Using an SEIQR model to show how the spread of a viral infection affects a population

Introduction

The early days of COVID-19 demonstrated how rapidly viruses can spread and their impact on population density. The first group of patients were identified on the 21st December 2019 in the city of Wuhan, which had a population of 11 million people. By 25th January 2020, an estimated 75,815 individuals had been infected, exhibiting just how quickly an epidemic can spread, escalate, and cause a global health crisis (Qu et al., 2020). In this investigation, we'll explore the spread of COVID-19 through a population, to better understand its epidemiology and spread using the complex computational model SEIQR in Python. This stimulates, visualises and forecasts the spread of an epidemic and its effect on population density. This representation of spread is based on the more simplistic, probabilistic SIR model within a cellular automaton (CA) and demonstrates how populations can change states corresponding to the chain of infection where the spread is proliferated longitudinally. SIR only employs the use of three states: susceptible, infected and recovered, whereas the SEIQR model includes additional states to create a variant model which promises a stronger precision in simulation. Therefore, the SEIQR is the model better suited to investigate the effect of spread of a viral infection on population density and effect of lockdown rules in different stages. This report will focus on how the interaction range affects viral spread and how the timing of regional lockdown measures impacts the pattern of transmission, where both the intrinsic nature of infection spread, and governmental epidemic management are considered.

Methodology

Model Description

The SEIQR model is initialised first where the parameters of the grid, which includes grid size, population density, number of initial exposed and initial infected, are set. This is in the form of a cell lattice with a defined size L × L, which when applied in this context is the population size. Each cell represents an individual that's assigned one of six states, where state refers to a distinct stage in the chain of infection and changes based on the states of its neighbouring cells (**Table 1**).

State	***Description***	***Transition Probability***
Susceptible (S)	Uninfected, can become exposed	$P_e = 0.5$
Exposed (E)	In contact with infected, not yet infectious	$P_i = 0.5$ after $T_i = 7$ days
Infected (I)	Actively infectious	$P_q = 0.1$ for quarantine
Quarantined (Q)	Isolated, still infected	$P_r = 0.12$ for recovery
Removed (R)	Recovered with immunity, or passed away	After $T_r = 21$ days

Table 1 *SEIQR model states, their meanings and assigned probability values*

Each cell has set number of neighbours surrounding them, its Moore neighbourhood. As the timesteps progress, every cell with at least one infected neighbour becomes infected based on an assigned probability value and if this value is not reached, the individual remains susceptible but uninfected.

Parameter	***Value***	***Description***
L x L	100 × 100	Grid size, meaning total population = 10,000
D	1	Moore neighbourhood radius (subject to change)
P_e	0.5	Probability of exposure
P_i	0.5	Probability of infection
P_q	0.1	Probability of quarantine
P_r	0.12	Probability of recovery
T_i	7 days	Incubation period
T_q	7 days	Time in quarantine
T_r	21 days	Recovery period

Table 2 *Parameters, assigned values and explanations*

Making the code specific to that of a CA requires that the probabilities of the assigned dynamic states are defined as a numerical value (**Table 2**) and to allow for the cells to interact with its Moore neighbourhood, where this is denoted as the range of interaction, D. Within the code, P_e is the probability of exposure, P_i is the probability of infection, P_q is the probability of quarantined, P_r is the probability of recovery. The array is augmented by loop instructions so that it can be iterated, which allows for the states of cells to be updated in accordance with the state transition rules (**Table 3**) and run over a series of timesteps where for there is a period of incubation T_i, period of quarantine T_q and period of recovery T_r.

S → E:	If an individual has an adjacent exposed or infected neighbour, with P_e.
E → I:	After T_i, an exposed individual will become infected via a P_i.
I → Q or R:	**I → Q:** After the timesteps $T_i + T_q$, an infected person could need to quarantine according to probability P_q. **I → R:** After timesteps $T_i + T_r$, an infected individual may recover with probability P_r.
Q → R:	After T_r timesteps, quarantined individuals recover with probability P_r.

Table 3 *Allowed state transitions and rules*

The simulation is then run to allow these iterations to take place, each timestep follows 3 steps:

1. Compute the state proportions (fractions of S, E, I, Q, R).

At the beginning of each timestep, the proportions of individuals in each state are calculated. The number of cells in each state is counted and then normalised by the total population. These are the values we later refer to as fractions and they'll help to illustrate the disease progression.

2. Apply state transition rules to update the grid.

Each cell is assessed to see if it can change state according to the aforementioned state transition rules (Table 3). The spread of the disease is determined by the cell's interactions with its Moore neighbourhood, the range of interaction (Davies, 1995). Each susceptible individual evaluates its surroundings, counting the number of exposed and infected individuals.

3. Record data for analysis

A separate grid is used for time-tracking to record how long every cell remains in a specific state. This lets the parameters T_i, T_q, and T_r occur within the correct timeframe, only resetting when a transition to another state occurs. At the end of each timestep, the updated proportions of individuals in each state are stored, allowing us to create plots such as the line graphs and spatial simulation plots seen in this report.

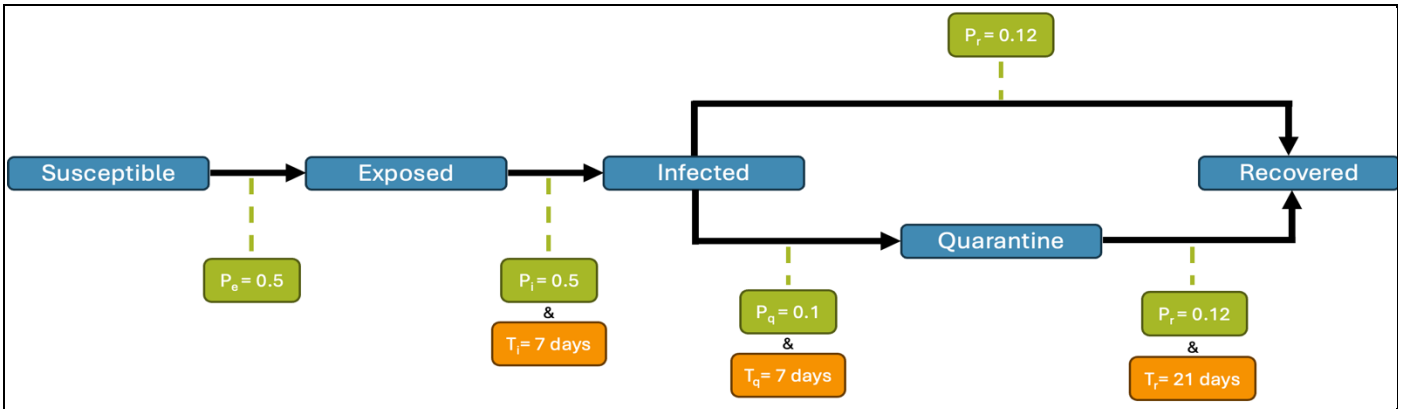

Figure 1a

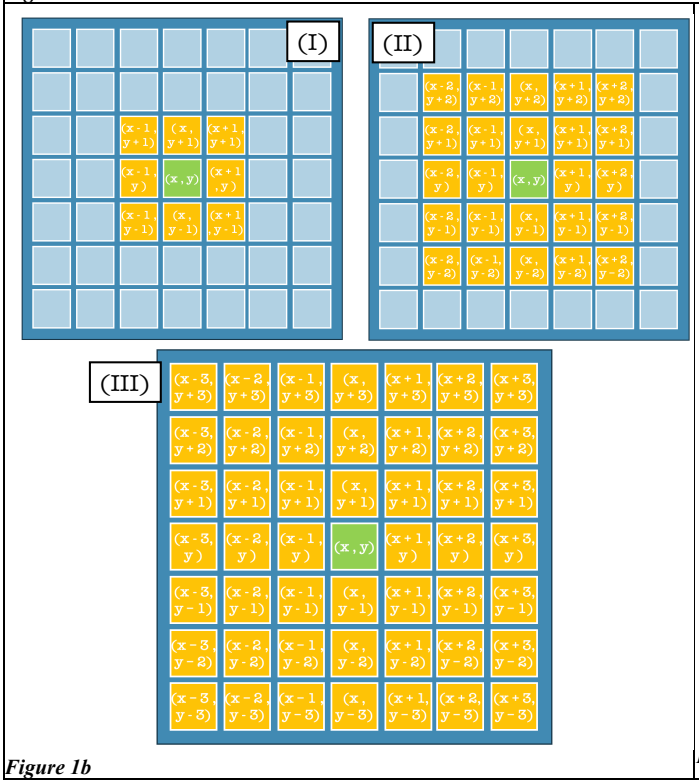

Figure 1b

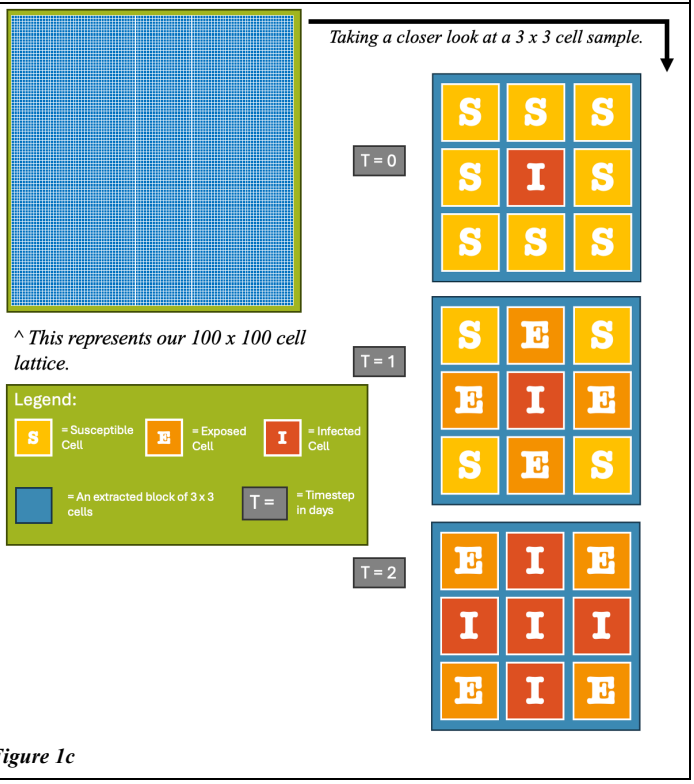

Figure 1c

Fig 1 a *SEIQR model visualisation* ***b*** *Representation of the Moore neighbourhood, where (I) has D = 1, (II) has D = 2 and (III) has D = 3.* ***c*** *A grid evolution sample, showing the behaviour of the viral spread in our model.*

Simulation and Results

We begin by creating a default simulation for the SEIQR model. Within this, the numerical values of the parameters follow those found in table 2, apart from D which acts as our dependent variable. We initially set D to 2 and later start to investigate the difference as D is changed to 1 and 3.

Figure 2.a shows the SEIQR epidemic trajectory over a 100 day from our default simulation. Initially, almost all individuals are susceptible. As the virus circulates, the exposed group rises, followed by a sharp increase in the infected fraction. Around day 15, Infected peaks at roughly 0.967, then gradually decreases as quarantine and recovery measures take effect. The number of quarantined individuals increase after the infection peak, while the recovered fraction accumulates steadily. From day 40 onward, the infected and quarantined fractions both decline sharply as new transmissions become negligible. Most individuals have by then either recovered or been removed from the infectious pool, so the disease's momentum is lost. Consequently, the curves for infected and quarantined remain near zero through to day 100, while the recovered fraction continues to rise and eventually stabilizes.

Initial Results:

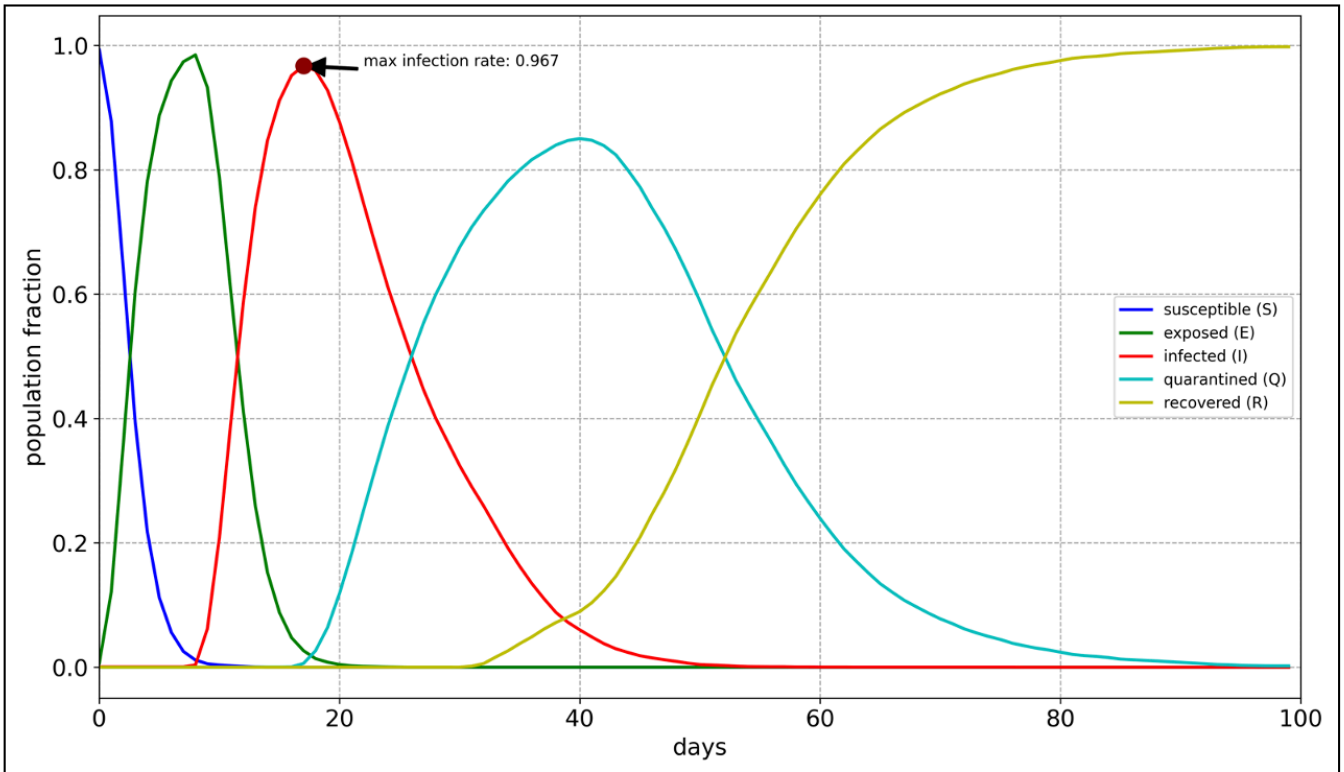

Figure 2a

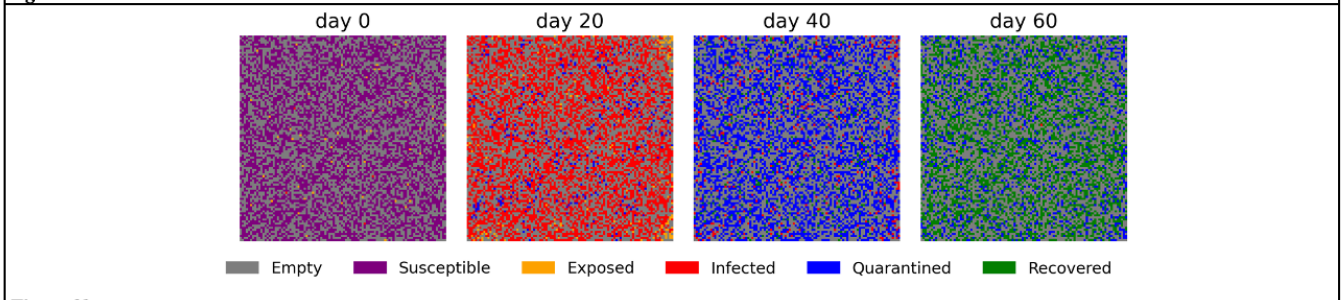

Figure 2b

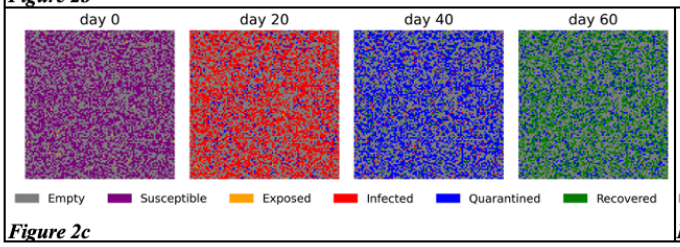

Figure 2c

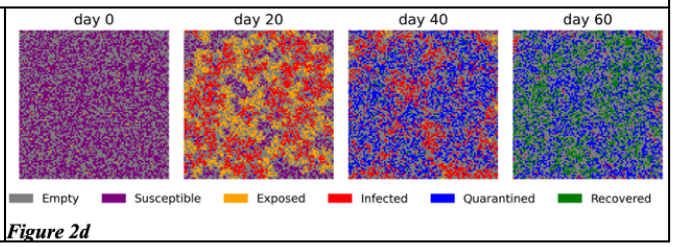

Figure 2d

Fig 2 a *Dynamics of the infection spread under default SEIQR parameters. Time evolution of the five subpopulations over 100 days, for D=2, P_e = 0.5, P_i = 0.5, P_q = 0.1, P_r = 0.1.* ***b*** *A model spatial spread simulation over 60 days, of the defult SEIQR model where D = 2.* ***c*** *A model spatial spread simulation of the SEIQR model where D is increased to 3 and all other variables are constant over 60 days.* ***d*** *A model spatial spread simulation of SEIQR model where D is decreased to 1 and all other variables are constant over 60 days.*

Effect of neighbourhood size on spatial spread dynamics
To explore how interaction range affects the speed and scale of virus spread, we adjusted the neighbourhood size while keeping all other variables constant. This parameter controls how many neighbouring individuals each person can interact with and therefore the potential to infect or become infected by the virus.

In the default case of D = 2 (**Fig 2.b**), the infection spreads steadily and reaches a peak of 0.967 (**Fig 2.a**) before being reduced by quarantine and recovery processes. **Fig 2.c** sees D = 3, meaning each individual has a higher interaction range, increasing spread of the virus. The infected regions grow more rapidly, and the outbreak transitions through the states in a shorter amount of time. In contrast, **Fig 2.d** shows that reducing the interaction range so D = 1 slows down the spread significantly. The infected areas expand more slowly so the peak of infection appears later.

Overall, the outbreak unfolds over more timesteps and lasts longer. These results suggest that a larger interaction range accelerates the spread of infection, while limiting contact allows for the rate of spread to decline—providing more time for intervention and response.

Effect of lockdown threshold
To study how lockdown rules effect the spread of a virus, the population area was divided into four blocks. Each block represents a distinct region of a city. When the number of infected people in one block reaches a certain threshold probability, that block goes into lockdown. This setup is used to compare the effects of early vs late intervention, specifically focused on block 1, where the virus starts, and block 4, which is the furthest, to see how the virus spreads within different locations. We present our observations on the spatiotemporal infection dynamics in a 100x100 lattice divided into four blocks, under two different lockdown threshold values: 1% and 10%, with all parameter's constant (**table 2**) and a neighbourhood value of 3. In this context, a lockdown threshold of 1% means that once any block observes 1% of its population actively infected, it goes into lockdown to restrict further spread. The blocks can still influence each other before crossing this threshold.

Fig 3.a shows that, under a 1% threshold, block 1 undergoes lockdown upon the infected fraction hitting 1%, then block-wide movement is sharply reduced, slowing further spread within block 1. This threshold is reached relatively early, only a small fraction of individuals become infected internally. However, the amount of spillover is limited if transmission connections form just before the lockdown fully takes effect. Consequently, smaller infected clusters (red patches) emerge near boundaries with the adjacent blocks. Either way, the overall infection remains spatially confined, as reflected by scattered red regions rather than a broad outbreak.

By contrast, in the scenario represented by **Fig 3.b** where the probability threshold is 10%, infection grows freely in block 1 until it hits this percentage of infected, generating dense red patches that extend into blocks 2 and 3 before any restrictions begin. While lockdown eventually prevents explosive growth inside block 1, a higher proportion of individuals are already infected. This leads to larger infected areas in blocks 2, 3, and 4, compared to the more controlled 1% scenario.

Figs 3.c and **3.d** compare infection trajectories in block 4 for the 1% and 10% thresholds respectively. Being further from the initial source that is block 1, block 4 is reached primarily via blocks 2 and 3. Under the 1% rule, early lockdown in block 1 greatly limits the influx of infected individuals into block 4; that block locks down quickly at 1%, so its epidemic curve stays well below the "no lockdown" baseline. Conversely, with a 10% trigger, each block experiences a higher internal outbreak prior to lockdown, sending larger waves of infection into block 4. By the time block 4 itself locks down, the infection curve has already crested much higher, settling above its 1% threshold counterpart.

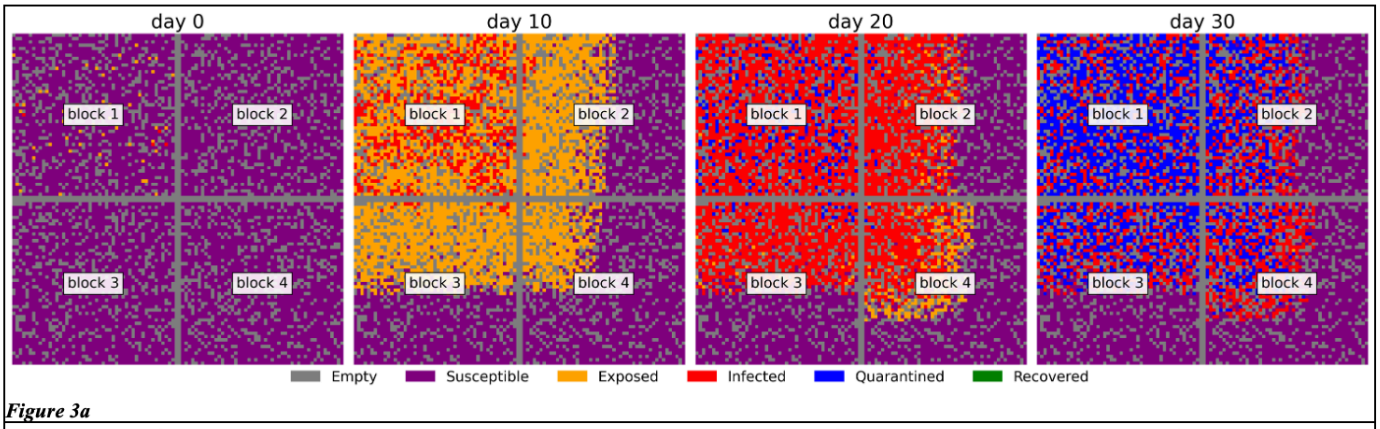

Figure 3a

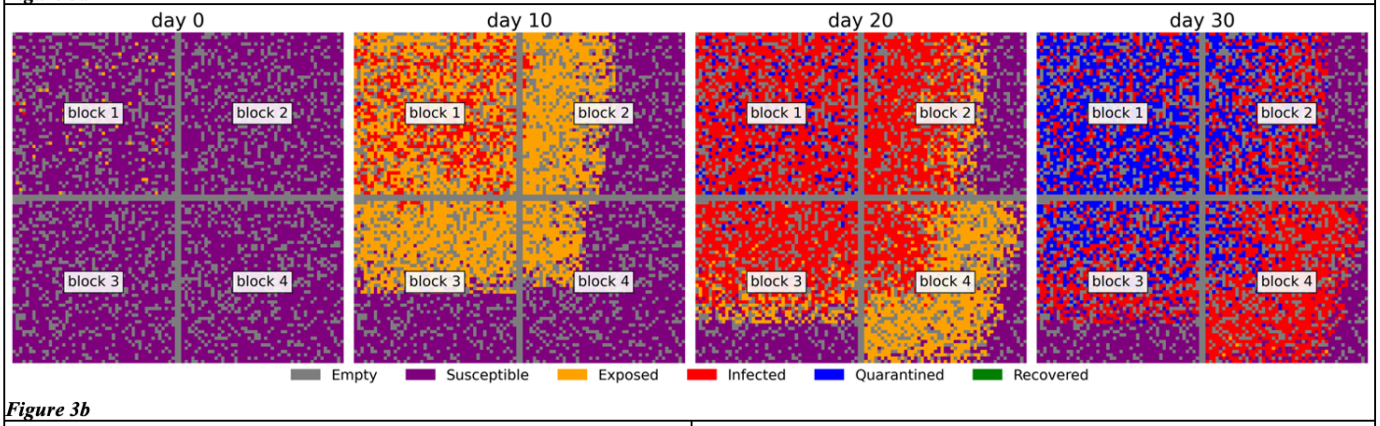

Figure 3b

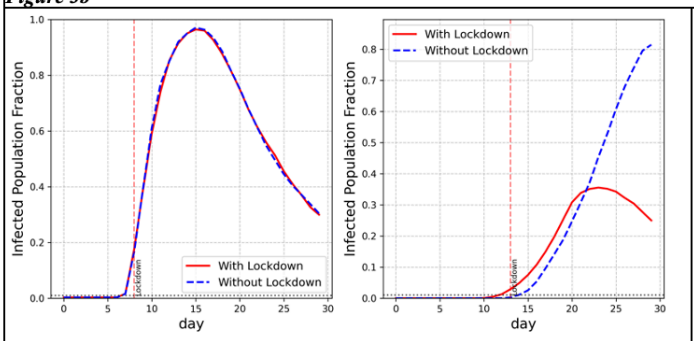

Figure 3c

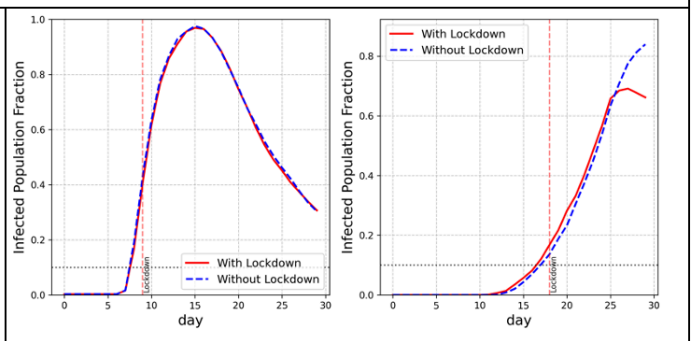

Figure 3d

Fig 3 a *A model spatial spread simulation of SEIQR model in a lattice divided into four blocks, where lockdown threshold is 1% and D =3 and all other variables are constant over 30 days.* ***b*** *A model spatial spread simulation of SEIQR model in a lattice divided into four blocks, where lockdown threshold is 10% and D =3 and all other variables are constant over 30 days.* ***c*** *Comparison of infection trajectories for Block 1 and Block 4, with and without lockdown, where lockdown threshold is 1%.* ***d*** *Comparison of infection trajectories for Block 1 and Block 4, with and without lockdown, where lockdown threshold is 10%.*

Discussion

Modelling epidemic spread across a spatial lattice allows for the complex transmission pathways shaped by both individual behaviours and policy interventions to be captured and these specifically effect the infection dynamics.
The initial varying of the lockdown threshold illustrates the importance of rapid intervention. A strict early lockdown, triggered at 1% of a block's population becoming infected, substantially limits internal transmission and reduces outward spread. By locking down block 1 (dense "downtown") early, few infections escape to adjacent blocks shown in **figure 3.a**. Consequently, more peripheral zones such as block 4 experience lower and delayed peaks. In contrast, a higher 10 % threshold postpones mobility restrictions until the block reaches a much larger infected fraction. Neighbouring areas face an influx of infections when the "downtown" block locks down late, propelling larger local epidemics even in relatively isolated blocks. Therefore, the timing of local lockdowns in pivotal transmission hubs can shape downstream contagion trajectories.

In addition, our experiments with varying the neighbourhood parameter reveals that broader contact radii accelerate virus proliferation. When D is high (**Fig 2.c**), each individual has a higher potential to infect more neighbours, causing a steeper and faster rise in infection levels. Reducing D (**Fig 2.d**), whether through physical distancing or other mobility constraints, slows disease transmission. This effect is beneficial for allocating time to deploy interventions such as testing, contact tracing, or localized lockdowns before infection causes an overwhelm on resources. Notably, restricting D alone does not eliminate spread but can mitigate the outbreaks peak and extend its duration, allowing health systems to respond more effectively.

Taken together, these results highlight how both macroscale policies (e.g. lockdown thresholds) and microscale behaviours (e.g. contact ranges) jointly determine epidemic outcomes. In a real city, a busy downtown which is represented by block 1 can rapidly seed infections into nearby neighbourhoods which are represented by blocks 2 and 3 (**Fig 3.a**). A real-world example of this can be observed in South Korea. In early 2022, the country experienced a sharp surge in COVID-19 cases, with daily new infections peaking at 600,000 around March (World Health Organization, 2024). This happened after delayed restrictions in central areas like Seoul, which allowed the virus to spread widely until strong measures were taken. As a result, even the outer regions with fewer direct links to the outbreak centre, like Daejeon, eventually faced large outbreaks. This supports our models' findings that a lockdown in a densely populated area can lead to widespread transmission across the entire system. Therefore, implementing strong lockdowns early in busy highly populated areas is imperative in order to prevent the epidemic from spreading outward.

Conversely, if lockdown is delayed until infection reaches 10%, the virus gains more opportunity to circulate, and distant areas like block 4, though poorly connected, may eventually experience a substantial outbreak. Such findings align with observed patterns in metropolitan regions, where late interventions in central districts lead to pronounced spillover effects in suburban or less-populated areas (**Fig 3.b**). From a policy standpoint, these simulations suggest that synchronizing early, stringent measures in dense hubs while simultaneously encouraging reduced contact rates can yield the great containment. While high testing coverage and quarantine are critical, regulating lockdown timing and physical distancing remains a robust strategy to minimize overall infections.

Limitations

As a mathematical model, SEIQR is subject to certain assumptions which limit the model's application to real life. We must assume that no birth nor death occur within the population as there would be too many factors to consider. Another assumption that needs to be made is that the cells are fixed in place and cannot travel between blocks, this is because the Moore neighbourhood is not large enough to infect a cell in another block and can only spread in one direction. Using this method also means that we assume that individuals can only affect those within a fixed spatial radius. Although this allows us to simplify the problem to apply our computational method, it doesn't account for the real-life complex dynamics of transmissions such as commuting and social gatherings. We also assume homogenous mixing within each neighbourhood; all individuals have the same likelihood of exposure, which again doesn't reflect real-life.

Additional limitation is that our framework simplifies the notion of "blocks," treating them as largely self-contained units with uniform population density. Many real-world regions exhibit diverse population clusters, complex transport networks, and transient populations, making transmission patterns more fluid. Furthermore, lockdown enforcement is another limitation, as within reality a lockdown isn't absolute. Many individuals require to move within the population, such as key workers.

Hence as a model, the SEIQR doesn't reflect the level of complexity found in reality, rather it serves as a simpler way to model how the spread of a viral infection affects a population, using only the states S, E, I, Q, and R.

Conclusion

In this project, we used an SEIQR model to simulate the spread of a viral infection across a spatially structured population. By adjusting key parameters such as interaction range and lockdown thresholds, we demonstrated how both individual-level behaviours and regional policies can significantly affect the outbreak's progression. Our results showed that earlier lockdowns and smaller neighbourhood interaction ranges help reduce infection peaks and limit spread into surrounding areas. Although our model simplifies many aspects of real-life disease dynamics, it provides a useful framework to test how timely interventions and movement restrictions can alter the course of an epidemic. These insights may help policymakers better understand the importance of early containment measures, especially in densely connected urban areas. Future work could improve realism by incorporating population mobility, asymptomatic transmission, and variable compliance with public health measures.

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