Master's thesis

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Epidemic spreading in an active matter model

Master's thesis in MTFYMA

Supervisor: Paul Gunnar Dommersnes

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Abstract

In this master thesis, we have studied how a virus can spread in a human population. This has been done by developing a numerical model that we have used to consider how different measures and restrictions can affect the way the disease spreads and how many in the population are infected. The measures we have considered are similar to the ones that are typically introduced by the governments in a country during an epidemic. Among these are making stricter rules regarding hygiene to reduce the infection probability, identifying and isolating the persons that are infected and setting a limit to the number of people that are allowed to meet in one group.

The numerical model that has been developed is an example of an active matter model with three types of interactions; inter-particle forces, alignment and information exchange. The particles are controlled by the inter-particle interactions and by a diffusion in the angular direction. The model is inspired by the Vicsek model. We have used exchange to simulate the transfer of a disease between human, but the model could also be used to consider information transfer in general, for example in cells in tissue.

We have found that by changing the mobility of the particles in the system we can reduce the number of persons that are infected. In a system where all the particles are restricted to a small area, fewer people are infected than if some of these are able to move freely in the entire system. This has also been confirmed by comparing the results of the active model to the results from a static lattice model where it is predefined which particles can interact with each other. From this result we can learn that the we can reduce the number of people that are infected by interacting only with people from our own area.

When trying different measures we found that all of them were effective in reducing the number of infected. Decreasing the infection probability to half of the original had an extensive effect on the number of infected. Identifying and isolation was also very effective. The measure that spread the epidemic over the longest time period was to limit the group sizes. All of these results will of course depend strongly on the parameters used in the simulation and they will also be affected by random fluctuations. The main finding is therefore that the model can be used for simulating an epidemic an be a tool to help understand what can slow down an epidemic.

Sammendrag

I denne masteroppgaven har vi studert hvordan et virus kan spre seg i en befolkning med mennesker. Vi har gjort dette ved å lage en numerisk modell hvor vi kan teste hvordan ulike tiltak og restriksjoner påviker antall smittede og hvordan sykdommen sprer seg. Tiltakene vi har sett på er typiske tiltak som blir innført i et land når det bryter ut en epidimi. Eksempel på slike tiltak kan være å stramme inn hygieneregler for å redusere sjangen for at en sykdom overføres fra en person til en annen, finne ut hvem som er smittet og isolere disse og å sette en grense for hvor mange som har lov til å samles på et sted.

Den numeriske modellen er et eksempel på en aktiv modell som har tre ulike typer vekselvirkninger mellom partiklene; krefter som tiltrekker eller frastøter partiklene, et dreiemoment som justerer retningen til partiklene og informasjonsutveksling mellom partiklene. Bevegelsen til partiklene blir styrt av disse vekselvirkningene i tillegg til en diffuderende oppførsel i retningsvinkelen. Modellen er inspirert av Vicsek modellen. Vi har brukt informasjonsoverføringen til å simulere spredning av smitte mellom to mennesker, men modellen kan også brukes til å studere informasjonsoverføring generelt, foreksempel i celler i vev.

Fra resultatet av simuleringen vi har gjort har vi sett at vi kan redusere hvor mange som blir smitted ved å endre på hvor mye partiklene i modellen kan bevege på seg. Dersom alle partiklene har et lite, begrenset område de kan bevege seg i er det ferre som blir smittet en dersom noen av partiklene kan bevege seg fritt i hele området. Vi har også sjekket dette resultatet ved å sammenligne den aktive modellen med en gitter-modell der partiklene ikke kan bevege seg, men har forhåndsdefinerte partikler de kan vekselvirke med. Dette resultatet forteller oss at vi kan redusere smitten ved å begrense kontakten med andre til de som bor rundt oss.

Når vi testet ulike tiltak i modellen vår fant vi at all tiltakene var effektive måter å bremse smitten på. Ved å halvere sannsyligheten for å overføre smitten mellom to personer ble antall personer smittet drastisk redusert. Å finne ut hvem som var smittet og isolere disse hadde også en positiv effekt på smittetallet. Å redusere antall personer som kunne møtes på samme sted var det tiltaket som spredde smitten mest over tid. Alle resultatene vi har funnet vil i stor grad avhenge av hvilke parametere som blir brukt i simuleringen og vil også bli påvirket av fluktuasjoner i systemet. Hovudfunnet er derfor at modellen kan brukes til å smiulere smitte og kan dermed være et verktøy for å stå hvordan en epidemi sprer seg.

Contents

List of Figures

Chapter A

Chapter 1

Preface

This work is the continuation of the master's project last semester, where a numeric active matter model we developed in Python. This model was used to simulate the collective migration of epithelial cells. The original plan was to continue the work with these cells in the master thesis and a model written in C++ was developed with this goal. The model was rewritten in C++ in order to improve the running time such that systems with more particles could be considered. During the work with the master thesis, the outbreak of the COVID-19 pandemic started. This caused us to consider if the model developed could be used for simulating an epidemic spreading in a population. It was found that with small changed the model could be used for this purpose. In March, we therefore decided to change our focus from epithelial cells to simulating an epidemic.

First of all, I would like to thank my supervisor Professor Paul Gunnar Dommersnes for admirable guidance while writing this thesis. He has shown excellent support, been a motivational factor during the whole period and provided me with thorough feedback. I would like to thank him for the opportunity to do this master thesis. In addition, Professor Ingve Simonsen has been available for questions and discussions regarding the numerical model through this semester. I appreciate the help and guidance.

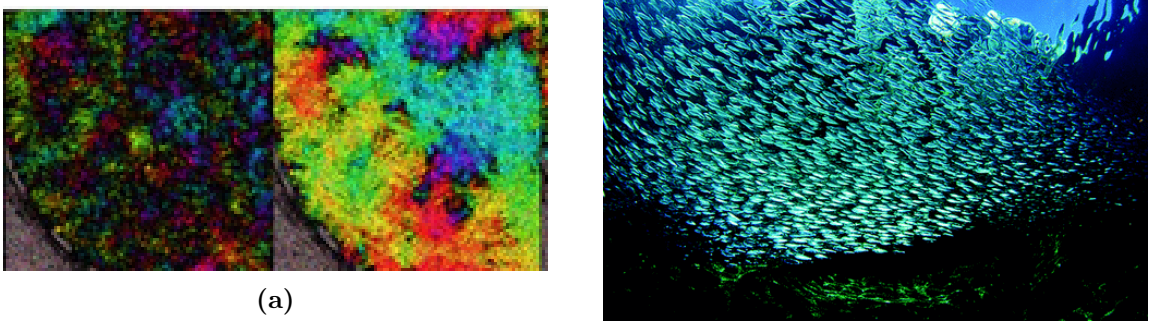
I would also like to thank my fellow student, Edvard Stubberud. The conversations and cooperation have been highly appreciated. Finally, I would like to mention my closest friends and family who have given me strength and support during this work. A special thanks to Harald Wilhelmesen for all the help with my computer and Shahzeb Syed for always being available for questions.

Chapter 2

Introduction

According to the World Health Organization (WHO), a global influenza pandemic was one of the ten most severe threats to global health in 2019 [1]. Among the other nine threats, we find noncommunicable diseases, antimicrobial resistance, Ebola and other high-threat pathogens, vaccine hesitancy, dengue and HIV. Five of these ten threats are directly concerning viruses and two of them, noncommunicable diseases and antimicrobial resistance, can strongly affect people's ability to survive a global outbreak of a virus. The threat of a pandemic became very clear to the world early in 2020 as the SARS-CoV-2 virus, causing the respiratory tract infection called COVID-19 started spreading across the world at a disturbing speed. The 11th of March 2020 WHO declared the virus a pandemic and the threat was real [2]. As long as the world population keeps growing and people keep travelling across the world, such pandemic will stay one of the most dramatic threats to humanity. There are still many things we do not know when it comes to how a virus spreads in a population and how we can slow down this spread with measures that do not affect people's lives too much. It is difficult to research this in a real population, since the consequences of making the wrong choices can be severe, and in worst case fatal for a large part of the population. Numerical models simulating the spread of a contagious disease in a population can, therefore, be an essential tool to help us learn more about this subject.

In this thesis, we will use an active matter model to simulate a population of humans. An active matter model can be of interest because the constituents are moving, and we can mimic different types of human behaviour in the system. In an active matter model, the constituents, called active agents, are living objects. These objects can be all from single-celled organisms such as bacteria to herd animals and humans. The challenge in simulating living objects is that these systems tend to be out of thermodynamic equilibrium, in contrast to most other systems that are thoroughly studied in physics. This deviation from thermodynamic equilibrium comes from the active agents' ability to absorb energy that is stored in the environment and transform this energy into direct motion [3, 4, 5]. This is one of the reasons why the active matter domain has been left out of conventional physics for a long time. We have seen a growing interest in this field in the later years, due to a growing interest in the field in the cell biology environment and due to better computational tools.



(a) **(b)**

Figure 2.1: The figure demonstrates how large groups of living entities can align their motion and thereby move in collective. a) Serum-stimulated collective migration in quiescent cell sheets. The color of the cells indicates the direction of motion, while the strength of the color indicates the velocity of the cells (reused from [7]). b) The figures shows a school of sardines that moves in the same direction. This collective motion in the same direction can be seen as a polarization in the system (reused from [4]).

Active systems consist of a large number of active agents. These active agents are characterized by their transduction of chemical energy into direct motion [6]. There can be many types of interactions between these agents. Repulsive and attractive forces are the most obvious. In this model, the forces working between the particles will prohibit them from moving into each other, but they can also induce clusters of different sizes in the system. The direction of motion is often aligned between particles in active systems. An example is cells, which tends to move in parallel or anti-parallel to each other, often due to their oblong shape. We can model this alignment by adding a torque working between the particles, making them adjust their directional motion with regards to each other. The alignment torque makes it possible to create a preferred way for the particles to move relative to each other. Another type of interaction is called information exchange. Information exchange can be used to model how information spreads through a population. Examples of this can be how a rumour spreads in a population or how cells communicate. In this model, we will use information exchange to simulate how a virus can spread from one infected person to a healthy person. In general, information exchange has been studied less than many other types of interactions, and even though the discussion in this thesis will be regarding epidemics, the model can be used for any type of information exchange. By using models with information exchange, we can understand more about the dynamics of a typical active matter system.

Typical every-day examples of active matter systems are birds flying in flocks, schools of fish and humans moving in a crowd. We also find active matter systems on a micro-scale, examples would be cells moving in collectives or bacteria in fluids. Figure 2.1 shows some examples of active matter systems.

In this thesis, we will build a model that simulates humans moving more or less randomly around in a restricted area. We start with a system where a few agents are infected by a contagious disease, and we will look at how this disease spreads in the population. The active model will be compared to a static lattice model. We will investigate how different factors, like the probability of spreading the disease and the distance at which the disease is contagious, will affect how the disease hits the population. Also, we will introduce some rules for how the active agents in the system are allowed to move. These rules will mimic the changes a government can introduce in a community when such a pandemic is a reality. By doing this, we wish to study what effect these measures have and which of them is most effective.

Chapter 3

Theory

In this chapter we will present the general theory of an active matter system, deriving the equations describing it. We will later use the theory to develop our active matter model for simulating a epidemic spreading in a human population. The theory is presented in sections 3.1 to 3.5. In the last section, 3.6, we will give an introduction to pandemics and present some important terminology commonly used when discussing pandemics. We will discuss specifics of how we can build an active matter model simulating a disease spreading in a population and the possible applications.

Active matter systems consist of a large number of individual units, also called self-propelled particles. Through this thesis, these particles will also be called people, humans, agents and objects. Active systems differ from passive ones in the fact that their units are able to find and absorb energy from their surroundings. This energy is converted into direct motion through an irreversible process. In an irreversible process, the probability of going from state A to state B is not the same as the probability to go from state B to state A. Because of this, active matter systems can not be described by equilibrium statistical mechanics, but they still have several similarities to statistical mechanics [8]. One important thing they have in common is the ability to be in different phases as the temperature and density are changed. A known phase transition for an active system is from a disordered/gas phase to a flocking/polar liquid phase. This is familiar from the Ising model, which experiences ferromagnetic phase transitions from disorder to order.

Another known quality in statistical mechanics is that all microscopic models showing the same symmetry and conservation laws will have the same macroscopic behaviour. This means that the big picture is more important than the details of the mechanics in a system. This motivates using a model that is as simple as possible, as long as the model can display a phase transition. A natural choice is, therefore, the Vicsek model, which are able to find phase transitions even though it is quite simple.

3.1 The Vicsek model

One of the most important and recognized models within active matter is the Vicsek model, proposed by T. Vicsek et al. in 1995 [9]. The model's popularity can be explained

by its rich, realistic results despite its simplicity. The system described by the model consists of N particles constrained in a two-dimensional square container with size $L \times L$. In the Vicsek model, periodic boundary conditions are used. The initial conditions are simple: the particles are placed at random locations within the square and their directional orientation, given by the angle θ, are chosen randomly. All the particles have the same absolute velocity, v, which is kept constant at all times. Each particle i has a position $\vec{r}_i$ and a velocity $\vec{v}_i$, where the orientation of the velocity is given by the angle, θ_i. The particle position at any given time only depends on the previous configuration, making the equation of motion simple.

$$\vec{r}_{i,n+1} = \vec{r}_{i,n} + \vec{v}_{i,n}\Delta t = \vec{r}_{i,n} + v\Delta t(\cos\theta_{i,n} + \sin\theta_{i,n}) \tag{3.1}$$

with a discrete time $t_n = \mathrm{n}\Delta t$.

The equation above contains mostly constant parameters, as the absolute velocity and the time step. The previous position is given, and thus the only varying parameter left is the angle, θ. The Vicsek model defines the angle in the following way

$$\theta_{i,n+1} = \langle\theta_{j,n}\rangle_{r_c} + \widetilde{\eta}. \tag{3.2}$$

$\langle\theta_{j,n}\rangle_{r_c}$ denotes the average angle of the neighbouring particles. This neighbourhood is limited by a circle with radius r_c around particle i, and the middle value also includes the angel of particle i itself. The second term, $\widetilde{\eta}$, is a stochastic term with a Gaussian distribution in the interval [-η, η]. The random noise, which can be interpreted as the temperature, competes with the energy-like alignment of the particles. The relation between these two competing factors will decide the dynamics in the system.

In their study, Vicsek et al. discovered a kinetic phase transition from a disordered system to a system with complete order. The phase transition can be seen by studying an order parameter for the system, Π. The definition of this parameter is given in the following.

$$\Pi \equiv \frac{1}{Nv}\Big|\sum_{i=1}^{N}\vec{v}_i\Big| \tag{3.3}$$

Π can be interpreted as the total degree of order in a system with N particles.

In the limit with zero noise, interpreted as zero temperature in the system, they found that the order parameter became unity, meaning that all the particles are moving in the same direction. In the other limit, letting the temperature go to infinity, given by $\eta = \pi$, the order parameter is zero. The high temperature in the system gives the particles enough energy to break free from the alignment and move in any direction they find favourable. The high energy dominates, and the particles in the system move randomly, creating chaos. In Vicsek et al. original work, they found that the phase transition from disorder to order happens at $\eta \approx 0.3\pi$. This can again be compared to statistical mechanics and the spin models, where the magnetization works as an order parameter.

3.2 Langevin equation

In the early twentieth century, Jean Perrin performed an experiment on colloidal grains suspended in water, aiming to describe their trajectories [10]. This made him one of the first physicists to ever try to extract quantitative information from such a system. His work motivated Paul Langevin, who in 1908 published an article describing the random movement of a particle in a fluid due to collisions in the fluid [11]. Langevin found that the motion could be described by two contributing terms: a mean drag force and a random force caused by collisions between the particle and the fluid surrounding it. By newtons second law this leads to the following equation

$$m\frac{d^2\vec{r}}{dt^2} = -\gamma\frac{d\vec{r}}{dt} + \vec{\xi}(t), \tag{3.4}$$

which today is know as the Langevin equation. This is a special case of Brownian motion. Here, m is the particle mass. The drag force opposes the movement and γ is the magnitude of the sum of viscous forces. $\vec{\xi}(t)$ represents the collisions in the system and is given as a random Gaussian noise.

The Langevin equation can be used to describe a system with passive Brownian particles. In passive systems, there are external forces and fields controlling the motion of the constituents. This gives a symmetric system which will end up in an equilibrium state. We also have some passive systems that can be said to break symmetry, like raindrops falling towards earth in a gravity field (even though if the whole world is considered, the raindrops will fall in the opposite direction on the other side, and there is global symmetry). In active systems, on the other hand, we find steady states that break symmetry without any external fields or forces, due to the energy absorption. We have to add a term describing the energy absorption to the Langevin equation to be able to model an active system.

3.3 Active systems

The self-propelled particles' ability to absorb energy from the system and convert this into direct motion can be regarded as a force working from the particle on the system. This means that the particles are exerting a net force on the system, pushing it out of equilibrium and making it break the symmetry. We call this force the self-propulsion force, F_p, which can be described by the particles self-propulsion velocity, $\vec{v}_p$. This absorption of energy also leads to a change in the net momentum in the system, which Vicsek et al. found to be the reason for phase transitions in active matter systems.

This self-propulsion force must be added to equation (3.4) in order to describe an active system. This gives a new equation of motion on the following form

$$m\frac{d^2\vec{r}}{dt^2} = -\gamma\frac{d\vec{r}}{dt} + \vec{\xi}(t) + F_p\vec{u}(t). \tag{3.5}$$

Here, $\vec{u}(t)$ is the direction of the self-propulsion.

When the Reynold number is small, the drag forces in the system dominate, and other forces can be neglected. This gives a special kind of Brownian motion, called overdamped Brownian motion. The result is

$$\frac{d\vec{r}(t)}{dt} = \frac{1}{\gamma} v\vec{u}(t) \tag{3.6}$$

with

$$\vec{u}(t) = (\cos\theta, \sin\theta) \tag{3.7}$$

in two dimensions. $\vec{u}$ gives the direction of the velocity. The Langevin equation also has a stochastic noise term, $\vec{\xi}$, but for active systems the diffusive motion of the self-propulsion swimming dominates, and the stochastic noise can be neglected.

As in the Vicsek model, the directional orientation of the particle is affected by the collisions between the particle and the fluid surrounding the particle. This can be described by the following equation

$$\frac{d\theta(t)}{dt} = \sqrt{2D_r}\eta(t) \tag{3.8}$$

The collisions are represented in the η term in equation (3.8). η is a random number drawn from a uniform distribution with the following properties,

$$< \eta(t) >= 0 \tag{3.9}$$

$$< \eta(t_1)\eta(t_2) >= \delta(t_2 - t_1) \tag{3.10}$$

The strength of the stochastic process in the fluctuations of the orientation is given by $\sqrt{2D_r}$ where D_r is the rotational diffusion coefficient. The rotational diffusion coefficient is defined such that $D_r = 1/\tau_0$, where τ_0 is the decorrelation time. Further, the decorrelation time can be used to define the persistent length.

$$l_p = v_0\tau_0 = \frac{v_0}{D_r}. \tag{3.11}$$

The persistent length is the distance a particle typically move before changing the direction it moves in. In a system with a small rotational diffusion coefficient, the particle will move in the same direction for a longer time than in a system with a larger diffusion coefficient. In the limit where D_r approaches zero, the persistent length will approach infinity, and the particle will move in a straight line as long as nothing else is affecting it. In the opposite case, $D_r \to \infty$, the persistent length, $l_p \to 0$ and the system is completely dominated by the stochastic term.

3.4 Interacting particles

In the Vicsek model, every particle is considered to be a point particle. In order to change the particles from being point particles to having some extent, we can add a repulsive force between every particle. By adding such a force, we can make sure that no two particles are closer to each other than some minimum distance, and this distance can then be seen as the diameter of the particles. A common way to do this is by adding a potential between the particles. A Lennard-Jones potential is often chosen for this purpose. The Lennard-Jones potential is given by

$$V(r) = 4\epsilon\Big[\Big(\frac{\sigma}{r}\Big)^{12} - \Big(\frac{\sigma}{r}\Big)^{6}\Big]. \tag{3.12}$$

Here ϵ is the potential strength, r is the particle-particle distance, and σ is the distance at which the inter-particle potential is zero. At particle-particle distances smaller than σ, the first term is dominating, and the potential is repulsive and rapidly growing as the inter-particle distance is decreasing. This term enforces excluded area for the particles, and σ can be interpreted as the particle diameter. For distances larger than σ, the potential is attractive, but decreasing as the distance increases. This term works as a force to induce clustering in the system.

3.4.1 Competing interactions

For the potential given in equation (3.12) it is expected that the particles will attract each other but not overlap. It is therefore reasonable to that the particles will cluster, but that the sizes for the clusters will depend on the relation between the potential strength and the noise term, and on random fluctuations in the system. Many system found in nature have competing interactions that give rise to clusters of different sizes. This can be imitated in the model by using both repulsive and attractive long-range forces. The strengths and ranges of these forces can be adjusted relative to each other to control the dynamic system.

$$V(r) = 4\epsilon\Big(\frac{\sigma}{r}\Big)^{12} - C_a e^{-r/R_a} + C_r e^{-r/R_r}. \tag{3.13}$$

Equation (3.13) is a general form of a potential that gives competing interactions. The first term is the short term repulsive part from the Lennard-Jones potential. This is kept to ensure that the particles have excluded area. The second term is attractive, and the third term is repulsive. The four parameters C_a, R_a, C_r and R_r in the potential can be used to control both the strength and the range of the two terms, in relation to each other. This potential gives more freedom and the ability to study different types of dynamics in the system. By choosing $R_r > R_a$, the attractive potential will die out faster than the repulsive one and therefore have a shorter range. This will make the particles in the system cluster, but limit the cluster size. In the opposite case, $R_a > R_r$, the repulsive potential will have the shortest range, and this leads to a system with larger clusters. We know that different types of living beings behave differently, and with a potential on this

form, we can adjust our model according to which objects we wish to study. While birds often fly in large flocks, humans tend to form smaller groups. At a micro-scale, we also see that for example, cells often have a preferred group size when they move in collective.

The equation of motion can be changed to include the inter-particle potential. This gives the following equation of motion for a given particle i

$$\frac{d\vec{r}_i}{dt} = v\vec{u}_i(t) + \frac{-\nabla V_i(\vec{r})}{\gamma_t}, \tag{3.14}$$

with γ_r as the translational friction coefficient and $V_i(\vec{r})$ as the potential between particle i and all the other particles in the system.

3.4.2 Alignment

In the Vicsek model, the direction of every particle is affected by the neighbouring particles' directions by simply letting it be the average of the neighbourhood direction and some noise term. A more recent approach to this is what was done by A.P.Solon et al. in 2015 [12]. In their article, *Pressure is not a state function for generic active fluids* they define the equation for the angle in the following way

$$\frac{d\theta_i}{dt} = \frac{1}{\gamma_r}\sum_{j=1}^{N} F(\theta_j - \theta_i, \vec{r}_i, \vec{r}_j) + \sqrt{2D_r}\eta. \tag{3.15}$$

Here F is an alignment torque and γ_t is the rotational friction coefficient. With the alignment torque used in the Vicsek model, the particles will strive to move in the same rotational direction. While this might be the case for some active system, there can also be other preferred ways to align. An example is rats which seem to prefer moving parallel or anti-parallel to each other. The way humans move is often more complex than the same for animals, but in many situations, it will be natural also for humans to move either parallel or anti-parallel to each other. Examples of such situations are when walking on the sidewalk, in a store or in a hallway. This type of behaviour can be induced by adding the following alignment torque to the equation

$$F_i = \frac{\gamma}{N_0}\sum_{j=1}^{N_0} \sin(2\theta_j - 2\theta_i). \tag{3.16}$$

Here, γ is the strength of the alignment and N_0 is the number of particles within some critical radius, r_c. The sum is over the neighbouring particles limited by a critical radius surrounding particle i. By removing the multiplication with 2 inside the sine function, the torque would again align the particles to be parallel to each other. This would, therefore, effectively do the same as in the Vicsek model.

3.5 Known properties

One of the great challenges with building a numerical model is to verify that it is true to reality. The model has to be able to recreate known results, and it is important that the results do not break any physical laws. In this section we will introduce some known properties that the model must have in order to say that the model is trustworthy.

3.5.1 Energy conservation

The first law of thermodynamics is one of the most fundamental laws of physics. The law states that energy can neither be created nor destroyed. The work done on the particles must cause a change in the total energy in the system. The energy balance of the system can be represented by three terms: the friction, the propulsion force and the mechanical force. With the Langevin equation as a base, this gives

$$\gamma_t \frac{d\vec{r}}{dt} = \vec{F}_p + \vec{F}_m, \tag{3.17}$$

which can be rewritten to the following equation

$$\int_0^t d\tau \left(\gamma_t \vec{v}^2 - \vec{F}_p \vec{v}\right) = \Delta V. \tag{3.18}$$

Here, the integration gives the work done by the self-propulsion and the friction in the system. The term on the right side is the change in the potential energy for the particles, given by the inter-particle potential V.

Because conservation of energy is such a fundamental part of physics, it is also a good way to test a model. Any derivation in the system energy has to come from either numerical errors or some more comprehensive mistake in the model. Such a deviation is a good measure of the accuracy of the model and can be used for picking good parameters and a small enough time step.

3.5.2 Expectation values

Another way of testing the model is to numerically calculate some expectation values and see that they are the same as the theoretical expectation values. There are several interesting expectation values to consider in a Brownian system. The first one is for the parameter which describes the orientation of motion, θ. For a free Brownian particle, the orientational direction is given by

$$\theta(t) = \int_0^t dt' \sqrt{2D_r}\eta(t'), \tag{3.19}$$

Here, the initial direction is chosen in the positive x-direction, $\theta(0) = 0$. From this, the expectation value must be

$$\langle \theta(t) \rangle = \int_0^t d\tau \sqrt{2D_r} \langle \eta(\tau) \rangle = 0, \tag{3.20}$$

given by the definition of η in equation (3.9).

The expectation value of the squared angle can also be used as a check of the model. This value is given by

$$\langle [\theta(t)]^2 \rangle = \int_0^t d\tau \int_0^t d\tau' 2D_r \langle \eta(\tau)\eta(\tau') \rangle = 2D_r a^2 t. \tag{3.21}$$

Here, η is picked from the interval [-η, η]. This is given by the second property of η, given in equation (3.10). The expectation value of the absolute value of the angle, $|\theta|$ is therefore proportional to $\sqrt{t}$.

The systems ability to move in a constant direction over time is captured by assuming that the correlation of the orientation decays exponentially in time [13]

$$< u_\alpha(t) u_\beta(0) >= \delta_{\alpha\beta} \frac{v^2}{d} e^{-t/\tau}. \tag{3.22}$$

Here, d is the spatial dimension, v is the absolute value of the self-propulsion velocity, and τ is the decorrelation time. This is the last expectation value we will use to test the model.

3.5.3 Phase transitions

All the previous tests focus mostly on the physics of the model and that the results follow physical and mathematical laws. This is, of course, important, but not sufficient. In addition, we have to test that the model behaves as expected with regards to simulating active systems. One of the demands we have is that the model should be able to display phase transitions, and a good test is to see if we can produce the same phase transition as Vicsek did from a disordered gas phase to a system with total order, $\Pi = 1$. This will be the last test we do on our model.

3.6 Simulating a pandemic in an active matter model

Now that the theory of an active matter system has been presented, we shift our focus to the more specific case of simulating a pandemic spreading in a human population. We begin by giving a short introduction to what a pandemic is.

The World Health Organization defines a pandemic as a world spread of a new disease [14]. There are several things that separate a pandemic from, for example, seasonal influenza. One of these is that no one or very few are immune to a pandemic since the virus is new. Another difference is that while the casualties of seasonal influenza mainly are elderly, pandemics tend to have more young victims. Pandemics are also generally more severe and have a larger impact on the world. This is much because of the lack of immune persons which causes the number of infected to be high. When the total number

of infected persons is high, the number of people that develop a severe illness can be dramatic, even if the disease is mild and harmless to most people. Since a pandemic is caused by a new disease, we do not have a vaccine against it as it breaks out. A goal is often to work to slow down the spread until such a vaccine is ready. To do this, we need to know how the disease spreads and how fast it spreads in a population, meaning how many can we expect will be infected by one infected person. This can be measured by something called the reproduction number.

3.6.1 The reproduction number

The reproduction number is a measure of how easy the disease is spread from one infected person to other susceptible persons. A susceptible person is a person that is healthy and not immune to the disease. The basic reproduction number, R_0, is defined as the number of new cases directly generated by one infected person in a population where everyone else is susceptible [15]. The calculation of the basic reproduction number is not trivial as it depends on several other factors that are difficult to estimate. These factors are given below.

- The amount of contact between people in the population.
- The probability that the disease spreads from one person to another during contact.
- The duration of infectiousness.

The duration of infectiousness can be estimated quite precisely at the early stages of an outbreak of a disease by studying the ones that are infected. The two other factors are more difficult to find a good estimation for. The basic reproduction number can change during an outbreak and can be different in different countries as measures to prohibit spreading the infection are done. Both the amount of contact and the probability of passing on the disease can be altered by quite simple measures in a community.

The basic reproductive number is mostly used for calculating what fraction of a population must be immune in order for the disease to die out by itself. When such immunity is achieved, we say that the population is protected by herd immunity. Herd immunity protects not only the proportion of the population that is immune but also the rest of the population through prohibiting the disease from spreading [16]. In simple models, this proportion is said to be 1 - $1/R_0$. Knowing how large this fraction must be is important since a common way to achieve herd immunity is through vaccination. Vaccinating everyone in a society would be both unnecessarily expensive and risky since herd immunity will have the same effect. Every vaccine has some risk of negative side effects, and it will be better to vaccinate as few as possible. In addition, there will always be someone in the society that should not be exposed to a vaccine due to, for example, reduced immune repository.

Further, we can use the basic reproduction number to the effective reproduction number, R. We have that

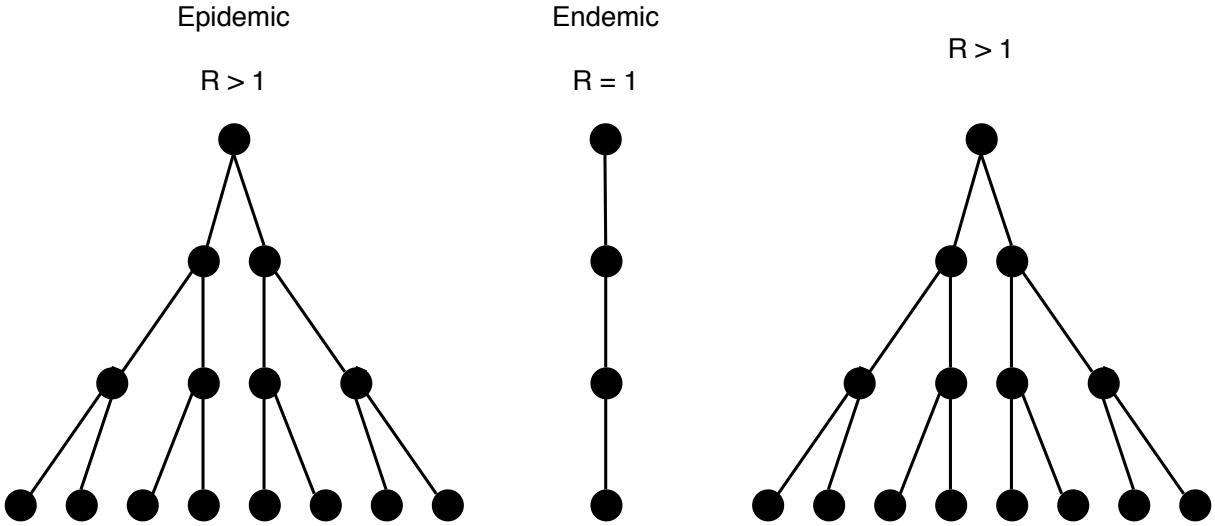


Figure 3.1: How the spread of diseases with different effective reproductive numbers, R, can look.

$$R = R_0 \frac{N_s}{N}, \tag{3.23}$$

where N_s is the number of people in a population that is susceptible while N is the total number of people in the population. The effective reproduction number depends only on the basic reproductive number and the fraction of people in the population that are susceptible. R will decrease as more of the population becomes immune from either having had the disease or vaccination. The effective reproduction number can tell us more about the situation at a given time than the basic reproduction number alone. Figure 3.1 shows three different cases for the effective reproductive number, resulting in different outcomes from a disease outbreak.

The goal during a pandemic is to minimize the number of casualties in the population. The number of casualties will be some fraction of everyone that is infected, but this fraction will depend on several things. The most obvious factor is how dangerous the disease is. Another factor is which part of the population is mostly infected. Every disease will have some group or groups that are less likely to survive it. It would save many lives if we were able to keep an influenza virus out of all nursing homes. The thing that we will focus most on here is the number of people that are infected at the same time. While a vaccine is developed, the goal would be that the disease spreads as slowly as possible, such that as few people are infected as possible. Still, the development of a vaccine can take several years or might not be possible at all. Another way to achieve herd immunity is through letting enough people get infected and immune. If only the part of the population most resistant to the disease is exposed to it, the population could achieve herd protection without infecting the most exposed part of the population and without a vaccine. This must be done by protecting the once that are likely not to survive the illness. It would still be important to slow down the spread as we expect some part of the most resistant part of

the population also to need medical attention. If too many are infected at the same time, there would not be enough resources to help the fraction of the infected that would need it. In addition, filling up the hospitals would also affect the capacity of helping others, as persons with heart attacks and other serious diseases that demand acute help. The goal is to slow down the spread by reducing the effective reproductive number, preferable to keep the effective reproduction number below one. This is the topic of the next subsection.

3.6.2 Slowing down the spread

During the work with the thesis, the world was hit by the outbreak of the coronavirus, which drastically changed the everyday life of most people. Strict rules where introduces by governments in almost every country in the world. Many of these countries closed their boundary to everyone except their own citizens. Schools were closed, cinemas, shops, hairdressers and similar services were not allowed to have costumers. In addition, there were strict rules regarding social interference, like a limitation of how many could meet in one group and how close one could be to other persons. In Norway, this was described as the strictest rules that have ever been, if not counting during wars [17].

Such rules might be necessary to slow down a pandemic and to get control over the situation even though such rules will also have other dramatic effects on a country. As businesses are not allowed to be open, many of them will go bankrupt. As businesses go bankrupt, many people will lose their jobs, leading to a weaker economic situation. As the economic situation is getting worse, people spend less money, leading to more bankruptcy, and so on. The overall consequence can be huge, and the way back to normal can be long.

Another consequence of such a "lockdown" is of a social matter. Children are no longer able to go to school and meet their friends. For many children, this can be challenging as they tend to have a large need for social interactions. This is especially hard on children who experience child abuse, parents that drink to much alcohol and so on. Missing many weeks of school could also lead to fewer children finishing school, which will again have consequences for the economy. The total of these consequences is therefore so severe that it is important to consider which of these rules are really necessary and which of them are the most effective.

The goal of this project is to recreate some of these changes that are done to a society during a pandemic in order to see what effect they have on the disease spreading. To do this, we have limited us to some of the measures that are commonly taken in a country, and these are given in the list below.

1. We can change the radius of infection. This radius describes an area surrounding any infected person where healthy persons can catch the virus. By increasing this radius, we can simulate how it affects the community when there are more total interactions between people or when people are more socially engaged. Decreasing the infection radius will, on the other hand, have the same effect as when people stop hugging, touching each other, touching their face etc.

2. The government are likely to introduces stricter rules for hygiene during a pandemic, both for all public places and services and for all persons. Better hygiene will reduce

the probability of both spreading and catching the virus. This will be modelled by reducing the infection probability.

3. During a pandemic it is common that everyone that know or have reason to believe that they are infected should isolate themselves. This can be modelled by removing everyone that is infected from the system after some time.

4. Another rule we wish to look at is limitations regarding how many people are allowed to meet. This number can vary a lot from country to country and at different stages in a pandemic. We will give one example of such a limitation. This will be done by using a potential that gives competing interactions and thereby limiting the cluster size of active agents.

3.6.3 Pandemic models

Active matter models have mostly been used to simulate constituents with reciprocal mechanical or rule-based interactions. Living entities can also interact through information exchange [18]. On a micro-level, there are different ways this information can be transmitted from one living entity to another. In nature, we see that microbes exchange information through biochemical signals [19]. Another form of information transportation is using light intensity to control and trigger collective behaviours in photo-kinetic bacteria [20]. Models using information exchange can also simulate how information spreads in a population. This is closer to what we are doing in this thesis. We want to use an information exchange model to transfer a disease from one person to another. Paoluzzi et al. have used a model based on information exchange which is discussed in their article *Collective dynamics of logic active particles*, published in February 2020 [18]. They have used AND and OR logic gates in a circuit to model the spreading of epidemics, among other things. This has been done in a SIS model (Susceptible, Infected, Susceptible), meaning that the constituents can be healthy, become infected and then become healthy again. When a constituent is healthy, it has no memory of any sickness, meaning that it can become sick once again. An alternative to this is using a SIR model (Susceptible, Infected, Removal/Recovery) [21]. In a SIR model, the constituents can become sick, and when they recover, they are immune to the decease. The motivation for studying the spread of a pandemic in our case was the coronavirus. Even though it is still unclear whether or not the COVID-19 virus gives immunity during this work, it is assumed that it does, since most viruses humans can recover from gives some kind of immunity. A SIR model is therefore used in this thesis.

3.6.4 Percolation

In the active model, the agents can move quite freely in the system, which also means that they can interact with any of the other agents. An alternative approach when modelling the spread of a virus is to pre-define which agents can interact with each other. This can be done by placing all the agents in a lattice and connecting them through a lattice

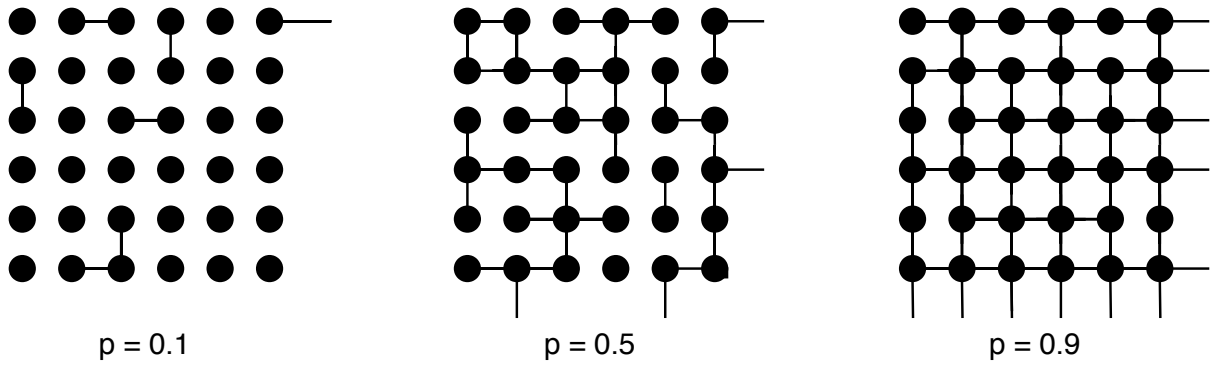


Figure 3.2: We place one active agent at each lattice site in the square lattice. These agents are connected to their nearest neighbours through bounds. Only some portion of these bonds is active, meaning able to transfer a disease. This portion is given by the probability p.

bond. This has been done in several papers earlier [22, 23, 24]. Because of this, we will also develop such a model to have something to compare the results of our active model with. Placing all agents in a lattice with a given number of neighbours can be seen as a percolation model. Percolation theory has many applications and can thus be seen from many different points of views. In general, we have some lattice with N sites and some probability, p, that something is true for each site. One concrete example of a percolation model is the forest fire model [25]. We can divide the forest into small squares where each square is represented by a lattice site. Each such square might or might not have a tree in it. We say that the probability that a random square contains a tree is given by p. Based on this, we can find how long the fire will last, and this time will depend on the probability, p. If almost every site has a tree, the fire will spread out quickly, and the entire forest can catch on fire. In the other case, almost every site is empty, and the fire would not be able to spread where there are large gaps. The fire would die out by itself. It has been shown that for some critical value of p, a phase transition happens. The time for the fire goes from being short to approaching infinity with just a small increase in the probability. We call this value for the percolation threshold, p_c [25].

We define our percolation model by placing one agent at each lattice site, and then we connect these agents to the four nearest neighbours through a lattice bond. In the example with the forest fire, the probability, p, is defined as the probability that there is a tree in a lattice site. In our case, we define p to be the probability that a such nearest neighbours band is active. We define an active bond in the following way: if the agent placed at a lattice site is infected, all other agents connected through an active bond will also get infected. By that definition, the probability p is the probability of spreading the disease from one infected person to a susceptible nearest neighbour of this person, and we will therefore also call p for the infection probability. This lattice and the bonds are shown in figure 3.2. In the figure, only the active bonds are shown, even though there is a bond between every nearest neighbour. The figure shows three different probabilities, $p = 0.1,\ p = 0.5$ and $p = 0.9$, resulting in 7, 36 and 65 active bonds between the 36 lattice sites.

We wish to investigate how the probability affects the spread of the disease in the system. The disease will, of course, spread faster and to more agents as the probability increase, but will this increase be linear as the probability grows? We expect there to be some critical threshold value for the probability, p_c, where we can observe a phase transition in the system, similar to the phase transition that can be found in the forest fire model. We want to find this critical threshold and see if we can change it by changing the model to a small-world network.

3.6.5 Small-World networks

In the percolation model, every agent is able to interact only with the nearest surrounding agents. Such a model can be interpreted as that no one is travelling far away from their home. For many persons, this is not so far away from the truth. Many people work close to where they live and are mostly at home, at work and at the store. In addition to these people, there will be some persons that travel further away from where they live. This can be due to business travels, vacation, working in another city than where they live etc. These people will interact with persons from other neighbourhoods than their own initial neighbourhood. This can be modelled by using a small-world network.

The theory of small-world networks was given by Duncan Watts and Steven Strogatz in 1998 [26]. The concept is built on the small-world phenomenon, popularly known as six degrees of separation. Six degrees of separation is the theory that any two persons on earth are six or fewer acquaintance links apart [27]. Mathematically speaking, in a small-world network, most nodes are not neighbours, but the neighbouring nodes of one node have a big probability of being neighbours themselves. In order to properly define a small-world network, we have to introduce two new words; the average shortest path and the clustering coefficient. The *average shortest path* is, as the name reviles, the average of the shortest path between every node in the network. A small average shortest paths mean that few steps are necessary to move between any two nodes on average. The clustering coefficient is a measure of the degree to which nodes tend to cluster together. The property that makes small-world networks unique is their small average shortest path length and that their clustering coefficient is significantly higher than what is expected by random chance.

Figure 3.3 demonstrates the difference between a regular network and a small-world network. The regular network is effectively the same as the lattice described in the previous subsection about percolation. In the network, all sites are connected to the four closest sites through a bond. In the small-world network, on the other hand, some of these nearest neighbours bonds are removed and replaced with bonds between sites that are further away from each other. The two networks have the same amount of lattice sites and bound, but we see that the number of steps needed to move across the lattice is expected to be lower for the small-world network.

When we add some longer-range bonds in our model, this opens up for spreading the disease from one infected area to a new area where everyone is susceptible. In the regular square lattice network, all the neighbours are in the same neighbourhood, and as the disease has spread for a while, there will be many neighbours that are already infected.

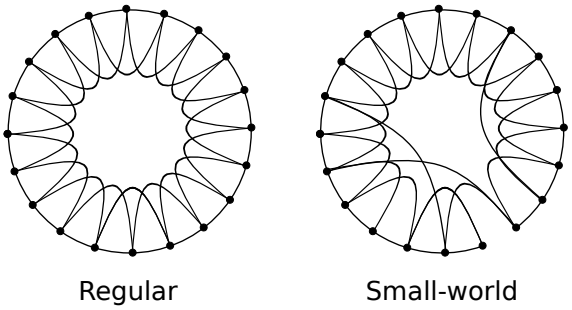


Figure 3.3: The figure demonstrates the difference between a regular network and a small-world network, in regards of which nodes are connected with bonds.

The probability of finding a neighbour to infect is therefore decreasing, and the disease could just die out by itself. In the small-world network, on the other hand, the disease can spread in different areas at the same time, and this can lead to a system where more agents are infected and where the disease reaches a larger part of the system.

We wish to see how adding some of these long-range bonds affects the system. The goal is, therefore, to find the threshold value, p_c for the small-world network and compare this with the one for the regular network. We keep the definition of p to be the probability that a bond between two bonded sites is active.

3.6.6 Challenges

It is very challenging to develop a realistic model of how humans move. Human behaviour is strongly affected by their ability to make independent choices but is also controlled by rules and norms in the society. A big difference between humans and microns is that humans follow patterns that depend on what time of day it is, what day of the week it is, and so on. While microns care little about whether it is night or day, most humans sleep and therefore stay still in the night. In the morning almost everyone is going either to school or to work and are therefore moving. People also tend to have the same routines on weekdays while their routines might be very different on the weekends. Many such patterns can be found when studying human behaviour.

In addition to the patterns typical for humans, there are also many things separating different types of humans. While some humans love social interactions, others tend to avoid big crowds. Some always ride their bicycle no matter where they go, others prefer to go by bus, and some always drive their own car. There are also big differences in office structures in different occupations. Some have large open offices shared by lots of people, others have individual offices and are maybe not in contact with anyone for a whole day.

All these things, and more, are factors that should be considered if the goal was to create a model of realistic movement among humans. This would be very complex and is out of the scope for this thesis. We make some assumptions that we know break with real-life to make a much simpler model. We assume that all constituents in the model move with a constant velocity and that they move continuously. This can be interpreted as that we are only interested in the part of the day when people are moving, while for example, the night is neglected. The movement of our agents will also be much more random than a realistic simulation of human behaviour would be. Even though the active agents are affected by each other, this is a large simplification of human movement. The model should therefore not be considered as an attempt to reconstruct human movement, but rather as a simple model for how the spread of a disease could be affected by changing different parameters. Our goal is to build a model that can demonstrate the effect of different rules and changes in the society on a qualitative scale, and we hope that the changes in our model will be similar to the changes we would expect in a society.

3.6.7 Area of use

During the COVID-19 pandemic, interactive models where one could see how a virus spreads in a population became very popular and almost every large newspaper had one. Some of the first models were given in the Washington Post by Harry Stevens [28] and in Melting Asphalt by Kevin Simler [29]. The purpose of these models can be seen as to make people understand what happens during a pandemic in a way they are not able to simply by listening to politicians and experts talk. People can use these models to see how different measures affect the population. In addition, they can change the parameters used in the simulations themselves. Examples of this would be to change the incubation time, the duration of the illness, how far people could travel etc. Some of the models also have small tasks, like "What's the largest transmission rate where the disease doesn't seem capable of spreading forever (e.g., reaching all four edges of the grid)?" [29]. These interactive articles are unique in the way they include the reader in the search for the answer, making the reader feel like he or she understands and thrusts the results.

The goal of this thesis is to make a similar, interactive model where it is easy to change the behaviour of the virus and the humans in the population. During a pandemic, most countries that have outbreaks of the disease will introduce new rules and routines. People breaking these rules can be punished by fines or even prison, but if they have already contributed to spreading the disease further, is can be too little too late. The challenge is to get everyone to join in on the war against the virus and follow the rules. One way to motivate people into doing this is to help them understand why these changes are important, and what happens if the rules are not obeyed. This is what we hope such a model can contribute to.

In comparison to most of the models published by newspapers, we have done two main changes that separate our model from the rest. The first one is that most of the models assume that once a new rule is suggested, everyone follows this rule. A more realistic scenario would be that most of the population follow the rules, while there is a smaller part of the population which keeps on their day as usual. We use quarantine rules as an

example. Even though everyone that are infected or have reason to believe they could be infected should be in quarantine, it is not realistic to expect that this will be the reality. Some part of the group will not know that they could be infected due to, for example, lack of symptoms. Others might get wrong test results or are not notified that they have been in contact with someone that is infected. In addition, someone will simply ignore it and keep on as usual. Therefore we have modified the model such that it is possible to decide how big part of the population that follows the rules. This can be a strong tool for helping people understand how many that can "cheat" before the restrictions no longer have the wanted effect. The second change we have done is to introduce clustering behaviour among our active agents. This way, we are able to control how large groups of people that are allowed to meet. In most models, there are only two cases; everyone can meet (normal circumstances), or no one can meet (social distancing). This is a weakness for several reasons. To expect that no one will be closer than some distance is unrealistic. Allowing everyone to meet is also unrealistic since humans tend to gather in groups of a limited size. This also gives us the opportunity to see how the limitation of maximum persons that are allowed to be together affects the population.

In addition to creating an interactive model that can be used by "everyone" to better understand what happens during a pandemic, we wish to use the model to gain more physical insight into a pandemic. This is done by comparing the active model to the lattice model, and by finding phase transitions for different systems.

Chapter 4

Method

We are now done with presenting the theory used and will proceed with giving the specifics regarding the numerical model used in the thesis. We start by finding the discrete forms of the equations of motion using Euler's method in section 4.1. Next, a description of the system is given in section 4.2. Some choices done regarding the running time is given in section 4.3 before the parameter values are explained in section 4.4. In the final chapters an overlook on the implementation is presented 4.5 and possible improvements are suggested 4.6.

4.1 Equations of motion

The first thing we need to do is to find a discrete form of the equations of motion. This is done using Euler's method. Euler's method is a first-order, explicit numerical solver for ordinary differential equations (ODEs). A first-order solver has a local error proportional to the square of the step size and a global error proportional to the step size [30]. An explicit method uses the current state of the system to calculate the state of the system at a later time, while an implicit method uses both the current state and a later one to find a later state. When solving a complex system, one usually wishes to use a high order numerical solver to reduce the error and achieve greater stability. The reason for choosing Euler's method in this project is the stochastic term in the equation of motion. Using a higher-order solver for stochastic differential equations is very complex and is therefore not done here. This is the reason why it is common to user Euler's method when solving active matter systems.

Euler's method is defined in the following way

$$y_{n+1} = y_n + \Delta t f(t_n, y_n), \tag{4.1}$$

where Δt is the time step [30]. We start out by applying Euler's method to equation (3.14). We end up at the following equation,

$$\vec{r}_{i,n+1} = \vec{r}_{i,n} + \vec{v}_{i,n}\Delta t - \mu_n \nabla V(\vec{r}_{i,n})\Delta t, \tag{4.2}$$

with $\mu_n = 1/\gamma_n$, where μ_n is the translation mobility. $\mathrm{V}(\vec{r}_{i,n})$ is the inter-particle potential. The self-propulsion velocity is defined as $\vec{v}_{i,n} = v\vec{u}_{i,n} = v(\cos\theta_{i,n}, \sin\theta_{i,n})$.

We also use Euler's method to derive the discrete version of the directional equation (3.8), giving the following,

$$\theta_{i,n+1} = \theta_{i,n} + \sqrt{2D_r\Delta t}\eta_{i,t} + \frac{\gamma}{N_0}\sum_{j=1}^{N_0}\sin(2\theta_j - 2\theta_i)\Delta t \tag{4.3}$$

where $\tilde{\eta}$ is drawn from a uniform distribution in the interval [-η, η].

Euler's method is explicit and uses only the current time step. As an alternative, we could have used another explicit method that uses both the current state and one or more previous states, for example, an Adam-Bashforth method. This was tried but did not give any more stability in our case and was therefore rejected as Euler's method is easier to implement and less computationally demanding.

4.2 System description

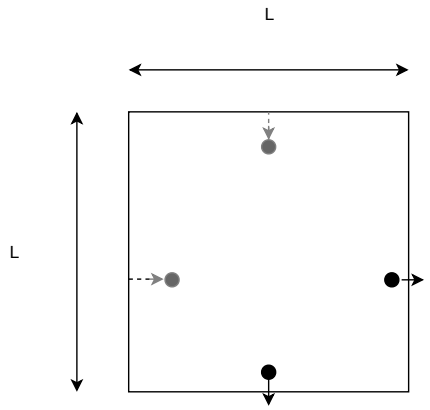
The system consists of N particles in a two-dimensional box with size $L \times L$, as for the Vicsek model. These particles interact with the other particles in the system. There are three different types of interactions in the system; the particle-particle alignment torque, the information exchange transmitting the disease and the inter-particle potential. The alignment is restricted to an area surrounding the particle given by a cutoff radius, r_c. The disease can only be transferred between persons closer to each other than the infection radius, which we will call r_{inf}. The inter-particle potential is not cut off in the same way but is decreasing as the distance between the particles is increasing. The range of the potential depends on the factors in equation (3.12) and (3.13). This will, for most systems, mean that the inter-particle force will be small or zero between many of the particles. An important parameter will, therefore, be the particle density. Higher density will give more interactions, which will affect not only the running time but also the results. To keep control of this, we introduce a parameter which we call the area fraction, describing the density of particles in the system. The area faction is defined as

$$\phi = \frac{N\pi r^2}{L \times L}. \tag{4.4}$$

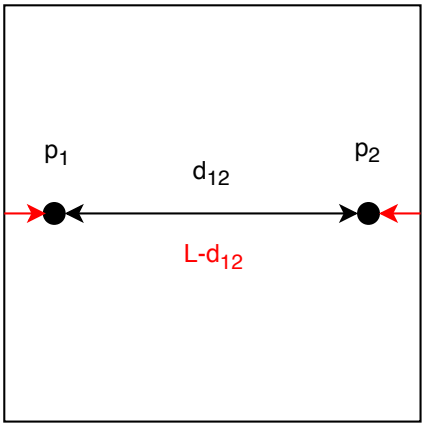
Here, r is the particle radius, which for the potentials given by equation (3.12) and (3.13) will be σ.

4.2.1 Boundary conditions

When defining the system, we considered two different ways to define the boundary conditions. One option would be to add a strong, repulsive potential to all the ends of the box, and thereby simulate the boundaries to be hard walls. The other option is periodic



(a) A particle can move through the "wall" and come out on the other side.



(b) The distance between two particles is always the shortest way between them, including going through the "walls" with periodic boundary conditions.

Figure 4.1: The figure shows the space where the particles can move, which has periodic boundary conditions in both directions. It demonstrates what happens when a particles tries to escape the area it is imprisoned in. The figure also shows how the distance between particles is defined due to the periodic boundary conditions.

boundaries conditions, which is the boundary conditions used in the Vicsek model. Because of this, we chose periodic boundary conditions as well. With periodic boundary conditions, active agents that move out of the box will reappear at the opposite end, effectively transforming the square into a torus where the agents can move on the surface. The particles will still be plotted as they are moving on a 2D square and "jumping" from one end to the other as they go out of bounds, as this is easier to interpret. A system with periodic boundary conditions can be interpreted as that the particles are moving on a surface that are much larger than the particle size. This allows us to focus on the particle interactions without having to be concerned about what happens in the boundary of the box. A system with periodic boundary conditions is illustrated in figure 4.1a.

The periodic boundary conditions do not only affect the positions of the particles but also how the distance between particles must be calculated. This is illustrated by figure 4.1b. Here, two particles, p_1 and p_2 has been places in a square. If the system had hard walls, the distance between these particles would simply be defined by the black line denoted as d_{12}. Due to the periodic boundary conditions, the distance is defined by the red line in the figure instead. This means that the maximum distance between two particles in the system is $d = \sqrt{\frac{L}{2}^2 + \frac{L}{2}^2} = \sqrt{\frac{L^2}{2}}$ instead of $d = \sqrt{2L^2}$ as it would be for the hard walls system. This makes it more complicated to calculate the distance between particles, and has a bad effect on the computational running time, as more checks have to be done.

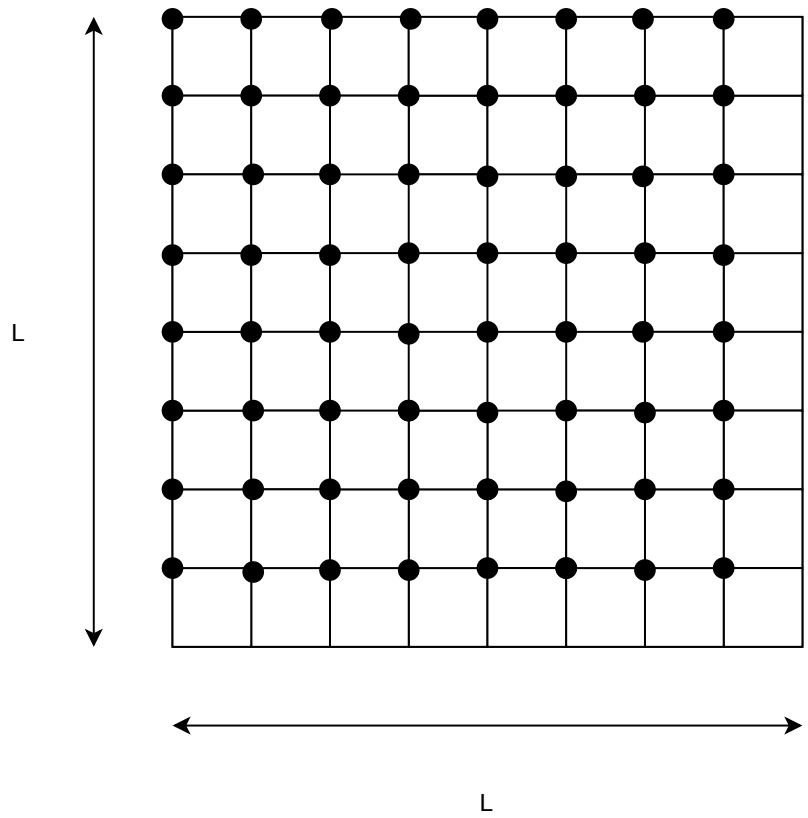


Figure 4.2: Demonstration of the initial position of the particles the system with size $L \times L$

4.2.2 Initial conditions

In the Vicsek model, the particles are placed randomly within the box as a starting position. This works fine for systems with low particle density, but as the density increases, so does the probability of placing two particles in the same spot. If two particles are initially placed too close to each other, they will repel each other so strongly that the particles are moved to a new, random position somewhere in the box. As this new position also is random, the chance of getting to close to another particle is again very high. The same problem occurs again, and the particles will just move around randomly at high speed. When such a problem first has occurred, the system is likely to never reach a steady-state solution.

To avoid the problem of overlapping particles, we have chosen to distribute the particles evenly on a grid as a starting state. This is demonstrated in figure 4.2.

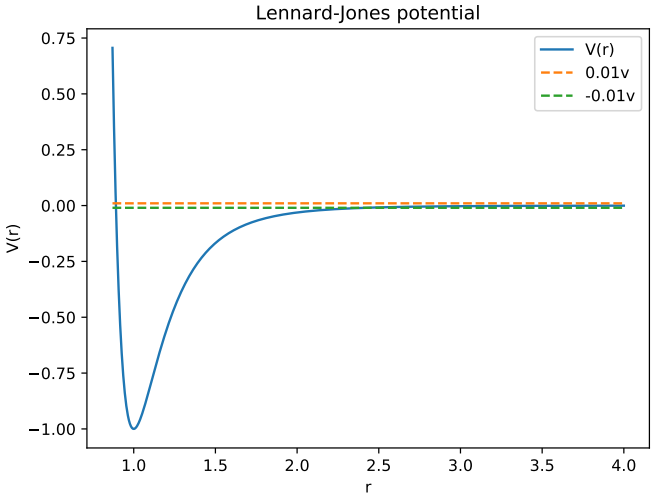
The advantage of this distribution is that no particle is placed within the radius of any other particle. The disadvantage of this is that the initial position is no longer random. The initial directions of the particles self-propulsion velocity, θ, is still randomly chosen, as for the Vicsek model. Since the direction is randomly drawn, the system will approach a steady-state after some time, and the initial conditions will not matter. The lack of randomness in the initial position is therefore not a problem if the running time is long

enough that the initial state is forgotten.

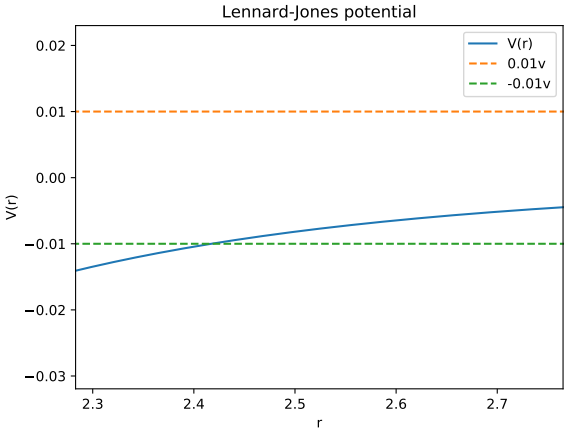
4.3 Running time

Solving active matter systems numerically can be computational demanding. If the code is written in a naive way, the interactions between every pair of particles must be calculated. This will quickly limit the number of particles in the system, N, that we are able to solve without the computational time becoming very long. We have already said that the alignment and the information exchange is cut off at a radius r_c and r_{inf} respectively. This makes it natural to calculate these interactions only for particles that are closer to each other than this critical distance. The force from the potential, on the other hand, is not cut off at a certain radius. This force slowly decreases at the distance increases but will be small for very many of the particle pairs. This force is also the most time demanding to calculate, as it is consists of high ordered exponents.

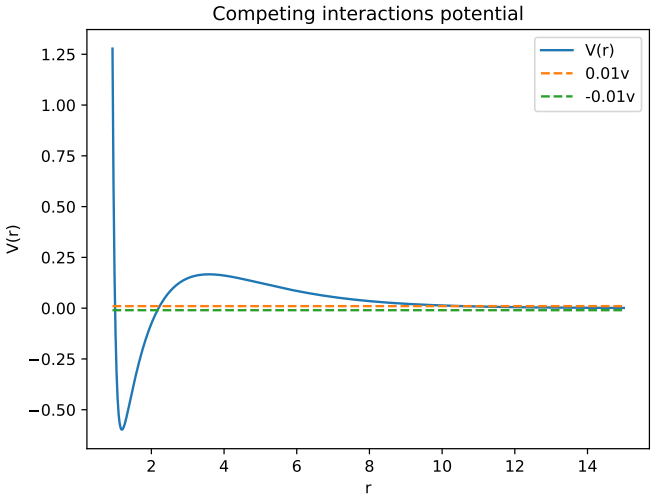
We wish to reduce the number of calculations needed, also for the inter-particle force, and we do this by setting a limit for where this force is small enough that it can be neglected. In figure 4.3 both the potentials defined in the theory by equation (3.12) and (3.13) are plotted as a function of the inter-particle distance, r. The sub-figures contains dotted lines indicating the absolute value of 0.01 times the self-propulsion velocity. In this project, we have chosen to neglect all forces that come from a potential smaller than 1% of the absolute value of v. This is because the effect of the force will be very small, but the computational time it takes to calculate it is long. By doing this, we are able to solve systems with a lot more particles than we would if all the forces should have been calculated precisely. A disadvantage with doing this is that our calculations become less accurate. The sudden cut off in the potential gives a loss of energy, causing a deviation in the energy conservation. If the main goal was to simulate and recreate movement, this would be a problem. In our model, we know that the movement of the active agents already is fare away from reality, and the goal is not to simulate accurate motion. Thus, we accept the deviation in energy as the running time is considerably improved.



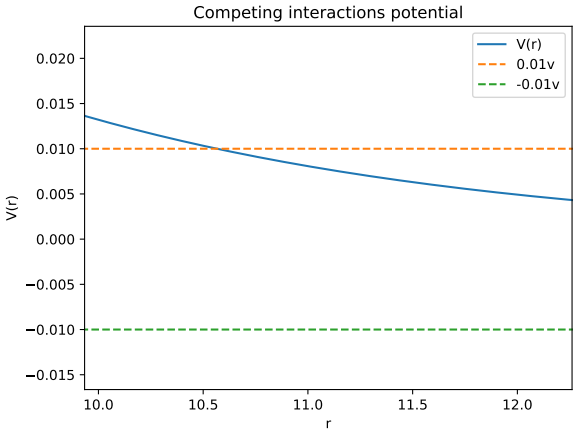
(a) The Lennard-Jones potential defined in equation (3.12) as a function of the inter-particle distance, r.



(b) The Lennard-Jones potential showed in a) zoomed in at the distance where the absolute value of the potential becomes smaller than 1% the absolute value of the self-propulsion velocity.



(c) The potential defined in equation (3.13) plotted as a function of the inter-particle distance, r.



(d) The competing interactions potential showed in c) zoomed in at the distance where the absolute value of the potential becomes smaller than 1 % the absolute value of the self-propulsion velocity.

Figure 4.3: The figure shows the different types of potential as a function of the distance between two particles. The dashed lines in the figures encapsulate the area where the absolute value of the potential is smaller than 0.01 of the absolute value of the self-propulsion velocity. Parameters used is: a and b) $\epsilon = 1$, $\sigma = 1/2^{1/6}$, v = 1 c and d) $\epsilon = 1$, $\sigma = 1/2^{1/6}$. $C_a = 6$, $R_a = 1$, $C_r = 2$, $R_r = 2$, v = 1

In figure 4.3, we can see that for the specific parameters used, the Lennard-Jones potential dies out much faster than the potential with competing interactions. While the Lennard-Jones potential crosses into the $|V(r)| < |0.01v|$ at $r = 2.4$, the other potential crosses this limit first at $r = 10.6$ This means that many more particle interactions must be considered when using the potential creating competing interactions. This potential will, therefore, only be used when we want to study different group sizes.

Figure 4.4 shows the running time for the program as a function of the number of particles in the system. The figure illustrates the difference in the running time when the system calculates the interactions between every particle pair in the system, and when these calculations are limited to the interactions between particles that are closer to each other than the critical distance for the different types for interactions. The graph is produced using the potential given in equation 3.13 and the critical radius was therefore chosen to be $r = 15$. With a box with lengths 100×100, it means that about 7% of the area is considered in each iteration. If the particles are evenly distributed in the system, this gives a considerable reduction in the running time.

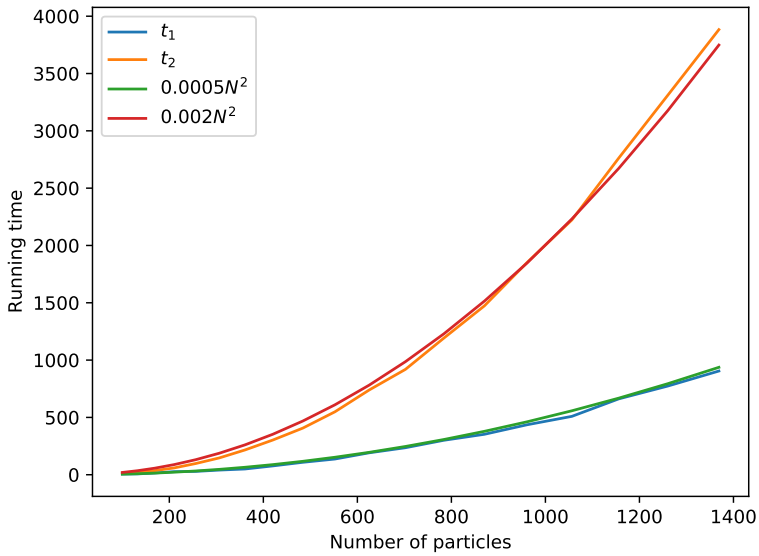


Figure 4.4: The running time of the same system as a function of the number of particles. The figure shows the running time both when a check of the particle-particle distance is done before calculating the force and when all inter-particles forces are calculated. The inter-particle potential is cut off at $r = 15$ in the case with check. Parameters used in the simulation: Time = 100, $\Delta t = 0.001$, $D_r = 0.01$, L = 100, $\gamma = 10$, R = 3, $\epsilon = 1$, $\sigma = 1/2^{1/6}$. $C_a = 1$, $R_a = 1$, $C_r = 1.5$, $R_r = 2$, v = 1

In figure 4.4 we have plotted the running time for 19 different number of particles, from $N = 100$ to $N = 1400$. We see from the figure that the running time is significantly improved by reducing the number of particle-particle interactions considered. The running time for both cases scales as a function of the square of the particle number, $\mathcal{O}(N^2)$. When

Table 4.1: Simulation parameters

Quantity	Symbol	Dimension	Value
Self-propulsion velocity	v	LT^{-1}	1
Time step	Δ t	T	0.01 - 0.001
Time	t	T	350
Rotational diffusion coefficient	D_r	T^{-1}	0.01
System size	L	L	70
Particle diameter	σ	L	$\frac{1}{2^{1/6}}$
Repulsion radius	R_r	L	2
Attraction radius	R_a	L	1
Repulsion strength	C_r	1	1/2
Attraction strength	C_a	1	1
Alignment cut off	r_c	L	2
Alignment strength	γ	1	To be varied
Number of particles	N	1	1024
Effective area fraction	ϕ	1	0.1 - 0.5
Infection probability	p	1	0.1 - 1
Infection time	t_{inf}	T	20
Infection radius	r_{inf}	L	4
Translational friction coefficient	γ_t	1	1

every particle pair in the system is checked the running time scales as 0.002 N^2, while for the limited case it scales as $0.0005N^2$.

4.4 Parameter values

Through the theory and the introduction into the numerical model, many parameters have been introduced. In this subsection, we take a closer look at these parameters, their dimensions and typical values. Table 4.1 shows the parameters defining the active matter system. The values given in the table are just an example of a typical value, as most of the parameters will be varied. The exact parameters used to produce a result will be given in the respective figure.

4.4.1 Time step

The time step is limited by the accuracy of the ODE solver. Since we have used Euler's method, a first-order ODE solver, we need to use a sufficiently small time step. As the time step increases, the length that the particles move in one time step will also increase. The inter-particle potentials used can grow very rapidly, especially for the Lennard-Jones potential. This means that two particles move towards each other with a large time step, they can go from having an attractive potential in one step to having a very large

repulsive potential in the next. If this repulsive potential is too large, we will experience the same as we discussed under the initial conditions, namely that the particle velocity will be huge. This will again give the potential problem of placing two particles in the same space, which can lead the system into chaos.

The problem with choosing the time step too small is that the running time of the program will grow very fast with the decreasing time step. The challenge thus lies in balancing accuracy and running time. The time step has mainly been chosen by initially guessing a suiting time step, running the program and checking the deviation in the energy conservation. If the deviation is acceptable, the time step is approved, if not, a smaller time step is chosen, and the simulation is done again. This way, the time step is chosen as large as possible without losing accuracy. It was found that a time step of $\Delta t = 0.01$ was sufficiently small for most simulations.

4.4.2 Number of particles

The number of particles in the system, N, is an important parameter. From a thermodynamic view, we would like this parameter to go to infinity, but this is obviously not possible to simulate. The particle number will be a compromise between accuracy and running time, as the time step. As we saw in the last section, the running time scales as $\mathcal{O}(N^2)$. Due to the central limit theorem, the error of the measured averages will only scale as $\mathcal{O}(1/\sqrt{N})$. The particle number will mostly be around 900 in this project, as this was seen as a good balance between precision and running time. Calculating the same measured averages of a system with different particle number (but all other parameters, including the particle density, kept the same) would give a good measure of the error. If the two calculations give the same result, the number of particles is large enough. If not, the particle number should be increased.

4.5 Implementation

The code was originally written in Python, but due to computational time, it was rewritten using C++. All the results presented in this report is therefore calculated using the C++ script, while all the figures have been plotted using matplotlib in python 3. The C++ script can be found on Github [1].

The system is solved using three for-loops. The outer one iterates over the time steps, the middle one iterates over the persons in the system, and the inner one again loops over all the persons. In the middle for-loop, the condition of the person, p_1 is considered. If the person is infected and has been so for the entire infection time, the condition is changed to immune. All the other persons are then looped over. Inside this loop, all the forces working on person p_1 from the other ones are calculated. Then, the condition of all the persons closer to person p_1 than the infection radius is checked. If any of them are infected, and p_1 is not infected, they can infect person p_1 with a probability p. After this is done, the new position of the person is calculated based on the forces in the middle

[1] https://github.com/siljeovstetun/masterThesis

for-loop. This is done for all the persons in the system, and then the next time step is considered.

The code is written with very few built-in functions and packages. The built-in functions used are mostly mathematical functions like sin, cos, tan, abs, sqrt and exp all from the cmath library.

The code has been parallelized using OpenMP. This was done by using pragma omp parallel on the second for-loop, over particles. This gave around five times speedup on the computer used for simulations, which has eight threads.

The numerical calculations have been done in C++ while the plots have been produced in Python. The data from the calculations have to be stored and read into the Python program. For systems with many particles and iterations the files can get large ($\sim$ 5GB in our case). Reading so large files in Python is time demanding if the wrong file types are used. In this project, we have used HDF5 files to store the data [31]. Every HDF5 file has a root group which can have one or more under-groups. Using HDF5 files is a good way to structure data which contain several data set. It is also much faster to read large HDF5 files in Python than an identical file of the format *dat* or *txt*.

4.5.1 Object-oriented programming

Object-oriented programming differs from functional programming in the uses objects that belong to a self-defined class. We have defined a class called *People*. The advantage of using object-oriented programming is that attributes can easily be acquired to each object. This was the main reason for choosing to use object-oriented programming in this project. Information as position, velocity and health condition can be added to each object, making it easy to keep control over every person's status.

Figure 4.5 shows an object in the class *People* and all the attributes belonging to the object. These attributes are the x and y position, the directional angle, θ, the self-propulsion velocity, v, the condition, the infection time and the number of persons the infection is spread to. The condition is given as an integer, where 0 means susceptible, 1 means infected and 2 means immune. For some simulations, we will also add the number 3 for quarantine. The infection time is given as the iteration where the person is infected and will not be given at all if the person is never infected. The number of infected is also an integer keeping count of how many persons are infected by this object. The number is set to 0 in the beginning and is increases during the process. Every object has the attribute velocity so that the self-propulsion velocity can be set to different values for different objects.

One of the advantages of using object-oriented programmed is that it is very easy to add more attributes to the objects as they are relevant. There are many things that affect how a person reacts to a virus. It could be useful to add things like age, other diseases, nationality etc. to make a more complex model. Object-oriented programming makes it much easier to change the model as new things are considered and are therefore much more dynamic. This is a big advantage for a complex system, as one often wished to build an easy model at first, and then expand the model to consider more things with time.

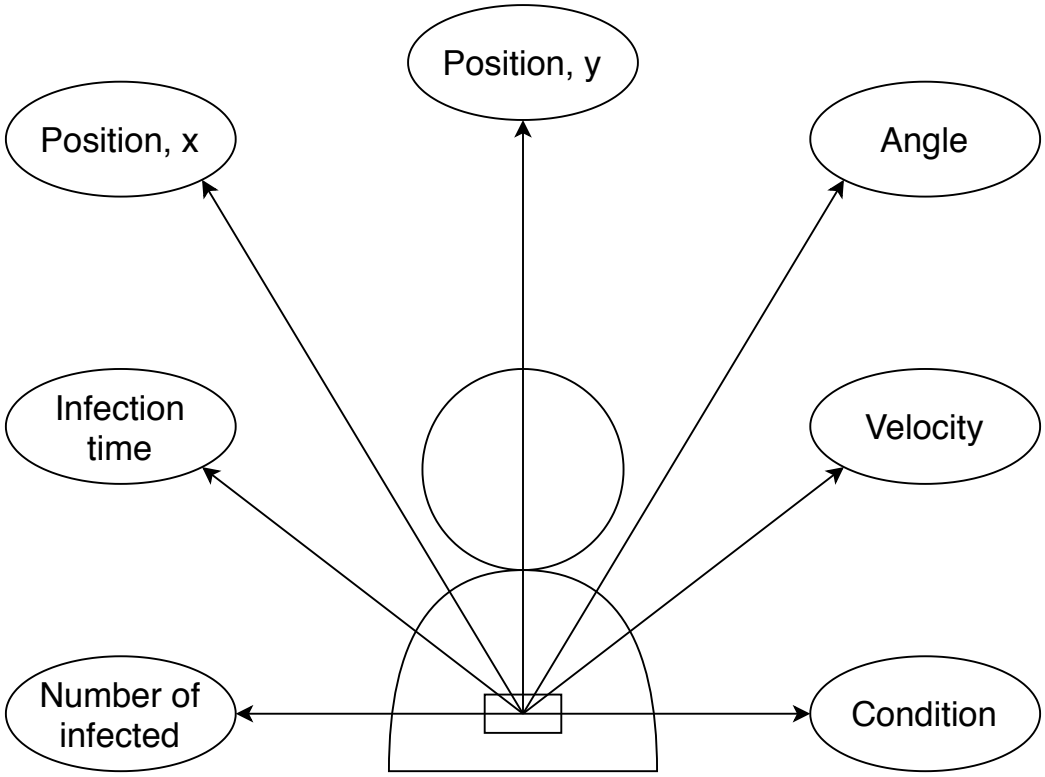


Figure 4.5: The figure shows an object of the class *People* with all the belonging properties in the active model.

4.5.2 Information exchange

We use the concept of information exchange to simulate the spread of a disease from one object to another. This is done by defining a probability, p_{inf}, that the disease spreads from an infected object to a susceptible person. We define this probability such that if $p_{inf} = 0.5$, there is a 50 % chance of spreading the disease from the infected object to the susceptible object if these two objects are closer than the critical distance throughout the entire duration of the infection time, t_{inf}. The infection time is the time from an object is infected to the same object has recovered, and we assume that the object can infect others throughout the entire infection time. The probability of spreading the disease in each time step will therefore be $p_{eff} = p_{inf}\Delta t/t_{inf}$. When an object is infected by the disease, the condition is changed from 0 (susceptible) to 1 (infected). The infection time is set to the current time step. In addition, the attribute *Number of infected* is increased by one for the object spread the infection. When the object has been infected for the entire infection time, the condition is again changed, this time to 2 (recovered). The object can no longer infect others and can not be infected again. We say the object is immune to the disease.

For some of the simulations we do, we wish to consider what happens when people that are infected are removed from the system. We consider this removal as that the objects are put in a quarantine where they are isolated from all other objects. This could

be done by automatically put everyone that is infected in quarantine at the same time that they are infected, but this will, of course, make the disease disappear before the first case can infect anyone. This is not realistic for several reasons; it will take some time from a person becomes infected to that person is put in quarantine. This is because it takes some time to develop symptoms and before that person will do a test. In addition, we have to assume that there will be some proportion of the infected ones that never develop symptoms, or that ignores them and never get tested. It is also realistic that even with a positive test, some of the infected ones will ignore the rules and go on as usual. Because of this, we have chosen the following strategy; When an object is infected, we let it stay in the system from some time. After this time we change the condition of the object to 3 (quarantine) with a probability, $p_{quarantine}$. The object is not really removed from the system but does not longer have the ability to infect others and are not shown in the plots.

4.5.3 Lattice model

In subsection 3.6.4 we discussed percolation theory. Since the particles in such a model do not have to move (the bonds predefined and moving the particles will not change anything) we have made a separate program for these simulations. The model is quite similar to the original model, but with some small changes that we will present in this subsection. The code used for this model can also be found on Github [2]

One of the main differences is the class *People* and the attributes of the objects in specific. Since the objects are no longer moving, there is no need for a velocity and a directional angle. Instead, we have added an array which contains the neighbours of the object, meaning the objects it is connected to through bonds. The new attributes are shown in figure 4.6. The infection probability is defined in the same way as for the initial model.

This model has a considerably better running time because we do not have to iterate through all the objects in the system twice, as we do with the original model. In this case, each object has information about who is their neighbours, and it is therefore only necessary to iterate through these four objects for each object. There is also no need to calculate forces and new positions since the objects are standing at the same positions throughout the simulation. In fact, the objects do not really need a position at all, and this is done in the interest of being able to simulate and plot the system in a meaningful way.

The objects are placed in a square lattice, as demonstrated in figure 4.2. Their neighbours are initially chosen as the four objects close to them, using periodic boundary conditions at the ends. When a small-world network is considered, as discussed in subsection 3.6.5, some of the bonds between the nearest neighbours are removed, and the same amount of bonds between two random objects are added. How many of these bonds are changed will affect the small-world network, and we, therefore, define a new parameter, ρ. We define ρ as the number of bonds that are changed divided on the total number of

[2] https://github.com/siljeovstetun/MasterThesisPercolation

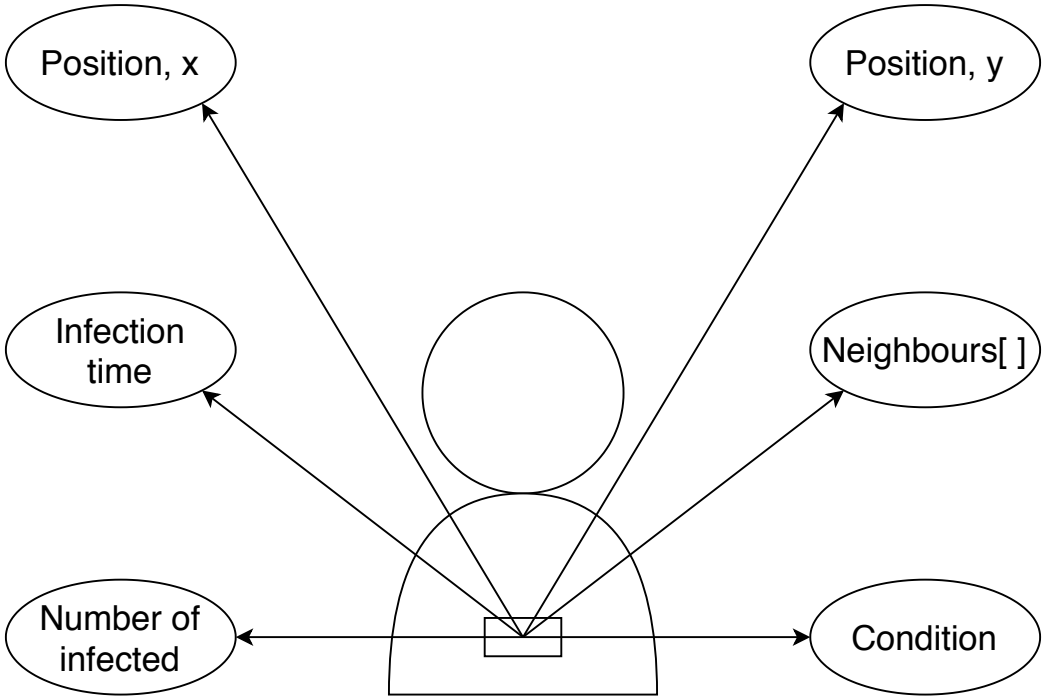


Figure 4.6: The figure shows an object of the class *People* with all the belonging properties in the lattice model.

bonds in the system.

4.6 Possible improvements in implementation

The numerical program used in this master thesis is quite simple and has room for improvements. This subsection will focus on some improvements that could have been done but have not been prioritized in this project.

4.6.1 Varying time step

The implementation of the program has been done in such a way that the time step is the same for all iterations in a simulation. This means that the time step has to be chosen small enough to handle all possible scenarios. It could be time-saving to change this so that the time step could be different for different iterations. A way to do this is by setting an initial time step that is quite high, but small enough that most iterations would be fine. For every iteration done, there should be some kind of check to see if the time step is small enough. This check could be if any of the particles move further than some maximum distance that is set in the start. If this is the case, the time step has been too large for some of the last iterations. The system should then be moved back some steps, and the iterations should be done again, this time with a smaller time step. The challenge here is to find a suitable timestep such that most iterations are approved, but

also such that the running time is improved.

In addition to potentially improving the running time, such an implementation provides a check in the system for each iteration. This check can help discover if something strange happens in any of the time steps. Such errors can be hard to detect otherwise because the error can be hidden if the system goes back to normal after some time. It can also happen that some of the particles jump out of the system (since we use periodic boundary conditions this will only happen if the position is set to NAN or inf) and this can also be hard to detect in a system with a high particle number. Such a check could, therefore, be helpful for several reasons.

4.6.2 Box system

We have already discussed that limiting the area where the inter-particle interactions are calculated reduces the computational time. The way this is limited in the program is by calculating the distance between the particles and then decide if the forces and alignment should be calculated and if the information exchange should be considered. Though this is time-saving, we still have to calculate many distances that are never used for anything. To further improve the running time, we could rewrite the program in a way such that these distances do not have to be calculated for every particle couple.

A way to do this is to divide our system into boxes, as demonstrated in figure 4.7. We have seen that we do not have to consider any of the particles further away than some critical radius, R since the potential between the particles is so small that it can be neglected. If all the particles are divided into imaginary boxes based on their positions, the particles near enough a given particle can be found from this box information rather than calculating the distance between the particles. This will be most time saving for large systems with high particle density.

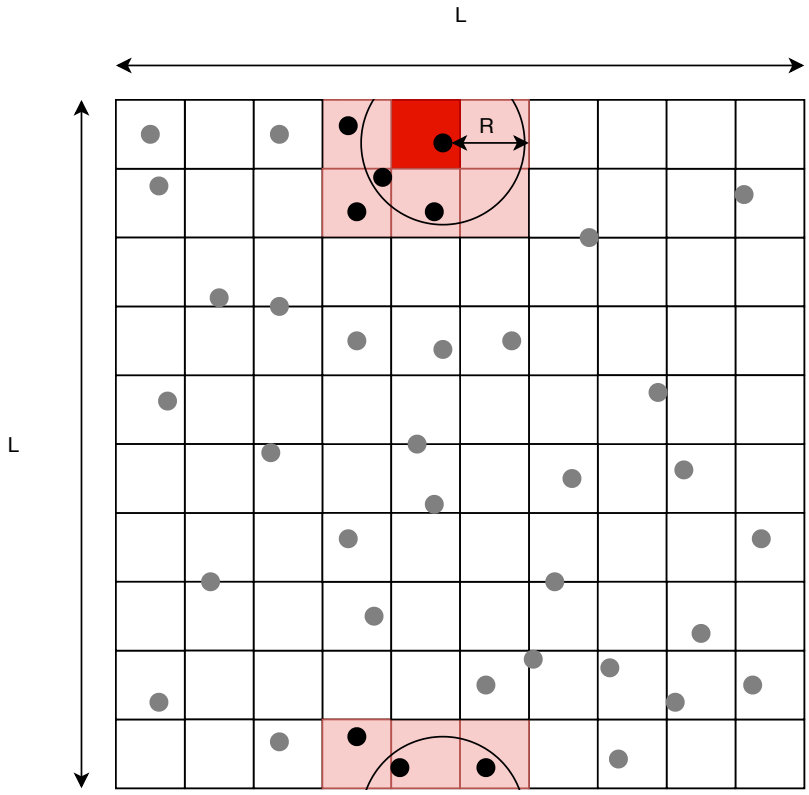


Figure 4.7: Illustration of how a box system could look.

Chapter 5

Results and discussion

In this chapter, the results will be presented and discussed. In the theory some properties we expect from our model was introduced. In the first section, several tests are done on the system to verify that these basic conditions are fulfilled. We then proceed with finding two threshold values, p_c, where a transition occurs for both the nearest neighbors square lattice and the small-world network. These threshold values are discussed in section 5.2. These results are compared with the threshold values for the active model in section 5.3. Finally, in section 5.4, some new rules are introduced in the system, and the effects of these are discussed.

5.1 Verification of the system

The first thing we want to do, it verifies that the system behaves as expected. We use the checks introduced in section 3.5. These checks do not cover all possible errors, but they give a good indication on whether our model is sufficiently reliable or not. We start by running the most basic test; if the energy in the system is conserved.

5.1.1 Energy conservation

The energy conservation in the system is given by equation (3.18). This test is a bit different from the other ones, as it strongly depends on the parameters used. If our goal was to simulate accurate motion, the energy conservation should be checked in every simulation. Since our model is not an attempt to simulate the movement of a real-world system, we have not prioritized this here. We have run several simulations where the energy has been conserved, but we have also had cases where there has been some deviation in the energy. The energy tends to deviate either when the particle density is high, the time step is large, or the potential is defined with a cut-off radius. The fact that we observe energy conservation from some combinations of parameters indicates that there are no fundamental errors in the model, even though we see that the energy is not conserved for all such combinations. Still, the model should not be seen as an attempt to correctly describe the motion of active particles as the energy conservation has not been

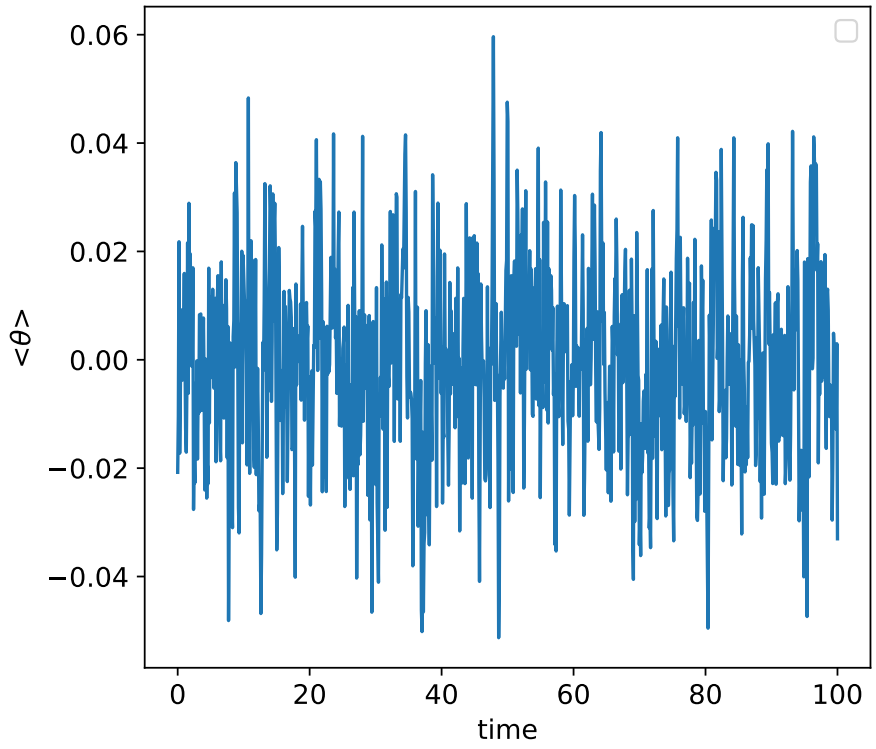


Figure 5.1: The expectation value of the directional orientation, θ. The figure shows that the directional orientation is drawn from a uniform distribution. The result is made by using only the repulsive part of the Lennard-Jones potential and no alignment. The parameters used is $N = 1024$, $dt = 0.001$, $D_r = 1$. The result has been averaged over ten simulations.

checked in every simulation.

5.1.2 Expectation values

In addition to energy conservation, we have used the expectation value of some of the parameters in the program as checks. One of these parameters is the directional orientation of the particles, θ. In equation (3.20), we saw that the expectation value of the angle should be zero. If the angles are chosen randomly, the probability of picking every value between $-\pi$ and π should be uniform, which gives an expected value of 0. This should always be the case if the particles in the system have no preferred direction. The particles align their direction when we add the torque, and will, therefore, have a preferred direction. This means that the expectation value is only valid for the system when $\gamma = 0$, meaning no alignment in the system.

Figure 5.1 shows the expectation value of the direction parameter, θ. We see that the expectation value is approximately zero, as the theoretical value. The expectation value deviates a bit from zero, but not much and it seems to be equally much deviation in both negative and positive direction. We expect the deviation to be smaller if the result had

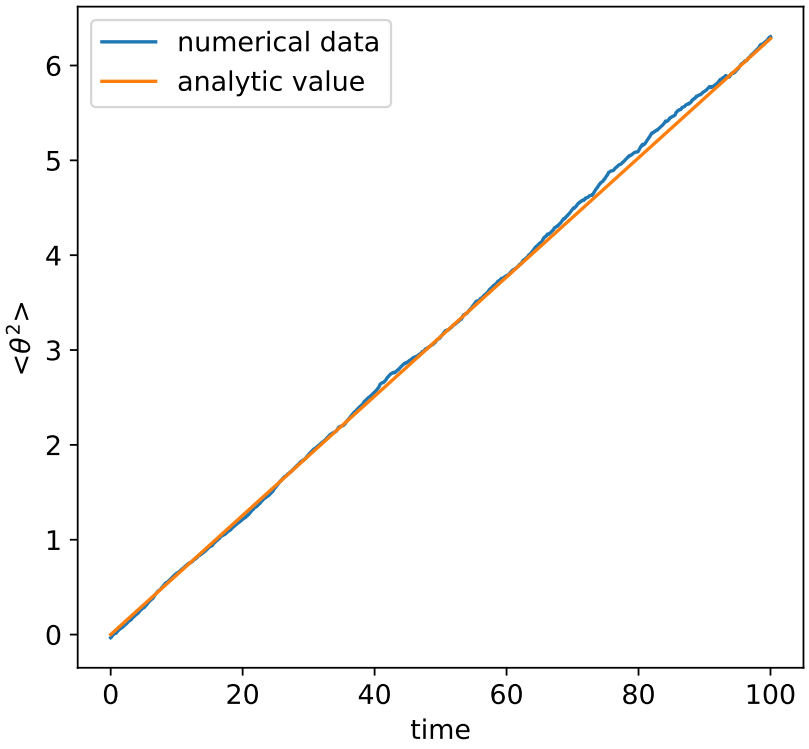


Figure 5.2: The figure shows both the theoretical and numerical value of the expectation value of the squared angle, $<\theta^2>$. We can see that the directional parameter, θ, has a diffusion behaviour. The parameters used is $N = 1024$, $dt = 0.001$, $D_r = 1$. The result has been averaged over ten simulations.

been averaged over more simulations (the result presented has been averaged over ten simulation), but due to running time and the clear tendency, we accept this results as proof that the angles are chosen randomly.

While the directional orientation is expected to average to zero, the squared angle, θ^2 is expected to be proportional to the time, t, where the constant of proportionality is connected to the rotational diffusion coefficient, D_r, as given by equation (3.21). This expectation value has been plotted in figure 5.2. We see that the numerical calculated data follows the theoretical value closely, and this gives a good confirmation that the directional orientation of the parameters is chosen right. In the figure the random noise is drawn from a uniform distribution in the interval $\eta \in [-\sqrt{\pi}, \sqrt{\pi}]$. The following equation gives the orange line

$$< \theta^2 >= 2D_r\pi t, \tag{5.1}$$

with $D_r = 0.01$.

The last expectation value we will use as a check is the correlation of the orientation. The theoretical value is given in equation (3.22). The persistent length, of the system, meaning the particles ability to move in straight lines for a given time, τ, is given by

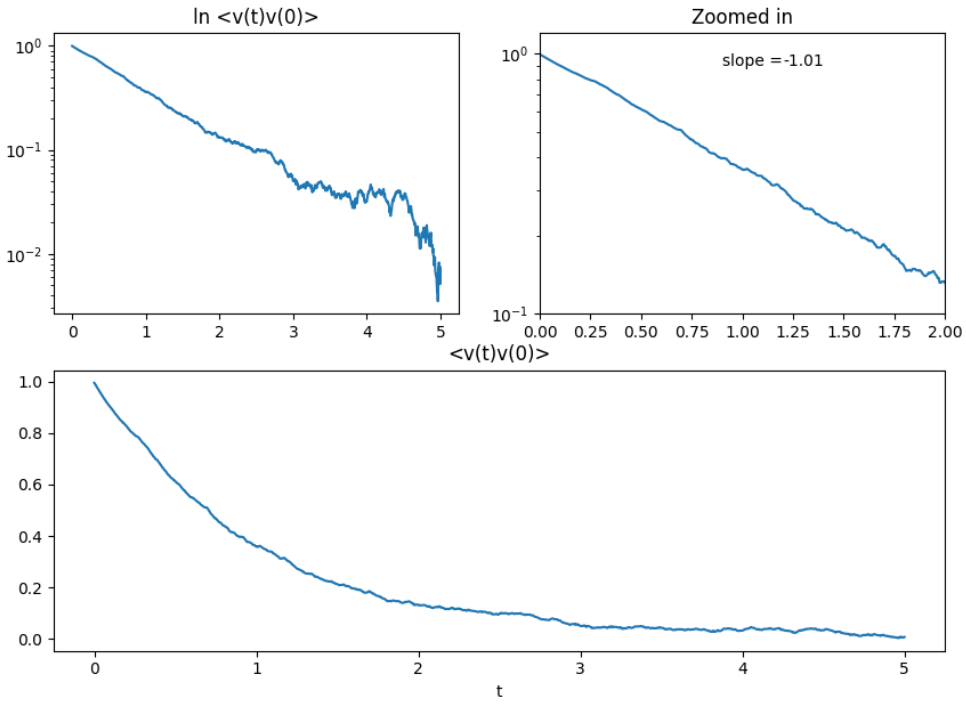


Figure 5.3: The figure shows the exponential decay of the autocorrelation function. The following parameters where used when producing the graph: number of particles = 1000, number of iterations=1000, v_0=1, dt=5e-3, D_r=1, $\eta = \sqrt{3}$

the fact that this correlation decays exponentially in time. This can be used to test our program. Figure 5.3 shows the correlation of the orientation as a function of time. The figure has three plots; the correlation of the orientation, the same on a log-scale and the log-scale zoomed in at the area where the logarithm of the dot product is linear. From equation (3.22) we find that the slope of the logarithmic graph should be the negative, inverse correlation time, $slope = -1/\tau$. From this we find the correlation time, $\tau = 0.99$. The correlation time has the following dependency to the rotational diffusion coefficient $D_r = 1/\tau$, which gives $D_r = 1.01$. The figure was simulated with a rotational diffusion coefficient $D_r = 1$, so this is as expected.

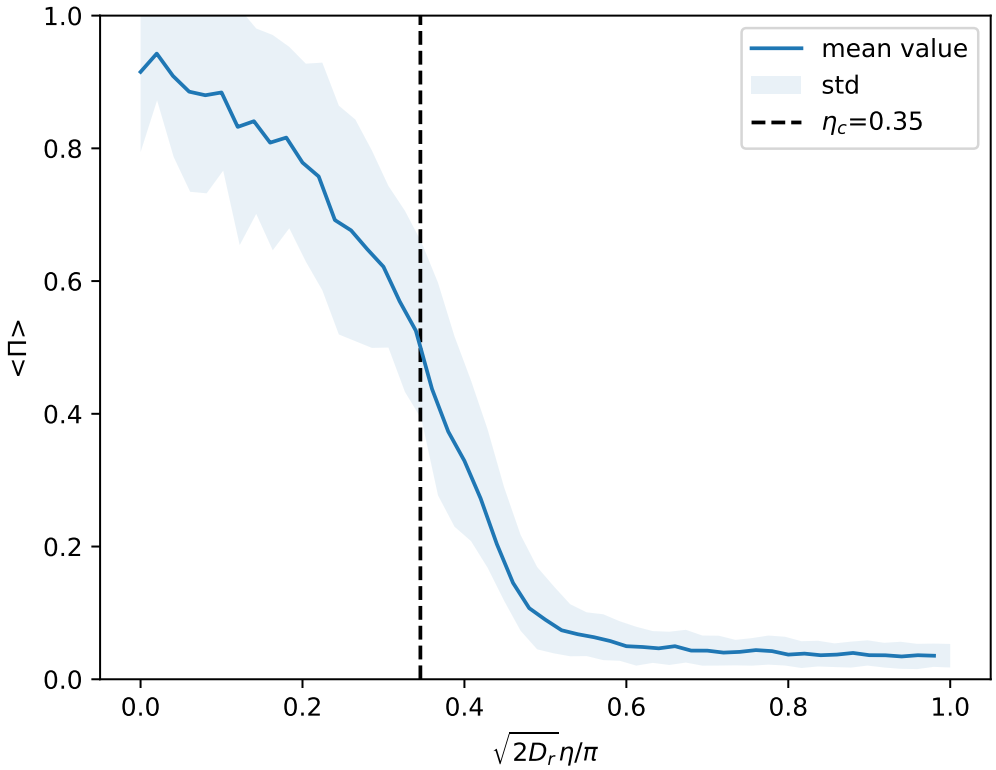


Figure 5.4: The figure shows the average order in the system as a function of the rotational diffusion. The steep curve in the figure indicated that there might be a phase transition in the system around $\sqrt{2D_r}\eta/\pi = 0.35$. The result is averaged over 100 simulations. Parameters used: $N = 900$, $t = 400$, $\Delta t = 0.01$, $L = 90$, $v = 1$, $\sigma = 1/2^{1/6}$, $\epsilon = 1$

5.1.3 Transition

We have now done several tests on our system that all indicate that the model follows the laws of physics. The last check we want to do is that our model also can display the transition found by Vicsek et al. in the Vicsek model. In work done by Vicsek, they found that there is a phase transition from disorder to order at $\eta = 0.3\pi$.

In figure 5.4, we see the average order in the system as a function of the rotational diffusion. The values are averaged over 100 simulations of the same system. Here, we have removed the factor of 2 inside the sine in the alignment torque in equation (3.15) to have the same alignment as in the Vicsek model. We see that the system goes through a transition from total order in the system to a system without order. This transition happens from $\sqrt{2D_r}\eta = 0.2$ to $\sqrt{2D_r}\eta = 0.5$. This steep fall in the order in the system can implicate that a phase transition occurs somewhere in this interval. In the figure we have marked the value $\eta_c = 0.35$ which is the value where $\Pi = 0.5$ Vicsek et al. found a phase transition at $\eta = 0.3\pi$ which is approximately the same as the critical value, η_c found in our model. The standard deviation is quite significant, which is expected as the

fluctuations can be substantial in active systems due to it not being an equilibrium system. Still, we accept this as a confirmation of that our model displays the same transition as the Vicsek model.

5.2 Lattice model

We start by looking at a system with particles placed in a square lattice, as explained in 3.6.4. The system consists of 900 particles, all with four nearest neighbours that are connected with through bonds. In the first simulation, we will let all the objects be connected to four other objects through lattice bonds. These connections are chosen by finding the four objects that are placed closest to each object in the lattice. In the initial state, two of the objects are infected. These two are randomly picked and can be placed anywhere in the system, also with regards to each other. After the simulations are done, the fraction of objects that are infected or immune is calculated. This tells us how big part of the system has been reached by the disease. This is done 101 times, for different values of the infection probability, with values evenly distributed from 0 to 1. The result of this simulation is shown in figure 5.5. Here we see how big part of the population has been infected by the diseases during the running time as a function of the probability than a random bond is active. The result has been produced by doing the same simulations 100 times and finding the average of the number of infected. The standard deviation is also plotted in the figure together with the threshold value, p_c.

From figure 5.5, we observe that for small values of the infection probability, p_{inf}, almost none of the objects in the system are infected. The fraction of infected does not start to increase until p is larger than 0.3, but here the curve increases rapidly. Half of the objects are infected when $p_{inf} = 0.53$, so we call this the threshold value in the system, $p_c = 0.53$. For probabilities larger than 0.7, every object in the system will be infected in most of the simulations. Even though the graph is produced using the mean value of 100 simulations, we can see that the curve is not smooth and that there are several large spikes. For the high probability values, we would expect the entire population to be infected after the running time, but even as $p_{inf} \approx 1$ there are spikes, which tells us that the disease is not able to spread to the entire system in some simulations. This is confirmed by the large standard deviation, which is also plotted in the figure. The standard deviation is large for almost all values of the probability, except for the area where $p_{inf} < 0.3$. One thing that affects the number of infected in the system is the placing of the two initial cases. If the two objects that are randomly chosen is close to each other, the infection is only able to spread in one area. Two close objects might have common neighbours, or their neighbours might have common neighbours. This means that there are less susceptible objects to infect for each of the initial cases. If they are spread in different areas of the system, on the other hand, the disease has to areas to spread in, and there will be more susceptible objects surrounding the infected ones. The standard deviation will also be affected by random fluctuations in the system, which will cause some outbreaks to die out by themselves even with high infection probabilities.

We have also plotted some snapshots of how the disease typically spreads in the system. The results is shown in figure 5.6. In this case, we have chosen to let only one of the objects be infected in the initial state, and we have chosen to place this object in the middle. The probability, p_{inf}, was set to 0.8. The figure shows that the infection spreads from the middle and moves towards the edges of the system. The spread happens in an almost

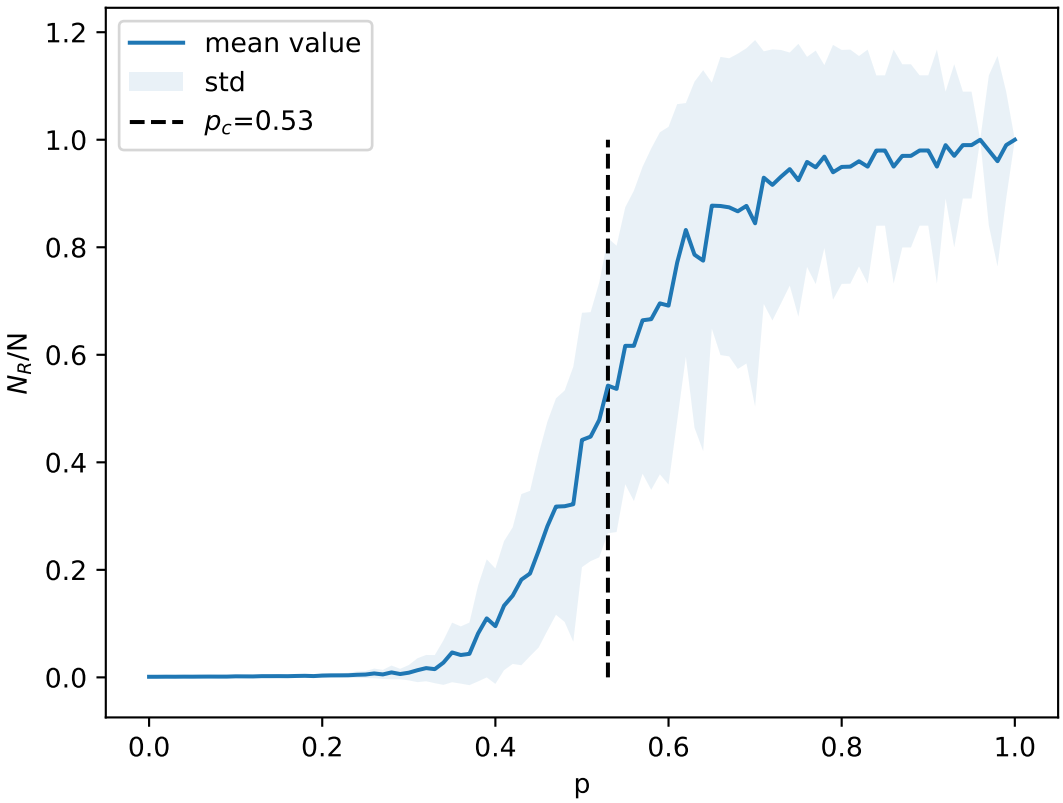


Figure 5.5: The figure shows how large proportion of the population has been infected during the running time as a function of the probability that a bond is active in a square lattice with nearest neighbours bonds. We see that the proportion of the population that is infected increases fast in a small interval of the probabilities. The result is the average over 100 simulations. The dotted line shows where the fraction of infected objects is 0.5, and thus, the threshold value p_c. Parameters used: $N = 900$, $t = 300$, $\Delta t = 0.01$, $L = 120$

a circular shape. The circumference of this "circle" consist of infected objects while it is filled with mostly immune and some susceptible objects. At the end of the running time, at $t = 500$, the disease has spread to the entire system, leaving some small clusters of susceptible objects spread around. It takes between 400 and 500 units of time before the last infected person has recovered.

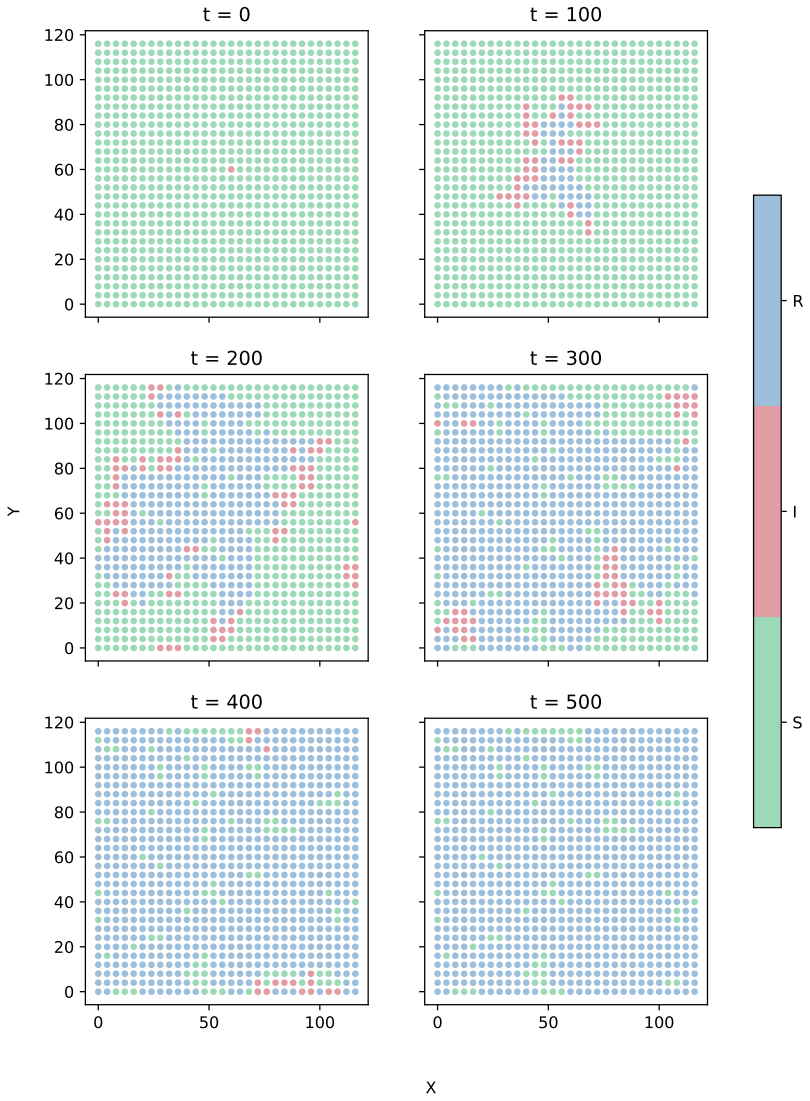


Figure 5.6: The figure shows snapshots of how the disease spreads in a square lattice with only one infected object placed in the middle in the initial state. We see that the disease spreads somewhat symmetric around the middle object and that the objects at the boundaries are infected last. Here S stands for susceptible, I for infected and R for recovered. Parameters used: $N = 900$, $t = 500$, $\Delta t = 0.01$, $L = 120$

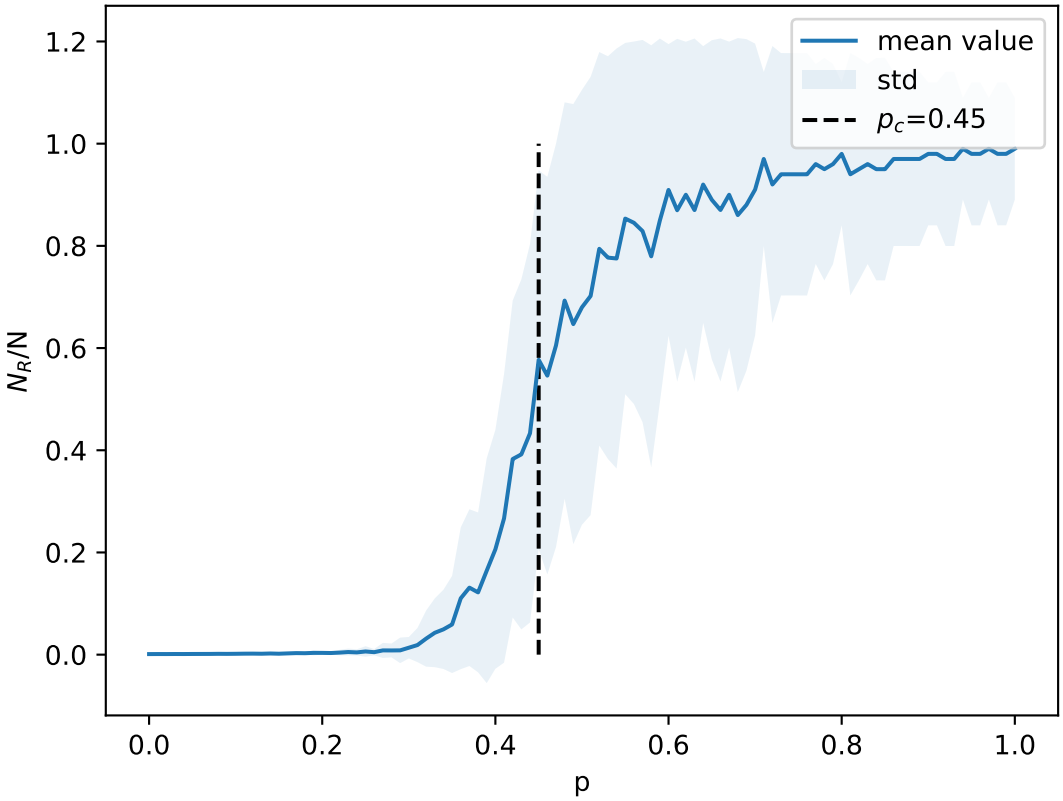


Figure 5.7: The figure shows how big proportion has been infected during the running time as a function of the probability that a bond is active in a small-world network. We see that small and large values of the probability gives little change in the number of people that are infected while the middle interval of probabilities give rise to a rapid change. The dotted vertical line is at the threshold value where over half the population is infected. The result is the average over 100 simulations. Parameters used: $N = 900$, $t = 300$, $\Delta t = 0.01$, $L = 120$, $\rho = 0.05$

By changing some of the bonds from being between nearest neighbours to connecting two random nodes, the square lattice becomes a small-world network. We would like to see whether this lowers the percolation threshold and if so, by how much. In figure 5.7, the proportion of the population that are infected are plotted against the infection probability for the small-world network. We have kept all the parameters from the last example in this simulation. The only change is that some of the bonds are now between objects that are not neighbours. With 900 objects, all connected to four other objects, there are a total of 1800 bonds in the system. In figure 5.7, 90 of these are changed to be between random nodes, giving $\rho = 0.05$. Once again, the result is the mean value of 100 simulations.

The figure shows that the small-world network also displays a phase transition from a system with very few infected to a state where almost every object is infected. The first probability value that leads to more than half the population caching the disease is $p_c = 0.45$. For small values of the probability, the disease dies out by itself before it can spread to the population. As the probability approaches one, the entire system is reached, and the objects are mostly infected. The standard deviation is slightly larger for the small-world network, which can be explained by the random altering of bonds. Which of the bonds are change and the reach of these new bonds are important factors to how the disease spreads in the system. Besides the shift in the threshold value and the slight increase in standard deviation, figure 5.5 and 5.7 are approximately identical, which was expected.

Again, we have also plotted some snapshot of the system to demonstrate how the disease typically spreads. These snapshots are presented in figure 5.8. The initial state has one infected object in the middle of the system, and the probability is $p_{inf} = 0.8$. The disease starts to spread from the middle, but we see that the long-range bonds spread the disease to new areas without infecting the objects between these areas. This leads to several local outbreaks of the disease happening at the same time. We see that in the small-world network, the disease is spread to the entire system much faster than in the regular network. The last infected case is recovered some time between $t = 300$ and $t = 400$. With approximately the same total number of infected, this must mean that more objects are infected at the same time in the small-world network. After the outbreak is over, there are some susceptible clusters in this system as well.

The current results show how the spread of the infection is affected by adding some long-range bonds. The most obvious change is that the threshold value is smaller for the small-world network than for the regular network, which was what we expected. This can be interpreted as that the regular network can handle a probability that is 0.08 larger than the small-world network in the critical area $0.3 < p < 0.7$, without getting more infected objects. This can mean that the spread of a disease can be severely slowed down without reducing the number of persons we interact with, but by merely restraining every one to keep close to their homes and avoid travelling. In addition to more objects being infected in the small-world network, we have seen that the infection travels faster, leading to more sick people at one time in this system. This has an adverse effect on the population because it can cause more people to need medical attention than the hospitals can manage. The last change we found was the increase in standard deviation in how many are infected, caused by another factor that can affect the infection, namely the random choosing of the long-range bonds. All in all, we see a clear trend; the society where everyone interacts with people from their neighbourhood only handles the outbreak of a pandemic better than the population where people can travel across the system.

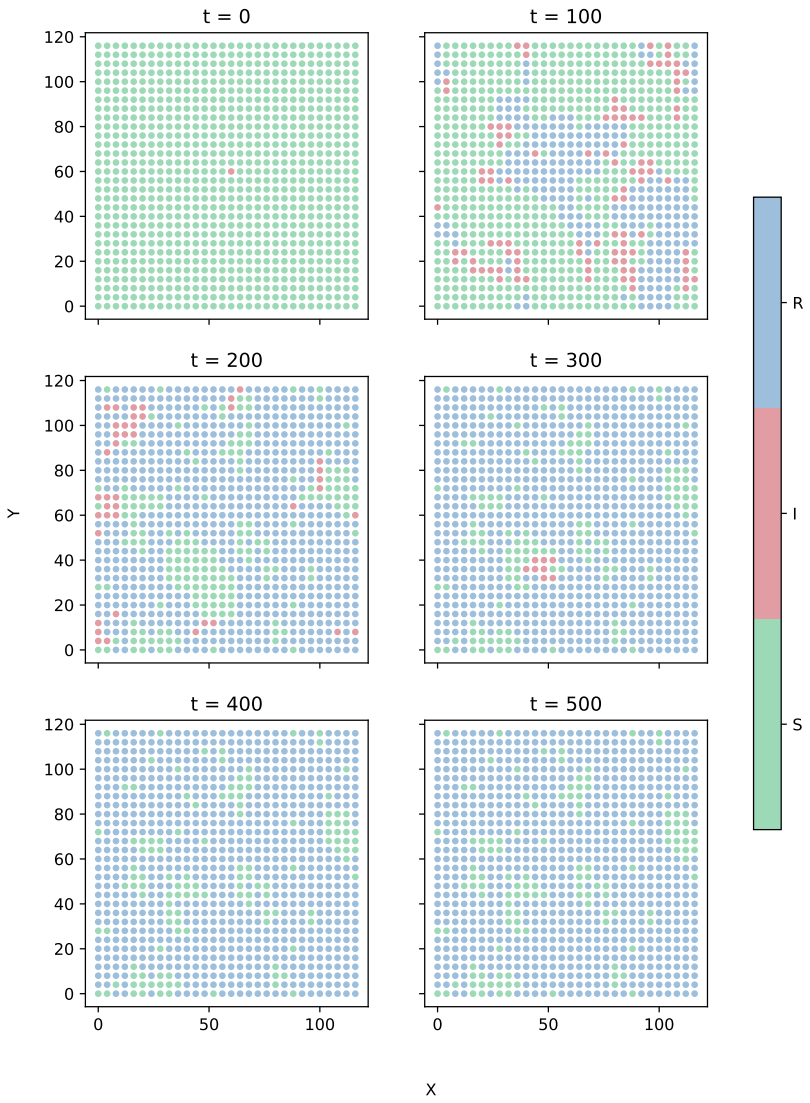


Figure 5.8: The figure shows snapshots of how the disease spreads in a small-world network with only one infected object placed in the middle in the initial state. We see that the disease spreads to different areas of the system in this case in contrast to in the previous system. This is caused by the long-range bonds in the small-world network. Here S stands for susceptible, I for infected and R for recovered. Parameters used: $N = 900$, $t = 500$, $\Delta t = 0.01$, $L = 120$, $\rho = 0.05$

5.3 Active agents

After having used the lattice model where all the objects are locked to one position throughout the running time, we wish to compare this to our active model where the objects can move around in the system. In order to compare the active model with both the square lattice model and the small-world network, we will look at two different cases. In the first case, we give all our objects a low self-propulsion velocity and let the rotational diffusion coefficient be high. A high rotational diffusion coefficient will give a short persistent length. Our system will then consist of active agents moving around in their neighbourhood, but the total average displacement of the agents will be low. We see this as the active equivalent to the regular square lattice model. To compare with the small-world network, we will give some proportion of the active agents a higher self-propulsion velocity and a lower rotational diffusion coefficient. This way, these active agents can move faster through the system, and they can interact with other agents that were not in their original neighbourhood. We want to check if these agents have the same effect on the system as the long-range bonds in the small-world network.

In the active system, there are no predefined rules of which objects can interact with each other. The objects will interact with every object that comes closer than the critical radius given for the different types of interactions. The density of particles in the system and the particle velocity will affect the spread of the disease. Since the particles are moving in this system, we have decreases the particle density by increasing the system size L. This is especially important for the "small-world" case as some of these particles are moving long distances and faster than the other particles. This is done to avoid collisions that shoot particles out of the system.

Figure 5.9 shows the transition from a low degree of infection to a high degree of infection in the active matter system. The system consists of 900 active agents, all with the same self-propulsion velocity, $v = 0.3$ and a rotational diffusion coefficient of $D_r = 0.5$. The combination of the low particle velocity and high rotational diffusion coefficient makes the agents move around in a restricted area around the initial position. The agents will therefore mostly interact with the agents they are initially placed next to, making this system quite similar to the square lattice system. The particles repel each other with a repulsive Lennard-Jones potential. A challenge with the active model is that much more calculations must be done, as the particles are moving and exerting forces on each other. These result presented in this section is therefore only averaged over 60 simulations, while the once in the lattice systems where run 100 times. This makes these results less accurate.

Figure 5.9 shows the fraction of the population is infected as a function of the infection probability in the active system. Once again, we see that small probability values lead to a disappearing fraction of infected objects. In this system, the increase in the fraction that is infected seems to start sooner than in the lattice model, at $p = 0.2$. This means that the transition happens slightly faster in the lattice model than in the active model. The threshold value seems to be at $p_c = 0.55$, which is approximately the same threshold value as the system with objects that are not moving. We also observe that the standard deviation is smaller in this system. This can be because the particles are moving such

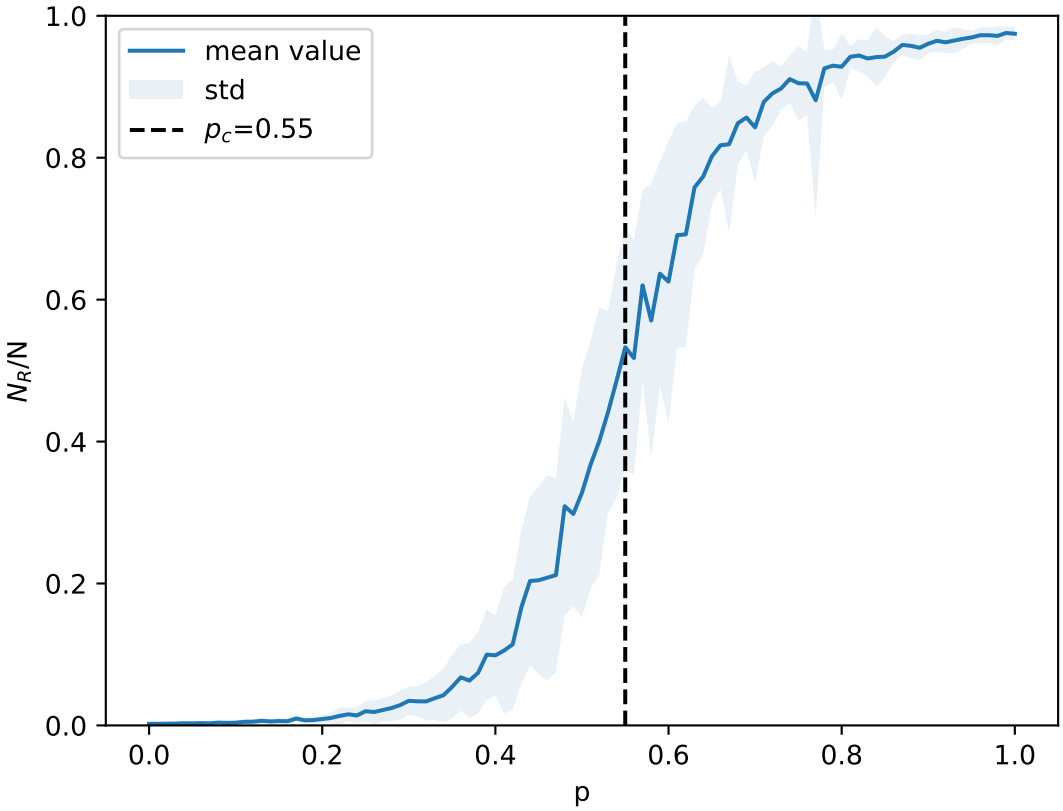


Figure 5.9: The figure shows how big proportion has been infected during the running time as a function of the probability that of infection. As for the previous cases, we see that there are an area in the middle where the growth of the number of infected are much larger than for the rest of the figure. The dotted vertical line is at the threshold value where over half the population is infected. The result is the average over 20 simulations. Parameters used: $N = 900$, $t = 500$, $\Delta t = 0.01$, $L = 200$, $D_r = 0.5$, $v = 0.3$, $R_{inf} = 10$, $\sigma = 1/2^{1/6}$, $\epsilon = 1$

that they interact with more other particles and are not bound by the lattice bonds. This can make the initial choice of which agents are infected less critical and thus reduce the standard deviation in the system.

In figure 5.10 four snapshots of a system with 900 particles are shown. The probability of infection is set to 0.8 and the particle density is $\phi = 0.013$. The particles are not plotted to scale as this would make them so small that they would "disappear". Also here there is only one infected object in the start, and this object is placed in the middle. We see that the spread of the disease in this system is similar to the square lattice system. The disease starts out in the middle and spreads out in a circle around the middle object. The biggest difference is that the disease dies out before having reached the edges and thereby leaving a larger part of the population susceptible. This also fits good with the fact that the threshold value is slightly larger for the active system than the lattice system.

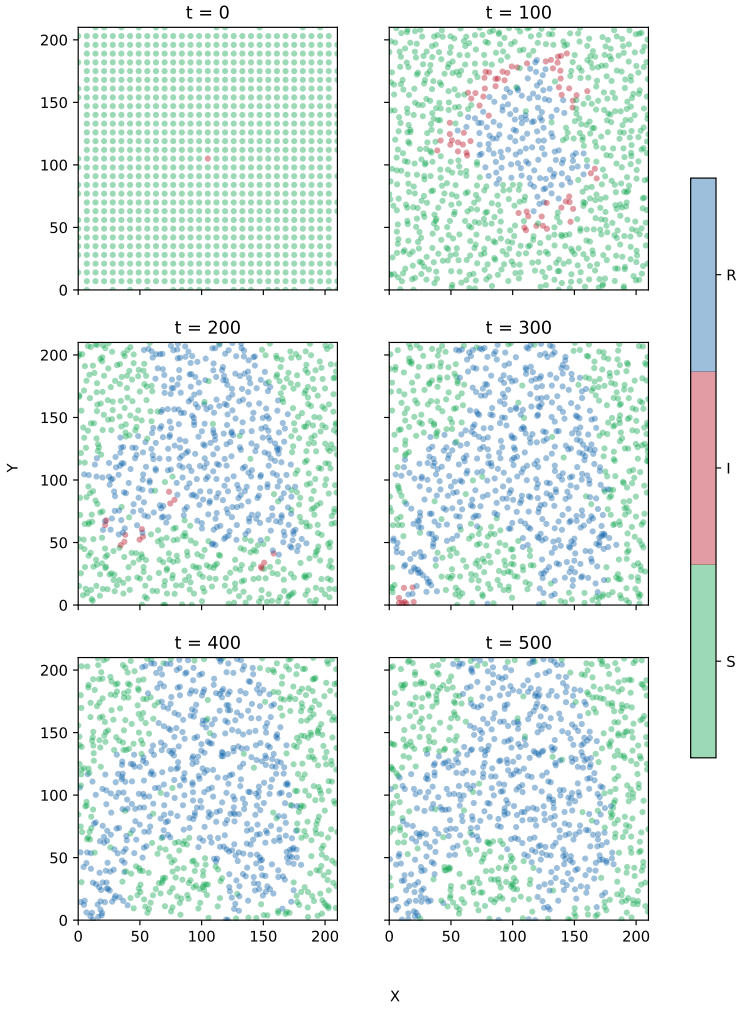


Figure 5.10: The figure shows snapshots of how the disease spreads in an active system with only one infected object placed in the middle in the initial state. The rotational diffusion coefficient are high to make the objects move around in an area surrounding the initial position. The figure reminds us of the way the disease spread in the regular lattice case, but we see that the objects at the edges are never infected. The inter-particle potential consists only of the repulsive part of the Lennard-Jones potential. Here S stands for susceptible, I for infected and R for recovered. Parameters used: $N = 900$, $t = 500$, $\Delta t = 0.01$, $L = 200$, $D_r = 0.5$, $v_0 = 0.3$, $R_{inf} = 8$, $\sigma = 1/2^{1/6}$, $\epsilon = 1$

The same simulation has been done once again, but this time a small proportion of the active agents has been given a larger self-propulsion velocity and a smaller rotational diffusion coefficient. This makes these particles able to move through the system while the other particles are still moving around in their initial neighbourhood. In figure 5.11

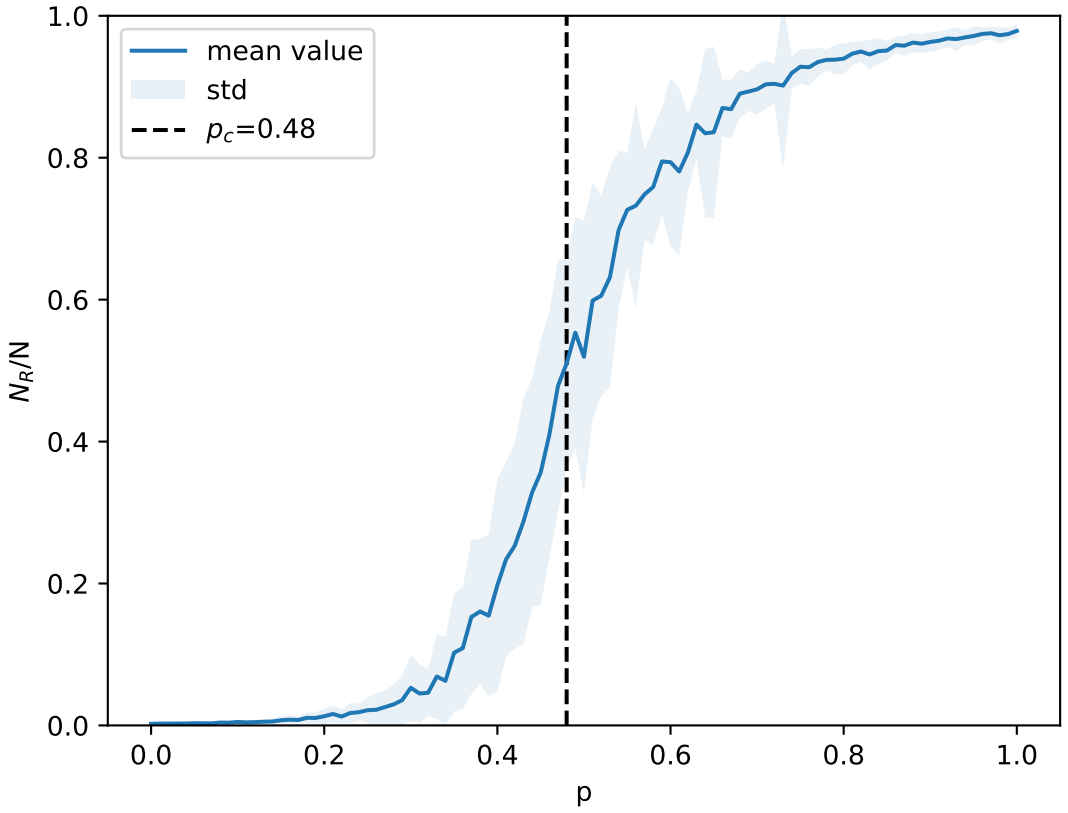


Figure 5.11: The figure shows how big proportion has been infected during the running time as a function of the probability that of infection. In this system, 20 of the 900 agents were able to move in the entire area, while the rest were restricted to their neighbourhood. The figure shows similar behaviour as the previous examples. The dotted vertical line is at the threshold value where over half the population is infected. The result is the average over 20 simulations. Parameters used: $N = 900$, $t = 500$, $\Delta t = 0.01$, $L = 200$, $D_r = 0.5$, $v = 0.3$, $R_{inf} = 10$, $\sigma = 1/2^{1/6}$, $\epsilon = 1$

20 of the 900 particles are able to move freely in the system while the remaining 880 particles are restricted to their neighbourhood. All other parameters are kept the same.

The threshold value for this active system is found to be $p_c = 0.48$, which is smaller than the value for the system where none of the particles was able to move outside their neighbourhood. Other than this shift of threshold value, the graph is mostly the same as the others. The standard deviation is of the same order as in the previous system with all the particles kept in the same area. The difference between the two threshold values is approximately the same as the difference between the square lattice and the small-world network. This indicates that we are able to provoke the same behaviour in our active model by letting some of the particles move freely as we see in the lattice system when we introduce long-range bonds.

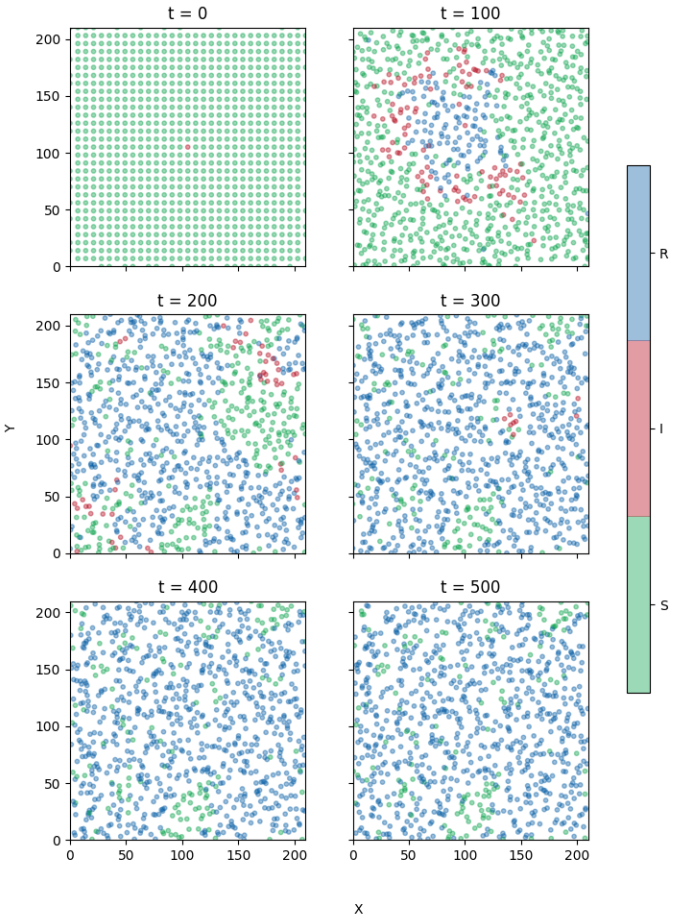


Figure 5.12: The figure shows snapshots of how the disease spreads in an active system with only one infected object placed in the middle in the initial state. We see that the disease again spreads in a more similar way as it did for the small-world network. The rotational diffusion coefficient are high for most of the particles, but lower for 20 of the 900 particles. This is to let a small portion of the particles move away from their initial position to make the same effect as the small-world network. The same particles also have a larger self-propulsion velocity. The inter-particle potential consists only of the repulsive part of the Lennard-Jones potential. Here S stands for susceptible, I for infected and R for recovered. Parameters used: $N = 900$, $t = 500$, $\Delta t = 0.01$, $L = 200$, $R_{inf} = 8$, $\sigma = 1/2^{1/6}$, $\epsilon = 1$. For the particle restricted to an area: $D_r = 0.5$, $v_0 = 0.3$, for the particles allowed to move in the entire system: $D_r = 0.05$, $v_0 = 5$

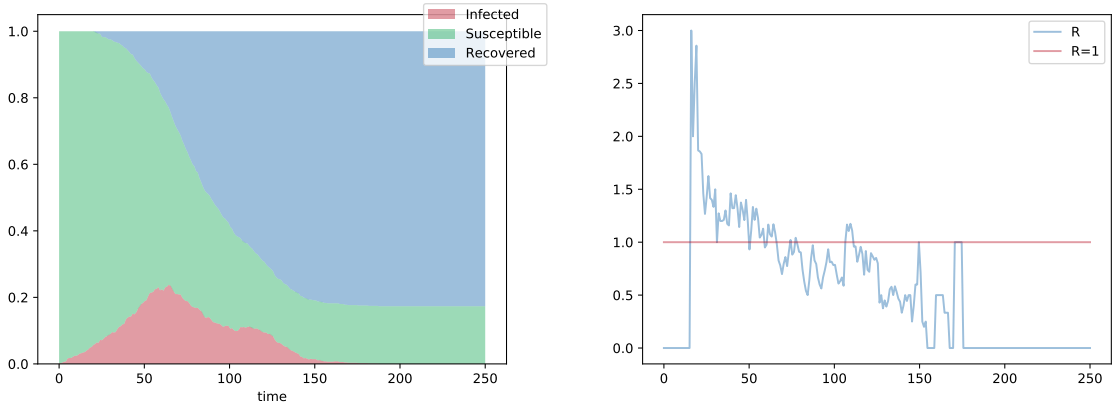
We want to see if this is also true for the way the disease spreads in the population. Snapshots of the same system during one of the simulations are shown in figure 5.12.

We see that this change has some of the same effects as the small-world network had in the lattice case. In the start, the disease spreads in a circle around the initial infected object in the middle, as in the previous case. After some time, we see that there are small

clusters with infected agents at different, more random, places in the system. This is due to the agents that are able to move from one neighbourhood to another. As a result of this, we see that the disease is spread to the entire system in contrast to the system in figure 5.10. As for the lattice cases presented in figure 5.6 and 5.8, there are some small clusters that are left susceptible also in this case. We see that the disease spreads fast in the system and that the last infected person recovers sometime between $t = 300$ and $t = 400$ as for the small-world network.

The results from both the active and the lattice model show that there are transitions in the systems. These transitions take the system from a state where the disease naturally dies out by itself without many cases of infection to a state where almost the entire population is infected. In the systems where the objects only interact with their neighbours, this transitions happens at a probability that is slightly larger than 0.5, $p_c = 0.53$ and $p_c = 0.55$ for the lattice model and the active model respectively. When some of these objects are allowed to interact with objects that are further away, the transition happens at a lower probability. For the small-world network we found the threshold value at $p_c = 0.45$ while the active model displayed a transition at $p_c = 0.46$. We also see a similarity in the change in the way the disease spreads in the system when the model is changed to include long-range interactions for both the lattice model and the active model.

These results can be interpreted as that during a pandemic, the spread of the disease can be drastically reduced without reducing the number of people we interact with, but with merely restricting these interactions to be in a given area surrounding us. An example of this would be to divide every city into areas where everyone are only allowed to move and interact with persons inside their own area. The results also show that the active model is a good alternative to the lattice model, as the same results can be produced. With the active model, we can do more changes in the model than we could have done in the lattice model, and more scenarios can be studied. Examples for such changes is to alter the infection radius, the cluster sizes etc. This is the topic of the next section.



(a) Proportion of the population in each category **(b)** Effective reproductive number, R

Figure 5.13: The figure shows the development in a system with N=900 objects. Two randomly chosen objects are infected in the initial state and the objects are able to move freely in the entire system. This figure will be used as an initial case such that we can compare it to other systems with different measures and restrictions. Parameters used:$N = 900$, $t = 250$, $\Delta t = 0.01$, $L = 200$, $R_{inf} = 12$, $\sigma = 1/2^{1/6}$, $\epsilon = 1$, $D_r = 0.01$, $v_0 = 1$, $p = 0.4$

5.4 Introducing restrictions in the system

We wish to use the model to simulate what happens when different restrictions are introduced in a society, similar to the models discussed earlier. The first simulation done is in a system with 900 objects moving around freely. The objects have a self-propulsion velocity of 1, and the rotational diffusion is low such that the particles can move anywhere in the container. The result of this simulation is shown in figure 5.13. Figure 5.13a shows how large proportion of the population that is infected, susceptible and recovered as a function of time. Figure 5.13b shows the effective reproduction number in the system as a function of time. Due to the way we calculate this number, there will be some lag in the value. The lag will be equal in all the simulations, and the results can still be compared to each other.

Figure 5.13 shows that most of the population is infected at some point of the running time, but that around 17 % is left susceptible in the end. The effective reproduction number peaks at 3, meaning that the average number of new cases as a direct consequence of one case is three at a maximum. This leads to a total of 23% of the population infected at the same time. The last infected object recovers at $t = 150$ according to figure 5.13a. Snapshot of the system is shown in figure 5.14. This figure shows how the disease spreads in the system. For this case, we see that the disease spreads fast and that a large portion of the population is infected or has recovered from being infected already after 100 units of time. We call this the initial case, and we imagine that this would be the result if no measures are taken to slow down the pandemic. Following there will be done several changes to the system, and the result of these changes will be compared with this initial case to see the effect.

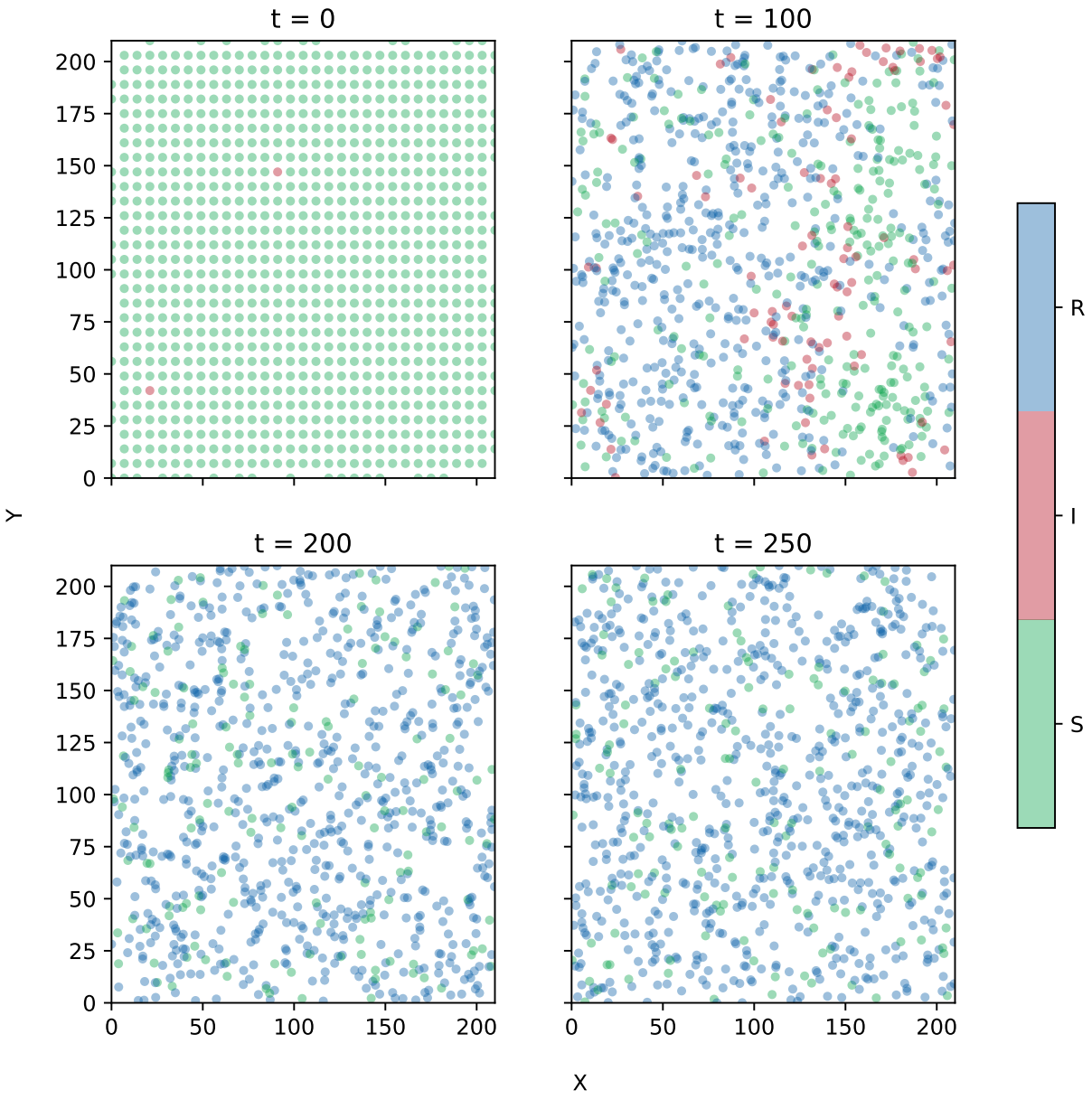
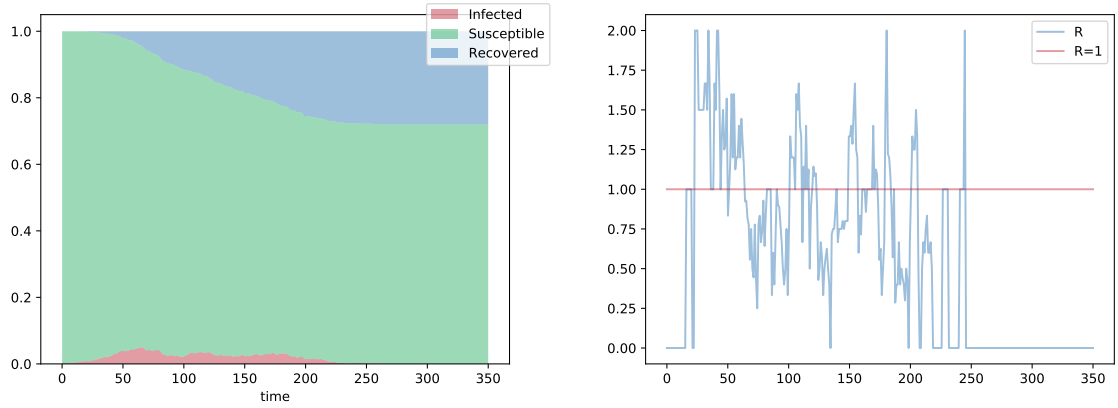


Figure 5.14: The four snapshots shows the condition of the objects in the system at four different times. This figure will be used as an initial case such that we can compare it to other systems with different measures and restrictions. Here S stands for susceptible, I for infected and R for recovered. Parameters used: $N = 900$, $t = 500$, $\Delta t = 0.01$, $L = 200$, $D_r = 0.01$, $v_0 = 1$, $R_{inf} = 12$, $\sigma = 1/2^{1/6}$, $\epsilon = 1$, $p = 0.4$

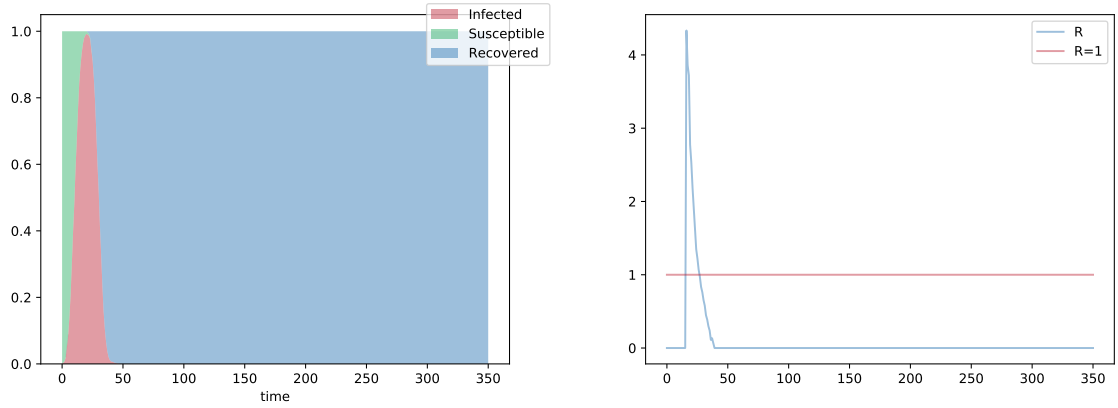


(a) Proportion of the population in each category **(b)** Effective reproduction number, R

Figure 5.15: The figure shows the development in a system with N=900 objects for a smaller infection probability than in figure 5.13. Two randomly chosen objects are infected in the initial state and the objects are able to move freely in the entire system. We see that the number of infected objects are much smaller than for the initial case. Parameters used:$N = 900$, $t = 350$, $\Delta t = 0.01$, $L = 200$, $R_{inf} = 12$, $\sigma = 1/2^{1/6}$, $\epsilon = 1$, $D_r = 0.01$, $v_0 = 1$, $p = 0.2$

5.4.1 Decreasing the infection probability

As a logical test, we decrease the infection probability to see what happens. Reducing the infection probability can be seen as introducing better rules for hygiene. The result of this change is shown in figure 5.15. Here the infection probability was decreased to half of the original, such that $p_{inf} = 0.2$. As expected, the total proportion of susceptible objects at the end are much larger in this system, meaning that the number of infected is much smaller. While a total of 83 % of the population is infected in the initial system, this proportion is only 28 % in the system with reduced probability for spreading the infection. Reducing the probability also decreases the peak of the reproduction number, which for this case is at 2. Also, the peak of the number of infected at the same time is much smaller for this case, at only 5 % of the population. Compared to the previous peak at 23 % this is a considerable improvement. Reducing the probability leads to a longer duration of the disease. We see that the last infected person does not recover until $t = 220$ in this case. The effective reproductive number is also larger than one for a longer time with the reduced probability, meaning that there is an increase in the number of infected for a longer time. While the effective reproduction number is less than 1 for t $\geqslant$ 120 for the initial case, the reproduction number does not stabilize below one until t = 250 for the reduced probability case. Decreasing the probability of infection is seen to be a very effective way to slow down the spread of the disease. Snapshots of the system can be found in the appendix in figure A.1.

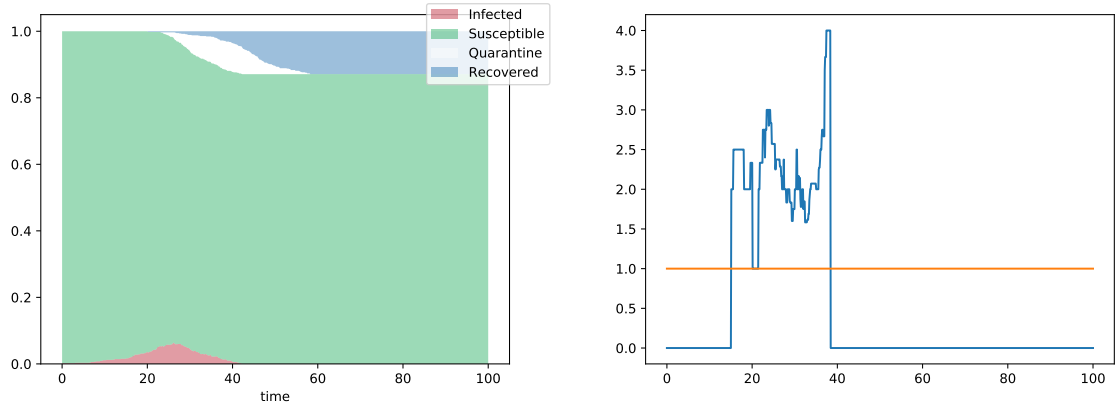


(a) Proportion of the population in each category **(b)** Effective reproductive number, R

Figure 5.16: The figure shows the development in a system with N=900 objects for a larger infection radius than in figure 5.13. Two randomly chosen objects are infected in the initial state and the objects are able to move freely in the entire system. We see that the number of infected objects are much higher than in the initial case, and that these objects are infected right after the first object is infected. Parameters used:$N = 900$, $t = 250$, $\Delta t = 0.01$, $L = 200$, $R_{inf} = 24$, $\sigma = 1/2^{1/6}$, $\epsilon = 1$, $D_r = 0.01$, $v_0 = 1$, $p = 0.4$

5.4.2 Increased infection radius

Similar to decreasing the infection probability, changing the infection radius is also expected to affect the outcome. Increasing the infection radius can be seen as people being more interactive with each other, for example, by hugging and kissing. It can also be interpreted as that there are more total interactions between people, meaning that everyone is interacting with more people. Figure 5.16 is a simulation done on the same system as the initial one, but with a larger infection radius. The infection radius has been increased from 12 to 24. By doubling the infection radius, we see that the outcome of the disease is drastically changed. During only 40 time units, the infection has spread to the entire system, leading to a huge amount of people infected at the same time. For the large radius case, all of the objects are infected at the same time, which could cause a serious overload on the medical services of a country. The effective reproductive number is larger for this case, but perhaps not so much that one would expect. This can be explained by the fact that so many are infected at once, so there is no one left to infect for most of the objects. Snapshot of the system are placed in the appendix in figure A.2 and shows that all the objects in the system are infected and has recovered after 100 units of time. From figure 5.16b, we see that the effective reproduction number drops to 0 already for t=50, meaning that the entire system is infected at this point of time.



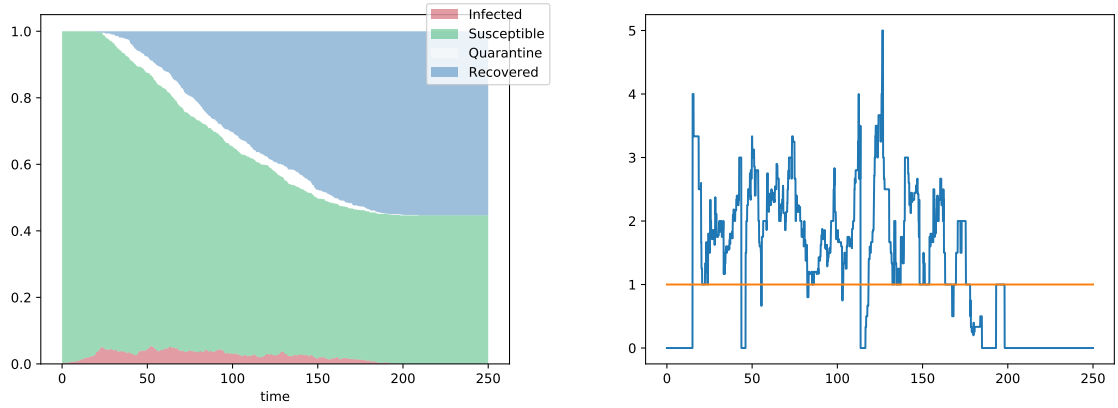
(a) Proportion of the population in each category **(b)** Effective reproductive number, R

Figure 5.17: The figure shows the development in a system with N=900 objects when all of the infected objects are isolated after the infection. Two randomly chosen objects are infected in the initial state and the objects are able to move freely in the entire system. We see that this kills the disease almost immediately and that there are few infected objects. Parameters used:$N = 900$, $t = 250$, $\Delta t = 0.01$, $L = 200$, $R_{inf} = 12$, $\sigma = 1/2^{1/6}$, $\epsilon = 1$, $D_r = 0.01$, $v_0 = 1$, $p = 0.4$

5.4.3 Quarantine

We have now seen the effect of two of the measures discussed in section 3.6.2, namely changing the infection probability and the infection radius. We proceed by introducing quarantine rules in our system. This is done by letting there be a probability, $p_{quarantine}$ that an infected object is discovered and therefore isolated in quarantine. We let this happen at four units of time after the object is infected, $t = t_{inf} + 4$. An object will spend the first 20 % of the time it is infected in the system as usual before it is removed with the probability $p_{quarantine}$. First, we let this probability be 1, meaning everyone who is infected is removed from the system. We have added a rule such that none of the objects is isolated until the number of infected reaches a critical value. This is to better simulate real societies, where such restrictions would not be introduced before the government is aware of the situation. In this case, we have said that 50 objects must be infected before any measures are taken. The result of this is shown in figure 5.17.

We see that it is very effective to diagnose and isolate everyone that is infected. We see that only about 10 % of the population is infected, leaving 90 % susceptible at the end of the running time. These 10 % are infected at almost the same time, also giving an infection peak of 10 % of the population. The effective reproduction number, shown in figure 5.17b, has a maximum at four which is quite high. This can be due to the fact that the quarantine rules does not kick in before enough objects are infected. We see that the disease dies out very fast and that there are no infected objects after the time $t = 40$.



(a) Proportion of the population in each category

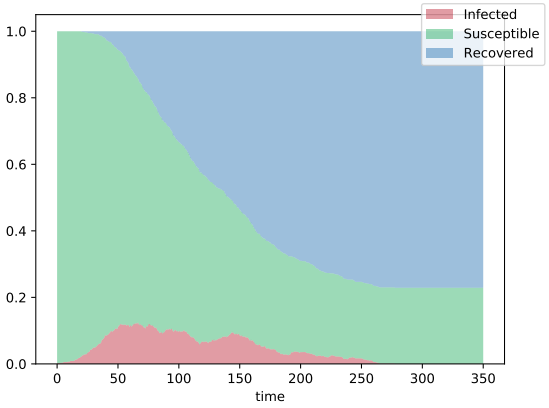
(b) Effective reproductive number, R

Figure 5.18: The figure shows the development in a system with N=900 objects when 70 % of the infected objects are isolated after the infection. Two randomly chosen objects are infected in the initial state and the objects are able to move freely in the entire system. More objects are infected here than when all infected objects are isolated, but the number of infected are still much lower than for the initial case. Parameters used:$N = 900$, $t = 250$, $\Delta t = 0.01$, $L = 200$, $R_{inf} = 12$, $\sigma = 1/2^{1/6}$, $\epsilon = 1$, $D_r = 0.01$, $v_0 = 1$, $p = 0.4$, $p_{quarantine} = 0.7$

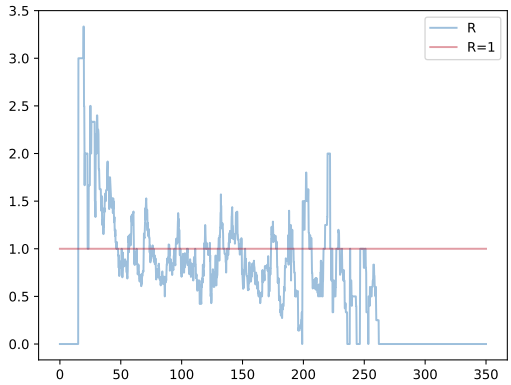
It was expected that discovering and isolating every infected object would be very effective, but this is unfortunately difficult to do in real life. We want to see what happens if instead 70 % of the infected objects are removed while the remaining 30 % are not discovered and therefore left in the system. This is a more realistic goal, as some of the infected persons would most likely not develop symptoms. This is simulated, and the results are shown in figure 5.18.

There are several large differences when only 70 % is discovered and isolated. The first thing we notice is that the disease is spread over a much larger time span. While the disease was over after $t = 40$ for the case where everyone was isolated, we have new cases of infection until $t = 200$ in this case. Even though the peak is a bit smaller here, we see that the total number of infected are much larger. At the end of the running time, 50 % of the population is infected, in contrast to the 10 % in the previous case. We also see that the maximum value for the effective reproduction number is larger here. The quarantine system is most effective when everyone is discovered, but this is not very surprising.

What might be more interesting is to compare the $p_{quarantine} = 0.7$ case with the initial case in figure 5.13. We remember that this first example had a total of 83 % infected after running time and that 23 % of the population was infected at the same time. From this number, we see that quarantines are very effective, even if 30 % of the infected objects are never detected. The effectiveness does, of course, increase if more of the infected ratio is detected. The most important consequence with the quarantine might be that number of infected are spread over a larger time, which will prevent the health system from being overloaded.



(a) Proportion of the population in each category



(b) Effective reproductive number, R

Figure 5.19: The figure shows the development in a system with N=900 objects. Here we have used a potential creating competing interactions induce clustering of a limited size in the system. Two randomly chosen objects are infected in the initial state and the objects are able to move freely in the entire system. We see that the number of infected is a bit better than for the initial case, but the main improvement is that the infection cases are spread over a longer time period. Parameters used:$N = 900$, $t = 250$, $\Delta t = 0.01$, $L = 200$, $R_{inf} = 12$, $\sigma = 1/2^{1/6}$, $\epsilon = 1$, $D_r = 0.01$, $v_0 = 1$, $p = 0.4$, $R_r = 2$, $R_a = 1$, $C_r = 4$, $C_a = 1$

5.4.4 Limited group size

The fourth and last item on the list of restrictions is to limit the number of people that are allowed to meet in one group. This can be modelled by using the potential that creates competing interactions given in equation (3.13). With this potential, the strengths and the ranges of the different terms can be adjusted to provoke the dynamic we want in the system. In the simulations that are done, these potential parameters are chosen such that most of the clusters consist of seven objects or less. All of the other parameters are kept the same as in the initial case. The objects can move between different clusters and the clusters will not consist of the same object at all times.

The proportion of the population that is infected in this simulation is shown in figure 5.19a. Compared to the initial case, the fraction that is not infected is a little bigger as 23 % of the population are never infected compared to the 17 % in the original case. The 77 % that are infected are spread over a longer time period, such that the maximum fraction that is infected at one time is approximately half of the initial one. The last infected object recovers after about 260 units of time. The effective reproduction number, shown in figure 5.19b, is stable around one for almost the entire time before the disease dies out. This means that the disease spreads at a controlled speed.

In figure 5.20, four snapshots of the particles in the system are shown. These snapshots show the dynamic and the size of the clusters in the system. Since the particles are small, many of the particles in the same clusters appear to be in the same position. It can, therefore, be difficult to see how many particles are gathered in the same cluster, but the

strength of the colours give a good indication (stronger colours means more particles). To be able to get a better understanding of the dynamics, the snapshot in the lower right corner has been magnified and is shown in figure 5.21. In this figure, we can see that most of the clusters consist of less than eight particles, but that there are also some larger clusters. We do not make any attempt to fix this, as we expect that everyone would not follow the rules in a real society either. The two figures also illustrate why the disease spreads slower in this system than in the initial one. While systems with a Lennard-Jones potential will have particles almost evenly distributed in the entire system, this system has clusters with particles that are spread further away from each other. In the initial system, some objects can be infected all the time, as particles meet continuously. In this system, on the other hand, it can be longer time periods where no objects from different clusters meet due to the larger distance between the clusters. It, therefore, takes longer before the disease can spread to the entire system.

Even though restricting the number of people that are allowed to meet did not reduce the total number of infected by very much, it was the change that had the largest effect on the time it took before the disease could reach the entire system. As one of the main goals with introducing such a measure could be to slow down the spread, we would say that this measure is very effective. How effective it is, would, of course, depend on the group sizes, and we could have tried several such cases to look at the difference. As the main goal here was to test our model was able to reveal such effects rather than the effect itself, we have only included this one case.

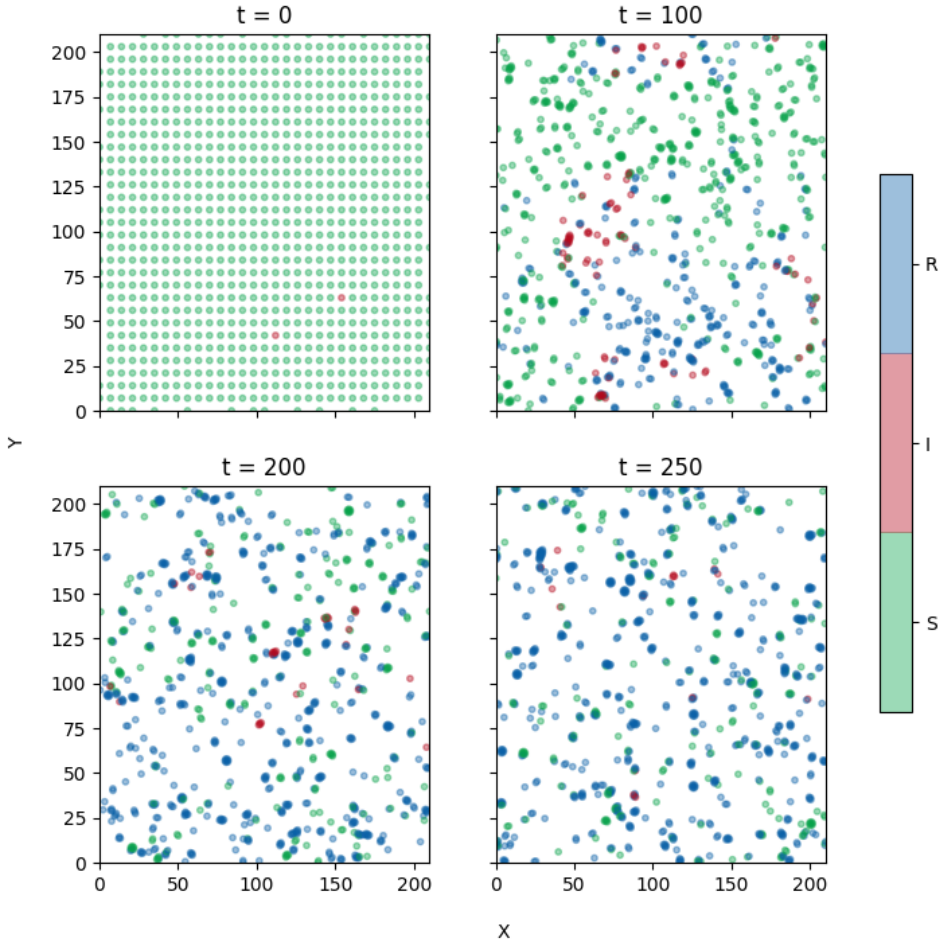


Figure 5.20: Snapshots of the system simulated in figure 5.19. We have used a potential inducing clusters of a limited size to model a society with limitations on how many are allowed to meet. Particles in cluster are overlapping in the figure and can therefore be spotted by the dark color. The infection is spread to the clusters more slowly than when the objects are spread in the system, but once an object in a cluster is infected, most of the objects in that cluster are also infected. Parameters used:$N = 900$, $t = 250$, $\Delta t = 0.01$, $L = 200$, $R_{inf} = 12$, $\sigma = 1/2^{1/6}$, $\epsilon = 1$, $D_r = 0.01$, $v_0 = 1$, $p = 0.4$ $R_r = 2$, $R_a = 1$, $C_r = 4$, $C_a = 1$

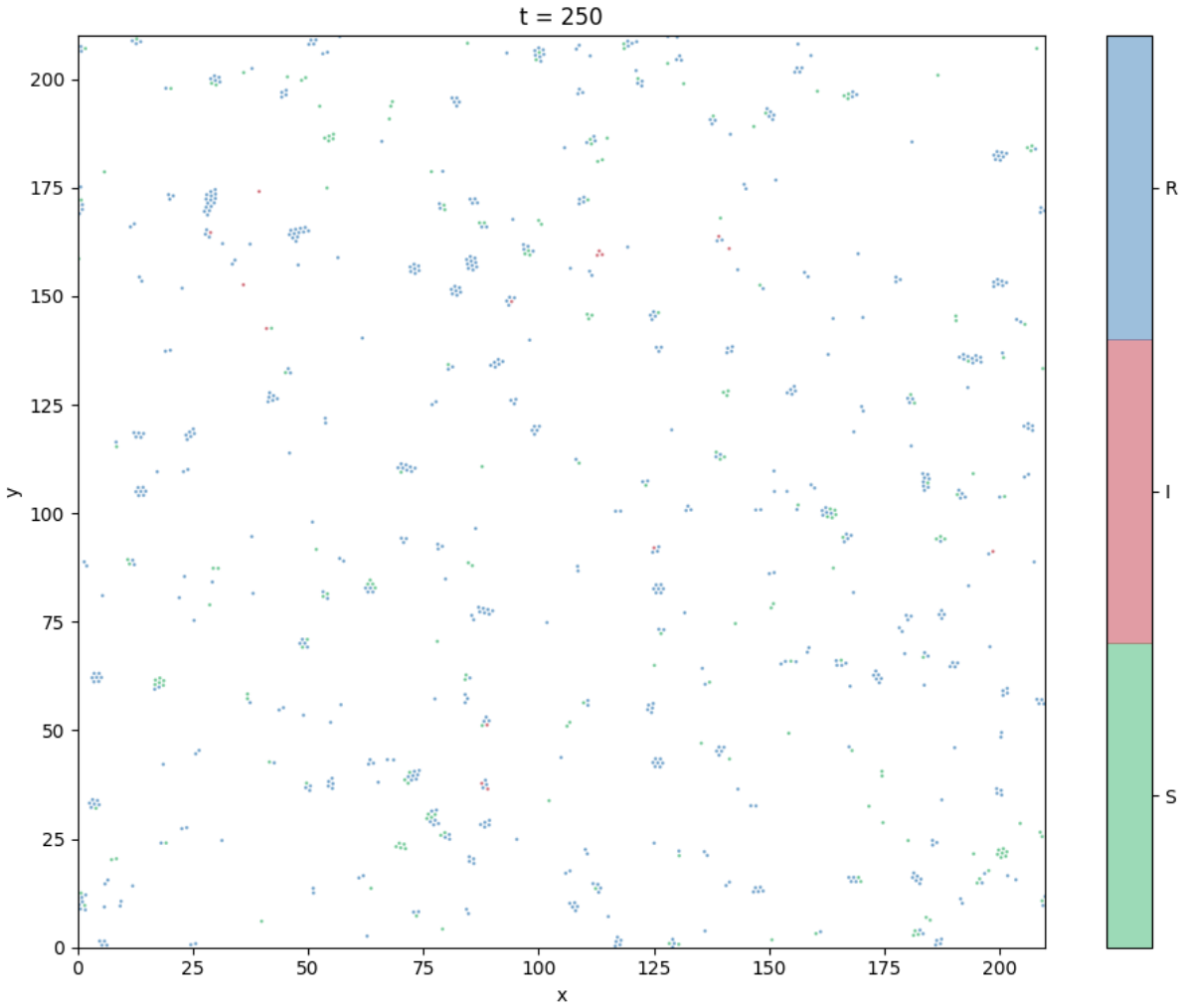


Figure 5.21: The figure shows the lower right snapshot from figure 5.20 but in a larger size such that the objects can be seen better. This is to be able to see how many particles the clusters consist of and how many of the particles in each clusters are typically infected. Parameters used:$N = 900$, $t = 250$, $\Delta t = 0.01$, $L = 200$, $R_{inf} = 12$, $\sigma = 1/2^{1/6}$, $\epsilon = 1$, $D_r = 0.01$, $v_0 = 1$, $p = 0.4$ $R_r = 2$, $R_a = 1$, $C_r = 4$, $C_a = 1$

Chapter 6

Conclusion

We have developed an active model that can be used to simulate an epidemic spreading in a population. This model has been used to see how large part of the population is infected for different probabilities of spreading the infection from one person to another. We found that the number of infected persons increases as the infection probability increases, as expected. A more interesting finding might be that the number of infected persons grows much faster for some values of infection probability than others. The number of infected grows very slowly for small and large values of infection probability, while it grows rapidly for the middle spectre of the infection probabilities. The specter where the curve was steepest was between $p_{inf} = 0.4$ and $p_{inf} = 0.7$ with our choice of parameters. This could indicate that the system goes through a phase transition somewhere in this spectre.

We have found the curve for the number of infected persons for different values of infection probability for two different systems. All the objects were given low ability to move far away from their initial place in the first example. This was done by giving the objects a low self-propulsion velocity and a high rotational diffusion. When the objects are not able to leave their initial neighbourhood, they can only interact with other objects initially placed in the same neighbourhood. This was compared with a regular square lattice system. We found that the infection curves for the active system and the lattice systems have a high resemblance. In the second system, some of the objects were able to move wherever they wanted in the container. This was done by giving them a larger self-propulsion velocity and a smaller rotational diffusion. This system was compared to a small-world network. When this change was done, we found that in general, more people were infected in the second system if the infection probability was the same. We have found that the results from the second simulation resemble the results from the small-world network. The differences between the first and the second active system match the differences between the regular network and the small-world network. We, therefore, conclude that the results support the use of an active matter model as an alternative to the lattice model, and that letting some of the objects move further than other can be seen as an active equivalent to a small-world network. From the fact that fewer people are infected in the regular network, we learn that the infection can be slowed down without reducing the level of interaction, but by restricting who is allowed to interact with each other.

We have also used the model to test several restrictions and measure that can be introduced in a society during a pandemic. One such measure is to introduce more strict rules regarding hygiene and thereby reducing the infection probability. Reducing the infection probability was the first change we did to our model, leading to a considerable improvement in the number of infected. Similar, the infection radius can be changed to see how this affects the spread. When we changed the infection radius to the double of the original the effect was clear; the entire system was infected almost instantly, and this lead to a much worse outcome. The third thing we considered was to identify and isolate the persons who were infected. Isolation was also very effective, even when only 70 % of the infected persons were discovered. The last restriction we have tested is to reduce the number of people gathered in the same group. This was done by using a potential that induces competing interactions. This reduction also turned out to be a quite effective way both to reduce the number of infected and to spread the infection over a longer time. From these results, we conclude that the model can be a starting point for developing a tool to demonstrate how such measures and restriction affect a society.

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Appendix A

System snapshots

Figure A.1 shows four snapshot of a system where the infection probability has been reduced.

Figure A.2 shows four snapshot of a system where the infection radius has been increased.

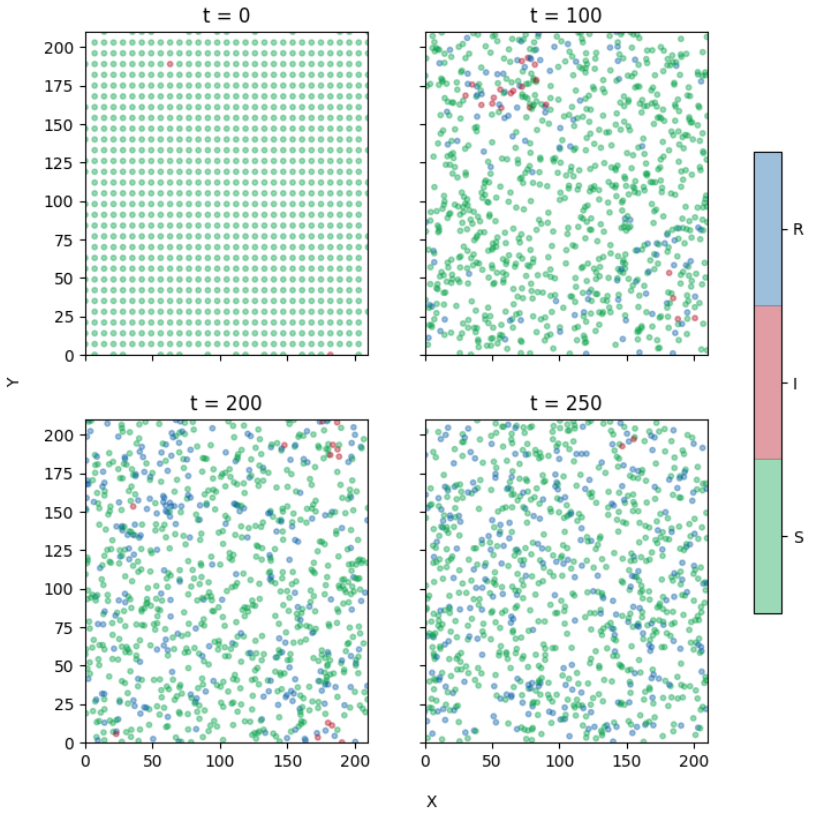


Figure A.1: Snapshots of a system where the probability of spreading the disease, p, is reduced. Parameters used:$N = 900$, $t = 350$, $\Delta t = 0.01$, $L = 200$, $R_{inf} = 12$, $\sigma = 1/2^{1/6}$, $\epsilon = 1$, $D_r = 0.01$, $v_0 = 1$, $p = 0.2$

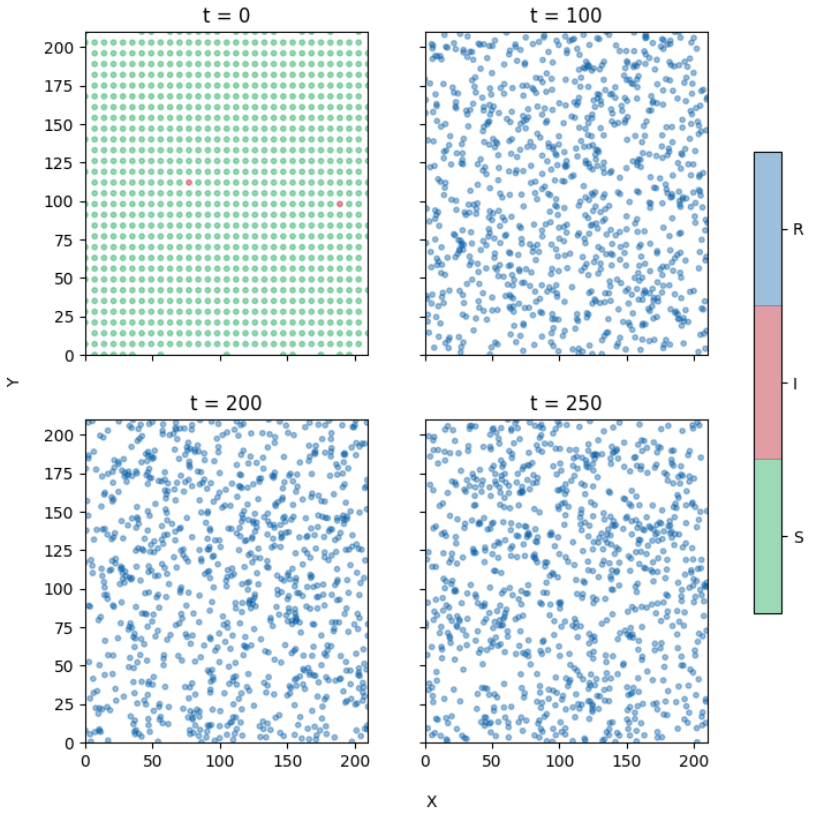
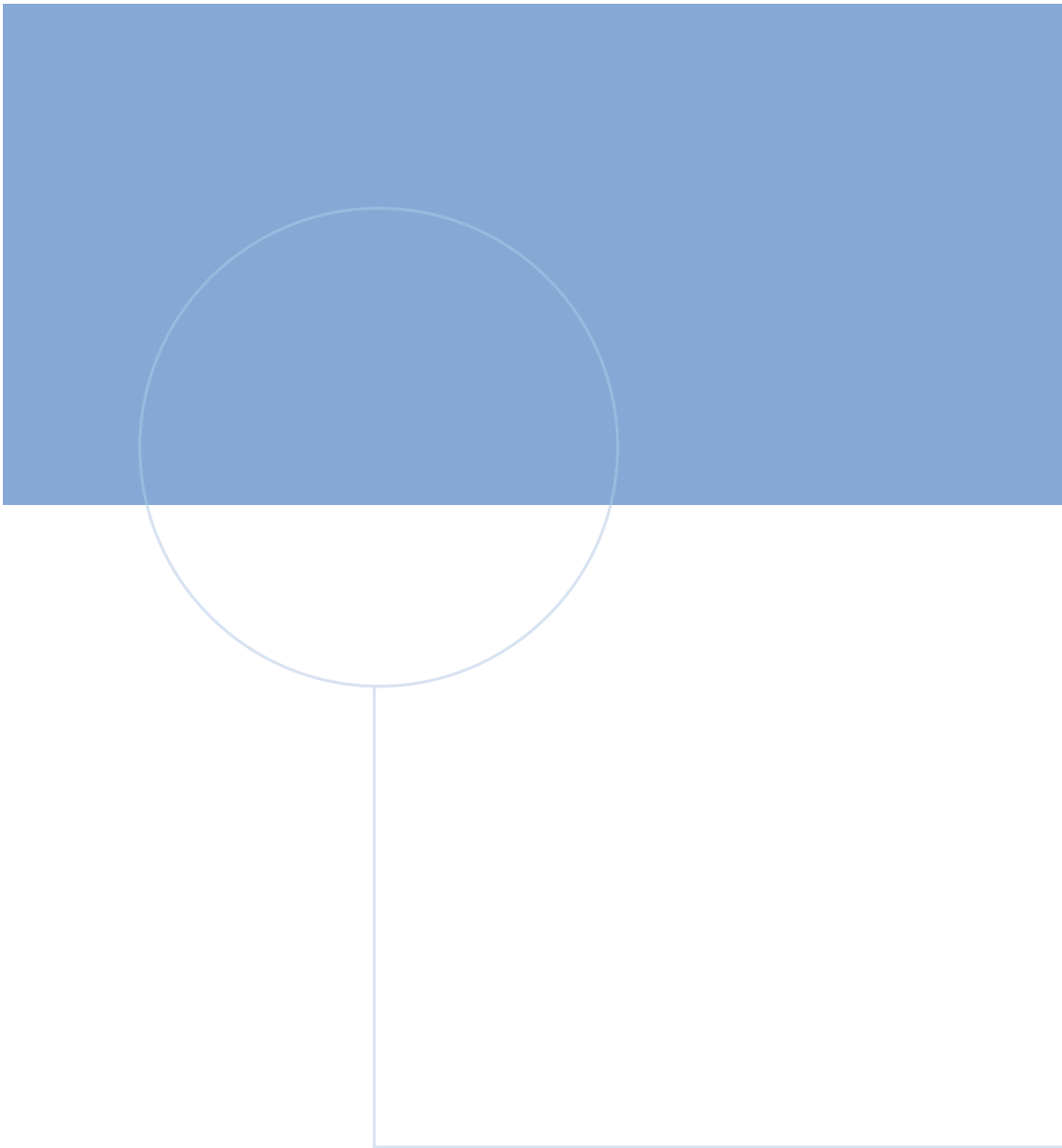


Figure A.2: Snapshots of a system where infection radius,r_{inf} is increased. Parameters used:$N = 900$, $t = 350$, $\Delta t = 0.01$, $L = 200$, $R_{inf} = 24$, $\sigma = 1/2^{1/6}$, $\epsilon = 1$, $D_r = 0.01$, $v_0 = 1$, $p = 0.4$



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