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Epidemics and their mathematical modelling

In this first contribution focusing on the mathematical modelling of epidemics, we will review some history, look at basic epidemiological models, and their parameters and what kind of information we can infer from them.

The current pandemic of Coronavirus *SARS-CoV-2* has inflicted a severe blow to our society and it has shown how vulnerable we are. It will be interesting to see what changes will take place in our modern society after this pandemic. Today, it is important to understand the physical, chemical and biological properties of this virus and how it is transmitted. The ultimate goal of the modern epidemiologists is not only to predict the course of a disease (at the present time the infection Covid-19 produced by SARS-CoV-2) but also developing methods to control it. Right now, many people are wondering how effective the current measures taken by different countries are in order to "flatten the curve". The answer of this question is not easy, we need more data (and time!).

1 Some history on epidemology

Humanity has always been affected by different pandemics. "Black Death" was arguably the worst pandemic, devastating Europe in the XIVth century, reaching its peak between 1346 and 1353. This disease originated in central Asia and was spread by fleas carried by rodents traveling in ships to Europe. There is a general consensus that the Black Death was indeed the bubonic plague, caused by the bacteria *Yersinia pestis*. Nonetheless, there are several authors who believe that this disease was a different kind of epidemic (see for example, the article [4]). It is calculated that between 30% and 40% of the whole population of Europe died because of this disease. The consequences of this pandemic were dramatic changes not only in the hierarchical and economic structure of the medieval society but also at intellectual level, giving rise to the Renaissance. The Black Death recurred regularly in Europe for almost 300 years. Nowadays, this disease (bubonic pest) can be cured with usual antibiotics.

You might be surprised to know that the first mathematical modelling of a disease was carried out by the Swiss mathematician and physicist Daniel Bernoulli in 1760 and published later in the article [3], where he studied the variolation against smallpox. Variolation was one of the first efforts to "vaccinate" people by inoculating a mild strand. This work was unfortunately better known in the actuarial literature than in the epidemiological one [1].
In the XIXth century, the physicians John Snow and William Budd studied the temporal and spatial patterns of cholera and typhoid, respectively. Their analysis brought light into understanding the transmission of these two diseases.
Later on, one the landmark works in epidemiology was published: Kermack and McKendrick proposed in 1927 [6], the first compartmental model. After this paper, they continued the study of these models in a series of articles. A slight variation of their proposed model will be briefly described in the next section.

2 Compartmental models

Simple epidemiological models split the population into different groups or compartments; it is also assumed that the individuals within each group possess the same characteristics. These models are called compartmental models.
The first compartmental model described by Kermack and McKendrick splits the total population N into 3 groups: susceptible (S), infected (I) and recovered (R). You will find in the literature many papers dealing with such SIR models. Each of the groups changes according to time, or in other words, all of them can be considered as a

function of the variable time t. They proposed a system of differential equations describing the rate of change of each group as follows:

$$\frac{d}{dt}S(t) = -\tau I(t)S(t) \tag{1}$$

$$\frac{d}{dt}I(t) = \tau I(t)S(t) - (\beta_1 + \beta_2)I(t) \tag{2}$$

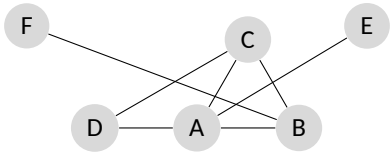
$$\frac{d}{dt}R(t) = (\beta_1 + \beta_2)I(t) \tag{3}$$

with certain initial conditions that are clear $S(0) > 0, I(0) > 0, R(0) \geq 0$ (we assume that at the beginning there exist indeed infected and susceptible individuals). In this case, τ is the transmission rate, β_1 the removal rate of actually reported infected individuals, and the coefficient β_2 is the removal rate of infected individuals occurring for other unreported causes such as mortality or other reasons. We will not enter into the details about how to obtain the solutions of these equations or how these solutions qualitatively behave. Most of the time researchers approximate the solutions using mathematical software. Usually neither $I(0)$ nor $S(0)$ are known and therefore simulation algorithms are used to calculate those values. One of the big challenges for the determination of the parameters τ, β_1, β_2 is how to identify unreported cases [7].

We now define the most import quantity in mathematical epidemiology, a threshold called *basic reproduction number* and that is (almost) everywhere denoted as R_0. The quantity R_0 is defined as $R_0 := \frac{\tau S(0)}{\beta_1+\beta_2}$, which translated to words is the expected number of new infection cases generated by one single person. As you may imagine, the coefficients τ, β_1 and β_2 (therefore also R_0) are determined from the data provided by the health systems. The main idea is that there is an epidemic outbreak whenever $R_0 > 1$. The basic reproduction number will change in the course of the epidemic and when this value decreases below 1, the epidemic will disappear.
There are several types of compartmental models, of course. Another very interesting model is SIS: susceptible, infected, susceptible. Here, it is assumed that the individuals recovering from the infection do not have immunity against the infectious agent. These models are better suited to describe infections by helminths and most of the sexually transmitted diseases.

3 Stochastic models

There are serious limitations with basic compartmental models such as those described by Kermack and McKendrick. These models assume that the sizes of the groups are big enough so that the mixing of members is homogeneous. However, in reality this is not the case since at the beginning of an outbreak there is only a reduced number of infected individuals, and the transmission of the disease is an stochastic event depending on the network of contacts of those infected persons.
Such stochastic events are well described by the so-called Galton-Watson processes. The network of contacts can be described by what we call a *graph*. In this graph, the members of the population are represented by vertices and the contacts between individuals are represented by edges. The number of edges at one vertex is what we call the degree of the vertex. We are interested especially in those vertices having a lot of edges, since the individuals in these vertices can potentially infect many people.
The next graph shows how we represent such networks.



In these models we assume that all contacts between an infective and a susceptible individual result in a new infection, however this assumption can be relaxed. It is also assumed that infective individuals make contacts independently of one another. The quantity p_ℓ denote the probability that the number of contacts by a randomly chosen individual is exactly ℓ, with the technical condition $\sum p_\ell = 1$. For this model, the epidemic outbreak begins when an infective transmits the infection to all of the individuals with whom he or she is in contact. This branching process outbreak is explained in more detail in [5].

There are different variants of this model, where the assumptions on the transmission and the functions that generate the model are slightly different.

If you want to dive more deeply into these interesting topics you can consult the book [2].

References

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