EFFICACY OF PULSE VACCINATION OVER CONSTANT VACCINATION IN COVID-19: A DYNAMICAL ANALYSIS

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Abstract. Today, the whole world is fighting against a dreadful pandemic COVID-19 with no substantial vaccination invented till now. Researchers, scientists are struggling hard to develop different vaccination strategies which can be of help to society. It's a novel idea to pursue multiple strategies and platforms because we really don't know what will work best. Keeping this in mind, in this paper we have developed a Susceptible-Infected-Removed(SIR) model of corona-virus with constant as well as pulse vaccination strategy in individuals to show that how vaccination strategy can also play an important role to control infection and block the virus production. The model is formulated for both constant vaccination and pulse vaccination by taking discrete and distributive delays and analysed the potency of vaccination along with delay. In the latter case, pulse Vaccination of the susceptible population takes place at periodic intervals. The system is studied for a special infection free case and is solved for a T-periodic solution. In both the systems, disease free equilibrium point is locally as well as globally attractive if the basic reproduction number($R_0 < 1$) is less then 1 and the endemic point is stable provided $R_0 > 1$. Further, we have compared the efficacy of constant and pulse vaccination plan and obtained an interesting result that pulse vaccination strategy has come up as a better strategy as it can lower the reproduction number comparatively and should followed frequently. In addition to it, if we extend the time period, it would be difficult to control the infection. Finally, all the theoretical results are verified with numerical simulation using MATLAB.

Keywords: discrete delay; distributive delay; constant vaccination; pulse vaccination; local and global stability; permanence.

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1. INTRODUCTION

The corona virus infection (COVID-19) represents a significant world wide danger. Corona is a vast group of enveloped RNA viruses, causes respiratory infection as SARS, MERS and COVID-19. Generally COVID-19 can be partitioned among three levels. Level I is the incubation time, where symptoms are shown or not. Level II is the not serious indicative brooding interval in which infection is visible. Level III is the extreme respiratory symptomatic with the maximum virus load[4]. Neither all individual residing in corona affected area are contaminated nor every single infected patient create serious respiratory ailment. So, it totally depends on individual's immunity which is developed by adaptive immune response to eradicate the virus and to prevent the disease to reaches up to severe stages[21]. Also, innate immunity plays a vital role in the detection of viral infection with the help of pattern recognition receptors(PRRs)[5]. Therefore, it is required to upgrade immune response. In addition to this for the improvement of internal immune response at the incubation period and severe stages, the individual ought to be healthy and a proper hereditary contrasts are notable for singular reaction to pathogens. When a defensive insusceptible reaction is weakened, virus will propagate and affect many tissues. A few control measures are being done to limit the spread of this infectious sickness like social separating, isolation, homely quarantine etc. Lock down is the best strategy but can't be incorporated at numerous stages[1]. [8] assumed that inoculation of BCG may provide defence against corona virus infection(COVID-19) as it helps to improve immune system. All these safety measurements can control the infection but can't eradicate it from roots as it is seen that after discharge from hospital some persons can again get infected where as some fully recovered. These situations happen only due to immunity. Vaccination is the only technique to control the infection as virus can never removes from our body. We can only make them weak by developing immunity against it. The infection will continue contaminating until a huge population creates in-susceptibility against it. According to WHO, people aged above 60 years, children below 10 years and people underlying with medical condition are at high risk of corona infection due to weaker immune response and current statistical data showed people of

aged $31 - 40$ years are highly infected due to frequent contact with people. So, it is required to vaccinate people either continuously at definite time interval with different dosage or vaccinate with a single dosage to boost up immunity against this dreadful disease. A vaccine BNT-162 has been developed by the joint effort of BioNTech and Pfizer for COVID-19 and the trial is going on whether it is effective with monotonous immunization with multiple dosage at different time interval or by constant vaccination with a single dosage for the age group $18 - 55$. NIAID(National Institute of Allergic and Infectious Diseases) has developed a vaccine named as mRNA-1273 and trial is going on. According to Dr. Anthony Fauci, it has two injections, second will be applied after 28 days of first injection with three dosage and should follow for one year. These results motivate us to think about pulse vaccination. It has always been a very important strategy which has eradicated several infectious diseases and as per current scenario, it should be done on continuous basis depending upon the nature of epidemic and environment of a region. For example: In May 1980, WHO announced the global eradication of smallpox. This dreadful disease was cured by pulse vaccination strategy which extended all over the world. Polio is also almost eradicated by PVS. Now, we have many vaccines which are efficient in preventing viral infection such as rabies, yellow fever, hepatitis B, measles etc. To begin with, various generalized mathematical models have been proposed till now to capture the dynamics of disease using compartment modelling approach. A mathematical model was developed in 20th century by [12] known as the SIR epidemic model. Some more important models were developed by many authors [3, 16]. [19] compared the effectiveness and cost between the pulse vaccination and concluded that pulse vaccination leads to epidemic eradication at low cost. [26, 20] discussed that duration of pulsing and rate of PVS is responsible for permanence of infection. [25] showed the effectiveness of vaccination doze and gap over the reproduction number and virus growth. Many authors suggested that PVS is a blessing in the explosion of viral diseases and should be done at a high rate as compared to continuous vaccination by using SIR model, SIRS model, SIS model, SEIRS model and it is observed that SIRS model is complicated then any other under PVS [2, 30, 6, 7]. [10] studied the complexity of SIRS epidemic with non linear incidence rate. [27, 28] discussed that PVS done at large rate will be helpful in eradication of disease by using SEIRS and SIRVS model. [13] presented the

application of PVS with horizontally and vertically communication and compared the effectiveness of constant and PVS strategy. [24] suggested that PVS and quarantine technique is helpful in eradication of disease with discrete delayed SEIQR model. [23] studied the SEIR epidemic with pulse vaccination at a non linear incidence rate and two time delays and found eradication condition. [22] studied SIR epidemic model with PVS and age structured. [17] studied SVEIR epidemic model under adaptive impulsive vaccination. Mathematical modelling has been playing a very important role in understanding the dynamics of diseases and helps to control them. Till now many mathematical model has been developed which focused about the treatment of disease and controlling of disease with the help of continuous vaccination but many theoretical results shows that dynamical behaviour of continuous vaccination is questionable but a planned pulse vaccination strategy, unites our framework to stable periodic solution with zero infective. To understand biologically, corona usually takes time to develop in the host as there is a specific time period of minimum $4-5$ days and maximum of $10-14$ days approximately between the time of contact with the infection and the first time when the symptoms are seen which is commonly known as the 'Incubation Period' and it is varied according to immunity level. This period is responsible for a time-lag in the infectious models. Therefore we are taking a typical example of a generalized S-I-R outbreak model along with distributed lag given as[15].

(1)
$$
\begin{cases}
\dot{S(t)} = -\beta I(t) \int_{-\infty}^{t} G(t-\tau)S(\tau)d\tau - bS(t) + b, \\
\dot{I(t)} = \beta I(t) \int_{-\infty}^{t} G(t-\tau)x(\tau)d\tau - \beta S(t)I(t) - bI(t), \\
\dot{R(t)} = \beta S(t)I(t) - bR(t),
\end{cases}
$$

$G(t) = ae^{-at}$, $a > 0$, $\int_0^{\infty} G(\tau)d\tau = 1$. Here, β and b are positive constants, where b is the recruitment rate of susceptible as well as the death rate of all the three populations and β is the contact rate.

A typical example of S-I-R outbreak model along with discrete lag is given as[14],

$$
\begin{cases}
\dot{S(t)} = \mu - \beta S(t)I(t) - \mu S(t), \\
\dot{I(t)} = \beta S(t)I(t) - \beta e^{-b\tau}S(t-\tau)I(t-\tau) - \mu I(t), \\
\dot{R(t)} = \beta e^{-b\tau}S(t-\tau)I(t-\tau) - \mu R(t),
\end{cases}
$$

where, τ refers to the convalescence period (infectious period). The above system is formulated using the assumptions that new comers as soon as they are born are transferred to 'susceptible' class (S). Individuals who have lived through the infectious period τ are transferred to the 'removed' class (R), the death rate during that period is also considered which is represented by '$\beta e^{-b\tau} S(t - \tau) I(t - \tau)$' term.

Keeping in mind the above literature review, we have formed an SIR model with both distributive and discrete delay and discussed the effect of two vaccination strategies on it which are as follows:

1. Pulse vaccination strategy : PVS can be characterized as the replicated utilization of vaccine over an age go. Expect that immunize a portion(say) μ of the whole affected community in only one pulse and is employed after T month.

2. Constant vaccination strategy : It is defined as the infants or any susceptible, vaccinated once to control the infection and lessen the death rate of susceptible with an extent(say) μ of those immunized effectively. The paper is organized in the following manner: In section 2, we have constructed SIR model with pvs along with the lemmas to establish the boundedness of the system. We have discussed the global attractivity of disease free case and permanence of the system. In section 3, S-I-R model with constant vaccination strategy is proposed and local and global stability of the steady state solutions are discussed. In section 4, we have compared the reproduction no. of cases without vaccination, with constant vaccination and pulse vaccination. Finally, theoretical results are verified using numerical simulations and we have given justification the significance of the results that we have obtained throughout our study in the conclusion.

2. S-I-R MODEL WITH PULSE VACCINATION STRATEGY

In this section, model with pulse vaccination is proposed.

$$
(2) \quad
\begin{cases}
\dot{S}(t) = -\beta I(t) \int_{-\infty}^{t} G(t-\tau)S(\tau)d\tau - bS(t) + b(S(t) + I(t) + R(t)) \\
\dot{I}(t) = \beta I(t) \int_{-\infty}^{t} G(t-\tau)S(\tau)d\tau - \beta e^{-b\tau}S(t-\tau)I(t-\tau) - bI(t), \; t \neq nT \\
\dot{R}(t) = \beta e^{-b\tau}S(t-\tau)I(t-\tau) - bR(t) \\
S(t^+) = (1-\mu)S(t), \\
I(t^+) = I(t), \; t = nT \\
R(t^+) = R(t) + \mu S(t)
\end{cases}
$$

where T, denotes the time when impulsive effect takes place, $n = 1, 2, 3, ...$ and following variables and parameters have been used throughout the process of model development.

Variables/Parameters	Relevance
S	Susceptible Class
I	Infected Class
R	Removed Class
τ	Infection period
$b > 0$	Recruitment rate of susceptible class
$\beta > 0$	Contact rate
$0 < \mu < 1$	Vaccination rate

Also, $N(t) = S(t) + I(t) + R(t) = 1$. Mathematical model is shown by the following diagram Since, the third class in system (2) does not appear in first two equations, our main focus is on first two equations only.

So, we use the chain transformation $Z(t) = \int_{-\infty}^{t} G(t-\tau)S(\tau)d\tau$. Since, $\int_{-\infty}^{t} G(t-\tau)d\tau = \lim_{A \to -\infty} \int_{A}^{t} ae^{-a(t-\tau)}d\tau = 1$ and $\int_{-\infty}^{t} G(t-\tau)S(\tau)d\tau$ is convergent, then $\Delta Z(t) = \int_{-\infty}^{t^+} G(t-\tau)S(\tau)d\tau - \int_{-\infty}^{t} G(t-\tau)S(\tau)d\tau = 0$.
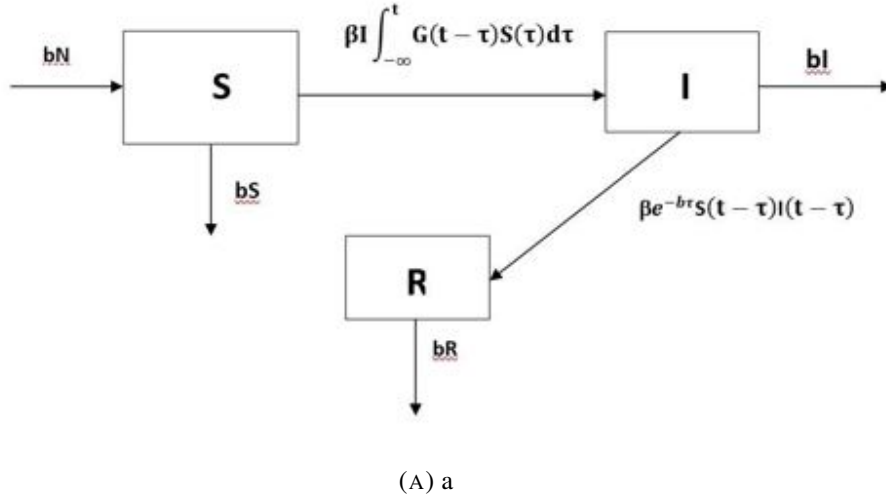
(A) a

FIGURE 1. Flowchart

So, reducing system (2) and introducing the transformation gives rise to following framework:

$$(3) \quad \begin{cases} \begin{cases} \dot{S}(t) = -\beta I(t)Z(t) - bS(t) + b, \\[2mm] \dot{I}(t) = \beta I(t)Z(t) - \beta e^{-b\tau}S(t-\tau)I(t-\tau) - bI(t), \ t \neq nT \\[2mm] \dot{Z}(t) = a(S(t) - Z(t)) \end{cases} \\[10mm] \begin{cases} S(t^+) = (1-\mu)S(t), \\[2mm] I(t^+) = I(t), \ t = nT \\[2mm] Z(t^+) = Z(t) \end{cases} \end{cases}$$

The applications of (3) to population dynamics motivate us to assume that solution of the (3) satisfies the initial conditions $\phi \in C_h{}^+$, and $\phi(0) \geq 0$ which lies in $C_h{}^+ = [\phi = (\phi_1(s), \phi_2(s), \phi_3(s)) \in C_h{}^+ : \phi_i(0) \geq 0 (i = 1, 2, 3)]$, where $\phi(s) > 0$, continuous and bounded function for $s \in [0, \infty)$.

2.1. Preliminaries.

In this section, we will define a few definitions, lemmas which would be required in the paper.

Definition 2.1[15] Let $V : R_+ \times R_+^3$, then $V \in V_0$, if:

(1) V is continuous in $(n\tau, (n+l)\tau] \times R_+^3$ and $((n+l)\tau, (n+1)\tau] \times R_+^3$, for each $z \in R_+^3, n \in Z_+$,

$V(n\tau^+, z) = \lim_{(t,y)\to(n\tau^+,z)} V(t,y), V((n+l)\tau^+, z) = \lim_{(t,y)\to((n+l)\tau^+,y)} V(t,y)$ exist.

(2) V is Locally Lipschitzian in z.

lemma 2.2[29]Let $V : R_+ \times R_+^3 \to R_+$ and $V \in V_0$. Assume that

$$\begin{cases} D^+V(t,x) \le g(t,V(t,x)), \ t \ne n\tau \\ V(t,x(t^+)) \ne \psi_n(V(t,x)), \ t = n\tau \end{cases}$$

where $g(t,u) : R_+ \times R_+^3 \to R_+$ is continuous in $(n\tau, (n+1+\tau] \times R_+^3$ and quasi-monotone non decreasing in u, for $v \in R_+^3$, $\lim_{(t,u)\to(n\tau^+,v)} g(t,u) = g(n\tau^+, v)$ exists, and $\psi_n : R_+ \to R_+$ is non decreasing. Let $r(t)$ be the maximal solution of the following vector impulsive differential system :

$$\begin{cases} \frac{du}{dt} = g(t,u), \ t \ne nT \\ u(t^+) = \psi_n(u(t)), u(t_0^+) = u_0 \ge 0, \ t = nT \end{cases}$$

existing on $[t_0, \infty)$. then

$$V(t_0^+, x_0) \le r(t), \ t \ge t_0,$$

lemma 2.3 Given,

(4)
$$\begin{cases} \begin{cases} \dot{p}(t) = b(1 - p(t)), \\ \dot{q}(t) = a(p(t) - q(t)), \ t \ne nT \end{cases} \\ \\ \begin{cases} p(t^+) = (1 - \mu)p(t), \\ q(t^+) = q(t), \ t = nT. \end{cases} \end{cases}$$

then (4) has unique T-periodic solution $(p^*(t), q^*(t))$ given by

(5)

$$\begin{cases} p^*(t) = 1 - \dfrac{\mu e^{-b(t-nT)}}{1-(1-\mu)e^{-bT}}, \\[2mm] q^*(t) = 1 + a\mu \dfrac{e^{-a(t-nT)}(1-e^{-bT}) - e^{-b(t-nT)}(1-e^{-aT})}{(a-b)(1-e^{-aT})(1-(1-\mu)e^{-bT})}, \\[2mm] p^*(0^+) = 1 - \dfrac{\mu}{1-(1-\mu)e^{-bT}}, \\[2mm] q^*(0^+) = 1 - a\mu \dfrac{(e^{-bT}-e^{-aT})}{(a-b)(1-e^{-aT})(1-(1-\mu)e^{-bT})}, \end{cases}$$

and for each solution, $p(t) \to p^*(t)$, $q(t) \to q^*(t)$ as $t \to \infty$.

Proof. Solving first equation of (4) yields,

(6)
$$p(t) = e^{-b(t-nT)}(p(nT^+) - 1) + 1, \; nT < t \le (n+1)T.$$

Solving second equation of (4) yields,

(7)
$$q(t) = q(nT^+)e^{-a(t-nT)} + \frac{a}{a-b}(p(nT^+) - 1)(e^{-b(t-nT)} - e^{-a(t-nT)}) + 1 - e^{-a(t-nT)}, \; nT < t \le (n+1)T.$$

The stroboscopic map of difference equation, gives
$$p((n+1)T^+) = (1-\mu)p((n+1)T) = (1-\mu)p(nT^+)e^{-bT} + (1-\mu)(1-e^{-bT}),$$

$$q((n+1)T^+) = q((n+1)T) = q(nT^+)e^{-aT} + \frac{a}{a-b}(p(nT^+) - 1)(e^{-bT} - e^{-aT}) + 1 - e^{-aT}.$$

The fixed points are,

$$p^* = 1 - \frac{\mu}{1-(1-\mu)e^{-bT}},$$

$$q^* = 1 - a\mu \frac{(e^{-bT} - e^{-aT})}{(a-b)(1-e^{-aT})(1-(1-\mu)e^{-bT})}.$$

Hence, the T-periodic solution $(p^*(t), q^*(t))$ comes out to be:

$$p^*(t) = 1 - \frac{\mu e^{-b(t-nT)}}{1-(1-\mu)e^{-bT}}, \; nT < t \le (n+1)T,$$

$$q^*(t) = 1 + a\mu \frac{e^{-a(t-nT)}(1-e^{-bT}) - e^{-b(t-nT)}(1-e^{-aT})}{(a-b)(1-e^{-aT})(1-(1-\mu)e^{-bT})}, \; nT < t \le (n+1)T.$$

Now, we show that the solution is attractive.

Let $(p(t), q(t))$ be the solution of (4). Then, for $t \in (nT, (n+1)T]$, we have,

$$p(t) = p^*(t) + (p(nT^+) - p^*)e^{-b(t-nT)},$$

$$q(t) = q^*(t) + (q(nT^+) - q^*)e^{-a(t-nT)} + \frac{a}{a-b}(p(nT^+) - p^*)(e^{-b(t-nT)} - e^{-a(t-nT)}).$$

by the recursion formula we have,

$$p((n+1)T^+) = (1-\mu)p(nT^+)e^{-bT} + (1-\mu)(1-e^{-bT}).$$

therefore,

$$p(nT^+) = (1-\mu)^n e^{-nbt} p(0^+) + [1 + (1-\mu)e^{-bT} + ... + (1-\mu)^{n-1}e^{-(n-1)bt}] \times (1-\mu)(1-e^{-bT})$$

$$= (1-\mu)^n e^{-nbt} p(0^+) + (1-\mu)(1-e^{-bT})\frac{1-(1-\mu)^n e^{-nbt}}{1-(1-\mu)e^{-bT}}$$

$$\to p^*(n \to \infty),$$

Thus, we have $p(t) \to p^*(t)$ when $n \to \infty$.

Also,

$$q((n+1)T^+) = q((n+1)T) = q(nT^+)e^{-aT} + \frac{a}{a-b}(p(nT^+) - 1)(e^{-bT} - e^{-aT}) + 1 - e^{-aT},$$

and $\lim_{n\to\infty} x(nT^+) = x^*$, for n large enough we have the following recursion formula:

$\therefore$ for any $l \in N^+$ we have,

$$q((l+N)T^+) = q(nT^+)e^{-lat} + [1 + e^{-aT} + ... + e^{-(l-1)at}] \times (\frac{a\mu(e^{-bT} - e^{-aT})}{(a-b)(1-(1-\mu)e^{-bT})} + (1-e^{-aT}))$$

$$= q(nT^+)e^{-lat} + \frac{1-e^{-lat}}{1-e^{-aT}} \times \left(\frac{a\mu(e^{-bT}-e^{-aT})}{(a-b)(1-(1-\mu)e^{-bT})} + (1-e^{-aT}) \right) \to \frac{a\mu(e^{-bT}-e^{-aT})}{(a-b)(1-(1-\mu)e^{-bT})}$$

$$+(1-e^{-aT})q(t) \to q^*(t)$$

as $(l \to \infty)$.

This completes the proof.   □

2.2. Local Stability : Infection Free Case

Our aim is to establish the conditions responsible for local stability and global attractivity of
the infection free case. After neglecting all the terms which include infection, our system is
reduced to the following:

(8)
$$\begin{cases} \begin{cases} \dot{S}(t) = -bS(t) + b, \\ \dot{Z}(t) = a(S(t) - Z(t)), \ t \neq nT, \end{cases} \\ \\ \begin{cases} S(t^+) = (1-\mu)S(t), \\ Z(t^+) = Z(t), \ t = nT \end{cases} \end{cases}$$

By Lemma (2.3), above system has a positive T-periodic solution. i.e. unique $(S^*(t), Z^*(t))$ is
specified as,

(9)
$$\begin{cases} S^*(t) = 1 - \frac{\mu e^{-b(t-nT)}}{1-(1-\mu)e^{-bT}}, \\ \\ Z^*(t) = 1 + a\mu \frac{e^{-a(t-nT)}(1-e^{-bT}) - e^{-b(t-nT)}(1-e^{-aT})}{(a-b)(1-e^{-aT})(1-(1-\mu)e^{-bT})}, \\ \\ S^*(0^+) = 1 - \frac{\mu}{1-(1-\mu)e^{-bT}}, \\ \\ Z^*(0^+) = 1 - a\mu \frac{(e^{-bT}-e^{-aT})}{(a-b)(1-e^{-aT})(1-(1-\mu)e^{-bT})}, \end{cases}$$

and $S(t)$ approaches $S^*(t)$ and $Z(t)$ approaches $Z^*(t)$ as t approaches ∞.

Theorem 2.4 [18] If $R = \dfrac{\beta(1-e^{-b\tau})[T - \frac{\mu(1-e^{-bT})}{b(1-(1-\mu)e^{-bT})}]}{bT} < 1$, then (3) has a solution $(S^*(t), 0, Z^*(t))$

which is locally stable.

Proof. We can verify that the solution of infection free system exists from the lemma (2.3), We will use method of small amplitude perturbations of the solution to show that the system is locally stable. Define $S(t) = S^*(t) + u(t)$, $I(t) = v(t)$, $Z(t) = Z^*(t) + w(t)$, where u, v, w are small perturbations, then equations of (3) can be expanded to a Taylor series and after neglecting the higher order terms we get following:

(10)
$$
\begin{cases}
\begin{cases}
\dot{u}(t) = -\beta v(t)Z^*(t) - b(S^*(t) + u(t)) + b, \\
\dot{v}(t) = [\beta(Z^*(t) - e^{-b\tau}S^*(t)) - b]v(t),\ t \neq nT \\
\dot{w}(t) = a(S^*(t) + u(t) - Z^*(t) - w(t)),
\end{cases} \\[2em]
\begin{cases}
u(t^+) = (1-\mu)u(t), \\
v(t^+) = v(t),\ t = nT \\
w(t^+) = w(t),
\end{cases}
\end{cases}
$$

If $\phi(t)$ is the fundamental matrix of (10), then it must satisfy

$$
\frac{d\phi(t)}{dt} = A\phi(t)
$$

where

$$
A(t) = \begin{pmatrix}
-b & -\beta Z^*(t) & 0 \\
0 & \beta(Z^*(t) - e^{-b\tau}S^*(t)) - b & 0 \\
a & 0 & -a
\end{pmatrix}
$$

The forth, fifth and sixth equations can be linearized as:

$$
\begin{pmatrix}
u(t^+) \\
v(t^+) \\
w(t^+)
\end{pmatrix}
=
\begin{pmatrix}
1-\mu & 0 & 0 \\
0 & 1 & 0 \\
0 & 0 & 1
\end{pmatrix}
\begin{pmatrix}
u(t) \\
v(t) \\
w(t)
\end{pmatrix}
$$

The stability of the periodic solution of $(S^*, 0, Z^*)$ is determined by the eigenvalues of

$$N = \begin{pmatrix} 1-\mu & 0 & 0 \\ 0 & 1 & 0 \\ 0 & 0 & 1 \end{pmatrix} \phi(t)$$

where, $\phi(t) = \phi(0)\exp(\int_0^t A(s)ds) \approx \phi(0)\exp(\tilde{A})$, $\tilde{A} = \int_0^t A(s)ds$ and

$$\phi(0) = \begin{pmatrix} 1-\mu & 0 & 0 \\ 0 & 1 & 0 \\ 0 & 0 & 1 \end{pmatrix}$$

If $\lambda_1, \lambda_2, \lambda_3$ are characteristic roots then $\lambda_1 = (1-\mu)e^{-bT} < 1$, $\lambda_2 = e^{\int_0^t (\beta(Z^*(t) - e^{-b\tau}S^*(t)) - b)d} < 1$, $\lambda_3 = e^{-aT} < 1$.

According to Floquet's theory [18] of impulsive differential equation the solution $(S^*(t), 0, Z^*(t))$ is locally stable when $\mod \lambda_2 < 1$.

Denote

$$R = \frac{\beta(1-e^{-b\tau})[T - \frac{\mu(1-e^{-bT})}{b(1-(1-\mu)e^{-bT})}]}{bT}$$

For $R < 1$, we have

$e^{\int_0^t (\beta(Z^*(t) - e^{-b\tau}S^*(t)) - b)d} < 0$ which leads to $\lambda_2 < 1$. Thus, $(S^*(t), 0, Z^*(t))$ is locally stable. □

Theorem 2.5 If $(S(t), I(t), Z(t))$ is any solution of (3), then if $R < 1$, the infection free periodic solution $(S^*(t), 0, Z^*(t))$ is globally asymptotically stable. Here, $R = \frac{\beta(1-e^{-b\tau})[T - \frac{\mu(1-e^{-bT})}{b(1-(1-\mu)e^{-bT})}]}{bT}$.

Proof. Since, $R < 1$, we have

$$\beta(1-e^{-b\tau})[T - \frac{\mu(1-e^{-bT})}{b(1-(1-\mu)e^{-bT})}] - bT < 0.$$

we choose an $\varepsilon > 0$ small enough such that, that $\psi = \beta(1-e^{-b\tau})[T(1+2\varepsilon) - \frac{\mu(1-e^{-bT})}{b(1-(1-\mu)e^{-bT})}] - bT < 0$

Note that, $\dot{S}(t) \leq b(1 - S(t))$, then by impulsive differential inequalities we have, $S(t) < S^*(t) + \varepsilon$ for all t large enough.

Hence, from third equation of (3) we have,

(11)
$$
\begin{cases}
\dot{Z}(t) \leq a(S^*(t) + \varepsilon - Z(t)), \ t \neq nT \\
\\
Z(t^+) = Z(t), \ t = nT
\end{cases}
$$

Again by impulsive differential inequalities we have,

$$Z(t) < Q^*(t) + \varepsilon,$$

Where $Q^*(t)$ is the periodic solution of the following

(12)
$$
\begin{cases}
\dot{Q}(t) = a(S^*(t) + \varepsilon - Q(t)), \ t \neq nT, \\
\\
Q(t^+) = Q(t), \ t = nT.
\end{cases}
$$

$$
Q^*(t) = 1 + a\mu \frac{e^{-a(t-nT)}(1 - e^{-bT}) - e^{-b(t-nT)}(1 - e^{-aT})}{(a-b)(1 - e^{-aT})(1 - (1-\mu)e^{-bT})} + \varepsilon, nT < t \leq (n+1)T.
$$

Again from second and fifth equation of (3), we have,

(13)
$$
\begin{cases}
\dot{I}(t) \leq \beta I(t)(Q^*(t) - e^{-b\tau}S^*(t) + \varepsilon) - bI(t), \ t \neq nT, \\
\\
I(t^+) = I(t), \ t = nT.
\end{cases}
$$

thus,

$$
I((n+1)T) \leq I(nT^+)exp\left(\int_{nT}^{(n+1)T} (\beta(Q^*(t) - e^{-b\tau}S^*(t) + \varepsilon) - b)dt\right)
$$

$$
= I(nT)exp\left(\int_{nT}^{(n+1)T} (\beta(Q^*(t) - e^{-b\tau}S^*(t) + \varepsilon) - b)dt\right)
$$

$$
= I(nT)e^{\psi}
$$

Hence $I(nT) \leq I(0)e^{n\psi}$ and $I(nT) \to 0$ as $n \to \infty$. Therefore, $I(t) \to 0$ as $t \to \infty$, since $I(t) \leq I(nT)$ for $nT \leq t \leq (n+1)T$.

So, $I(t) \to 0$ as $n \to \infty$.

Now, we show that $S(t)$ approaches $S^*(t)$, $Z(t)$ approaches $Z^*(t)$ as $t \to \infty$. For all $\varepsilon > 0$, there must exist a $T_a > 0$ such that $0 \leq I(t) < \varepsilon$ for $t > T_a$. Without loss of generality, we assume that $0 \leq I(t) < \varepsilon \; \forall \; t \geq 0$, then from (3) we have $b(1 - S(t)) - \beta\varepsilon \leq \dot{S}(t) \leq b(1 - S(t))$. We can also rewrite this as $x(t) \leq \dot{S}(t) \leq S^*(t) + \varepsilon_1$. and $x(t) \to x^*(t)$ as $t \to \infty$, where $x(t)$ is the solution of

(14)
$$
\begin{cases}
\dot{x}(t) = b(1 - x(t)) - \beta\varepsilon, \; t \neq nT, \\[2mm]
x(t^+) = (1 - \mu)x(t), \; t = nT, \\[2mm]
x(0^+) = S(0^+), \\[2mm]
x^*(t) = \frac{(b - \beta\varepsilon)}{b}[1 - \frac{\mu e^{-b(t-nT)}}{1 - (1-\mu)e^{-bT}}], \; nT < t \leq (n+1)T.
\end{cases}
$$

Thus, for any $\varepsilon_1 > 0$, $\exists$ a $T_b > 0$ s.that $x^*(t) - \varepsilon_1 < S(t) < S^*(t) + \varepsilon_1$. Let $\varepsilon \to 0$; we have $S^*(t) - \varepsilon_1 < S(t) < S^*(t) + \varepsilon_1$ for large t. Hence, $S(t) \to S^*(t)$ as $t \to \infty$.

Similarly, we can prove $Z(t) \to Z^*(t)$ as $t \to \infty$. This completes the proof. $\qquad\square$

2.3. Permanence. In this section, we will settled permanence of the system (3).

Theorem 2.6 The system (3) is permanent if $R > 1$, i.e $\exists \; m_1, \; m_2, \; m_3$ such that $S(t) \geq m_1$, $I(t) \geq m_2$, $Z(t) \geq m_3$ for t large enough.

Proof. We start by assuming $(S(t), I(t), Z(t))$ to be any solution of (2.2). The second equation of (3) can be rewritten as:

$$
\dot{I}(t) = [\beta(Z(t) - e^{-b\tau}S(t)) - b]I(t) + \beta e^{-b\tau}\frac{d}{dt}\int_{t-\tau}^{t} S(\theta)I(\theta)d\theta.
$$

Define,

(15)
$$
K(t) = I(t) - \beta e^{-b\tau}\frac{d}{dt}\int_{t-\tau}^{t} S(\theta)I(\theta)d\theta,
$$

Where,

$$\dot{K}(t) = b[\frac{\beta(Z(t) - e^{-b\tau}S(t))}{b} - 1]I(t) \tag{16}$$

We set,

$$m^* = [t - \frac{\mu(1 - e^{-bT})}{b(1 - (1 - \mu)e^{-bT})}] - \frac{bT}{\beta(1 - e^{-b\tau})}$$

$m^* > 0$ follows from the fact that $R > 1$, thus there exists $\varepsilon_1 > 0$ such that

$$\frac{\beta(\xi - e^{-b\tau}\rho)}{b} > 1$$

Where,

$$\rho = 1 - \frac{\mu}{1 - (1 - \mu)e^{-bT}} + \varepsilon_1$$

$$\xi = \frac{b - m^*\beta}{b} + \frac{b - m^*\beta}{b}[a\mu \frac{e^{-aT}(1 - e^{-bT}) - e^{-bT}(1 - e^{-aT})}{(a - b)(1 - e^{-aT})(1 - (1 - \mu)e^{-bT})}] - \varepsilon_1$$

CLAIM: $I(t) < m^*$ cannot hold for $t \geq t_0$, for any positive constant t_0. Proof is by contradiction .i.e. let there exists a t_0 such that $I(t) < m^*$ for $t \geq t_0$. Considering the first equation of (3)

$$\begin{cases} \dot{S}(t) \geq b - m^*\beta - bS(t), \ t \neq nT \\ S(t^+) = (1 - \mu)S(t), \ t = nT. \end{cases} \tag{17}$$

$\therefore S(t) \geq g(t)$ and $g(t) \to g^*(t), t \to \infty$, where $g(t)$ is solution of

$$\begin{cases} \dot{g}(t) = b - m^*\beta - bg(t), \ t \neq nT \\ g(t^+) = g(t), \ t = nT \\ S(0^+) = g(0^+). \end{cases} \tag{18}$$

where, $g^*(t) = \frac{b - m^*\beta}{b}[1 - \frac{\mu e^{-b(t - nT)}}{1 - (1 - \mu)e^{-bT}}], \ nT < t \leq (n + 1)T$.

which is a unique globally asymptotically positive periodic solution.

Therefore, $\exists\, T_a \geq t_0 + \tau$, for $t \geq T_a$ such that,

(19)
$$S(t) \geq g^*(t) - \varepsilon_1 \geq g^*(0^+) - \varepsilon_1$$

where,

(20)
$$g^*(0^+) - \varepsilon_1 = \frac{b - m^*\beta}{b}\left[1 - \frac{\mu e^{-bT}}{1 - (1-\mu)e^{-bT}}\right] - \varepsilon_1 = \eta$$

$\therefore$ we have,

(21)
$$S(t) > \eta.$$

Now, from (19) and third equation of (3) we have,

(22)
$$\begin{cases} \dot{Z}(t) \geq a(g^*(t) - \varepsilon_1 - Z(t)), \ t \neq nT \\ Z(t^+) = Z(t), \ t = nT \end{cases}$$

and $Z(t) > h^*(t) - \varepsilon_1$, here

$$h^*(t) = \frac{b - m^*\beta}{b} + \frac{b - m^*\beta}{b}\left[a\mu\frac{e^{-a(t-nT)}(1 - e^{-bT}) - e^{-b(t-nT)}(1 - e^{-aT})}{(a-b)(1 - e^{-aT})(1 - (1-\mu)e^{-bT})}\right] - \varepsilon_1$$

Also,

(23)
$$Z(t) \geq h^*(t) - \varepsilon_1 \geq h^*(0^+) - \varepsilon_1,$$

and

$$h^*(0^+) - \varepsilon_1 = \frac{b - m^*\beta}{b} + \frac{b - m^*\beta}{b}\left[a\mu\frac{e^{-aT}(1 - e^{-bT}) - e^{-bT}(1 - e^{-aT})}{(a-b)(1 - e^{-aT})(1 - (1-\mu)e^{-bT})}\right] - \varepsilon_1 = \xi$$

thus,

(24)
$$Z(t) > \xi$$

Again from first equation of (3), we have

(25)
$$\begin{cases} \dot{S}(t) \le b(1 - S(t)), \ t \ne nT \\ S(t^+) = (1 - \mu)S(t), \ t = nT \end{cases}$$

So $S(t) \le l(t)$ where,

(26)
$$\begin{cases} \dot{l}(t) = b(1 - l(t)), \ t \ne nT, \\ l(t^+) = (1 - \mu)l(t), \ t = nT \\ S(0^+) = l(0^+). \end{cases}$$

then by lemma (2.2),

(27)
$$S(t) \le l^*(t) - \varepsilon_1 \le l^*(0^+) + \varepsilon_1$$

where,

(28)
$$S(t) < l^*(0^+) + \varepsilon_1 = 1 - \frac{\mu}{1 - (1 - \mu)e^{-bT}} + \varepsilon_1 = \rho$$

From (16), (24) and (28), we have,

(29)
$$\dot{I}(t) > b[\frac{\beta(\xi - e^{-b\tau}\rho)}{b} - 1]I(t), t \ge T_a$$

Suppose

(30)
$$I^m = \min_{t \in [T_a, T_a + \tau]} I(t).$$

CLAIM : $I(t) \ge I^m \ \forall \ t \ge T_a$.

If not then $\exists \ T_b > 0$ such that $I(t) \ge I^m$ for $t \in [T_a, T_a + \tau + T_b]$ and $I(T_a + \tau + T_b) = I^m$. Now from (29) we have,

$$I(T_a + \tau + T_b) > \frac{\beta(\xi - e^{-b\tau}\rho)}{b}I^m > I^m,$$

hence, a contradiction. Thus we get $I(t) \ge I^m > 0 \ \forall \ t \ge T_a$. From (29), we have

$$\dot{I}(t) > b[\frac{\beta(\xi - e^{-b\tau}\rho)}{b} - 1]I^m > 0.$$

then $\exists\, T_a^* \geq T_a$ s.that $t(t) > 0\; \forall\, t \geq T_a^*$. By (29) and the definition of $K(t)$, we obtain for $t \geq T_a^*$,

$$\dot{K}(t) > b[\frac{\beta(\xi - e^{-b\tau}\rho)}{b} - 1]K(t),$$

therefore, $K(t) \to \infty$ as $t \to \infty$, hence a contradiction. Therefore, for any $t_0 > 0$, $I(t) < m^*$ cannot hold for $t \geq t_0$.

If $I(t) \geq m^*\; \forall\, t$ very large, we are done. Otherwise it is oscillatory about m^* .i.e. there exist constants $\hat{t}$, α such that

$$I(\hat{t}) = I(\hat{t} + \alpha) = m^*$$

and

$$I(t) < m^*, \hat{t} < t \leq \hat{t} + \alpha.$$

$S(t) > \eta$ holds true for $\hat{t}$ large enough and also for $\hat{t} < t \leq \hat{t} + \alpha$. the positive solutions of (3) are bounded and $I(t)$ not being affected by impulses, $I(t)$ is uniformly continuous.

$\therefore \exists$ a constant T_c independent of $\hat{t}$ and lying in $(0, \tau)$ such that $I(t) > \frac{m^*}{2}\; \forall\, \hat{t} < t \leq \hat{t} + T_c$. If $\alpha \leq T_c$, we are done. If $T_c < \alpha \leq \tau$, for $\hat{t} + T_c < t \leq \hat{t} + \alpha$,

$$I(t) \geq \beta \int_{t-\tau}^{t} (Z(\theta) + e^{-b(t-\theta)}S(\theta))d\theta$$

$$\geq \beta \int_{\hat{t}}^{\hat{t}+T_c} (Z(\theta) + e^{-b(t-\theta)}S(\theta))d\theta$$

$$> \frac{\beta(\xi + e^{-b\tau}\eta)T_c m^*}{2} = m_1^*.$$

Set

$$m_1 = \min\{\frac{m^*}{2}, m_1^*\}$$

So, $I(t) > m_1\; \forall\, \hat{t} < t \leq \hat{t} + \alpha$. If $\alpha > \tau$, we can again have $I(t) > m_1$ for $\hat{t} < t \leq \hat{t} + \tau$. then, following same approach as above, we can have $I(t) > m_1$ for $\hat{t} + \tau < t \leq \hat{t} + \alpha$. $\because$ this type of

interval $[\hat{t}, \hat{t} + \alpha]$ is selected in a way which is arbitrary when $\hat{t}$ is large enough, we can conclude

that $I(t) > m_1$ for large t.

From first and fourth equation of (3) we have,

(31)
$$\begin{cases} \dot{S(t)}l \geq b - bS(t) - \beta, \; t \neq nT \\ S(t^+) = (1 - \mu)S(t), \; t = nT \end{cases}$$

So, $S(t) \geq g_1(t)$ where,

(32)
$$\begin{cases} \dot{g_1(t)} = b - bg_1(t) - \beta, \; t \neq nT, \\ g_1(t^+) = (1 - \mu)g_1(t), \; t = nT \\ S(0^+) = g_1(0^+). \end{cases}$$

Suppose $g_1^*(t)$ is unique globally attractive positive solution of (32), then for $\varepsilon > 0$ and t large

enough $S(t) \geq g_1^*(t) - \varepsilon_1 \geq g_1^*(0^+) - \varepsilon = m_2 > 0$.

From third and sixth equation of (3), we have

(33)
$$\begin{cases} \dot{Z(t)} > a(m_2 - Z(t)), \; t \neq nT \\ Z(t^+) = Z(t), \; t = nT \end{cases}$$

Consider

(34)
$$\begin{cases} \dot{h_1(t)} = a(m_2 - h_1(t)), \; t \neq nT \\ h_1(t^+) = h_1(t), \; t = nT \end{cases}$$

Clearly, $h_1(t) \to m_2$ as $t \to \infty$, $\exists \, \varepsilon > 0$ such that

$$Z(t) \to m_2 - \varepsilon = m_3 > 0.$$

$\square$

3. SIR MODEL WITH CONSTANT VACCINATION STRATEGY

The system (3), in case of constant vaccination with constant infectious rate under the assumption that death and birth rate are same can be written as:

(35)
$$\begin{cases} \dot{S}(t) = -\beta I(t)Z(t) - bS(t) + b - \mu S(t) \\ \dot{I}(t) = \beta I(t)Z(t) - \beta e^{-b\tau}S(t-\tau)I(t-\tau) - bI(t) \\ \dot{Z}(t) = a(S(t) - Z(t)) \end{cases}$$

3.1. Existence of equilibrium points.
The system has two equilibrium points: disease free and endemic equilibrium.

- The disease free equilibrium $E_0(\frac{b}{(b+\mu)}, 0, \frac{b}{(b+\mu)})$.

- Endemic equilibrium $E^*(\frac{b}{(b+\mu)R_0}, \frac{b+\mu}{\beta}(R_0 - 1), \frac{b}{(b+\mu)R_0})$, exist when $R_0 > 1$. where, $R_0 = \frac{\beta(1-e^{-b\tau})}{b+\mu}$.

3.2. Local Stability of equilibrium points.

- The jacobian of system (35) at E_0 is

$$J(E_0) = \begin{pmatrix} -b-\mu & -\beta & 0 \\ 0 & \beta - e^{-b\tau} - b & 0 \\ a & 0 & -a \end{pmatrix}$$

E_0 is locally asymptotically stable if $\beta - \beta e^{-b\tau} - b - \mu < 0$, i.e. $\frac{\beta(1-e^{-b\tau})}{b+\mu} < 1$ or stable if $R_0 < 1$ and unstable if $R_0 > 1$.

Where, $R_0 = \frac{\beta(1-e^{-b\tau})}{b+\mu}$ is the basic reproduction number.

- Jacobian of the system (35) at $E^*(\frac{b}{(b+\mu)R_0}, \frac{1}{R_0}(R_0 - 1), \frac{b}{(b+\mu)R_0})$ is

$$J(E^*) = \begin{pmatrix} -b-\mu-\lambda & -\beta Z^* & -\beta I^* \\ -\beta e^{-b\tau}I^* & \beta Z^* - e^{-b\tau}S^* - b - \lambda & \beta I^* \\ a & 0 & -a-\lambda \end{pmatrix}$$

The characteristic equation is $\lambda^3 + a_0\lambda^2 + a_1\lambda + a_2 = 0$.

where, $a_0 = a + 2b + \mu - \beta z^* + \beta e^{-b\tau}S^* + a = a + b + \mu > 0$,

$a_1 = 2ab + \mu(a+b) + b^2 + a\beta I^* + \beta^2 e^{-b\tau}S^* + \beta^2 e^{-b\tau}\mu S^* - \beta^2 e^{-b\tau}I^*Z^* - a\beta S^* - b\beta S^* - \mu\beta S^*$

$= ab + a\mu + \frac{\beta(R_0-1)}{R_0}[a - (a+b)e^{-b\tau}] > 0$, if $R_0 > 1$ and $a > (a+b)e^{-b\tau}$

$a_2 = +a\mu b + ab\beta I^* + ab\beta e^{-b\tau}S^* + ab^2 + a\beta e^{-b\tau}\mu S^* - ab\beta Z^* - a\mu\beta Z^*$

$= ab(b+\mu)(R_0 - 1) > 0$, if $R_0 > 1$.

3.3. Global Stability of equilibrium points.

In this section, by using Lyapunov function global stability is established.

Theorem 3.1 If $R_0 \leq 1$, then the disease free equilibrium point of the system is globally asymptotically stable on ω.

Proof. For disease-free equilibrium points Let us consider the Lyapunov function $V : \omega \to R, V(S,I) = I(t)$.

$\frac{dV}{dt} = \frac{dI}{dt}$

$\frac{dV}{dt} = \beta IZ - \beta e^{-b\tau}SI - bI$

From the third equation of the system (35), $Z(t) = S(t)$

$\frac{dV}{dt} = bI[\frac{\beta(1-e^{-b\tau})}{b}S - 1] = bI(\frac{R_0(b+\mu)S}{b} - 1) = bI(R_0 - 1)$

$\frac{dV}{dt} < 0$, if $R_0 < 1$.

Hence, if $R_0 < 1$ then $\frac{dV}{dt} < 0 \leftrightarrow I(t) = 0$ and if $R_0 = 1$ then $\frac{dV}{dt} = 0 \leftrightarrow S(t) = 1$.

Hence by Lassale invariance principle, the disease free equilibrium is globally asymptotically stable on ω. □

Theorem 3.2 The endemic equilibrium point $E^*(S^*, I^*, Z^*)$ of the system is globally asymptotically stable on ω_+.

Proof. We construct a Lyapunov function $L : \omega_+ \to R$ where, $\omega_+ = S(t), I(t) \in \omega, S(t) > 0, I(t) > 0$ given by

$L(S,I) = w_1(S - S^*)^2 + w_2[I - I^*ln(\frac{I}{I^*})]$

Where, w_1 and w_2 are positive constants to be chosen later.

Then the time derivative of the Lyapunov function is given by

$\frac{dL}{dt} = 2w_1(S-S^*)\frac{dS}{dt} + w_2\frac{(I-I^*)}{I}\frac{dI}{dt}$

From the third equation of the system (35), $Z^* = S^*$

$\frac{dL}{dt} = -[(2+\mu)w_1(S-S^*)^2 + 2w_1\beta(I-I^*) + 2w_1(\beta I^* + bS^* + \mu S^* - b)(S-S^*) - w_2 R_0(b+\mu)(I-I^*)(S-S^*)]$

Taking, $w_1 = \frac{1}{2}, w_2 = \frac{1}{R_0(b+\mu)}, a_1 = (S-S^*)$ and $a_2 = (I-I^*)$

$\frac{dL}{dt} = -[(a_1-a_2)^2 + (\sqrt{\mu}a_1+a_2)(\sqrt{\mu}a_1-a_2) + ba_1(S^*-1) + \beta z_2 + \beta I^* a_1 + \mu a_1 S^*]$ Thus,

$\frac{dL}{dt} < 0$ in ω_+, if $S^* > 1$ and $\sqrt{\mu}a_1 > a_2$, also $\frac{dL}{dt} = 0$ at $S = S^*$.

Hence by Lassale invariance principle, endemic equilibrium is globally asymptotically stable on ω_+. $\qquad\square$

4. COMPARISON BETWEEN THE REPRODUCTION NUMBERS

Reproduction number in pulse vaccination is least and reproduction number. of without vaccination strategy is maximum which can be visible from the following: In the case of without vaccination, reproduction number. is $R_1 = \frac{\beta(1-e^{-b\tau})}{b}$.

In case of constant vaccination, reproduction number is $R_0 = \frac{\beta(1-e^{-b\tau})}{b+\mu} = \frac{bR_1}{b+\mu} < R_1$.

In case of pulse vaccination, reproduction no. is $R = \frac{\beta(1-e^{-b\tau})[T-\frac{\mu(1-e^{-bT})}{b(1-(1-\mu)e^{-bT})}]}{bT}$

$= R_1 - \frac{\mu\beta(1-e^{-bT})(1-e^{-b\tau})}{b^2T(1-(1-\mu)e^{-bT})} = \frac{(b+\mu)R_0}{b} - \frac{\mu\beta(1-e^{-bT})(1-e^{-b\tau})}{b^2T(1-(1-\mu)e^{-bT})}$.

So, we get $R < R_0 < R_1$. Thus, we have proved it analytically that the reproduction number in the case of pulse vaccination would give less number of infective than constant vaccination.

5. NUMERICAL SIMULATION

In this section, we have plotted three sets of figures by taking Recruitment rate of susceptible class (b) and contact rate (β), same for all to verify our results. We consider the hypothetical set of parameters.

Case I: Without Vaccination

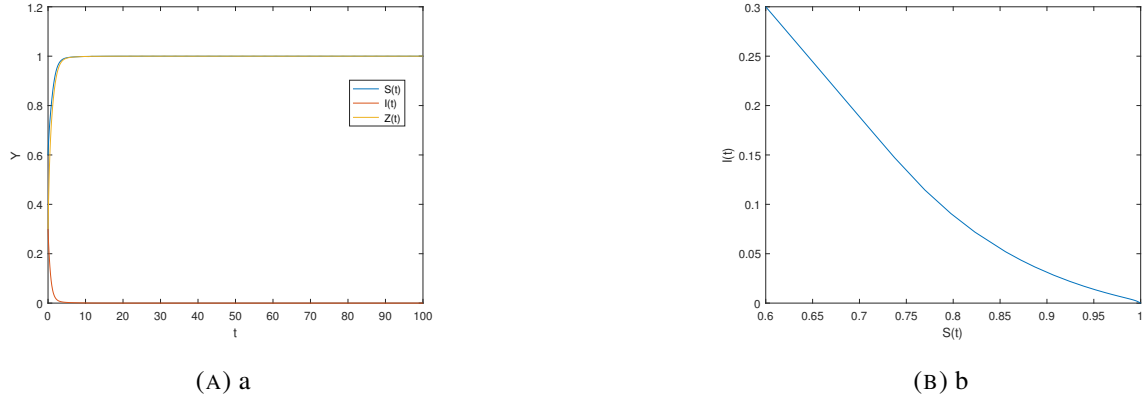We obtained disease free system for the following set of parameters $b = 1.7$, $\beta = 2.8$, $a =$

(A) a

(B) b

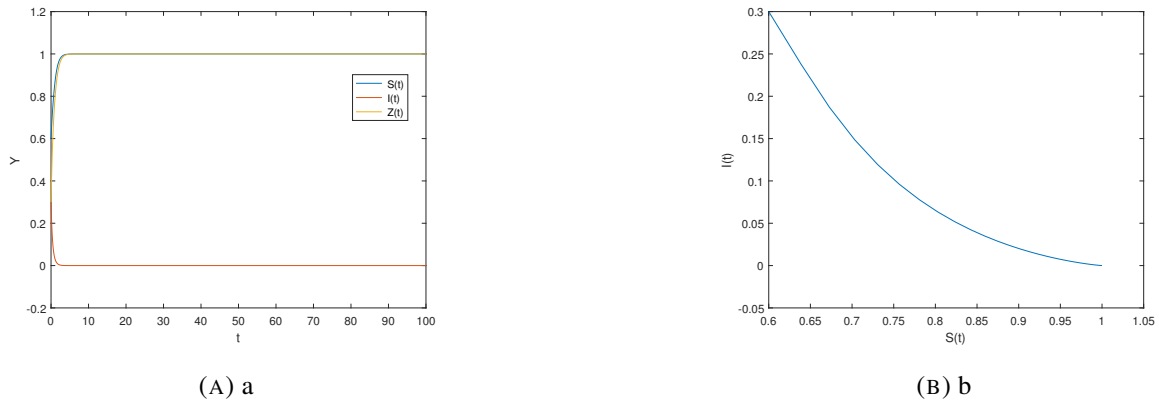FIGURE 2. Behaviour of disease free system without vaccination at $\tau = 0.5$

(A) a

(B) b

FIGURE 3. Behaviour of disease free system without vaccination at $b = 1.7, \beta = 2.8, a = 0.3, \tau = 0.1$, gives $R_1 = 0.25$

3.0, $\tau = 0.5$ and $(S_0, I_0, Z_0) = (0.6; 0.3; 0.3)$ (Figure 2a) . Further, it is stable provided $R_1 = \frac{\beta(1-e^{-b\tau})}{b} = 0.94 < 1$. for the same initial condition, as we increase delay to $\tau = 0.6$, keeping the rest parameters same, we obtained endemic equilibrium point (Figure 3a) with $R_1 = \frac{\beta(1-e^{-b\tau})}{b} = 1.05 > 1$.

Case II: With constant vaccination strategy

As we introduced constant vaccination, we obtained disease free system with the parameters $b = 1.7$, $\beta = 2.8$, $a = 3.0$, $\tau = 0.9$ and $\mu = 0.6$ for same initial condition (Figure 6a)with $R_0 = 0.95 < 1$ but as soon we reduced the rate of vaccination to $\mu = 0.4$, endemicity exists in the

(A) a

(B) b
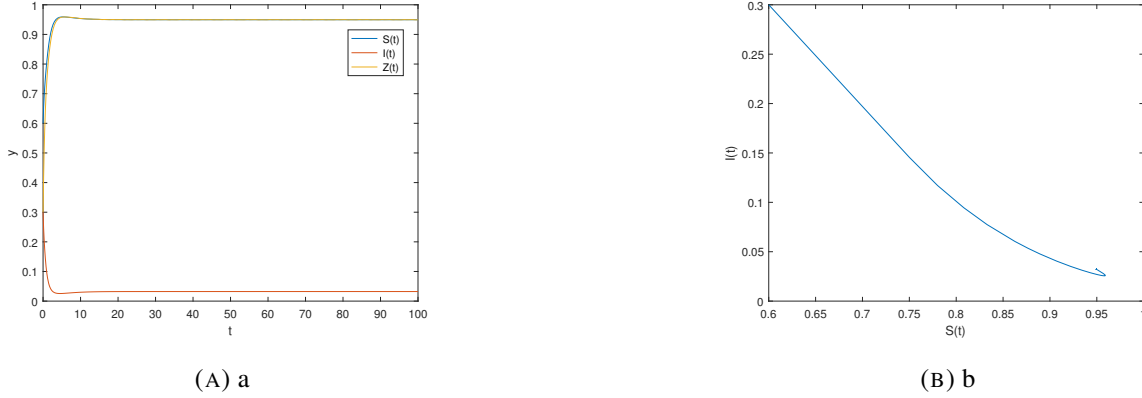
FIGURE 4. Behaviour of the endemic system without vaccination at $\tau = 0.6$

(A) a

(B) b
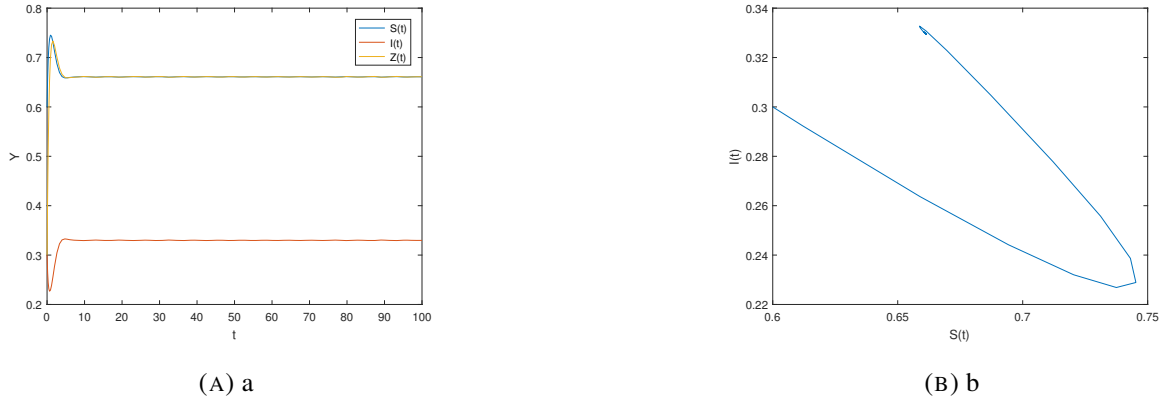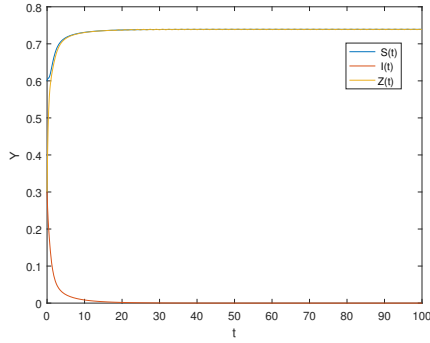
FIGURE 5. Behaviour of the endemic system without vaccination at $b = 1.7$, $\beta = 2.8$, $a = 3.0$ and $\tau = 2.0$, gives $R_1 = 1.59 > 1$.

system (Figure 8a)with $R_0 = \frac{\beta(1-e^{-b\tau})}{b+\mu} = 1.04 > 1$. Interestingly, as we introduced vaccination, for higher value of delay also we are able to control disease and obtain the disease free case.

Case III: With pulse vaccination strategy

As pulse vaccination approach is applied and proportion of susceptible population are vaccinated periodically after equal intervals, we observed that for $b = 1.7$, $\beta = 2.8$, $a = 3$, $\tau = 1.1$, $T = 1$, $\mu = 0.8$ and at same initial conditions, $R = \frac{\beta(1-e^{-b\tau})[T-\frac{\mu(1-e^{-bT})}{b(1-(1-\mu)e^{-bT})}]}{bT} = 0.72 < 1$ for disease free equilibrium and for the same set of parameters and with $\tau = 2$, $T = 1$ and $\mu = 0.2$,
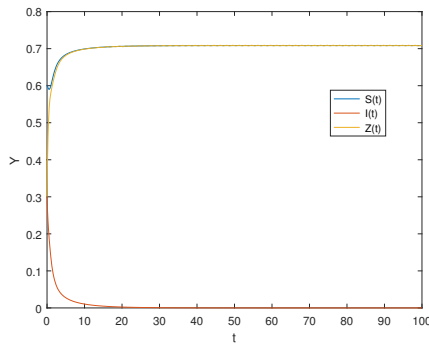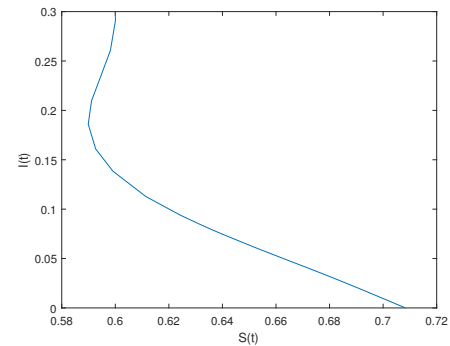
(A) a
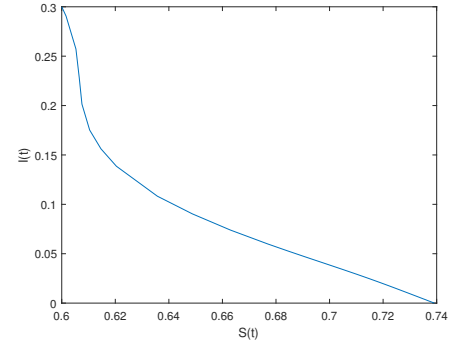
(B) b

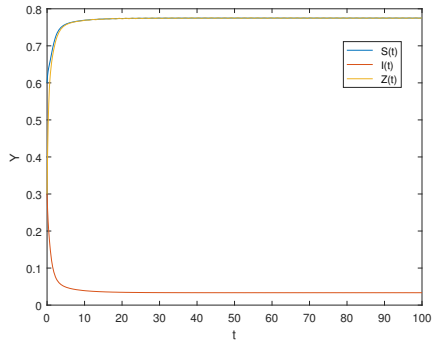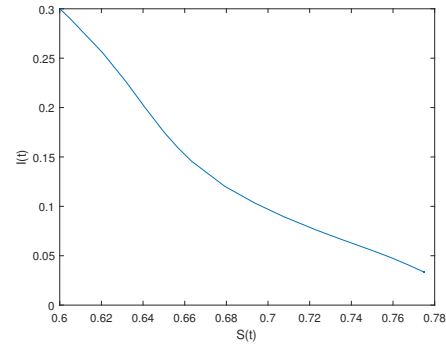FIGURE 6. Behaviour of disease free system with constant vaccination at $\tau = 0.9$

(A) a

(B) b

FIGURE 7. Behaviour of disease free system with constant vaccination at $b = 1.7, \beta = 2.8, a = 3, \mu = 0.8, \tau = 1.1$, gives $R_0 = 0.94 < 1$

we obtained $R = 1.1 > 1$ which confirms the endemicity of the system. This results conclude that although for increased delay also we are able to achieve the disease free state, but the vaccination rate has to be increased and the periodic interval of vaccination should be less because as soon we increase this interval, it would be difficult to control the infection. This result is very much in lines with all the presumed vaccinations in progress across world for corona virus till now.
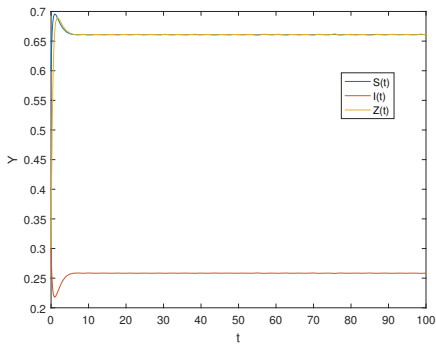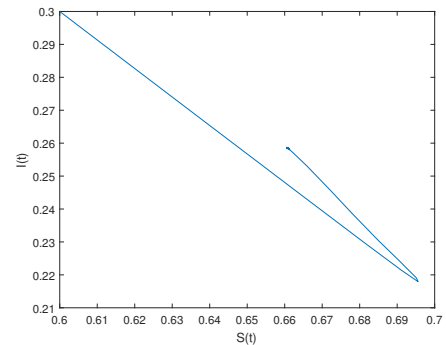
(A) a   (B) b

FIGURE 8. Behaviour of endemic system with constant vaccination at $\tau = 0.9$
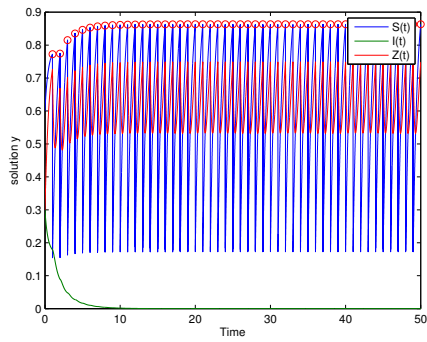

(A) a   (B) b

FIGURE 9. Behaviour of endemic system with constant vaccination at $b = 1.7$, $\beta = 2.8$, $\tau = 2.0$, $a = 3.0$, $\mu = 0.2$, gives $R_0 = 1.36 > 1$

.

TABLE 1. Without Vaccination

τ	0.1	0.5	0.6	1.1	2.2
R_1	0.25	0.94	1.05	1.39	1.59

TABLE 2. With Constant Vaccination

τ	0.9	0.9	1.1	0.9	2.0
μ	0.6	0.8	0.8	0.4	0.2
R_0	0.95	0.87	0.94	1.04	1.36

(A) a
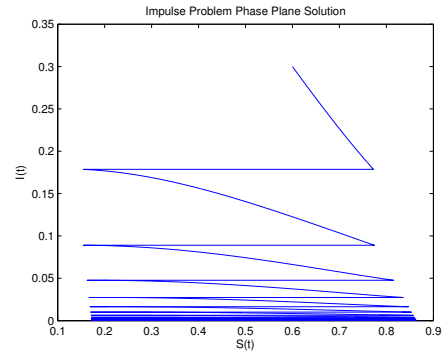
(B) b

FIGURE 10. Behaviour of the disease free system with PVS
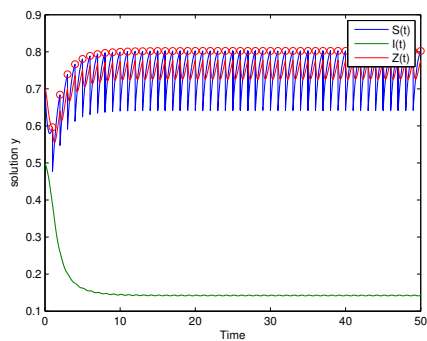
(A) a
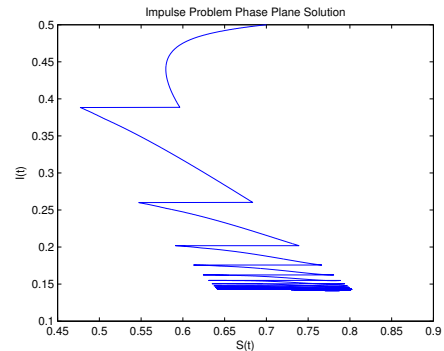
(B) b

FIGURE 11. Behaviour of the endemic equilibrium system with pvs

TABLE 3. With Pulse Vaccination at T = 1

τ	0.9	0.9	1.1	2.0
μ	0.6	0.8	0.8	0.2
R	0.88	0.15	0.72	1.1

TABLE 4. With Pulse Vaccination at T = 2

τ	0.9	0.9	1.1	2.0
μ	0.6	0.8	0.8	0.2
R	1.06	1.54	1.07	1.49

5.1. Result analysis.

- it is seen that low vaccination rate and large infection span is accountable for infection permanence in both constant and pulse (Figure 6a, 7a, 8a, 9a, 10a, 11a).

- Comparative study of Case I and Case II : From Table 1, it is concluded that disease free situation can be obtained at $\tau \le 0.5$ when no vaccination is given to an individual which means that if the infectious period is less, only then we can control the infection without vaccination. From Table 2, we observed that, we get a disease free situation at $\tau \le 0.6$. But, in this case vaccination dosage has also equal importance like τ. Further, even if we increase $\tau = 0.9$ which is higher than the case when no vaccination is applied, we can still manage to obtain a disease free state due to vaccination. However, if we decrease the dosage than it would be difficult to control.

- Comparative study of Case II and Case III : From Table 2 and Table 3, we have noticed that disease free state is attained at a low vaccination dosage ($\mu = 0.6$) in both the case of constant and pulse vaccination but the reproduction number is comparatively less in pulse vaccination and if at the same delay(0.9), μ is increased, reproduction number. becomes 83% less while in constant, it is only 8%. It is also seen that on same set of parametric values, in case of disease free case, number of susceptible are more in the case of pulse vaccination (Figure 7a, 10a) and furthermore in endemic state, susceptible individuals are more and infective individuals are less in pulse vaccination as contrast with constant vaccination (Figure 9a, 10a).

- From Table 2, Table 3 and Table 4, we have also observed that, when pulse interval is less ($T = 1$), than the pulse vaccination is performing better but if the interval is large due to any reason like production etc, it would be hard to control infection.

6. Conclusion

In this paper, we have analyzed the effect of time-delay with constant vaccination as well as pulse vaccination strategy on a corona-epidemic model and obtained the conditions for controlling as well as permanence of the infection. Our model has an upper edge over the reviewed models as, it inculcates both the delays i.e discrete as well as distributive and their effects on the model. Central idea behind these delays are to lift up the problem to a more pragmatic platform as the distributive delay is used to showcase the dependency of infectious class on the full history of susceptible. The local and global stability of disease free and endemic equilibrium point has been discussed for both the cases and also numerically validated with an example. Our analysis shows that reduction in vaccination dosage and increment in delay τ, result infection persistence in the environment in both the cases : pulse as well as constant vaccination strategy. Another significant finding is the relation between recovery period/infectious period and the vaccination strategy. A small infectious period requires constant vaccination whereas long period of infection needs pulse vaccination which is also in lines with the current scenarios of patients as susceptible having better immunity can resist the virus and will require a less dosage but people who are already suffering with other diseases like diabetes, cancer, flu etc, pulse vaccination strategy is a better approach. To further explain, as we know that the endemic SARS (Severe Acute Respiratory Syndrome) happened in China in 2003, MERS (Middle East Respiratory Syndrome) happened in Saudi Arebia in 2012 and pandemic COVID-19, all these infectious disease belong to same virus i.e. corona virus. So, vaccination is the only technique to fight against this infection. Under the states of constant vaccination, it would be helpful for people with strong immunity but as by recent evidences [9] mutation can revive the virus to overcome corona promoting stress, if we go towards pulse vaccination strategy by applying some boosters, chances of infection would be controlled and also this strategy would be cost effective and easy to manage as compare to constant vaccination, if pulse interval is small. Since this crown infection is extremely infectious and has immediately spread all around and furthermore cross species hindrance. Hence present scenario of COVID-19 and the research has been done till now shows, the helpful methodology to manage this pandemic is pvs, enforced at regular and short interval to control the contamination. Numerically, we also attained the

interval of pulsing $(T = 1)$, which is fundamental for applying immunization system effectively and suggests that pvs would be simpler to be control this pandemic.

This paper may help the government to make new policies to control this dangerous infectious sickness around the globe.

CONFLICT OF INTERESTS

The author(s) declare that there is no conflict of interests.

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