Introduction to Stochastic SIR Model

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SIR model is used to model the infection of diseases. It is short for Susceptible-Infected-Recovered. It is important to address that SIR model does not work with all diseases. It has several basic assumptions:

1. The total population is fixed. That is, the birth rate is equal to the death rate;
2. A person from the susceptible group cannot be immune to the disease. Only the recovered persons are immune and they are immune lifelong;
3. New births belong to the susceptible group. That is, there is no inherited immunity or mother-to-fetus transmission;
4. Gender, age and race do not affect this model;
5. The members of the groups mix homogeneously.

Details of the model are as following: there is a susceptible population of size $S$, an infective population of size $I$ and a population of recovered populations of size $R$. The evolution of the populations is according to the rates

\[
\text{transition rate} \quad \text{rate} \\
S \rightarrow S+1 \quad \mu N
\]
\[
S \rightarrow S+1 \quad \beta SI / N + \mu S \\
I \rightarrow I+1 \quad \beta SI / N \\
I \rightarrow I-1 \quad (\gamma + \mu)I \\
R \rightarrow R+1 \quad \gamma I \\
R \rightarrow R-1 \quad \mu R \\
\]

where \( N \) (total population size) is fixed and equal to \( S+I+R \). \( \mu \) is the birth or death rate per individual, \( \gamma \) is the recovery rate and \( \beta \) is the average number of transmissions from an infected person per unit time.

We define the basic reproductive number \( R_0 \),

\[
R_0 = \frac{\beta}{\mu + \gamma}
\]

is the number of new infectives produced by one infective introduced in a completely susceptible population.

The formulas of the model are expressed as

\[
\frac{dS}{dt} = \mu(N - S) - \beta \frac{SI}{N} \quad (1) \\
\frac{dI}{dt} = \beta \frac{SI}{N} - (\gamma + \mu)I \\
R = N - I - S
\]

The equations can be interpreted: the increase of the susceptible population equals the new births minus the death from the susceptible minus the new infected; the increase of the infected group is the new infected minus the death and the recovered from the infected group.

If \( R_0 > 1 \), the system has a unique nontrivial stable equilibrium
We introduce the dimensionless variables

\[ u = \frac{S - S_{eq}}{S_{eq}}, \quad v = \frac{I - I_{eq}}{I_{eq}}, \]

and rescale time \( t \) to \( \Omega t \), where \( \Omega = \sqrt{\frac{\alpha \beta}{R_0} (R_0 - 1)} \). Then we can get an approximate solution of the system

\[
\begin{pmatrix}
  u \\
  v
\end{pmatrix}
\sim C_1 e^{-\epsilon^2 t} \begin{pmatrix}
  b \cos t \\
  \sin t
\end{pmatrix} + C_2 e^{-\epsilon^2 t} \begin{pmatrix}
  b \sin t \\
  -\cos t
\end{pmatrix}
\]

where \( C_1 \) and \( C_2 \) are two constants yet to be determined and \( \epsilon^2 \) is a constant much smaller than 1. Both \( u \) and \( v \) show exponentially decay with time, which is not reasonable because disease will not extinct in reality. To explain such phenomenon, we should pay attention to the fact that the birth, death, infecting, recovery process are all stochastic. Mathematically, they yield poisson processes with zero mean and certain variances. That is, we need to rewrite the equations in stochastic form:

\[
dS = (\mu (N - S) - \beta \frac{SI}{N}) dt + dz_1 - dz_2 \\
dl = (\beta \frac{SI}{N} - (\gamma + \mu) I) dt + dz_2 - dz_3
\]

where \( dz_i (i = 1, 2, 3) \) are poisson variables (stochastic terms). \( dz_1 \) is corresponding to the birth and death process. \( dz_2 \) infecting process and \( dz_3 \) recovery process and death process. Their variances are
\[ \mu(N+S) dt, \beta \frac{SI}{N} dt, (\gamma + \mu) I dt \] respectively.

In order to develop the theory we need to replace the poisson variables by Brownian variables \(dW_i\), with the same means and variances. Actually, for small \(dt\) and large \(N\), wiener process (Brownian) is a well approximation of poisson process. So the general SIR equations are:

\[
\begin{align*}
\text{d}S &= \left(\mu(N-S) - \beta \frac{SI}{N}\right) \text{d}t + G_1 \text{d}W_1 - G_2 \text{d}W_2, \\
\text{d}I &= \left(\beta \frac{SI}{N} - (\gamma + \mu)I\right) \text{d}t + G_2 \text{d}W_2 - G_3 \text{d}W_3,
\end{align*}
\]

\[G_1 = \sqrt{\mu(N+S)}, \quad G_2 = \sqrt{\beta \frac{SI}{N}}, \quad G_3 = \sqrt{(\gamma + \mu)I}\]

We reform them to be:

\[
\begin{align*}
\text{d} \begin{pmatrix} u \\ v \end{pmatrix} &= M \begin{pmatrix} u \\ v \end{pmatrix} \text{d}t + G \begin{pmatrix} \text{d}W_1 \\ \text{d}W_2 \\ \text{d}W_3 \end{pmatrix}, \quad \text{(2)}
\end{align*}
\]

\[
M = \begin{pmatrix} -\frac{\mu R_0}{\Omega} & \frac{R_0 - 1}{\Omega} \\
\frac{\beta}{\Omega R_0} & 0 \end{pmatrix},
\]

\[
G = \begin{pmatrix} \frac{\mu}{\Omega S_{eq}^2} (N + S_{eq}) & -\frac{\beta I_{eq}}{\Omega NS_{eq}} & 0 \\
0 & \frac{\beta S_{eq}}{\Omega N I_{eq}} & -\frac{\mu + \gamma}{\Omega I_{eq}} \end{pmatrix}
\]

\[= \begin{pmatrix} g_1 & -b^2 g_2 & 0 \\
0 & g_2 & -g_2 \end{pmatrix}\]
We consider an approximation for solving the stochastic model,

\[ \begin{pmatrix} u \\ v \end{pmatrix} \sim A(T) \begin{pmatrix} b \cos t \\ b \sin t \end{pmatrix} + B(T) \begin{pmatrix} b \sin t \\ -\cos t \end{pmatrix}, \quad T = \mathcal{E}^2 t, \quad (3) \]

where \( A(T) \) and \( B(T) \) contain both the slow decay and stochastic part, and assume that all the stochastic effect is absorbed into \( A(T) \) and \( B(T) \).

By substitution of (3) into (2), the system becomes

\[ du = (-2b\mathcal{E}^2 [A \cos t + B \sin t] + b[-A \sin t + B \cos t])dt + g_1dW_1 - b^2g_2dW_2, \]

\[ dv = [A \cos t + B \sin t]dt + g_2dW_2 - g_2dW_3, \quad (4) \]

Since \( A(T) \) and \( B(T) \) contain stochastic term, we could express them in

\[ \begin{pmatrix} dA \\ dB \end{pmatrix} = \begin{pmatrix} f_1 \\ f_2 \end{pmatrix} dT + \begin{pmatrix} dN_1(T) \\ dN_2(T) \end{pmatrix}, \quad (5) \]

which \( dN_1, dN_2 \) represent the stochastic form, and \( f_1, f_2 \) serve as the drift term (deterministic term).

Combined with Ito’s formula,

\[ du = \frac{\partial u}{\partial A} dA + \frac{\partial u}{\partial B} dB + \frac{\partial u}{\partial t} dt + \frac{1}{2} Q_1 \frac{\partial^2 u}{\partial A^2} dt + \frac{1}{2} Q_2 \frac{\partial^2 u}{\partial A \partial B} dt + \frac{1}{2} Q_3 \frac{\partial^2 u}{\partial B^2} dt, \]

and ignoring the second derivative term because they are all equal to zero, another expression for \( du \) and \( dv \) could be found by combining (3) and (5):

\[ \begin{pmatrix} du \\ dv \end{pmatrix} = \begin{pmatrix} b \cos t & b \sin t \\ \sin t & -\cos t \end{pmatrix} \begin{pmatrix} f_1 \\ f_2 \end{pmatrix} dT + \begin{pmatrix} -bA \sin t + Bb \cos t \\ -bA \sin t + Bb \cos t \end{pmatrix} dt + \begin{pmatrix} b \cos t & b \sin t \\ \sin t & -\cos t \end{pmatrix} \begin{pmatrix} dN_1(T) \\ dN_2(T) \end{pmatrix}. \]

Here comes the core part of this method. We use multiscale analysis to
separate the first order \( t \) and the second order \( T \) and stochastic term, similar to perturbation theory. By comparing the coefficient of (3) and (5), the drift coefficient is shown to be \( f_1=\alpha \), \( f_2=\gamma \) which A and B here are pure deterministic term. By introducing the covariance matrix, the stochastic amplitude in (4) can now be written as

\[
\left( \frac{dA}{dB} \right) = \begin{pmatrix} -1 & 0 \\ 0 & -1 \end{pmatrix} \begin{pmatrix} A \\ B \end{pmatrix} dT + \frac{\delta}{\sqrt{2\varepsilon}} \begin{pmatrix} 1 & 0 \\ 0 & 1 \end{pmatrix} \begin{pmatrix} dw_1 \\ dw_2 \end{pmatrix},
\]

\[ \frac{\delta}{\sqrt{2\varepsilon}} = \sqrt{\frac{\alpha + \gamma}{4N\alpha}} \left( 1 + \frac{R_0 + 1}{R_0 - 1} + 2 \frac{\alpha + \gamma}{\alpha(R_0 - 1)} \right). \]

The \( dw_1 \) and \( dw_2 \) are independent wiener process term. We did not show the exact derivation but just presenting the idea.

Equation (7) is exactly the Ornstein-Uhlenbeck type process. By changing variables to \( y = Ae^T \), we have

\[ dy = \frac{\delta}{\sqrt{2\varepsilon}} e^t dw_1, \]

with solution

\[ A(T) = A(0)e^{-T} + \frac{\delta}{\sqrt{2\varepsilon}} \int_0^T dw_1(s)e^{-(T-s)} \]

\[ B(T) = B(0)e^{-T} + \frac{\delta}{\sqrt{2\varepsilon}} \int_0^T dw_2(s)e^{-(T-s)}. \]

The rule of changing variables from \( u \) to \( v \) \((u ,t)\) in Ito’s formula is

\[ dv = \left( \frac{\partial v}{\partial t} + f \frac{\partial v}{\partial u} + \frac{1}{2} g^2 \frac{\partial^2 v}{\partial u^2} \right) dt + g \frac{\partial v}{\partial u} dW, \]

given that \( du = f(u, t)dt + g(u, t)dW. \)

Finally, after inserting (8) into (3), we got the general solution of \( u \) and \( v \).
The way to see the dynamics of S and I is to evaluate the average power:

$$\text{Average power} = \langle u^2(t) \rangle.$$  

However, instead of directly viewing it, power spectral density (PSD), defined as Fourier Transformation of the average power, would present more details. Fig. 1 shows the PSD versus frequency. By transforming the Gaussian-like PSD back, the average power will evolve sinusoidally at centered frequency, which is also called coherence resonance. If the PSD graph looks like fig. 2, then there is no resonance, and the average power will evolve as exponentially decay, just like the deterministic solution.

**Fig. 1**

a) $R_0=15$, $\alpha=1/55$, $\gamma=20$, $N=500000$

b) $R_0=15$, $\alpha=1/55$, $\gamma=25$, $N=500000$

**Fig. 2**

$$PSD \propto \frac{1}{A^2 + \omega^2}$$

Average power exponentially decays.
Comparing the Infective process between deterministic and stochastic solution, it matches to our expectation (Fig. 3). Red curve represents the deterministic solution, which exponentially decays to zero. Black curve shows the stochastic solution, which is sinusoidal evolved.

Conclusion

From the basic definition of SIR model, we got the deterministic solution, which is a wave function term combined with exponential decay. However, it seems not that reasonable. Normally, both susceptible and infective population should not decay to zero with time going. Therefore,
we introduce the stochastic term together with the deterministic differential equation. By using multiscale method combing with the covariance matrix, we got the solution of both \( A(T) \) and \( B(T) \). We plug both coefficients into equation (2) and Fourier transform it to get the PSD graph, which shows a Gaussian-like curve. The Gaussian curve PSD means that the original function would evolve as sustained oscillation, which makes more sense. In this case, both the susceptible and infective population will keep oscillating instead of decaying to zero, remaining a dynamic equilibrium.

**References**