Contagion – Disease Spreading Modelling as Complex Networks

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Seminar Presentation – Complex Network Dynamics

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Introduction What's the importance of understanding how disease spread?

Past two years of COVID-19 pandemic showed the importance of disease control for reaching different public health goals (i.e., prevent infections until a vaccine arrives, limiting stress on healthcare system, …)

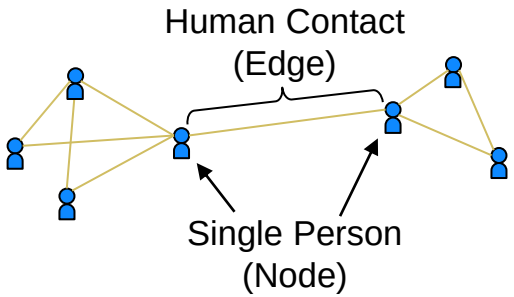
But to control a disease, the infection dynamics need to be tangible. Information can be…

- Predicting epidemic parameters like the reproductive number $R_{(0)}$
- Whether and when to expect threshold behavior
- Having a (holistic) model that emulates the real scenario as good as possible

Contents

Contagion Model On An Individual Level [1] (or Local Diffusion)

Individual Level = Looking at disease transmission from person to person



Now, assumptions have to be made, i.e.

- Social network is scale-free
- Disease grants immunity after infection
- Infinite-size population

SIR model

Case 1
(May & Lloyd)[1]

Negation of these statements can also theoretically be modelled!

[1] Infection dynamics on scale-free networks: May & Lloyd (2001)

Contagion Model On An Individual Level [1] (or Local Diffusion)

Equations that govern this model:

$$\frac{dx_i}{dt} = -x_i \cdot (\sum_j \beta_{ij} \cdot y_j)$$

$$\frac{dy_i}{dt} = x_i \cdot (\sum_j \beta_{ij} \cdot y_j) - \gamma \cdot y_i$$

Infection Rates

Recovery Rate

$$\gamma = \frac{1}{D}$$

with D average duration of infection

x_i : fraction of nodes susceptible with i neighbours

y_i : fraction of nodes infected with i neighbours

Differentiation between node connectivity (nodes with i neighbours) because later calculation done dependent on this

[1] Infection dynamics on scale-free networks: May & Lloyd (2001)

Contagion Model On An Individual Level [1]

Case 1
(May & Lloyd) [1]

Key figure of epidemiological theory:

basic reproductive number R_0
(average secondary infections)

Exhibits **threshold behaviour**
$R_0 > 1$ leads to epidemic outbreak

For defined heterogenous networks:

$R_0 \rightarrow \infty$

with

$\rho_0 = \beta D < k >$

(average nr of infections by single infected into naive population)

For scale-free network with infinite population, $R_0 \rightarrow \infty$ so an **outbreak can always occur**

$C_V \rightarrow \infty$ due to variance being infinite

(coefficient of variation of connectivity distribution)

[1] Infection dynamics on scale-free networks: May & Lloyd (2001)

Contagion Model On An Individual Level [1]
Further important metrics…

Final epidemic size I (total number of infected during an epidemic)

$$I = < 1 - e^{-k\alpha} > \text{ where } \alpha = \frac{<k(1-e^{-k\alpha})>}{<k>^2}$$

Case 1 (May & Lloyd) [1]

Scale-free network ($\rightarrow P(k) \propto k^{-\nu}$)

Take **contineous** distribution $P(k) = 2m^2/k^3$ (where m connectivity of least connected nodes)

$$I = 2m^2 \int_m^\infty dk \frac{1}{k^3} (1 - e^{-k\alpha})$$

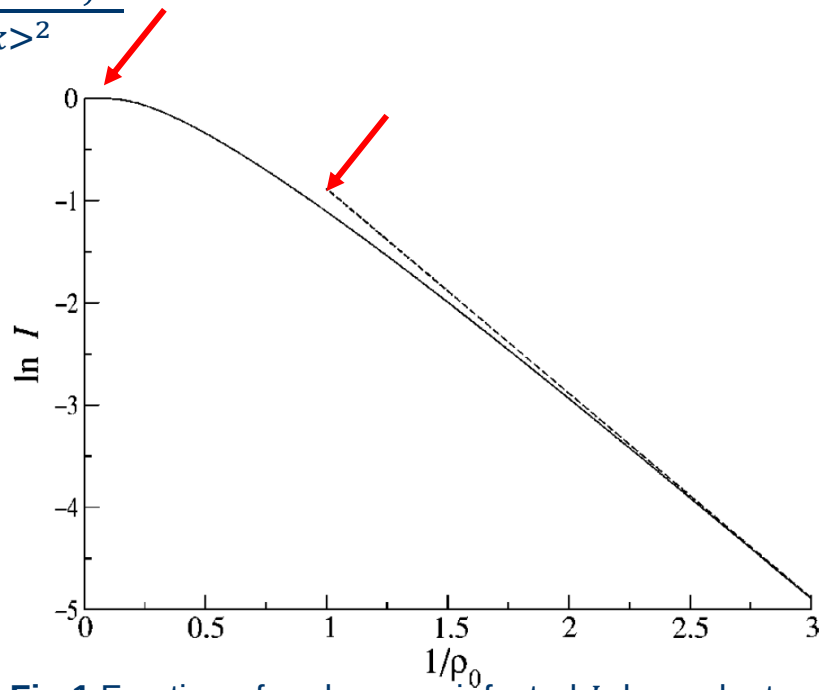


Fig 1 Fraction of nodes ever infected I dependent on $1/\rho_0$ for Case 1 with $\nu = 3$ and $m = 4$, dotted line being approximation. Graphic from May & Lloyd [1].

[1] Infection dynamics on scale-free networks: May & Lloyd (2001)

Contagion Model On An Individual Level [1]
Further important metrics…

Fraction of Nodes ever infected and differentiating between connectivity k

→ set of nodes with lower connectivity have mostly significantly fewer infections

→ at certain connectivity, practically all nodes will be infected (depending on ρ_0)

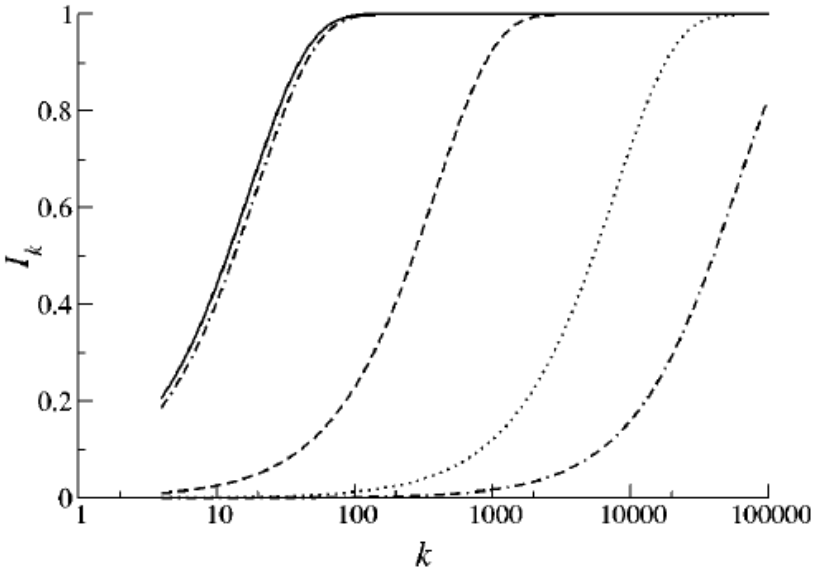


Fig 1 Fraction of nodes ever infected I for different connectivities for Case 1 for $\rho_0 = \{1, 0.4, 0.25, 0.2\}$ from left to right, dotted line being approximation. Graphic from May & Lloyd [1].

[1] Infection dynamics on scale-free networks: May & Lloyd (2001)

Contagion Model On An Individual Level [1]

Further notable conclusions:

- Fewer individuals with low-connectivity while mostly all individuals with high-connectivity are infected during an epidemic
- Effects of finite-size populations lead to finite R_0 and exhibits threshold effects
- Discretisation of population (individuals, no continuum) and assortative mixing (individuals tend to interact with individuals with similar connectivity) also needs to be assessed

But this model is still deterministic, not stochastic!

[1] Infection dynamics on scale-free networks: May & Lloyd (2001)

Contagion Model On A Global Scale [2]

Perspective so far on temporal development…

What about geographical spread and occurring spatial effects?

What about transmission and recovery being stochastic and lack of fluctuation?

Describing this with probability $p(s, j; t)$ of finding s susceptible and j infected in a population (size N) at time t with…
(assuming process is Markovian)

Master equation

$$\partial_t\, p(s,j;t) = \frac{\beta}{N}(s+1)(j-1)\ \cdot p(s+1, j+1; t) + \gamma(s+1)\cdot p(s, j+1; t) - \left(\frac{\beta}{N} sj \cdot p\ - \beta \cdot j\right) p(s,j;t)$$

+ initial conditions

[2] Forecast and control of epidemics in a globalized world: Hufnagel L, Brockmann D, Geisel T (2004)

Contagion Model On A Global Scale [2]

For large but finite population, one can approximate (for large but finite populations):

Deterministic

$$\frac{ds}{dt} = -s \cdot \beta \cdot j$$

$$\frac{dj}{dt} = s \cdot \beta \cdot j - \gamma \cdot j$$

$$(x \to s\,; y \to j)$$

(for comparison)

Stochastic

Gaussian white noise (fluctuations)

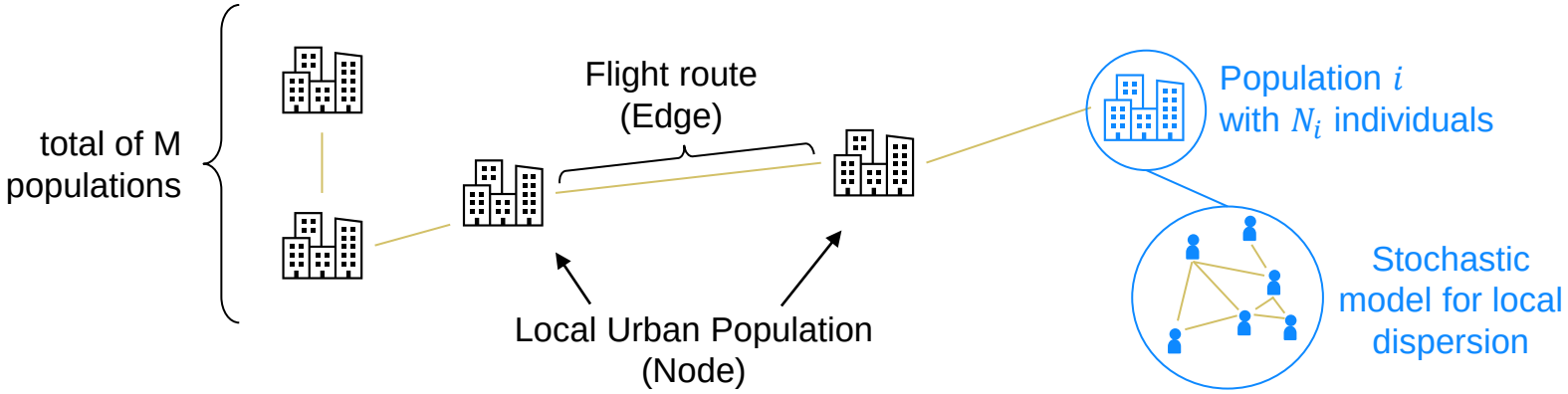
$$\frac{ds}{dt} = -s \cdot \beta \cdot j + \frac{1}{\sqrt{N}}\sqrt{s\beta j} \cdot \xi_1(t)$$

$$\frac{dj}{dt} = s \cdot \beta \cdot j - \gamma \cdot j - \frac{1}{\sqrt{N}}\sqrt{s\beta j} \cdot \xi_1(t) + \frac{1}{\sqrt{N}}\sqrt{\gamma\, j} \cdot \xi_2(t)$$

[2] Forecast and control of epidemics in a globalized world: Hufnagel L, Brockmann D, Geisel T (2004)

Contagion Model On A Global Scale [2]

Global Scale → quantifying the dispersals through aviation network



Transition from population i to j with transition matrix Ψ_{ij}
+
Assuming, individuals remain in urban area for some time
+
Time dependent $\rho_0(t)$

Case 2 (Hufnagel, Brockmann & Geisel)

[2] Forecast and control of epidemics in a globalized world: Hufnagel L, Brockmann D, Geisel T (2004)

Contagion Model On A Global Scale [2]

Case 2
(Hufnagel, Brockmann & Geisel) [2]

To validate model:

1,000 simulations were run and final epidemic size $< I(t) >$ was calculated at each node i

+

Comparison with SARS epidemic 2003 (outbreak in Hong Kong)

Results show:
for two confined populations A (infected) & B (uninfected) and transition rate ψ:

Probability of outbreak

$$p(\psi) = 1 - exp(-\frac{\psi}{\psi^*})$$

with ψ^* critical transition rate

[2] Forecast and control of epidemics in a globalized world: Hufnagel L, Brockmann D, Geisel T (2004)

Contagion Model On A Global Scale [3]

For similar, more recent model (Brockmann & Hebling[3]), an additional figure is introduced, **the effective distance d_{nm}**

Idea: process is governed by set of most probably paths, reducing the redundancies and multiplicity of paths

→ $d_{nm} = (1 - \log(\Psi_{nm}) \geq 1$ with Ψ_{nm} representing the flux of passengers from n to m

$d_{nm} \neq d_{mn}$

[3] The Hidden Geometry of Complex, Network-Driven Contagion Phenomena: Brockmann D, Helbring D (2013)

Contagion Model On A Global Scale [3]

Graphic representation of effective distance reveals the otherwise not noticeable wavefront of epidemic;
here modelled for disease starting in Hong Kong

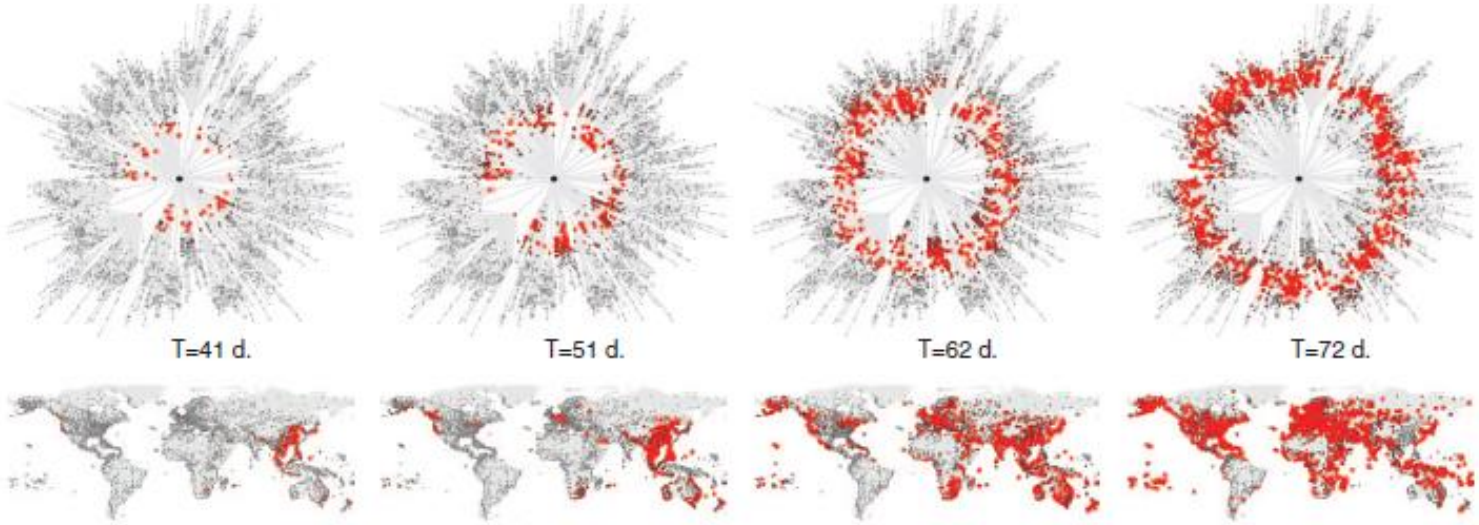


Fig 2 Visual representation of spread of simulated disease, redness signaling significant prevalence, with outbreak in Hong Kong. Representation with effective distance (top, radial distance equals effective distance) or geographical (bottom). Graphic from Brockmann & Helbring [3].

[3] The Hidden Geometry of Complex, Network-Driven Contagion Phenomena: Brockmann D, Helbring D (2013)

Contagion Model On A Global Scale [3]

Fig 3 Visual video-based representation of spread of simulated disease, redness signaling significant prevalence, with outbreak in Atlanta. Representation with effective distance (left) or geographical (right).
Video via https://rocs.hu-berlin.de/project/viz-event-horizon/ by Dirk Brockmann; referencing Brockmann & Helbring [3].

[3] The Hidden Geometry of Complex, Network-Driven Contagion Phenomena: Brockmann D, Helbring D (2013)

Derived Containment Strategies [1,2,3] (selection only)

Local duffison processes

- Containment strategies most effective when targeted towards highly connected individuals (vs random)
- Using for example vaccinations to reduce the effective reproductive number below threshold of 1

On Global Scale

- Global Mobility Network can be used to estimate percentage of vaccinations needed to prevent an epidemic spread
- Quick response to initial outbreak necessary to prevent need for global vaccination
- Prediction of arrival time can help estimate when to impose preventive containment measure

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[2] Forecast and control of epidemics in a globalized world: Hufnagel L, Brockmann D, Geisel T (2004)
[3] The Hidden Geometry of Complex, Network-Driven Contagion Phenomena: Brockmann D, Helbring D (2013)

Gillespie Algorithm And Spreading Models [4]

Previous stochastic models often used equidistant time steps and transition rates and looked at whether a **possible event took place after that time increment** (Monte-Carlo Simulation)

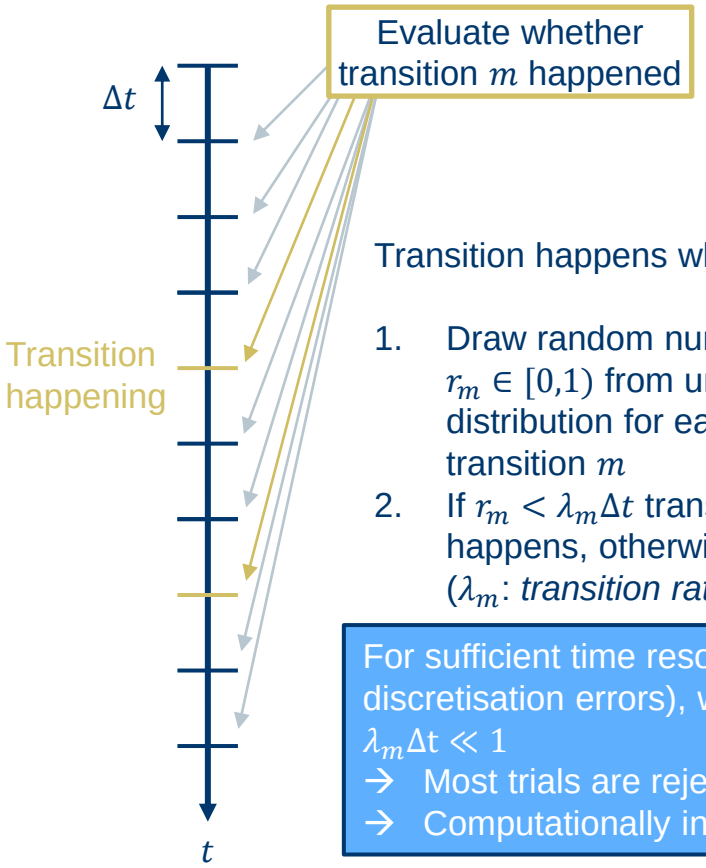
Gillespie Algorithm instead replicates a continuous-time process and uses the **time until the next event** takes place and therefore improving computational time

However, application to non-static networks (such as spreading) not trivial, yet possible

[4] Temporal Gillespie Algorithm: Fast Simulation of Contagion Processes on Time Varying Networks: Vestergaard CL, Génois M (2015)

Gillespie Algorithm And Spreading Models [4]

Monte Carlo

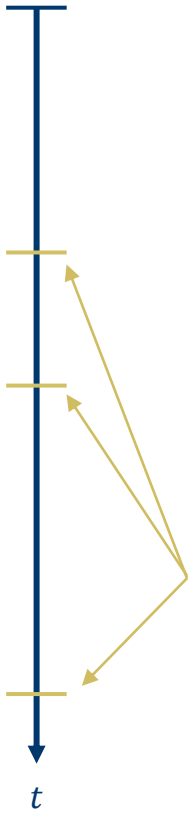


Transition happens when:

1. Draw random number $r_m \in [0,1)$ from uniform distribution for each transition m
2. If $r_m < \lambda_m \Delta t$ transition happens, otherwise not. (λ_m: *transition rate*)

For sufficient time resolution (→ no discretisation errors), we require $\lambda_m \Delta t \ll 1$
→ Most trials are rejected
→ Computationally inefficient

Gillespie Algorithm



For Poisson processes in **static** networks, probability that transition m **didn't** happen after time τ:

$$S_m(\tau) = e^{-\lambda_m \tau}$$

λ_m: transition rate

Probability, that no transitions happened after τ:

$$S(\tau) = e^{-\Lambda\tau} \text{ with } \Lambda = \sum_{m=1}^{M} \lambda_m$$

Cumulative transition rate

Draw τ from distribution of waiting times τ (probability density):

$$p(\tau) = \Lambda \cdot e^{-\Lambda\tau}$$

or for transition m:

$$p(\tau) = \lambda_m \cdot e^{-\Lambda\tau}$$

[4] Temporal Gillespie Algorithm: Fast Simulation of Contagion Processes on Time Varying Networks: Vestergaard CL, Génois M (2015)

Gillespie Algorithm And Spreading Models [4]

For non-static/Temporal Gillespie Algorithm

Problem: Network and its transitions varying over time, independently of transitions (example survival rate: nodes become infected only when in contact with another infected node → exponential distribution not applicable)

Therefore:

$$S_m(\tau; t^*) = \exp(-\int_{t^*}^{t^{**}} I_m(t)\lambda_m \, dt \,) \text{ with } \mathrm{I_m}(t) = \begin{cases} 1 \text{ for } m \text{ may takes place} \\ 0 \text{ for } m \text{ may not take place} \end{cases}$$

t^*: last transition that took place

$t^{**} = t^* + \tau$: next transition that takes place

If changes in network only depend on process itself, $\mathrm{I_m}(t)$ only changes with transitions and static Gillespie Algorithm can be applied

[4] Temporal Gillespie Algorithm: Fast Simulation of Contagion Processes on Time Varying Networks: Vestergaard CL, Génois M (2015)

Gillespie Algorithm And Spreading Models [4]

Analogously to static Gillespie Algorithm (now time dependent):

Cumulative Transition Rate: $\Lambda(t) = \sum_{m \in \Omega(t)} \lambda_m$

Introduce normalised waiting time: $\tau' = \int_{t^*}^{t^{**}} \Lambda(t)\, dt$

→ $S(\tau') = e^{-\tau'}$ from which we can draw and implicitly have information about time of next transition t^{**}

Then, either…
(1) transition m happens, t^{**} can be calculated and Λ and Ω need to be updated before drawing again or…
(2) τ' needs to be adjusted and redrawn…

depending on where you land on normalised timeline.

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Gillespie Algorithm And Spreading Models [4]

Advantages of Gillespie Algorithm:

- Both, static and non-static are stochastically exact, Monte Carlo technically only for $\lim_{\lambda_m \Delta t \to 0}$
- Significantly faster processing, for exemplary contagion model with only contact-based transmission by factor 2

[4] Temporal Gillespie Algorithm: Fast Simulation of Contagion Processes on Time Varying Networks: Vestergaard CL, Génois M (2015)

Conclusion and Outlook [1,2,3]

Contagion Models exist in various forms like…

… for local diffusion processes:

- where for infinitely large populations no threshold behavior is noticeable
- where final epidemic size can be exactly and approx. calculated

… for today's global scale spreading processes:

- where the modelling of the global mobility network leads to improvement of the quality of prediction
- where relative distance can reveal properties like wavefronts in the network

[1] Infection dynamics on scale-free networks: May & Lloyd (2001)
[2] Forecast and control of epidemics in a globalized world: Hufnagel L, Brockmann D, Geisel T (2004)
[3] The Hidden Geometry of Complex, Network-Driven Contagion Phenomena: Brockmann D, Helbring D (2013)

Conclusion and Outlook [1,2,3]

- Different containment measures then can be deducted from these models, respectively, which in a future step could be evaluated with regards to the current pandemic.

- Furthermore, quality of models and computational time can always be further advanced.

- One problem of interest may be impact evaluation of different containment measures where the current pandemic could again serve as a point of reference

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[2] Forecast and control of epidemics in a globalized world: Hufnagel L, Brockmann D, Geisel T (2004)
[3] The Hidden Geometry of Complex, Network-Driven Contagion Phenomena: Brockmann D, Helbring D (2013)

Conclusion and Outlook [4]

- Additionally, advantages of the Gillespie Algorithm vs rejection sampling models (Monte Carlo) has been shown
- Deeper investigation of the computational superiority could be another topic for future research

[4] Temporal Gillespie Algorithm: Fast Simulation of Contagion Processes on Time Varying Networks: Vestergaard CL, Génois M (2015)

Bibliography

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3) The Hidden Geometry of Complex, Network-Driven Contagion Phenomena: Brockmann D, Helbing D. Science Vol 343 (2013). DOI: 10.1126/science.1245200
4) Temporal Gillespie Algorithm: Fast Simulation of Contagion Processes on Time Varying Networks. Verstergaard CL, Génois M. PLOS Computational Biology (2015). DOI:10.1371/journal.pcbi.1004579