以小世界社會網路為基礎的流行病模擬模型

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摘要

本研究提出一個新的小世界模型—具有分身點概念的細胞自動機，以進行流行病模擬。利用分身點的概念，可以直觀地描述在真實社會中，個體藉著交通工具長距離移動與每天在固定地點活動的行為，例如：家庭、工作場合、捷運站、或餐廳。本研究從社會學與流行病學兩個層面依次說明如何將分身點的概念應用在傳統細胞自動機上。然後以實驗分析證明本模型具有社會特質，也就是能夠表達出小世界的特性（低分隔度與高群聚度）。之後，與傳統流行病模型所推導出的 R0 再傳染參數做比較，說明本模型亦能夠展現 R_0 參數的特性，證明本模型可以正確地套用在流行病學的模擬上。最後以 2003 年在全世界爆發的 SARS 為例證，證明本模型適合可以用來做流行病模擬。

A Small-World Model for Epidemic Simulation

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Abstract

The author validates a new small world model consisting of cellular automata with mirror identities of daily-contact social networks for purposes of epidemiological simulations. The mirror identity concept was established to integrate human long-distance movement and daily visits to fixed locations into the model. After showing that the model is capable of displaying small-world effects (i.e., low degree of separation and relatively high degree of clustering) on a societal level, we offer proof of its ability to display R_0 properties, which are considered central to epidemiological studies. A simulation of the 2003 SARS outbreak serves as our primary example of how the proposed model functions.

Keywords: Epidemic network model, Small-world model, Epidemic simulation, Mobile individual problem, Epidemic model validation

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最後，感謝我的父母，在我自小求學至今，不斷地給我支持、鼓勵和關懷。我以此論文，表達我對你們的謝意，我想並不足夠，但是，希望你們開心與驕傲。

目錄

圖目錄

1. Introduction

Increasingly sophisticated computer and mathematical models are being used to simulate medical epidemics that result from a broad range of complex factors that include underlying social networks, contagious disease properties, and public health policies. The literature is filled with efforts to find a suitable model for simulating epidemics of various types and sizes and for predicting the movement of new epidemics as soon as their identification is confirmed. If such a model can be created and refined, it would make it possible for public health authorities to choose the best policies for containing the spread of a new virus such as SARS[1, 2], or for targeting specific human behaviors that affect the spread of sexually transmitted diseases[3].

Societal and epidemiological knowledge must be gathered before any simulation of an epidemic can be performed. On a societal level, we know that individuals come into contact with other neighbors in ways that can be measured geographically. Neighbor clusters include family members, classmates, fellow commuters (on trains and buses), and diners in restaurants. They often form triadic closures[4] (See Appendix1.1)within a dynamic process leading to high levels of local clustering that increase the speed of epidemic transmission. Furthermore, epidemics are now being

spread via the long-distance movement of individuals via airplanes, trains, and cars.

In their efforts to determine who is most likely to become infected, epidemiologists focus on target populations (e.g., livestock or the elderly), transmission routes (e.g., airborne, contact with skin, sexual contact), temporal properties, and the presence and location of heterogeneous individuals. An example of temporal segmentation that must be considered when designing a simulation is incubation—between 4 and 6 days for SARS, up to 10 years for HIV. Individual heterogeneity is measured in terms of health status, immunity, and recovery rates.

We have found that the compartmental models used in classic epidemiological research make it very easy to study state transformations and changing numbers of individuals in various populations [5]. Furthermore, these models can be used to calculate R0—the basic case reproduction number that is considered a core epidemiological parameter[6]. However, they fail to accurately assess social phenomena resulting from human interactions. Many of the network models that use sites to represent individuals and links to represent their interactions fail to acknowledge long-distance movement. We established our mirror identity concept to solve the problem of the mobile individual. A complete description will be offered in the two sections that follow (See Appendix1.2).

Compartmental epidemiological models such as SIR, SIS, and SEIR focus on the

transformation of global parameters in a society and use stochastic processing and differential equations[7] to derive R0 numbers. Furthermore, they use continuous segments of populations rather than individuals. Population compartments are needed if the models are to generate data that correspond to the statistical data collected by epidemiologists. These models may be suitable for simulating the transmission dynamics of infectious diseases, but since they consider all individuals with the same status as a single population, they overlook the fact that social phenomena result from interactions between individuals. In the case of infectious diseases, epidemics are the consequences of numerous contacts, interactions, and transmissions. Traditional models ignore the spatial, interactive, and local properties of social issues, which makes it difficult to study public health policies as control measures. On the other hand, these models are useful for collecting, verifying, and comparing disease parameters; we therefore considered them useful in terms of improving the accuracy of our proposed model.

Simple network models emphasize relations, links, and interactions among individuals and assume that they remain independent of other links or interactions [5]. Several models simulate epidemics on two-dimensional lattices, with lattice points representing heterogeneous individuals and links representing interactions among them [8]. Two-dimensional lattices make it easier to describe neighborhoods and their

local properties, but they still fail to address the issue of long-distance movement. Despite many attempts, researchers have yet to solve this particular problem.

The rest of this paper is organized as follows: in the next section we will define the mirror identity concept and our overall model in societal and epidemiological contexts. In the third section we will offer proof that our model is a small world model by calculating average vertex-vertex distances and clustering coefficients. In the fourth section we will use a contagious disease example to show how population size and average number of mirror identities affect the spread of an epidemic. In Section 4 we will also show how our model is capable of displaying the epidemiological properties of R0 by comparing our model parameters with the R0 derived by a compartmental statistical model. In the fifth section we will simulate the 2003 SARS outbreak to show how our proposed model can be used to simulate the movement of a contagious disease, and compare our simulation results with actual data from the SARS outbreaks that occurred in Taiwan and Singapore.

2. Description of cellular automata with mirror identity model

We created the mirror identity concept in an attempt to accurately portray the daily activities of people living in modern societies—that is, the movement of individuals among various geographic locations. The concept treats individuals as agents with mirror identities that are logically abstracted in the form of places they regularly visit—for instance, residences, train stations, workplaces, and restaurants. Small clusters of neighbors consist of family members, coworkers, fellow commuters on trains and buses, and diners in restaurants. The mirror identities of any individual can be viewed as "hyper-links" that act in the same manner as weakly connected properties in small-world social networks (Figure 1). We believe that mirror identities more accurately reflect the low degree of separation that exists among individuals who travel long distances (See Appendix2.3).

On a societal level, each cell in a cellular automata(See Appendix2.1) can be viewed as one mirror identity, with the term of links used to describe relations among individuals or with their mirror identities. Links can represent many types of relations, including friendships, simple message transmissions, contacts between infected and healthy people, and sexual relations. Neighborhoods are categorized as either von

Neumann or Moore [9], depending on the social issue being studied (Figure 2). We will use the Moore concept for our discussion due to its ability to display triadic closures among neighbors (See Appendix2.2).

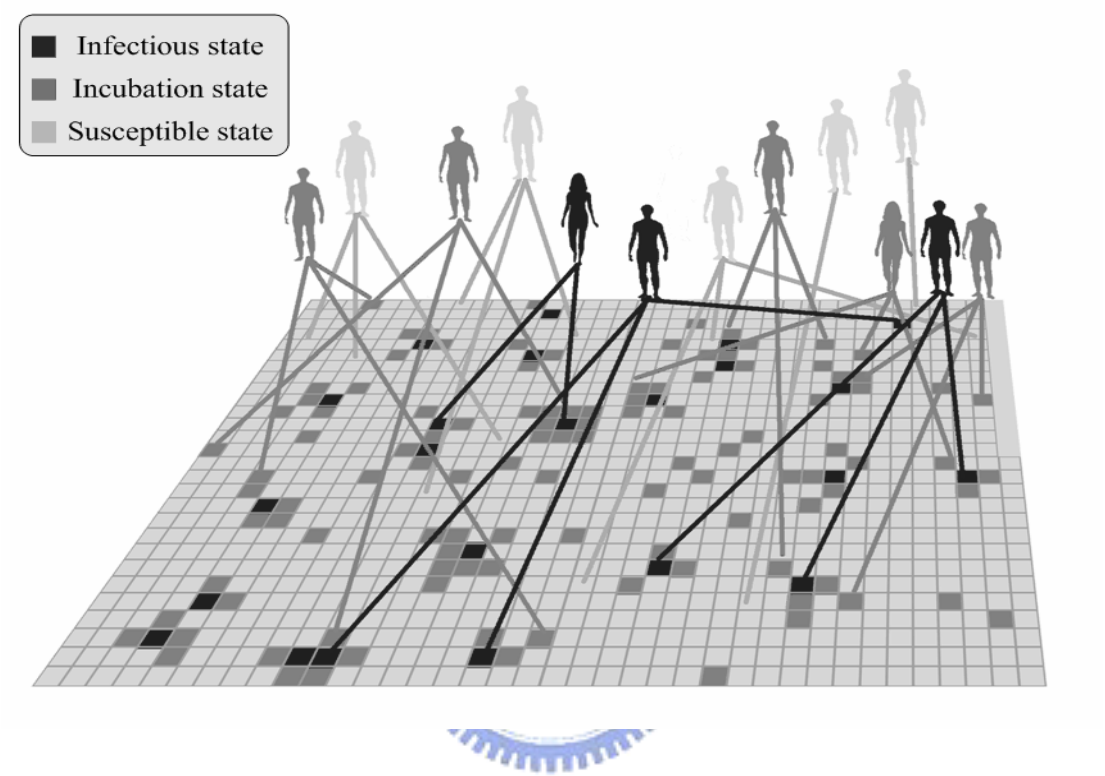

Figure 1. Social mirror identity on a two-dimensional lattice model

In terms of epidemiology, the independent status of all cellular automata sites and links makes it easier to study heterogeneous individuals as well as the interactive and temporal properties of infectious diseases discussed above. The SEIR model that we adopted acknowledges four disease statuses: susceptible (S), exposed (E), infected (I), and removed (R). All individuals are assumed as starting with a susceptible disease status, which changes according to contact history and the temporal development of the infectious disease in question. Any time an individual's disease

status changes, so do the statuses of all associated mirror identities.

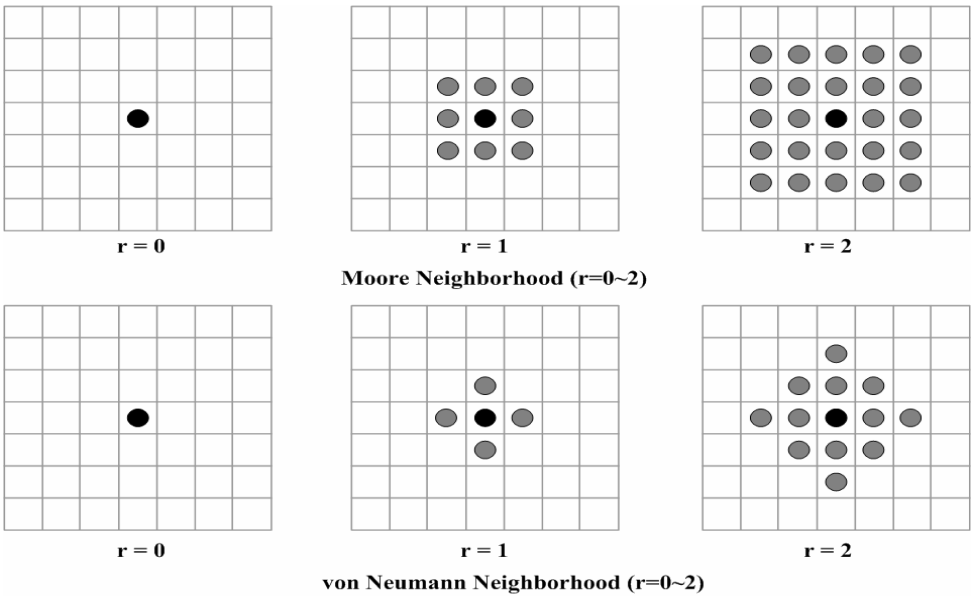

Figure 2. Examples of von Neumann and Moore Neighborhoods

In addition to addressing interactions among heterogeneous individuals, our model also makes use of the neighborhood concept and spatial properties to describe such important geographic locations as homes and healthcare centers (Figure 1). This reflects the reality that an individual not only has a disease status, but also geographic mobility and social identity during an epidemic outbreak. In the SARS scenario, isolation in a hospital means that we can disable the geographic mobility of all mirror identities but one (i.e., as a hospital patient) (See Appendix2.4); this allows for a restricted research focus on the movement of healthcare workers. A similar example entails a home quarantine policy involving family members as the only neighbors having access to the quarantined individual.

3. The small-world phenomenon

In 1998, Watts and Strogatz described the effects of Milgram's small-world phenomena on multiple interactions among social individuals [10, 11]. It is now generally accepted that the topological structure of social networks exerts a strong influence on the development of social issues [12]. Since individuals move over long distances and express aggregate behaviors, geographic location and distance are considered secondary disease transmission factors in societies with small world properties [13-15].

The two most important factors for measuring small world phenomena are degree of separation and clustering. Our rapidly increasing tendencies to come into contact with individuals over long distances by airplane, train, and car reduces the actual degree of separation among humans—what Milgram has described as "six degrees of separation." Most researchers use average vertex-vertex distances and clustering coefficients (i.e., average pair fractions of neighbors, either with a designated individual or with each other) in random graph theorems to calculate separation and clustering. Because real-world neighbors form triadic closures so easily, their relations tend to produce large clustering coefficients. Accordingly, the two most important factors in small world networks are low degree of separation (compared to regular networks) and high degree of clustering (compared to random

networks). A typical small world phenomenon is the logarithmic (instead of geometric) increase in degree of separation that occurs with the expansion of network size [16] (See Appendix3.1).

Watts and Strogatz's β-model is arguably a better fit than other models in terms of real-world parameters. Although the original β-model is a one-dimensional lattice, it does reflect a certain degree of randomness—for instance, randomly rewired links with β probabilities [10]. These networks are still mostly regular when β values are small, but some links are rewired to long-distance vertexes. In contrast, when β equals 1, all of the original network links are rewired to new vertexes, thus changing the original network to a random network (Figure 3). Watts and Strogatz believe that a modified network can be considered a small world network whenever the normalized average vertex-vertex distance is smaller than the clustering coefficient[10] (See Appendix3.2).

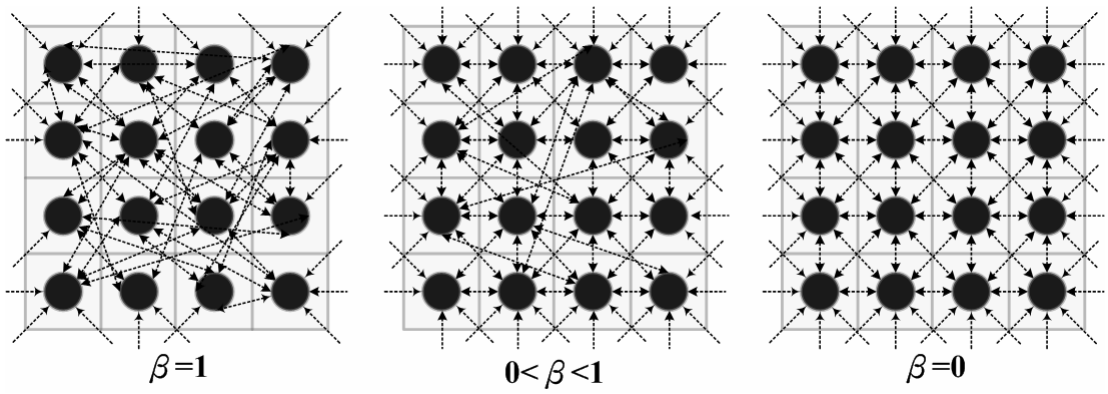

Figure 3. Creating a small world and random network from a regular network on a two-dimensional lattice

We used the mirror identity concept in order to utilize cellular automata for simulating interactions among mobile individuals. The use of mirror identities preserves the properties of individuals that interact with neighbors within cellular automata, thus acknowledging both long-distance movement and daily visits to fixed locations. The most important factor in our proposed model is the average number of mirror identities; combined with population size, it determines cellular automata size as follows:

$$Cellular\_Automata_{size} = Population_{size} \times Mirror\_\ identities_{average\_number}$$

We used an average number of mirror identities to create a normal distribution of mirror identities per individual, then allocated each mirror identity to cellular automata so that each cell contains a single mirror identity for one individual (See Appendix3.3.1).

To use our proposed model to analyze small world phenomena, we changed the values for population size and number of mirror identities to observe changes in average vertex-vertex distances and clustering coefficients. As shown in Figure 4, degree of separation increased with population size regardless of the number of mirror identities used. However, as shown in Figure 5, even when population size increased, the average clustering coefficient did not change with increased population size. In this situation, the degree of clustering was affected by the number of neighbors;

obviously, the degree of clustering remains constant with a fixed number of neighbors, as is the case when the first layer of a Moore neighborhood is utilized. On the other hand, a typical small world effect is noted in Figure 4, due to the logarithmic increase in degree of separation according to network size[16](See Appendix3.3.2).

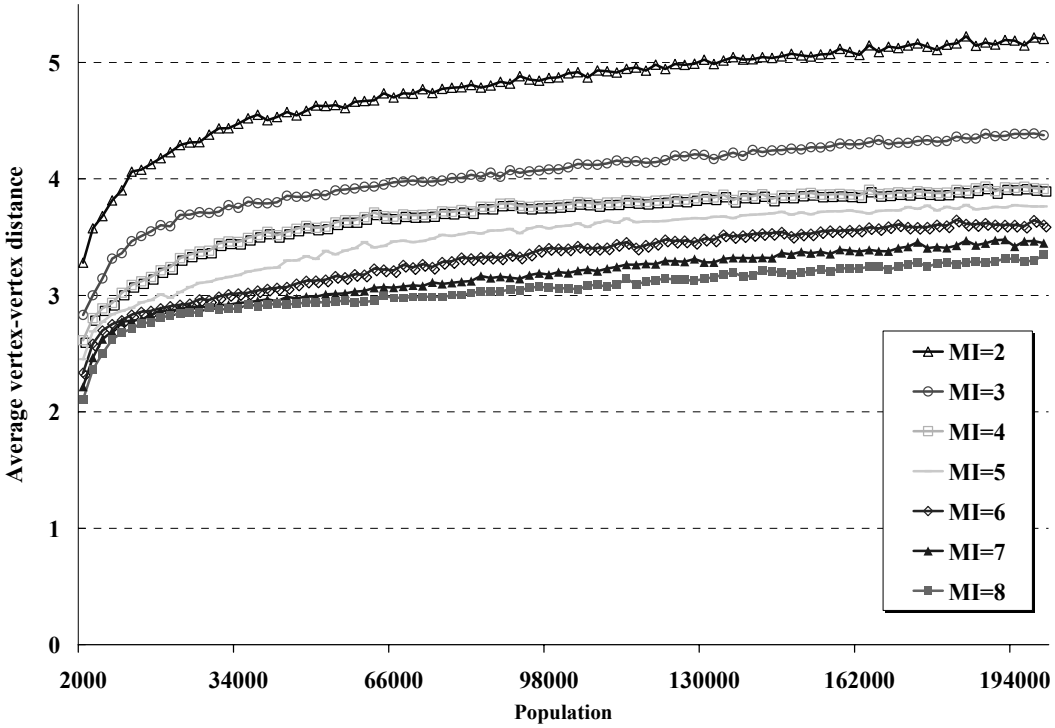

Figure 4. Population effect on average vertex-vertex distance (MI: average number of mirror identities)

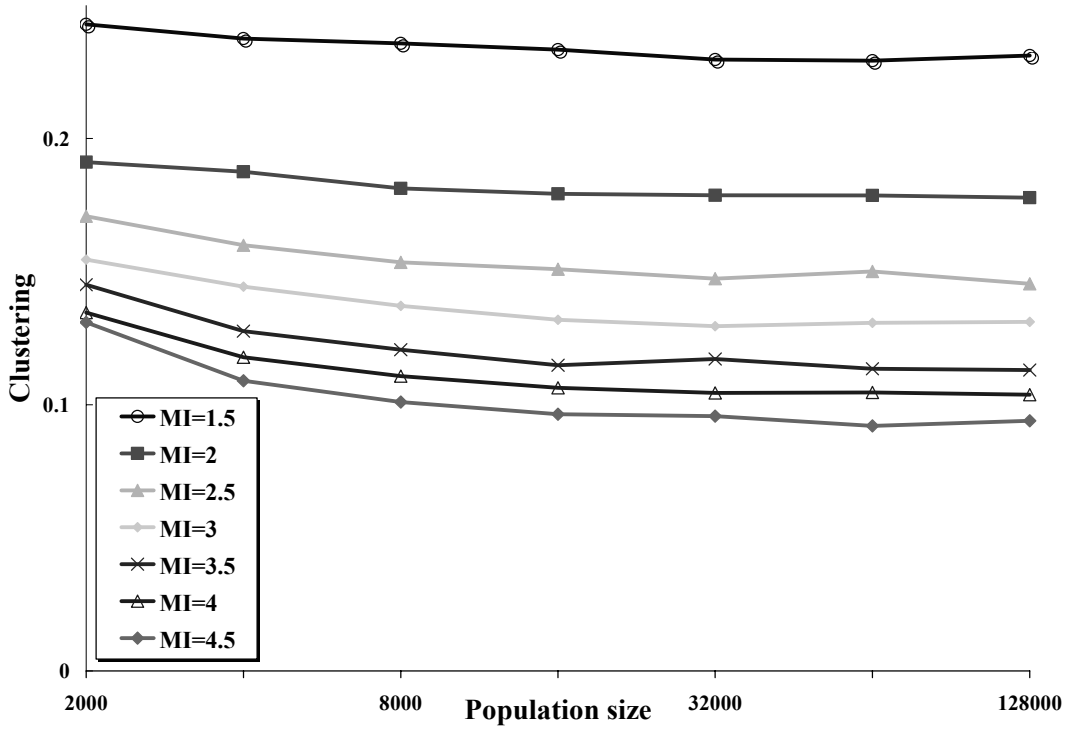

Figure 5. Population effect on clustering coefficient (MI: average number of mirror identities)

When the number of mirror identities increased, the average vertex-vertex distance quickly decreased to 6 or 7 steps (Figure 4); this low degree of separation is considered a small world phenomenon. Furthermore, we observed that the normalized clustering coefficient was larger than the normalized average vertex-vertex distance (Figure 6). By comparing the results of the two clustering and separation curves with increasing β in the β-model[10], we verified that our proposed model could display the same small world properties. We offer this as evidence that our simulation model is capable of displaying real-world properties (See Appendix3.3.3).

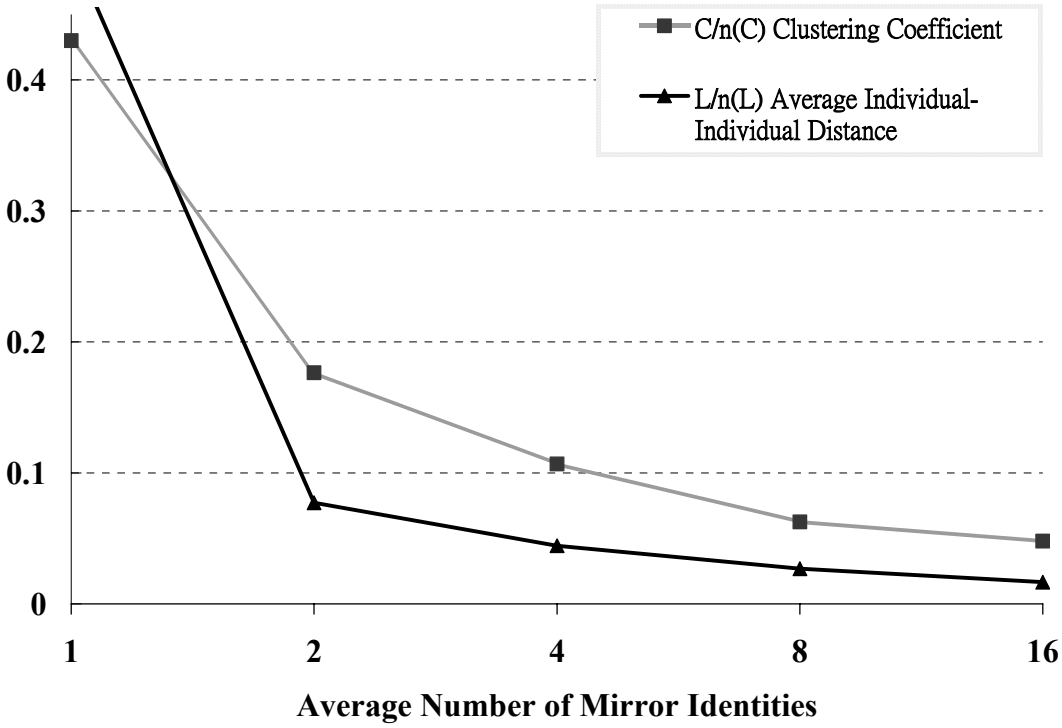

Figure 6. Mirror identity effect on average vertex-vertex distance (normalized) and clustering coefficient

4. Validating contagious disease simulation and parameter sensitivity analysis

In this section we will address two issues: a) how to integrate classic epidemiology with cellular automata and mirror identities to build a simulation network model for contagious diseases, and b) how mirror identity number and population size affects contagious disease transmission dynamics. Since contagious diseases spread through daily contact among individuals, the underlying contagious disease network is clearly a contact network. A model that incorporates cellular automata with mirror identities is suitable for analyzing contact networks.

An individual's state in a society entails a) epidemiological status, for which we adopted the SEIR model of susceptible, incubation, proper infection, and recovered periods; and b) geographic mobility, using such categories as "normal," "quarantined," and "isolated." Transitions are determined by probabilistic causes—for instance, the probabilities of contact with sick persons (contact rate), of catching a disease (transmission rate), of being detected as having a fever (detection rate), recovery rate, or the conditions of neighbors (Figure 7)(See Appendix4.1). If the state of one mirror identity changes, the states of all mirror identities associated with the same individual also change. Even though the mirror identities associated with a

certain individual share a common infection status and geographic mobility, each mirror identity is situated in a different location on the lattice. The local properties of a mirror identity (e.g., whether a location is next to an infected site) have the potential to change an individual's disease state. Once a transition is determined, it is immediately applied to all of the mirror identities of that individual.

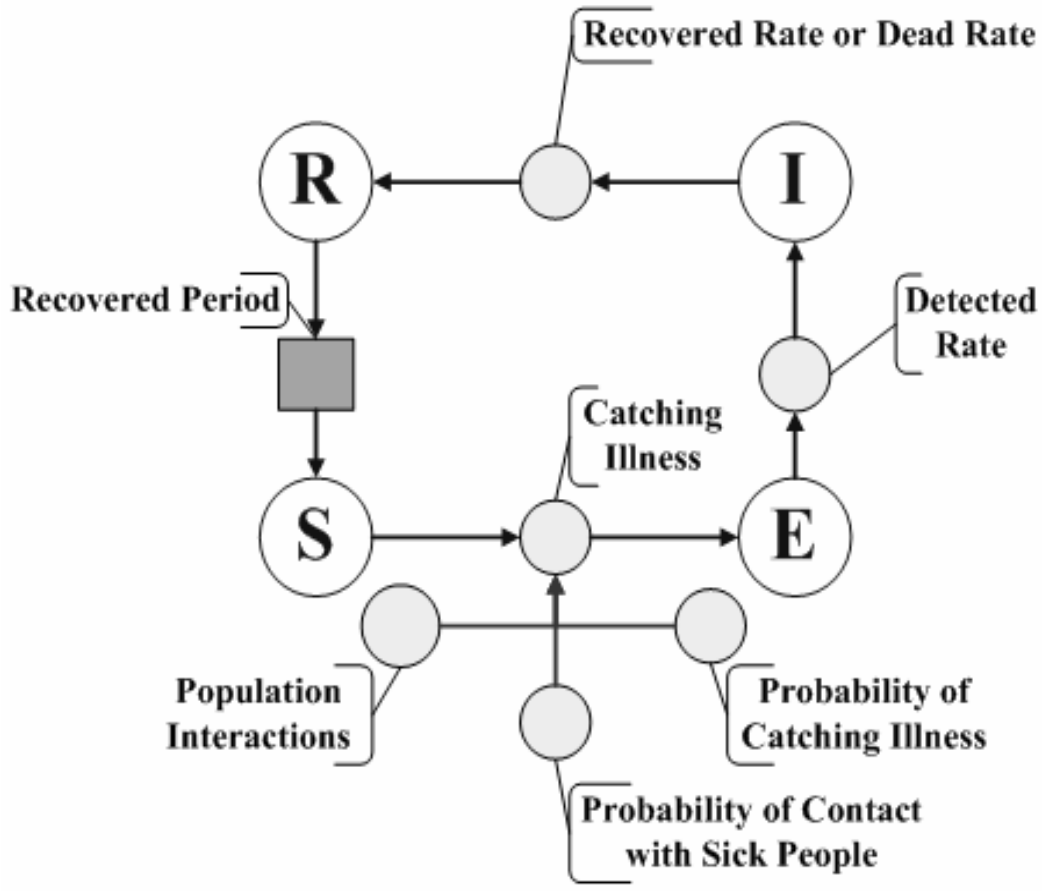

Figure 7. SEIR state transformation and transforming probabilities

In a previous project that used the transmission model to simulate a contagious disease[1, 5], these projects reduced R0 to a simple relational expression such as c·β·D[17]; when all neighbors of an infected individual are susceptible, R0 equals the contact rate × transmission probability × duration of infection. After integrating

mirror identities with cellular automata, we modified the original expression to obtain

$$R_0 = (C_{rate} \times C_{time}) \times (T_{rate} \times T_{period}) \times (Avg.\ Mirror \times Num.\ of\ Neighbors)$$

where Crate stands for the rate of contact between an individual and his or her respective neighbors, Ctimes stands for the number of contacts between individuals and neighbors in one day, Trate the average transmission rate of infected individuals, Tperiod the average transmissible period of an infected individual, Avg. Mirror the average number of mirror identities, and Num. of Neighbors the number of neighbors for each mirror identity.

Because we adopted the first layer of the Moore neighborhood for our model, Num. of Neighbors = 8 and Crate = 1/8. In addition to adopting R0 and transmission periods derived by epidemiologists, we also adopted the average number of mirror identities and numbers of neighbors as suggested by sociologists. We then integrated parameters derived from R0 to simulate the contagious disease and to derive a transmission rate.

To determine how the average number of mirror identities and population size affect transmission dynamics, we manipulated both factors while simulating the outbreak of a contagious disease epidemic. Since the average number of mirror identities exerts a strong influence on degrees of separation and clustering while population exerts a weak influence, we assumed that the average number of mirror

identities would have a greater impact on the course of the epidemic.

For our simulation trigger, we used the number of imported SARS cases according to a timeline established for the SARS outbreak in Singapore. We found that increasing population size had a small impact on the spreading of the disease, but increasing the average number of mirror identities had a large impact (Figures 8, 9). After comparing this with the considerable impact of average number of mirror identities on degrees of separation and clustering, we concluded that degrees of separation and clustering exert very strong influences on epidemic growth. Furthermore, because the average number of mirror identities has a larger impact than decreasing the degree of clustering on shortening the degree of separation, the total number of infected individuals increased in step with an increasing average number of mirror identities.

In terms of validating our proposed network model, we attempted to replicate an important R0 property—that is, when R0>1, a disease becomes epidemic; if R0=1, the disease becomes endemic; if R0<1, the disease is easily suppressed. By deconstructing R0, it should be possible to choose an appropriate suite of simulation parameters once a value for the transmissible period of a disease is provided by epidemiologists. Since parameter sensitivity to an epidemic model varies, it is possible to use R0 properties to find appropriate suites. In Figure 10, the R0 properties

appear when Tperiod = 6, Avg. Mirror = 4, and Ctime = 4, 3, or 2. We divided the R0 into the six parameters we used to build our model in order to perform a contagious disease simulation. We believe the model can also be used to display the epidemiological properties of R0 (See Appendix4.2).

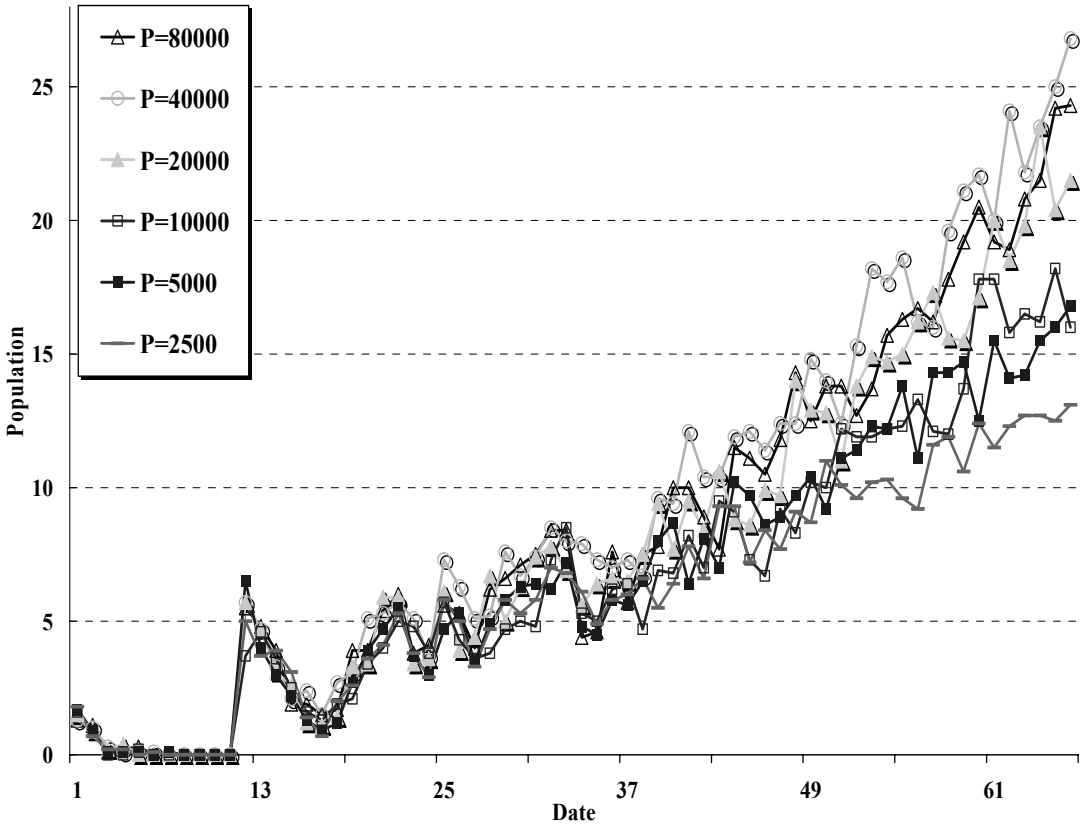

Figure 8. Effects of variable population size (P) on an epidemic curve

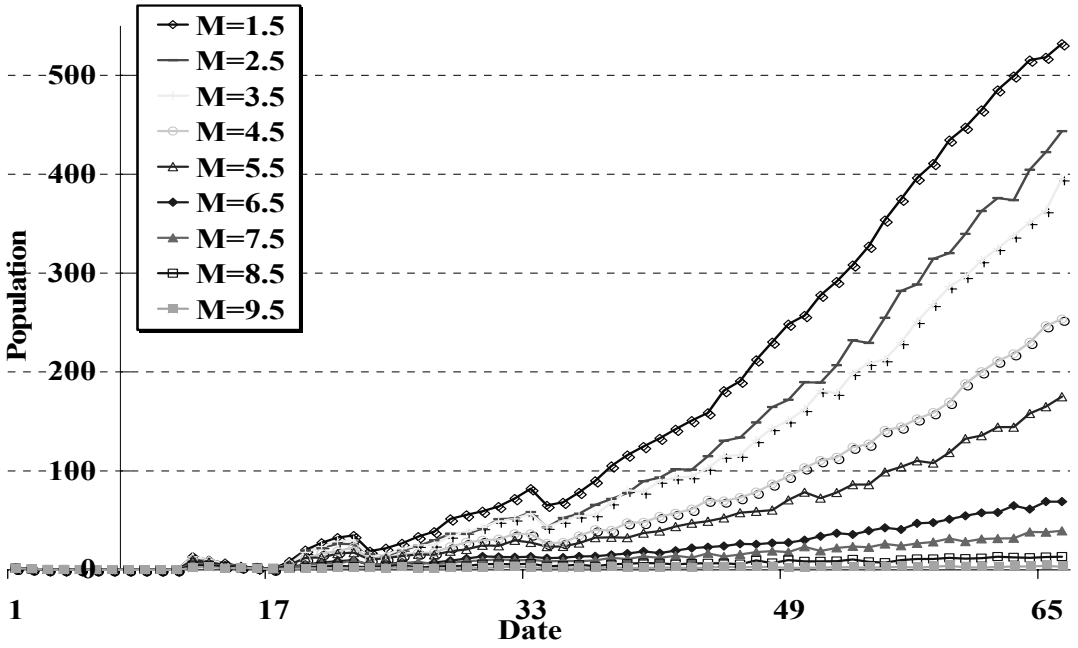

Figure 9. Effects of variable average number of mirror identities (M) on an epidemic curve

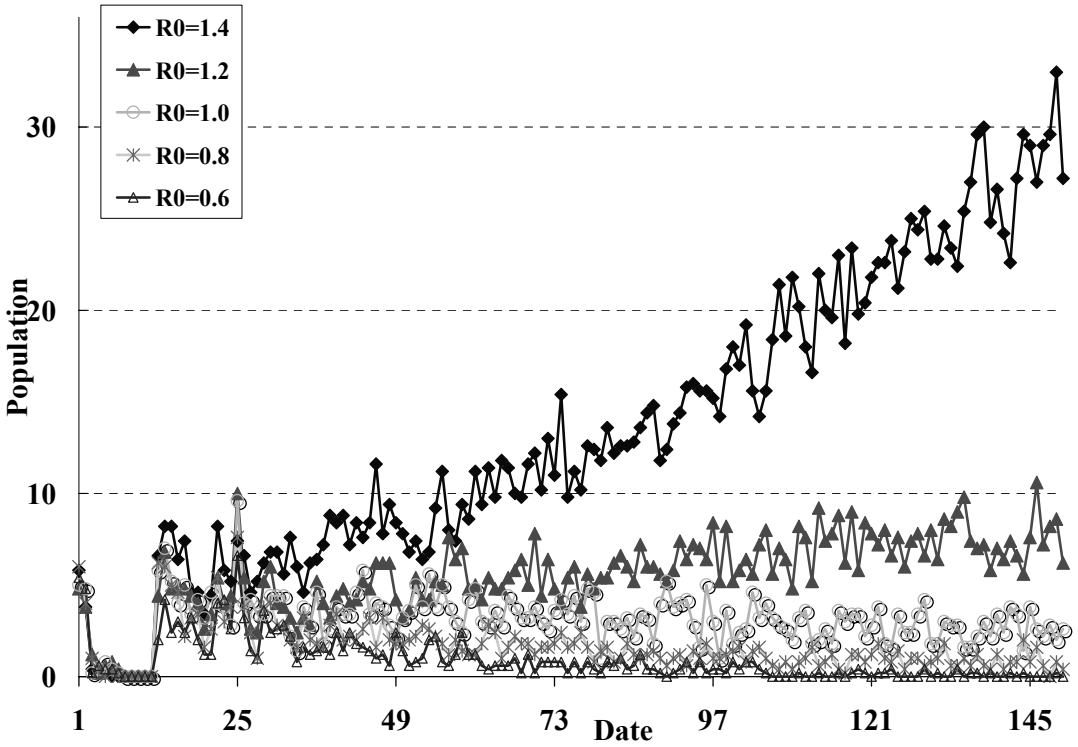

Figure 10. Epidemic curves(R0=1.4, 1.2, 1.0, 0.8, 0.6) when Tperiod=6, Avg. Mirror=4, Ctime=4.

5. SARS application

After verifying our proposed model's societal and epidemiological properties, we performed transmission simulations of the 2003 SARS outbreaks in Singapore and Taiwan, focusing on the effectiveness of those countries' respective public health policies. Since SARS is a short-distance contagious disease, its underlying transmission network was identified as a contact network with moving individuals, making it suitable for a simulation involving cellular automata with mirror identities. According to the specific epidemiological and social conditions associated with the SARS outbreak, our simulations began with imported cases acting as triggers. Both simulations then proceeded using data on public health policies collected from the two countries. Results were organized in chart form for analysis and comparison (See Appendix5.1).

In the Singapore simulation, the epidemic curve was very similar to that published by the Singaporean health authority in June, 2003 (Figure 11). The curve reflects the SARS epidemic in that city between March and May of the same year, during which two major outbreaks occurred. Our simulation took into account several policies enacted by the health authority on March 24 (e.g., home quarantines, closing hospitals, and banning hospital patient visitation). Based on our experience with the

Singapore simulation, we restricted our Taiwan simulation to cases reported in metropolitan Taipei and policies enforced by the Taipei city government. Our results fit well with the epidemic curve published by the Taipei Health Department on September 28, 2003—that is, one major infection climax followed by several small-scale outbreaks (Figure 12). The Taipei epidemic curve was more concentrated than Singapore's; a likely explanation for this difference is the late discovery of other imported cases in Taipei—for instance, those involving travelers returning from Hong Kong.

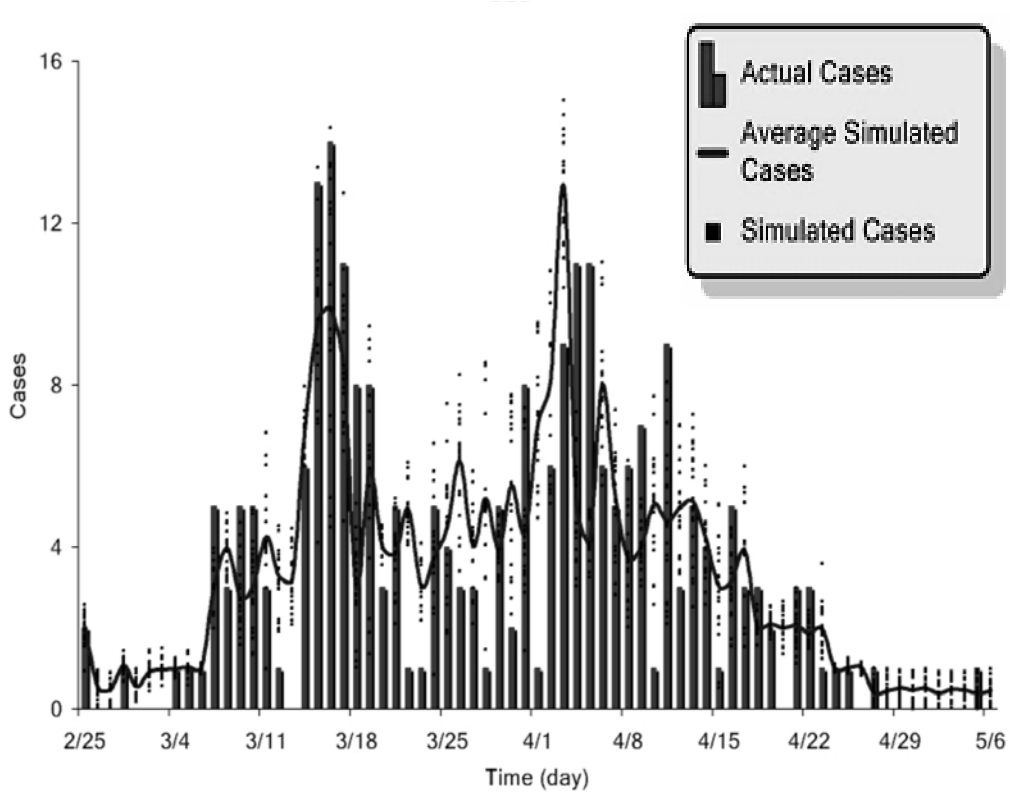

Figure 11. Singapore SARS curve and simulation results

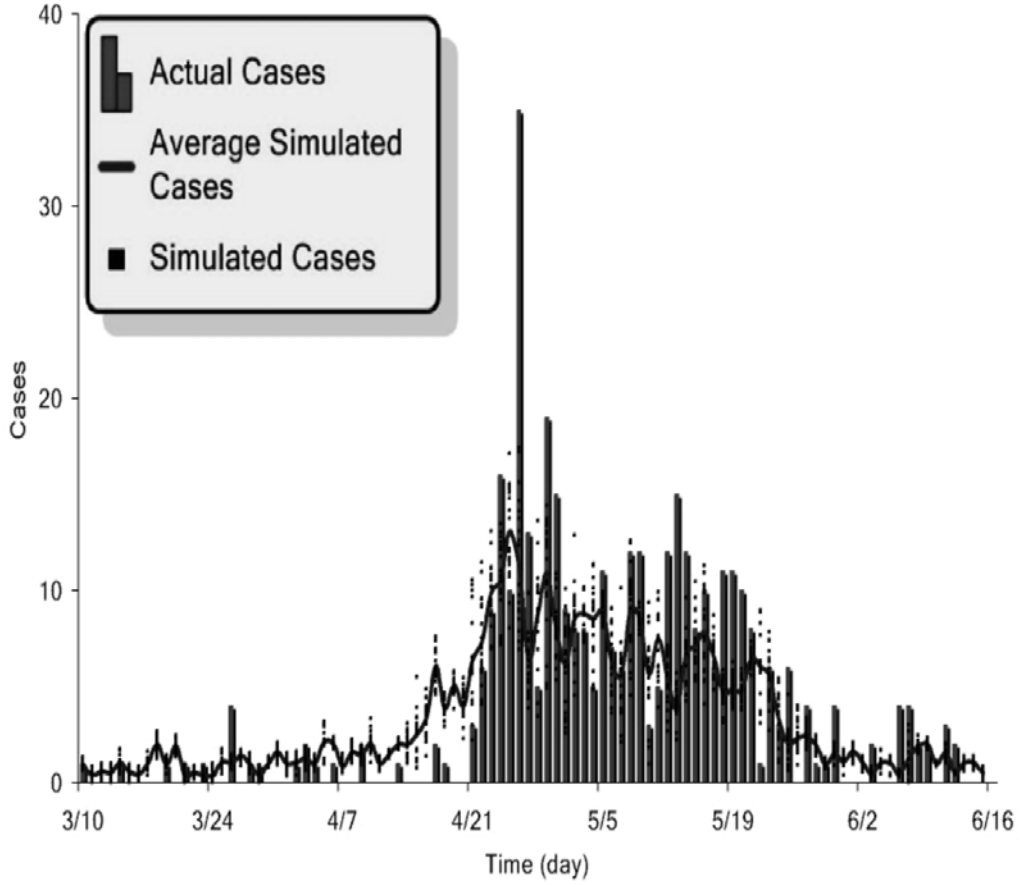

Figure 12. Taiwan SARS curve and simulation result

In addition to producing epidemic curves for infected areas, our proposed model may assist health authorities and researchers better understand the efficacies of public health policies (See Appendix5.2.1). As part of our simulation, we iteratively tested various combinations of public health policies in an effort to identify the best suite of policies for curbing the epidemic (See Appendix5.2.2). The four policies we tested were the wearing of surgical masks by the general public, the wearing of surgical masks by healthcare workers, quarantines, and large-scale efforts to measure body temperature. Reduced public contact and hospital closures served as control measures.

According to our simulation results, the combination of mask wearing by the general public and reduced public contact would be very effective in suppressing the epidemic (Figure 13).

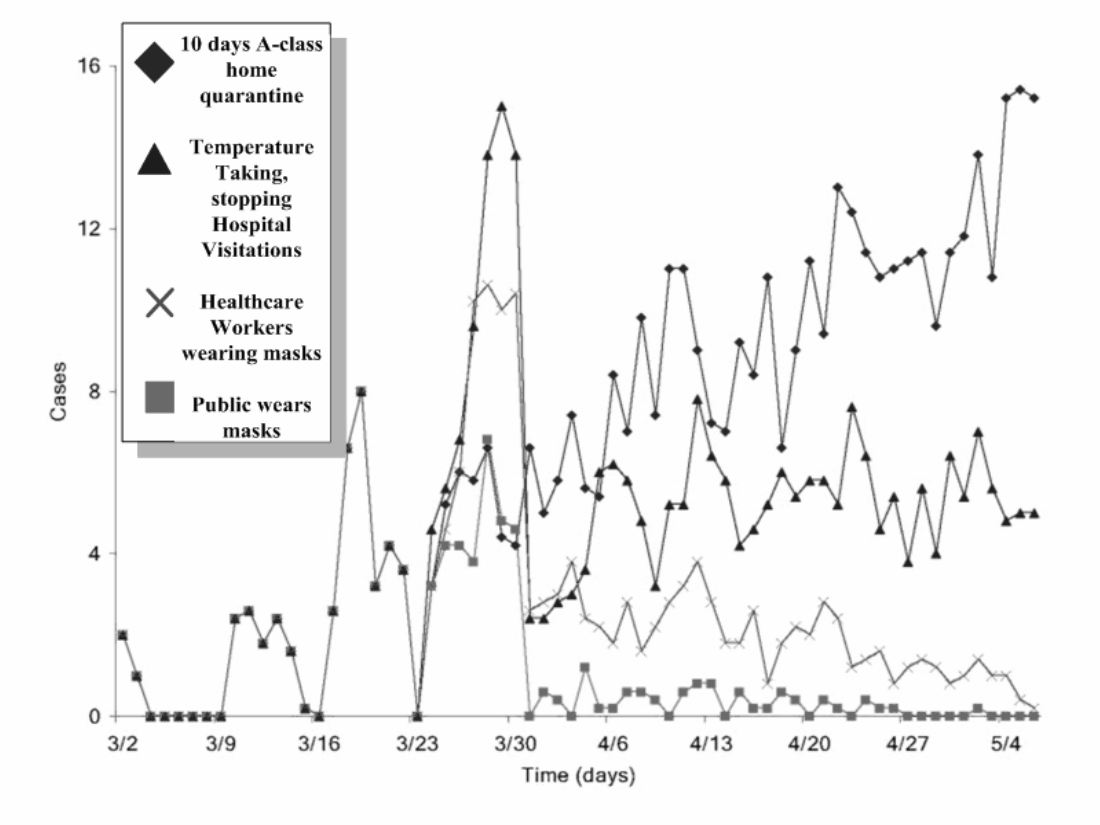

Figure 13. Effects of various public health policy suites

6. Conclusion

We used this paper to propose a small world model that integrates cellular automata with mirror identities, then verified its small world properties in terms of average vertex-vertex distance and clustering coefficients. Based on an analysis of individual parameter sensitivities from deconstructed R0, we observed the same R0 properties in our model. We therefore suggest that our model is capable of displaying the societal and epidemiological properties of epidemics. We then presented and discussed results from simulations of the 2003 SARS outbreaks in Singapore and Taiwan.

Since our model is based on social network models with simple geographic properties, and since it applies the concept of mirror identity, we were able to describe several special terms associated with epidemic outbreaks—for instance, hospitals, families, and heterogeneous individuals with varying geographic mobility. Finally, the results show that our simulation model is very flexible and can be applied to a wide range of diseases with different transmission routes. It can be used to simulate influenza, smallpox, HIV, or enterovirus disease epidemics using such parameters as specific definitions of individuals, special interaction rules between individuals, and the progress of other epidemics. Since our model can be used to display small world

properties, it is also suitable for simulating other social issues that involve individual movement.

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附錄

A1 社會中的三角閉合與流行病模擬模型

A1.1三角閉合

三角閉合（Triadic closure）的觀念由 Anatol Rapoport 在 1957 年時提出。他們的研究團隊當時瞭解到，在真實社會中的人際關係網路模型不等於隨機網路模型，他們注意到個體在社會中，往往會呈現物以類聚的關係。例如在同公司的職員、同班的學生，彼此間互相認識的機率就比其他在社會中的人來的大；或是天天坐同一班公車的上班族，彼此間互相接觸的機率也比一般人來的大，這些接觸可能是言語或所聽見的事情，或疾病的飛沫傳染、接觸傳染等；常常上同一個網站或留言版，彼此之間有訊息交流的可能也比其他人來的大。以上，不管是朋友關係、日常生活接觸關係或訊息交流，就過去的隨機網路模型而言，沒有辦法描述這樣的概念。這些人會互相認識，乃是他們之間具有一些共通點的關係，這造成了人際社會中許多不同關係網路的區域群聚性。但更有趣的是，例如若 A 認識 B，B 又認識 C，那麼 A 認識 C 的機會會比較大嗎？是的，這就是 Rapoport 所強調的三角閉合。例如說社會中某個體的家人，與該個體在公司的同事認識的機率，應該就比其他一般人大的多。而當個體隨著社會的動態演化，個體與個體之間，會傾向於三角閉合的觀念。

A1.2流行病模擬模型的發展

A1.2.1　傳統統計學流行病模型（Compartmental Model）

該模型為統計學模型，如同此模型的名稱一樣，此模型將各種不同疾病狀態的個體分群，各群中的個體是連續且都是一致的（identical），例如：潛伏群（ incubation）、感染群（infectious）、免疫群（immunity），之後計算在每個群與群之間個體數的變化，以瞭解整個疾病的趨勢，並計算其轉變參數。可以瞭解每一個個體，在康復或死亡之前，平均會引起多少人次的二次傳染。在此模型中並沒有個體的概念，因此也不容易討論潛在的異質性個體對感染所造成的影響，如：超級傳染者。在這方面著名的流行病模型有 SIS、SIR、SEIR 等模型。

A1.2.2　簡單網路流行病模型（Simple Network Model）

由於社會議題或事件，例如整個流行病的趨勢，就是由許多個體間的互動所累積構成的，也可以解釋為整個社會所表現出來的現象，其實是很多個體片段的行為所累積浮現出來，所以在做流行病或其他社會議題模擬的時候，必須要考慮到個體與個體之間的互動。而網路模型所重視的就是以個體與個體之間的固定連結，來代表個體與個體之間的關係。在網路模型的社會中，每個個體擁有固定數量的連結，連結到其他個體，每個個體的行為與其他個體的行為，接觸、訊息溝通或流行病的傳染，都是獨立的且以機率來決定。在早期以網路作為流行病模擬或其他社會議題探討的時候，所使用的多半是強調其隨機的隨機網路模型

（random network model），或是強調其簡單且易觀察的有序網路模型（regular network model）。隨機網路強調的是模型中個體與個體之間的隨機連結機率，但是卻也難以掌控，尤其難以呈現在真實社會中的區域性高群聚度的現象。有序網路強調的是對於區域性的描述，例如易於描述個體與周圍個體之間的關係，但是真實社會並非如同有序網路般，有整齊一致的脈絡。所以，如何呈現社會特性，就成為以網路模型來研究流行病模擬或其他社會議題的重要課題。

A1.2.3　小世界網路流行病模型（Small-World Network Model）

自從 1967 年 Milgram 提出小世界現象（Small-World Phenomenon），在 1998 年由 Watts 與 Strogatz 加以發揚光大後，小世界現象成為研究社會議題一個重要的指標。若相關於社會網路與社會中的個體對整個社會的影響，都可以考慮這些社會議題的底層網路是否可以用小世界效應來探討。他比原先的網路模型多的是，展現真實社會中一些社會現象的特性，也就是低分隔度與高群聚度的特性，而人際社會中的低分隔度與高群聚度的特性，將在本附錄的 3-1 節中做詳細討論。

經由小世界網路，疾病可以藉著高群聚度的特性，快速地傳染給周圍區域性的個體，再經由低分隔度的特性，快速在整個社會中流竄。Newman 與 Watts 曾以二維晶格加上捷徑（shortcut）的方法，作為模擬流行病底層的小世界網路模型，而在此環境上疾病的傳染，則以滲透（percolation）的概念來進行。但是目前此模型被證明出來其在決定流行病為鍵結滲透（bond percolation）或地基滲透（site percolation）時，常常導致類似的流行病得到迥然不同的預測。

A2 具有分身點的細胞自動機

A2.1細胞自動機

本論文的模型利用了人工智慧中的細胞自動機，加上論文中所提出的分身點的概念。以二維細胞自動機來說，底層架構原本就是一個具有空間性，且能夠展現異質性個體之間的關係的二維晶格。我們很容易的就可以在這上面定義個體的性質與鄰居的關係。再加上參考細胞自動機中，每個晶格擁有自己的晶格狀態的概念，在流行病學中，一個染病個體就具有潛伏期、發病期與康復期等不同的狀態，並且根據流行病狀態轉變的規則而轉變；而若以兩黨制的政治選舉為例的話，可發現群眾起碼會分為三種不同的狀態，偏其中一個政黨，或成為中間選民。細胞自動機所注重的是由片段晶格的互動，所導致整體的浮現現象，而許多的社會議題也是如此。所以在進行社會模擬的時候，很適合用細胞自動機來做模擬的平台。但是其實只要底層的模型能夠展現出前述的社會特性就適合作為模擬的模型，然後再去定義上面個體的性質，和互動的關係。

A2.2鄰居關係的定義

鄰居關係有很多種，有 Moore Neighborhood 和 von Neumann Neighborhood，但是在後面的模擬中皆採用 Moore Neighborhood 為例，因為 Moore Neighborhood 的第一層鄰居關係共有八名鄰居，而且周圍的鄰居與該個體具有附錄 1.1 所提及

的三角閉合關係。以 Figures 2, 14 為例，可發現在 Moore Neighborhood 的第一層鄰居關係就能夠展現三角閉合的現象。但是第一層的 von Neumann Neighborhood 則沒有三角閉合的現象。兩種鄰居關係的第二層鄰居個數和真實社會的鄰居個數比起來又過多（von Neumann: 12 人，Moore: 24 人），所以本論文選擇用第一層的 Moore Neighborhood 為鄰居關係。在經由附錄 2.3 節對分身點的描述後，我們可以發現，運用分身點的概念，可以展現異質性的鄰居關係。由於在一天之中，個體在社會上具有多個多重身份，所以個體的鄰居關係不侷限在於一個點的周圍，而是在一群點的周圍，這可以修改以往在討論細胞自動機的時候，只針對每一個點所提出的鄰居概念。

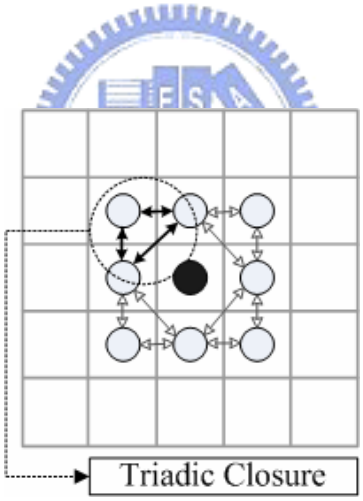

Figure 14. 第一層的 Moore Neighborhood 所呈現的三角閉合關係

A2.3社會分身點與個體

在本節將清楚定義每一個個體與其分身點之間的關係，以及我們如何使用分身點的概念，在細胞自動機上呈現小世界的特性。我們可以把個體與分身點的關係簡單的分為上下兩層（如 Figure 1）：

上層是抽象層，每一個點代表一個個體，一個個體可以擁有數個分身點，在

本層個體的身份是唯一的。上層只描述了個人身份和分身點的從屬關係，並不具有地域觀念，例如下頁 Figure 15 中的 A 與 B 雖然在上層的距離看似很遠，但是卻因為底層有分身點分佈的關係，其實 A 與 B 是鄰居，有可能是公司同事，或是天天在公車站一起等公車的人。

底層是實體的模型層面，在二維細胞自動機上的每一個晶格恰可以代表一個分身點，每個分身點唯一屬於一個個體，但是很多分身點會從屬於某一個個體。底層最大的特色是具有區域性，在底層相當靠近的個體，就代表其在地理位置上相當接近。但是經由上下層關係的對照後，因為個體的遠距離移動與每天的定點移動，會有某些遠距離的個體，其實日常接觸關係的接觸相當接近。例如 Figure 15 中的 C 與 E 雖然間隔很遠，兩人的分身點也互不為鄰居，但由於 D 和 C 是鄰居，D 和 E 也是鄰居，所以兩個人的接觸關係只透過一個 D，所以兩個人的實際日常接觸關係距離相當接近，不受到地域的影響。

在社會特性的呈現上，在底層的分身點與其該分身點周圍的鄰居，呈現出在 Moore Neighborhood 中的三角閉合現象，而本研究所注重的遠距離移動與日常定點移動的現象，則是由上下層的個體身份和各分身點的對應來表示。如同本論文在第三章所證明的，加上分身點概念後的細胞自動機，具有區域群聚性，又能表達出移動性個體（Mobile Individual）的行為。

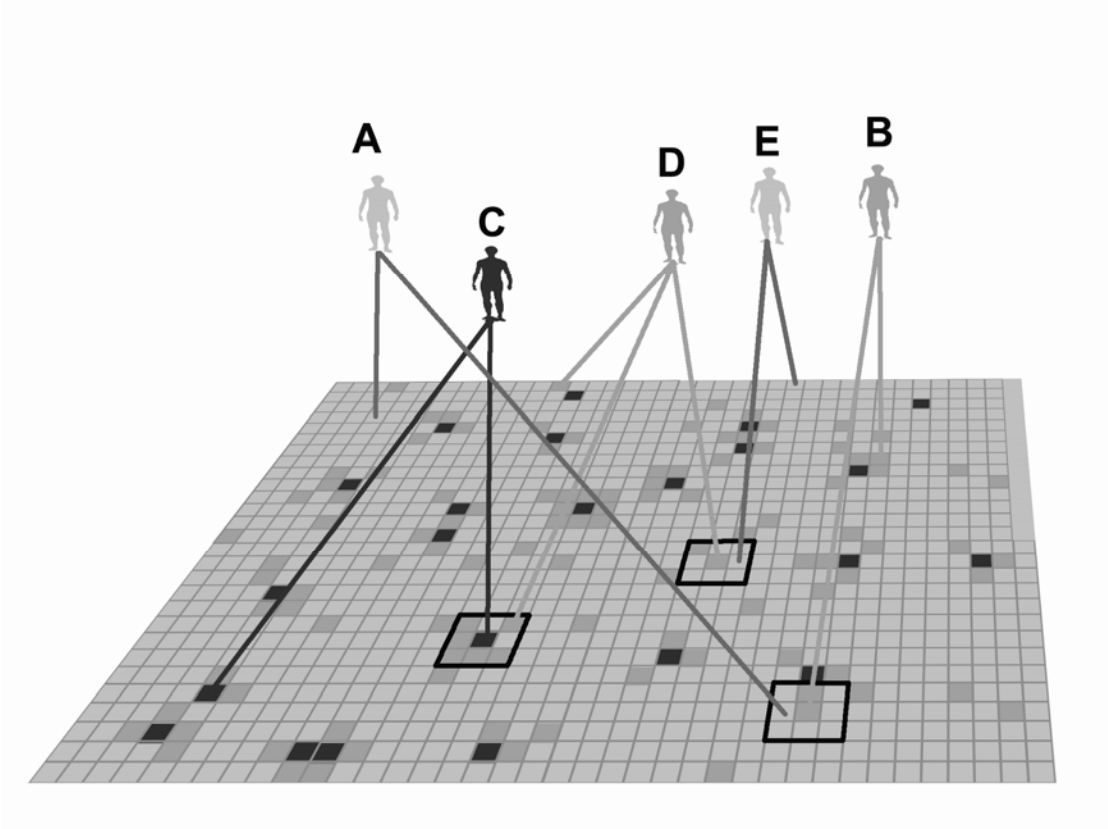

Figure 15. 社會中個體和其分身點的關係示意圖

A2.4社會行動力

利用本模型的抽象個體，與在實際模型上的分身點，可以表達出很多在流行病爆發時期的特有現象。例如「醫院隔離」、「居家隔離」。和流行病中的特殊身份「醫護人員」、「被隔離的家人」。社會行動力（Social Mobility）就是指一個人所能活動的能力，在討論流行並模擬時，特別用來討論的就是那些因為隔離或醫院治療被限制行動的個體。

「醫院隔離」：當染病個體（E）尚未被發現時，其會在社會中到處行動將疾病傳染給周圍的個體，但是以 SARS 而言，染病個體往往病狀會非常嚴重（發燒、乾咳），所以很容易被發現。被發現後，染病個體就會被送進醫院隔離，進

入染並且隔離的狀態（I）。其在細胞自動機上的所有分身點，只留下一個分身點能夠與周圍的鄰居繼續保持接觸，其他的分身點的接觸活動均停止，代表此人的活動被限制在一個地方。而那些能和該分身點互動的周圍鄰居，就可以被視為是醫護人員。所以藉著分身點的概念，我們不僅定義了醫院隔離的現象，也定義了周圍醫護人員的狀況，並以相同的方式，定義封院時，醫護人員被留在病人周圍，所引發的高傳染率現象。

「居家隔離」：被居家隔離的個體是有可疑病狀的個體，在這次 SARS 疫情下，多國政府啟動居家隔離政策，有可疑病狀的個體會被強迫居家隔離。在本模型中被居家隔離的個體行為和被醫院隔離相似，除了這時候個體的染病狀態應該是可疑的（S）或略有症狀但卻未被發現（E）。所有屬於該個體的分身點，只留下一個分身點能夠繼續和周圍鄰居接觸，其他均停止。而和此尚能活動的分身點相鄰的個體，就可以被視為該可疑個體的家人。所以我們也可以模擬出家人的觀念。

A3 小世界現象與社會性的驗證

A3.1小世界現象和傳統的小世界模型

在 Milgram 於 1967 年以實驗說明小世界現象時，就已經注意到社會中的個高群聚度與低分隔度的現象，所謂人際社會中的的低分隔度與高群聚度，可以想像在人與區域周圍的鄰居之間，往往會因為三角閉合的關係形成高群聚度的鄰居關係，但是若這個社會只有高群聚度現象的時候，照道理來說，遠距離的個體，應該彼此間的分隔度也會很高。比方說在早期社會，由於交通不便，訊息若要傳到遠方的話，必須要以人工或郵件來傳送，於是與遠處之間的訊息溝通就會相當貧乏，往往只限於區域性的訊息溝通。但是在現代的社會中，由於有便利的交通和訊息溝通工具，人在一天之內，很容易就可以將訊息快速地散播到各地。若模擬的時候以一天為時間單位，就彷彿此人在一天之中同時在很多地方發表他的意見，會大大的降低人和人之間訊息溝通的分隔度。而又以人的朋友關係為例，早期社會人與人之間的朋友關係大部分只侷限於該生長的村落，所以群聚度會非常高，與遠方的村落則沒有朋友關係。但是在今日交通便利的社會，往往人一天就會藉著交通工具從家庭到工作場合、餐廳、或朋友家等地，A 是 B 的家庭成員，而 B 是 C 的工作主管，雖然工作和家庭的地方區域距離很遠，但是 A 和 C 的分隔其實很低。所以，整個社會在交通便利與訊息網路便利後，造成了現在社會中，如：疾病、訊息、思想觀念、朋友關係等等議題原本是區域性傳播的問題，藉著

現代社會所提供低分隔度現象，使得這些議題得以藉由低接觸次數就快速擴散開來。

要討論這樣的問題，我們必須把整個社會視為很大的一個網路，那麼社會中的個體就是網路上的節點，而我們所要討論的個體關係，就是在節點與節點之間的連結。可以發現，在有序網路中很容易可以藉著各種鄰居關係來表現區域群聚性，但是在有序網路中，就難以呈現小世界現象，因為個體與個體之間的分隔度，與他們的地理位置分隔成正比。相反的，在隨機網路中，我們難以控制其中的區域群聚性，但是隨機網路卻常常具有六度分隔的現象（非必然）。所以，在建構一個小世界網路的時候，我們可以嘗試由一個有序網路開始，藉由描述真實社會的特殊行為（人的移動、通訊、或每日的定點移動），增加圖形中的隨機性。從有序網路到隨機網路之間，應可以找到一塊區域，具有有序網路的優點，容易描述區域群聚性，又有隨機網路的優點，具有六度分隔的現象，利用這樣的組合，建構出小世界網路，以模擬社會議題，下一節 Watts 和 Strogatz 所提出的 β 模型就是利用這樣的想法來架構一個小世界網路。

A3.2Watts 和 Strogatz 與他們的 β 模型

Watts 和 Strogatz 的 β-model 以晶格作為基本模型，本論文的底層模型亦為一二維晶格，晶格間的距離長度皆同，很容易可以討論地理相關的問題。Watts 和 Strogatz 一開始以一維晶格來討論，並且首尾相接，成為一個環狀一維晶格，這就不用解釋在環境中邊界晶格的問題，本論文的二維晶格亦是如此，二維的晶

格上下相接，左右相接形成一環面。Watts 和 Strogatz 的研究在於要在有序與無序網路之間找到中間狀態的小世界網路，而並非要找到一個完全掌握社會關係的網路。他們利用 β 值來選擇是否重新更改原先在有序網路上的連結，當 β=0，那麼所有有序網路上的連結均不需被更動，若 β=1，所有的連結均被隨機更動，所產生的就是一個隨機網路模型，而當 β 值介於 0 和 1 之間的時候，既保有原先有序網路的高群聚度，也有低分隔度的現象，就是 Watts 和 Strogatz 所提出的小世界網路。在 Watts 和 Strogatz 發表的論文中，他們比較經過正規化的平均分隔度和群聚度後，平均分隔度會隨著 β 值急遽下降，然後趨於平緩；而群聚度則會先漸漸降低，然後急遽下降（Figure 16）。他們所提出的小世界網路就是當隨著 β 越來越大，群聚度大於平均分隔度的時候，所呈現出來的就是小世界網路。並非每一個小世界網路的平均分隔度與群聚度的趨勢都像 β 模型一樣，但是可以肯定的是，小世界網路具有高群聚度與低分隔度的現象。

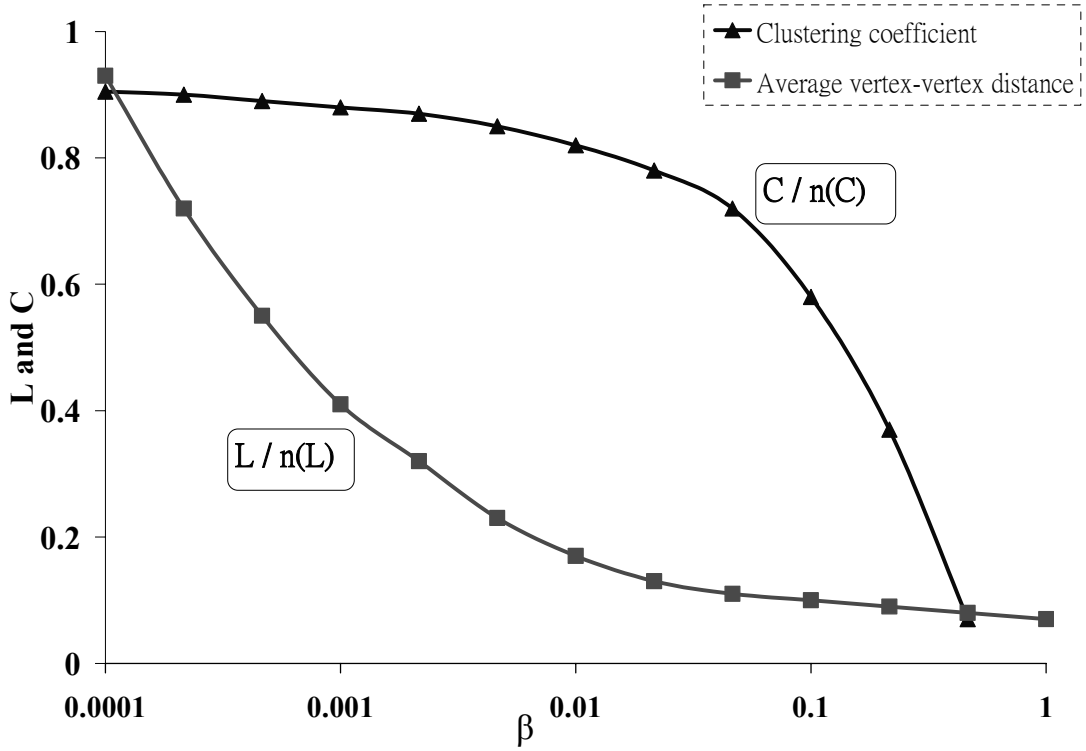

Figure 16. Watts 和 Strogatz 的 β 模型中的平均分隔度（平均個體與個體之間的距離）與群聚度的曲線圖（正規化後）

A3.3本模型在社會性的證明

A3.3.1　分配分身點的方法

在本節中將描述我們如何分配分身點，還有平均分身點數對於整個環境大小的影響。首先要決定平均分身點數，然後以平均分身點數來做常態分配，隨機分配給每個個體固定數量的分身點，所有個體的分身點總和除以個體的總數會等於其平均分身點數量。當決定了平均分身點數後，再決定個體數量，平均分身點數乘上個體數，就會等於整個環境中所有分身點的數量，就等於細胞自動機所有的晶格數量，由於我們在一個正方形的環境上做模擬，就會導出二維細胞自動機的長和寬，並且進行模擬。例如：平均分身點數為 4，個體數量為 10000 時，可以

計算細胞自動機的長和寬應為 200×200。所以由上面的步驟我們知道每個個體有幾個分身點，我們就針對細胞自動機上每一個晶格，將每一個晶格隨機分配給某一個個體，若該個體所應擁有的分身點數已經滿的話，就再重新隨機分配給另一個體，我們就可以將所有的晶格恰分配給一個分身點。

A3.3.2 分身點對分隔度與群聚度的影響

在測量分隔度（Degree of Separation or Diameter）時，大多採用計算該圖形的平均點對點的距離（Average vertex-vertex distance），在本模型中，由於個體所擁有的分身點不唯一，所以上面的點對點的距離，指的是個體與個體之間的距離。而計算群聚度的時候，則是某個體的所有鄰居中，鄰居和鄰居間真正存在連結的數量，除以鄰居兩兩之間可能發生的所有連結的數量。在計算群聚度的時候，要計算的是個體與個體之間的群聚度，所以必須要考慮到所有分身點的鄰居之間的群聚度，也有可能有某個體的兩個分身點，和另外一個個體的兩個分身點皆為鄰居的情況，舉例來說就是一對夫婦在同一公司上班。在 Figures 4、5 中可以發現，族群大小（population size）對群聚度和分隔度的影響不會很大，在此族群的大小指的是模擬的環境中的個體總數，而非分身點總數。在 Figure 4 中的可發現族群大小增加時，平均分隔度呈現對數成長，證明本模型也具有典型的小世界現象「當族群大小增加時，平均分隔度只呈現對數成長」。且也顯示了只要平均每個人有兩個分身點的時候，舉例來說每人每天都定點移動到兩個地方，公司和家庭，平均分隔度就會只剩下 2~6。

A3.3.3　本模型中所呈現的小世界現象

本模型隨著平均分身點數的增加，計算分隔度和群聚度的結果，在正規化後，與 β-model 相較之下，發現也有群聚度高於分隔度的現象，但是所得的群聚度曲線趨勢和 β-model 所得到的不一致，在 β-model 的群聚度曲線是呈現平緩最後陡降的趨勢，而本模型是陡降再平緩。在根本上，β-model 是依照 β 機率，隨機改變其原本有序網路上的連結，本模型則是以增加分身點的方式，讓原本每個晶格都是獨立一個個體，變成一個個體擁有好幾個晶格（分身點），其描述小世界現象的基本模型原本就很不同，若和後來 Watts 所提出的二維晶格加上捷徑（Shortcut）的方法比較下，其所代表的意義也不同，加上捷徑的方法保留了原本每一個晶格就代表一個個體的特色，但是隨機地加上一些遠連結，這樣的方法很容易可以描述經由電話或電子郵件所傳遞的訊息網路，但是仍和本論文所提出的概念上有很大的差距。本模型的分隔度與群聚度趨勢的一個合理解釋是，當分身點一多後，假若其中一個分身點在公司，一個在家庭，原本家庭和公司就是兩個不同的群聚，這兩群個體之間的群聚度很低，所以只要分身點一增加，群聚度就會大大降低，然後隨著分身點越多，越來越平緩，以 Figure 17 為例，一個個體若只有一個分身點，若周圍的分身點所屬的個體只擁有該分身點（也就是周圍個體都只擁有一個分身點，就是相鄰的那八個。），那麼這八個個體的群聚度是 12/C(8, 2)=0.43，但若該個體有兩個分身點的時候，若周圍十六個分身點皆屬於其他不同的個體，且這些個體也是只有一個分身點，這十六個個體的的群聚度就

只剩下 2×12/C(16, 2)=0.2，群聚度急遽減小。

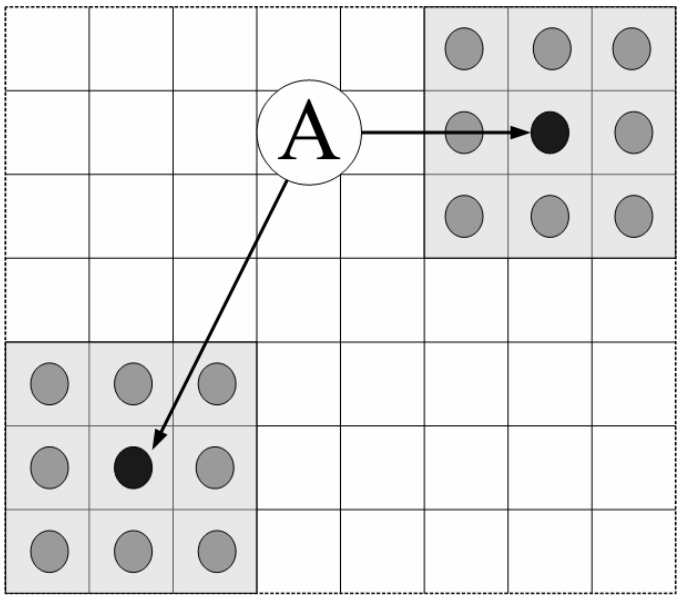

Figure 17. 在兩個分身點周圍的十六個不同個體的群聚度計算

A4 SEIR 染病進程與 R_0 參數特性的驗證

A4.1SEIR 染病進程

SEIR 模型是統計模型中的其中一種,本節將解釋在本論文中用來模擬 SARS 的 SEIR 模型狀態（Susceptible、Exposed、Infectious、Recovered）的轉變（見 Figure 7）：

S → E：當個體的疾病狀態為 S 的時候，若鄰居有 I 狀態的個體時，每天的染病機率為每天可能的次數（Population Interactions），乘上接觸成功的機率（Probability of Contact with Sick People），再乘上接觸後的傳染率（Probability of Catching Illness）。若該個體的某一分身點經過此三個機率決定被傳染後，則該個體與其所有分身點進入疾病狀態 E。

E → I：當個體的疾病狀態為 E 的時候，由於該個體為染病，但未被發現，代表該個體帶著病原暴露在社會中，其被發現的機率為一偵測率（Detected Rate），一旦被偵測後，則該個體進入疾病狀態 I。若以 SARS 為例，由於 SARS 病狀嚴重，所以在一定的期間內，一定會就醫，所以另一種可能就是經過一段時間後，進入 I 的狀態。在本論文所提出的模型中，一旦進入 I 狀態的個體，其代表的是進醫院治療，則該個體的社會活動力狀態會進入附錄 A2.4 所述的醫院隔離狀態，除了留在醫院的分身點外，其他所有分身點暫時不和鄰居互動。

I → R：I 狀態的個體，以死亡率（Mortal rate）決定該個體是否死亡，若沒

有死亡的話，則康復，康復的個體的所有分身點再度恢復與鄰居的互動。死亡的個體在本模型中則是以新的個體替代其所有分身點，所以隨著死亡人數的增加，本模型的總人口數也會不斷增加，R 狀態代表有可能是死亡或是康復。

R → S：通常康復的個體暫時不會染病，通常在本模型中都以兩週為週期（Recovered Period），康復個體在兩週後，可視為一全新的個體，也就是轉變為 S 狀態。若是該個體死亡，當新的個體替代死亡的個體後，則所有分身點立刻具有活動力，不必經過兩週的週期。

A4.2R_0 參數特性的驗證

本論文除了證明具有分身點的細胞自動機能呈現個體日常接觸網路的低分隔度與高群聚度外，本模型在第四章中也提供了和傳統流行病學中 R_0 參數的比較。在第四章的 Figure 10 中，本論文以平均傳染期等於 6（Tperiod=6）、平均分身點數等於 4（Avg. Mirror=4）、每日接觸次數等於 4（Ctime）的 R_0 曲線為例子，可以看出當 R_0=1.4 和 1.2 時，該接觸性傳染病均處於爆發的狀態，具有傳統流行病學中 R_0>1 的特性；當 R_0=1.0 時，可以發現傳染病的傳播到後面就已經開始漸漸收斂。當 R_0=0.8 和 0.6 時，則在早期就可明顯的發現疾病傳播的收斂。這樣的結果，證實本模型可呈現傳統流行病學中的 R_0 參數性質。但是也意味著這些參數也不能隨便設定。

經由徵詢流行病學專家，我們可以知道每一場傳染病爆發的平均傳染期，然後由於在本模型上的鄰居數固定，接觸率也固定，經由傳統的流行病統計模型算

出 R_0 參數後，我們給予恰當的兩個參數，平均分身點數和每日接觸次數，就可以推算出在模型中的傳染率。並非每一組參數都是恰當的，因為每一個參數對於本模型的參數敏感度不同。在本模型中傳染率的敏感度大於平均分身點數，所以當高估了平均分身點數的時候，相對的傳染率太小，可能有一流行病 R_0=1.2 或 1.4 以上，並不會像 R_0>1 時，呈現出爆發的傾向，而會收斂，像這樣的參數設定就是不恰當的。另一方面地，若平均分身數太小的話，那麼相對的傳染率就會太大，在 R_0=0.8 或 0.6 以下的時候，也會產生錯誤的傳染病爆發的結果。所以，在選擇要進行模擬的平均分身點數時，針對不同的平均傳染期，必須要謹慎選擇。經由實驗，在模型中可以找到類似 Figure 10 中的參數對應，只要針對每一個流行病學家所給的平均傳染期，我們就可以找出相對應的一組參數（平均分身點數, 每日接觸次數）來進行模擬（該粗體字組為 Figure 10 的參數）。

平均傳染期	**合乎 R_0 參數性質的(Avg. Mirror, Ctimes)對**
4	**(5.0, 4), (5.0, 3), (5.0, 2).**
5	**(5.0, 5), (5.0, 4), (5.0, 3), (5.0, 2),** **(4.5, 4), (4.5, 3), (4.5, 2).**
6 (SARS)	**(5.0, 5),** **(4.5, 5), (4.5, 4), (4.5, 3), (4.5, 2),** **(4.0, 4), (4.0, 3), (4.0, 2).**
7	**(4.5, 5),** **(4.0, 5), (4.0, 4), (4.0, 3), (4.0, 2),** **(3.5, 4), (3.5, 3), (3.5, 2).**
8	**(4.0, 5),** **(3.5, 5), (3.5, 4), (3.5, 3), (3.5, 2),** **(3.0, 4), (3.0, 3), (3.0, 2).**
9	**(3.5, 5),** **(3.0, 5), (3.0, 4), (3.0, 3), (3.0, 2).**

A5 SARS 模擬

A5.1 進行 SARS 模擬的步驟與方法

在本論文的 SARS 模擬實驗中，我們模擬在 2003 年初在台灣與新加坡爆發的 SAR 疫情，我們以輸入病例作為感染源，並且考慮到各國公衛政策的實施，我們比較模擬的結果與 SARS 疫情中每日的通報人數，來證明本論文所提出的方法適用於 SARS 這樣的近距離接觸傳染病上。本節將介紹本論文中進行 SARS 模擬的步驟，包含疫情資料的取得與種類、模擬的過程、模擬的結果比較等部分。

A5.1.1 建立適合的流行病模擬模型

在模擬的一開始，必須要先建立一個合乎該傳染病傳染特性的社會模型，並且採用適當的疾病狀態轉變來建立模型。以 SARS 為例，我們觀察到 SARS 是近距離接觸傳染，所以我們可以採用具有分身點概念的細胞自動機來進行模擬；SARS 患者在發病之後，到處移動或就醫的現象，容易造成疾病迅速的擴散，而這些現象在急性傳染病中是相當重要的，包含尚未被醫院隔離，到處就醫的行為會造成大幅度的院與院之間的傳播。所以我們採用 SEIR 疾病狀態的轉變來進行模擬。

A5.1.2 環境參數（社會性的參數、流行病學參數）的初始化

在確定了模型與流行病的狀態轉變後，我們分社會學與流行病學兩方面來初

始化整個模型的環境。在流行病學方面，我們要確定的是該疾病的 R_0 參數與平均傳染天數。以 SARS 為例，R_0 參數約為 2.6~3.0，每個疫情國的平均傳染天數不同，但大約為 4~6 天。根據我們所要模擬的疫情國家的平均傳染天數和 R_0 值，可以利用第四章所做的 R_0 參數分解，與在附錄 4.2 中所找出來的合理 R_0 值來找出適當的兩個社會學參數，每日接觸次數和平均分身點數。依據該兩個參數，我們可以建立起疫情模擬的底層社會網路，並推算出在本模型中，個體傳染給個體的傳染率，以進行模擬。其他的流行病環境初始資料則有潛伏期天數、得病後免疫與否、死亡率與超級傳染者的比例與致病力等等。

A5.1.3 流行病的疫情資料輸入與模擬的步驟

建立模型並且初始化模型後，就可以開始進行模擬。模擬時程以一天為時間單位，在模擬的時候，由於 SARS 起源於中國大陸廣東省，其他國家皆為外來病例，所以本模型以各國衛生機構所公布的輸入病例來誘發感染，每個病例的特徵包含其在模擬時程中的第幾天輸入，和其為傳染期輸入或潛伏期輸入等。除了參照各國的輸入病例資料外，為了實際模擬各國的疫情曲線，我們仿照各國所執行的公衛政策，在本模型上制訂出相對的公衛政策的實施，以觀察本模型所模擬的疫情曲線是否能與在疫情後各國修訂過後的疫情曲線相符。在模擬的過程中，就依照時程加入輸入病例，並且啟動公衛政策及其效度。

A5.1.4　真實疫情與疫情模擬的比較

經由模擬之後，我們統計每日由 E 狀態轉變為 I 狀態的人數，也就是被送進醫院隔離的人數，在與實際資料比較上，就是疫情國家每日所通報為 SARS 病例的人數。經由流行病的模擬，我們可以不斷重複模擬該國疫情，並且和實際的每日資料做曲線比對，在模擬的結果中（Figures 11, 12），本模型所模擬出的曲線皆能與在新加坡與台灣兩個國家所爆發的疫情曲線相符。

A5.2公衛政策的比較與討論

A5.2.1　模擬公衛政策的目標

在 2003 年 SARS 爆發期間，最受注目的除了每日的通報人數，就是各國政府所實施的公衛政策，共有「居家隔離」、「醫院隔離」、「禁止醫院探訪」、「全民帶口罩」、「全民量體溫」等，每種公衛政策所實施的目的與對象皆不同。居家隔離的目的是防止可疑病患（S 或 E）造成擴大感染；醫院隔離是防止染病者（I）到處散播病毒；禁止醫院探訪的目的是隔絕一般民眾因為醫院探訪所造成的院內感染或將病毒帶出院外；全民帶口罩則是雙向地防止染病者（I）傳染，與一般大眾（S）被傳染；全民量體溫則是篩檢措施，避免未知的 SARS 病患（E）到處散播病菌。

諸項公衛政策實施的效果與所耗費的成本皆有不同，很難在傳染病發生的同時，不斷地重複討論公衛政策的效度與成本。且在 SARS 期間，多項公衛政策同

時遍施全國，有可能少數染病者逃過隔離檢疫措施，或被錯認為是染病者的情況，在實際上也難以做統計來進行公衛政策的效果討論。利用建模模擬的方法，不僅能夠反覆地模擬公衛政策以觀察其效果，加入所耗費的社會成本考量，更能夠模擬單一公衛政策實施的效果，除了討論單一公衛政策不同實施程度的效果，也容易比較政策與政策之間的效度。

A5.2.2 模擬公衛政策的方法

在單一公衛政策比較的部分，由於該公衛政策實施的效果與普及率皆不同，例如在民主專制政府的國家越南可以強制實施醫院關閉的法令，禁止所有進出醫院的行為，就能有效地將病患控制在院內，在一般國家則必須要考慮到人權問題，所實施的法令的強制力可能就不如專制國家來的高。或全民戴口罩的命令，也要考慮民眾接受戴口罩政策的接受率和口罩本身的效果。所以，針對每個公衛政策，我們必須要制訂其實施的普遍率與該公衛政策的有效率。我們可以藉由反覆模擬，來討論普遍率（在全人口中實施的比例）與效率（公衛政策的防制效果）兩個參數的敏感度，以選擇適當的參數組合來做疾病模擬。所以，在討論單一公衛政策的時候，我們可以在模型上依循某個國家的輸入病例時程，輸入病例來誘發流行病的爆發。本論文在做公衛政策模擬的時候，採用新加坡八個輸入病例的時程，並在新加坡政府啟動大量公衛政策，也就是在本模擬時程中的第二十四天，啟動單一的公衛政策，反覆地模擬實驗，改變其普遍性和效度，觀察該公衛政策對疫情散播的影響。