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Stochastic Modeling of Infectious Disease

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Abstract **– Here we present three versions of an SIR model including death, birth, infection, and recovery terms. We began with an unforced model, which we non-dimensionalized and analyzed. It was found to have two steady states: one where the entire population has become susceptible and another with less easily interpretable population levels. The states were mutually exclusive, depending on the infection and recovery terms and featured slightly different behaviors. We then altered the model in an attempt to resemble data from the CDC. First, a seasonal forcing term was added that had a period of half of a year. With this term, the model produced a profile that resembled a CDC yearly profile in that the infected population of both varied on the order of a year. Next, a stochastic or random model was created that treated each term of our non-dimensionalized equation as random events. A random event was triggered by each time step, creating profiles that appeared to replicate the randomness of CDC data.**

Keywords **– Vital Dynamics, Infectious Disease, Stochastic Model, Seasonal Forcing, SIR Model.**

I. Introduction to the SIR Model

In 1927, W.O. Kermack and A.G. McKendrick created an epidemiological model in which they considered a fixed population with three compartments [5, 6]. Their model is presented here without change:

$$\frac{dx}{dt} = -kxy$$

$$\frac{dy}{dt} = kxy - \tau y$$

$$\frac{dz}{dt} = \tau y \qquad (1)$$

where x represents the population susceptible to disease, y represents the population infected and z represents those removed from the infectious pool. The constants κ and τ represent the effective contact rate and the recovery rate, respectively, and are discussed in further detail below.

These equations form the basis of what is known today as the SIR model wherein:

- $s(t)$ represents the number of individuals susceptible to the disease.
- $i(t)$ represents the number of individuals who have been infected and are capable of spreading the disease to those in the susceptible category.
- $r(t)$ represents the number of individuals who have been infected and are no longer contagious, either due to death or immunity.
- β^0 represents the effective contact rate. In Kermack and McKendrick's model, the function $F = \kappa x$ models the transition rate from the compartment of susceptible individuals to the compartment of infectious individuals; it may be called the force of infection [6]. We consider a force of infection that depends on the

fraction of the infectious with respect to the constant population N: $F = \beta' \frac{i}{N}$

The constant β^0 is in units of time^{-1} and is the number of contacts made per unit time.

- γ^0 represents the removal rate. This constant, represented by τ in Kermack and McKendrick's equations, models the rate of movement from the infected compartment to the recovered compartment. This is the inverse of the duration of infection and is in units of time^{-1}.

The flow of this model is considered in a closed population N: $s \to i \to r$.

Thus, we have the following model:

$$\frac{ds}{dt} = -\beta' \frac{i}{N} s,$$

$$\frac{di}{dt} = -\beta' \frac{i}{N} s - \gamma' i,$$

$$\frac{dr}{dt} = \gamma' i. \tag{2}$$

II. Basic Model

A. *Vital Dynamics*

Vital dynamics refers the effects of natural birth and death on the model [9]. Consider a birth rate (μ) equal to the death rate. Then the model becomes:

$$\frac{ds}{dt} = -\beta' \frac{i}{N} s + \mu N - \mu s,$$

$$\frac{di}{dt} = -\beta' \frac{i}{N} s - \gamma i - \mu i,$$

$$\frac{dr}{dt} = \gamma' i - \mu r. \tag{3}$$

The term μN represents the introduction of new susceptible individuals into the population by birth. Each compartment has a negative death term ($-\mu s$, $-\mu i$, $-\mu r$), which represents the removal of individuals due to natural death. We can see that the total population N remains constant [9].

Instead of modelling birth and death, we can use μ to represent the natural influx and efflux of individuals from the model. For example, let's consider an outbreak of the flu at New Mexico Tech. Suppose there are 2000 people in the student population and suppose that the average "lifetime" of a student is 2 years thus N = 2000 and μ = 1/(2·365). Furthermore, suppose each student makes 10 meaningful contacts per day and that each infected person remains contagious for 3 days so that β^0 = 10 and γ^0 = 1/3. For initial conditions let's set $s(0)$ = 1900, $i(0)$ = 10 and $r(0)$ = 90. The progression of disease for the basic SIR model with μ = 0 is shown in figure 1. An initial epidemic affecting most of the population occurs within 2 days. Within 2 weeks, only a small fraction of students remain infected.

In figure 2 we consider the effects of $\mu > 0$ on the model. The graph is shown with the y–axis on a logarithmic scale. Over the course of a year we have a similar initial epidemic followed by outbreaks of decreasing amplitude. We will show later that a small fraction of the population remains infected as $t \to \infty$.

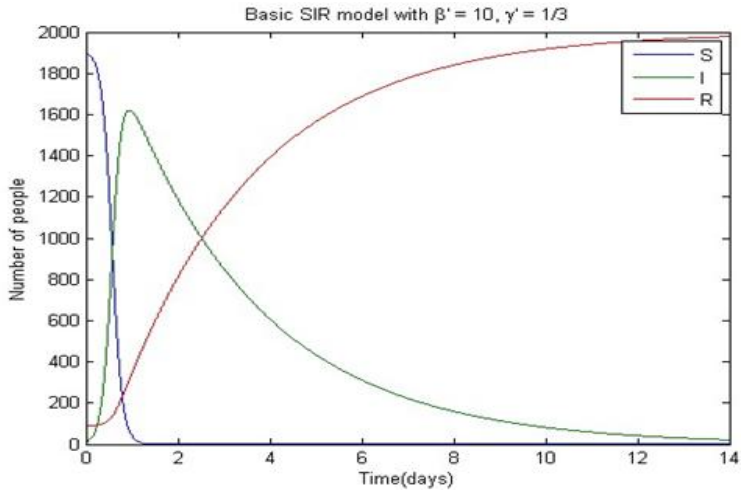

Fig. 1. The basic SIR model.

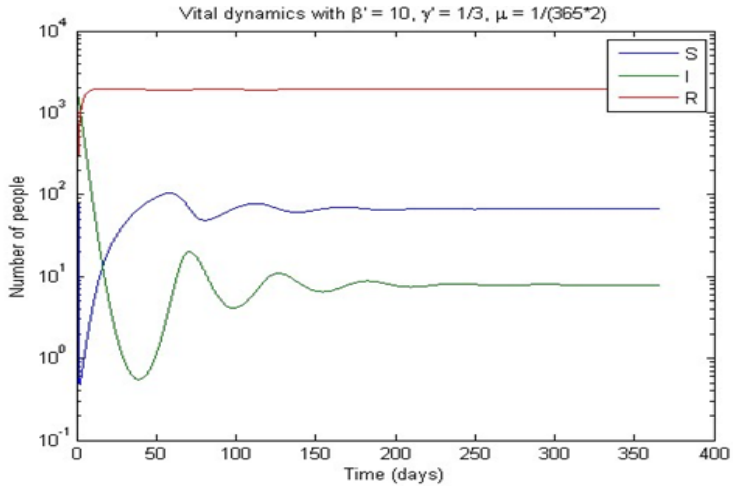

Fig. 2. Vital dynamics.

B. *Non-dimensionalization*

To get a non–dimensionalized version of the model in (3), let $S = \frac{s}{N}$, $I = \frac{i}{N}$, $R = \frac{r}{N}$. So that $S + I + R = 1$.

To eliminate μ, let $\beta = \frac{\beta'}{\mu}$, $\gamma = \frac{\gamma'}{\mu}$, $r = \mu t$.

Thus $\frac{d}{dt} = \frac{d}{d\tau}\frac{d\tau}{dt} = \frac{d}{dt}\mu$, So we have the non-dimensionalized model [10]:

$$\frac{dS}{d\tau} = 1 - \beta SI - S,$$

$$\frac{dI}{d\tau} = \beta SI - \gamma I - I,$$

$$\frac{dR}{d\tau} = \gamma I - R. \qquad (4)$$

C. *Equilibrium States and Stability*

To determine the equilibrium states and the conditions for their stabilities, we begin by setting the equations in (4) equal to 0 [7].

$$\frac{d}{d\tau}\begin{pmatrix} S \\ I \\ R \end{pmatrix} = \begin{pmatrix} -S(\beta I + 1) - 1 \\ I(\beta S - \gamma - 1) \\ \gamma I - R \end{pmatrix} = \vec{0}$$

One solution is $\{S = 1, I = 0, R = 0\}$. This solution is important because it corresponds to 100% of the population being susceptible and none being infected. Now we linearize the system around this equilibrium state. We calculate the Jacobian matrix of partials: $J(S, I, R) = \begin{pmatrix} -\beta I - 1 & \beta I & 0 \\ -\beta S & -\beta S - \gamma - 1 & \gamma \\ 0 & 0 & -1 \end{pmatrix}$

Now we evaluate at the equilibrium state (1, 0, 0) whence this matrix takes the form:

$J(1,0,0) = \begin{pmatrix} -1 & 0 & 0 \\ -\beta & -\beta-\gamma-1 & \gamma \\ 0 & 0 & -1 \end{pmatrix}$ Next, subtract λ down the diagonal, take the determinant and set it to zero.

$$\begin{vmatrix} -1-\gamma & 0 & 0 \\ -\beta & \beta-\gamma-1-\lambda & \gamma \\ 0 & 0 & -1-\lambda \end{vmatrix} = -(\lambda+1)\begin{vmatrix} \beta-\gamma-1-\lambda & \gamma \\ 0 & -1-\lambda \end{vmatrix} = (\lambda+1)^2(\beta-\gamma-1-\lambda) = 0.$$

Let $\alpha = \beta - \gamma - 1$. Then we have eigenvalues $\lambda_1 = \lambda_2 = -1$ and $\lambda_3 = \alpha$. For stability, we need negative eigenvalues, that is $\alpha < 0$ so $\beta - \gamma - 1 < 0$. Thus the condition for our first equilibrium state is $\beta - \gamma < 1$.

A second equilibrium state, calculated in Maple, occurs when $\left\{S = \frac{\gamma+1}{\beta}, I = \frac{\alpha}{\beta(\gamma+1)}, R = \frac{\alpha}{\beta(\gamma+1)\gamma}\right\}$.

Suppose $\alpha < 0$, the condition of stability for our first equilibrium state. But then I and R are negative, which is meaningless considering the physical constraints of our model. So we consider the case when $\alpha > 0$, where our first equilibrium state is unstable. Thus the Jacobian is as follows: $J\left(\frac{\gamma+1}{\beta}, \frac{\alpha}{\beta(\gamma+1)}, \frac{\alpha}{\beta(\gamma+1)\gamma}\right)$

$$\begin{pmatrix} \frac{\alpha}{\gamma+1} - 1 & \frac{\alpha}{\gamma+1} & 0 \\ -(\gamma+1) & 0 & \gamma \\ 0 & 0 & -1 \end{pmatrix}.$$

As before we subtract λ down the diagonal and take the determinant, this time using a cofactor expansion along the bottom row.

$$\begin{vmatrix} \frac{\alpha}{\gamma+1} - 1 - \lambda & \frac{\alpha}{\gamma+1} & 0 \\ -(\gamma+1) & -\lambda & \gamma \\ 0 & 0 & -1-\lambda \end{vmatrix} = -(\lambda+1)\begin{vmatrix} \frac{\alpha}{\gamma+1} - 1 - \lambda & \frac{\alpha}{\gamma+1} \\ -(\gamma+1) & -\lambda \end{vmatrix} = -(\lambda+1)\left[\lambda^2 + \lambda\left(1 - \frac{\alpha}{\gamma+1}\right) + \alpha\right] = 0$$

We have our first eigenvalue $\lambda_1 = -1$. To find the other eigenvalues, we use the quadratic formula so

$\lambda = \frac{\left(1-\frac{\alpha}{\gamma+1}\right) \pm \sqrt{\left(1-\frac{\alpha}{\gamma+1}\right)^2 - 4\alpha}}{2}$ We require $\alpha > 0$ thus, $\lambda = \frac{\left(1-\frac{\alpha}{\gamma+1}\right) \pm \sqrt{\left(1-\frac{\alpha}{\gamma+1}\right)^2 - 4\alpha}}{2}$. $-4\alpha < 0$

$$\Rightarrow \left(1 - \frac{\alpha}{\gamma+1}\right)^2 - 4\alpha < \left(1 - \frac{\alpha}{\gamma+1}\right)^2 \Rightarrow \sqrt{\left(1 - \frac{\alpha}{\gamma+1}\right)^2 - 4\alpha} < \left|1 - \frac{\alpha}{\gamma+1}\right|.$$

Let's suppose the quantity inside the absolute value is negative, that is $1 - \frac{\alpha}{\gamma+1} < 0$. Then we have

Also, $\lambda_2 = \frac{\left(1-\frac{\alpha}{\gamma+1}\right) \pm \sqrt{\left(1-\frac{\alpha}{\gamma+1}\right)^2 - 4\alpha}}{2} < 0 - \left(1 - \frac{\alpha}{\gamma+1}\right) > \sqrt{\left(1 - \frac{\alpha}{\gamma+1}\right)^2 - 4\alpha} \Rightarrow \left(1 - \frac{\alpha}{\gamma+1}\right) + \sqrt{\left(1 - \frac{\alpha}{\gamma+1}\right)^2 - 4\alpha} <$

0

So as long$1 - \frac{\alpha}{\gamma+1} < 0$, our third eigenvalue, λ_3, is negative and this equilibrium state is stable. Then since $\alpha = \beta - (\gamma + 1)$ we have: $1 < \frac{\alpha}{\gamma+1} = \frac{\beta-(\lambda+1)}{\gamma+1} \Longrightarrow \gamma + 1 < \beta - (\gamma + 1)$

Finally, this simplifies to $\beta - 2\gamma > 2$, which is our stability condition for this equilibrium state

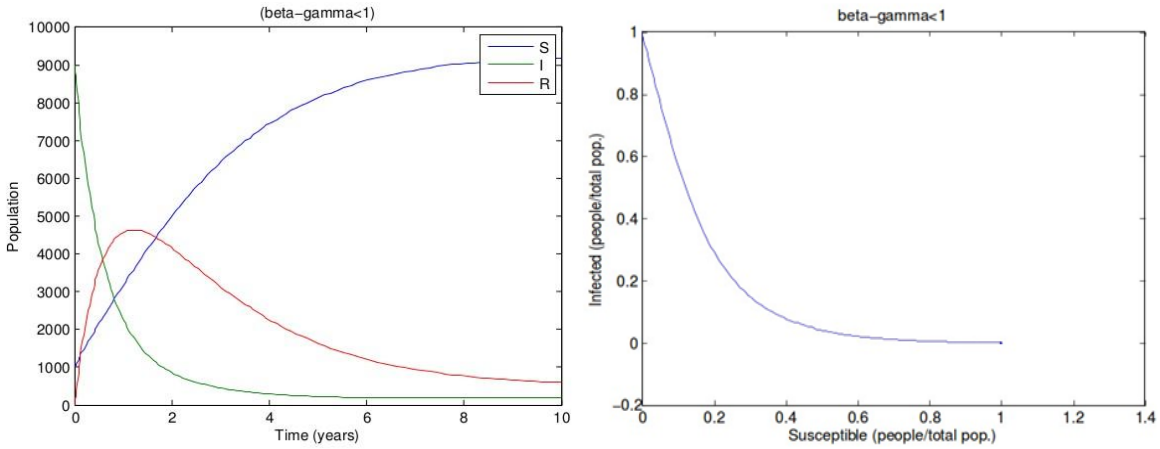

Fig. 3. An example of the first equilibrium state and its phase plot.

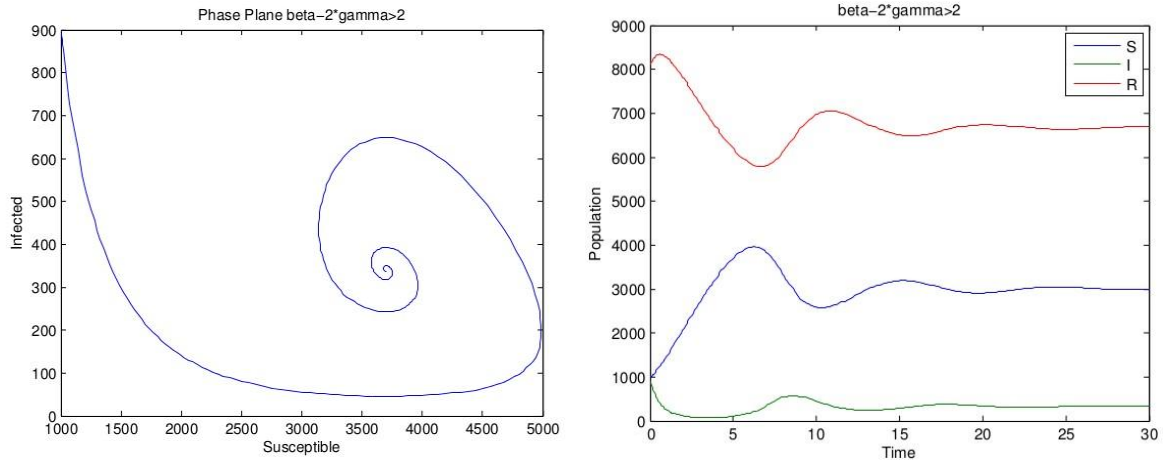

Fig. 4. An example of the second equilibrium state and its phase plot.

In the above figures, the profile approaches the first equilibrium point (1, 0, 0) quickly.

III. SEASONAL FORCING

The above model produces behavior that is reasonable on small time scales, but constant equilibrium states are not realistic. The number of infections should vary with time, as usually more flu cases are documented in the winter and fall than spring and summer [8]. To attempt to replicate this seasonal dependence, β may be changed to

$$\beta = \beta_0(1 + \beta_1 \cos(2\pi t/365)) \quad (5)$$

where $t = days$, so that for small timescales, $\cos(e) \approx 1$ and β reduces to a constant. The constant term (β_0) and periodic term $\beta_0(1 + \beta_1 \cos(2\pi t))$ can be separately varied.

The equilibrium states for this model are the same as the previous model, but they are now functions of time. We should then expect not constant values of S, I, and R for all time, but periodic behavior.

In figure 5, the left plot has a biannual period, so the argument of the cosine term in β is $(\pi t/365)$. The variables were also changed so that the solution is stable. The right plot, is obviously less stable over a range of 50 years.

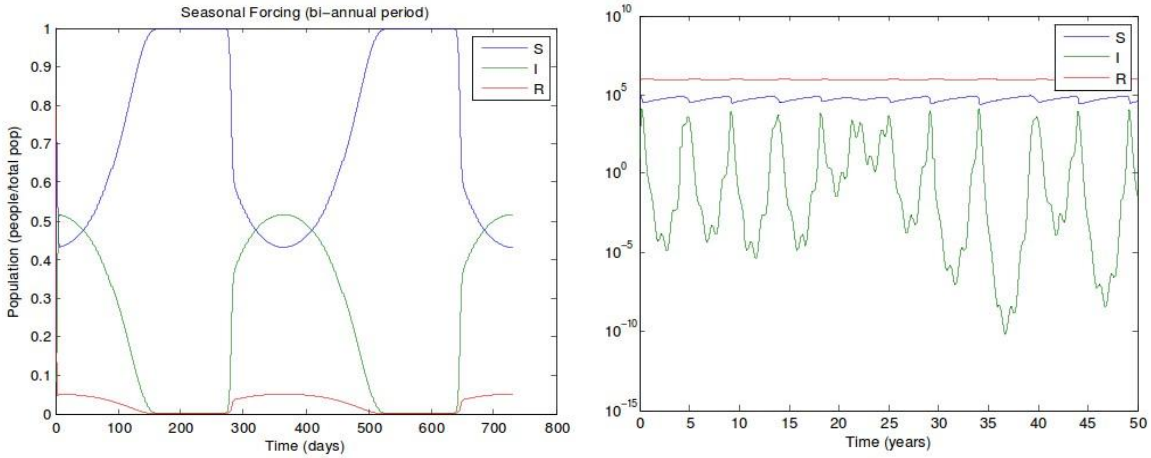

Fig. 5. Two runs of a model with seasonal forcing. The left has biannual forcing and was only run for two years, while the right plot has a different forcing period and was run for longer.

If the left plot is compared to data from the CDC (figure 6), the periodic behavior of the real data is replicated in the model.

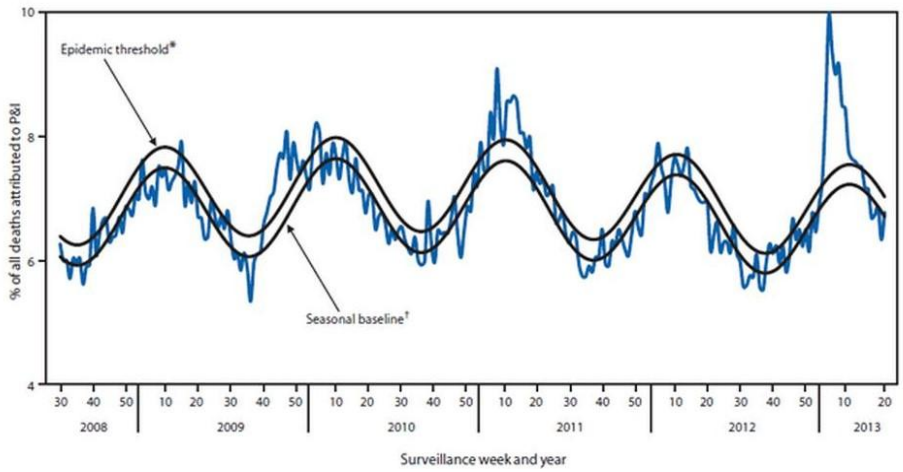

Fig. 6. A plot of flu and pneumonia deaths as a function of time from CDC data.

IV. Stochastic Model

The two models above have complicated but determined behaviors for specific values of each variable. If this dependence could be made more random, perhaps the model output could approach a realistic behavior [2].

We took each term of the non-dimensonalized system (births, deaths, infections, recoveries) and called them events. Our model then stepped through time and created a random number at each timestep. Depending on the value of the number, an event would happen. In this way, random occurrences of life could be modelled.

In figure 7, each of the runs appears to have some periodicity. It should be noted that the model code included no periodic forcing, so this is purely coincidental. The periods and smaller scale behavior differs between each plot, as seen when they are plotted on the same axes (left plot of figure 8).

In figure 8, the two plots have different time domains, as our model covered up to 14 years and had four runs, while the CDC data covers around a year and has six years of data. Our model resembles the CDC data, as the over plotted profiles are all different, but there are peaks. If enough of our runs were averaged, we may be able to produce profiles more similar to the CDC data, but since the model is purely random, mostly any profile could be replicated in this way. Nonetheless, this stochastic model is a useful approximation of realism.

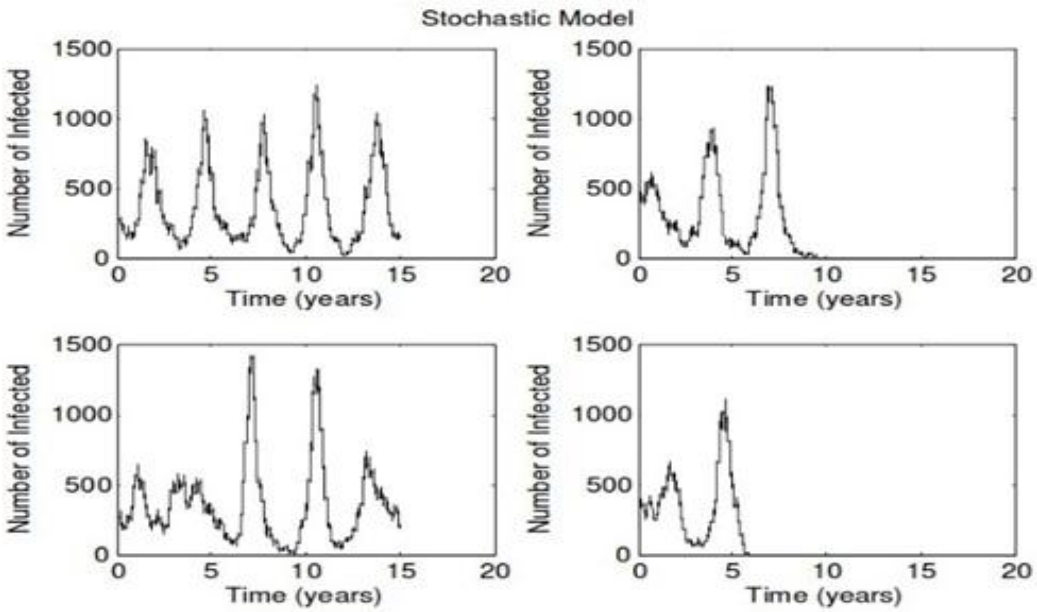

Fig. 7. Four runs of our Stochastic model. Each run had identical initial values but evolved differently.

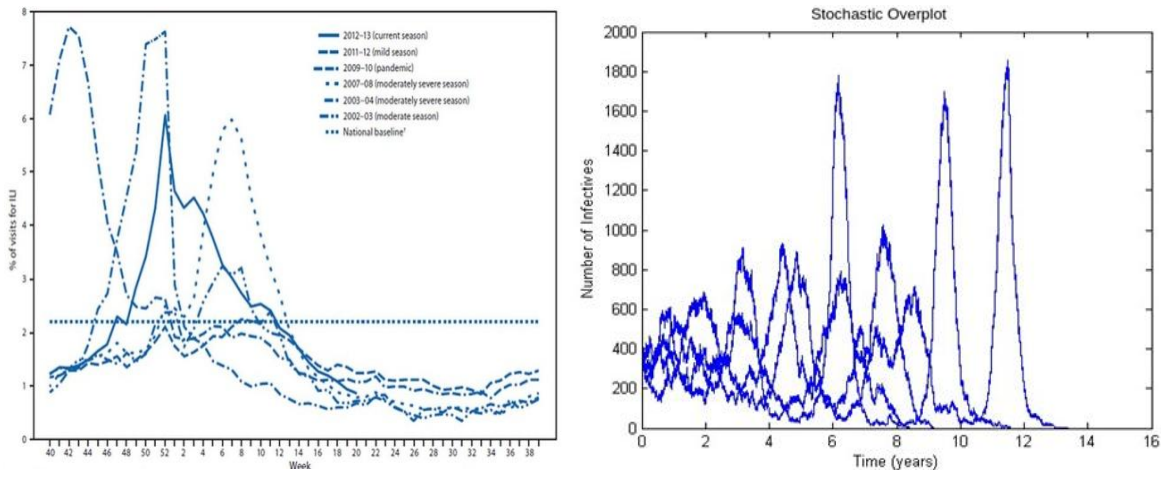

Fig. 8. Left: Overplotted model runs. Right: A plot of flu and pneumonia cases as a function of time from CDC data for multiple years.

V. Conclusion

We found that in our unforced model, the two equilibrium states are that of the entire population becoming susceptible and a more complicated state. Requiring the population to be positive made it so that only one point was stable at a time. In both cases, the population levels in each compartment varied slightly and approached the steady state, where they stayed. The more complicated state featured increased oscillation before settling to the equilibrium point. Which state was stable depended on the relative values of the interaction term β and the recovery term γ. This is reasonable, as if no one recovers, they either stay infected or die and, on average, new

susceptible people are born. Thus, the infected and recovered populations die out and the total population becomes susceptible. Alternatively, if recovery is widespread, there is enough movement between the three levels to allow for a steady state with a non–zero infected population. Our alterations to the model resulted in profiles that featured periodicity and variety similar to those of CDC plots. These properties are required for realism, as neither of the CDC profiles had steady states with constant infected levels.

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