**Outline**

**Goal of this project**

Epidemic spread is characterized by exponentially growing dynamics, which are intrinsically unpredictable. The time at which the growth in the number of infected individuals halts and starts decreasing cannot be calculated with certainty before the turning point is actually attained; neither can the end of the epidemic after the turning point. A susceptible-infected-removed (SIR) model with confinement (SCIR) illustrates how lockdown measures inhibit infection spread only above a threshold that is shown later in the presentation. The existence of that threshold has major effects in predictability: A Bayesian fit to the COVID-19 pandemic in Spain shows that a slowdown in the number of newly infected individuals during the expansion phase allows one to infer neither the precise position of the maximum nor whether the measures taken will bring the propagation to the inhibition regime. There is a short horizon for reliable prediction, followed by a dispersion of the possible trajectories that grows extremely fast. The impossibility to predict in the midterm is not due to wrong or incomplete data, since it persists in error-free, synthetically produced datasets and does not necessarily improve using larger datasets.

The parameters of the model can be estimated within a relatively narrow range using data available from COVID-19 pandemic. Yet, unavoidable uncertainties in those parameters, which determine the time at which growth is halted or the overall duration of the pandemic, propagate to the predicted trajectories, preventing reliable prediction of the intermediate and late stages of epidemic spread. This is the main message of this article because it transcends the model of choice.

This model does an excellent job in reproducing past data, but instead of taking most likely parameters values to draw a prediction, we estimate compatible ranges of variations in the parameters.

The main conclusion is that the deterministic nature of SIR-like models is misleading if aimed at describing the actual course of any pandemic: prediction of the past is achieved through suitable fitting of data, and different functions may work, but prediction of the future in the midterm cannot be trusted.

**SCIR: SIR model with confinement**

The SCIR model includes the usual states of a SIR model plus a class C for individuals sent to confinement that are susceptible, but not infected. **See figure**.

Figure description:

Diagram of the epidemic model along with the equations ruling the dynamics. *Susceptible* individuals (S) can enter and exit *confinement* © or become *infected* (I). Infected individuals can *recover* ® or *die* (D). *N* is the total population. Rates for each process are displayed in the figure; *q* depends on specific measures restricting mobility and contacts, while *p* stands for individuals that leave the confinement measures (e.g. people working at essential jobs like food supply, health care, policing, etc.), as well for defection.

Here is performed a fit of I to data on officially diagnosed cases, which are automatically quarantined. The underlying assumption is that the real, mostly undetected, number of infections is proportional to the diagnosed cases.

In a sufficiently large population, the number of infected individuals at the initial stages of the infection is well below the population size. If we assume I(t)/N≪1 (1), then we can neglect the nonlinear term in the equation for the number of susceptible individuals and solve the model analytically. Within this approximation the equations determining the dynamics are:

The first two equations imply an approximate relation consequence of assumption (1), so we can write and reformulate the equations as

That are two *1° Order Constant Coefficient Linear ODEs* and they can be solved with separation of variable method (resolution).

The solution of this system is:

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Where:

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is the effective basic reproduction number modulated by the confinement—R0 being its value at the beginning of the epidemic. All the behaviour of the epidemic is enclosed in this magnitude. At its initial stages, , so the epidemic spreads when  (as is the case of COVID-19), and the larger , the faster it does. When confinement sets in,  gets tamed, eventually dropping to the value . An important epidemiological message follows from this simple fact: only if the confinement is strong enough (p and q are sufficiently different so that ) can the epidemic be controlled; otherwise, it spreads until eventually decaying due to the standard SIR mechanism—the exhaustion of susceptible individuals.

**Fitting COVID-19 data**

The data span two different regimes: unconstrained propagation of the epidemic, with , and a lockdown phase with effective parameters for the transition to the confined state. Since separated data for the number of recovered and deaths was unreliable, we have merged these two compartments and jointly fitted , which become a single variable in practice. Here it is used a Bayesian approach to fit the data, assuming that the numbers of infected and recovered + dead are log-normally distributed with unknown variance and mean given by the expression for  obtained from the model. At the very early stages of the epidemic (before any recovery or death event), the total number of confirmed cases grows as  independently of the chosen model. Analysing this initial growth for every country in the world, it appears that  everywhere (doubling times larger than one day are reported in all cases). Thus, we use informative priors for β and  (uniform distributions from 0 to 1 days−1) and vague priors for the rates q and p (uniform distributions from 0 to 5 days−1). Also, we use noninformative priors for the variances.