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UNIVERSITÀ DEGLI STUDI DI PADOVA

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Master Degree in Physics (LM-17)

Final Dissertation

Modelling COVID-19 spreading in a network

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Contents

Abstract

The usual simplified description of epidemic dynamics predicts an exponential growth. This is due to the mean field character of the dynamical equations. However, a recent paper (Thurner S, Klimek P and Hanel R 2020 Proc. Nat. Acad. Sci. 117, 22684) [**?**] showed that in a network with fixed connectivity, the nodes become infected at a rate that increases linearly rather than exponentially. Experimental data for COVID-19 seem to validate this approach. In this thesis we plan to study this model by tuning its parameters. In particular, we monitor the effect induced by a significant presence of hubs in the network.

Introduction

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Goals:

1. As is claimed in [**?**], we find that for any given transmission rate (β) there exists a critical number of social contacts (D_c) which describes a phase transition of the disease growth:
   - for $D < D_c$, linear growth, i.e. low infection prevalence, occurs;
   - for $D > D_c$, "classical" exponential growth as predicted by the SIR model.

   In particular, by setting our parameters to the empirical estimates of β and μ, $D_c \simeq 7.2$.
2. Assuming that the every-day-life contact network has $\langle k \rangle \simeq 5$; while lockdown measures reduce it to the household size of $\langle k \rangle \simeq 2.5$, we reproduced the infection curves of two classes of states with respect to NPIs intervention plans. The first is represented by the United States of America, which did not imposed measures; and the second by Austria, which planned a immediate severe lockdown. It's a relevant remark that no other parameters where introduced and fined-tuned.

At 8 May 2020, none of the affected Covid-19 states have reached "herd immunity" [1], but still they have reached the "epidemic peak" due to the containment restrictions on social contacts. The most striking observation is that the epidemic curves, especially when not NPIs are introduced, exhibit a linear growth for an extended time interval in contrast with the "S-shaped" logistic curve as predicted by the standard compartmental models. The extend of the linear regime depends on the onset of the measures; while for early stages, as it was the case for many countries [**?**] (8 May 2020), an exponential growth dominates the spreading of the disease. By taking care of the modification of the underlying social network structure, it's possible to recover the spreading trends for the states that have or haven't applied the NPIs measures.

Infection may occur for two reasons:

1. interaction between an infected and a susceptible person;
2. contact is "intense" (e.g. long, close,...) enough to lead to a disease transmission.

So, the rationale behind social distancing is that it takes to a reduction of both of these factors. Moreover, the standard SIR model assumes that there's the same probability that an individual encounters an infected person ("well-mixed population") and all the nodes have the same number of neighbours ($N-1$ or $\langle k \rangle$). However, since their first introduction, considering the complex network structure as provided remarkable results. e.g. how the vaccination threshold depends on the network topology [**?**]. However, no focus was put on the spreading of the disease below the epidemic threshold, since few works where published trying to analyze the lockdown effects, e.g. [**?**]. In this work, we try to grasp the relevant features of a complex social network. In fact, they present richer structures with respect to the well-known model ("Erdoes-Rényi","Scale-Free", etc.), including

[1] The total infected cases at the first peak were 0.3% of the total population, remarkably low with respect to the SARS reported level that are $0.5\% - 0.8\%$ of the entire population.

multilevel organization; weak ties between communities; and temporal aspects that suggest a degree of fluidity, however, with stable social cores. To grasp these feature, we used a Poissonian "small-world" network which enabled us to taking care of degree heterogeneity (i.e. different social contacts), family-clusters and their overlaps, small-world feature, given the fact that "leisure" activities may connect nodes that are diametrically opposite. Moreover, by changing the number of the average degree D we may pass from light to severe NPIs measures, i.e. strict lockdown.

Model

The main assumptions of the model are:

- fixed number of total individuals N connected by a social link;
- "undirected" adiaciency matrix A, where $A_{i,j} = 1$ if there's a link between the ith- and jth-node or $A_{i,j} = 0$ otherwise;
- transmission per day probability r and d days for an infected individual to be recovered, i.e. immune or naturally dead;
- average degree D and shortcut (or rewiring) probability ϵ

As one may expect, the r and d parameters are in a one-to-one map with to the SIR ones, respectively, for the recovery and transmission rates:

$$\gamma = 1/d, \quad \beta = rD/N. \tag{2.1}$$

At each time step (day), the infection curve of positive cases, $P(t)$, is the cumulative sum of the number of new cases $C(t)$. So, if $D > D_c$ the "well-mixed" (or homogeneous/mean-field) approximation may be applied, with the result that $C(t)$ is associated to $R(t)$ up to a time-shift of d, since all the infected are predicted to become recovered after d days.

Results

3.1 Infection Dynamics

To gain some intuition about the importance of modelling the underlying network, we've made the disease spread for two different values of D but fixing $N_{total} = 1000$, $D = 8$, $\epsilon = 0.1, d = 6\,\text{days}, r = 0.1$ and 10 initially infected nodes as seeds ($N_{seeds} = 10$).
The results are the following:

- In the large D and ϵ limit, the mean-field condition is fulfilled and the model resembles the compartmental SIR model. By mapping the parameters as done in **??**, we may compare $P(t)$ from the network model with the compartmental $R(t)$ by shifting it of $-d$ days. As a relevant reference for the comparison to the "small D" evolution, we've plotted the histogram of the daily cases which shows the typical peak in the early-exponential stages and, then, a decrease towards herd immunity, which for [**?**] has been reached at the 98% of the overall population.
- By reducing the average degree to $D = 3$, we've find a changing in the spreading behaviour, since $P(t)$ increases almost linearly for a remarkable timespan. At the end of the infection, nearly $1/5$ of the entire population has been infected which is noticeably smaller than the SIR predicted herd immunity nearly at 80%.

With this rough perception of the main results by considering the underlying network structure, we've further analyzed the parameter dependence.

3.2 Parameter Dependence and Phase Transition

The aim of this section is to obtain the value of the "critical average degree" D_c, such that, for $D < D_c$, the infection curve is (roughly) linear. In order to take care of the two growth trends, we've defined an order parameter

$$\mathbb{O} := SD(C(t)) \tag{3.1}$$

as the standard deviation of the daily new cases, having removed the days without new infected. In this way, $\mathbb{O}! = 0$ signals the presence of a nonlinear increase of the daily new cases and, therefore, of its cumulative sum $P(t)$. Other way around, $\mathbb{O} = 0$ corresponds to a linear growth of $P(t)$.

Appendix

4.1 Notes on Network book of A.Barabasi

4.1.1 Compartimental Models 10.2

SI model

At (10.1) β is the probability of 1 spread only. So, $\frac{\beta \langle k \rangle S(t)}{N}$ expresses the probability of 1 spread over $\langle k \rangle S(t)/N$ neighbors.
$C := \ln \frac{i_0}{1-i_0}$

SIS model

In (10.7), define $v_{SIS} := 1 - \frac{\mu}{\beta * \langle k \rangle} = 1 - R_0^{-1}$ the "characteristic velocity". If $v > 0$ spread; otherwise it dies out. So, $\tau^{SIS} = \frac{1}{\beta \langle k \rangle v_{SIS}} = \frac{\tau^{SI}}{v_{SIS}}$ In, SI model $v_{SIS} = 1$ [**?**]
The endemic fraction of infected is given by $\frac{\beta - \mu}{\beta}$ slides 16 of [**?**].

4.1.2 Epidemics on Nets 10.3

SI Model

10.16 :

$$\tau_n^{SI} = \frac{\tau_c^{SI}}{\frac{\langle k^2 \rangle - \langle k \rangle}{\langle k \rangle^2}}$$

10.17 : is obtained by imposing the initial condition $i(t=0) = i_0$. Anyway, $i_k = i_0 + i_0 f(t)$

SIS

$$\tau_N^{SIS} = \frac{\tilde{k}^2}{\beta \langle k \rangle - \mu \tilde{k}^2}$$

where $\tilde{k}^2 := \frac{\langle k \rangle^2}{\langle k^2 \rangle}$ and ???? if the network is homogeneous $\langle k \rangle^2 = \langle k^2 \rangle$. Check better what's the behaviour of $\langle k^2 \rangle$ wrt the considered network.

4.1.3 10.B

If a nets lacks degree of correlation, i.e. $e_{ij} = q_i q_j$, the probability $p_{kk'}$ is independent on k. Thus,

$$p_{kk'} = \frac{k' p'_k}{\langle k \rangle}.$$

Typo under (10.50) multiply by kp_k not $k-1)p_k$

Tight Binding model

5.1 Second Quantization formulation of the SIR Model