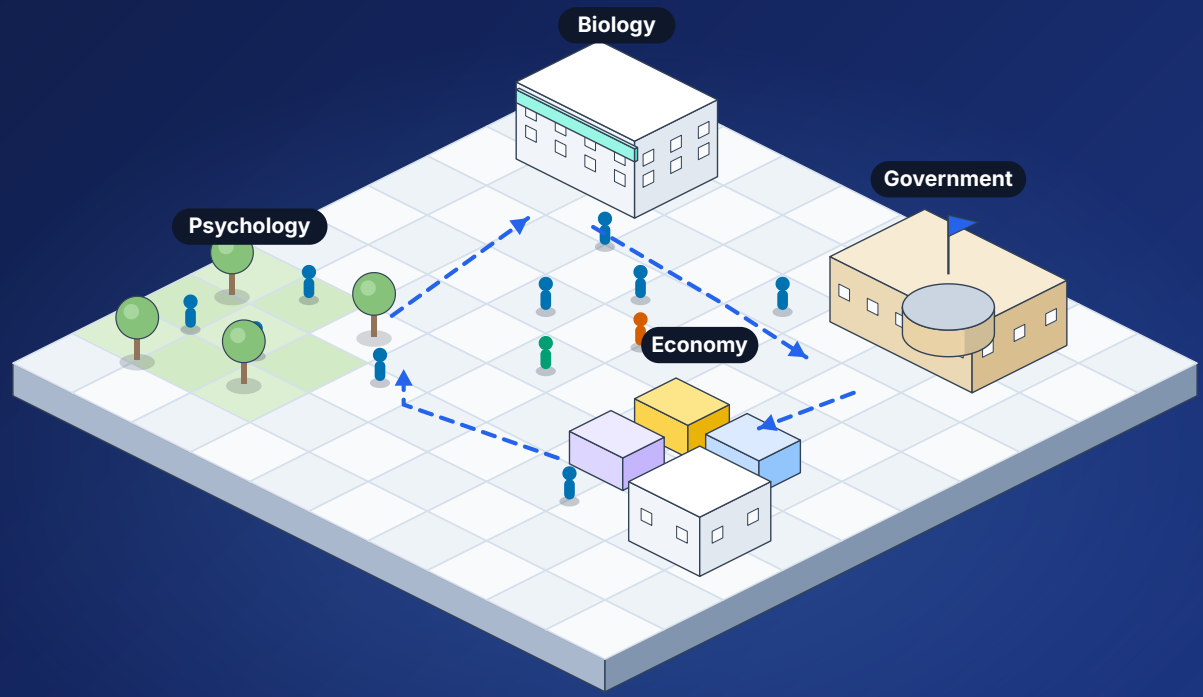
Applied Computational Mathematics Lab

EPIDEMIC MODELING · MODULE 2

Beyond the virus

Biology, psychology, economics and governance



3 EPISODES

1. How infections spread, and the biology behind it
2. Fear, trust and money
3. Governments and feedback loops

Based on Chapter 2 of T. Lazebnik, Epidemic Modeling: Principles, Algorithms, and Applications (Academic Press, 2026)

acml.teddylazebnik.com

BEFORE WE START

Warm-up: true or false?

CHECK YOURSELF

(a) The reproduction number R is a fixed property of a virus.

(b) If eight in ten sick people isolate, an outbreak always shrinks.

(c) Most of the drop in shopping early in COVID-19 was caused by official orders.

HOW TO USE THIS

Guess now. (a) and (b) are settled in Part 1, (c) in Part 2; all answers are at the end of the deck.

WHY THIS MODULE

A model is only as good as its picture of the world around the virus. This module builds that picture: the biology, the people, the money and the institutions that every later model simplifies.

BEFORE WE START

This module's map

PART 1

How infections spread

Transmission vs interruption, who meets whom, uneven spread, space, delays, and the biology underneath.

PART 2

Fear, trust and money

Perceived risk, norms, trust, misinformation, fatigue, and the economics of who can protect themselves.

PART 3

Governments and feedback loops

What must exist before a crisis, timing, policy packages, late data, and what models can honestly do.

GOALS

By the end you should be able to explain why R depends on the setting, show how timing in the body and in policy changes outcomes, and describe the feedback loop between disease, behavior and policy.

ONE PHRASE

Coupled system: parts that change each other. A pandemic couples biology, contacts, psychology, economics and governance.

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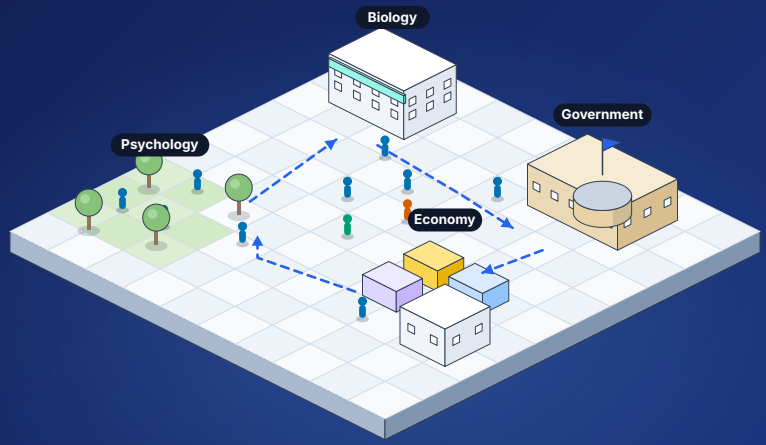
PART 1

How infections spread

From one infection to an outbreak, and the biology underneath.

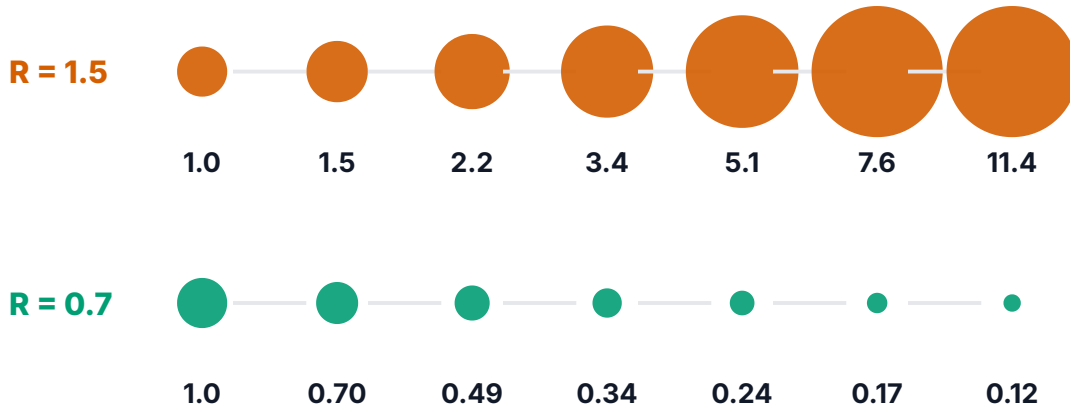


A tug of war: transmission vs interruption

- **Transmission**: an infectious person meets a susceptible one, and the pathogen gets across.
- **Interruption**: people become immune, cases are isolated sooner, or contacts change.
- The balance is summed up by the **reproduction number R**: the average number of new infections caused by one typical case, in a given setting.
- Above 1, each generation is bigger than the last; below 1, chains shrink.

R_0 AND R(T)

R_0 is R at the very start, when almost everyone is susceptible. R(t) is its value later, after immunity and behavior change.



expected cases in each generation, starting from one case

Our arithmetic: $1.5^6 \approx 11.4$ and $0.7^6 \approx 0.12$.

R depends on the setting

WHAT CHANGES	WHY R MOVES	EXAMPLE
How people mix	more meetings, more chances to pass it on	crowded housing, packed transport
How fast cases are found	a case isolated on day 2 infects fewer people than one found on day 6	fast testing, contact tracing
What is in place	each meeting becomes less risky	ventilation, masks, vaccination
Who is still susceptible	immune people break chains	after a wave, or after vaccination

IN PLAIN WORDS

R is not a property of the virus alone. The same virus has a different R in a different city, season or year.

TRY IT

A virus has R = 2 in a city. Contacts fall by 30% and nothing else changes. Roughly what is R now? Does the outbreak still grow?

Who meets whom

- People do not mix evenly: the same few people at home every day, groups at school and work, strangers now and then.
- Contact surveys (for example the large European POLYMOD study, Mossong et al., 2008) show strong age patterns: children mostly meet children, working adults meet each other, homes mix ages.
- Where no survey exists, researchers project contact patterns from census and household data.
- So closing schools and closing offices are different levers: they cut different cells of the contact matrix.

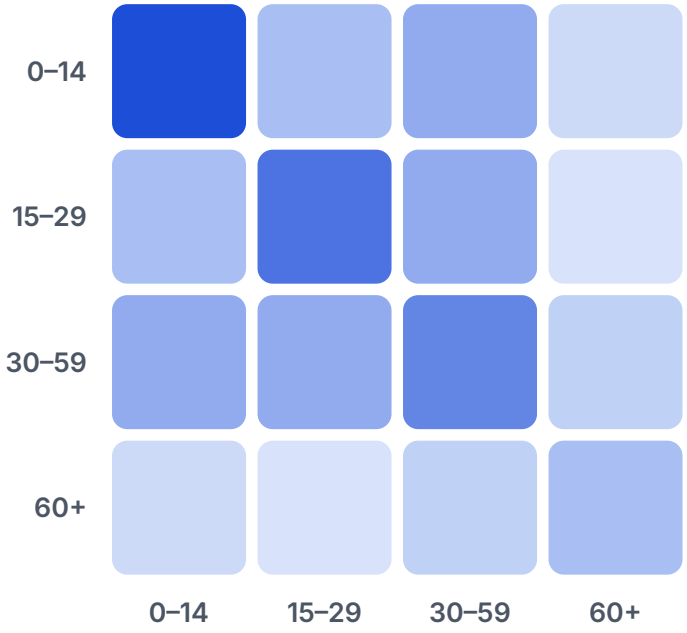
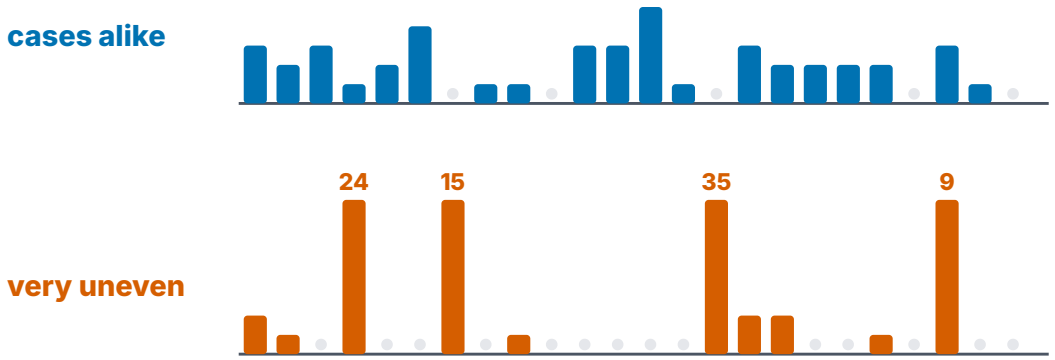


illustration of the usual pattern, not survey data

TRY IT

In the illustration, which row would a school closure shrink most? Which cells would working from home shrink?

Uneven spread



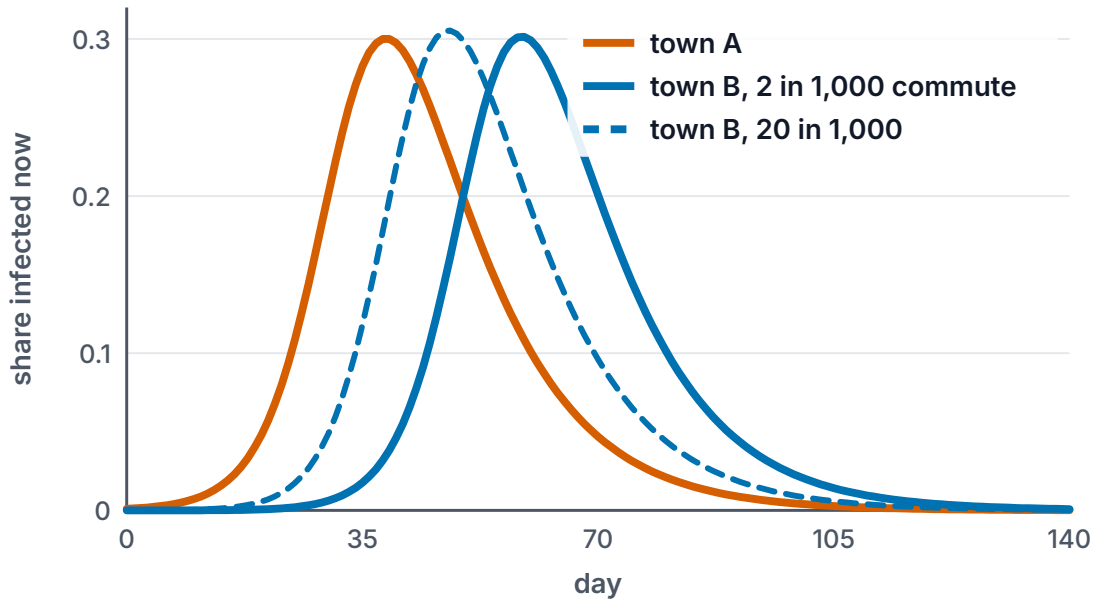
new infections caused by 24 cases from each simulated outbreak (bars cut at 8)

Our simulation, 200,000 cases per model: Poisson with mean 2 vs negative binomial with mean 2 and dispersion 0.1.

- Same average (2 new infections per case), very different outbreaks.
- In the uneven model, **74%** of cases infect nobody, and **9%** of cases cause 80% of all infections (alike: 53%).
- One imported case dies out on its own **89%** of the time, against 20% when cases are alike.
- So: rarer outbreaks, but more explosive when they take off. Preventing high-risk settings can do more than measures that assume everyone is average.

The idea of individual variation: Lloyd-Smith, Schreiber, Kopp and Getz, Nature 2005.

Epidemics move through space



Our run: two towns of equal size; a share of each town's contacts happen in the other town.

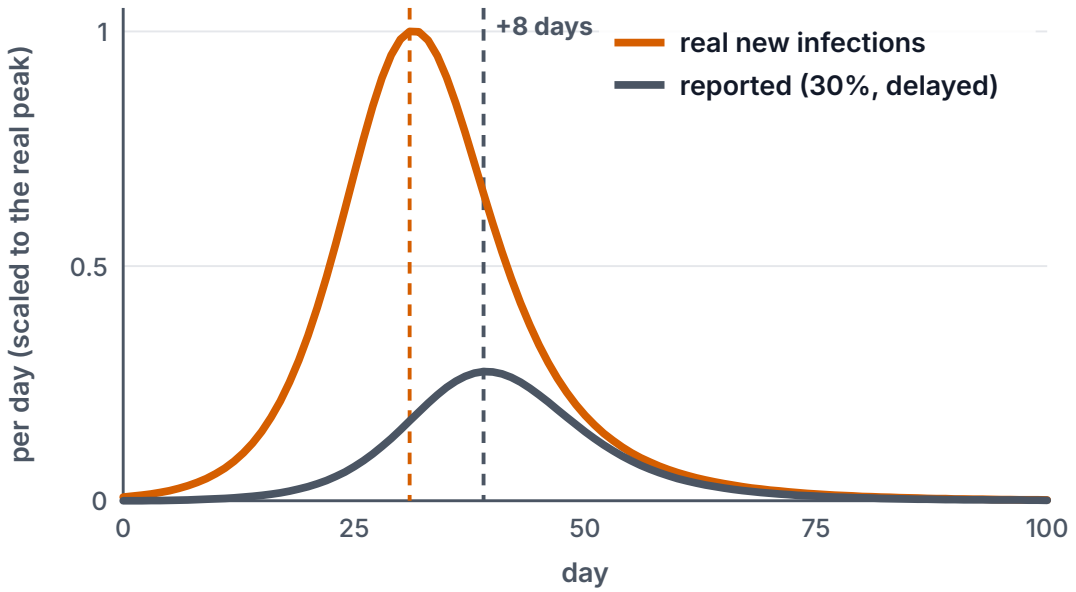
- Commuting ties nearby places together; long trips seed outbreaks far away.
- Two people in 1,000 commuting: town B peaks **20 days** after town A. Twenty in 1,000: **9 days** after.
- More travel synchronizes nearby places; less travel spreads the peaks out in time.
- So one national number can hide a surge in one region and a decline in another.

LOOKING AHEAD

Module 4 builds these "cities linked by travel" models properly, with networks and continuous space.

What happens vs what we see

- Infection comes first; then symptoms, a test, a result, a report. Each step adds delay and drops some cases.
- Our run: incubation of about 5 days and about 4 more days to report (on average 8.0 days in all), and only 30% of infections are ever reported.
- The reported curve peaks **8 days** after the real one, and is much smaller.
- Epidemiologists estimate a time-varying R(t) from such data, correcting for the delays and for the typical gap between linked infections.



IN PLAIN WORDS

Case counts measure two things at once: transmission, and the surveillance system itself.

Kinds of pathogens

VIRUSES

must enter our cells

They copy themselves only inside host cells: influenza, coronaviruses, measles.

BACTERIA

can grow on their own

In tissue or in the environment: cholera, plague, tuberculosis.

FUNGI

yeasts and moulds

Mostly a threat to people with weak immune systems.

PROTOZOA

single-celled parasites

Malaria, often carried by an insect.

HELMINTHS

parasitic worms

Long infections, often linked to water and soil.

PRIONS

misfolded proteins

No genes at all, yet transmissible.

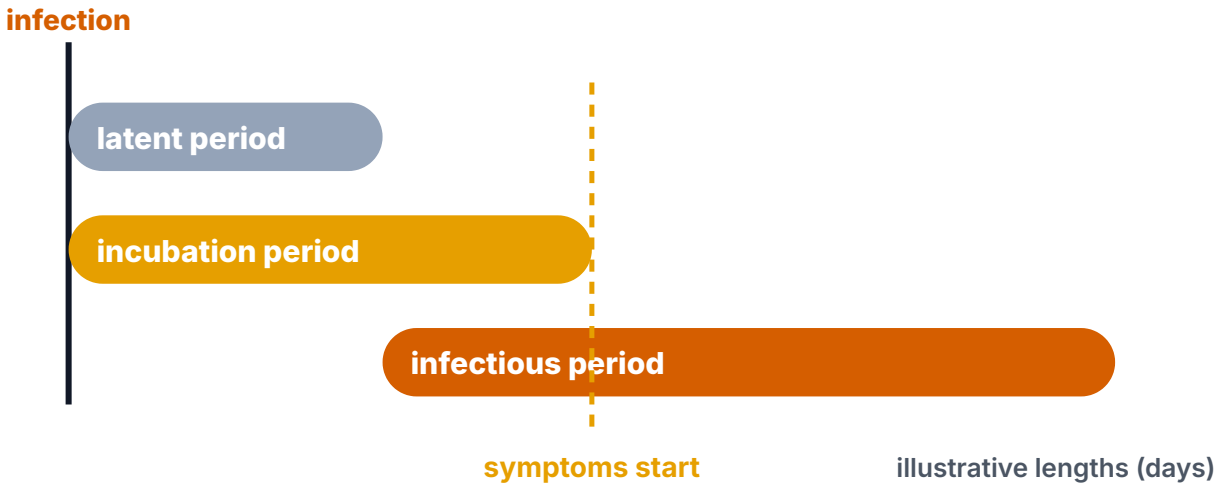
INFECTION IS NOT DISEASE

Infection: the pathogen is present and multiplying. **Disease**: the clinical consequences, such as symptoms. A person can be infected without being ill, and still pass it on.

IN PLAIN WORDS

Most fast pandemics come from viruses and some bacteria: short, acute infections that spread when contacts are frequent.

Three clocks



CLOCK	ENDS WHEN…
latent period	you become infectious
incubation period	symptoms start (if they ever do)
infectious period	you can no longer pass it on

- All three start at infection.
- Each varies from person to person: think of them as **distributions**, not single numbers.
- What matters most for control is their **order**. If infectiousness starts after symptoms, isolating sick people works well.
- If much of the spread happens before symptoms, or from people who never get symptoms, symptom-based control is far weaker.

Timing in the body becomes timing in policy

$$R = R_0 \left(1 - (1 - \theta)\, c\, e\right)$$

- θ: share of spread that happens before symptoms.
- c: share of sick people who isolate at symptom onset. e: share of their later spread that isolation stops.
- Toy numbers: $R_0 = 2.5$, $c = 0.8$, $e = 0.9$.
- $\theta = 0.05$: R $\approx$ **0.79**, the outbreak shrinks.
  $\theta = 0.4$: R $\approx$ **1.42**, it still grows.

IN PLAIN WORDS

Same effort, opposite outcomes. Whether symptom-based isolation can control a disease depends on biology.

TRY IT

With the same R_0, c and e, what share of presymptomatic spread θ puts R exactly at 1?

The idea: Fraser, Riley, Anderson and Ferguson, PNAS 2004.
Numbers: our arithmetic.

Routes, and their defenses

ROUTE	SHAPED BY	MATCHING DEFENSES
Through the air	indoor air, crowding, time together	ventilation, filtration, fewer people indoors, masks
Direct contact	touch, close care	hand hygiene, protective gear
Contaminated objects	how long the pathogen survives on surfaces	cleaning, hand hygiene
Water and food	sanitation, water treatment	clean water, sewage systems, food safety
Blood or sex	needles, transfusions, sexual networks	screening, safe injection, condoms
Insects and other vectors	mosquito and tick habitats	bed nets, repellent, draining breeding sites

IN PLAIN WORDS

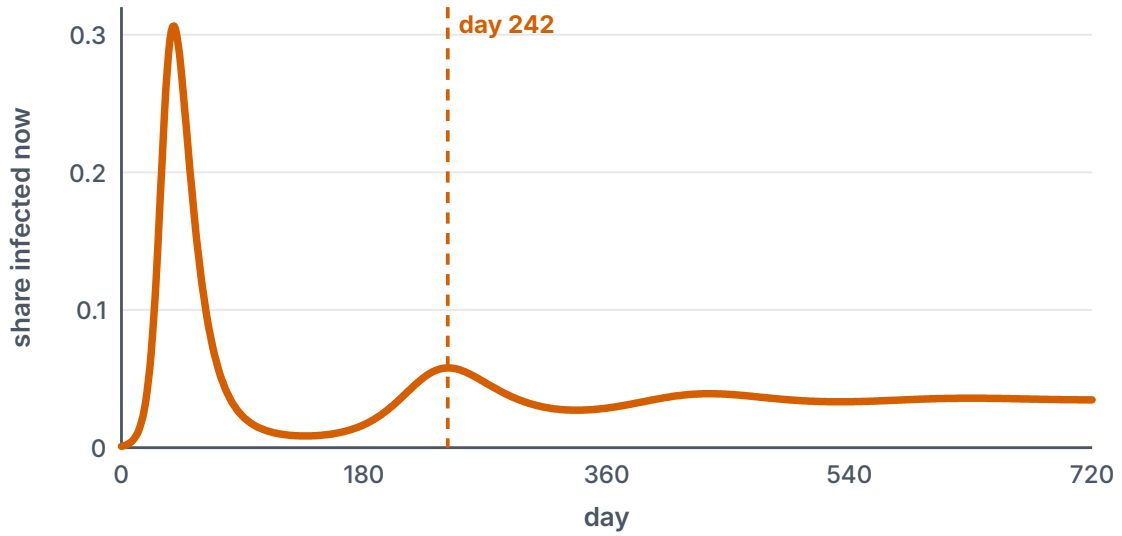
To spread, a pathogen must leave one body, survive the trip, and reach another in a big enough dose. Each route is a different weak point.

ENVIRONMENT

Temperature, humidity and sunlight change how long a pathogen survives outside the body, so the same pathogen can lean on different routes in different settings.

Severity and immunity

- **Virulence**: how much harm a pathogen does. Severe disease fills hospitals, and also changes behavior, which feeds back into spread.
- There is no rule that pathogens evolve to become milder: it depends on how they spread and how hosts behave.
- **Immunity** is not an on-off switch. It can block infection, cut onward spread, soften illness, or a mix, and it can fade.
- Our run: when protection fades after about 180 days on average, a second wave arrives around day **242**, smaller than the first (6% vs 31% infected at once).



Our SIRS run: β = 0.3, γ = 0.1 per day, waning after 180 days on average.

Spillover: from animals to people



- Many pandemics begin with **zoonotic spillover**: a pathogen crosses from animals into people.
- It must clear a chain of barriers; sustained person-to-person spread is the last and hardest step.
- Human activity (land use, farming, wildlife trade, travel) can lower every barrier, even when the pathogen itself has not changed.

TRY IT

Pick one barrier in the chain. Name one human activity that lowers it and one policy that raises it again.

Check yourself: Part 1

CHECK YOURSELF

1. Give two reasons why the same virus can have a different R in two cities.

2. Two outbreaks have the same mean number of new infections per case. Why might one die out far more often?

3. Why does reported data peak later than real infections?

4. In $R = R_0(1 - (1 - \theta)ce)$, what happens to R as θ grows?

TIP

Answers are at the end of the deck. Try them before you look.

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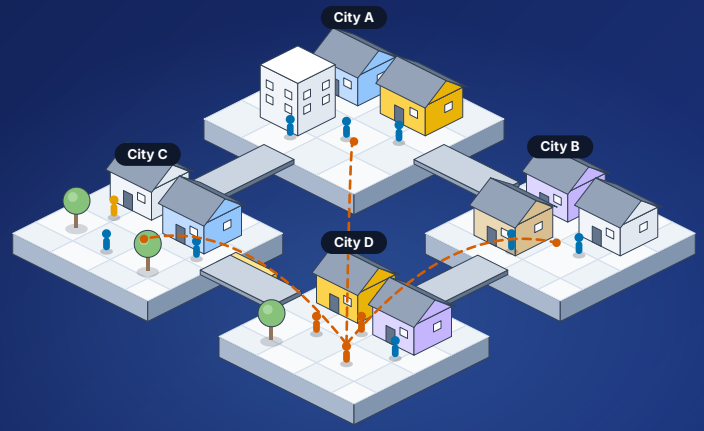
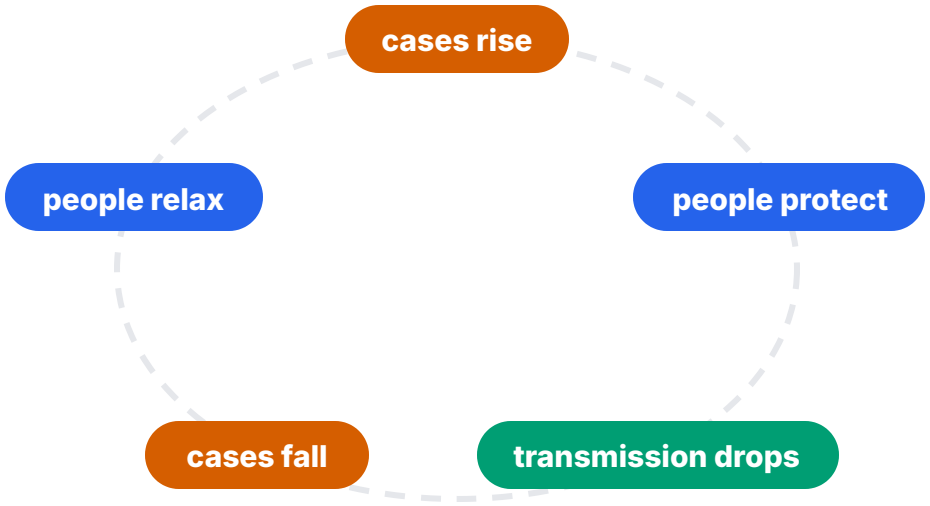
PART 2

Fear, trust and money

Behavior is part of the epidemic.



The behavior loop



- Disease changes behavior, and behavior changes disease.
- As cases rise, many people stay home, avoid crowds, wear masks or get tested; transmission falls.
- As cases fall, people relax; transmission rises again.
- This two-way coupling explains why outbreaks can accelerate, plateau or surge again with the pathogen unchanged.

IN PLAIN WORDS

A model that holds contact rates fixed leaves out half of this loop.

Perceived risk and the step to action

- People react to the risk they **perceive**, not the true risk.
- Mental shortcuts: vivid, recent and heavily covered events feel more likely; slow, quiet risks feel smaller until they come close.
- So two towns with the same epidemic can behave very differently, depending on what they believe is happening.

FROM BELIEF TO INTENTION

A classic model of behavior (the theory of planned behavior) says intention comes from three things: **attitude** toward the action, what you think **others expect**, and whether you feel **able** to do it.

EXAMPLE

Someone believes the disease is dangerous, but thinks masks don't work and cannot afford to stay home. Attitude and perceived control both block action.

TRY IT

Name one policy that works on each of the three ingredients.

PART 2 · PSYCHOLOGY

Norms, trust and misinformation

NORMS

we copy each other

A visible, normal habit spreads through workplaces and schools. When it becomes rare, people drop it.

IDENTITY

a badge of one side

When a measure becomes linked to a group, behavior can follow loyalty more than health beliefs.

TRUST

who says it matters

Trusted messengers get advice followed, and changes in advice accepted. Openness about uncertainty builds trust.

MISINFORMATION

a second contagion

False stories spread farther and faster than true ones on Twitter, mostly through people sharing what feels new.

IN PLAIN WORDS

Information spread and disease spread become entangled: beliefs change behavior, and behavior changes transmission.

Misinformation study: Vosoughi, Roy and Aral, Science 2018.
Vaccination-communication guidance: World Health Organization.

PART 2 · PSYCHOLOGY

Fatigue, and our behavior model

- The World Health Organization describes **pandemic fatigue** as an expected response to long restrictions: motivation wears down.
- Our model adds behavior to SIR. Protection p cuts transmission by up to 90%.
- **Fear**: people react to infections reported 7 days late and remember them for about 5 weeks (perceived risk P).
- **Fatigue** F builds while people protect and fades when they stop.

$$\beta(t) = \beta_0\,(1 - 0.9\,p), \qquad p = \frac{P}{P + 0.0005}\,(1 - F)$$

$$\frac{dF}{dt} = 0.005\,p\,(1 - F) - 0.005\,F$$

SETTINGS

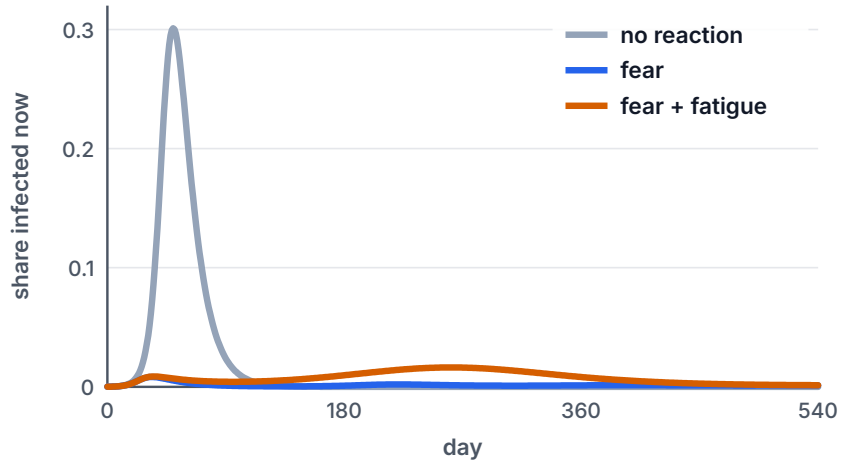
β_0 = 0.3 and γ = 0.1 per day (R_0 = 3), one case in 10,000 at the start, 540 days. Code: production/explains/sims/epi_m02_sims.py. The lab runs the same model in your browser.

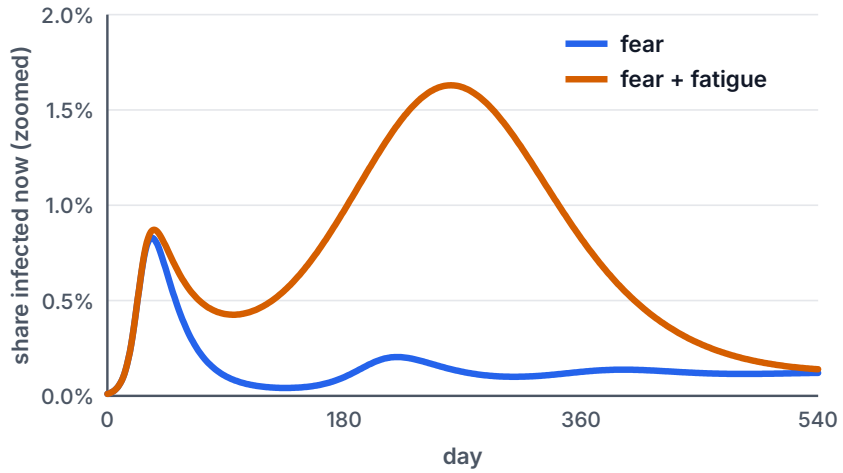
IN PLAIN WORDS

All parameter values are ours, chosen to show the mechanism. They are not estimates for any real disease.

PART 2 · PSYCHOLOGY

Fear flattens, fatigue brings the wave back





OUR RUN	PEAK INFECTED AT ONCE	INFECTED BY DAY 540
no reaction	30%	94%
fear	0.8%	9%
fear + fatigue	0.9%, then 1.6% (day 261)	40%

- Fear alone cuts the peak from 30% to under 1%.
- Fatigue lets the curve climb back into a second, bigger wave about nine months in.

TRY IT

In the lab, make fatigue build twice as fast. Does the second wave come sooner, and is it bigger?

Most infections happen in ordinary economic life

- Working, shopping, commuting, caring: the activities that make income also make contacts.
- Someone who can work from home cuts contacts and keeps their pay; someone whose job is on site faces a trade-off between health and earnings.
- Unequal infection rates across jobs and neighborhoods often reflect **constraints**, not preferences.

EXTERNALITY

An **externality** is a cost (or benefit) of my choice that falls on others. A sick worker who goes in keeps their pay; coworkers and customers carry the risk.

IN PLAIN WORDS

Because these costs are not priced, private choices need not match what is best for everyone, even when everyone acts sensibly.

NAME OF THE FIELD

Economic epidemiology studies this coupling: risk changes activity, and activity changes risk.

Sick pay changes the math

$$R = R_0\,(1 - s\,e\,q)$$

- s: share of spread after symptoms (0.6). e: share of it that staying home stops (0.9). q: share of sick workers who can afford to stay home.
- $R_0 = 2.5$, $q = 0.4$: R $\approx$ **1.96**. $q = 0.8$: R $\approx$ **1.42**.
- Sick pay alone does not end an outbreak, but it moves R a long way.

EVIDENCE

A study of the US emergency paid sick leave introduced in 2020 linked it to slower growth in confirmed cases (Pichler, Wen and Ziebarth, Health Affairs 2020).

TRY IT

With these numbers, what share q would bring R down to 1.5?

Pulling back, and two kinds of shock

- People pull back on their own: early in COVID-19, visits to businesses fell sharply, and official orders explained only part of the drop (Goolsbee and Syverson, J. Public Economics 2021).
- So a recession is not only caused by mandates; fear also cuts spending and work.
- **Supply**: workers sick, caring for others, or kept home. **Demand**: restaurants, travel and entertainment collapse while delivery and remote tools grow.
- Production networks pass shocks along: one closed supplier can stall many factories downstream.

DOWN	UP
restaurants, travel	delivery, logistics
events, tourism	video calls, remote work
in-person services	some health goods

IN PLAIN WORDS

A pandemic is a demand shock and a supply shock at once, in different mixes over time and place.

PART 2 · ECONOMICS

Who pays, and targeting

- Costs fall unevenly: jobs that cannot be done remotely, few savings, crowded housing, small firms with little credit, children who lose school time.
- Those groups carry more risk and more loss, and if they cannot protect themselves, transmission stays high for everyone.
- Unevenness can also be used: reducing contacts where they are densest buys more per unit of cost.

Toy town with proportionate mixing: R is proportional to the mean of contacts squared divided by the mean of contacts.

OUR TOY TOWN (R_0 2.5)	R AFTER
remove a quarter of all contacts, everywhere	1.88
remove the same quarter, only in busy settings (1 in 5 people, 4× the contacts)	1.33

CAUTION

Targeting is not always easy or fair. But ignoring differences gives blunt policies that cost more and work less.

WHAT COUNTS

Deaths are one outcome. Long illness, mental health, lost schooling and delayed care also count, and so does who bears each cost.

Check yourself: Part 2

CHECK YOURSELF

5. Name the three ingredients of intention in the theory of planned behavior, and one barrier for each.

6. In our behavior model, why does fear alone lower the peak so much, and what does fatigue change?

7. Explain the externality of going to work sick in one sentence.

8. With $R_0 = 2.5$, $s = 0.6$, $e = 0.9$ and $q = 0.6$, what is R?

TIP

Answers are at the end of the deck.

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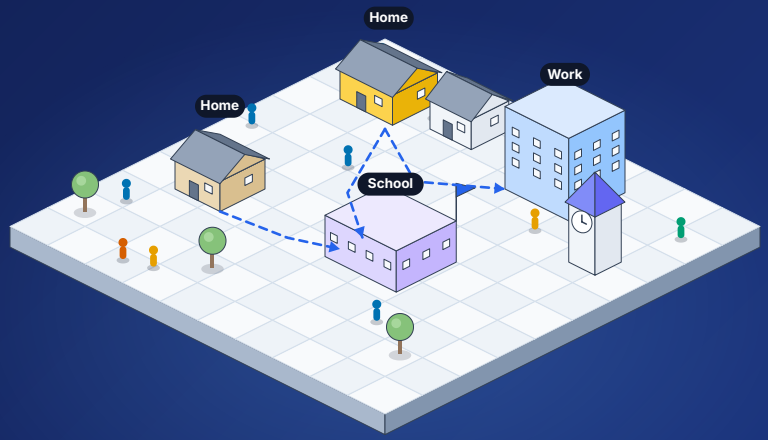
PART 3

Governments and feedback loops

Coordination, timing, and decisions on late data.



A collective action problem

- Even when everyone knows what helps, it is hard to keep up without coordination, good information and practical support.
- Governments provide what individuals and markets cannot: **public goods** (surveillance, lab networks, emergency logistics, clear rules) and management of **shared resources** such as hospitals and medicines.
- In infectious disease, one person's choices affecting many others is the norm, not the exception.

BEFORE

foundations

legal authority, trained staff, labs that test and sequence, data systems that share, agencies that know each other

DURING

decisions on epidemic time

reduce uncertainty fast, then interrupt transmission, then sustain it

INTERNATIONAL RULES

Under the International Health Regulations (2005), countries have agreed to build core public health capacities and to report unusual events that could cross borders.

Legitimacy, and the levers

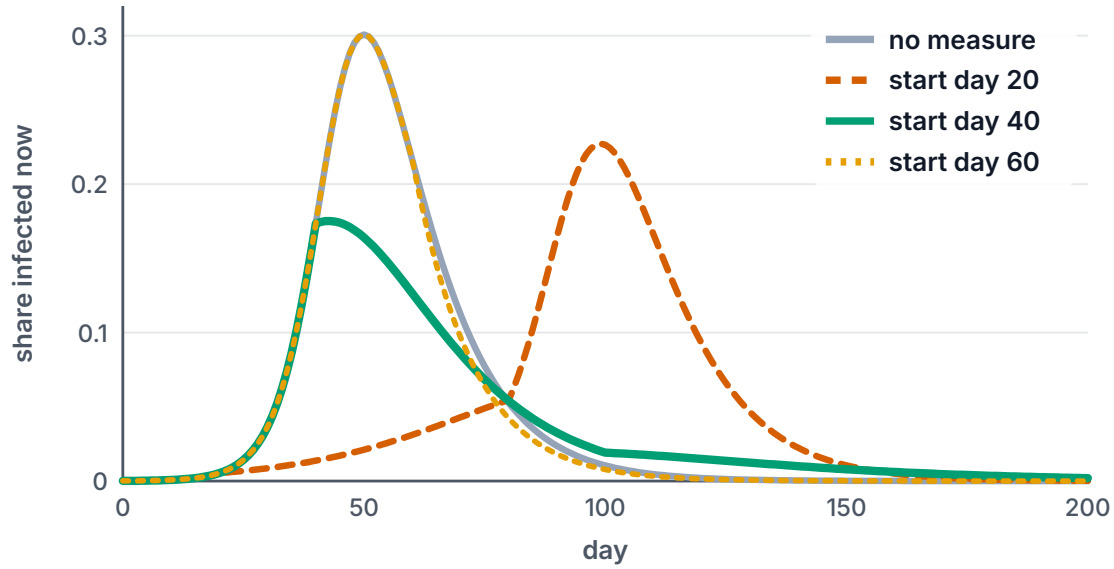
- **Legitimacy**: when people see the system as competent and fair, the same epidemic effect needs much less force. Trust is a variable that changes transmission.
- **Early**: reduce uncertainty fast: testing, case definitions, reporting pipelines, data people can read, and quick ways to update guidance.
- **Sustained spread**: change the environment the virus needs: how often people meet, where, how risky each meeting is, and how fast cases are found.

LEVER	WHAT IT CHANGES
isolation support, contact tracing, testing	how fast cases are found
ventilation, masks	risk per meeting
limits on crowding, temporary restrictions	how often and where people meet
hospital surge capacity, protecting staff	harm per infection

IN PLAIN WORDS

The effect depends on timing and adherence, not on the announcement.

Timing decides



Our runs: transmission halved for 60 days; β = 0.3, γ = 0.1 per day; one case in 10,000 at day 0.

START DAY	PEAK	INFECTED OVERALL
none	30%	94%
20	23%	92%
40	18%	77%
60	30%	90%

- Too early: the wave waits and returns when the measure lifts.
- Too late: the peak has passed.
- Day 40: the same 60 days cut both the peak and the total.

TRY IT

Why does the day-20 run end with almost the same total as no measure at all?

Policies interact

$$R = R_0\,(1 - s\,d\,c)$$

- $s = 0.6$: share of spread that can be stopped once a case is found. d: share of cases detected. c: share who can isolate, with support such as paid leave and food delivery.
- Testing helps only if people can isolate; isolation support helps only if cases are found.

R (R_0 2.5)	LITTLE SUPPORT, C = 0.4	STRONG SUPPORT, C = 0.95
little testing, d = 0.3	2.32	2.07
lots of testing, d = 0.7	2.08	1.5

IN PLAIN WORDS

Good governance is less about one perfect measure and more about a package that fits together and can be delivered.

TRY IT

Which single change (d or c) helps more here? Why is the combined effect bigger than the sum of the two single effects?

Delivery, equity and supplies

- **Delivery** fails in ordinary ways: tests people cannot reach, isolation advice without pay, tracers overwhelmed, beds without staff. Pathogens exploit every gap.
- **Equity**: if the most exposed groups cannot protect themselves, transmission stays high for everyone. Strong measures must be justified, proportionate and safeguarded.
- **Supplies**: buying tests, protective gear and vaccines, cold chains and last-mile delivery, and fast but careful approvals.
- **Borders**: governments answer first to their own people, which makes global sharing hardest when it is needed most.

THE COST OF A WEEK

If cases double every 3.5 days, one week of delay means $2^{7/3.5}$ = **4×** as many cases.

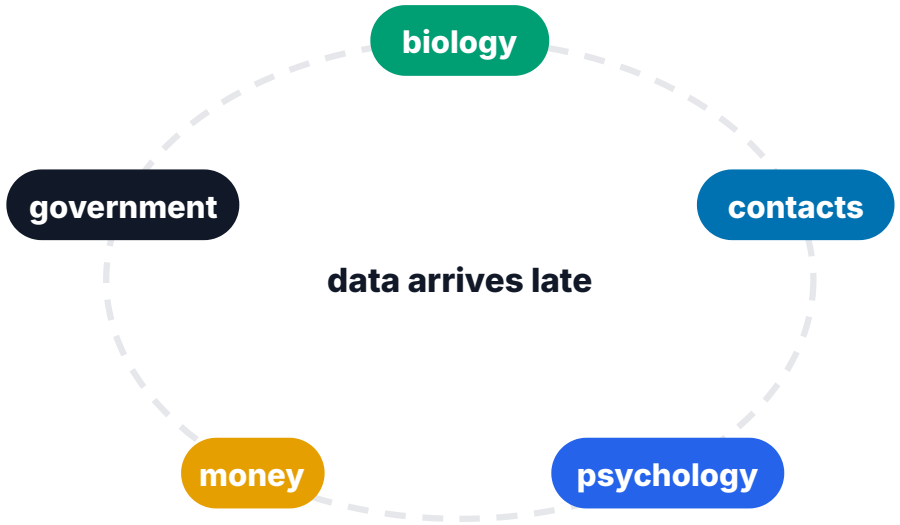
GLOBAL ACCESS

Pooled procurement mechanisms try to share scarce vaccines and treatments between countries; they depend on national cooperation.

TRY IT

If cases double every 5 days instead, how much does a two-week delay multiply them?

One coupled system

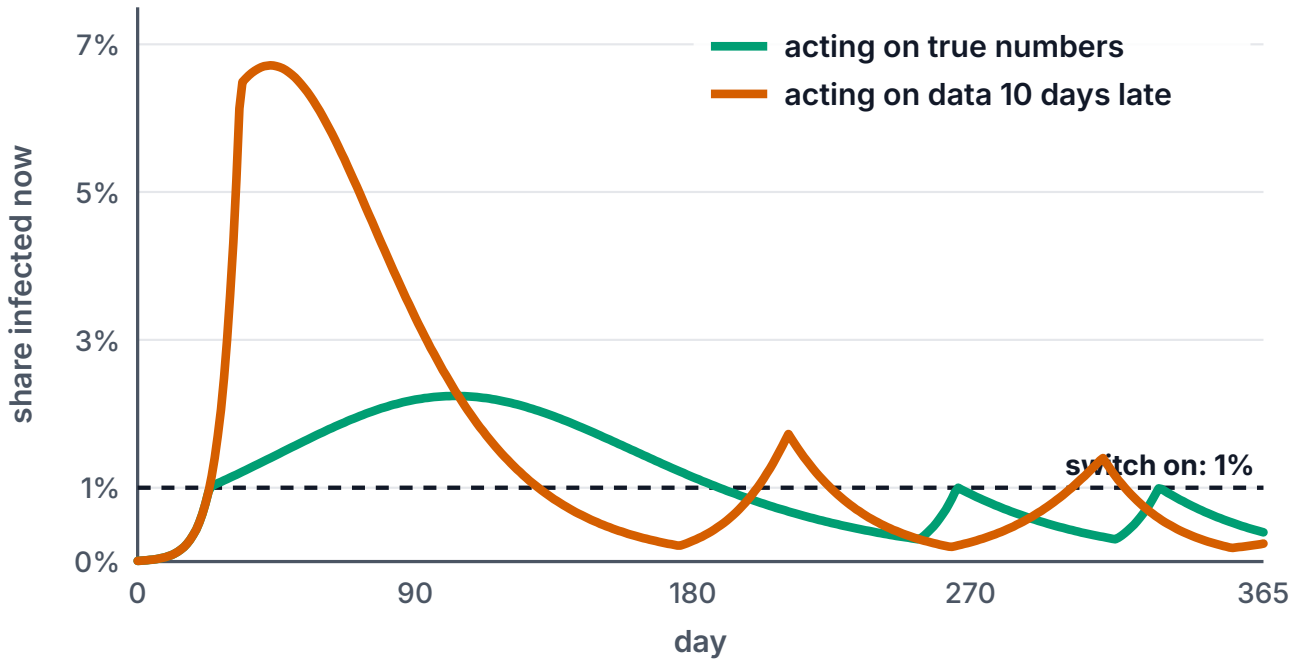


- Biology sets the clocks. Contacts turn biology into spread. Psychology changes contacts. Money decides who can change. Government coordinates and sends signals.
- Government also reacts to data that arrives late, after incubation, testing, reporting and clinical progression.
- So decisions are made on information that reflects transmission from days or weeks earlier.

IN PLAIN WORDS

Every arrow is a modeling choice: include it, simplify it, or leave it out and say so.

Acting on late data



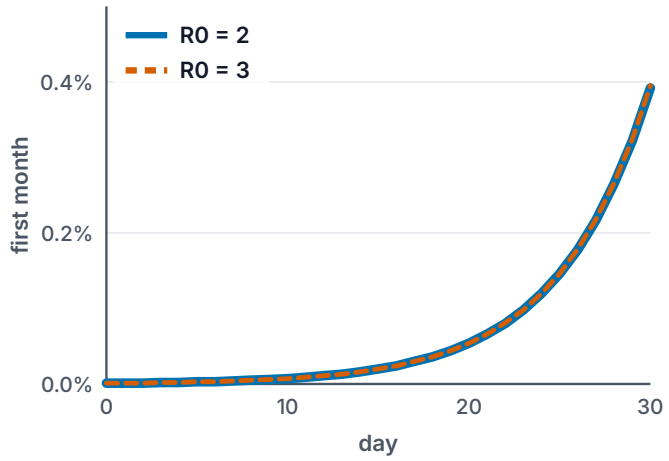
Our runs: restrictions switch on above 1% infected and off below 0.3%, and cut transmission by 60%.

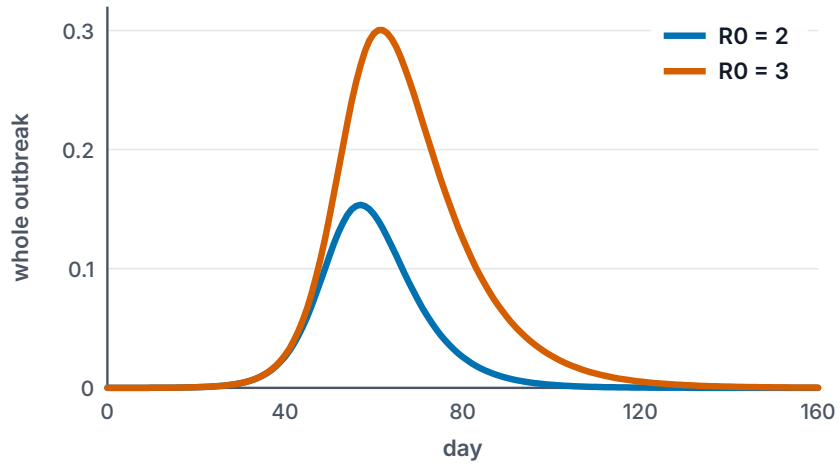
- Acting on true numbers, the peak stays near **2.2%**.
- Acting on data 10 days late, it reaches **6.7%**, about three times higher.
- The epidemic keeps growing during the lag, so each switch comes too late, and switching off comes too late as well.

TRY IT

Name two ways to shorten the lag between infection and decision.

Why modeling this is hard





- Early on, exponential growth turns small timing differences into big gaps; chance, like one superspreading event, can decide what happens next.
- Key quantities are never observed directly: true infections, real contact rates, who really isolates.
- Different assumptions can fit the same data. Our runs: R_0 2 (β 0.4, γ 0.2) and R_0 3 (β 0.3, γ 0.1) both grow 20% a day at first and are almost identical for a month; then the peaks are 15% and 30%.
- Uncertainty comes from parameters, structure, measurement, delays, and adaptive behavior and policy.

IN PLAIN WORDS

Matching past data does not prove the mechanism, and does not guarantee a good forecast under new policies.

What models can honestly do

1

Map plausible futures

Scenarios under clearly stated assumptions, not one prediction.

2

Test what must be true

What would have to hold for a policy to succeed?

3

Find the unknowns that matter

Which uncertainties change the decision, and are worth measuring?

IN PLAIN WORDS

Models stay indispensable: they make invisible feedback loops visible. Their value depends on being explicit about what was simplified away.

NEXT

Module 3 starts writing this system in mathematics: compartments, rates, R_0, and how behavior and seasons enter the equations.

Check yourself: Part 3

CHECK YOURSELF

9. Give two things that must exist before a crisis, and explain why they cannot be built quickly during one.

10. In the timing runs, why does starting on day 20 help so little?

11. With $R_0 = 2.5$, $s = 0.6$, $d = 0.5$ and $c = 0.8$, what is R?

12. Why can two models fit the same early data and still disagree about the peak?

TIP

Answers are on the next slides.

Answers: warm-up and Part 1

- **(a)** False: R depends on mixing, detection, measures and immunity. **(b)** False: with 40% of spread before symptoms, our toy numbers give R ≈ 1.4. **(c)** False: studies found official orders explained only part of the drop.
- **1.** Any two of: different contact patterns (crowding, age mix), speed of finding cases, measures in place, share already immune, season.
- **2.** With very uneven spread most cases infect nobody, so a single introduction often fizzles (89% vs 20% in our runs).
- **3.** Infection is followed by incubation, testing and reporting delays; and only part of the infections is ever reported.
- **4.** R grows: less of the spread happens after symptoms, where isolation can stop it.

TRY-IT ANSWERS

Try-it answers: $R \approx 2 \times 0.7 = 1.4$, still growing. θ at $R = 1$: $1 - (1 - \theta) \times 0.72 = 0.4$, so $\theta \approx 0.17$.

Answers: Parts 2 and 3

- **5.** Attitude (thinks masks are useless), what others expect (nobody around me does it), perceived control (cannot afford to stay home).
- **6.** People react strongly as soon as infections are seen, so transmission drops before many are infected. Fatigue slowly erodes protection, so the curve climbs back into a second wave.
- **7.** The sick worker keeps the income; the risk falls on coworkers and customers, who did not choose it.
- **8.** $R = 2.5 \times (1 - 0.6 \times 0.9 \times 0.6) \approx 1.69$.
- **9.** Any two of: legal authority, trained staff, labs, data systems, working relationships. They take years of investment and trust.
- **10.** The measure ends while most people are still susceptible, so the wave simply happens later.
- **11.** $R = 2.5 \times (1 - 0.6 \times 0.5 \times 0.8) = 1.9$.
- **12.** Early growth depends on $\beta - \gamma$; the peak and final size depend on $R_0 = \beta / \gamma$, which the early curve does not pin down.

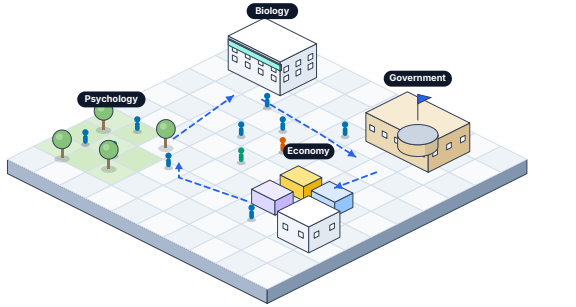
TRY-IT ANSWERS

Try-it answers: sick pay $q = 0.74$ gives $R = 1.5$. Policy packages: raising d alone gives 2.08, raising c alone 2.07; both together 1.5 because the terms multiply. Doubling every 5 days for 14 days: $2^{2.8} \approx 7\times$.

CREDITS

Sources and credits

- This deck accompanies Chapter 2 of T. Lazebnik, Epidemic Modeling: Principles, Algorithms, and Applications (Academic Press, 2026), the course textbook. It follows the chapter's topics in our own words; no text, figures or tables are copied from the book.
- The text, figures, examples and simulations were written, drawn and run for ACML; nothing is copied from the book. Numbers labelled "our run" come from our own code; counts from agencies and papers are credited where they appear.
- Studies cited by name: Mossong et al. 2008 (POLYMOD contact survey); Lloyd-Smith et al., Nature 2005; Fraser et al., PNAS 2004; Vosoughi, Roy and Aral, Science 2018; Pichler, Wen and Ziebarth, Health Affairs 2020; Goolsbee and Syverson, J. Public Economics 2021; World Health Organization on pandemic fatigue and the International Health Regulations (2005).
- All numbers, curves and toy calculations are our own runs (production/explains/sims/epi_m02_sims.py), chosen to show mechanisms, not to estimate any real disease.
- Companion videos: **Epidemic Modeling**, module 2, episodes 4–6. More from the lab: acml.teddylazebnik.com



Applied Computational Mathematics Lab

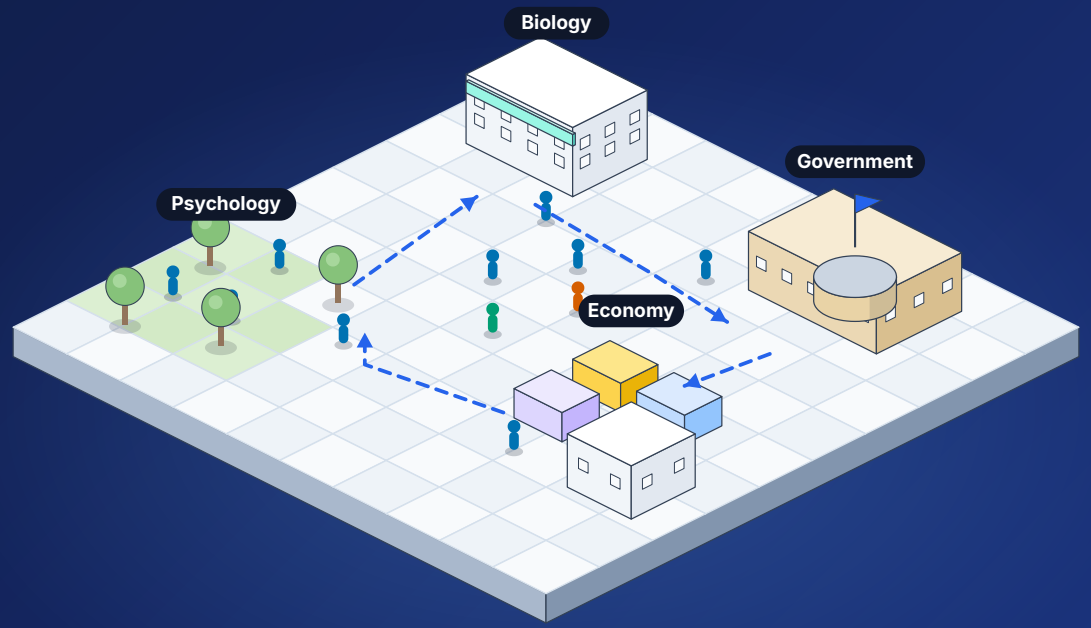
END OF MODULE 2

Beyond the virus

YOU COVERED

- How infections spread, and the biology behind it
- Fear, trust and money
- Governments and feedback loops

Module 2 complete



UP NEXT · MODULE 3

Compartmental models

SIR and its family, R_0 and the next-generation matrix

Based on Chapter 2 of T. Lazebnik, Epidemic Modeling: Principles, Algorithms, and Applications (Academic Press, 2026)