Remarks on Epidemic Spread

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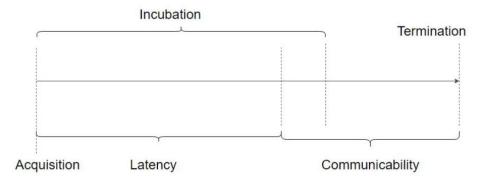


Figure 1: Timeline of an illness presenting latency, incubation, and communicability

Introduction

Epidemic spread may in general depend on both climate related factors such as air temperature, and population related factors such as movement of units between locations.

The article [TJH⁺18] analyses BTV, a disease of ruminants spreading in Northern Europe as a result of Climate Change, introduced for the first time in 2006 and resulting in the eventual infection of thousand of farms across many countries.

The authors show that the UK outbreak could have potentially been much larger had the infection been introduced into the west of England, either directly or as a result of the movement of infected animals from East Anglia before the first case was detected. Hence we are given a first clue that Network Topology, that is, the combinatorial contingency of the geographical placement of locations may play a crucial role in the spread, but also in the containment of animal epidemics.

The concrete explanation together with an in-depth analysis and the full details of the case study that is the starting point for our analysis is found in [TJH⁺18].

The importance of our project is considerable and applications may not be restricted to one particular domain. The context of the problem is amenable to abstraction. The setting may be applied to any problem supposing locations, fixed dwelling places for animal populations, and interactions in the form of movement of units between these locations.

Our intention is for the framework we are developing to be applied to illnesses presenting disjoint periods of latency and communicability, and periods of incubation.

During the latency period, if specific to the illness in question, the illness is acquired and not transmittable.

During the incubation time individuals infected do not display any sign of the illness and the illness is not detectable macroscopically.

During the communicability time, a period that may overlap in part with the period of incubation, individuals infected may or may not display any sign of the illness while the illness becomes transmittable.

During our initial consultations and assessment of the problem, it has become clear we need to place a large amount of our efforts on the analysis of the combinatorial properties of the networks and of the processes that act upon these networks.

To be quite concrete, as in a strategic board game, some maps may be good, some may be easy, some may require great skill in managing the associated problems, and so on. The specific details we are and we plan to be concentrating on are the main matter for the present report.

Attainable Objectives

Our discipline and approach is mathematical in nature, with focus on Discrete Processes and Combinatorics. Hence the main subject of our investigation is the structural assessment of epidemic outbreaks, the interactive process that leads to these outbreaks, together with the topology of the networks on which these interactions take place.

One aim is to present a model of disease detection within this setting such that the model obtained has both explanatory power, in the sense of tracing the history of an illness within a geographical area, which may also be used for prediction and containment purposes.

We study disease spread as flowing through a particular type of connected temporal network, also known as a temporal graph.

We are given a geographical map in the form of a list of geographical coordinates that essentially presents the spatial information relating to the fixed locations we are considering.

Therefore, in its abstract form, our problem generalizes to any setting in which we have locations, that is - spatially-limited dwelling places for animal populations, and animal movement between these locations.

We aim to model disease spread within this setting such that the model obtained has not only explanatory power, but may also be used for prediction purposes.

For example, we may isolate or identify problem parameters which can be global or local, such that one can react in real-time when an epidemic breaks and through a process of optimization of these parameters one can isolate infected locations effectively such that the outbreak will be minimal. Spread can be modelled with several different layers of structured data, e.g.

- unit-disk graphs for geometric locations U
- temporal graphs of movements M
- aggregated temporal graph of the dynamics of spreading $U \circ M$
- a probabilistic generalization of this model, comprising probabilistic state transitions.

Two of the questions we posed during our initial assessment for which we expect to design efficient solutions are the following.

Is it possible to "localize" the source of an infection in real time, by observing detection data on the given graph?

Hence, is it possible to predict the fact that other locations are infected, by observing a detected location at a given time?

The main application of our study is in policy-making. One of our earliest observations was that locations on the underlying map may hypothetically be arranged in a way that minimizes spread of disease.

Furthermore, identifying certain preferable network topologies, both for the underlying map of locations and for the economic exchanges between them may dictate rigorous strategies in performing trade between locations.

We shall attempt a description of our problem in terms of game theoretical terminology below.

Background of Research in the Area

The earliest efforts towards a mathematical analysis of epidemics are found in Bernoulli [DH00] who used statistical methods to analyze smallpox and identified a cyclical behavior in the spread of epidemics.

An early serious attempt towards a rigorous model of transmission is found in a paper by P.D. Enko published in Russian in 1889 [Die88].

The modern mathematical theory of epidemics is one century old now. Hamer and Soper elaborate on the periodical nature of disease prevalence [Sop29].

We also mention the early work of Kermack and McKendrick [KM91b, KM91a, KM91c]. Compartmental models, that is, models assuming a change of states between susceptibility, infection and possibly recovery originate in these early attempts.

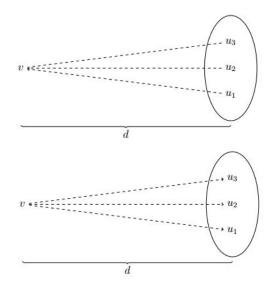
Furthermore, Ross elaborates at the beginning of the last century on the laws governing malaria and fifty years later Macdonald develops a quantitative theory of control that Ross had been attempting [SBH⁺12].

Comparisons between concepts in computing and epidemic spread are found more recently, and go in both directions, including an analysis of databases update [DGH⁺87] and applications of information, gossip and rumor dissemination within a network [DK64, Bum81].

The structural properties of the potential spread due to local interaction and global movements could be described by a model of Temporal Graphs. The main idea here is that edges of a graph are augmented with labels specifying the time when they are enabled. A temporal journey through this graph then becomes a path respecting the natural order of time. The classical problems of Graph Theory translate to Temporal Graphs resulting usually in harder computational problems [Mic16]. Temporal Network Epidemiology is one of the newest domains under the umbrella of disciplines that propose a mathematical study for epidemic spread. The monograph [ABVdDW08] already contains a chapter on Network Epidemiology, while Masuda and Holme edit a collection of papers specifically on this topic [MH17].

Our locations grid may be indeed viewed as either an online, or offline, temporal graph in which edges between locations are available at all times if the radius between them is very small, and other edges become available as soon as there exist movement of units between locations. Probabilities may be added to the edges in the spirit of the model presented in [TJH⁺18]. A (probabilistic) journey through this temporal graph will then correspond to a possible scenario of spread.

Considering the fact that the source of an illness may relate to both vector transmission due to geographical proximity of locations, and animal movements, we need to be able to compose the disk graph representing the spatial coordinates of our locations with the map that represents the movement between these locations. The resulting structure is a temporal graph in which we allow some of the edges to have permanent life-time. Connectivity for Temporal Graphs is studied in [KKK02]. Temporal exploration is a very debated topic in the area [EHK15]. Viewing specific problems at a higher resolution, the technique found for example in [CKNP18] may be used for efficient computation of certain network properties on which viral propagation may depend. The mentioned paper uses the ladder technique to compute very efficiently the diameter of a temporal graph, among other properties. The phenomenon of intractability may be avoided in searches by only considering a temporal radius dictated by the periods of Latency and Incubation for the illness.



We assume an indivisible temporal radius d on which the transmission mechanism is enabled. This means in practice that searching for infected locations must halt after a fixed period of time, making such a search procedure fixed-parameter tractable in the number of locations with Incubation time as parameter.

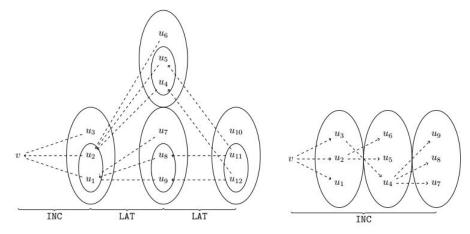
Figure 2: Inner and Outer Operators

Initial Elaborations

Classical Patient-zero. History of Spread. Radius of spread. Prediction We are in the possession of a formal description of classical epidemiological detection applied in the context of Temporal Network Epidemiology. Our algorithm comprises of two stages: the back-detection phase, aiming at building an accurate past history of disease spread within the network, and a forward-detection phase, aiming at identifying the compromised locations on the network, i.e. the nodes of the network in direct and indirect contact with a source of infection, after the source has acquired the illness in question.

The patient zero problem refers to tracing the primary origin of spread within a certain geographical area, while predictions regarding the possibly exposed locations can be made with a positive degree of accuracy. We elaborate on the process of detecting a given source of infection given there exist one or several detected locations.

We outline the procedure for approximating a *history of spread*, that is, a time-line for a given illness, including a solution for a possible "patient zero" problem and prediction of possibly exposed locations.



Detection mechanisms are well-understood in Epidemiology. What often makes this task harder is lack of precise surveillance information, which in our case does not constitute an issue.

Figure 3: Back detection (Patient Zero), followed by forward prediction (Detection)

The classical procedure is as follows.

- given a macroscopically infected location, trace its contacts with other locations, on a temporal radius of roughly the Latency period of the illness
- repeat the previous step for all locations found, until no other locations can be found within the network
- now trace the contacts the identified locations have had meanwhile, forwardly, again sensitive to the relevant temporal radius, function of Incubation and Latency.

Other attempts at a Patient-zero problem are complicating the issue somewhat and sometimes attempt to model situations with imperfect information [ABD⁺14].

In our case however, computing these potential infections is not expensive in terms of resources and we may safely say that isolating possible infections may be done with a very high degree of accuracy.

Regarding infections by proximity, that is, an infected location lying too close to healthy locations, it is possible to identify locations lying within a certain radius of spread.

Intuition suggests there exists, for each location and each period of time $t - t_0$, a manageable geographical radius of spread beyond which infection started at time t_0 will not have spread by the time t.

Relativized Patient-zero. Viral strains Next, we define and analyze two problems that can be viewed as relativizations of the patient-zero problem in a temporal graph with respect to a set of vertices of the graph. We observe the source of non-vector infection for a given location to be another location that sent units to the first, such that there exists a temporal journey between the two, at a temporal distance equal roughly to the latency period of the illness.

A least temporal source for two vertices is a set of vertices that lie the closest on journeys to the two vertices.

Define the least temporal source for a set of vertices as the intersection of least temporal sources for the vertices in the set, taken pairwise.

A greatest temporal source for two vertices is a set of vertices that lie the farthest on journeys to the two vertices.

Define the greatest temporal source for a set of vertices as the intersection of greatest temporal sources for the vertices in the set, taken pairwise.

Therefore we are able to define the corresponding problems of finding the set of least and greatest temporal sources for a given set of nodes in the Temporal Network.

These problems are related directly to the concept of relativization of the patient-zero problem, in the intuitive sense that the set of least temporal sources will most likely contain locations infected with strains most common to the nodes we take as input, while the set of greatest temporal sources, if lying at a temporal distance greater than the latency period, will contain the patient-zero relative to all of the input nodes.

Numerical figures The data is contained in files outlining the geographical locations, and the transactions between locations, both for the year 2013. As outlined previously, a search was performed on the data and the following was found. There were 110,000 farms recorded. A number of distinct animal moves of 2,658,619 and a number of non-distinct animal moves of 3,336,089. Distinct here means repeated pairs were ignored. The number of sending farms was 72,200 while the number of receiving farms was 52,685, out of which the number of distinct senders and receivers was 82,127. Starting on some farm we proceeded with DFS and restarted the search when no more farms could be reached, iterating this process until all of the farms were visited by the search. We have found 9,115 components on the receiving farms, while 19,814 components were found on the sending farms.

One Preliminary Analysis

Incubation As usual we assume a period of incubation for the illness. During this period, an infected unit cannot be easily detected through macroscopic observation. Hence a possible infection cannot be contained in a cost-effective manner.

Infection Infection may be due to animal movements, or due to proximity of locations. In the case of infection through proximity we assume the radius of infection on which we elaborated above.

Rounds Trade between locations will hence only be done in rounds. The first round of unit moves is done at time t_1 while the second at time $t_2 \ge t_1 + d + d'$. In general, $t_k \ge t_{k-1} + d + d'$, where the value of d depends on the Incubation time for the illness while the value of d' will be a function of the Incubation time for the nodes lying the farthest away from the infection epicenter but still within the geographical radius defined above.

Infection response When and if locations become detected, these detected locations are taken out of the market for a suitable number of rounds,

until they have fully recovered, after which they will be allowed to trade again. The goal of this Epidemic Game is to schedule the next round of trade between locations in such a way as to allow the Network to recover from potential infections.

Question Are we able to formulate a strategy minimizing the number of infected locations?

Formalization We are given the disk graph and we may be given the movements graph in off-line or on-line form. The movements graph specifies the order of the moves between locations. The game has one player, the Controller, having access to the movement file, acting against a randomized Environment. After each move from the movements graph, the Environment chooses randomly one of the two actions available: they may choose to label a location as infected, or they may not exercise this action. Player Controller decides the exact point in time the next round of trade will take place. Constraints may be placed that result in a form of censorship for the actions of the Controller.

Game Description Starting with the specified disk graph, the game is described as follows.

- rounds are indexed by the time of their occurrence;
- in any round, movements occur between locations according to the movements graph;
- for each movement (u, v) within the round the Environment specifies randomly if this results in an infection of v by u;
- any infected location stays infected for a period of time determined by a fixed recovery parameter;
- an infection in a round n becomes macroscopic only at time n + d where d is the Incubation time, in which case the count of the loss function is incremented;

- any location which is macroscopically infected is taken out for a number of rounds that span a period of time determined by a given recovery parameter;
- the task of the Controller is to flag locations as "exposed", and decide when the next round of trade will occur,
- it is assumed that locations flagged are taken out of trade, but also that it is impossible for a flagged location to infect by proximity.

In specifying a strategy we are seeking to give the values for a scheduling and a flagging that minimize some possible loss. An initial theoretical analysis of this framework pinpoints three types of strategy.

Strong apriori strategy A strong apriori schedule minimizes the value of the loss globally. The Controller has no access to the movements file in this setting.

Apriori strategy An apriori strategy minimizes the value of the loss globally. The Controller has access to the movements file in this setting in an offline fashion.

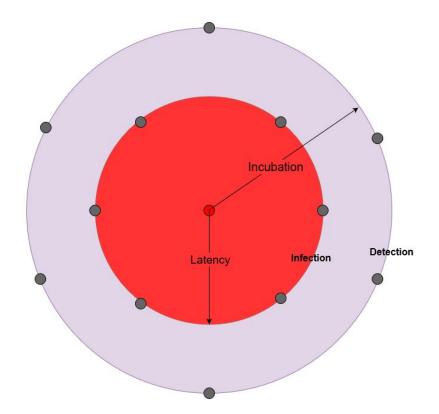
Greedy strategy A greedy strategy minimizes the value of the loss locally. The Controller has access to the movements file in this setting in an online fashion.

Questions How to compute apriori and greedy strategies? How to compute them efficiently?

Example For any detected node in the graph, we are able to find the nodes within the radius of spread relative to the time interval since the node was infected, and estimate their time of infection; to the most recent time of potential infection, we add the time of Incubation of the illness, repeat this operation for every node in the graph and take the maximum value calculated, to obtain the parameter Δ . Let the trivial strategy be

specified by the schedule function adding to the time of the current round the amount of time corresponding to this Δ , while flagging as exposed every node in the radius of spread of already detected nodes.

The trivial strategy is greedy; since the time interval between rounds exceeds the incubation time of the latest infected location, potential infections are flagged before the time of the next round of trade.



The key note here is that performing Rounds of Trade within the temporal equivalent of the red area is a factor contributing to Epidemic spread.

Figure 4: Relationship between Detection and Latency.

We identify the worst-case scenario for the Trivial Strategy. This corresponds to performing Rounds of Trade on or before the Detection time for the illness.

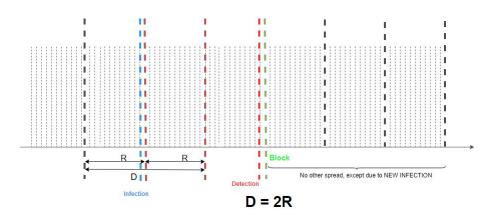


Figure 5: Trivial strategy analysis. Worst case variant.

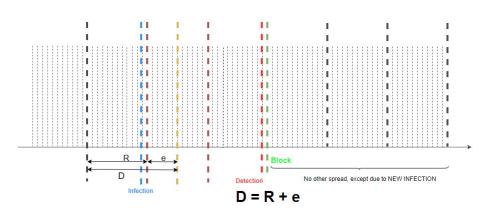


Figure 6: Trivial strategy analysis. Worst case variant.

The Optimal scenario is performing trade after the period of Detection has passed.

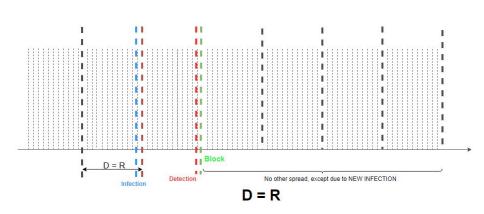


Figure 7: Trivial strategy analysis. The optimal case.

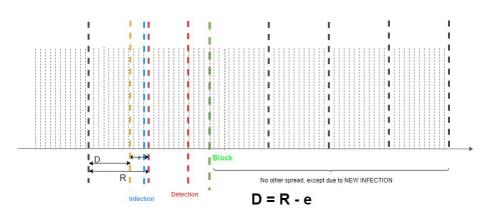


Figure 8: Trivial strategy analysis. Best case variant.

Axiomatic Epidemiology

Since there exist Mathematical Theories that study the domain, it is but natural to attempt Axiomatizations of Epidemic Spread.

Some of the questions one is hypothetically able to answer under the model we construct are the following.

do given observations imply a given hypothesis, e.g. there exist "natural immunity" to an infection?

is a given hypothesis, e.g. the patient zero hypothesis, contradictory with a given theory of epidemics?

is a certain scenario, e.g. Armageddon, possible?

how likely, under a certain theory of epidemics and given a set of observations, is a certain scenario, e.g. Armageddon?

does there exist a computable necessary and sufficient condition for a given scenario?

can this condition be tractably decided?

Basic notions

Primitives

units Units are undividable biological macro-organisms.

$$u, u', u'' \dots v, v', v'' \dots$$

time Temporality is discrete. Time instances are natural numbers.

$$t, t', t'' \dots$$

We make use of the following integer parameter

NOW

This parameter divides time into, roughly-speaking, three equivalence classes.

probabilities computable reals ranging in [0, 1].

$$p, p', p'' \dots$$

populations collections of units.

 $\gamma, \gamma', \gamma''$

clusters collections of populations.

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\Gamma, \Gamma', \Gamma'' \dots
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regions collection of clusters.

$$\rho, \rho', \rho' \dots$$

continents collection of regions.

$$P, P', P'' \dots$$

universe of discourse collection of continents.

Υ

Primitive predicates unit v is susceptible at time t

unit **u** is exposed to unit **v** at time t

unit u is infected by unit v at time t

unit **v** is detected at time t

D(v,t)

We note the operator immunized, which may be taken either as primitive or as a redundant operator – unit v is immunized at time t

$$V(v,t) \equiv \neg S(v,t)$$

Temporal predicates

temporal overlap We provide a basic treatment of temporality under which time intervals may overlap.

$$O(\gamma, \gamma', t) = \{ u \colon \exists t' < t [u \in \gamma(t) \land u \in \gamma'(t')] \}$$

population overlap Note that populations may overlap, in a certain temporal sense. These overlapping conditions are the most fundamental factors for disease spread.

$$O(\gamma, \gamma') = \{u \colon O(\gamma, \gamma', NOW)\}\$$

Axioms on units

definitions to be exposed at time t is to be susceptible at an earlier time

$$E(v, u, t) \implies \exists t' < t[S(u, t')]$$

to be infected at time t is to be exposed at an earlier time

 $I(v, u, t) \implies \exists t' < t[E(u, t')]$

to be detected at time t is to be infected at an earlier time

$$D(v, u, t) \implies \exists t' < t[I(u, t')]$$

Spread

susceptibility susceptibility

$$\forall u \exists t < NOW[S(u, t)]$$

immunity strong immunization

$$V(u,t) \implies \forall t' > t[\neg S(u,t)]$$

weak (seasonal) immunization

$$V(u,t) \implies \exists t' \forall t'' \in [t,t'][\neg S(v,t'')]$$

exposure exposure is reflexive

$$\forall t[E(v,v,t)]$$

exposure is symmetric

$$\forall t[E(v,u,t) \implies E(u,v,t)]$$

infection infection is weakly-transitive

$$\forall t < t' < t''[I(v,v',t) \land I(v',v'',t') \implies I(v'',t'')]$$

Axioms on populations

Definitions

$$E(v,t) \longleftrightarrow \exists u[E(u,v,t)]$$

$$I(v,t) \longleftrightarrow \exists u[I(u,v,t)]$$

$$S^{\gamma}(k,t) \longleftrightarrow \exists u_{1}u_{2} \dots u_{k} \in \gamma[\bigwedge_{i=1}^{k} S(u_{i},t)]$$

$$E^{\gamma}(p,t) \longleftrightarrow p = k/|\gamma| \land \exists u_{1}u_{2} \dots u_{k} \in \gamma[\bigwedge_{i=1}^{k} E(u_{i},t)]$$

$$D^{\gamma}(p,t) \longleftrightarrow p = k/|\gamma| \land \exists u_{1}u_{2} \dots u_{k} \in \gamma[\bigwedge_{i=1}^{k} D(u_{i},t)]$$

$$I^{\gamma}(p,t) \longleftrightarrow p = n/|\gamma| \land \exists u_{1}u_{2} \dots u_{k} \in \gamma[\bigwedge_{i=1}^{k} I(u_{i},t)]$$

Probabilities

$$D^{\gamma}(p,t) \implies \exists r \ge p[I^{\gamma}(r,t)]$$

$$\begin{split} I^{\gamma}(p,t) \implies \exists r \geq p[E^{\gamma}(r,t)] \\ \\ E^{\gamma}(p,t) \implies \exists r \geq p[S^{\gamma}(r,t)] \end{split}$$

Uncertainty

operator SE

$$\begin{array}{c} SE^{\gamma}(u,t) \\ \longleftrightarrow \end{array}$$

 $\exists t^{\prime\prime\prime} < t^{\prime\prime} < t^{\prime} < t \\ \exists u^{\prime\prime\prime} \\ \exists \gamma^{\prime} \\ \exists u^{\prime\prime} \\ \in \gamma^{\prime} \\ \exists u^{\prime\prime} \\ \in \gamma \\ \{ S(u,t^{\prime}) \land \{ [E(u^{\prime\prime},u,t)] \land \{ [I(u^{\prime\prime\prime},u^{\prime\prime},t)] \land [E(u^{\prime\prime},u^{\prime\prime},t)] \\ \} \\ \}$

$$SE^{\gamma}(t) \longleftrightarrow \exists u \in \gamma[SE^{\gamma}(u,t)]$$

 $SE^{\gamma} \longleftrightarrow SE^{\gamma}(NOW)$

operator SI

 $SI^{\gamma}(u,t)$

 \longleftrightarrow

 $\exists t^{\prime\prime\prime} < t^{\prime\prime} < t^{\prime} < t \exists u^{\prime\prime\prime} \exists \gamma^{\prime} \exists u^{\prime\prime} \in \gamma^{\prime} \exists u^{\prime} \in \gamma \{S(u,t^{\prime}) \land \{[E(u^{\prime},u,t)] \land [I(u^{\prime\prime\prime},u^{\prime\prime},t)]\}\}$

 $SI^{\gamma}(t) \longleftrightarrow \exists u \in \gamma[SI^{\gamma}(u,t)]$ $SI^{\gamma} \longleftrightarrow SI^{\gamma}(NOW)$

operator EI

$$EI^{\gamma}(u,t)$$

 \longleftrightarrow

 $\exists t''' < t'' < t \exists u''' \exists \gamma' \exists u'' \in \gamma' \exists u' \in \gamma \{ S(u,t') \land \{ [E(u',u,t)] \land \{ [I(u''',u'',t)] \land [E(u'',u',t)] \} \}$

$$EI^{\gamma}(t) \longleftrightarrow \exists u \in \gamma[EI^{\gamma}(u,t)]$$
$$EI^{\gamma} \longleftrightarrow EI^{\gamma}(NOW)$$

Control measures

quarantine

$$Q^{\gamma}(u,t,t') \longleftrightarrow \forall t'' \in [t,t'] \longrightarrow \neg \exists v \in \gamma E(u,v,t')$$

isolation

 $Q^{\gamma}(t,t') \longleftrightarrow \forall u \in \gamma$

block

 $Q^{\Gamma}(t,t') \longleftrightarrow \forall u \in \gamma$

border

 $Q^{\rho}(t,t') \longleftrightarrow \forall u \in \gamma$

restriction

 $Q^P(t,t') \longleftrightarrow \forall u \in \gamma$

Computational complexity

Our model is decidable due to the finiteness of the structures employed.

We expect to observe NP-complete Problems at Cluster Level due to a reduction from k-SAT or HyperGraph Search

We expect to observe Polynomial Time Problems at Population Level and PSPACE, EXPTIME above Cluster Level.

Using Cellular Automata

A two-dimensional cellular automaton M is a septuple

$$M = (N, W, A, I, \Delta, \bar{S}, T)$$

hence M comprises of a finite two-dimensional array A over a bounded integer set I, a fixed neighborhood function $N_j^i \to \mathcal{P}(A)$ together with a collection Δ of rules δ of the form

$$(\bar{N}_j^i, \bar{W}_j^i, \bar{A}_{i,j}) \to q \in I$$

It is assumed |N(i,j)| = k for some fixed k. Hence the transition rules are defined for all collections of tuples $\bar{r} \in I^k$.

W(i, j) is a list corresponding to the weather parameters in the neighborhood N(i, j), according to which infection may propagate in different ways.

A configuration of the automaton is an array of values \bar{A} . \bar{S} is the starting configuration.

A transition $\bar{A} \to \bar{A}'$ takes in total a fixed T time units to propagate and it is any pair of arrays (\bar{A}, \bar{A}') such that if $\bar{A} = \bar{A}$ then

$$\bar{A}' = \begin{bmatrix} (\bar{N}_1^1, \bar{W}_1^1, \bar{A}_{11}) & (\bar{N}_2^1, \bar{W}_2^1, \bar{A}_{12}) & \dots & (\bar{N}_n^1, \bar{W}_n^1, \bar{A}_{1n}) \\ (\bar{N}_1^2, \bar{W}_1^2, \bar{A}_{21}) & (\bar{N}_2^2, \bar{W}_2^2, \bar{A}_{22}) & \dots & (\bar{N}_n^2, \bar{W}_n^2, \bar{A}_{2n}) \\ \dots & \dots & \dots \\ (\bar{N}_1^d, \bar{W}_1^d, \bar{A}_{d1}) & (\bar{N}_2^d, \bar{W}_2^d, \bar{A}_{d2}) & \dots & (\bar{N}_n^d, \bar{W}_n^d, \bar{A}_{dn}) \end{bmatrix}$$

A computation is a sequence $\bar{A}_1 \bar{W}_1 \bar{A}_2 \bar{W}_2 \bar{A}_3 \bar{W}_3 \dots$ such that each weather configuration \bar{W}_i is given at step *i* and

$$\bar{A}_1 = \bar{S}$$
 and $\bar{A}_i \to \bar{A}_{i+1}$

We say the sequence $\bar{A}_1 \bar{W}_1 \bar{A}_2 \bar{W}_2 \bar{A}_3 \bar{W}_3 \dots$ stabilizes if the sequence $\bar{A}_1 \bar{A}_2 \bar{A}_3 \dots$ stabilizes, that is if $\bar{A}_i = \bar{A}_{i+1}$ for some K and all i > K.

We say the sequence weakly-stabilizes if the sequence $\bar{A}_1 \bar{A}_2 \bar{A}_3 \dots$ contains a sub-sequence that stabilizes.

We may choose to consider a computation as ended in the case in which it stabilizes or weakly-stabilizes.

Modeling jumps

In the case of modeling the jumps, the automaton becomes

$$M = (U, W, A, I, \Delta, \bar{S}, T)$$

where $U(i,j) = N(i,j) \cup J(i,j)$ for any specified $J(i,j) \subseteq A$.

In other words, any collection of cells of the grid may become, for a predetermined period of time, part of the neighborhood of cell A_{ij} . The rules δ now have the form

$$(\bar{U}^i_j, \bar{W}^i_j, \bar{A}_{i,j}) \to q \in I$$

The form of a transition becomes $\bar{A} \to \bar{A}'$ such that

$$\bar{A}' = \begin{bmatrix} (\bar{U}_1^1, \bar{W}_1^1, \bar{A}_{11}) & (\bar{U}_2^1, \bar{W}_2^1, \bar{A}_{12}) & \dots & (\bar{U}_n^1, \bar{W}_n^1, \bar{A}_{1n}) \\ (\bar{U}_1^2, \bar{W}_1^2, \bar{A}_{21}) & (\bar{U}_2^2, \bar{W}_2^2, \bar{A}_{22}) & \dots & (\bar{U}_n^2, \bar{W}_n^2, \bar{A}_{2n}) \\ \dots & \dots & \dots \\ (\bar{U}_1^d, \bar{W}_1^d, \bar{A}_{d1}) & (\bar{U}_2^d, \bar{W}_2^d, \bar{A}_{d2}) & \dots & (\bar{U}_n^d, \bar{W}_n^d, \bar{A}_{dn}) \end{bmatrix}$$

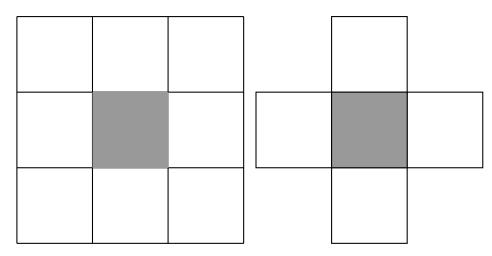


Figure 9: Moore and Von Neumann Neighborhoods

Finally, the form of the computation becomes $\bar{A}_1 \bar{W}_1 \bar{J}_1 \bar{A}_2 \bar{W}_2 \bar{J}_2 \bar{A}_3 \bar{W}_3 \bar{J}_3 \dots$

Again, we assume \overline{W}_i and \overline{J}_i are given as inputs at step *i*.

Computation timing

Transitions $\bar{A}_1 \to \bar{A}_2 \to \bar{A}_3 \to \cdots \to \bar{A}_i \to \bar{A}_{i+1} \cdots$ each happen in discrete time $1, 2, 3 \dots i, i+1 \dots$ The practical meaning of this is that we may observe temperature changes and animal movements at every single unit of time.

The timing parameters of the automaton $T(\bar{N})$ may however be larger than one time unit. This parameter describes the time an infection takes to propagate from the neighborhood periphery to the center.

Neighborhoods

The Moore Neighborhood M(i, j) of $A_{i,j}$ is defined as the set of cells

$$A_{i+1,j-1}, A_{i,j-1}, A_{i-1,j-1}, A_{i+1,j}, A_{i,j}, A_{i-1,j}, A_{i-1,j+1}, A_{i,j+1}, A_{i+1,j+1}$$

The Von Neumann Neighborhood V(i, j) of $A_{i,j}$ is defined as the set of cells

$$A_{i,j-1}, A_{i+1,j}, A_{i,j}, A_{i-1,j}, A_{i,j+1}$$

We note that special conditions may be defined for neighborhoods of boundary cells, as the grid is not assumed to be infinite.

Future Work

Expanding our theoretical analysis: finding a list of reasonable constraints to be satisfied by the choices of the Controller. Construct a probabilistic generalization of the Game.

Topics in Network Centralization. Immunization Centralization seeks to provide a ranking on the nodes according to their importance within the Network.

There exist three types of Centralization in the classical sense, Degree Centralization, Closeness Centralization and Betweeness Centralization. The first is purely local and considers degree of nodes.

The second is semi-global and considers relative position of nodes to other nodes. The last is fully global and considers the prevalence of nodes within all-pairs shortest paths.

This concept plays a central role in the choice of locations for which immunization may prove to be mandatory.

Questions in Temporal Network Epidemiology Is it possible to produce different temporal graph layers for the aggregated automaton that will generate the same computation?

Hence, under what conditions do different sequences of jumps and different weather parameters produce the same computation?

Similarly, under what conditions do different sequences of moves and different weather parameters produce a substantially different computation? Hence, is it possible to split the geographical graph into "equivalence classes" such that an infection within such an equivalence class may produce similar results to any other infection within the same partition?

What is the "worst" possible temporal graph, of bounded degree, in terms of disease spread, given a fixed location graph? We shall pose this question, after identifying reasonable constraints on the graph of movements.

Hence, under what restrictions can disease spread be delayed as much as possible?

For example, we may be able to show formally that limiting the number of farms that can receive animals from any given farm, i.e. placing a bound on the out-degree of any node in the movements graph, would cause a significant delay in the spread of BTV, hence allowing for a natural end to the disease before reaching disastrous amplitudes.

The gist of these questions concerns Network Configuration. What exactly characterizes a Network favoring spread of disease?

Can we classify bad networks? Can we measure their badness?

Can we, by any chance, rank the Networks by attempting to create a Topological Space through defining a measure of "distance" between them? **Axiomatization** Advanced topics in the Axiomatization part would be the definition of the following predicates

- Infectious Injection, the assumption of maliciously infecting units or populations,
- Patient Zero Hypothesis, the assumption that there existed a unit infected first during the course of an Epidemic,
- Exposure Rate, the definition of the rate at which units or populations are exposed,
- Typhoid Mary Predicate, the definition of a reservoir of infection,
- False Positive, and True Negative screening, the assumption that detection can be fallible,
- Harmless Exposure, the assumption that Exposure can be harmless,
- Fallible immunization, the assumption that immunization can fail.

Cellular Automata Produce different inputs for the cellular automaton that will generate the same computation.

Compute the conditions for different sequences of jumps and different weather parameters to produce the same computation.

Hence, separate equivalence classes for computation, some desirable and some undesirable.

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