A possible smeared phase transition in epidemic track-and-trace

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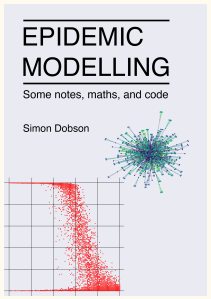
Introduction

Exploring epidemics using network science [1]

- Disease models and parameters
- Contact structures
- Countermeasures



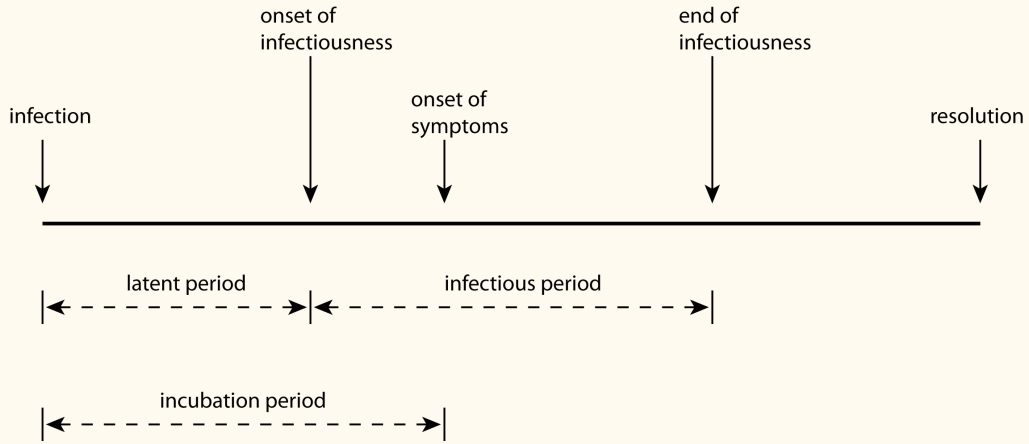
In the course of writing about these ideas I stumbled across something I can't readily explain

- A change on behaviour that should be crisp, but doesn't seem to be
- Deserves more exploration

[1] S. Dobson. *Epidemic modelling – Some notes, maths, and code*. Independent Publishing Network, 2020. ISBN 978-183853-565-0. URL https://simoninireland.github.io/introduction-to-epidemics/

Structure of this talk

Real diseases – 1

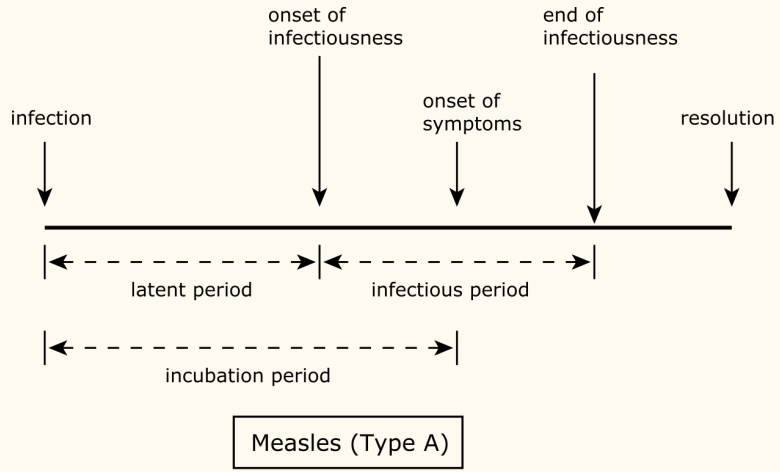


Different periods

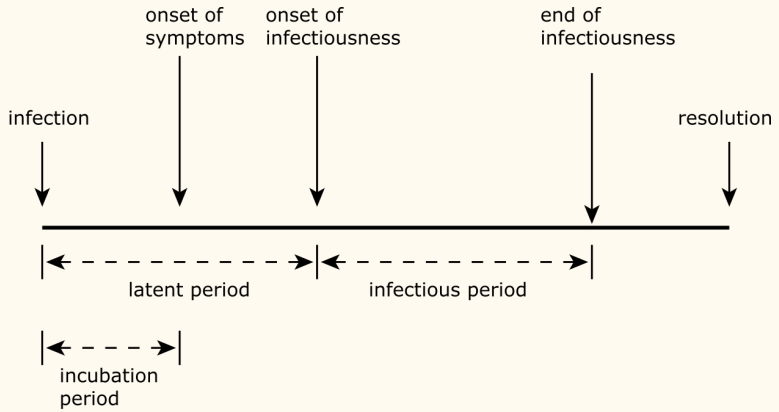
- *Incubation*: from infection to onset of symptoms
- *Latent*: from exposure to infectiousness
- *Infectious*: overlapping with symptoms (usually)

Periods defined by biology, of both disease and host

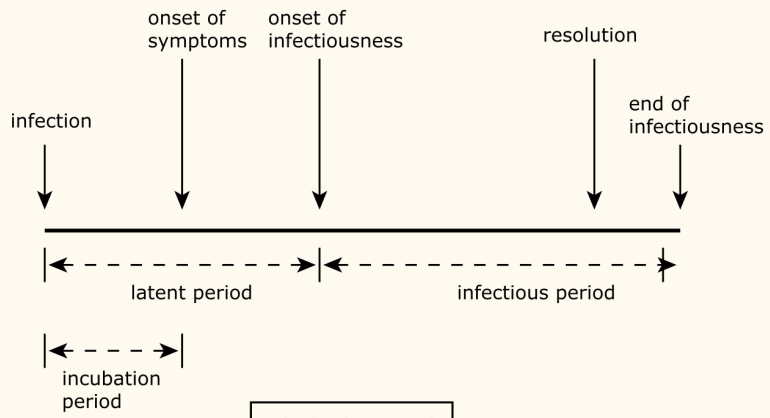
Real diseases – 2



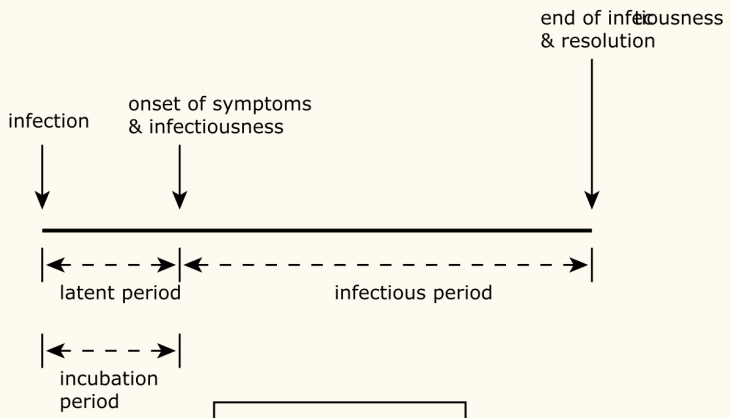
Measles (Type A)



SARS (Type B)



Ebola (Type B)



Zombie apocalypse

$\mathcal{R}$ and all that [2]

$\mathcal{R}$, the case reproduction number

- Number of secondary cases per primary
- Especially $\mathcal{R}_0$, reproduction absent countermeasures

r, the case reproduction rate

- Doubling time for an epidemic
- Also sometimes see T_g, the inter-generation time

k, the overdispersion parameter

- Essentially a variance of the distribution around R
- Small number of individuals can drive most cases

[2] Royal Society SET-C group. Reproduction number (R) and growth rate (r) of the COVID-19 epidemic in the UK: methods of estimation, data sources, causes of heterogeneity, and use as a guide in policy formulation, August 2020. URL https://royalsociety.org/-/media/policy/projects/set-c/set-covid-19-R-estimates.pdf

The "wickedness" of covid-19

$\mathcal{R}_0 \approx 3$ is not particularly infectious

- Straightforward to get $\mathcal{R} \approx 1.5$; harder to get $\mathcal{R} < 1$
- Significant overdispersion
- Infection seems to convey only temporary immunity

Case fatality rate is about 1%

- Too large to comfortably ignore, but not so large as to admit no arguments

Substantial asymptomatic transmission

- Asymmetric costs (spreading *vs* dying, "long covid")
- Effective countermeasures are collective (and expensive)

The goals of modelling

What are we trying to find out?

- Concrete: how will this *particular* outbreak behave, in this *particular* population?
- Abstract: how can diseases behave *in general*? Are there common mathematical structures?

Compartmented models

Traditional epidemic modelling uses the framework of a *compartmented model* of a disease

- A number of compartments holding some fraction of the population
- (Can also think of this as the state of each individual)
- Rules on how these fractions change over time

Continuous SIR

The model

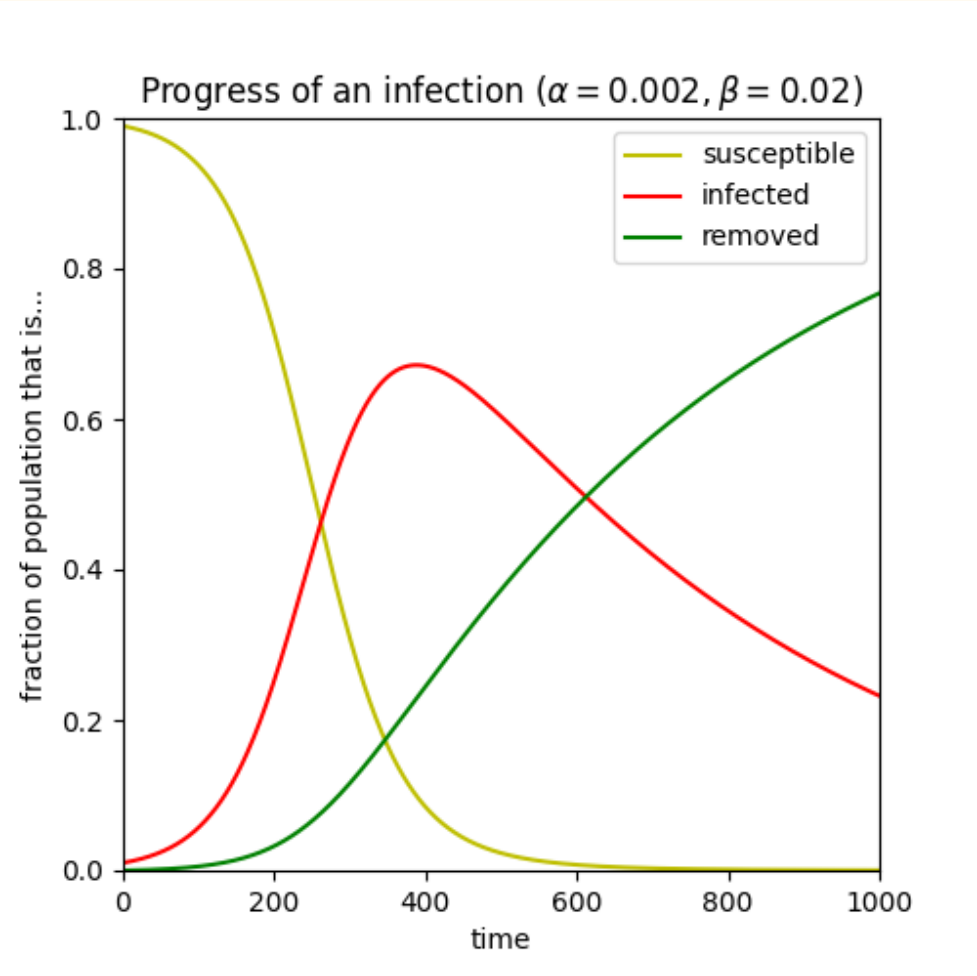
- *S*usceptible individuals can catch the infection from *I*nfected individuals
- …who then are *R*emoved from the dynamics by recovery (or death)

Epidemic dynamics

- Susceptibles infected per contact with probability β
- Infecteds removed with probability α

$$\frac{dS}{dt} = -\beta SI \qquad \frac{dI}{dt} = \beta SI - \alpha I \qquad \frac{dR}{dt} = \alpha I$$

Solution


Progress of an infection ($\alpha = 0.002, \beta = 0.02$)
susceptible
infected
removed
fraction of population that is...
time
0.0
0.2
0.4
0.6
0.8
1.0
0
200
400
600
800
1000

A range of models with increasing complexity

Can model different disease structures [3]

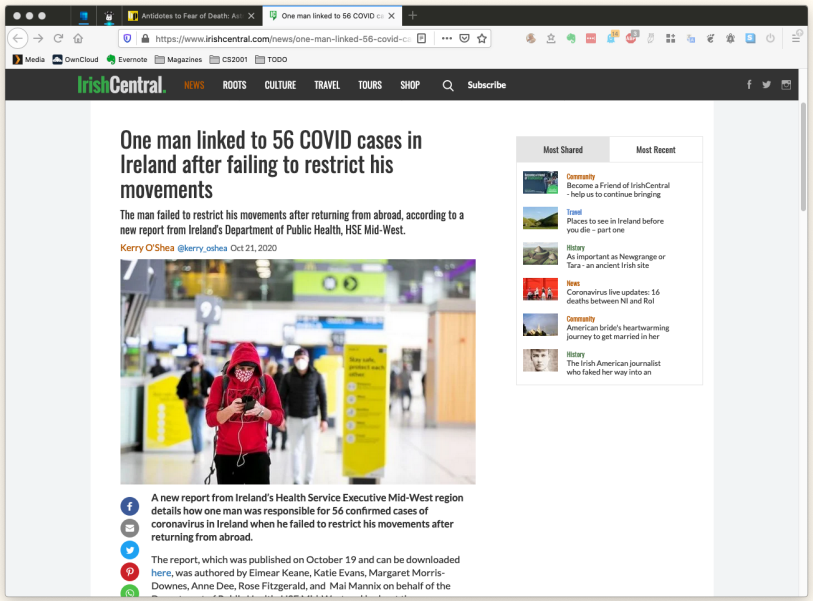
- SIR – simple infection offering complete immunity post-infection
- SIS – infection confers no immunity
- SEIR – exposed individuals are infectious before symptoms
- MSEIR – initial immunity passed from mother to child
- SEIRS – immunity wears off with time
- …

[3] H. Hethcote. The mathematics of infectious diseases. *SIAM Review*, 42(4):599–653, December 2000. URL doi://10.1137/S0036144500371907

Epidemics on networks – 1

Complete homogeneous mixing implies populations have no structure, which clearly isn't true

- Not everyone meets everyone else
- Some are massively more (or less) connected than average



Epidemics on networks – 2

Use a network as the substrate for the epidemic [4]

- Only nodes that are adjacent can interact
- Compartment = label on node

Leads to some more modelling decisions

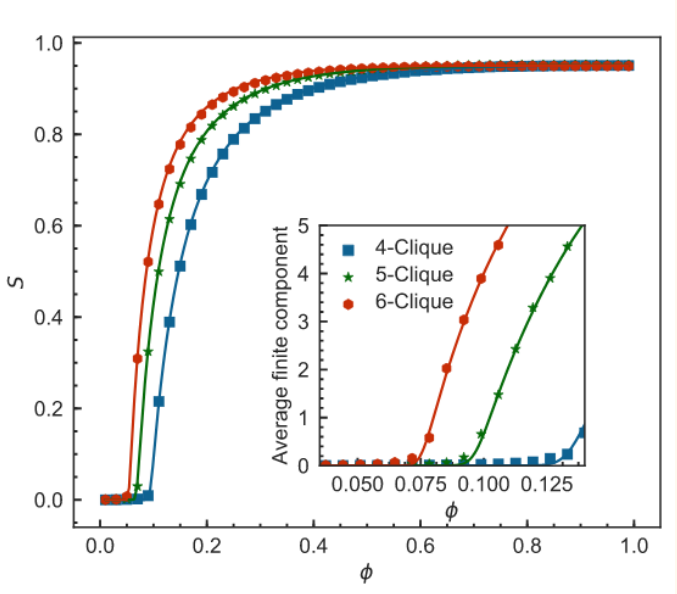
- Degree distribution: probability p_k of node having degree k
- Often treat the mean degree $\langle k \rangle$ as "representative"
- Topological fine structure: loops, modules, layers, …
- Adaptive behaviour to change these features in response to the progression of the disease (*e.g.*, quarantine)

[4] M. Newman. Spread of epidemic disease on networks. *Physical Review E*, 66, July 2002. URL doi://10.1103/PhysRevE.66.016128

How to do analysis

The "gold standard" is an analytic model with numerical validation

- Find an analytic description for what happens under different infection parameters
- Run process on random networks with the given topology
- Lots of repetitions to squeeze out variance
- (Hopefully) sample points land on solutions of the equations [5]



[5] P. Mann, V. A. Smith, J. Mitchell, and S. Dobson. Percolation in random graphs with higher-order clustering. URL https://arxiv.org/abs/2006.06744. Under review by Physical Review E. Preprint available on arXiv

How to do numerical validation

A networked population

- Set all to susceptible
- Change a fraction to infected, chosen at random
- Run the model rules until equilibrium

Discrete-event simulation [6]

- After a time τ we see an event e (infection, removal, ...)
- Draw the next time and event from joint distribution $P(\tau, e)$

[6] D. Gillespie. Exact stochastic simulation of coupled chemical reactions. *Journal of Physical Chemistry*, 81(25): 2340—2361, 1977

Field lacks standard tooling

So we built some :-)

- epydemic: models, standard topologies, ...
- epyc: experiments, parallelism, dataset management

```
lab = epyc.ClusterLab(profile='hogun')

lab[epydemic.ERNetwork.N] = 10000
lab[epydemic.ERNetwork.KMEAN] = 40

lab[epydemic.SIR.P_INFECTED] = 0.001
lab[epydemic.SIR.P_REMOVE] = 0.002
lab[epydemic.SIR.P_INFECT] = numpy.linspace(0.00001, 0.0002, num=50)

m = epydemic.SIR()
g = epydemic.ERNetwork()
e = epydemic.StochasticDynamics(m, g)

lab.runExperiment(epyc.RepeatedExperiment(e, 100))
```

https://github.com/simoninireland

Structure of this talk

What do human contact networks look like?

Networks with particular contact structure

- Tend to have cycles (friends of friends)
- Highly variable numbers of contacts
- Often modular, having different local and global structures

Construction

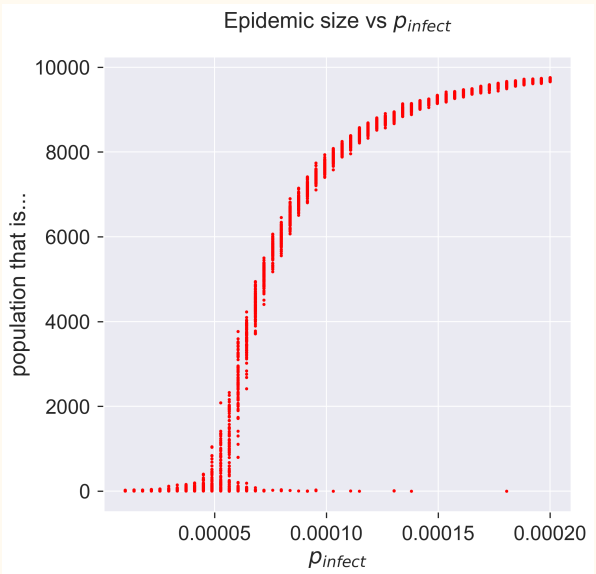
- Theoretically, using a process to generate the structures
- Empirically, structured from survey data [7]

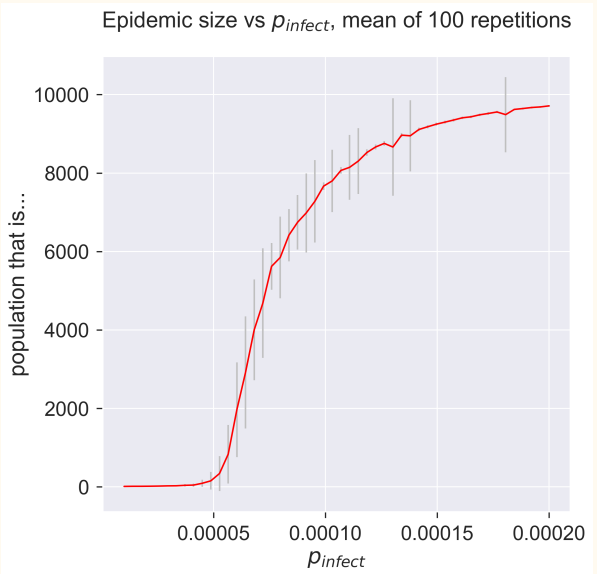
[7] K. Prem, A. Cook, and M. Jit. Projecting social contact matrices in 152 countries using contact surveys and demographic data. *PLOS Computational Biology*, 13(9), 2017. URL https://doi.org/10.1371/journal.pcbi.1005697Ed

ER networks and the epidemic threshold

Erdős-Rényi (ER) networks

- For N nodes build the complete network K_N
- For each edge, retain ("occupy") it with probability p_{infect}
- Leads to p_k normally distributed around $\langle k \rangle = p_{infect} N$

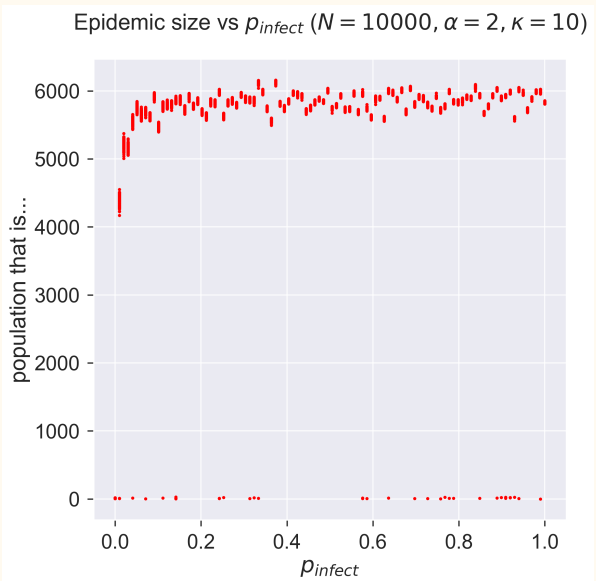


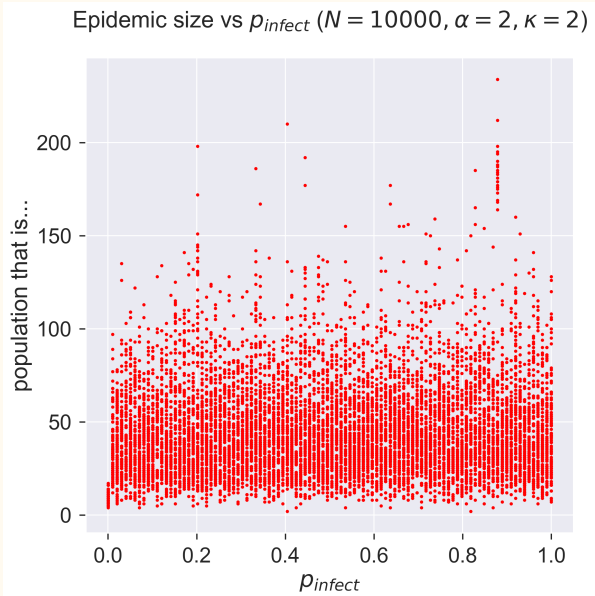


Not all networks behave like this

Too "even" to be a good model of human contacts

- Powerlaw with cutoff, $p_k \propto k^{-\alpha} e^{K/\kappa}$



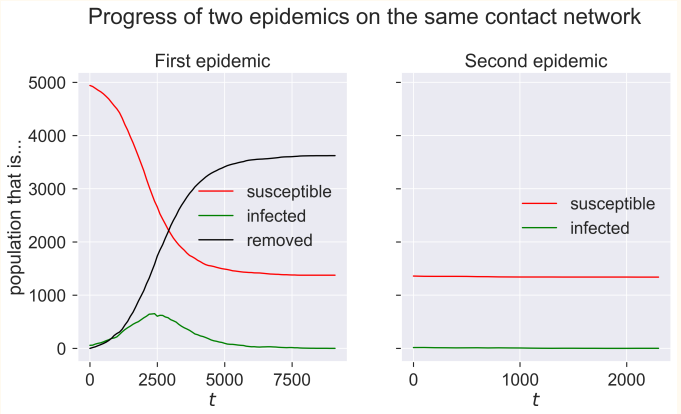


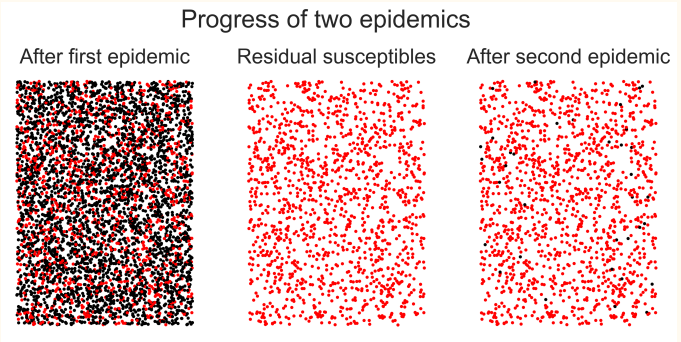
- Relatively insensitive to p_{infect}, but sensitive to α and κ

Herd immunity

Sufficient immune/recovered individuals to stop an epidemic propagating

- Infecteds never adjacent to enough susceptibles
- First epidemic changes the effective topology
- "Effective" $\langle k \rangle$ drops from 20 to 5.5
- (Interested to explore other topological changes)





Why this is a bad idea

Herd immunity has been seriously advocated as a strategy for COVID-19[8]

Ignores some rather inconvenient facts

- A 1% death rate = 700K UK deaths, about one year's excess
- At a rate that would collapse health services
- Immunity may not be permanent – which makes herd immunity behave differently (or not appear at all)
- Long COVID not accounted for in the costs

[8] See the "Great Barrington Declaration", https://gbdeclaration.org

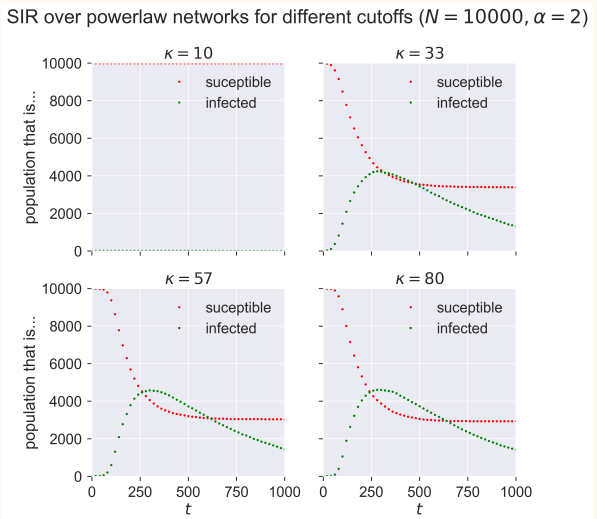
Vaccination

"Herd immunity without the bad bits"

- Aim for the herd immunity threshold, generally about 60% of the population
- …without anyone actually being infected

Epidemic proceeds at different rates depending on topology

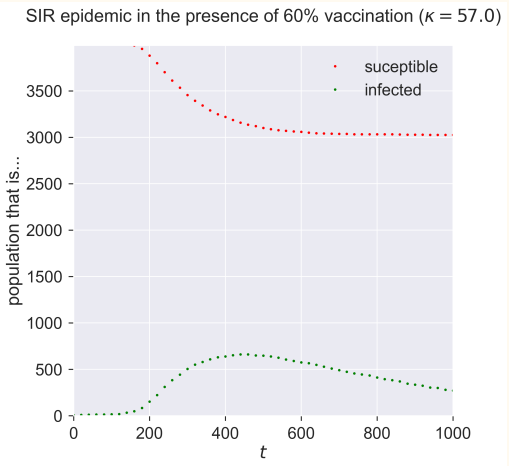
- "Enough" contacts stabilise the size of outbreak



Vaccination strategies

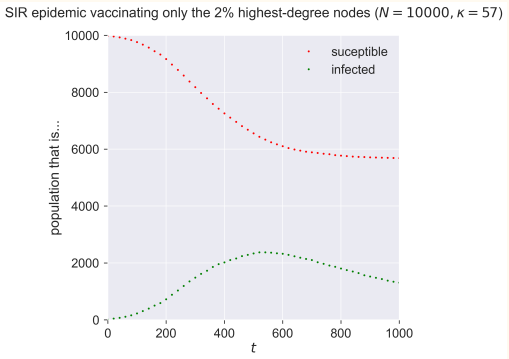
Randomly vaccinate 60% of the population

- Massive reduction in epidemic size
- Only catching high-degree nodes at random
- Sensitive to missing people



If we target vaccination we can reduce the threshold needed to get the same effect

- Target highest-degree 2% of nodes
- Take out the super-spreaders



Physical distancing

What does a physically-distanced network look like?

- Good question
- (I have two SH projects looking at this at University level)

One possible model

- Normally-distributed, fully connected family "bubbles" of mean size 4
- A couple of members with outside contacts
- Exponentially-distributed connections between the contacts in different bubbles

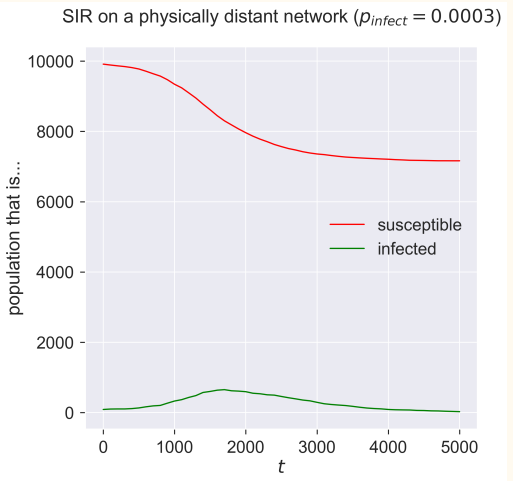
Lockdown changes propagation

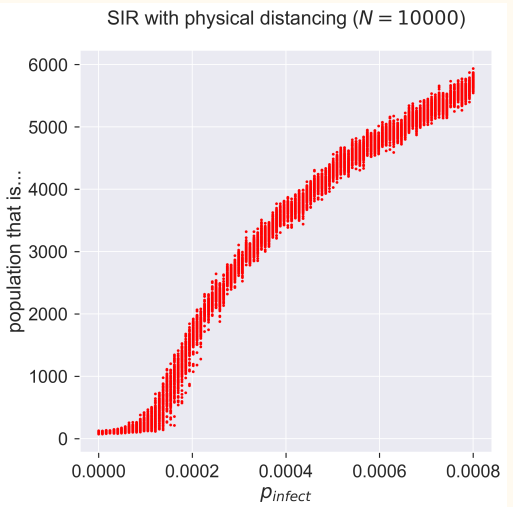
Changes the epidemic threshold compared to an ER network

- Needs a higher infectivity to take off

Slower take-off

- Not like a powerlaw network
- Get bursts of infection if the infection gets into a bubble





Structure of this talk

Asymptomatic transmission

Because covid-19 is essentially SEIR (or maybe SEIRS) it invites other countermeasures

- Self-isolating on showing symptoms is ineffective
- Try to find the asymptomatic carriers

This is the basis for track-and-trace

- Identify contacts of that person
- Quarantine them if they're infected – means we catch infecting individuals before they knew to self-isolate
- Quarantine the symptomatic individual too

Track and trace in practice

A large-scale procedure, unlike the local procedure of self-isolation when symptomatic

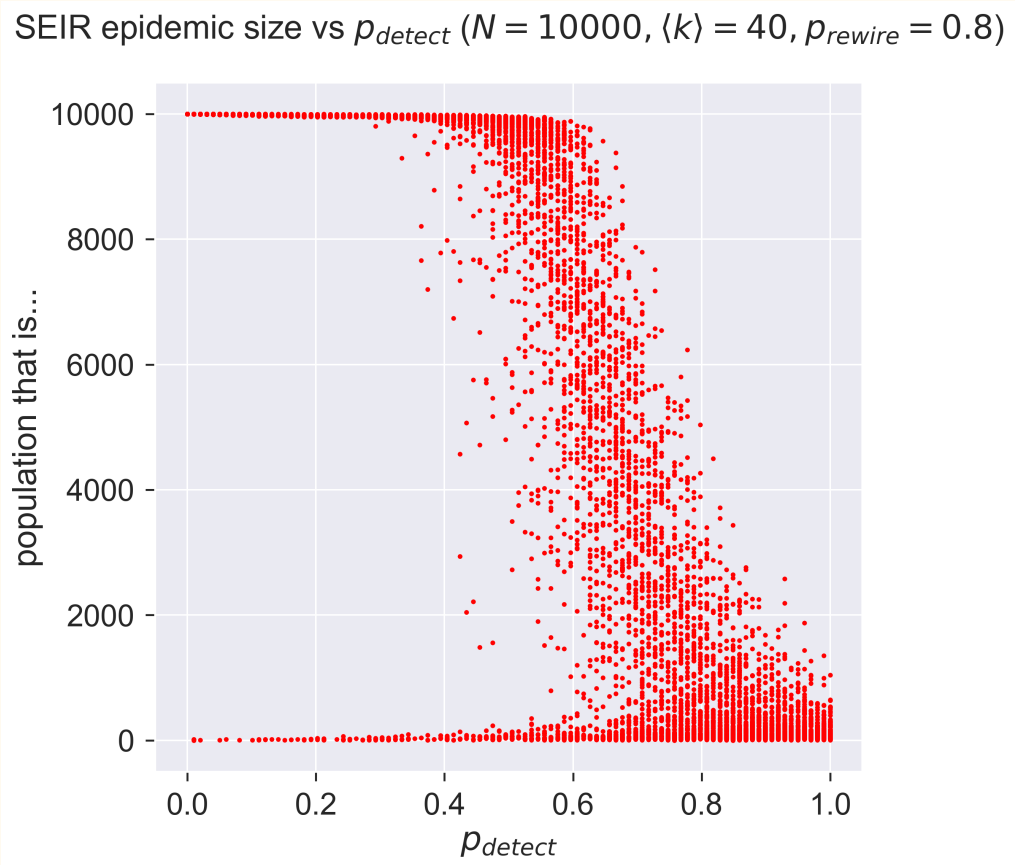
- Requires organisation by some authority
- What can possibly go wrong?…

Unlikely to be fully accurate even if done competently

- Some proportion of people don't quarantine? (p_{rewire})
- Only test some proportion of contacts? (p_{detect})

The impact of detection rates

Hold p_{rewire} constant and vary p_{detect}



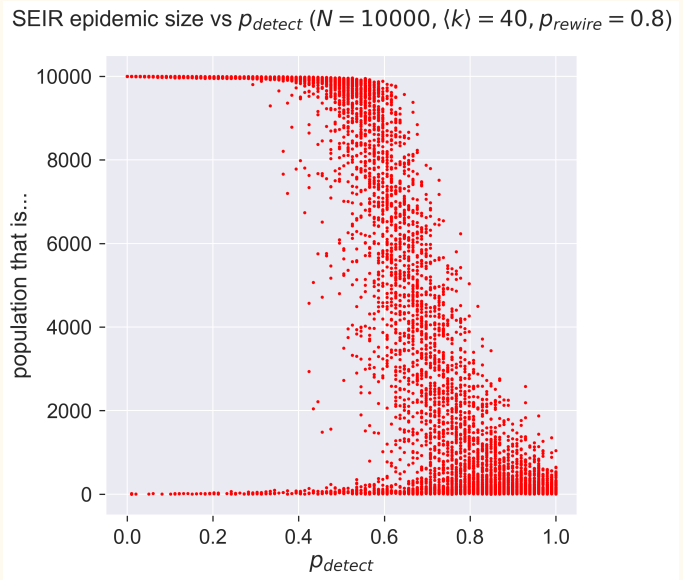
Interpreting the plot

Very dependent on detection rate

- Quarantine can still sometimes damp epidemic anyway
- High detection is very effective

Seems to be unstable in the mid range

- All sizes of epidemic are possible
- No clean break
- Phase transition is "smeared out" rather than occurring cleanly



What's happening?

This phenomenon only entered the literature last year [9]

Possible explanations

- It's an artefact of ER networks
- It's an artefact of not simulating long enough to crush the variance
- It's a function of network fine structure like clustering
- SEIR propagation is really driven by asymptomatic infection

[9] L. Hébert-Dufresne and A. Allard. Smeared phase transitions in percolation on real complex networks. *Physical Review Research*, 1, August 2019. URL https://doi.org/10.1103/PhysRevResearch.1.013009

Possible implications

Designing track-and-trace

- Very sensitive to the proportion of contacts tested
- Need to check at least 40% to have any effect at all

Effectiveness

- Even very effective tracing doesn't guarantee elimination
- Although it does reduce the peak significantly once above about 80%
- Understanding the smearing might let us improve the test strategy

Four things to take away

1. Epidemic spreading still isn't fully understood, even now – there's lots of exciting work still to do, mathematically and computationally
2. Explore of possible public policy decisions
3. Need (in my copious free time) to understand what's going on with this possible smeared phase transition
4. Especially interested in how small-scale topological structures affect network-based processes

References

S. Dobson. *Epidemic modelling – Some notes, maths, and code*. Independent Publishing Network, 2020. ISBN 978-183853-565-0. URL https://simoninireland.github.io/introduction-to-epidemics/.

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L. Hébert-Dufresne and A. Allard. Smeared phase transitions in percolation on real complex networks. *Physical Review Research*, 1, August 2019. URL https://doi.org/10.1103/PhysRevResearch.1.013009.

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K. Prem, A. Cook, and M. Jit. Projecting social contact matrices in 152 countries using contact surveys and demographic data. *PLOS Computational Biology*, 13(9), 2017. URL https://doi.org/10.1371/journal.pcbi.1005697Ed.

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