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The effect of heterogeneous distributions of social norms on the spread of infectious diseases

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E-mail: daniele.vilone@gmail.com**Keywords:** epidemics, social norms, compartmental models

Abstract

The COVID-19 pandemic, caused by the SARS-CoV-2 virus, suddenly erupted in China at the beginning of 2020 and soon spread worldwide. This has resulted in an outstanding increase on research about the virus itself and, more in general, epidemics in many scientific fields. In this work we focus on the dynamics of the epidemic spreading and how it can be affected by the individual variability in compliance with social norms, i.e. in the adoption of preventive social norms by population's members, which influences the infectivity rate throughout the population and through time. By means of theoretical considerations, we show how such heterogeneities of the infection rate make the population more resistant against the epidemic spreading. Finally, we depict possible empirical tests aimed to confirm our results.

1. Introduction

The outbreak of COVID-19 pandemic in early 2020, caused by the SARS-CoV-2 virus, represented a challenge for public health worldwide. Indeed, it has produced an outstanding increase in research about the epidemic spread in many scientific fields [1].

Since mitigating the spread of COVID-19 requires large-scale behaviour change, insights from the social and behavioural sciences have been proven crucial to help align human behaviour with the recommendations of epidemiologists and public health experts [2–7]. A large literature has found social norms—the unwritten social rules that regulate behaviour in everyday contexts [8–10]—to be crucial in solving these challenges [11–14] and determining the behaviour of people during the emergency [14, 15] (but see [16] for a reflection on the limits of social norms to guide behaviour during a pandemic).

Apart from research in virology seeking for vaccines against COVID-19, an important topic is the analysis of the dynamics in the spread of infections during the pandemic. One established model to study the spread of infectious diseases is the ‘susceptible-infected-recovered’ (SIR) model [17]. Here, we use a modified SIR model and, based on the recent literature, we explore the effect of social norms on the spreading of the virus throughout the population.

As a first step, in the next sections we define the model we are adopting in our work.

1.1. Compartmental models and SIR(S) models

The most common way to model epidemic spread is to divide the exposed population in classes or compartments according to the state of the individuals. How these compartments are determined and how the agents change their status following the evolution of the epidemics varies per model [18]. The most famous and long-standing of these is the SIR model [17], where the compartments are three: Susceptibles (S), Infected (I), and Recovered (R). The first compartment includes the ‘healthy’ individuals, who have not been infected (yet). Infected (second compartment) and Recovered (third compartment) are the subsequent

stages of the process of epidemic spread. An S agent can be infected by an I agent with rate β , an I agent recovers with rate γ reaching the R status. The R status is the final, frozen status—in SIR, recovered individuals persist in their condition and do not become susceptible again.

Therefore, at any given time, the state of the system is given by the densities of susceptible and infected individuals, $x = x(t)$ and $y = y(t)$, respectively; the recovered density $z = z(t)$ is given by the normalization: $z = 1 - x - y$ (if the time scale of disease spreading is much shorter than average human life, and neglecting phenomena as migration, it is reasonable to assume the population size is constant in time). Assuming mean-field approximation, which is equivalent to consider the population set on a complete graph (*i.e.*, every agent is directly connected with everyone else), the dynamics of the epidemics will be given by the following system of differential equations [17, 18]:

$$\begin{cases} \dot{x} = -\beta xy \\ \dot{y} = \beta xy - \gamma y \\ \dot{z} = \gamma y. \end{cases} \quad (1)$$

An explicit solution of the previous system cannot be obtained analytically due to its non-linearity, but it can easily be solved numerically. In short, the main features of the model are the following. First, it is straightforward that in the final state no infected agents are present, since $y_\infty = 0$ is a necessary condition for reaching equilibrium. Therefore, in the final state there will be a part of the population who has never been infected (let x_∞ be the final density of it), and the remaining part consists of recovered people ($z_\infty = 1 - x_\infty$).

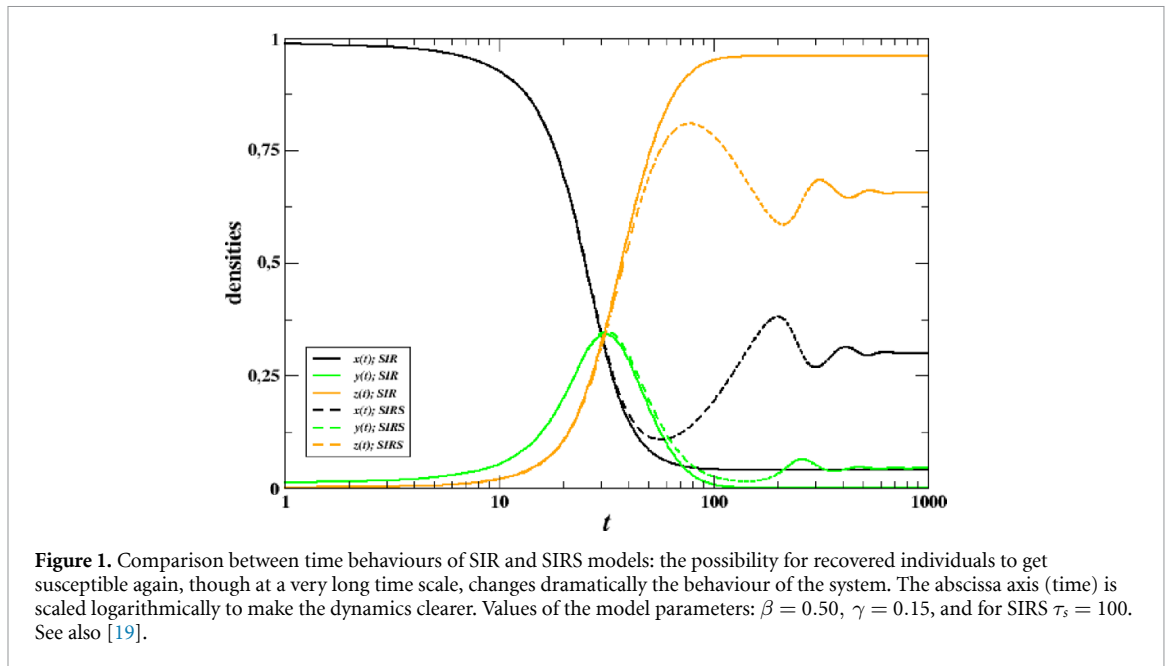
In general, we observe a non-trivial dynamic, where the evolution of epidemic spread depends on the precise values of the parameters at stake. Indeed, infected density $y(t)$ may undergo an exponential growth stage before vanishing, or go directly to zero. In particular, the quantity $R = \beta/\gamma$ (called *basic reproduction number*) is crucial to determine the behaviour of $y(x)$: if $R < 1$, $y(t)$ vanishes rapidly; if instead $R > 1$, there is an initial exponential growth of the number of infected people [17] (in figure 1 we show an instance of such behaviour). The SIR model turns out to be a useful description of an epidemic when some core conditions are met, in particular a quick dynamic with respect to average human life and the impossibility for individuals to become susceptible again once recovered (*i.e.* recovered agents have become resistant to the virus).

In order to broaden the scope of the validity of this approach, the SIR model has to be suitably generalized. Therefore, let us now consider a modified SIR model, which is better known as SIRS [19]. Like the original SIR defined by equations (1), it models a population whose members can be susceptible to the infection through direct contact among individuals. The compartments are the same, susceptible (S), infected (I), and recovered (R), but in this model recovered individuals become susceptible again after an average time τ_s . Therefore, in this case the system of differential equations describing the dynamics have the form

$$\begin{cases} \dot{x} = -\beta xy + \frac{1}{\tau_s} z \\ \dot{y} = \beta xy - \gamma y \\ \dot{z} = \gamma y - \frac{1}{\tau_s} z. \end{cases} \quad (2)$$

Of course, in the limit $\tau_s \rightarrow +\infty$ the recovered individuals are practically immune and the model reduces to the classical SIR.

SIRS more realistically represents the dynamics of infections in situations where people who have been infected become susceptible again after a while, as is the case for COVID-19 [20]. In this model the system can end up to a final, mixed state where the population size of all the three kinds of agents are in general non-zero. The possibility of recovered people to get infected again is actually a key point which determines the fate of the system [19]: indeed, while in the SIR model a requirement for every possible equilibrium state is that there are no infected agents left ($\lim_{t \rightarrow \infty} y(t) = 0$), in the SIRS model with a finite value of τ_s final states generally have a constant infected rate larger than zero. More importantly, the convergence to the equilibrium presents oscillations of vanishing amplitude, as shown in figure 1. This property of the SIRS model compared to the SIR shows how fundamental is to know whether infected people get immune once recovered or not. For instance, Vespignani and coauthors in [21] carry out an evaluation of the effectiveness of a ban on international flights departing from China with an epidemic model that assumes the immunization after recovering from the virus, which for the SARS-CoV-2 virus and its variants is the case only in the short term [20]. Therefore, models with no possibility of reinfection are useful only on short time scales.



1.2. The role of topology

The results of the SIR and SIRS models summarized above hold in complete graphs (mean-field), where all agents are directly connected with each other. In reality, epidemics happen in complex network topologies, determined by the actual relations (or lack thereof) among individuals. In general, the strength of the pandemic is at its maximum in complete graphs, because the more the agents are connected, the easier the infection spreads from an infected to a susceptible agent, and this holds true for most compartmental models [18, 22]. This feature holds true also in the model we defined, as we will illustrate in the next sections.

1.2.1. Social norms and epidemic spreading

Social norms are the unwritten rules that regulate our everyday life [23]. They are expectations about the behaviour of other individuals in one's reference groups and about what these individuals think is the socially appropriate behaviour to adopt in that situation [8, 9, 11]. They have been shown to be effective in promoting cooperative behaviour in many situations and have played an important role also in the adoption of preventive behaviour, such as mask wearing, social distancing, vaccine uptake etc during the COVID-19 pandemic [12, 14, 24, 25].

When studying the effect of social norms on behaviour, the common approach is to focus primarily on mean behaviour of others. Recently, scholars have acknowledged the importance of accounting also for the distribution of behaviour [26, 27]. Consider for instance a norm of cooperation. If on average a cooperation norm is present, this average might be the result of everyone cooperating, but also of half of the group free-riding and the other half overcompensating [28]. These two scenarios will likely impact an individual's decision regarding norm compliance differently.

The importance of variance in characterising social norms and norm compliance is emphasised by Gelfand *et al* [29], who distinguish between tight and loose norms, arguing that this distinction can help to understand systematic differences across cultures. Tight cultures are characterised by clearly defined rules of behaviour, while loose cultures show larger behavioural variance.

1.2.2. Heterogeneous infection rates

Here we explore how the shape of the distribution of behaviour compliant with COVID-19 preventive measures influences the dynamics of the contagion. In particular, we focus on the effect of the standard deviation and examine situations where everyone shows the same degree of compliance with preventive behaviour compared to others where instead there is a large variability throughout the population (e.g. norm compliance is very high for some individuals and very low for others) to examine whether different distributions may produce different outcomes.

The parameters of the classical compartmental models as SIR (1) and SIRS (2) systems are constant real numbers: that is, the rates β and γ , and the average recovery time τ_s are considered uniform throughout the population and constant in time. This is in many cases a useful approximation, but here we aim to refine the

model and consider the individual variability in space and time. Since our primary interest is in the influence of social norms over the spreading of the pandemic, it is convenient to start considering the infection rate β as non-uniform throughout the population.

2. The model

In standard compartmental models, the infection rate is uniform. That is, the probability to switch from a Susceptible state to an Infected state when interacting with an already infected agent is the same for everyone. In general, this is often not the case, for both physiological [30] and behavioural reasons [12, 31]. In the real world every individual has his/her own β . As a first refinement of the model, we consider here the parameter β as an individual one, defining the distribution $\{\beta_i\}_{i=1,\dots,N}$. Since in this case each agent has a different infection rate, when a susceptible individual a meets an infected individual b , the probability that a becomes infected will be the geometric average of the two infection rates: $\beta_{b \rightarrow a} = \sqrt{\beta_a \beta_b}$. This because an agent who perfectly respects all hygienic measures should not, ideally, infect nor be infected.

In order to test the effect of the heterogeneity of the infection rate in the population, we conduct simulations where we keep fixed the average $\langle \beta_i \rangle$ and the values of the remaining parameters, and only vary the the variance of the distribution $\{\beta_i\}_i$. This way, we will be able to sort out the role of fluctuations in infectivity in the dynamics of epidemic spread.

2.1. Symmetric distributions

We start by considering only symmetric distributions of β . Since $\beta \in [0, 1]$, that means we consider distributions where the probability density is such that

$$F(\beta) = F(1 - \beta) . \quad (3)$$

It follows that we also have $\langle \beta \rangle = 1/2$. We consider the following distributions with average $1/2$:

- Delta distribution centered in $\beta = 1/2$, such that every agent has infectivity ratio equal to $1/2$ and the variance is $\sigma^2 = 0$.
- Uniform distribution in the real interval $[0, 1]$ ($\sigma^2 = 1/12$).
- A derived distribution given by a Gaussian centered in 0 and squeezed in the interval $[0, 1]$ by this rule:

$$\mathcal{P}(x) = \mathcal{G}_\Sigma \left(\frac{1 - \tanh(x)}{2} \right) \quad (4)$$

where $\mathcal{G}_\Sigma(x)$ is the zero-mean Gaussian distribution with variance Σ . In figure 2 some examples of such distribution with different variances are shown.

- A bimodal distribution such that the only possible values of β are $x_0 \in [0, 1/2)$ and $1 - x_0$, each with probability $1/2$; the variance is here $\sigma^2 = 1/4 - x_0(1 - x_0)$.

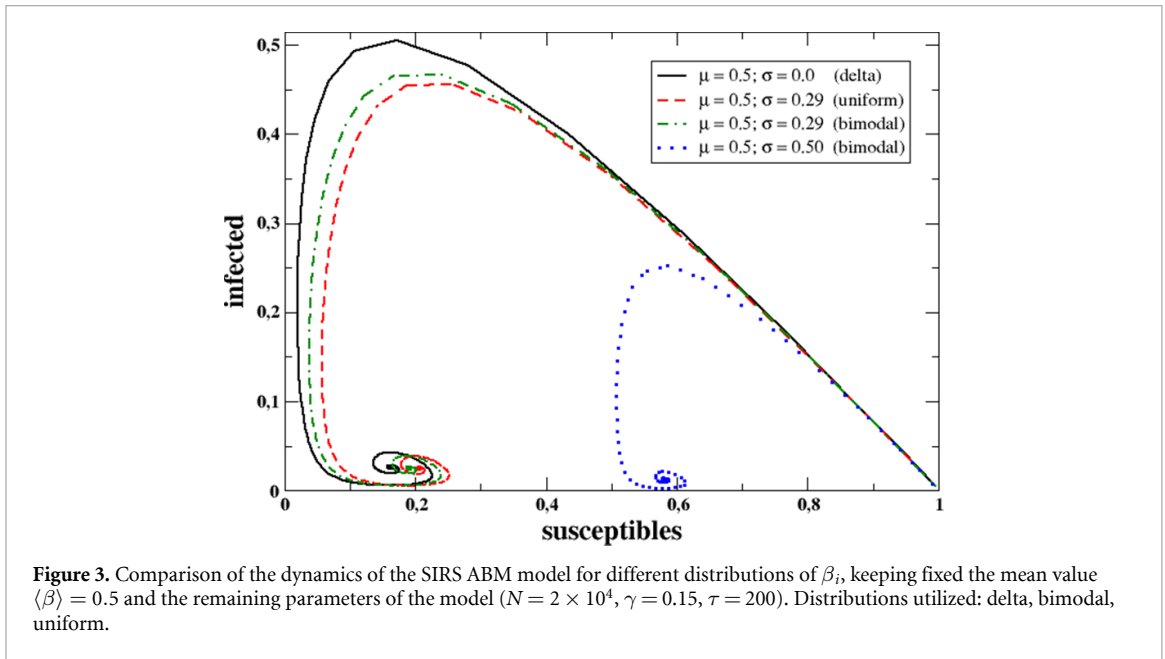
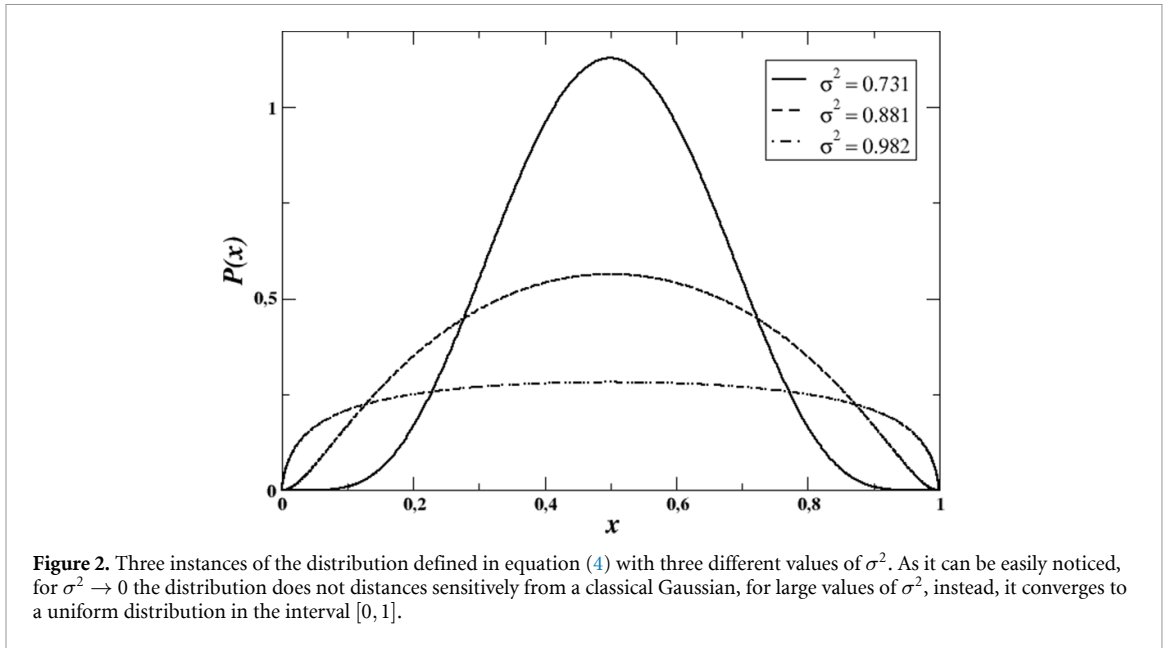
We checked the model behaviour keeping fixed all parameters but the variance of the distribution $\{\beta_i\}_i$.

2.1.1. Results

We conducted simulations experiments to single out any possible effect on dynamics due to the variance of the distribution of β . Therefore, we first focused on different distributions with the same mean value, and subsequently considered the same distribution for different values of σ^2 . We focus on the case $R > 1$, since here we are interested in the effect of heterogeneity on the dynamics of a rapidly spreading pandemic. How the epidemic threshold is affected by a non-uniform infection rate distribution is left to future work.

In figure 3, we show the time behaviour of the model in the I - S plane (i.e. we show the orbits in the space defined by the infected and susceptible densities), for different distributions, and for $\langle \beta \rangle = 0.5$. As is easy to discern, when the variance increases, the maximum of the infected ratio decreases. That is, having fixed the average, the variability of the distribution helps the system to handle the pandemic. The effect of higher-order moments is very small, because the difference between a uniform distribution and a bimodal one with the same variance is barely perceptible.

In figure 4, on the other hand, we consider only tanh-distributions. Again, the effect of changing the variance is quite strong, especially in the limit $\sigma \rightarrow 0.5$. In the inset of the same figure, we further clarify this point by showing the maximum level of infection during the whole dynamics as a function of σ .

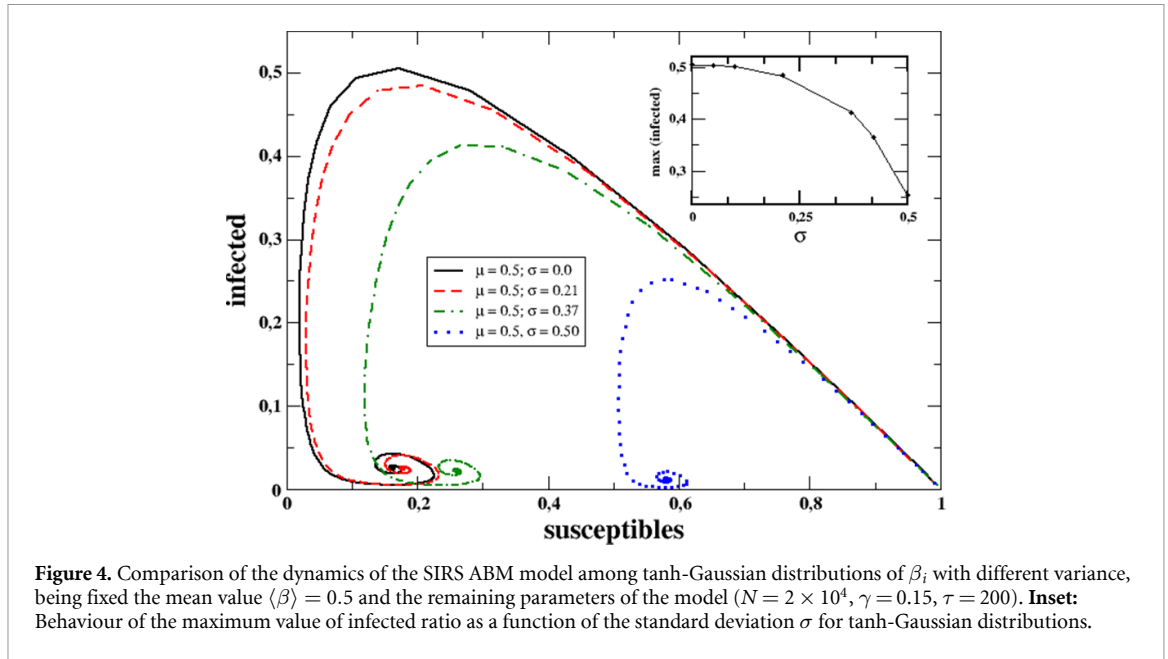


2.2. Theoretical analysis

As demonstrated in the previous section, the heterogeneity of the distribution $\{\beta_i\}_i$ strongly affects the dynamics of the epidemic and the level of damage it can reach. In particular, it shows that, keeping the average fixed, higher heterogeneity implies less global infectivity. We can figure out the mechanism behind this phenomenon if we consider the limiting case of the extreme bimodal distribution with $x_0 = 0$. In this case, half of the population has exactly $\beta = 0$. That is, all these individuals cannot infect nor be infected at all, they are practically isolated from the rest of the individuals. Therefore, even though half of the population is heavily subjected to the infection, the presence of such ‘invisible to contagion’ agents makes the system much more resistant than a population where everyone has $\beta = 0.5$.

The previous conclusions can also be justified analytically. For simplicity, let us take into consideration a bimodal distribution of $\{\beta_i\}$ with mean value $\langle \beta \rangle = 1/2$ defined as follows:

$$\beta_i = \begin{cases} \beta_+ = \frac{1}{2} + \varepsilon & \text{with probability } \frac{1}{2} \\ \beta_- = \frac{1}{2} - \varepsilon & \text{with probability } \frac{1}{2} \end{cases} \quad \varepsilon \in \left(0, \frac{1}{2}\right] \quad (5)$$



whose standard deviation is indeed $\sigma = \varepsilon$. Now, we can split the population in two subpopulations with higher and lower infection rate, respectively, so that each density is also split: $x_{\pm}, y_{\pm}, z_{\pm}$, where of course we have $x_+ + x_- = x, y_+ + y_- = y, z_+ + z_- = z$ being the densities, with the subscript \pm referring to the agents with $\beta = \beta_{\pm}$. Therefore, each differential equation of the system (2) doubles. For example, the dynamics of Infected will be described by the following pair of differential equations:

$$\begin{cases} \dot{y}_+ = \beta_+ x_+ y_+ + \sqrt{\beta_+ \beta_-} x_+ y_- - \gamma y_+ \\ \dot{y}_- = \sqrt{\beta_+ \beta_-} x_- y_+ + \beta_- x_- y_- - \gamma y_- \end{cases} \quad (6)$$

At the beginning of the dynamics we can assume $y_{\pm} \ll x_{\pm} \simeq \frac{1}{2}$, so that previous system can be rewritten in a more compact form as follows:

$$\dot{y} = \dot{y}_+ + \dot{y}_- \simeq -\gamma y + \frac{1}{2} \left(\frac{1}{2} + \varepsilon \right) y_+ + \frac{1}{2} \left(\frac{1}{2} - \varepsilon \right) y_- + \frac{\sqrt{1 - \varepsilon^2}}{4} y, \quad (7)$$

which yields

$$\dot{y} = \gamma [R(\varepsilon) - 1] y + \frac{\varepsilon}{2} (y_+ - y_-), \quad (8)$$

where

$$R(\varepsilon) \equiv \frac{1 + \sqrt{1 - \varepsilon^2}}{4\gamma}. \quad (9)$$

At very early stages of the dynamics we can assume the last term at the right side of equation (8) to be negligible with respect to the first one: at $t = 0$ the initial cluster of infected individuals is made up indifferently by agents of both kinds, because at the beginning of the contagion nobody is aware of the risk and behavioural norms to cope with it are not implemented yet, so that it is reasonable to pose $|y_+ - y_-| \ll y$ (notice that the assumption becomes exact in the limit $\varepsilon \rightarrow 0^+$). Therefore, reminding that the quantity ε is simply the standard deviation of the distribution defined in equation (5), we finally obtain

$$\dot{y} = \gamma [R(\sigma) - 1] y. \quad (10)$$

Equation (10) is formally the same as in the homogeneous case, but here the basic reproduction number depends on the standard deviation of the distribution $\{\beta_i\}$. As is easy to understand from equation (9), $R(\sigma)$ is a decreasing function of its argument, meaning that the higher the heterogeneity of the distribution, the less the epidemic spreads throughout the system. We stress the fact that equation (10) is valid only at early

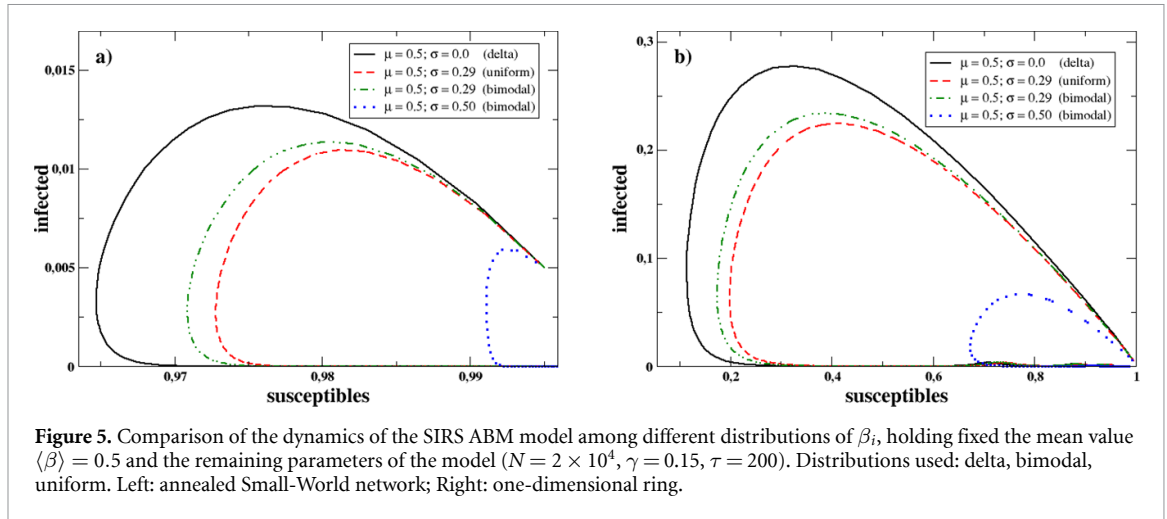


Figure 5. Comparison of the dynamics of the SIRS ABM model among different distributions of β_i , holding fixed the mean value $\langle \beta \rangle = 0.5$ and the remaining parameters of the model ($N = 2 \times 10^4$, $\gamma = 0.15$, $\tau = 200$). Distributions used: delta, bimodal, uniform. Left: annealed Small-World network; Right: one-dimensional ring.

stages of the dynamics, in particular as long as the approximation $|y_+ - y_-| \ll y$ holds: the faster the quantity y_+ increases, the sooner this approximation is no longer true, but the impact of the infection process as a function of σ is well described by its initial behaviour.

2.3. Effect of topology

The role of topology has been studied for SIR-like models [32–34]. Here we aim for a comparison between topology and heterogeneous infectivity effects in SIRS ABM. In general, complex topology can be considered a tool to model top-down measures to control epidemic spread (limiting physical contacts among individuals is equivalent to changing the network in which the population is embedded); on the other hand, the heterogeneity of the infection rates accounts for individual differences in physiological characteristics (how prone one is to get and/or transmit the pathogen) and the variability in complying with social norms about health and hygiene (the more agents observe and comply with the norms, the less is likely they can infect or be infected), as we have already illustrated in previous sections. Indeed, as is clearly shown in figure 5, the qualitative behaviour of the model is the same (the larger the variance, the better is the system’s response to the epidemics), but the share of the population infected is much smaller.

It must be noticed that a complex topology can be also used to model non-uniform social mobility, because agents who more often get in touch with others have a higher probability to infect or get infected. However, this can also be seen as an individual feature contributing to the infection rate β_i of each agent, just like other personal behaviours.

3. Time heterogeneity

Another strong assumption usually adopted by compartmental models is the time invariability of the parameters involved, in particular the infection rate β . In this section we take into consideration the possibility of non-constant rates. Initially, we neglect the distributions throughout the population, going back to the delta-distribution case.

In [35, 36] data about the spread of the COVID-19 pandemic in Japan are fitted by considering the so-called Avrami equation [37, 38] for describing the time behaviour of the infected density. The original Avrami equation can be written in the form [39]

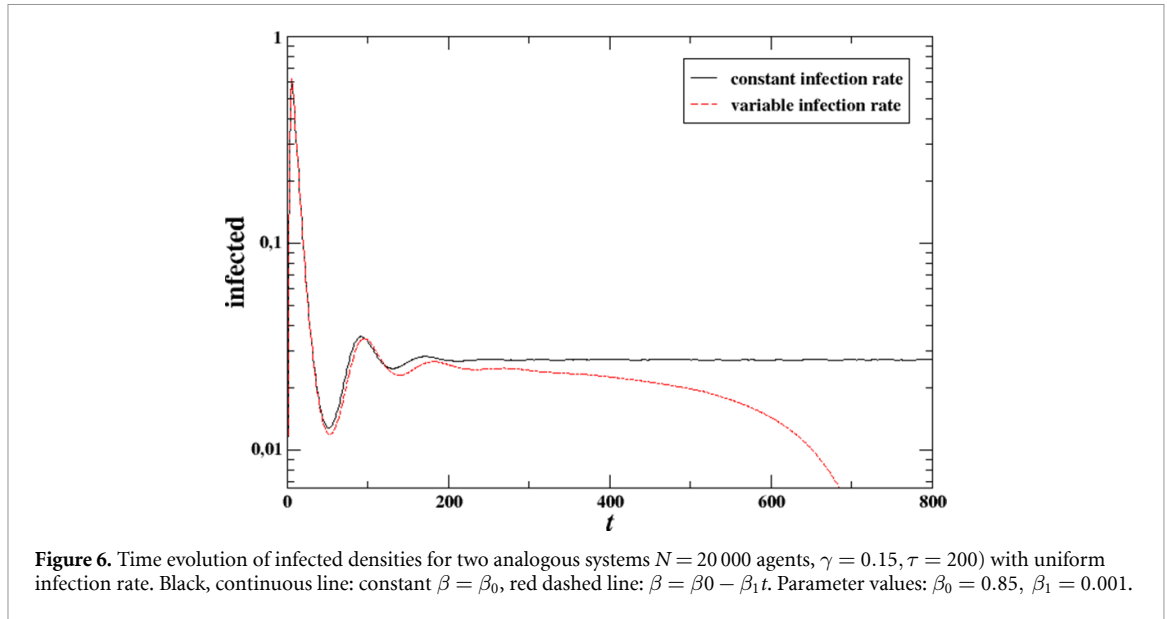
$$y(t) \propto \exp(-Kt^m), \tag{11}$$

where $m > 1$ and K is the fitting parameter. Historically, the Avrami equation was first written down to describe phase transitions at constant temperature, but equation (11) can be obtained by the SIR/SIRS equation for the infected density at initial times (when $x \simeq 1$) with both β and γ proportional to the same power of time:

$$\dot{y} = \beta_0 t^n \cdot y - \gamma_0 t^n \cdot y = (\beta_0 - \gamma_0) \cdot t^n y, \tag{12}$$

where $n \in \mathbf{R}^+$, and whose solution is actually

$$y(t) = y_0 \cdot \exp\left(\frac{\beta_0 - \gamma_0}{n + 1} t^{n+1}\right). \tag{13}$$



It is clear that it is quite unrealistic to assume that β and γ share the same time behaviour and that they start from zero and increase with time. In fact, for our purposes we have to characterise better the time dependence of the rates. A more realistic choice for time-dependent β and γ is the following:

$$\gamma = \text{constant}; \quad \beta(t) = \begin{cases} \beta_0 - \beta_1 t^n & t \leq T \\ \beta_0 - \beta_1 T^n & t > T \end{cases} \quad \beta_0 \in (0, 1]; \quad 0 < \beta_0 - \beta_1 T \geq 0 \quad (14)$$

where $T \leq \sqrt[n]{\beta_0/\beta_1}$ is the time needed to reach the minimum value of the infection rate. Here we have split β in a constant and a variable part, which takes into account the modifications of the infection rates due to the change in agents' behaviour. For simplicity we set $\beta_1 > 0$, meaning that we assume the emergence of the epidemic to make people behave more cautiously, decreasing the infection rate. The recovery rate is left constant in time, since it is less influenced by norm-induced behaviour of the agents and it is reasonable to assume that it varies more slowly with respect to β . Inserting relations (14) in the equation for the infected density at initial times, for any $t < T$ we obtain

$$\dot{y}(t) = (\beta_0 - \beta_1 t^n) y(t) - \gamma y(t) = \gamma_0 (R_0 - 1) y(t) - \beta_1 t^n y(t) , \quad (15)$$

whose general solution is

$$y(t) = y_0 \cdot \exp \left[\gamma (R_0 - 1) t - \frac{\beta_1}{n+1} t^{n+1} \right] , \quad (16)$$

being $R_0 \equiv \beta_0/\gamma$. Equations (15) and (16) correctly reduce to their classical SIR/SIRS versions in the limit $\beta_1 \rightarrow 0^+$.

It is straightforward to see that for $R_0 < 1$ the solution (16) is always decreasing for $t \geq 0$, so that the infection gets rapidly reabsorbed. On the other hand, if $R_0 > 1$ the time behaviour of the infected density becomes much more interesting. As a matter of fact, $y(t)$ is initially increasing up to the instant t^* , after which it decreases. Such critical time is

$$t^* = \left(\frac{|R_0 - 1|}{R_1} \right)^{\frac{1}{n}} , \quad (17)$$

where we defined $R_1 \equiv \beta_1/\gamma$. Of course, it must be that $t^* < T$, otherwise $y(t)$ remains increasing. It is easy to understand that t^* actually exists if $\beta(T) = \beta_0 - \beta_1 T < \gamma$, that is, if $R(t) \equiv \beta(t)/\gamma$ manages to become smaller than the recovery rate. Finally, it must be reminded that these results hold while the approximation $y(t), z(t) \ll 1$ is still true.

Finally, we show in figure 6 the behaviour of the ratio of infected agents in a population with time-changing infection rate, compared with its analogous with constant β . As is easy to see, and reasonable to expect, if the infection rate decreases in time, the strength of the contagion likewise decreases.

4. Discussion

SIR models are nowadays considered essential to provide insights on the dynamics of infectious disease outbreaks and the potential impact of interventions [40–42]. Nevertheless, they often overlook human behaviour, which, especially during the COVID-19 pandemic, has been largely influenced by adherence to social norms advocating for compliance with preventive measures like vaccination, wearing face masks, and self-isolation [12, 14, 16, 43–46]. The model we present here makes it possible to explore the effect of social norms on the spreading of the virus throughout the population. Since compliance with social norms is not homogeneous and for the same average can have different distributions [27, 29], we used our model to test how different distributions of compliance with social norms (i.e. low vs high variance) affects the spread of infectious diseases.

As a first step, we focused on the contagion rate from infected to susceptible individuals by considering its variability throughout the population (and also in time). We set every population member with their own infection rate, a personal value that is determined also by the degree to which individuals obey to the preventive social norms, e.g. compliance with hygienic norms, quarantines, etc. Our simulation results show that, keeping constant the average level of compliance with social norms, infections spread less in groups in which compliance with preventive behaviour is more polarised. As already hinted in section 2.2, a large subgroup of individuals who respect strict sanitary social norms acts as a shield against the infection spreading throughout the whole population. For example, let us consider the extreme situation where 50% of individuals are fully compliant so that they can be considered ideally impenetrable to the infection spreading. Then at most only half of the population can be infected independently from the behaviour of the non-fully compliant agents.

Certainly this model in its current form is not aimed to be a prediction tool, in the sense of forecasting the future course of an ongoing epidemic. The purpose of the model is to compare the effect of different distributions of compliance with social norms sustaining COVID-19 preventive behavior on the spread of infectious diseases. However, while the model is not a forecasting instrument, it can be useful for identifying interventions to limit the spread of viral epidemics or pandemics. For instance, results from our model suggest that it might be more effective to target specific subgroups with the goal of making them completely obedient to social norms underlying health preventive behaviour, rather than trying to make the entire population averagely obedient.

In the future, it would be interesting to test or calibrate the model with heterogeneity in infectivity and social norm compliance on empirical data. To test the variability of norm compliance, data would be needed for multiple subpopulations—either through cross-national comparisons or through regional comparisons, preferably over multiple time points. Unfortunately, most existing data either allows for subgroup comparisons in a single time point [46] or allows for a comparison of the evolution of the pandemic and social norm compliance over time within a single population [16], thus limiting the possibilities of a calibration exercise. In the absence of another epidemic/pandemic, one could also test this model experimentally by assigning to participants a random infectivity risk and letting them interact in a SIRS environment where they have to choose between complying with costly preventive social norms or violating these norms and thereby increasing their payoff but also their risk of infection (see [47] for an example of an experimental test of a SIRS model with endogenous network formation).

Finally, from a theoretically point of view, testing the combined effect of space and time heterogeneity, that is, the effect of infection ratios dependent on both individuals and time, will be another goal for future work.

Data availability statement

All data that support the findings of this study are included within the article (and any supplementary files).

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