Misra AK, Rai RK, Takeuchi Y (2018) Modeling the control of infectious diseases: effects of TV and social media advertisements. Math Biosci Eng 15(6):1315. https://doi.org/10.3934/mbe.2018061

Musa SS, Zhao S, Hussaini N, Usaiyi S, He D (2021) Dynamics analysis of typhoid fever with public health education programs and final epidemic size relation. Results Appl Math 10:100153. https://doi.org/10.1016/j.ram.2021.100153

Njankou SDD, Nyabadza F (2017) Modelling the potential role of media campaigns in Ebola transmission dynamics. Int J Diff Equ 2017:3758269. https://doi.org/10.1155/2017/3758269

Nyabadza F, Chiyaka C, Mukandavire Z, Hove-Musekwa SD (2010) Analysis of an HIV/AIDS model with public-health information
campaigns and individual withdrawal. J Biol Syst 18(02):357–375. https://doi.org/10.1142/S0218339010003329
Shanta SS, Biswas MHA (2020) The impact of media awareness in controlling the spread of infectious diseases in terms of SIR model. Math Model Eng Prob 7(3):368-376 https://doi.org/10.18280/mmepe.070306
Shukla JB, Naresh R, Verma SR, Agarwal M (2020) Modeling the effect of sanitation in a human habitat to control the spread of bacterial diseases. Model Earth Syst Environ 6(1):39–49. https://doi.org/10.1007/s40808-019-00653-4
Sundar S, Mishra AK, Naresh R (2018) Modeling the impact of awareness programs on mitigation of carbon dioxide emitted from automobiles. Model Earth Syst Environ 4(1):349–357. https://doi.org/10.1007/s40808-017-0401-1
Swachh Bharat Mission-Grameen (2017) Department of Drinking Water and Sanitation, Government of India. https://swachhbharatmission.gov.in/sbcmcms/index.htm
Tchuenche JM, Dube N, Bhunu CP, Smith RJ, Bauch CT (2011) The impact of media coverage on the transmission dynamics of human influenza. BMC Public Health 11(1):1–14. https://doi.org/10.1186/1471-2458-11-S1-S5
Tiwari PK, Rai RK, Gupta RK, Martcheva M, Misra AK (2022) Modeling the control of bacterial disease by social media advertisements: effects of awareness and sanitation. J Biol Syst 30(01):51–92
Tripathi A, Tripathi RN, Sharma D (2021) A mathematical model to study the COVID-19 pandemic in India. Mod Earth Syst Environ 1–12. https://doi.org/10.1007/s40808-021-01280-8
Wang X, Gao D, Wang J (2015) Influence of human behavior on cholera dynamics. Math Biosci 267(2015):41–52. https://doi.org/10.1016/j.mbs.2015.06.009
WHO, World Health Organisation, Media Centre, Pneumonia - Fact Sheet No 331, (2012). http://www.who.int/mediacentre/factsheets/fs331/en/index.html
WHO, World Health Organisation, Media Centre, Diarrhoeal Disease - Fact Sheet No 330, (2009). http://www.who.int/mediacentre/factsheets/fs330/en/index.html
WHO, World Health Organisation, (2019). https://www.who.int/news-room/fact-sheets/detail/sanitation
Yang C, Wang X, Gao D, Wang J (2017) Impact of awareness programs on cholera dynamics: two modeling approaches. Bull Math Biol 79(9):2109–2131. https://doi.org/10.1007/s11538-017-0322-1
Publisher’s Note Springer Nature remains neutral with regard to jurisdictional claims in published maps and institutional affiliations.

Springer Nature or its licensor holds exclusive rights to this article under a publishing agreement with the author(s) or other rightsholder(s); author self-archiving of the accepted manuscript version of this article is solely governed by the terms of such publishing agreement and applicable law.