Bachelor thesis - Simulation of a spatial SEIR model

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1 Introduction and Motivation

In 2020, the whole world witnessed an epidemic - COVID-19 epidemic. Although very much of daily life has been made worse by this, a situation arises with this situation additional incentive to examine this problem scientifically.

In particular, various mathematical models have been developed for the question of the spread of an epidemic. Probably the best known class of such models are the so-called compartment models. They are an important tool for analysis and disease control; after specifying the model and the variables In reality, it is possible to draw various conclusions about the epidemiological situation. Furthermore, enable simulations of such models to build and evaluate theories, examine causal connections, and answer specific questions. Furthermore, with enough experience and intuition, it is possible that models of this kind teach about legislative decisions, particularly those intended to prevent the spread of a disease. Exactly this last point is from of central importance in this work.

Namely, we build and simulate a geometric compartment model. Such models are of great relevance in this area as they are a natural extension of the classical deterministic models. Classic models work with installments and since the population is considered homogeneous; ie any two individuals are in the same relationship to each other. In particular, no 'location' is assigned to the individuals, what is already the case in reality.

The main questions are: when will the disease be under control and when will it die out? When and under what conditions does a sick person cause only a local cluster in their area? After we specify some stochastic properties is it is possible to make very elegant and meaningful mathematical guesses about this question. We shall see that these conjectures reflect reality well.

Once we can say with certainty that the model represents a legitimate (but obviously simplified) version of the epidemiological situation, we can ask how to can use the new findings meaningfully in real life. The important question you to this simulation is: Do movement restrictions make sense in the context of Preventing the Spread of a Disease? If so, how exactly should they be defined? Despite her, do global clusters sometimes emerge? Does it matter how long the incubation is and the phase in which one is contagious last? Does it matter how many people a person meets on average in a day? How to model the probability that you get sick yourself after meeting a sick person?

Exactly this problem we will solve with this simulation, which is made by combining knowledge from computer science and mathematics, examine and comment on the main results. First we present the basics of the theory of Compartment models and some variations thereof. After that, let's focus on those actual simulation: the mechanisms of infection spread and the simulation results. The simulation was written entirely in C++.

2 compartment models

Compartment models in epidemiology are simplified mathematical models of an infectious disease. The population is assigned to different categories (compartments) with different names. In the elementary case, the compartments are vulnerable (S - Susceptible), Infected (I - Infected) and Removed (R - Removed). The order in which the compartments are indicated, often points out how the circulation between the Compartments is modeled.

2.1 The classic SIR model with ODE's [1]

A well-known and relevant approach to modeling the dynamics between the compartments is that with ordinary differential equations. Then you model them Transition rates between the compartments depend on the levels of the compartments itself, as well as some externally defined constants. So, a population of N ÿ N Individuals will be allocated to the compartments indicated above; starting from a zero time point, the number of individuals in the respective compartment is taken as a time-dependent one Function defined: S(t), I(t), R(t). Further one defines constants ÿ, ÿ ÿ R+, which the model transition rates. Accordingly, such a model can have the following form:

$$\begin{aligned} \frac{dS}{dt} &= \frac{\ddot{y}\ddot{y}IS}{N} \\ \frac{dI}{dt} &= \frac{\ddot{y}IS}{N} \; \ddot{y} \; \ddot{y}I \\ \frac{dr}{dt} &= \ddot{y}I \end{aligned} \tag{1}$$

German

With the initial conditions:

$$\begin{aligned} S(0) &= S0 \; \ddot{y} \; 0 \\ I(0) &= I0 \; \ddot{y} \; 0 \\ R(0) &= R0 \; \ddot{y} \; 0 \end{aligned} \tag{2}$$

So, the rate of transition from S to I is negatively dependent on both the level of S as well as that of I - the more susceptible and infected there are, the more newly infected there is. Symmetrically, I grows at the same rate (here it is positive); a share from I, depending on the level of I, switches to R.

It is important to clarify that in this model all components are deterministic (no uncertainty, no stochastic modelling) and that the geometric location of individual individuals plays no role or is not specified at all.

Figure 1: SIR circulation

2.1.1 Theorem about uniqueness of the solution of SIR [2]

Sentence:

The differential equation system in the SIR model is uniquely solvable.

Proof:

After dividing the equations in (1) by the constant population size N, we get one:

$$\frac{ds}{dt} = \ddot{y}\ddot{y}is$$
$$\frac{tu}{} = \ddot{y}is\ \ddot{y}\ \ddot{y}i\ dt \qquad (3)$$

With the initial conditions:

$$s(0) = s0\ \ddot{y}\ 0$$
$$i(0) = i0\ \ddot{y}\ 0 \qquad (4)$$

with r(t) = 1 ÿ s(t) ÿ i(t), where s(t), i(t), r(t) are the shares of the compartments. The Triangle T in the si phase plane is expressed as:

$$T = \{(s, i)|s\ \ddot{y}\ 0, i\ \ddot{y}\ 0, s + i\ \ddot{y}\ 1\} \qquad (5)$$

is positively invariant and unique solutions exist in T for all positive times, so the model is mathematically and epidemiologically well defined. [12]

2.1.2 The number of reproductions: ÿ [3]

An important measure of this model, on which the dynamics of the I compartment depends, is the so-called reproduction number, $\ddot{y} = \frac{\ddot{y}}{g}$. It denotes the expected number of Neuin infections from a single infection in a population where all are susceptible. After If you rewrite the second differential equation, you get:

$$\frac{dI}{\text{German}} = (\ddot{y}\ \frac{S}{N}\ \ddot{y}\ 1)\ddot{y}I \qquad (6)$$

This implies the following:

$$ÿ\ ÿ\ S(0) > N = ÿ \quad \frac{dI}{dt}(0) > 0 \tag{7}$$

That means a real outbreak is happening and the number of infected is increasing.

On the other hand, one also has:

$$ÿ\ ÿ\ S(0) < N = ÿ \quad \frac{dI}{dt}(0) < 0 \tag{8th}$$

That means no real outbreak happens and the number of infected people decreases.

In the special case, in which (almost) all individuals initially belong to S, one can use the above
Also write formulas in the following way:

$$\begin{array}{ll} dIÿ > 1 = ÿ & \frac{dI}{dt}(0) > 0 \\ ÿ < 1 = ÿ & \frac{dI}{dt}(0) < 0 \end{array} \tag{9}$$

2.1.3 The limit of the part of S: sÿ

Set: [2]
Let (s(t), i(t)) be the solution of (3) in T. If ÿ ÿ s0 > 1, then i(t) ÿ 0, as t ÿ ÿ. If ln(ÿÿs0) ÿ ÿs0 ÿ 1, then i(t) has its maximum imax = i0 +s0 ÿ and then i(t) ÿ 0, as t ÿ ÿ. The susceptible part is the unique solution in (0,) of the equation:

$$i0 + s0 ÿ sÿ + \ln(s0 \frac{sÿ}{}) / ÿ = 0 \tag{10}$$

Proof: The proof is omitted here.
The interpretation of this is similar to that in 2.1.2. If almost all individuals are susceptible at the beginning and the reproduction number is greater than 1, all individuals will get sick in a moment. That is, in the end there are no vulnerable individuals. Conversely, if the reproductive number is less than or equal to 1, not all individuals will be infected and a proportion of the population will remain susceptible 'forever'.

2.1.4 Solvability of the SIR model [4]

In 2014, Harko et al. a pseudo-analytic solution (contains integrals that have to be solved numerically) to the SIR model is derived. She looks like this:

$$\begin{aligned} S(u) &= S(0)u \\ I(u) &= N ÿ R(u) ÿ S(u) \\ R(u) &= R(0) ÿ ÿ ÿ \ln(u) \end{aligned} \tag{11}$$

..

for the parameterization:

$$t = \frac{N}{\ddot{y}} \int_{\text{and}}^{1} \frac{\text{youÿ}}{\ddot{y} = u\ \ddot{y} I(u\ \ddot{y})\ \ddot{y}} \ddot{y} N, \quad \text{——} \tag{12}$$

with the initial requirements:

$$(S(1), I(1), R(1)) = (S(0), N\ \ddot{y}\ R(0)\ \ddot{y}\ S(0), R(0)),\ uT < u < 1, \text{ where } I(uT) = 0 \tag{13}$$

2.1.5 Examples of SIR trajectories

First we present two SIR trajectories with different parameters. In the first, the disease dies out, i.e. ÿ ÿ 1. In the second, nevertheless, all individuals are infected, i.e. ÿ > 1.

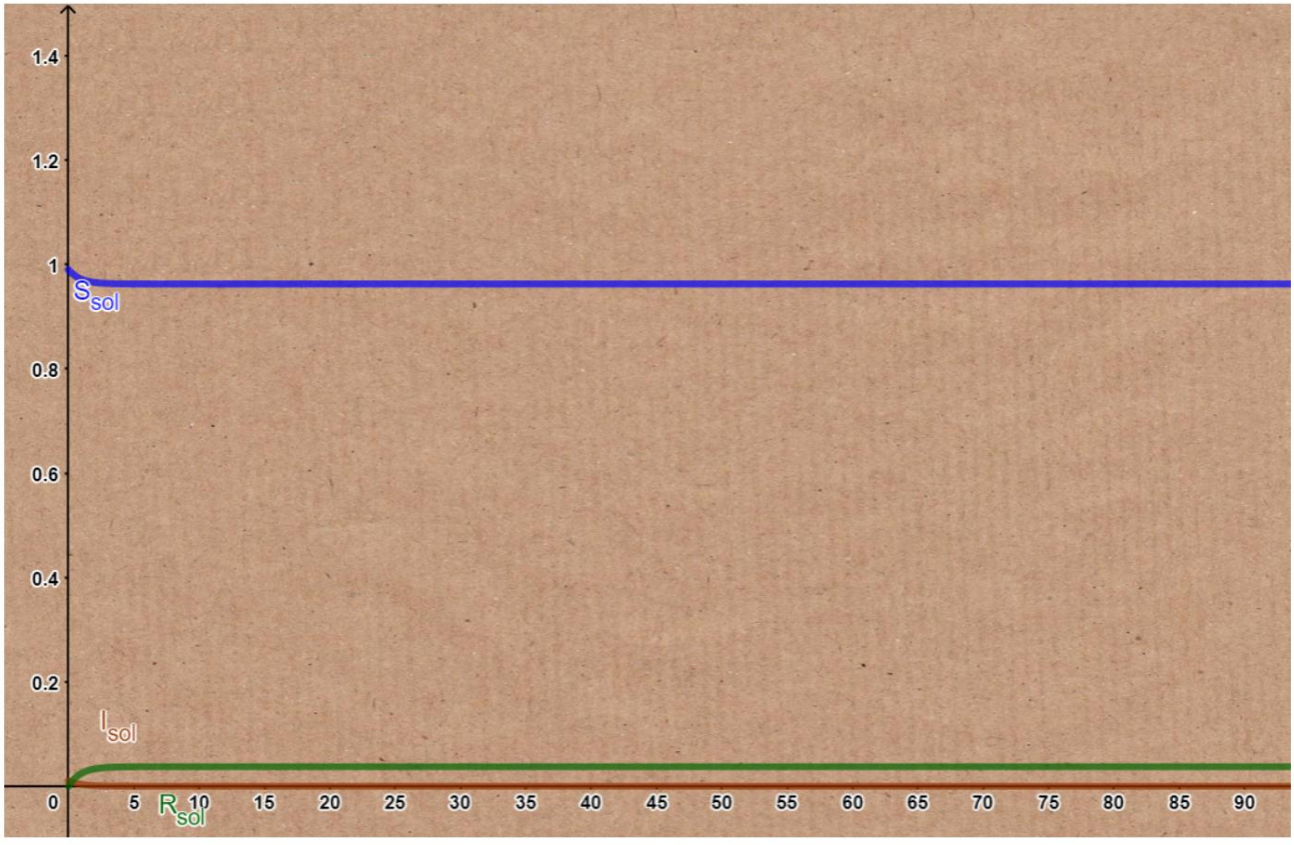

Figure 2: A SIR trajectory with ÿ = 3, ÿ = 4 [10]

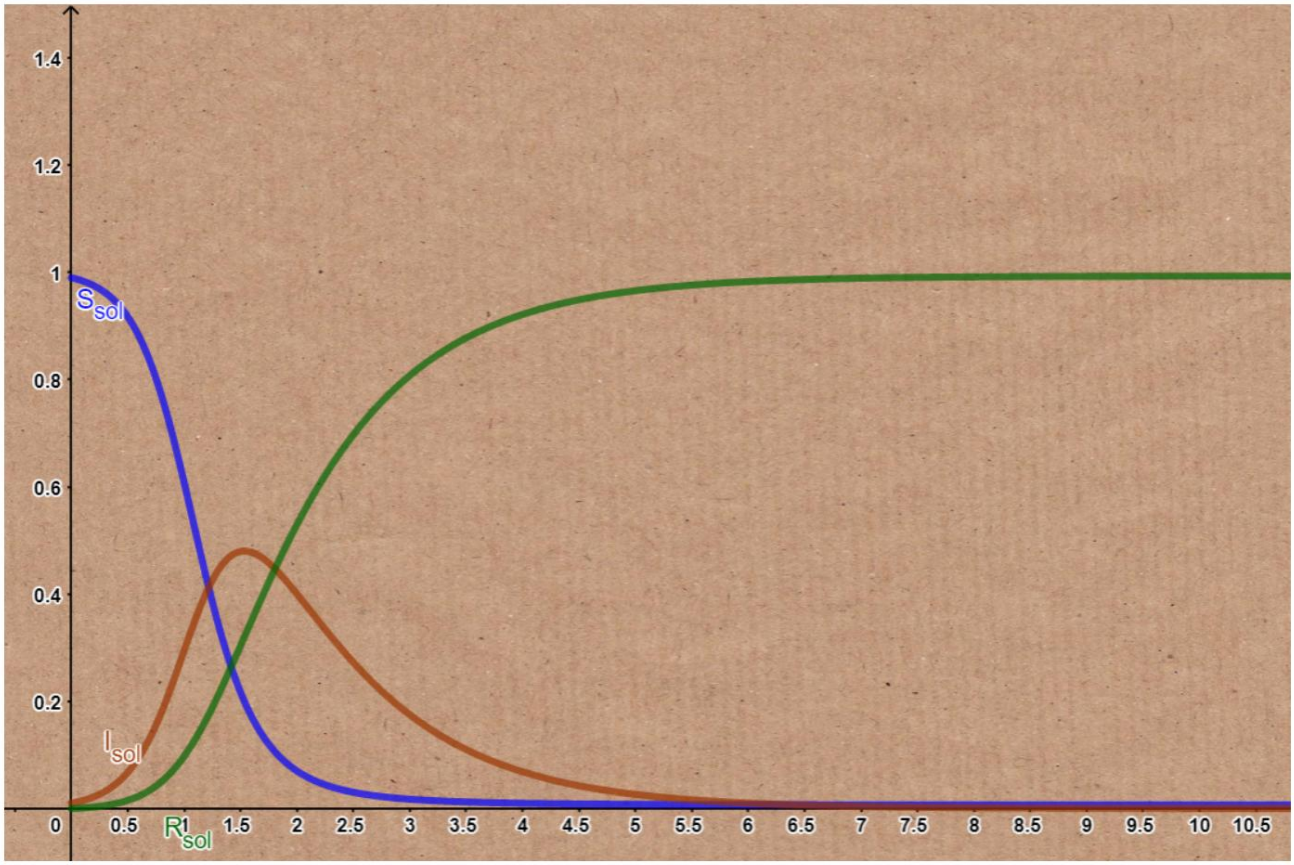

Figure 3: A SIR trajectory with ÿ = 5, ÿ = 1 [10]

2.2 Other variations of SIR modelling

2.2.1 SIS model

Long-term immunity is not developed for some diseases. In this case, the R compartment is left over, so you switch forever between S and I.
The following form is recommended:

$$\begin{aligned} \frac{dS}{dt} &= \frac{\ddot{y}\ddot{y}IS}{N} + \ddot{y}I \\ \frac{dI}{dt} &= \frac{\ddot{y}IS}{N} \ddot{y}\ddot{y}I \end{aligned} \tag{14}$$

It is easy to see from everyday life that the SIR and SIS structures for ¨
oversimplify the majority of infectious diseases. On the one hand, many diseases
have more complicated stages: people develop immunity, some get it
disease again, viruses mutate, etc. On the other hand, as already mentioned, take into account ¨
these two models the population as a homogeneous set, which is also problematic.

2.2.2 Models with the trans Ager (C) Compartment

Some people who have an infectious disease never fully recover and passively carry the disease on. This leads to an alternative to the R compartment: the C compartment (carrier).

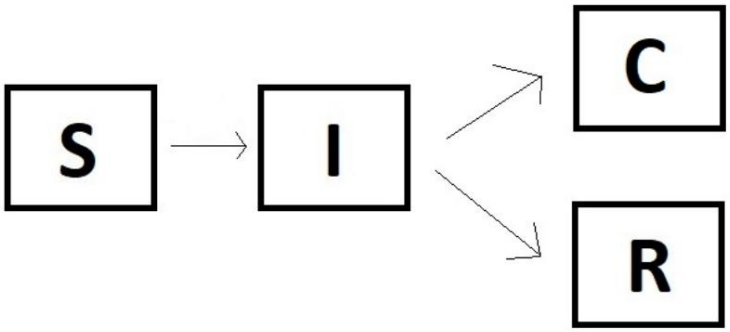

Figure 4: SIRC circulation

Such a structure can be modeled as follows, where ÿ1, ÿ2, ÿ1, ÿ2 ÿ R+

$$\begin{aligned} \frac{dS}{dt} &= (\ddot{y}\ddot{y}1I \ddot{y} \ddot{y}2C) \quad \frac{S}{N} \\ \frac{dI}{dt} &= (\ddot{y}1I + \ddot{y}2C) \quad \frac{S}{N} \ddot{y} (\ddot{y}1 + \ddot{y}2)I \\ \frac{dr}{dt} &= \ddot{y}1I \\ \frac{dC}{dt} &= \ddot{y}2I \end{aligned} \tag{15}$$

2.2.3 SEIR model

It is often the case that, after an infection, one is not immediately infectious to others. Thus one has to introduce an additional compartment between S and I: E - Exposed. In addition to the objects defined in 2.1 , let ÿ, µ ÿ R+. Thus the following differential equation system is possible:

$$\begin{aligned} \frac{dS}{dt} &= \mu N \ddot{y} \mu S \ddot{y} \quad \frac{\ddot{y}IS}{N} \\ \frac{dE}{dt} &= \frac{\ddot{y}IS}{N} \ddot{y} (\mu + \ddot{y})E \\ \frac{dI}{dt} &= \ddot{y}E \ddot{y} (\ddot{y} + \mu)I \\ \frac{dr}{dt} &= \ddot{y}I \ddot{y} \mu R \end{aligned} \tag{16}$$

Here, ÿ models the duration of the E phase and µ the rate of mortality and fertility.
We will use this compartment structure later in the actual simulation model.

For this version, the reproduction number is defined as follows:

$$\ddot{y} = \frac{a}{\mu + \ddot{y}} \ddot{y} \frac{\ddot{y}}{\mu + \ddot{y}} \quad (17)$$

It is possible to find results about ¨ sÿ for this model analogous to those for SIR. [2]

2.3 Geometric models

Unlike the class of models discussed above, in geometric models plays
the location of an individual does play a role in the spread of the disease. Here will
'the world' is usually defined as a (large) matrix, where each element of the matrix represents a person. Then a number of epidemiologically relevant ones are specified
Properties that are then stochastically assigned to the individuals. Depending on the distribution under which these properties lie, happens as you can imagine
can, various phenomena. It is precisely these dependencies that are often of central importance in such models, and this work is no exception in this respect.

3 'The' model

3.1 Preparation: Distributions

3.1.1 Discrete uniform distribution (abbr. Unif) [5]

With this distribution, every possible outcome occurs with the same probability.
The basic idea of a uniform distribution is that there is no preference. Let ÿ be a non-empty finite set. Then, given a uniform distribution, the probability P(A) is one
Event A with A ÿ ÿ defined by the following formula:

$$P(A) = \ddot{y}|\ddot{y}| \quad \frac{|A|}{} = \frac{\text{Number of elements in A}}{\text{Number of elements in ÿ}} \tag{18}$$

3.1.2 Poisson distribution (abbr. Poiss) [6]

The Poisson distribution is a discrete probability distribution. She becomes through a real parameter ÿ > 0 determines the expected value and at the same time the variance of the distribution describes. It is defined as follows, a random variable X is Poisson distributed with parameters ÿ ÿ R+ if:

$$P\ddot{y}(X = k) = k! \quad \ddot{y}^{k} \quad e^{\ddot{y}\ddot{y}}, \quad \ddot{y}k \ddot{y} N0, \ddot{y} > 0 \tag{19}$$

As already mentioned, the following applies to the first two moments:

$$E[X] = \ddot{y}, \mathrm{Var}[X] = \ddot{y} \tag{20}$$

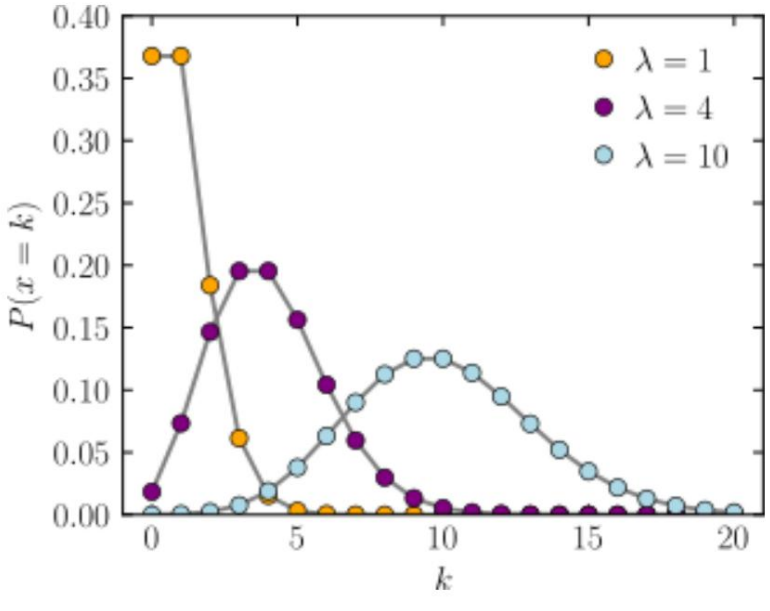

Figure 5: Illustration of Poisson's density for different parameters

3.1.3 Exponential distribution (abbr. Exp) [7]

The exponential distribution is a continuous probability distribution over the set of non-negative real numbers given by an exponential function. she will
defined as follows, a random variable Y is exponentially distributed with parameter ÿ ÿ
R+ if it has the following density:

$$fÿ(y) = 1\{y>0\}ÿeÿÿy \tag{21}$$

The following applies to the first two moments:

$$E[Y] = \frac{1}{ÿ}, Var[Y] = \frac{1}{ÿ^2} \tag{22}$$

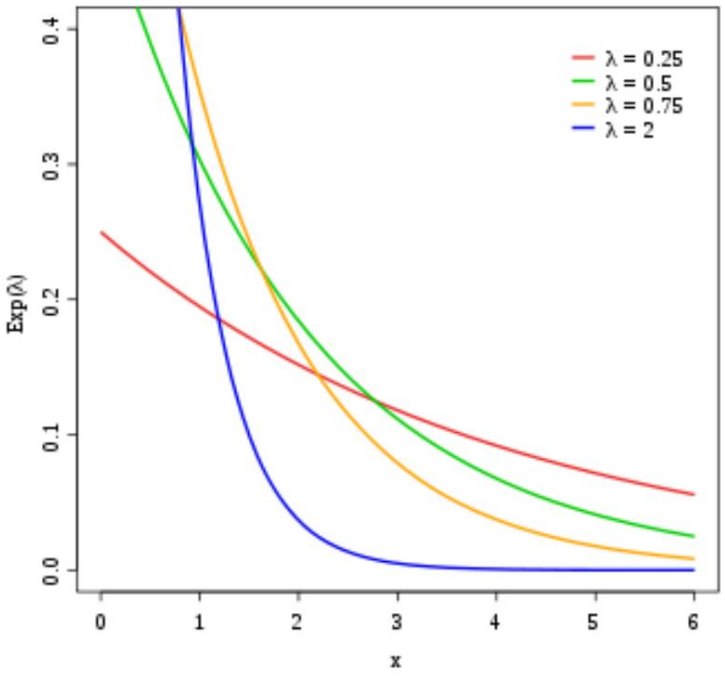

Figure 6: Illustration of the density of the exponential distribution for different parameters

3.1.4 Pareto distribution [8]

The Pareto distribution is a continuous probability distribution on a right-tailed
infinite interval [xmin,ÿ] with parameters ÿ > 1, xmin ÿ 1. It is scale-invariant and
satisfies a power law. For the special case that xmin = 1 (which will be relevant for us later
is) it is defined as follows; a random variable Z is Pareto distributed with parameters
ÿ > 1 if it has the following density:

$$f_{\ddot{y}}(z) = 1\{z>1\}(\ddot{y}\ \ddot{y}\ 1)\ \ddot{y}\ z^{\ddot{y}\ddot{y}} \tag{23}$$

Lemma:

For the density $f_{\ddot{y}}(z)$ the following holds:

$$\ddot{y} > 2 \Rightarrow \ddot{y}\ddot{y}\ E[Z] < \ddot{y}$$

$$\ddot{y} > 3 \Rightarrow \ddot{y}\ddot{y}\ Var[Z] < \ddot{y}$$

Proof:

$$E[Z] = \int_1^{\ddot{y}} z\ \ddot{y}\ f(z)\,dz = \int_1^{\ddot{y}} z\ \ddot{y}\ (\ddot{y}\ \ddot{y}\ 1)\ \ddot{y}\ z^{\ddot{y}\ddot{y}}\,dz = \int_1^{\ddot{y}} (\ddot{y}\ \ddot{y}\ 1)\ \ddot{y}\ z^{1\ddot{y}\ddot{y}}\,dz =$$

$$= (\ddot{y}\ \ddot{y}\ 1)\int_1^{\ddot{y}} e.g^{1\ddot{y}\ddot{y}}\,dz = \ddot{y}\ (E[Z] < \ddot{y} \Rightarrow \ddot{y}\ddot{y}\ \ddot{y} > 2) \tag{24}$$

$$Var[Z] = E[(Z\ \ddot{y}\ E[Z])2\ ] = E[Z^{2}]\ \ddot{y}\ E[Z]^{2} =$$

$$= (\ddot{y}\ \ddot{y}\ 1)\int_1^{\ddot{y}} e.g^{2\ddot{y}\ddot{y}}\,dz\ \ddot{y}\ ((\ddot{y}\ \ddot{y}\ 1)\int_1^{\ddot{y}} e.g^{1\ddot{y}\ddot{y}}\,dz)^{2} = \ddot{y}\ (V\ ar[Z] < \ddot{y} \Rightarrow \ddot{y}\ddot{y}\ \ddot{y} > 3) \tag{25}$$

Furthermore, for the first two moments, as soon as they exist, the following holds:

$$E[Z] = \ddot{y}\ \frac{a\ \ddot{y}\ 1}{\ddot{y}\ 2}\ ,\ Var[Z] = \ddot{y}\ \frac{a\ \ddot{y}\ 1}{\ddot{y}\ 3}\ \ddot{y}\ (\frac{\ddot{y}\ \ddot{y}\ 1}{(\ddot{y}\ \ddot{y}\ 2}\ )^{2} \tag{26}$$

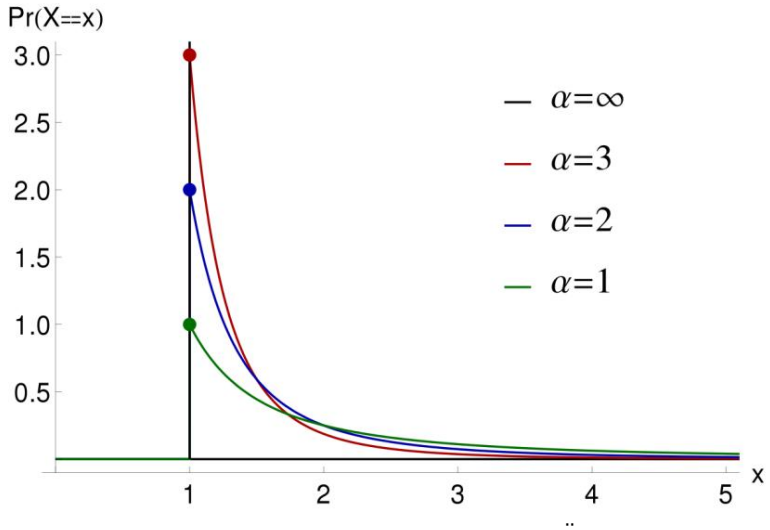

Figure 7: Illustration of the Pareto density for different parameters

3.2 Interpretation and disadvantages of the SIR model

The classic SIR model explains many things, but some strong ones emerged in its design
assumptions made. It works with rates that change the transitions between compartments
explain. Although these rates partly reflect the interaction between individuals from different
Explain compartments well, but simplify the situation in one aspect.

The population in the SIR is considered to be a homogeneous set of individuals. In particular, the structure of the model implies that all susceptible individuals at any given time have an equal probability of getting the disease. Ie, it will be under ..
Equal distribution 'tossed' which susceptible individuals actually get sick.

However, we know that the actors in an epidemiological model rarely point to the same
Ways to interact with all other actors, and that accordingly the relationship
between two individuals already plays a role in how likely it is that a
individual becomes ill because of interaction with the other. Here in particular the
emphasizes the geometric aspect, i.e. the distance between two individuals.

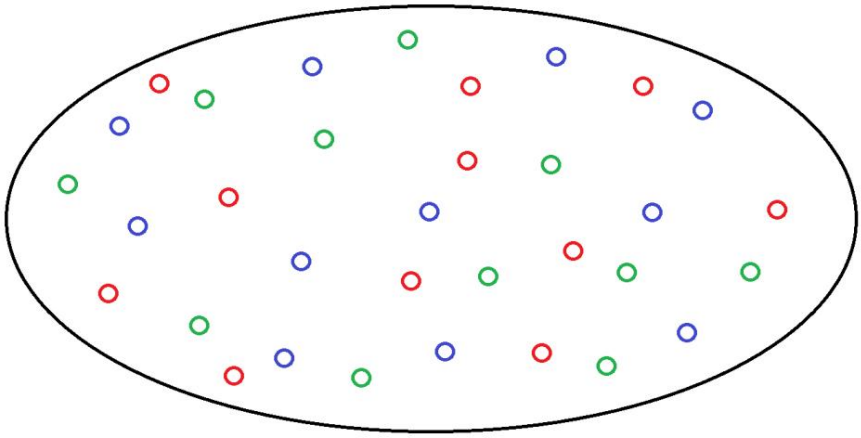

Figure 8: Illustration of the homogeneous structure of the SIR model; The color system
is eg S - blue, I - red, R - green

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This leads us to the actual geometric model of the simulation. Namely, we define
a unique location for each individual in the population. The epidemiologically relevant
Interactions will then also depend on these locations.

3.3 Description of the model

The simulation takes the following parameters as input:

$$n \ddot{y} N, \ddot{y} > 1, \ddot{y}meet > 0, \ddot{y}inc > 0, \ddot{y}inf > 0, days \ddot{y} N \quad (27)$$

The population in which we are simulating an epidemic is defined as an n^2 Matrix shown and we use the SEIR structure (2.2.3). The adjacency rules are like on a torus; the person (n, n) has eg the following neighbors: (n ÿ 1, n),(n, n ÿ 1),(1, n),(n, 1).

Each individual is assigned the following random numbers once:

A ÿ Exp(ÿinc): the duration of the E phase once the individual becomes infected

B ÿ Exp(ÿinf): the duration of the I phase once the E phase of the individual is over is

days are simulated. At the beginning of the simulation, all individuals are healthy except for one: the so-called 'patient-zero'. In the language of compartment models is called that all individuals except one are in the S compartment. You set the 'patient-zero' oBdA to the center of the matrix and it is assumed that it is already contagious. So, it belongs to the I compartment.

Each individual is assigned the following random number each day:

M ÿ P oiss(ÿmeet): The number of other individuals that the individual at the Day visited (Visits are interpreted as interactions; after meeting an S and I individual, the E-phase begins for the first.)

Each individual is assigned the following random numbers for each of the M visits:

D ÿ P areto(ÿ): The distance to the individual to be visited

W ÿ Unif({0, 1, ..., D}): The horizontal part of the movement for the visit

H ÿ Unif({ÿ1, 1}): The orientation of the horizontal part of the motion

V ÿ Unif({ÿ1, 1}): The orientation of the vertical part of the motion

Thus, for a given visit, an individual is located H ÿ W to the right and V ÿ (W ÿ D) down and hits a person there.

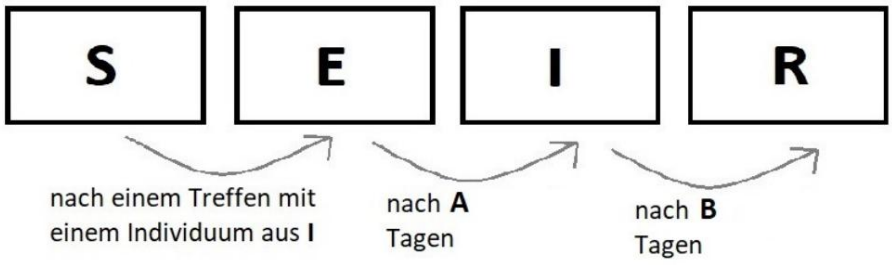
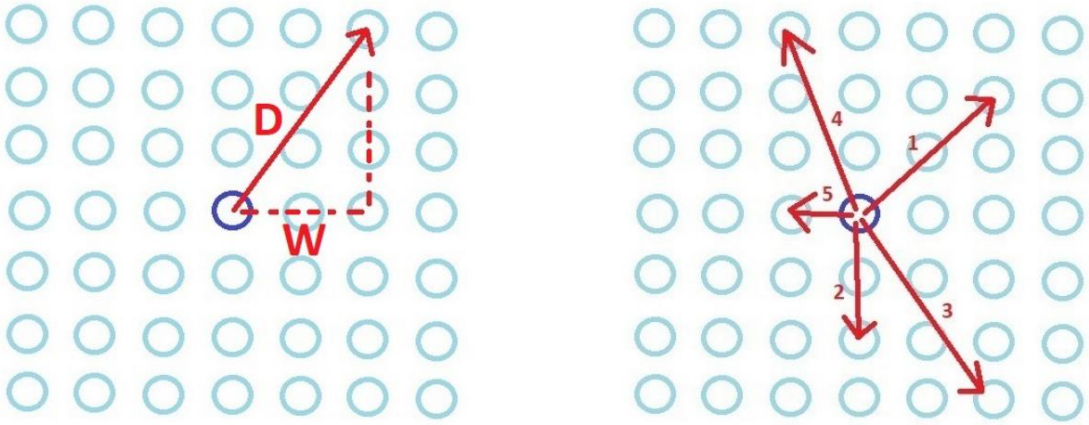


Figure 9: Sketch of the SEIR mechanism



(a) Outline of a specific visit; the case when D = 5, W = 2, H = 1, V = ÿ1

(b) sketch of all visits by an individual in one day; the case when M = 5

Figure 10: Sketch of the spatial mechanism

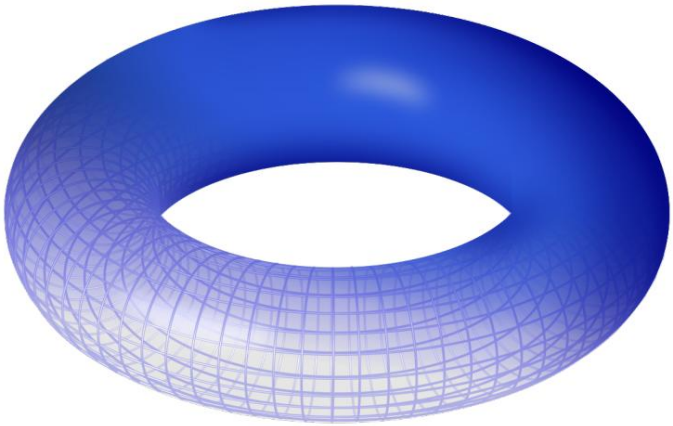


Figure 11: Illustration of a torus [11]; one can imagine the spatial configuration of the Think of the population as the area of a torus

3.4 Justification for the choice of distributions

In the model we use four distributions:

Uniform distribution 3.1.1

We use them for random numbers where there is no pure preference: for the horizontal part of the movement for a visit and the orientations of the respective movement components

Poisson distribution 3.1.2

We use them for the number of individuals that an individual visits in a day. When one needs to model random natural numbers, the Poisson distribution is often a plausible choice; it has only one parameter, and the expectation and variance correspond exactly to that parameter.

Exponential Distribution 3.1.3

It is primarily used as a model when answering the question of the length of random time intervals. Therefore it is also a good and plausible choice for our two phase durations.

Pareto distribution 3.1.4

We use this distribution to decide how far an individual to visit is. The main idea behind this is to 'punish' large distances exponentially. The distribution also has only one parameter, ÿ > 1, which regulates how far individuals move move in the model.

3.5 Possible Generalizations

There are, of course, some generalizations that are useful in addition to the model could import:

Probably the first generalization one can expect is that the simulation does not work in discrete points in time, but that the model works in continuous time is built.

One can consider a broader class of distributions for each mechanism than just one. The distributions used above are of course not the only natural choice.

The assumption of secure and eternal immunity can also be revoked and this Roll property stochastically in an elegant way.

Analogous to this, it is possible to model that the transmission of the disease does not occur at every visit, but that this event can also be stochastic instead Are defined.

The 'world' in this model is defined as an n ÿ n matrix, n ÿ N, ie a subset of Z2
. One can generalize the whole thing in this regard and define the 'world' as a subset of Zm, m ÿ N and adjust all mechanisms accordingly. However, the interpretation of the results from such a model would be presumptive
more difficult.

3.6 Assumptions

We are particularly interested in the influence of two variables:

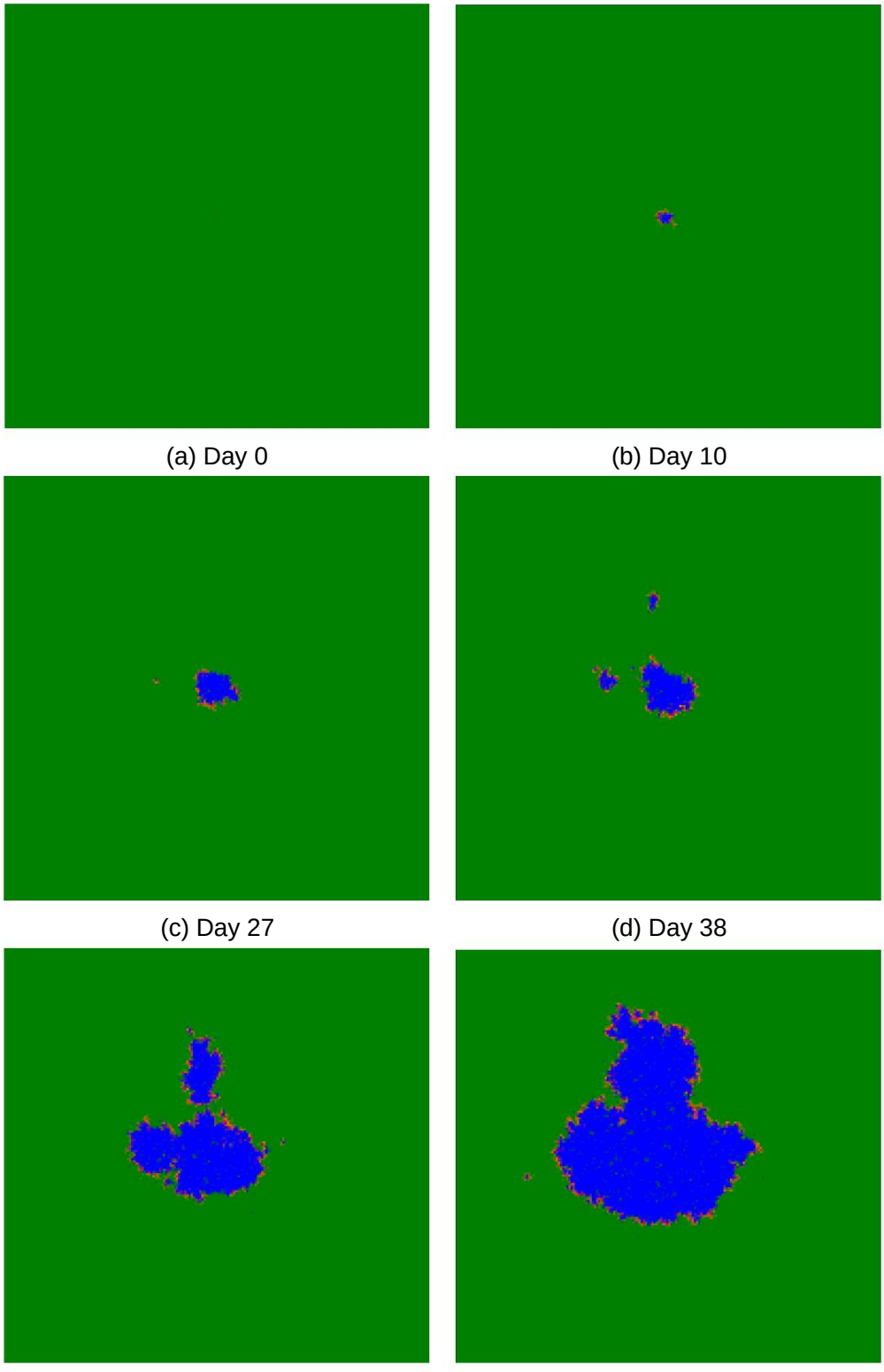
ÿ: As already shown in 3.1.4 , the corresponding Pareto distribution has exactly
then a finite variance if ÿ > 3. This can be interpreted in our model in such a way that the individuals stay close to their location in a controlled manner. In that case
the spread of the disease should also be reasonably stable.

ÿmeet: This variable plays a similar role in the simulation model as the reproduction number does in the classic SIR model. In the expected value, an individual hits
exactly ÿmeet other individuals. The assumption is that the disease should die out,
if ÿmeet ÿ 1.

So, we're basically looking at the results of what happens if we ceteris paribus
the two parameters mentioned above change iteratively (taking into account interesting marginal values, of course).

3.7 Graphical results [9]

First we present some simulation results for the following parameter settings: n = 400, ÿmeet = 3, ÿinc = 1, ÿinf = 1, days = 100. It is simulated three times for 3 choices
for the 'distance parameter': ÿ ÿ {4, 3, 2.5}. The color system is: S - green, E - yellow, I - red, R - blue. Only selected days are shown for compactness. Here is
the focus on the variable ÿ. Therefore we 'insure' that the disease will not die out,
by choosing a 'safe' ÿmeet .

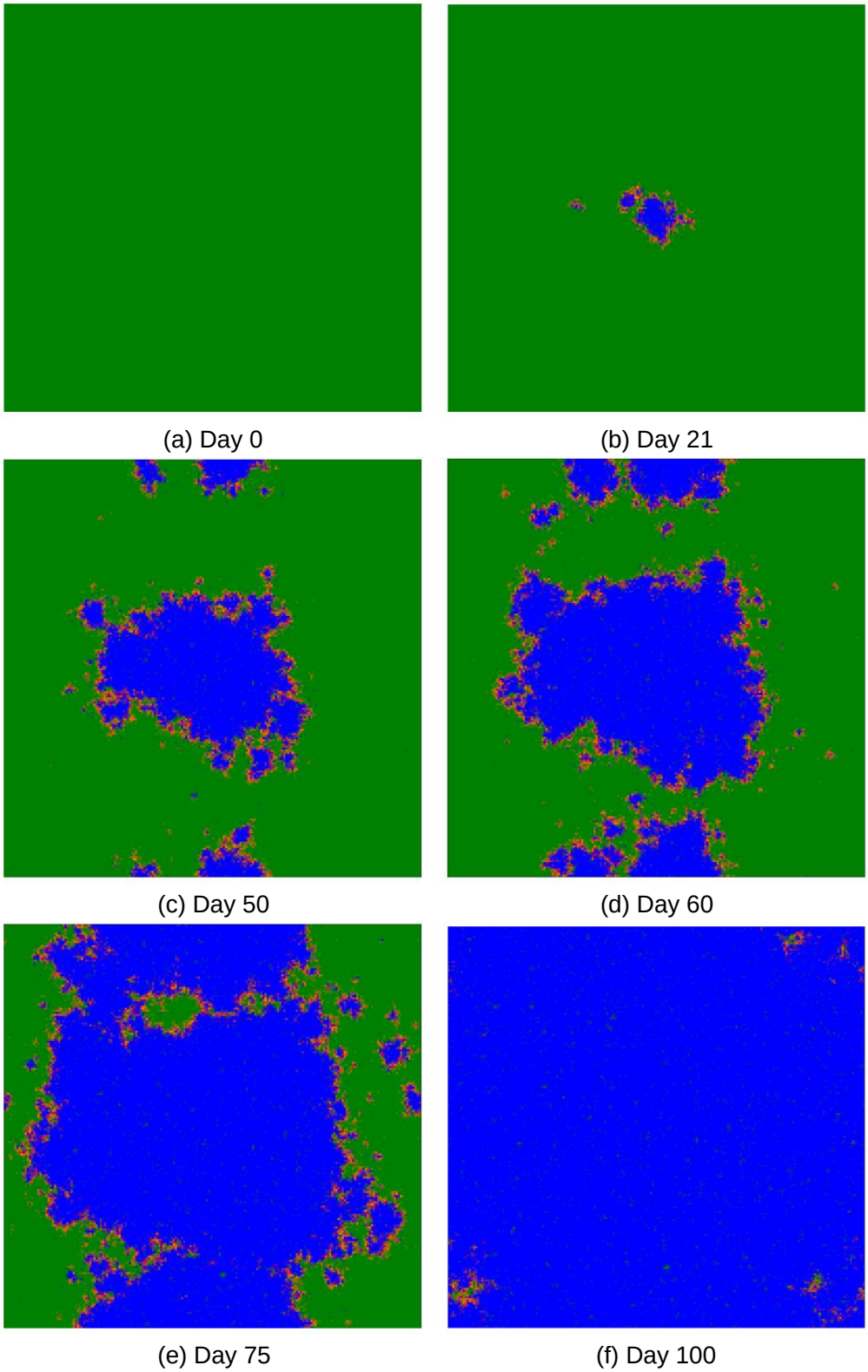


(a) Day 0 (b) Day 10

(c) Day 27 (d) Day 38

(e) Day 60 (f) Day 88

Figure 12: Simulation result for ¨ÿ = 4

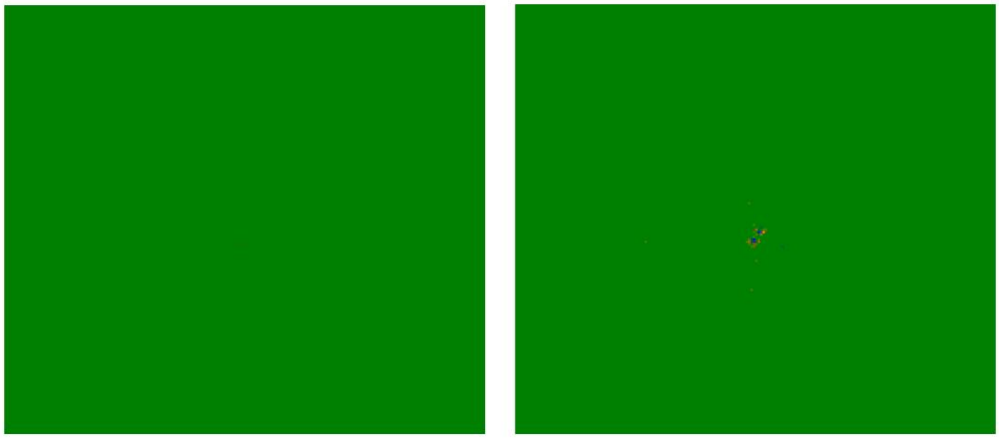


(a) Day 0 (b) Day 21

(c) Day 50 (d) Day 60

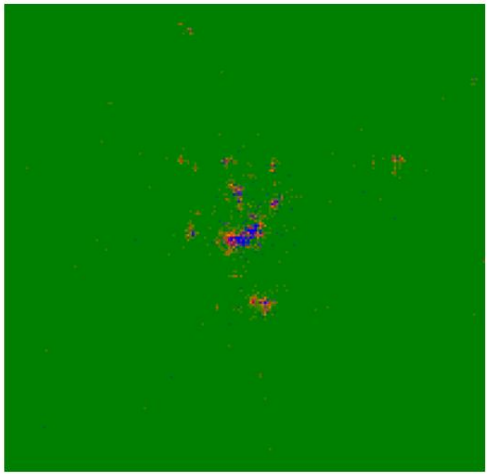
(e) Day 75 (f) Day 100

Figure 13: Simulation result for ¨ÿ = 3

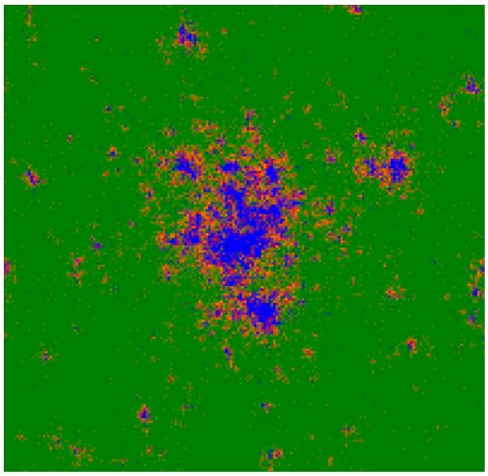


(a) Day 0

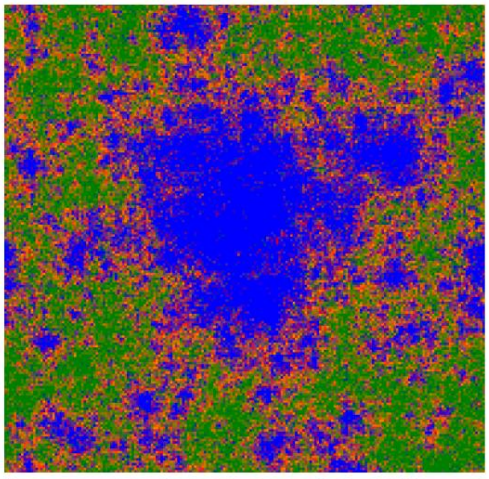
(b) Day 6



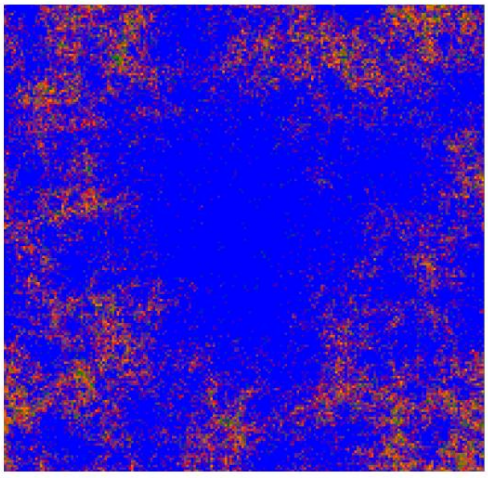
(c) Day 12



(d) Day 18



(e) Day 24



(f) Day 28

Figure 14: Simulation result for ¨ÿ = 2.5

3.8 Conclusions I

We annotate each simulation separately:

ÿ = 4: Since ÿ is significantly larger than 3, one sees, as announced, a local spread of the disease.

ÿ = 3: In this marginal case one also sees marginal behavior. The dissemination looks reasonably local, but new clusters are emerging as well.

ÿ = 2.5: Since ÿ is significantly smaller than 3, one sees, as announced, a lightning-fast global spread of the disease.

The remaining parameters (apart from ÿmeet) do not affect the 'locality' of the propagation, only the speed at which it occurs. For these parameters we make the following remarks:

n: The larger n is, the larger the 'world' in which we simulate. It is natural it is desirable that this parameter is large in order to ensure the plausibility of the propagation to allow. On the other hand, large n's also involve large initialization costs.

ÿinc: The longer the E phase (aq. as the larger ÿinc is), the slower it is Spread because it takes longer for an individual to become infectious.

ÿinf: The longer the I phase (aq. as the larger ÿinf is), the faster the spread, since individuals remain infectious longer.

In the next chapter we deal more with the parameter ÿmeet.

3.9 Quantitative Results

Now we simulate more than once to show some asymptotic results. First let's deal with 'big' ÿ's where propagation is controlled, ie if ÿ > 3. Then we consider what happens if the propagation is more flexible, ie if a ÿ 3.

3.9.1 Results for 'big' ¨ ÿ's

Here we consider cases where ÿ > 3. It is simulated 100 times each and us interested in what proportion of the population was sick at any given moment. Because of this Let's see who belongs to compartments I or R at the end of each simulation. It is simulated for the following parameters:

$$\text{days} = 500,\ n = 400,\ \ddot{y}\text{inc} = 1,\ \ddot{y}\text{inf} = 1,\ \ddot{y}\ \ddot{y} \in \{4, 5\},\ \ddot{y}\text{meet}\ \ddot{y} \in \{1, 2, 3\}, \tag{28}$$

The comments on the respective histogram of the simulation are located under the respective image.

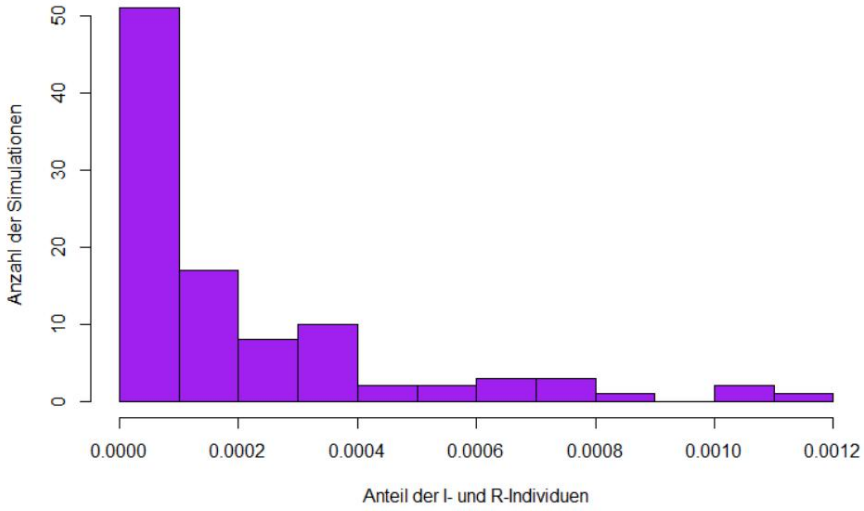


Figure 15: Simulation results for ¨ÿ = 4, ÿP oisson = 1

In this case, the disease practically died out, almost no one was sick.

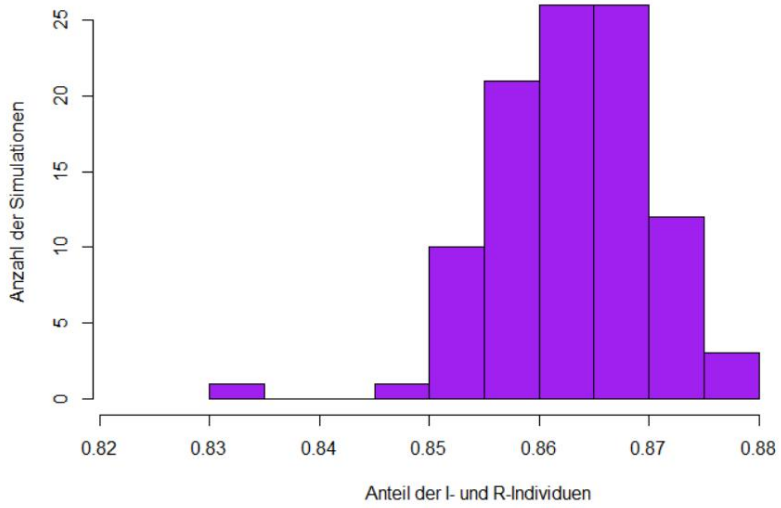


Figure 16: Simulation results for ¨ÿ = 4, ÿP oisson = 2

Here a significant proportion of the population was ill, presumably everyone would be in I or R, if we were to simulate several days. ¨

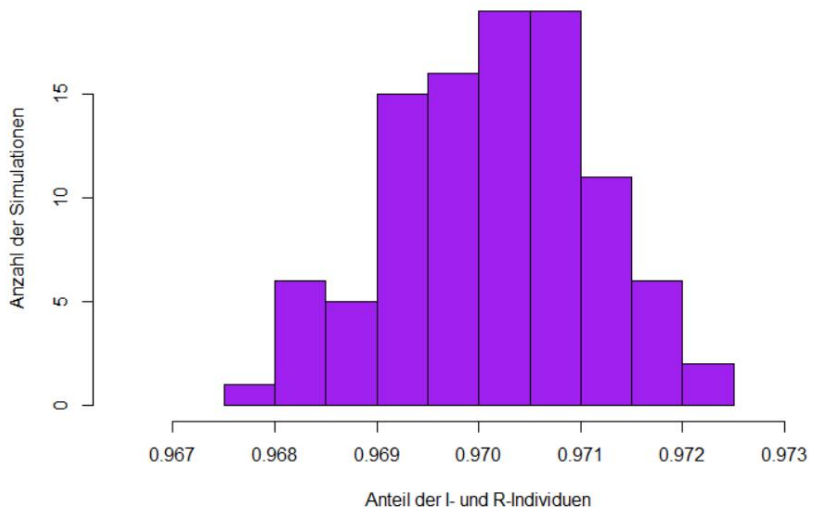


Figure 17: Simulation results for ¨ÿ = 4, ÿP oisson = 3

In this simulation, practically all individuals have become ill.

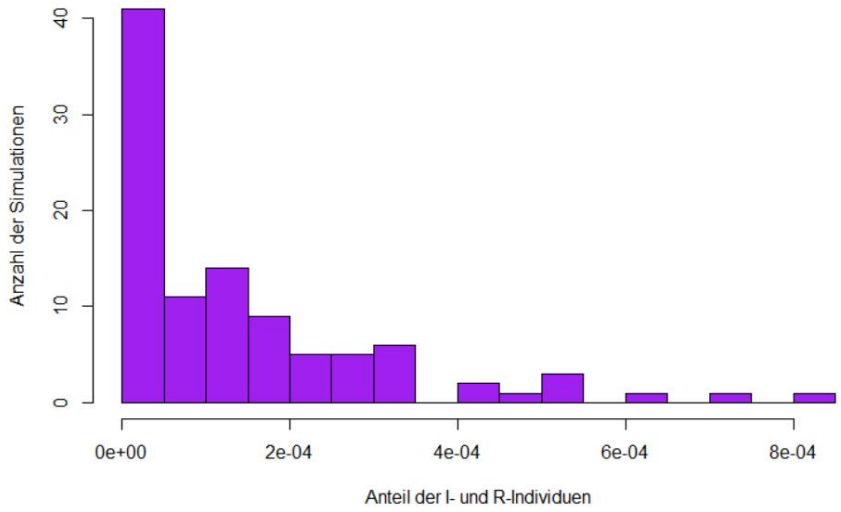


Figure 18: Simulation results for ¨ÿ = 5, ÿP oisson = 1

Similar to the case for ¨ÿ = 4, ÿmeet = 1, effectively nobody got sick.

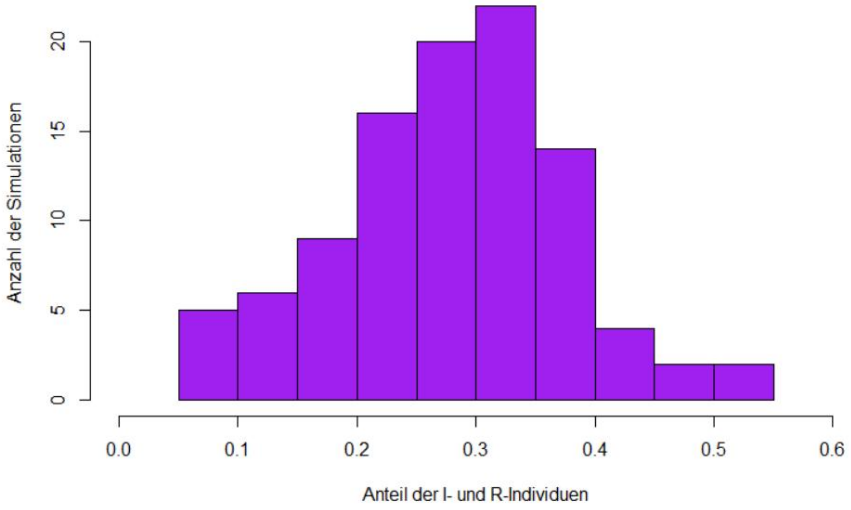


Figure 19: Simulation results for ¨ÿ = 5, ÿP oisson = 2

A significant proportion of the population has also fallen ill here, but this proportion is smaller than in the case for ¨ ÿ = 4, ÿmeet = 2. However, one can expect that all individuals will get sick sooner or later.

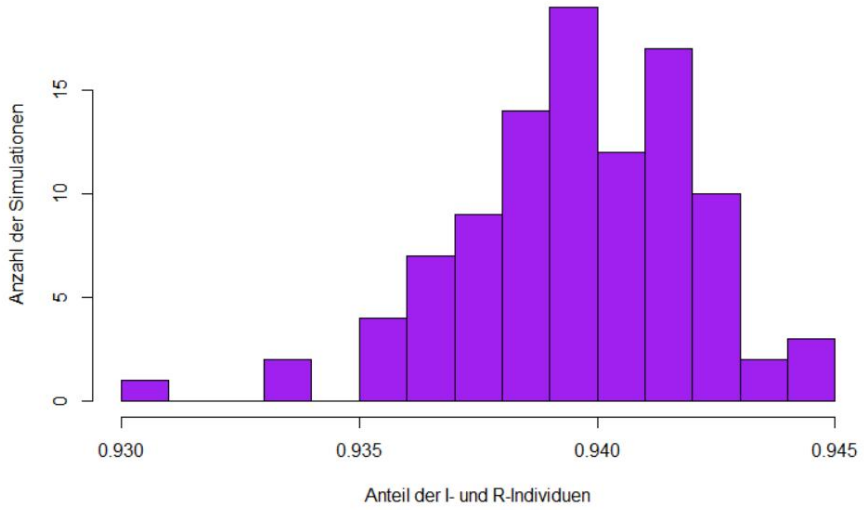


Figure 20: Simulation results for ¨ÿ = 5, ÿP oisson = 3

Again, analogous to the case for ¨ÿ = 5, ÿmeet = 3, more or less all individuals are ill been.

3.9.2 Results for 'small' ¨ ÿ's

Now we simulate distributions for ¨ ÿ < 3. In this chapter we do not show histograms,
but a table with the most important statistical information. The size of the (I or
C) Clusters is defined as follows:

a small cluster: less than 5% of the population is in I or C

a medium-sized cluster: between 5% and 95% of the population is in I or C

a large cluster: more than 95% of the population is in I or C

..

It is simulated 100 times for the following parameters:

days = 500, n ÿ {100, 400}, ÿinc = 1, ÿinf = 1, ÿ ÿ {2, 2.5}, ÿmeet ÿ {1, 2} (29)

n	100				400			
a	2.5		2		2.5		2	
ÿmeet	1	2	1	2	1	2	1	2
Small Clusters Medium	0.46 0.04 0.27 0.04 0.02 0.00 0.01 0.00							
Clusters 0.54 0.20 0.73 0.00 0.98 0.00 0.99 0.00								
Large clusters 0.00 0.76 0.00 0.96 0.00 1.00 0.00 1.00								
Emp. Expected value 0.30 0.92 0.50 0.93 0.53 0.95 0.69 0.96								
Emp. Standard deviation 0.28 0.19 0.31 0.19 0.08 0.00 0.07 0.00								

3.10 Conclusions II

In both classes of results one sees a strong influence of the parameter
ÿmeet. For the sake of compactness, we assign the comments to the respective class:

Results for 'big' ¨ÿ's 3.9.1:
It can be seen that in this case it is crucial whether ÿmeet ÿ 1. The distance parameter is already strict and causes controlled propagation. If it
so it is also the case that a sick person infects no more than one new one,
this disease usually dies out.

Results for 'small' ¨ÿ's 3.9.2:
Here the situation is a little more complicated. In principle, implies a small enough
ÿmeet the extinction of the disease, but a small enough ÿ (hectic spread) may well negate this effect. Also, larger n's teach the 'naturalness' of propagation.

In both cases, it is essential to realize that the interaction of the two parameters discussed is central if the spread of the infection is to be analyzed in this model
want to investigate.

3.11 Discussion

No model is a perfect reflection of reality, and this is of course the case here. Therefore, we indicate some limitations that this simulation suffers from.

There are other, more complicated relationships between individuals. The exact rules for how people meet each other on a daily basis are almost undefinable,
accordingly, it's pretty clear that we're taking the situation seriously in terms of it
simplify.

The world defined for the simulation is only pseudo-infinite. That is, after n steps
in one direction you are still in the same place. Although this, strictly
taken, also applies to our world, this modeling is not the absolutely ideal choice in the epidemiological context.

The individuals in the simulation become permanently immune. Because this medical
The problem is still being discussed and since there are already cases of e.g. COVID-19
If there was that one had fallen ill for the second time, one could also use this restriction
and stochastically model this property meaningfully.

Processes in nature are continuous, our model is discrete. The time is not
discretized, every observation we make or simulate is only a temporal one
'Snapshot' of an object that is a subject of continuous change. Therefore the results of this simulation are to be taken 'cum grano salis'.

Contents of the enclosed CD

The enclosed CD contains a digital (PDF) version of this work as well as a C++ file - the actual simulation.

literature

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Declaration of Honor

I hereby declare on my honor that I have completed this work independently; thoughts taken directly or indirectly from external sources are marked as such. The work has not yet been submitted to any other testing authority and also not yet published.

Munich, September 15, 2021 Place and date

Signature (Vjekoslav Drvar)