- Toy models are, for example in physics, extremely simple models and highly idealized, used to get an idea of a phenomenon that is in reality much more complex

  The most famous toy model used in physics is perhaps the armonic oscillator

- Let's talk about epidemic spreading models

  Tomorrow we will introduce a network of contacts

- Let's start with the SI model, the simplest model in which you have two compartments: infected I and susceptible S (can be infected)

  The number of I and S is constant: nobody dies (it's a toy model!)

$$\frac{dI}{dt} = +\beta \frac{S(t) I(t)}{N_0} \qquad \frac{dS}{dt} = -\beta \frac{S(t) I(t)}{N_0}$$

$$\boxed{\frac{dI}{dt} + \frac{dS}{dt} = 0}$$

$N_0(t) = N_0 = \text{costant}$

β = a certain constant

There's no detail about the specific virus spreading in the population

- It's easy to find an analytical solution, by considering that $S(t) + I(t) = N_0$

$$\Rightarrow S(t) = N_0 - I(t)$$

if $x(t) = \frac{I(t)}{N_0}$

$$\Gamma x(t) = \frac{I(t)}{N_0} \rfloor$$

$x(t) \propto e^{\beta t}$ There's an indefinite growth!

Nobody dies, and nobody recovers! This model is pretty bad

- An attempt to improve the model is the SIR (susceptable, infected, recovered), there's a third compartment

R is a group of people that either recover from the pathology or die

$$\frac{dS(t)}{dt} = -\beta \frac{S(t)}{N_0} I(t) \qquad \frac{dR(t)}{dt} = +\gamma \frac{I(t)}{N_0}$$

$$\frac{dI(t)}{dt} = +\beta \frac{S(t)}{N_0} I(t) - \gamma \frac{I(t)}{N_0}$$

$$\frac{dS(t)}{dt} + \frac{dI(t)}{dt} + \frac{dR(t)}{dt} = N_0 = \text{Costant}$$

γ, β = Costant parameter

This model has still a problem, someone that recovers could be infected again, but this model don't presuppone that.

We can solve numerically the equations produced by this model

The asyntotic value of r can be obtained from the numerical analysis

$$r_\infty \approx 1 - S_0 e^{-\frac{\beta r_\infty}{\gamma}} \qquad \text{with} \quad r_0 \approx 1$$

$$R_0 = \frac{\beta}{\gamma}$$

With R_0 <1 the disease would die out exponentially

With R_0 >1 the disease will grow exponentially

This is the same R_0 as the Covid pandemic!

We assume that the probability of remaining infected decrease exponentially with time.

$$\underbrace{p(\tau)\,d\tau}_{1} = \gamma e^{-\gamma t} d\tau$$

probability of remainig infected for a time τ and then recovering in a time interval between τ and $\tau + d\tau$

In the covid pandemic this was verifyed, this is how we measured γ for calculating the R_0

- The SIS model is an attempt to include the possibility of re-infection

In this model the recovery from the pathology does not confer immunity

There are again two compartment

$$\begin{cases} \frac{dy}{dt} = \gamma x - \beta yx \\ \frac{dx}{dt} = \beta yx - \gamma x \end{cases}$$

this factor is new!
It presuppones the possibility of recovering from the infection but becoming again subsceptible to it

$$\frac{dy}{dt} + \frac{dx}{dt} = \text{costante}$$

It can be solved numerically, again

$$x_\infty = \frac{(\beta - \gamma)}{\beta} < 1$$

It's different from SI!

At a certain point, we reach an equilibrium point between people that go from S to I and people that go from I to S

Another model is the SIRS! Individual recover from the infection but the immunity is only temporary

$$\frac{ds}{dt} = \delta r - \beta s x$$

$$\frac{dx}{dt} = \beta s x - \gamma x \qquad s + x + r = 1$$

$$\frac{dr}{dt} = \gamma x - \delta r$$

This model has not analytical solution. Approximate solutions are found by linearizing the model, with tools of nonlinear dynamics or by numerical integration.

- Let's try applying our models on real datas

$$S I R$$

$$s(t) + x(t) + r(t) = 1$$

We can make an assumption, a strong one, that the rate of recovery γ is costant
This is the same to say that the probability to recover DOES NOT depend on the time an individual has already been infected

$$P(T_I > t) \propto e^{-\gamma t}$$

Dove T_I rappresenta la durata del periodo di infettività.

γ = costante

THIS IS AGAINST EMPYRICAL EVIDENCE! But it's a """"necessary"""" simplification to keep things simple

We make another assumption (Ansaz), at the start of an epidemic the number of supsceptible people is almost constant $S(t) \sim S_0$

Those thing make so we have an exponential growth in the number of infected

$$x(t) = x_0 e^{(\beta s_0 - \gamma)t}$$

Dalla teoria approssimata ho: $x(t) = x_0 e^{(\beta s_0 - \gamma)t}$

Dall'analisi empirica ho: $x(t) = x_0 e^{rt}$

Pertanto: $r = \beta s_0 - \gamma$ $\quad \frac{r}{\gamma} = \frac{\beta}{\gamma} s_0 - 1$ $\quad \frac{r}{\gamma} = R_0 s_0 - 1$ $\quad R_0 = \frac{\beta}{\gamma}$

One can also introduce the constant R_e defined as:

$$R_e = R_0 s_0$$

which incorporates the information about the initial fraction of susceptible.
Since we know that $R_0 = \beta/\gamma$, one finally gets:

$$R_e = 1 + \frac{r}{\gamma}$$

which gives an empirical way to measure the infection rate.

$R_e \approx R_0$

- By some numerical analysis, we can get 3 parameters:

$$A = \gamma x_0$$

$$B = -\gamma(1 - R_0 + x_0 R_0)$$

$$C = -\frac{\gamma}{2}(1 - x_0)R_0^2$$

And then...

⇒

$$r(t) = \frac{-B}{2C} + \frac{\sqrt{-B^2 + 4AC}}{2C} tg\left(\varphi + \frac{\sqrt{-B^2 + 4AC}}{2} t\right)$$

$$\varphi = arctg\left(\frac{B}{\sqrt{-B^2 + 4AC}}\right)$$

```
Plot[(apprr[t] /. {alpha → alfa, phi → fi}) /. {R0 → 1.5, x0 → 0.001, gamma → 0.2}, {t, 0, 100}]
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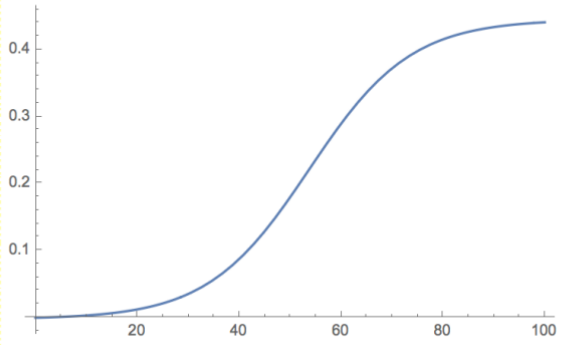

This kind of approximation was used by Kermack and McKendrick in 1927 to modelize the epidemic of Bombay (1905-1906)

$$\frac{dR}{dt} = \frac{890}{cosh^2\ (0.2t - 3.4)}$$

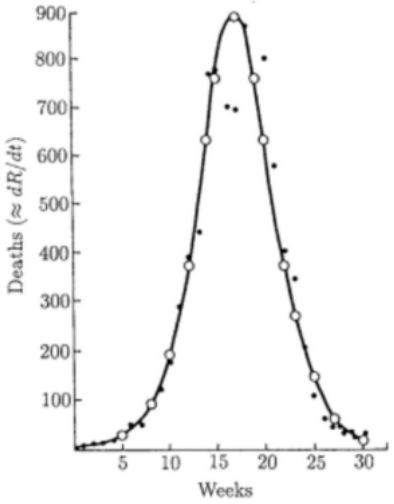

Figura 2.3: Epidemia di Bombay del 1905 - 1906). Confronto fra i morti registrati (•) e previsti dalla teoria (○) per settimana.

This kind of result is why the SIR model was popular!

Prof. tried to apply the SIR model with real data from COVID, but it's impossible

Those described above are models that follow the traditional approach where each individual potentially interacts with all other possible other individuals with the same probability.
This is the so-called **fully-mixing approach**.

However, this is not a realistic representation of the way people interact with each other. In fact, it is nowadays very well accepted that individuals interact through **network of contacts**. In other words, individuals interact with a small fraction of all the possible individuals and such contacts are not chosen at random. Rather, these contacts are the result of choices that are both personal and dictated by the environment where each of us operates.

Networks of contacts therefore play a crucial role in the way epidemics spread over a given society.

That's where complex networks come into play