The effect of heterogeneous distributions of preventive social norms on epidemic spreading

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Introduction

The emergence due to the outbreak of the CoVid-19 disease, caused by the SARS-CoV-2 virus, suddenly erupted at the beginning of 2020 in China and has soon spread worldwide. This has caused an outstanding increase on research about the virus itself [1] and, more in general, epidemics in many scientific fields. In this work we will focus on the dynamics of the epidemic spreading and how it can be affected by the dynamics of Social Norms.

SIRS Model

Our study is grounded in compartmental modelling of epidemics [2], in particular we focus on a generalization of the well known SIR model called SIRS [3]. Both SIR and SIRS models describe a population whose members can be susceptible to the infection through direct contact among individuals. Three types of people are considered, the susceptible (S), whose density in time is indicated by $x(t)$, the infected (I), whose density is $y(t)$, and the recovered ones (R), to which corresponds the density $z(t)$. If the time scale of spreading of the disease is much shorter than average human life, and neglecting phenomena as migration, it is reasonable to assume the amount of population constant in time, so that $x(t) + y(t) + z(t) = 1\ \forall t$.
In the SIRS case, the time evolution of the densities is given by the following system of differential equations:

$$\begin{cases} \dot{x} = -\beta x y + \frac{1}{\tau_s} z \\ \dot{y} = \beta x y - \gamma y \\ \dot{z} = \gamma y - \frac{1}{\tau_s} z\ , \end{cases} \qquad (1)$$

where the dots represent the derivatives with respect to time ($\dot{w} \equiv dw/dt$), β is the infection rate (per interaction) controlling how often a susceptible-infected contact results in a new infection, γ the rate an infected recovers and moves into the resistant phase, and τ_s the average time before a recovered becomes susceptible again: in the limit $\tau_s \to +\infty$ the recovered individuals are become immune and the model reduces to the classical SIR, which does not consider reinfection. The possibility of recovered people to get infected again is a key point which determines the fate of the system: indeed, while in the SIR case the every possible equilibrium state has no infected ($\lim_{t\to\infty} y(t) = 0$), with finite value of τ_s we have in general final states with a constant infected rate larger than zero, in addition the convergence to the equilibrium presents oscillations of vanishing amplitude [3].

Agent Based Model approach

We start by reproducing the analytical model given in Eqs. (1) with an Agent Based Model simulation. To this aim, we consider initially a population of N agents on a complete graph (*i.e.*, mean field configuration). Each agent can be in one of three possible states, susceptible, infected, or recovered. At each elementary step of the dynamics one agent i is picked up at random, and one of the $N-1$ remaining agents, which we call j, is on its turn chosen randomly. If i is recovered, or susceptible and j susceptible or recovered, nothing happens. If i is susceptible and j infected, or vice-versa, the susceptible gets infected with probability β. Moreover, if i is infected, it gets recovered with probability γ, while if i is recovered it gets back susceptible with probability $1/\tau_s$. One time unit is given by N elementary steps as describes before. Of course, in mean field the simulations give back the analytical results of Eqs. (1).

Heterogeneous infection rate — In the original SIR(S) models the infection rate is uniform, that is, it is the same for all the agents. In general, this is not the case, and in the real world every individual has his/her own β. Indeed, besides plausible physiological considerations, we have also to link this parameter also to other factors, such as individual compliance with preventive social norms such as keeping social distancing, wearing masks, washing/sanitising hands, etc. [4]: the more a person respects such norms, the less he/she is likely to infect or be infected. Therefore, we define at the beginning the distribution $\{\beta_i\}_{i=1,\dots,N}$. Since in this case every agent has a different infection rate, when a susceptible individual a meets an infected one b, the probability that a is in its turn infected will be the geometric average of the two infection rates: $\beta_{b\to a} = \sqrt{\beta_a \beta_b}$. This because an individual following perfectly all the preventive measures should not, ideally, infect nor be infected.

We performed a set of simulations in order to single out any possible effect on dynamics due to the variance of the distribution of the infection rate. Therefore, we focused on different distributions with the same average $\langle\beta\rangle$ but different values of σ^2.
In Figure 1 we show the time behaviour of the model in the plane infected-susceptibles, for different distributions with $\langle\beta\rangle = 0.5$. As it is easy to discern, as the variance increases, the maximum of the infected ratio decreases: that is, having fixed the average, the variability of the distribution helps the system resist the pandemics (the effect of higher-order moments is very small, since the difference between uniform distribution and a bimodal with the same variance is barely perceptible). The heterogeneity of the distribution $\{\beta_i\}_i$ appears to affect heavily the dynamics of the epidemics and the level of damage it can reach. In particular, higher heterogeneity implies less global infection rate. We can figure out the mechanism such that this phenomenon takes place by considering the limiting case of the extreme bimodal distribution with $x_0 = 0$: in this case, half population has exactly $\beta = 0$, that is, all these individuals cannot infect nor being infected at all, they are practically isolated from the rest of individuals. Therefore, even though half population is heavily subject to the infection, the presence of such "invisible to the contagion" agents makes the system much more resistant than a population where everyone has $\beta = 0.5$.

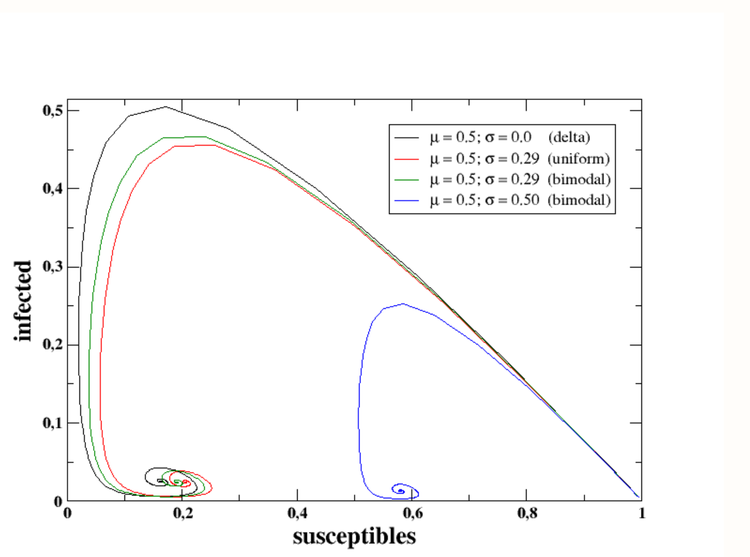

Figure 1: Comparison of the dynamics of the SIRS ABM model among different distributions of β_i, being fixed the mean value $\langle\beta\rangle = 0.5$ and the remaining parameters of the model. Distributions utilized: delta, uniform and two bimodals (where $\beta_i = \{x_0, 1 - x_0\}$; two cases are shown, the one with $x_0 = 0.211$, having the same variance of the uniform distribution, and the extreme one for $x_0 = 0$).

Summary and conclusions

Our simulation results indicate that heterogeneous distributions of the infection rate throughout the population allow a more efficient response to the spreading of the virus: keeping constant the average infection rate, the spread of the virus is more difficult to control when each individual violates a bit preventive social norms (homogeneous distribution), then when the distribution is heterogeneous, with systems populated with individuals fully complying with social norms or fully violating them. At the moment there are not available data yet to test rigorously this hypothesis, but empirical work on tax evasion points to similar results [5]. Therefore, we also propose new studies and data collections to verify the robustness of our hypothesis.

References

[1] Already on April 2nd 2020, only in the arXiv 264 preprints had "CoVid-19" in their title and/or abstract. A graphics showing the increasing of research in epidemics after the outbreak of the pandemics, up to the end of February, can be found at https://medium.com/@tomaspueyo/coronavirus-the-hammer-and-the-dance-be9337092b56 (chart 10).

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