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Chaos, Solitons and Fractals 138 (2020) 109953

Contents lists available at ScienceDirect

Chaos, Solitons and Fractals

Nonlinear Science, and Nonequilibrium and Complex Phenomena

journal homepage: www.elsevier.com/locate/chaos

A nonlinear epidemiological model considering asymptotic and quarantine classes for SARS CoV-2 virus

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ARTICLE INFO

Article history:
Received 11 May 2020
Revised 23 May 2020
Accepted 29 May 2020
Available online 4 June 2020

2010 MSC:
92-02
92B99

Keywords:
Reproduction number
Lyapunov's function
Prophylaxis

ABSTRACT

In this article, we develop a mathematical model considering susceptible, exposed, infected, asymptotic, quarantine/isolation and recovered classes as in case of COVID-19 disease. The facility of quarantine/isolation have been provided to both exposed and infected classes. Asymptotic individuals either recovered without undergo treatment or moved to infected class after some duration. We have formulated the reproduction number for the proposed model. Elasticity and sensitivity analysis indicates that model is more sensitive towards the transmission rate from exposed to infected classes rather than transmission rate from susceptible to exposed class. Analysis of global stability for the proposed model is studied through Lyapunov's function.

1. Introduction

In December 2019, first case of a new virus named severe acute respiratory syndrome novel coronavirus 2 (SARS CoV-2) detected in Wuhan, a city of China. The infection of virus prompted a continuous flare-up and an exceptional global health emergency. The number of contaminated individuals is increasing globally and it is likely to be underestimated the actual data of infection worldwide. The infections show 5% – 80% likely to asymptotic makes hard to detect and combat the infections. The leaders of different countries are making strategies to prevent the infections like social distancing, lockdown and isolation from world. Such measures cost economically too. Virus acting as double-edged sword as it is threatening human life and also making worse the remaining people of the world.

The virus identified as zoonotic, similar to SARS and MERS. The different studies [1] show the basic reproduction number for the disease ranges from 1.4 to 6.49 with a mean of 3.28, a median of 2.79 and interquartile range of 1.16. However, Zhang and their collaborators [2] have estimated maximum likelihood value of basic reproduction number: 2.28. Another early dynamics of daily reproduction number estimated as 2.35 which is reduced upto 1.05 after implementing lockdown [3]. The effect of virus is much severe in United States with 1,292,879 infected on 08 May, 2020 [4]. Another study [5] concluded that fatality was highest in persons aged 85 years, ranging from 10% to 27%, followed by 3% to 11% among persons aged 65–84 years in United States from February 21-March 16, 2020. In the case of Wuhan city in China, incubation period for the novel coronavirus estimated as 6.4 days and epidemic doubling time 6.4 days [6]. The reproduction number has estimated to 2.56 with 95% confidence interval when unreported cases for the virus has taken into account [7]. In the context of India, Mandal et al. [8] and Mishra et al. [9] have found basic reproduction number being 1.5 under the situation in March 2020 by considering SEIR model. They have also suggested control measures in order to prevent from the disease.

A key feature of the disease is significant proportion of asymptotic individuals. A specific study in Japan [10] shows 41.6% of individuals are asymptotic. Such high degree of hidden infections isolates this virus among others. A basic model in epidemiology, SIR model, can be used for estimation of total size of infected population. However, these models are not so much accurate to predict the prevalence for COVID-19 disease.

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https://doi.org/10.1016/j.chaos.2020.109953
0960-0779/

Deterministic compartmental models often used to design the infectious disease in epidemiology. Wei et al. [11] designed model for vector borne disease which has direct method of transmission in addition to vector mediated infection. Martcheva and Horst [12] presented a model for a disease with an advancing and a silent exposed class and variable susceptibility to super-infection. Similarly, Kribs et al. [13] designed a model related to acute and chronic infective disease. They have discussed the effect of vaccination upon the spread of non-fatal disease. Nuno et al. [14] modelled two influenza strains under different degree of obstructions. They built up cross resistance and host seclusion lead to intermittent pandemic outbreak in the multi-strain framework. In particular, a mathematical modelling has analysed in Ndaïrou et al. [15] using a compartmental model taking hospitalised and asymptomatic cases as extra compartments for COVID-19 disease. In [16], authors used a new spatiotemporal approach (SBDiEM) to explain the phenomenon of the SARS CoV-2 virus. In an advanced stage, Khan and Atangana [17] used fractional integral approach [18] to describe the epidemiology of COVID-19 disease and estimated $R_0 = 2.4829$. Martcheva and Carlos [19] studied Hepatitis C with a chronic infectious stage and variable population size.

Mathematical modelling is one of the significant tools to provide valuable insight into the epidemic problems and various other real world problems, for instant see recent work [20–25]. This work presents a deterministic compartmental model to explain SARS CoV-2 virus in some extent. We incorporate asymptotic infection and quarantine/isolation into SEIR model.

2. Proposed model

In the proposed model, S (susceptible) is a healthy population which undergo contagion and move to E class (exposed). After infection from virus, some of them are asymptotic and are categorised into A group (asymptotic). Others who shows symptoms are moved to I group (infected). Asymptotic are not showing any visible symptom of the disease and it is much difficult to identify them. As per study available, SARS CoV-2 virus has a key feature of asymptotic individuals amounts to 5–80% of the exposed people [10]. We denote this probability to p. Asymptotic people are assumed to be infectious with reduced (or enhanced) transmission rate $q\beta_0$. An individual from asymptotic group either shows symptom after some duration and moves to infected group or remain asymptotic and recovers from disease. Let γ denote the recovery rate of asymptotic individuals. The infected may go under treatment with isolation. In this case, they move to recovered group through Q-group (quarantine/isolation). But some of individuals may not go to isolation and directly move to recovered group. Let ν denote the recovery rate from infected group and δ denote recovery rate from quarantine/isolation. The quarantine rate from infected to isolation group is denoted by α. We are assuming that dead are not infectious. Hence the recovered group contains both dead and recovered people and separated from susceptible after the process. We also assume that once an individual is recovered from disease, he develops the immunity from the virus and will not undergo the cycle.

Mathematically the model is expressed as the following autonomous system:

$$\begin{aligned}
\frac{dS}{dt} &= \Lambda - \beta_0 \frac{S(I+qA)}{N-Q} - \mu S \\
\frac{dE}{dt} &= \beta_0 \frac{S(I+qA)}{N-Q} - (\eta+\theta+\mu)E \\
\frac{dI}{dt} &= p\eta E - (\alpha+\nu+\mu)I + \rho A \\
\frac{dQ}{dt} &= \alpha I + \theta E - (\delta+\mu)Q \\
\frac{dA}{dt} &= (1-p)\eta E - (\rho+\gamma+\mu)A \\
\frac{dR}{dt} &= \gamma A + \delta Q + \nu I - \mu R
\end{aligned} \tag{1}$$

The total size of population is assumed to be N which are logistically increasing at a rate of Λ and decreasing by natural mortality rate μ. β_0 denotes transmission rate from susceptible to exposed compartment. η is infection rate for the model and θ is isolation rate of individual. Force of infection is related to prevalence $(I+qA)$ with a linear relation as

$$\lambda = \frac{\beta_0(I+qA)}{N-Q}$$

In order to make relation simple we substitute $\beta = \beta_0/(N-Q)$. β is per capita transmission rate.

2.1. Initial conditions

The population is disease free until $I(0)$ number of infected enter into the population. At the time $t = 0$, $R(0) = 0, S(0) = N, Q(0) = 0, A(0) = 0$ and $E(0) = 0$. The feasible region of the system will be

$$\{(S, E, I, Q, A, R) : S > 0, E > 0, I > 0, Q \geq 0, A \geq 0, R \geq 0\}$$

2.2. Incubation period

From the system (1), $S'(0) < 0$ for all $t > 0$ implies that $S(t)$ is positive, monotone and bounded by N. Hence, the final size of epidemic is

$$\lim_{t\to\infty} S(t) = S_\infty.$$

In the same way, R' is positive, bounded and monotonic which implies that

$$\lim_{t\to\infty} R(t) = R_\infty.$$

The trivial equilibrium point for the model is $(\Lambda/\mu, 0, 0, 0, 0, 0)$. For sufficiently large time, number of infected exponentially decay

$$\frac{dI}{dt} = -(\alpha+\nu+\mu)I \tag{2}$$

for $t \to \infty$. This implies that

$$I(t) = I_0 e^{-(\alpha+\nu+\mu)t} \tag{3}$$

Probability of individuals who recovered from the disease, indicate the cumulative distribution function $F(t)$:

$$F(t) = 1 - e^{-(\alpha+\nu+\mu)t}. \tag{4}$$

To find the probability density function $P(t)$ from cumulative distribution, we must use fundamental theorem of calculus:

$$P(t) = \begin{cases} (\alpha+\nu+\mu)e^{-(\alpha+\nu+\mu)t}; & t \geqslant 0 \\ 0; & t < 0 \end{cases}. \tag{5}$$

First moment about the origin gives the expectancy of time spend in infectious class:

$$E[t] = \int_{-\infty}^{\infty} tP(t)dt = \frac{1}{(\alpha+\nu+\mu)} \tag{6}$$

The incubation period is thus, the inverse of sum of transmission rates α, ν, μ.

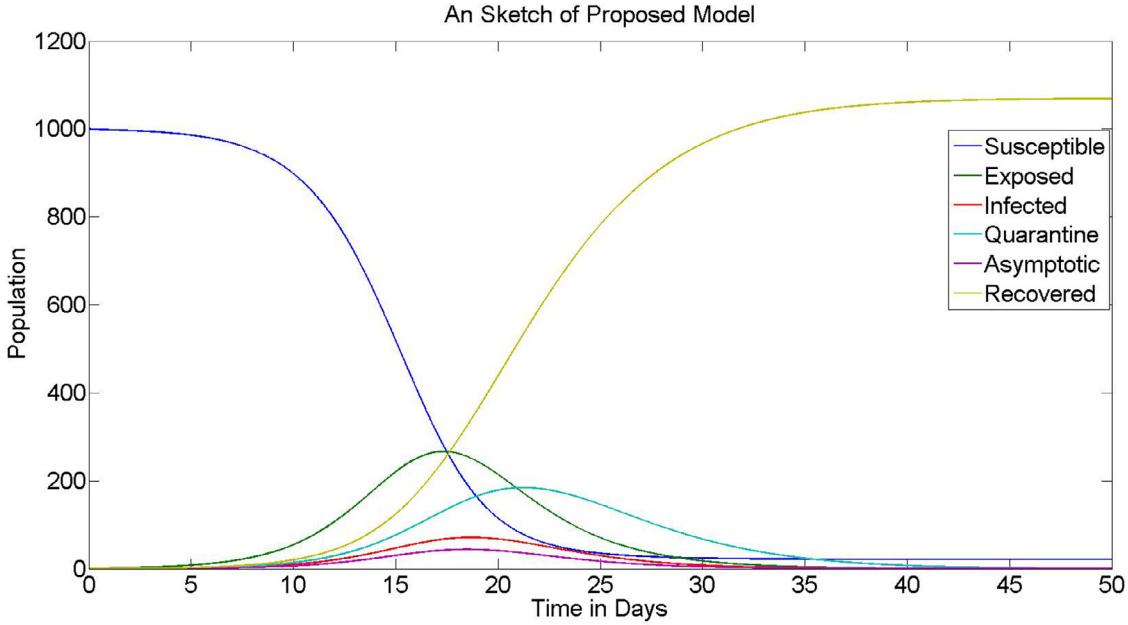

Fig. 1. Sketch of population with time.

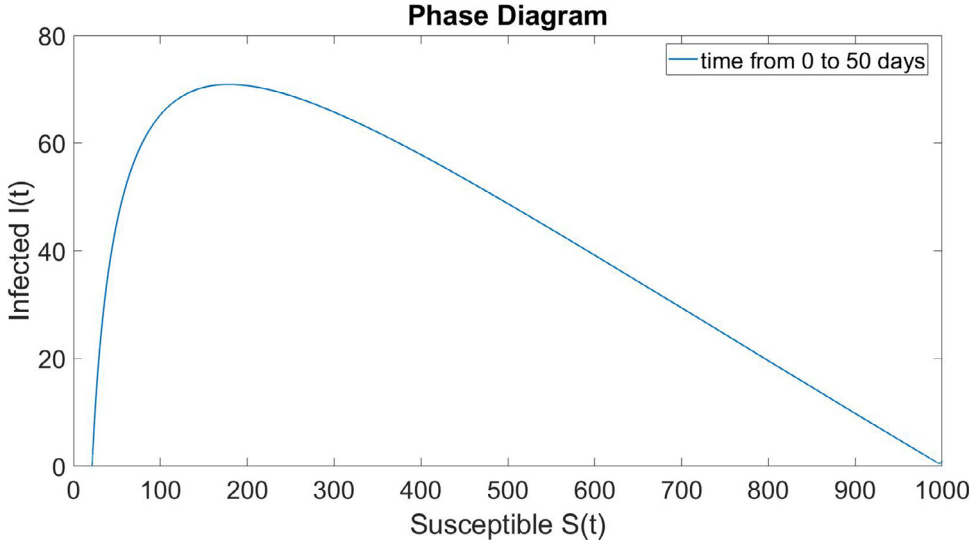

Fig. 2. Phase plane.

2.3. Graph of system

In this subsection, we sketch a graph of the model. Let us take total population be $N = 1000$. The birth rate of the population is assumed to $\Lambda = -0.2\%$ per year and mortality rate $\mu = 0.87\%$ per year. $\beta = 2.5/(N - Q)$ where Q is number of quarantine/isolation people. Other parameter are assumed as $p = 0.56, q = 1, \nu = 10/228, \eta = 0.32, \theta = 0.01, \rho = 0.5, \alpha = 0.9, \delta = 0.03,$ and $\gamma = 0.5$. In this scenario, the graph of system can be sketched in Fig. 1.

2.4. Equilibrium points

In Epidemiology, we often interested to know the long term behaviour of system. We assume that population is not open thus the quantity we deal in modelling are finite one. A graph between S and I with time as parameter is call orbits or trajectories and graph is often called Phase plane (Fig 2). If we look the system in long term, the system gets steady state equilibrium and at this point

$$dS/dt = dI/dt = dE/dt = dA/dt = dQ/dt = dR/dt = 0.$$

The system possesses two singular points (equilibria points). One, at disease free equilibrium, when $I = 0$ and then, the point is $(\Lambda/\mu, 0, 0, 0, 0, 0)$. Disease free equilibrium is used as boundary condition for the system so named it boundary equilibrium. Other point at equilibrium is endemic equilibrium and represented by $\mathcal{E}_0 = (S^*, E^*, I^*, A^*, Q^*, R^*)$. After the infection in the population, we show interest in endemic equilibrium. $\mathcal{E}_0$ exists when the basic reproduction number is greater than one.

$$S^* = \frac{\Lambda}{\mu} - \frac{(\eta + \theta + \mu)}{\mu} E* \tag{7}$$

$$I^* = \frac{1}{\alpha + \nu + \mu}\left[p\eta + \frac{(1-p)\eta\rho}{\gamma + \rho + \mu}\right] E* \tag{8}$$

$$Q^* = \frac{1}{\delta + \mu}\left[\theta + \frac{\alpha}{\alpha + \nu + \mu}\left(p\eta + \frac{(1-p)\eta\rho}{\gamma + \rho + \mu}\right)\right] E^* \tag{9}$$

$$A^* = \frac{(1-p)\eta}{\gamma+\rho+\mu}E^* \tag{10}$$

$$R^* = \left[\frac{(1-p)\eta}{\gamma+\rho+\mu}\left(\gamma + \frac{\rho\nu}{\alpha+\nu+\mu} + \frac{\alpha\delta\rho}{(\delta+\mu)(\alpha+\nu+\mu)}\right) + \frac{\delta\theta}{\delta+\mu} + \frac{\gamma p\eta}{\alpha+\nu+\mu} + \frac{\alpha\delta p\eta}{(\delta+\mu)(\alpha+\nu+\mu)}\right]\frac{E*}{\mu} \tag{11}$$

where E^* is

$$E^* = \frac{\Lambda}{\mu(\eta+\theta+\mu)} - \left(\frac{p}{\alpha+\nu} + \frac{(1-p)q}{\gamma}\right)^{-1} \tag{12}$$

2.5. Reproduction number

The reproduction number generally defined as number of secondary infections appear from one infected individual. It provides a threshold condition for the stability of the system. Finding reproduction number through Jacobian approach using linearization of the system often does not work for complex system. We use next generation approach, known as Van den Driessche and Watmough approach [26], here.

In this approach, we first decompose system (1) into infected compartment:

$$\begin{aligned}
\frac{dE}{dt} &= \beta S(I+qA) - (\eta+\theta+\mu)E\\
\frac{dI}{dt} &= p\eta E - (\alpha+\nu+\mu)I + \rho A\\
\frac{dQ}{dt} &= \alpha I + \theta E - (\delta+\mu)Q\\
\frac{dA}{dt} &= (1-p)\eta E - (\rho+\gamma+\mu)A
\end{aligned} \tag{13}$$

and non-infected compartment:

$$\begin{aligned}
\frac{dS}{dt} &= \Lambda - \beta S(I+qA) - \mu S\\
\frac{dR}{dt} &= \gamma A + \delta Q + \nu I - \mu R.
\end{aligned} \tag{14}$$

We arrange infected compartment such that

$$\frac{d}{dt}\begin{bmatrix} E\\ I\\ Q\\ A\end{bmatrix} = \mathcal{F} - \mathcal{V} \tag{15}$$

The matrices $\mathcal{F}$ and $\mathcal{V}$ chosen such that

$$\mathcal{F} = \begin{bmatrix}\beta S(I+qA)\\ 0\\ 0\\ 0\end{bmatrix} \tag{16}$$

and

$$\mathcal{V} = \begin{bmatrix}(\eta+\theta+\mu)E\\ p\eta E - \rho A + (\alpha+\nu+\mu)I\\ -\theta E - \alpha I + (\delta+\mu)Q\\ -(1-p)\eta E + (\gamma+\rho+\mu)A\end{bmatrix}. \tag{17}$$

Note that various combinations of $\mathcal{F}$ and $\mathcal{V}$ are possible, however, the functions defined in (16) and (17) satisfy the following conditions:

- New infections in the populations are secondary infections, that is, $\mathcal{F} = 0$ whenever $E = I = A = Q = 0$.
- There is no immigration from susceptible population, that is, $\mathcal{V} = 0$, whenever $E = I = A = Q = 0$.
- Total output from infected compartments is positive.

Moreover, feasibility condition on $\mathcal{F}$ allows only non-negative output and feasibility condition on $\mathcal{V}$ allows only non-positive output. Non-infected compartment provides a unique disease free equilibrium at $\mathcal{E}_0 = (\Lambda/\mu, 0, 0, 0, 0, 0)$ when $S = R = 0$.

Matrices of partial derivatives of $\mathcal{F}$ and $\mathcal{V}$ are respectively,

$$F = \begin{bmatrix}0 & \beta S^* & 0 & \beta q S^*\\ 0 & 0 & 0 & 0\\ 0 & 0 & 0 & 0\\ 0 & 0 & 0 & 0\end{bmatrix} \tag{18}$$

and

$$V = \begin{bmatrix}\eta+\theta+\mu & 0 & 0 & 0\\ -p\eta & \alpha+\nu+\mu & 0 & -\rho\\ -\theta & -\alpha & -\delta+\mu & 0\\ -(1-p)\eta & 0 & 0 & \gamma+\rho+\mu\end{bmatrix}. \tag{19}$$

Next generation matrix is defined as $K = FV^{-1}$. Van den Driessche and Watmough approach suggests the reproduction number $\mathcal{R}_0$ = spectral radius of K.

$$K = FV^{-1} = \begin{bmatrix}a_1 & a_2 & 0 & a_3\\ 0 & 0 & 0 & 0\\ 0 & 0 & 0 & 0\\ 0 & 0 & 0 & 0\end{bmatrix} \tag{20}$$

with

$$a_1 = \beta S^*\left[\frac{p\eta}{(\eta+\theta+\mu)(\alpha+\nu+\mu)} + \frac{(1-p)\eta[\rho+q(\alpha+\nu+\mu)]}{(\eta+\theta+\mu)(\alpha+\nu+\mu)(\gamma+\rho+\mu)}\right] \tag{21}$$

$$a_2 = \frac{\beta S^*}{(\alpha+\nu+\mu)} \tag{22}$$

$$a_3 = \beta S^*\left[\frac{\rho}{(\alpha+\nu+\mu)(\gamma+\rho+\mu)} + \frac{q}{(\gamma+\rho+\mu)}\right] \tag{23}$$

Reproduction number can be defined as

$$\mathcal{R}_0 = \frac{(1-p)\eta\beta S^*\left[\rho+q(\alpha+\nu+\mu)\right]}{(\gamma+\rho+\mu)(\eta+\theta+\mu)(\alpha+\nu+\mu)} + \frac{p\eta\beta S^*}{(\alpha+\nu+\mu)(\eta+\theta+\mu)}. \tag{24}$$

The reproduction number $\mathcal{R}_0$ can be visualised as sum of two reproduction numbers. The term

$$\mathcal{R}_0^a = \frac{(1-p)\eta\beta S^*\left[\rho+q(\alpha+\nu+\mu)\right]}{(\gamma+\rho+\mu)(\eta+\theta+\mu)(\alpha+\nu+\mu)} \tag{25}$$

related to asymptotic class and represents number of secondary infection because of asymptotic individuals during its life span in the susceptible population. This reproduction number $\mathcal{R}_0^a$ is a sum of two terms. The term $(1-p)\eta\beta S^*\rho/[(\gamma+\rho+\mu)(\eta+\theta+\mu)(\alpha+\nu+\mu)]$ represents the number of secondary infected people due to one asymptotic individual. The time unit an asymptotic individual remains in Asymptotic group is $1/(\gamma+\rho+\mu)$. The term $(1-p)\eta q\beta S^* q(\alpha+\nu+\mu)/[(\gamma+\rho+\mu)(\eta+\theta+\mu)]$ indicates the number of secondary infections who get recovered without any treatment because of an asymptotic individual.

Second right side expression of Eq. (24)

$$\mathcal{R}_0^i = \frac{p\eta\beta S^*}{(\alpha+\nu+\mu)(\eta+\theta+\mu)} \tag{26}$$

represents number of secondary infections caused by an infected individual during his life span. The time units spend an individual in infected compartment is $1/(\alpha+\nu+\mu)$. The term βSI in first and second equation of the system (1) is the incidence of infected. The number of secondary infections that will be produced in the

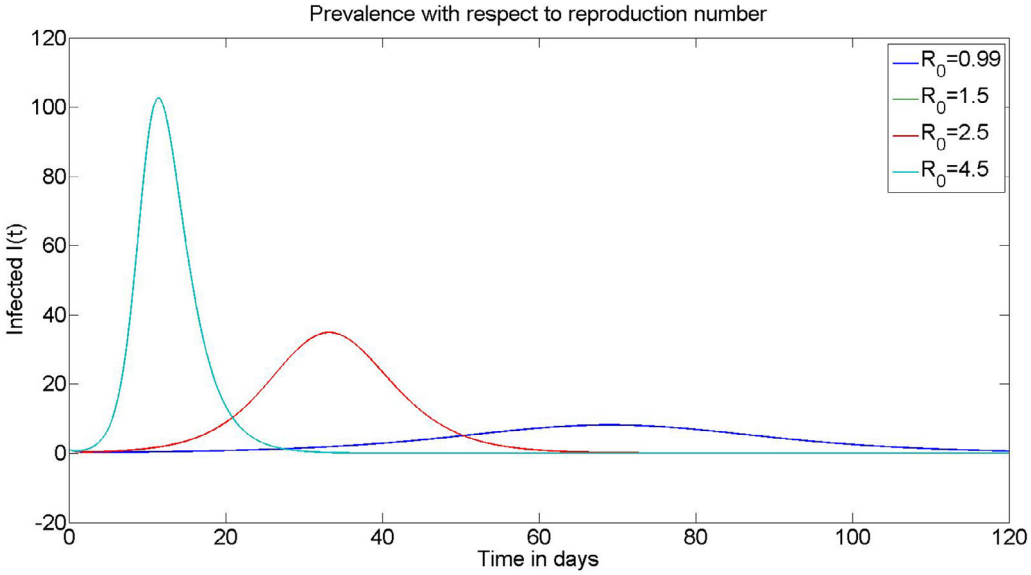

Fig. 3. A graph showing the infection for different reproduction number.

susceptible population per unit of time is βS^*. However, the fraction of the newly infected population produced by an individual is $p\eta/(\eta+\theta+\mu)$.

In Fig. 3, infected population has been plotted for different values of reproduction number. Fig. 3 shows higher the value of reproduction number (from unity), the greater peak of infected population.

2.6. Sensitivity analysis

The proposed model has five transmission rates, namely, β, η, θ, ρ, α and three recovery rates δ, ν, γ apart from Λ, μ and two probabilities p, q. One of the primary mandate of the epidemiological model is to provide an effective tool to control the transmission. Sensitivity analysis suggests that which transmission rate we should control sensitively in order to prevent the transmission. To perform global sensitivity analysis we need to grow the framework to incorporate all parameter involved in the system. This is a computationally hard issue, especially for a multi-dimensional model [27]. We focus here on local sensitivity analysis first.

Suppose we need a local sensitivity analysis of infected with respect to the transmission rate β. We introduce a new variable

$$Z_S = \frac{\partial(dS/dt)}{\partial\beta}.$$

Other variables Z_E, Z_I, Z_Q, Z_A, Z_R are defined in the same fashion by replacing S to respectively E, I, Q, A, R. Our interest is to simulate Z_I and for this, we need to solve the following system of differential equations

$$Z_S' = -(1+\beta Z_S)(I+qA) - \beta S(Z_I + qZ_A) - \mu Z_S \tag{27}$$

$$Z_E' = S(I+qA) + \beta Z_S(I+qA) + \beta S(Z_I+qZ_A) - (\eta+\theta+\mu)Z_Q \tag{28}$$

$$Z_I' = p\eta Z_E + \rho Z_A - (\alpha+\nu+\mu)Z_I \tag{29}$$

$$Z_Q' = \theta Z_E + \alpha Z_I - (\delta+\mu)Z_Q \tag{30}$$

$$Z_A' = (1-p)\eta Z_E - (\gamma+\rho+\mu)Z_A \tag{31}$$

$$Z_R' = \gamma Z_A + \delta Z_Q + \gamma Z_I - \mu Z_R \tag{32}$$

with initial conditions $Z_S(0) = Z_E(0) = Z_Q(0) = Z_A(0) = Z_R(0) = 0$ and $Z_I(0) = 1$. The solution of the system (27)–(32) provides local sensitivity with respect to β. Same can be performed to find local sensitivity with respect to other transmission rates.

Finding the solution of the system (27)–(32) is rather a complicated approach, we can observe the local sensitivity of a parameter by plotting the infected for different values of the parameter.

In Fig. 4, the number of infected have been plotted for different values of $\beta = \beta_0/(N-Q)$. Graph indicates that decreasing the value of β, the number of infected decreases and the curves are also shifting towards right. This implies that lowering the value of β not only reduces the infection but also delays to attend the peak of curve. The sensitivity of η can be viewed in Fig. 5. Graph Fig. 5 shows a decrease of 0.1 value in η affects the highest number of infected with an effective manner. This implies that η is a better tool to control the transmission. However, a decrease in ρ does not contribute in the infection as compare to η (Fig. 6). Quarantine rate θ is showing opposite behaviour (Fig. 7). We observe that the increase upto the value 0.01 in θ lowers the peak of infection leaving the position of peak unaltered.

2.7. Elasticity of reproduction number

Elasticity of a static quantity with respect to a parameter measures the percentage change in the quantity with respect to percentage change in parameter. Positive elasticity represents positive correlation in the quantity and parameter and negative elasticity shows negative correlation. It is a dimension less quantity.

From Eq. (24), elasticity of reproduction number with respect to β is

$$\mathfrak{E}^{\beta}_{\mathcal{R}_0} = 1. \tag{33}$$

Furthermore, elasticity of reproduction number with respect to η

$$\mathfrak{E}^{\gamma}_{\mathcal{R}_0} = \frac{\theta+\mu}{\eta(\eta+\theta+\mu)}. \tag{34}$$

Expression (34) shows reciprocal relation of η, θ and μ. The elasticity of reproduction number with respect to ρ is the following:

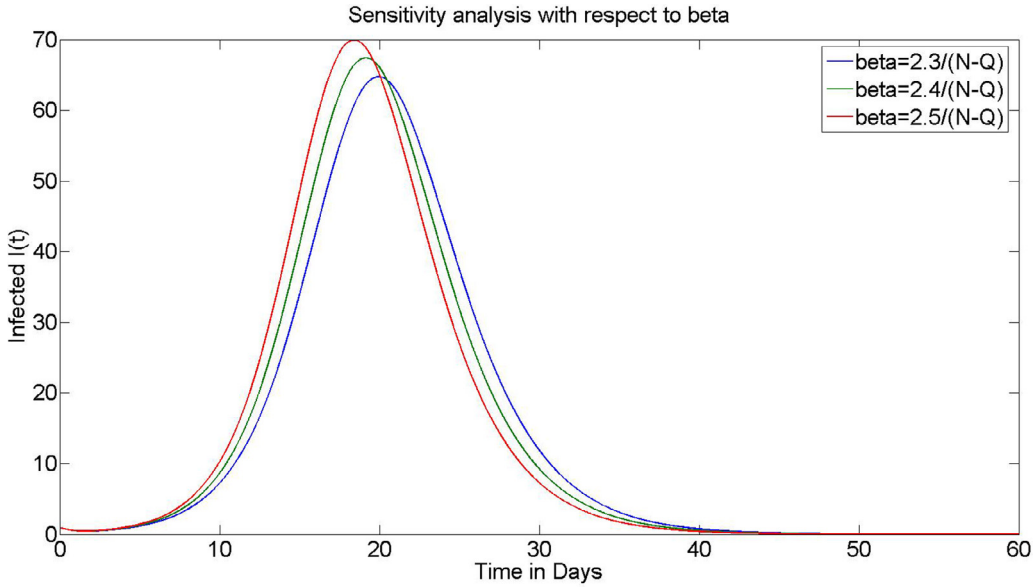

Fig. 4. Sensitivity analysis with respect to β.

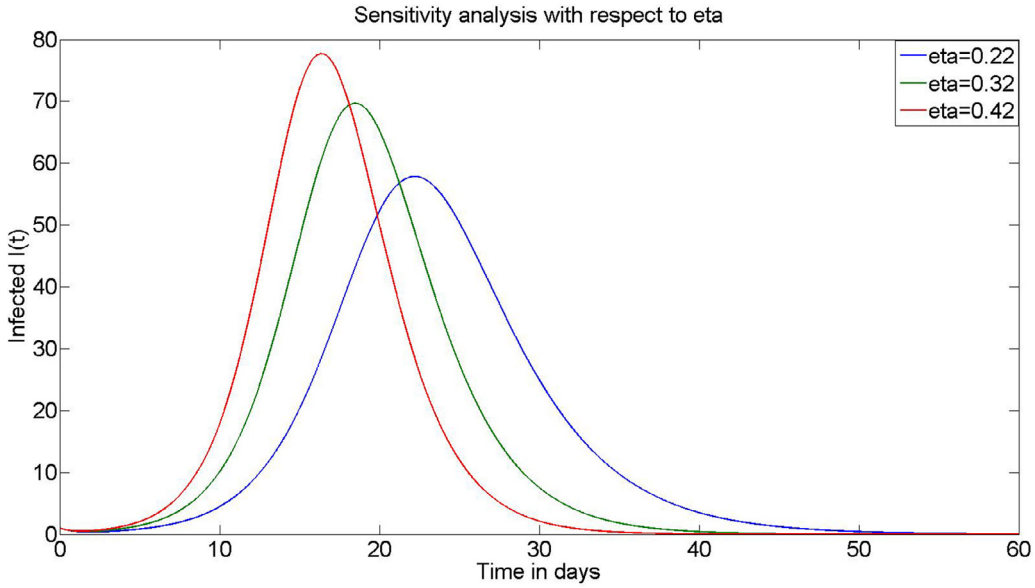

Fig. 5. Sensitivity analysis with respect to η.

$$\mathfrak{E}^{\rho}_{\mathcal{R}_0} = \frac{\gamma+\mu-q(\alpha+\nu+\mu)}{\big(\rho+q(\alpha+\nu+\mu)\big)(\gamma+\rho+\mu)}. \tag{35}$$

Note that Eq. (26) is independent from ρ. This means $\mathcal{R}_0$ and $\mathcal{R}_0^a$ have same elasticity. Elasticity of reproduction number with respect to θ and α are respectively

$$\mathfrak{E}^{\theta}_{\mathcal{R}_0} = -\frac{1}{(\eta+\theta+\mu)}. \tag{36}$$

and

$$\mathfrak{E}^{\alpha}_{\mathcal{R}_0} = -\frac{1+\rho}{(\alpha+\nu+\mu)\big(1+\rho+q(\alpha+\nu+\mu)\big)}. \tag{37}$$

This investigation reveals that increase in ρ, that is, identification of more infected from asymptotic class helps to reduce the reproduction number. The same is with η and θ.

2.8. Global stability using Lyapunov's function

Global stability of the system can be determined using Lyapunov's function. Lyapunov's stability theorem states that a globally positive definite and radially unbounded Lyapunov's function whose derivative is negative on entire feasible region except a point x^*, possesses the globally stable equilibrium at x^*. Construction of suitable Lyapunov's function is tricky. Krasovkii-LaSalle theorem helps to establish such function for an autonomous system.

Suppose (S^*, E^*, I^*, Q^*, A^*, R^*) is an endemic equilibrium point stated in Eq. (7)–(12). We define

$$L(t) = k_1\left(S - S^* - S^* ln\left(\frac{S}{S^*}\right)\right) + k_2E + k_3I + k_4Q + k_5A \tag{38}$$

It is clear from the definition that function defined in Eq. (38) are positive definite and radially unbounded. The values of k_2, k_4 and

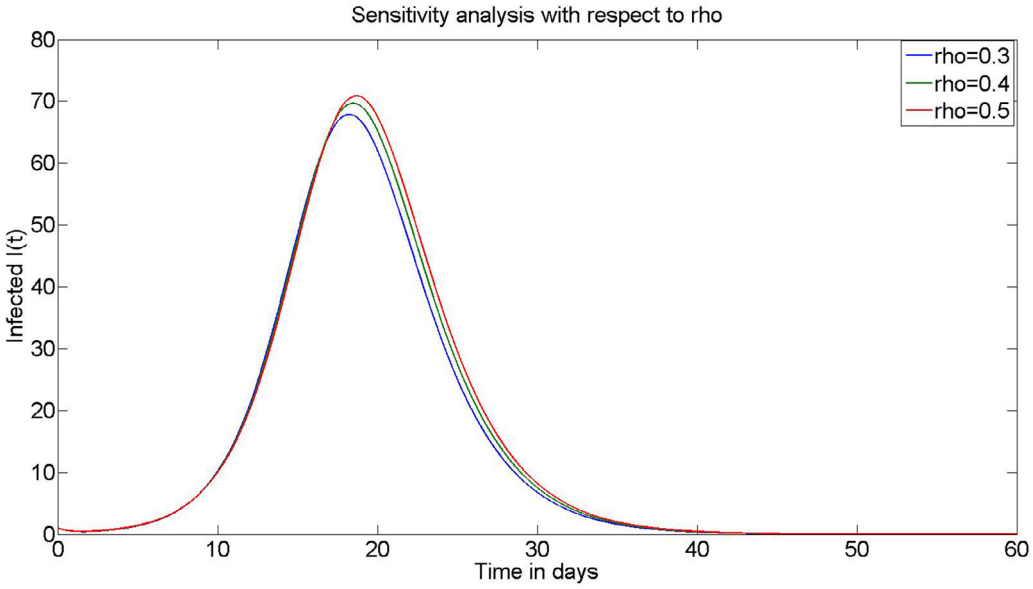

Fig. 6. Sensitivity analysis with respect to ρ.

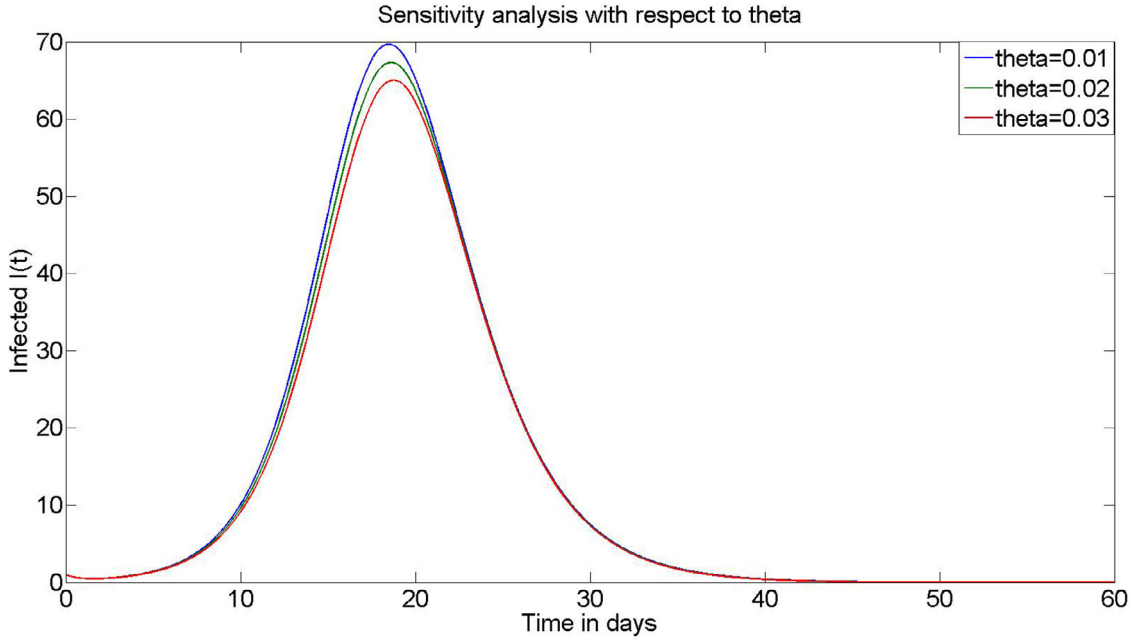

Fig. 7. Sensitivity analysis with respect to θ.

k_5 are assumed to be

$$k_2 = \frac{k_3}{\eta+\theta+\mu}\left(p\eta + \frac{\theta\alpha}{\alpha+\nu+\mu} + \frac{(1-p)\eta\rho}{\rho+\gamma+\mu}\right) \tag{39}$$

$$k_4 = k_3\frac{\alpha}{\alpha+\nu+\mu} \tag{40}$$

$$k_5 = k_3\frac{\rho}{\rho+\gamma+\mu}. \tag{41}$$

In order to establish global asymptotic stability, $S < S^*$ and k_1 must be chosen such that

$$k_1\left(1-\frac{S^*}{S}\right) > k_2.$$

It is worth mentioning here that this global asymptotic stability shall be disease free.

2.9. Prophylaxis

Prophylaxis are off-medical control strategies in order to prevent the spread of the disease. Such measures include social distancing, mask wearing, hand-wash, sanitization and lockdown. Prophylaxis reduces the transmission rates. Lockdown reduces person to person contact in susceptible and exposed group which leads to reduction of β and η. This we observe reduction of reproduction number from Eq. (24).

If we see the relationship between reproduction number and isolation rate α, we get a graph (Fig. 8). Smaller value of β provides the steeper decline in reproduction number. The reproduction number is a decreasing function of the isolation rate α. The

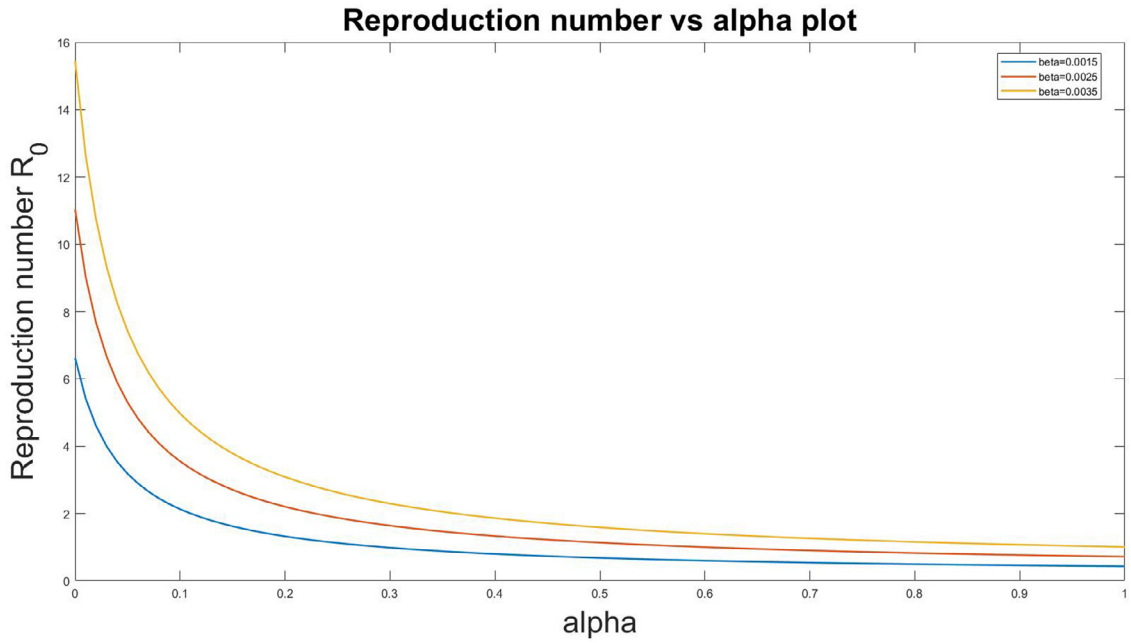

Fig. 8. The reproduction number as a decreasing, concave up function of α for several values of β.

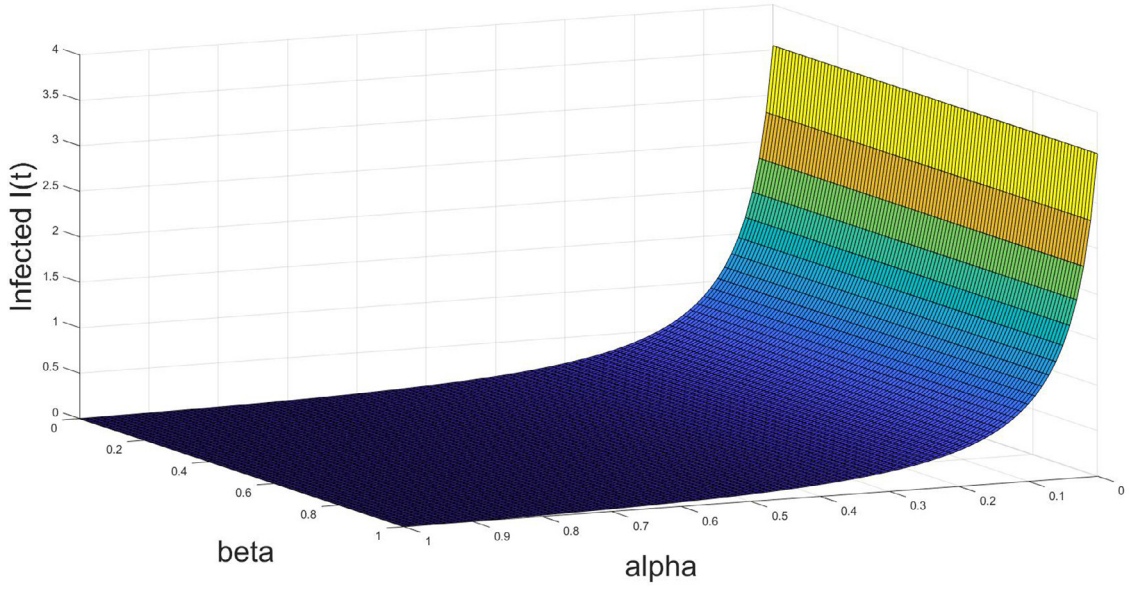

Fig. 9. Surface plot of infected I(t) with β and α.

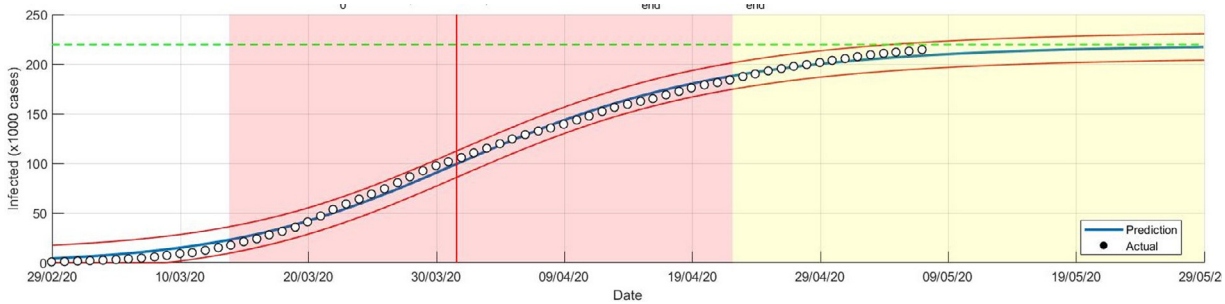

Fig. 10. Plot of cumulative infected vs. date for Italy.

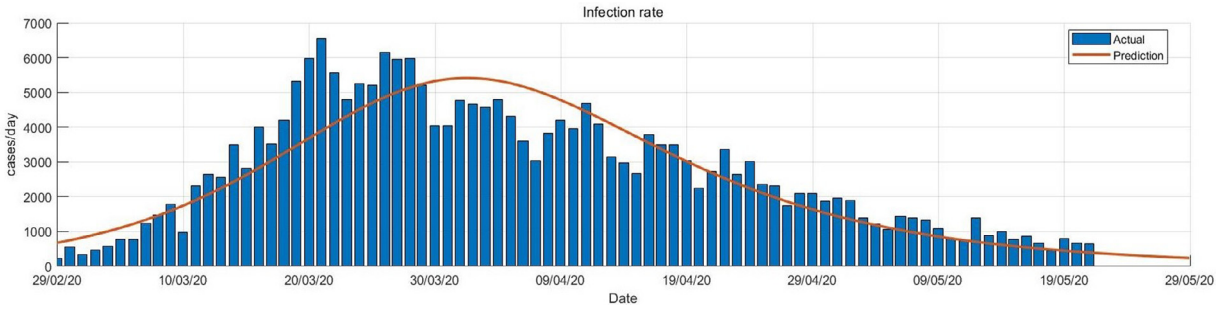

Fig. 11. Plot of reported infection per day vs. date for Italy.

critical isolation rate that gives $R_0 = 1$, is given by

$$\alpha* = \frac{\eta\beta S^*\big(\rho + q(\nu+\mu)\big) - (\gamma+\mu)(\eta+\theta+\mu)(\gamma+\rho+\mu)}{(\eta+\theta+\mu)(\gamma+\rho+\mu) - (1-p)q\eta\beta S^*}. \tag{42}$$

The Eq. (42) provides an information that one can control the reproduction number by applying the prophylaxis in a proactive manner. Variation of infected with respect to β and α is shown in Fig. 9 which is a decreasing, concave up function of α.

In the proposed model, we have considered isolation and quarantine as a separate compartment. Isolation has been studied in different disease model and found that it destabilize the dynamics and lead to oscillations [28,29] and thus, suggested as potential intrinsic mechanism to combat the disease.

3. COVID-19 data from Italy

In Italy, 228, 003 confirmed cases and 32, 486 deaths are reported for SARS-CoV-2 virus [4] on 22 May 2020 and 134, 560 peoples have been recovered. Out of total confirmed cases, 59% have been recovered and the fatality rate is estimated to be 14.24%. A plot of cumulative infections vs. time is shown in (Fig. 10). The Outbreak of epidemic started from 21 February 2020 and accelerated on 14 March 2020. The point of inflation are on 02 April 2020. The growth of population became steady on 25 April 2020. The phase ending of the epidemic is estimated to be third week of June 2020 by extrapolating the data. A plot of infection rate with dates are shown in (Fig. 11).

4. Discussion

Proposed model considers six stages of populations: susceptible (S), exposed (E), asymptotic (A), infected (I), quarantine/isolation (Q) and recovered (R). The model discriminates between infected and asymptotic people depending upon whether infected people from virus do not show symptom or otherwise. As per study, transmission of COVID-19 disease through asymptotic population is an evident feature. The quarantine and isolation in the population are carrying out from both stages: from exposed and infected. In the model, we consider that dead do not transmit the disease and a person who recovered from the disease will not undergo again through infection.

However, reproduction number is an effective tool to control the disease. From (Fig. 3), it is clear that larger the value of R_0 invite the disaster in a large amount quickly.

The sensitivity analysis of the model advocates to reduce the value of η in order to control the disease in an efficient way. Transmission rate β also controls the infection but in less sensitive manner than η. Changing ρ and θ do not affect in the number of infected in a large scale.

If we enhance the isolation rate α as given by Eq. (42), the disease will disappear from population. Thus it provides a threshold value of transmission which prevent the spreading of the disease using isolation only.

Credit author statement

All authors make equal contributions

Declaration of Competing Interest

The authors hereby declare their intensions to publish the attached manuscript in Chaos, Solitons and fractals.

Acknowledgments

The authors thank the referee for his concrete suggestions which resulted in a better organization of this article.

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