Markov Models for Infectious Diseases

Marcelo Coca Perraillon

University of Colorado
Anschutz Medical Campus

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Outline

- Common models in economic evaluations
- Trying to make sense of terminology
- Microsimulations: discrete event simulations, agent-based models
- Teaching in the Time of the Coronavirus: dynamic Markov models in epidemiology
  - Susceptible Infected Recovered (SIR)
  - Susceptible Exposed Infected Recovered (SEIR)
- Excel examples
- Further extensions
- Models versus reality

Big Picture

- Not a complete catalog; there are many types of models that are hard to classify
- Most common in EEs: "microsimulations" like **discrete event simulation** and **agent-based** models (confusingly, also called Monte Carlo simulation models)
- We will try to make sense of some jargon today
- We'll explore the basics of infectious disease models
- There are many models out there but we will focus on the **Susceptible-Infected-Recovered (SIR)** model because it's relatively easy to do using Excel
- Another variant is the Susceptible-Exposed-Infected-Resistant (SEIR). **This model was used to understand some COVI-19 scenarios in Colorado**

Patient-level simulations

- Patient-level simulations (or **microsimulations**) model the transitions of an **individual patient** rather than a cohort
- Very useful to make transition probabilities dependent on patient history, not just cycle time
- For example, we could take into account that the probability of an event depends on age, sex, disease severity, or time with the disease
- These models are all about memory, so we do not have to worry about the **memoryless assumption assumption of Markov models**
- Some of these models take into account how people **interact with each other** (key aspect of infections diseases)

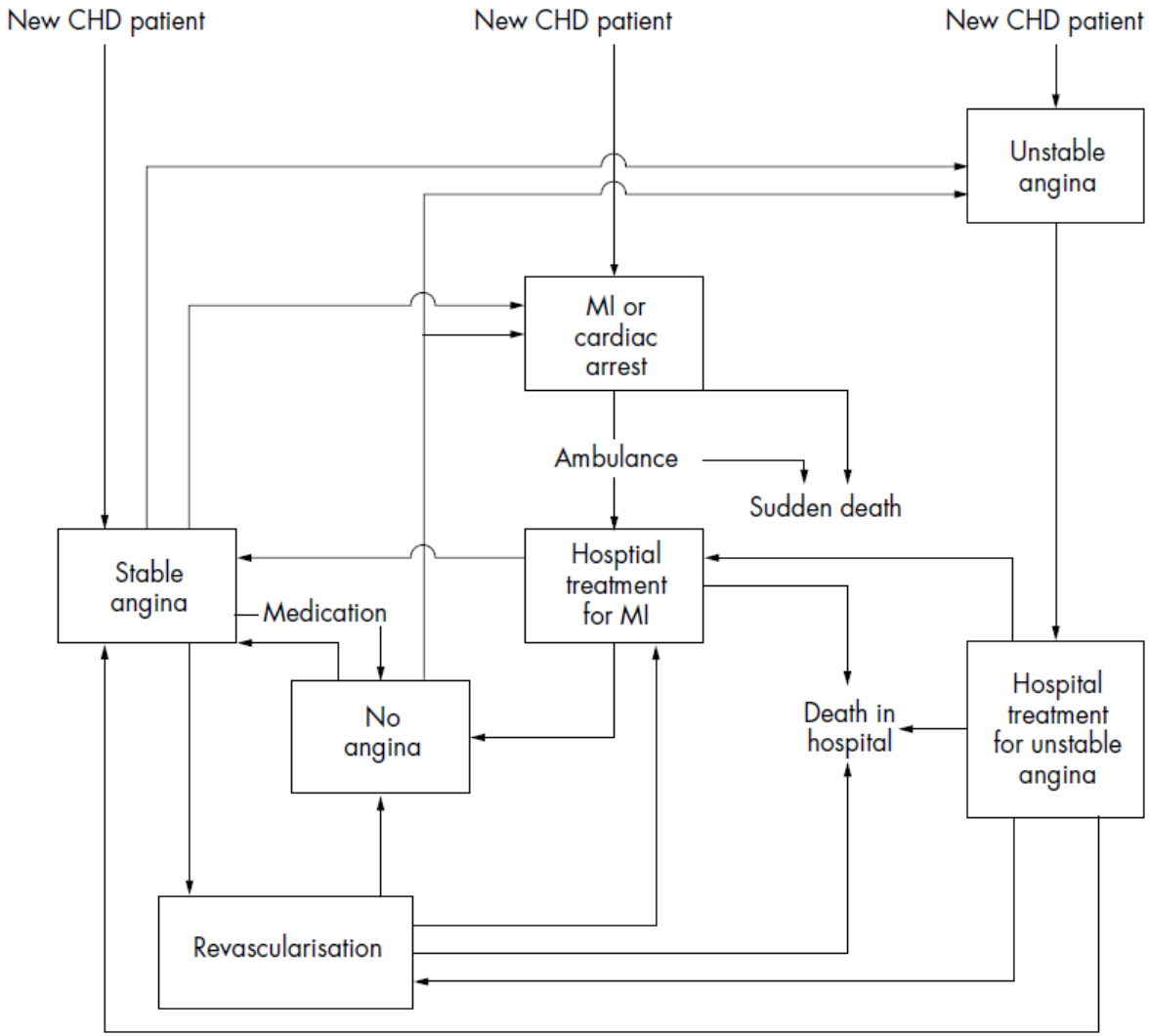
Discrete event simulation (DES)

- Origins in **operations research** (design and optimization of industrial processes). As a result, they use different terminology
- Like Markov models, events happen in time (**discrete periods**, like cycles)
- It models **entities** (e.g. patients, hospitals) that have **attributes** (e.g. age, sex, disease history)
- Attributes can change in the model and they determine how entities interact with their **environment** and how they react to **events**
- **Events** are "things" that could happen to an entity in an environment (e.g. death, infection, MI, stroke)
- Costs and outcomes (e.g. life years, QALYs) can be added

Example

- Cost effectiveness of improving ambulance and thrombolysis response times after myocardial infarction (Chase et al, 2006) (available on Canvas)
- "Interventions: Improving the ambulance response time to 75% of calls reached within 8 minutes and the hospital arrival to thrombolysis time interval (door-to-needle time) to 75% receiving it within 30 minutes and 20 minutes, **compared to best estimates of response times in the mid-1990s**"
- Below is a graphical representation of the model

DES model


New CHD patient
New CHD patient
New CHD patient
Unstable angina
MI or cardiac arrest
Ambulance
Sudden death
Stable angina
Medication
Hosptial treatment for MI
No angina
Death in hospital
Hospital treatment for unstable angina
Revascularisation

Disadvantages

- It may not be obvious but **more realism is not always better**. Remember Einstein (if he actually said it):

  Everything should be made as simple as possible, but not simpler
- More realism means that **a lot more data** is needed
- How do we get transition probabilities that depend on age, sex, disease history?
- **Many times the type of model follows the data**. Given the data we have, what is the best model we can build?
- Sensitivity analyses are more difficult in DES models (specially probabilistic sensitivity analyses), which often can't be done
- Computational burden can be a big problem

Infectious diseases and vaccinations (static versus dynamic models)

- Microsimulation models have a clear advantage when considering infectious diseases and vaccinations studies
- The likelihood of contracting an infection depends on the number of people already infected
- Vaccines can be effective even if not all people are vaccinated (**herd immunity**)
- In **static** models, the rate of infection (i.e. transition probability) is fixed. In **dynamic** models, rate of infection is not fixed and may depend on the number of people already infected (i.e. possible to model epidemics)
- Discrete event simulations are useful in these situations; agent-based models are a variation. But our trusty Markov model can be adapted (more in 10 minutes)

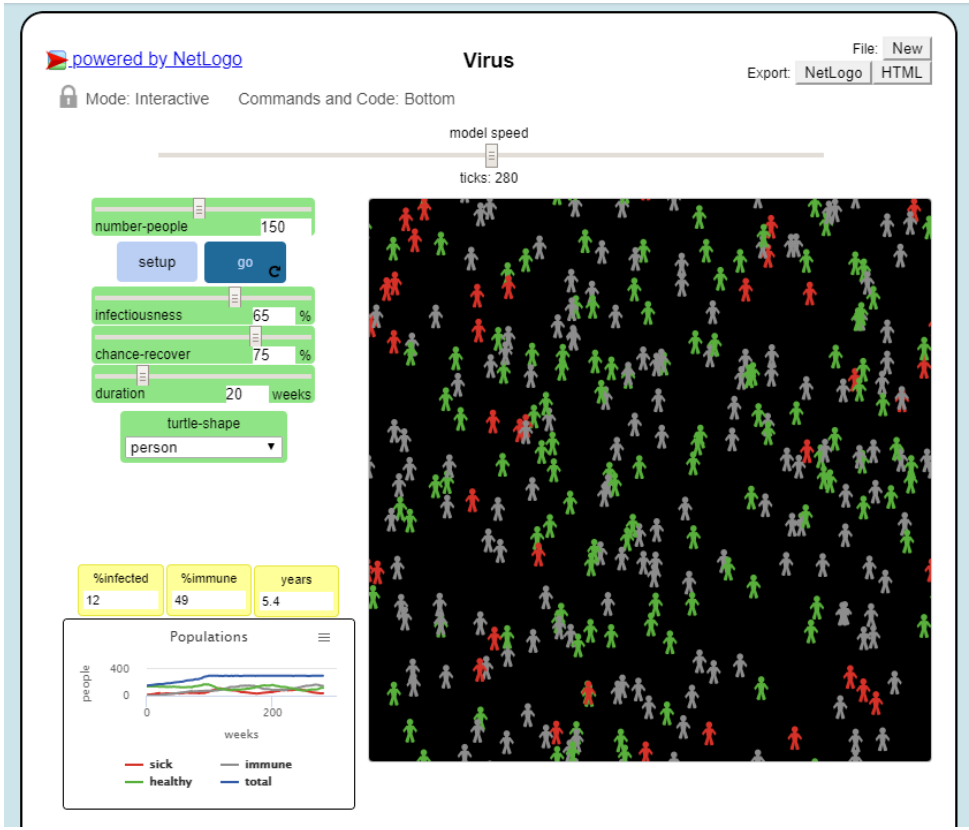
More on types of models

Table 1. Pharmacoeconomic model types.

Model type	Description	Pros and cons
Decision tree models	Depicts patient health state changes with sequential probability branches from initial state or intervention	*Pros*: intuitive, very versatile and flexible, easy to depict clinical pathways graphically *Cons*: data requirements easily expand beyond available information, does not allow direct accounting of time spent in various states of health
Markov models	Depicts patient health state progression over multiple time cycles with fixed health state transition probabilities (could vary probabilities)	*Pros*: all costs and outcomes easily calculated for every time interval, parameter uncertainty and sensitivity analysis easy to incorporate, parsimonious information requirements *Cons*: assumes that transition history does not matter, ~~state transition probabilities are fixed over time~~
Discrete event simulation models	Depicts patient health state progression over multiple time cycles with variable health state transition probabilities	*Pros*: costs and outcomes calculated at each time interval, more realistic than fixed transition probability models *Cons*: can be difficult to compute results and sensitivity analyses, has greater data requirements
Monte Carlo simulation models could be called agent-based	Depicts patient health state progression through computer simulation of multiple individuals with random pathways based on model probabilities	*Pros*: extremely flexible and realistic *Cons*: most computationally difficult, requires the most data

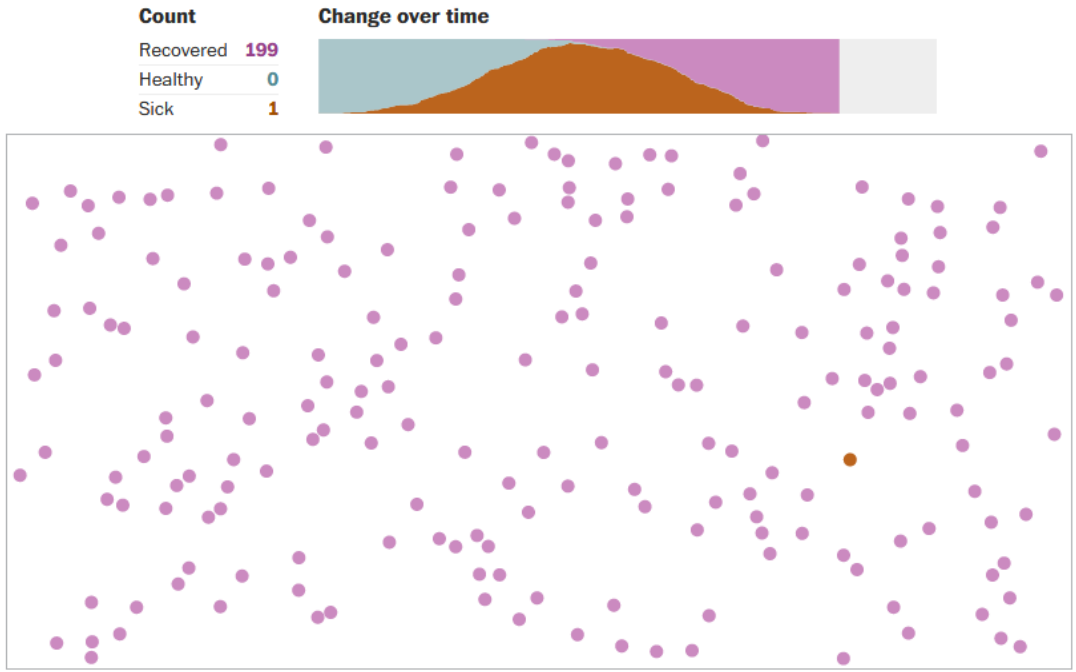
- Adapted from Hay (2004). Trying to make sense of modes, with my notes in red
- Software: Net Logo, Arena, and @Risk

Classic Logo



- Basic (and graphical) agent-based modeling of bacterial infection. I used to program Logo when I was a kid
- Source: `http://netlogoweb.org/`

Agent-based Coronavirus simulation



- Very popular (viral?) animation in the Washington Post: `https://www.washingtonpost.com/graphics/2020/world/corona-simulator/`
- Very nice graphs but **not possible to study the simulation analytically**

Summary

- Many modeling techniques are used in cost effectiveness but by far the most common are decision trees and Markov models
- They come under a myriad of names. Methods originate in different fields
- Today we will cover a classic model for infectious diseases extensively used in epidemiology
- They are not called Markov models in epi, but they are in fact Markov models – **dynamic Markov models**
- See Blackwood and Childs (2018) for an excellent introduction (on Canvas)
- We can use the same tools we have learned during the last month to understand and simulate these models

Susceptible-Infected-Recovered (SIR) model

- This model assumes people can be in 3 **states**: susceptible to become infected, infected, or recovered
  1. **Susceptible** people have no immunity to the disease. These people can become infected by coming into contact with an infected person
  2. **Infected** people have the disease and they can spread it
  3. **Recovered** people survive the disease and have immunity
- These are states as we saw before but in these models they are referred as "compartments." For this reason models like these are called **compartmental disease models**
- There is not much agreement on names, but you can think of these models as **dynamic Markov models** that come in two flavors, deterministic and stochastic. We'll focus on deterministic models
- **Modeling nomenclature is all over the place**

Dynamics

- They are "dynamic" because people move from one compartment (state) to the other at different rates over time. In other words, the **transition probabilities are not fixed, they change over time**
- The transition probabilities are themselves a function of other parameters in the model (which we will cover soon)
- The math can get complicated. The rates at which people go from one state to the other are derivatives; the model is actually a **system of differential equations**
- The equations were derived by Kermack and McKendrick almost 100 years ago, in 1927
- In **stochastic dynamic Markov** models, the parameters are also given a probability distribution
- But we don't have to "solve" a system of differential equations. We'll use simulations to understand the key insights of the model

Transition Diagram

- The **force of infection** depends on the proportion of the population who is infected and the transmission rate. We will denote it by λ (lambda)
- The **recovery rate** is the rate at which people recover from the infection. We will denote it by γ (gamma)
- (I'm following the convention in this type of model, but there should be an arrow to itself from the Recovered state. Nowhere else to go because of immunity.)

Force of Infection

- The force of infection (the equivalent of a transition probability) is not a constant as in decision trees or the other Markov models we covered
- It is defined as $\lambda(I) = \beta \frac{I}{N}$, where β is the transmission rate, I is the number of infected, and N is the total population
- This is a realistic way of modeling the chances a person is infected. The larger the proportion infected ($\frac{I}{N}$) the more likely it is people can become infected
- β, the transmission rate, is a function of the **rate of contact** and **probability of transmission** given contact
- COVID-19 has a high β. Lockdowns, "social distancing" (should be physical distancing), masks, etc are attempts to lower either the rate of contact or the probability of transmission (therefore, lowering β)
- In this simple model, we capture all these features with the parameter β, but we could define $\beta = rc \times p(T)$

How are things going to change over time?

- Here is the part where the math of the model can be complicated because I need to show you derivatives, **but derivatives are just rates of change**
- Over time the rate of change in the number of susceptible people is going to go down as a proportion of the force of infection (negative slope):
  $\frac{dS}{dt} = -\lambda(I)S = -\beta\frac{I}{N}S$
- $\frac{dS}{dt}$ is read as "the derivative of S with respect to time, t,"
- Read it as the change in the number of Susceptible over unit of time, where t could be days, weeks, or months (technically, continuous)
- Same as with the other Markov models. We need to adjust parameters depending on whether we use days, weeks, or months

How are things going to change over time?

- The *change* in the number of infected (I) per unit of time depends on the susceptible becoming infected and the infected recovering
- Again, don't lose sight of the big picture: we are now modeling the **transition probabilities**. In previous classes we assumed they were fixed numbers given by the transition matrix
- So the change in the infected people over time is:
  $\frac{dI}{dt} = \beta \frac{I}{N} S - \gamma I$
- The first term is the same as the previous slide –susceptible people becoming infected as a proportion of the proportion infected. The second term, γI, are the people recovering after being infected
- That's the most important equation (more on this later)

How are things going to change over time?

- **Where are the death?** In this baby version of the SIR model we are not modeling it directly
- We could just add a Dead state, but it's not strictly necessary: the death will be a proportion of the infected
- Or we could assume, as usual, that people can die in any state
- The last component is the change in the Recovered:
  $\frac{dR}{dt} = \gamma I$
- One more thing, although in this basic model is not really needed: at any point the total number of people (N) must add up:
  $N = I + S + R$

SIR equations

- To summarize the simple SIR model:
  1. $\frac{dS}{dt} = -\beta \frac{I}{N} S$
  2. $\frac{dI}{dt} = \beta \frac{I}{N} S - \gamma I$
  3. $\frac{dR}{dt} = \gamma I$
  4. $N = I + S + R$
- Except for 4), a constraint, that's a system of differential equations

Fine-tuning the model and variations

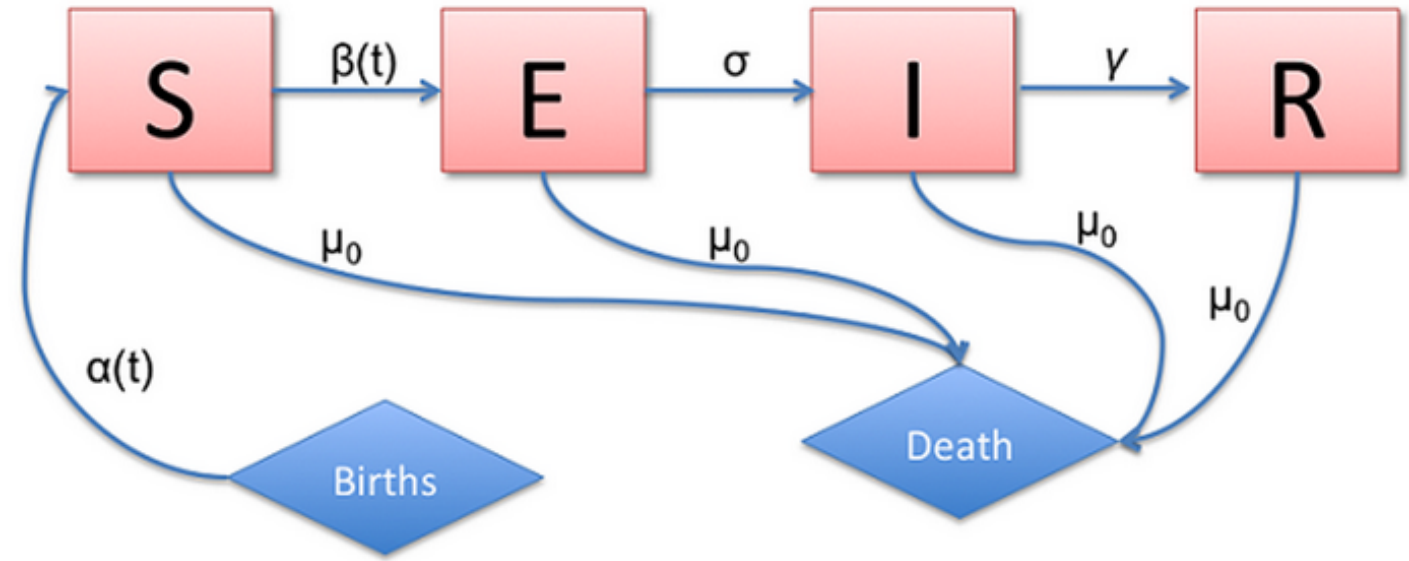
- If you google the SIR model be aware that many variations exist
  1. Some versions explicitly add mortality, which could flow from any of the states or just the infected state. For the coronavirus, mortality could be a function of the number of people hospitalized. Higher if too many in hospitals without ventilators. Or a function of hospital capacity. Or just a proportion of the infected (I)
  2. Some incorporate vaccines. A proportion of the Susceptible could become vaccinated at a rate ν (nu) (good for modeling measles for example)
  3. Some versions use proportions of people as inputs, so $\frac{I}{N}$ instead of I, but the end result is the same
  4. Some versions could add more realism by making parameters a function of other parameters. For example $\beta = cr \times p(T)$, where cr is the rate of contact and $p(T)$ is the probability of transmission
- Of course, an important rule of modeling: **more realism involves more needed information**
- Coronavirus models have used an extension of the SIR model, the SEIR version

Susceptible-Exposed-Infected-Recovered (SEIR)

- This version adds another state, the Exposed state
- The idea is to explicitly model one aspect of infections that is important: the incubation period
- In SEIR, there is an incubation rate; the rate at which exposed people become infectious
- Adding a latency or incubation period delays the initial spread of the disease –the SEIR model is more realistic than the SIR model because of the incubation period
- SEIR model is another example of the time-honored trick we covered in our last class. Want to incorporate more features? **Add more health states!!**

Susceptible-Exposed-Infected-Recovered (SEIR)

- This is an example of a SEIR model incorporating births and death. Births, of course, are fairly irrelevant to model the coronavirus



Transfer diagram for the SEIR model with seasonality in birth rate and transmission.

- Source: Dorélien, Audrey M., Sebastien Ballesteros, and Bryan T. Grenfell. "Impact of birth seasonality on dynamics of acute immunizing infections in Sub-Saharan Africa." PLoS One 8, no. 10 (2013).

World Health Organization Coronavirus model

- The WHO developed a SEIR model for COVID-19 incorporating other features like travel from China. See here for more details: `https://triplebyte.com/blog/modeling-infectious-diseases`
- WHO model parameters

Parameter	Definition
$S(t)$	Number of susceptible individuals at time point t
N	Total population
R_0	Basic reproductive number
D_I	Mean infectious period
$I(t)$	Number of infectious individuals at time point t
$z(t)$	Force of infection in baseline scenario
$L_{I,W}$	Average daily number of international inbound air passengers to Wuhan
$L_{C,W}$	Average daily number of domestic inbound travelers to Wuhan from mainland China
$L_{W,I}$	Average daily number of international outbound air passengers from Wuhan
$L_{W,C}$	Average daily number of domestic outbound travelers from Wuhan to mainland China
$E(t)$	Number of latent individuals at time point t
D_E	Mean latent period

The Colorado Model

Model description. We used an age-structured susceptible, exposed, infected, recovered (SEIR) model to project the number of people with COVID 19 needing hospitalization, critical care and the number of deaths in Colorado under different intervention scenarios (Figure 1).

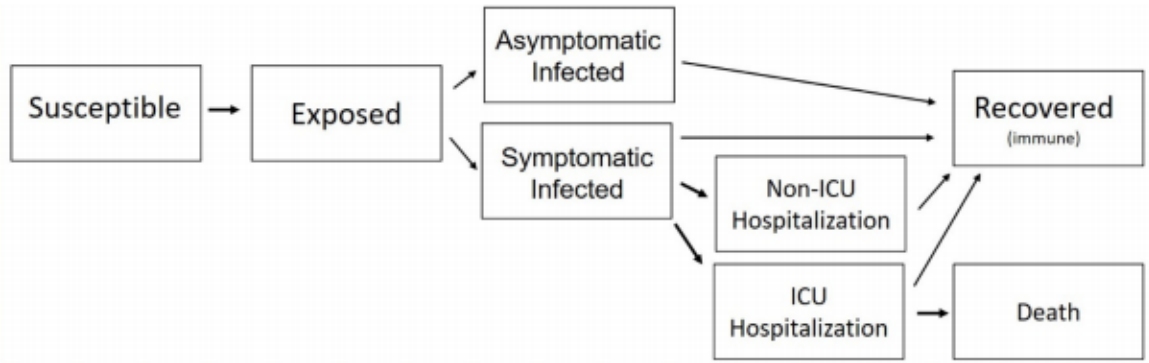


Figure 1. Structure of the deterministic SEIR model used. Infected individuals are separated into asymptomatic and symptomatic individuals. Symptomatic individuals may recover without hospitalization, experience a non-ICU hospitalization or an ICU hospitalization.

- Split Infected into two groups states (asymptomatic, symptomatic). Death and Recovered are absorbing states. Stratified by age
- Source: `http://www.ucdenver.edu/academics/colleges/PublicHealth/coronavirus/Pages/coronavirus.aspx`

The Central Role of R_0

- There is a model number that is central for understanding **how contagious a disease is**, the famous R_0 ("R-nought")
- R_0, called the **basic reproductive number**, is defined as the"average number of secondary cases arising from a typical primary case in an entirely susceptible population" (Blackwood and Childs, 2018)
- Think of this as the average (expected) number of cases generated by one case, or the average number of individuals an infected person infects
- For example, studies of measles have determined that R_0 for measles is about 12 to 18 (super high!). Mumps 4-7. Influenza? 1.4-1.6, but it depends on strains, as low as 0.9.
- COVID-19? **We don't know**. Best guesses, anywhere from 1.4 to 4

Department of Major (MAJOR!) Confusion

- R_0 **is not a fixed number**. How many people (on average) an infected person infects depends on the **features of the disease** and the **behavior** of people
- If all of the population is under (draconian) quarantine (like in Bolivia), then R_0 will go down
- The numbers I mentioned above are based on studies because we have had plenty of time to study other diseases, so they are average numbers based on past outbreaks
- Public health interventions are trying to lower R_0. But in the context of the basic SIR model, R_0 has to depend on the force of infection β and the recovery γ
- So changing β and γ means changing R_0, but it's not that COVID-19 has a natural, fixed R_0 that is out there to be found
- Or think about it this way: the R_0 in CO is different than that of FL or Bolivia or China. The virus is the same (so far)

Back to the simple SIR model

- Again, intuitively it makes sense that R_0 should depend on the force of transmission and the recovery rate
- The higher the recovery rate, the less time a person stays infected and stops transmitting the disease
- The higher the force of infection, the higher R_0
- Recall that the rate of transmission depends on the probability of transmission after contact (feature of the disease) and the contact rate (behavior driven): $\beta = cr \times p(T)$
- I'm saying here that $p(T)$ is a feature of the disease, but it depends how you define it. Wearing masks is trying to reduce the probability of transmission given contact, so it could depend on behavior as well

Back to the simple SIR model

- **In both our simple SIR model and the SEIR model, it turns out that** $R_O = \frac{\beta}{\gamma}$ (R_0 varies depending on the structure of the model)
- Another way to write this is $R_O = \beta \times \frac{1}{\gamma}$
- β is how fast a person is infected (transmission rate). $\frac{1}{\gamma}$ can be viewed as **how long a person is in the infected state**
- So the interplay between how fast a person is infected (contact rate, probability of transmission given contact) and how long a person is infected are **key to determine the dynamics of the model**

"Solving" the SIR model

- The system of differential equations can be studied analytically to understand the features of the model: How quickly will people become infected? How many? For how long? How can this be changed?
- A key concept in any model like this is the **equilibria** of the model
- Essentially, how does the model look like when there is **stability** and nothing is changing? By that I mean that S, I, and R stop changing over time
- Mathematically, equilibrium means that all the differential equations are equal to zero (that is, no more change)
- These stability scenario could mean that there are no more infections (**disease-free** equilibrium) or the disease becomes **endemic** (infections steadily persist; endemic equilibrium)
- (I'm skipping one part that is not that important in the simple SIR/SEIR model: the other important part in the analysis of differential equations is whether the equilibria are stable or not)

"Solving" the SIR model

- In our simple SIR model, all the population is susceptible, with no vaccine. So the only possible equilibrium is no more infections because at **some point everybody will be infected** ("disease-free" equilibrium)
- There could be an opportunity to stop an infection, by making β zero, but that means changing the parameter, which is not an outcome of the model, but an input (**this distinction is very important**)
- We are NOT going to solve the model analytically. We will play with a simulation, same stuff we did with our Markov models
- I'll just say that R_0 is critical to understanding the behavior of the SIR/SEIR models
- If $R_0 < 1$, the disease stops, slows spreading. If $R_0 = 1$ the disease becomes endemic (infections change steadily). If $R_0 > 1$, the disease will continue to spread unless something structurally changes that lowers R_0

SIR model in discrete time

- To use Excel we need to make a slight change: we need to make time discrete, not continuous
- The discrete time version of the model is similar, but now derivatives become discrete changes
- So $\frac{dS}{dt}$ becomes, for example, $\frac{\Delta S}{\Delta t}$. "Delta" S from one period to the other is just $S_{t+1} - S_t$. To make things simple, time will change by 1 unit. So $\frac{dS}{dt}$ is now $\frac{S_{t+1} - S_t}{1}$
- $t + 1$ is next period, t is current period
- So we have the same system of equations written in a slightly different way:
  1. $S_{t+1} - S_t = -\beta S_t$
  2. $I_{t+1} - I_t = \beta S_t - \gamma I_t$
  3. $R_{t+1} - R_t = \gamma I_t$
  4. $N_t = S_t + I_t + R_t$
- Why is this helpful? Because we can program those formulas in Excel

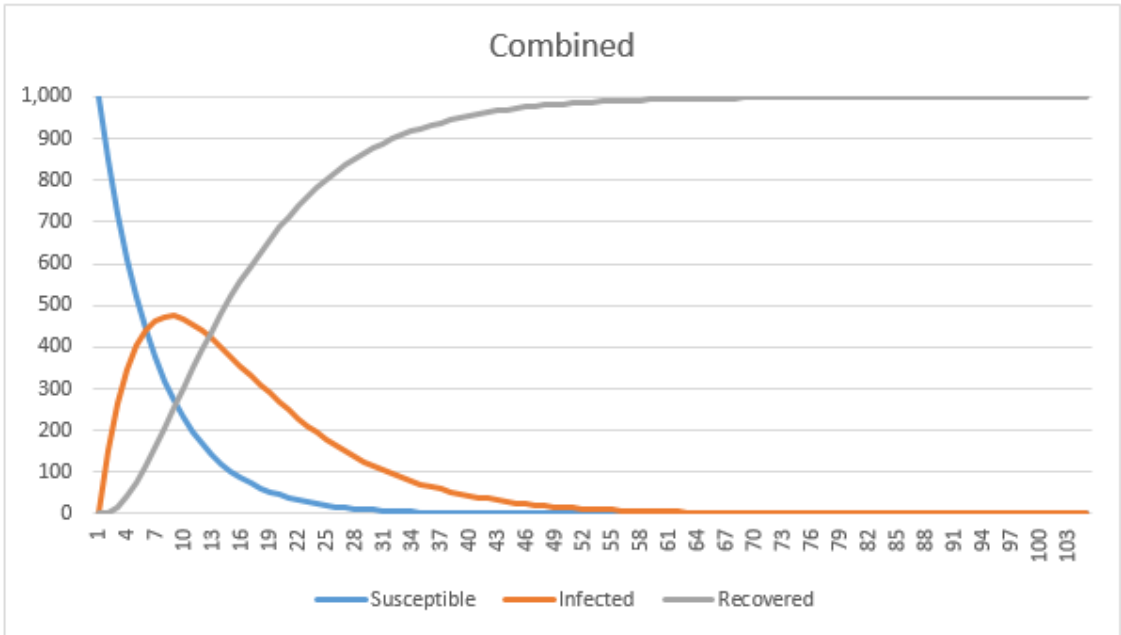
Excel

- We'll start with $N = 1000$, with one person infected, so $I_0 = 1$
- Check out the Excel file. **Note that we are modeling cumulative numbers, not the changes**
- So instead of $I_{t+1} - I_t = \beta S_t - \gamma I_t$, I'm using $I_{t+1} = \beta S_t - \gamma I_t + I_t$

Time	Susceptible	Infected	Recovered	Check	Parameters			
0	999	1	0	1,000	N	1,000		
1	499.5	500.4	0.100	1,000	I (start)	1		
2	249.75	700.11	50.140	1,000				
3	124.875	754.974	120.151	1,000	beta	0.5	R_0	5
4	62.4375	741.9141	195.648	1,000	gamma	0.1		

SIR graphically

- These curves should look familiar by now
- Try changing β, from 0.05 to 0.5. That would make R_0 go from 0.5 to 5
- Keep $\gamma = 0.1$ when changing β but check how it changes too



Insights

- So what did we gain from these models? So many things...
- Epidemics can come fast, with a large number of people becoming infected extremely quickly. Infections grow exponentially. Worse with a new, highly contagious virus – everybody in the population is susceptible
- Interventions that lower β, and therefore R_0, can "flatten" the curve. Remember, a month ago leaders (and the public) didn't seem to understand these issues
- Flattening the curve is important because if people do not become infected at the same time we have enough hospital capacity to treat them. If not, mortality is going to be higher

Insights

- The curve can be flattened by changing the contact rate (lockdowns, 6 ft apart), the probability of transmission given contact (masks, shields), and improving the recovery rate (ventilators, experimental medications)
- Note something else: **we have a public health crisis and we have not even modeled mortality**
- It may seem as it was 10 years ago, but about a month ago some politicians were arguing that COVID-19 was not a big deal because mortality is the same as the flu
- We actually don't know if mortality is the same. The number of cases means little. We do not know the actual number of infections (denominator). Testing has been a failure
- But we could have a crisis regardless. And if we have a capacity crisis, more people will die

Insights

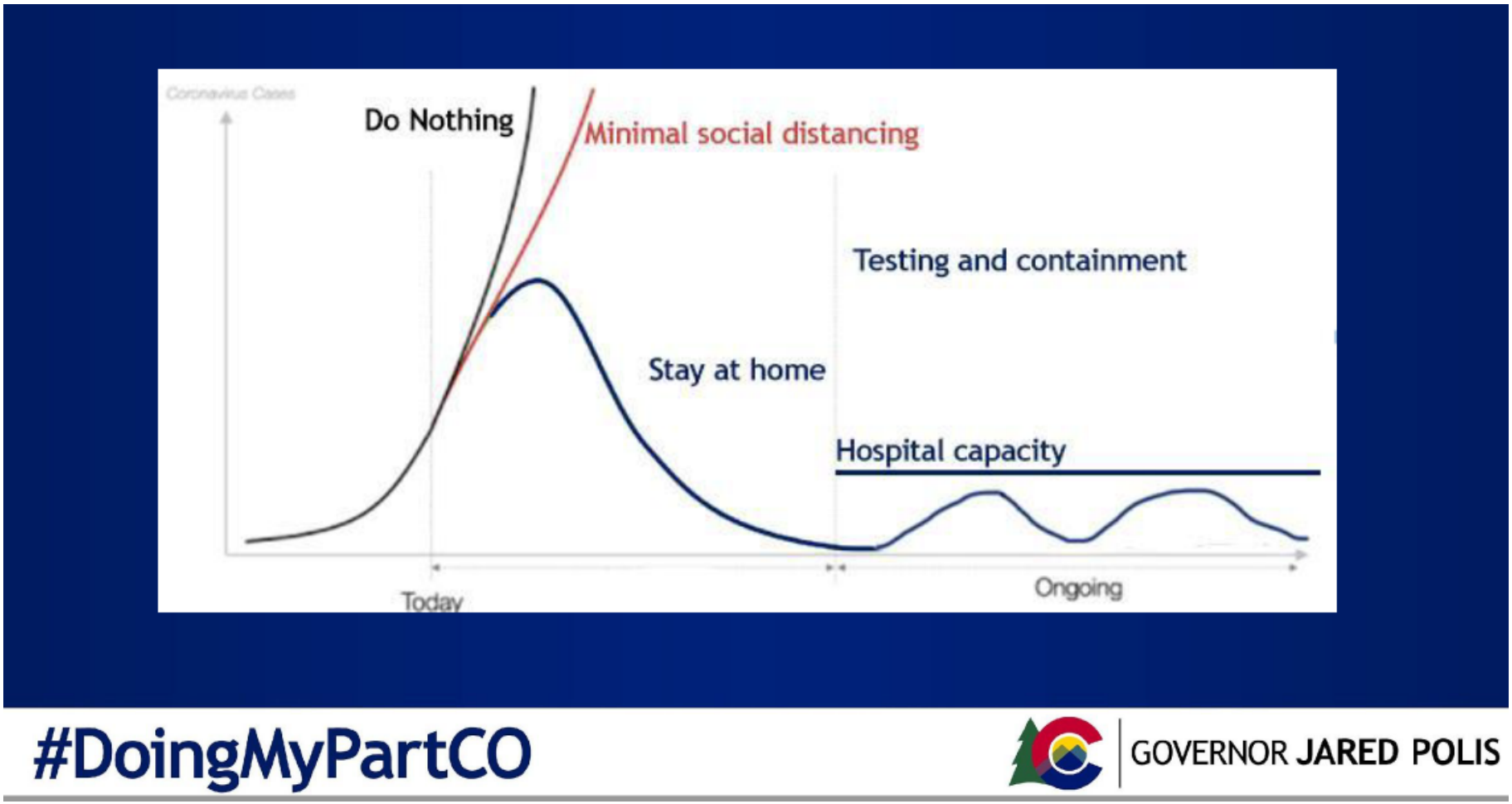
- Flattening the curve doesn't mean that the disease is over. With no cure, we are **extending the epidemic**. *Flattening the curve implies a longer outbreak*
- In our SIR model the equilibrium is disease-free because everybody eventually will get sick (high R_0). Nothing in the model stops transmission other than running out of susceptible people. There is no vaccine
- Why is there a peak? Because the virus starts infecting people but at some point the susceptible numbers start to come down; the fraction of infected in the population then decreases. At some point there is more recovering than getting infected
- Stare at the equation for the infected for a while: $\frac{dI}{dt} = \beta \frac{I}{N} S - \gamma I$
- That equation is positive at the start, then turns negative when $\gamma I > \beta \frac{I}{N} S$. Rewrite as $\gamma > \beta \frac{S}{N}$

Models are abstractions, not reality!!

- It's great that now everybody and their grandmothers understand flattening the curve and health system capacity
- But models are models, they are not reality
- In a couple of years, models will be "calibrated" and we will have a better estimation of the parameters. Right now, we are guesstimating
- As usual with models, people are starting to think that the model is reality. **In particular, there is no reason to believe the actual data will be a clean curve**
- It's likely to be more like rolling hills. Once the worse has passed, we need to relax the lockdown. That implies *increasing* β in the middle of the simulation. What will happen? We'll probably get another peak since there is no vaccine yet and we have plenty of susceptible people in the population
- I love how somebody "drew" rolling hills in Gov Polis slides

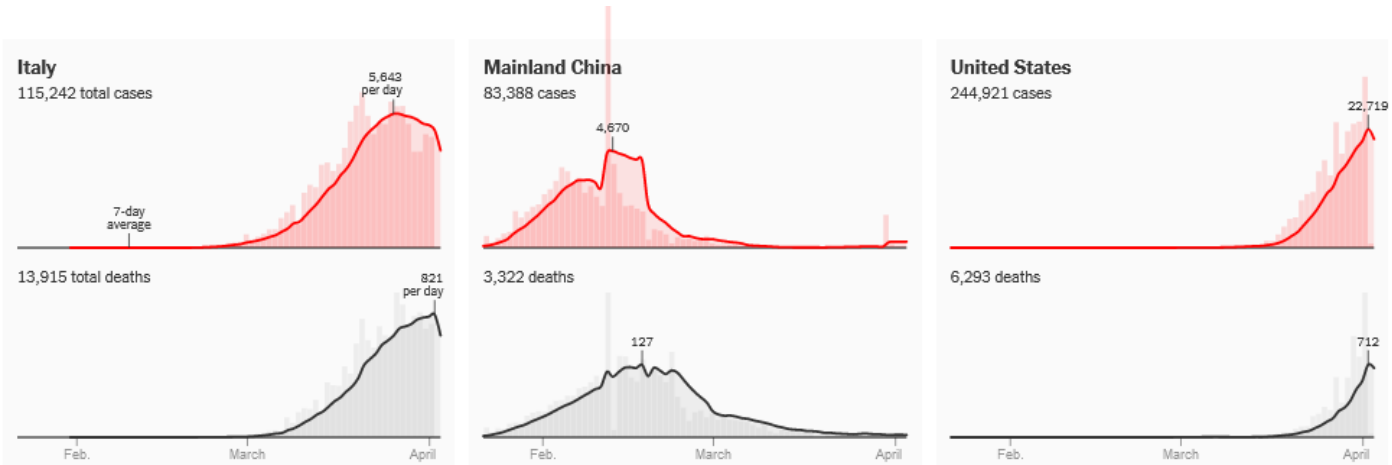
Photoshop simulation?

- Rolling hills, unsteady hand version. The rolling hills should have been drawn early in the simulation, though



Real data

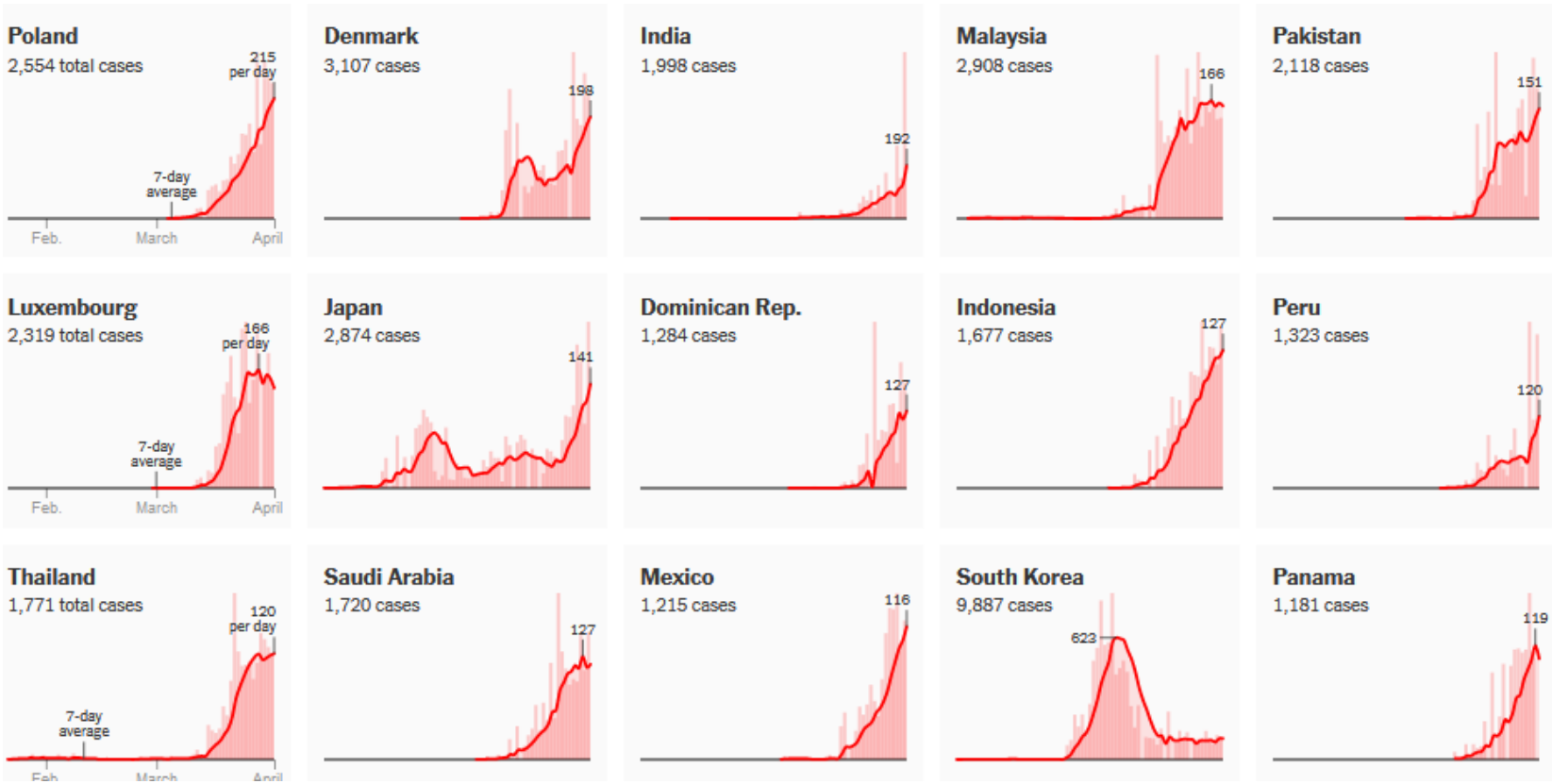
- The New York Times published some nice graphs (hard to tell if China's data are reliable)
- Mortality is mostly a proportion of cases



- Source: `https://www.nytimes.com/interactive/2020/04/03/world/coronavirus-flatten-the-curve-countries.html`

Real data

- See Japan and South Korea (longer since first case)



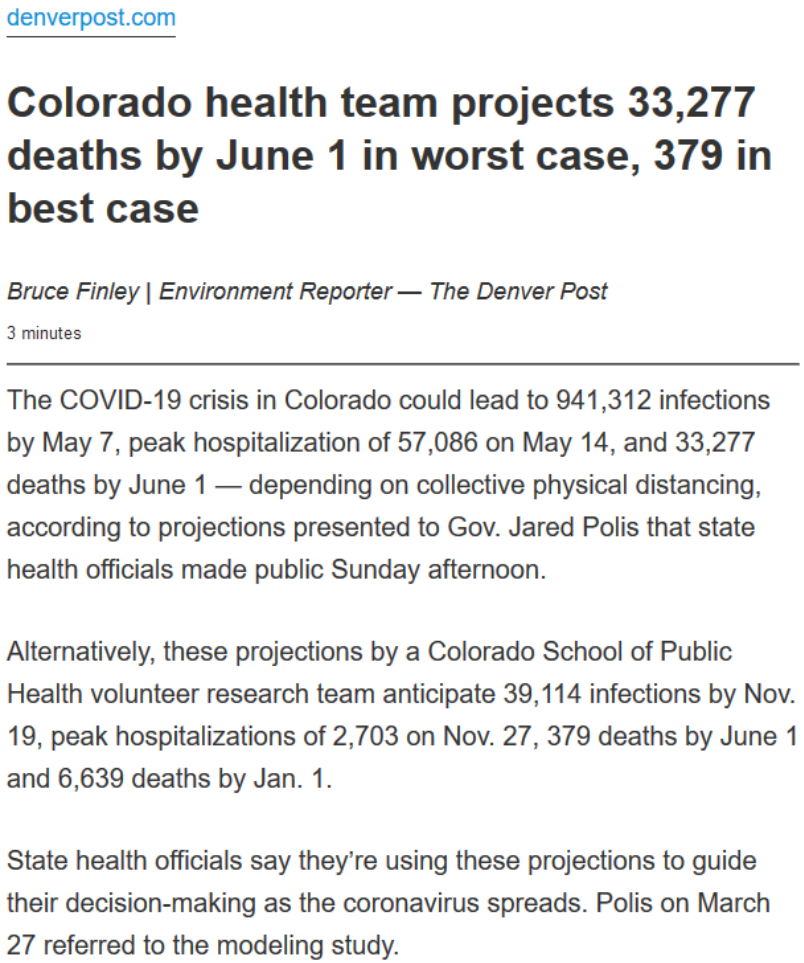
- Source: `https://www.nytimes.com/interactive/2020/04/03/world/coronavirus-flatten-the-curve-countries.html`

Should we use these models to make predictions?

- Yes and no. Yes in the sense that we can make predictions about the effects of policy changes
- But no in the sense that even more sophisticated versions of these models do not reflect actual reality. We have a lot of **parameter uncertainty** (we'll cover this next week). **And with exponential growth, small changes can make a world of difference**
- The numbers projecting 100,000 to 200,000 deaths are crude estimates at best – most likely, they are meaningless (already revised as of 4/8)
- We would be better-off using actual data rather than simulation models to make projections. In particular, we need to take into account demographics. SEIR models being used are state-level
- This article in the Atlantic about modeling is good: `https://www.theatlantic.com/technology/archive/2020/04/coronavirus-models-arent-supposed-be-right/609271/`

Colorado

- Notice the uncertainty



denverpost.com

Colorado health team projects 33,277 deaths by June 1 in worst case, 379 in best case

Bruce Finley | Environment Reporter — The Denver Post

3 minutes

The COVID-19 crisis in Colorado could lead to 941,312 infections by May 7, peak hospitalization of 57,086 on May 14, and 33,277 deaths by June 1 — depending on collective physical distancing, according to projections presented to Gov. Jared Polis that state health officials made public Sunday afternoon.

Alternatively, these projections by a Colorado School of Public Health volunteer research team anticipate 39,114 infections by Nov. 19, peak hospitalizations of 2,703 on Nov. 27, 379 deaths by June 1 and 6,639 deaths by Jan. 1.

State health officials say they're using these projections to guide their decision-making as the coronavirus spreads. Polis on March 27 referred to the modeling study.

- Source: `https://www.denverpost.com/2020/04/06/colorado-projected-coronavirus-covid-deaths/`

How does this end?

- Recall that in a SIR/SEIR model without vaccination and $R_0 > 1$, the stable equilibrium means that there are no more infections because the virus ran out of susceptible people
- Policy in some states has managed to lower β (the rate of transmission). Remember, to run the simulation we didn't change beta, therefore, R_0
- If we could test everybody in the country at the same time, we could isolate infected people, thus lowering β. It's a bit unrealistic but better testing for sure will help
- Most likely, when things get better, we'll relax, and then tighten, lockdowns from time to time. **Relaxing lockdowns doesn't mean going back to normal**
- There won't be normalcy until a vaccine is developed
- Note how all these predictions are informed by the SIR/SEIR model **but are not model outcomes from one simulation**

Resources

- This is changing daily but some models have come to the top
- The White House paid attention to the IHME model from U of Washington: `http://www.healthdata.org/covid`
- Two obnoxious things about that model: black box. Can't find an explanation of their methods anywhere. Nice job with the marketing, though. Another thing: they show a clean curve. Misleading
- The other model is from the Imperial College in London. They have good documentation `https://www.imperial.ac.uk/media/imperial-college/medicine/sph/ide/gida-fellowships/Imperial-College-COVID19-NPI-modelling-16-03-2020.pdf`
- They have scenarios with more peaks

Summary

- Many types of models are used in economic evaluations
- Usually models give us important insights; insights that were not clear before constructing a model
- But models are just models. Always be careful with models. Understand the difference between an **assumption** versus an **outcome** of the model. *It's not easy*
- Do not take models too seriously. Particularly, do not make the mistake of believing that a model is reality
- Read this article by Paul Krugman on the role of models in economics `http://web.mit.edu/krugman/www/formal.html`