Modelling CoVid-19 Epidemic Spread on Social Networks

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Abstract

In this project, we have attempted to study the time evolution of the ongoing Covid-19 pandemic. We have worked with the **SAIRD model** and compared the simulated data with real epidemic data of New Zealand. Moreover, we have undertaken two distinct approaches-deterministic and stochastic, to simulate a human social network and their interactions. Also, we have incorporated the impact of different lockdown scenarios in mitigating the spread of disease. The understanding of epidemics in complex networks might deliver new insights in the spread of information technological networks which often seem to possess similar network topology.

1 Introduction

The Covid-19 pandemic has been unprecedented in terms of the sheer impact it has had on the human population and institutions. As of June 2020, the disease has spread to almost every nation and proved to be quite a challenge for governments alike. Scientists across domains are currently working to develop feasible solutions to tide over this tough period. Apart from medical studies and vaccine development studies, there has been significant emphasis on modelling studies which can predict the trends of the pandemic and help policy-makers in making correct decisions to tackle this challenge. Study of epidemics in human populations is complicated by the fact that human societies are complex and stratified and human interactions are stochastic to some extent. Nonetheless, there has been an abundance of studies simulating the disease spread on networks and fitting the parameters to explain actual data.

In this project, we have proposed an epidemic model called the SAIRD model. We have proceeded to model a human social network structured into families, societies and communities etc. and simulated the spread of the disease on the same network through deterministic and stochastic approaches. The parameters are fitted once again to obtain a better agreement to the actual data. The results are summarised in the sections that follow.

2 The SAIRD Model

The SAIRD model is a variant of the SLIRD model proposed by Sheng et al.[2].This model considers the population of N individuals categorised into 5 mutually exclusive classes:

- **S - Susceptible**
  The native population of the model who are at the risk of contracting the disease when they come into contact with infected individuals.
- **A - Asymptomatic**
  The individuals exposed to the disease who possess a strong immune symptom and do not develop symptoms and eventually recover without hospitalisation. However, due to the infectiousness of the pathogen, they are capable of spreading the disease through their contacts or while they cough, sneeze etc. Such individuals may not necessarily be detected by the medical system and can continue spreading the disease for long.
- **I - Infected**
  The individuals exposed to the disease who eventually require hospitalisation and intensive care. They carry a non-zero probability of succumbing to the disease. They are also capable of spreading the disease.
- **R - Recovered**
  The individuals who recover from the disease.
- **D - Dead**
  The individuals who succumb to the disease and die.

3 Differential Equations

The differential equations governing the change of the various categories are as follows:

$$\frac{dS}{dt} = -\beta_A \frac{S(t)}{N} A(t) - \beta_I \frac{S(t)}{N} I(t) \tag{1}$$

$$\frac{dA}{dt} = \beta_A \frac{S(t)}{N} A(t) - \alpha_A A(t) \tag{2}$$

$$\frac{dI}{dt} = -\alpha_I I(t) - \gamma_I I(t) + \beta_I \frac{S(t)}{N} I(t) \tag{3}$$

$$\frac{dR}{dt} = \alpha_I I(t) + \alpha_A A(t) \tag{4}$$

$$\frac{dD}{dt} = \gamma_I I(t) \tag{5}$$

To get a coarse estimate of the various parameters, we solve the differential equation numerically using standard RK4 algorithm while varying the parameters. A few arguments are presented in section 5.1 for the determination of the coefficients.
Armed with a preliminary estimate of the parameter values, we then proceed to solve the

system of differential equations in the case of a more realistic scenario of a structured human population. To accurately model the disease spread in a human population, the structure of human society also needs to be taken into account. Human interactions are complex in nature and network analysts have spent considerable effort in modelling them. Ever since Milgram's experiments, several network models have been proposed which capture social networks. Such network models are referred to as small world networks and notable among them are Watts and Strogatz, and Barabasi and Albert model.[1]

Such models incorporate the fact that human social networks are often observed to possess high clustering coefficients, low geodesic path distance and power-law degree distributions.

But as we will see below, such power-law degree distributed network are not suitable for epidemic modelling.

4 Network Structure

4.1 Adjacency matrix

The most dominant reason for spread of infection is because of proximity and physical contacts between individuals in a population. Based on this argument our initial attempt will be to **categorize these physical interactions**. We can consider the following scenarios: interactions of members inside a family, members of different families in a building, members living in the same locality and so on. This interaction system hints at a community structure in the network, where we can group the individuals at different community levels.

Thus we propose the following adjacency matrix in Figure 1.

The shades in the adjacency matrix denote the weights of the links between the individuals. The darkest shaded regions in the matrix corresponds to the strongest connections within a level 1 community(family), the level 2 connections are denoted by the lighter shaded regions in the matrix and so forth. Thus we would say, level 1 communities consist of 2 individuals, level 2 consists of 3 level 1 communities, level 3 consists of 4 level 2 communities in the network. The community structure of the adjacency matrix shown in figure 1 is (2,3,4).

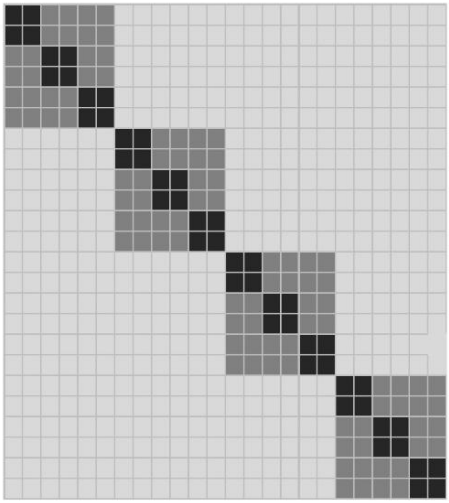


Figure 1: Proposed Structure of Adjacency Matrix

4.2 Determining the entries of the adjacency matrix

It is quite evident that to fill the entries in the adjacency matrix we need to use a function which is dependent on the level of interaction such that weight of the connections decrease as we move to higher levels. This can be effectively modelled by an exponential function, where the argument of the exponent contains the level of interaction i.e.

$$f(c, x) = e^{-cx} \tag{6}$$

where c, denotes the level of interaction

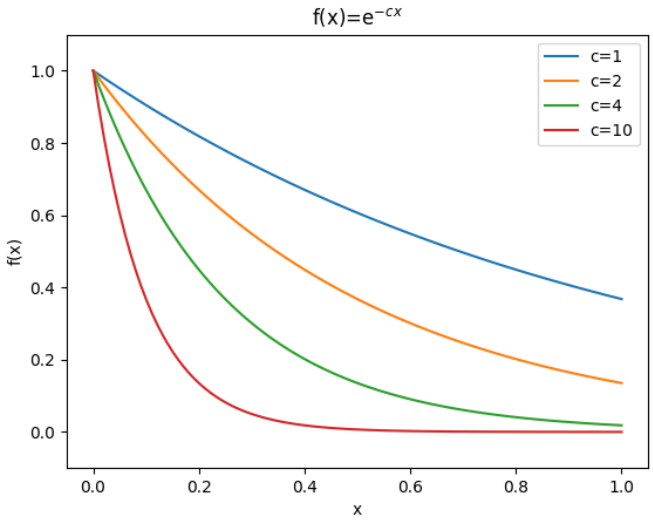


Figure 2: Probability Distribution Function

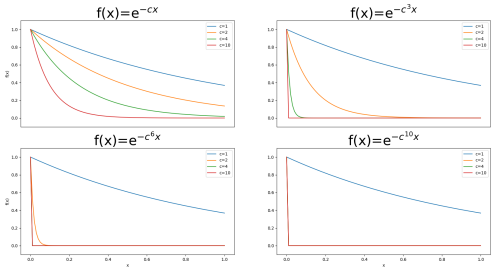
If the argument x is a uniformly generated random number between 0 to 1, then, **higher the level of interaction, lower is the probability of making a strong link and lower the level of interaction, higher is the probability of making a strong link**. An adjacency matrix with the entries as weights of the connections, as shown in figure 1, can be generated by using a particular value of x instead of using random numbers. The uniform random number as an argument of the exponential gives us variability in the interactions that take place in the same level which depicts the variability observed in real physical interactions.

4.3 Lock-Down in the Network

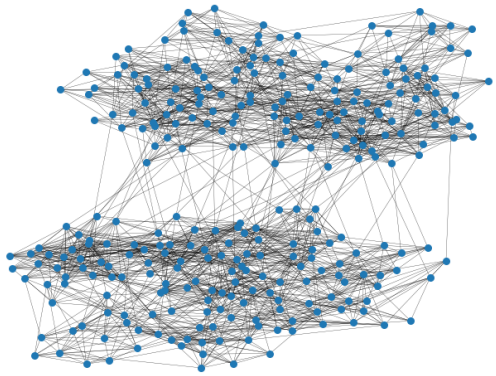
Considering the effects that Lock-Down has on social interactions that are physical in nature, we need to modify our function defined in the previous subsection such that the **links in level 1 remains unaffected, while the links get increasingly hampered for higher level interactions**. This can be effectively modelled by a modification to the above function,

$$f(c, ld, x) = e^{-c^{(ld+1)}x} \tag{7}$$

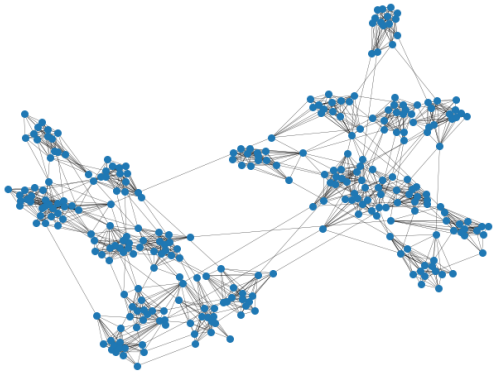
where, (ld) loosely characterizes the strength of the lock-down. Note that the exponent is $-c^{(ld+1)}x$.



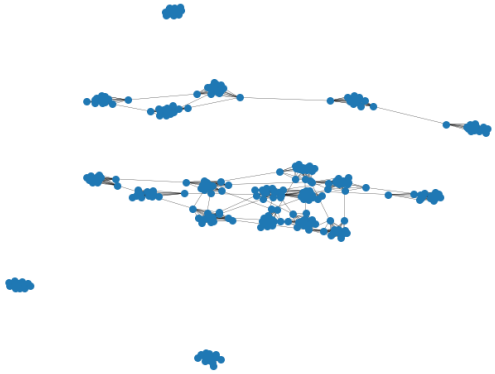
(a) Probability Distribution function for various levels of Lock-down



(b) Demo of a network having community structure (15,10,2) with (ld)=4



(c) Community Structure (15,10,2) with (ld)=6



(d) Community Structure (15,10,2) with (ld)=8

5 Deterministic Simulation

The set of differential equations mentioned in section 3 depicts our model of infection in a society where every node has an equal probability to interact with any node of the network. Be warned of a caveat in the differential equations of section 3. On close inspection equation 1,2 and 3 reveal that the increment of I is brought about by the term $\beta_I \frac{S(t)}{N} I(t)$ and increment of A is brought about by the term $\beta_I \frac{S(t)}{N} A(t)$. So, if one considers the initial population to consist of only one I(A) at the start and zero A(I), then the population evolves to have a considerable amount of I(A) but no A(I). This also means that an **individual affected by an I(A) will turn out to be an I(A) in a later time step**, this is not at all acceptable. Thus, we add another extra stage in the model which will couple A and I, namely L(latent). Thus, our set of differential equations take the form:

$$\frac{dS}{dt} = -\beta_A \frac{S.A}{N} - \beta_I \frac{S.I}{N} \tag{8}$$

$$\frac{dL}{dt} = \beta_A \frac{S.A}{N} + \beta_I \frac{S.I}{N} - \theta_I L - \theta_A L \tag{9}$$

$$\frac{dI}{dt} = \theta_I L - \alpha_I I - \gamma_I I \tag{10}$$

$$\frac{dA}{dt} = \theta_A L - \alpha_A A \tag{11}$$

$$\frac{dD}{dt} = \gamma_I I \tag{12}$$

$$\frac{dR}{dt} = \alpha_I I + \alpha_A A \tag{13}$$

L denotes the stage, where the individuals who have contracted the virus initially arrive at. Then, depending on the value of θ_A and θ_I the contracted individuals are bifurcated into Asymptomatic or Infected.

5.1 Simulation with No Population Structure

The estimate of the constants have to be such that the end results match with the observed values in the real world. There are a few constraints on the parameters which are as follows:

- $\theta_I + \theta_A < \beta_A + \beta_I$: L is just a stage which we have included to counter the problems that are explained before. But this state can be advantageously used to loosely compare the average 5 days required for the virus to show its symptoms.

- $\gamma_I < \alpha_I + \alpha_A$: This has to be followed since the mortality rate of the virus is found to be within 10%, with average somewhere near 3-4%.

- $\theta_I < \theta_A$: There are very few severe cases which require hospitalization.

- $\theta_{A/I} > \alpha_{A/I}$: Since we need the individuals to stay in the stages I and A so that they can pass the virus to S.

Considering these, we have chosen the following set of parameters in solving the differential equations, $\beta_A = 0.6, \beta_I = 0.1, \theta_I = 0.04, \theta_A = 0.08, \alpha_I = 0.015, \alpha_A = 0.015, \gamma_I = 0.02$.

As we can see, the preliminary plots without any network structure gives us a very good match with the observed data of infected and recovered individuals, with a mortality rate of 4.08%.

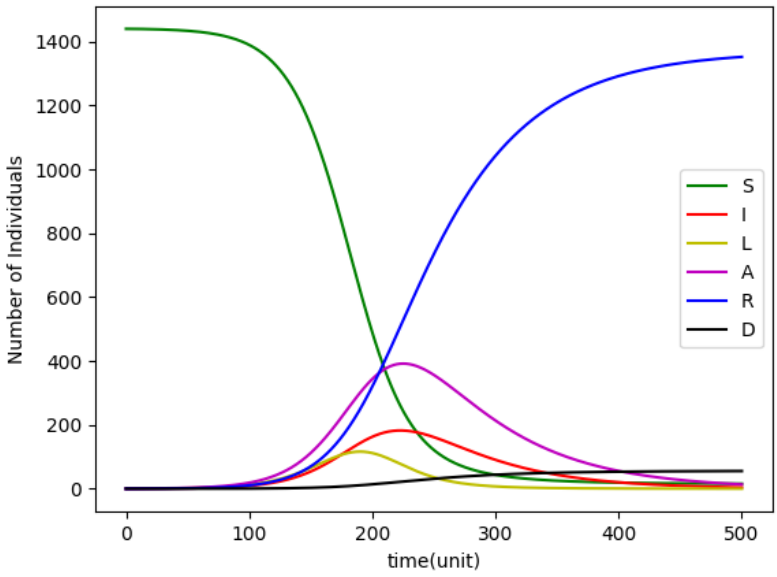


Figure 3: Plot of the different stages of the infecton vs time

5.2 Simulation with a Population Structure

The differential equations in the previous subsection corresponds to a well mixed population with no underlying network. To incorporate the adjacency matrix in the model we need to consider the terms in the differential equation which are dependent on the linking of the underlying network. On inspecting the equations, it is quite evident that **the contact terms between S I and S A are the ones which are dependent on the underlying network structure**. Hence, $\frac{dS}{dt}$ and $\frac{dI}{dt}$ can be written as [1]:

$$\frac{d\vec{S_i}}{dt} = -\beta_A \vec{S_i} \sum_j (Adj)_{ij} \vec{A_j} - \beta_I \vec{S_i} \sum_j (Adj)_{ij} \vec{I_j} \tag{14}$$

$$\frac{d\vec{L_i}}{dt} = \beta_A \vec{S_i} \sum_j (Adj)_{ij} \vec{A_j} + \beta_I \vec{S_i} \sum_j (Adj)_{ij} \vec{I_j} - \theta_I \vec{L_i} - \theta_A \vec{L_i} \tag{15}$$

The other equations can similarly be written with a slight modification by adding the node subscript given by 'i'. A few comments for the simulation are in order. This can be qualitatively understood by considering the 'i'th entry of S,L,I,A,R and D column matrix as the amount of the character that the node i has.

- **Comment 1**: In simulation of the deterministic equations on a network structure the probability distribution function has been modified such that higher level connectivity in nodes are greatly diminished to counter the positive feedback loop of infection. The feedback mechanism has been explained in the comment 2.

$$f(c, x) = e^{-(15(c-1)+1)^{2+(ld)}x} \tag{16}$$

A qualitative argument is, f(x) needs to capture the strong connections in level 1 communities while vastly decreasing the connectivity in higher levels. Although the use of 15 for scaling can be argued.

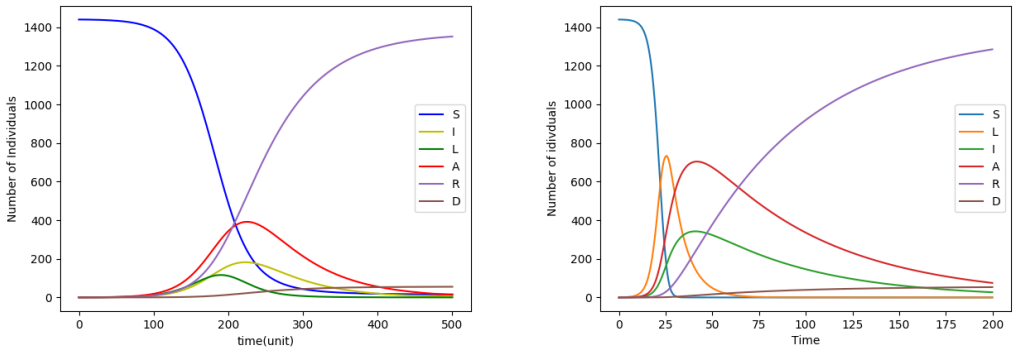
- **Comment 2**: If we consider the edges to be weighted edges then a non-zero value of L will be attained by all the nodes in the network. Since, the diagonal entries are 0 in the adjacency matrix, thus there is no sink for L or A or I (except D and R, that requires going through I or A), which will create **a strong positive feedback loop in the level 1 communities in the network promoting infection even after a strict Lock-Down. Thus, instead of using weighted edges we have fixed a threshold value for the entries of the adjacency matrix. The threshold acts as a step function s.t. entries of the matrix having value below it will be 0 and above it will be 1**. The threshold is set to be 0.5. This along with the modified function resolves our problem of uncontrolled leaking of the virus.

- **Comment 3**: Following the above argument, it is natural that implementation of Lock-Down will not abruptly decrease the spread of infection because of the strong feedback loop in the lower level connections.

In the plots to follow, a few choices were made concerning the restructuring of the network and implementation of the Lock-Down.
The network structure is chosen considering the constraint that we have limited computational strength and there should be enough levels to counter the infection after Lock-Down.

5.2.1 No Lock-Down

The scenario in which the individuals in the network are indifferent regarding the spread of the infection, no Lock-Down(LD) is observed, ld=0. The network is restructured every 25 time steps.



(a) Spread on a structured network (3,4,5,6,2,2), restructuring time period=25

(b) Spread without a network

As we can see, there are a few difference in the evolution of the epidemic in a network structure and without a network.

Observations

- **Difference in duration of epidemic** : The epidemic takes 200 time steps to affect the entire population, whereas, without a network structure it takes 500 time steps for completion.
- **Sharp spike in L** : The L stage has a spike in case of a network structure, while it does not in case of no network.
- **Asymmetry in inflow and outflow for I and A** : In case of no underlying network, the the graphs for I and A are almost symmetric; but in the scenario of an underlying network the nodes spend substantially more amount of time in the transition states of I and A after getting infected very fast.

Explanation

In the absence of a population network, the nodes are equally likely to interact with all the other nodes in the network, with a probability 1/N; while, **in the presence of a network structure, only the neighbours of a node are involved in the interaction**. Now if we consider the complete network with weights 1/N, then imagining the adjacency matrix, **the overall degree of each node can be calculated to give** $\frac{N-1}{N} \approx 1$, whereas, **in case of a network structure the overall node of a degree is a few times more than 1 which is hugely responsible for spreading the disease at a very fast rates through lower level connections**.

Absence of LD and constant restructuring at regular periods also facilitates a very rapid spread of the virus at higher level connections. This can explain the faster evolution, the spike in L and the asymmetry in the inflow and the outflow rate of stage I and L.

5.2.2 Successful(?) Lock-Down

Currently there has been a lot of success in containing the virus in countries like Italy, Germany, New Zealand etc. In this subsection we have used our model and our network structure to simulate a LD in the population which will contain the epidemic as seen in the real world.

The parameter constants of our model selected in 5.1 is used in the simulation with additional parameters involving the network restructuring that are argued and discussed below. The LD in these cases is dictated by two params: the strength of the LD, (ld) and the time period of the network restructuring within the LD.

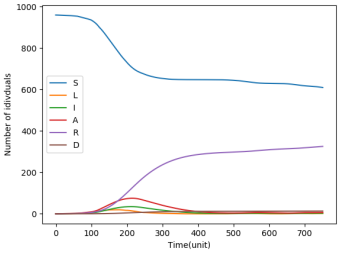
- **Strength of LD** It is quite evident that the strength of LD, (ld) as shown in section 4.3 in equation 7 decreases the probability of the formation of a link with distant neighbours. But after a certain value of (ld) only the level 1 families will be left, thus further increase of (ld) will not have any effect on the system.

- **Restructuring time period** Restructuring of a network mixes the links between the nodes and lower level communities, implies a higher rate of restructuring can intensify the spread of the virus. There are two types of restructuring to be considered here, restructuring before LD (ReBLD), and restructuring after LD (ReALD). Since a moderately high value of ld can decompose the network into just the level 1 communities ReALD does not have a vital role, whereas ReBLD has a vital role in the epidemic magnitude.

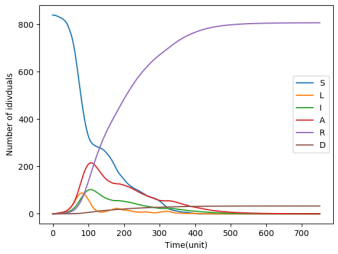
- **Community Structure** The community structure has a vital role in the epidemic. Let us consider a **community structure with descending order of level numbers e.g. (5,4,3,2) is more prone to spreading due to an overall high degree and clustering coefficient. Whereas, in networks with the opposite structure e.g. (2,3,4,5), the average degree of the nodes are much lower with very low clustering coefficient, making it very successful in containing an epidemic**.

Note 1 : **There is a very subtle balance between the community structure used and the strength of LD. If the level values are in an ascending order then low ld values are enough to contain the spread, whereas, community structure in a descending order requires very high values of ld to contain the epidemic**.
Note 2 : The restructuring time period before the imposition of LD (ReBLD) has a very dominant contribution on the overall magnitude of the epidemic. The descending level structure e.g. (5,4,3,2) will have a much more considerable spread than the ascending level structure of (2,3,4,5); which is quite expected and obvious considering **the higher clustering coefficient can spread minute quantities of the virus in several regions of the network before an imposition of LD**.
A few plots have been attached which explores the parameter space of the network and shows that proper selection of these 4 parameters corresponding to individual countries can give us a really good estimate of the epidemic magnitude in those respective countries.**The parameter space is explored by fixing three parameters and varying the other**.
The contribution of a parameter on the outcome of the epidemic is shown in a descending fashion. The threshold value for the imposition of a LD is set at 10 individuals.

Note that a shortcoming of our simulation is the computational power. Our simulation runs over a small community structure which implies a total population of 500-1500, which is nowhere close to the million inhabitants in a country. Thus, most of our S population gets infected while in the real scenario the total number of I and A people is a minute percentage of the total S in the country. To determine the epidemic growth in a particular country we require a substantial amount of data to understand the community structure at play in the country.

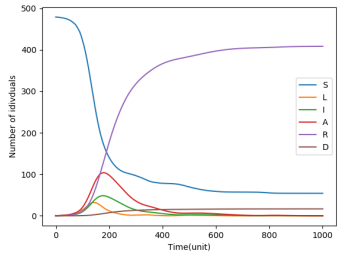


(c) ld=25, ReALD=25, ReBLD=25, Community Structure=(3,4,5,4,4)

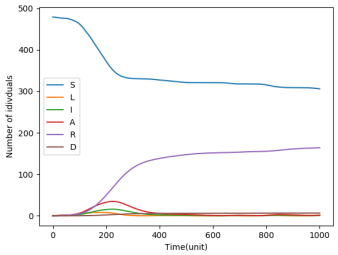


(d) ld=25, ReALD=25, ReBLD=25, Community Structure=(8,7,6,3)

Figure 4: Variation in the community structure affecting the outcome of the epidemic, refer to note 1

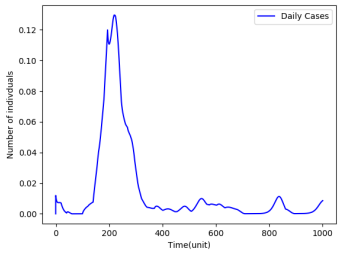


(a) ld=100, ReBLD=5, ReALD=500, Community Structure=(3,4,5,4,2)

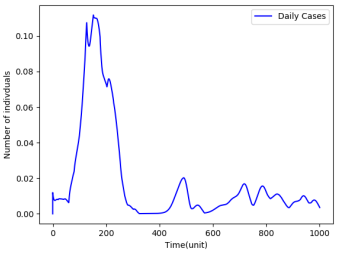


(b) ld=100, ReBLD=25, ReALD=500, Community Structure=(3,4,5,4,2)

Figure 5: Epidemic magnitude depending on ReBLD, refer to note 2

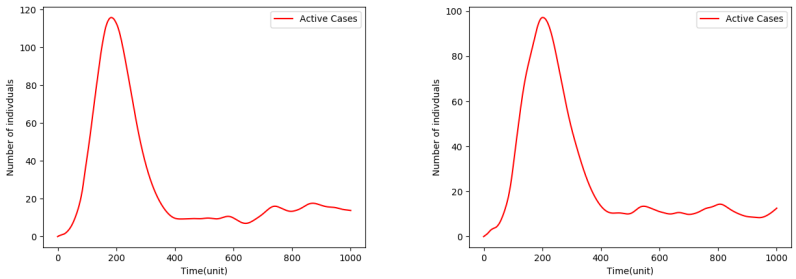


(a) ld=100, ReALD=25, ReBLD=25, Community Structure=(3,4,5,4,4)



(b) ld=5, ReALD=25, ReBLD=25, Community Structure=(3,4,5,4,4)

Figure 6: Increase in the fluctuation of Daily Cases brought by the LD parameter (ld) due to sudden links among isolated families



(a) ld=4, ReALD=5, ReBLD=25, Community Structure=(3,4,5,4,4)

(b) ld=4, ReALD=150,ReBLD=25, Community Structure=(3,4,5,4,4)

Figure 7: Increase in the peak of Active cases because of lowering the Restructuring time period during LD. This is the least dominant contribution as it depends on the value of (ld).

5.2.3 Recurring Outbreaks

Currently we have been seeing the possibility of a second wave of the infection in countries like China, South Korea, Japan, New Zealand etc. This recurring spread of infection can be quite effectively modelled in our simulation by controlling the network restructuring. For simplicity we have assumed that **a constant threshold value (chosen to be 8 in the figure below) of infected individuals will decide if a Lock-Down will be imposed or removed**. The following plot was obtained on a (3,4,5,6,2) network with restructuring before LD (ReBLD)=30 time steps and ReALD=150 time steps, showcasing transition between strict LD and no LD.

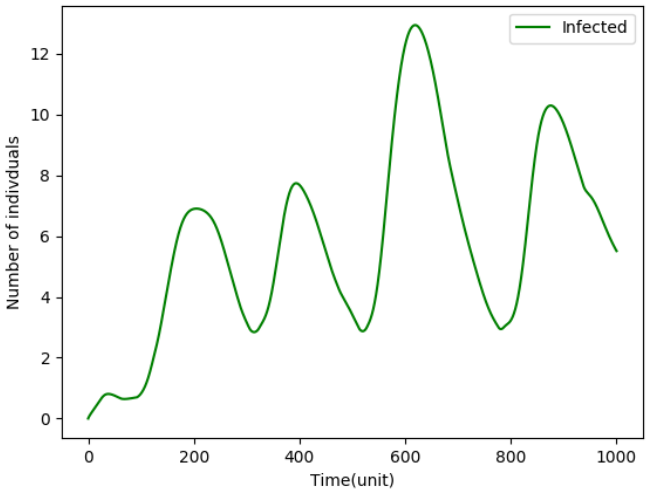


Figure 8: Waves of epidemic, increasing the infected individuals at regular time intervals

As we can see, the epidemic returns in waves as concluded by other studies. This primarily happens due to the immediate spread of the virus on removal of LD. In the real world we

can expect such a situation to happen, considering the presence of untested assymptomatic cases and influx of virus carying individuals.

These deterministic simulations on a network are partially stochastic because of the network, which give rise to the fluctuations that we can see in the plots.
A detailed study of the fully stochastic process is carried out in the following section.

6 Stochastic Simulation

The stochastic approach is another commonly used alternative to model disease spread phenomenon. Unlike the deterministic case where rate of infection is governed by a set of differential equations, here the states of the population vary randomly. The determining factor is the infection probability,β when two nodes are connected by an edge.

We have made some extra assumptions to simplify the calculations and conserve computing resources. Firstly, infection is spread when two nodes are in contact with a probability of β i.e. β is the probability a Susceptible (S) in contact with either Asymptomatic(A) or Infected(I) contracts the disease.

We have assumed that β is same for both A and I spreading the infection but I are able to spread the disease for a maximum of 5 days since exposure, because they are isolated/quarantined once they start exhibiting more serious symptoms. However, A do continue to spread the disease throughout their infectious period until recovery. We will later see how this leads to a runaway effect of rapid increase of infections within a very short period of time. We hypothesise that this is the likely cause of what is known in epidemiology as "Community Transmission."

Secondly, with a probability of γ, S exposed to either A or I, become A. We choose γ close to 1 as global trends reveal asymptomatic outnumber people with symptoms.

Thirdly, the recovery time is modelled with a gamma distribution with mean=14 days for A and mean=18 days for I with a common shape parameter k=4.[4] Since the gamma distribution is right skewed, so we have truncated it appropriately at 20 days and 25 days respectively so as the loop does not run infinitely. Also, we have argued that as recovery time of I is directly correlated with their health, so individuals taking more time to recover are likely to succumb eventually. We have defined a 'death probability function' as:

$$g(t) = \frac{1}{1 + e^{-t+c}} \quad \text{where, t = time since infected} \tag{17}$$

c is fitted from actual data to obtain an empirical estimate of the death probability at time = t days.

We have applied our stochastic model to the case of New Zealand. New Zealand has shot into global limelight for its success against the pandemic. Strict measures and responsible actions of its citizens has helped ensure to keep its cases low and deaths to a minimum. In fact, New Zealand was one of the first countries to impose a strict lockdown even when its case numbers were quite low.

Our social network algorithm allows us to create a social network structure with any number of strata and any number of units in each strata. Low population of the country

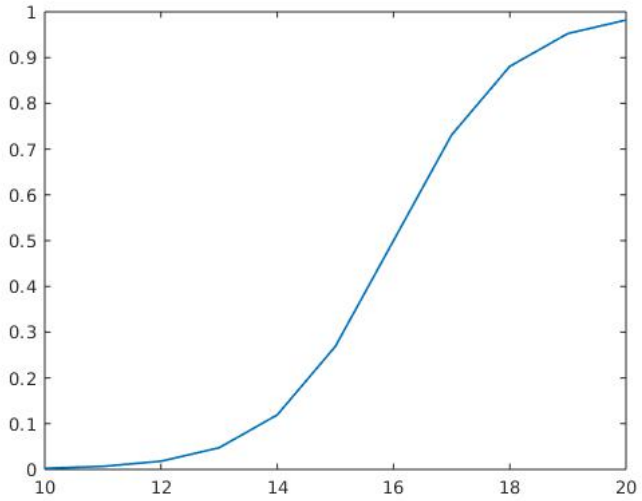


Figure 9: Plot of f(t); (as t nears day 20, probability of death increases to almost 99%)

has made it possible to simulate its social structure and study the spread of the disease. We have created the network structure of New Zealand after several considerations as follows: A closer look at the data reveals that the most number of cases were detected in the largest of the 5 cities.[5] Also, with an average family size of 3 [5], we have considered a community structure of 3,10,10,5 i.e. 3 individuals constitute a family,10 of them a neighborhood, 10 such neighbourhoods where infection spread in the 5 largest cities to capture the maximum possible data. Needless to say, this is somewhat an oversimplification and the community structure is merely a hypothesis.

Once all the considerations were taken into account, we developed a code to evolve the nodes of the network. At each time step, assign them a status $\in$ S,A,I,R,D.

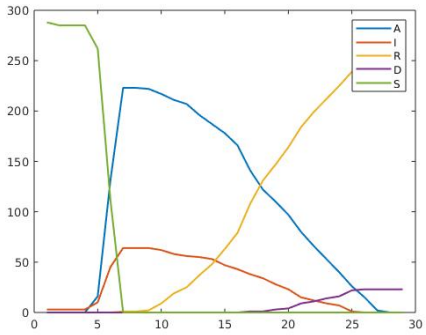
- If the node is S, calculate the number of nodes which are A(say n_1) and number of nodes which are I and time since infection is less than 5 days (say n_2), then probability of exposure $= 1 - (1 - \beta)^{n_1+n_2}$. Probability of turning into $A = \gamma(1 - (1 - \beta)^{n_1+n_2})$.
- Probability of recovery of A at t days $= \frac{F(t+0.5)-F(t-0.5)}{F(20)}$ where F(t) is the cdf of gamma$(4, \frac{14}{4})$ .The scale parameter is obtained by dividing mean by shape parameter.
- Probability of removal of I at t days $= \frac{F(t+0.5)-F(t-0.5)}{F(25)}$ where F(t) is the cdf of gamma$(4, \frac{18}{4})$. Further, the P(death | removed at t days) = g(t).

We introduce the germ in 5 individuals initially. We generate uniform random numbers in (0,1) to simulate probabilities and determine the status of the nodes accordingly. We run the code in a loop until all members are either R, D or S. We have also incorporated the effects of three different lockdown scenarios in our code – no lockdown, multiple lockdowns and strict total lockdown. Since the New Zealand data series holds for the third scenario,

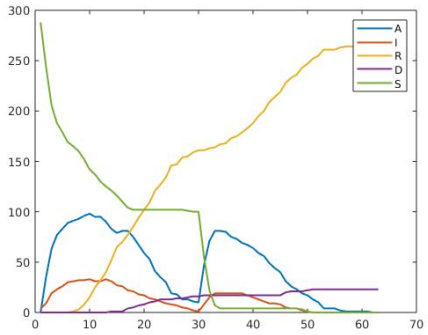
we proceed accordingly and execute the code. As with any stochastic approach, the code is run several times and the average is plotted to reflect the trends.

6.1 Observations

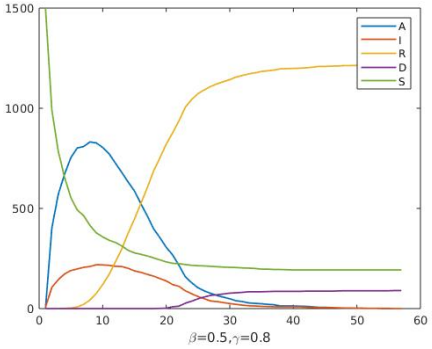
The stochastic approach yields a relatively good fit to the actual data. After several runs of the code, we arrive at a value of c = 18 in g(t) to match the observed death rate. The real shortcoming is that with our population of 1500 individuals, a high fraction of individuals contract the disease; however with a population of 5 million , New Zealand's cases are only a fraction of the total population. It is worth noting however that the entire population may not belong to a single giant component of the network, so infection cannot spread far. Also , with a strict lockdown, New Zealand restricted its population to a great degree as our LockDown code does.



(a) No lockdown



(b) Multiple successive lockdowns



(c) Total Strict Lockdown

Figure 10: Plots of CoVid trends in different scenarios

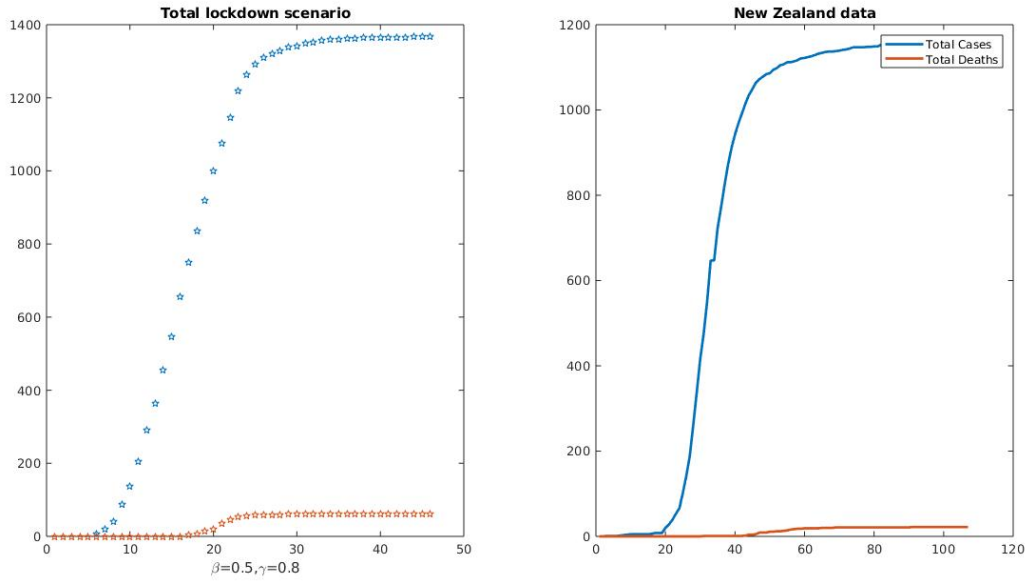


Figure 11: A comparison of real and simulated data. After several iterations, the values of β, γ and c in g(t) were decided to be 0.5, 0.8 and 18 respectively.

Since the model is probabilistic in nature, the code needs to be run for several iterations to discern and compare any trends in the infection data. We have discovered that the infection statistics converge in mean to the real data available for New Zealand. This validates our assumptions, to some extent, about the various probability distributions of infection, death and recovery. The best fit is obtained with the values of $\beta = 0.5$ and $\gamma = 0.8$ respectively.

The model reinforces what epidemiologists have advocated since the beginning of the pandemic: only a strict total lockdown can keep Covid positive cases in check and restrict fatalities to a minimum, while with no lockdown, a large section of the population is infected and death rates are commensurately higher. In fact, in our simulations almost every individual is infected, though we have considered a complete network with edges of varying weights. Also the rise in number of cases is exponentially fast, as was expected from available knowledge. Multiple lockdowns, if implemented at the right time, can also prevent cases from rising beyond unmanageable levels. We look forward to apply our models and social network construction techniques to India with a diverse population, vast expanse and widely differing lockdown strategies in every state.

A Concluding Remark

The stochastic and the deterministic methods give us equally good insight into the epidemic modelling. But a key challenge in applying these ideas into predicting the trends of the epidemic evolution of a country lies in the selection of the community structure.

Contributions

Anuran Pal : Code and Idea of the Determinsitc Simulation on networks and without network. Code and Idea of the Community Structure. Sections 4 and 5 of the report.
Anirban Dey : Code and Idea of the Stochastic Simulations
Anubhav Sur : Sections 1, 2, 3 and 6 of the report.
Abhinav Patra : Fit of the deterministic equation on New Zealand Data in the presentation.

7 References

[1] M.E.J.Newman, Networks : An Introduction.

[2] Sheng Bin, Gengxin Sun , Chih-Cheng , Spread of Infectious Disease Modeling and Analysis of Different Factors on Spread of Infectious Disease based on Cellular Automata.

[3] https://ourworldindata.org/coronavirus_source_data

[4] Andrew W. Byrne et al, Inferred duration of infectious period of SARS-CoV-2: rapid scoping review and analysis of available evidence for asymptomatic and symptomatic COVID-19 cases.

[5] https://stats.gov.nz