EPIDEMIC SPREADING: TAILORED MODELS FOR COVID-19

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A very simple epidemic model proposed a century ago is the linchpin of the current mathematical models of the epidemic spreading of the COVID-19. Nowadays, the abstracted compartmentalisation of the population in susceptible, infected and recovered individuals, combined with precise information about the networks of mobility flows within geographical territories, is the best weapon of the physics community to forecast the possible evolution of contagions in the current pandemic scenario.

Epidemic spreading usually refers to the territorial diffusion of an infectious disease that affects a large fraction of the human population in a relatively short time. The high mortality caused by infectious diseases has boosted mathematical research since the XVIII century. But it took until 1927 for the biochemist W.O. Kermack and the physician A.G. McKendrick to propose what we know today as the "SIR model" [1] and derive one of the main results of epidemiology: the existence of a threshold point that separates the growth of an epidemic from its extinction. Almost a century later, our capabilities for epidemic modeling have been complemented with extraordinary computational resources, which has allowed to tailor the basic models to more sophisticated tools to forecast the epidemic course, but the fundamental idea of the SIR model still remains.

The linchpin of epidemiological modeling: the SIR model

In epidemiology, compartmental models are those models that assume that the population is divided into groups (compartments) such that individuals belonging to the same compartment are epidemiologically equivalent. The SIR model divides the N individuals of a population in three classes: in the "S" compartment people are susceptible to infection (they can get the disease), the "I" compartment consists of people who are infected (and infectious, they can infect others), and the "R" compartment contains those individuals who have recovered from the infection, although "R" can also stand for "removed" to account for people who die from an infection (hence the acronym SIR). Note that at any time t, it holds that $N = S(t) + I(t) + R(t)$.

The SIR model is used to represent the spreading of diseases that yield immunity, no reinfections of individuals are allowed. It specifies the different transitions among these epidemic compartments, according to the most relevant parameters of the particular disease transmission. These transitions can be expressed by a simple *law of conservation of mass*, equivalent to a stoichiometric approach: $S+I \xrightarrow{\beta} 2I \quad I \xrightarrow{\mu} R$.

The evolution of these variables is determined by two essential parameters: the probability of infection per contact β, and the recovery rate μ. According to the previous expression, if a susceptible individual (S) encounters an infected one (I), the former will transit to the *Infected* compartment with probability β. Additionally, individuals in the *Infected*

compartment will move to the *Recovered* compartment with probability μ. These probability rates are specific for each disease, and they depend on so many different factors that it is almost impossible to find accurate values for them by studying the biology of the infection only, making it necessary to rely on statistics once the epidemics evolves.

Despite its simplicity, the SIR model is able to give us some interesting insights. *An essential outcome of the model is the existence of a phase transition whose critical point separates two regimes: one where the epidemic dies out and the other where the epidemic becomes endemic.* Considering the previous simple formulation for the SIR (which assumes a well-mixed population where everybody is statistically equivalent and makes $<k>$ contacts), this critical point happens when $\beta<k>/\mu = 1$. When this ratio (also known as the Basic Reproductive Number R_0, indicating the average number of individuals that one infected individual will generate in an otherwise totally susceptible population) is below 1, one infected individual will generate less than one new infected and thus the epidemic dies out. Conversely, when $R_0>1$ the epidemic will grow. The usefulness of this number went unnoticed until 1979, when Anderson and May applied it to study epidemic control strategies ensuring that R_0 is kept below 1. In Figure 1 (top plot) we show this transition, as well as two different temporal evolutions (bottom plots) for two different values of R_0.

Including the crucial role of mobility

An important aspect when modeling epidemic spreading is mobility. This is a crucial factor given that the virus is able to travel from one location to another when the host does so. To introduce mobility in the previous model, we adopt the level of description of "metapopulations", where the full population is decomposed in distinguishable geographical areas named "patches" (see Figure 2). So now, but considering the general quantities $S(t)$, $I(t)$ and $R(t)$, we will have $S_i(t)$, $I_i(t)$ and $R_i(t)$, for each patch i. Also, the subpopulations are not isolated, instead they are connected through a network of mobility flows W, where W_{ij} is the weight of the connection between patch i and j. Considering this new scenario, the mobility and epidemic dynamics are as follows: (I) each individual belongs to (or resides in) a patch; (II) an individual will travel outside its patch with probability p and its destination patch is chosen according to the mobility flows W; (III) the individual will contact a fraction of individuals in the destination patch; (IV) the mobility patterns are recurrent, meaning that after traveling, the individual will return to its original patch, implying that the next travel will start out from the same patch of origin. Regarding the epidemics, these mobility dynamics imply that one individual might get infected either if he does not travel and gets infected by someone resident of (or that has traveled to) his origin patch; or if the individual travels and gets infected in the destination patch either by a resident or by somebody that has traveled to that patch. See [2] for the full formulation of this model.

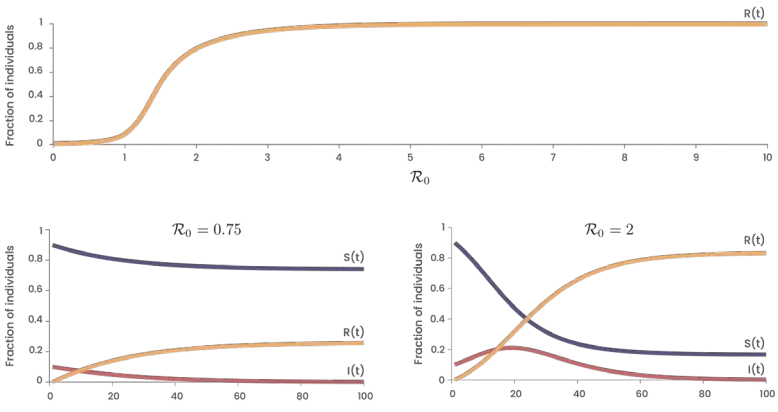
This new scenario allows us to account for a realistic setup in epidemics, one where individuals move across territories; and gives us some non-trivial insights about the role of mobility. Indeed, following the same rationale as with the original SIR model, we can calculate the critical point of the phase transition between epidemic extinction and epidemic growth as a function of the mobility parameters, and therefore learn what interventions in mobility should be enforced if we want to ensure the epidemic is receding. One surprising conclusion that we learn from this model is that a higher mobility probability does not always imply a higher spreading in the epidemic. This is counterintuitive, as higher mobility implies a higher mixing between the subpopulations, and this should foster the spread of the virus. However, despite the higher spatial diffusion, we found that, in some cases, mobility can reduce the number of potential interactions made by infectious individuals, which leads to a higher value of the epidemic threshold needed to observe an outbreak [2,3].

Towards a tailored model for COVID-19

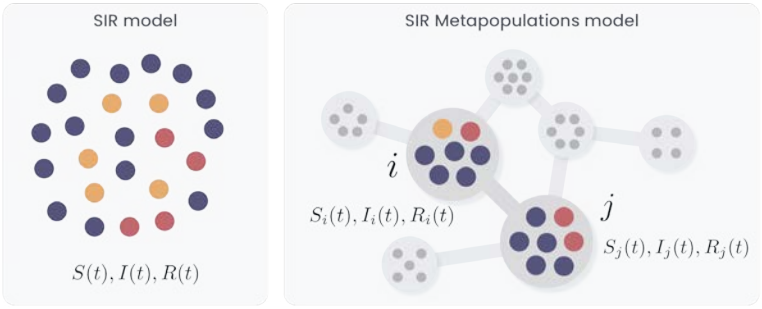
Once we have a basic framework to represent any SIR-like disease and account for mobility, we can move on to the challenge of modeling COVID-19 [7,8]. For this particular disease the epidemic compartments available in the SIR are not enough to capture the complexity of the dynamics and the timescales of transmission of COVID-19, so we start out by including more epidemiological compartments and new transitions among them. The following is a summarised list of the key additions that one has to consider for modeling COVID-19, as exposed in [2-5].

First, we introduce the *Exposed* compartment (E), that accounts for individuals that are in the incubation stage (already infected but not yet infectious). Therefore, when a susceptible individual contacts an infectious one, the former will transit to the *Exposed* compartment, and will remain in this state until the incubation time has passed.

Second, a crucial aspect of the COVID-19 is the existence of *Asymptomatic* individuals (A): individuals that are infected and infectious but that do not show any symptoms. Far from being a clinical feature only, this class is epidemiologically very relevant: an asymptomatic individual is rarely aware of its infectious potential and therefore his or



▼ **FIG. 1:** Results for the SIR model in a well-mixed population. Top: fraction of recovered individuals in the steady state as a function of R_0. Bottom: Fraction of individuals in each compartment as a function of time, for two values of R_0, one below the epidemic threshold (R_0=0.75) and the other above the epidemic threshold (R_0=2). In both cases the initial fraction of infected individuals is 10%.



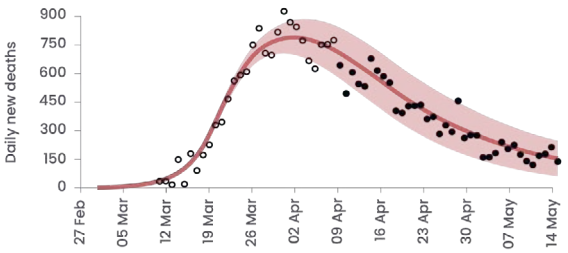
▲ **FIG. 2:** Schematic of the SIR metapopulations model compared to the basic SIR model. In the metapopulation model we consider multiple populations so that now the quantities $S_i(t)$, $I_i(t)$ and $R_i(t)$ refer to the fraction of Susceptible, Infected and Recovered individuals in patch *i*. These quantities can also be interpreted as the probability that a resident of patch i is in the Susceptible, Infected or Recovered state. The colours are identical to those used in Fig. 1.

her social and mobility patterns remain unchanged, fostering the spread of the pathogen across the territory.

Third, at variance with usual SIR-like models, we include compartments that capture the clinical evolution of those infected individuals that need hospitalisation in ICU, distinguishing between those that have a favourable prospect and will transit to Recovered after the hospitalisation time has passed, and those having a fatal outcome, transiting to a *Deceased compartment. This way, besides forecasting the epidemic trajectory, the model allows us to assess different clinical aspects associated to the pandemic such as the health system overload or the number of fatalities.*

Another major feature of COVID-19 models lies in the addition of age compartments. Indeed, it is known that age influences not only the symptomatology of the disease, but also the individual's prospect. Besides this clinical factor, the mobility patterns also depend on the age of the individual (*e.g.* retired individuals do not commute to work). Therefore, we deem necessary to account for age compartments, and we choose to divide the population in three segments: young (Y), adult (M), and elderly (O). To accommodate the age strata, each of the previously mentioned compartments is triplicated, and we use a contact matrix to model the contacts that are established between age compartments.

Once all the aforementioned particularities of COVID-19 are accounted for, the resulting model becomes a very powerful tool for surveillance and policy making. For example, starting out from a completely susceptible population, we can seed infection in certain patches (mimicking the first imported infection cases) and let the system evolve, observing to what other territories the disease spreads to, effectively forecasting community transmission. From this setup, one can calculate the expected amount of new cases, hospitalisations or deaths (see Figure 3). Besides merely observing the outcome of the system, one can also simulate containment measures, like restricting the number of contacts of the elderly population (emulating the confinement of this age segment), and observing if this implies a reduction in hospital load, for example. But most importantly, we can, as we did with the previous simpler models, calculate the critical threshold of the phase transition and discern which values of the parameters drive the epidemic to extinction [6,7].



▶ **FIG. 3:** Daily new deaths for the first wave of COVID-19 in Spain in 2020. The red line represents the outcome of the model, while dots represent real data as reported by the Spanish health authorities. Hollow dots represent the data points used to calibrate the parameters of the model, and solid dots are data used for validation.

Summarising, the SIR model established a solid foundation to model the spreading of infectious diseases in a population. Despite its simplicity, it still remains at the core of most of the current tools for epidemic forecasting. *Including the epidemiological traits of the disease we wish to model and considering the role of human mobility, one is able to build very effective tools that reveal us what are the crucial mechanisms behind the spreading of a particular disease, giving us the opportunity to anticipate its outcome and change the course of epidemics.*

About the authors

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