on Computer Simulations of the Ising Model, and its possible use in modelling the spread of pathogenic diseases

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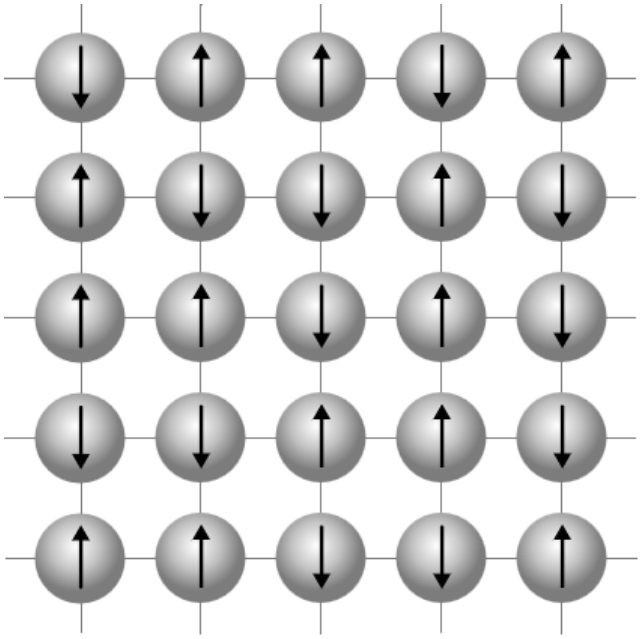

Ising spin lattice (anti-ferromagnetic)

Abstract

This investigation sees the simulation of a 2-dimensional Ising Model using Markov Chain Monte Carlo Methods such as the Metropolis Algorithm applied to ferromagnetic materials as a function of temperature using Python 3.6.0. The average energy, average magnetisation, magnetic susceptibility, and specific heat capacity of the lattice were investigated over a range of temperatures. It was found that a phase transition occurs in the 2-D lattice at a critical, Curie Temperature, determined to be 2.4 ±0.1 J/Kb (in good agreement with literature results of Onsager, 1944), and the ground state energy of the lattice was determined to be 2J where J is the exchange energy between spin sites. Finally the model and its methods were adapted to create a visual model of the spread of pathogenic diseases as a function of their virility, mortality rate, and associated immunity of the population.

Introduction

The Ising Model

The Ising model, named for Enst Ising, a PhD student of Wilhelm Lenz is a theoretical model used to explain ferromagnetism; that is magnetism not due to applying a current through a conductor. The model is a simple one which attributes ferromagnetism to atomic spins. The model suggests that a lattice of atoms can have their spins align with that of their nearest neighbours, which yields a net magnetic moment which can be detected on the macroscopic scale.

In our understanding of the Ising Model, let us introduce an atomic lattice, of NxN sites, each of which has a spin, s_i of either +1, or -1. The overall energy of a site is lower when it is in alignment with its nearest neighbours, and this effect carries throughout the lattice. This effect can be attributed the Pauli exclusion principle, more specifically Pauli repulsion: nearby electrons cannot have both the same spin and energy states, however as the atoms to which they belong become closer, one must move to a higher energy state, keeping its spin the same. Although an electron has moved to a higher energy state, the overall energy of the system is lowered by this interaction. The nature of the energy is electrostatic, and very short range; this is why we consider the state of only the nearest neighbour spins. As the spins align, due to the instrinsic spin propeties of electrons, there is a net magnetic moment, which can be large enough to be experienced on a macrscopic scale.

More rigourously[5]:

we label the lattice site index of an NxN matrix, [i]
the spin of a site is $S_i \in -1, 1$.
the spin matrix is given by S=$(S_1, S_2, ..., S_{n-1}, S_n)$
the Hamiltonian of the system can be given by:

$$H(S) = -J[\sum_{i,j} S_i S_j] - h[\sum_i S_i] \qquad (1)$$

where J is the exchange energy,
h is a magnetica field applied externally,
(i,j) represents the sum over the nearest lattice neighbours of spin S_i.

For a ferromagnet J >0, whereas for an anti-ferromagnet J<0

The model is based upon the ideas of Wilhelm Lenz. Lenz suggested that "atoms are like dipoles which turn over between two positions", however this was incompatible with the atomic models of the time. He discussed his theory with a PhD student, Ernst Ising, who suggested a matrix formulation of squares to solve the problem. Ising went on to write his thesis on the 1 dimesnional variation of the model, and noting that there was no phase transition, incorrectly concluded that there was no phase transition to the n^{th} dimension (this was disproved in 1936 by Rudolf Peierls who demonstrated the existence of a phase change in the 2-D Ising model). The critical temperature was the "Curie temperature", outlined by Pierre Curie in his doctoral thesis 1895- a temperature beyond which magnetic materials undergo a change in their magnet properties.[2]

The Ising model by virtue of its simplicity is incredibly adaptable and applications can be seen in almost every field: ferromagnetism (of course), the study of co-operativity between ion channels (Liu Y. et al), and even urban segregation in American communities (Stauffer, 2008). In this investigation however a truly novel and potentially extremely application has been found: using the Ising Model to describe the spread of pathogenic diseases throughout a population, as a function of the disease virility and the herd immunity and how the use of antibiotics can eradicate widspread disease

The Metropolis Algorithm

The Metropolis(-Hastings) algorithm falls under the umbrella of Monte-Carlo methods- simulations which create distobutions of possible outcomes rather than a discrete, defined hard outcome. The Metropolis algorithm can be described generally as a Markov Chain Monte Carlo (a Markov chain is a model which describes the sequence in which events will occur, where the probability of each state depends only on the state attained in the prior event [1]). Markov chain Monte Carlo methods follow a defined set of steps:

- Start from some initial parameter value.
- Evaluate un-normalised posterior.

- propose a new parameter value.
- evaluate the new un-normalised posterior.
- decide whether the new value should be accepted.[3]
- repeat.

In simulating the Ising Model computationally, the metropolis algorithm was the key component in evaluating whether a spin should be 'flipped' or not depending upon the spin state of its nearest neighbours. If the energy of the system was lowered by a spin, the spin was permitted, otherwise the spin was permitted only with a certain probability, P. Should a randomly generated value fall below the probability P, the change was rejected, and a new lattice site was evaluated.

0.1 Observable Properties

This investigation saw 4 primary observables reproduced as a function of temperature, and this data was used to calculate the Curie temperature (T_c) at which the phase transition occurs for a 2-dimensional Ising Model.

The 4 observables investigated were:

- The total magnetisation of the system
- The magnetic susceptibility of the system
- The average energy of the system
- The specific heat of the system

The magnetisation of the system is a macroscopically observable quantity, for a given temperature can be given by:

$$M(T) = \sum_i < S_i >= \frac{1}{N^2} \sum_i S_i \tag{2}$$

Where M(T) is the magnetisation as a function of temperature,
N is the lattice size..

Magnetisation $< M >$ can be defined simply as the density of the magnetic dipole moments of a system, induced when placed near a magnet, due to the spin state of nuclei and/or electrons. Practically speaking magnetisation against iterations is a strong indicator of the sufficient number of sweeps of the metropolis algorithm required for a given matrix size; we simply print the value of sweeps after which the value of magnetisation becomes constant for that given lattice size.

The magnetic susceptibility ¡χ¿ of a system is tied closely to the definition of the magnetisation of that system. Loosely, the susceptibility describes the extent to which a system can be magnetised in relation to the magnetic field applied to it.[4] The susceptibility can be given as χ:

$$\chi = \frac{\partial < M >}{\partial H} \tag{3}$$

$$\chi = \frac{1}{K_B T}(< M^2 > - < M >^2) \tag{4}$$

The average energy ¡E¿ of the system is equal to the average value of the Hamiltonian divided by two to avoid the double counting of each lattice site:

$$< E >=< \sum_{<i,j>} H_{i,j} >= 1/2 < \sum_{i,j} H_{i,j} > \tag{5}$$

We expect the value of the observable ¡E¿ to be equal to (-2)*J in the case that the lattice is homogeneous. We expected a negative result only for a ferromagnetic material. Were the material to be antiferromagnetic, J would be equal to -1 and so the ¡E¿ at homogeneous solution should be equual to (+2)*J. Since we expect the system to move from 'order' to 'disorder with temperature, as we plot temperature against the average energy, we expect a change in the nature of the curve at the phase transition point, that is, at the Curie temperature for the system.

Finally we can define the specific heat capacity C_v:

$$C_v = \frac{1}{K_b T^2}[< E^2 > - < E >^2] \tag{6}$$

0.2 Biological Application: Modelling the Spread of Pathogenic Disease

The Ising model simulation outlined above, or more specifically the metropolis algorithm is *incredibly* versatile with applications across almost every field of study given that the Markov chain Monte Carlo methods are not extremely computer resource intensive, and a probabilistic representation is more representative of real life systems. As such this investigation modifies the metropolis algorithm and the Ising Model such that models which show the spread of pathogenic diseases with varying mortality rates and virility affect the population. A second, 'lightweight' Metropolis algorithm was used to model the application of antibiotics to the population, assuming uniform distribution.

The model allows one of three states:

- healthy; assigned a numerical value of +1 (green)
- infected; assigned a numerical value of -1 (yellow)
- Dead; assigned a numerical value of -2 (red)

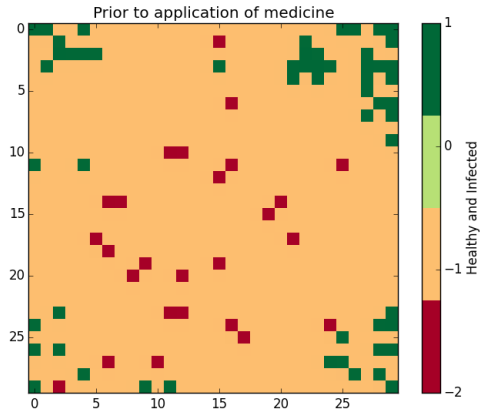

fig 1: This graph shows the state of a population ravaged by a low-fatality disease, prior to medical treatment. The purpose of this graph is to illustrate the difference between the three states graphically

The nearest neighbour function of the Ising model extends well to disease spread, however, rather that an energy evaluation, a lattice site will become infected IF a nearest neighbour is infected AND the virility of the disease is greater than the immunity of the population. Once infected a second conditionality statement is applied relating to mortality: IF a randomly generated number is below the mortality rate, the lattice site will 'die'. This is a terminal state. The dead stay dead. Assuming the application of the model to a developed country with good hygiene practices is crucial; the assumption that a 'healthy' nearest neighbour site CANNOT be infected by a dead site has been made, although this is easily modified, should one wish to apply the model to a less developed area with less rigorous hygiene practices.

Finally, a recovery function has been added such that the 'infected stage' is not terminal. The effectiveness of the applied anti-biotic/remedy has been assigned, such that for a given number of sweeps, if a randomly generated number is less than the recovery rate (normalised), the site will recover.

1 Methodology

The simulations were written in Python 3.6.0, with each simulation having an associated module and script. The module stored all pertinent functions and procedures, which were then, in turn called within the script. The Metropolis algorithm, which was key to this investigation, as mentioned previously, is outlined below, for both an Ising model investigation of magnetism, and for the adapted simulation to model the spread of pathogenic bacterial/viral disease:

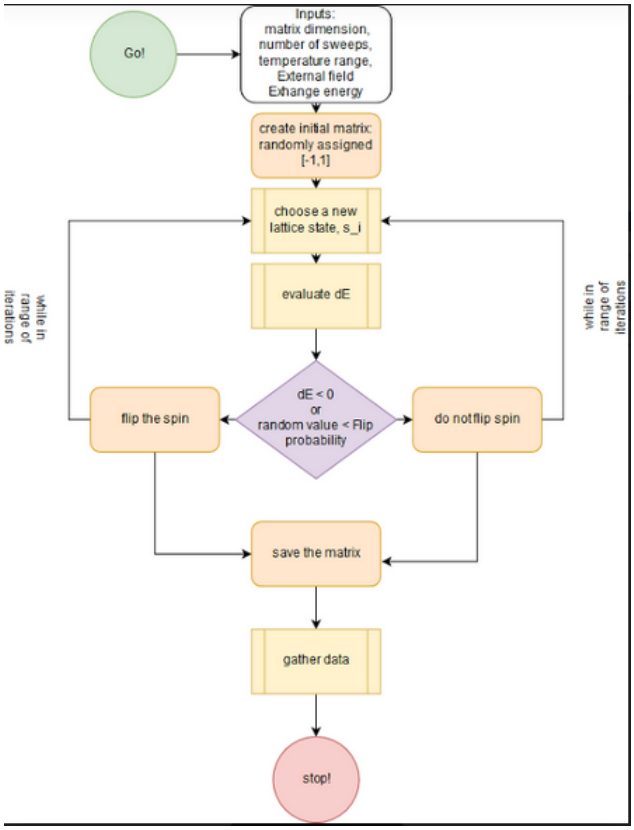

fig 2:Flowchart illustrates the metropolis algorithm used in simulating the Ising Model of Ferromagnetism

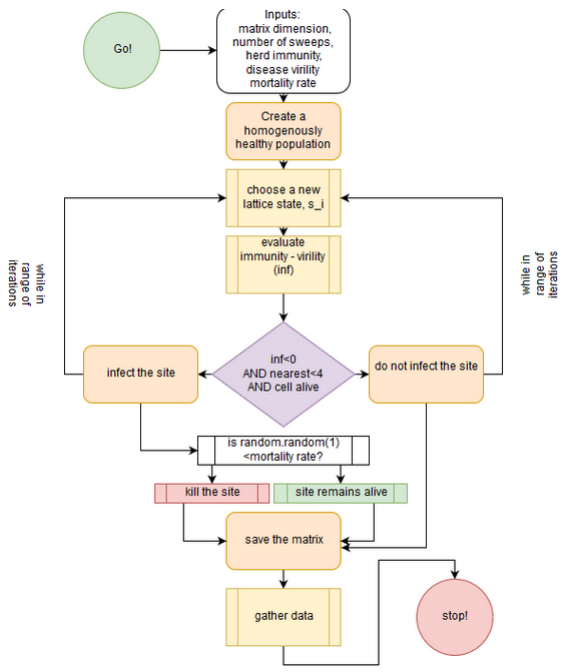

fig 3:Flowchart illustrates the metropolis algorithm used in simulating the spread of pathogenic diseases

There were supplemental functions within the code of both simulations to obtain observable data, or to model the effect of recovery, in the case of pathogenic disease spread. The full set of functions can be found at:

`www.github.com/cumminj1/ising-desktop`

in the lattice and medical python codes respectively.

2 Results and Discussion

The results for ferromagnetism and pathogenic disease spread have been reported separately

2.1 Ising Model of Ferromagnetism

An animated .gif extension file illustrating the effect of increasing temperature on a 60 x 60 Ising lattice can be found at:

`github.com/cumminj1/ising-desktop/blob/master/isingmodeltemp.gif`

however, a four panel summary can be seen below:

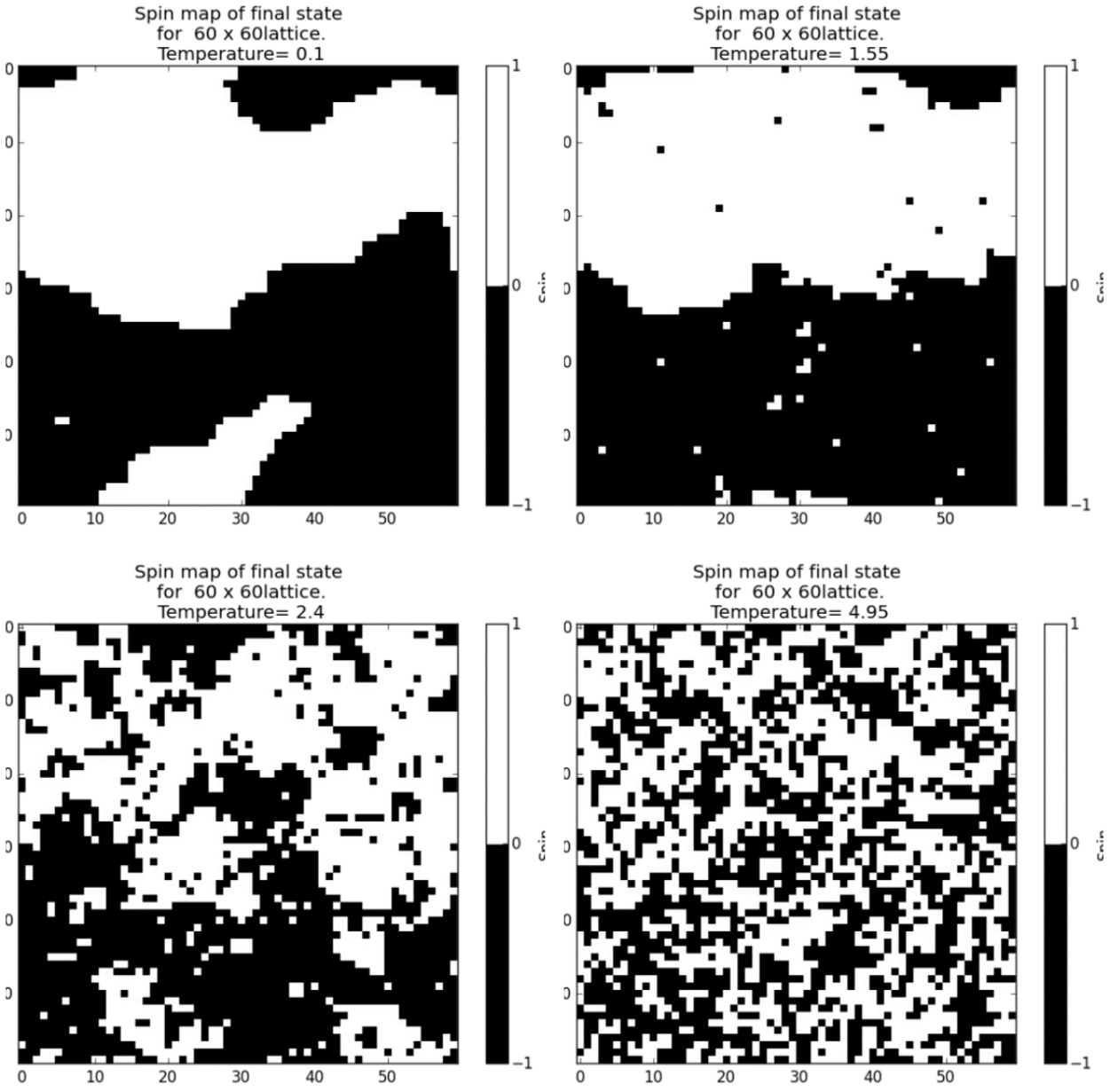

fig 4:The Spin states of a 60x60 ising lattice at four temperatures; very low, $T < T_c$,T_c, $T > T_c$

We expect that below the critical temperature, T_c that the lattice will display Ferromagnetic behaviour, i.e. will tend to align the spin of each lattice site with that of its neighbours. The first panel of figure 4, at low temperature values, this behaviour is exactly what we observe, although the lattice has not reached *full* spin equilibrium, suggesting that for this lattice size, the number of sweeps through the Metropolis algorithm (750) was not sufficient. This is easily remedied in theory- by simply adding more sweeps, however in reality the result obtained took a large amount of time due to a less than powerful processor (intel i3 $\approx$ 9 yrs old). The issue could also be remedied by simply reducing the size of the lattice under investigation.

As the temperature rises (remaining below T_c), we

note that some sites do not conform to the configuration of their nearest neighbours. As the temperature of the lattice increases, so too does the probability that a site will remain energetically unfavourable to the system given the additional heat energy available. This noted, the system still conforms to what we would expect of a material exhibiting ferromagnetism.

At the critical temperature, T_c, shown here in the third panel of fig 4. The critical temperature denotes a phase change in the material, with the magnetic qualities of that material changing considerably; no longer does the lattice conform well to a model of ferromagnetism, however AT the critical temperature the system does not have sufficient energy (for the sweep limit) to tend towards disorder. This result is consistent with the Ising model of ferromagnetism.

Finally we examine the lattice at a higher temperature and we can conclude that the system has become disordered due to the higher available energy in the system due to heat energy. This disorder is consistent with the theoretical model of the lattice as the temperature rises above the Curie Temperature.

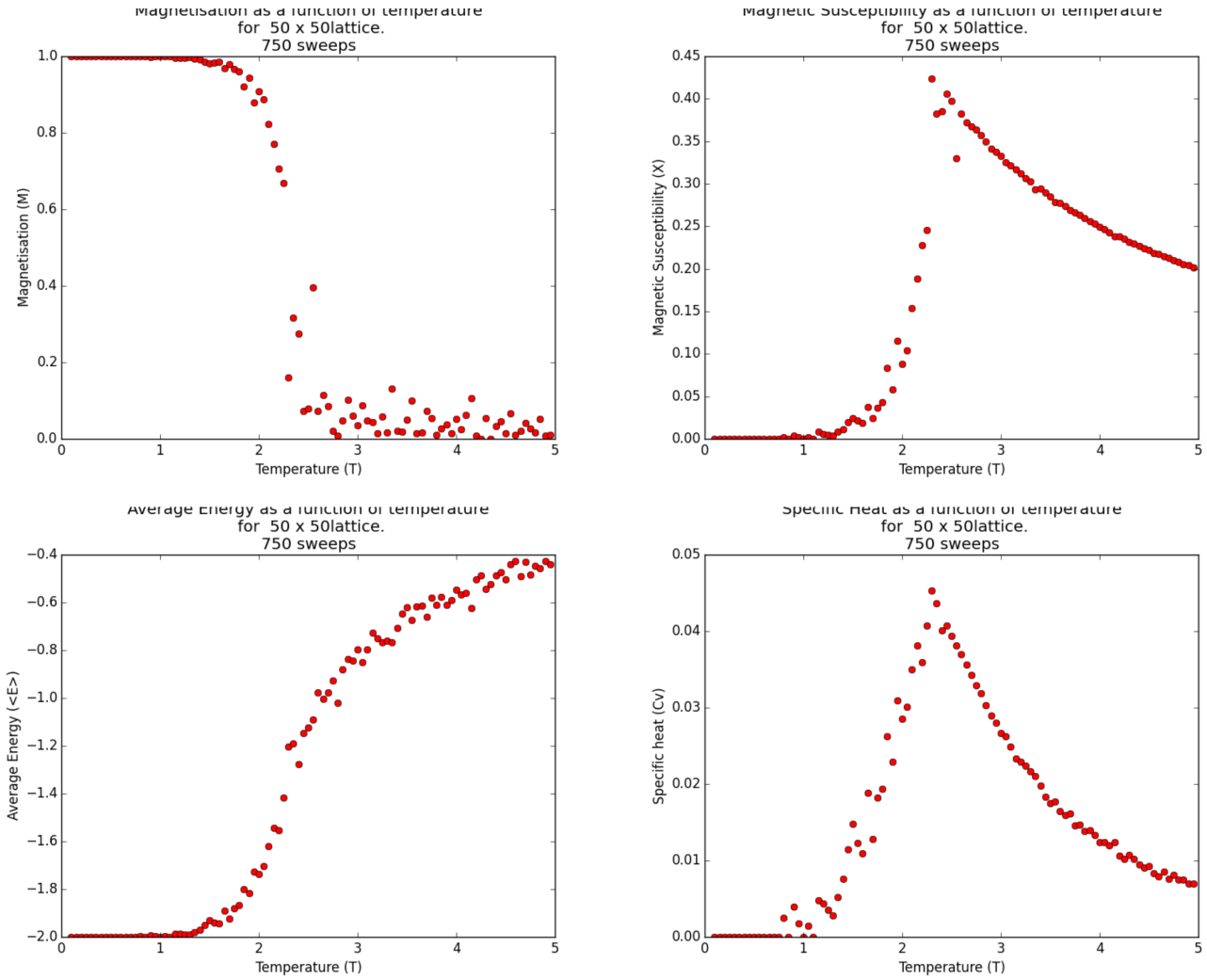

fig 5:Shows the observables as a function of Temperature for a 2-dimensional Ising Model

Above, fig 5 shows how the observables of the system relevant to this investigation vary with temperature. The results here are based upon simulations of a 50x50 lattice with 750 sweeps through the Metropolis Algorithm, and as can be see, these conditions allow for clear and relatively noiseless graphs.

Magnetisation: The first panel of fig 5 shows the average magnetisation of the lattice sites as a function of temperature. We note that initially the magnetisation remains constant at 1 for low temperatures (note the absolute value of magnetisation is taken). This suggests that at low temperatures the lattice remains homogeneous with regards to the spin of each lattice site, which is consistent with theory. As temperature rises, some sites begin to flips due to the additional energy in the system, and that at ≈ 2.4-$T - c$, the average value of magnetisation is 0.5 - neither ferro nor paramagnetic. This result is once again consist with theory. Finally we note that the magnetisation approaches 0 at higher temperatures.

We do however note that there is much fluctuation of the average magnetisation at these higher temperatures. This is due to the inherent fluctuating nature of paramagnetic materials, and were we to ignore the absolute values and allowed for values below zero, we would indeed observe that the average of these large fluctuation was 0.

Magnetic Susceptibility: [4] The magnetic susceptibility, as seen in the second panel of fig 5, behaves as predicted (for the most-part). The susceptibility begins at a constant 0 for low temperature, which is expected given that it is the derivative of the magnetisation which was constant over this temperature range. As the temperature approaches T_c the magnetic susceptibility begins to rise. The susceptibility of the lattice is not defined at T_c, exhibiting a discontinuity at this temperature, suggesting that the phase transition undergone is of second order. The value of susceptibility then declines post-T_c, although it must be noted that the graph does not decline as quickly as theory suggests; the theoretical model suggest that the susceptibility will be represented by a symmetric curve (similar to a delta function). We attribute this error to the presence of excessive noise in the late values of magnetisation (a slower rate of change in the slope!).

Average Energy: the average energy of the system behaves rather like the magnetisation of the system, but with a different sign; the average energy begins at a non-zero value and then tends towards 0, with a point of inflection at T_c. The graph is somewhat noisier than magnetisation however, given the probabilistic nature of the energy distrobution.

Specific Heat Capacity: given that energy behaves like magnetisation, it is therefore no surprise that specific heat behaves like susceptibility, given that it is the derivative of average energy. The data begins constantly 0 at low values of temperature, then increases as it approaches the Curie Temperature, with a discontinuity at T_c, and then declines symmetrically about this temperature. We note that the decline in specific heat behaves more like the predicted result than susceptibility, due to the lesser variation in Energy data due to noise. The discontinuity at T_c re-enforces the notion that the phase transition in a 2-dimensional lattice is of second order.

The Curie Temperature: graphically speaking, we can observe rather clearly that the Curie temperature of this system lies just below 2.5 J/K_b (although the units are somewhat arbitrary given that constants were all set to be equal to 1). The computationally returned value is 2.4±0.1 J/K_b. This result was obtained by evaluating the temperature index at which the maximum susceptibility occurred. The value compares well with a literature value of ≈ 2.3 J/K_b [Onsager, 1944]. The result could be improved further with both larger lattice sizes and increased sweeps, although this would be computationally quite demanding.

2.2 Pathogenic Diseases

Given the complexity of pathogenic diseases (with dependencies upon whether the pathogen is bacterial or viral, whether or not the pathogen has a capsid, whether or not the genetic information of the pathogen in DNA-based or RNA-based), models can often be difficult to construct. As such this model assumes that data about a species of pathogen has already been gathered, and normalised. For example: this model takes a virility rating of a pathogen (0-100), the immune rating of the population against this disease (0-100), the fatality rate of the disease (0-100 percent), and the response of the pathogen to anti-biotic treatment (0-100), as inputs. To illustrate the effectiveness of the model, we examine below 4 theoretical pathogens:

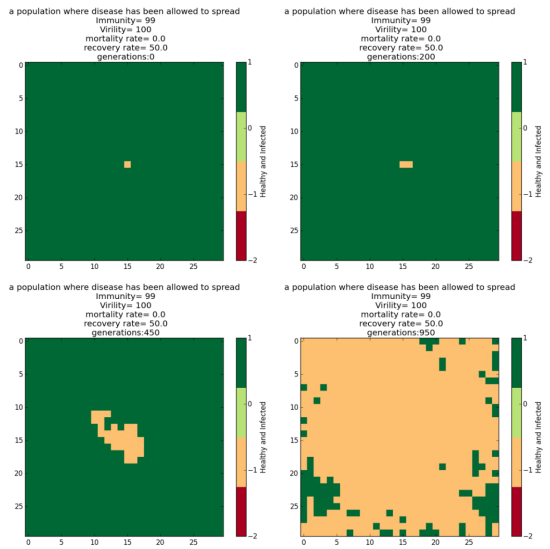

fig 6:Shows the spread of a virile, but non-fatal disease through a 30x30 population

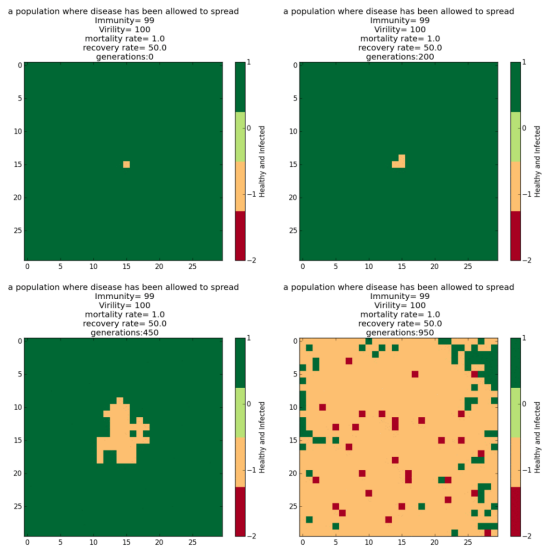

fig 7:Shows the spread of a virile, low fatality rate disease through a 30x30 population

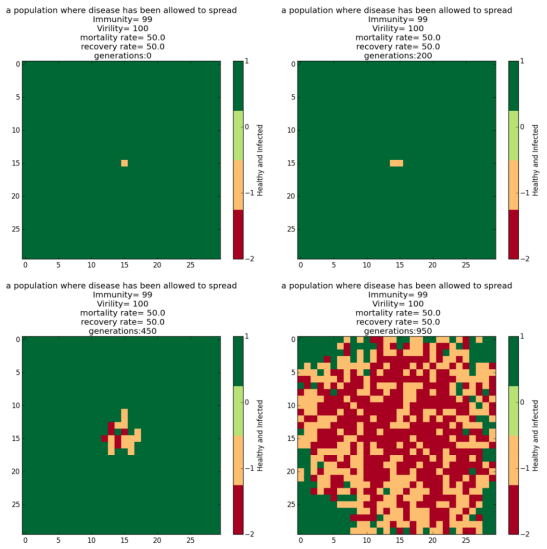

fig 8:Shows the spread of a virile, mid fatality rate disease through a 30x30 population

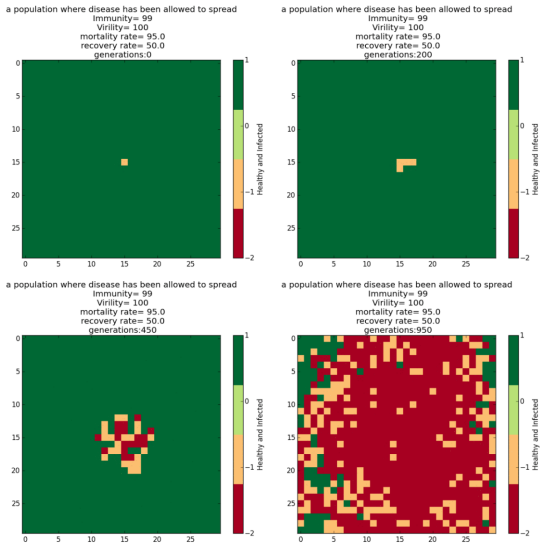

fig 9:Shows the spread of a virile, high fatality rate disease through a 30x30 population

Virility and Spread of Infection: we note from all theoretical pathogens that the spread of disease is non-linear, and non-identical (even for identical time-steps). This might seem trivial, but it is in fact incredibly important; the non-linearity of the growth is a result of the *natrual* growth mechanisms of living bacteria, which increases in number exponentially when left unchecked. We note that an exponential spread is present, DESPITE never having this spread rate explicitly specified. This means that given the correct set of processes, will describe models found in reality. The non-identical nature of identical timesteps is of import too- the 200th generation of different pathogens should not look the same in every case. This randomness can be attributed to the Markov Chain Monte Carlo methods which uses probability distributions to model results.

It was important to this model that a site can only become infected if it comes into contact with another (non-dead) infected site. As such a healthy population was created, and then a secondary function would add one seed of disease to each healthy population. The position of this seed can be randomised, but for visual clarity it has been placed at the center of the lattice.

Death: Modelling the death behaviour of the population was the most difficult section of this model to create, however success was achieved. The criteria for population death were as follows:

- Site can only die if already infected
- Infected site will only die provided the conditions of the mortality rate are met
- Dead sites DO NOT infect other sites
- Dead sites cannot change their state under *any* condition

The issues were however solved, using a combination of a series of conditional statements which had to be met before the death of the site could be allowed. The final condition proved troublesome; despite giving no protocol outside of what had already been specified, the dead were returning to life. This problem was finally rectified by adding an additional conditional statement to the flip conditions of the modified Metropolis algorithm, which required that the state not be equal to -2 for any further procedures to take effect.

The final product is a good model of death conditions, although this cannot be seen in 4 panels alone. Please visit the readme.md of

`www.github.com/cumminj1/ising-desktop`

to see animated gifs of the effect of each of these theoretical pathogens.

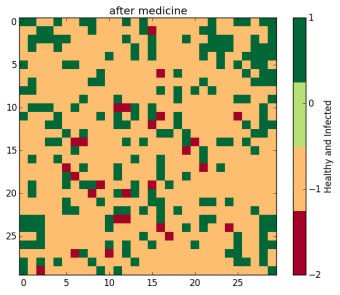

fig 10:Shows the effect a virile, low-fatality rate disease through a 30x30 population over an extended period of time

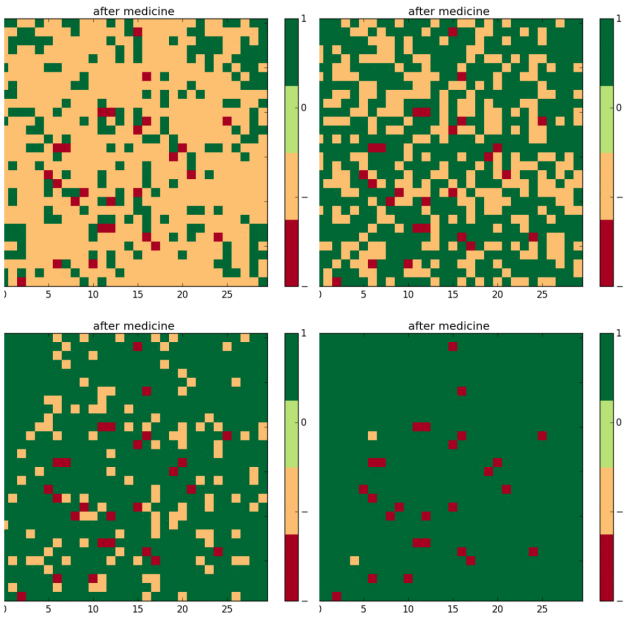

fig 11:Shows the recover of a population from a virile, low-fatality rate disease through a 30x30 population over an extended period of time, after the application of a middling efficiency antibiotic throughout the lattice

Recovery from Infection: A function was added to the code for the biological model which allowed a return to health from an infected (non-dead) state. The model assumes that the application of anti-biotics to a population is immediate and uniform, and the efficiency of the antibiotic is rated, with higher ratings treating the population more quickly. fig 10 shows a population which is mostly infected, prior to the application of anti-biotics. Fig 11 shows stages of recovery of that population after the application of the antibiotics.

The primary criteria for the recovery model have been met and can be observed in fig 11:

- The medicine's application does not account for the state of the nearest neighbours, unlike the infection.
- The medicine is incapable of resuscitating the dead

Both criteria were met, and so we can consider the recovery model a success.

3 Conclusions

The Ising spin lattice model was implemented successfully, with the findings that there is a phase transition in the lattice which occurs at a set temperature- the Curie Temperature, determined computfationally to be 2.4±0.1 J/Kb, which agrees well with the literature value of 2.26 J/Kb. The magnetisation, magnetic susceptibility, average site energy, and specific heat capacity were all modelled as a function of temperature. It was found that the magnetisation is 1 for temperatures below T_c, and approaches 0 above T_c, with the point of inflection of the curve at the Curie Temperature. The average energy also had an inflection point at this temperature, and the ground state energy was found to be -2, which is consistent with a J of 1. The magnetic susceptibility was found to have a discontinuity at the Curie Temperature, as did the specific heat. As such we conclude that the phase transition in a 2-D Ising Model is a second order transition.

The Ising Model and associated methods were then adapted to model the spread of virile, fatal disease throughout a population, and although the model is quite basic, it is very effective given the limited data it uses. The pathogenic spread can also be subject to antibiotics and recovery from illness can be modelled. Theoretically, with further development this model could be used to predict fatalities in a population due to a pathogen, regardless of type. The model is limited however, in that it assumes a homogeneous population with a homogeneous population density. In order for this model to become practically useful, these two assumptions should be rectified. Additionally, this model has great potential as a class with pathogens being objects. Then, given a library of pathogens the program could model almost any pathogenic disease outbreak.

Both models could be improved greatly in their precision by use of both a larger lattice and an increased number of sweeps, however this increases the computation time considerably, and so with all computational simulations, one must strike the balance between precision and computational time.

The pathogenic model of disease spread, despite its basic nature, provides us with an interesting insight into the development of diseases throughout a population and the speed at which that development takes place. The model is quite limited in its current form, however the model is promising and should be developed further into the future.

4 References

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