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Abstract

Introduction to the *hows* and *whys* of mathematical modeling.

Most of the text is about the details, especially of the COVID-19 disease. **Remarks about the general *hows* and *whys* of modeling** are set in boldface.

Modeling

First, we need to talk about *models*.

- **A model is a *structure* for thinking about something complex.**
- **But models are always *incomplete* and *idealized* to some extent.** We say they are *abstract*.
- Models may be purely *informative*, as in artist's models. An artist's model is often not sitting for a portrait. The purpose of the model is so that the artist can support the *art* with a certain amount of realism.
- Models can guide our behavior, often prescriptively (or *normatively*). That's why we talk about "model students." Teachers want other students to *emulate* those models.
- A map (graphic image) is a model.
- In mathematical logic, a model is a map (function).

There was a model for this painting ...

Removed due to copyright concerns. Please use the Google search below to view a whole page of examples of paintings of people that are not portraits, by Pablo Picasso.

`https://www.google.com/search?q=picasso+woman+images`

Modeling in Policy and Planning Science

- **Models** may be *causal* or *policy-oriented*, that is, they **help us to *control* some phenomenon.**
- In statistics, there are usually *two* models. One model comes from a scientific domain, and describes the policy-relevant aspects. The other part is purely statistical, and uses randomness to describes aspects we cannot control, and usually cannot even observe.

Mathematical modeling

- Consider a 3D printer, or any printer—an image is also a model of the thing it depicts. How does it work? You feed it numbers to tell it where to put tiny drops of plastic (3D) or ink (2D).
- **The image produced by the printer is a physical representation (model) of the object. The list of numbers the computer sends to the printer is a quantitative (mathematical) representation of the object.**
- Of course, once we have a quantitative representation, we can abstract further by using *functions* to generate the numbers.
- Once we have a functional representation, we can use *algebra*, *calculus*, and even more advanced mathematics to analyze our model (because, of course, the function is another representation, or model, of whatever we are studying).

Causal modeling

- In science, and even in daily life, the most important models are *causal models*. They explain behavior we observe in terms of causes and effects.
- If we ask an attractive person to go out with us, and they say no, we can *ask* them *why* to directly acquire a model of their behavior. But if they say yes, we may *imagine* they find us attractive too. Imagining is a very dangerous way to acquire models. Consider: that's how marriage frauds make money!
  - **We have no choice but to *start* with our imaginations. But we should not *rely* on such models until we have *verified* them.**
- The point of science is to ask *why*. *E.g.*, why does a person become sick with the *disease* COVID-19? Our model is that the *necessary* condition is infection with the *virus* SARS-CoV-19. Both cause and effect are actually in the name: "SARS" stands for "severe acute respiratory syndrome" which tells us about the disease, "CoV" stands for "corona virus," and "19" for the year of discovery, 2019.
  - Showing cause and effect is why scientists like names like "SARS-CoV-19" although ordinary people just say "the coronavirus."

Modeling COVID-19

Let's think about an example of building a model.

- What do we know about disease, for example, the flu? It's pretty much the same every year nowadays: in the autumn we go to the doctor, get vaccinated, a few people get sick, a very few get very sick and die, and then we repeat the following year.
- **This is the *very* simplest model: a *constant*.** What we *expect* to happen next time is what happened last time.
- COVID-19 is new (that's the "novel" in "novel corona virus"). **By that very fact, the idea of a constant model** (next year will be the same is this year) **is undermined.** Next time (tomorrow, with COVID-19) is not going to be the same as last time (yesterday, before COVID-19). We see that in the papers every day: new cases—and new deaths.
- Yet the constant model plays a fundamental part in the political economy of the pandemic.

A bad model

- The political argument is that we see that the business shutdown orders had a big economic effect:
  - before the order, most people took a few precautions and then went to school, work, and play, but
  - after the order, many businesses shut, people stayed home, and
  - we see a very large negative impact on the economy.
  - Therefore, it was a bad idea to shut all the businesses before we were sure that the virus was spreading explosively.
- This is based on the *constant model*. Why? Because the standard of comparison for "very large negative effect" is *last period's GDP* (or *employment*, *etc.*).
- The implied assumption is "if we had no shutdown order, the economy would work the same as last year," *i.e.*, the constant model.

Rejecting the bad model

- OK, it's the constant model. Is that *bad*? After all, it works for the flu, and business activity was going on as before.
- The problem is that **we know** from the experience of Wuhan (China), Bergamo (Italy), and New York (US) **that COVID-19 is different from the flu.** We *don't* have *any* vaccine, a relatively *large fraction* of the population gets sick, and relatively *many* of them get sick enough to die.
- Finally, it had a great effect on the economy even before shutdown orders in those places, even if you *only* count economic losses due to sick workers and shortages from falling production, and the like.
- **The constant model is *untenable*** (we can't defend it) **in the case of COVID-19.**
- To estimate the costs (economic and otherwise) of business shutdowns, **we need to evaluate a *counterfactual***: "What would the level of economic activity be *with* COVID-19 but *without* the shutdown?

Counterfactuals

- How do we evaluate a counterfactual? You guessed it, I'm sure. We build a model. *We have no choice but to build a model.* **Working with counterfactuals *always* involves models.**
- Sometimes we can avoid explicit modeling. If we're lucky, we have appropriate data and we use analogical reasoning: *this* situation is like *that* situation, so the outcome *this* time should be like the outcome *that* time.
  - This is just an alternative description of the constant model.
- Building a model of the effect of COVID-19 on a national economy is hard. So hard that I don't know of any professionals willing to publish theirs yet.
- Part of economic modeling must be modeling the disease's *medical effects*, which is not something we can do in Shako, especially not this class.
- But there's a component of that model that we can at least get started on: the *epidemiology* of the virus (*i.e.*, the scientific study of how it spreads). **We *decompose* the problem and model the parts we *can* understand.**

Simplest epidemiological model, *cont.*

We need to keep asking "why?" and what that means for our model.

- Why can't we use the constant model? We know from the worst-hit cities that the SARS-CoVID-19 virus has *faster than linear growth*, which varies from place to place (and responds to policy) and is estimated to have a *doubling time* of 2–7 days.
- Assuming we take no special action, and a doubling time of 2 days (worst case), starting from *one* infected person, we get the table on the next slide.
- If one of us is sick today, in a week the whole zemi has gotten sick, in a month the whole university is infected, in 5 weeks all of Tsukuba, in 7 weeks all of Kanto, 5 days after that all of Japan, in 9 weeks all of Asia, 2 days after that the whole world, and 2 days after that … uh, wait …() oops.
- **Lesson #1:** You can run, but you can't hide from exponential growth.
- **Lesson #2:** Exponential growth is a bad model of an epidemic. It predicts *impossible* outcomes.

Table: Doubling Time of 2 Days

day	0	2	4	6	8	10	12	14	16	18	20	22	24
count	1	2	4	8	16	32	64	128	256	512	1,024	2,048	4,096

day	26	28	30	32	34	36	38
count	8,192	16,384	32,768	65,536	131,072	262,144	524,288

day	40	42	44	46	48	50
count	1,048,576	2,097,152	4,194,304	8,388,608	16,777,216	33,554,432

day	52	54	56	58	60
count	67,108,864	134,217,728	268,435,456	536,870,912	1,073,741,824

day	62	64	66	68
count	2,147,483,648	4,294,967,296	8,589,934,592	17,179,869,184

Table 1: Exponential growth model

Shifting gears to define a better model

- As we'll see later, the exponential growth model is not a bad model in the way that the constant model is. The constant model is *just plain bad* because with data, it can only predict the same thing (or an average), and *without* data, it predicts anything you can imagine.
  - I can imagine 10 million people in Tokyo with viral pneumonia from COVID and 10,000 ventilators—two weeks later Tokyo would literally be a ghost town.
  - Politicians seem to imagine 5000 people sick with COVID, and plenty of ventilators for people with asthma (like me) too.

  Arguing about our imaginings is useless.
- But **to see that exponential growth is a somewhat useful model it's helpful to use different mathematics**, namely *continuous time*. Our calculations were done with *discrete time*, calculating only for every two days. What about odd days? Can we say something about hours or minutes?

Discrete time to continuous time

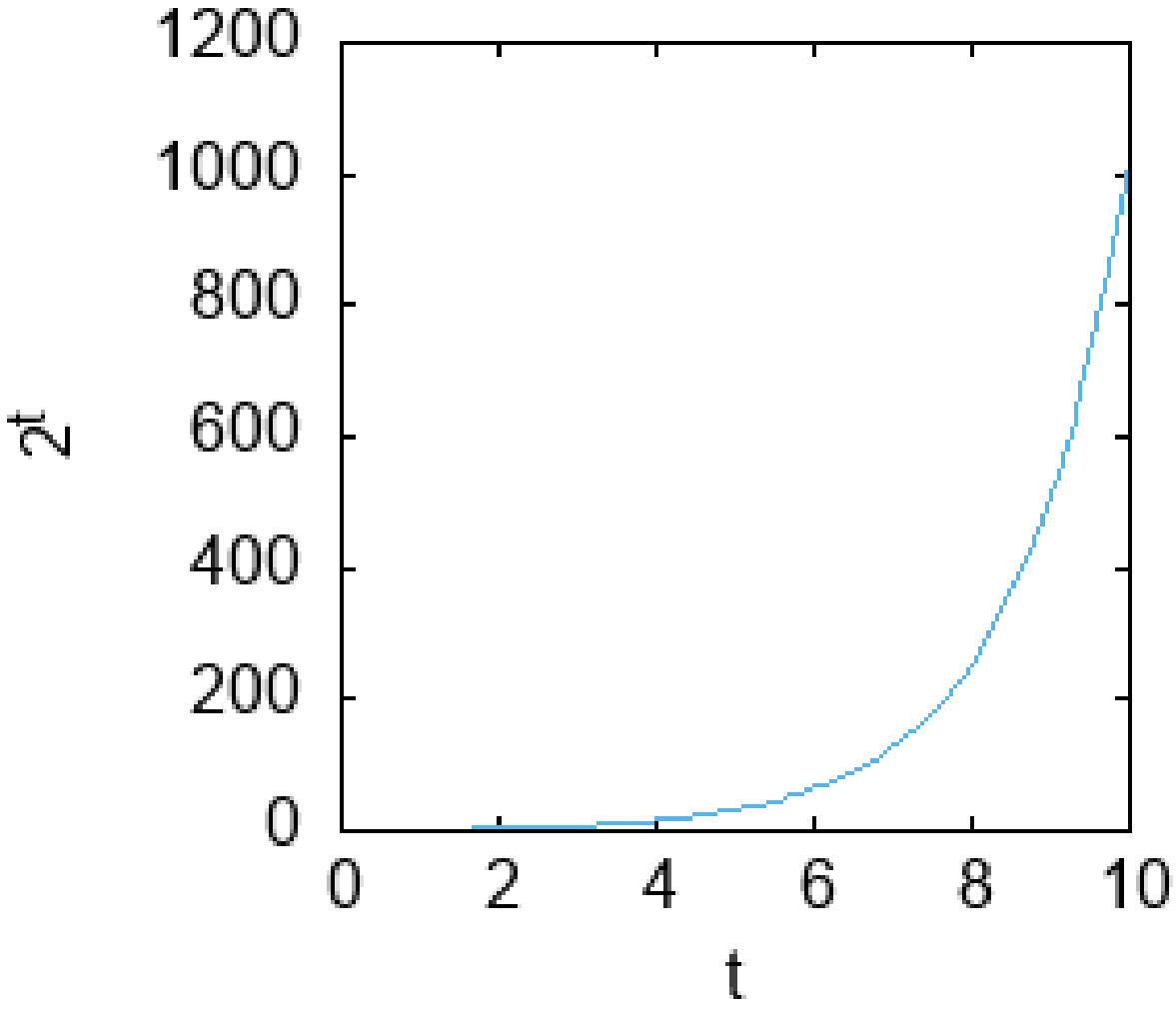
- A bit of thought will show that after t days, we have $2 \times 2 \times \cdots \times 2$ (multiply by 2, $\frac{t}{2}$ times), or $N = 2^{\frac{t}{2}}$ infected individuals. So if we allow t to be odd numbers, or fractional numbers, we can still calculate this (with a computer).
- But this is not very helpful. The first change we're going to make in our model is to change the doubling time so that we can't have more sick people than we have people!
- To express this, the trick is to look at the relationship between the *number of infected individuals* and the *rate of increase of infected individuals.*
  - On any day we can count the infected individuals N.
  - Two days later we'll have $2N$.
  - The *rate of increase* is $2N - N = N$ (with the unit of time being 2 days).

  We have expressed the rate of increase directly in terms of N.
- In continuous time, we use calculus and write $\frac{dN}{dt} = N$.
- If we integrate, we get *the* exponential function $N(t) = \exp(t) = e^t$.

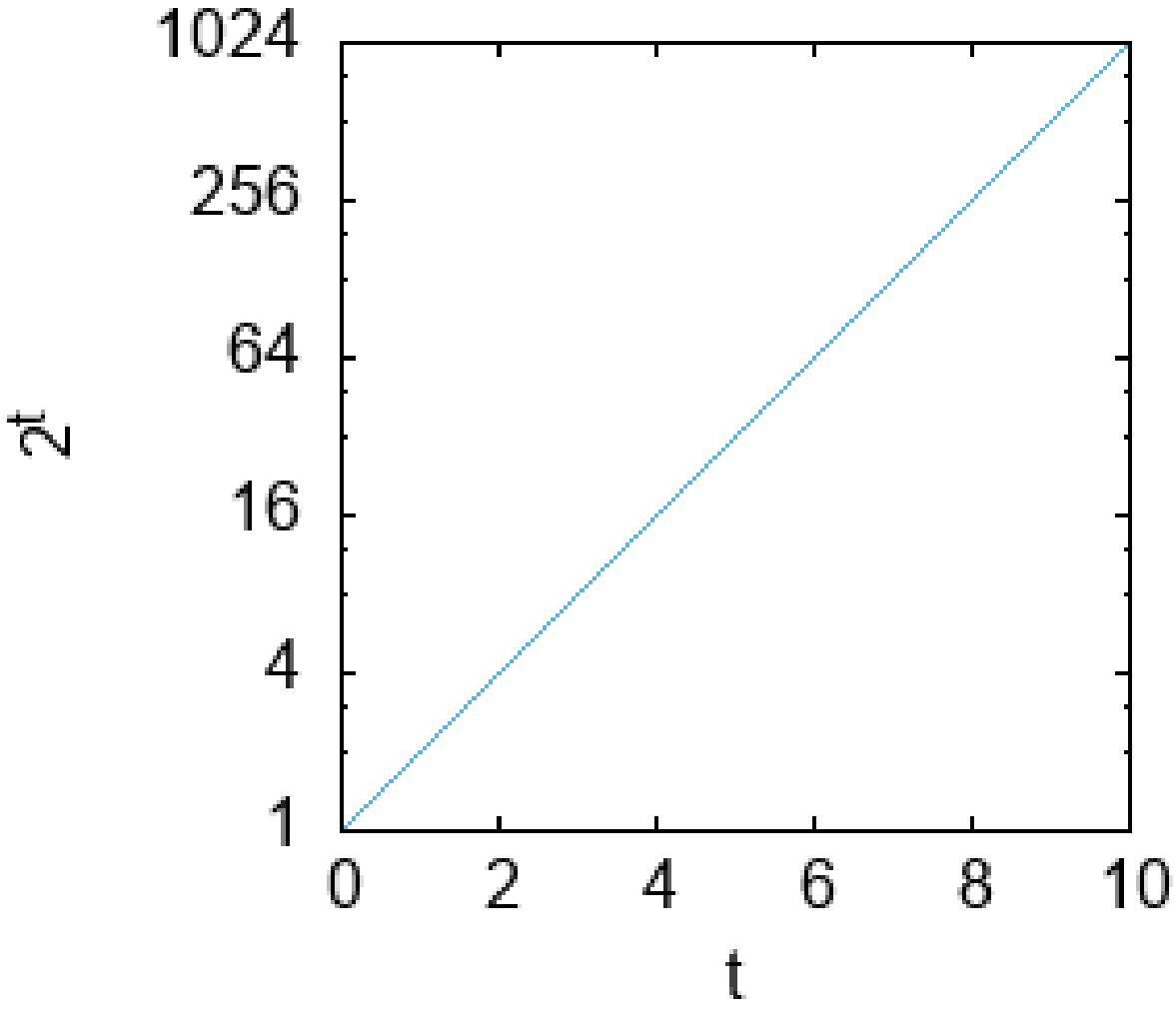
Exponential functions

- For our purpose, *the* exponential function e^t is *defined* by the *differential equation* $\frac{dN}{dt} = N$.
- e is an irrational number, approximately 2.718281828459045 according to my iPhone.
- There are many exponential functions, such as $2^{\frac{t}{2}}$ (and 2^t), but e^t is *the* exponential function because the differential equation is so simple.
- In fact, all exponential functions can be expressed as $f(t) = Ae^{\alpha t}$, and they all have linear differential equations $\frac{df}{dt} = \alpha f(t)$.
- Although the *rate of increase* $\frac{df}{dt}$ changes over time, the *rate of growth* $\frac{df/dt}{f} = \alpha$ does not.
- Try the exponential function $2^{\frac{t}{2}} = e^{0.346573590279973\,t}$ with even numbers t!

Graph of exponential growth

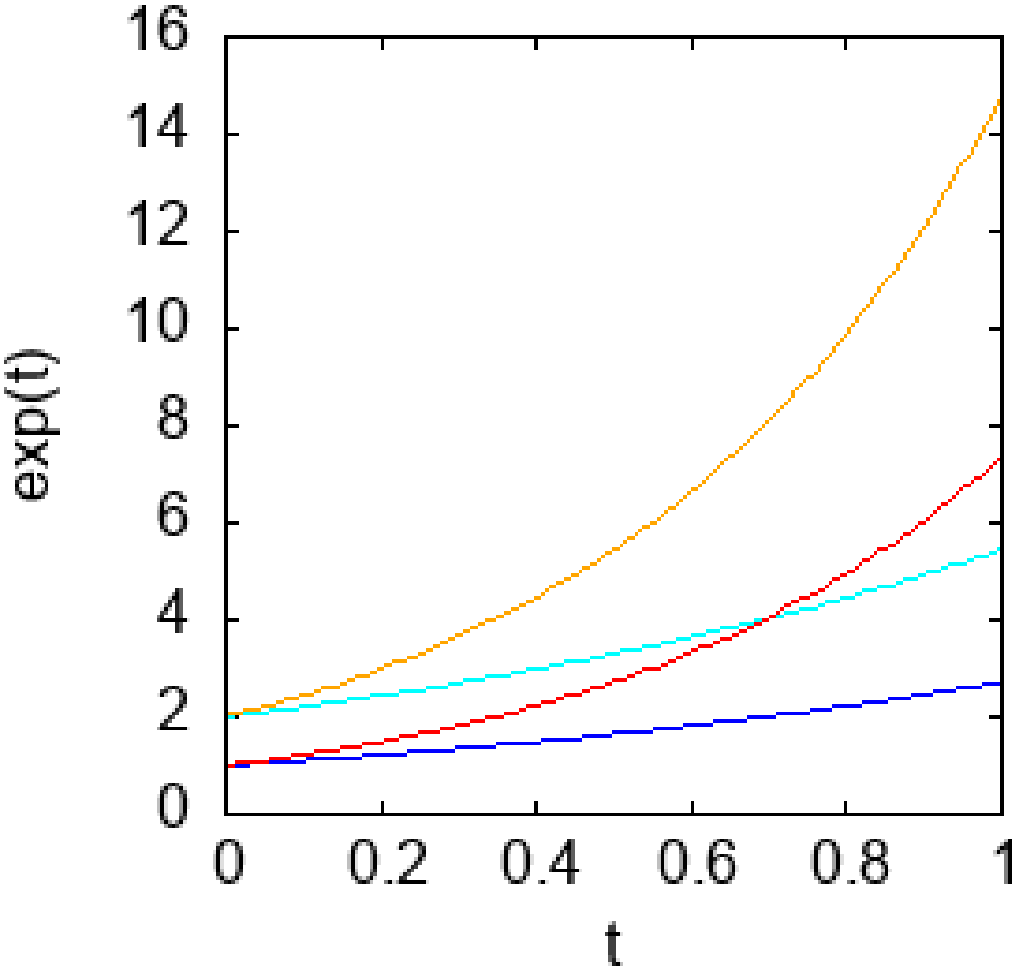


Logarithmic-scaled graph of exponential growth



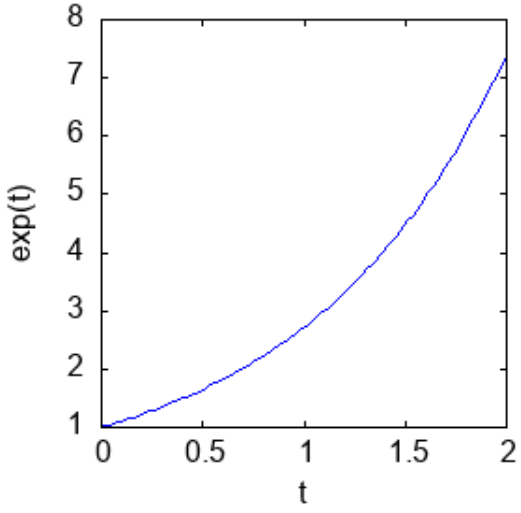
More exponentials

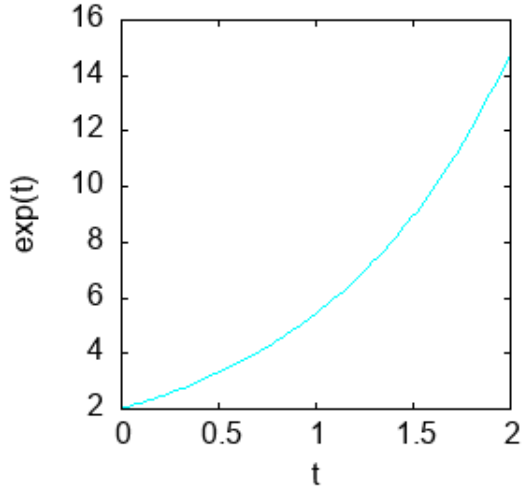
The generic exponential function is $Ae^{\alpha t}$. *The* exponential function e^t has $A = \alpha = 1$. Here are four exponentials with $A = 1$ or $A = 2$, and $\alpha = 1$ or $\alpha = 2$.

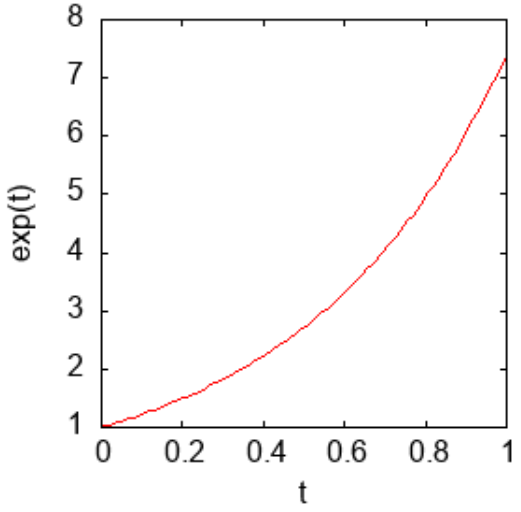


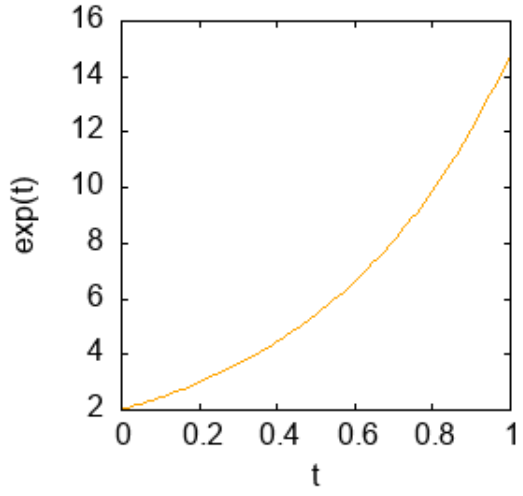
Now, on split-screen: There's only one

Can you see the differences?







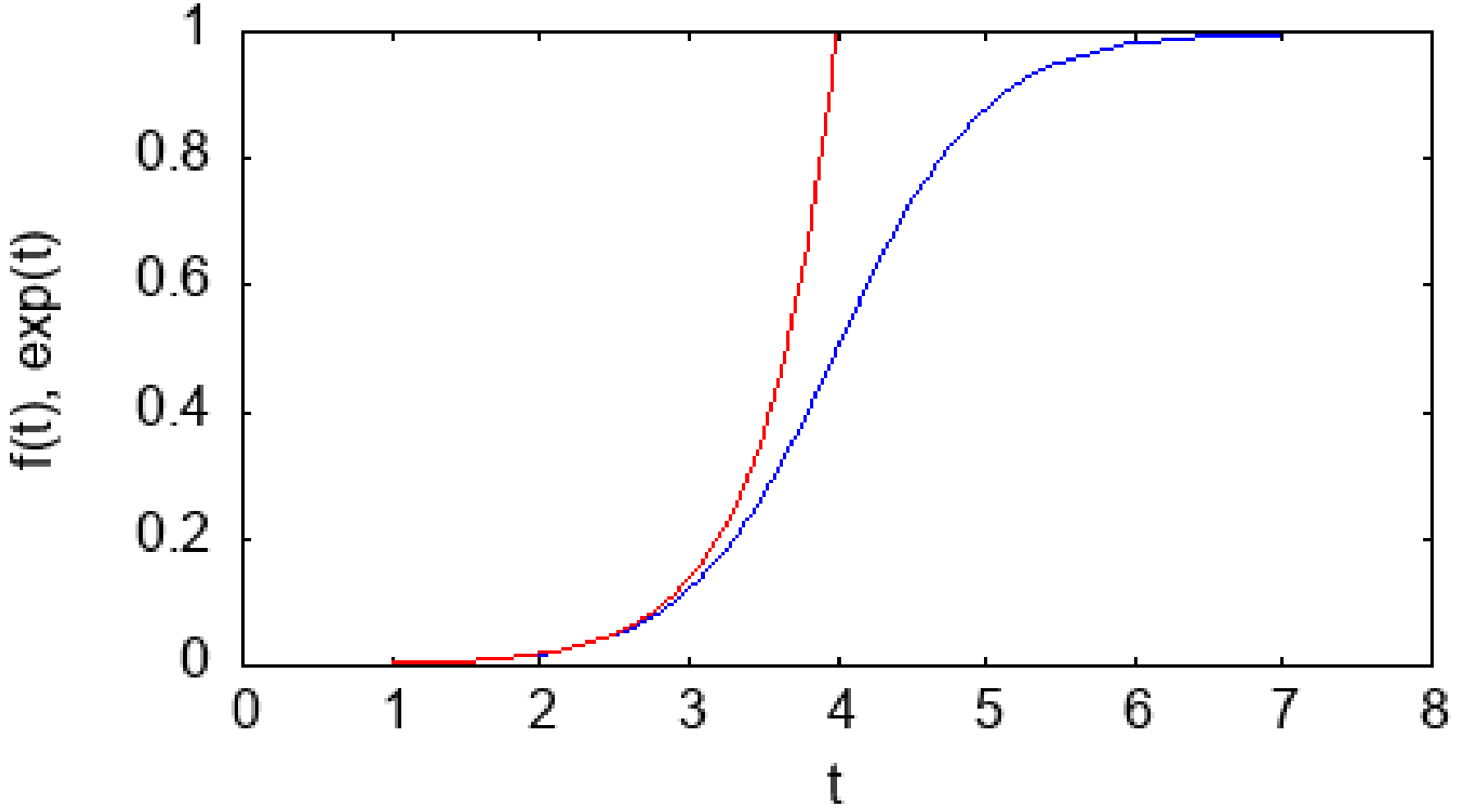


Logistic growth

- How is it that exponential growth crashes through the upper limit of population? One explanation is that it "doesn't know" that only individuals who *aren't infected* can *change to* infected.
- One way to generate a better mathematical model is to consider α as the *hazard rate*: the chance that an infected person will infect someone they run into. Since in the exponential model there are $N(t)$ infected individuals meeting other people, the rate at which infections increase is $\frac{dN}{dt} = \alpha N$.
- But already infected individuals don't "get" infected, so the chance that the individual some infected individual meets is uninfected is $\frac{\bar{N}-N}{\bar{N}}$, where $\bar{N}$ is the total population (the bar on the top symbolizes "maximum infections").
- Now the hazard rate for infections is $\alpha\frac{\bar{N}-N}{\bar{N}} = \alpha(1-\beta N)$, where $\beta = \frac{1}{\bar{N}}$, and the differential equation is $\frac{dN}{dt} = \alpha(1-\beta N)N$.
- This modification would be very tedious in discrete time.

The solution to logistic growth

Compare logistic growth $f(t) = \frac{e^t}{e^t + e^{-t}}$ (blue) with exponential growth (red).
(The exponential growth curve is rescaled to match the logistic curve.)



Policy variables and parameters

- **A policy *variable* is something that the authorities can change to get a better outcome.**
  - In a dynamic model like this one, we sometimes distinguish policy *parameters*, which are policy variables that do *not* change over time, from time-varying policy *variables*.
- Our hazard rate α is a sort of policy parameter. We talk about "flattening the curve" by reducing the hazard rate.
  - Why "flattening"? Because as a proportion of population, for any α the logistic curve tends to 1 as $t \to \infty$. We can't stop that but we can make the slope smaller.

Matching model variables to real policies

- But what are the real policies?
- We don't control the rate of infection directly. Instead we use *social distancing* (meeting fewer people per day) and *prevention* (masks).
- **For a more *realistic* model we can decompose a descriptive parameter $\alpha = \alpha_d \alpha_m$ into policy-relevant components** α_d, the rate of meeting people, and α_m, the leakage rate of masks.
  - Note that in this case, we can do all the math with α and substitute the decomposition later! **More realism doesn't always make the math harder.**
- We could further decompose α_m into the effectiveness *vs.* inhaling (not very good) and the effectiveness *vs.* exhaling (important), but that's not very useful.

I am still not happy with logistic growth

- **To *adapt* our model to the policy needs, we ask *why* do we care** so much about COVID-19? Because some infected individuals get sick and *die*, a minor model change ($\bar{N}$ changes over time).
- That's the worst case, but infected individuals usually *recover*.
  - When recovered, they may be *susceptible* (they can get infected again), decreasing N, **or**
  - When recovered, they may be *immune* (they cannot get infected again), decreasing N where it means "infectious individuals" (the second factor in the right hand side of the differential equation), but not N where it means "nonsusceptible individuals" (the N in the third factor), **or**
  - they may be partially or temporarily immunne.

  Any of these would change the model a lot, making the mathematics harder.

Interpreting logistic growth

- **Can we interpret the** logistic growth **model realistically** for a disease?
- Yes! **But it's not a very intuitive way to think about it.** It doesn't match the way we think about public health.
- In the discussion of hazard rate, we need to think about *why* people are removed from the susceptible population. *E.g.*, with a new virus, it may make sense to class people who never had it as "susceptible" and those who have ever had it as "not susceptible". This is not a good generic model of disease (*e.g.*, you can catch the flu or a cold many times, you can be immune to measles), but it logically generates the logistic model.
  - Note: **The standard for *good model* is domain-specific**: "useful for public health policy." "Logically acceptable" isn't good enough!
- The logistic model may be the best well-founded single equation model, although it's not so helpful to predict hospital demand. **Sometimes a simple model gives a simple answer, and we're satisfied with that.** *E.g.*, the logistic model is *sufficient reason* to fear a devastating spike in cases, even though cases and rate of infection are bounded.

The SIR model

- The SIR model divides the population into three subpopulations (SIR = Susceptible, Infected, Recovered/Immune). Only the Infected population requires treatment, and only the Infected population can infect others.
- Then the equations are

$$\begin{aligned} \frac{dN}{dt}(t) &= \alpha I(t)(1 - \frac{N(t)}{\bar{N}}) \\ R(t) &= N(t-14) \\ I(t) &= N(t) - R(t) \\ S(t) &= \bar{N} - N(t) \end{aligned}$$

- The equation $R(t) = N(t-14)$ makes this model a *delay differential equation* model. The detailed theory of these models is difficult, but **in differential equations, simulations are usually good indicators of system behavior** because the model has a unique solution.

Compartmental models

- SIR is a *compartmental model*. **Compartmental models are frequently a good basis for statistical analysis.**
- Sometimes our subpopulations are fixed (*e.g.*, male *vs.* female), sometimes individuals move among them as in SIR.
- When the compartments are based on age, and individuals move among them in lockstep, we call the compartments *cohorts*.

Variations on the SIR model

- Because with COVID we fear spikes in infections will overwhelm the medical system, and **because we have poor** testing **data** about distribution in the population, **we want more flexible models that can accurately predict system behavior with different component behavior**. Here "components" are the disease and its transmission.
- In SIR, we *abstract from* (ignore) the facts that degree of sickness ranges from "asymptomatic" to "fatal", that we don't know recovery confers immunity, and that the periods of illness and of infectiousness may differ.
  - *Asymptomatic vs. symptomatic* infected individuals are out of scope here, because we don't model burdens on the economy.
  - If recovered individuals are immediately susceptible to reinfection with the SARS-Cov-2 virus, we could use the *susceptible-infectious-susceptible* (SIS) model, which is even simpler than SIR.
  - There is an *incubation period* between the time they are infected and when they show symptoms. This leads to the *susceptible-exposed-infectious-recovered* (SEIR) model.

Beyond SIR, *cont.*

In the case of COVID-19, there are other complications that public health policy requires we add to the model.

- If we had thorough testing, the test itself would be a symptom for epidemiological purposes, but the burdens would still differ from sick individuals.
- We believe that an individual becomes *infectious* before displaying symptoms of COVID-19. In general, infectious capacity may vary over time. (Models with this property were not discussed on Wikipedia.)
- The English Wikipedia article on *Compartmental models in epidemiology* is quite good:

`https://en.wikipedia.org/wiki/Compartmental_models_in_epidemiology`

Still not good enough

- An important phenomenon in *pandemics* is that the *epidemic* is not uniform, it *clusters*.
- *Why* do people get infected (and then sick)? Partly it's an individual characteristic. We handle that statistically by modeling it with *randomness*. We don't know enough about the *pathology* (the way the virus infects us), and it's good enough for epidemiology.
- In epidemiology, people are infected when they meet others. But meetings are not uniform, expressible by a single hazard rate. In fact, the probability of meeting depends on *both* individuals in an encounter. This requires a *social network model*, which generally can't be solved by algebra or calculus, a very big change in the model.

- The SIR model is good enough to give a picture that looks a lot like the graphs used to demonstrate the "flatten the curve" effect of social distancing.
- Can't we stop there? Unfortunately not. We know that for individual and economic reasons, amount of social distancing varies greatly among individuals. We must look at a social network model to *estimate* the flattening effect we can get from a policy (more counterfactuals!)
- We also have experience with multiple "waves" of a disease, both very regular ones (the annual "flu season"), and single episodes (the 1918 influenza pandemic, where the second wave caused the most deaths).
- In fact, the 1918 pandemic also teaches us that different policies give different curves, and quite different second and third waves. We need models to predict those too.
- **When do we stop modeling?** This is a pragmatic question, and the answer is "when we run out of time, budget, or motivation." For example, we learned a lot about the *first* SARS virus, but we never developed a vaccine or an antiviral treatment for SARS-like corona viruses due to lack of motivation: SARS was defeated quite quickly (as these things go).