

Perfect Epidemics

2nd Workshop, UK Research Network in Stochastics
University of Liverpool

W S Kendall S B Connor

Warwick, York

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Introduction

“Once we came to accept the photographic image as reality, the way to its future simulation was open.”
[Lev Manovich]



Handout is on the web: use the QR-code or visit
wilfridskendall.github.io/talks/PerfectEpidemics.

Work on perfect simulation ([CFTP](#)) for epidemics, now being written up.
WSK acknowledges the support of UK EPSRC grant EP/R022100.

1. Introduction to Perfect Simulation

- ➊ Propp & Wilson (1996) invented exact simulation / Coupling from the Past ([CFTP](#)) / perfect simulation;
- ➋ The term “perfect simulation” (WSK, 1998) was chosen to encourage you to be suspicious: perfection is never achieved!
- ➌ Key ideas of “classic [CFTP](#)”:
 - ▶ extend simulation *backwards* through time not forwards;
 - ▶ exploit monotonicity (*couple* maximal and minimal processes);
 - ▶ seek coalescence.
- ➍ Simplest possible example: *random-walk-CFTP*
(can boost to use Ising model to do simple image reconstruction).

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Here is a very brief summary of [CFTP](#)/ perfect simulation.

- ➊ Jim Propp described the discovery of [CFTP](#) as like walking down the street and suddenly noticing a 50\$ bill lying on the ground.
- ➋ In particular, “exact simulation” cannot somehow miraculously defeat numerical approximation error :-). And, as with all simulation, its validity depends on correctness of code!
- ➌ We will illustrate these key ideas by considering a single very specific example.
- ➍ Propp & Wilson (1996) show how to vary random walk [CFTP](#) to get exact samples for a [critical](#) Ising model (Persi Diaconis: “Like seeing the landscape of Mars for the first time”). The Ising model with an external field can be used to model images, hence [CFTP](#) can be used for image reconstruction.

Classic CFTP for a simple random walk (I)

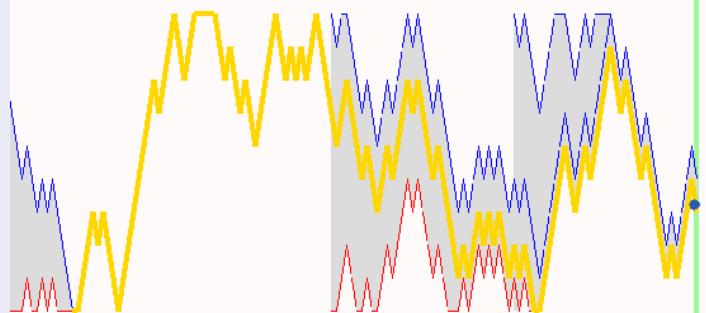
- ① Consider a simple random walk on $0:9 = \{0, 1, 2, 3, 4, 5, 6, 7, 8, 9\}$.
 - ▶ $\mathbb{P}[+1 \text{ jump}] = p \in (0, 1)$, while $\mathbb{P}[-1 \text{ jump}] = 1-p$, except that at state 9 replace the +1 jump by “staying still”, and at state 0 replace the -1 jump by “staying still”.
- ② Conventional MCMC picks a starting point, then runs the simple random walk for long time till approximate equilibrium.



- ③ How long? One way to *estimate* this is to run two (or several?) coupled copies till they meet. If probability of meeting by time T is high, then deviation of X_T from equilibrium is statistically small;
- ④ Generally **not true** that location *at* coupling is a draw from equilibrium.

Classic CFTP for a simple random walk (I)

- ① Start at top (9) and bottom (0) at negative time $-T$, run to time 0.



- ② If not coupled by time 0, then back-off to time $-2T$ and repeat.
NB: re-use randomness!
- ③ May need to iterate back-off doubling several times.
- ④ When coupled, top and bottom yield a common value at time 0.
- ⑤ The common value (golden thread) is an exact draw from equilibrium!

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This random process is asymmetric simple random walk on the integers, modified by forbidding any transitions outside the specified state-space $0:9$.

- ① The process is irreducible *and* aperiodic, so equilibrium is a truncated Geometric distribution (hint: use reversibility to check this!).
- ② Of course even conventional MCMC is overkill here: simulation directly from the equilibrium distribution is simple and efficient.
- ③ This observation was originally formalized by Aldous. Here we use “synchronous” coupling – variants are possible but not particularly useful.
- ④ In the case of reflecting simple random walk, coupling can only occur at the boundary points 0 or 9! So here the blue and red evolutions can only couple at a boundary; thus self-evidently the value at time of coupling cannot be a draw from the equilibrium!

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- ① Ideally one needs to choose T neither too small nor too large. But the result is not particularly sensitive to this.
- ② Very important in this binary back-off that one doesn't use different jump proposals for the same time $-t$! Couple to **re-use randomness**.
- ③ *Binary back-off procedure*, so if initially T too small then at most four-fold extra work compared to conventional MCMC!
- ④ Re-use of randomness means there is no point in extending the binary backoff beyond coalescence: the common value at time 0 will be the same however far we extend into the past with further back-offs.
- ⑤ Why is the common value an exact draw from equilibrium? Informally, because one would get the same result however far one backed-off: therefore the draw is effectively a draw from time $-\infty$. The golden thread can be viewed as a perfect draw from the last segment of such a simulation (this sort of device is very well-known to ergodic theorists). Remarkably, this can easily be converted into a fully rigorous proof!

2. A little theory about CFTP-

- ① What if monotonicity fails? or there isn't a sensible “maximal” process?
Ideas (WSK, 1998):

- ▶ cross-couple upper and lower envelope processes;
- ▶ dominate by amenable “dominating process” (time-reversible, can draw from equilibrium, can couple target processes below dominating process).

- ② Theoretical limits: *in principle*

- ▶ Classical CFTP equivalent to uniform ergodicity (Foss & Tweedie, 1998);
- ▶ Dominated CFTP achievable under geometric ergodicity (WSK, 2004);
- ▶ Dominated CFTP can work in some non-geometrically ergodicity cases (SBC & WSK, 2007a; *nb* corrigendum SBC & WSK, 2007b).

- ③ Dominated CFTP delivers perfect simulation for stable point processes (WSK & Møller, 2000);
④ Detailed expositions: WSK (2005), Huber (2015).
(Want to implement CFTP in R? see WSK, 2015.)

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└ 2. A little theory about CFTP-

- ① To be computationally effective, WSK (1998) ideas still require a (perhaps weak) notion of an associated partial order, and also the ability to simulate the dominating process.
- ② Basic ideas: use the notion of regenerative sets (“small sets”), and (Foster-)Lyapunov arguments. Note that the resulting recipes tend not to be computationally practical: they simply suggest the *possibility* of (possibly computationally infeasible) CFTP. They are intended to frame a challenge: when can one find *practical* methods?
- ③ (Locally finite) point processes are *generally* not accessible via Classic CFTP; the “top” pattern would have points *everywhere*. The “in principle” results encourage us to search for CFTP for a wide variety of challenge problems [for example, $M/G/k$ multiserver queues; see SBC & WSK (2015); SBC (2020)].
- ④ This ends the visual introduction to CFTP. People who are interested in practical CFTP may find it useful to work through WSK (2015).

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3. Perfect Epidemics: a challenge problem for CFTP

S-I-R deterministic epidemic:

based on susceptibles s , infectives i , removals r :

$$\begin{aligned} s' &= -\alpha s i, \\ i' &= (\alpha s - \beta) i, \\ r' &= \beta i. \end{aligned}$$

Constant total population $s + i + r = n$.

S-I-R stochastic epidemic: a Markov chain (S, I, R) with transitions

| | | | | |
|-------------------|-------------------------|-----------------------|---------|----------------|
| Infection: | $S \rightarrow S - 1$, | $I \rightarrow I + 1$ | at rate | $\alpha S I$, |
| Removal: | $I \rightarrow I - 1$, | $R \rightarrow R + 1$ | at rate | βI . |

Both make an **unrealistic assumption**: homogeneous mixing.

In contrast, Fraser & Others (2023) use a UK model with 10^6 agents!

There are *many* important inferential questions (Cori & Kucharski, 2024).

Perfect Epidemics

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- Two classical models (one deterministic, one stochastic) from mathematical epidemiology. Even the exceedingly simple case of deterministic S-I-R permits only *partial* closed-form solution.
- The assumption of homogeneous mixing of population greatly simplifies mathematical *and* statistical issues. Without this assumption one risks huge numbers of parameters, very bad news statistically speaking. Much research dealing with more realistic models, for example, UK model of Fraser & Others (2023) with 1000000 agents.
- The work presented in this talk considers only perfect simulation for homogeneous mixing. Before running, one must learn to walk!
- Cori & Kucharski (2024) provide a broad and stimulating perspective on statistical challenges from a very practical point of view!

The first question asked about a new epidemic

“What is the R-number?”

The R-number is $\alpha s_0 / \beta$: mean number of new infectives produced per infective at *start* of epidemic with initially s_0 susceptibles.

Whittle (1955)’s threshold theorem: R-number $\gg 1$ means strongly positive chance of epidemic infecting significant proportion of the population.

Wikipedia: “The British-registered *Diamond Princess* was the first cruise ship to have a major [COVID-19] outbreak on board, with the ship quarantined at Yokohama from 4 February 2020 for about a month. Of 3711 passengers and crew, around 700 people became infected and 9 people died.”

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- I am skipping over very influential early work on the *Diamond Princess* incident, which particularly focussed on the incubation period (a time when the subject is infectious, perhaps at time-varying rate, before symptoms appear). This is related to drawing inferences about β .
- See Mizumoto *et al* (2020) for very early analysis of the *Diamond Princess* outbreak.
- Question:** is there a dataset for the *Diamond Princess* outbreak? and how detailed is it? Rocklöv *et al.* (2020) focusses on R-number and refers to some public sources of cumulative daily totals.

Inference on the R-number

Important, because the R-number controls severity of epidemic. However:

- Modelling is **tough**. *Either* massive assumptions (homogeneous mixing) *or* very many parameters;
- Inference is **really tough**: hard to get information about infection times;
- It is all **especially tough** in early stages. Answers are most needed when hardly any information is available (a simplified example for a Warwick UG second-year statistics module shows how tough this can be);
- Markov chain Monte Carlo (MCMC) can be used (see next slide) but what about burn-in?
- Can we use **perfect simulation**?

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➎ Can we use **perfect simulation**?

- The “R-number” was the subject of continuing interest throughout the COVID-19 pandemic!
- Notice that the “R-number” is as much a social construct as a biological one, and in real situations will interact in subtle and complicated ways with behaviour.
- We will seek to make headway in using perfect simulation to aid inference.
- The PhD thesis of Bensoussane (2025) is a useful source of references.
- Here is a promising setting for demanding challenge problems.

An easier question

An absurdly simple variant of contact tracing:

“When did the infections occur, supposing we only observe removals?”
 (Gibson & Renshaw, 1998; O’Neill & Roberts, 1999; Gibson & Renshaw, 2001)

Important first step: think about generation of an *unconditioned* epidemic.

- ➊ Suppose n, α, β are known. Eventually removal times are observed, but unobserved infection times **must be inferred**.
- ➋ Visualize n timelines, along which incidents are scattered:
 - ▶ potential removals, activated if timeline is infected;
 - ▶ potential infections, activated if timeline is infected *and* if designated target timeline is lowest uninfected timeline.
- ➌ Poisson point processes of *appropriate rates* yield an S-I-R epidemic.
- ➍ First step: evolve whole S-I-R trajectory in *algorithmic time* (alter potential infections and removals using immigration-death in discrete algorithmic time).
- ➎ Result: *trajectory-valued chain*, unconditioned S-I-R as equilibrium.

From incidents to unconditioned epidemic trajectories (1/3)

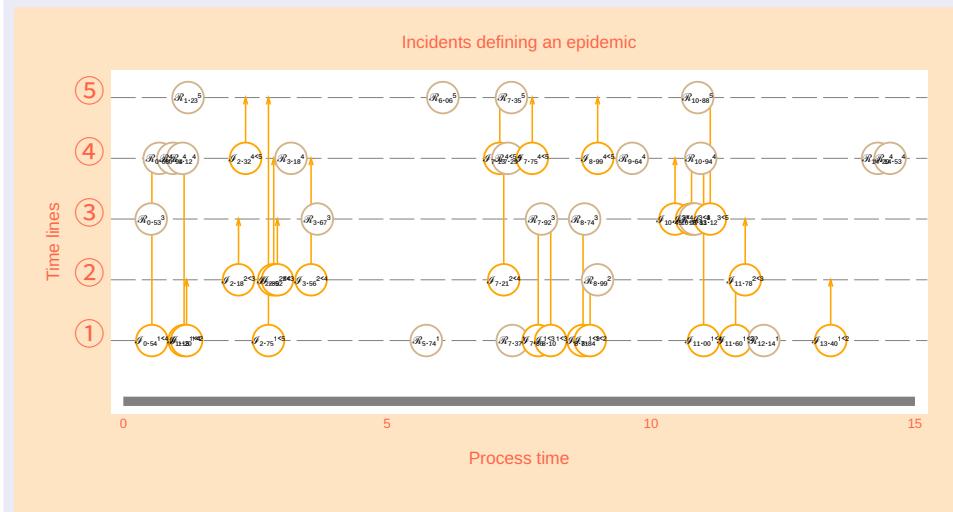


Figure 1: Light-orange circles denote potential infections (arrows point upwards to targets); light-brown circles denote potential removals.

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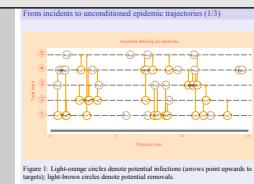
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Focus on just one aspect of inference:

- ➊ Avoid parameter inference for now; consider the large number of nuisance parameters given by (typically unobserved) infection times.
- ➋ Timelines: instead of tracking individuals as in the stochastic epidemic model, track the numbers of individuals in various categories.
- ➌ Poisson point processes generate incidents, which then collectively influence the S-I-R trajectory.
- ➍ I learned the phrase “algorithmic time” from Andrew Stuart.
 Move from Poisson points spread out along timelines to discrete immigration-death processes evolving in algorithmic time.
- ➎ Exploit the classic connection for reversible Markov processes, between conditioning and restriction of state space.
- ➏ Work in *continuous* algorithmic time, obtain a dynamically reversible Markov process, prove restriction=conditioning still works.

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As implied above, incidents comprising a single innovation are produced by Poisson point processes on each of the timelines.

Colour-coding distinguishes infections from deaths. Note that infections need to specify which timeline is going to be infected.

- Rates of Poisson processes generating the (proposed) incidents are chosen to ensure that numbers of actual susceptibles, infectives and removals form a Markov chain with S-I-R epidemic rates.
- For an infection incident to be activated, it must sit on an infected portion of its timeline and must point to the least of the uninfected timelines (calculating at the process time of the incident). (This is computationally expensive, but simplifies monotonicity arguments.)
- For a removal incident to be activated, it must sit on an infected portion of its timeline.

From incidents to unconditioned epidemic trajectories (2/3)

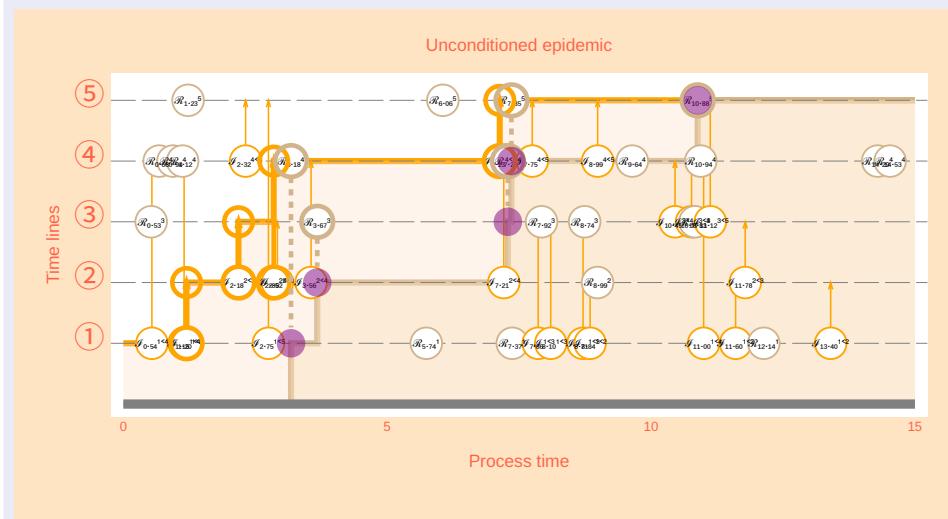
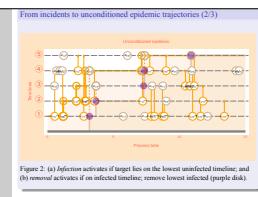


Figure 2: (a) *Infection* activates if target lies on the lowest uninfected timeline; and (b) *removal* activates if on infected timeline; remove lowest infected (purple disk).

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- Here is the resulting evolution of an epidemic.
- Note that here we specify the initial number of infectives (here we specify $I_0 = 1$).
- However we can do better: given a prior for I_0 , at the start of each cycle we could introduce an accept-reject move which alters I_0 while respecting detailed balance.
- Options include use of an independence sampler, or of a random-walk Metropolis sampler.

From incidents to unconditioned epidemic trajectories (3/3)

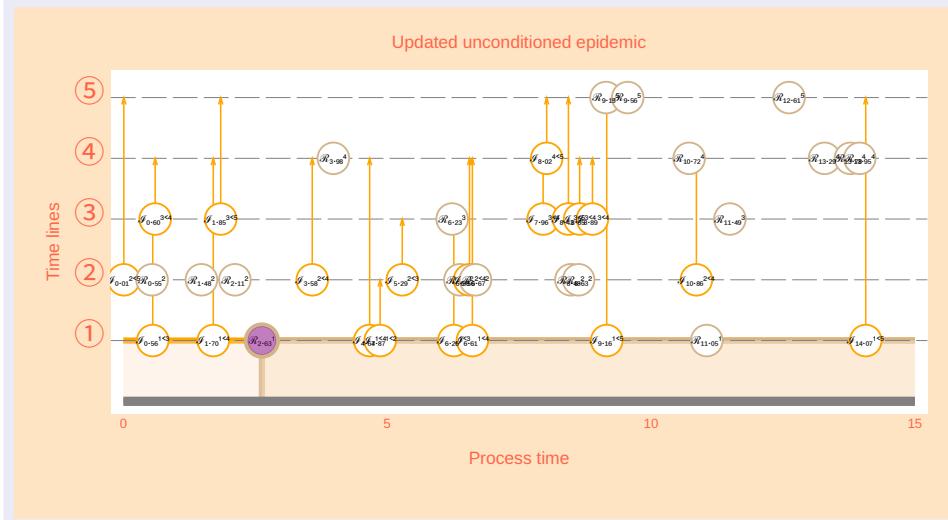
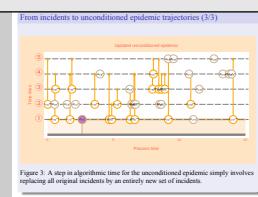


Figure 3: A step in algorithmic time for the unconditioned epidemic simply involves replacing all original incidents by an entirely new set of incidents.

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- An update in algorithmic time, if there is no conditioning, simply involves replacement of one set of incidents by another. (Here there is a radical change, as epidemic dies out at an early stage.)
- But we need to refine this if we are to take account of conditioning: a simple accept/reject procedure will almost always reject proposals of *entire* innovations involving all of both infection and removal incidents (and I_0 if we update this too).
- We need to *localize* the proposal, by considering one change at a time. (Intuitively, a “pixel-by-pixel” analysis working along the time axis.)

Crucial technical point

- Updates in algorithmic time τ are then (algorithmic-)*time-reversible*: so restriction to a subset S of state-space (the *activated / conditioned* removals to occur precisely at the specified set of times) implies a new equilibrium which is the old equilibrium conditioned to lie in S .
- For later purposes it is convenient to stage the replacement as follows:
 - ① Replace removals (\mathcal{R}_S);
 - ② Re-sample timelines (though not times) of \mathcal{R}_S ;
 - ③ Replace infections (\mathcal{I}_S).
- Re-express using *continuously varying* τ . Process time runs over $[0, T]$.
 - ① For $2nT < \tau < (2n+1)T$, update old \mathcal{R}_S with times in $(0, \tau - 2nT)$;
 - ② For $\tau = (2n+1)T$, resample timelines (not times) of \mathcal{R}_S ;
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- Thus the original update is expressible as a (continuous) composition of updates, each of which satisfies detailed balance in equilibrium.
- The connection “restriction=conditioning” holds (**needs proof**).
- Crucially, re-sampling step 2 ensures composite evolution is irreducible over S ! (So equilibrium under conditioning is unique.)

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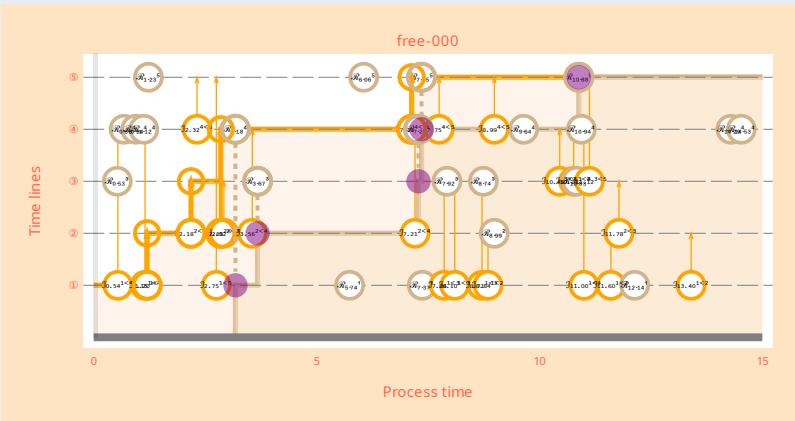
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It is convenient to express the algorithmic update in *continuous* time: applying a given innovation over a continuous-time cycle of length $2T$. The result is no longer reversible, but each individual steps satisfies detailed balance, so the important connection between conditioning and constraint remains valid.

- In the case of discrete algorithmic time, arranging for conditioning would involve horrendous computations. Instead we unroll a “pixel-by-pixel” analysis into continuous algorithmic time.
- Intuitively, we analyze first removals (τ runs over algorithmic time range $(0, T)$) and then infections, (τ runs over algorithmic time range $(T, 2T)$) each working “pixel-by-pixel” along the process time axis.
- It turns out to be convenient to work *forwards in process time* for removals, then *backwards in process time* for infections – and to re-assign random timelines (but not times) for removals in between these two phases (at algorithmic time $\tau = T$).

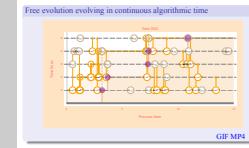
Free evolution evolving in continuous algorithmic time



GIF MP4

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GIF MP4

Conditioning not applied here!

- Start with initial configuration, use immigration-death process of removals to replace old removals. **To avoid premature extinction, condition on no removals in timeline 1!**
- Because we are not yet conditioning, all old removals have now been removed, and all new removals have been added.
- Once all removals have been processed, resample timelines of all removals. Jitter back and forth here to emphasize this important step.
- Now we resample infections, *but this time working backwards through process time*.
- At the end all removals and infections have been removed and replaced by new ones.

4. Conditioning on observed removals

- The trajectory-valued chain is *dynamically reversible*, in *continuous algorithmic time*.
 - Irreducibility is *vital* (otherwise equilibrium depends on starting point). Consequently:
 - conditioned removals must be able to change timeline (but not time of occurrence);
 - Forbidding removal of observed removals, and forbidding creation of new activated removals, yields a modified chain whose invariant probability measure conditions on observed pattern of removals.
- Implications:**
- a removal can be introduced only if it doesn't activate;
 - a conditioned removal timeline can be altered only if it doesn't de-activate;
 - an infection cannot be removed if that action loses a conditioned removal;
 - an infection can be introduced only if no new observed removals result.
- Does this produce a *feasible* and suitably *monotonic* algorithm?
 - Housekeeping details used to establish that monotonicity still works: *laziest feasible epidemic* (LFE) and *no-fly zone* (NFZ).

Initial conditioned epidemic

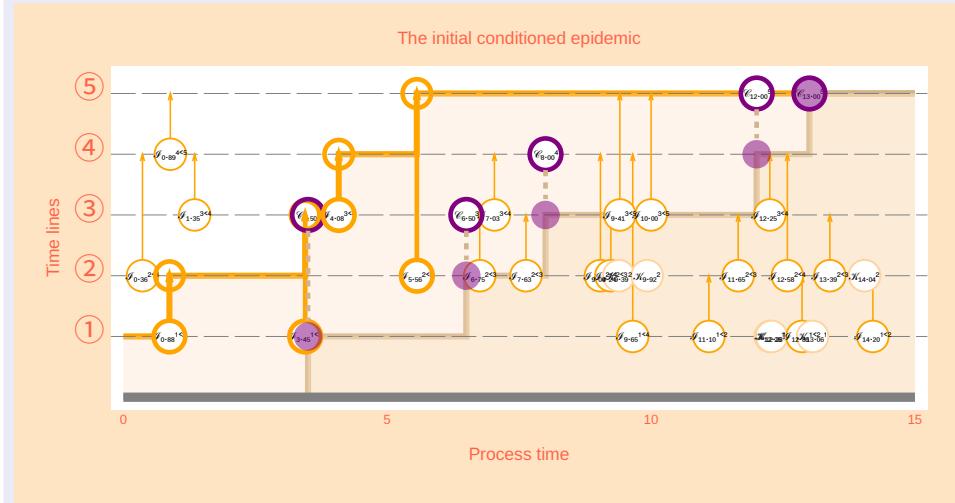


Figure 4: Initial conditioned epidemic, with conditioned removals indicated using purple circles (and purple disks when non-target timelines are infected).

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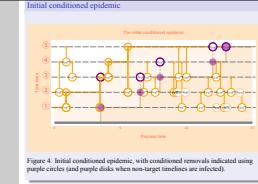
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 - Infection is *vital* (otherwise equilibrium depends on starting point). Consequently:
 - conditioned removals must be able to change timeline (but not time of occurrence);
 - Forbidding removal of observed removals, and forbidding creation of new activated removals, yields a modified chain whose invariant probability measure conditions on observed pattern of removals.
- Implications:
- a removal can be introduced only if it doesn't activate;
 - a conditioned removal timeline can be altered only if it doesn't de-activate;
 - an infection cannot be removed if that action loses a conditioned removal;
 - an infection can be introduced only if no new observed removals result.
- Does this produce a *feasible* and suitably *monotonic* algorithm?
- Housekeeping details used to establish that monotonicity still works: *laziest feasible epidemic* (LFE) and *no-fly zone* (NFZ).

Monotonicity is a key concept here. (Note however that we can still sometimes generate perfect simulation algorithms even when monotonicity does not obtain: we don't need to do this here but should bear it in mind for more general problems in the future.)

- The heart of the matter lies in establishing (some variant of) monotonicity.
- Additionally, for a complicated algorithm such as this one, it is important to test the underlying logic by implementing the algorithm in computer code (which often reveals the falsity of hidden assumptions!).
- LFE and NFZ are useful vocabulary for formulating how this works out, also for arguing for monotonicity, and are also computationally helpful.

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Here is a different initial epidemic, including *conditioned removals*, indicated by purple circles.

- This evolves in algorithmic time as in the unconditioned case (working ‘pixel-by-pixel’), but forbidding any change that breaks conditioning.
- Dealing with removals amounts to removing all inactivated removals and replacing them with a new set of removals, and then resampling the conditioned removal timelines, accepting only those changes which do not de-activate the conditioned removals.
- Dealing with *infections* requires more care.

Conditional epidemic update

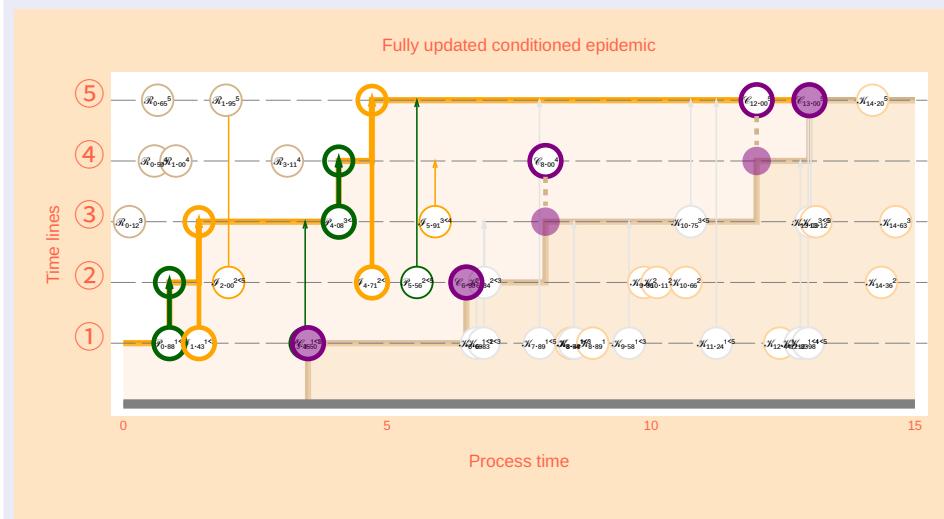
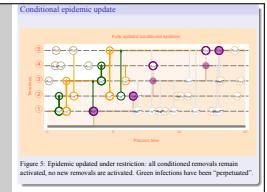


Figure 5: Epidemic updated under restriction: all conditioned removals remain activated, no new removals are activated. Green infections have been “perpetuated”.

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The update proceeds as follows:

1. Accept inactivated removals only if targets avoid infected region.
2. Accept conditioned removals only if targets fall in infected region.
3. Accept new infections only if this doesn't result in infecting an inactivated removal.
4. Retain old infections only if rejection would result in a conditioned removal *not* being infected, in which case retain old infection as *perpetuated* infection.

Options 1 and 2 are directly imposed by the requirement of not changing the set of conditioned removals.

For options 3 and 4 there is a useful correspondence to two recursively-defined geometric structures: the *laziest feasible epidemic* (LFE) and the *no-fly zone* (NFZ).

Laziest feasible epidemic (LFE)

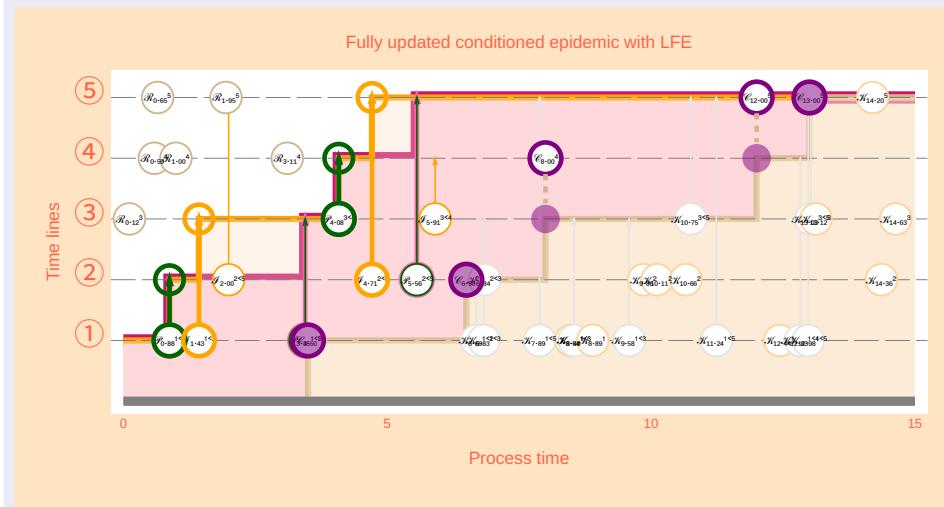
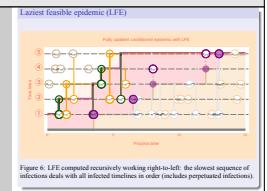


Figure 6: LFE computed recursively working right-to-left: the slowest sequence of infections deals with all infected timelines in order (includes perpetuated infections).

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The LFE is a collection of intervals $[t_j, \infty)$, (j indexing timelines). It is recursively defined, working *backwards* in process time. It is computable at process time t from the updated system of incidents over $[t, \infty)$. It is the latest time t_j of an infection with target in infected region and infectee $j+1$ and

$$\begin{aligned} t_j &\leq t_{j+1}, \text{ and} \\ t_j &\leq \inf\{s : s \text{ is the time of a conditioned removal with target } j\}. \end{aligned}$$

- In the example the LFE boundary differs from the epidemic trajectory, over time intervals $[1.43, 3.45]$, $[4.71, 5.56]$.
- At times 0.88, 3.45, 4.08, 5.56 there are perpetuations (green arrows: an old infection has to be retained to ensure conditioning is not broken); two of these perpetuations (0.88, 4.08) end up forming part of the epidemic trajectory. Note it is in fact possible for a new infection to be part of the LFE but *not* part of the epidemic trajectory.

No-fly zone (NFZ)

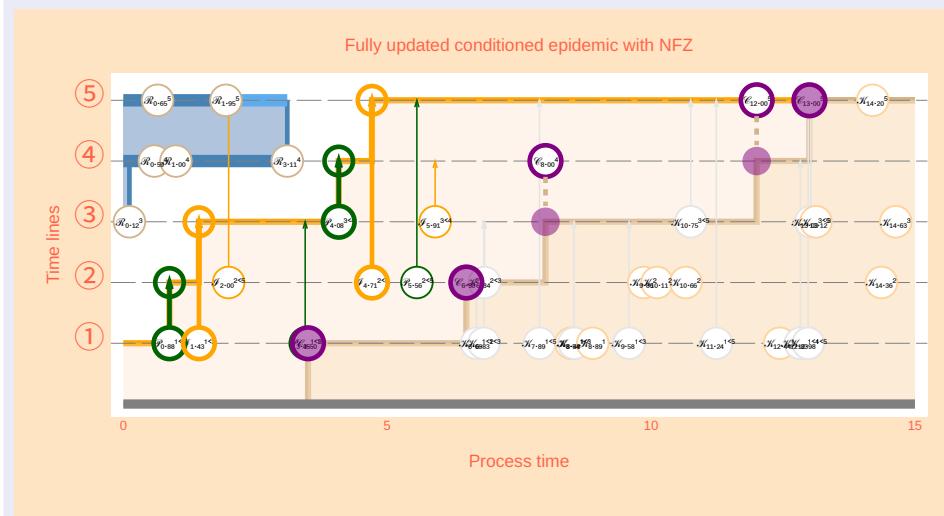
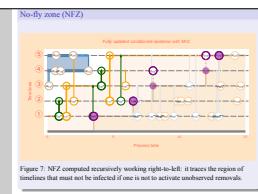


Figure 7: NFZ computed recursively working right-to-left: it traces the region of timelines that must not be infected if one is not to activate unobserved removals.

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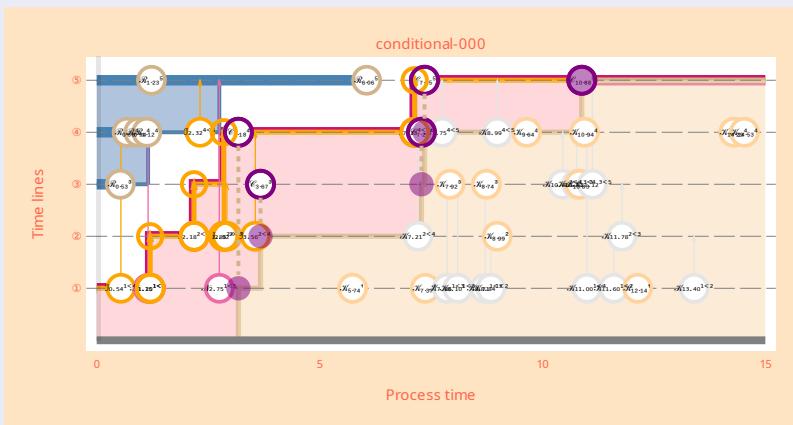
The **NFZ** is used to decide whether to accept proposals of infections targeting the susceptible region:

- ① New infections with targets in the infection zone are assessed to see whether their introduction would activate removals, by connecting the infectious zone to a timeline leading to such an activation (a “no-fly” portion of the timeline);
- ② An infection with target in the susceptible zone may be such that it makes a portion of its timeline *before* its time into “no-fly”.

In this particular case neither of these two mechanisms play a part.

The **NFZ** is evaluated recursively, working backwards in process time, and again is computable at process time t from the updated system of incidents over $[t, \infty)$. Inductive argument shows that **NFZ** and **LFE** depend monotonically on the epidemic trajectory. This implies monotonicity of the algorithmic-time evolution of the conditioned epidemic: hence permitting **CFTP**!

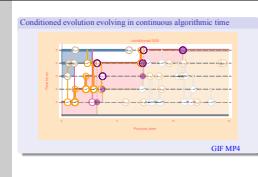
Conditioned evolution evolving in continuous algorithmic time



GIF MP4

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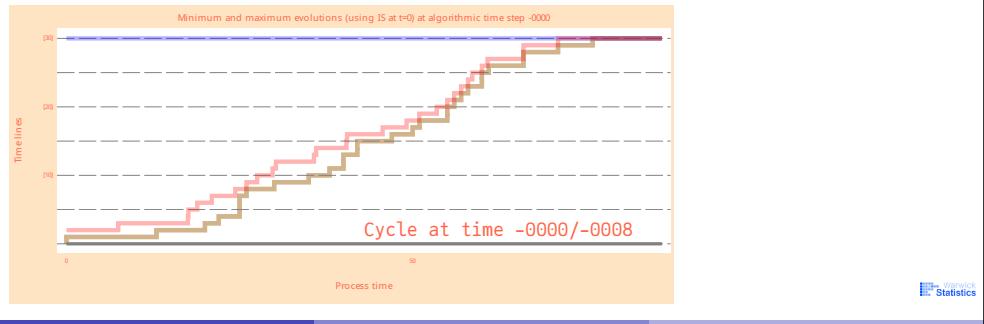


Conditioning applied here!

- Start with initial configuration, use immigration-death process of removals to replace old removals **except** in the infected region.
- All old removals have now been removed, and some new removals have been added.
- Resample timeline of each removal if this does not violate conditioning. Jitter back and forth here.
- Now resample infections, *but this time working backwards through process time*. If addition proposed, add only if pattern of observed removals are unchanged. If deletion proposed, and deletion would change pattern of observed removals, then *perpetuate*.

5. Example

- Smallpox outbreak in a closed community of 120 individuals in Abakaliki, Nigeria (much studied! see page 125 of Bailey, 1975).
- Assume**
 - first observed removal is also the first removal: under a plausible improper prior we can then deduce what is the distribution of infectives I_0 at time 0;
 - all* removals are recorded;
 - no further removals after last observed removal (makes life easier).
- Coding in *julia* (Bezanson *et al.*, 2017), we obtain a **GIF** or an **MP4** of the **perfect simulation** yielding a draw from unobserved pattern of infections.



W S Kendall, S B Connor (Warwick, York)

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27 June 2025

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So what?

- Why this emphasis on unobserved infections given fixed α and β , when we need inference on R-number n/β for *unknown* α and β ?
- Good question. But a re-weighting argument allows us to get (unbiased) estimates based on *different* α and β . The perfect simulation provides exact simulation-based computation to integrate out pattern of unobserved infections.
- So (next steps after SBC & WSK, 2024)**
 - estimate likelihood test statistic for specified α and β ;
 - construct steepest ascent algorithm (in effect, variant of Robbins-Monro stochastic optimization) to find *maximum a posterior* estimates of α and β ;
 - or even, with some computational effort, compute the entire posterior joint density for α and β !
- Finally: can we generalize to other suitable compartment models?

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└ 5. Example

We could vary the assumption concerning the initial and final numbers of infectives while still using essentially the same perfect simulation algorithm! This amounts to presuming some Bayesian prior knowledge.

- How do we allow for the (unobserved) number of initial infectives being unobserved?
- Use an independence sampler: draw from the (unconditioned) number of initial infectives, accept if this leads to observed removals exactly at the list of conditioned removals.
- (One can alternatively employ a random-walk Metropolis sampler, if the prior for the initial number of infectives is suitably monotonic.)

Technical note:

The language *julia* (Bezanson *et al.*, 2017), is chosen because it allows for rapid development by using an expressive type-based syntax (very useful for involved algorithms), and can deliver remarkably fast execution using very good “just in time” compilation techniques.

5. Example

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- Finally: can we generalize to other suitable compartment models?

In effect, perfect simulation supplies a well-behaved stochastic integration mechanism, reducing an MCMC algorithm to use a technique equivalent to a much more amenable Monte Carlo calculation. Other compartment models presenting varying degrees of challenge: a preliminary discussion is given in an appendix.

- ① Split population into a small number of interacting sub-populations;
- ② (For purposes of insight even if impractical.) Treat each individual as a sub-population on its own;
- ③ Allow parameters to change at specific times of day or week;
- ④ Allow for some activated removals to be unobserved!

Conclusion

- If MCMC burn-in is a concern, try to build a perfect simulation!
- CFTP works even for significantly complex and relevant models of real-life phenomena.
- Of course detailed models resist perfect simulation (but it will be helpful to compare with a simpler model using fewer parameters).
- Still to be done: seek faster CFTP; statistical estimation of parameters, generalization to other compartment models.
- Thank you for your attention! **QUESTIONS?**



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└ Conclusion

There is much still to be done!

At the Warwick talk in January 2025, Paul Jenkins asked whether this might be related to the “look-down” argument in population genetics (**Donnelly & Kurtz, 1996**)? My provisional answer, after recalling Donnelly & Kurtz (1996) later in the day, is:

as far as I can tell, the “look-down” argument is related in the sense that it also involves a particle model as discussed in Liggett (1985). However I have not yet found a closer connection.

Conclusion

- If MCMC burn-in is a concern, try to build a perfect simulation!
- CFTP works even for significantly complex and relevant models of real-life phenomena.
- Of course detailed models resist perfect simulation (but it will be helpful to compare with a simpler model using fewer parameters).
- Still to be done: seek faster CFTP; statistical estimation of parameters, generalization to other compartment models.
- Thank you for your attention! **QUESTIONS?**



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Image information

| Image | Attribution |
|--|---|
| Terry Pratchett Classic CFTP for a simple random walk | Luigi Novi Result of code written by WSK |
| Diamond Princess Epidemic CFTP images and animation | Alpsdale Result of code written by WSK |
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Previous instances of this talk

| Date | Title | Location |
|----------|-------------------------|-----------------------------------|
| 19/04/24 | Perfect Epidemics | Short Research Talk |
| 15/05/24 | McMC+Perfect Simulation | Graduate Seminar, Aristotle Univ. |
| 17/01/25 | Perfect Epidemics | Applied Probability Seminar |
| 27/06/25 | Perfect Epidemics | UK Research Network Stochastics |

Appendix A: A “near-maximal” configuration

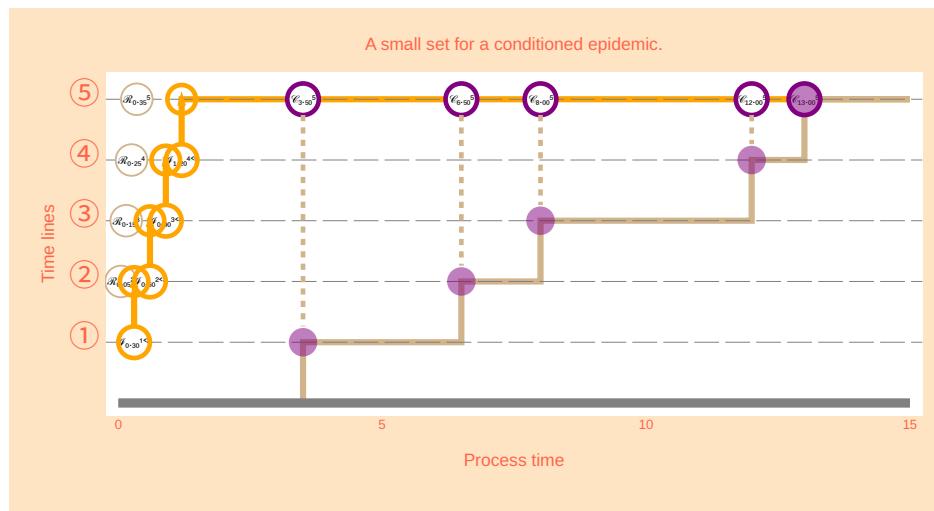
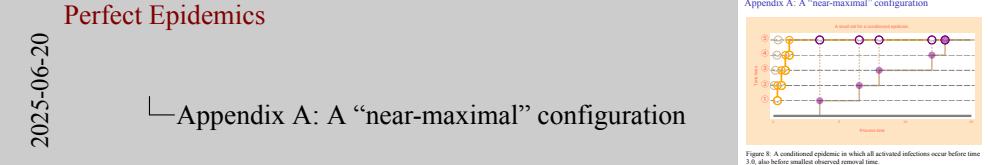


Figure 8: A conditioned epidemic in which all activated infections occur before time 3.0, also before smallest observed removal time.



- ① For a “near-maximal configuration”, we set relevant infections to occur in order on or before a fixed small time $\delta = 1.5 > 0$, where 2δ is smaller than any of the conditioned removal times.
- ② All inactivated removals have to occur before their respective infections (in order to avoid activation).
- ③ Variants on this construction can be used to show that CFTP for the perfect epidemic must coalesce in finite time;
- ④ However to get a sensible stochastic bound on coalescence time would require a lot more work!

Appendix B: Notes towards a monotonicity proof

Summary of plan of proof:

- ➊ Let $EPI_{\tau=0}^{\pm}$ represent two epidemic trajectories (\pm) at algorithmic time $\tau=0$, viewed as subsets of “timeline-space” $\{(k, [0, T)) : k = 1, 2, \dots\}$.
- ➋ Suppose at algorithmic time $\tau=0$ the *fast* $EPI_{\tau=0}^+$ is never later than the *slow* $EPI_{\tau=0}^-$ so $EPI_{\tau=0}^+ \supseteq EPI_{\tau=0}^-$; additionally suppose monotonicity holds for conditional removal marks: if $C_{\tau=0}^{\pm}$ are conditional removals at fixed process time t then $C_{\tau=0}^+ \text{ timeline} \geq C_{\tau=0}^- \text{ timeline}$.
- ➌ Then a related monotonicity holds for the laziest feasible epidemics: $LFE_{\tau=1}^+ \leq LFE_{\tau=1}^-$ at algorithmic time $\tau=1$.
- ➍ Likewise a similar monotonicity (but reversing the set-theoretic inclusion!) holds for no-fly zones: $NFZ_{\tau=1}^+ \subseteq NFZ_{\tau=1}^-$.
- ➎ Now prove $EPI_{\tau=1}^+ \supseteq EPI_{\tau=1}^-$ moreover if $C_{\tau=1}^+$ matches $C_{\tau=1}^-$ at process time t then $C_{\tau=1}^+ \text{ timeline} \geq C_{\tau=1}^- \text{ timeline}$.

$LFE_{\tau=1}^+$: recursive construction

Let $LFE_{\tau=1}^{\pm}(k)$ be the (process) time of the latest infection of timeline k needed if all C^{\pm} s of $EPI_{\tau=1}^{\pm}$ are to be infected.

- ➊ For the top timeline n , $LFE_{\tau=1}^{\pm}(n)$ must precede any C^{\pm} on timeline n ; set $LFE_{\tau=1}^{\pm}(n) = T$ if no such C^{\pm} .
- ➋ For $k < n$ with $LFE_{\tau=1}^{\pm}(k+1) = T$, again $LFE_{\tau=1}^{\pm}(k)$ must precede any C^{\pm} on timeline k ; set $LFE_{\tau=1}^{\pm}(k) = T$ if no such C^{\pm} .
- ➌ Suppose n_0 is largest k with $LFE_{\tau=1}^{\pm}(k) < T$. Working downwards through $\ell = n_0 - 1, \dots, 1$, $LFE_{\tau=1}^{\pm}(\ell)$ is the time of the latest infection targeting $\ell + 1$ and based in the infected region such that
 - ➌(a) $LFE_{\tau=1}^{\pm}(\ell) \leq LFE_{\tau=1}^{\pm}(\ell + 1)$;
 - ➌(b) $LFE_{\tau=1}^{\pm}(\ell)$ precedes any C^{\pm} on timeline ℓ .
- ➍ Equivalently, $LFE_{\tau=1}^{\pm}(\ell)$ is the time of the latest potential infection targeting $\ell + 1$ such that
 - ➍(a) $LFE_{\tau=1}^{\pm}(\ell) \leq LFE_{\tau=1}^{\pm}(\ell + 1)$;
 - ➍(b) $LFE_{\tau=1}^{\pm}(\ell)$ precedes any C^{\pm} on timelines $\ell, \ell + 1, \dots, n$.

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└ Appendix B: Notes towards a monotonicity proof

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| Appendix B: Notes towards a monotonicity proof Summary of plan of proof: <ul style="list-style-type: none"> ➊ Let $EPI_{\tau=0}^{\pm}$ represent two epidemic trajectories (\pm) at algorithmic time $\tau=0$, viewed as subsets of “timeline-space” $\{(k, [0, T)) : k = 1, 2, \dots\}$ ➋ Suppose at algorithmic time $\tau=0$ the <i>fast</i> $EPI_{\tau=0}^+$ is never later than the <i>slow</i> $EPI_{\tau=0}^-$ so $EPI_{\tau=0}^+ \supseteq EPI_{\tau=0}^-$; additionally suppose monotonicity holds for conditional removal marks: if $C_{\tau=0}^{\pm}$ are conditional removals at fixed process time t then $C_{\tau=0}^+ \text{ timeline} \geq C_{\tau=0}^- \text{ timeline}$. ➌ Then a related monotonicity holds for the laziest feasible epidemics: $LFE_{\tau=1}^+ \leq LFE_{\tau=1}^-$ at algorithmic time $\tau=1$. ➍ Likewise a similar monotonicity (but reversing the set-theoretic inclusion!) holds for no-fly zones: $NFZ_{\tau=1}^+ \subseteq NFZ_{\tau=1}^-$. ➎ Now prove $EPI_{\tau=1}^+ \supseteq EPI_{\tau=1}^-$ moreover if $C_{\tau=1}^+$ matches $C_{\tau=1}^-$ at process time t then $C_{\tau=1}^+ \text{ timeline} \geq C_{\tau=1}^- \text{ timeline}$. |
|---|

- ➊ Expressing epidemics in terms of subsets of “timeline-space” (corresponding infected regions) eases discussion of monotonicity. We use EPI_{τ}^{\pm} , LFE_{τ}^{\pm} et cetera to refer simultaneously to fast (+) and slow (-) entities at algorithmic time τ .
- ➋ The timeline monotonicity for conditional removals is important!
- ➌ The LFE are viewed as non-decreasing sequences of infection times. Monotonicity for LFE follows by a relatively straightforward recursive argument. **Notation:** $LFE(k)$ is the intersection of LFE with timeline k .
- ➍ Monotonicity for NFZ requires analysis that is slightly more involved.
- ➎ The $LFE_{\tau=1}$ and $NFZ_{\tau=1}$ monotonicities depend only on monotonicity for the preceding $EPI_{\tau=0}$; monotonicity for $EPI_{\tau=1}$ uses these plus monotonicity for $EPI_{\tau=0}$ to show monotonicity for $EPI_{\tau=1}$.

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| LFE_{τ=1}[±]: recursive construction <p>Let $LFE_{\tau=1}^{\pm}(k)$ be the (process) time of the latest infection of timeline k needed if all C^{\pm}s of $EPI_{\tau=1}^{\pm}$ are to be infected.</p> <ul style="list-style-type: none"> ➊ For the top timeline n, $LFE_{\tau=1}^{\pm}(n)$ must precede any C^{\pm} on timeline n; set $LFE_{\tau=1}^{\pm}(n) = T$ if no such C^{\pm}. ➋ For $k < n$ with $LFE_{\tau=1}^{\pm}(k+1) = T$, again $LFE_{\tau=1}^{\pm}(k)$ must precede any C^{\pm} on timeline k; set $LFE_{\tau=1}^{\pm}(k) = T$ if no such C^{\pm}. ➌ Suppose n_0 is largest k with $LFE_{\tau=1}^{\pm}(k) < T$. Working downwards through $\ell = n_0 - 1, \dots, 1$, $LFE_{\tau=1}^{\pm}(\ell)$ is the time of the latest infection targeting $\ell + 1$ and based in the infected region such that <ol style="list-style-type: none"> ➌(a) $LFE_{\tau=1}^{\pm}(\ell) \leq LFE_{\tau=1}^{\pm}(\ell + 1)$; ➌(b) $LFE_{\tau=1}^{\pm}(\ell)$ precedes any C^{\pm} on timeline ℓ. ➍ Equivalently, $LFE_{\tau=1}^{\pm}(\ell)$ is the time of the latest potential infection targeting $\ell + 1$ such that <ol style="list-style-type: none"> ➍(a) $LFE_{\tau=1}^{\pm}(\ell) \leq LFE_{\tau=1}^{\pm}(\ell + 1)$; ➍(b) $LFE_{\tau=1}^{\pm}(\ell)$ precedes any C^{\pm} on timelines $\ell, \ell + 1, \dots, n$. |
|--|

- ➊ For the top timeline, we need only be sure that any C s on this time line are activated. If there are no such C s then we can set $LFE_{\tau=1}^{\pm}(n) = T$.
- ➋ We apply similar reasoning working down through the timelines k : we can continue setting $LFE_{\tau=1}^{\pm}(k) = T$ until we encounter a C .
- ➌ After that, timeline ℓ must be infected early enough to insure:
 - ➌(a) infection of the previous $LFE_{\tau=1}^{\pm}(\ell+1) < T$ (there must be an early enough infection to activate the LFE for timeline $\ell+1$), as well as
 - ➌(b) infection of any C s on the current timeline ℓ .
- ➍ Expanding “any C^{\pm} on timeline ℓ ” to “any C^{\pm} on timelines $\ell, \ell+1, \dots, n$ ” doesn’t affect $LFE_{\tau=1}^{\pm}(\ell)$, since “any C^{\pm} on timelines $\ell+1, \ell+2, \dots, n$ ” will be subsequent to $LFE_{\tau=1}^{\pm}(\ell+1) \geq LFE_{\tau=1}^{\pm}(\ell)$.

LFE _{$\tau=1$} : monotonicity

- ① Re-sample \mathcal{C}^\pm timelines by accept-reject: same proposals for both \pm . As $EPI_{\tau=0}^+ \supseteq EPI_{\tau=0}^-$, so \mathcal{C} timelines for $EPI_{\tau=1}^+$ no lower than for $EPI_{\tau=1}^-$.
- ② In particular, if $LFE_{\tau=1}(k) = T$ then $LFE_{\tau=1}(k) = T$ also.
- ③ Now work inductively. Suppose monotonicity holds for $k+1, \dots, n$. Then $LFE_{\tau=1}^+(k) \leq LFE_{\tau=1}^+(k+1) \leq LFE_{\tau=1}^-(k+1)$. But the set of “times of \mathcal{C}^- on timelines $\ell, \ell+1, \dots, n$ ” is a subset of the set of “times of \mathcal{C}^+ on timelines $\ell, \ell+1, \dots, n$ ”. So if b_k^\pm is the resulting right-constraint on $LFE_{\tau=1}^\pm(k)$ then $b_k^+ \leq b_k^-$.
- ④ Suppose $EPI_{\tau=0}^\pm$ infects timeline $k+1$ at time $a_k^\pm: a_k^+ \leq a_k^- \leq b_k^-$ by monotonicity for $EPI_{\tau=0}^\pm$. If no $\tau=1$ infections infect timeline $k+1$ in $[a_k^\pm, b_k^\pm]$, then $LFE_{\tau=1}^\pm(k)$ perpetuates a_k^\pm using $EPI_{\tau=0}^\pm(k)$. Then argue case-by-case:
 - (a) no perpetuation occurs (use fact, all infections are shared);
 - (b) $LFE_{\tau=1}^-(k)$ is perpetuated (so no useful infections after perpetuation);
 - (c) only $LFE_{\tau=1}^+(k)$ is perpetuated (then use $\tau=0$ monotonicity).

NFZ _{$\tau=1$} : iterative construction

- ① Set $NFZ_{\tau=1}^{\pm,*}$ to be union of regions $(k, [0, t])$ for all \mathcal{R} s of $EPI_{\tau=1}^\pm$, for timeline k and time t of \mathcal{R} . Set $NFZ_{\tau=1}^{\pm,*} = \{(k, [0, t_k^*]) : t_k^* > 0\}$.
- ② Set $NFZ_{\tau=1}^{\pm,0} = \{(k, [0, t_k]) : t_k > 0\}$ as monotonic envelope of $NFZ_{\tau=1}^{\pm,*}: \{t_k\}$ is smallest non-decreasing sequence majorizing $\{t_k^*\}$.
- ③ Work backwards through new “non-removed” \mathcal{J} s. At step j , time t ,
 - (a) **accept** \mathcal{J} if it targets $NFZ_{\tau=1}^{\pm,j-1}$ at timeline k but infection fails: set $NFZ_{\tau=1}^{\pm,j} = NFZ_{\tau=1}^{\pm,j-1} \cup \{(k-1, [0, t])\}$; otherwise set $NFZ_{\tau=1}^{\pm,j} = NFZ_{\tau=1}^{\pm,j-1}$ and
 - (b) **reject** \mathcal{J} if it would infect part of $NFZ_{\tau=1}^{\pm,j-1}$;
 - (c) **accept** \mathcal{J} if it doesn’t target $NFZ_{\tau=1}^{\pm,j-1}$.
- ④ Set $NFZ_{\tau=1}^\pm = NFZ_{\tau=1}^{\pm,j}$ if a total of j new \mathcal{J} s are proposed for $EPI_{\tau=1}^\pm$, so no more \mathcal{J} s remain!

NB: ignore \mathcal{J} proposals targeting $NFZ_{\tau=1}^{\pm,j-1}$: either these are rejected ((b) above) or $NFZ_{\tau=1}^{\pm,j}$ is modified ((a) above) so they aren’t relevant! *Relevant* accepted \mathcal{J} s are exactly those *not* targeting the final $NFZ_{\tau=1}^\pm$.

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LFE _{$\tau=1$} : monotonicity

- ➊ Re-sample \mathcal{C}^\pm timelines by accept-reject: same proposals for both \pm . As $EPI_{\tau=0}^+ \supseteq EPI_{\tau=0}^-$, so \mathcal{C} timelines for $EPI_{\tau=1}^+$ no lower than for $EPI_{\tau=1}^-$.
- ➋ In particular, if $LFE_{\tau=1}^+(k) = T$ then $LFE_{\tau=1}^-(k) = T$ also.
- ➌ Now work inductively. Suppose monotonicity holds for $k+1, \dots, n$. Then $LFE_{\tau=1}^+(k) \leq LFE_{\tau=1}^+(k+1) \leq LFE_{\tau=1}^-(k+1)$. But the set of “times of \mathcal{C}^+ on timelines $\ell, \ell+1, \dots, n$ ” is a subset of the set of “times of \mathcal{C}^+ on timelines $\ell, \ell+1, \dots, n$ ”. So if b_k^\pm is the resulting right-constraint on $LFE_{\tau=1}^\pm(k)$ then $b_k^+ \leq b_k^-$.
- ➍ Suppose $EPI_{\tau=0}^\pm$ infects timeline $k+1$ at time $a_k^\pm: a_k^+ \leq a_k^- \leq b_k^-$ by monotonicity for $EPI_{\tau=0}^\pm$. If no $\tau=1$ infections infect timeline $k+1$ in $[a_k^\pm, b_k^\pm]$, then $LFE_{\tau=1}^\pm(k)$ perpetuates a_k^\pm using $EPI_{\tau=0}^\pm(k)$. Then argue case-by-case:
 - (a) no perpetuation occurs (use fact, all infections are shared);
 - (b) $LFE_{\tau=1}^-(k)$ is perpetuated (so no useful infections after perpetuation);
 - (c) only $LFE_{\tau=1}^+(k)$ is perpetuated (then use $\tau=0$ monotonicity).

- ➊ Proposals are accepted only when higher than relevant infected region. Proposals *more easily* accepted for fast epidemic ($EPI_{\tau=0}^+ \supseteq EPI_{\tau=0}^-$).
- ➋ Suppose timelines $k, k+1, \dots, n$ are clear of \mathcal{C}^+ s. Then same true of \mathcal{C}^- s, whose timelines are no higher than corresponding \mathcal{C}^+ s.
- ➌ The subset argument follows because \mathcal{C} s in EPI^+ are never on lower timelines than corresponding \mathcal{C} s in EPI^- .
- ➍ The arguments here are case-by-case, using $a_k^+ \leq a_k^-$ and $b_k^+ \leq b_k^-$:
 - (a) no perpetuation: same potential infections so monotonicity!
 - (b) perpetuation at $LFE_{\tau=1}^-(k)$: no infections between $LFE_{\tau=1}^-(k)$ and b_k^- , thus forcing monotonicity;
 - (c) perpetuation only at $LFE_{\tau=1}^+(k)$: so $LFE_{\tau=1}^-(k)$ arises by infection, so has to lie in $(b_k^+, b_k^-]$, so forcing monotonicity:

$$LFE_{\tau=1}^-(k) \geq EPI_{\tau=0}^-(k) \geq EPI_{\tau=0}^+(k) = LFE_{\tau=1}^+(k).$$

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NFZ _{$\tau=1$} : iterative construction

- ➊ Set $NFZ_{\tau=1}^{\pm,t}$ to be union of regions $(k, [0, t])$ for all \mathcal{R} s of $EPI_{\tau=1}^\pm$, for timeline k and time t of \mathcal{R} . Set $NFZ_{\tau=1}^{\pm,t} = \{(k, [0, t]) : t > 0\}$.
- ➋ Set $NFZ_{\tau=1}^{\pm,0} = \{(k, [0, t_k]) : t_k > 0\}$ as monotonic envelope of $NFZ_{\tau=1}^{\pm,t}$.
- ➌ While $NFZ_{\tau=1}^{\pm,t}$ is not empty, propose new \mathcal{J} s. At step j , time t ,
 - (a) accept \mathcal{J} if it targets $NFZ_{\tau=1}^{\pm,j-1}$ at timeline k but infection fails: set $NFZ_{\tau=1}^{\pm,j} = NFZ_{\tau=1}^{\pm,j-1} \cup \{(k-1, [0, t])\}$;
 - (b) reject \mathcal{J} if it would infect part of $NFZ_{\tau=1}^{\pm,j-1}$;
 - (c) accept \mathcal{J} if it doesn’t target $NFZ_{\tau=1}^{\pm,j-1}$.
- ➍ Set $NFZ_{\tau=1}^\pm = NFZ_{\tau=1}^{\pm,j}$ if a total of j new \mathcal{J} s are proposed for $EPI_{\tau=1}^\pm$, so no more \mathcal{J} s remain!

NB: ignore \mathcal{J} proposals targeting $NFZ_{\tau=1}^{\pm,j-1}$: either these are rejected ((b) above) or $NFZ_{\tau=1}^{\pm,j}$ is modified ((a) above) so they aren’t relevant! Relevant accepted \mathcal{J} s are exactly those targeting the final $NFZ_{\tau=1}^\pm$.

Note: \mathcal{R} s of $EPI_{\tau=1}^\pm$ are all determined *before* considering new \mathcal{J} s!

- ➊ Infections proposed for $EPI_{\tau=1}^\pm$ are rejected if they would infect part of $NFZ_{\tau=1}^{\pm,*}$. We reject proposals directly infects part of $NFZ_{\tau=1}^{\pm,*}$, but must also reject if *indirect* chains of infection arise ...
- ➋ The extension to $NFZ_{\tau=1}^{\pm,0}$ is a **short-cut**, justified because an \mathcal{J} can infect its target if and only (b) it is based in the infected region and (b) the timeline immediately below the target timeline is infected at the relevant time. So no direct infection of $NFZ_{\tau=1}^{\pm,0}$ by $EPI_{\tau=0}^\pm$ is possible.
- ➌ $NFZ_{\tau=1}^{\pm,j-1}$ is changed to $NFZ_{\tau=1}^{\pm,j} = NFZ_{\tau=1}^{\pm,j-1} \cup \{(k-1, [0, t])\}$ (extending k^{th} timeline to $[0, t]$) only if \mathcal{J} hits $NFZ_{\tau=1}^{\pm,j-1}$ at target timeline k but *either* timeline $k-1$ is *not* currently infected at t *or* \mathcal{J} is *not* based in the current infected region.

(Note: infected subregion of time interval $[0, t]$ agrees with that for $EPI_{\tau=0}^\pm$ if no new \mathcal{J} s prior to t have yet been considered!)

NFZ_{τ=1}: monotonicity

Establish monotonicity for NFZ_{τ=1}^{±,*}, NFZ_{τ=1}^{±,0}, NFZ_{τ=1}^{±,1}, NFZ_{τ=1}^{±,2}, ... in turn:

- ① Since EPI_{τ=0}⁺ ⊇ EPI_{τ=0}⁻ and the set of \mathcal{R} s for EPI_{τ=1}[±] are formed by intersecting the same \mathcal{R} pattern with the complements of EPI_{τ=0}[±], it follows that NFZ_{τ=1}^{+,*} ⊆ NFZ_{τ=1}^{-,*}.
 - ② Monotonicity for NFZ_{τ=1}^{±,0} is a direct consequence.
 - ③ Given NFZ_{τ=1}^{+,j-1} ⊆ NFZ_{τ=1}^{-,j-1}, create NFZ_{τ=1}^{±,j} by proposing \mathcal{J} at time t targeting timeline k , based in EPI_{τ=0}⁺ infected region. Then $NFZ_{\tau=1}^{+,j} = NFZ_{\tau=1}^{+,j-1} \cup \{(k-1, [0, t])\}$ exactly when \mathcal{J} fails to infect in EPI_{τ=1}⁺. Then
 - (a) we know k timeline at t is in $NFZ_{\tau=1}^{-,j-1} \supseteq NFZ_{\tau=1}^{+,j-1}$;
 - (b) infection fails for EPI_{τ=1}⁺ because timeline $k-1$ is not infected at t in EPI_{τ=0}⁺. But we know EPI_{τ=0}⁺ ⊇ EPI_{τ=0}⁻, so timeline $k-1$ is not infected at t in EPI_{τ=0}⁻ either. So infection in EPI_{τ=1}⁻ also fails.
- Thus $NFZ_{\tau=1}^{+,j} = NFZ_{\tau=1}^{+,j-1} \cup \{(k-1, [0, t])\}$ implies $NFZ_{\tau=1}^{-,j} = NFZ_{\tau=1}^{-,j-1} \cup \{(k-1, [0, t])\}$ and so $NFZ_{\tau=1}^{+,j} \subseteq NFZ_{\tau=1}^{-,j}$.

EPI_{τ=1}: monotonicity (I)

Consider:

- there is epidemic monotonicity at algorithmic time $\tau=0$ ($EPI_{\tau=0}^+ \supseteq EPI_{\tau=0}^-$, also \mathcal{C} s in $EPI_{\tau=0}^+$ are never lower than in $EPI_{\tau=0}^-$);
- at algorithmic time $\tau=1$ there is monotonicity of laziest feasible epidemic ($LFE_{\tau=1}^+(k) \leq LFE_{\tau=1}^-(k)$ for all timelines k);
- and there is monotonicity of no-fly zone ($NFZ_{\tau=1}^+ \subseteq NFZ_{\tau=1}^-$).

Deduce epidemic monotonicity at algorithmic time $\tau=1$

$(EPI_{\tau=1}^+ \supseteq EPI_{\tau=1}^-)$.

Two cases to consider. Use ∂NFZ to represent right-most boundary for a NFZ, and similarly use ∂EPI to represent left-most boundary for a EPI):

- ① $\partial NFZ_{\tau=1}^+ \leq LFE_{\tau=1}^+ \leq \partial NFZ_{\tau=1}^- \leq LFE_{\tau=1}^-$;
- ② $\partial NFZ_{\tau=1}^+ \leq \partial NFZ_{\tau=1}^- \leq LFE_{\tau=1}^+ \leq LFE_{\tau=1}^-$;

In the first case there is nothing to be done: simply use the remark

$$\partial NFZ_{\tau=1}^\pm \leq \partial EPI_{\tau=1}^\pm \leq LFE_{\tau=1}^\pm.$$

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| NFZ _{τ=1} ^{-,*} : monotonicity |
| Establish monotonicity for NFZ _{τ=1} ^{+,*} , NFZ _{τ=1} ⁰ , NFZ _{τ=1} ¹ , NFZ _{τ=1} ² , ... in turn: |
| Since EPI _{τ=0} ⁺ ⊇ EPI _{τ=0} ⁻ and the set of \mathcal{R} s for EPI _{τ=1} [±] are formed by intersecting the same \mathcal{R} pattern with the complements of EPI _{τ=0} [±] , it follows that NFZ _{τ=1} ^{+,*} ⊆ NFZ _{τ=1} ^{-,*} . |
| Monotonicity for NFZ _{τ=1} ⁰ is a direct consequence. |
| Given NFZ _{τ=1} ^{+,j-1} ⊆ NFZ _{τ=1} ^{-,j-1} , create NFZ _{τ=1} ^{±,j} by proposing \mathcal{J} at time t targeting timeline k , based in EPI _{τ=0} ⁺ infected region. Then $NFZ_{\tau=1}^{+,j} = NFZ_{\tau=1}^{+,j-1} \cup (k-1, [0, t])$ exactly when \mathcal{J} fails to infect in EPI _{τ=1} ⁺ . Then |
| we know timeline t is in $NFZ_{\tau=1}^{-,j-1} \supseteq NFZ_{\tau=1}^{+,j-1}$. |
| infection fails for EPI _{τ=1} ⁺ , because timeline $k-1$ is not infected at t in EPI _{τ=0} ⁺ . But we know EPI _{τ=0} ⁺ ⊇ EPI _{τ=0} ⁻ , so timeline $k-1$ is not infected at t in EPI _{τ=0} ⁻ either. So infection in EPI _{τ=1} ⁻ also fails. |
| Thus $NFZ_{\tau=1}^{+,j} = NFZ_{\tau=1}^{+,j-1} \cup (k-1, [0, t])$ implies $NFZ_{\tau=1}^{-,j} = NFZ_{\tau=1}^{-,j-1} \cup (k-1, [0, t])$ and so $NFZ_{\tau=1}^{+,j} \subseteq NFZ_{\tau=1}^{-,j}$. |

It remains important that the infected region over time interval $[0, t)$ agrees with that for EPI_{τ=0}⁺ if no new \mathcal{J} s prior to t have yet been considered!

- ④ Monotonicity of NFZ_{τ=1}^{±,*} is immediate.
- ⑤ For NFZ_{τ=1}^{±,0} use monotonicity of monotonic envelope operation.
- ⑥ is an argument using induction.
 - (a) Use $NFZ_{\tau=1}^{+,j-1} \subseteq NFZ_{\tau=1}^{-,j-1}$.
 - (b) Use the note above, concerning agreement of initial segments of epidemics, to see that the epidemic monotonicity up to time t carries over from EPI_{τ=0}⁺ to the set-up when considering the \mathcal{J} proposal at time t .

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| EPI _{τ=1} ⁺ : monotonicity (I) |
| Consider: |
| • there is epidemic monotonicity at algorithmic time $\tau=0$ ($EPI_{\tau=0}^+ \supseteq EPI_{\tau=0}^-$, also \mathcal{C} s in $EPI_{\tau=0}^+$ are never lower than in $EPI_{\tau=0}^-$); |
| • at algorithmic time $\tau=1$ there is monotonicity of laziest feasible epidemic ($LFE_{\tau=1}^+(k) \leq LFE_{\tau=1}^-(k)$ for all timelines k); |
| • and there is monotonicity of no-fly zone ($NFZ_{\tau=1}^+ \subseteq NFZ_{\tau=1}^-$). |
| Deduce epidemic monotonicity at algorithmic time $\tau=1$ ($EPI_{\tau=1}^+ \supseteq EPI_{\tau=1}^-$). |
| Two cases to consider. Use ∂NFZ to represent right-most boundary for a NFZ, and similarly use ∂EPI to represent left-most boundary for a EPI): |
| • $\partial NFZ_{\tau=1}^+ \leq LFE_{\tau=1}^+ \leq \partial NFZ_{\tau=1}^- \leq LFE_{\tau=1}^-$; |
| • $\partial NFZ_{\tau=1}^+ \leq \partial NFZ_{\tau=1}^- \leq LFE_{\tau=1}^+ \leq LFE_{\tau=1}^-$; |
| In the first case there is nothing to be done: simply use the remark |
| $\partial NFZ_{\tau=1}^\pm \leq \partial EPI_{\tau=1}^\pm \leq LFE_{\tau=1}^\pm$. |

⚠ Warning

Need to verify some “obvious” facts:

- ① NFZ dominance over LFE: $\partial NFZ_{\tau=1}^\pm \leq LFE_{\tau=1}^\pm$;
- ② EPI sandwiching: $\partial NFZ_{\tau=1}^\pm \leq \partial EPI_{\tau=1}^\pm \leq LFE_{\tau=1}^\pm$.

EPI _{$\tau=1$} : monotonicity (II)

In the second case argue as follows. Localize to particular timeline k :

- ① If the EPI _{$\tau=1$} ⁻(k) infection is perpetuated, it must agree with LFE _{$\tau=1$} ⁻(k) and so monotonicity follows.
- ② If the EPI _{$\tau=1$} ⁻(k) infection is no earlier than LFE _{$\tau=1$} ⁺(k) then again monotonicity follows.
- ③ If the EPI _{$\tau=1$} ⁻(k) infection is not perpetuated and occurs earlier than LFE _{$\tau=1$} ⁺(k) then it is available as a possible candidate for EPI _{$\tau=1$} ⁺(k) and so here too EPI _{$\tau=1$} ⁻(k) is no earlier than EPI _{$\tau=1$} ⁺(k).

This completes the proof of monotonicity for EPI _{$\tau=1$} .

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EPI _{$\tau=1$} : monotonicity (II)
In the second case argue as follows. Localize to particular timeline k :
• If the EPI _{$\tau=1$} ⁻(k) infection is perpetuated, it must agree with LFE _{$\tau=1$} ⁻(k) and so monotonicity follows.
• If the EPI _{$\tau=1$} ⁻(k) infection is no earlier than LFE _{$\tau=1$} ⁺(k) then again monotonicity follows.
• If the EPI _{$\tau=1$} ⁻(k) infection is not perpetuated and occurs earlier than LFE _{$\tau=1$} ⁺(k) then it is available as a possible candidate for EPI _{$\tau=1$} ⁺(k) and so here too EPI _{$\tau=1$} ⁻(k) is no earlier than EPI _{$\tau=1$} ⁺(k).
This completes the proof of monotonicity for EPI _{$\tau=1$} .

⚠ Warning

Verify the following:

- ① Need explicit proof: perpetuated infections must be part of LFE;
- ② Use EPI sandwiching again.
- ③ In fact EPI _{$\tau=1$} avoids NFZ _{$\tau=1$} ⁻ and therefore NFZ _{$\tau=1$} ⁺; the portion earlier than LFE _{$\tau=1$} ⁺(k) provides a possible epidemic path for EPI _{$\tau=1$} ⁺ and therefore should be no earlier than EPI _{$\tau=1$} ⁺ at any timeline level.

💡 Tip

It should be possible to extend all this to cases where EPI⁺ has **more** initial infectives than EPI⁻: important when varying initial condition!

Appendix C: Naïve approach to compartment models fails

- Consider a modification of this approach to the case of compartmentalized populations.
- Focus on the extreme case in which every individual j has infectivity (outgoing to individual k) $\alpha_{j,k}$ and removal parameter β_j .
- Suppose the conditioning on removals is specifically about named individuals j being removed at specified times r_j ; suppose also there are no “occult” (unobserved) removals for any other individuals.
- This would apply, for example, in the case of the *Diamond Princess* if α , β depended on age and location of cabin on the ship.

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Appendix C: Naïve approach to compartment models fails
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• Focus on the extreme case in which every individual j has infectivity (outgoing to individual k) $\alpha_{j,k}$ and removal parameter β_j .
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• This would apply, for example, in the case of the *Diamond Princess* if α , β depended on age and location of cabin on the ship.

└ Appendix C: Naïve approach to compartment models fails

We want to model $\alpha_{j,k}$ and β_j in terms of a simple parametric model using explanatory variables that are observable *ab initio*.

Timelines and incidents for the compartmental generalization

- ① Individuals no longer exchangeable, so S-I-R state space is unsuitable.
- ② Given population $N \in \mathbb{N}$, the ground space is a tuple of n timelines $(i, [0, T])$, one timeline per individual i , where T is the final time of observation of the epidemic.
- ③ Typical element of state-space: a locally-finite point pattern of
 - ▶ infections $\mathcal{I} = \mathcal{I}_{i,j}(t)$, marked by timelines i at various times t , each infection marked by a target timeline j other than its mark k ;
 - ▶ (inactivated) removals $\mathcal{R} = \mathcal{R}_i(t)$, marked by timelines i at times t ;
 - ▶ conditioned removals $\mathcal{C} = \mathcal{C}_i(t)$, marked by timelines i at times t .
- ④ Initial stipulations:
 - ▶ the \mathcal{I} s, \mathcal{R} s and \mathcal{C} s all occur at different times;
 - ▶ there is at most one \mathcal{C} per timeline;
 - ▶ for convenience, no \mathcal{I} or \mathcal{R} occur after a \mathcal{C} on the same timeline;
- ⑤ Epidemic can be viewed as a union of intervals on different timelines;
 - ▶ intervals end at the \mathcal{C} in the timeline or at time T ;
 - ▶ intervals on initially infected timelines start at time 0;
 - ▶ intervals on eventually infected timelines start at the first time t an \mathcal{I} targets the timeline while marked by a timeline infected at t .

W S Kendall, S B Connor (Warwick, York)

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Process dynamics

Recall that infections and removals *after* a conditioned removal have been censored out. A valid configuration must satisfy the following, derived from the process dynamics:

- ① initially infected timelines i possess no \mathcal{R}_i : if they possess a (single) $\mathcal{C}_i(t)$ then they contribute $(i, [0, t))$ to the epidemic, otherwise $(i, [0, T))$;
- ② other timelines k only contribute if they possess $\mathcal{I}_{k,i}(s)$ such that timeline k is infected at time s , in which case the earliest s is chosen and the contribution is $(i, [s, t))$ when $\mathcal{C}_i(t)$ is the conditioned removal of the timeline, otherwise $(i, [s, T))$;
- ③ every inactivated removal $\mathcal{R}_i(t)$ satisfies $t < s$, where s is the time of first infection of the timeline i .
- ④ every conditioned removal $\mathcal{C}_i(t)$ is at the right t of an infected interval. So each timeline is divided into a *susceptible interval* (empty if it is initially infected), an *infected interval* (empty if it is never infected), and a *removed interval* (empty if it has no conditioned removal).

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| Timelines and incidents for the compartmental generalization | |
|---|---|
| • Individuals no longer exchangeable, so S-I-R state space is unsuitable. | Individuals no longer exchangeable, so S-I-R state space is unsuitable. |
| • Given population $N \in \mathbb{N}$, the ground space is a tuple of n timelines $(i, [0, T])$, one timeline per individual i , where T is the final time of observation of the epidemic. | Given population $N \in \mathbb{N}$, the ground space is a tuple of n timelines $(i, [0, T])$, one timeline per individual i , where T is the final time of observation of the epidemic. |
| • Typical element of state-space: a locally-finite point pattern of infections marked by $\mathcal{I}_{i,j}(t)$, marked by timelines i at various times t ; each infection marked by a target timeline j other than its mark k ; | Typical element of state-space: a locally-finite point pattern of infections marked by $\mathcal{I}_{i,j}(t)$, marked by timelines i at various times t ; each infection marked by a target timeline j other than its mark k ; |
| • (inactivated) removals $\mathcal{R} = \mathcal{R}_i(t)$, marked by timelines i at times t ; | (inactivated) removals $\mathcal{R} = \mathcal{R}_i(t)$, marked by timelines i at times t ; |
| • conditioned removals $\mathcal{C} = \mathcal{C}_i(t)$, marked by timelines i at times t . | conditioned removals $\mathcal{C} = \mathcal{C}_i(t)$, marked by timelines i at times t . |
| • Initial stipulations: | Initial stipulations: |
| ▪ the \mathcal{I} s, \mathcal{R} s and \mathcal{C} s all occur at different times; | there is at most one \mathcal{C} per timeline; |
| ▪ there is at most one \mathcal{C} per timeline; | there is at most one \mathcal{C} per timeline; |
| ▪ for convenience, no \mathcal{I} or \mathcal{R} occur after a \mathcal{C} on the same timeline; | Epidemic can be viewed as a union of intervals on different timelines; intervals end at the \mathcal{C} in the timeline or at time T ; |
| • Epidemic can be viewed as a union of intervals on different timelines; | intervals on initially infected timelines start at time 0; |
| ▪ intervals end at the \mathcal{C} in the timeline or at time T ; | intervals on eventually infected timelines start at the first time t an \mathcal{I} targets the timeline while marked by a timeline infected at t . |
| ▪ intervals on initially infected timelines start at time 0; | |
| ▪ intervals on eventually infected timelines start at the first time t an \mathcal{I} targets the timeline while marked by a timeline infected at t . | |

- ① “S-I-R state space”: $\{(s, i, r) : s, i, r \in \mathbb{N}, s + i + j = n\}$;

- ② one timeline per individual;

- ③ locally-finite point pattern of incidents, derived from several independent Poisson processes on $[0, T]$ for each timeline i ;

- intensity of $\mathcal{I}_{i,j}$ is $\alpha_{i,j}$;

- intensity of \mathcal{R}_i is β_i ;

- the \mathcal{C}_i are not random but stipulated by the conditioning.

- ④ “different times” follows from independent Poisson process assumption. We censor \mathcal{I} or \mathcal{R} occurring after a \mathcal{C} on the same timeline, as they will play no part in the epidemic dynamics.

- ⑤ the simple epidemic trajectory is replaced by a set! We can re-order individual so that (for example) conditionally removed timeline come first, and initially infected timelines take priority in the two categories of “conditionally removed” and “not conditionally removed”.

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| Process dynamics | |
|--|--|
| Result that infections and removals <i>after</i> a conditioned removal have been censored out. A valid configuration must satisfy the following, derived from the process dynamics: | Result that infections and removals <i>after</i> a conditioned removal have been censored out. A valid configuration must satisfy the following, derived from the process dynamics: |
| • initially infected timelines i possess no \mathcal{R}_i : if they possess a (single) $\mathcal{C}_i(t)$ then they contribute $(i, [0, t))$ to the epidemic, otherwise $(i, [0, T))$; | • initially infected timelines i possess no \mathcal{R}_i : if they possess a (single) $\mathcal{C}_i(t)$ then they contribute $(i, [0, t))$ to the epidemic, otherwise $(i, [0, T))$; |
| • other timelines k only contribute if they possess $\mathcal{I}_{k,i}(s)$ such that timeline k is infected at time s , in which case the earliest s is chosen and the contribution is $(i, [s, t))$ when $\mathcal{C}_i(t)$ is the conditioned removal of the timeline, otherwise $(i, [s, T))$; | • other timelines k only contribute if they possess $\mathcal{I}_{k,i}(s)$ such that timeline k is infected at time s , in which case the earliest s is chosen and the contribution is $(i, [s, t))$ when $\mathcal{C}_i(t)$ is the conditioned removal of the timeline, otherwise $(i, [s, T))$; |
| • every inactivated removal $\mathcal{R}_i(t)$ satisfies $t < s$, where s is the time of first infection of the timeline i . | • every inactivated removal $\mathcal{R}_i(t)$ satisfies $t < s$, where s is the time of first infection of the timeline i . |
| • every conditioned removal $\mathcal{C}_i(t)$ is at the right t of an infected interval. So each timeline is divided into a <i>susceptible interval</i> (empty if it is initially infected), an <i>infected interval</i> (empty if it is never infected), and a <i>removed interval</i> (empty if it has no conditioned removal). | • every conditioned removal $\mathcal{C}_i(t)$ is at the right t of an infected interval. So each timeline is divided into a <i>susceptible interval</i> (empty if it is initially infected), an <i>infected interval</i> (empty if it is never infected), and a <i>removed interval</i> (empty if it has no conditioned removal). |

- ① a \mathcal{R}_i on an initially infected timeline would violate conditioning by being activated.

- ② the first \mathcal{I} connecting from an infected timeline will infect the target timeline if it is not already infected.

- ③ inactivated removals simply occur as a Poisson point pattern on the initial uninfected interval of each timeline not initially infected.

Dynamics in algorithmic time

This closely corresponds to the evolution of the S-I-R epidemic above, but does not resample the mark i for each conditioned removal \mathcal{C}_i ;

- ➊ Remove all \mathcal{R} s, and re-sample inactivated removals on the susceptible intervals of each timeline. Recompute **NFZ** as a union of $(i, [0, t_i])$ using the latest time $t_i = t$ of re-sampled $\mathcal{R}_i(t)$.
- ➋ List in *time-reverse order* original infections together with sampled *new candidate* $\tilde{\mathcal{I}}$ s in complements of the removed intervals.
- ➌ Work iteratively through this list. Would discarding original $\mathcal{I}_{i,j}(t)$ result in failure to infect a conditioned removal? If so, **retain** $\mathcal{I}_{i,j}(t)$ as *perpetuated infection* $\mathcal{P}_{i,j}(t)$, otherwise **discard**.
- ➍ Otherwise, at $\tilde{\mathcal{I}}_{a,b}(u)$, consider the latest update of **NFZ**.
 - ▶ If a infected at u and u is in b component of latest **NFZ**, **discard** $\tilde{\mathcal{I}}_{a,b}(u)$;
 - ▶ If a is not infected at u but u lies on b component of latest **NFZ**, **update** **NFZ** by adding/replacing interval of **NFZ** at a using $(a, [0, u))$;
 - ▶ Otherwise **retain** $\tilde{\mathcal{I}}_{a,b}(u)$ as $\mathcal{I}_{a,b}(u)$.

As in S-I-R case, the conditioned epidemic is the unique equilibrium.

Requirements for monotonicity

For **CFTP** we need to know that, for coupled iterations (using the same pattern of innovations of new \mathcal{I} s and \mathcal{R} s), if two variants are started so that the infected region of one contains the other, then this persists through development of the algorithmic time.

It would suffice to prove two technical results:

- ➊ If the old infected region of one contains the other, then the **NFZ** of the one is contained in the **NFZ** of the other;
- ➋ If the old infected region of one contains the other, and the **NFZ** of the one is contained in the **NFZ** of the other, then the new infected region of the one is contained in the other.

Then **CFTP** would make sense, and it would only be necessary to show that accessibility of a set of near-maximal configurations guarantees eventual coalescence.

Perfect Epidemics

2025-06-20

| Dynamics in algorithmic time | |
|---|--|
| This closely corresponds to the evolution of the S-I-R epidemic above, but does not resample the mark i for each conditioned removal \mathcal{C}_i ; | |
| ➊ Remove all \mathcal{R} s, and re-sample inactivated removals on the susceptible intervals of each timeline. Recompute NFZ as a union of $(i, [0, t_i])$ using the latest time $t_i = t$ of re-sampled $\mathcal{R}_i(t)$. | ➋ Resample the marks i for each conditioned removal \mathcal{C}_i on the remaining susceptible intervals of each timeline. Recompute NFZ as a union of $(i, [0, t_i])$ using the latest time $t_i = t$ of re-sampled $\mathcal{R}_i(t)$. |
| ➌ List in <i>time-reverse order</i> original infections together with sampled <i>new candidate</i> $\tilde{\mathcal{I}}$ s in complements of the removed intervals. | ➌ List in <i>time-reverse order</i> original infections together with sampled <i>new candidate</i> $\tilde{\mathcal{I}}$ s in complements of the removed intervals. |
| ➍ Work iteratively through this list. Would discarding original $\mathcal{I}_{i,j}(t)$ result in failure to infect a conditioned removal? If so, retain $\mathcal{I}_{i,j}(t)$ as <i>perpetuated infection</i> $\mathcal{P}_{i,j}(t)$, otherwise discard . | ➍ Work iteratively through this list. Would discarding original $\mathcal{I}_{i,j}(t)$ result in failure to infect a conditioned removal? If so, retain $\mathcal{I}_{i,j}(t)$ as <i>perpetuated infection</i> $\mathcal{P}_{i,j}(t)$, otherwise discard . |
| ➎ Otherwise, at $\tilde{\mathcal{I}}_{a,b}(u)$, consider the latest update of NFZ . | ➎ Otherwise, at $\tilde{\mathcal{I}}_{a,b}(u)$, consider the latest update of NFZ . <ul style="list-style-type: none"> ➏ If a infected at u and u is in b component of latest NFZ, discard $\tilde{\mathcal{I}}_{a,b}(u)$; ➏ If a is not infected at u but u lies on b component of latest NFZ, update NFZ by adding/replacing interval of NFZ at a using $(a, [0, u))$; ➏ Otherwise retain $\tilde{\mathcal{I}}_{a,b}(u)$ as $\mathcal{I}_{a,b}(u)$. |
| As in S-I-R case, the conditioned epidemic is the unique equilibrium. | |

The S-I-R epidemic resamples the marks of \mathcal{C} s to maintain irreducibility. The conditioning here means irreducibility does not fail.

- ➊ Use Poisson process of intensity β_i on susceptible interval of timeline i . The **NFZ** will be iteratively updated in what follows.
- ➋ Use a Poisson process of intensity $\alpha_{a,b}$ to generate the candidate $\tilde{\mathcal{I}}_{a,b}$.
- ➌ When computing whether removal of an infection results in uninfected conditioned removals, use current pattern of infections. The S-I-R case uses **LFE**; the necessary reformulation in set-theoretic terms means more computation is required here. (But note the transition from $\mathcal{I}_{i,j}(t)$ to $\mathcal{P}_{i,j}(t)$ can occur only if timeline b transits from susceptible to infected at time t , and even then only if no subsequent infection happens early enough!).
- ➍ Note how **NFZ** grows in extent as this iteration proceeds, so as to prohibit infection of \mathcal{R} s previously encountered.

Perfect Epidemics

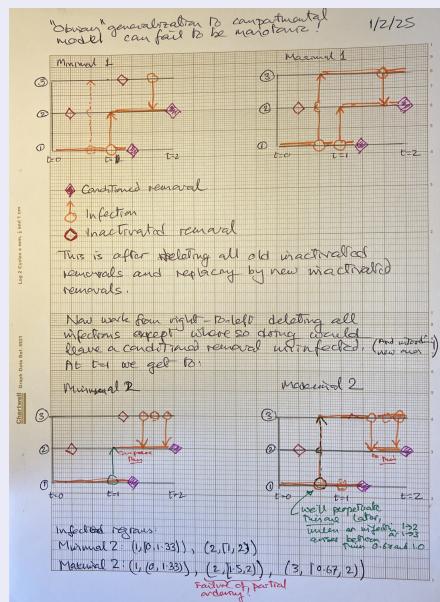
2025-06-20

| Requirements for monotonicity | |
|---|--|
| For CFTP we need to know that, for coupled iterations (using the same pattern of innovations of new \mathcal{I} s and \mathcal{R} s), if two variants are started so that the infected region of one contains the other, then this persists through development of the algorithmic time. | |
| ➊ If the old infected region of one contains the other, then the NFZ of the one is contained in the NFZ of the other; | For CFTP we need to know that, for coupled iterations (using the same pattern of innovations of new \mathcal{I} s and \mathcal{R} s), if two variants are started so that the infected region of one contains the other, then this persists through development of the algorithmic time. |
| ➋ If the old infected region of one contains the other, and the NFZ of the one is contained in the NFZ of the other, then the new infected region of the one is contained in the other. | <ul style="list-style-type: none"> ➏ If the old infected region of one contains the other, then the NFZ of the one is contained in the NFZ of the other; ➏ If the old infected region of one contains the other, and the NFZ of the one is contained in the NFZ of the other, then the new infected region of the one is contained in the other. |

Unfortunately, although

- ➊ the first result can be proved using a case-by-case analysis;
- ➋ **nevertheless the second result is not true**: see next slide for a counterexample!

Counterexample to monotonicity



W S Kendall, S B Connor (Warwick, York)

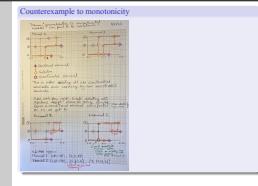
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There is a counterexample for a population of size 3:

- in the minimal case only timelines 1 and 2 are infected, and on the second step it turns out that the $1 \rightarrow 2$ infection must be perpetuated;
- however in the maximal case there is an early $1 \rightarrow 3$ infection and a late $3 \rightarrow 2$ infection which survives to step 2 with a minor modification of the $3 \rightarrow 2$ infection time: the presence of this means the $1 \rightarrow 2$ infection is *not* perpetuated, but it turns out (because there happen to be no suitable new infections) that the $1 \rightarrow 3$ infection must be perpetuated.

We find that the Minimal 2 infection region is not a subset of the Maximal 2 infection region! (though a renumbering of the Minimal 2 population *would* be a subset – not a surprise given our previous results!).

Question: is there a way round this using the CFTP “crossover” trick?

Other technical information

Software used in computations

| Software | Version | Branch | Last commit |
|---------------------|-----------|---------|--------------------------|
| quarto | 1.6.39 | — | |
| Running under julia | 1.11.5 | — | |
| EpidemicsCFTP | 2.2.514 | develop | Fri Mar 21 10:43:55 2025 |
| EpidemicsUtilities | 0.1.2.174 | main | Tue Mar 4 16:32:10 2025 |
| This quarto script | 0.2.2.713 | develop | Wed Mar 12 14:27:50 2025 |

Project information

Version: 0.2.2.715 (develop)

Author: Wilfrid Kendall <W.S.Kendall@warwick.ac.uk>
Date: Fri Jun 20 15:42:30 2025 +0100

Comment:

Preparation for Liverpool seminar