A Susceptible-Infected-Vaccinated Model for Influenza Infection Dynamics

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Abstract

A ongoing challenge in public health is our inability to reliably forecast the timing and intensity of seasonal Influenza. Current models for infectious diseases like SIS (susceptible-infected-susceptible) and SIR (susceptible-infected-recovered) models inadequately account for the seasonal dynamics of Influenza and the time-limited effectiveness of vaccination. In this paper, we propose an SIV (susceptible-infected-vaccinated) model which takes into account both the seasonal behavior of Influenza outbreaks as well as the time-limited effectiveness of vaccination. Additionally, we use relevant clinical and epidemiological data to inform the choice of model parameters. Given sufficiently informed parameters, the SIV model may provide useful insight towards the behavior of influenza outbreaks.

1 Introduction

Influenza, commonly known as the flu, is a contagious respiratory illness caused by influenza viruses. It presents a significant public health concern globally due to its high transmissibility and potential to cause severe illness and death, particularly among vulnerable populations. Influenza viruses are categorized into four main types: A, B, C, and D, with types A and B being the most common and responsible for seasonal epidemics. Type A influenza viruses are further classified into subtypes based on the combinations of hemagglutinin (HA) and neuraminidase (NA) proteins on their surfaces, such as H1N1 and H3N2, which have been associated with major pandemics and seasonal outbreaks [1, 2].

The history of influenza is marked by several devastating pandemics. The 1918 Spanish flu, caused by the H1N1 virus, resulted in an estimated 50 million deaths worldwide, including significant mortality in India . Subsequent pandemics, such as the 1957 Asian flu (H2N2), the 1968 Hong Kong flu (H3N2), and the 2009 H1N1 pandemic, have also led to substantial global health and economic impacts [1]. In India, the 2009 H1N1 pandemic saw over 34,000 confirmed cases and more than 1,600 deaths by July 2010, illustrating the severe impact of influenza on the country [3–6].

Seasonal influenza epidemics occur annually and are influenced by various factors, including climatic conditions, population density, and vaccination coverage. In temperate regions, influenza activity typically peaks during the winter months, while in tropical regions, influenza can circulate year-round with peaks often coinciding with the monsoon season [7–11].

In India, usually there are two peak seasons, one is in January to March, other is during the post-monsoon season August to October [12].

India faces challenges in managing influenza outbreaks due to its large and diverse population, varying healthcare infrastructure, and differences in regional health policies. Continuous surveillance and timely reporting are crucial for influenza management. The Indian government, through agencies like the Ministry of Health and Family Welfare (MoHFW) and the Indian Council of Medical Research (ICMR) monitor influenza activity and guide public health interventions.

Vaccination remains the primary tool for preventing influenza and reducing its impact. In India, influenza vaccination coverage is relatively low compared to developed countries, posing a challenge to controlling the disease's spread. By understanding the virus's dynamics, historical context, and regional variations, public health authorities and government can better prepare for and respond to influenza outbreaks, ultimately reducing morbidity and mortality associated with it.

2 Mathematical Model

We aim to expand upon existing epidemiological models for infection dynamics. One such model, the SIR model, separates a population into three disjoint sets, compartments, **Susceptible**, **Infected**, and **Recovered** [13]. The SIR

model is given by the following system of differential equations:

$$\begin{aligned} \frac{dS}{dt} &= -\alpha IS \\ \frac{dI}{dt} &= \alpha IS - \beta I \\ \frac{dR}{dt} &= \beta I \end{aligned} \tag{1}$$

where α represents the rate of infection and β represents the rate of recovery. One key assumption is that the total population remains constant. The sum of derivatives, $\frac{dS}{dt} + \frac{dI}{dt} + \frac{dR}{dt} = 0$, implies that the derivative of the sum is also equal to 0. Thus, the total population does not change.

Another key assumption of the SIR model is that once individuals are "Recovered", they have lasting immunity to the disease. While appropriate for certain diseases like chicken pox or measles [14], the SIR model fails to take into account the ability of certain viruses to escape the immune response. An alternative model which addresses the time-limited conferred immunity is the SIS model, where individuals transition between being **Susceptible**, **Infected**, and **Susceptible** again [13–15]. The SIS model is given by the following system of differential equations:

$$\begin{aligned} \frac{dS}{dt} &= -\alpha SI + \beta I \\ \frac{dI}{dt} &= \alpha SI - \beta I \end{aligned} \tag{2}$$

Like the SIR model, the SIS model also assumes that the population remains constant. However, unlike the SIR model, the SIS model takes into account the ability of individuals to be infected multiple times. Notably, the standard SIS model is blind to the passage of time – future behavior is entirely determined by the current state of the system [16]. Due to this property, SIS models are unable to capture the seasonal behavior of certain viral diseases like Influenza, which is well known to follow "flu seasons" [7].

It stands to reason that a model that takes into account both the time-limited immunity conferred by recovery or vaccination, as well as the seasonal behavior of Influenza, may have more predictive power than current SIV and SIS models.

According to standard modeling epidemiological problems, we'll base our SIV model on a system of ordinary differential equations [15, 17]. We propose that the SIV model be given by the following system of differential equations:

$$\begin{aligned} \frac{dS}{dt} &= -\alpha SI + \beta I - \gamma S + \kappa V + r\sin(\frac{2\pi}{12}t) \\ \frac{dI}{dt} &= \alpha SI - \beta I - \gamma I - r\sin(\frac{2\pi}{12}t) \\ \frac{dV}{dt} &= \gamma S + \gamma I - \kappa V \end{aligned} \tag{3}$$

For the model to be mathematically well defined, we'll impose that this system must be equipped with initial conditions $S(0)$, $I(0)$, and $V(0)$. Additionally, we'll require that initial conditions must be positive and non-zero and that the values of $S(t)$, $I(t)$, and $V(t)$ must also remain positive and non-zero for all time. The α parameter represents the rate of infection, the β parameter represents the rate of recovery, the γ parameter represents the rate of vaccination, and the κ parameter represents the rate at which immunity is lost. Lastly, the r coefficients are multiplicative terms for the amplitude of their respective sine functions, which are used to introduce periodic forcing as a simulation of seasonal outbreaks. We use $(\frac{2\pi}{12}t)$ as the argument for the sine functions, as we want the period of the external forcing to be 12 months, since "seasonal" outbreaks are annual [5, 8, 18]. Our model uses months as a unit of time to directly compare with clinical and epidemiological data which has been studied in the papers [5, 7, 18–20].

Note that only the β terms are dependent on the value of I. We can interpret this as: the rate of recovery is proportional to the number of infected people. The"reasonable" value of β is less than 1, since individuals do not immediately recover from Influenza. Similarly, "reasonable" values for α, γ, and κ are all much less than 1. Additionally, it wouldn't make biological sense if any of the rate parameters were negative, so we'll bound the rate parameters to be between 0 and 1.

Notably, to keep the population constant, it must be that the sum of all three differential equations is 0. We denote the total population as $N(t)$, with the condition that $\frac{dN}{dt} = 0$. Since, we've a constant total population, disjoint subsets of the population, and defined rates of transition, we can visualize the SIV model as a "compartmental model", in which S, I, and V refer to disjoint compartments that an individual can be assigned to [21]. In Figure 1, we show the flowchart associated with this "compartmental model", with boxes representing the individual compartments, and arrows labeled with the respective rate of transition between states. A key feature of the system as in Figure 1 is that the system is closed, meaning that **all changes in any of the three compartments are due to an equal and opposite change in another compartment.**

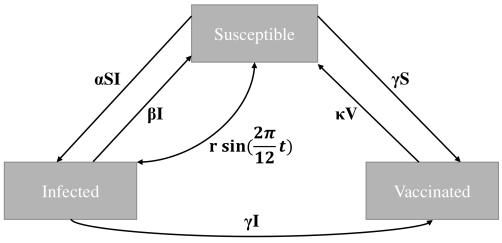

Figure 1: Flowchart of SIV Compartmental model.

3 Assumptions

We make several assumptions in the SIV model. We believe that these assumptions are reasonable due to constraints on time and computational resources.

Firstly, we'll make several biological assumptions about the nature of viral diseases and epidemiology. Similar to the SIV and SIS models, we'll retain the assumption that population remains constant. Additionally, we make the assumption that "Susceptible", "Infected", and "Vaccinated" groups are disjoint, with the transition between these groups treated as instantaneous. More realistically, the immunity provided by vaccination is non-instantaneous, and the susceptibility of vaccinated individuals towards losing their immunity is more aptly described with a time-dependent continuous probability distribution, rather than a population-dependent differential equation [22]. By treating transitions as instantaneous, another inherent assumption we must make is that "Infected" individuals are immediately infectious. In reality, when individuals are infected with a virus, they typically undergo an "**incubation period**" wherein they do not exhibit symptoms of the disease, but are still able to infect others [23].

Furthermore, we make several assumptions about behavior specific to Influenza. Currently, two major strains of Influenza circulate throughout Human populations, H1N1 and H3N2, however, our model will only consider a single strain [8]. Also, due to Influenza's extreme rate and diversity of viral evolution, it has been noted to "jump" between different species, like the 2008-2009 Swine Flu epidemic [24]. Due to this unique ability of Influenza, so-called "genetic shifts" result in more extreme outbreaks than typical seasonal behavior, because it is much less likely for individuals to have existing immunity to novel strains. Our model does not take a genetic shift into account, and we will assume that Influenza will continue to follow current and past seasonal patterns without breakout pandemics.

Lastly, we'll make several assumptions about our simulated population. We'll not take into consideration any concept of locality or regionality when determining the respective rates of lost immunity, infection, and vaccination – rather, we'll assume that the future state of the model is only dependent on time and the current state. Additionally, we'll assume that the rate of vaccination is determined similarly for both infected and susceptible individuals. More realistically, we expect that infected individuals experiencing symptoms are more likely to visit a healthcare centre and thus are more likely to be vaccinated.

These assumptions may limit the effectiveness of our model due to the various factors that are ignored or otherwise not taken into consideration. However, we hope that by referencing existing clinical and epidemiological data, we can select informed parameters that will offset the penalty to model accuracy caused by said assumptions.

4 Solution and Analysis

Due to the presence of sine terms, we are unable to solve this system with the same methods as the SIS and SIR models – the presence of time-dependent external forcing results in the system no longer having fixed points. For the purposes of solving the model (i.e., finding the fixed points), we will ignore the external forcing and solve for the behavior of the system without the sine terms. Thus, we've the following system of equations, which we'll denote as the "non-forced SIV model":

$$\begin{aligned} \frac{dS}{dt} &= -\alpha SI + \beta I - \gamma S + \kappa V \\ \frac{dI}{dt} &= \alpha SI - \beta I - \gamma I \\ \frac{dV}{dt} &= \gamma S + \gamma I - \kappa V \end{aligned} \tag{4}$$

4.1 Trivial Fixed point

A trivial fixed point is to take the initial condition $(S, I, V) = (0, 0, 0)$, which doesn't provide any useful mathematical or biological information. We can find other fixed points by analyzing the system with the Jacobian set to 0. Note that, since we're ignoring the sine-terms, the trivial fixed point is always stable.

4.2 "Disease-Free Equilibrium (DFE)" Fixed point

We can rewrite the system of equations as in 4 as the following matrix:

$$\begin{bmatrix} \frac{dS}{dt} \\ \frac{dI}{dt} \\ \frac{dV}{dt} \end{bmatrix} = \begin{bmatrix} (-\alpha I - \gamma)S + \beta I + \kappa V \\ \alpha SI + (-\beta - \gamma)I + 0 \\ \gamma S + \gamma I - \kappa V \end{bmatrix}$$

Thus, we can take the Jacobian of the non-forced SIV model as follows:

$$J_f = \begin{bmatrix} -\alpha I - \gamma & \beta & \kappa \\ \alpha I & -\beta - \gamma & 0 \\ \gamma & \gamma & -\kappa \end{bmatrix}$$

In mathematical epidemiology, we typically analyze a fixed point at **disease-free equilibrium**, or "DFE" [15,25]. *This fixed point occurs when the initial condition of the system is* $(S, I, V) = (N_1, 0, N_2)$*, with* $N = N_1 + N_2$*, that is, the initial condition where there are no diseased individuals in the population, and thus the entire population is either susceptible or vaccinated.* Note that, aside from the DFE fixed point, there is one globally stable fixed point known as **"endemic equilibrium"**, in which there is equilibrium between all three compartments [26,27]. Unlike the endemic fixed point, the DFE fixed point can be unstable. *When the DFE initial condition is unstable, then the system will globally tend towards endemic equilibrium.*

Since the DFE initial condition suggests that the susceptible and vaccinated compartments are in equilibrium, we can find this fixed point by setting:

$$\begin{aligned} -\alpha S^* I^* + \beta I^* - \gamma S^* + \kappa V^* &= \gamma S^* + \gamma I^* - \kappa V^* \\ I^* &= 0 \\ -2\gamma S^* &= -2\kappa V^* \\ S^* &= \frac{\kappa}{\gamma} V^* \\ S^* + V^* &= N \\ \frac{\kappa}{\gamma} V^* + V^* &= N \\ (1 + \frac{\kappa}{\gamma}) V^* &= N \\ \boxed{V^* = \frac{N}{1 + \frac{\kappa}{\gamma}}} & \\ V^* &= \frac{\gamma}{\kappa} S^* \\ V^* + S^* &= N \\ \frac{\gamma}{\kappa} S^* + S^* &= N \\ (1 + \frac{\gamma}{\kappa}) S^* &= N \\ \boxed{S^* = \frac{N}{1 + \frac{\gamma}{\kappa}}} & \end{aligned}$$

Thus, the DFE fixed point is given by $\boxed{(S^*, I^*, V^*) = (\frac{N}{1 + \frac{\gamma}{\kappa}}, 0, \frac{N}{1 + \frac{\kappa}{\gamma}})}$.

As an example, Figure 2 demonstrates the long-run behavior of the non-forced SIV model given (assumed based on [10, 25]) parameters $\alpha = 0.0035$, $\beta = 0.56$, $\gamma = 0.0056$, $\kappa = 0.0028$, and DFE initial conditions. We take total population $N = 1000$.

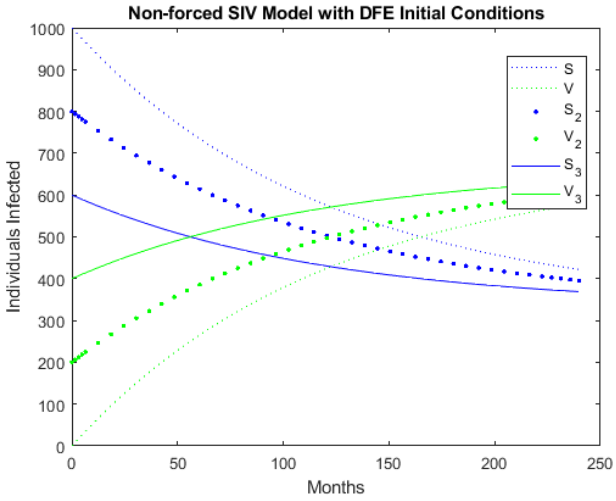

Figure 2: Long-run behavior of non-forced SIV model with DFE initial conditions.

Note from figure 4.2 that even when given different initial conditions, S and V tend to the fixed point at $(\frac{N}{1+\frac{\gamma}{\kappa}}, 0, \frac{N}{1+\frac{\kappa}{\gamma}})$, which should be around $(333, 0, 667)$ according to our rate parameters, and our plot also approximately tends to our calculated values.

Next, we'll evaluate the stability of the DFE fixed point. Substituting in the DFE initial condition into the Jacobian gives us:

$$J_f^{DFE}(N_1, 0, N_2) = \begin{bmatrix} -\gamma & \beta & \kappa \\ 0 & -\beta-\gamma & 0 \\ \gamma & \gamma & -\kappa \end{bmatrix}$$

We can find the eigenvalues of this matrix by taking the determinant and expanding upon the middle row:

$$det(J_f^{DFE}(N_1, 0, N_2) - \lambda) = \begin{bmatrix} -\gamma-\lambda & \beta & \kappa \\ 0 & -\beta-\gamma-\lambda & 0 \\ \gamma & \gamma & -\kappa-\lambda \end{bmatrix}$$

This gives us

$$\begin{aligned} 0 - (-\beta-\gamma-\lambda) \cdot det(\begin{bmatrix} -\gamma-\lambda & \kappa \\ \gamma & -\kappa-\lambda \end{bmatrix}) + 0 &= -(-\beta-\gamma-\lambda) \cdot det(\begin{bmatrix} -\gamma-\lambda & \kappa \\ \gamma & -\kappa-\lambda \end{bmatrix}) \\ &= (\beta+\gamma+\lambda)((-\gamma-\lambda)\cdot(-\kappa-\lambda)) - (\kappa\gamma) \\ &= (\beta+\gamma+\lambda)(\lambda^2 + \kappa\lambda + \gamma\lambda + \kappa\gamma - \kappa\gamma) \\ &= (\beta+\gamma+\lambda)(\lambda^2 + \kappa\lambda + \gamma\lambda) \\ &= \lambda^3 + (k+\gamma^2+2\gamma+\beta)\lambda^2 + (\beta\kappa+\beta\gamma+\kappa\gamma)\lambda \end{aligned}$$

Since there is no constant term, so from the expression, we can say that one of the eigenvalues is 0. We can find the other two eigenvalues by factorisation, rejecting a $\lambda \neq 0$.

$$\lambda^3 + (k+\gamma^2+2\gamma+\beta)\lambda^2 + (\beta\kappa+\beta\gamma+\kappa\gamma)\lambda \to \lambda^2 + (k+\gamma^2+2\gamma+\beta)\lambda + (\beta\kappa+\beta\gamma+\kappa\gamma)$$

We use the quadratic formula to find the other two eigenvalues:

$$\lambda^2 + (\kappa+\gamma^2+2\gamma+\beta)\lambda + (\beta\kappa+\beta\gamma+\kappa\gamma) = 0$$

$$\lambda = \frac{-(\kappa+\gamma^2+2\gamma+\beta) \pm \sqrt{(\kappa+\gamma^2+2\gamma+\beta)^2 - 4(\beta\kappa+\beta\gamma+\kappa\gamma)}}{2}$$

Note that, as we've discussed in section 2, reasonable values of α, β, γ, and κ are all much less than 1. Thus, we can expect $(\kappa+\gamma^2+2\gamma+\beta)^2$ and $4(\beta\kappa+\beta\gamma+\kappa\gamma)$ to be fairly small numbers, such that $\sqrt{(\kappa+\gamma^2+2\gamma+\beta)^2 - 4(\beta\kappa+\beta\gamma+\kappa\gamma)} < \kappa+\gamma^2+2\gamma+\beta$. Also, all rate parameters are positive. Thus, the real component of the non-zero eigenvalues for the DFE initial condition is always negative, which suggests that, for this model, this is always a stable fixed point [28–30]

4.3 Endemic Fixed point

Next, let us examine the endemic fixed point. We can solve for S^* using the differential equation for I.

$$\begin{aligned}
\alpha S^* I^* - \beta I^* - \gamma I^* &= 0 \\
\alpha S^* I^* &= \beta I^* + \gamma I^* \\
\alpha S^* &= \beta + \gamma \\
\boxed{S^* = \frac{\beta + \gamma}{\alpha}}
\end{aligned}$$

We can solve for V^* using the differential equations for S and I:

$$\begin{aligned}
-\alpha S^* I^* + \beta I^* - \gamma S^* + \kappa V^* &= 0 \\
\alpha S^* I^* - \beta I^* - \gamma I^* &= 0 \\
\hline
-\alpha S^* I^* + \beta I^* - \gamma S^* + \kappa V^* + \alpha S^* I^* - \beta I^* - \gamma I^* &= 0 \\
\hline
-\gamma S^* + \kappa V^* - \gamma I^* &= 0 \\
V^* &= \frac{\gamma(S^* + I^*)}{\kappa} \\
V^* &= \frac{\gamma(S^* + I^*)}{\kappa} \\
V^* &= \frac{\gamma}{\kappa}(N - V^*) \\
V^* &= \frac{\gamma}{\kappa} N - \frac{\gamma}{\kappa} V^* \\
(1 + \frac{\gamma}{\kappa}) V^* &= N \\
\boxed{V^* = \frac{N}{1 + \frac{\gamma}{\kappa}}}
\end{aligned}$$

Now, we can solve for I^* using the definitions of S^* and V^* and the differential equation for V:

$$\begin{aligned}
\gamma S^* + \gamma I^* - \kappa V^* &= 0 \\
\gamma I^* &= \kappa V^* - \gamma S^* \\
I^* &= \frac{\kappa}{\gamma} V^* - S^* \\
\boxed{I^* = \frac{\kappa}{\gamma} \frac{N}{1 + \frac{\gamma}{\kappa}} - \frac{\beta + \gamma}{\alpha}}
\end{aligned}$$

Thus, the endemic fixed point is given by $\boxed{(S^*, I^*, V^*) = (\frac{\beta + \gamma}{\alpha}, \frac{\kappa}{\gamma} \frac{N}{1 + \frac{\gamma}{\kappa}} - \frac{\beta + \gamma}{\alpha}, \frac{N}{1 + \frac{\gamma}{\kappa}})}$.

Figure 3 demonstrates the long-run behavior of the non-forced SIV model given parameters $\alpha = 0.0035$, $\beta = 0.56$, $\gamma = 0.0056$, and $\kappa = 0.0028$ and non-DFE initial conditions.

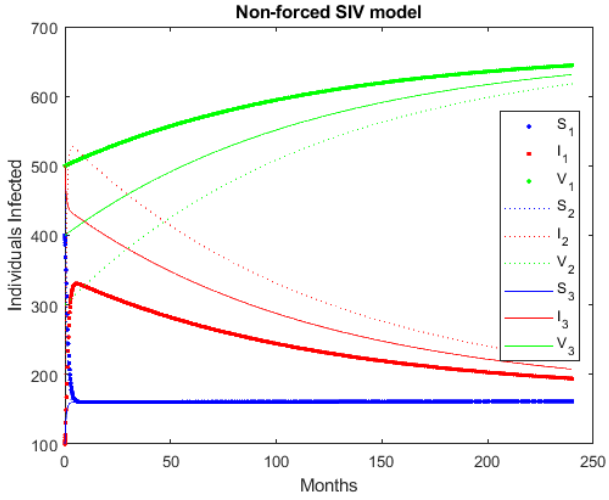

Figure 3: Long-run behavior of non-forced SIV model.

Note in figure 3 regardless of initial conditions, S, I, and V tend to the fixed point at $(\frac{\beta+\gamma}{\alpha}, \frac{\kappa}{\gamma}\frac{N}{1+\frac{\gamma}{\kappa}} - \frac{\beta+\gamma}{\alpha}, \frac{N}{1+\frac{\gamma}{\kappa}})$, which should be approximately $(161, 172, 667)$ given our rate parameters.

As previously mentioned, the endemic fixed point is globally stable, given reasonable rate parameters [25–27, 31]. The proof for why the endemic fixed point is globally stable involves a Lyapunov function.

Although we cannot explicitly solve the system when including the external forcing, we can observe that the long-run behavior is still similar. Figure 4 demonstrates the long-run behavior of the forced SIV model given parameters $\alpha = 0.0035$, $\beta = 0.56$, $\gamma = 0.0056$, $\kappa = 0.0028$, and $r = 15$.

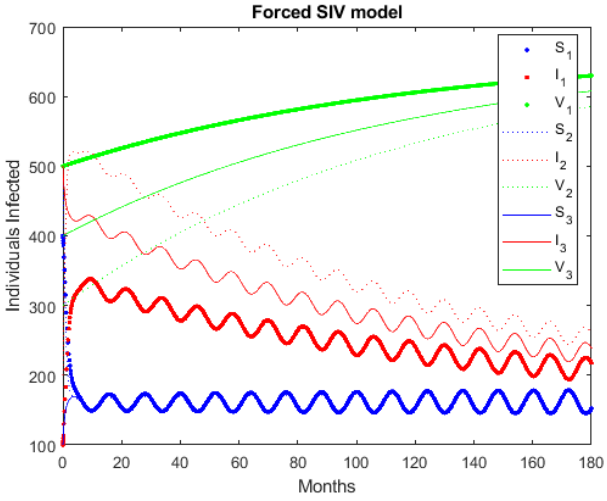

Figure 4: Long-run behavior of forced SIV model.

Note that it appears to reach periodic equilibrium around the same fixed points as in the non-forced SIV model. This may suggest that the long-run behavior of the forced SIV model cycles about the fixed points of the non-forced SIV model. Additionally, note that even when given different initial conditions, S, I, and V still tend towards the same long-run behavior.

5 Results and Discussion

We will now discuss the application of this model in predicting the timing and intensity of seasonal Influenza. The Indian Health Service reports that approximately 36% of the population of India has an effective vaccine against seasonal Influenza [32]. From this, we can estimate that approximately 64% of the population is susceptible to Influenza. The average number of infected individuals recorded per month over the past 15 years is 1108 [3, 33].

We'll assume that there is a limit to the number of people that individuals can infect, due to geographical boundaries or otherwise. So, to simulate the infection dynamics in India, we'll take a small sample population, with $N = 10000$. To have long-run behavior similar to the reported rates of vaccination and disease incidence, we use the parameters $\alpha = 0.00001$, $\beta = 0.045$, $\gamma = 0.005$, $\kappa = 0.006$, and $r = 15$. Based off of our findings in 4, these parameters should result in long-run behavior with S, I, and V approaching periodic equilibrium around $(\frac{\beta+\gamma}{\alpha}, \frac{\kappa}{\gamma}\frac{N}{1+\frac{\gamma}{\kappa}} - \frac{\beta+\gamma}{\alpha}, \frac{N}{1+\frac{\gamma}{\kappa}})$, which is approximately $(5000, 500, 4500)$.

Figure 5 details the SIV model using the informed parameters, as run with three different sets of initial conditions, being $(8000, 1000, 1000)$, $(1000, 2000, 7000)$, and $(5500, 500, 4000)$.

Note that even when given different initial conditions are imposed, S, I, and V tend towards periodic equilibrium around the fixed points of the non-forced SIV model. Additionally, in using informed parameters, the long-run behavior of the model somewhat resembles the published percentages of vaccination and disease incidence.

Figure 6 details clinical and epidemiological data published by the NCDC regarding the number of individuals infected with seasonal Influenza H1N1, each year from 2010 to 2024 (31 March) [3, 33].

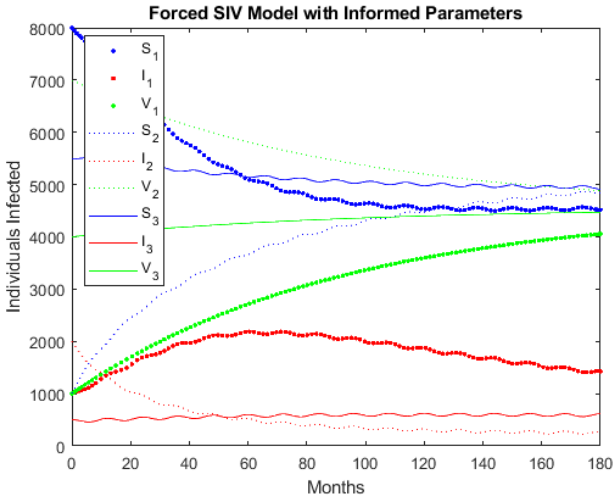

Figure 5: SIV model with informed parameters over 15 years.

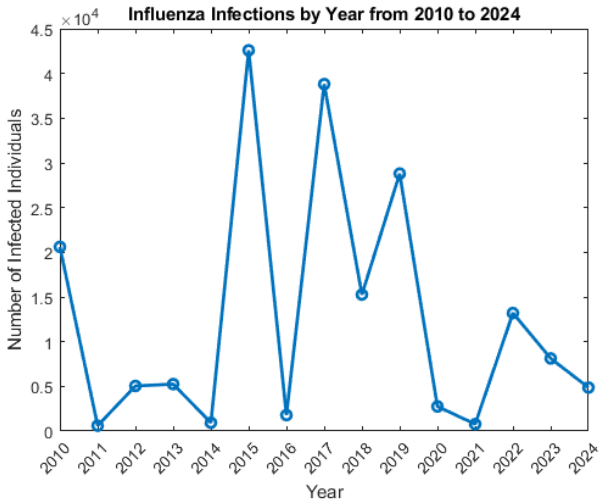

Figure 6: Influenza infections by year from 2010 to 2024 (31 March).

The long-run behavior of the "Infected" compartment in the SIV model somewhat resembles the clinical data published in literature. More specifically, the SIV model appears to be able to capture some elements of the seasonal behavior of Influenza, as seen in the periodicity of its long-run behavior. However, it appears that the SIV model was unable to capture relevant information about the varied intensity of seasonal outbreaks, as the amplitude of the external forcing is constant. This differs from the above clinical data, in which different years have seasonal outbreaks of varying intensity.

6 Possible Improvements

It appears that a shortcoming of the SIV model is an inability to account for variation in the intensity of Influenza outbreaks, we've taken the amplitude of external forcing is constant. This variance in intensity could be explained by one of the biological or epidemiological implications [34–37] that we ignored. For example, the SIV model is only capable of interpreting the behavior of one strain of Influenza. As mentioned in section 3, a more realistic model would take into consideration multiple strains concurrently. When a certain strain has been dominant in Human populations for an extended period of time, individuals tend to more quickly lose immunity to the non-dominant strains [8]. A current example of this effect is seen between the H1N1 and H3N2 strains of Human Influenza – H3N2 has been the dominant strain in Human populations for most recent years, but H1N1 has been shown to escape immune response at a faster rate [38]. One way of improving the SIV model could be by taking more of these biological and epidemiological assumptions into consideration, such that the model would be able to detect the factors that play into the varying intensity of Influenza outbreaks. This would likely make the model more accurate, however, it would also likely add additional parameters to the model, which would increase computational complexity. Adding parameters would also increase the number of decisions that must be made for the model.

7 Conclusions

We've presented a susceptible-infected-vaccinated model which attempts to take into consideration both the seasonal dynamics of Influenza as well as the time-limited immunity conferred through vaccination. Due to the presence of a time-dependent external forcing term, the fixed points of the system of ordinary differential equations could not be found, however, we were able to solve for the fixed points of the system when ignoring the periodic component. From this, we've shown that **the long-run behavior of the "forced SIV model" cycles about the fixed points of the "non-forced SIV model".**

By consulting relevant literature as well as publicly available clinical and epidemiological data, we've identified a set of model parameters which result in output that somewhat resembles infection dynamics as observed in the past 15 years (upto 31^{st} March'2024). Additionally, although the epidemiological behavior of seasonal influenza is periodic, the shape of the data shown in Figure 6 does not appear to resemble any sinusoidal curve. This suggests that a sine term is likely not the most appropriate function to represent the seasonal aspect of Influenza. The model could be improved by introducing a more suitable function to represent the external forcing associated with the seasonal behavior of influenza. A possible candidate for an alternative external forcing term could be a parabolic function.

Although the SIV model appears to have captured some elements of the seasonal behavior of Influenza infection dynamics, further work is necessary to account for the varying intensity of seasonal outbreaks.

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