

A Brief Primer on SIR Models in Economics

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What is a SIR model?

- compartmental epidemiological model
- population of size N passes over time t through three stages (more can be added)
 - **S**usceptible, S_t
 - **I**nfectious, I_t
 - **R**esolved, R_t (recovered or dead)
- $N = S_t + I_t + R_t$ for all t
- initial condition: some infected, $I_0 > 0$; the rest $N - I_0$ susceptible

SIR Equations

- **S to I**

$$\frac{dS_t}{dt} = -\beta S_t \frac{I_t}{N}$$

- **I to R**

$$\frac{dI_t}{dt} = \beta S_t \frac{I_t}{N} - \gamma I_t$$

- **R (absorbing state)**

$$\frac{dR_t}{dt} = \gamma I_t$$

Key equation

$$\frac{dI_t}{dt} = \beta S_t \frac{I_t}{N} - \gamma I_t \quad (*)$$

- for a susceptible agent $i \in S_t$, the probability (rate) of being infected is

$$Prob(\mathbf{S} \rightarrow \mathbf{I}) = \beta \times \frac{I_t}{N}$$

- β captures
the probability/rate of infection conditional on contact with I person
 \times the contact rate
 - some authors write $\beta = \beta_0 M_t$
- $\frac{I_t}{N}$ is the **probability/rate of contact with I person** (uniform mixing)

Key parameters

- “infectiousness”, β
 - tricky, since product of biology (the chance of passing the infection upon meeting 1 person) and behavior/policy (the contact rate)
- “removal rate”, γ
 - (mostly) biological – how fast people recover and how many infected die (fraction $\mu\gamma$, approx. 0.3%–0.6% for COVID-19)
 - measured in time^{-1} ($1/\gamma$ is the expected time to recovery/death, e.g., in days)

Dynamics

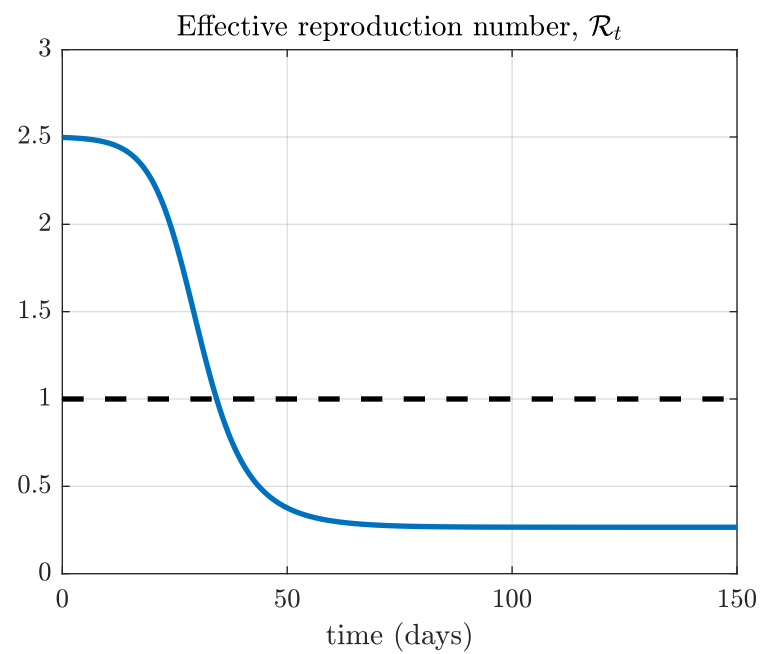
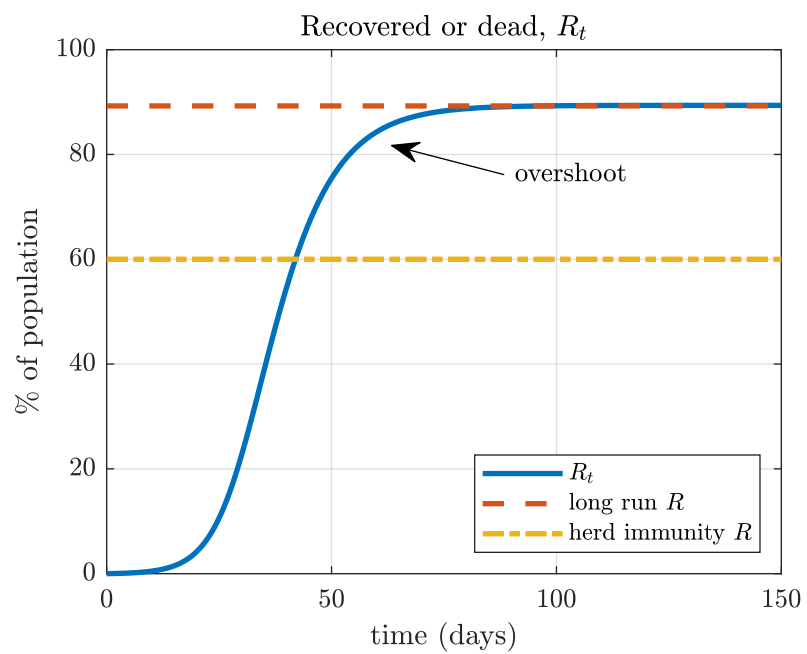
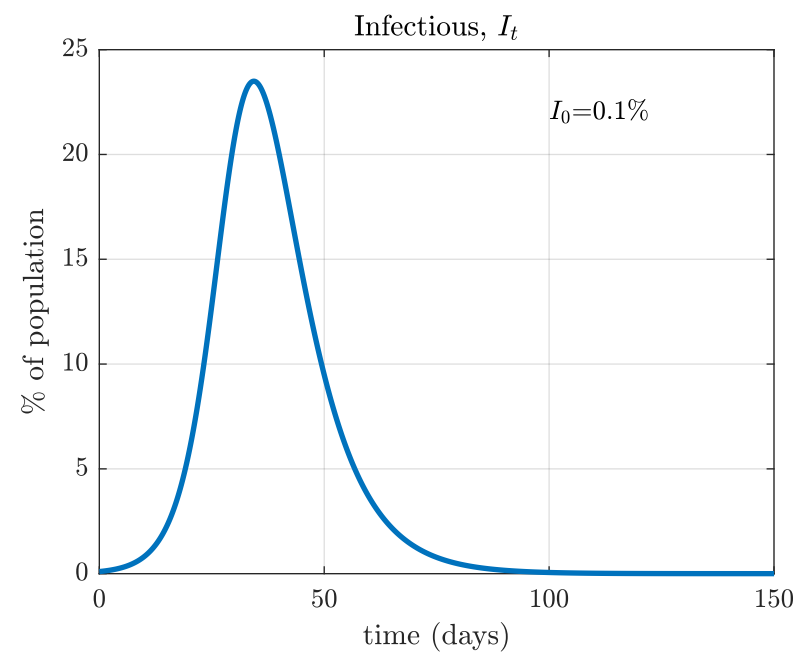
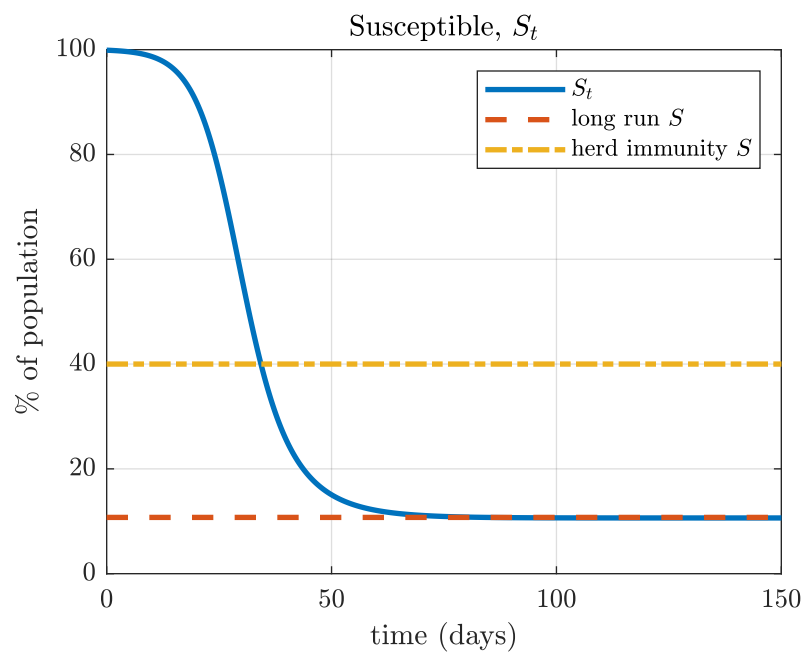
- re-write SIR equation (*) as:

$$\begin{aligned}\frac{dI_t}{dt} &= \beta S_t \frac{I_t}{N} - \gamma I_t = \\ &= I_t \gamma \left(\frac{\beta S_t}{\gamma N} - 1 \right)\end{aligned}\tag{1}$$

- the current number of infected I_t will grow, $\frac{dI_t}{dt} > 0$ if

$$\frac{\beta S_t}{\gamma N} > 1\tag{2}$$

and decrease otherwise



Basic reproduction number \mathcal{R}_0

\mathcal{R}_0 = expected new infections per unit of time generated by the first infected person, when $S_0 \simeq N$

note the confusing notation with R_t (resolved)

- from (1) we see that

$$\mathcal{R}_0 = \frac{\beta}{\gamma}$$

- hence the **importance of $\mathcal{R}_0 = \beta/\gamma$ being larger or smaller than 1**
 - if $\mathcal{R}_0 < 1$ the epidemic never takes off, even at $S_0 = N$, see (2)
- also, note that the early ($S_t \simeq N$) infection growth rate is

$$\frac{dI_t}{dt}/I_t \simeq \beta - \gamma$$

Effective reproduction number

- as the epidemic evolves in time (assuming $\mathcal{R}_0 > 1$) there are less susceptibles
- so, the expected new infections per 1 person and unit of time,

$$\mathcal{R}_t \equiv \frac{\beta S_t}{\gamma N}$$

decrease over time

- \mathcal{R}_t is called the **effective reproduction number**
- note that if *interventions* (e.g., lockdown, distancing, testing and quarantine) or *behavioral responses* affect β or S_t, I_t they affect \mathcal{R}_t too

Herd immunity

- when $\mathcal{R}_t = 1$ (inflow into state **I** equals outflow from **I**) it is said that “herd immunity” is reached (unstable steady state)

- herd immunity occurs when

$$\frac{dI_t}{dt} = 0$$

- that is, when the remaining fraction of susceptibles is

$$\frac{S_t}{N} = \frac{1}{\mathcal{R}_0}$$

- or, equivalently, when the fraction of recovered or dead (who were infected at some point) is

$$\frac{R_t}{N} = 1 - \frac{1}{\mathcal{R}_0}$$

- Example: if $\mathcal{R}_0 = 2.5$, need **60% of the population** to have been infected to reach herd immunity

Overshoot

- 60% may sound bad enough but the total number of infected, $\int I_t dt$ (and dead) *continues to grow after* herd immunity is reached
- this is known as **overshoot**

Why?

- $\frac{dI_t}{dt} < 0$ after herd immunity is reached means only that there are fewer daily **new** infections (I_t decreases) but $I_t > 0$ still
- important role for isolating known infectious persons

Extensions

- “Exposed” state – incorporate incubation time (around 5 days for COVID-19)
 - SEIR model
- “Quarantine” state – e.g., if an individual tests positive and is isolated from contacts
 - reduces $\frac{I_t}{N}$ in $\beta S_t \frac{I_t}{N}$
- “symptomatic” vs. “asymptomatic” infectious states

SIR model and social networks

- the standard SIR model assumes population-level, uniform mixing
- abstracts from locality and the fact that many infections occur via social contacts (broadly defined)

Network SIR model (NSIR)

- represent the population by graph G (nodes with social links among them)
- probability of a susceptible person i becoming infected at time t depends on i 's social contacts

$$Prob(\mathbf{S} \rightarrow \mathbf{I}) = \beta \frac{\sum_{j \in C_G(i)} \mathbf{1}_{x_{jt}=\mathbf{I}}}{\#C_G(i)}$$

- where $C_G(i)$ is the set of person i 's contacts (other nodes in G)
- $\#C_G(i)$ is node i 's degree (number of contacts)

Effective reproduction number in the NSIR model

- note that the graph structure affects the infection dynamics
- in the standard SIR model, the probability of **S** person i meeting an **I** person is **uniform**, $\frac{I_t}{N} \forall i$ (“representative agent model”)
- in the network model this probability is **heterogeneous** and depends on i ’s social contacts

$$\sigma_{it}(G) \equiv \frac{\sum_{j \in C_G(i)} \mathbf{1}_{x_{jt}=\mathbf{I}}}{\#C_G(i)}$$

- can model ‘superspreaders’, clusters, local outbreaks, etc.

Policy interventions

- essentially, trying to make smaller the term $\beta S_t \frac{I_t}{N}$
- main benefit: “**flatten the curve**” I_t
 - reduce its peak (ICU capacity constraint)
 - reduce the overshoot and total deaths
- cost: can prolong the epidemic duration

Policy interventions

- simplest, reduced-form way
 - *time-dependent* β , e.g., $\beta_t = (1 - l_t)\beta$ (Atkeson, 2020; Moll, 2020)
 - * equivalent to reducing the effective reproduction number \mathcal{R}_t
 - model “distancing” or “lockdown” as period of lower β_t
- alternative way: reduce the rate of meetings with infectious persons $S_t \frac{I_t}{N}$
 - e.g., $(1 - \lambda)S_t$ meet with $(1 - \lambda)I_t$ where λ is fraction locked down (Alvarez et al. 2020)
 - different meeting technology (Acemoglu et al., 2020)

Testing and quarantine

- introduce state **P** (tested positive) which evolves as

$$\frac{dP_t}{dt} = \theta I_t - \gamma P_t$$

where θ is the testing rate per unit of time

- Note: the “new cases” reported in the news every day correspond to P_t and **not** to I_t (infectious)
- Quarantine or (self-)isolation:
 - assume **P** agents are removed from meetings
 - or meet on a restricted-contacts graph G_Q

Contact tracing

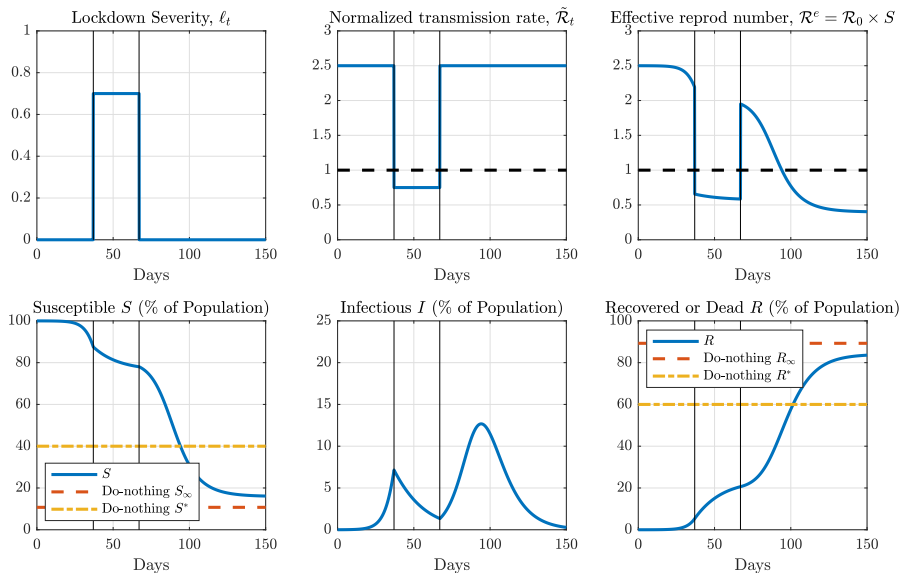
- key role for the graph G
- tracking, testing and isolating infected social contacts of positive (**P**) agents

$$Prob(\mathbf{I} \rightarrow \mathbf{P}) = \theta + \phi \sum_{j \in C_G(i)} \mathbf{1}_{x_{jt}=\mathbf{P}}$$

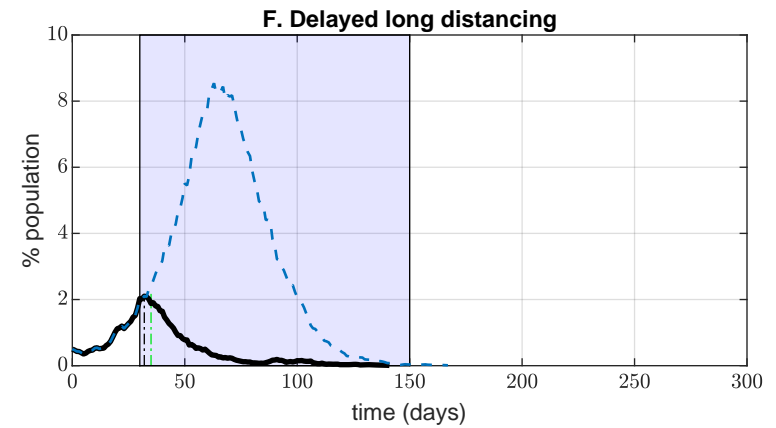
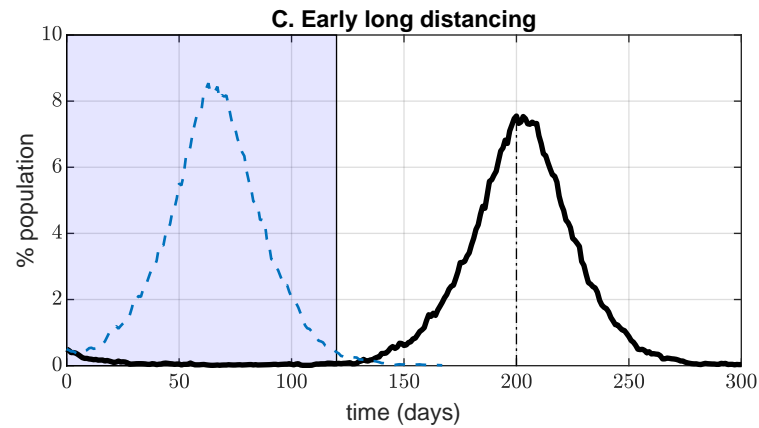
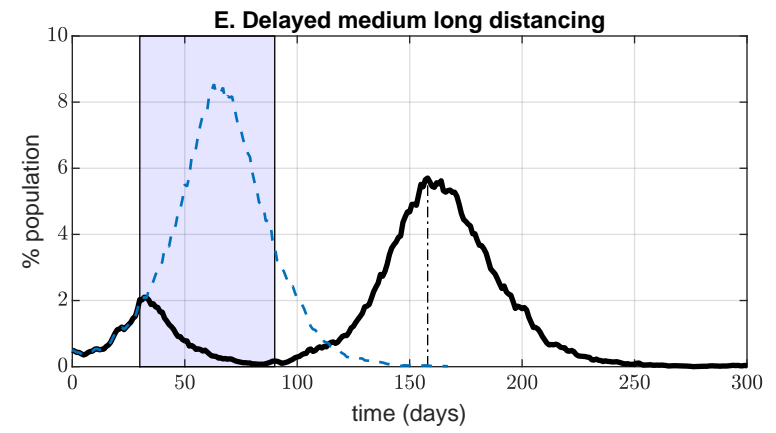
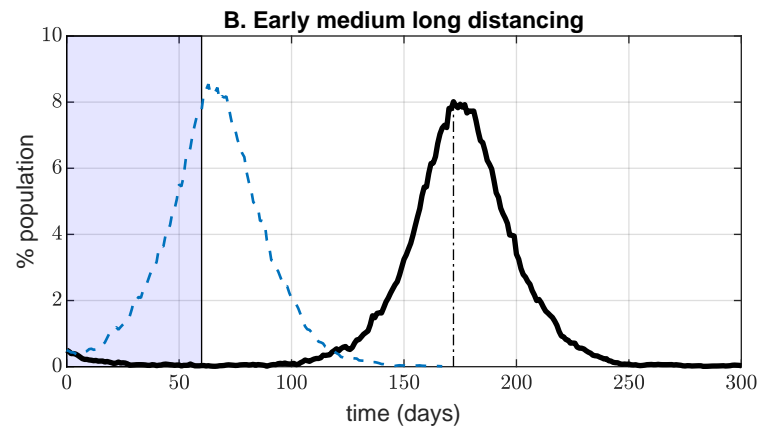
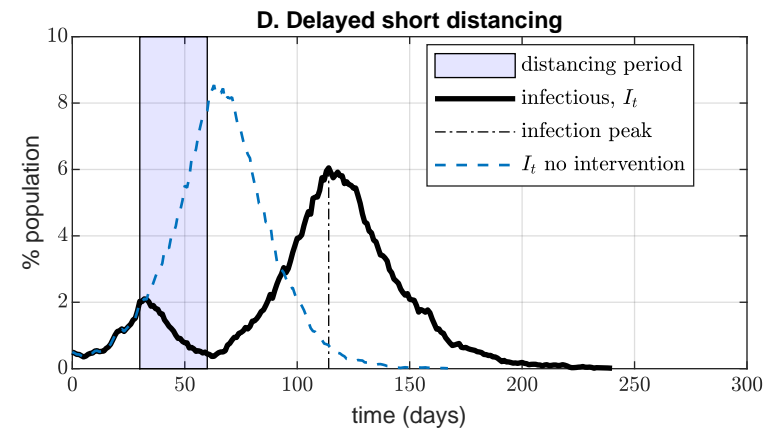
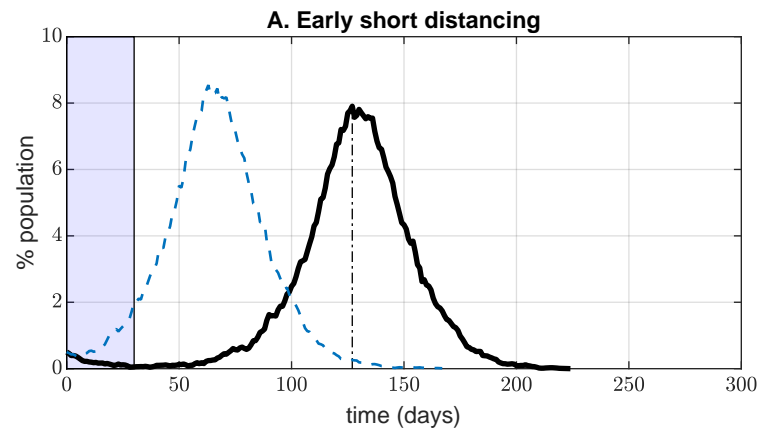
where ϕ is the contact tracing rate

Examples and discussion

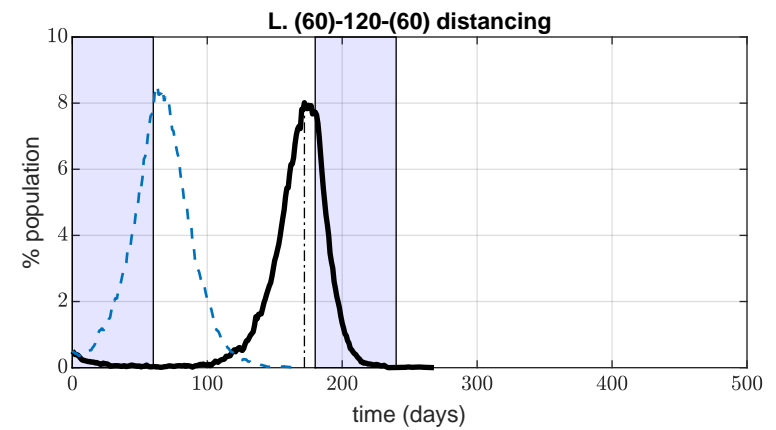
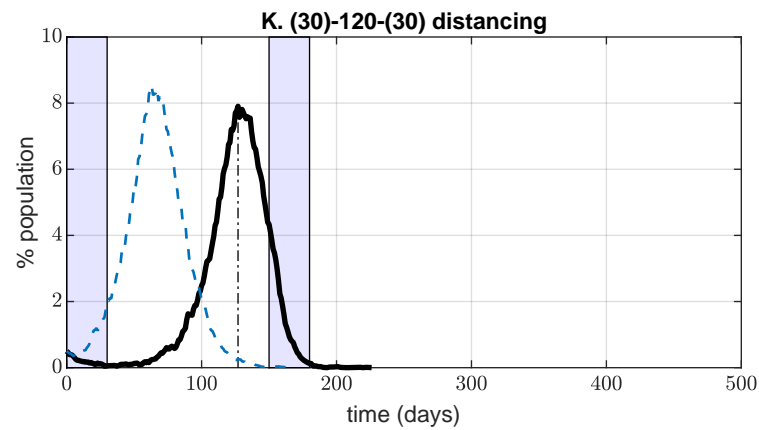
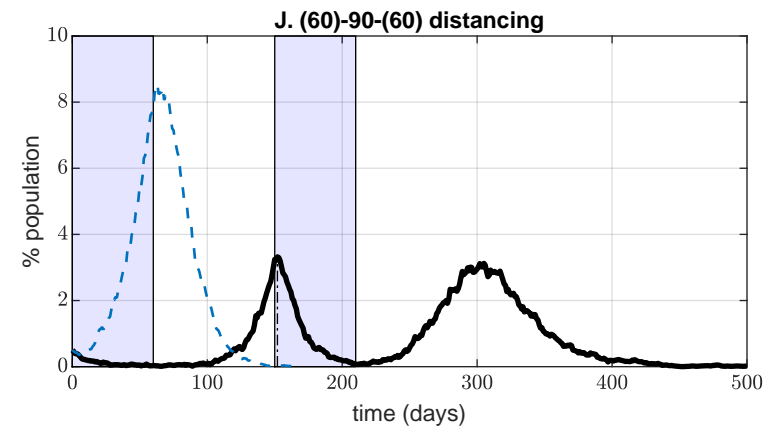
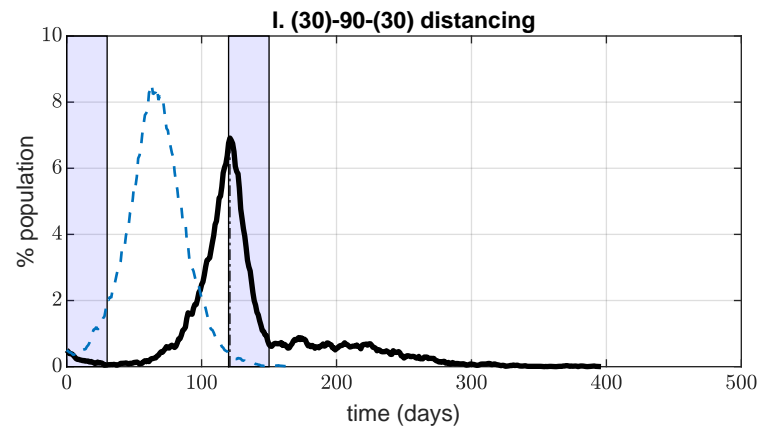
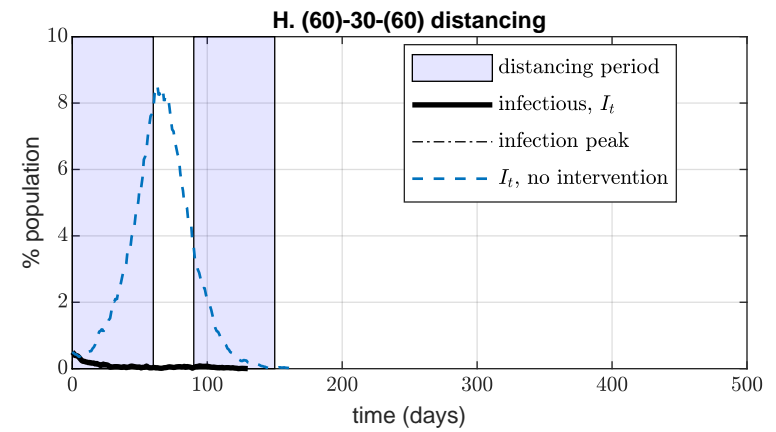
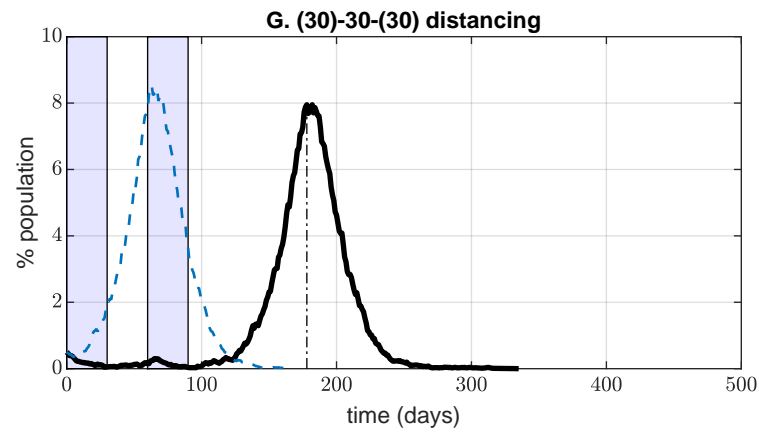
Short and Tight Lockdown



One-off or intermittent distancing

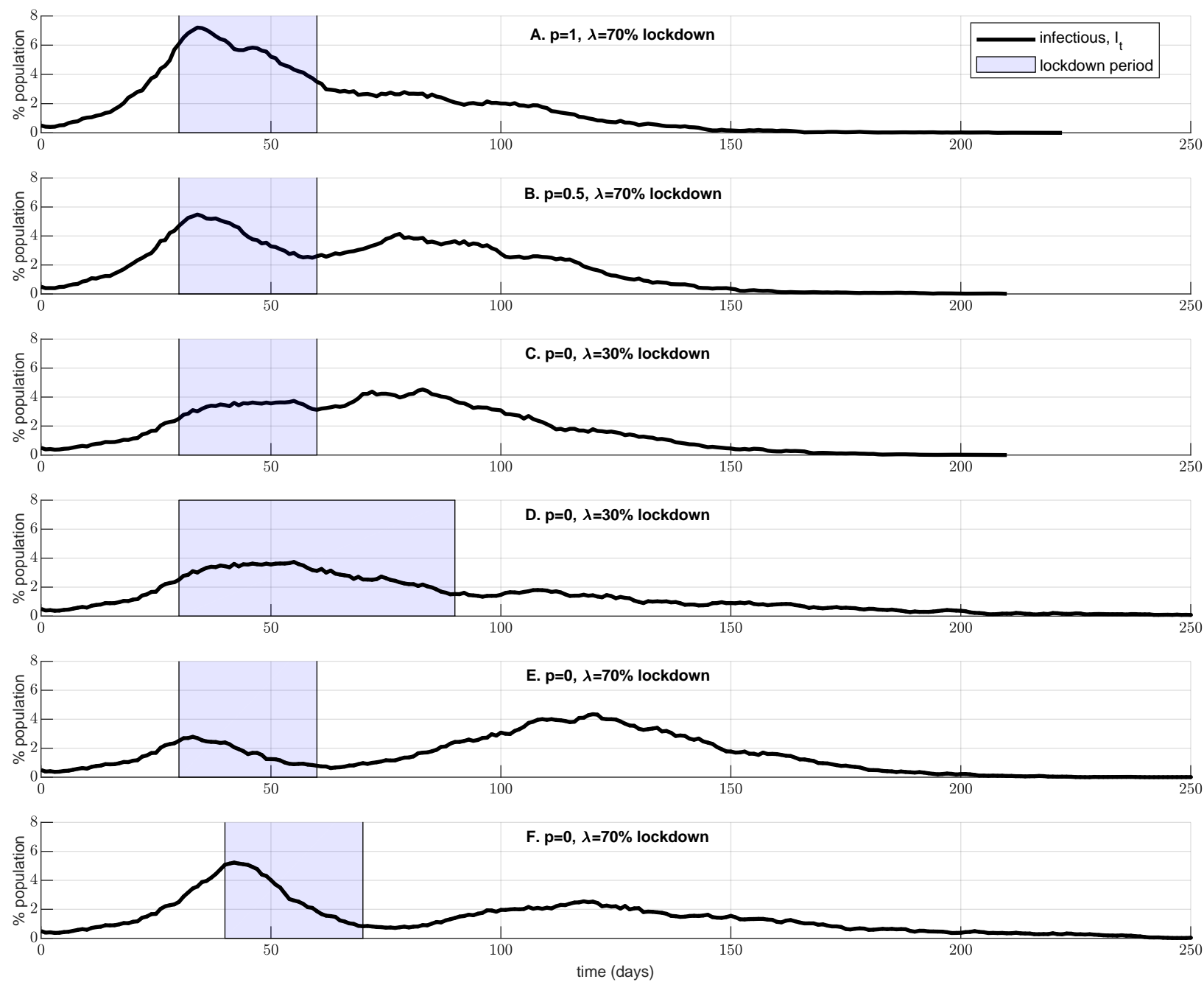


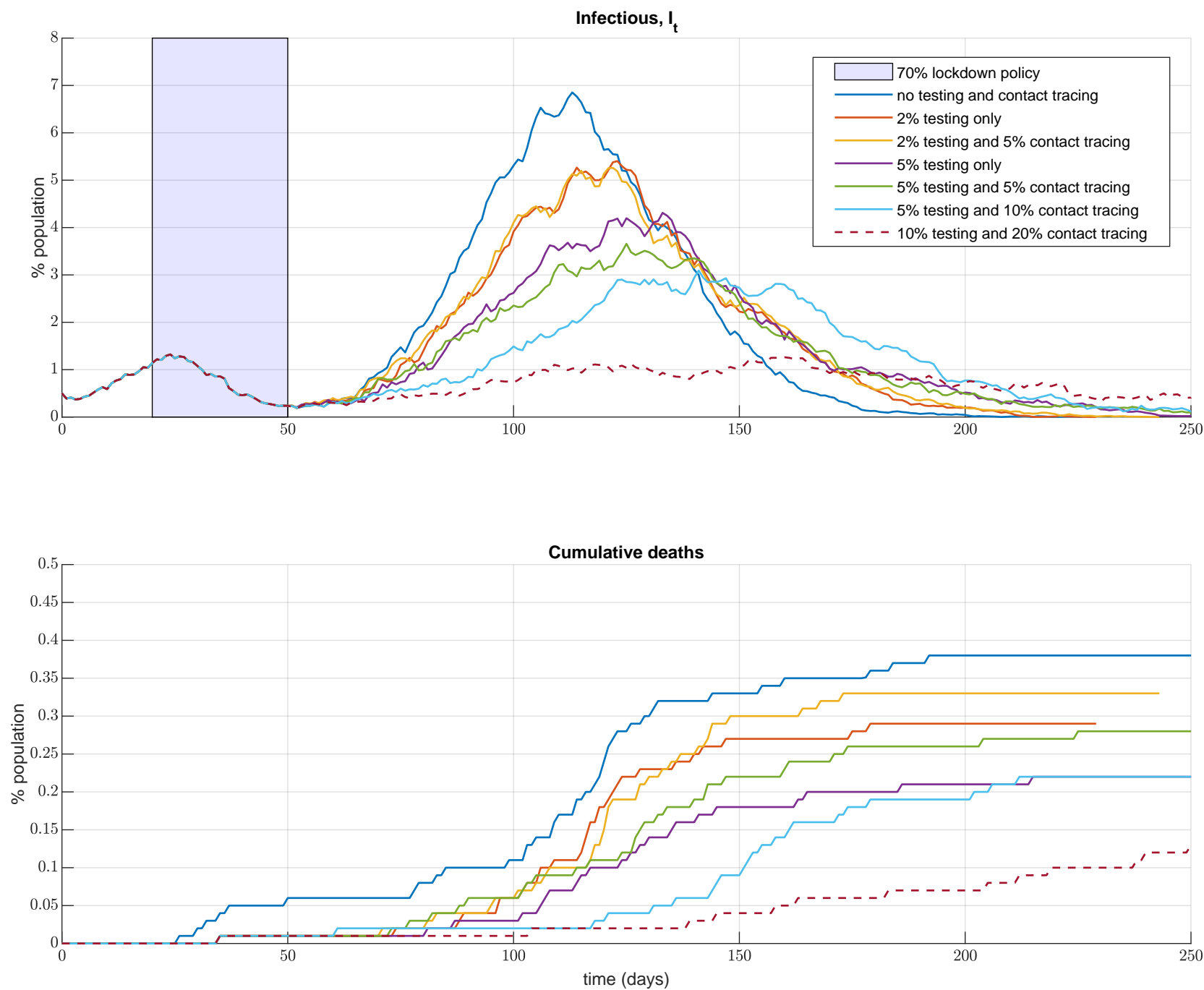
Note: assumed initial infection rate 0.5%



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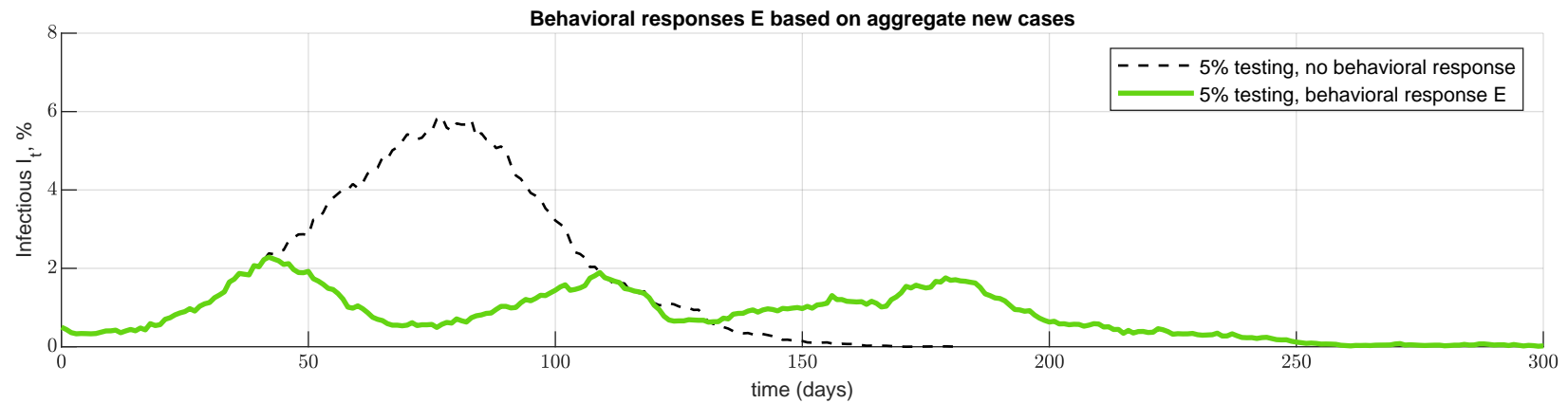
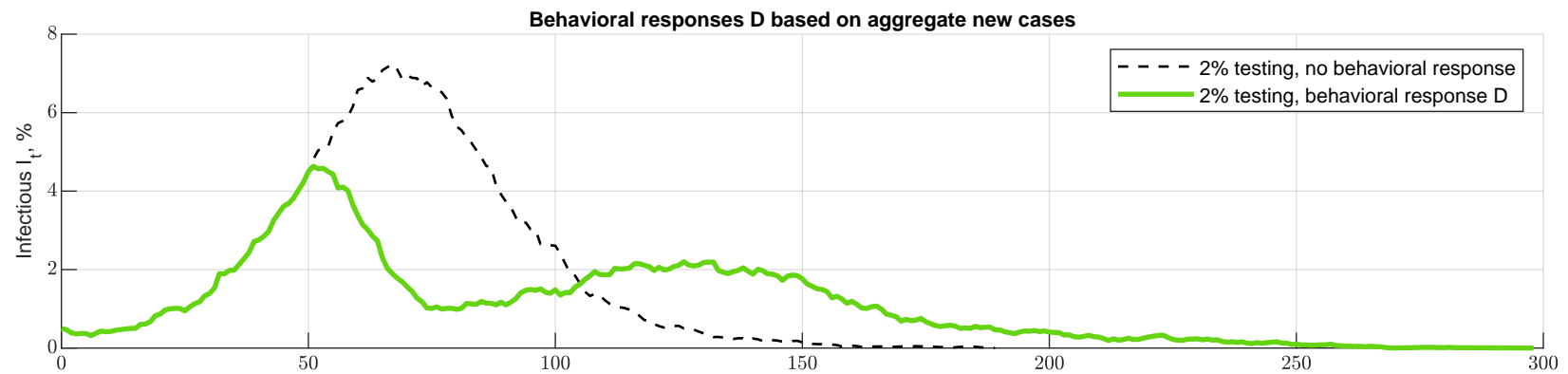
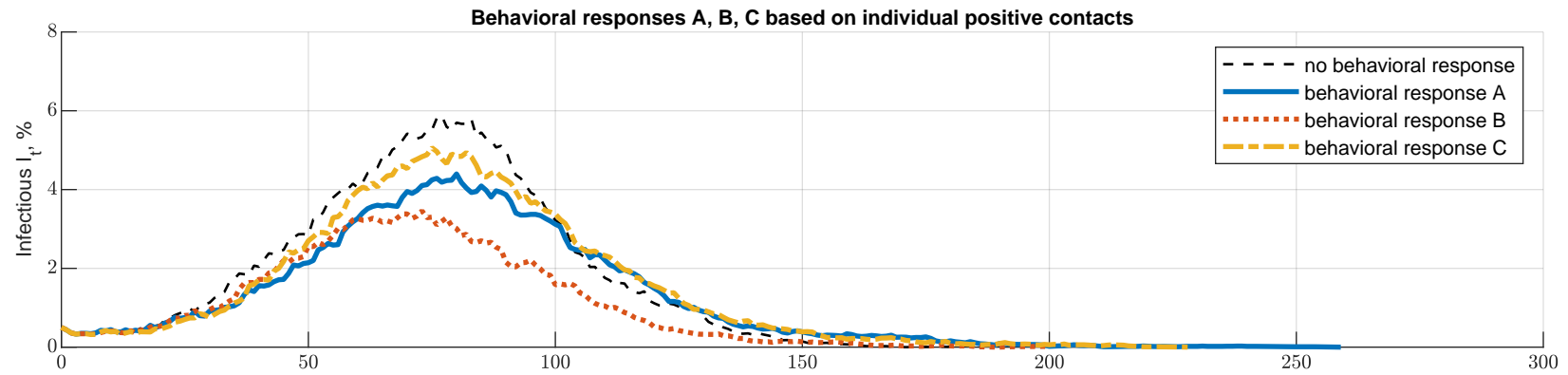
Lockdown exit





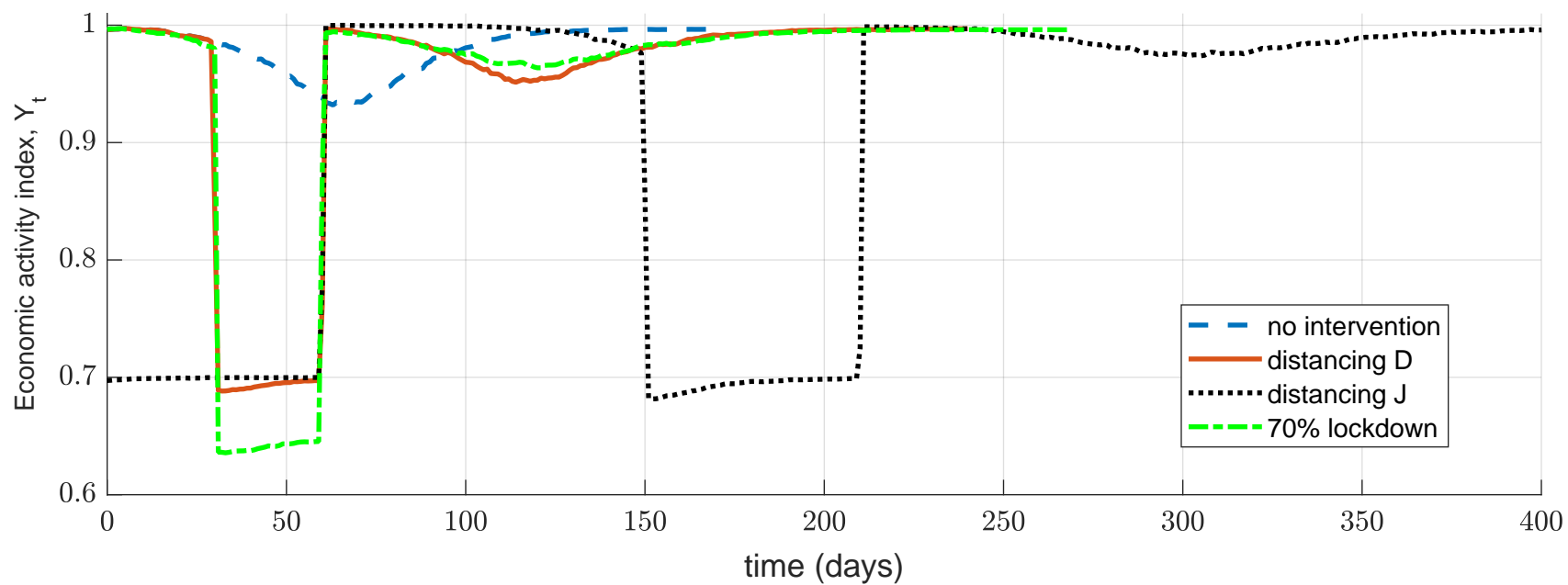
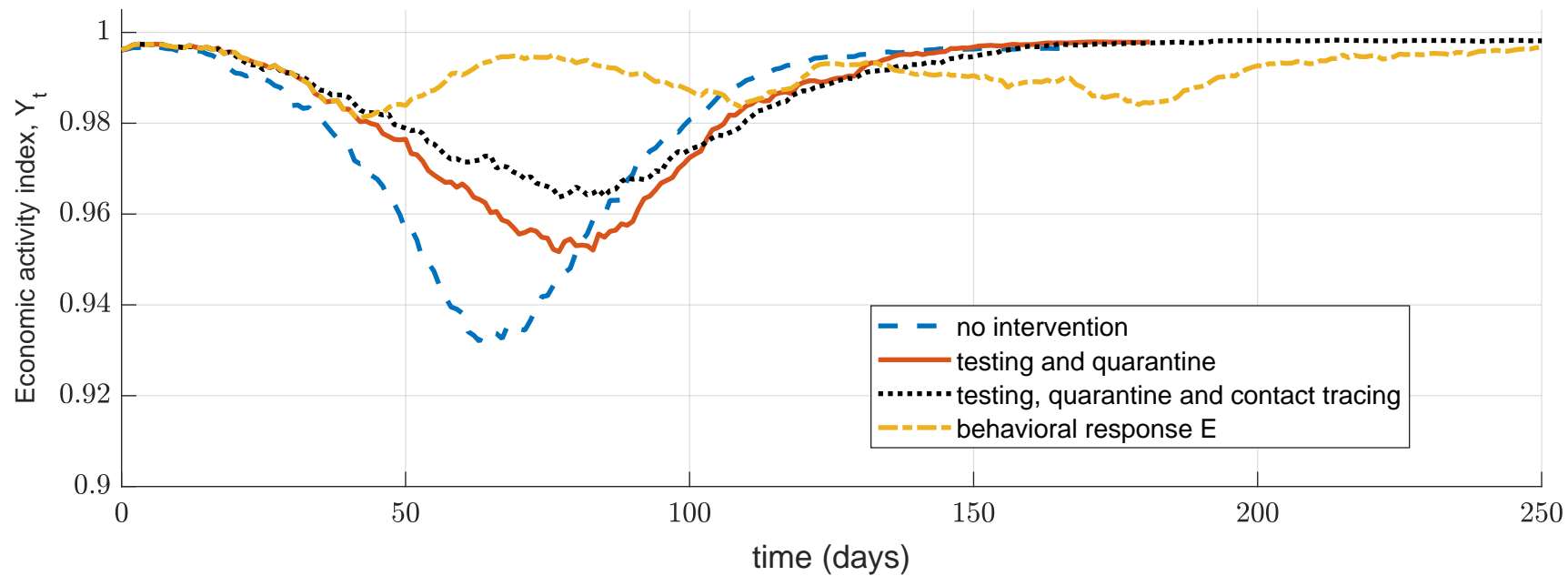
Behavioral responses

- assume timely information on positive cases is available
- responses (A, B, C) based on individual circumstances – e.g., a contact who tested positive
 - permanent reduction in contacts (upper bound)
 - temporary reduction (while a contact is positive)
- responses D, E based on aggregate data (x new cases in the past y days)



Impact on the economy?

- requires (heroic) assumptions about
 - productivity in lockdown and/or when infectious (symptomatic vs. asymptomatic)
 - value of life / years
- current frontier working papers look at interventions and outcomes by segments
 - by industry: e.g., retail vs. IT jobs are affected very differently
 - or, by population cohorts: elderly vs. work-age vs. school-age



Some Unpleasant Lockdown Arithmetic

- If lockdowns only option, how long do effective ones need to last?
- Key: **need to reach herd immunity**. So: how long to reach that?
- Optimistically assume perm immunity, $\mathcal{R}_0 \downarrow$ to 2 (better hygiene...)

herd immunity threshold = $1 - 1/\mathcal{R}_0 = 50\%$
- **Simple back of envelope calculation for U.S.** Assumptions:
 1. 10% have had disease \Rightarrow need additional 40% \approx 100 million
 2. lockdown suppresses \mathcal{R}_t^e to 1, infections rolled over (sl 40-41)
 ($\mathcal{R}_t^e = 1$ close to current US estimate)
 3. 200,000 new infections per day (current official count \approx 30,000)
- \Rightarrow need **some** sort of lockdown / control for

$$\frac{100 \text{ million}}{200,000} = 500 \text{ days}$$
- Note: optimistic calculation assuming low \mathcal{R}_0 , permanent immunity

References

- Acemoglu, D., V. Chernozhukov, I. Werning and M. Whinston (2020), "A Multi-Risk SIR Model with Optimally Targeted Lockdown"
- Alvarez, F., D. Argente and F. Lippi (2020), "A Simple Planning Problem for COVID-19 Lockdown"
- Atkeson, A. (2020), "What will be the Economic Impact of COVID-19 in the US? Rough Estimates of Disease Scenarios"
- Azzimonti, M., A. Fogli, F. Perri and M. Ponder (2020), "Social Distance Policies in Network Cities"
- Berger, D., K. Herkenhoff and S. Mongey (2020), "An SEIR Infectious Disease Model with Testing and Conditional Quarantine"
- Chari, V.V., R. Kirpalani and C. Phelan (2020), "The Hammer and the Scalpel: On the Economics of Indiscriminate versus Targeted Isolation Policies during Pandemics"
- Farboodi, M., G. Jarosch and R. Shimer (2020), "Internal and External Effects of Social Distancing in a Pandemic"
- Fernandez-Villaverde, J. and C. Jones (2020), "Estimating and Simulating a SIRD Model of COVID-19 for Many Countries, States, and Cities"
- Karaivanov, A. (2020), "A Social Network Model of COVID-19", http://www.sfu.ca/~akaraiva/Karaivanov_covid2020.pdf
- Kermack, W. and A. McKendrick (1927), "A contribution to the mathematical theory of epidemics", *Proceedings of the Royal Society A*, 115(772), p.700-721
- Kuchler, T., D. Russel and J. Stroebe (2020), "The geographic spread of COVID-19 correlates with structure of social networks as measured by Facebook"
- Moll, B. (2020), "Lockdowns in SIR Models", slides