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A mathematical model of epidemiology in presence of vaccination for the spread of contagious diseases transmitting without vector

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Abstract. In the present work, we have established a mathematical model of epidemiology in presence of vaccination for contagious diseases which can transmit directly in absence of any vector taking into account the non-vaccinated but still uninfected population, vaccinated population and infected population. We have made a search for equilibrium points for the proposed system and have discussed their local asymptotic stabilities. Efforts have been made to find the solution of the proposed system. On the basis of extensive analysis, relevant comments have been made on mutual co-existence of the different groups stated above. Effort has also been made to establish a mathematical expression for the herd immunity threshold value.

Keywords: epidemiology, vaccination, equilibrium points, local asymptotic stability, mutual co-existence, herd immunity threshold value

1 Introduction

Europe witnessed Plague in 14th Century which killed 24 million lives. Aztecs lost half of 3.5 million to smallpox. A devastating influenza epidemic of 1919 snatched 20 million people. At present 1 million deaths occur per year due to malaria; 1 million deaths per year due to measles and 2 million deaths per year due to tuberculosis. Presently available records say that billions of people get infected with these diseases[18].

In the year 1760 Daniel Bernoulli showed that inoculation against smallpox would improve life expectancy of French. Ross developed Simple Epidemic Model in 1911. In the Simple Epidemic Model Ross assumed the population to be large and constant and it comprises of two groups, viz. i) group of susceptible and ii) group of infected individuals. In his model he considered that there is no birth, death, immigration or emigration, no recovery, no latency. Mixing was supposed to be homogeneous and infection rate is proportional to the number of infectives. From his model we can observe that as time goes to infinity each individual in the population gets infected[4, 5, 18, 21]. Later on in the year 1927 Kermack and McKendrick developed General Epidemic Model[18]. In the General Epidemic Model Kermack and McKendrick divided the population into three groups viz. i) Susceptible individuals, ii) Infected individuals and iii) Recovered individuals taking into account the same assumptions as in Simple Epidemic Model with only one alteration that the rate of recovery from the disease is constant. They proposed the concept of the basic reproductive number and they showed that if this number is greater than 1 then we have an epidemic[4, 5, 21]. In the year 1937 Fisher introduced a mathematical model of the spread of a gene in a population. This model was based on the same assumptions as in the Simple Epidemic Model with only one additional assumption that individuals disperse by a diffusion process with diffusion constant. His model comprised of a pair of partial differential equations and for certain initial conditions, the solutions of the partial differential equations tend to the travelling wave with

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minimum wave speed[6, 12, 18, 22, 26]. Capasso and Paveri-Fontana developed a mathematical model[9] describing the 1973's cholera epidemics in Italy. In their version, two equations described the dynamics of infected people in the community and the dynamics of the aquatic population of pathogenic bacteria. Codeco[10] proposed a mathematical model, together with numerical simulations which was an extension of the model of Capasso and Paveri-Fontana [9] where the dynamics of the susceptible population was taken into account to study long-term dynamics. Although in Codeco's model emigration and immigration were not taken into consideration. Furthermore, in that model birth rate and death rate in the group of susceptible were taken to be same which in reality may not be true. Roberts and Heesterbeek [25] concluded with an examination of techniques for modelling transmission dynamics in structured populations. Cai and Li [8] showed that if the basic reproduction number is less than unity, the disease-free equilibrium is globally asymptotically stable and in such a case the endemic equilibrium does not exist. Moreover, they showed that if the basic reproduction number is greater than unity, the disease is uniformly persistent and the unique endemic equilibrium of the system with saturation incidence is globally asymptotically stable under certain conditions. Zou, Gao and Zhong [23] formulated an SEIRS epidemic model with two time delays and pulse vaccination. By estabishing stroboscopic map, they obtained the exact infection free periodic solution of the impulsive epidemic system. Huo and Ma [16] proposed a delayed epidemic model with non-monotonic incidence rate which describes the psychological effect of certain serious on the community when the number of infective is getting larger.

For all the models discussed above the total population in the whole time duration of spread of epidemic is supposed to be fixed. This kind of simplification is very helpful to get a solution out of it smoothly but it seems to narrow the scope of these models. During the effective time span of the spread of any epidemic the population changes significantly due to the birth and death in the group of susceptible and death in the group of infected individuals. Furthermore, emigration and immigration were not allowed in those previous models. But real situations don't go in this simple way. To overcome this problem Ghosh[14] prescribed a model of epidemiology in which the changes in population during the process were allowed by taking into account birth, death, emigration and immigration of the individuals for those contagious diseases, which can transmit directly in absence of any vector and in absence of any vaccination. Dumrongpokaphan et al. [27] also proposed a SIR model with varying population size.

The most effective way to protect our society from several contagious diseases is to vaccinate individuals in a population as much as possible so that their immunal system can combat the factors causing those diseases. Always it is not possible to execute preventive vaccination and there are some diseases, which cannot be prevented by vaccination for a lifetime. So we need intervention whenever contagious diseases like tuberculosis, plague, small pox etc. start spreading or various cases manifest in a short time period. Intervention by mass vaccination is the only way out at these situations to stop the disease from taking the form of an epidemic. But in this process a question may arise: "Is it possible to vaccinate all individuals or is it necessary?" The term "Herd Immunit"[1, 2, 11, 15, 17, 24] appears as an answer to these questions. It is a proportion of uninfected individuals, which depends on the nature of the disease as well as on the vaccine. Sometimes it is enough for some diseases to vaccinate 60% of the population, but sometimes the proportion may reach 95%. Gandon and Day [13] proposed some useful qualitative predictions regarding the outcome of the competition between different types of vaccine favoured variants. Song, Jiang and Wei [28] studied a SVEIRS infectious disease model with pulse vaccination strategy and two time delays. Buonomo and Lacitignola [7] prescribed a compartmental epidemic model which incorporates a nonlinear incidence rate and an imperfect preventive vaccine given to susceptible individuals. Sen, Ibeas and Quesada [19, 20] proposed linear vaccination-based control strategy for a SEIR (susceptible plus infected plus infectious plus removed populations) disease propagation model.

In the present context we have advanced the model prescribed by Ghosh [14] by including the process of vaccination and this model may be applicable for all contagious diseases, which can transmit directly in absence of any vector. We have searched for the equilibrium points for the proposed system and have discussed their local asymptotic stabilities. We have also proposed a solution of the present system. Effort has also been made to establish a mathematical expression for the herd immunity threshold value. In this connection we mention the reference of our earlier work[3] in which too we made an effort to find an expression for the herd immunity taking into account the time-dependent series solution for the number of infected individuals. But

that the calculation was made under the assumption that all the higher degree terms after the second degree in the series are getting negligibly small. But the real world situations may not be always played by this simplified rule. The work on the herd immunity in the present paper is a generalization and to some extent rectification of this old work[3] on herd immunity. Additionally, in the present work, on the basis of extensive analysis, relevant comments have been made on mutual co-existence of the susceptible and infected groups. One additional feature of the present work is that it has not involved the idea of basic reproduction number as in the situation where the total population is varying the concept of basic reproduction number can be misleading.

2 Theory

2.1 The mathematical model of epidemiology in presence of intervention

Let $S(t)$ and $I(t)$ be the number of uninfected and infected individuals respectively at time t. Initially let there be 'n' uninfected and 'a' infected individuals in the community so that $S(0) = n, I(0) = a$. We consider the entire situation in the presence of vaccination/interventions to control the infection. Let p be the proportion of the uninfected mass, which is vaccinated. So effectively $(1-p)S$ can be taken as the non-vaccinated but still uninfected mass, i.e. in other words it can be taken as the number of susceptible. Growth of the number of infected individuals and decrease in the number of susceptible individuals are proportional to the product of the susceptible and infected individuals and this rate is assumed to be $\beta(1-p)SI$. We also assume that each individual becomes susceptible since birth (unless vaccinated) and death of each infected individual occurs due to the epidemic disease only. As an individual gets infected, it is not generally expected from him/her to give birth to a new child and even if it happens, that number is very small. So we can ignore that number. Again, immigration and emigration are very common phenomena for the group of uninfected, but these are not expected in the infected groups and if they exist in this group, then the corresponding numbers are too small to be considered. Let i be the uniform immigration rate and e be the uniform emigration rate in the group of uninfected. Let b be the rate of birth and d be the rate of death of uninfected per individual. We also consider that an infected person can recover and become susceptible again at a rate γI and infected persons are removed by hospitalization or by death at a rate αI. So, we have[3]

$$\begin{cases} \frac{\mathrm{d}S}{\mathrm{d}t} = -\beta(1-p)SI + (b-d)S + (i-e) + \gamma I, \\ \frac{\mathrm{d}I}{\mathrm{d}t} = \beta(1-p)SI - \gamma I - \alpha I. \end{cases} \tag{1}$$

Taking $b-d=c$, $i-e=k$ and $\beta' = \beta(1-p)$ we have

$$\begin{cases} \frac{\mathrm{d}S}{\mathrm{d}t} = -\beta' SI + cS + k + \gamma I, \\ \frac{\mathrm{d}I}{\mathrm{d}t} = \beta' SI - \gamma I - \alpha I. \end{cases} \tag{2}$$

Here the parameters α, β, γ and c are all positive while k can take any sign including 0. In the ideal case, for $c=0$ (i.e. birth rate=death rate), $k=0$ and $\alpha=0$ we have $\frac{\mathrm{d}S}{\mathrm{d}t} + \frac{\mathrm{d}I}{\mathrm{d}t} = 0$ which leads to the conclusion that the total population remains constant throughout. This situation has no real life implementation in large time domain.

From Eq. (2) we have $\frac{\mathrm{d}N}{\mathrm{d}t} = \frac{\mathrm{d}S}{\mathrm{d}t} + \frac{\mathrm{d}I}{\mathrm{d}t} = cS + k - \alpha I = cN + k - (c+\alpha)I$ where $N = S + I = total\ population$ at time t. To get $\frac{\mathrm{d}N}{\mathrm{d}t} > 0$ we must have $N > (1+\frac{\alpha}{c})I - \frac{k}{c}$. This condition leads to an increasing profile of the total population with time. Again to obtain $\frac{\mathrm{d}N}{\mathrm{d}t} < 0$ we must have $N < (1+\frac{\alpha}{c})I - \frac{k}{c}$. But, in reality, for any time t we must satisfy $N \geq I$. So, for consistency, we must have $I \leq N < (1+\frac{\alpha}{c})I - \frac{k}{c}$ and this will happen if $\alpha I - k > 0$ (as $c > 0$). If $k \leq 0$ this is obvious. Otherwise, if $k > 0$ we must have $I > \frac{k}{\alpha}$. This leads to a decreasing profile of the total population. Again, to investigate the stationarity of N we consider $\frac{\mathrm{d}N}{\mathrm{d}t} = 0$ which gives $N = (1+\frac{\alpha}{c})I - \frac{k}{c}$.

2.2 Search for equilibrium points

For equilibrium point we must have

$$\begin{cases} \frac{\mathrm{d}S}{\mathrm{d}t} = -\beta' SI + cS + k + \gamma I = 0, \\ \frac{\mathrm{d}I}{\mathrm{d}t} = \beta' SI - \gamma I - \alpha I = 0. \end{cases} \tag{3}$$

Solving, we get the points of equilibrium as $(-\frac{k}{c}, 0)$ and $\left[\frac{\gamma+\alpha}{\beta'}, \{\frac{c(\gamma+\alpha)}{\beta'} + k\} \cdot \frac{1}{\alpha}\right]$. The first equilibrium point exists only if $k \leq 0$ and the second equilibrium point exists only if $k \geq -c\frac{(\gamma+\alpha)}{\beta'}$.

2.3 Analysis of local asymptotic stability of equilibrium points

For the present system we have the characteristic equation

$$\begin{vmatrix} -\beta' I + c - \lambda & -\beta' S + \gamma \\ \beta' I & \beta' S - \gamma - \alpha - \lambda \end{vmatrix} = 0.$$

For the disease-free equilibrium point $(-\frac{k}{c}, 0)$, we have

$$\begin{vmatrix} c - \lambda & \frac{\beta' k}{c} + \gamma \\ 0 & \frac{-\beta' k}{c} - \gamma - \alpha - \lambda \end{vmatrix} = 0,$$

which gives, $\lambda = c, -(\frac{\beta' k}{c} + \gamma + \alpha)$. If $-c\frac{(\gamma+\alpha)}{\beta'} < k \leq 0$, then one eigen value is positive and the other is negative. So it is a saddle point. If $k < -c\frac{(\gamma+\alpha)}{\beta'}$, then both the eigen values are positive. So it is an unstable node in this case.

Again for the interior equilibrium point, $\left[\frac{\gamma+\alpha}{\beta'}, \{\frac{c(\gamma+\alpha)}{\beta'} + k\} \cdot \frac{1}{\alpha}\right]$, we have the characteristic equation

$$\begin{vmatrix} -\frac{\beta'}{\alpha}\left\{\frac{c(\gamma+\alpha)}{\beta'} + k\right\} + c - \lambda & -\beta'\frac{(\gamma+\alpha)}{\beta'} + \gamma \\ \frac{\beta'}{\alpha}\left\{\frac{c(\gamma+\alpha)}{\beta'} + k\right\} & \frac{\beta'(\gamma+\alpha)}{\beta'} - \gamma - \alpha - \lambda \end{vmatrix} = 0,$$

which gives

$$\lambda = \frac{-(c\gamma + k\beta') \pm \sqrt{(c\gamma + k\beta')^2 - 4\alpha^2(c\gamma + k\beta' + c\alpha)}}{2\alpha}.$$

If $k \geq 0$ we have $c\gamma + k\beta' > 0$ as well as $c\gamma + k\beta' + c\alpha > 0$. In this case, if the discriminant $(c\gamma + k\beta')^2 - 4\alpha^2(c\gamma + k\beta' + c\alpha) \geq 0$, both the eigen values are negative and thus the equilibrium point becomes a stable node and if $(c\gamma + k\beta')^2 - 4\alpha^2(c\gamma + k\beta' + c\alpha) < 0$, the equilibrium point becomes a stable focus. We can also have $-c\frac{(\gamma+\alpha)}{\beta'} \leq k < 0$ for the validation of this equilibrium point which in turn gives $c\gamma + k\beta' + c\alpha > 0$. Under this consideration, if $c\gamma + k\beta' < 0$ and $(c\gamma + k\beta')^2 - 4\alpha^2(c\gamma + k\beta' + c\alpha) \geq 0$ both the eigen values are positive and consequently it is an unstable node. On the other hand, if $c\gamma + k\beta' < 0$ and $(c\gamma + k\beta')^2 - 4\alpha^2(c\gamma + k\beta' + c\alpha) < 0$, the equilibrium point is an unstable focus. Here we must note that the parameters c, α, β', γ are all positive. But k is unrestricted in sign: for some communities it can be positive, somewhere it can be negative even zero also. Hence $(c\gamma + k\beta')$ can take both the signs. If $(c\gamma + k\beta') > 0$, obviously $c\gamma + k\beta' + c\alpha > 0$ as $c\alpha > 0$ for all circumstances. But if $c\gamma + k\beta' < 0$, then we may have $c\gamma + k\beta' + c\alpha > 0$ or it may be $c\gamma + k\beta' + c\alpha \leq 0$, depending on the magnitude of $c\alpha$.

3 Search for the solution of the system

Remembering $S(0) = n$ and $I(0) = \alpha$, we consider

$$\begin{cases} S = n + p_1 t + p_2 t^2 + p_3 t^3 + \cdots, \\ I = a + q_1 t + q_2 t^2 + q_3 t^3 + \cdots. \end{cases} \tag{4}$$

Then

$$\begin{cases} \frac{\mathrm{d}S}{\mathrm{d}t} = p_1 + 2p_2 t + 3p_3 t^2 + \cdots, \\ \frac{\mathrm{d}I}{\mathrm{d}t} = q_1 + 2q_2 t + 3q_3 t^2 + \cdots. \end{cases} \tag{5}$$

From the system of Eq. (2), we have

$$\frac{\mathrm{d}S}{\mathrm{d}t} + \frac{\mathrm{d}I}{\mathrm{d}t} = cS + k - \alpha I. \tag{6}$$

By Eq. (6) using Eq. (4) and Eq. (5), we get

$$\begin{cases} p_1 + q_1 = cn + k - \alpha a, \\ 2(p_2 + q_2) = cp_1 - q_1, \\ 3(p_3 + q_3) = cp_2 - q_2, \end{cases} \tag{7}$$

and so on.

Again using Eq. (4) and Eq. (5) in the first equation of Eq. (2), we get

$$\begin{cases} p_1 = -\beta' na + cn + k + \gamma a, \\ p_2 = \frac{1}{2}\Big[cp_1 + \gamma q_1 - \beta'(nq_1 + ap_1)\Big], \\ p_3 = \frac{1}{3}\Big[cp_2 + \gamma q_2 - \beta'(nq_2 + ap_2 + p_1 q_1)\Big], \end{cases} \tag{8}$$

and so on.

From Eq. (7) and Eq. (8), we can determine the expressions for p_1, p_2, p_3, q_1, q_2, q_3, etc. Finally we can write the solution as

$$\begin{cases} S = n + (-\beta' na + cn + k + \gamma a)t + \frac{1}{2}\Big[-2can\beta' + 2\beta'\gamma na + \alpha\beta' na \\ +c^2 n + ck + \gamma ac - \alpha\gamma a - \gamma^2 a - \beta'^2 n^2 a + \beta'^2 n a^2 - ka\beta' - \beta'\gamma a^2\Big] t^2 + \cdots, \\ I = a + (\beta' na - \alpha a + \gamma a)t + \frac{1}{2}\Big[can\beta' - 2\alpha\beta' na - 2\beta'\gamma na \\ +\alpha^2 a + 2\alpha\gamma a + \gamma^2 a + p^2 n^2 a - \beta'^2 n a^2 + ka\beta' + \beta'\gamma a^2\Big] t^2 + \cdots. \end{cases} \tag{9}$$

4 Mutual co-existence of the uninfected and infected individuals

From Eq. (2), we have

$$\frac{\mathrm{d}I}{\mathrm{d}S} = \frac{\beta' SI - \gamma I - \alpha I}{-\beta' SI + cS + k + \gamma I}. \tag{10}$$

Case A: $\frac{\mathrm{d}I}{\mathrm{d}S} > 0$

Sub-Case (i): $\frac{\mathrm{d}I}{\mathrm{d}t} > 0$ and $\frac{\mathrm{d}S}{\mathrm{d}t} > 0$ i.e. $\beta/SI - \gamma I - \alpha I > 0$ and $-\beta' SI + cS + k + \gamma I > 0$.

In this case, we have $S > \frac{\gamma+\alpha}{\beta'}$ and we consider $S = \frac{\gamma+\alpha}{\beta'} + h$ for $h > 0$. From the other inequality $-\beta' SI + cS + k + \gamma I > 0$, we have

$$I < \frac{1}{(\alpha + \beta' h)}\left[c\frac{(\gamma+\alpha)}{\beta'} + k + ch\right].$$

We take $f(h) = \frac{1}{(\alpha+\beta' h)}\left[c\frac{(\gamma+\alpha)}{\beta'} + k + ch\right]$.

So that $f'(h) = \frac{-(c\gamma + k\beta')}{(\alpha+\beta' h)^2}$.

If $c\gamma + k\beta > 0$, $f(h) < 0$. So in that case, $f(h)$ is a decreasing function of h. Hence $f(h) < f(0)$ for $h > 0$ i.e.

$$\frac{1}{(\alpha + \beta' h)}\left[\frac{c(\gamma+\alpha)}{\beta'} + k + ch\right] < \frac{1}{\alpha}\left[\frac{c(\gamma+\alpha)}{\beta'} + k\right].$$

Hence $I < \frac{1}{\alpha}\left[\frac{c(\gamma+\alpha)}{\beta'} + k\right]$.

If $c\gamma + k\beta' < 0$, then $f(h) > 0$. In that case, $f(h)$ is an increasing function of h. Hence $f(h) > f(0)$ for $h > 0$, i.e.,

$$\frac{1}{(\alpha + \beta' h)}\left[\frac{c(\gamma+\alpha)}{\beta'} + k + ch\right] > \frac{1}{\alpha}\left[\frac{c(\gamma+\alpha)}{\beta'} + k\right].$$

So, we are unable to get any specific range for I.

Sub-Case (ii): $\frac{\mathrm{d}I}{\mathrm{d}t} < 0$ and $\frac{\mathrm{d}S}{\mathrm{d}t} < 0$ i.e.

$$\beta' SI - \gamma I - \alpha I < 0 and - \beta' SI + cS + k + \gamma I < 0.$$

In this case, we have $S < \frac{\gamma+\alpha}{\beta'}$ and we consider $S = \frac{\gamma+\alpha}{\beta'} - h$ where $h > 0$. from the other inequality $-\beta' SI + cS + k + \gamma I < 0$, we get

$$I > \frac{1}{(\alpha - \beta' h)}\left[\frac{c(\gamma+\alpha)}{\beta'} - ch + k\right],$$

provided $\alpha - \beta h > 0$, i.e., $h < \frac{\alpha}{\beta'}$ or in other words $S > \frac{\gamma}{\beta'}$. Hence for $\frac{\gamma}{\beta'} < S < \frac{\gamma+\alpha}{\beta'}$ we have $I > \frac{1}{(\alpha-\beta' h)}\left[\frac{c(\gamma+\alpha)}{\beta'} - ch + k\right]$.

Now, we take $g(h) = \frac{1}{(\alpha-\beta' h)}\left[\frac{c(\gamma+\alpha)}{\beta'} - ch + k\right]$ So that, $g'(h) = \frac{c\gamma + k\beta'}{(\alpha-\beta' h)^2}$.

If $c\gamma + k\beta' > 0$, $g(h) > 0$. In that case $g(h)$ is an increasing function. Hence $g(h) > g(0)$ for $h > 0$ i.e.

$$\frac{1}{(\alpha - \beta' h)}\left[\frac{c(\gamma+\alpha)}{\beta'} - ch + k\right] > \frac{1}{\alpha}\left[\frac{c(\gamma+\alpha)}{\beta'} + k\right].$$

Hence, $I > \frac{1}{\alpha}\left[\frac{c(\gamma+\alpha)}{\beta'} + k\right]$.

If $c\gamma + k\beta' < 0$, $g(h) < 0$. In that case, $g(h)$ is a decreasing function. Hence $g(h) < g(0)$ for $h > 0$, i.e.,

$$\frac{1}{(\alpha - \beta' h)}\left[\frac{c(\gamma+\alpha)}{\beta'} - ch + k\right] < \frac{1}{\alpha}\left[\frac{c(\gamma+\alpha)}{\beta'} + k\right].$$

Hence, in this case we fail to get any specific range for I.

Case B: $\frac{\mathrm{d}I}{\mathrm{d}S} < 0$

Sub-Case(i): $\frac{\mathrm{d}I}{\mathrm{d}t} < 0$ and $\frac{\mathrm{d}S}{\mathrm{d}t} > 0$ i.e. $\beta' SI - \gamma I - \alpha I < 0 and - \beta' SI + cS + k + \gamma I > 0$. In this case, we have $S < \frac{\gamma+\alpha}{\beta'}$ and we consider $S = \frac{\gamma+\alpha}{\beta'} - h$ where $h > 0$. From the other inequality $\beta' SI + cS + k + \gamma I > 0$, we get

$$I < \frac{1}{(\alpha - \beta' h)} \left[\frac{c(\gamma+\alpha)}{\beta'} - ch + k \right],$$

provided $\alpha - \beta/h > 0$, i.e., $h < \frac{\alpha}{\beta'}$ or in other words $S > \frac{\gamma}{\beta'}$. Hence for $\frac{\gamma}{\beta'} < S < \frac{\gamma+\alpha}{\beta'}$, we have $I < \frac{1}{(\alpha-\beta' h)} \left[\frac{c(\gamma+\alpha)}{\beta'} - ch + k \right]$.

Now, by previous arguments in sub-case (ii) of Case A, we have for $c\gamma + k\beta' > 0$,

$$\frac{1}{(\alpha - \beta' h)} \left[\frac{c(\gamma+\alpha)}{\beta'} - ch + k \right] > \frac{1}{\alpha} \left[\frac{c(\gamma+\alpha)}{\beta'} + k \right].$$

This means we are not getting any specific range for I. Again, if $c\gamma + k\beta' < 0$, we have as in the previous case,

$$\frac{1}{(\alpha - \beta' h)} \left[\frac{c(\gamma+\alpha)}{\beta'} - ch + k \right] < \frac{1}{\alpha} \left[\frac{c(\gamma+\alpha)}{\beta'} + k \right].$$

Hence we have, $I < \frac{1}{\alpha} \left[\frac{c(\gamma+\alpha)}{\beta'} + k \right]$.

Sub-Case (ii): $\frac{\mathrm{d}I}{\mathrm{d}t} > 0$ and $\frac{\mathrm{d}S}{\mathrm{d}t} < 0$, i.e., $\beta' SI - \gamma I - \alpha I > 0$ and $\beta' SI + cS + k + \gamma I < 0$.

In this case, we have $S > \frac{\gamma+\alpha}{\beta'}$. We take $S = \frac{\gamma+\alpha}{\beta'} + h$ where $h > 0$. Then from the other inequality $-\beta' SI + cS + k + \gamma I < 0$, We have $I > \frac{1}{(\alpha+\beta' h)} \left[\frac{c(\gamma+\alpha)}{\beta'} + ch + k \right]$.

But, previously in sub-case (i) of Case A we got that for $c\gamma + k\beta' > 0$,

$$\frac{1}{(\alpha + \beta' h)} \left[\frac{c(\gamma+\alpha)}{\beta'} + ch + k \right] < \frac{1}{\alpha} \left[\frac{c(\gamma+\alpha)}{\beta'} + k \right].$$

Hence we are not getting any specific range for I here also. Again, if $c\gamma + k\beta' < 0$, we have previously obtained

$$\frac{1}{(\alpha + \beta' h)} \left[\frac{c(\gamma+\alpha)}{\beta'} + ch + k \right] > \frac{1}{\alpha} \left[\frac{c(\gamma+\alpha)}{\beta'} + k \right].$$

In that case, $I > \frac{1}{\alpha} \left[\frac{c(\gamma+\alpha)}{\beta'} + k \right]$.

Keeping in view all the above facts we have the following table:

Situations 1 and 2 are analogous to the model in which both the groups of uninfected and infected mutually co-exist by increasing their numbers. In situations 3 and 4, both the groups gradually reduce their numbers. In situations 5 and 6, the group of uninfected individuals increases its number, but the number of infected individuals gradually decreases. Situations 7 and 8 are very alarming where the group of uninfected individuals reduces its number but the group of infected individuals increases with time. These situations can be called as "Critically Epidemic situations". We must carefully handle the situation arising at the serial 6 in the above table. Here as $c\gamma + k\beta' < 0$, we may have $\frac{1}{\alpha} \left[\frac{c(\gamma+\alpha)}{\beta'} + k \right] \leq 0$. In that case, '$I$' becomes a negative quantity, which is absurd. So, this situation must not arise. The situation is possible only if $\frac{1}{\alpha} \left[\frac{c(\gamma+\alpha)}{\beta'} + k \right] > 0$. From the above table it is also clear that in all possible cases we are getting specific ranges for 'S' in the positive half which argues for the consistency of the model. But we can find four such cases where we are unable to get any specific range for 'I'. Out of these in the situations 2 and 7 we have $\frac{\mathrm{d}I}{\mathrm{d}t} > 0$ which at least assures that once 'I' gets started with a nonnegative value it retains its nonnegative nature throughout. But that may not be assured in the situations 4 and 5 where $\frac{\mathrm{d}I}{\mathrm{d}t} < 0$. These cases are to be handled carefully and we must stop the mathematical scheme once we get $I = 0$.

Sign of $\frac{dI}{dS}$	Serial No.	Sign of $\frac{dI}{dt}$	Sign of $\frac{dS}{dt}$	Sign of $c\gamma + k\beta$	Range of S	Range of I
+ve	1	+ve	+ve	+ve	$S > \frac{\gamma+\alpha}{\beta'}$	$I < \frac{1}{\alpha}\left[\frac{c(\gamma+\alpha)}{\beta'+k}\right]$
	2	+ve	+ve	−ve	$S > \frac{\gamma+\alpha}{\beta'}$	No specific range for I
	3	−ve	−ve	+ve	$\frac{\gamma}{\beta'} < S < \frac{\gamma+\alpha}{\beta'}$	$I > \frac{1}{\alpha}\left[\frac{c(\gamma+\alpha)}{\beta'+k}\right]$
	4	−ve	−ve	−ve	$\frac{\gamma}{\beta'} < S < \frac{\gamma+\alpha}{\beta'}$	No specific range for I
−ve	5	−ve	+ve	+ve	$\frac{\gamma}{\beta'} < S < \frac{\gamma+\alpha}{\beta'}$	No specific range for I
	6	+ve	+ve	−ve	$\frac{\gamma}{\beta'} < S < \frac{\gamma+\alpha}{\beta'}$	$I < \frac{1}{\alpha}\left[\frac{c(\gamma+\alpha)}{\beta'+k}\right]$
	7	+ve	−ve	+ve	$S > \frac{\gamma+\alpha}{\beta'}$	No specific range for I
	8	+ve	−ve	−ve	$S > \frac{\gamma+\alpha}{\beta'}$	$I > \frac{1}{\alpha}\left[\frac{c(\gamma+\alpha)}{\beta'+k}\right]$

5 Herd immunity

We can precisely categorize the epidemic diseases into two fundamental classes: i) natural epidemic and ii) behavioral/acquired Epidemic. Class (i) means those contagious diseases, which spread unwillingly by means of vectors or via some media. But class (ii) categorizes those diseases, which spread by some physical process, or interaction, which can be prevented if human beings are careful, aware and conscious. For example HIV/AIDS, spreads mainly due to unsafe sexual interaction, which is completely a behavioural process. Although HIV/AIDS can spread due to infected blood transfusion, contaminated needles or from infected mothers to newly born babies, we mainly consider the spread of HIV/AIDS as a behavioural/acquired process. We here deal with the process of vaccination only for natural epidemic diseases. The concept of Herd Immunity for this particular category can be mapped as a dependent parameter involving implicitly the efficiency of the entire intervention process as well as the characteristic of the disease. Herd immunity describes a type of immunity that occurs when vaccination of a portion of a population (or Herd) provides protection to unvaccinated individuals[1]. The more immune individuals present in a population the lower the possibility of an uninfected individual in that population[2]. It is notable that to introduce the Herd Immunity we have not used basic reproduction number unlike the earlier communications[8, 15, 17, 23, 24]. This is due to the fact that in the situation where the total population is varying the concept of basic reproduction number can be misleading.

Although no vaccination offers 100% protection, virologists have found that when a certain percentage of a population is vaccinated the spread of the disease is expected to stop[11]. This critical percentage is called "Herd Immunity Threshold Valu"[1, 17]. In the present study we make an attempt to estimate this threshold value by the following manner:

At the threshold we must have $\frac{dI}{dt} = 0$ which gives, using Eq. (2) $\beta' S = \gamma + \alpha$. This in turn gives

$$p = 1 - \frac{\gamma + \alpha}{\beta S}. \tag{11}$$

The above expression for p determines the herd immunity threshold value at a certain instant at which the number of the susceptibles is S. The present expression confirms that as S increases the value of p increases. In addition to that, as p depends on the parameters α, β and γ that characterize the epidemic disease it can be interpreted that the herd immunity threshold value depends significantly on the characteristics of the disease.

6 Discussion

In the present work, we have presented a mathematical model which provides an insight to intervention (vaccination) of epidemic diseases like tuberculosis, anthrax, plague etc. transmitting without any vector. But it is to be noted that the present model does not work for diseases like HIV which spreads by sexual interaction.

In this particular work, we have tried to connect the parameter p for the process of intervention (vaccinating adults); not significantly for the process of prevention (vaccinating children). Again, as the value of p depends significantly on the characteristics of the disease the present parameter p is to be used carefully in practical situations.

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