Multi-Agents Computational Experiment on Twice-Linked Model of Unconventional Emergencies Evolution Mechanism

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***Abstract*—Based on the cellular automaton principle and multi-agents theory of complex systems, the generation and the evolutionary mechanism of unconventional emergencies have been studied and the twice-linked model of he evolutionary mechanism of unconventional emergencies has been established with calculating experiments being carried out in this study. The conclusion is reached that the internal causes of unconventional incident is the accumulation of internal energy. Evolution process of twice-linked systems is a process that the constantly-accumulated energy in the promoted system is first suddenly released, and then the internal energy in the linkage system is stimulated and also released in a large-scale. These two outbreaks are almost at the same time, and the eruption of the linkage system can be more than twice as powerful as that of promoted system. The linkage effect of unconventional emergencies could be totally prevented only when no transfer possibility or 100% ceasing ability in linkage system or no network space density occur.**

***Index Terms*—Unconventional Emergencies, Twice-Linked Evolution Mechanism, Cellular Automaton, Multi-Agents, Promotion System, Linkage System**

I. INTRODUCTION

Unconventional emergency refers to the emergency of which the premonition is insufficient, with obvious complex characteristics and potential secondary derivative hazards, and that has destructive efforts and the routine supervisor modes cannot deal with [1, 2], such as 911, SARS, the Indian Ocean tsunami, the New Oreland hurricane, the Wenchuan Earthquake, the H1N1 influenza, the east Japan earthquake, etc. These events brought great hazards to our human society and have been highly valued all over the world. Consequently, the issue on how to carry on the management of manage unconventional emergency has become an extraordinary challenging subject [3-7].

The research on the evolution mechanism of unconventional emergencies has become the one of the core basic science issues of emergency management [1-4].There are a quantity research work of the evolution law of emergencies, but most of them aim at some specific disaster events, few comprehensively considered the multiple emergencies and the interaction among them. The comprehensive study about the formation mechanism of the social system vulnerability, how the conventional disasters evolve into serious crisis events, and the behavior law of individual, colony, and organization in emergency management has just started [8].

Through multi-agents technology, based on criticality theory of complex system, this paper composed the cellular automaton model of emergency evolution mechanism and imitated the generation and the evolutionary process of the unconventional emergencies. On the basis of this evolution characteristics, it constructed active immunization defense model and analyze the appearance of group phenomenon by observing individuals, then discover the essence for evolution of the emergency and provide scientific evidences for preventing and controlling the emergency.

II. EVOLUTION MODEL OF UNCONVENTIONAL EMERGENCIES

A. The Classification of Emergencies Evolution

The evolution process of unconventional emergencies can be divided into one time criticality evolution and repeatedly criticalities evolution. One time criticality evolution refers to the situation that the breakout of a certain emergency will not spread to another emergency, while repeatedly criticalities evolution refers to a certain emergency causes another one's breakout, even a series of emergencies' occurrence. For example, on 11th March 2011, the east Japan earthquake not only brought disaster to humans by itself, but also caused F island nuclear crisis.

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This paper mainly discusses the subject of twice-linked evolution.

B. Twice-linked Evolution Model

This paper constructs emergency evolution model on cellular automata theory. Cellular Automata is a common tool used to describe the complex phenomena in nature and is initially applied to simulate the special self-replication phenomenon of life system. In the early 1950s, the founder of the modern computer, Von Neuman, proposed the rudiment of Cellular Automata in order to simulate the cell's self-replication during the biological development process [9]. In 1970, J. H. Conway at the University of Cambridge designed a computer game, the game of life [10, 11], which was a Cellular Automata model equipped with the ability to generate dynamic pictures and dynamic structure, attracting many scientists' interests. Later, Wolfram carried on detailed and thorough studies on Cellular Automata [12-14].

Suppose two sorts of cells in the system constitute their subsystem respectively, A is the promoted system while B is the linkage system, both of which have the same space structure and size of the grid. There are cell interactions in the subsystems and inside the subsystems themselves. The interaction condition is cell A_i and cell B_i which are in the same coordinate position but in different grid cells. When A_i is in outbreak state, it can stimulate B_i into latent state or outbreak state. The evolution of B_i is caused by the transferring efforts of A_i or other cells of the same sort B_i.

As Cellular Automata is able to evolve emerging events based on simple rules, when the evolution rules are set, the model in this paper can judge whether the "emerging"-unconventional emergency happens in the system according to the system's state and can analyze its evolution rules.

(1) System Construction

Suppose that this A system consists of M amount cells and B system M_2 amount cells while they are in the space whose number of the grids for M = world$X$ × world$Y$, world$X$ and world$Y$ are boundaries of the space.

$M_2 \le M$, the amount of M_2 is decided by space density ρ of system B, ρ is input value within [0, 1]. Every cell in the space has a coordinate (xPos, yPos) and Cell A_i fill up total grid units of its gird which are originally all in stable state except one random cell with gene $N_{(A)}$ whose first bit equals to 1 and others equal to 0 as latent state. Cell B_i scatter randomly in the grid in a stable state.

(2) State Parameter

Cell A_i is dynamic subsystem with a certain energy, whose state parameters are gene $N_{(A)}$, energy value $E_{(A)}$ and current state. $N_{(Ai)}$ is a binary digits with certain length $L_{(A)}$. 0 and 1 represent stability and disorder respectively. Suppose that the length of all Cell A_i's gene is $L_{(A)}$.

$$N_{(A)}=\begin{cases}00000\ldots\ldots000 & \text{stable state } (state=1)\\ 11110\ldots\ldots000 & \text{latent state } (state=2)\\ 11111\ldots\ldots111 & \text{outbreak } (state=3)\end{cases} \quad (1)$$

$E_{(A)}$ is decided by the number of 1 in attribute $N_{(A)}$, in which n represents 0 or 1 in $N_{(A)}$. Assuming energy value of each bit is 0.1, the minimum value of $E_{(A)}$ is 0, the maximum is $0.1L_{(A)}$.

$$E_{(A)}=\sum(0.1\ n) \qquad n=0 \text{ or } 1 \quad (2)$$

Total energy of system A as:

$$G=\sum E_{(Ai)}=\sum\sum(0.1n) \quad (3)$$

The state parameters of cell B_i are almost the same as cell A_i except for alterable length $L_{(B)}$ of gene and energy transfer e.

Energy transfer e is accumulated energy transferred from A_i to B_i in every cycle depicted as an accumulation parameter.

$$e_{(t+1)}=\begin{cases}e_t+(m(ab)-(L_{(B)}-v))\times 0.1 & m(ab)\ge L_{(B)}-v>0\\ e_t+m(ab)\times 0.1 & v=L_{(B)}\\ e_t+0 & m(ab)<L_{(B)}-v\end{cases} \quad (4)$$

$m(ab)=E_{(A)}\times c\times 10$, is the intensity of cell A_i interacting with cell B_i which have the same coordinates in every cycle. c is the transfer rate of state change in B_i between 0 and 1 incurred by cell A_i. 10 indicate the unit force, while. v means the number of 1 in cell B_i's gene N.

Total cumulative energy of system B as:

$$G_2=\sum E_{(Bi)}=\sum\sum(0.1n)_)+\sum e \quad (5)$$

(3) Effect Transfer

A_i connects with A_j in system A by a node. The node transfer operator is noted as C_{ij}, while No inverse operator (namely, supposed a single connection is one-sided). Among them m_0 is input value which suggests changed bits in cell gene $N_{(A)}$ within one cycle.

$$C_{ij}=\begin{cases}m_0 \text{ bits 0 changed to 1 in } N_{(A)} & state_{(Aj)}=1\\ \text{No change} & state_{(Aj)}=2 \text{ or } 3\end{cases} \quad (6)$$

$C_{i\,i}$=add k bits 0 changed to 1 in $N_{(A)}$
state$_{(Ai)}$=2 and C_{ji}=No change (7)

The one-sided transfer function of cellular in system B is in common with that in system A. The effect function between cell A_i and B_i is: the transfer operator which the cell A_i transfer to B_i which is in the same coordinate location with A is C_{2ii}. The impedance value that the cell B product to prevent cell A happening the effect transfer is called z. Its range is [0, 1], which is input value, meaning its range from 0 to1. 0 means No ability to prevent, and 1 means prevent completely, namely, never trigger any state changes of cell B. The random number is called a, whose scope is [0, 1], and it is used to product random simulation results comparing with z.

$$C_{2ii}=\begin{cases}m_2 \text{ bits 0 changed to 1 in } N_{(Bi)} & \alpha\le 1-z\\ \text{No change} & \alpha>1-z\end{cases} \quad (8)$$

$$m_2 = \begin{cases} L_{(B)}- v & m(ab)\geq L_{(B)}- v>0 \\ 0 & v = L_{(B)} \\ m(ab) & m(ab)< L_{(B)}- v \end{cases} \quad (9)$$

m_2 is the change of bits of cell gene $N_{(Bi)}$ in cell B_i if the cell A_i has made effect transfer to B_i.

(4) Neighborhood

The neighborhood of System A is built up by all cells connected with cell A_i. The cells work only in neighborhood shown in table 1, namely, one cell is adjacent to the other four cells.

TABLE I.

NEIGHBORHOOD

		△		
	△	A_i	△	
		△		

The neighborhood of system B is the same with that of system A.

The neighborhood which cell A_i to B_i is the grid cell of cell B_i whose location is corresponding with the coordinates of A_i. Shown in Figure 1, the corresponding coordinate of A_i may be B_i or nothing.

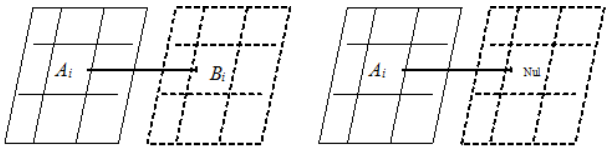


Figure 1. Neighborhood A in correspondence with B.

(5) Cell Evolvement

Equations the internal evolvement of system A can be described as follows: the process can be divided into T periods, in which the system will evolve according to the effect of the transferring operator. Cell A_i in the t+1 step is determined by the elements which contains state parameter (X_{it}), the sum of input to A_i from the other cells (H_{it}), increment of time (Δt) and evolvement formulation (F_i), the p_0 is input value, which presents the possibility of energy distribution, ranging from 0 to 1, β *is* random a*mong* 0 to Max($p_0E_{(Ai)}$), which is used to produce the simulated results, formulation of the system is:

$$X_{i(t+1)} = F_i\,(X_{it}, H_{it}, \Delta t) = \begin{cases} \text{No change} & \beta > p_0E_{(Ai)} \\ \sum C_{ij} & \beta \leq p_0E_{(Ai)} \end{cases} \quad (10)$$

Symbols the evolvement of system B contains the interaction among cell B_i and that from cell A_i to cell B_i. The formulation of formal evolvement is the same as that 6 expressed above. The mode of the later one is: a single cell A_i will activate cell B_i when the A_i is under eruption, then the energy level ($E_{(Ai)}$) of A_i will turn to 0 once B_i is excited. The running cycle of cell last for T and the system will develop according to the effect of the transferring operator.

Cell B_i in the t+1 step is determined by the elements which contains state parameter (X_{2it}), the sum of input to B_i from the other cells (H_{2it}), increment of time (Δt) *and* evolvement formulation (F_{2i}), formulation of the system is:

$$X_{2i(t+1)}=F_{2i}\,(X_{2i\,t}, H_{2i\,t}, \Delta t) = \begin{cases} \text{Formula (6)} & \text{(energy form } B_i\text{)} \\ \sum C_{2ii} & \text{(activated by } A_i\text{)} \end{cases} \quad (11)$$

(6) Evolvement Control

Accomplishment of Evolvement Control is achieved through controlling and pre-defining the state parameters, internal and external operator from A_i and B_i, the internal structure of evolvement can be seen in Figure 2 (the state of A_1 and A_6 is latent state, the rest are stable state), the depiction of activation between A_i and B_i is seen as Figure 3.

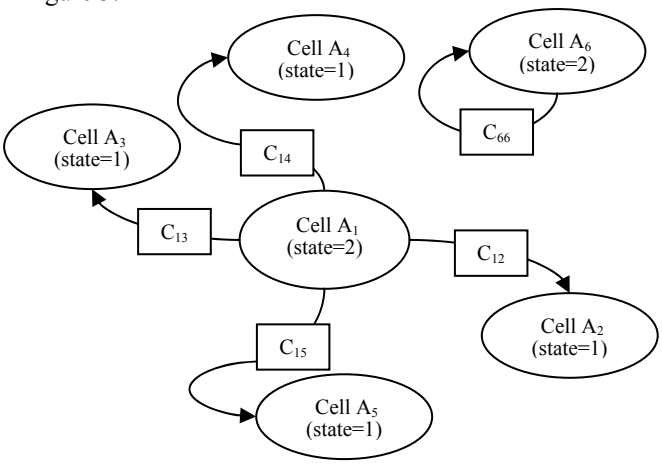


Figure 2. One time criticality evolution mode of cellular automaton (in the case of cell A_i).

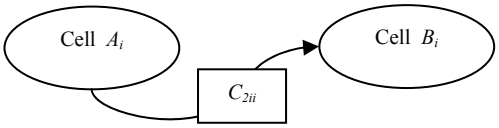


Figure 3. Twice-linked evolution mode of cellular automaton.

III. CONSTRUCTION AND REALIZATION OF MULTI-AGENTS MODEL OF THE UNCONVENTIONAL EMERGENCIES

The model is based on Multi-Agents in swarm 2.2. The term "Agent" was put forward by Minsky [15] in *Society of Mind* which refers to self-adaptive and self-autonomous entities, in order to cognize and simulate human intelligence behaviors, as follows:

Agent:= $\{S_m, Ag_i\}$

S_m refers to the internal state of Agent while Ag_i is its function or interaction with the external.

This model includes three subjects: Agent cell A_i and cell B_i, the model subject agent-model swarm that is used for constructing the interaction rule objects of cells, the environmental subject agent-environment swarm that is

the grid space where the cells are. The basic parameters settings are:

M = world$X$×world$Y$=21×21=441

$L_{(A)}$=15

Then, according to the transfer operators in the model in this paper, the simulation algorithm was constructed to carry the energy between cell A_i and cell B_i and in them and to simulate the process of emergencies outbreaks, and the process of simulation is shown in Figure 4.

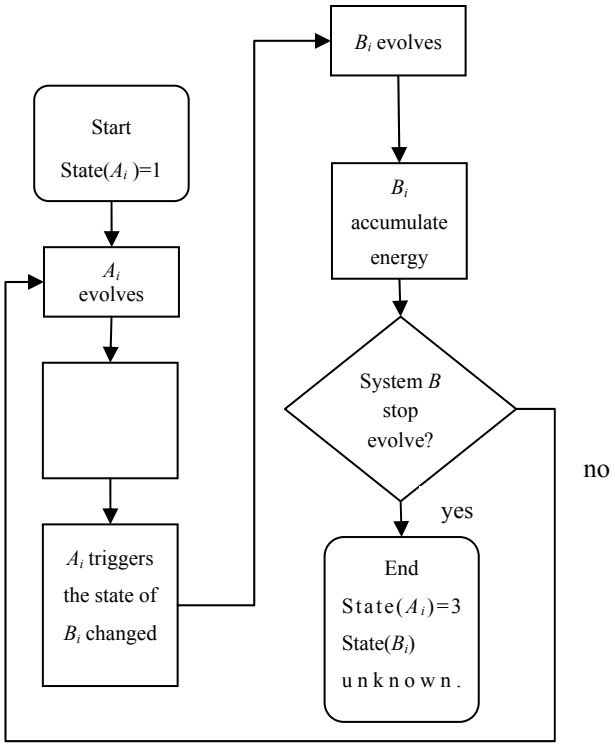


Figure 4. The computational experiment flow diagram.

IV. Computational Experiment Results Of The Unconventional Emergencies

Assume that the transfer function m_0=1 in system A, transfer possibility p_0=1, and the transfer function in system B m_1=1 and p_1=1. Set the critical factors caused emergency in system B as resistance z, the transfer function from A_i to B_i as $m(ab)$ =$E_{(Ai)}$×c×10 and the transfer function rate as c , and the space density of system B as ρ. If we consider the intensity of emergency in system B, we should add gene length $L_{(B)}$ of B_i. So four variables can be regulated to observe different results in this computational experiment, that is z, ρ, c, $L_{(B)}$.

The simulation results are the curves of three kinds of observations——latent value, outbreak value and destructive force (the total accumulated energy), d and d_2 mean the latent values of system A and B separately, f and f_2 mean the outbreak values of system A and B, and G and G_2 mean the destructive forces of system A and B. The change of system B is always later than system A.

(1) No. 1 experiment: input values are ρ=0.8, z=0.5, c=0.5, $L_{(B)}$ =15

These input values stand vulnerable to linkage emergency level. Total number of cells in system B is world$X$×world$Y$×ρ=21×21×0.8≈353. Internal total energy of system A reach M (as amount of cells)×$L_{(A)}$×0.1=441×15×0.1=661.5, while internal total energy of system B equals (amount of cells)×$L_{(B)}$×0.1=353×15×0.1=529.5, which is shown in Figure 5.

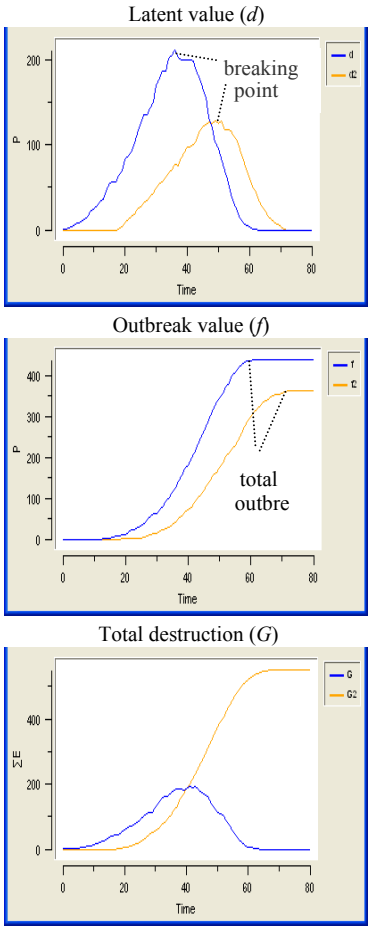


Figure 5. Contrast evolution of experiment result 1: ρ=0.8, z =0.5, c=0.5, $L_{(B)}$=15.

Analysis of latent values from the simulation results, maximum latent value of system A is 210, while maximum of system B is 120 where time point reaches 50 cycles which are about 15 cycles later than that of system A.

Outbreak values of the cell A and cell B in the simulation results show curves of each are approximate, which means interval time of the linkage outbreak is extremely short, as associated system can make rapid reaction so as to cause internal energy outbreaks with higher energy than owns.

The destruction in simulation results show that system A turns to be in disorder at 40 cycles with maximum destruction force as 200. The internal reason for above is that partial energy of cell A_i transfer to cell B_i, which means system B outbreaks by gaining partial energy from cell A_i after system A outbreaks. When comprehensive outbreaks of system A occurs, destruction force in system B reaches maximum 600, which is higher than the total internal energy in system B as 529.5.

(2) No.1 series of experiments: input values are $\rho \in [0, 100\%]$, step length is 1%, z=0.5, c=0.5, $L_{(B)}$ =15

The purpose of No.1 series of experiment is to study the effect of the change of spatial density of System B made to the results of evolution. Series of experiments show that the outbreak of system B is greatly influenced by the spatial density of system B. The greater the value is, the bigger outbreak value and destruction force of System B will be (as shown in Figure 5). If the value is 1, the maximum of outbreak value can achieve 400 and eventually equal the value of system A with the damage to maximum 700. If the value is small (if ρ=0.1, its simulated result as shown in Figure 6), it will not cause linkage emergency. If value is 0, no emergency at all.

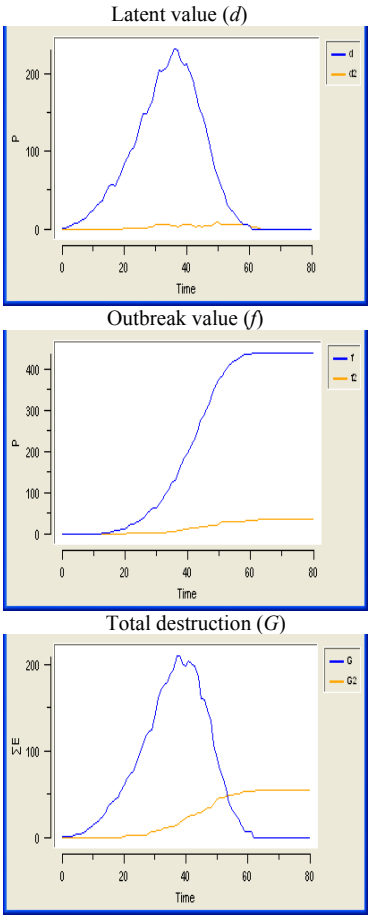


Figure 6. Contrast evolution of experiment result 2: ρ=0.1, z =0.5, c = 0.5, $L_{(B)}$ = 15.

Compared with Figure 6 and Figure 5, there is a big change in evolution curves. In the system B, latent value, outbreak value and destruction force have been reduced significantly. That is mainly because the lower density directly reduce the number of cells and the total energy within system B. Owing to the low energy, latent fluctuation of the value is not clear, that is, there is basically no outbreak point in system B so that no secondary linkage emergency consequently .

(3) No.2 Series of experiments: input values are ρ=0.8, $z \in [0,100\%]$, step length is 1%, c=0.5, $L_{(B)}$ =15

The purpose is to see the effect or influence resistance make on evolvement in the second experiment. The influence on the evolvement has a little connection with the effect cell B_i makes on A_i while resistance increase will affect system B positively. For example, when the resistance starts to rise from 0.10, the maximum eruption and destruction of system B declines slowly from 360 and 550 until resistance increases to 0.90. The result can be seen from chart 7 and chart 8, the impact is not obvious until the resistance charged, then it can deter the eruption of the series of events.

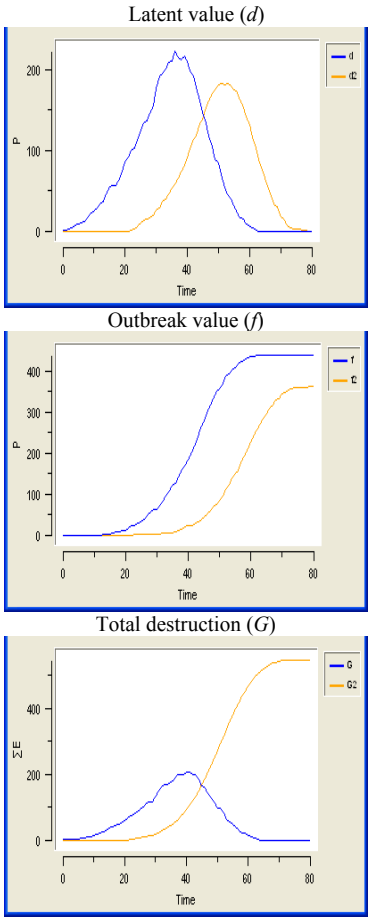
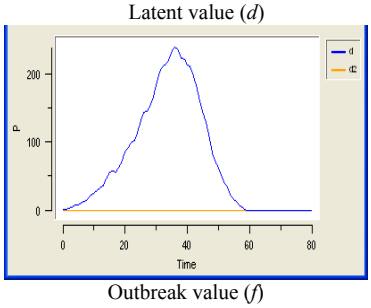
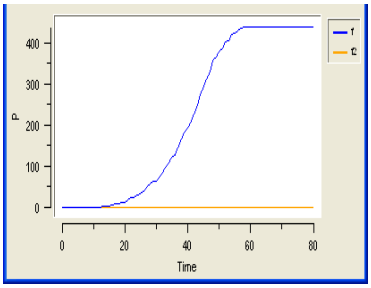


Figure 7. Contrast evolution of experiment result 3: ρ=0.8, z =0.9, c = 0.5, $L_{(B)}$ = 15.

We can see from the chart 7, the curve of latent value in system B fluctuates greatly, however, the other curves resemble structure in chart 5 where latent value of system B rises from 120 to 190, meanwhile, the eruptional point and destructive point changes a little.





Total destruction (G)

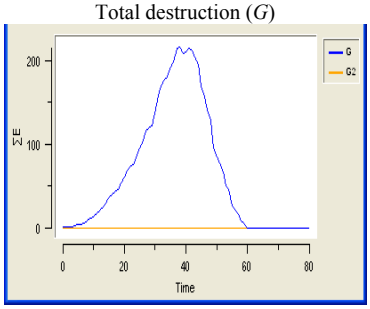
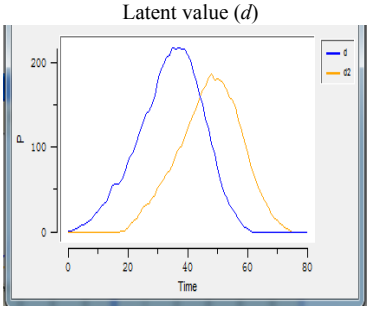


Figure 8. Contrast evolution of experiment result 4: ρ=0.8, z =1.0, c=0.5, $L_{(B)}$=15.

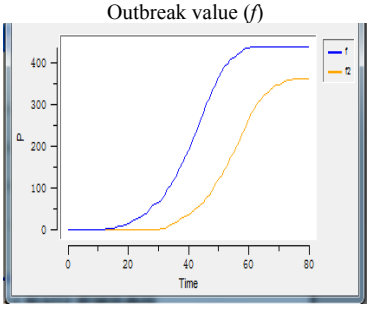
(4) No. 3 series of experiments: input values are ρ=0.8, z=0.5, $c\in$[0, 100%], step length is 1%, $L_{(B)}$ =15

No.3 series of experiments is for the purpose of investigating the impacts that the change of transfer function rate from cell A_i to cell B_j has on the results of evolution. The result shows that the impacts are various and prominent. When the transfer function rate becomes lower, the impact that system A has on system B in per period is reduced, and the evolution process of system B slows down, leading to the outbreak number increase in system B, the less power in total destruction transferred from system A and the stronger destruction from itself as shown in Figure 9 (c=0.1). When c becomes bigger, the impact is opposite to the above. The Figure 10 is the result when c is 0.9.

Latent value (d)



Outbreak value (f)



Total destruction (G)

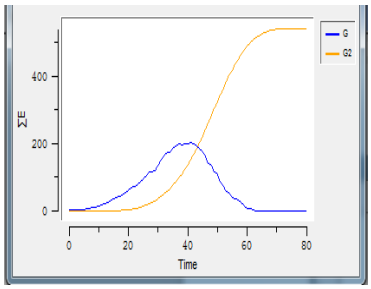
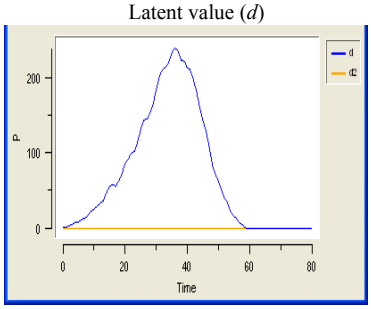
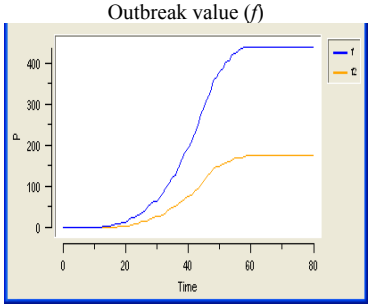


Figure 9. Contrast evolution of experiment result 5: ρ=0.8, z =0.5, c=0.1, $L_{(B)}$=15.

Latent value (d)



Outbreak value (f)



Total destruction (G)

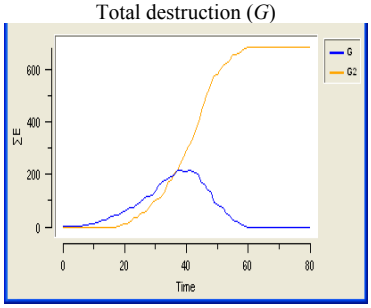


Figure 10. Contrast evolution of experiment result 6: ρ=0.8, z =0.5, c=0.9, $L_{(B)}$=15.

The improvement of the transfer function rate from cell A_i to B_i reduces the outbreak numbers of linkage system, but leads to stronger destruction caused by out broken cell B_i that means outbreak intensity increase dramatically.

(5) No.4 series of experiment: input values are ρ=0.8, z=0.5, c=0.5, $L_{(B)}\in$[1,25], step length is 1

The purpose of the No.4 series of experiments is to observe affect to the events evolution when the B cell gene length changed. B cell gene length can have great influence to the event, when length is lesser, each cell B will subsequently evolve into the state of outbreak, so the latent value of system B is always 0 and the outbreak value quickly into maximum value, even to a maximum from beginning and no longer change through the end,

which is shown in Figure 11 (length is 1) with limited destructive force of the B system. Until the length to 15 (as shown in Figure 5) could lead to the widespread outbreaks of emergency.

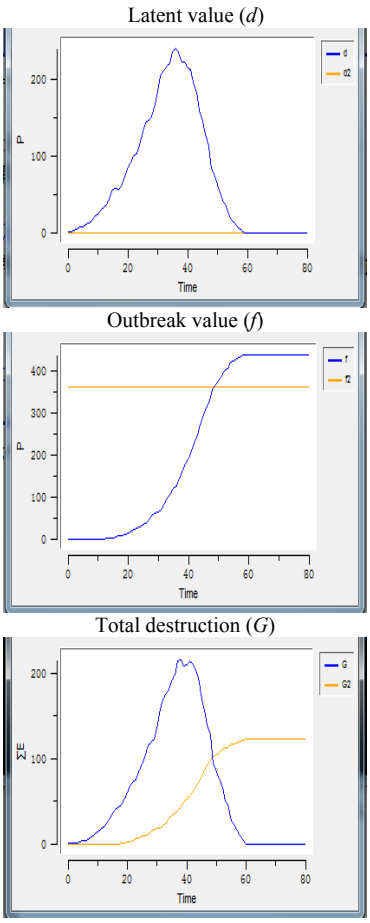
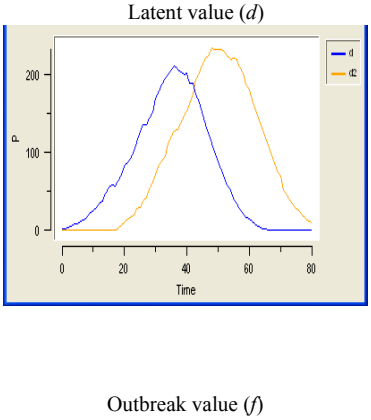


Figure 11. Contrast evolution of experiment result 7: ρ=0.8, z =0.5, c=0.5, $L_{(B)}$=1.

To increase the gene length of B cell continuously will lengthen the time for internal transfer effect, increase internal total energy, and make the system more latent B until the latent value more than the peak of A system. And the energy of cell B_i from the A system transfer becomes larger too, so the system B could have the extremely strong destructive force in the end, far more than A system's as shown in Figure 12 ($L_{(B)}$=25).



Outbreak value (f)

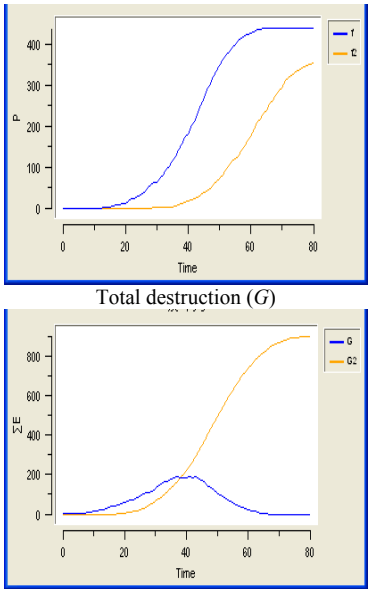


Figure 12. Contrast evolution of experiment result 8: ρ=0.8, z =0.5, c=0.5, $L_{(B)}$=25.

Compared with Figure 5, Figure 12 shows the system of B has great changes in latent value. We can see from the curve that in the 50th the latent value reaches maximum more than the A system, achieving 200 above, which means when the B system breaks out, the latent individual significantly increased, but the system breakout time was not changed. From the destructive curve, destructive force could be extremely as strong as 900.

V. Conclusion

The generation time of the twice-linked evolution is greatly shortened, the second emergency occurred almost at the same time with the first emergency, if there are two or more associated systems, it may linked to an emergency, the happening of the subsequent linkage event is difficult to control, the most effective prevention can only be blocking the association, if the association can't be blocked, the resistance of the associated system must be strengthened and the internal density or the internal transfer should be decreased.

The specific conclusions are summarized as follows:

(1) The time of the linkage evolution of emergency is greatly reduced from the critical state to the outbreak state, almost simultaneously following the outbreak of system A, which immediately leads to the outbreak of the system B. In the extreme cases, not even through the critical state it directly enters the outbreak of the state, so it is difficult to change the evolutionary process if action to stop the system B is taken until the outbreak of the system A.

(2) z, ρ, c, $L_{(B)}$ respectively represent the prevention ability of linkage system, linkage system space density, the transfer function rate of linkage evolution and internal gene length of linkage system, these four factors have influence on the evolution of events, among these factors ρ has the largest sensitivity, the higher of the value, the greater the harm of linkage evolution's occurrence, and vice versa. Secondly c and $L_{(B)}$ has greater sensitivity; but

the sensitivity of z is minimal, it has limited impact on the linkage evolution.

(3) To strengthen the preventing capacity of the linkage system to the transferring system cannot prevent the number of individuals and the explosive power of the outbreak of the linkage system effectively, however, the more effective measure is to reduce the individual density of the linkage system, such as to avoid more buildings in the seismic zone, etc.

(4) The transfer function rate of linkage evolution has a greater impact on the system evolution, which mainly reflects that it can strengthen the individual capacity strength of linkage system, with fewer individuals producing greater capacity. Since no change of the overall destructive power and no obvious critical state in the linkage system, it is more difficult to prevent.

(5) The internal gene length increase of linkage system $L_{(B)}$ will lead to that of the internal energy and internal transfer function time, thus the individuals number increase in a latent state. Namely, the more latent individuals are discovered, if we can take early prevention, situation will be improved; otherwise, the trend of the outbreak of the linkage system cannot be bettered effectively.

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