DYNAMIC SIMULATION OF A SEIQR-V EPIDEMIC MODEL BASED ON CELLULAR AUTOMATA

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Abstract. A SEIQR-V epidemic model, including the exposure period, is established based on cellular automata. Considerations are made for individual mobility and heterogeneity while introducing measures of vaccinating susceptible populations and quarantining infectious populations. Referencing the random walk cellular automata and extended Moore neighborhood theories, influenza A(H1N1) is used as example to create a dynamic simulation using Matlab software. The simulated results match real data released by the World Health Organization, indicating the model is valid and effective. On this basis, the effects of vaccination proportion and quarantine intensity on epidemic propagation are analogue simulated, obtaining their trends of influence and optimal control strategies are suggested.

1. Introduction. Outbreaks of epidemics have disastrous effects on the economy and livelihood of large populations, for which the most effective methods of prevention and control are predominantly vaccination and quarantine strategies (Jian Liu et al. 2014), thus, studies of effective vaccination and quarantine strategies are of great importance.

Cellular Automata (CA) (Von Neumann, 1966) is a model of dynamical systems, where space and time are discrete, and local interactions induce global variations. This is highly similar to the propagation of disease through transmission between individuals, thus CA has been used as an alternative method of modelling epidemics.

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G. Ch. Sirakoulis et al. (2000) has studied the effects of population movement and vaccination on epidemic propagation; C. Guan et al. (2011) has studied the propagation process under medical intervention; Some researchers considered the individual mobility and heterogeneity, and simulated process of epidemic propagation (Jiatai Gang et al. 2013; Xinxin Tan et al. 2014 ).

However, the majority of epidemic models employing Cellular Automata had not considered medicinal prevention during the exposure period to restrain propagations; and there are few studies on epidemic propagation with considerations of vaccination in conjunction with quarantine strategies. In actuality, general infectious disease such as parotitis, sexually transmitted diseases and influenza A(H1N1) all possess exposure period, in which infectiousness already exist (Sanling YUAN et al. 2001; Guangliang Li et al. 2013). This study will establish a SEIQR-V epidemic model including the exposure period based on CA principles, incorporating the effects of vaccinating the susceptible population and quarantine strategies for the infectious population on propagation. Digital software is used to create a dynamic analogue simulation, to study the effect of two measures and provide government departments with scientific foundations as basis for formulating optimized epidemic prevention and control strategies.

2. Establishment of Epidemic Model.

2.1. SEIQR-V Epidemic Model. It is assumed that during the propagation period, the studied population can be classified into seven states:

- Susceptible ($S$): healthy but non-immune and non-infectious;
- Exposure ($E$): infected, displays certain symptoms but has not been attacked by the disease with a certain amount of infectiousness;
- Vaccination ($V$): vaccinated but yet to produce antibodies, susceptible, non-infectious;
- Infection ($I$): displays disease symptoms with strong infectiousness;
- Quarantine ($Q$): infectious individuals under quarantine treatment, infectiousness nullified with loss of external contact;
- Recovery ($R$): successfully treated or vaccinated, retains significant immunity but can be susceptible after the immune cycle;
- Death ($D$): the subject is removed from the transmission process. Transition between the seven states can be seen in FIGURE 1.

When in state $S$, the individual is randomly mobile from daily activities, and will change into state $E$ by the probability of infection when in contact with infectious individuals; after a period of propagation, to restrain the spread of disease, susceptible individuals are vaccinated by probability $q$, transitioning into state $V$. 

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**Figure 1.** Propagation flow chart
In state $V$, before producing antibodies, the individual is still susceptible to disease when in contact with those infectious, changing into state $E$; of those vaccinated which are not infected after the vaccine’s response time $T_\nu$, a proportion of $\mu$ individuals will produce antibodies and change into state $R$, those that does not will change into state $S$.

In state $E$, to restrain propagation, the individuals are quarantined by probability $q_1$ within the exposed period $T_e$, changing into state $Q$; those not quarantined after $T_e$ will be diagnosed by probability $p_1$, changing into state $I$. Those uninfected will again become susceptible, changing into state $S$.

In state $I$, during course of the disease, infected individuals are quarantined by probability $q_2$, changing into state $Q$; of those not quarantined, after the period of disease, those uncured will die by casualty rate $d_1$, changing into state $D$, those cured will change into state $R$.

In state $Q$, within the quarantine period $T_r$, those will be cured by probability $\gamma$, changing into state $R$, those uncured die by casualty rate $d_2$, changing into state $D$.

In state $R$, the individual still possess immunity within the immunity period $T$ and will not be re-infected. After $T$ the individual becomes susceptible and changes into state $S$.

When in state $D$, the individual is removed from the transmission process due to death.

### 2.2. Neighborhood form.
A two dimensional cellular automaton (CA) usually takes 2 neighborhood forms: Von Neumann neighborhoods and Moore neighborhoods. However in real life scenarios, people interact with random population outside their social sphere of family, neighbors and workmates, such as visiting friends or travel vacations. This study references the random walk cellular automata (Xiaodong Duan et al. 2012; Jiatai Gang et al. 2013; Xinxin TAN et al. 2013) and the extended Moore neighborhood theories, to produce a neighborhood form for a cellular $C_{i,j}$ as shown in FIGURE 2.

Divide the space into $n$ by $n$, the individual random movement each time is by certain percentage, thus the number of mobile cellular each time is $\text{Num} = n \times n \times \text{percentage}$. Randomly sweep $\text{Num}$ amount of non-repeating cellular $C_{i,j}$ ($i, j$ the cellular co-ordinate, of which $i = 1, 2, \ldots, n, j = 1, 2, \ldots, n$) within the cellular space network, and produce $\text{Num}$ sets of discreet random number $(d_{i'}, d_{j'})$ (of which $|d_{i'}|, |d_{j'}| \leq n, i' = 1, 2, \ldots, \text{Num}, j' = 1, 2, \ldots, \text{Num}$), then randomly exchange cellular $C_{i+d_{i'}, j+d_{j'}}$ at $(i + d_{i'}, j + d_{j'})$ $(0 \leq i + d_{i'} \leq n, 0 \leq j + d_{j'} \leq n)$ with cellular $C_{i,j}$ to complete a random movement.

### 2.3. Probability of infectious contact.
Close range contact is a fundamental way for spreading infectious diseases. Generally, further the distance between two individuals, the smaller likelihood of being infected. This can be depicted through distance impact factors (Guan C et al. 2011; Xiaodong Duan et al. 2012; Jiatai Gang et al. 2013). Determine the distance impact factor of cellular $C_{m,n}$ on cellular $C_{i,j}$ and it is shown in equation (1).

$$D_{C_{i,j}, C_{m,n}} = \begin{cases} \frac{1}{|C_{i,j} - C_{m,n}|}, & C_{m,n} \in N_{C_{i,j}} \\ 0, & C_{m,n} \notin N_{C_{i,j}} \end{cases}$$  \hspace{1cm} (1)
Of which, $d_{C_{i,j}C_{m,n}}$ is the Euclidean distance between $C_{i,j}$ and $C_{m,n}$, $N_{C_{i,j}}$ is the neighborhood set of $C_{i,j}$.

Apart from distance affecting transmitting disease, the heterogeneity of susceptible individuals will also affect the transmission. Heterogeneity is $R_{C_{i,j}}$, the varying resistance to disease of susceptible individuals; infectious individuals have varying infectiousness $f_{C_{m,n}}$, both obey (0, 1) uniform distribution. As exposed individual infectiousness is less than the infected individual, the exposed infectiousness obey uniform distribution at (0, 0.5) while the infected obey uniform distribution at (0.5, 1).

In conclusion, the infectiousness of cellular $C_{m,n}$ on cellular $C_{i,j}$ is $f_{C_{i,j}C_{m,n}}$, thus the probability $P^{t}_{C_{i,j}C_{m,n}}$ of $C_{m,n}$ infecting neighboring cellular $C_{i,j}$ at time $t$ can be depicted in equation (2).

$$P^{t}_{C_{i,j}C_{m,n}} = \sqrt{D_{C_{i,j}C_{m,n}} \times f_{C_{i,j}C_{m,n}} \times (1 - R_{C_{i,j}})} , \quad (m,n) \neq (i,j) \quad (2)$$

This study takes the maximum infectiousness of all neighboring cellular of $C_{i,j}$ as $P^{t}_{C_{i,j}}$ (Jiatai Gang et al. 2013), the probability of infection by contact at time $t$, shown in equation (3).

$$P^{t}_{C_{i,j}} = \max_{(m,n) \neq (i,j)} \{ P^{t}_{C_{i,j}C_{m,n}} \} , \quad C_{m,n} \in N_{C_{i,j}} . \quad (3)$$

In simulation, when any susceptible cellular within a cellular space contacts a neighborhood where exposed or infected individuals exist, that cellular will, by the definition above, become an exposed individual by probability $P^{t}_{C_{i,j}}$. All cellular are synchronously updated at each simulated state, the state of $C_{i,j}$ at time $t + 1$ is dependent on the state of $C_{i,j}$ and its neighbor’s state at $t$.

3. **Medical intervention procedures and implementation.**

3.1. **Vaccination.** The objective of vaccination is to induce antibodies against the virus within the vaccinated individual, thus increasing immunity. In simulation, the effects of various proportions of vaccination on epidemic propagation are reflected by changing the value of vaccinated proportion $\nu$. Considering limitations of medical conditions and the population which is not suitable for the vaccine, such as those
with allergies or in early stages of pregnancy, the parameter of vaccinated proportion is $0 < \nu < 1$.

3.2. Quarantine. The objective of quarantine is to prevent transmission of disease through quarantine treatment from neighboring individuals. As exposed individuals also have a degree of infectiousness, while the infected are under quarantine, quarantine strategies should also be implemented for those in exposure.

The probability of quarantining infectious individuals is quarantine intensity, $q_1, q_2$ represents that of the exposed and infected individual respectively. In simulation, the effects of quarantine on epidemic propagation are reflected by changing the value of quarantine intensity. Considering the discrepancies of medical conditions, numbers of medical staff and limitations in medical supplies across different countries, parameters are set as $0 < q_1 < 1$ and $0 < q_2 < 1$.

4. Dynamic simulation and analysis of the results.

4.1. Simulation parameters. Simulation parameters vary for different infectious diseases, this study uses the case of Influenza A(H1N1).

As influenza A(H1N1) has a comparatively broad propagation scope, population during propagation is assumed constant (i.e. birth rate is equivalent to death rate). The immune cycle is set as $T = 365$ as infected individuals remain substantially immune after being cured and cases of relapse has not yet occurred. The size of cellular space is $N = 100 \times 100$, proportion of initial exposed individuals is $0.0001$, the remaining population are set as susceptible. Mobility ratio parameter $percentage = 0.001$. Other parameter definitions and values are as shown in TABLE 1.

| Parameter | Definition | Reasons | Unit | Value range | Value |
|-----------|------------|---------|------|-------------|-------|
| $T_e$     | exposure period | actual epidemic reports | day | 1 ~ 7 | 4 |
| $T_i$     | average course of disease | actual epidemic reports | day | 7 | 7 |
| $T_q$     | quarantine period | average course of disease + 48hrs observation | day | - | 9 |
| $T_v$     | vaccine response time | actual epidemic reports | day | 14 ~ 21 | 15 |
| $\gamma$  | cure rate in quarantine | actual epidemic reports | - | - | 99.5% |
| $p_1$     | probability of diagnose | rate of change from $E$ to $I$ | - | $14\% \sim 100\%$ | 25.0% |
| $d_1$     | infected casualty rate | actual epidemic reports | - | - | 1.0% |
| $d_2$     | quarantined casualty rate | reference 11 | - | $0.4\% \sim 0.5\%$ | 0.5% |
| $\mu$     | vaccine protection rate | actual epidemic reports | - | $70\% ~ 90\%$ | 85% |

4.2. Example. As the successful development of influenza A(H1N1) vaccine was in September 2009 and the selected data was prior to September, in analogue simulation the proportion of vaccination is $\nu = 0.00$. Initial quarantine intensity is set $q_1 = 0.45$, $q_2 = 0.65$. Based on the above parameters, Matlab is used to simulate curvature of change in cumulative deaths and infections. FIGURE 3(a) and 3(b) represent the simulated cumulative deaths and infections respectively, in comparison to the actual data of Influenza A(H1N1) in Mexico between May and mid-July (World Health Organization 2009).

It can be observed that the simulated results match that of Mexico, simulated data under $T test$ has shown no significant variance from real data at 95% confidence level, the correlation coefficient between simulated cumulative number of deaths and the real data is 0.978579, between simulated cumulative number of infections and the real data is 0.993441, indicating the model is valid and effective.
4.3. **Dynamic simulation.** In this section, the model parameters: vaccination proportion and quarantine intensity are discretely adjusted and tested, to analyze the patterns of each parameter’s influence on epidemic propagation, and to find an effective optimal control strategy.

**Simulation analysis of variation in vaccination proportion**

This study assumes that the influenza A(H1N1) vaccine has already been developed, and it is transported to the temporary vaccination point after 4 days. As influenza A(H1N1) vaccines are in single doses, assuming the temporary vaccination point’s basic conditions (e.g. waiting room, consulting room, vaccination room) and number of vaccination staff is enough to complete one vaccination. Set proportion $\nu$ to be vaccinated on the day 5 of epidemic propagation and quarantine intensity $q_1 = 0.45$, $q_2 = 0.65$, for other parameters reference TABLE 1. FIGURE 4 shows the change in cumulative number of infections against time when $\nu$ is 0.00, 0.15, 0.25, 0.55, 0.60 and 0.65.

**Figure 4.** Cumulative number of infections against time under various proportions of vaccination
It can be observed in FIGURE 4 that as proportion of those vaccinated increase, the cumulated number of infections decrease, and the speed of reduction slows down. In comparison to $\nu = 0.55$, when $\nu = 0.60$ the number of infected individuals stabilized 3 days in advance, and the cumulative number of infections decreased by 35.34%; in comparison to $\nu = 0.60$, when $\nu = 0.65$ the number of infected individuals stabilized 7 days in advance, however, the cumulative number of infections decreased by only 8.00%. In reality, when restraining the epidemic propagation, not only the reduction of infected individuals but also cost in implementing vaccinations. As $\nu$, the vaccination proportion of susceptible individuals, reach optimal value of 0.60, both propagation and resources can be significantly restrained.

As Cellular Automata can visually depict the propagation process, this study took dynamic analogue simulation images of the propagation of influenza A(H1N1) both with no vaccination and $\nu = 0.60$, at when $t = 12$ days, 30 days and 60 days respectively, as shown in FIGURE 5.

Two groups of images corresponding to the cases of no vaccination and vaccination, respectively. By comparing image sets in FIGURE 5, we can visually deduce that vaccination can effectively control the propagation of disease. On day 12, the vaccinated individuals have not yet produced antibodies, therefore the situation is almost the same as that in which individuals were non-vaccinated. on day 30, vaccinated individuals are immune due to production of antibodies and change into state $R$, cumulative number of infections and casualties decreased by 103 people and 1 people respectively; as time pass, the number of infected gradually decrease. On day 60, epidemic propagation has stabilized, at this point the cumulative number of infections and casualties decreased by 424 people and 69 people respectively.

**Simulation analysis of variation in quarantine intensity**
After outbreaks of general infectious disease, the time taken to develop and produce vaccines usually takes 28 to 35 days, but for new epidemics such as the 2009 outbreak of influenza A(H1N1), the development process took as long as 3 months. In circumstances where transmission cannot be controlled through vaccination, extent of propagation may develop. To investigate the effects of vaccinating exposed individuals on epidemic propagation, this study sets $\nu = 0.00$, $q_2 = 0.65$, for other parameters refer to TABLE 1. Variations in cumulative number of infections due to changing the quarantine intensity parameter $q_1$ by using dynamic simulation of Matlab can be seen in FIGURE 6.

![Figure 6](image)

**Figure 6.** Changes in cumulative number of infection under various quarantine intensity

From FIGURE 6, it can be observed: as $q_1$ increase, cumulative number of infected individuals decreases at a decelerated pace. For quarantine intensities $q_1 = 0.60$, $q_1 = 0.65$ and $q_1 = 0.70$, comparing the former two, the number of infected individuals stabilized 7 days in advance, and the cumulative number of infected individuals decreased by 38.50%; the latter two only decreased by 3.03%, and stability was delayed by 6 days. When the quarantine intensity is greater than $q_1 = 0.65$, increase of medical facilities and staff does not cause significant change to the number of infected individuals, but expends significant labor and resources. This shows that when the quarantine intensity of exposed individuals $q_1$ approaches optimal value 0.65, the epidemic propagation can be most effectively controlled.

Considering their significant infectiousness, infected individuals should be quarantined to prevent further propagation. To study the effect of propagation under various quarantine intensities of infected individuals, first set $\nu = 0.00$, refer to table 1 for other parameters, and change quarantine intensity value $q_2$ under $q_1 = 0.45$ and $q_1 = 0.65$, as seen in FIGURE 7.

FIGURE 7 shows: As $q_2$ increase, cumulative number of infected individuals decreases significantly. In FIGURE 7(a), when $q_1 = 0.45$, in comparing $q_2 = 0.65$, $q_2 = 0.70$ and $q_2 = 0.75$, the cumulative number of infected individuals has decreased by 10.68% and 5.02% respectively. In FIGURE 7(b), when $q_1 = 0.65$, in comparing $q_2 = 0.70$, $q_2 = 0.75$ and $q_2 = 0.80$, the cumulative number of infected
The cumulative number of infections at various quarantine intensities has decreased by 10.34% and 3.84% respectively. In this study it is found when \( q_2 \) is greater than 0.75, increase of medical facilities and staff creates strain on labor and resources without causing significant change to the number of infected individuals. Thus when the quarantine intensity of infected individuals \( q_2 \) approaches optimal value 0.75, the epidemic propagation can be most effectively controlled.

**Integrated simulation analysis of vaccination and quarantine**

From the above, while keeping other initial parameters constant and proportion of vaccinated individuals at optimum, the initial and optimum values of \( q_1 \) (quarantine intensity of exposed individuals) and \( q_2 \) (quarantine intensity of infected individuals) are configured as the following four permutations:

1. \( \nu = 0.60, q_1 = 0.45, q_2 = 0.65; \)
2. \( \nu = 0.60, q_1 = 0.65, q_2 = 0.65; \)
3. \( \nu = 0.60, q_1 = 0.45, q_2 = 0.75; \)
4. \( \nu = 0.60, q_1 = 0.65, q_2 = 0.75. \)

Simulated using Matlab software, changes in cumulative number of infected individuals under different permutations are shown in FIGURE 8.

FIGURE 8 shows: Comparing (1) and (2), infected individuals dropped from 75 to 21 people, decreasing by 72.00%; comparing (1) and (3), infected individuals decreased from 75 to 65 people, by 25.33%. This shows under optimal vaccination strategies, independently increasing quarantine intensity of exposed individuals is more effective than increasing that of infected individuals. Comparing (2) and (4), infected individuals dropped from 21 to 19 people, by 9.52%. This shows that taking optimal values for all three parameters does not create significant change to cumulative numbers of infected individuals in comparison to taking optimum value of vaccination proportion and quarantine intensity of exposed individuals.

From above, considering that the quarantine process costs more than that of vaccination, when change in cumulative numbers of infected individuals are the same, the optimal choice for controlling epidemic propagation is to increase proportion of vaccinated individuals and quarantine intensity of exposed individuals, which restrains propagation most effectively while cutting down medical labor and resources.
5. Conclusion. This paper considers the state of exposure, quarantine and vaccination in addition to the classic epidemic propagation model based on Cellular Automata (CA) to establish a SEIQR-V model in which there is an infectious exposure period. A more realistic neighborhood mode is achieved by referencing the random walk cellular automata and extended Moore neighborhood theories. Heterogeneity, the varying individual resistance to disease, varying levels of individual infectiousness and lesser infectiousness of the exposed individual in comparison to the infected individual, is also considered to define the individual’s probability of infection by contact at any given time.

This paper also considers the measures of vaccinating susceptible population and quarantining the infectious population (exposed and infected individuals). Using influenza A(H1N1) as example in combination with its propagation characteristics, a dynamic simulation is created with Matlab software to calculate the cumulative numbers of infected individuals and deaths. The simulated results match actual epidemic reports released by the World Health Organization of the Mexican influenza A(H1N1) outbreak during the period May from mid-July 2009. Simulated results by T testing has no significant difference from real data at 95% confidence lever, with correlation coefficient reaching 0.978579 and 0.993441, indicating the model is valid and effective.

In analogue simulation, one of three parameters out of proportion of vaccinated individuals, quarantine intensity of exposed individuals and quarantine intensity of infected individuals are modified while the remaining two stays constant to find its effect on propagation and effective optimal control strategies. The results indicate when the proportion of vaccinated susceptible individuals approach 0.60, the control of propagation is most effective; when the population is unvaccinated and quarantine intensity of infected individuals remains constant, the optimal quarantine intensity for exposed individuals $q_1$ is 0.65; optimal quarantine intensity of infected individuals $q_2$ is 0.75 when quarantine intensity of incubate individuals remains constant.
Simultaneously, this study integrates the effects of vaccination and quarantine on propagation by changing quarantine intensities $q_1$ and $q_2$ when vaccination proportion is at optimal value and other initial parameters remains constant. The results show: under optimal strategies, independently increasing quarantine intensity in exposed individuals is more effective than increasing that of infected individuals; while there is no significant change in number of infected individuals when all three parameters are optimized in comparison to optimizing only the vaccination proportion and quarantine intensity of exposed individuals. Therefore, in consideration of quarantine costing higher than vaccination, the most effective and resource efficient propagation control is increasing vaccination proportion and quarantine intensity of exposed individuals. These findings can provide government departments with scientific foundations as basis for formulating optimized epidemic prevention and control strategies.

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