Perfect Epidemics

Seminar at University College Dublin

W S Kendall S B Connor

Warwick, York

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Introduction

Homage to Dublin
(Book of Kells, 9th century)

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

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

Plan of talk

Gregory: Is there any other point to which you would wish to draw my attention?
Holmes: To the curious incident of the dog in the night-time.
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from "The Adventure of Silver Blaze", Sir Arthur Conan Doyle (1892).

(a) Introduction to perfect simulation:
(b) A little theory about CFTP;
(c) Epidemics and the R-number;
(d) "Contact tracing" (inferring infection pattern if removals observed);
(e) Example with real data.

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- Epidemics and the R-number;
- "Contact tracing" (inferring infection pattern if removals observed);
- Example with real data.

The Sherlock Holmes quote emphasizes how information can be deduced from what we *don't* see.

Here is the plan of the talk:

(a) A rapid visual review of CFTP ("perfect simulation"),
(b) Touching on some relevant results.
(c) A brief survey of the epidemiological context.
(d) Main part of talk: "Contact tracing", describing the use of perfect simulation when inferring infection pattern if one has observed all epidemic removals.
(e) How CFTP for epidemics ("Perfect Epidemics") works in practice when applied to real data.

1. A Visual Introduction to Perfect Simulation

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

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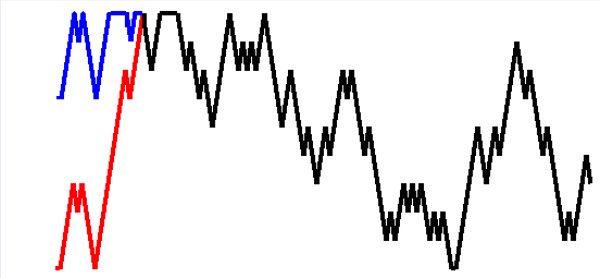
Very brief history of CFTP/ perfect simulation.

1. Jim Propp described the discovery of CFTP as like walking down the street and suddenly noticing a 50$ bill lying on the ground. He also said exact simulation "was in the air": many related ideas
2. In particular, "exact simulation" cannot somehow miraculously defeat numerical approximation error `:-)`. And, as with all simulation, the validity of "exact simulation" depends on correctness of code!
3. We illustrate these key ideas by considering a single very specific and simple example: CFTP for a simple random walk.
4. Propp & Wilson (1996) build on random walk CFTP to get exact samples for a critical Ising model: "Like seeing Mars for the first time through a telescope" (Persi Diaconis, 2009). 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.

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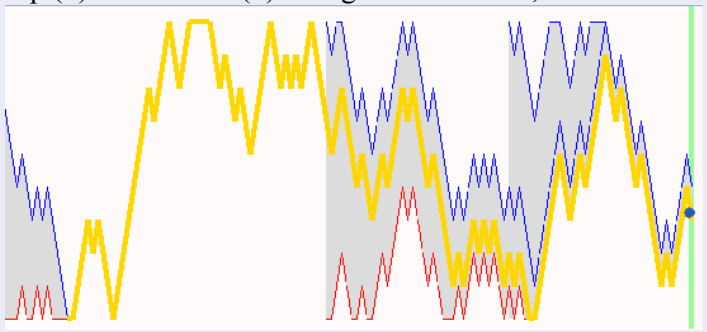
- Generally not true that location *at* coupling is a draw from equilibrium.

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

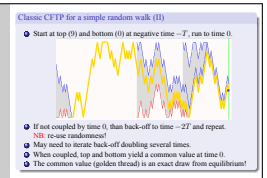
1. The process is irreducible *and* aperiodic, so equilibrium is a truncated Geometric distribution (hint: use reversibility to check this!).
2. Of course even conventional MCMC is overkill here: simulation directly from the equilibrium distribution is simple and efficient.
3. This observation was originally 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!

Classic CFTP for a simple random walk (II)

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



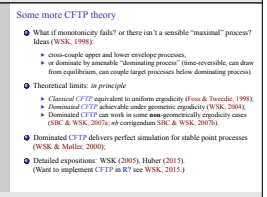
2. If not coupled by time 0, than back-off to time $-2T$ and repeat.
   NB: re-use randomness!
3. May need to iterate back-off doubling several times.
4. When coupled, top and bottom yield a common value at time 0.
5. The common value (golden thread) is an exact draw from equilibrium!



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 T is initially 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 gets essentially the same result however large the back-off, so the draw is effectively a draw from time $-\infty$. The golden thread can be viewed as a perfect draw from the end of the simulation (this sort of argument is very well-known to ergodic theorists). Remarkably, this can easily be converted into a fully rigorous proof!

Some more CFTP theory

1. What if monotonicity fails? or there isn't a sensible "maximal" process? Ideas (WSK, 1998):
   - cross-couple upper and lower envelope processes,
   - or 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.)



1. To be computationally effective, the ideas of WSK (1998) still require a (possibly very weak) notion of an associated partial order, and also the ability to simulate the dominating process.
2. Basic plan: 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 as in Sigman (2011), 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).

2. Perfect Epidemics: a challenge problem for CFTP

S-I-R deterministic epidemic: differential equation system for (s, i, r)

Susceptible:	s'	$=$	$-\alpha\, s\, i\,,$
Infected:	i'	$=$	$(\alpha\, s - \beta\,)\, i\,,$
Removed:	r'	$=$	$\beta\, i\,.$

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

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

Infection:	$S \to S-1\,,$	$I \to I+1$	at rate	$\alpha\, S\, I\,,$
Removal:	$I \to I-1\,,$	$R \to R+1$	at rate	$\beta\, I\,.$

Both models share an unrealistic assumption: homogeneous mixing.

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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 *et al* (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 $\gg 1$ implies 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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Whittle (1955) uses a coupling argument that doesn't mention coupling!

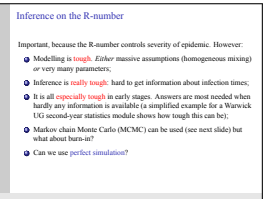
Some recent history

- 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) focus 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:

1. Modelling is tough. *Either* massive assumptions (homogeneous mixing) *or* very many parameters;
2. Inference is really tough: hard to get information about infection times;
3. 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);
4. Markov chain Monte Carlo (MCMC) can be used (see next slide) but what about burn-in?
5. Can we use perfect simulation?



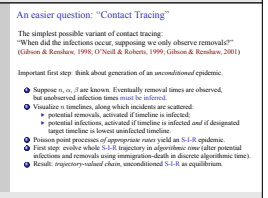
- The "R-number" was the subject of continuing interest throughout the COVID-19 pandemic!
- Notice that the "R-number" is as much a social construct as a biological one, and in real situations will interact with behaviour in subtle and complicated ways.
- We will seek to make headway in using perfect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

An easier question: "Contact Tracing"

The simplest possible 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.

1. Suppose n, α, β are known. Eventually 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.



Focus on just one aspect of inference:

1. Avoid parameter inference for now; consider the large number of nuisance parameters given by (typically unobserved) infection times.
2. Timelines: instead of tracking individuals as in the stochastic epidemic model, track the numbers of individuals in various categories.
3. Poisson point processes generate incidents, which then collectively influence the S-I-R trajectory.
4. 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.
5. Exploit the classic connection for reversible Markov processes, between conditioning and restriction of state space.
6. Work in *continuous* algorithmic time, obtain a dynamically reversible Markov process, prove restriction=conditioning still works.

From incidents to unconditioned epidemic trajectories (1/3)

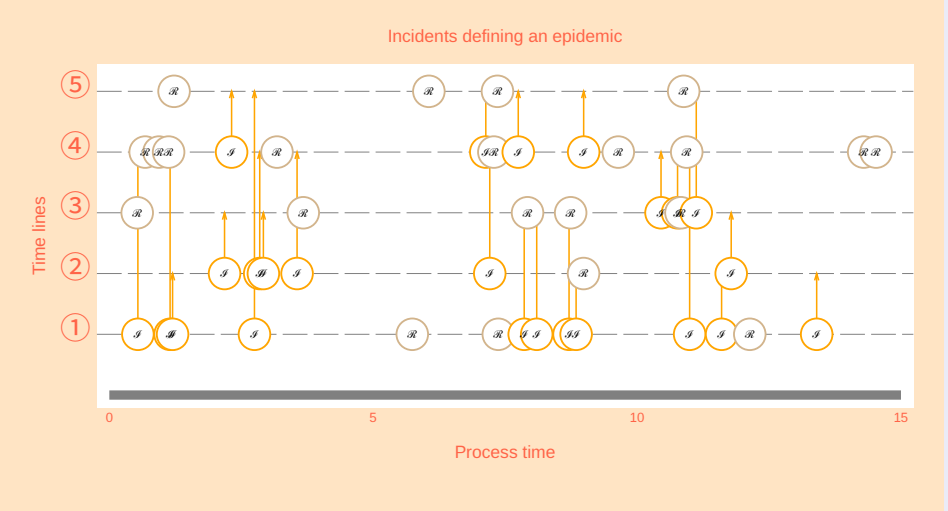
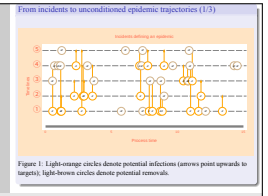


Figure 1: Light-orange circles denote potential infections (arrows point upwards to targets); light-brown circles denote potential removals.



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

Infections and removals are distinguished by colour-coding. Incidents specify time and target timeline: infections also point to the timeline to be infected.

- Intensities 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.
- An infection incident is *activated* exactly when it sits on an infected portion of its timeline and points to the least of the uninfected timelines (as calculated at the process time of the incident). This is computationally expensive, (cost is cubic in αT, not quadratic!) but simplifies monotonicity arguments.
- A removal incident is activated exactly when it sits on an infected portion of its timeline.

From incidents to unconditioned epidemic trajectories (2/3)

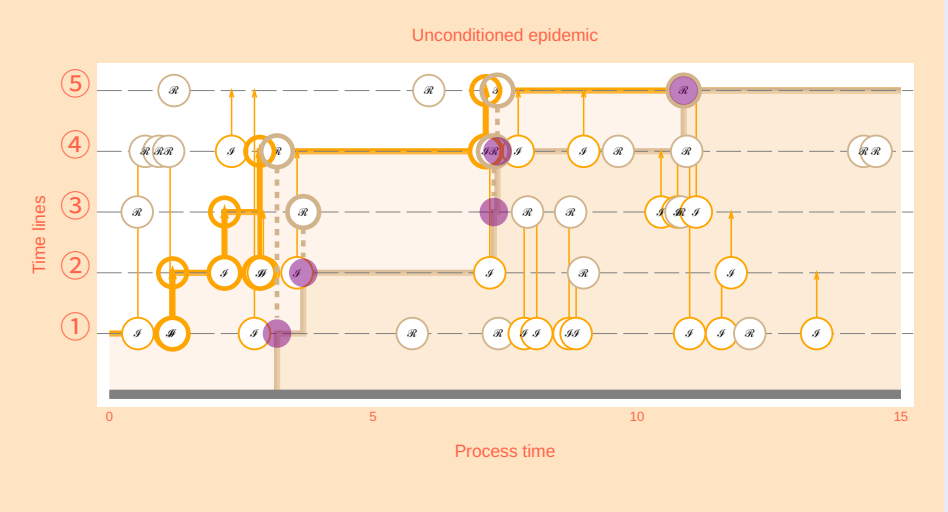
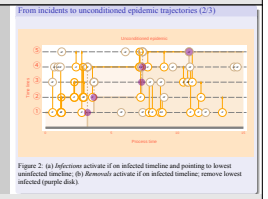


Figure 2: (a) *Infections* activate if on infected timeline and pointing to lowest uninfected timeline; (b) *Removals* activate if on infected timeline; remove lowest infected (purple disk).



- Here is the epidemic evolution resulting from an innovation.
- Note that the initial number of infectives must be specified (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, or more subtle forms of independence sampler.

From incidents to unconditioned epidemic trajectories (3/3)

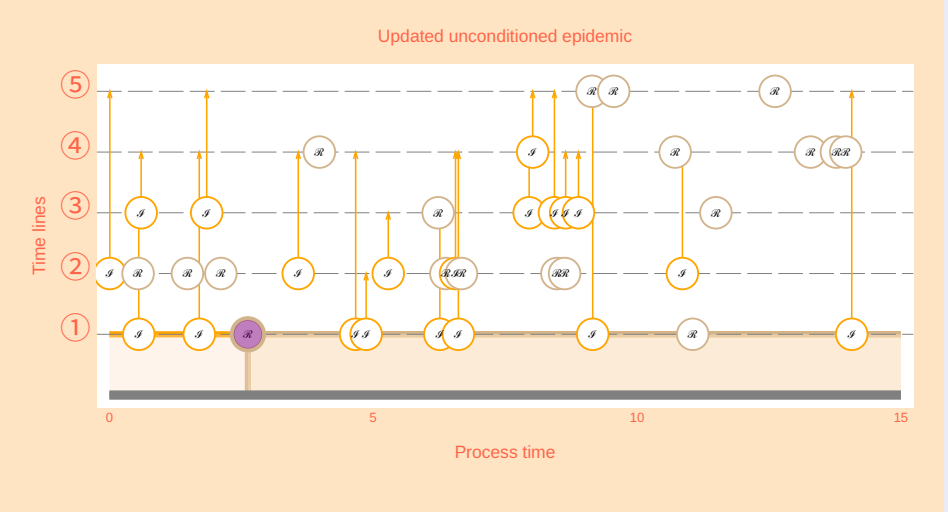
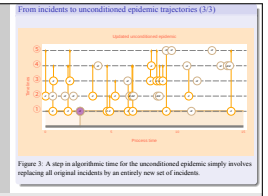


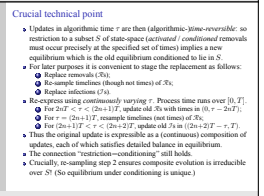
Figure 3: A step in algorithmic time for the unconditioned epidemic simply involves replacing all original incidents by an entirely new set of incidents.



- An update in algorithmic time, if there is no conditioning, simply involves replacement of one set of incidents by another. (A radical change occurs in this illustration: epidemic dies out at 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

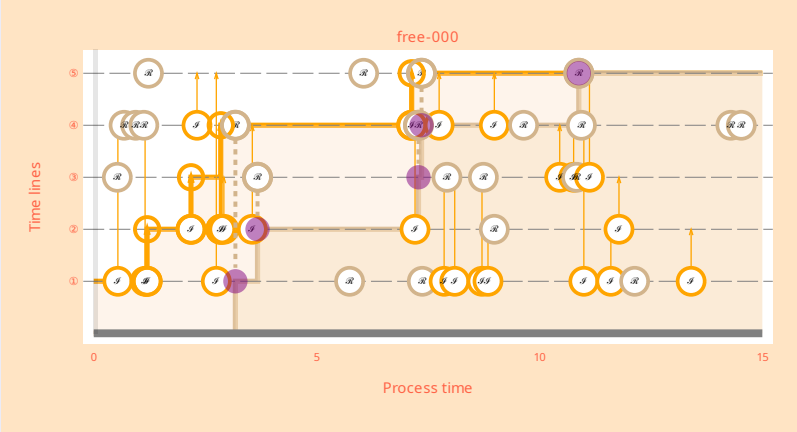
- Updates in algorithmic time τ are then (algorithmic-)*time-reversible*: so restriction to a subset S of state-space (*activated* / *conditioned* removals must occur precisely at the specified set of times) implies a new equilibrium which is the old equilibrium conditioned to lie in S.
- For later purposes 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;
  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, re-sampling step 2 ensures composite evolution is irreducible over S! (So equilibrium under conditioning is unique.)



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



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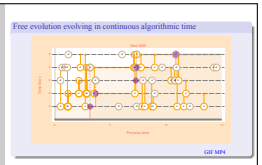
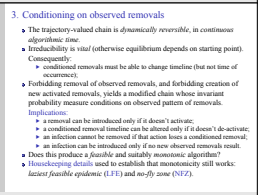


We discuss a single step in algorithmic time. Conditioning not applied here!

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

3. 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).



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.)

1. "Restriction=monotonicity needs careful proof in the continuum limit: discretization,"convergence stationnaire" ("parking convergence").
2. The heart of the matter lies in establishing (some variant of) monotonicity.
3. 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!).
4. Geometric notions of LFE and NFZ provide a useful vocabulary for formulating how this works out. They facilitate a rigorous argument for arguing for monotonicity, and are also computationally helpful.

Initial conditioned epidemic

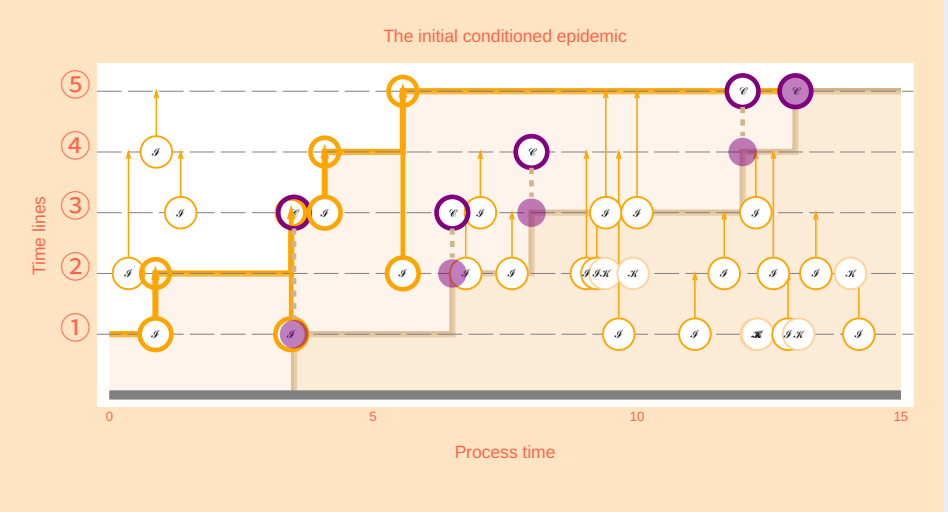
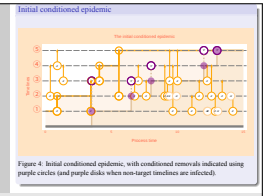


Figure 4: Initial conditioned epidemic, with conditioned removals indicated using purple circles (and purple disks when non-target timelines are infected).



Here is a different initial epidemic, including *conditioned removals* ($\mathcal{C}$s), indicated by purple circles.

- $\mathcal{C}$s have fixed times, so removed zone does not change.
- Evolution in algorithmic time as in the unconditioned case (working "pixel-by-pixel"), but forbidding any change that breaks conditioning.
- Dealing with $\mathcal{R}$s amounts to removing all inactivated $\mathcal{R}$s and replacing them with a new set of $\mathcal{R}$s, and then resampling the $\mathcal{C}$ timelines, accepting only those changes which do not de-activate the $\mathcal{C}$s.
- Dealing with *infections* ($\mathcal{I}$s) requires more care.

Conditional epidemic update

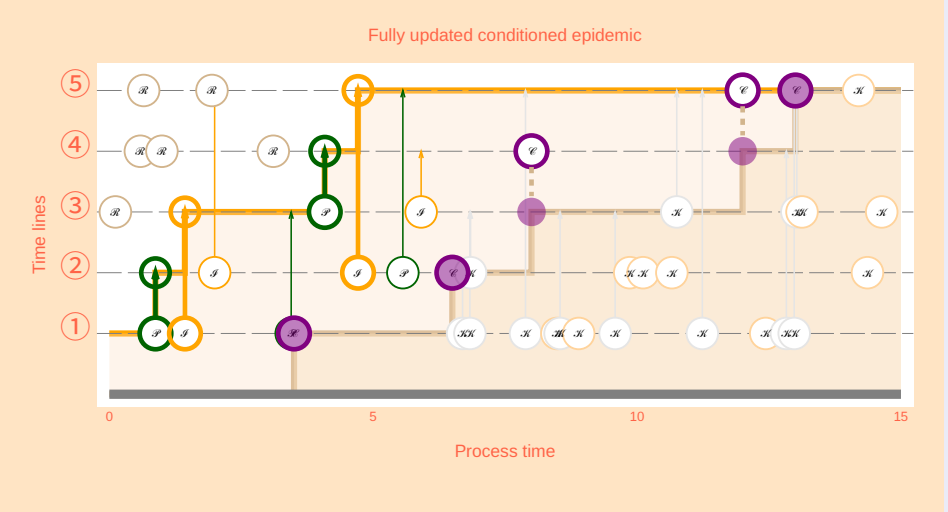
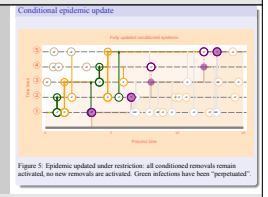


Figure 5: Epidemic updated under restriction: all conditioned removals remain activated, no new removals are activated. Green infections have been "perpetuated".



The update proceeds as follows:

1. Accept inactivated $\mathcal{R}$s only if targets avoid infected region.
2. Accept $\mathcal{C}$s only if targets fall in infected region.
3. Accept new $\mathcal{I}$s only if this doesn't result in infecting an inactivated $\mathcal{R}$.
4. Retain old $\mathcal{I}$s only if rejection would result in a $\mathcal{C}$ *not* being infected, in which case retain old $\mathcal{I}$ as *perpetuated infection* ($\mathcal{P}$).

Options 1 and 2 are directly imposed by the requirement of not changing the set of $\mathcal{C}$s.
For options 3 and 4 there is a useful correspondence to two recursively-defined geometric structures: the *laziest feasible epidemic* (LFE: *slowest possible* epidemic compatible with $\mathcal{C}$s) and the *no-fly zone* (NFZ: forbid all $\mathcal{I}$s that might infect this zone).

Laziest feasible epidemic (LFE)

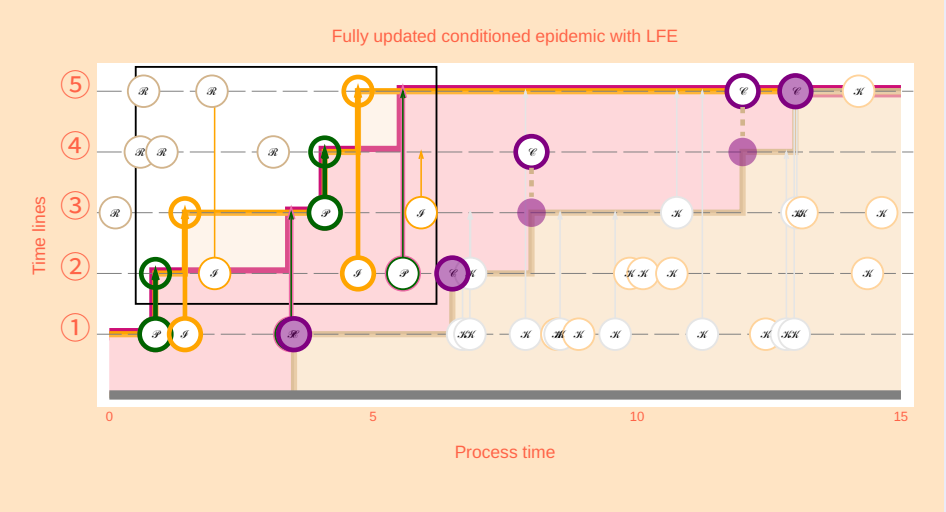
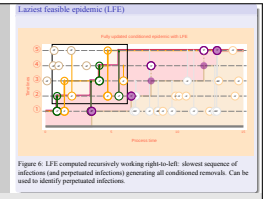


Figure 6: LFE computed recursively working right-to-left: slowest sequence of infections (and perpetuated infections) generating all conditioned removals. Can be used to identify perpetuated infections.



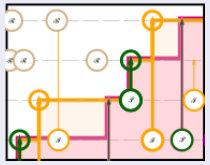
The LFE is the slowest sequence of $\mathcal{P}/\mathcal{I}$s generating all $\mathcal{C}$s. Suppose timeline i is infected by LFE at time s_i. Set $s_N = T$. Backwards recursion: LFE infects timeline i using $\mathcal{P}_{s_i}^{j<i+1}/\mathcal{I}_{s_i}^{j<i+1}$ for maximum s_i with

$$s_i \leq \min\left\{s_{i+1}, \inf\{s \,:\, \text{there is a } \mathcal{C}_s^i\}\right\} .$$ (see next slide!)

- Here LFE and epidemic differ on time intervals $[1.4, 3.5]$, $[4.7, 5.6]$;
- There are $\mathcal{P}$s at times 0.9, 3.5, 4.1, 5.6 (green arrows: old $\mathcal{I}$s retained to ensure conditioning is not broken);
- Two $\mathcal{P}$s (at times 0.9, 4.1) form part of the new epidemic trajectory;
- All $\mathcal{P}$s *have* to form part of the LFE;
- $\mathcal{P}$s will *not* form part of the new epidemic trajectory when preceded by *new* activated $\mathcal{I}$;
- so the LFE includes a (new) $\mathcal{I}$ instead of a $\mathcal{P}$ if it stops $\mathcal{P}$ forming.

The LFE is rather like an epidemic run *backwards* through process time.

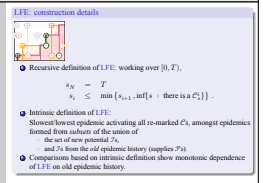
LFE: construction details



1. Recursive definition of LFE: working over $[0, T)$,

$$\begin{aligned} s_N &= T \\ s_i &\leq \min\left\{s_{i+1}, \inf\{s \,:\, \text{there is a } \mathcal{C}_s^i\}\right\} . \end{aligned}$$

2. Intrinsic definition of LFE:
   Slowest/lowest epidemic activating all re-marked $\mathcal{C}$s, amongst epidemics formed from *subsets* of the union of
   - the set of new potential $\mathcal{I}$s,
   - and $\mathcal{I}$s from the *old* epidemic history (supplies $\mathcal{P}$s).
3. Comparisons based on intrinsic definition show monotonic dependence of LFE on old epidemic history.



Exercise: show the two definitions agree.

The intrinsic definition helps prove monotonic dependence of the LFE on the old epidemic history (using the same sets of potential incidents).
Compare the effects of slower and of faster old epidemic histories (using the same sets of $\mathcal{C}$s and of new potential $\mathcal{I}$s)

Consider:

(i) Construction based on slower old epidemic history with new potential $\mathcal{I}$s and re-marked $\mathcal{C}$s based on slower old epidemic history;
(ii) Construction based on faster old epidemic history with new potential $\mathcal{I}$s and re-marked $\mathcal{C}$s based on slower old epidemic history;
(iii) Construction based on faster old epidemic history with new potential $\mathcal{I}$s and re-marked $\mathcal{C}$s based on faster old epidemic history.

We can show that moving from (i) to (ii) to (iii) increases LFE at each step.

No-fly zone (NFZ)

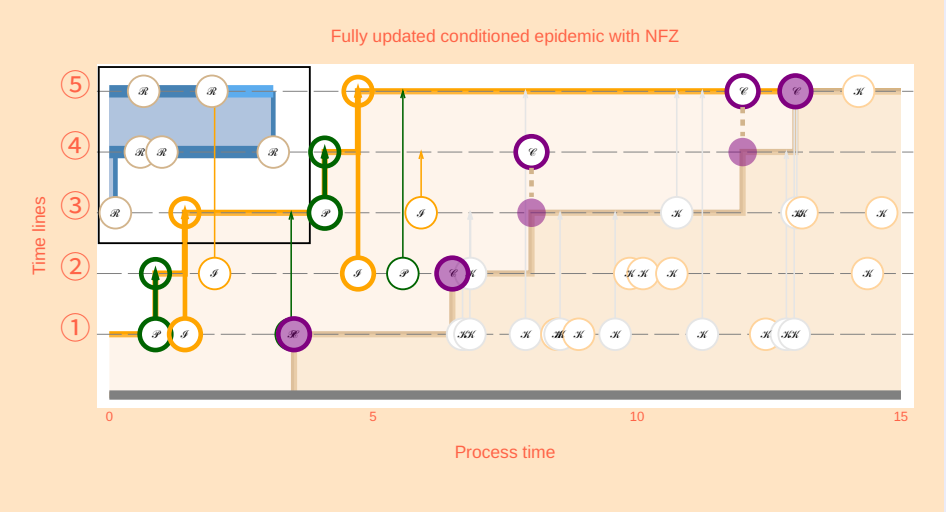
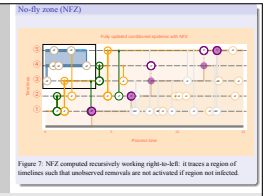


Figure 7: NFZ computed recursively working right-to-left: it traces a region of timelines such that unobserved removals are not activated if region not infected.

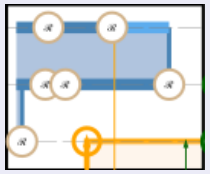


The NFZ is used to decide whether to accept proposals of $\mathcal{I}$s targeting the susceptible region:

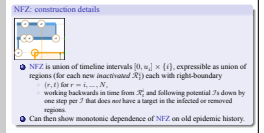
1. New $\mathcal{I}$s with targets in the infected zone are assessed to see whether their introduction would activate $\mathcal{R}$s, by connecting the infected zone to a timeline leading to an $\mathcal{R}$ (hence "no-fly" portion of timeline);
2. An $\mathcal{I}$ with target in susceptible zone may make 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.

NFZ: construction details



1. NFZ is union of timeline intervals $[0, u_i] \times \{i\}$, expressible as union of regions (for each new *inactivated* $\mathcal{R}_t^i$) each with right-boundary
   - (r, t) for $r = i, \ldots, N$,
   - working backwards in time from $\mathcal{R}_t^i$ and following potential $\mathcal{I}$s down by one step per $\mathcal{I}$ that does *not* have a target in the infected or removed regions.
2. Can then show monotonic dependence of NFZ on old epidemic history.



N is the population number!

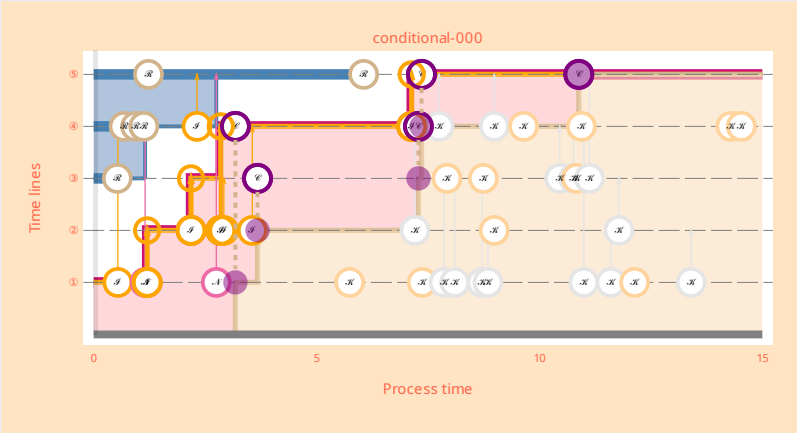
There is also a recursive definition which we do not give here.

It is then relatively easy (though slightly more involved than for the LFE) to show that the NFZ also depends monotonically on the old epidemic history (using the same sets of potential incidents).

All the above implies monotonicity of the algorithmic-time evolution of the conditioned epidemic: hence permitting CFTP! Key points:

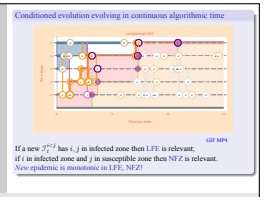
(i) LFE and NFZ together determine whether updates of I_{0-} are permitted, leading to monotonicity if the update mechanism for I_{0-} is monotone.
(ii) Monotonicity for new epidemic history then follows by establishing it for *constant* I_{0-}.

Conditioned evolution evolving in continuous algorithmic time



GIF MP4

If a new $\mathcal{I}_t^{i<j}$ has i, j in infected zone then LFE is relevant;
if i in infected zone and j in susceptible zone then NFZ is relevant.
New epidemic is monotonic in LFE, NFZ!

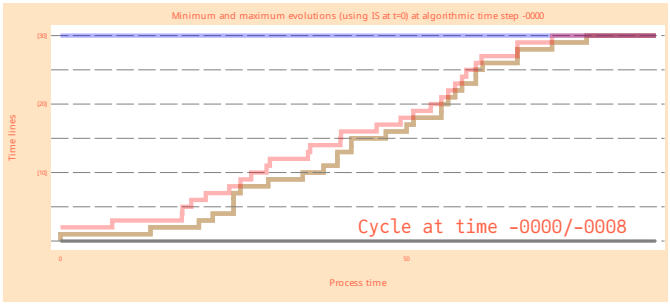


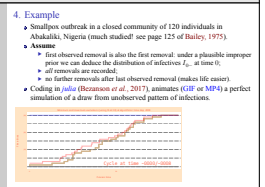
Conditioning applied here!

- Start with initial configuration, use immigration-death process of $\mathcal{R}$s to replace old $\mathcal{R}$s except in the infected region.
- All old $\mathcal{R}$s have now been removed, and some new $\mathcal{R}$s have been added.
- Resample timeline of each $\mathcal{R}$ if this does not violate conditioning. Jitter back and forth here.
- Now resample $\mathcal{I}$s, *but this time working backwards through process time.* If addition proposed, add only if pattern of observed $\mathcal{R}$s are unchanged. If deletion proposed, and deletion would change pattern of observed removals, then *perpetuate*. Use backwards recursive definitions of LFE and NFZ to facilitate online computations for these decisions.

4. 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 deduce 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), animates (GIF or MP4) a perfect simulation of a draw from unobserved pattern of infections.





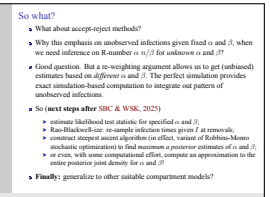
We could vary the assumption concerning the initial and final numbers of infectives while still using essentially the same perfect simulation algorithm! This amounts to presuming some Bayesian prior knowledge.

- Blue is maximal, red is minimal, start at extremal epidemics.
- Suppose we only have a prior for the number of initial infectives?
  - Use independence sampler: draw from (unconditioned) number of initial infectives, accept if this leads to observed removals exactly as conditioned;
  - Or random-walk Metropolis sampler, if prior for initial number of infectives is suitably monotonic;
  - Or modified independence sampler and optimal scaling heuristic.

Technical note: The language `julia` (Bezanson *et al.*, 2017), allows for rapid development by using an expressive type-based syntax (very useful for involved algorithms), and delivers remarkably fast execution using "just in time" compilation techniques.

So what?

- What about accept-reject methods?
- Why this emphasis on unobserved infections given fixed α and β, when we need inference on R-number $\alpha\ n/\beta$ 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, 2025)
  - estimate likelihood test statistic for specified α and β;
  - Rao-Blackwell-ize: re-sample infection times given I at removals;
  - 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 an approximation to the entire posterior joint density for α and β!
- **Finally:** generalize to other suitable compartment models?

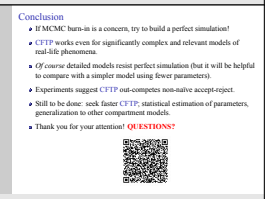


Accept-reject approach for non-trivial problems requires *much* more computation.
Perfect simulation supplies a *well-behaved* stochastic integration mechanism, using repeated MCMC samples in a Monte Carlo calculation. Rao-Blackwellization available (interpolate between conditioned removals using nonlinear growth process bridges!).
Other compartment models presenting harder challenges:

1. Split population into 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!
5. Current recipe then becomes non-monotonic! Perhaps cross-over arguments might help?

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).
- Experiments suggest CFTP out-competes non-naïve accept-reject.
- Still to be done: seek faster CFTP; statistical estimation of parameters, generalization to other compartment models.
- Thank you for your attention! **QUESTIONS?**



There is much work still to be done!

Notes from previous discussions:
Warwick talk (January 2025). Paul Jenkins asked whether this relates to the "look-down" argument in population genetics (Donnelly & Kurtz, 1996)?

Provisional answer (after rereading Donnelly & Kurtz, 1996): the "look-down" argument is *related* to this CFTP algorithm because both involve a particle model as in Liggett (1985). I cannot yet see a closer connection.

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Image information

Image	*Attribution*	
Book of Kells	Huber Gerhard	*CC BY 4.0*
Classic CFTP for a simple random walk	Result of code written by WSK	
Diamond Princess	Alpsdake	*CC BY-SA 4.0*
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	12mn	Warwick
15/05/24	McMC+Perfect Simulation	Graduate Seminar, Aristotle Univ.	50mn	Thessaloniki
17/01/25	Perfect Epidemics	Applied Probability Seminar	50mn	Warwick
27/06/25	Perfect Epidemics	UK Research Network Stochastics	45mn	Liverpool
20/10/25	Perfect Epidemics	Seminar		Dublin

Other technical information

Software used in computations

Software	*Version*	*Branch*	*Last commit*
`quarto`	`1.6.39`	—	
Running under `julia`	`1.12.0`	—	
`EpidemicsCFTP`	`2.2.532`	`develop`	`Tue Jul 8 17:13:42 2025 +0100`
`EpidemicsUtilities`	`0.1.2.177`	`main`	`Fri Sep 26 15:35:26 2025 +0100`
This `quarto` script	`0.2.2.725`	`2025-10-09-Dublin-preparation`	`Tue Oct 14 18:01:39 2025 +0100`

Project information

Version:	`0.2.2.727` (`2025-10-09-Dublin-preparation`)
Author:	Wilfrid Kendall <`W.S.Kendall@warwick.ac.uk`>
Date:	Wed Oct 15 19:53:35 2025 +0100

Comment:
Near-final preparation for Dublin talk 20 October 2025. Minor edits.