Bi-stability of SUDR+K model of epidemics and test kits applied to COVID-19

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Motivated with various responses of world governments to COVID-19, here we develop a toy model of the dependence epidemics spreading on the availability of tests for disease. Our model, that we call SUDR+K, is based on usual SIR model, but it splits the total fraction of infected individuals into two components: those that are undetected and those that are detected through tests. Moreover, we assume that available tests increase at a constant rate from the beginning of epidemics but are consumed to detect infected individuals. Strikingly we find a bi-stable behavior between a phase with a giant fraction of infected and a phase with a very small fraction. We show that the separation between these two regimes is governed by a match between the rate of testing and a rate of infection spread at given time. We also show that the existence of two phases does not depend on the mathematical choice of the form of the term describing the rate at which undetected individuals are tested and detected. Presented research implies that a vigorous early testing activity, before the epidemics enters into its giant phase, can potentially keep epidemics under control, and that even a very small change in rate of testing can increase or decrease the size of the whole epidemics of various orders of magnitude. For the real application of realistic model to ongoing epidemics, we would gladly collaborate with field epidemiologists in order to develop quantitative models of testing process.

I. INTRODUCTION

The recent outbreak of the SARS-CoV-2virus and the associated illness COVID-19 has triggered, in this century, unprecedented containment measures around the world including the complete lock-down of the populations of all towns in Italy and and China, [1]. The World Health Organization has declared the diffusion of COVID-19 to be a pandemics and issued a strong warning of a severe global threat[2]. In the case of the COVID-19 epidemics there is also an *infodemic* of true and false news about the danger, the diffusion and the treatments of COVID-19 [3]. This context muddles the attempts to understand the epidemics and confuses the people. At the same time we assist to lively debates among scientists following the epidemics on all social media and platforms. Some of important questions are: *(i)* how many infected people are undetected? *(ii)* how the number of tests and testing policies affects the dynamics of epidemics? *(iii)* Is there a benefit in early testing? Some of those questions are addressed with different methods in the context of different epidemics or are recently addressed without explicit modeling effort. In [4], authors statistically evaluate different strategies of testing in the context of ebola epidemics and show the importance of early testing. They found that availability of early testing would reduce epidemics by one third. In [5] authors review laboratory testing for influenza, which is often mentioned as similar to SARS-CoV-2in methods of spreading, and lay out all the possible ways in which early tests can be used in fighting the diffusion of such a disease. In [6] authors conclude that undocumented infections present main channel of geographic spread of SARS-CoV-2.

There is an ongoing effort in estimating modelling the dynamics of this epidemics and to set the values of the model parameters significantly affecting the diffusion [7–9]. In this letter we adopt the available numerical estimates published in this studies. Parameters, whose calibration is impossible due to lack of data are implicitly kept within realistic ranges.

In order to explicitly take into account the different impacts on the spreading dynamics of undetected and detected infected individuals, and the contribution of the available number of testing kits to put the epidemics under control, here we extend the usual SIR model to a novel "SUDR + K" one. In the model we propose four states of population - S (susceptible), U (undetected), D (detected) and R (removed), and one additional variable K which models the number of available test kits. Susceptible are those in population which can acquire disease. Infected individuals can be detected or undetected, therefore $I = U + D$. Detected are those that are positively tested, and undetected are infected of which no one knows of although some may be suspected for infection. Removed are those individuals that either healed and acquired immunity or are deceased. Total number of people in population is N. Lower case letters represent fraction of population, $s+u+d+r = 1$ ($u+d = i = I/N$), and $k = K/N$ represents available number of tests per capita.

Even though in reality there are different kinds of tests (including Nasopharyngeal and oropharyngeal swabs, Bronchoalveolar lavage, serum testing, CT etc. [10]), we gather all the kinds in a single family of tests.

The model we propose is defined by the following equations:

$$\dot{s} = -\beta s u \tag{1}$$
$$\dot{u} = \beta s u - \delta u k - \gamma u \tag{2}$$
$$\dot{d} = \delta u k - \gamma d \tag{3}$$
$$\dot{r} = \gamma(u + d) \tag{4}$$
$$\dot{k} = \alpha - \epsilon \delta u k \tag{5}$$

Equation (1) is just the usual equation of SIR model that represents the dynamics from susceptible to infected after exposure. Here we put u instead of i, because we assume that after detection the probability of contagion becomes negligible [11].

Equation (2) needs a more detailed explanation. The first term just represents the fraction of individuals that changed their state from susceptible to infected. The second term models the change of undetected to detected by random testing. If there are no tests no one can get detected, if there are no undetected again no one can get detected. It is then proportional to both the numbers of undetected and of kits. It is motivated by the idea that infected individuals report to hospital on the basis of symptoms (proportional to u) and get tested with higher probability if there is abundance of kits or lower if there is a scarcity of kits. The third term represents just the fraction of individuals that gets removed without ever been detected. Equation (3) has terms of opposite sign with respect to the second and the third previous equation and and additional removal term of detected individuals. Although The removal of a undetected individual happens only through healing (direct death without a transition to d can be neglected), while the removal of a detected individual can be due to both healing and death, it is reasonable that both detected and undetected individuals are removed with equal probability (4), to reduce the number of parameters. Equation (1) represents growth of fraction of kits. The first term in the equation represents a constant growth of the number of kits (fixed produced kits per time unit). The second term means that kits are used proportionally to the number of undetected individuals and the number of available kits, which makes sense and also prevents the number of kits to become negative. The parameter ϵ measures how many more tests have to be done to switch an undetected individual to detected, this term always has to be equal or larger than the corresponding term $\delta u k$ in equations (2) and (3).

Of course there can be higher order contributions in all equations, however in our opinion Eqs. (1)-(5) are the simplest possible to get plausible dynamics.

II. MODELS FOR DETECTION

An alternative model for detection can be obtained in the following way. First, let us assume that for each time

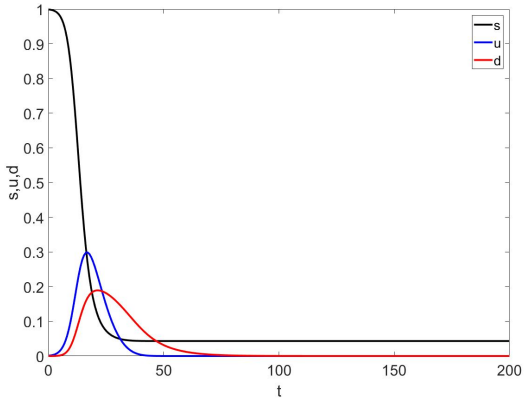

FIG. 1. Red line represents detected fraction of population, through time; blue - undetected and black - susceptible for parameters $\beta = \ln 2$, $\delta = 0.5$, $\gamma = \ln 2/7$, $\alpha = 0.02$, $\epsilon = 1$.

increment K new kits are produced, and that a fraction of $0 < \delta' < 1$ of available kits is used for people accepted in the hospitals. This means that the number of kits used on hospitalized people is $\delta' K$ On the other hand the number of people arriving at hospitals with symptoms is proportional to number of undetected therefore $\delta' = \delta u$. Moreover let us hypothesize that each of this newly detected individuals had previously infected other βs individuals and therefore we could expect that the number of newly detected is

$$\Delta D = \delta u K(1 + \beta s) = u\Phi(K, s\delta, \beta) \tag{6}$$
$$\Delta d = \Delta D/N = u\phi(k, s, \delta, \beta)\,. \tag{7}$$

Consequently the model equations become:

$$\dot{s} = -\beta s u \tag{8}$$
$$\dot{u} = \beta s u - u\phi - \gamma u \tag{9}$$
$$\dot{d} = u\phi - \gamma d \tag{10}$$
$$\dot{r} = \gamma(u + d) \tag{11}$$
$$\dot{k} = \alpha - \epsilon\phi\,. \tag{12}$$

Alternative couplings between detected and undetected can in principle be also:

$$u^\delta k = u\phi(u^{\delta-1}, k)\,, \tag{13}$$

which is a term often used in chemical kinetics in $A+B \to C$ [12], and the

$$k\frac{u}{\delta s + u} = u\phi(k, s, u, \delta) \tag{14}$$

which is also typical in the kinetics of chemical reactions [12]. As $\delta < 1$, the interpretation is that each unit of kits will be used on either susceptible or undetected people, but undetected individuals are more probable to be tested, therefore δ reduces the susceptible cohort. The number of new detected subjects in a single time step is given by the ratio between u and all of the people subjected to tests (which can be either susceptible or undetected). The rate of finding is then proportional to k and this factor. In Eq. (10) we have assumed that spreading of disease and testing happen at the same time. More

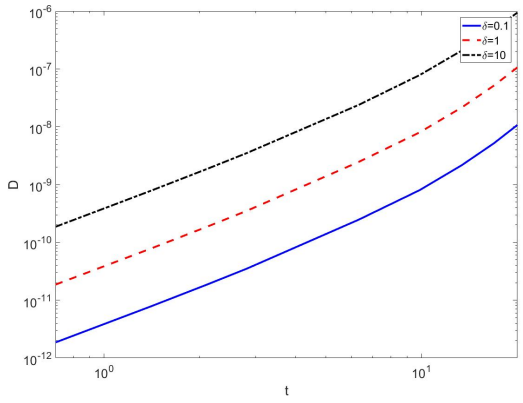

FIG. 2. For different values of parameter δ , using $\beta = 0.25$,

realistic model could include expected incubation time τ and then the term would become delayed with

$$\delta u k(1 + \beta s(t - \tau)) = u\phi(k, s, \beta, \tau). \tag{15}$$

All the above terms can be collected in a single function

$$u\phi(u, k, \delta, \beta, s)\,, \tag{16}$$

that we will use later.

III. RESULTS

Whichever model we choose, we qualitatively observe the same qualitative behavior. Generally speaking, we find a difference between the two different peaks of detected and undetected individuals both in size and in their position, as seen in Figure 1. Depending on the values of the parameters we choose values and ordering of peaks heights can vary, but the peak of undetected individuals is always higher than the peak of detected ones.

In Figure 2 we show that, for the chosen parameters values, the initial growth of detected subjects is initially a power-law with exponent ≈ 2 in line with results by Maier and Brockman [13]. The reason for this is very similar to their model in the sense that there is a reduction of the epidemic spreading for those individual that enter into this new compartment. This is also checked from the analytical point of view and an expression very similar to the one found in [13] is obtained. However one can see that the fraction of infected individuals in such power-law regime, multiplied by the Italian population predicts less than one single individual, and therefore this very initial theoretical regime is unobserved in real data for Italy. On the contrary, in Figure 3 one can see that for a range of parameters values a successive exponential growth is obtained as expected in any epidemics dynamics. In this respect it is noteworthy that the exponential rate in the model is always smaller than the one observed in real cases. Parameters which are modeled for SIR have $\beta - \gamma$ as exponent on the onset of epidemics, while ours is given by $\beta - \phi - \gamma$. Using the parameters measured for SIR changes the slope (depending on the strength of testing term).

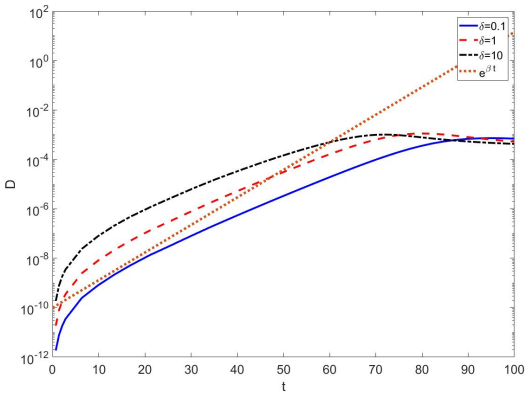

FIG. 3. Red squares are detected through time, blue circles are undetected and black diamonds are susceptible for parameters $\beta = \ln 2$, $\delta = 0.5$, $\gamma = \ln 2/7$, $\alpha = 0.05$, $\epsilon = 0.15$.

One of the most interesting aspects of our new model is the appearance of two different peaks in the dynamical evolution of the densities of the two sub-classes of infected people, undetected and detected. The peak related to undetected individuals is in general occurring before the peak of detected ones. The earlier the peak of detected happens the smaller is the number of total infected at the end of epidemics. We have found a very interesting relationship between the time $t_{D,max}$ at which the peak of detected occurs and the parameter α giving the production rate of the testing kits:

$$t_{D,max} \sim \alpha^{-\eta}. \tag{17}$$

This relationship is very clear in Figure 4.

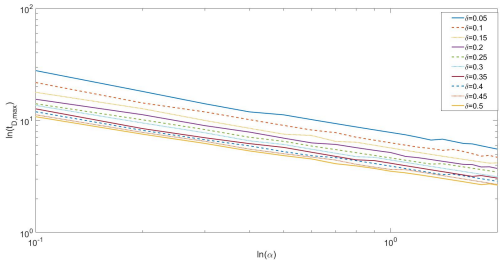

FIG. 4. For $\beta = \ln 2$ and $\gamma = 0.099$ and for different α and δ, one can observe the relationship 17

The most surprising result of our model is represented by Figures 5 and 6.

In Figure 5, we can see that for very realistic values of α, one can observe a switching behavior between two phases, one with a full blow epidemics, and the other one

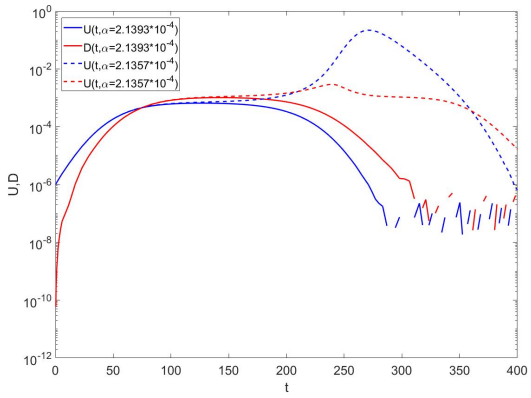

FIG. 5. For the usual choice of parameters $\beta = \ln 2$, $\gamma = 0.099$, $\delta = 10$ we see bi-stability and a strong response of the system to jump from phase of full blown epidemics to almost disappearing one for 2 close but different α. Both detected and undetected are depicted.

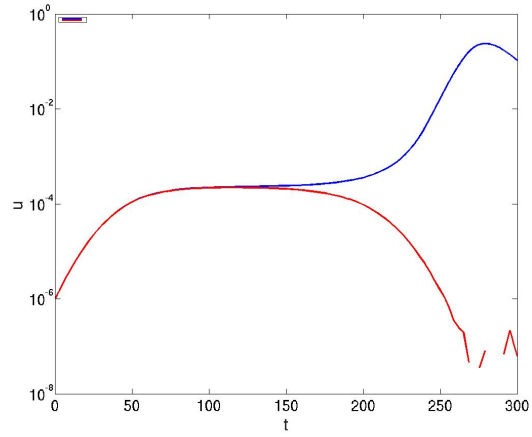

FIG. 6. For the usual choice of parameters $\beta = \ln 2$, $\gamma = 0.099$, $\alpha = 7.14 \cdot 10^{-5}$ we see bi-stability and a strong response of the system to jump from phase of full blown epidemics to almost disappearing one for two different δ. Only undetected persons are depicted.

in which epidemics practically disappears before macroscopic spreading in the population. The values of α for which we observe this bifurcation between these two very different behaviors are very close one to each other and are suggesting the possibility of a huge effect on the epidemics diffusion even for a change of few percentiles of the number of new available testing kits per day.

In Figure 6, we can see that in a certain range of values of the δ parameter, one can also observe a switching behavior between the two phases, one with a full blow epidemics, and other in which epidemics diffusion stays limited and then vanishes. These observations strongly suggest that the coupling between kits and the fractions of undetected and detected individuals is crucial for the possible evolution of the epidemics.

Since a similar behavior is observed also for other kinds of coupling, that we have seen in Sect. II, we give a more general argument for this behavior about Eq. (2). By focusing on the temporal location of the maximum of the fraction of undetected subjects, obtained by solving the equation $\dot{u}(t_c) = 0$, we notice that

$$s(t_c) = \frac{\delta}{\beta} k(t_c) + \frac{\gamma}{\beta}. \tag{18}$$

In Fig. 7 the right side of the equation 18 is depicted for both choice of parameters that lead to exploding or suppressed phase that are represented with the number of susceptible individuals through time., and it is clear that it give place to a switch between the two aforementioned phases of the epidemics. In order to generally explain this transition, we make use of the general coupling term (16) in the model equations. As long as the function ϕ is strictly positive and continuous we will have the same behavior, but with changed temporal location of the switch. In that case the switch will arise naturally by setting $\dot{u}(t_c) = 0$ which, from Eq. (2), means through the solution t_c of the equation :

$$\phi(u(t_c), k(t_c), \alpha, \delta) = \beta s(t_c) - \gamma \tag{19}$$

In order to proceed to a classification of the two phases, we have to study the second time derivative of the fraction of undetected individuals $\ddot{u}$:

$$\ddot{u}(t_c) = \beta \dot{s} u - u\dot{\phi}. \tag{20}$$

Clearly t_c will be location of local maximum if $\ddot{u}(t_c) < 0$, therefore for

$$\beta \dot{s} < \dot{\phi} \tag{21}$$

growth of undetected is subdued. Equation 21 says that the change of the rate βs with which new undetected are produced has to be smaller then the change of the rate ϕ with which new undetected are found.

IV. DISCUSSION

A simple interpretation of this result is that when the rate of successful testing and the rate of recovery equals the rate of transmission of the infection (i.e. transformation of individuals from susceptible to the infected state), and the changes of this rates also coincide, the pandemic enters into a dynamical stationary state. Note that this does not mean that there are no newly infected, but simply that the number of new undetected per day is kept below a certain value. When the two rates equate, we have a clear separation between the region with small and manageable population of u and full blow up of the epidemics. The repercussion of this result is that testing can have an immense impact if it is done in time and in a smart calibrated on the rate of transmission of the infection in the population. Indeed it is important

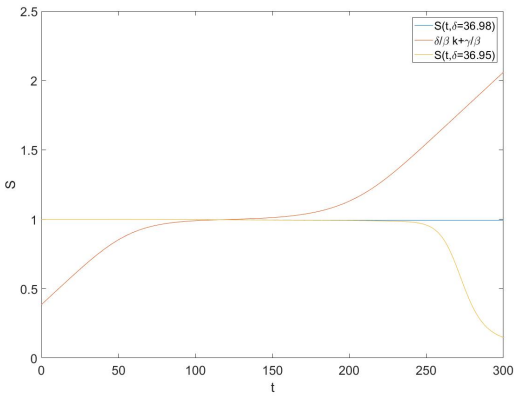

FIG. 7. Two different phases of susceptible and Eq. 18 for parameters that lead to the phase with extinguishing epidemics.

to stress that the way Singapore handled the Covid-19 crisis [14] is very similar to our model. Moreover Japan and Hong Kong are also managing well the diffusion of the epidemics during the writing of this paper: indeed $\alpha = 0.0002$, as reported for the Hong Kong case [15], is within the meaningful range of parameters we used in this model. This leads us to believe that developed countries which are adopting testing policies postponing a widespread testing activity until they have full blown epidemics are probably wrong. This result would also suggest that sharing of tests among nations is fundamental in order to mitigate the epidemics diffusion.

In the end we would like to once again stress that here we present toy model which is not calibrated and suitable to any kind of quantitative predictions. We believe that the testing strategy, and the modeling of detection of cases is of fundamental importance for the epidemics of COVID19 as well as for all possible future epidemics of unknown pathogens, and we would love to collaborate with institutions and researchers which are working on real testing to model it as best as possible.

V. ACKNOWLEDGEMENTS

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