Emergence of SIR Model in the Vicsek Model

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1 Introduction

Collective motion or the dynamics of active matter has been the focus of study for quite some time. It involves a group of agents displaying long range collective behaviour often away from its state of *equilibrium*. This often takes place without an external interaction and does not follow a set direction of motion hence breaking the continuous symmetry of the system, spontaneously. This behaviour is often seen in 'aquatic flocking', 'bacterial growth' etc. To understand this behaviour the pioneering work was carried out by Vicsek, et al. [1] in 1995. The theory that explains this behaviour is briefly explained in below sections.

The Vicsek model to study complex behaviour in human/animal populations alike. It provides a template within which the dynamics of the population can be largely classified into three *states*:

1. Disordered (Random motions, could be called chaotic),
2. Ordered (organised behaviour)
3. Transition between the two previous states (1 and 2) that can be perceived as a state of panic.

In this project we decided to use the Vicsek model as a template to simulate the movement of population (with focus on human population) Susbsequently, we decided to study how disease spreads in the population. In order to incorporate the spread of disease we used a certain simple set of *rules* that could propagate the disease in the population.

The objective was then to compare the results with the standard SIR model [6] that describes the spread in a population by taking into account the infection rate of the disease. The SIR model will be detailed below. Once the comparison of the results from the Vicsek model were found to be in agreement our next objective was to study how changing *rules* mentioned before would enable us to understand how the dynamics of the population could slow-down/fasten the rate at which infection spreads. To study this we broadly divided the population into two more categories: (a) High density (b) Low density. The results obtained along with the analysis are presented in this project. However, do note that among the aforementioned states only state 1 and 2 are being explored, though the state 3 is of quintessential importance, we have not ventured into it's analysis in this project.

2 The Model

2.1 The Vicsek Model

The model describes the dynamics of a set of N self-propelled particles, in lattice of length L. Each particle i is characterized by a position (x, y) and a direction θ. They all move with the same constant speed v_0, following the update:

$$\vec{x}_i(t+1) = \vec{x}_i(t) + \vec{v_i}(t)\Delta t \tag{1}$$

Particles tend to align with the direction of motion of the neighbors, that are elements in the circle of radius r centered in i. The update is given by:

$$\theta(t+1) = \langle\theta(t)\rangle_r + \Delta\theta \tag{2}$$

A term $\Delta\theta$ has been added and represents the amount of noise in the update. We sampled $\Delta\theta$ from an uniform distribution in the interval $(-\eta/2, +\eta/2)$, where η is defined as the noise of the system. We chose as temporal unit = 1, as radius of the neighborhood $r = 1$ and as velocity $v_0 = 0.03$. The model behavior is then depending only on two *control parameters*: the noise η and the density $\rho = N/L^2$. Of course it is possible to generalize this model to more dimensions $v = L^d$, but for the rest of the paper d will be fixed to 2. By increasing the noise from zero on, the system shows a transition between a synchronized phase, where all the particles point to the same direction, and an a-synchronized one, where randomicity rules. In order to describe this transition, we analyzed the absolute value of the average normalized velocity of the entire set:

$$v_a = \frac{1}{Nv}|\sum_{i=1}^{N}\vec{v}_i|. \tag{3}$$

Furthermore we evaluated the *susceptibility* of the system χ that, as usual in this models, near the critical point presents a peak. It is given by

$$\chi = L^2(\langle v_a^2\rangle - \langle v_a\rangle^2). \tag{4}$$

2.2 Finite size scaling

By doing finite-size scaling relations [5] for ψ and χ, we have:

$$\begin{aligned} v_a &= L^{-\frac{\beta}{\nu}} v_{a(+,-)}(L^{1/\nu}\epsilon), \\ \chi &= L^{\frac{\gamma}{\nu}} \chi_{(+,-)}(L^{1/\nu}\epsilon), \\ C &= C_{(+,-)}(L^{1/\nu}\epsilon), \end{aligned} \tag{5}$$

where $\epsilon = (\eta - \eta_c)/\eta_c$ and $C_{(+,-)}$ are the scaling functions (+ means $\eta < \eta_c$, - means $\eta > \eta_c$. By derivating the scaling functions by η we have

$$\frac{dC}{d\eta} = \frac{dC_{(+,-)}(L^{\frac{1}{\nu}}\epsilon)}{d\eta} = L^{\frac{1}{\nu}}\frac{C_{(+,-)}(L^{\frac{1}{\nu}}\epsilon)}{\eta_c}. \tag{6}$$

Then, the critical exponent ν (rate of vanishing of the order parameter), can be evaluated by plotting $log(\frac{dC(_c)}{d\eta})$ against $log(L)$, indeed ν will be the slope of the straight line.

2.3 The SIR Model

The SIR model is a compartmental model with 3 different groups of populations: Susceptibles, Infected and Recovered. The dynamics are ruled by the following system of ordinary

differential equations:

$$\begin{cases} \dfrac{dS}{dt} = -rSI \\ \dfrac{dI}{dt} = rSI - aI \\ \dfrac{dR}{dt} = aI \end{cases} \tag{7}$$

where $r > 0$ is defined as the infection rate and $a > 0$ as the recovery rate. The total size of the population is constant in time. By simply studying the the behaviour of the infected population at time $t = 0$ we have:

$$\begin{aligned} [\frac{\partial I}{\partial t}]_{t_o} = I(rS_0 - a) > 0 \implies S_0 > \frac{a}{r} = S_c \\ [\frac{\partial I}{\partial t}]_{t_o} = I(rS_0 - a) < 0 \implies S_0 < \frac{a}{r} = S_c \end{aligned} \tag{8}$$

Hence, by defining *reproduction rate* $R_0 = S_0 r/a$ this result can be generalized as the following theorem:

Theorem 1. *Given an homogeneous population* $N = S(t) + I(t) + R(t)$ *with the initial conditions:*

$$\begin{cases} S(0) = S_0 \geq 0 \\ I(0) = I_0 \geq 0 \\ R(0) = 0 \end{cases}$$

and an infection with a reproduction rate equal to $R_0 = rS_0/a$*, then :*
If $R_0 \leq 1$*, then* $I(t)$ *decreases monotonically to zero as* $t \to \infty$*.*
If $R_0 > 1$*, then* $I(t)$ *starts increasing, reaches its maximum, and then decreases to zero as* $t \to \infty$*. We call this scenario of increasing numbers of infected individuals an epidemic. It follows that an infection can invade and cause an epidemic in an entirely susceptible population if* $R_0 > 1$ *or* $r > a$*.*

3 Methods

3.1 The Vicsek Model

In order to reproduce the Vicsek Model, we simulated N particles in a 2-dimensional lattice of length L by using periodic boundary conditions. The system is initialized by choosing random position and initial angle.

Algorithm 1: *Initialize()*

```
dtheta = np.random.uniform(-eta/2, eta/2, size=N)
x_0 = np.random.uniform(0, L, size=N)
y_0 = np.random.uniform(0, L, size=N)
        if (t == 0):
            for i in range(0, N):
```

```
x[i][t] = PBC(x_0[i]);
y[i][t] = PBC(y_0[i]);
theta[i][t] = theta0[i];
vx[i][t] = v_0 * np.cos(theta[i][t]);
vy[i][t] = v_0 * np.sin(theta[i][t]);
```

Each particle has a position defined by *(*x[i][t],y[i][t]) and an angle of direction *theta[i][t]*. The parameter *eta* represents the noise of the system and defines the collective behaviour of it. On the other hand, the update will be constructed on the base of [1] and built as:

Algorithm 2: *Update()*

```
else:
    dtheta = np.random.uniform(-eta/2, eta/2, size=N)
    for i in range(0, N):
        for j in range(0, N):
            if (j != i):
             if (distance(x[i][t-1], y[i][t-1]) < 1.0):
                sum_sintheta += np.sin(theta[j][t - 1]);
                sum_costheta += np.cos(theta[j][t - 1]);
                nn = nn + 1;
        av_sin[i][t - 1] = sum_sintheta / nn;
        av_cos[i][t - 1] = sum_costheta / nn;
        av_theta[i][t - 1] = np.arctan2(av_sin[i][t - 1],av_cos[i][t - 1]);
        sumtheta = 0;
        sum_sintheta = 0;
        sum_costheta = 0;
        nn = 1;
        theta[i][t] = av_theta[i][t - 1] + dtheta[i];
        x[i][t] = PBC(x[i][t - 1] + vx[i][t - 1] * dt);
        y[i][t] = PBC(y[i][t - 1] + vy[i][t - 1] * dt);
        vx[i][t] = v_0 * np.cos(theta[i][t]);
        vy[i][t] = v_0 * np.sin(theta[i][t]);
```

Furthermore, the subroutine to evaluate the order parameter v_a is given by:

Algorithm 3: *Measure()*

```
for t in range (0, maxtime):
    for k in range(0, N):
        v_ax[t] += (vx[k][t])
        v_ay[t] += (vy[k][t])
    v_a_final[t] = (np.sqrt((v_ay[t] * v_ay[t]) + (v_ax[t] * v_ax[t])))
    v_a_final[t] = v_a_final[t] / (N * v_0);
    v_a[e] += v_a_final[t]
v_a[e] = v_a[e]/maxtime
```

Finally the main has the form:

Algorithm 4: *Main()*

```
for t in range(0, maxtime):
    if (t == 0):
            Initialize()
    else:
            Update()
```

Measure()

3.2 Vicsek Model + SIR Model

Subsequently we assigned to each particle the vector c[i][t], in order to take track of their state respect to the infection. If $c[i][t] == 0$, the particle i at time t can be considered as *susceptible*, if $c[i][t] == 1$ as *infected* and $c[i][t] == 2$ as *removed*. The disease is introduced in the population by infecting I_0 random elements as shown in the following subroutine:

Algorithm 5: $Initialize_infection()$

```
def init_infection(t):
    a1 = rndm.sample(range(N), N)
    for i in range(0, I_0):
        temp = a1[i]
        for o in range(0, maxtime):
            c[temp][o] = SIR[1]
```

After the first step, at each step every particle will have a probability r of infecting a neighbor, defined as already done in the Vicsek part. The new update function is given by:

Algorithm 6: *Initializeinfection()*

```
dtheta = np.random.uniform(-eta / 2, eta / 2, size=N)
for i in range(0, N):
    for j in range(0, N):
        if (j != i):
            if (dist(x[i][t-1],x[j][t-1])<1):
                sum_sintheta += np.sin(theta[j][t - 1])
                sum_costheta += np.cos(theta[j][t - 1])
                nn = nn + 1;
                if (c[i][t] == SIR[1] and c[j][t] == SIR[0]):
                if (rndm.uniform(0, 1) < r):
                for o in range(t, maxtime):
                    c[j][o] = SIR[1]
                    if(S!=0):
                        S = S - 1
                        I = I + 1
    av_sin[i][t - 1] = sum_sintheta / nn
    av_cos[i][t - 1] = sum_costheta / nn
    av_theta[i][t - 1] = np.arctan2(av_sin[i][t - 1] , av_cos[i][t - 1])
    sumtheta = 0
    sum_sintheta = 0
    sum_costheta = 0
    nn = 1
    theta[i][t] = av_theta[i][t - 1] + dtheta[i]
    x[i][t] = PBC(x[i][t - 1] + vx[i][t - 1] * dt)
    y[i][t] = PBC(y[i][t - 1] + vy[i][t - 1] * dt)
    vx[i][t] = v_0 * np.cos(theta[i][t])
    vy[i][t] = v_0 * np.sin(theta[i][t])
decay_infection(t)
```

Where decayinfection() is given by:

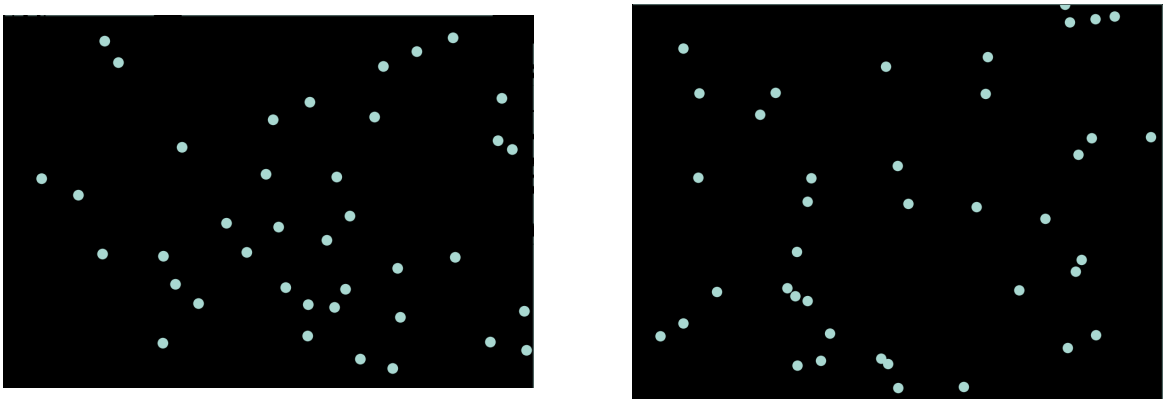
Algorithm 7: *Decayinfection*

```
def decay_infection(t):
    global R
    global I
    global S
    for i in range(0,N):
        if (c[i][t] == SIR[1]):
            if(rndm.uniform(0, 1) < a):
                for o in range(t, maxtime):
                    c[i][o] = SIR[2]
                R = R + 1
                I = I - 1
```

Because of this function, every infected particle will have a probability a/K to recover at each time step.

4 Results and Discussion

In this section we present the results of this project. First we will present the results that were obtained from implementing the Vicsek model alone and discussing how various parameters of the Vicsek model changes the dynamics of the system. Then we will proceed to show what were obtained after we introduced infected specimen into the system.



(a) $\eta = 6$ (b) $\eta = 0.1$

Figure 1: Snapshots of a simulation

4.1 Order parameter in Vicsek Model

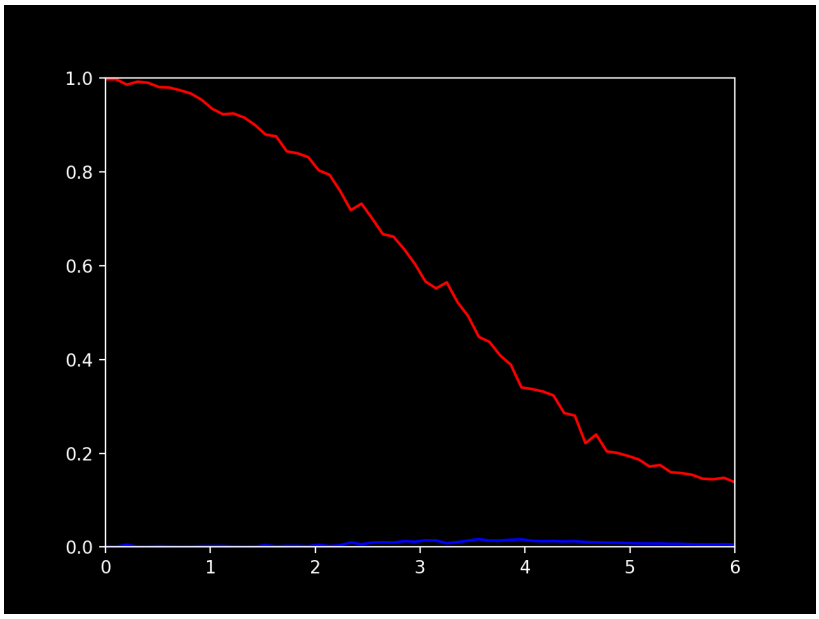


Figure 2: Order parameter v_a vs. noise η, $\rho = 4.44$ (N=40, L=3)

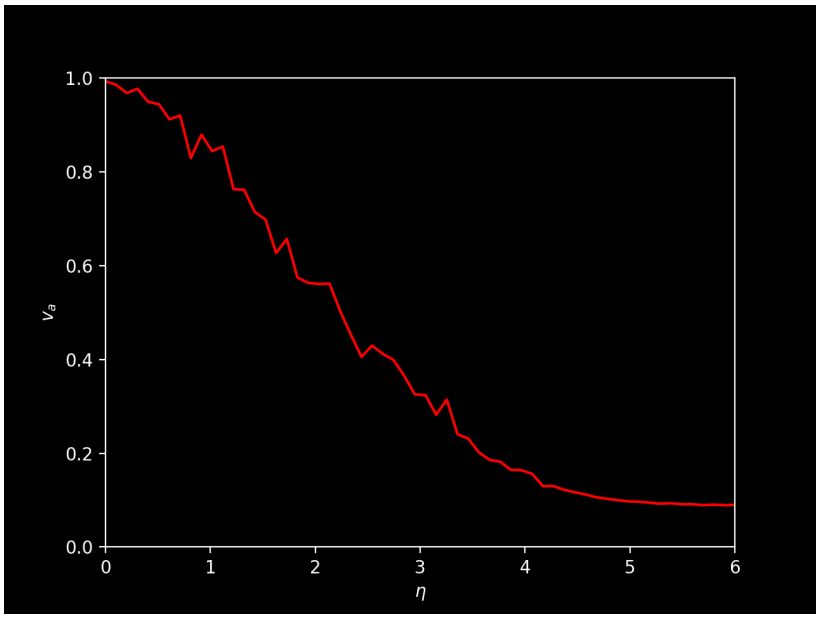


Figure 3: Order parameter v_a vs. the noise η, $\rho = 1$ (N=100, L=10)

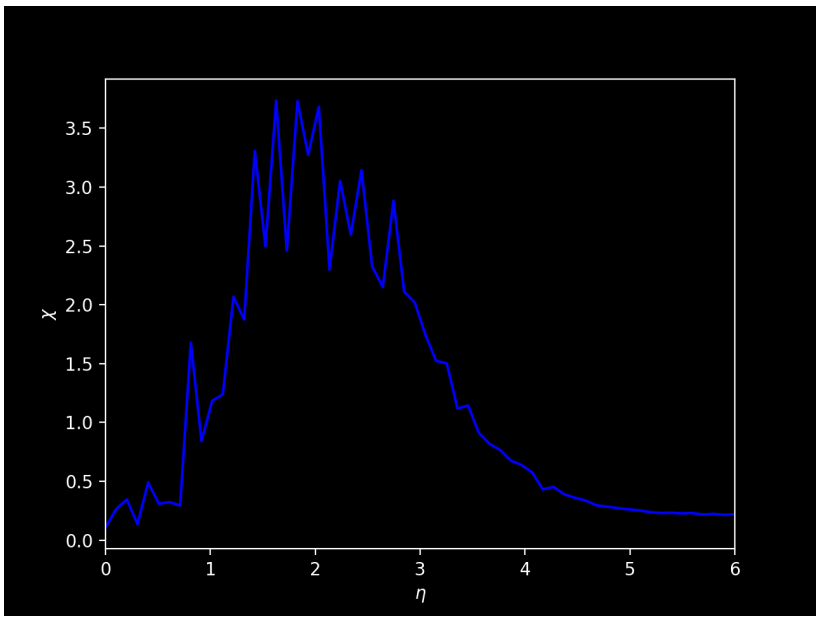


Figure 4: Susceptibility for various values of noise. Peak for a noise of $\eta \approx 1.9$

In *Figure 2* we have shown the plot between the average velocity of the system (oder parameter) against the noise in the system. As we see, as the noise is increased the order parameter goes from unity to almost zero. This change in order parameter indicates that the system has undergone phase transition from an *ordered state* to a *disordered state*. In *Figure 3* however, the order parameter falls much steeply. This is because this was done for a system that is of lower density. This tells us that decreasing the density of the system promotes the decrease of order parameter. The reason for this is presented in the following section. In *Figure 4* we have presented the susceptibility given by,

$$\chi = L^2(\langle v_a^2 \rangle - \langle v_a \rangle^2). \tag{9}$$

This was done to compute the critical value [5] of the noise at which the system undergoes phase transition [1]. We found the critical value for our system to be $\eta \approx 1.9$. This is the noise at which the order parameter variance peaks. We have presented few more systems with different densities and their noise to order parameter plots in *Figure 6*.

4.2 Order parameter vs. the density of the system

Now we saw that the density promotes the sharp decrease in order parameter. To investigate this further we can study the change in order parameter while varying density with fixed noise. *Figure 5* shows the plot between the order parameter and the density of the system for a noise of $\eta = 2.9$.

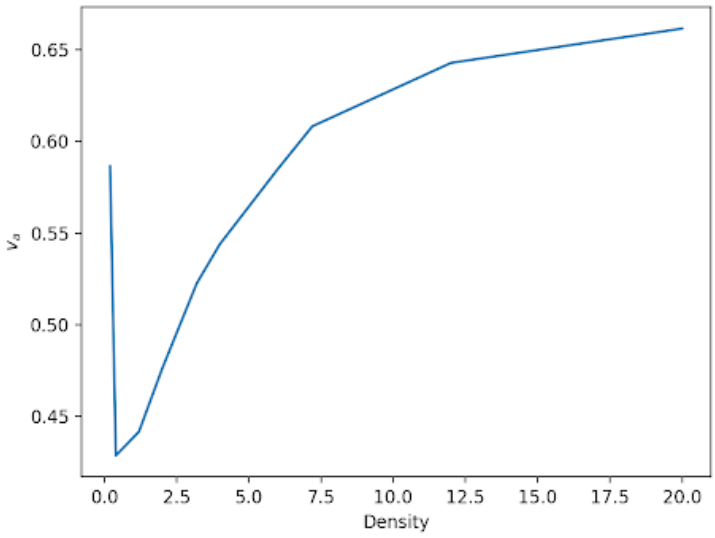


Figure 5: Density ρ versus order parameter v_a for fixed noise $\eta = 2.9$.

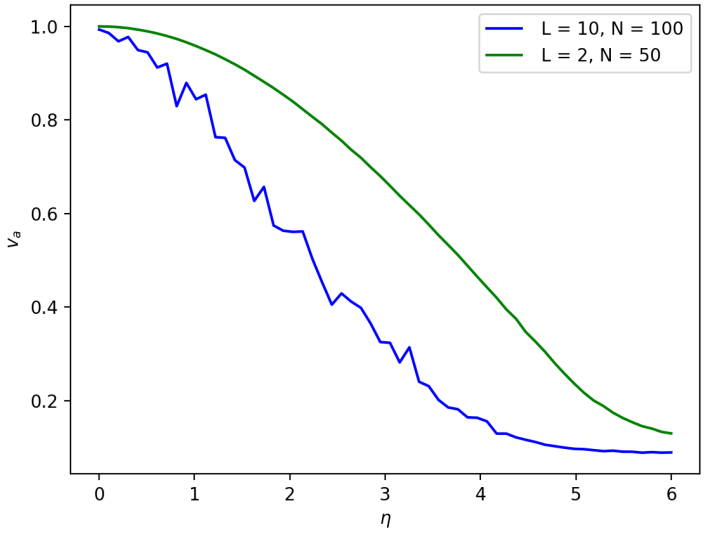


Figure 6: Order parameter v_a vs η for two different densities.

As we can observe from this figure, for a fixed noise, as we increase the density, the order parameter starts increasing. This means we have two means of controlling the order parameter. One way is to change the noise in the system and the other is to change the density of the system. This is exactly the reason why for a low density system the in *Figure 3* the order parameter falls much steeply as opposed to the high density system in *Figure 2*.

4.3 Estimation of critical exponent of the noise

We can assume that the kinetic phase transition in our system is similar to the continuous phase transitions in equilibrium system in the thermodynamic limit and the relationship between the order parameter and the noise takes the following form.

$$v_a \sim [\eta_c(L) - \eta(L)]^{\beta} \tag{10}$$

β is the critical exponent that we wish to calculate. To do this we plot the $\ln v_a$ against $(\eta_c(L) - \eta)/\eta_c$. Then we find the slope of the plot by performing a linear fit which is shown in *Figure 7*. The linear fit gives a critical exponent value of $\beta = 0.33$.

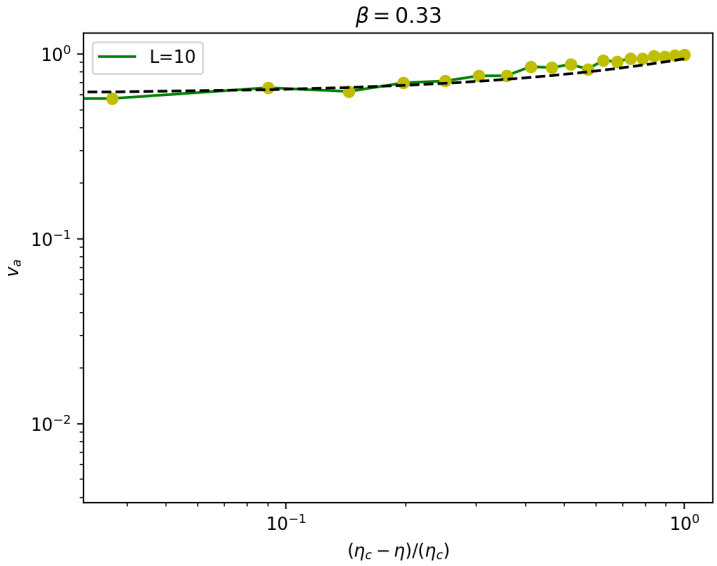
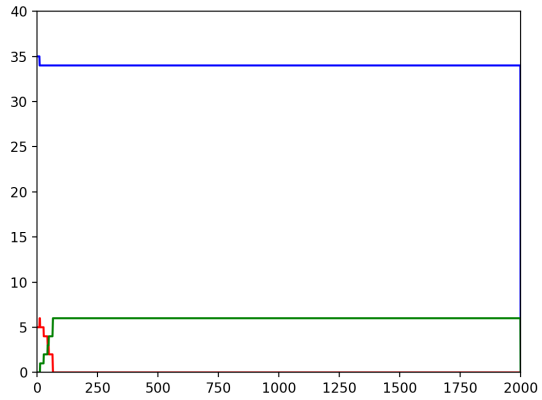


Figure 7: Order parameter v_a vs $\frac{\eta_c - \eta(L)}{\eta_c}$ in log scales for fixed value of density. The linear fit shows $\beta = 0.33$

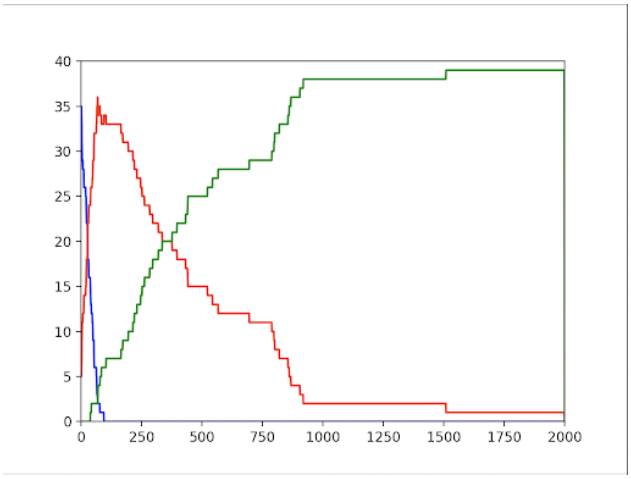
4.4 Vicsek model with the infected population

Now we will present the results for a system in which the dynamics of the population is modelled using the Vicsek parameters. First we introduced an infection in a population that is in an ordered state which is characterized by a low noise in the system. We have further categorized the population as High density (high population density) and Low density (low population density).

4.4.1 Infection in population that is ordered (Low noise) and Low density ($\rho = 4.4$)



(a) No epidemic scenario with $R_0 = 0.77$.



(b) Epidemic scenario with $R_0 = 105$.

Figure 8: Recovered population Susceptible population. Infected population.
Initial values: $S(0) = 35, I(0) = 5, R(0) = 0$. Noise η fixed as 0.1.

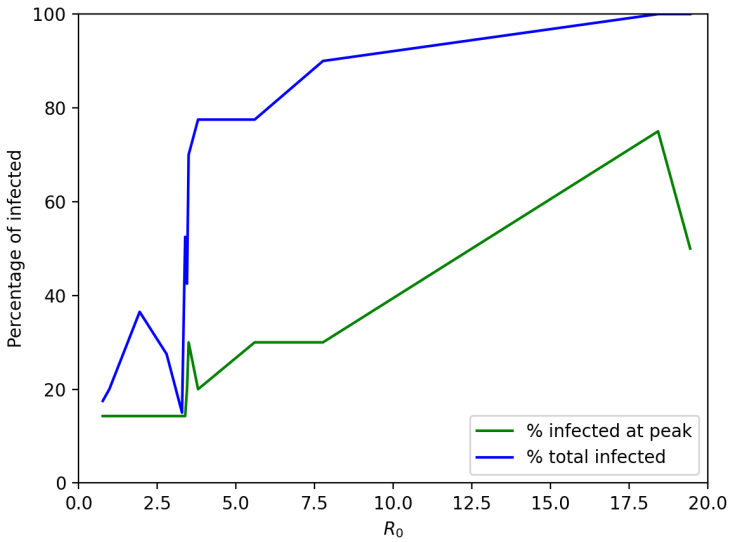


Figure 9: % population infected at peak of epidemic and % population infected in entire course of epidemic..
On the x-axis the reproduction rate R_0. At $R_0 = R_c \approx 3.5$ we have the critical reproduction rate.

Here we have plotted the results in *Figure 9* for the percentage of population infected when the epidemic reaches its peak and the total percentage of population infected in the entire

course of the epidemic for various values of reproduction rate R_0. Around $R_0 = R_c = 3.5$ we have the critical reproduction rate where happens a transition from a no-epidemic state to an epidemic one.

We have presented some more data for percentage population affected in *Figure 10* and peak time for infection in *Figure 11*.

In *Figure 8 a)* we show how the infection spreads in the population for a fixed value of $R_0 = 0.77$ over a period of time steps. As our critical value for reproduction rate is 3.5, a value below this will not result in an epidemic. The blue curve represents the Susceptible population, the red curve the infected and the green are the recovered and as we can see in this case the number of infected population does not grow over the period of time. However in *Figure 8 b)* we see that the number of people who are infected grows rapidly under a short period of time and this is due to the high reproduction rate ($R_0 \sim 105$).

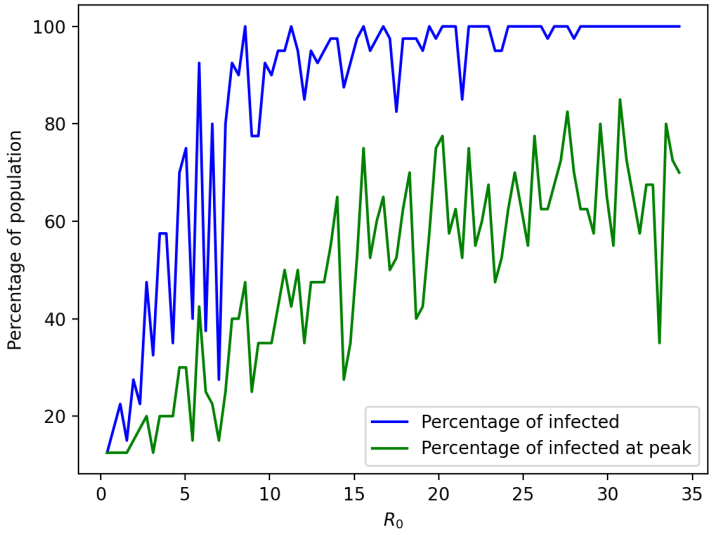


Figure 10: % population infected at peak of epidemic and % population infected in entire course of epidemic..
On the x-axis the reproduction rate R_0 at noise $\eta = 0.1$

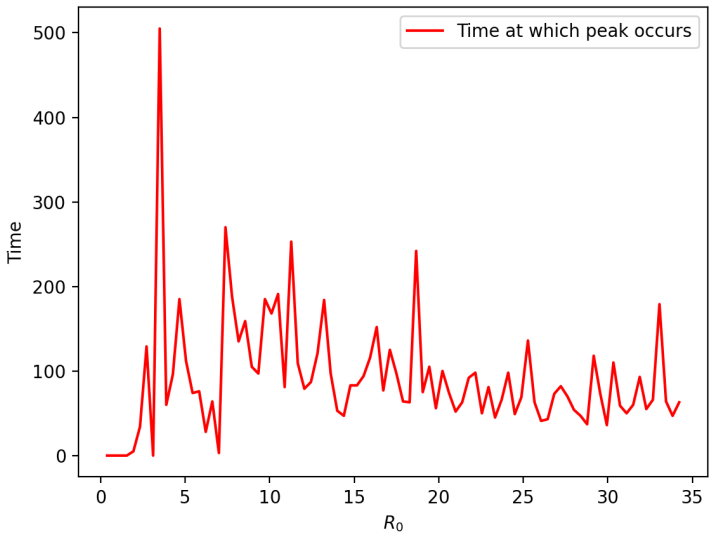
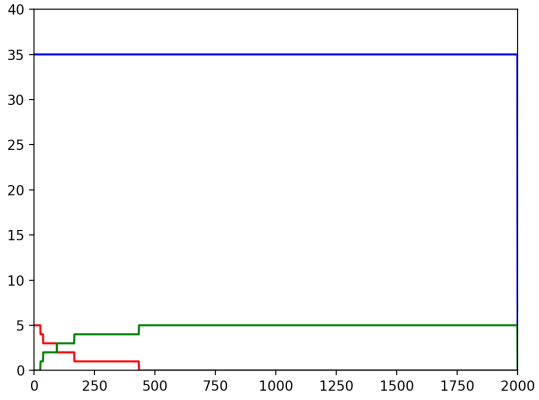


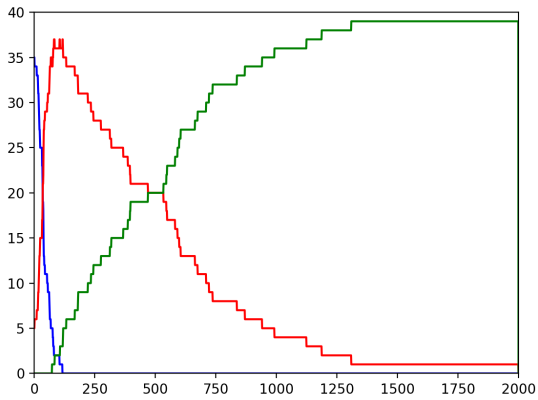
Figure 11: Peak infection time vs. reproduction rate R_0 at noise $\eta = 0.1$

The plots in *Figure 10* and *Figure 11* have been done with higher number data sets.

4.4.2 Infection in population in chaotic/disordered scenario (High noise) and Low density ($\rho = 4.4$)



(a) No epidemic scenario with $R_0 = 0.77$.



(b) Epidemic scenario with $R_0 = 105$.

Figure 12: Recovered population Susceptible population. Infected population.
Initial values: $S(0) = 35, I(0) = 5, R(0) = 0$. Noise fixed as $\eta = 5$.

Now we discuss the situation where the density of the system is Low as in section 4.4.1 . Contrarily here the system is having a high noise, hence completely random dynamics. In

Figure 13 we have again plotted the percentage population that is infected at the peak of infection (green) and total percentage of population infected during the entire course of the epidemic (blue).

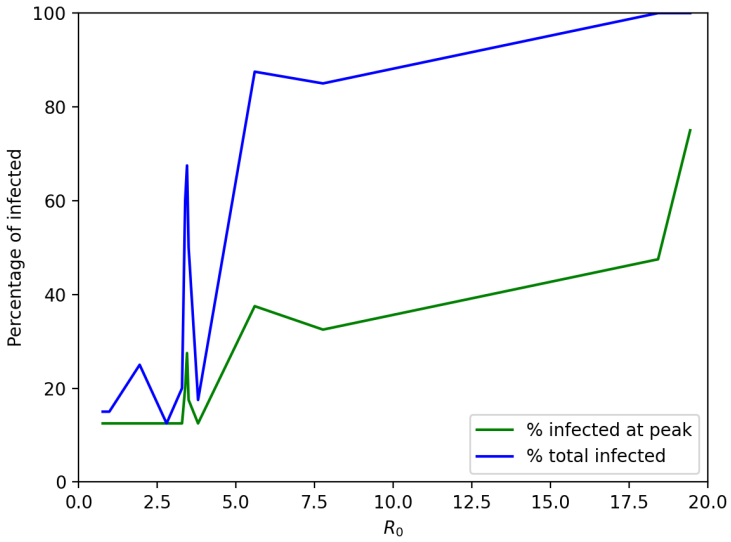


Figure 13: % population infected at peak of epidemic and % population infected in entire course of epidemic..
On the x-axis the reproduction rate R_0 at noise $\eta = 6$.

Also presenting some more data in *Figure 14* and *Figure 15* for the percentage population infected and the time taken for the population to reach its peak respectively.

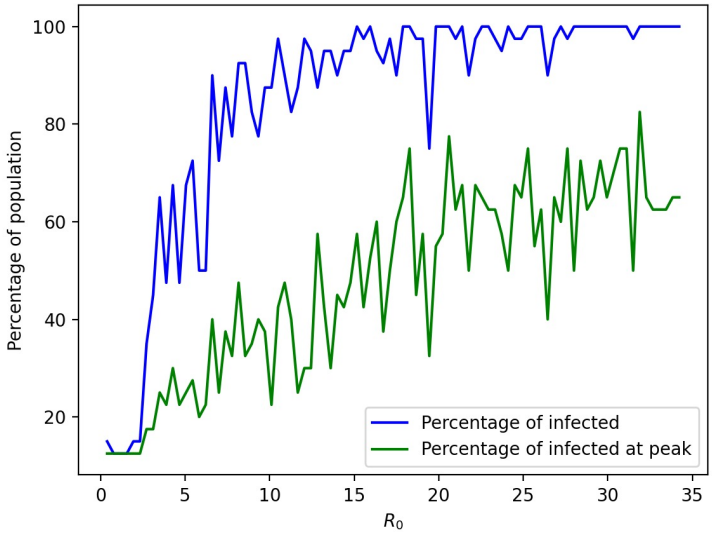


Figure 14: % population infected at peak of epidemic and % population infected in entire course of epidemic..
On the x-axis the reproduction rate R_0 at noise $\eta = 6$

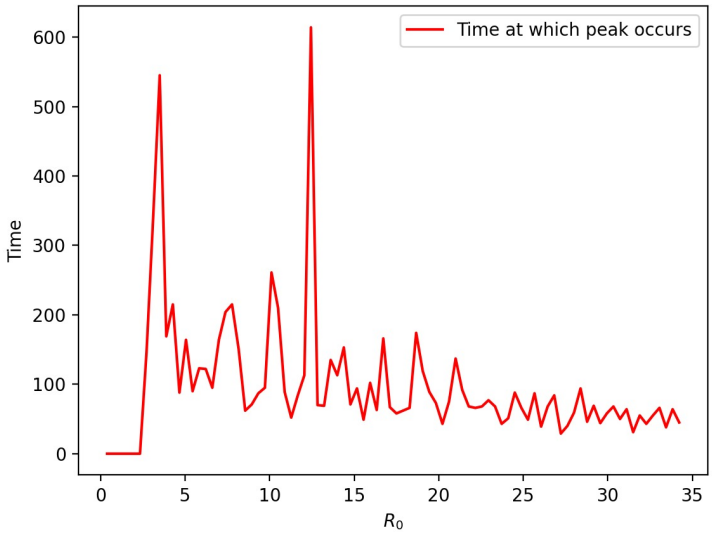


Figure 15: Peak infection time vs. reproduction rate R_0 for high noise of $\eta = 6$

4.4.3 Infection in population that is ordered (Low noise) and High density ($\rho = 11.11$)

Now we consider a system that is of low noise which can be seen as the movement of population to be ordered or in a disciplined manner. However we have now changed the density

of the population and have presented the results for a system that has a high population density. In *Figure 16* we have presented the percentage of populations that are affected at the peak of the epidemic (green) as well the total percentage of population affected in the entire course of the epidemic (blue). We now see the critical value for the reproduction rate has shifted in this system to a value of $R_c \approx 2.2$ which means that the transition to a state of epidemic happens earlier in this case.

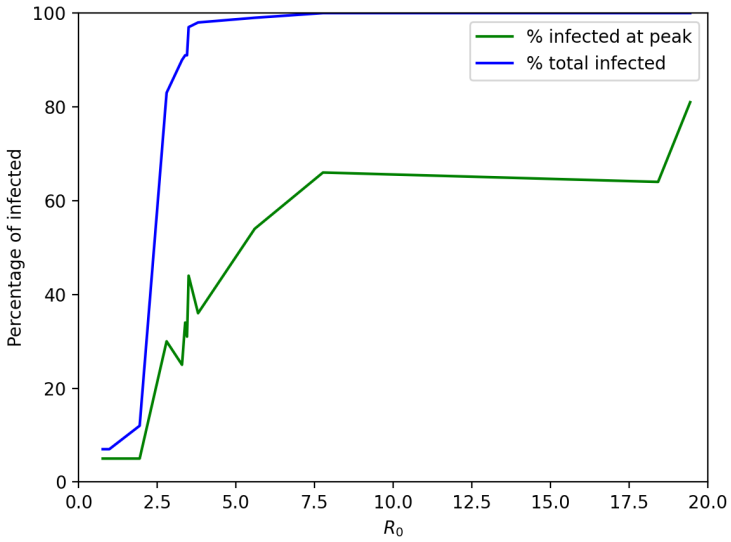


Figure 16: % population infected at peak of epidemic and % population infected in entire course of epidemic..
On the x-axis the reproduction rate R_0. At $R_0 = R_c \approx 2.2$ we have the critical reproduction rate.

In *Figure 17* we have shown the time taken for infection to reach its peak for various values of reproduction rate R_0

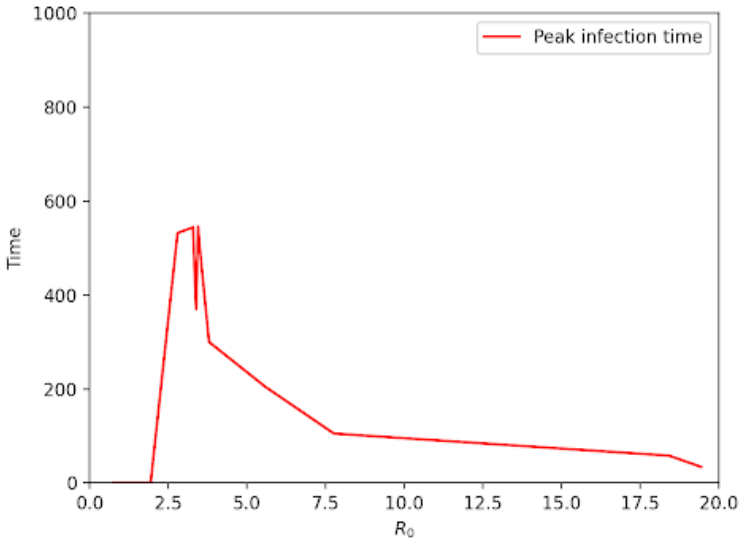


Figure 17: Peak infection time vs. reproduction rate R_0

4.4.4 Infection in population that is disordered/chaotic (High noise) and High density ($\rho = 11.11$)

Now we look at a system that is of high noise or a population that has a random or chaotic movement. We have again changed the density of the system to model a population that is densely packed. In *Figure 18* we have presented the percentage of population affected at peak of the epidemic (green) and percentage of total population affected over the course of epidemic (blue)

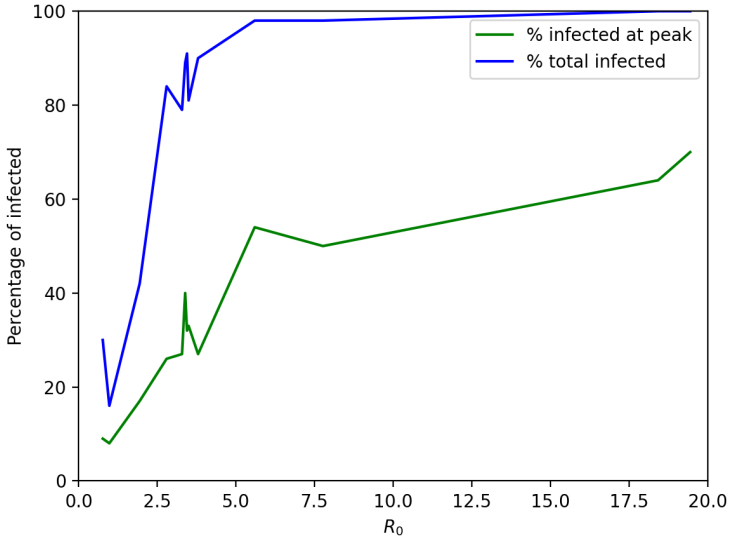


Figure 18: % population infected at peak of epidemic and % population infected in entire course of epidemic..
On the x-axis the reproduction rate R_0. At $R_0 = R_c \approx 2.5$ we have the critical reproduction rate.

And as usual, we also have the time taken for the epidemic to reach its peak shown in *Figure 19*.

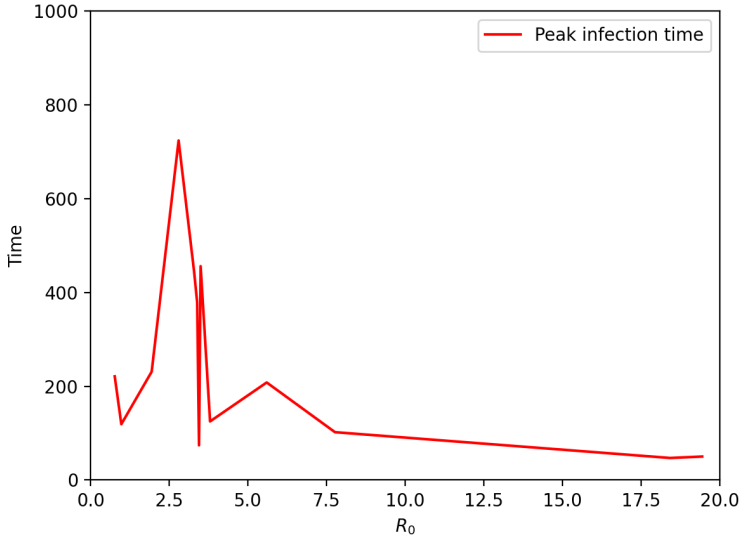


Figure 19: Peak infection time vs. reproduction rate R_0

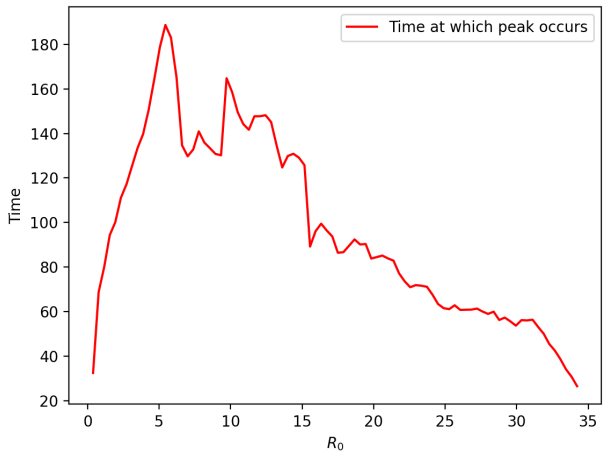
4.5 Discussion of results

4.5.1 Emergence of SIR results

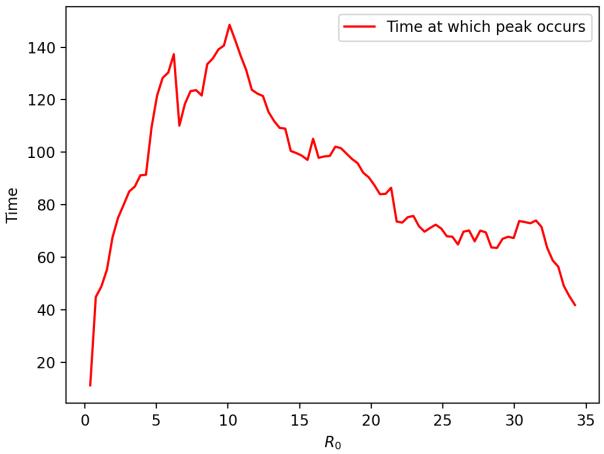
We have discussed the dynamics of an epidemic in the results section 4.4.1 and from *Figure 8a* and *8b* we see how introducing an infection to a population modelled by the Vicsek model naturally gives rise to the SIR model [6] without solving the ordinary differential equations provided in equation *(8)* and *(9)*. This is noteworthy as this is an assurance that Vicsek model is a fine template to model population dynamics. Also note, how by changing the reproduction rate R_0 the simulation gives us the epidemic or no epidemic condition as predicted in SIR model in *Theorem 1* in section 2.3. Although here the critical value for R_0 is different from the value in the theorem, but this can be normalized to unity.

4.5.2 Comparison of results in the Low density case

Comparing the Low density cases first we can look at the *Figure 9* and *13* we see that the percentage of total population that is infected (indicated by the blue curve), is slightly lower for the case where the population movement is chaotic (high noise) whereas the percentage of population infected at the peak of the epidemic (green curve) is almost same in both cases except that, at lower reproduction rates, the population that is disordered (high noise) is slightly better that the population that is ordered/disciplined (low noise). However when it comes to understanding the time taken for the epidemic to reach its peak, we can observe *Figure 20a* and *20b*, that show the moving average for the time taken to reach the peak. Comparing these two we see that for the case where population movement is disordered (High noise), the time is greater for all values of reproduction rate R_0, as opposed to the ordered/disciplined case where the time taken is less. This tells us that the infection reaches its peak much quickly in population that follows a disciplined/ordered state. However we must mention that in populations with a chaotic/disordered state (High noise) the order parameter is also less, indicating that the population is not moving much when compared to the low noise case where the population is moving a great deal of distance. Disordered/chaotic (High noise) can be seen as a form of *isolation*. We must note that the time taken in the low noise case although less to reach the peak is still not significantly lesser than the high noise case.



(a) High noise case ($\eta = 0.1$)



(b) Low noise case ($\eta = 0.1$)

Figure 20

4.5.3 Comparison of results in the High density case

First we look at the percentage of people infected at the peak of epidemic denoted by the green curves in *Figure 16* and *18*. We observe that the percentage of population infected at peak of epidemic is roughly the same for both the cases where the movement of population is chaotic/random (High noise) or coordinated/disciplined (Low noise). Moving ahead we take a look at *Figure 17* and *19*. We observe for populations that have random or disordered movement the time at which the epidemic reaches its peak is roughly 700 time steps whereas for the population that are coordinated(low noise) reach their peak at a time step of 550 time steps. This indicates that to prolong the peak epidemic it is better to have a population that follows chaotic behaviour if the population density is higher. However this significant difference in time is only noticeable at a reproduction rate that is closer to critical reproduction rate $R_0 \sim R_c$, at rest of the reproduction rates, the time to reach the peak of epidemic is roughly the same.

5 Conclusion

In this work we firstly presented and discussed the main features of the Vicsek Model. In analogy with many other models of this kind, we observed a continuous phase transition with the emergence of cooperative motion. Despite the small system used (N=100, L=10) it has been estimated a critical noise of $\eta_c = 1.9$ and a critical exponent of $\beta = 0.33$, not too far from the results obtained in the original paper ($\eta_c = 2.9, \beta = 0.45$). We observed that low density and high density systems have a very similar behaviour when subjected to an infection. In both the systems, populations moving in an ordered (low noise) or disordered (high noise) manner, the percentage of population getting infected at the peak of epidemic, as well as throughout the entire course of the epidemic, is comparatively the same. However

when we look at the time taken for the epidemic to reach its peak, populations with a disordered state (high noise) is higher when compared to the ordered states case. This happens because, when the movement is chaotic, individuals do not spread across the lattice (less diffusive motion). In terms of population dynamics this can be seen as a situation in which people are isolated with restricted movement, only in the immediate neighborhood. Whereas, when the movement is synchronized, even though there is clustering happening, the spread of the population is not restricted to their immediate neighborhood but to the entire lattice (diffusive motion). This gives more room for epidemic to grow. Further analysis can be explored by going more in depth in this different diffusive behaviour. As a conclusion, we can say that the Vicsek model can be used as a template to study population dynamics, where by changing the two control parameters (density and noise) we can observe different interesting scenarios. Here, we applied this to epidemics, showing that it is possible to predict the behaviour of an epidemic depending on how the density or the noise. With a higher level of abstraction, the noise can be modeled as the degree of social regulations (as countermeasure to stop the transmission) and further analysis can be developed.

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