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On some probabilistic epidemic models and the new fractional Brownian motion

Khairia El-said El-nadi[1], Mohamed El-shindidy[2], Aleya Abdelghany Lashen[3]
Department of mathematics and computer science- Faculty of science Alexandria University- Alexandria- Egypt
1- *khairia_el_said@hotmail.com, khairia.elnady@alexu.edu.eg*
2- elshandidy@yahoo.com
3- Aleya.abdelghany@gmail.com, aleya.abdelghany@alexu.edu.eg

Abstract
In this paper, access to some probabilistic models about epidemics will greatly help in understanding the extent of the spread and the influencing factors in terms of age, gender, health status, social status...etc. and through the models, it will help to reach some solutions to combat and reduce some of the diseases that destroy and destroy the living organism, which in turn affects the world.
Keywords: Epidemic models- Fractional stochastic models- New fractional Brownian motion.
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I. Introduction

Probabilistic modeling is a statistical approach that uses the effect of random occurrences or actions to predict the possibility of future results. It is a quantitative modeling method that projects several possible outcomes that might even go beyond what has happened recently.

Using Mathematical Modeling in epidemiology, models of how they progress in an epidemic are based on a set of assumptions and statistics which are used to establish a set of parameters that inform how effective intervention will be (for example, social distancing or mass vaccination).

Where an epidemic is defined as an unforeseen rise in disease cases in a particular region. Examples of epidemics include smallpox, measles, polio, yellow fever, and smallpox. An epidemic disease doesn't need to be communicable. We present a fractional stochastic model for the diffusion of epidemic. The stochastic process, which represents the solution of the considered model, is obtained in fractional stochastic models, we are to study the growth models for epidemic. Where the aim of the research is to reach models to combat the epidemic and work to develop chances of surviving the disease and to know the factors that are favorable and non-influencing and to reach on some probabilistic epidemic models.

Among some of the modern diseases is cancer of all kinds (leukemia, lung cancer, breast cancer, ... etc.) Diabetes and Hypertension and the Corona Virus Disease 19 pandemic that appeared in late 2019, which caused many deaths and imposed a ban on individuals not to spread and reduce deaths as much as possible, which had a role in changing the world in terms of political, economic and health conditions. And many other diseases that had an effect.

Access to some probabilistic models about epidemics will greatly help in understanding the extent of the spread and the influencing factors in terms of age, gender, health status, social status...etc.

Through the models, it will help to reach some solutions to combat and reduce some of the diseases, that destroy and destroy the living organism, which in turn affects the world.

Infectious diseases continue to pose a significant risk to human health. Although advances in medicine and public health have helped control many endemic diseases, a World Health Organization (WHO) study on the global burden of diseases found that by 2002, infectious diseases were responsible for more than one-quarter of approximately 57 million deaths worldwide (World Health Organization (WHO), 2004).

II. Fractional Stochastic System

Consider the following fractional stochastic system

$$S(t)=S(0)+\frac{1}{\Gamma(\alpha)}\int_0^t(t-\Theta)^{\alpha-1}[-\frac{\beta}{N}S(\Theta)+I(\Theta)b(I(\Theta)+R(\Theta))]d\Theta+\frac{\sigma_1}{\Gamma(\alpha)}\int_0^t(t-\Theta)^{\alpha-1}S(\Theta)dW(\Theta),$$

$$I(t)=I(0)+\frac{1}{\Gamma(\alpha)}\int_0^t(t-\Theta)^{\alpha-1}[\frac{\beta}{N}S(\Theta)I(\Theta)-I(\gamma+b)]d\Theta+\frac{\sigma_2}{\Gamma(\alpha)}\int_0^t(t-\Theta)^{\alpha-1}I(\Theta)\,dW(\Theta)$$

$$R(t)=R(0)+\frac{1}{\Gamma(\alpha)}\int_0^t(t-\Theta)^{\alpha-1}[\gamma I(\Theta)-bR(\Theta)]d\Theta-\frac{1}{\Gamma(\alpha)}\int_0^t(t-\Theta)^{\alpha-1}[\sigma_1 S(\Theta)+\sigma_2 I(\Theta)]dW(\Theta) \rightarrow(1)$$

Where $W(t)$ is the standard Wiener process, $0 < \alpha \leq 1$,
S stands for susceptible
I stands for infected individual
R stands for recovered individual
β stands for transmission rate (from susceptible state to infected state)
γ stands for the rate that an infected individual moves from the state to the recovered state.
Where Wiener process is used to represent the integral of a white noise Gaussian process, and so is useful as a model of noise in electronics engineering (see Brownian noise), instrument errors in filtering theory and disturbances in control theory.
σ_1 is the volatility of S(t)
σ_2 is the volatility of I(t)
b is defined as the rate of birth from infected state to susceptible state.
Let us assume that,
a) S(0) > 0, I(0) > 0 and R(0) = 0.
b) Population size N is constant and it is equal to the sum of individuals in three classes.
c) The ratio between birth and death is one.
d) The rate of moving directly from the infectious state to the susceptible state is equal to that from the recovered to susceptible states.

III. Stochastic integral epidemic model

Consider the case when α=1:

•S(t)=S(0)+$\int_0^t[-\frac{\beta}{N}S(\Theta)+\mathrm{I}(\Theta)\mathrm{b}(\mathrm{I}(\Theta)+\mathrm{R}(\Theta))]\mathrm{d}\Theta+\sigma_1 S(\Theta)dW(\Theta)$,

•I(t)=I(0)+ $\int_0^t[\frac{\beta}{N}S(\Theta)\mathrm{I}(\Theta)-I(\gamma+b)]d\Theta+\sigma_2\int_0^t\mathrm{I}(\Theta)\mathrm{dW}(\Theta)$,

•R(t)=R(0)+$\int_0^t[\gamma I(\Theta)-bR(\Theta)]d\Theta-\int_0^t[\sigma_1 S(\Theta)+\sigma_2 I(\Theta)]\mathrm{dW}(\Theta)$ →*(2)*

We notice that
S(t)+I(t)+R(t)=N=S(0)+I(0)+R(0), see [1-4].
Theorem 1. If β < γ+b, then
$\lim_{t\to\infty}E[I(t)]=\lim_{t\to\infty}E(R(t))=0\,,\quad\lim_{t\to\infty}E(S(t))=N$
Proof. Consider the equation
v(t)=I(0)+ $\int_0^t(\beta-\gamma-b)v(\Theta)d\Theta+\sigma_2\int_0^t v(\Theta)dW(\Theta)$.
Consider the following stochastic differential equation,
dX(t)=$[\frac{\sigma_2^2}{2}-(\beta-\gamma-b)]\mathrm{X(t)dt}-\sigma_2 X(t)dW(t)$,
dY(t)= $[\frac{\sigma_2^2}{2}+(\beta-\gamma-b)]\mathrm{Y(t)dt}+\sigma_2 Y(t)dW(t)$
The solution of these equations are given by
X(t)=$e^{-\sigma_2 w(t)-(\beta-\gamma-b)t}$
Y(t)= $e^{\sigma_2 w(t)+(\beta-\gamma-b)t}$
Set $v^*(t)=X(t)v(t)$
$dv^*(t)$=X(t)$dv(t)+v(t)dX(t)+G_1(t)G_2(t)dt$
$=X(t)\big[\big((\beta-\gamma-b)t\big)v(t)dt+\sigma_2 v(t)dw(\Theta)\big]+v(t)\left[\frac{\sigma_2^2}{2}-(\beta-\gamma-b)\right]X(t)dt-\sigma_2 X(t)dw(t)-$
$\sigma_2^2 v(t)X(t)dt$
$dv^*=-\frac{\sigma_2^2}{2}\mathrm{dt}\quad,\ v^*(t)=e^{-\frac{\sigma_2^2}{2}t}I(0)$,
$v(t)$=Y(t)$e^{-\frac{\sigma_2^2}{2}t}I(0)$
$=e^{\sigma_2 W(t)+(\beta-\gamma-b)t}e^{-\frac{\sigma_2^2}{2}t}I(0)$,
E$[v(t)]$ =$e^{(\beta-\gamma-b)t}$I(0)
Thus $\lim_{t\to\infty}E[I(t)]=0$
We notice that:
E[R(t)]=R(0)+ $\gamma\int_0^t E[I(\Theta)]d\Theta-b\int_0^t E[R(\Theta)]d\Theta$,
$consequently$,
$\frac{d}{dt}e^{bt}E[R(t)]=\gamma e^{bt}E[I(t)]$,
e^{bt}E[R(t)]= $\gamma\int_0^t e^{b\Theta}E[I(\Theta)]d\Theta$,
e^{bt}E[R(t)] ≤ $\gamma\int_0^t e^{b\Theta}e^{-(b+\gamma-\beta)}I(0)d\Theta$,

E[R(t)]≤ $\frac{\gamma}{\beta-\gamma}[e^{(\beta-b-\gamma)t} - e^{-bt}]I(0)$.
If β < b+ γ then
$\lim_{t\to\infty} E[R(t)] = 0$
Now S(t)+I(t)+R(t)=N= population size is continuous
E[S(t)]+E[I(t)]+E[R(t)]=N
Thus $\lim_{t\to\infty} E[S(t)] = N$
Theorem 2.
Let S(t), I(t), R(t) be the solutions of the fractional stochastic equation (1).
If $R_0 < 1$,
Then
$\lim_{t\to\infty} E[S(t)] = N$,
$\lim_{t\to\infty} E[I(t)] = 0$, $\lim_{t\to\infty} E[R(t)] = 0$
Proof, consider the equation
$v(t) = I(0) +)+ \frac{1}{\Gamma(\alpha)}\int_0^t (t-\Theta)^{\alpha-1}(\beta-\gamma-b)v(\Theta)d\Theta + \frac{\sigma_2}{\Gamma(\alpha)}\int_0^t (t-\Theta)^{\alpha-1}\, v(\Theta)dW(\Theta)$,
$E[v(t)]$=I(0)+ $\frac{1}{\Gamma(\alpha)}\int_0^t (t-\Theta)^{\alpha-1}(\beta-\gamma-b)E[v(\Theta)]d\Theta$
According to [2], we get
$E[v(t)]=\int_0^\infty \xi_\alpha(\Theta)e^{(\beta-\gamma-b)t^\alpha\Theta}$I(0)d$\Theta$

$=\sum_{j=0}^{\infty}\frac{((\beta-\gamma-b)t^\alpha)^j}{\Gamma(1+\alpha j)}\ I(0)$ 'Mittage leffer function,
So,
$E[I(t)] \le E[v(t)] \to 0$
as t→∞

IV. A model with new fractional Brownian motion

Consider the following model:
dS(t) = $[\mu N - \mu S - W_\alpha^* SI + \gamma I]dt - \sigma SIdW_\alpha(t)$,
dI(t)=$[W_\alpha^* SI - \mu I - \gamma I]dt + \sigma SIdW_\alpha(t)$.
where $W_\alpha(t)$ is the new fractional Brownian motion, constructed by El-Borai and El-Nadi, see [5-14], σ is the standard deviation of the noise and $W_\alpha^*(t)$ is the fractional Ornstein Uhlenbeck process defined by
d$W_\alpha^*(t) = \sigma_3(\beta - W_\alpha^*(t))dt + \sigma_4 dW_\alpha(t)$, see [15],
Where $\sigma_3 > 0$ is the speed of reversion, $\sigma_4 > 0$ is the intensity of volatility.
Set $v(t) = W_\alpha^*(t) - \beta$, we get $dv(t) = -\sigma_3 v(t)dt + \sigma_4 dW_\alpha(t)$
$v(0) = \beta_0 - \beta$, $\beta_0 = W_\alpha^*(0)$.
The solution is given by

$$v(t) = e^{-t\sigma_3}(\beta_0 - \beta) + \sigma_4\int_0^t e^{-(t-s)\sigma_3}\, dW_\alpha(s)$$

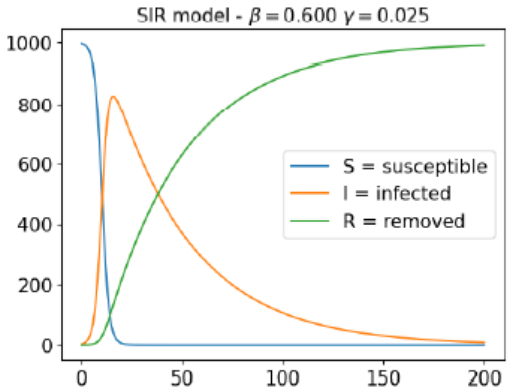
Thus
$W_\alpha^*(t) = \beta_0 + e^{-t\sigma_3}(\beta_0 - \beta) + \sigma_4\int_0^t e^{-(t-s)\sigma_3}\, dW_\alpha(s)$.
The expectation of $W_\alpha^*(t)$ is given by
$E[W_\alpha^*(t)] = \beta_0 + e^{-t\sigma_3}(\beta_0 - \beta)$.
And the variance of $W_\alpha^*(t)$ is:
$var[W_\alpha^*(t)]= \sigma_4^2\int_0^t e^{-2(t-s)\sigma_3}\frac{s^{\alpha-1}}{\Gamma(\alpha)}ds$.
Notice that at α=1,
$E[W_\alpha^*(t)] = \beta_0 + e^{-t\sigma_3}(\beta_0 - \beta)$
$var[W_\alpha^*(t)] = \frac{\sigma_4^2}{2\sigma_3}[1 - e^{-2t\sigma_3}]$
Let us try to find a deterministic function $G(t,\alpha), such\ that\ \left[\int_0^t e^{-(t-s)\sigma_3}\, dW_\alpha(s)\right]dt = G(t,\alpha)dW_\alpha(t)$.
We notice that:
$\int_0^t\left[\int_0^\eta e^{-(\eta-s)\sigma_3}\, dW_\alpha(\eta)\right]d\eta = \int_0^t G(\eta,\alpha)\ dW_\alpha(\eta)$=
$=\frac{1}{\sigma_3}\int_0^t[1 - e^{-(t-s)\sigma_3}]\ dW_\alpha(s)$,
$\frac{1}{\sigma_3^2}E[\{\int_0^t[1 - e^{-(t-s)\sigma_3}]\ dW_\alpha(s)\}]^2$=$E[\{\int_0^t G(\eta,\alpha)\, dW_\alpha(s)\}^2]$= $\int_0^t G^2(\eta,\alpha)\frac{\eta^{\alpha-1}}{\Gamma(\alpha)}d\eta$

Thus
$\frac{1}{\sigma_3^2}\int_0^t\left(1-e^{-(t-s)\sigma_3}\right)^2\frac{s^{\alpha-1}}{\Gamma(\alpha)}ds=\int_0^t G^2(s,\alpha)\frac{s^{\alpha-1}}{\Gamma(\alpha)}ds$, consequently
$\frac{t^{\alpha-1}G^2(t,\alpha)}{\Gamma(\alpha)}=\frac{2}{\sigma_3}\int_0^t[e^{-(t-s)\sigma_3}-e^{-2(t-s)\sigma_3}]\frac{s^{\alpha-1}}{\Gamma(\alpha)}ds.$

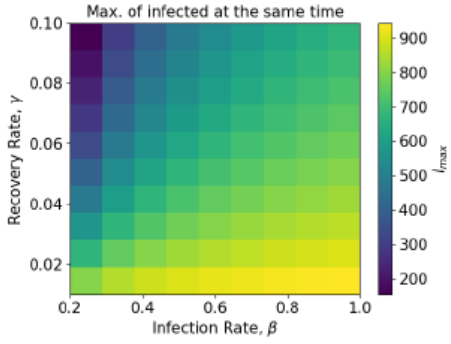
We have now the following expression for the stochastic process $W_\alpha^*(t)$:
$W_\alpha^*(t)dt=[\beta+(\beta_0-\beta)e^{-t\sigma_3}]dt+\sigma_4\mathrm{G(t,}\alpha)\,dW_\alpha(t).$
Since given that S(t)+I(t)=N, it is sufficient to study the fractional stochastic equation for I(t):
$dI(t)=[\{\beta+(\beta_0-\beta)e^{-t\sigma_3}\}I(N-I)-I\mu-I\gamma]dt+\sigma_4\sigma\mathrm{I(N-I)}dW_\alpha(t)$ (2)
With the initial condition $I(0)=I_0\in(0,N)$
Theorem. For any given intial value $I_0\in(0,N)$, the fractional stochastic differential equation (2) has a unique global positive solution $I(t)\in(0,N)$ for all $t\geq 0$, with probability one,
$p(I(t)\in(,N),\forall t\geq)=1$
Proof. Consider the following fractional stochastic model
$du=\{\beta_0+(\beta_0-\beta)e^{-t\sigma_3}(N-e^u)\}du+\left\{-(\mu+\gamma)dt-\frac{1}{2}\sigma^2\sigma_4^2\frac{t^{\alpha-1}}{\Gamma(\alpha)}(N-e^u)^2\right\}dt$ (3),
With the initial value $u(0)=I_0$
It is clear that the coefficient of model (3) satisfy the local Lipschitz condition ,thus there is a local solution $u(t)$, of the model (3), see [16].
Therefore it is easy to check that $I(t)=e^{u(t)}$ is the positive solution of model (2) with the initial value I_0.

V. Some examples of SIR Models

Let
S = y[0] ==> Number of individuals not yet infected
I = y[1] ==> Number of individuals infected
R = y[2] ==> Number of individuals recovered or killed by the disease
beta = Infection Rate (0.6)
gamma = Recovery Rate (0.025)

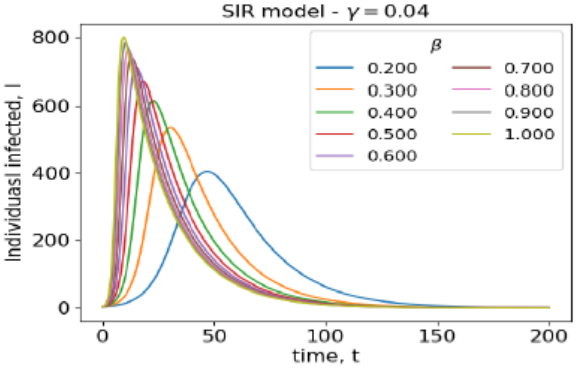


Plot a colormap for maximum number of individuals infected



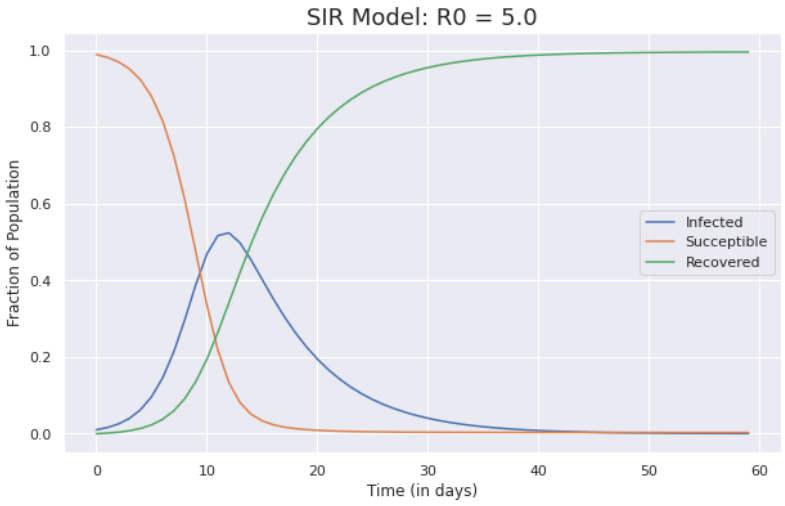
The frame

Plots for gamma = 0.04,
beta= 0.2,0.3,…,1



The framework separates the population into categories.
S: Susceptible refers to a group of persons who are susceptible to the infection.
I: Infectious population refers to the group of persons who can spread the viral infection.
R: The recovered (also known as removed) population is made up of persons who are no longer infectious; this comprises both the recovered population and those who died as a result of the epidemic.
A major assumption of the concept is that a recovered individual develops immunity to the pandemic.
We consider The total population (N) is considered to be one.The initial state for the infectious category (I0) is the proportion of the whole population that is infected at time T0. The initial state for the susceptible population (S0) is the remaining population (N-I0), presuming no one has been vaccinated. It is also believed that there is no found individual at the start. The model also assumes that the population remains constant, meaning that there are no additional births or deaths due to causes other than the pandemic.
Epidemic begins to spread. New cases emerge. The pandemic spreads when a susceptible individual comes into contact with an infectious person.The number of new infections is proportional to both the infection and vulnerable populations. New infection = beta * S * I -> (1), where beta represents the transmission rate.New recoveries happen. A subset of the infectious group ceases to be infectious as they die or recover (we will refer to them as the recovered population). The number of recoveries is determined by the total number of infected people. New recovery = gamma * I -> (2), where gamma is the recovery rate.
How do new infections and recoveries affect the S, I, and R groups?
The susceptible population shrinks as new infections emerge.
S[T+1] = S[T] - new infections.[T]
The infectious population grows with new infections and declines with new recoveries.
I[T+1] = I[T] plus new infections.[T] - New Recovery.
The recovered population grows as new recoveries occur.
R[T+1] = R[T] + New Recovery
Epidemic or not?
An infection becomes an epidemic when it spreads over time, meaning that the number of new infections exceeds the number of new recoveries.
New infections lead to new recoveries (beta * S * I > gamma * I), as seen in (1) and (2).
The effective reproductive number is defined as S * beta / gamma > 1.
At the start of an epidemic, almost the entire population is vulnerable, resulting in S=S0=1.
Reproduction number = beta / gamma, often known as the fundamental reproduction number (R0).
If R0 > 1, the infection becomes an epidemic; otherwise, it dies off.

SIR Model for R0=5
R0 for Covid-19 is estimated to be between 2.2 to 5.7 (Source Forbes Report)
We will model a scenario where R0 is 5 and Initial state of infectious population is 1%.
We use beta=0.75 and gamma=0.15 to obtain the desired R0 of 5.0



Maximum Infectious population at a time :52.43%
Total Infected population :99.69%

Observation:

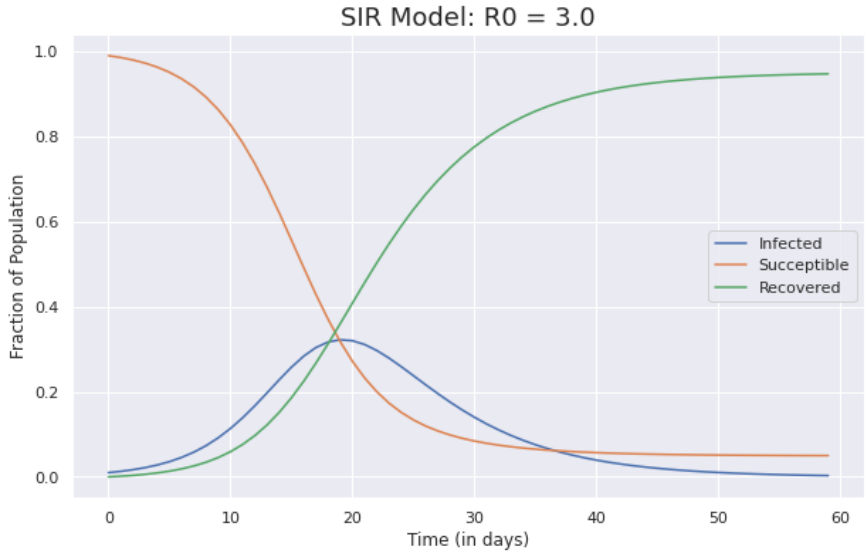
- Peak infectious population is 52.43% around 12th day
- Epidemic gets over with entire population getting infected
-

SIR Model for R0=3

Now let's examine a case where R0 is reduced to 3.
Since R0=beta/gamma, R0 reduction is achieved by:

- a. Decreasing beta
- b. Increasing gamma
- c. Both a and b simultaneously

Here we will just reduce beta to 0.45 keeping gamma at previous rate of 0.15 to reduce R0 to 3

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Maximum Infectious population at a time :32.22%
Total Infected population :95.01%
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Observation:

- Peak infection population gets reduced from around 50% to around 30%
- Delay in peak of infection, infection peaked at around 12th day when R0=5, whereas it took around 20 days to reach peak of infection when R0 is reduced to 3.
- Around 5% of the total population remains uninfected when the infection ends.

SIR Model for R0=1.5

Let's explore the nature of epidemic when R0 is furter reduced to 1.5.
We will achieve this by setting gamma=0.3


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Maximum Infectious population at a time :7.28%
Total Infected population :60.07%
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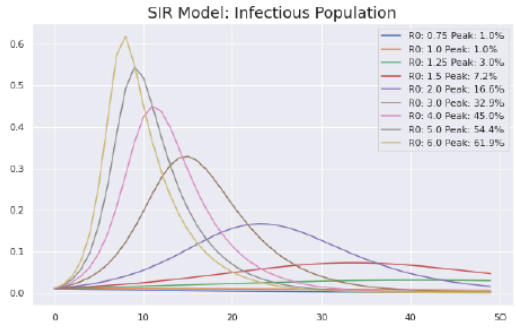
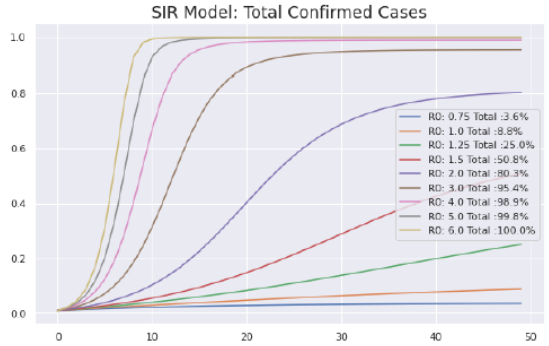
Observation:

- Peak level of infectious population is only 7.28% which is much lower than the previous case.
- Infection peaks around 25th day.
- Only around 60% of the total population is infected.

Flatten the curve.

Flatten the Curve is a well-known phrase nowadays, and this is exactly what we did above. We can see that decreasing R0 reduces the transmission of the virus and the highest level of infected people. Decreasing R0 will also postpone the peak level of infection, giving us crucial time to plan to combat the illness efficiently. Let's now look at how the peak level and extent of infection vary with R0.





As R0 drops, the curve for infectious population flattens. When R0 is 6, more than 60% of the population becomes infected by the eighth day, whereas when R0 is 2, the highest infectious population is 16.7% by the twenty-fourth day. That means a reduction in R0 from 6 to 2 provides us an extra two weeks to prepare for the peak infection. It is also worth noting that when R0 falls, so does the degree of infection. When R0=6, the entire population becomes infected, however when R0=2, 20% of the population remains uninfected after the epidemic has ended.

Despite being a relatively non-lethal infection, COVID-19 wrought devastation around the world since it quickly spread and caught several countries off guard, overwhelming their health-care systems. Health infrastructure is considered to be overwhelmed when the number of infected patients exceeds the number of hospital beds available. Yes, the health infrastructure may be rapidly expanded by transforming existing structures into makeshift hospitals and rehiring retired health workers, among other things. Many countries follow as well, but they will be unable to keep up if the number of instances increases rapidly. The faster the infection curve climbs, the sooner the local health-care system becomes overburdened beyond its capacity to treat patients. As we observe in Italy, Spain, and the United States, an increasing number of new patients may be forced to leave without ICU beds, and hospitals may run out of the basic resources required to respond to the outbreak.

VI. Conclusion

*Some stochastic fractional epidemic models are considered. The existence and properties of solutions of the considered models are studied. Also stochastic models with the new fractional Brownian motion are studied***.**

Conflicts of interest

The authors have declared that no competing interests exist.

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