

Perfect Epidemics

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

W S Kendall S B Connor

Warwick, York

18 June 2025



Introduction

“Maybe the only significant difference between a really smart simulation and a human being was the noise they made when you punched them.”
(The Long Earth, Pratchett & Baxter, 2012)



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

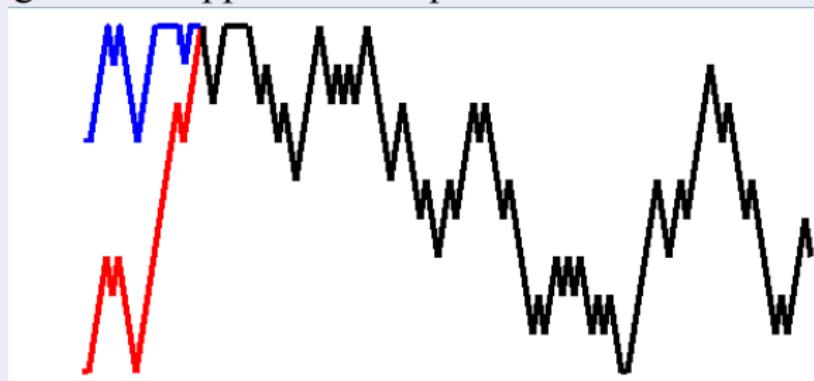
Work on perfect simulation ([CFTP](#)) for epidemics, now being written up.
WSK acknowledges the support of UK EPSRC grant EP/R022100.

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).

Classic CFTP for a simple random walk (I)

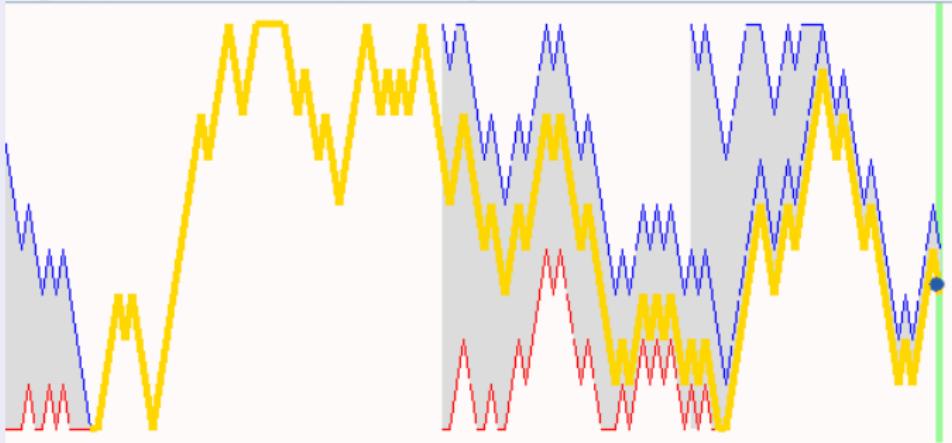
- ① Consider a simple random walk on $0:9 = \{0, 1, 2, 3, 4, 5, 6, 7, 8, 9\}$.
 - ▶ $\mathbb{P}[+1 \text{ jump}] = p \in (0, 1)$, while $\mathbb{P}[-1 \text{ jump}] = 1-p$, **except that**
 - ▶ at state 9 replace the +1 jump by “staying still”, **and**
 - ▶ at state 0 replace the -1 jump by “staying 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!

2. A little theory about CFTP-

- ① What about cases where 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* we can show that
 - ▶ 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** can be used to carry out 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**.)

3. Perfect Epidemics: a challenge problem for CFTP

S-I-R deterministic epidemic:

susceptibles s , infectives i , removals r (constant total population $s + i + r = n$):

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

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).



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 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.”

Evidently $\alpha s_0 / \beta \gg 1$ – as was sadly later confirmed, a sorrow for us all.



Inference on the R-number

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

- ➊ Modelling is **tough**. *Either* massive assumptions (homogeneous mixing) *or* very many parameters;
- ➋ Inference is **really tough**: hard to get information about infection times;
- ➌ 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);
- ➍ Markov chain Monte Carlo (MCMC) can be used (see next slide) but what about burn-in?
- ➎ Can we use **perfect simulation**?

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)

An important step on the way concerns generation of an *unconditioned* epidemic.

- ① Thus n, α, β are known, removal times are observed,
but unobserved infection times **must be inferred**.
- ② 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.
- ③ Poisson point processes *of appropriate rates* yield an S-I-R epidemic.
- ④ First step: evolve whole S-I-R trajectory in *algorithmic time* (alter potential infections and removals using immigration-death in discrete algorithmic time).
- ⑤ Result: *trajectory-valued chain*, unconditioned S-I-R as equilibrium.

From incidents to unconditioned epidemic trajectories (1/3)

Incidents defining an epidemic

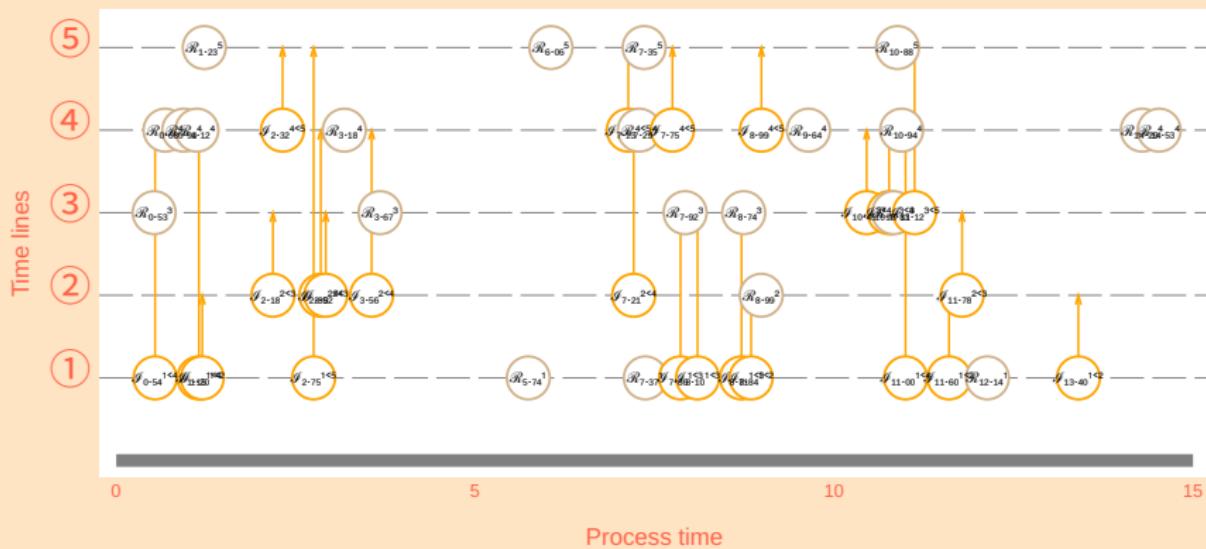


Figure 1: Light-orange circles denote potential infections (arrows point upwards to targets); light-brown circles denote potential removals.

From incidents to unconditioned epidemic trajectories (2/3)

Unconditioned epidemic

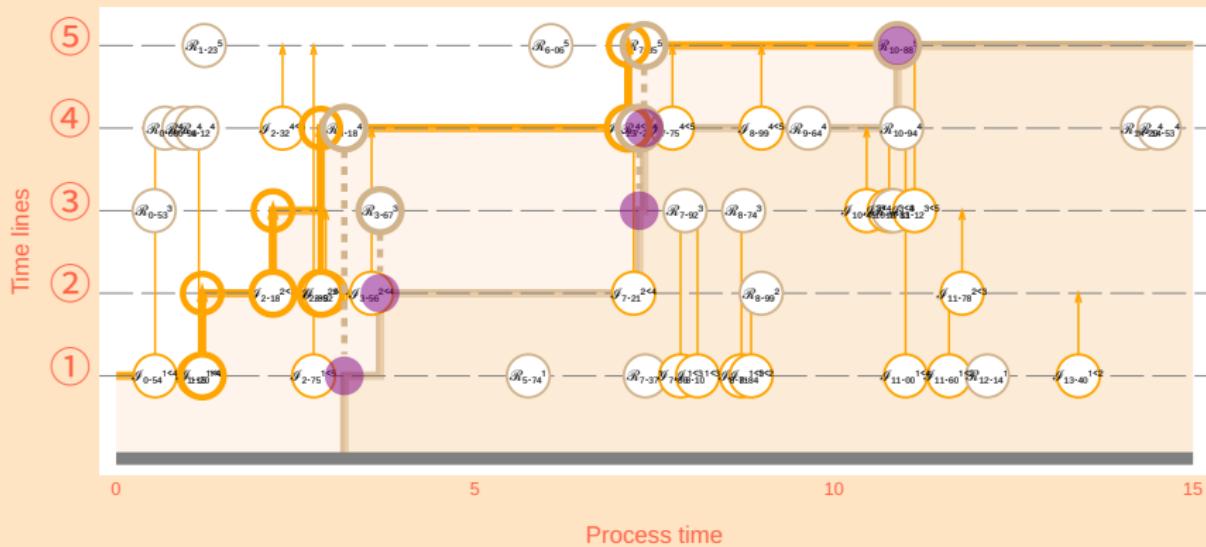


Figure 2: (a) *Infection* activates if target lies on the lowest uninfected timeline; and (b) *removal* activates if on infected timeline; remove lowest infected (purple disk).

From incidents to unconditioned epidemic trajectories (3/3)

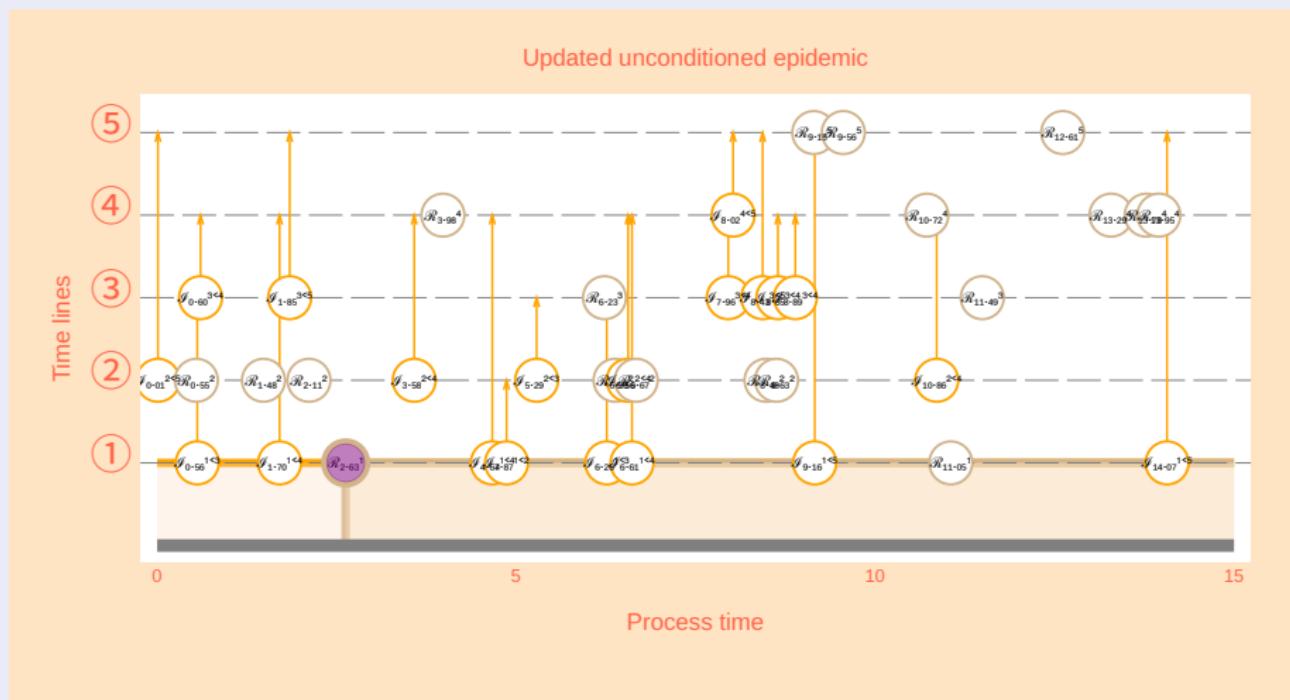


Figure 3: A step in algorithmic time for the unconditioned epidemic simply involves replacing all original incidents by an entirely new set of incidents.

Crucial technical point

- Updates in algorithmic time τ are then (algorithmic-) *time-reversible*: so restriction to subset S of state-space (the *activated / conditioned* removals must occur precisely at specified set of process 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;
 - ③ For $(2n+1)T < \tau < (2n+2)T$, update old \mathcal{I} s in $((2n+2)T - \tau, T)$.
- Thus the original update is expressible as a (continuous) composition of updates, each of which satisfies detailed balance in equilibrium.
- The connection “restriction=conditioning” still holds.
- Crucially, step 2 ensures composition action is irreducible over S !
(So equilibrium under conditioning is unique.)

Free evolution evolving in continuous algorithmic time

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

The initial conditioned epidemic

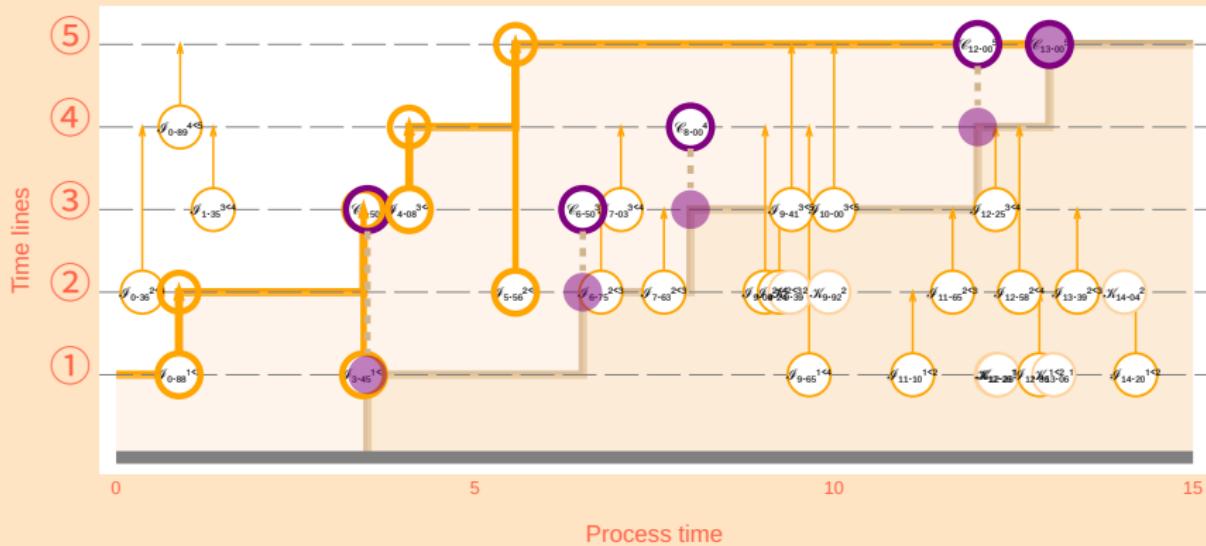


Figure 4: Initial conditioned epidemic, with conditioned removals indicated using purple circles (and purple disks when non-target timelines are infected).

Conditional epidemic update

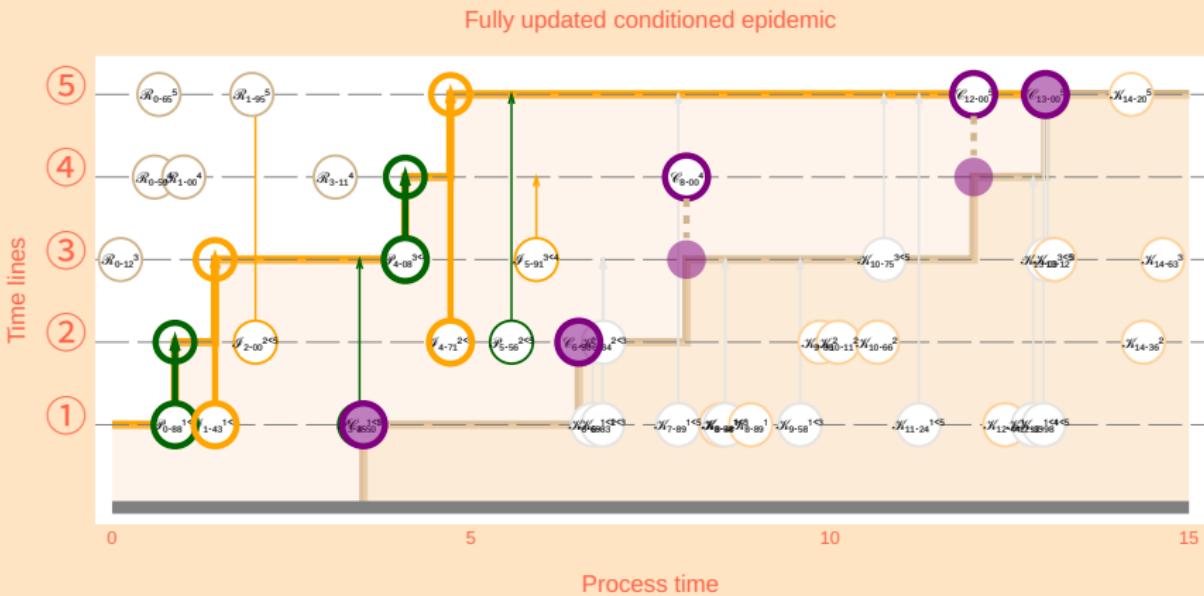


Figure 5: Epidemic updated under restriction: all conditioned removals remain activated, no new removals are activated. Green infections have been “perpetuated”.

Laziest feasible epidemic (LFE)

Fully updated conditioned epidemic with LFE

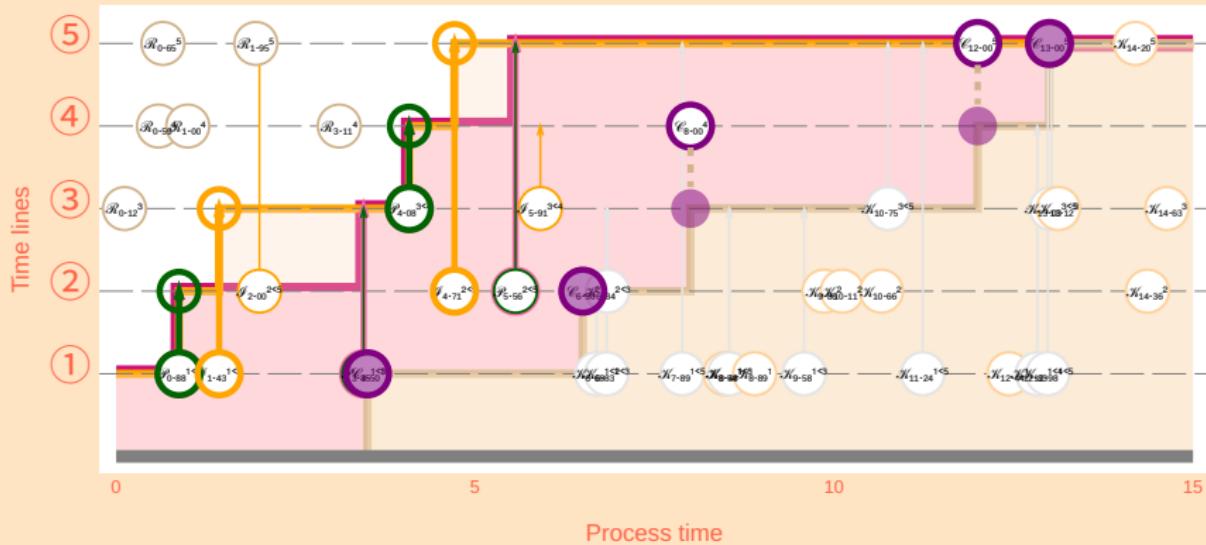


Figure 6: LFE computed recursively working right-to-left: the slowest sequence of infections deals with all infected timelines in order (includes perpetuated infections).

No-fly zone (NFZ)

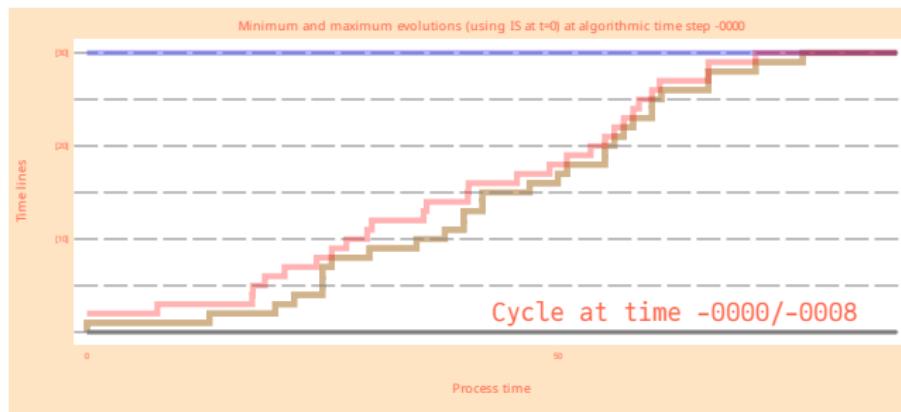
Fully updated conditioned epidemic with NFZ



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.

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 perfect simulation **GIF** or **MP4** yielding a draw from unobserved pattern of infections.



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?

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: statistical estimation of parameters, generalization to other compartment models.
- Thank you for your attention! **QUESTIONS?**



References I

- Bailey, N.T.J. (1975) *The mathematical theory of infectious diseases and its applications*, 2nd Ed. ed. Griffin.
- Bensoussane, H. (2025) Bayesian Individual-level Epidemic Models : Accounting for Missing Data and Utilising Covariate Information (PhD No. January).
- Bezanson, J., Edelman, A., Karpinski, S., & Shah, V.B. (2017) Julia: A Fresh Approach to Numerical Computing. *SIAM Review*, **59**, 65–98.
- Cori, A. & Kucharski, A. (2024) Inference of epidemic dynamics in the COVID-19 era and beyond. *Epidemics*, **48**, 100784.
- Donnelly, P. & Kurtz, T.G. (1996) A countable representation of the Fleming-Viot measure-valued diffusion. *The Annals of Probability*, **24**, 698–742.
- Foss, S.G. & Tweedie, R.L. (1998) Perfect simulation and backward coupling. *Stochastic Models*, **14**, 187–203.
- Fraser, C. & Others (2023) OpenABM-Covid19: Agent-based model for modelling the Covid-19 and Contact-Tracing.
- Gibson, G.J. & Renshaw, E. (1998) Estimating parameters in stochastic compartmental models using Markov chain methods. *Mathematical and Medical Biology*, **15**, 19–40.
- Gibson, G.J. & Renshaw, E. (2001) Likelihood estimation for stochastic compartmental models using Markov chain methods. *Statistics and Computing*, **11**, 347–358.

References II

- Huber, M.L. (2015) *Perfect Simulation*. Boca Raton: Chapman; Hall/CRC.
- Liggett, T.M. (1985) *Interacting Particle Systems*, Grundlehren der mathematischen wissenschaften. Berlin, Heidelberg: Springer Berlin.
- O'Neill, P.D. & Roberts, G.O. (1999) Bayesian Inference for Partially Observed Stochastic Epidemics. *Journal of the Royal Statistical Society Series A: Statistics in Society*, **162**, 121–129.
- Pratchett, T. & Baxter, S. (2012) *The Long Earth*. Doubleday.
- Propp, J.G. & Wilson, D.B. (1996) Exact sampling with coupled Markov chains and applications to statistical mechanics. *Random Structures and Algorithms*, **9**, 223–252.
- Rocklöv, J., Sjödin, H., & Wilder-Smith, A. (2020) COVID-19 outbreak on the Diamond Princess cruise ship: estimating the epidemic potential and effectiveness of public health countermeasures. *Journal of Travel Medicine*, **27**, 7 pp.
- SBC (2020) Omnithermal Perfect Simulation for Multi-server Queues. *ACM Transactions on Modeling and Computer Simulation*, **30**, 1–15.
- SBC & WSK (2007b) Perfect simulation for a class of positive recurrent Markov chains (corrigendum). *Annals of Applied Probability*, **17**, 1808–1810.
- SBC & WSK (2007a) Perfect simulation for a class of positive recurrent Markov chains. *Annals of Applied Probability*, **17**, 781–808.
- SBC & WSK (2015) Perfect simulation of M/G/c queues. *Advances in Applied Probability*, **47**, 1039–1063.

References III

- SBC & WSK (2024) Perfect Epidemics.
- Whittle, P. (1955) The outcome of a stochastic epidemic—a note on Bailey's paper. *Biometrika*, **42**, 116–122.
- WSK (1998) Perfect Simulation for the Area-Interaction Point Process. *Probability towards 2000* (Accardi, L. & Heyde, C.C. eds). Springer-Verlag, pp. 218–234.
- WSK (2004) Geometric ergodicity and perfect simulation. *Electronic Communications in Probability*, **9**, 140–151.
- WSK (2005) Notes on Perfect Simulation. Singapore: World Scientific, pp. 93–146.
- WSK (2015) Introduction to CFTP using R. *Stochastic geometry, spatial statistics and random fields, Lecture notes in mathematics*. Springer, pp. 405–439.
- WSK & Møller, J. (2000) Perfect simulation using dominating processes on ordered spaces, with application to locally stable point processes. *Advances in Applied Probability*, **32**, 844–865.

Image information

Image	Attribution	
<i>Terry Pratchett</i>	Luigi Novi	<i>CC BY 3.0</i>
Classic CFTP for a simple random walk	Result of code written by WSK	
<i>Diamond Princess</i>	Alpsdake	<i>CC BY-SA 4.0</i>
Epidemic CFTP images and animation	Result of code written by WSK	

Previous instances of this talk

Date	Title		Location	
19/04/24	Perfect Epidemics	Short Research Talk	12min	Warwick
15/05/24	McMC and Perfect Simulation	Graduate Seminar, Aristotle Univ.	50min	Thessaloniki
17/01/25	Perfect Epidemics	Applied Probability Seminar	50min	Warwick

Appendix A: A “near-maximal” configuration

A small set for a conditioned epidemic.

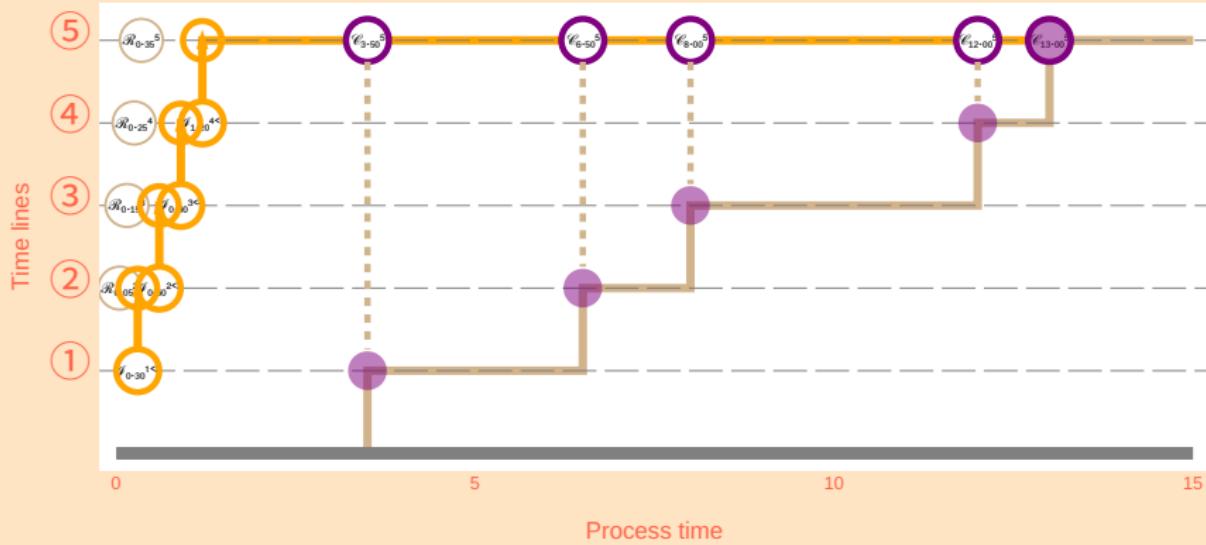


Figure 8: A conditioned epidemic in which all activated infections occur before time 3.0, also before smallest observed removal time.

Appendix B: Updating a conditioned epidemic

Animation of evolution of conditioned S-I-R epidemic in continuous algorithmic time.

Halfway through the animation we jitter between before and after the second re-sampling of timelines for Removal incidents.



Appendix C: Outline of the monotonicity proof

Summary of plan of proof:

- ① Let $\text{EPI}_{\tau=0}^{\pm}$ represent two epidemic trajectories (\pm) at algorithmic time $\tau=0$, viewed as subsets of “timeline-space” $\{(k, [0, T)) : k = 1, 2, \dots\}$.
- ② Suppose at algorithmic time $\tau=0$ the *fast* $\text{EPI}_{\tau=0}^+$ is never later than the *slow* $\text{EPI}_{\tau=0}^-$ so $\text{EPI}_{\tau=0}^+ \supseteq \text{EPI}_{\tau=0}^-$; additionally suppose monotonicity holds for conditional removal marks: if $\mathcal{C}_{\tau=0}^{\pm}$ are conditional removals at fixed process time t then $\mathcal{C}_{\tau=0}^+ \text{ timeline} \geq \mathcal{C}_{\tau=0}^- \text{ timeline}$.
- ③ Then a related monotonicity holds for the laziest feasible epidemics: $\text{LFE}_{\tau=1}^+ \leq \text{LFE}_{\tau=1}^-$ at algorithmic time $\tau=1$.
- ④ Likewise a similar monotonicity (but reversing the set-theoretic inclusion!) holds for no-fly zones: $\text{NFZ}_{\tau=1}^+ \subseteq \text{NFZ}_{\tau=1}^-$.
- ⑤ Now prove $\text{EPI}_{\tau=1}^+ \supseteq \text{EPI}_{\tau=1}^-$ moreover if $\mathcal{C}_{\tau=1}^+$ matches $\mathcal{C}_{\tau=1}^-$ at process time t then $\mathcal{C}_{\tau=1}^+ \text{ timeline} \geq \mathcal{C}_{\tau=1}^- \text{ timeline}$.

$\text{LFE}_{\tau=1}$: recursive construction

Let $\text{LFE}_{\tau=1}^{\pm}(k)$ be the (process) time of the latest infection of timeline k needed if all \mathcal{C}^{\pm} 's of $\text{EPI}_{\tau=1}^{\pm}$ are to be infected.

- ① For the top timeline n , $\text{LFE}_{\tau=1}^{\pm}(n)$ must precede any \mathcal{C}^{\pm} on timeline n ; set $\text{LFE}_{\tau=1}^{\pm}(n) = T$ if no such \mathcal{C}^{\pm} .
- ② For $k < n$ with $\text{LFE}_{\tau=1}^{\pm}(k+1) = T$, again $\text{LFE}_{\tau=1}^{\pm}(k)$ must precede any \mathcal{C}^{\pm} on timeline k ; set $\text{LFE}_{\tau=1}^{\pm}(k) = T$ if no such \mathcal{C}^{\pm} .
- ③ Suppose n_0 is largest k with $\text{LFE}_{\tau=1}^{\pm}(k) < T$. Working downwards through $\ell = n_0 - 1, \dots, 1$, $\text{LFE}_{\tau=1}^{\pm}(\ell)$ is the time of the latest infection targeting $\ell + 1$ and based in the infected region such that
 - (a) $\text{LFE}_{\tau=1}^{\pm}(\ell) \leq \text{LFE}_{\tau=1}^{\pm}(\ell + 1)$;
 - (b) $\text{LFE}_{\tau=1}^{\pm}(\ell)$ precedes any \mathcal{C}^{\pm} on timeline ℓ .
- ④ Equivalently, $\text{LFE}_{\tau=1}^{\pm}(\ell)$ is the time of the latest potential infection targeting $\ell + 1$ such that
 - (a) $\text{LFE}_{\tau=1}^{\pm}(\ell) \leq \text{LFE}_{\tau=1}^{\pm}(\ell + 1)$;
 - (b) $\text{LFE}_{\tau=1}^{\pm}(\ell)$ precedes any \mathcal{C}^{\pm} on timelines $\ell, \ell + 1, \dots, n$.

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

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

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

Establish monotonicity for NFZ _{$\tau=1$} ^{±,*}, NFZ _{$\tau=1$} ^{±,0}, NFZ _{$\tau=1$} ^{±,1}, NFZ _{$\tau=1$} ^{±,2}, ... in turn:

- ① Since EPI _{$\tau=0$} ⁺ \supseteq EPI _{$\tau=0$} ⁻ and the set of \mathcal{R} s for EPI _{$\tau=1$} [±] are formed by intersecting the same \mathcal{R} pattern with the complements of EPI _{$\tau=0$} [±], it follows that NFZ _{$\tau=1$} ^{+,*} \subseteq NFZ _{$\tau=1$} ^{-,*}.
- ② Monotonicity for NFZ _{$\tau=1$} ^{±,0} is a direct consequence.
- ③ Given NFZ _{$\tau=1$} ^{+,j-1} \subseteq NFZ _{$\tau=1$} ^{-,j-1}, create NFZ _{$\tau=1$} ^{±,j} by proposing \mathcal{J} at time t targeting timeline k , based in EPI _{$\tau=0$} ⁺ 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 _{$\tau=1$} ⁺. 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 _{$\tau=1$} ⁺ because timeline $k-1$ is not infected at t in EPI _{$\tau=0$} ⁺. But we know EPI _{$\tau=0$} ⁺ \supseteq EPI _{$\tau=0$} ⁻, so timeline $k-1$ is not infected at t in EPI _{$\tau=0$} ⁻ either. So infection in EPI _{$\tau=1$} ⁻ 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}.

$EPI_{\tau=1}$: 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

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

$EPI_{\tau=1}$: 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 $EPI_{\tau=1}$.

Appendix D: 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.

- 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 .

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).

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:

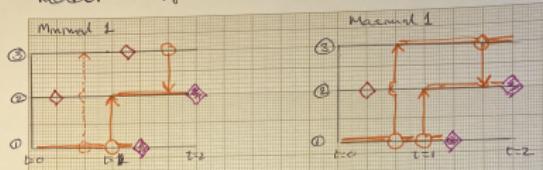
- ① If the old infected region of one contains the other, then the **NFZ** of the one is contained in the **NFZ** of the other;
- ② If the old infected region of one contains the other, and the **NFZ** of the one is contained in the **NFZ** of the other, then the new infected region of the one is contained 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.

Counterexample to monotonicity

"Observe" generalization to compartmental model can fail to be monotone!

1/2/25



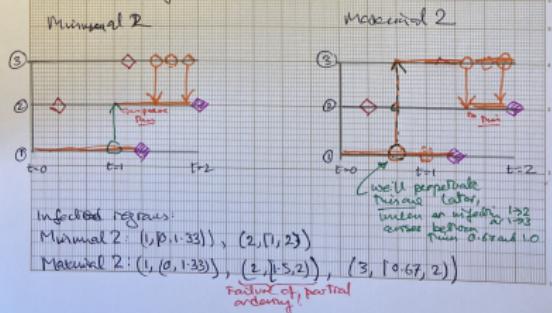
◆ Conditioned removal

◆ Infected

◆ Inactivated removal

This is after deleting all old inactivated removals and replacing by new inactivated removals.

Now work from right-to-left deleting all infections except where so doing would leave a conditioned removal uninfected. (and updating)
At t=1 we get to:



Infected regions:

Marmot 1: $(1[0,1.33]), (2[1,2])$

Marmot 2: $(1,(0,1.33)), (2,[1.5,2]), (3,[0.67,2])$

Failure of partial ordering

Other technical information

Software used in computations

<i>Software</i>	<i>Version</i>	<i>Branch</i>	<i>Last commit</i>
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:	0.2.2.713 (develop)
Author:	Wilfrid Kendall <W.S.Kendall@warwick.ac.uk>
Date:	Wed Mar 12 14:27:50 2025 +0000

Comment:

Checked against new Infection inscriptions.