# Analysis of An SEIQRS Epidemic Infectious Disease Model with Non-Pharmaceutical Interventions (NPIs) through Media Coverage as Control Strategy

Smriti Agrawal<sup>1</sup>, Nimisha Mishra\*

Amity School Of Applied Sciences, Amity University, Lucknow, Uttar Pradesh 226028, India.

#### **Abstract**

In this paper, a SEIQRS non-linear numerical model for the impacts of awareness programs on the spread of irresistible illnesses has been proposed and broke down. In the modelling system, it is expected that sickness spreads because of the contact among susceptibles and infectives as it were. The growth rate of awareness programs affecting the population is thought to be corresponding to the number of infective people. It is additionally expected that because of the impact of media, susceptible populations from a different class and stay away from contact with the infectives. The model is examined by utilizing the stability theory of differential equations. The model analysis shows that the spread of an irresistible infection can be constrained by utilizing awareness programs yet the sickness stays endemic because of movement. The reproduction examination of the model affirms the analytical outcomes. The results indicate that media coverage can reduce the burden of the epidemic and shorten the duration of the disease outbreak.

**Keywords:** Epidemic model, Non-Pharmaceutical Interventions (NPIs), Fundamental reproduction number, Global stability, Local stability, Sensitivity Analysis.

#### 1. INTRODUCTION

Controlling the spread of irresistible sicknesses to decrease the impacts of disease on a population is a significant command of general wellbeing. Irresistible infectious prevention can be accomplished through different methodologies including inoculation, the utilization of medication treatment, hand washing, wearing masks and social separating - eliminating oneself as much as could be expected from the population. Immunizations and medication treatments, be that as it may, might be ineffectual or inaccessible. Subsequently, hand washing, wearing masks and social separating rehearses, which can be utilized consistently during an irresistible illness episode are basic in diminishing the likelihood of contracting and sending disease. Broad communications missions can be utilized to give data on current and viable inoculation, drug treatment and social separating measures. General wellbeing instruction crusades, that incorporate enlightening writing, banners, paper articles and notices, radio and TV messages, and web-based media outlets are utilized every day to advise the general population on current medical problems. Broad communications outlets can help in the scattering of this data. Investigations of broad communications crusades and sound conduct have revealed that broad communications missions can evoke positive conduct change and even forestall negative conduct change in people. It is hence presumed that broad communications missions ought to be utilized to educate the public so conduct change can result. It has been demonstrated that data passed on by the media is turning into the basic factor concerning whether an immunization mission will succeed [1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 11].

The media inclusion is clearly not the main factor liable for the transmission of the irresistible illness, however it is a vital issue which must be dealt with truly. On account of countless tainted cases, on one hand, the media inclusion may cause the frenzy of the general public, while then again, it can surely diminish the chance and likelihood of contact transmission among the alarmed defenseless populations, which thus assists with controlling and keep the sickness from additional spreading [12, 13, 14, 15].

Media inclusion about a scourge gives a sense about the danger level and the overall requirement for insurances in hazard regions and urge the general population to take prudent steps against the illness like wearing veils, staying away from public spots, evading travel when debilitated, continuous hand washing, and so on. This is critical in the beginning phases of a scourge, when drug intercessions are not regularly conceivable in light of the fact that treatment or inoculation alternatives have not yet been created. Numerous scientists researched the effect of media mindfulness utilizing mathematical modelling. Cui utilized transmission coefficient capacity of the structure  $\beta(I) = \beta e^{-mI}$  and set up that different positive equilibria are conceivable

when the media impact is adequately solid. Non direct capacity of the structure  $\beta(I) = c_1 - c_2 f(I)$  is joined in the transmission term to research the impact of media inclusion, where  $f(I) = \frac{I}{m+I}$  [?, 13, 14, 15, 16, 17, 18, 19, 20].

In the mopdelling of irresistible illnesses, the rate work assumes a vital part, it can decide the ascent and fall of scourges. In numerous pestilence models, the bilinear rate  $\beta\widetilde{S}\widetilde{I}$  and the standard rate  $\frac{\beta\widetilde{S}\widetilde{I}}{\widetilde{N}}$  are regularly utilized, where  $\beta$  gauges the impact of both the irresistibleness of the sickness and the contact transmission rates. Anyway these rate capacities don't consider the effect of media inclusion to the spread and control of irresistible illnesses. The utilization of suggested non-drug intercessions through media inclusion and alarm has been discovered valuable for lessening illness trouble in some irresistible infections. Liu and Cui utilized media incited transmission rate of the structure  $\beta(I) = \beta e^{-mI}$  which has two significant restrictions. We consider media actuated transmission rate as  $\beta(I) = \beta e^{-m\frac{I}{N}}$  in the proposed model which is more reasonable than  $\beta(I) = \beta e^{-mI}$ , on the grounds that  $\beta e^{-mI} \to 0$  as  $I \to \infty$ , free of the estimation of m. Since the media inclusion and readiness are not the inherent deterministic factor answerable for the transmission, thus it is sensible to expect that the transmission rate can't be decreased under a specific level only through media alert. Also, in any event, for a fixed m, the base transmission rate varies from various population sizes, paying little mind to the comparability in friendly construction and climatic condition, which isn't sensible. Then again,  $\min\{\beta e^{-m\frac{1}{N}}\} = \beta e^{-m}$  that stays unaltered regarding the absolute population size [21, 22, 23, 24, 25, 26, 27, 28, 29, 30].

The point of this paper is to explore the effect of media inclusion to the spread and control of irresistible sicknesses in a given locale. The rest of this paper is coordinated as follows: In Section 2 We proposed a numerical model as the ODE framework and characterized all parameters utilized in the model. In Section 3, we calculate all possible steady state and basic reproduction numbers. Graphical portrayals are introduced In Section 4 and furthermore numerical simulations are performed to check the outcomes, in the examination, biologically relevant parameter values are used. In Section 5, sensitivity analysis and furthermore found highly sensitive parameters. At long last, in Section 6, the outcomes are talked about.

#### 2. FORMULATION OF MATHEMATICAL MODEL

In this section, we introduce SEIQRS pandemic infectious disease system with induced media transmission rate  $\beta e^{-m\frac{\tilde{I}}{\tilde{N}}}$ . We divided total population into five compartments at time  $\tilde{t}$ , which are Susceptible( $\tilde{S}$ ), Exposed( $\tilde{E}$ ), Infected( $\tilde{I}$ ), Quarantine( $\tilde{Q}$ ) and Recovered ( $\tilde{R}$ ). Let us consider that the total population at time  $\tilde{t}$  is  $\tilde{N}(\tilde{t}) = \tilde{S}(\tilde{t}) + \tilde{S}(\tilde{t})$ 

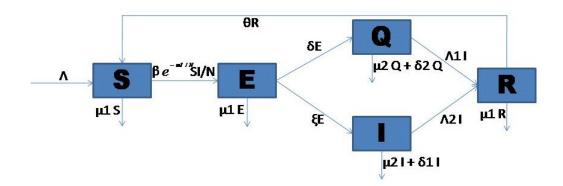


Figure 1: Schematic flow of proposed an SEIQRS epidemic Model.

 $\tilde{E}(\tilde{t}) + \tilde{I}(\tilde{t}) + \tilde{Q}(\tilde{t}) + \tilde{R}(\tilde{t})$ . The schematic flow diagram of the presented epidemic infectious disease mathematical system model including the coverage of media for homogeneously population which is shown in Figure 1, the model is presented with ODEs which are given below:

$$\frac{d\tilde{S}}{d\tilde{t}} = \Lambda - \tilde{\beta}e^{-m\frac{\tilde{I}}{\tilde{N}}}\frac{\tilde{S}\tilde{I}}{\tilde{N}} - \tilde{\mu}\tilde{S} + \tilde{\theta}\tilde{R}, \tag{1}$$

$$\frac{d\tilde{E}}{d\tilde{t}} = \tilde{\beta}e^{-m\frac{\tilde{I}}{\tilde{N}}}\frac{\tilde{S}\tilde{I}}{\tilde{N}} - (\tilde{\mu} + \tilde{\xi} + \tilde{\delta})\tilde{E}, \tag{2}$$

$$\frac{d\tilde{I}}{d\tilde{t}} = \tilde{\xi}\tilde{E} - (\tilde{\mu} + \tilde{\delta}_1 + \tilde{\Lambda}_1)\tilde{I},$$
(3)

$$\frac{d\tilde{Q}}{d\tilde{t}} = \tilde{\delta}\tilde{E} - (\tilde{\mu} + \tilde{\delta}_2 + \tilde{\Lambda}_2)\tilde{Q}, \tag{4}$$

$$\frac{d\tilde{R}}{d\tilde{t}} = \tilde{\Lambda}_1 \tilde{I} - \tilde{\Lambda}_2 \tilde{Q} (\tilde{\mu} + \tilde{\theta}) \tilde{R}, \tag{5}$$

with initial conditions:

$$\tilde{S}(0) = \tilde{S}_0 > 0, \ \tilde{E}(0) = \tilde{E}_0 > 0, \ \tilde{I}(0) = \tilde{I}_0 > 0, \ \tilde{Q}(0) = \tilde{Q}_0 > 0, \ \tilde{R}(0) = \tilde{R}_0 > 0.$$
 (6)

Now, we suppose the initial conditions for the solution which are inside feasible biologically region

$$\Omega = \left\{ (\tilde{S}, \tilde{E}, \tilde{I}, \tilde{Q}, \tilde{R}) : 0 \leq \tilde{S}, \tilde{E}, \tilde{I}, \tilde{Q}, \tilde{R} \leq \frac{\Lambda}{\tilde{\mu}}, \right\}$$

We study model system (1)-(6) and assert that region  $\Omega$  is positively invariant which is bounded with respect to presented model (1)-(6).

.

Parameter	Description	Unit
Λ	Recruitment rate of S	$days^{-1}$ $days^{-1}$
$\tilde{eta}$	Contact rate of I with S	$\rm days^{-1}$
$\mid m \mid$	Coefficient of media coverage or awareness	
$\mid  ilde{\mu} \mid$	Natural death rate for all	$\rm days^{-1}$
$ ilde{\delta}$	Rate of quarantine for exposed individuals	days <sup>-1</sup> days <sup>-1</sup>
$  ilde{\xi} $	Rate of infectious for exposed individuals	$\rm days^{-1}$
$ ilde{\Lambda_1}$	Rate of recovery for infectious individuals	$\rm days^{-1}$
$ ilde{\Lambda_2}$	Rate of recovery for quarantine individuals	days <sup>-1</sup>
$\mid  ilde{ heta} \mid$	Rate of transfer for recovered individuals to susceptible individuals	days <sup>-1</sup>
$\delta_1$	Death rate induced by disease for infectious individuals	days <sup>-1</sup>
$\delta_2$	Death rate induced by disease for quarantined individuals	$\rm days^{-1}$

Table 1: Definition of parameters for the system (7)-(11)

Also, the total population

$$\tilde{N}(\tilde{t}) = \tilde{S}(\tilde{t}) + \tilde{E}(\tilde{t}) + \tilde{I}(\tilde{t}) + \tilde{Q}(\tilde{t}) + \tilde{R}(\tilde{t}),$$

satisfies

$$\frac{d\tilde{N}}{d\tilde{t}} = \Lambda - \tilde{\mu}\tilde{N} - \tilde{\delta}_1\tilde{I} - \tilde{\delta}_2\tilde{Q}.$$

Then,

$$\frac{d\tilde{N}}{d\tilde{t}} < \Lambda - \tilde{\mu}\tilde{N},$$

by applying Birkhoff's and Rota's theorem [1, 10], as  $\tilde{t}\to\infty$ , also  $\tilde{N}^0=0\leq \tilde{N}(\tilde{t})\leq \frac{\Lambda}{\tilde{\mu}}$ . Hence, the model system's solution (1)-(6) is bounded

Now, we use the model system (1)-(6) for non-dimensionalise,

$$S = \frac{\tilde{S}}{\tilde{N}}, \ E = \frac{\tilde{E}}{\tilde{N}}, \ I = \frac{\tilde{I}}{\tilde{N}}, \ Q = \frac{\tilde{Q}}{\tilde{N}}, \ R = \frac{\tilde{R}}{\tilde{N}}, \ N = \frac{\tilde{N}}{\tilde{N}^0}, \ t = \tilde{\mu}\tilde{t}.$$

Since S = 1 - (E + I + Q + R), we can drop equation,

$$\frac{dS}{dt} = \frac{1}{N} - \beta e^{-mI}SI + \theta R - \frac{S}{N} + \delta_1 SI + \delta_2 SQ,$$

the identical non-dimensional model system is following:

$$\frac{dE}{dt} = \beta e^{-mI} (1 - E - I - Q - R)I - \xi E - \frac{E}{N} + \delta_1 EI + \delta_2 EQ,$$
 (7)

$$\frac{dI}{dt} = \xi E - (\delta_1 + \Lambda_1)I - \frac{I}{N} + \delta_1 I^2 + \delta_2 IQ, \tag{8}$$

$$\frac{dQ}{dt} = \delta E - (\delta_2 + \Lambda_2)Q - \frac{Q}{N} + \delta_1 IQ + \delta_2 I^2, \tag{9}$$

$$\frac{dR}{dt} = \Lambda_1 I + \Lambda_2 Q - \theta R - \frac{R}{N} + \delta_1 R I + \delta_2 Q R, \tag{10}$$

$$\frac{dN}{dt} = 1 - (1 + \delta_1 I + \delta_2 Q)N,\tag{11}$$

where

$$\beta = \frac{\tilde{\beta}}{\tilde{\mu}}, \; \delta = \frac{\tilde{\delta}}{\tilde{\mu}}, \; \xi = \frac{\tilde{\xi}}{\tilde{\mu}}, \; \Lambda_1 = \frac{\tilde{\Lambda_1}}{\tilde{\mu}}, \; \Lambda_2 = \frac{\tilde{\Lambda_2}}{\tilde{\mu}}, \; \delta_1 = \frac{\tilde{\delta_1}}{\tilde{\mu}}, \; \delta_2 = \frac{\tilde{\delta_2}}{\tilde{\mu}}, \; \theta = \frac{\tilde{\theta}}{\tilde{\mu}},$$

and with the initial condition:

$$E(0) = E_0 > 0, I(0) = I_0 > 0, Q(0) = Q_0 > 0, R(0) = R_0 > 0, N(0) = N_0 > 0.$$
(12)

#### 3. ANALYSIS OF THE MODEL

In this part, we will analyze the basic reproduction number of  $calR_0$ , the equilibrium of all feasible states, and the local and global stability of these two states (disease-free and endemic).

Analyze that feasible biologically state for the model system is,

$$\Gamma = \{ (E, I, Q, R, N) : 0 \le S, E, I, Q, R, N \le 1 \},$$

that is positively invariant for the model system (7)-(12). The model system (7)-(12) always has DFE  $E^0 = (0, 0, 0, 0, 1)$ .

# 3.1. Basic reproduction number

The basic reproduction number,  $\mathcal{R}_0$ , is defined as the expected number of secondary cases produced by a single (typical) infection in a completely susceptible population. Suppose,

x = (E, I, Q, R), then from model (7)-(12), it follows:

$$\frac{dx}{dt} = \mathcal{F} - \mathcal{V},$$

where,

Suppose,

 $x=(E,\;I,\;Q)$ , from system model (7)-(12), where:  $\frac{dx}{dt}=\mathcal{F}-\mathcal{V},$  where

$$\mathcal{F} = \begin{pmatrix} \beta e^{-mI} (1 - E - I - Q - R)I \\ 0 \\ 0 \end{pmatrix}$$

and

$$\mathcal{V} = \begin{pmatrix} \xi E + \frac{E}{N} - \delta_1 E I - \delta_2 E Q \\ -\xi E + \frac{I}{N} + \Lambda_1 I + \delta_1 I - \delta_1 I^2 - \delta_2 I Q \\ -\delta E + \frac{Q}{N} + \Lambda_2 I + \delta_2 I - \delta_1 I Q - \delta_2 Q^2 \end{pmatrix}.$$

We have,

$$\mathbf{F} = D\mathcal{F}|_{E^0} = \left( egin{array}{ccc} 0 & 0 & eta \ 0 & 0 & 0 \ 0 & 0 & 0 \end{array} 
ight)$$

and

$$\mathbf{V} = D\mathcal{V}|_{E^0} = \begin{pmatrix} \xi + 1 & 0 & 0 \\ -\xi & 1 + \Lambda_1 + \delta_1 & 0 \\ -\delta & 0 & 1 + \Lambda_2 + \delta_2 \end{pmatrix}.$$

The next generation matrix for the reproduction number of the model is given by

$$K = FV^{-1} = \begin{pmatrix} \frac{\beta\xi}{(1+\xi)(1+\delta_1+\Lambda_1)} & \frac{\beta}{1+\delta_1+\Lambda_1} & 0\\ 0 & 0 & 0\\ 0 & 0 & 0 \end{pmatrix}.$$

 $\mathcal{R}_0$  denotes the basic reproduction number, which is explained as the radius of spectral of the matrix of next generation  $K = FV^{-1}$ , which is,  $\mathcal{R}_0 = \rho(FV^{-1})$ . Hence,  $\mathcal{R}_0$  for model system (7)-(12) is given by,

$$\mathcal{R}_0 = \frac{\beta \xi}{(1+\xi)(1+\delta_1+\Lambda_1)}.$$
 (13)

## 3.2. Interior Equilibrium Points

Further, the system (7)-(12) has an endemic equilibrium which is given by,

$$\bar{E} = (E^*, I^*, Q^*, R^*, N^*),$$

where,

$$E^* = \frac{1 + \delta_1 + \Lambda_1}{\xi} I^*,$$

$$Q^* = \frac{\delta(1 + \delta_1 + \Lambda_1)}{\xi(1 + \delta_2 + \Lambda_2)} I^*,$$

$$R^* = \frac{\Lambda_1 + \frac{\Lambda_2 \delta(1 + \delta_1 + \Lambda_1)}{\xi(1 + \delta_2 + \Lambda_2)}}{1 + \theta} I^*,$$

$$N^* = \frac{1}{1 + \delta_1 I^* + \frac{\delta_2 \delta(1 + \delta_1 + \Lambda_1)}{\xi(1 + \delta_2 + \Lambda_2)}} I^*,$$

The solution of the equation the value of  $I^*$  is given by,

$$\frac{e^{mI^*}}{\mathcal{R}_0} = 1 - \left(1 + \frac{1 + \delta_1 + \Lambda_1}{\xi} + \frac{\delta(1 + \delta_1 + \Lambda_1)}{\xi(1 + \delta_2 + \Lambda_2)} + \frac{\Lambda_1 + \frac{\Lambda_2\delta(1 + \delta_1 + \Lambda_1)}{\xi(1 + \delta_2 + \Lambda_2)}}{1 + \theta}\right)I^*. \tag{14}$$

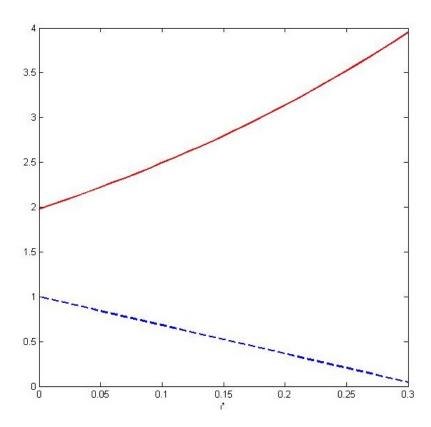


Figure 2: EE not exists for values of parameters  $\tilde{\beta}=1, m=2.3, \mathcal{R}_0=0.4545<1.$ 

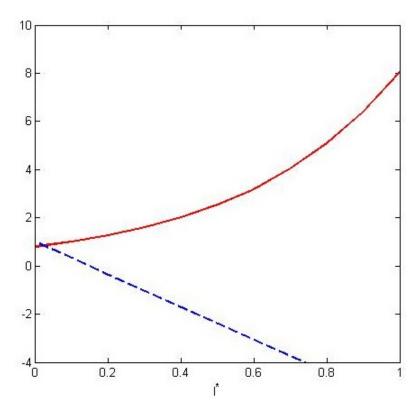


Figure 3: EE exists for values of parameters  $\tilde{\beta} = 8, m = 2.3, \mathcal{R}_0 = 3.6363 > 1.$ 

## 3.3. Local Stability Analysis of Disease-free and Endemic Equilibrium

In this subpart of the chapter, we analyze the local stability of the system (7)-(12) for both the states endemic and disease-free equilibria which are analyzed as following:

## 3.3.1 Local stability of disease-free equilibrium

The matrix of variational at disease-free equilibrium is shown by,

$$\mathbf{J_0} = \begin{pmatrix} -1 - \xi & \beta & 0 & 0 & 0 \\ \xi & -1 - \delta_1 - \Lambda_1 & 0 & 0 & 0 \\ \delta & 0 & -1 - \delta_2 - \Lambda_2 & 0 & 0 \\ 0 & \Lambda_1 & \Lambda_2 & -1 - \theta & 0 \\ 0 & -\delta_1 & -\delta_2 & 0 & -1 \end{pmatrix}. \tag{15}$$

The characteristic equation of  $J_0$  is shown by,

$$(1+\lambda)(1+\theta+\lambda)(\Lambda_2+\delta_2+1+\lambda) \lambda^2 + \lambda(2+\xi+\Lambda_1+\delta_1) + (\xi+1)(\Lambda_1+\delta_1+1)(1-\mathcal{R}_0) = 0.$$
 (16)

Clearly, the all five eigen values have real negative parts of  $J_0$ , if  $\mathcal{R}_0 < 1$  and the four eigen values have real negative parts and only one eigen value has real positive part of  $J_0$ , if  $\mathcal{R}_0 > 1$ . therefore, the disease-free equilibria is unstable, if  $\mathcal{R}_0 > 1$  and locally asymptotically stable, if  $\mathcal{R}_0 < 1$ .

#### 3.3.2 Local stability of endemic equilibrium

The bifurcation parameter's critical value  $\beta$  at the  $\mathcal{R}_0=1$  is  $\beta^*=\frac{(1+\xi)(1+\delta_1+\Lambda_1)}{\xi}$ . This can easily satisfied the  $\mathbf{J_0}$  at  $\beta=\beta^*$  has one right eigenvector which is shown by:

$$\mathbf{W} = (w_1, w_2, w_3, w_4, w_5)^T,$$

where,

$$\begin{split} w_1 &= 1 + \delta_1 + \Lambda_1, \ w_2 = \xi, \ w_3 = \frac{\delta(1 + \delta_1 + \Lambda_1)}{1 + \delta_2 + \Lambda_2}, \\ w_4 &= \frac{1 + \delta_1 + \Lambda_1}{1 + \theta} \left[ \frac{\Lambda_1 \xi}{1 + \delta_1 + \Lambda_1} + \frac{\Lambda_2 \delta}{1 + \delta_2 + \Lambda_2} \right], \\ w_5 &= -(1 + \delta_1 + \Lambda_1) \left[ \frac{\delta_1 \xi}{1 + \delta_1 + \Lambda_1} + \frac{\delta_2 \delta}{1 + \delta_2 + \Lambda_2} \right]. \end{split}$$

Further, the left eigenvector components are,

$$\mathbf{V} = (v_1, v_2, v_3, v_4, v_5),$$

must be verify the given equalities  $\mathbf{V}.\mathbf{J_0}=0$  and  $\mathbf{V}.\mathbf{W}=1$ , thus, we have conclude,

$$v_1 = \frac{1}{2+\xi+\delta_1+\Lambda_1}, \ v_2 = \frac{\xi+1}{\xi(2+\xi+\delta_1+\Lambda_1)}, \ v_3 = v_4 = v_5 = 0,$$

The non-zero partial derivatives of  $\mathbf{F}=(f_1,f_2,f_3,f_4)^T$  at the disease-free equilibria and  $\beta=\beta^*$  is shown as

$$\frac{\partial^2 f_1}{\partial E \partial I} = \frac{\partial^2 f_1}{\partial I \partial E} = \delta_1 - \frac{(1+\xi)(1+\delta_1+\Lambda_1)}{\xi}, \quad \frac{\partial^2 f_1}{\partial E \partial Q} = \frac{\partial^2 f_1}{\partial Q \partial E} = \delta_2, \quad \frac{\partial^2 f_1}{\partial E \partial N} = \frac{\partial^2 f_1}{\partial N \partial E} = 1,$$

$$\frac{\partial^2 f_1}{\partial I^2} = \frac{-2(1+m)(1+\xi)(1+\Lambda_1+\delta_1)}{\xi}, \quad \frac{\partial^2 f_1}{\partial R \partial I} = \frac{\partial^2 f_1}{\partial I \partial R} = \frac{\partial^2 f_1}{\partial I \partial Q} = \frac{\partial^2 f_1}{\partial Q \partial I} = -\beta, \quad \frac{\partial^2 f_2}{\partial I^2} = 2\delta_1,$$

$$\frac{\partial^2 f_2}{\partial I \partial Q} = \frac{\partial^2 f_2}{\partial Q \partial I} = \delta_2, \quad \frac{\partial^2 f_2}{\partial I \partial N} = \frac{\partial^2 f_2}{\partial N \partial I} = 1, \quad \frac{\partial^2 f_3}{\partial Q \partial N} = \frac{\partial^2 f_3}{\partial N \partial Q} = 1, \quad \frac{\partial^2 f_3}{\partial I \partial Q} = \frac{\partial^2 f_3}{\partial Q \partial I} = \delta_1,$$

(18)

$$\frac{\partial^2 f_3}{\partial Q^2} = \delta_2, \ \frac{\partial^2 f_4}{\partial R \partial N} = \frac{\partial^2 f_4}{\partial N \partial R} = 1, \ \frac{\partial^2 f_4}{\partial I \partial R} = \frac{\partial^2 f_4}{\partial R \partial I} = \delta_1, \ \frac{\partial^2 f_4}{\partial Q \partial R} = \frac{\partial^2 f_4}{\partial R \partial Q} = \delta_2,$$

$$\frac{\partial^2 f_5}{\partial I \partial N} = \frac{\partial^2 f_5}{\partial N \partial I} = -\delta_1, \ \frac{\partial^2 f_5}{\partial Q \partial N} = \frac{\partial^2 f_5}{\partial N \partial Q} = -\delta_2, \ \frac{\partial^2 f_1}{\partial I \partial \beta} = 1.$$

Here we used  $x_1 \equiv E, x_2 \equiv I, x_3 \equiv Q, x_4 \equiv R, x_5 \equiv N$ . Thus, we get

$$\mathbf{a} = \sum_{k,i,j=1}^{5} v_k w_i w_j \frac{\partial^2 f_k}{\partial x_i \partial x_j} (0,0)$$

$$= v_1 [2w_1 w_2 \delta_1 - \frac{(1+\xi)(1+\delta_1+\Lambda_1)}{\xi} + 2w_1 w_3 \delta_2 + 2w_1 w_5 + 2w_2 w_2$$

$$-\frac{2(1+m)(1+\xi)(1+\Lambda_1+\delta_1)}{\xi} + 2w_2 w_4 (-\beta) + 2w_2 w_3 (-\beta)]$$

$$+ v_2 [2w_2 w_5 + 2w_2 w_2 (2\delta_1) + 2w_2 w_3 \delta_2]$$

$$= \frac{-2(1+\delta_1+\Lambda_1)}{(2+\xi+\delta_1+\Lambda_1)(\delta_2+\Lambda_2+1)} [\xi(\Lambda_1+1) + (1+\delta_2\delta+\delta_2\xi+\delta\xi+\delta)(\delta_1+\Lambda_1+1)$$

$$+\xi(\Lambda_2\delta_1+\delta_1\delta_2+\delta_1+\delta\delta_2) + \frac{1}{\delta_1+\Lambda_1+1} \delta_1(\delta_2+\Lambda_2+1)(\xi^2+3\xi+2)$$

$$+(\xi^2+\xi)\delta_2(\delta_1+\Lambda_1+1) + \frac{1}{1+\theta}$$

$$2\xi(\xi+1)(1+m)(\delta_2+\Lambda_2+1)(1+\theta) + (1+\xi)(\delta_2+\Lambda_2+1)\xi\Lambda_1 + (\xi+1)(\delta_2+\Lambda_2+1)\delta\Lambda_2],$$

$$(17)$$

$$\mathbf{b} = \sum_{k,i=1}^{5} v_k w_i \frac{\partial^2 f_k}{\partial x_i \partial \phi} (0,0)$$

$$= v_1 w_2$$

$$= \frac{\xi}{2+\delta_1+\xi+\Lambda_1}.$$

$$(18)$$

Since a < 0 and b > 0 at  $\beta = \beta^*$ , hence, there is a bifurcation which is transcritical happens at  $\mathcal{R}_0 = 1$  and unique pandemic equilibria is Locally Asymptotically Stable at  $\mathcal{R}_0 > 1$ .

#### 3.4. Global stability analysis of disease-free equilibrium

Under this part, we analyze the global stability of the disease-free equilibrium. Let us consider that the death rate induced by disease  $\delta_1 = \delta_2 = 0$ , then  $\frac{dN}{dt} = 1 - N$ . In the case of limiting, we have  $N(t) \to 1$ , as  $t \to \infty$ . Hence, the model system which is reduced is shown as:

$$\frac{dE}{dt} = \beta e^{-mI} (1 - E - I - Q - R)I - (1 + \xi)E, \tag{19}$$

$$\frac{dI}{dt} = \xi E - (1 + \Lambda_1)I, \tag{20}$$

$$\frac{dQ}{dt} = \delta E - (1 + \Lambda_2)Q, \tag{21}$$

$$\frac{dE}{dt} = \beta e^{-mI} (1 - E - I - Q - R)I - (1 + \xi)E, \tag{19}$$

$$\frac{dI}{dt} = \xi E - (1 + \Lambda_1)I, \tag{20}$$

$$\frac{dQ}{dt} = \delta E - (1 + \Lambda_2)Q, \tag{21}$$

$$\frac{dR}{dt} = \Lambda_1 I + \Lambda_2 Q - (\theta + 1)R$$

and the modified  $\mathcal{R}_0$  is  $\mathcal{R}_0 = \frac{\beta \xi}{(\xi+1)(1+\Lambda_1)}$ .

Let  $Z=(E,\ I,\ Q),\ X=(R),$  here  $Q_0=(R^0,\ 0),$  where  $R^0=0.$  We have  $\frac{dX}{dt}=F(X,\ Z)=\Lambda_1I+\Lambda_2Q-(\theta+1)X.$  At  $R=R^0,\ G(X,\ 0)=0.$  Now  $\frac{dX}{dt}=F(X,\ 0)=-(\theta+1)X,$  as  $t\to\infty,\ X\to X^0.$  Thus,  $X=X^0(=R^0=0)$  is g.a.s. Now,  $G(X, Z) = BZ - \widehat{G}(X, Z)$ 

$$= \begin{pmatrix} -1 - \xi & \beta & 0 \\ \xi & -1 - \Lambda_1 & 0 \\ \delta & 0 & -1 - \Lambda_2 \end{pmatrix} \begin{pmatrix} E \\ I \\ Q \end{pmatrix} - \begin{pmatrix} \beta I (1 - e^{-mI}) + \beta I e^{-mI} (E + I + Q + R) \\ 0 \\ 0 \end{pmatrix}.$$

This may be easily satisfied that matrix

$$B = \begin{pmatrix} -1 - \xi & \beta & 0 \\ \xi & -1 - \Lambda_1 & 0 \\ \delta & 0 & -1 - \Lambda_2 \end{pmatrix}.$$

is a M-matrix and 
$$\widehat{G}(X,\ Z)=\left( egin{array}{ccc} eta I(1-e^{-mI})+eta Ie^{-mI}(E+I+Q+R) \\ 0 \\ 0 \end{array} 
ight) \ \geq \ 0.$$

Therefore, it verifies both the condition, thus the disease-free equilibrium  $E^0$  is globally asymptotically stable if  $R_0 < 1$ .

## 3.5. Uniform Persistence

The system (7)-(12) is said to be uniformly-persistent if there exists a constant c such that any solution (S(t), E(t), I(t), Q(t), R(t)) satisfies

$$\liminf_{t\to\infty} S(t) \geq c, \ \liminf_{t\to\infty} E(t) \geq c, \ \liminf_{t\to\infty} I(t) \geq c, \ \liminf_{t\to\infty} R(t) \geq c$$

provided that  $(S(0), E(0), I(0), Q(0), R(0)) \in \Gamma$ .

Similarly as in [12], the system (7)-(12) is uniformly-persistent in  $\Gamma$  if and only if  $\mathcal{R}_0 > 1$ . Since, the necessity of  $\mathcal{R}_0 > 1$  follows from global stability of disease-free equilibrium and the fact that the asymptotical stability of  $E^0$  precludes any kinds of

Table 2: Parametric values which are used for the numerical simulation of the model system (7)-(12)

Parameters	Values		
Λ	0.4		
$ ilde{eta}$	[1,8]		
m	2.3		
$ ilde{\mu}$	0.005		
$ ilde{\delta}$	0.8		
$egin{array}{c}  ilde{\mu} \  ilde{\delta} \  ilde{\xi} \  ilde{\Lambda_1} \end{array}$	1		
$ ilde{\Lambda_1}$	0.09		
$ ilde{\Lambda_2}$	0.04		
$ ilde{ heta}$	0.01		
$ ilde{\delta_1}$	0.01		
$ ilde{\delta_2}$	0.001		

persistence. The sufficiency of the condition  $\mathcal{R}_0 > 1$  follows from a uniform persistence result. Now, we demonstrate that the system (7)-(12) satisfies all the conditions, when  $\mathcal{R}_0 > 1$ , let  $X = \mathbf{R}^4$  and  $E = \Gamma$ . The maximal invariant set N on the boundary  $\partial \Omega$  is the singleton  $\{E^0\}$  and is isolated. Thus hypothesis (H) of holds for system (7)-(12). Therefore, in the setting of (7)-(12), the necessary and sufficient condition for uniform persistence is equivalent to  $E^0$  being unstable.

#### 4. NUMERICAL SIMULATION

In this section, with the help of ODE solver Matlab we perform the numerical simulation, to verify the analytical findings of previous sections. The parametric values used for numerical simulation of the model system for infectious disease are listed in Table 2 taking unit time in days.

now, we can suppose following cases:

- Case (a): When m=2.3 and  $\tilde{\beta}=1$ , then  $\mathcal{R}_0=0.909091<1$  and disease-free equilibrium is Globally Asymptotically Stable which is given in Figure 4, which is according to the results in ??.
- Case (b): When m=2.3 and  $\tilde{\beta}=8$ , then  $\mathcal{R}_0=3.63636>1$  and pandemic equilibrium is locally asymptotically stable which in shown in Figure 5, which is according to result in ??.

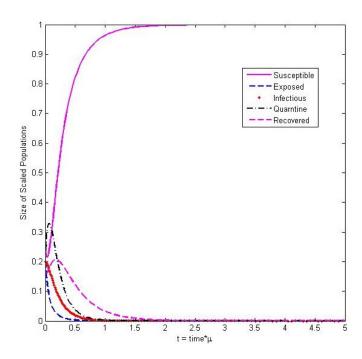


Figure 4: Scaled population in scaled-time's variation, taking m=2.3 and  $\tilde{\beta}=1$  with  $\mathcal{R}_0=0.4545<1.$ 

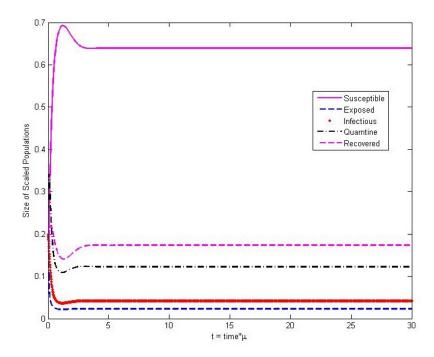


Figure 5: Scaled population in scaled-time's variation, taking m=2.3 and  $\tilde{\beta}=8$  with  $\mathcal{R}_0=3.6363>1.$ 

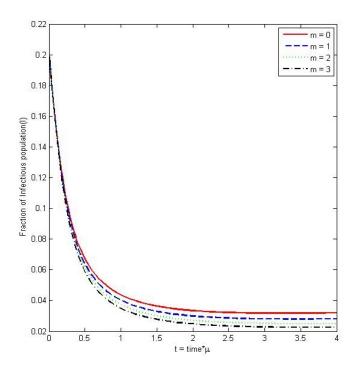


Figure 6: Effect of the m on I at  $\tilde{\beta}=1$  with  $\mathcal{R}_0=0.4545<1$ .

Table 3: The sensitivity indices,  $\Upsilon^{\mathcal{R}_C}_{y_j} = \frac{\partial \mathcal{R}_C}{\partial y_j} \times \frac{y_j}{\mathcal{R}_C}$ , of  $\mathcal{R}_C$  to the parameters,  $y_j$  for parameteric values given in Table 2

Parameter $(y_j)$	Sensitivity index of $\mathcal{R}_C$ w.r.t. $y_j$ ( $\Upsilon_{y_j}^{\mathcal{R}_C}$ )		
β	+1.000		
ξ	+0.5000		
$\delta_1$	-0.00909091		
$\Lambda_1$	-0.0818182		

The effect of m on the fraction of infectious and quarantine individuals (I) and (Q) is given in Figure 6 and Figure 7 taking same values of parameters as in cases (a) and (b), respectively with the different values of m.

## 5. SENSITIVITY ANALYSIS

In this part, we analyze the sensitivity analysis of effective reproduction number  $\mathcal{R}_C$  and endemic equilibrium taking parametric values given in Table 2. The normalized sensitive indices of  $\mathcal{R}_C$  the effective reproduction number with respect to all parameters are shown in 5

From the Table 3.3, we can observe that  $\beta$  and  $\xi$  have positive impact on  $\mathcal{R}_C$  and

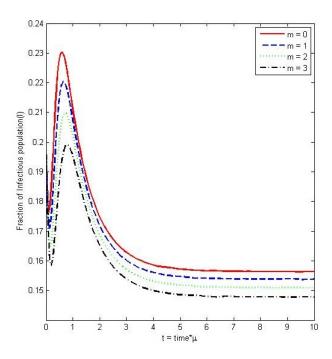


Figure 7: Effect of the m on I at  $\tilde{\beta} = 8$  with  $\mathcal{R}_0 = 3.6363 > 1$ .

remaining parameters have negative impact. Again, the parameters  $\beta$  and  $\delta$  are most sensitive to  $\mathcal{R}_C$ , hence we can see that significant change in  $\mathcal{R}_C$  by small changes in these parameters.

Now, we analyze a sensitivity analysis of state variables at endemic equilibrium on model system parameters. Sensitivity indices of state variables at endemic steady state are shown in Table 3.4 using parametric values shown in Table 2.

#### 6. RESULTS AND DISCUSSION

The media is broadly recognized as a critical device for affecting individuals practices towards the sickness to devise appropriate strategies for controlling the scourge. Awareness programs through media cause individuals to know about the sickness and play it safe to lessen their odds of being tainted. In this paper, a non-linear SEIQRS mathematical model has been proposed and broke down to contemplate the impacts of awareness programs driven by the media on the spread of irresistible sicknesses in a variable population. It has been viewed as that the development pace of aggregate thickness of awareness programs is relative to the number of infectives. It has been accepted further that awareness causes some susceptibles to confine themselves from infectives shaping a different subclass in the population. The model has shown that the disease-free equilibrium is steady until, the basic reproduction number, R<sub>i</sub>1. The

-0.35140

-0.35140

variable etc., $y_j$ , for parameter values shown in Table 2									
	$y_j$	$\Upsilon^{E^*}_{y_j}$	$\Upsilon^{I^*}_{y_j}$	$\Upsilon^{Q^*}_{y_j}$	$\Upsilon^{R^*}_{y_j}$	$\Upsilon^{N^*}_{y_j}$			
	β	23.0000	23.0000	23.0000	23.0000	-0.00355			
	ξ	11.1450	12.1450	11.1450	11.8719	0.85298			
	$\delta$	-0.28645	-0.28645	0.71354	-0.01335	0.28648			
	$\delta_1$	-0.20586	-0.20586	-0.20586	-0.20338	-0.00355			
	$\delta_2$	0.00027	0.00027	-0.00068	0.00001	0.01484			
	$\theta$	0.00039	0.00039	0.00039	-0.00950	0.00950			
	$\Lambda_1$	-1.88182	-1.88182	-1.88182	-1.13257	0.16393			
	$\Lambda_2$	0.00009	0.00009	-0.03833	0.26270	0.11518			

Table 4: The sensitivity indices,  $\Upsilon^{x_i}_{y_j} = \frac{\partial x_i}{\partial y_j} \times \frac{y_j}{x_i}$ , of state variables,  $x_i$ , at EE with respect to all the parameters,  $y_j$ , for parameter values shown in Table 2

DFE becomes temperamental for  $R_{i,1}$ , which prompts the presence of an endemic equilibrium. The investigation shows that an endemic equilibrium is locally just as non-linearly stable under specific conditions. The model examination further shows that awareness programs through the media battling are useful in diminishing the spread of irresistible sicknesses by confining a fraction of susceptibles from infectives.

-0.35140

-0.35140

+0.01520

#### REFERENCES

#### **REFERENCES**

- [1] Collinson S.,Khan K., and Heffernan J. M., (2015), "The Effects of Media reports on Disease Spread and Important Public Health Measurements", PLoS ONE, 10(11).
- [2] Roth D. Z., and Henry B., (2011), "Social Distancing as a Pandemic Influenza Prevention Measure", National Collaborating Centre for Infectious Diseases.
- [3] Breban R., (2011), "Health newscasts for increasing influenza vaccination coverage: An inductive reasoning game approach", PLoS ONE.
- [4] Jung M., Lin L., and Viswanath K., (2013), "Associations between health communication behaviours, neighbourhood social capital, vaccine knowledge, and parents' H1N1 vaccination of their children", Vaccine, 31(42), pp. 4860-4866.
- [5] Lau J.T.F., Yang X., Tsui H., and Kim J.H., (2003), "Monitoring community responses to the SARS epidemic in Hong Kong: from day 10 to day 62", J Epidemiol Community Health, 57, pp. 864-870.

- [6] Majumder M., Kluberg S., Santillana M., Mekaru S., and Brownstein J.S., (2015), "Ebola Outbreak: Media eventstrack changes in observed reproductive number", PLoS Curr., 7.
- [7] Hornik R., (2002), "Public Health communication: Evidence for behavior change Routledge".
- [8] Catalan-Matamoros D., (2011), "The role of mass media communication in public health. Health Management Different Approaches and Solutions", INTECH Open Access Publisher, Dr. Smigorski (Ed.) pp. 296-298.
- [9] Wakefield M.A., Loken B., and Hornik R.C., (2010) "Use of mass media campaigns to change health behaviour", The Lancet, 376 (9748) pp. 1261-1271.
- [10] Funk S., Knight G.M., and Jansen V.A.A., (2014), "Ebola: the power of behaviour change", Nature, 515(7528), pp. 492.
- [11] Wakefield M.A., Spittal M.J., Yong H.H., Durkin S.J., and Borland R., (2011), "Effects of mass media campaign exposure intensity and durability on quit attempts in a population-based cohort study", Health Education Research, 26(6), pp. 988-997.
- [12] Cui J., Sun Y., and Zhu H., (2007), "The impact of media on the control of infectious diseases", Journal of Dynamics and Differential Equations, 20(1), pp. 31-53.
- [13] Glasser J.W., Hupert N., McCauley M., and Hatchett R., (2011), "Modeling and public health emergency responses: lessons from SARS", Epidemics, 3(1), pp. 32-37.
- [14] Pang J., and Cui I.A., (2009), "An SIRS epidemiological model with nonlinear incidence rate incorporating media coverage", Second International Conference on Information and Computing Science, IEEE, pp. 116-119.
- [15] Lipsitch M., Cohen T., Cooper B., Robins J.M., Ma S., James L., Gopalakrishna G., Chew S.K., Tan C.C., Samore M.H., Fisman D., and Murray M., (2003), "Transmission dynamics and control of severe acute respiratory syndrome", Science, 300(5627), pp. 1966-1970.
- [16] Sun C., Yang W., Arino J., and Khan K., (2011), "Effect of media-induced social distancing on disease transmission in a two patch setting", Mathematical Biosciences, 230(2), pp. 87-95.

- [17] Bell D., (2006), "Non-pharmaceutical interventions for pandemic influenza, international measures", Emerging Infectious Diseases, 12(1), pp. 81-87.
- [18] Suess T., Remschmidt C., Schink S.B., Schweiger B., Nitsche A., Schroeder K., Doellinger J., Milde J., Haas W., Koehler I., and Krause G., (2012), "The role of facemasks and hand hygiene in the prevention of influenza transmission in households: results from a cluster randomised", BMC Infect Diseases, 12(1), pp. 26.
- [19] Donnelly C.A., Finelli L., Cauchemez S., Olsen S.J., Doshi S., Jackson M.L., Kennedy E.D., Kamimoto L., Marchbanks T.L., Morgan O.W., Patel M., Swerdlow D.L., and Ferguson N.M., (2011), "Serial intervals and the temporal distribution of secondary infections within households of 2009 pandemic influenza A (H1N1): implications for influenza control recommendations", Clinical Infectious Diseases, 52(1), pp. 123-130
- [20] Funk S., Gilad E., Watkins C., and Jansen V.A.A., (2009), "The spread of awareness and its impact on epidemic outbreaks", Proceedings of the National Academy of Sciences of the United States of America, 106(16), pp. 6872-6877.
- [21] Liu R., Wu J., and Zhu H., (2007), "Media/ psychological impact on multiple outbreaks of emerging infectious diseases", Computational and Mathematical Methods in Medicine, 8(3), pp. 153-164.
- [22] Cui J.A., Tao X., and Zhu H., (2008), "An SIS infection model incorporating media coverage", Rocky Mountain Journal of Mathematics, 38(5), pp. 1323-1334.
- [23] Tchuenche J.M., Dube N., Bhunu C.P., Smith R.J., and Bauch C.T., (2011), "The impact of media coverage on the transmission dynamics of human influenza", BMC Public health, 11, pp. 5.
- [24] Blendon R.J., Benson J.M., DesRoches C.M., Raleigh E., and Taylor-Clark K., (2004), "The public's response to severe acute respiratory syndrome in Toronto and the United States", Clinical Infectious Diseases, 38(7), pp. 925-931.
- [25] Liu Y., and Cui J.A., (2008), "The impact of media coverage on the dynamics of infectious diseases", International Journal of Biomathematics, 1(1), pp. 65-74.
- [26] Mushayabasa S., Bhunu C.P., and Smith R.J., (2012), "Assessing the impact of educational campaigns on controlling HCV among women in prison settings", Communications in Nonlinear Science and Numerical Simulation, 17(4), pp. 1714-1724.

- [27] Kiss I.Z., Cassell J., Recker M., and Simon P.L., (2010), "The impact of information transmission on epidemic outbreaks", Mathematical Biosciences, 225(1), pp. 1-10.
- [28] Valle S.D., Hethcote H., Hyman J.M., and Castillo-Chavez C., (2005), "Effects of behavioral changes in a smallpox attack model", Mathematical Biosciences, 195(2), pp. 228-251.
- [29] Tracht S.M., Valle S.Y.D., and Hyman J.M., (2010), "Mathematical modeling of the effectiveness of facemasks in reducing the spread of novel influenza A (H1N1)", PLoS ONE, 5(2).
- [30] Sahu G.P., and Dhar J., (2015), "Dynamics of an SEQIHRS epidemic model with media coverage, quarantine and isolation in a community with pre-existing immunity", Journal of Mathematical Analysis and Applications, 421, pp. 1651-1672.