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Why should we be mindful of nonlinear dynamics in the midst of a global pandemic

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Abstract. While nonlinear dynamics is no stranger to modeling epidemics, the present COVID-19 crisis has underlined the inadequacy of simple models of spreading contagia in describing the epidemic as it evolves in real life. It suggests that we need to augment our models which had so far focused on the complexity of interactions by incorporating the complexity in the components themselves, e.g., by considering strategic decision making in agents choosing between different actions.

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There is nothing like a raging global pandemic to make nonlinear dynamics popular among scientists, as well as non-scientists. Let us take the current example of the COronaVIrus Disease 2019 (COVID-19) epidemic, which was reported for the first time on Dec. 31, 2019, in Wuhan, China, and from that time has grown rapidly in scale to cover the entire world. From mid-February onwards, there has been a deluge of papers appearing in preprint servers and online journals that look at the epidemic through the lens of modeling. Moreover, mass media around the world have been reporting on the findings reported from these modeling studies as people try to understand the disease and how the epidemic is likely to develop in the coming weeks and months (and possibly, years). The bulk of such studies appear to be using different variations of the classic SIR compartmental model describing the time-evolution of an epidemic proposed by Kermack and McKendrick in 1927 [1]. It assumes that the members of a given population can be classed into three categories: susceptible (S), i.e., those who have not yet contracted the disease but have the potential to do so; infected (I), i.e., those who have the disease and can pass it on to others through contact; and recovered/removed (R), i.e., those who can no longer be infected as they either possess (temporary) immunity through earlier exposure to the disease or have died because of it. The size of each subpopulation evolves according to

$$\frac{dS}{dt} = -\beta SI, \qquad \frac{dI}{dt} = \beta SI - \gamma I, \qquad \frac{dR}{dt} = \gamma I, \tag{1}$$

where β and γ are the rate of infection transmission and that of recovery (or removal), respectively. As it is easy to check, the model assumes that the total population size $N = S + I + R$ is invariant in time. This constraint implies that only two of the coupled equations in eq. (1) are independent.

This simple model is in fact one of the staples of an introductory nonlinear dynamics course, along with the Verhulst logistic equation for population growth and the Lotka–Volterra (LV) model for prey–predator interaction [2]. In fact, the LV model has a close resemblance to eq. (1), with the S category identified as prey and I as predator. The only difference between the two is the appearance of a growth term for the prey population in the LV model which has no analog in the SIR model where the epidemic is assumed to occur over a short enough time that demographic changes in the population can be neglected. However, this apparently minor difference gives rise to very different dynamical outcomes in the two models. While the LV model shows periodic oscillations in the prey and predator populations, in the SIR model S shows an exponential decay while I rises exponentially until it reaches a peak and then exponentially decreases, eventually becoming zero, which brings the dynamics to a halt.

Indeed, this pulse-like appearance of I in its time-evolution has sometimes led to comparisons with excitable media models [3] – which also feature regularly in nonlinear dynamics courses – that are characterized by the existence of three states, viz, resting, excited and recovering/refractory, which are analogous to S, I and R, respectively. While ostensibly there is no explicit threshold in the SIR model unlike the situation in excitable systems, there is indeed an effective threshold that governs whether an epidemic will occur (i.e., the size of the infected pool will increase) or not (in which case the infection dies out with a bulk of the population remaining susceptible). As can be deduced immediately, assuming that one starts from a state where the population is almost entirely susceptible ($N \approx S$), the condition for an epidemic (i.e., $dI/dt > 0$) is given by $N\beta/\gamma > 1$. More intuitively, an epidemic can only arise if the number of new infections that occur at any point of time overwhelms the recoveries that are happening at the same time. Indeed, this is identical in content to the so-called mosquito theorem proposed by Sir Ronald Ross much earlier, suggesting that to eliminate malaria one only needs to reduce the mosquito population sufficiently (i.e., it is not necessary to drive mosquitos to extinction to check the epidemic) [4].

The above argument also leads to one of the most talked about metrics associated with an epidemic, viz, its basic reproduction number R_0. Described as the average number of secondary infections that are caused by a primary infection at the early stage of an epidemic (when the bulk of the population is in S), it is easy to see that for the mean-field SIR model, $R_0 = N\beta/\gamma$. The underlying assumption here of homogeneous mixing, i.e., any person is likely to be infected by any other in the population, is of course not tenable in any real setting and thus, substantial work that has been done on understanding how diseases spread over a social contact network. Thus, we now understand that R_0 is determined by multiple factors that range from biological to demographic and social. These are (i) the generation time (loosely interpreted as the period for which an individual is free to pass the infection to others), (ii) the average number of contacts that a susceptible person has with infectious individuals, (iii) the probability that a contact between an infectious and a susceptible individual will result in the latter becoming infected, and (iv) the number of people in the population who are susceptible. Intriguingly, this also suggests multiple ways in which one can try to control an epidemic. For example, rapid identification through mass testing and consequent isolation of infectious individuals focuses on the first factor, physical distancing and quarantine (or the massive countrywide lockdowns declared by governments in different countries), that targets the second factor, promoting hygienic practices (such as washing of hands) and changing climatic factors (such as higher temperature or humidity), that reduces the role of the third factor, while the final factor comes into play if a vaccine is available.

Knowing R_0 allows estimating the proportion of the population who would be affected by the disease before the epidemic eventually comes to an end if there are no substantial changes in the infection transmission rate over the entire course of the epidemic. Thus, knowing that R_0 for India is 1.83 ± 0.03 (which we estimated over the period between March 4, when the number of active cases in India first reached double digits, and April 11) implies that about 74% of the population will be affected eventually by the disease. Given that the case fatality rate for COVID-19 in India has been around 3%, allowing the epidemic to progress unimpeded (e.g., as Sweden chose to do [5]), is simply not an option because of the unacceptably large number of deaths that will result from such a policy. Many countries have therefore chosen to impose stay-at-home orders (referred to as 'lockdown' by the media) to contain the disease and break the chain of infection much before the epidemic reaches saturation naturally. Such unprecedented control measures have altered substantially the natural pattern of evolution of the epidemic (as indeed was the purpose of the lockdown), which is reflected in the decrease in the effective reproduction number R over time: for India, it dropped to 1.29 ± 0.01 during April 3–May 10, then further to 1.22 ± 0.02 during May and from June 10–26 has been 1.11 ± 0.01 (figure 1). While it is good in terms of better public health outcomes, it also means that dynamical models have not been able to provide accurate long-term forecasts because of these significant perturbations to the natural evolution of the epidemic.

Indeed, the plethora of modeling papers that have appeared on COVID-19 seems to have resulted in an entire spectrum of possible forecasts, possibly more confusing than clarifying to the lay public. So does it reveal an inadequacy in the enterprise of mathematically modeling epidemics, or more specifically, the nonlinear dynamics approach to understand how a disease spreads through contact? While the media and the general public may be most interested to know when the epidemic is likely to peak and by when it will end, I think it is unfair to expect models to be able to say what may likely happen six months (or for that matter, even two months) down the line, given the large role played by unanticipated interventions like lockdowns, as well as the impact of essentially unknown factors, such as how faithfully are people in different places adhering to the various norms put in place to reduce the transmission

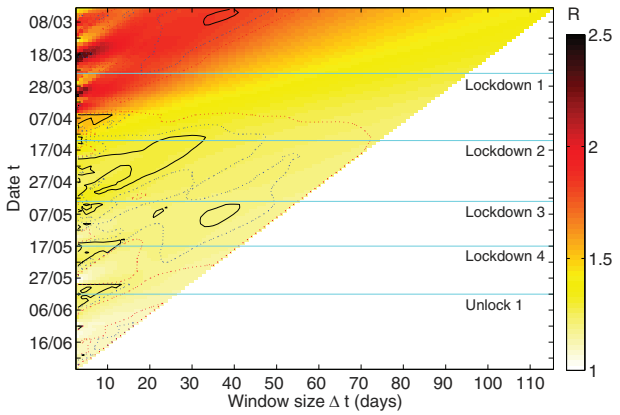

Figure 1. The effective reproduction number R (see color bar at right) for the COVID-19 pandemic in India, estimated from the time-series of the number of active cases by using moving windows having different starting dates (T) and interval lengths (Δt). Only those intervals are considered to yield robust estimates for which the correlation coefficient r between time and logarithm of the number of active cases is extremely high, e.g., the black, blue and red dotted contour lines indicate the choices of T and Δt for which $r > 0.998$, 0.995 and 0.99, respectively. The measure of significance for all these choices shows $p < 0.001$. The different phases of the stay-at-home order (lockdown) imposed by the government are demarcated by horizontal lines.

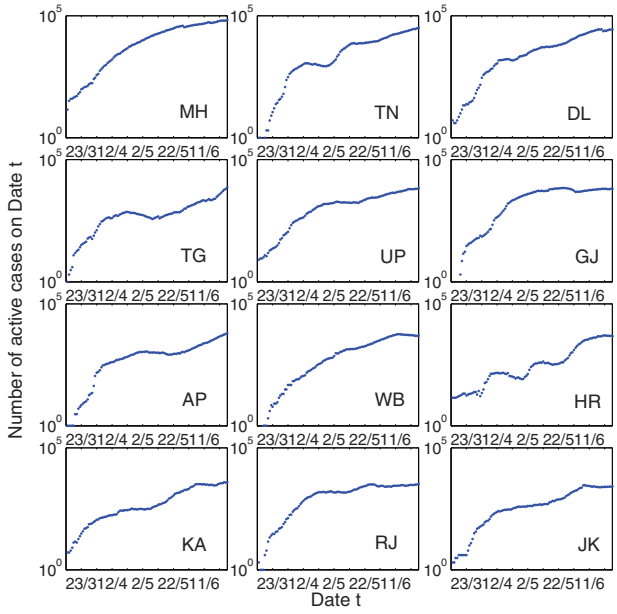

Figure 2. The progress of COVID-19 epidemic in 12 states of India that have the highest number of active cases as of June 26, 2020 (starting from top left, shown in descending order) with the number of active cases shown using a logarithmic scale (so that a linear trend in the graph corresponds to an exponential increase).

rate. Indeed, why should there be a single peak, as several models that incorporate the realistic structure of social networks along which a disease spreads have shown that such peaks may be recurrent [6, 7]? One look at how the number of active cases has changed over time should convince anyone how differently the progress of a real epidemic behaves from the smooth exponential rise and decline seen in models. (Figure 2 shows the time-evolution for different Indian states.) Thus, nonlinear dynamics would seem to be not such a great tool if the only purpose of models is to make predictions, as quite reliable short-term forecasts for expected case numbers can be done through statistical analysis alone (e.g., using techniques discussed in Ref. [8]).

Introducing more realism into the dynamical models by adding age-specific classes, details of transportation networks, etc., is unlikely to improve the accuracy of forecasts as, first, this will necessitate more parameters whose values then need to be estimated from data that is limited, as well as noisy, and second, factors such as whether there will be complete or partial lockdowns in the future are essentially unknowable. Instead, I propose that the true purpose of these models is to answer deeper questions about the epidemic, such as the efficacy of various control strategies that have been proposed, by testing the different competing proposals *in silico* and then making an informed choice on the basis of the simulation results. We also need to incorporate into the nonlinear dynamical models, which usually focus only on the complexity of the interactions, the complexity of the components themselves. Unlike the inert entities that we are used to in other contexts, the agents here are individuals who have their own objectives, that may sometimes be in conflict with that of society at large [9]. It is crucial to take such competing interests into account when, for example, investigating how lockdowns are likely to affect the progress of the disease. Owing to their differing circumstances, individuals will have very different risk perceptions from each other and thus, will have sharply divergent incentives in adhering to measures such as a lockdown. Not taking these into account (for instance, by incorporating game-theoretic decision making on the part of the simulated agents in the dynamical models) and imposing a one-size-fits-all measure may well result in a humanitarian crisis, as the events of the past months have shown us. In fact, if the events of the past few months have taught those of us who do nonlinear dynamics anything, it is how much of a complex system an epidemic truly is. To understand its evolution, it is not adequate to just add bells and whistles to dynamical models that look at it only as a spreading process, but rather to take into account the socio-psychological imperatives that drive the actions of individuals, as well as, of institutions and governments.

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