台灣二ＯＯ五年國際科學展覽會

科　　別：醫學與健康科學

作品名稱：對抗無尺度流行病傳染之新方法

得獎獎項：大會獎佳作

學　　校：臺中縣私立明道高級中學

作　　者：梁辰瑋

評語與建議事項：

所應用之程式，應考慮不同型態之傳染病的傳染路徑，以有效化程式所計算形成之數據。

作者簡介

梁辰瑋，1989 年 6 月生於台中市，今年十六歲，目前就讀於明道中學高中部(直升班)一年級。父親任教於逢甲大學環工系；母親任職於自來水公司第四區處；哥哥就讀於台灣大學生工系二年級，曾代表我國參加 2004 年美國 Intel 國際科展，並獲獎。由於受到父親與哥哥的薰陶，從小就喜歡科學與善於思考，小學電腦課被老師指定為助教，國中常被理化老師說喜歡打破砂鍋問到底。最喜歡科目是物理，目前參加國立中興大學物理系「高中物理資優生」為期一年的研習班。另外曾學過多年小提琴，因此平常喜歡聽音樂，也喜愛打籃球。一年多來，隨美籍之個人英文教師勤學英文，已具備相當的英文基礎，期盼未來能任職於美國 NASA，探索宇宙新知。

摘 要

流行病的傳染過程如同一個無尺度網路，但較一般無尺度網路有著更多的變數而明顯差異，因此無法直接應用一般的無尺度網路模式來描述其傳染途徑。我建立一個新模式「無尺度流行病模式」，經由比較模擬結果與疾病管制局的數據，證實此「無尺度流行病模式」是正確與確切可用，且適用於短期暴發性傳染病與長期流行病。SARS 案例研究結果，顯示影響 SARS 疾病傳染因子的大小是：$\psi > m > \gamma$。其中降低ψ值可使 SARS 確定病例至 5 月 31 日止降爲 143 人(減少確定病例 190 人，相當於減少死亡 21 人)；僅提高防疫使$\gamma = 5$，亦可使確定病例減至 307 人(減少確定病例 26 人，相當於減少死亡 3 人)。因此強化隔離措施以減少傳染天數最爲重要，且可以有效控制每日 SARS 新增病例，避免發生高侵襲率的現象。HIV/AIDS 案例研究結果，獲知採用ψ值來進行月份模擬，則至 2005 年 12 月 HIV(+)與 AIDS 分別爲可減少 2,715 與 285 人。而進行年度模擬結果，則至 2014 年底 HIV(+)與 AIDS 分別爲可減少 41,936 與 5,328 人。無尺度流行病模式可以協助所需警戒的程度與政策決定的計畫結果。因此無尺度流行病模式在幫助政府評估社會經濟成本與健康憂慮上的有用之工具。當面臨一個全然無知的新病毒的侵襲時，如何減少死亡與傷害人數？是本研究之最終目的。因此，本研究結合了流行病、無尺度網路與灰預測，建立面對病毒侵襲，一個確切可行的對抗無尺度流行病傳染新方法，並詳細說明運作流程。

Abstract

The course of epidemic infections resembles a scale-free network. However, they are different due to more variables in the epidemic infection. Therefore, the model of scale-free networks is not enough to satisfy the reality epidemic infections. In this study, I propose a new the Scale-Free Epidemic Model. Comparison of the simulation results with Taiwan CDC report data for SARS and HIV/AIDS cases show that the Scale-Free Epidemic Model is accurate and useful. This model can be used in the short-term outbreak of infectious diseases and for the longer-term epidemics. In the SARS case study, the results show that the sequence of effect of the epidemic factors was: $\psi > m > \gamma$. The SARS confirmed cases would decrease to 143 cases (reduced 190 confirmed cases or 3 death cases) calculated to May 31, 2003, if the average infection time was reduced to two days (an optimum value of ψ). Therefore, vigorous action in isolation quarantine and treatment for SARS cases is most effective policy; the number of new cases and the attack rate would also decrease. In the HIV/AIDS case study, the simulation results of the Scale-Free Model indicates that the reduced numbers of HIV(+) and AIDS in the monthly simulation calculated to December 2005 are 2,310 and 361 and the annual simulation by December 2014 are 27,161 and 3,710. The Scale-Free Epidemic model can help determine the level of caution needed and the projected results of policy decisions. Therefore it is a useful tool in assisting the government to balance socio-economic and health concerns. The fight against a new epidemic and how to reduce the number of deaths is the main purpose of this study. So, a new method to fight against epidemics is proposed. Detailed procedures of this method are explained.

一、前 言

(一)、動機

閱讀到”Scientific American”雜誌上一篇討論無尺度網路(scale-free network)文章[1]，內容談到現行的網際網路(Internet)甚至我們日常的許多行爲，都是可以歸納爲無尺度網路的一種，同時由該文章的延伸閱讀[2,3]，更獲知無尺度網路之冪次定律分布(the power law distributions)可同等應用在探討細胞(cellules)、電腦(computers)、語言(linguistics)與社會(society)之上。同時，無尺度網路知識未來更可以在運算(operation)、醫學(medicines)、商業(business)上有著眾多可發揮之處。如此，引起我對於應用無尺度網路知識於理解流行病(epidemic)的傳染(infection)，甚至進一步的預防(preventive)與抑制(suppression)，有著深厚的興趣，便著手蒐集相關資料，投入其中進行研究。

(二)、目的

流行病的傳染途徑亦如同一個無尺度網路傳播，但與前述的網路有著諸如無迴路(loopless)、單一傳入(single-incoming)但多方傳出(multiple-outgoing)、非自組與非線性成長(non-self-organization and non-linear growing)、節點隔離或死亡(node isolation or death)、個體內變異性(intra-individual variability)、種族差異(racial difference)等等許多的不同，因此無法直接應用一般的無尺度網路來描述其傳染途徑，而必須將這些新參數(parameters)或特徵(characteristics)納入網路中，重新建立一個屬於流行病的無尺度網路理論，這便是本研究第一個目的。

另外，對於一全新病毒所造成的流行病傳染初期，往往造成民眾重大的死亡與恐慌(如 2003 年的 sever acute respiratory syndrome, SARS)；然而在傳染初期，疾病防治單位所擁有相關資料既少又匱乏，不足以迅速拿出有效的對策來減少死傷。灰色理論(Gray theory)具有僅使用少量數據就可進行預測(predication)、關聯性分析(relational analysis)與決策(decision marking)的能力。因此，期望能在流行病傳染初期，運用所建立「流行病傳染無尺度網路模式」結合灰色理論來預測傳染途徑，並從中尋求有效的預防與抑制策略，是本研究第二個目的。

二、方 法

(一)、研究程序(Study process)

閱讀相關書籍、文獻與蒐集資料是本研究最先進行並一直持續的工作，從中獲知流行病學之 Reed-Frost 模式與無尺度網路之 Barabási-Albert 模式結合，經必要的修改，可以建構一套全新的流行病傳染無尺度網路模式。再將蒐集自各處的傳染病資料代入新模式中，及應用灰色理論之 GM(1,N)與 GM(1,1)模式來獲取各參數在傳染過程關聯性與預測式，進行必要的模式驗證。最後進行以 SARS 爲案例的研究，求取不同灰色決策下的結果，以獲得最佳預防措施，並評估成效。詳細研究流程如圖 1 所繪。

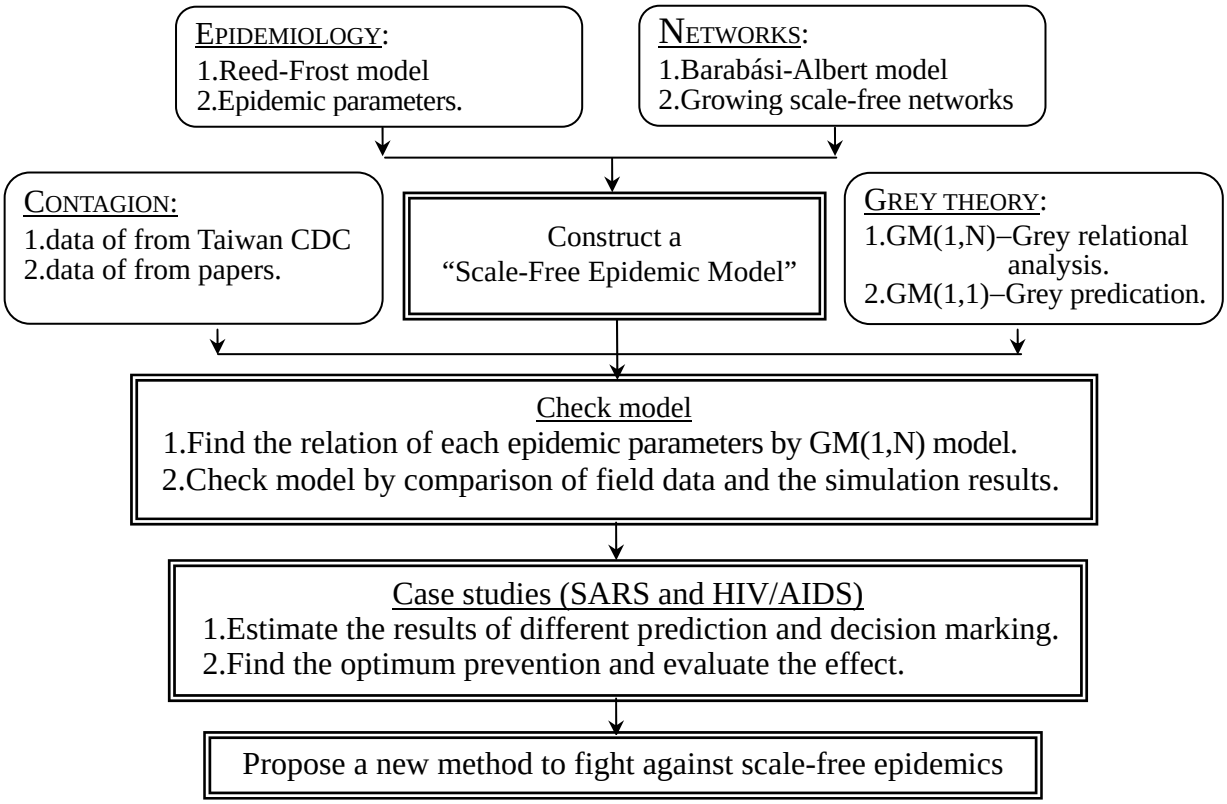

Fig. 1. Study procedures

(二)、網路(Networks)

一般可將網路區分爲：1.小世界網路(small-word networks)，2.隨機網路(random networks)與 3.無尺度網路(scale-free networks)三種[2,3]。其中小世界網路就如以教堂爲中心的區域交誼網，範圍小與連結(link)單純。其中節點的連接度分布(connectivity distribution, $P(k)$)可採用(1)式之 WS 模式(The Watts-Strogatz model, WS-model)[2,4]。

$$P(k)=\sum_{\alpha=0}^{f(k,K)} C_{K/2}^{\alpha}(1-p)^{\alpha}\, p^{K/2-\alpha}\,\frac{(pK/2)}{(k-K/2-\alpha)!}e^{-pK/2},\ \text{for } k\geq K/2 \qquad (1)$$

其中 k 爲連接度(degree)；K 爲鄰席數(number of neighbors)；$K/2$ 表示單邊鄰席數(neighbors on either side)；p 爲節點連接機率(probability of connection)；C 爲叢聚係數(clustering coefficient)。

隨機網路有如公路系統，在此系統中節點的連接度分布($P(k)$)遵守鐘形的(belled)帕松分布(Poisson distribution)，且大部份的節點具有的連接數都差不多[1-3]。(2)式爲 EP 模式(The Erdös-Rényi model, ER-model)[2,3,5]中隨機網路的連接度分布式。

$$P(k)=\begin{cases} C_{N-1}^{k}p^{k}(1-p)^{N-1-k} \\ P(k)=\dfrac{e^{-\bar{k}}\bar{k}^{k}}{k!} \qquad \text{for large N} \end{cases} \qquad (2)$$

其中 N 爲節點總數(total number of nodes)；$\bar{k}=p(N-1)$ 爲平均連接度(average degree)。

無尺度網路具有集散點(hub)，系統中節點與節點的連接度分布($P(k)$)遵守冪次定律分布(power law distribution)，其中大部份的節點只有具少數連接，但有些節點的連接數

(numbers of connection)卻十分龐大[1-3]。(2)至(5)式爲 BA 模式(The Barabási-Albert model, BA-model)[2,3,6,7]中無尺度網路的機率、連接度分布式與連接成長($k_i(t)$)爲

$$P(k) \propto k^{-\gamma} \tag{2}$$

$$P(k) = \begin{cases} \dfrac{k-1}{k+2} P(k-1) & \text{for } k \geq m+1 \\ 2/(m+2) & \text{for } k = m \end{cases} \tag{3}$$

具有

$$P(k) = \frac{2m(m+1)}{k(k+1)(k+2)} \tag{4}$$

$$k_i(t) = m\left(\frac{t}{t_i}\right)^{1/2} \tag{5}$$

其中γ爲冪次(exponent)，m爲新節點與舊節點連接度，t與t_i分別爲時間與引入(introduced) i節點的時間。

流行病傳染亦爲無尺度的成長網路(scale-free growing networks)，此類相關文獻計有 Dorogovtesev 等氏[8]探討成長網路結構的 BA 模式正解，計算成長位置的最初特性與求取精確的 $P(k)$，以及位置(s)與瞬時(t)之平均連接度$\bar{k}(s,t)$；Dorogovtesev 等氏[9]證實無尺度網路之 $P(k,t)$具有共通尺度(generic scale)－斷絕(cut- off)於$k_{out} \sim t^{\beta}$，其中 t 與 β 分別爲網路大小與同 $P(k) \sim k^{\beta}$ 之冪次[6]，並提出無尺度成長網路之尺度關係式(the scaling relation form)與連接度分布式；.Klemm 與 Eguíluz[10]提出無尺度網路節點之成長與衰退模式(growth and deactivation model)，在時間不連續動態(time-discrete dynamics)下的任何階段，由於新節點的加入，使一舊節點(node j)衰退(deactivate)的機率(deactivation probability, $D(k_j)$)式；Klemm 與 Eguíluz[11]介紹一簡單動態模式(a simple dynamical model)，適用於具有連接度無尺度分布(scale-free distribution of degree)與小世界效應(small-word effect)共通特點(generic features)之實際網路。

另外，Albert 與 Barabási[12]探討如新節點的加入與連結重置等局部事件(local events)所引發網路之成長與發展。其中依據連續定理(continuum theory)來預測尺度函數(scaling function)與指數(exponential)二種機制，並導出以節點之新連接機率(p)與終點機率(q)求取$k_i(t)$之方程式

$$k_i(t) = \left[A(p,q,m) + m + 1\right]\left(\frac{t}{t_i}\right)^{1/B(p,q,m)} - A(p,q,m) - 1 \tag{6}$$

其中 $A(p,q,m) = (p-q)\left(\dfrac{2m(1-q)}{1-p-q}+1\right)$ and $B(p,q,m) = \dfrac{2m(1-q)+1-p-q}{m}$ (7)

雖然流行病傳染亦具有前述之網路成長(growing)、衰退(deactivate)、損壞(crash)、高叢聚(high clustering)與小世界效應(small-word effect)等特性，但各文獻所提出之無尺度網路數學式，仍無法直接全然描述流行病傳染途徑，必須再更深入的考慮與納入流行病的一些獨特的參數或特徵，方足以完美的建立流行病無尺度網路模式(the epidemic scale-free network model)，而這正是本研究之重點工作。

(三)、流行病(The Epidemiology)

感染可分爲共同感染(common source infection)與連鎖感染(propagated infection)：

1.共同感染

流行曲線(epidemic curve)向右偏斜，且涵蓋的時段比較短，通常是一、二個潛伏期(incubation period)，大都屬暴發性流行(outbreak)，如食物中毒(food-borne)。由於潛伏期與誘導期(induction period)相當短，易於探尋流行原因。一般共同感染是隨機發生(random occurrence)，且具帕松分布(Poisson distribution)[13]，而發生機率(probability, Pr($A=a$))可由(8)式求得。

$$\Pr(A=a)=\frac{e^{-\mu}\mu^{a}}{a!} \tag{8}$$

其中 A 指病例(cases)在某特定人時(person-time)量下可變動數(variable number)；μ 爲病例期望數(expected number)。比較(8)與(2)式，獲知共同感染途徑如同隨機網路。

2.連鎖感染

流行曲線向左偏斜，且涵蓋時段往往超過二個潛伏期，傳染病(contagion)直接(immediate)或間接(mediate)由患者(sickness)或帶原者(carrier)傳染(contagion)至其他可感染宿主(susceptible host)。除人對人傳染外，也包含蟲媒(insect)、體液(body fluids)的傳染。連鎖感染會受到傳染間代(generation time)、集團免疫力(herd immunity)與二次侵襲率(secondary attack rate)的影響。本研究欲建構的流行病無尺度網路模式，是用以解析連鎖感染。

連鎖感染如僅限於人對人的傳遞，其流行狀況較接近 Reed-Frost 模式的條件，條件如下：(1).該疾病(disease)僅由發病者經特定途徑傳染於可感染宿主；(2).任何可感染宿主受感染後，僅在下一傳染代(generation)發病(disease onset)並具傳染力(infectious)，但在此傳染代後，不再具有傳染力；(3).每一個體(person)在每一個傳染代與任何個體的有效接觸機率(effective contact rate)是固定的；(4).每一個體必須與族群(population)以外的任何人隔離(isolation)；(5).以上條件在流行期(epidemic stage)必須成立。Reed-Frost 模式[13,14]如下：

$$H_{t+1}=S_t\left(1-q^{H_t}\right) \tag{9}$$

其中H_t與H_{t+1}爲第t與t+1 代的發生病例數(numbers of incident cases)；S_t爲第t代可感染宿主數；$q=1-C_R$ 爲未被新發病例接觸的機率，其中C_R爲接觸率(contact rate)，而接觸率可由啓始可感宿主數(S_o)與流行結束殘存可感宿主數(S_m)求得，列於(9)式。

$$C_R=\frac{\ln(S_m/S_o)}{S_m-S_o} \tag{10}$$

另外，一些與本研究有關之流行病學參數[13~15]也將被用於建立流行病無尺度網路模式中。這些參數爲：(1).侵襲率(attack rate)、(2).發生率(incidence rate)、(3).死亡率(mortality)、(4).致死率(fatality)與(5).集團免疫力(herd immunity)等。

(四)、灰色模式(Grey model)

本研究需逐一探討各流行病學參數在流行病無尺度網路模式中伴演的角色與重要性，即需進行各參數對傳染病傳播之關聯性分析(relational analysis)，而灰色理論之GM(1,N)模式的運用可以達到此目的。本研究運用 GM(1,N)模式的流程如圖 2 所繪。

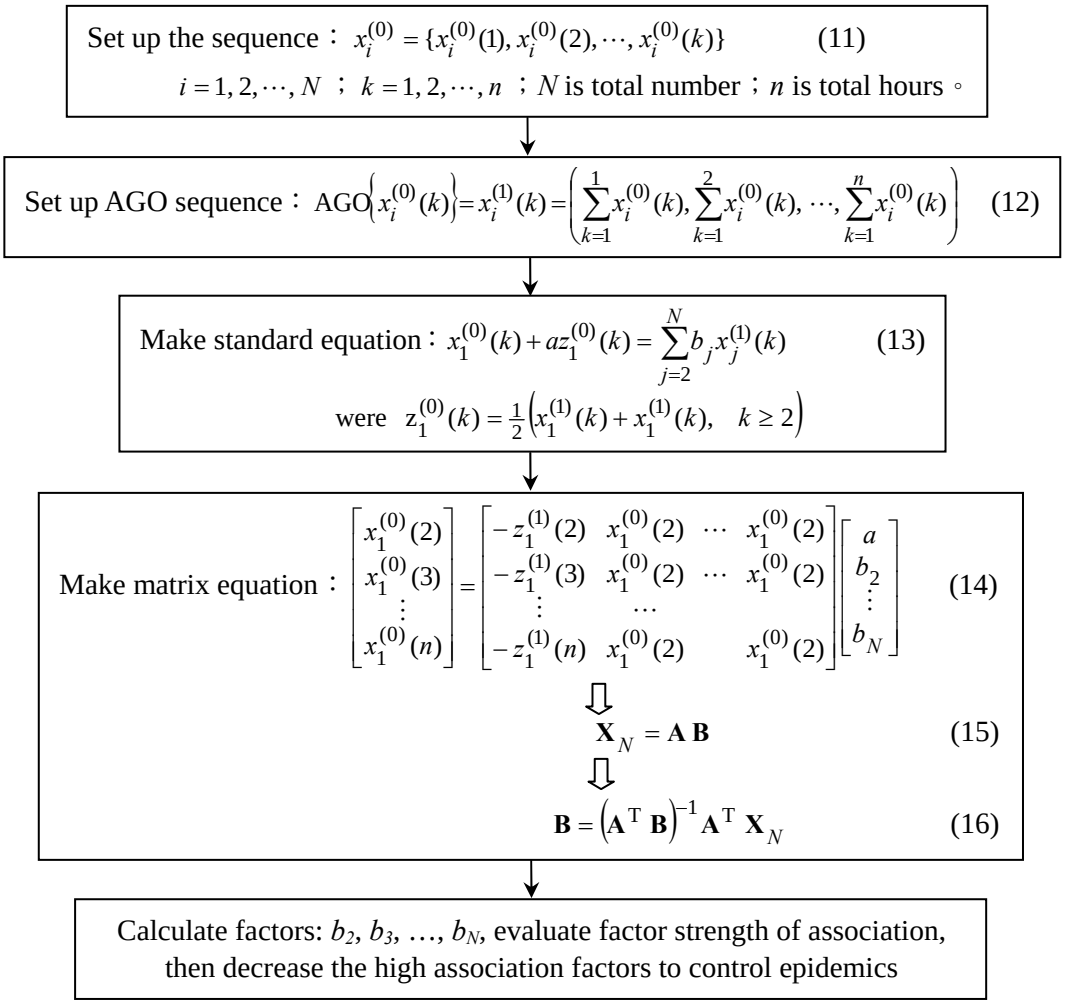

Fig. 2. GM(1,N) model operation procedures of this study.

不同於 GM(1,N)模式的多變數(參數)輸入與關聯性運算，GM(1,1)模式僅輸入單一變數來運算，求出該變數序列各個元素之未來動態狀況，達到預測目的。而預測的結果，是訂定決策的依據，即所謂灰色決策(gray decision marking)。研究中，依據 GM(1,N)模式運算結果，選定幾個關聯性較大的參數，適當調整其值並代入所建立「流行病無尺度網路模式」，算出調整後的發生病例數，如此重新建立原始序列，再依運用圖 3 之程序，求取發生病例數預測方程式(即所謂灰色模式建構(gray model construction))，應用此方程式進行灰色預測，以獲得未來之可能結果。若滿意此結果，則輸出調整後各參數值，並訂定防範傳染策略；若不滿意則重回調整參數值之步驟，直到滿意為止。值得一提的是GM(1,N)與 GM(1,1)模式運算所需之軟體，隨書[16,17]贈送。

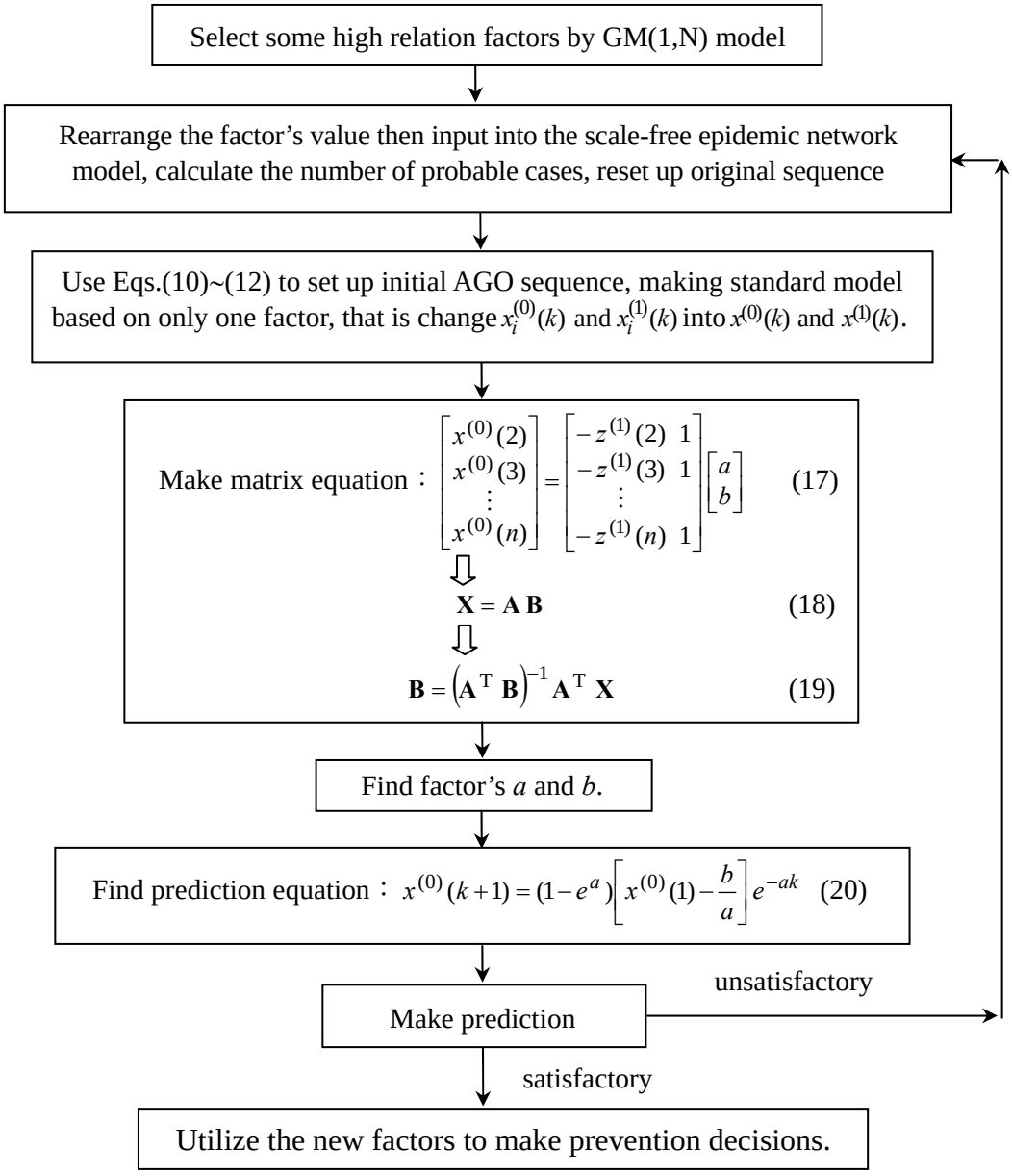

Fig. 3. GM(1,1) model and gray decision marking operation procedures of this study.

三、結果與討論

(一)、流行病無尺度網路模式建構(Epidemic scale-free network model construction)

如同前面所言，流行病傳染途徑雖亦爲一無尺度網路傳播，但仍有如無迴路、單一傳入但多方傳出、節點隔離或死亡、個體內變異、種族差異等的不同，因此無法直接應用一般無尺度網路數學式，而必須將流行病參數或特徵納入網路中，重新建立一個新的無尺度網路模式。基於此，本研究嘗試繪出足以完整描述流行病的傳染途徑的無尺度網路圖(圖 4)。圖中，流行病的無尺度網路區分爲二階段：尚屬於地方性(endemic)的流行前期(pre-epidemic stage)與已達大流行(pandemic)的流行期(epidemic stage)。另外，傳染過程中也會出現不同層叢聚(multiple stratum clusters)與突變病例(sudden change cases)等，因此傳輸途徑與小世界、隨機或無尺度網路截然不同[1~12]。

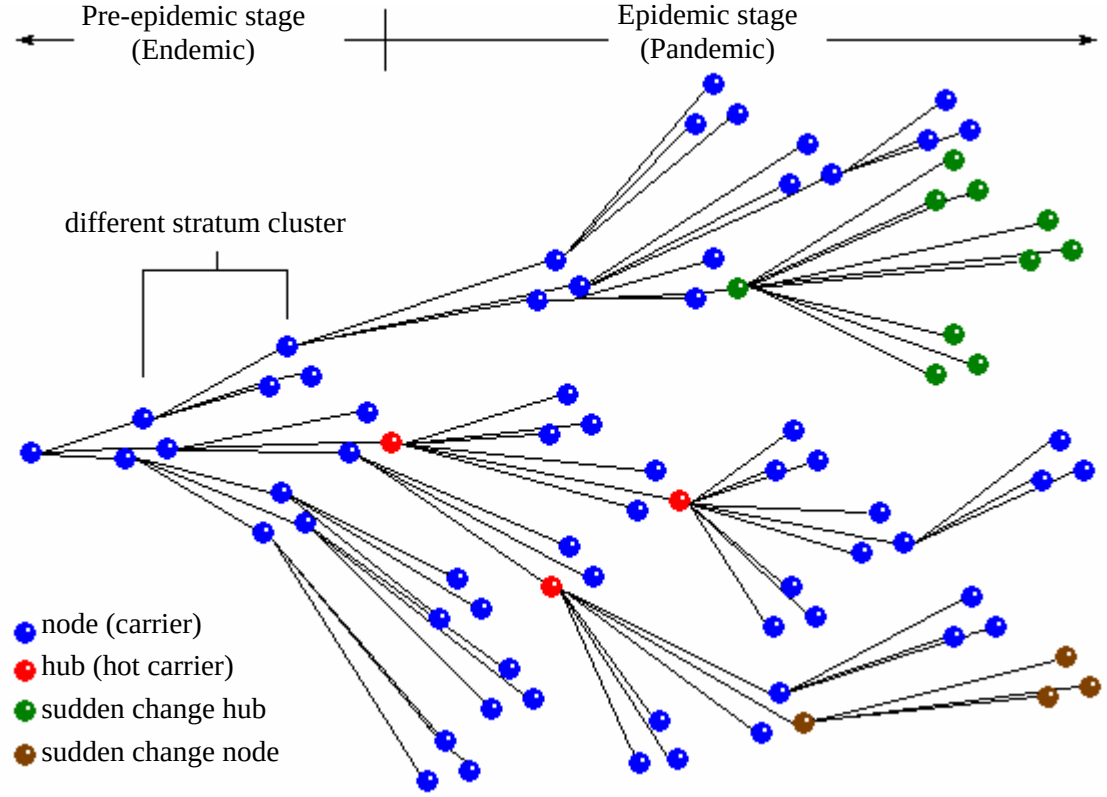

Fig. 4. The schematic structure of a epidemic scale-free network

流行病無尺度網路具有：(1).侵襲率隨發病或帶原時間而變化，(2).病患因隔離、治癒或死亡而不再傳染疾病，(3).病毒具有不同之突變率，且突變後之毒性可能改變，但不會改變其傳染途徑，(4).侵襲率隨集團免疫力提高而降低。考量上述特性，則本研究建立「無尺度流行病模式」如下：

$$k_i(t) = (m+1)\left(t - t_i + 1\right)^{m/(2m+\gamma)} - (m+1), \quad i = 1, 2, 3, \cdots, n \tag{21}$$

$$\text{where } m = A_t S_t - A_{t-1} S_{t-1} \quad \text{and} \quad \begin{cases} \gamma < 2 & \text{at high attack rate} \\ 2 < \gamma < 4 & \text{at middle attack rate} \\ \gamma > 4 & \text{at low attack rate} \end{cases} \tag{22}$$

$$N_n = N_0 + \sum_{i=1}^{n-1} \sum_{t=t_i}^{n-1} \psi \left[(m+1)\left(t - t_i + 1\right)^{m/(2m+\gamma)} - (m+1) \right] \tag{23}$$

$$\text{where } \psi = \begin{cases} 0 & \text{if the case is death, recoved, or in quarantine.} \\ \text{variable} & \text{if it is function of infection.} \\ 1 & \text{elsewhere} \end{cases} \tag{24}$$

其中N_n爲累積病例數；k_{t_i}爲時間t_i發病之病患在時間t之增加病例數；γ爲無尺度傳染冪次，其值爲$2 \le \gamma \le 3$ [6]；ψ爲病患隔離程度，其值可以爲常數或變數；$(t - t_i + 1)$爲發病後天數；A_t與S_t分別爲在時間t之侵襲率與隔離人數；N_0爲起算日病例數。

比較「無尺度流行病模式」與 Reed-Frost 模式之差異，本模式不僅可求出每一時間的病例增量(k_{t_i})，並可以預測未來時日的累積病例數，足以做爲一預估模式；而 Reed-Frost 模式，則僅爲事件後由固定接觸率配合可感染宿主數來計算病例數。

(二)、案例研究一：SARS (Case study-I: SARS)

1. *2003 年台灣 SARS 傳染(SARS epidemics in Taiwan in 2003)*

台灣 2003 年 3 月出現第一例境外移入 SARS 病患後，開始一段為期三個月的流行期，造成相當的恐慌與死傷。2003 年台灣 SARS 流行可分二個階段[18]，第一階段自 3 月初至 4 月初，特別為每日病例數少於五例，都有清楚的接觸史與感染區旅遊史；第二階段自 4 月中至 6 月中，經和平醫院發生大規模院內感染以及之後散播至台灣北部、南部，到 6 月底才停止流行。其中，台北地區 3~4 月間 SARS 傳染來源及途徑之分析如圖 5 所繪，包含境外移如與醫院內感染。

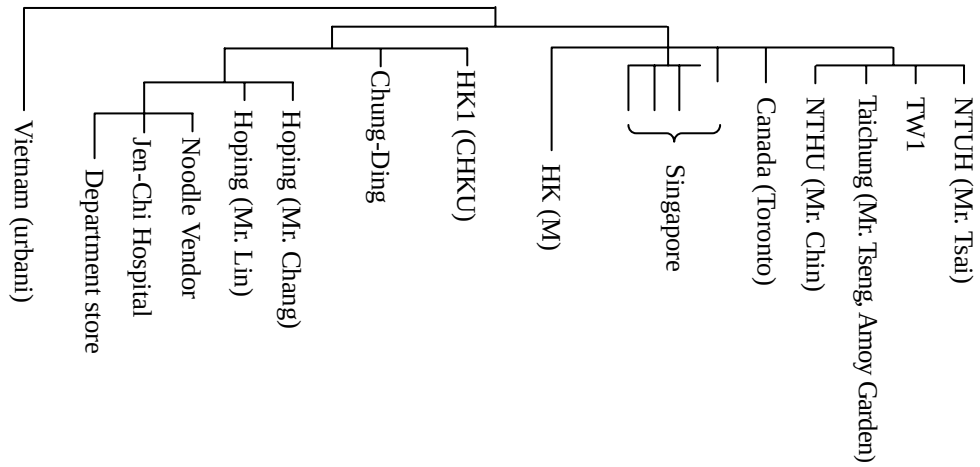

Fig. 5. Phylogenic tree analysis of SARS CoVs from three SARS Clusters in Taipei [18].
(*Source*: Memoir of Severe Acute Respiratory Syndrome Control in Taiwan, September 2003)

一些 SARS 病毒感染重要資料如下[18-21]：

(1) 台灣地區 SARS 病例 90%來自醫院內感染，4 月下旬醫護人員感染率達 32%。
(2) SARS 病毒感染潛伏期 3~10 天，沒有證據顯示潛伏期的感染者會將病毒傳給他人。
(3) SARS 病毒複製於發病後 7~10 天，病毒量達到高峰，是最危險的傳染期。
(4) 發病後三週，90%病人體內即無病毒。
(5) SARS 病人發燒消退後 10 天即不具感染力，可解除隔離。
(6) SARS 病人的抗體反應比一般傳統病毒感染延遲約 1 至 2 週。
(7) 高危險群或 A 級接觸者約有 0.22%的可疑感染，另有約 2%為無症狀的感染者。
(8) SARS 通報病例中，可能病例 58%與 SARS 病毒有關。

另外，本研究將 2003 年 SARS 在台灣傳染期間，由疾病管制局統計發佈之死亡病例數、生還數、可能病例數、累積可能病例、累積確定病例、累積死亡病例，蒐集繪整繪於圖 6(a)中，以瞭解每日的疫情變動。圖 6(b)則繪出 3 月 27 日至 6 月 15 日間每日隔離與解除隔離總人數，圖中因 4 月 13 日起改變統計方式，所以隔離總數略為下降；另外 5 月 21 後之隔離與解除隔離人數，則為 A 級與 B 級隔離數之和(因未取得該時段疾病管制局之數據，資料改用來自當時之聯合報)，因此隔離人數呈現明顯變動。

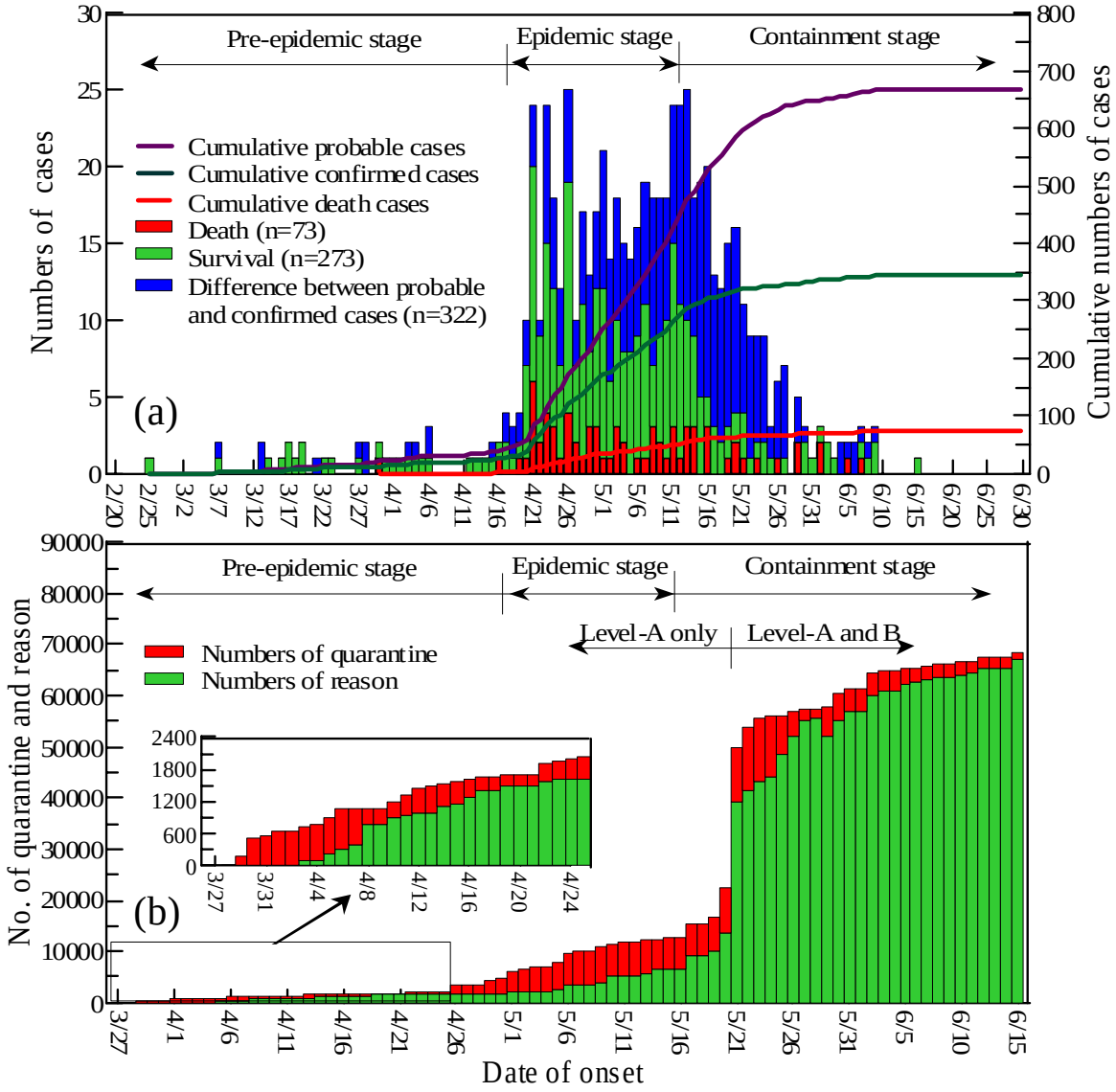

Fig. 6. (a) Epidemiological curves of SARS cases and (b) numbers of quarantine and reason for SARS in Taiwan in 2003. (*Data from*: The publications and the reports of Center for Disease control [18-21], Department of Health Web [22] and Union Daily [23], Taiwan)

定義每日之累積確定病例與累積隔離人數之比值爲侵襲率(attack rate)，每日之累積死亡病例與累積隔離人數之比值爲死亡率(mortality)，以及每日之累積死亡病例與累確定病例之比值爲死亡率(fatality rate)，而繪製出圖 7 之各比率動態變動圖。圖 7(a)與(b)顯示侵襲率與死亡率之動態變化非常近似。值得注意是 3 月 27 日至 4 月 7 日間侵襲率與死亡率均呈現明顯的上升趨勢，意味著 SARS 有高傳染力與致死力，若未採取有效防疫措施，恐暴發大流行，而之後也確實出現和平醫院暴發的大流行。因 4 月 8 日之後採較嚴格的隔離與封院措施，使侵襲率與死亡率呈現不同階段式的下降。

另外，由於 SARS 病毒有 3~10 天的潛伏期與約 2 週的發病期，因此致死率的上升會較侵襲率與死亡率落後 2 週或更多(參見圖 7(c))，且由於接連暴發多起院內感染事件(和平醫院、仁濟醫院、台大醫院、高雄長庚醫院等)，使致死率自 4 月 24 日起維持在 21%左右。6 月 10 日台灣地區 SARS 傳染已有效控制，所以侵襲率與死亡率已明顯下降

與持平於 0.5%與 0.1%左右。圖 7(d)爲每日確定病例與可能病例之比率變化，其中 3 月 28 日至 4 月 2 日與 4 月 17 日至 4 月 22 日兩上升趨勢，一則配合反映侵襲率與死亡率的上升，另一則反應合平醫院暴發大流行與封院後確定病例突增的現象。

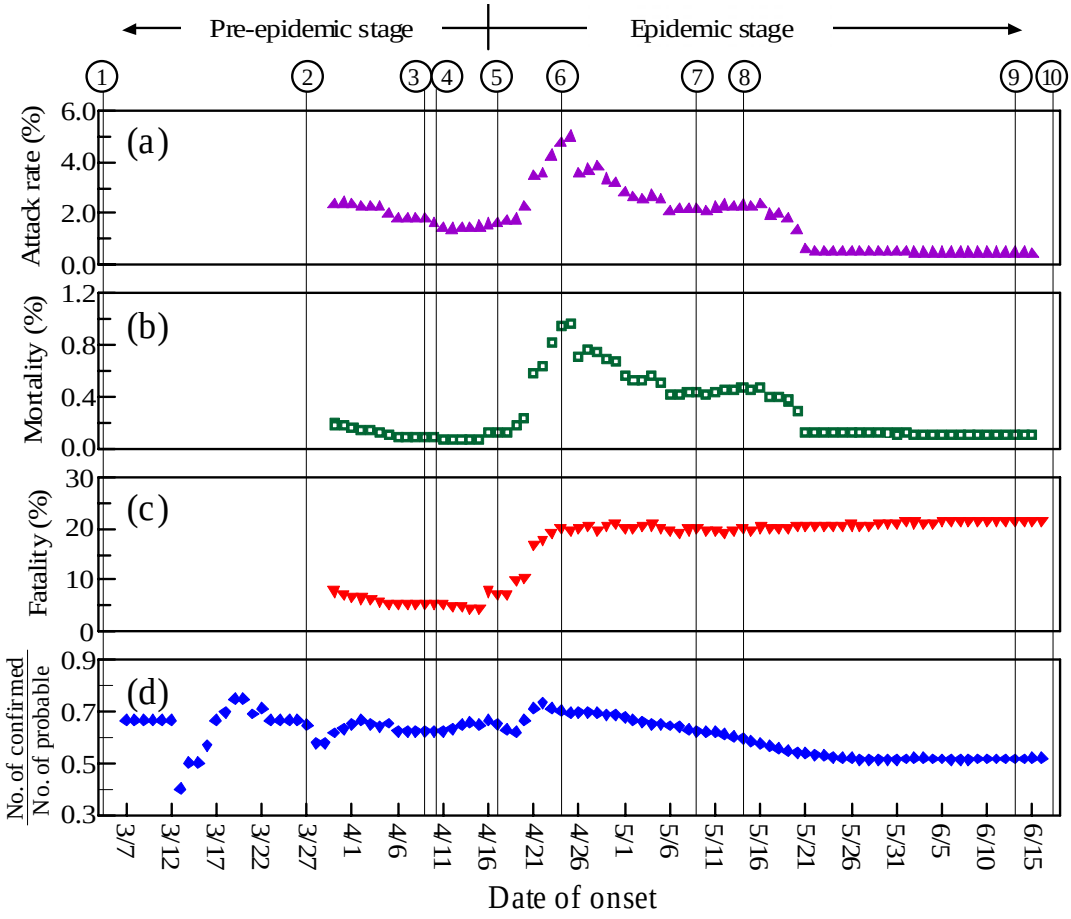

Fig. 7. SARS attack, morbidity and fatality rates and the ratio of numbers of confirmed cases and numbers of probable cases in Taiwan in 2003. ① First suspect SARS case was reported by CDC employee Ming-Chu Kuo; ② Home quarantine measures were announced; ③ The incubation period in the Hoping hospital (9-16 April); ④ Inbound travelers to Taiwan are requested to have their body temperature taken; ⑤ The nosocomial period in the Hoping hospital (17-23 April); ⑥ The hospital closure period (24 April to 8 May); ⑦ The isolation/containment period (since 9 May) and WHO website listed Taipei as the areas with recent local transmission; ⑧ Clustering infections in the NTUH and the Chang-Gung hospitals. ⑨ WHO raised Taiwan travel advisory to level B; ⑩ Taiwan was lifted from WHO's travel advisory list.

2. *模式驗證* (*Check model*)

運用 SARS 數據代入本研究所建立之「無尺度流行病模式」((20)~(23)式)以驗證模式之正確性。依據 2003 年台灣 SARS 傳染情況(參見圖 7)，本研究將發病期程再分爲傳染前期(pre-epidemic period, March 30~April 16, 2003)、傳染期(epidemic period, April 17~May 8, 2003)與抑制期(containment period, May 15~June 15, 2003)。

本研究選定每日的致死率爲參考序列，每日的死亡率、侵襲率與確定病例數和可能病例數比爲比較序列來進行灰關聯分析，以瞭解這些流行病參數對於「無尺度流行病模式」的重要性，所得結果列於表 1 中。表 1 顯示三個比較序列以侵襲率無論在傳染前期、

傳染期、抑制期或全部傳染期間的關聯性均最高，而死亡率則均爲最差。尤其值得一的是侵襲率在傳染前期與抑制期的關聯性均高於 0.9，顯現本研究所建立之「無尺度流行病模式」以侵襲率爲最主要參數，是正確的。

Table 1. Summary of the results of Grey relational analysis of four epidemic parameters.

Period	Dynamic mortality	Dynamic attack rate	No. of confirmed cases / No. of probable cases
Pre-epidemic (March 30~April 16)	0.907	0.945	0.918
Epidemic (April 17~May 14)	0.580	0.639	0.585
Containment (May 15~June 15)	0.885	0.913	0.894
Full epidemic (March 30~June 15)	0.555	0.601	0.565

在「無尺度流行病模式」驗證中，本研究採用之病患隔離程度 ψ 與無尺度傳染冪次 γ ，如(25)式所列

$$\psi = \begin{cases} 1 & \text{if } t - t_i + 1 \le 3 \\ 0 & \text{if } t - t_i + 1 > 3 \end{cases} \quad \text{and} \quad \gamma = \begin{cases} 3.5 & \text{at pre-epidemic and containment periods} \\ 3.0 & \text{at epidemic period} \end{cases} \tag{25}$$

其中病患隔離程度 ψ 爲一階梯函數，即每一病例發病至住院隔離天數爲三天，在這三天內具有逐漸增高的傳染率，此點可以吻合 2003 年台灣 SARS 傳染情況。在傳染前期與抑制期的 SARS 傳染情況並不嚴重，每日增加之確定病例數小於 5 例(見圖 6)，因此本研究採用 $\gamma = 3.5$ ；傳染期中每日有較高的確定病例數，因此採用 $\gamma = 3.0$ 。

圖 8 繪出模式模擬結果。由圖中顯現在傳染前期，模擬結果與疾病管制局公佈之確定病例數的吻合度非常高，顯示在傳染前期採用 $\gamma = 3.5$ 是相當合理與正確；傳染期的 4 月 26 日起模式模擬的結果，略高於公佈之確定病例數，雖然這段期間暴發台北市合平醫院感染(該院於 4 月 24 日緊急封院)，每日新增之確定病例尚不足以使 γ 大幅降低，而採用 $\gamma = 3.0$ 可能仍略高，但綜觀各期間模擬結果均與疾病管制局公佈之病例數相近，差異僅 5 個病例左右，因此採用(24)式的 ψ 與 γ 值是相當合理與正確。

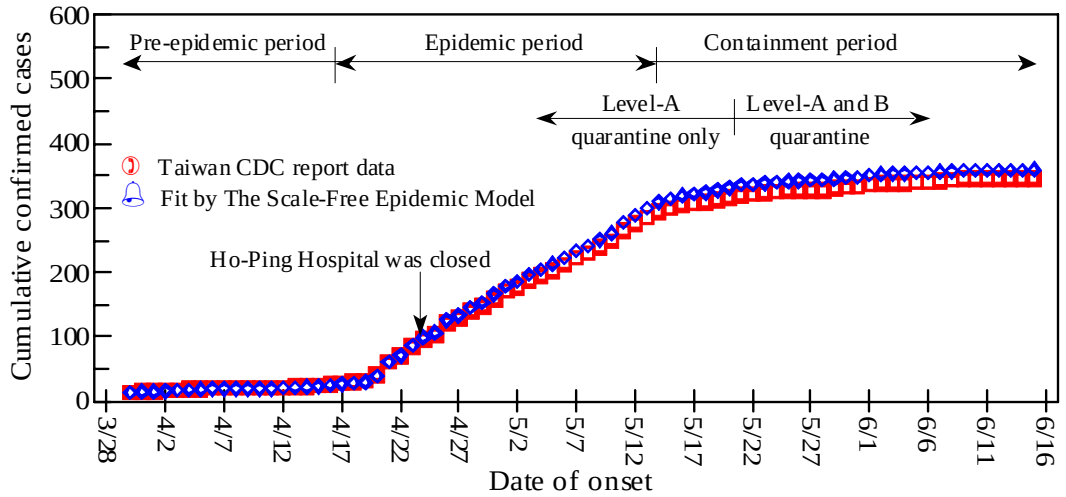

Fig.8. Comparison the simulation results by Eq. (23) with Taiwan CDC report data for cumulative confirmed cases. The values were used in the simulation that $\psi = 1$ at $t - t_i + 1 \le 3$ and $\psi = 0$ at $t - t_i + 1 > 3$. The values of γ in pre-epidemic, epidemic, and containment periods are 3.5, 3, and 3.5, respectively.

經由灰關聯分析(表 1)與模式模擬(圖 8)結果，均顯示本研究建構「流行病無尺度網路模式」所選用之流行病參數(侵襲率)與完成後的模式((20)~(22)式)，不僅正確可用，同時也可以反映事件發生時之數據的狀況，達到本研究之最大目標。

3.*預測與決策* (*Predication and decision marking*)

本研究使用所建立之「無尺度流行病模式」與灰預測法，利用改變 ψ、γ 與 m 參數值，來探討這三個參數對流行病傳染的影響，所得結果繪於圖 9 中。

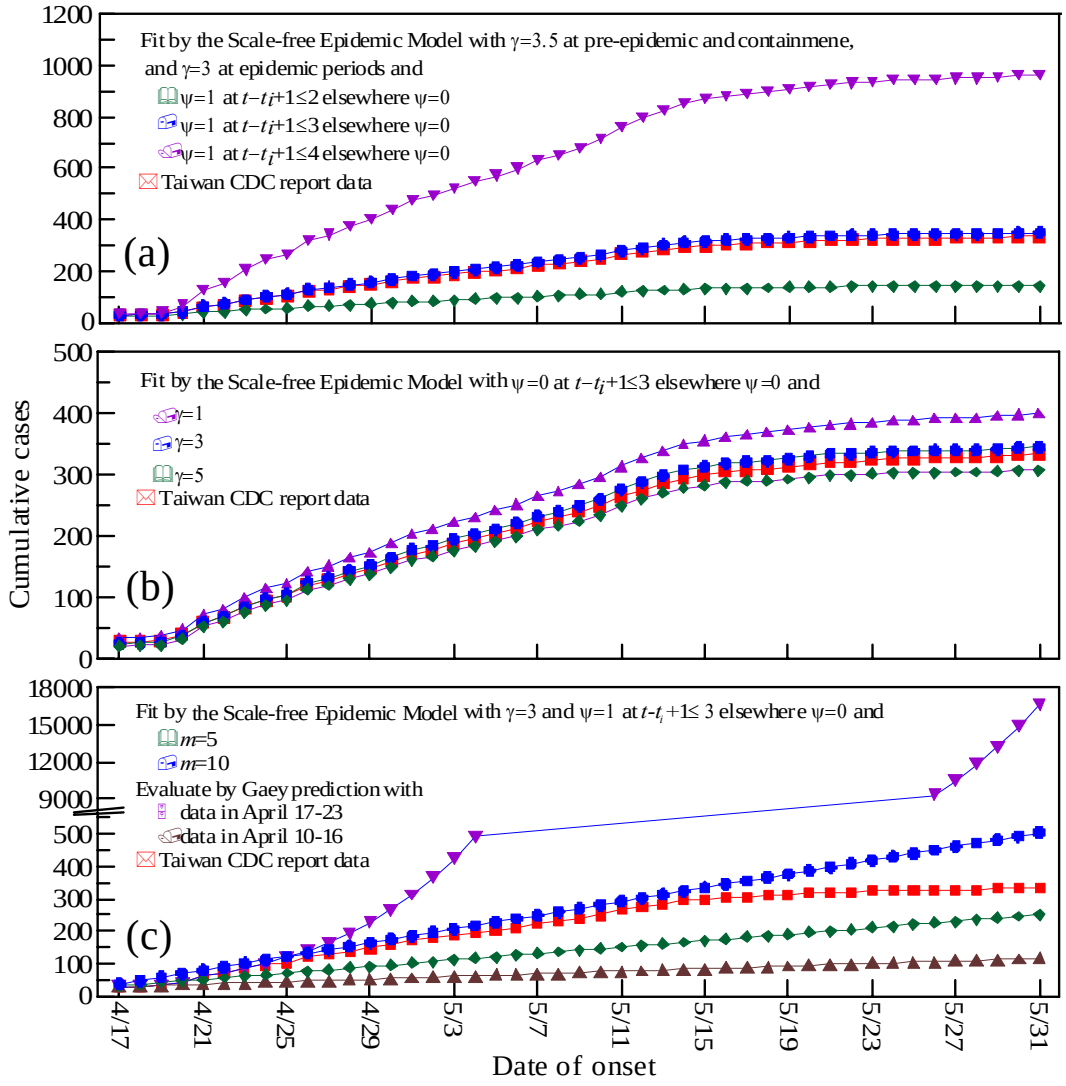

Fig. 9. The effects of ψ, γ, and m on reducing the numbers of SARS confirmed cases. (a) Changed ψ, (b) changed γ, and (c) changed m. Condition-1, 2, and 3 are $\psi = t - t_i + 1 \le 2$, ≤ 3, and ≤ 4, respectively, elsewhere $\psi = 0$.

(1). ψ 的影響 (Effect of ψ)

圖 9(a)繪出在三種 ψ 條件下(參見(25)式)所得之確定病例數，當加強隔離措施使感染天數縮短為 2 天，其結果如綠線，則截至 5 月 31 日止確定病例降為 143 人(**－190** 人)；但若放鬆隔離措施使平均感染增為 4 天，則至確定病例增至 963 人(**＋630** 人)。足見迅

速且有效隔離 SARS 病患，相當重要，二者相差 **820** 確定病例數。而強化宣導，加強通報，並嚴格要求發現 SARS 可能病患立即送醫院隔離，是達到有效隔離 SARS 病患的不二法門。

$$\left.\begin{array}{l}\text{Condition-1}: \quad \psi = 1 \text{ at } t - t_i + 1 \le 2, \text{ elsewhere } \psi = 0 \\ \text{Condition-2}: \quad \psi = 1 \text{ at } t - t_i + 1 \le 3, \text{ elsewhere } \psi = 0 \\ \text{Condition-3}: \quad \psi = 1 \text{ at } t - t_i + 1 \le 4, \text{ elsewhere } \psi = 0\end{array}\right\} \tag{26}$$

(2). γ 的影響 (Effect of γ)

回顧(2)式，獲知當 γ 值愈大，無尺度網路的連接機率 $P(k)$ 愈小，亦即流行病傳染率(infection rate)或侵襲率愈小。因此，本研究分別採用無尺度傳染冪次 $\gamma = 1, 3, \text{or } 5$ 來計算確定病例的變化，其結果繪於圖 9(b)中。圖中顯示當 $\gamma = 1$ 時，會使確定病例增至 399 人(**＋66** 人)；而當 $\gamma = 5$ 時，則確定病例減至 307 人(**–26** 人)。由於 γ 值與侵襲率成反比關係，而避免與 SARS 病患接觸可以降低受侵襲的機會。

(3). m 的影響 (Effect of m)

圖 9(c)顯示若使用嚴重感染週(4 月 17~26 日)數據來進行灰預測，且完全放任 SARS 傳染，則至 5 月 31 日可使病患增至 16,649 人(**+16,316** 人)；使用輕度感染週(4 月 10~16 日)數據來進行灰預測，則至 5 月 31 日病患僅爲 116 人(**–217** 人)。

以「無尺度流行病模式」來進行預測，則若讓每日新增確定病例保持 10 人($m = 10$)，則病患增至 503 人(**+270** 人)；而保持 $m = 5$ ，病患減至 250 人(**–83** 人)。

綜合前述結果，顯示影響 SARS 疾病傳染因子的大小是：

$$\psi > m > \gamma$$

事實上對於高傳染率 SARS 疾病而言，有效的就醫與隔離來降低 ψ 值，也是達到降低新增病例數 m 與侵襲率的有效方法，因此強化隔離措施以減少傳染天數最爲重要，且可以有效控制每日 SARS 新增病例，避免發生高侵襲率的現象。致於更深入的探討如何有效降低傳染天數，確定每一個防範與管制細節，避免特定 SARS 病患成爲大的感染源(hub in Fig. 4)，可以另行研究之。

(三)、案例研究二：HIV/AIDS (Case study-II: AIDS)

1. *1984~2004 年台灣 HIV/AIDS 傳染* (*HIV/AIDS epidemics in Taiwan in 1984~2004*)

台灣 1984 年出現 9 位人類免疫缺乏病毒(human immunodeficiency virus)感染者(HIV(+))，1986 年出現 2 位後天性免疫缺乏症候群(acquired immunodeficiency syndrome)發病者(AIDS)。截至 2004 年底累積 HIV(+)、AIDS 與死亡者分別有 6762、1880 與 1025 人；其中 HIV(+)與 AIDS 的死亡率分別爲 15.16%與 54.52%。女性與男性 HIV(+)所佔比例分別爲 6.94%與 93.06%；女性與男性 AIDS 所佔比例分別爲 7.55%與 92.45%；女性與男性死亡者所佔比例分別爲 7.80%與 92.20%[24]。

圖 10(a)列出 1984 年至 2004 年 10 月間逐年之累積 HIV(+)與累積 AIDS，以及每年

新增之 HIV(+)與 AIDS。圖 10(b)則列出近三年(2002 年 1 月至 2004 年 10 月)間逐月之累積 HIV(+)與累積 AIDS，以及每月新增之 HIV(+)與 AIDS。圖中顯示台灣地區每年新增的 HIV(+)與 AIDS，正快速的往上竄升，值得我們警惕，以及積極尋求有效的防範對策。

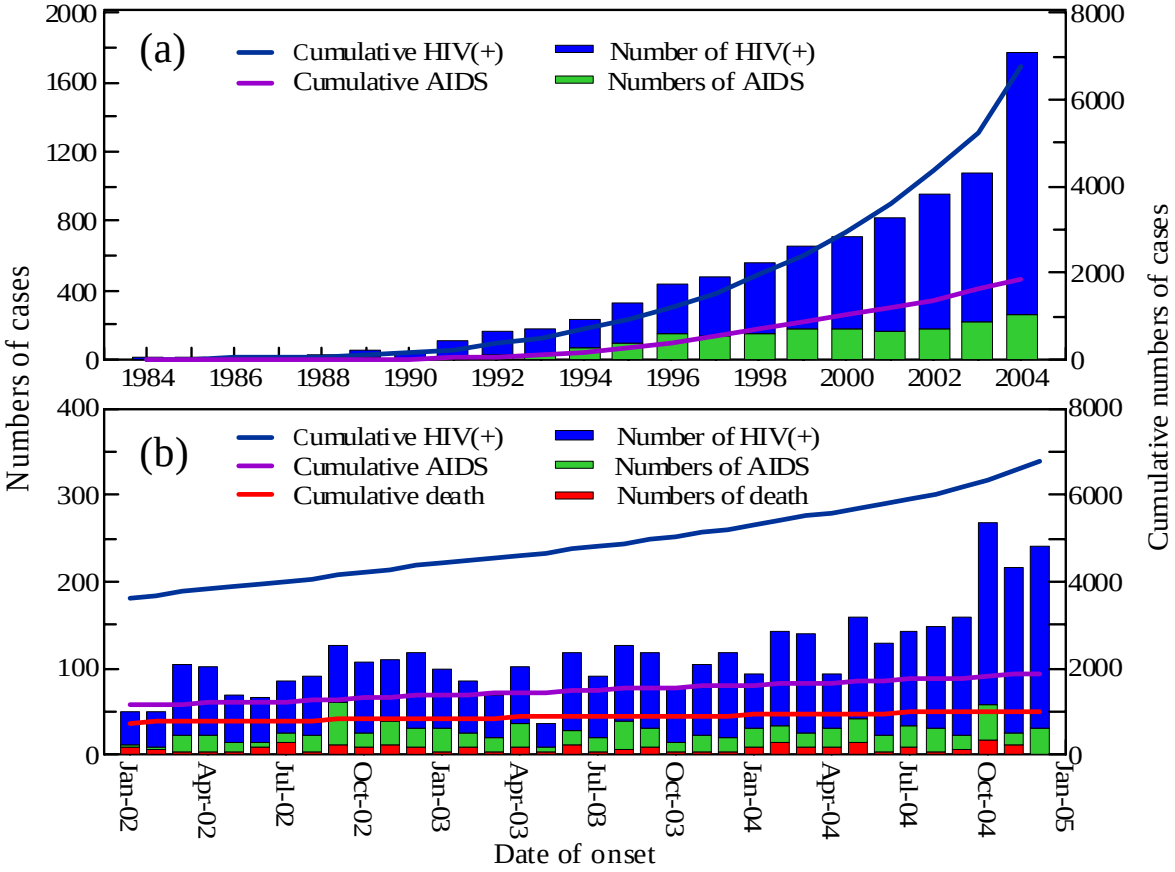

Fig. 10. Epidemiological curves of HIV(+) and AIDS in Taiwan. (a). From 1984 to 2004 and (b) from January 2002 to December 2004. (*Data from*: Center for Disease Control, Taiwan, web site [24])

2. *模式模擬* (*Model simulation*)

HIV 病毒的傳染主要是由於性接觸，根據 Liljeros 等氏之研究[25]，提出當性伴侶數(numbers of sexual partners)大於 20 位，則女性與男性無尺度性接觸網路冪次分別爲 $\gamma = 2.1 \pm 0.3$ 與 $\gamma = 1.6 \pm 0.3$。由於台灣地區 HIV(+)與 AIDS 男性所佔比例均高於 93%，因此在「無尺度流行病模式」模擬中，本研究採用偏向男性的 $\gamma = 1.6$ 來運算。

另外，模擬中之病患隔離程度 ψ，則列於模擬結果之圖 11 中。其中值 ψ 亦爲一階梯函數，即 HIV(+)與 AIDS 之傳染期在年度或月份的無尺度時間均爲 3 ($t = 3$ years or 3 months)，且在這 3 年或 3 月間的傳染率因自我警惕與健康逐漸變差而遞減(見圖 11(a)與(b))，此點可以吻合台灣地區 HIV 的傳染情況。

圖 11 繪出模式模擬結果。圖中顯現無論以「無尺度流行病模式」來模擬年度或月份的 HIV(+)與 AIDS 變動，均有極佳的吻合度，年度與月份的誤差率分別爲<0.41%與<0.17%，顯示本研究採用 $\gamma = 1.6$ 與如圖 11 中所列之 ψ 值，是相當合理與正確，同時再次證實本研究所發展出的「無尺度流行病模式」，非常正確與確切可用。

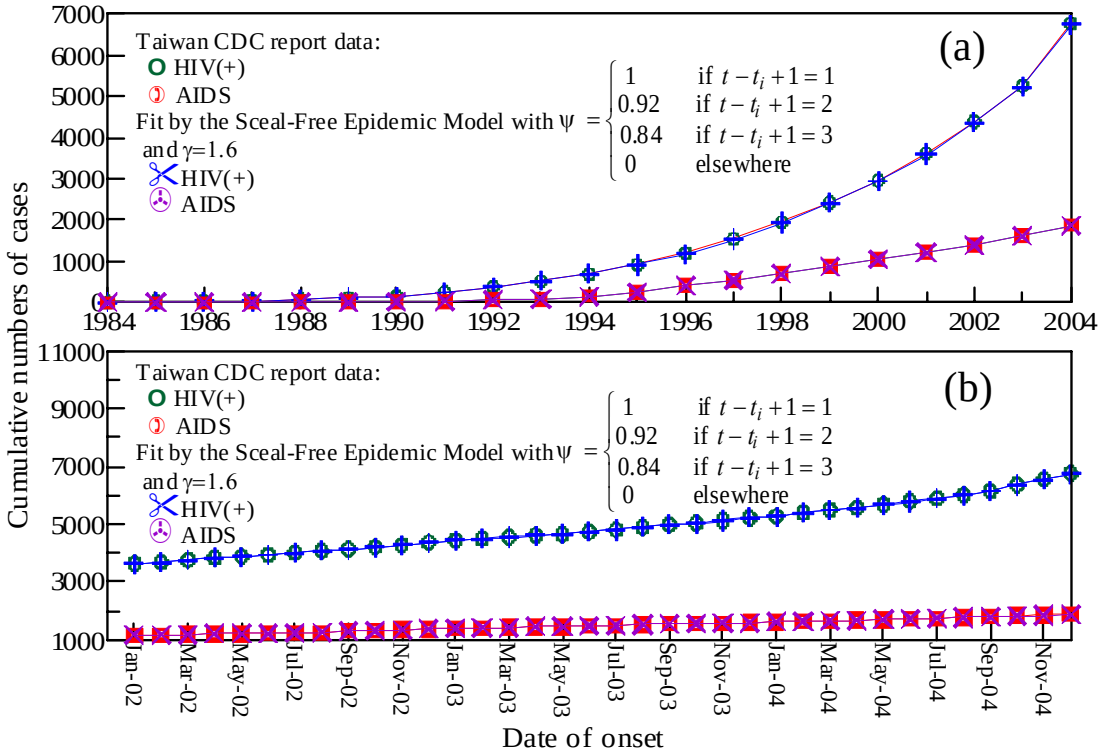

Fig. 11. Comparison the simulation results from the Scale-Free Epidemic Model with CDC reported data for HIV (+) and AIDS. (a) Annual simulation and (b) monthly simulation.

3.*預測與決策* (***Predication and decision marking***)

本研究應用所建立之「無尺度流行病模式」與灰預測法，利用改變ψ參數值，來探討 HIV(+)與 AIDS 在年度或月份的無尺度流行病傳染的影響，所得結果繪於圖 12 中。其中ψ值之變動列於(26)式中，而$\gamma = 1.6$。

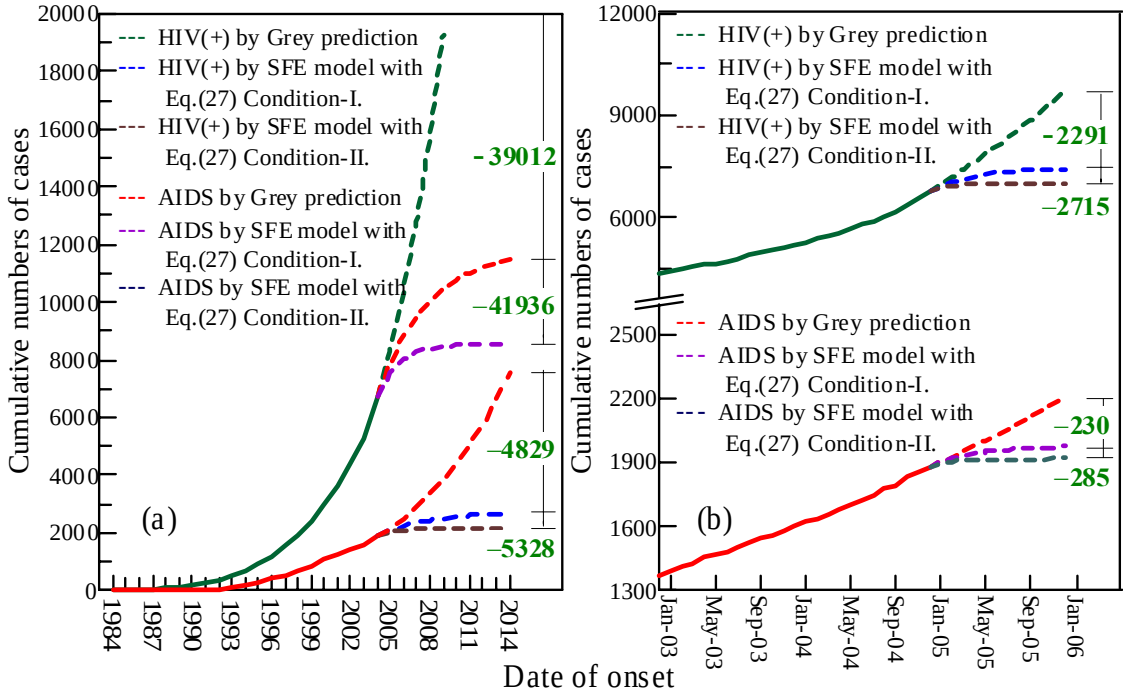

Fig. 12. The effects of ψ on reducing the numbers of HIV(+) and AIDS. SFE Model is the Scale-Free Epidemic Model. (a). Annual simulation and prediction and (b) monthly simulation and prediction

$$\text{Condition-I}:\ \psi=\begin{cases}1 & \text{if } t-t_i+1=1\\ 0.8 & \text{if } t-t_i+1=2\\ 0.6 & \text{if } t-t_i+1=3\\ 0 & \text{elsewhere}\end{cases}\ \text{and}\ \ \text{Condition-II}:\ \psi=\begin{cases}1 & \text{if } t-t_i+1=1\\ 0.6 & \text{if } t-t_i+1=2\\ 0.4 & \text{if } t-t_i+1=3\\ 0 & \text{elsewhere}\end{cases} \tag{27}$$

由圖 12(a)獲知採用(27)式 Condition-I 來模擬，至 2005 年 12 月 HIV(+)與 AIDS 分別爲減少 2,291 與 230 人；改用 Condition-II 值來模擬，則 HIV(+)與 AIDS 分別爲減少 2,715 與 285 人。圖 12(b)獲知採用 Condition-I 來模擬，則至 2014 年底 HIV(+)與 AIDS 分別爲減少 41,936 與 5,328 人；改用 Condition-II 來模擬，則 HIV(+)與 AIDS 分別爲減少 41,936 與 5,328 人。然而如何將 ψ 值降至(27)式的狀態，是值得我們深入探討。

(四)、對抗無尺度流行病傳染之新方法(A new method to fight with scale-free epidemics.)

經由模式之驗證與二個案例的研究，證實本研究所建立之「無尺度流行病模式」((21)~(24)式)相當正確，可以準確的模擬一個新病毒所引發之流行病傳染。然而，當面臨一個全然無知的新病毒的侵襲時，如何減少死亡與傷害人數？是本研究之最終目的。因此，本研究結合了流行病、無尺度網路與灰預測，建立面對新病毒來臨時，一個確切可行的對抗無尺度流行病傳染新方法，詳細運作流程繪於圖 13 中。

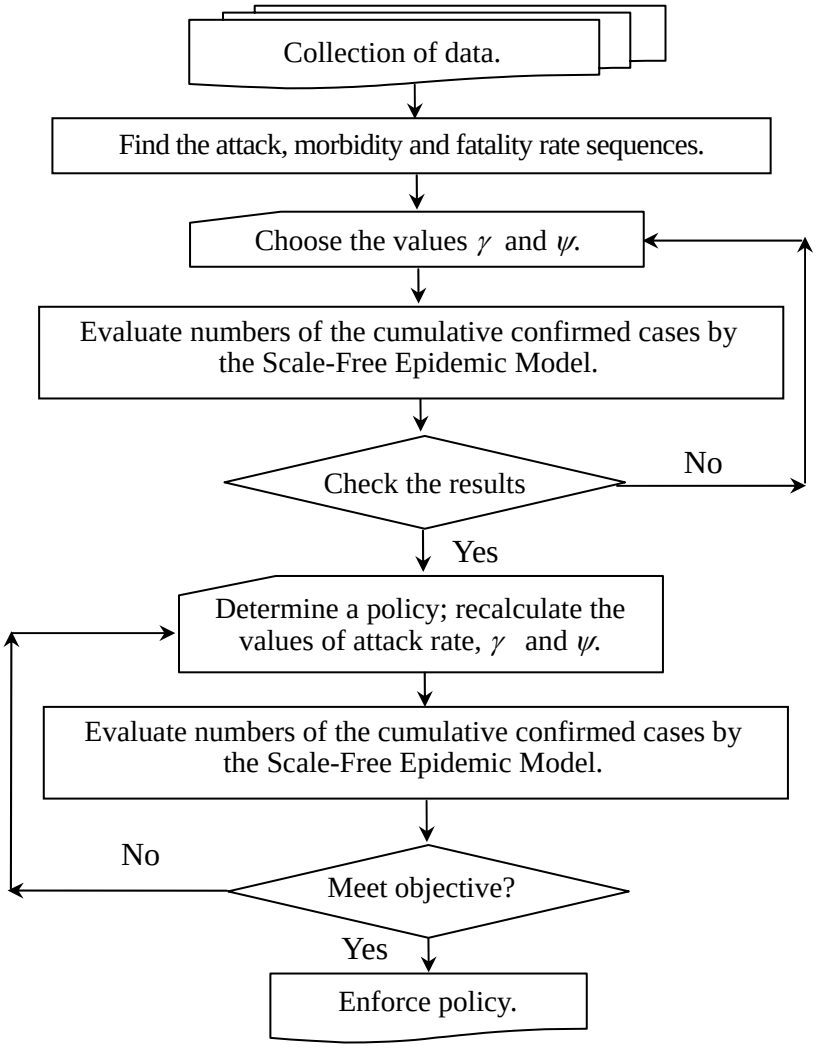

Fig.13. A new method to fight against scale-free epidemics

四、結 論

流行病的傳染過程如同一個無尺度網路，但較一般無尺度網路有著更多的變數而明顯差異，因此無法直接應用一般的無尺度網路模式來描述其傳染途徑。本研究建立一個新模式「無尺度流行病模式」。

SARS 案例研究結果，顯示影響 SARS 疾病傳染因子的大小是：$\psi > m > \gamma$。其中降低ψ值可使 SARS 確定病例至 5 月 31 日止降為 143 人(減少確定病例 190 人，相當於減少死亡 21 人)；僅提高防疫使$\gamma = 5$，亦可使確定病例減至 307 人(減少確定病例 26 人，相當於減少死亡 3 人)。因此強化隔離措施以減少傳染天數最為重要，且可以有效控制每日 SARS 新增病例，避免發生高侵襲率的現象。

HIV/AUDS 案例研究結果，獲知採用(26)式之ψ值來進行月份模擬，則至 2005 年 12 月 HIV(+)與 AIDS 分別為可減少 2,715 與 285 人。而進行年度模擬結果，則至 2014 年底 HIV(+)與 AIDS 分別為可減少 41,936 與 5,328 人。然而如何將ψ值降至(27)式的狀態，是值得我們深入探討。

經由比較模擬結果與疾病管制局的數據，證實此「無尺度流行病模式」是正確與確切可用。此模式可以協助所需警戒的程度與政策決定的計畫結果。因此「無尺度流行病模式」在幫助政府評估社會經濟成本與健康憂慮上的有用之工具。

當面臨一個全然無知的新病毒的侵襲時，如何減少死亡與傷害人數？是本研究之最終目的。因此，本研究結合了流行病、無尺度網路與灰預測，建立面對病毒侵襲，一個確切可行的對抗無尺度流行病傳染新方法，並詳細圖示運作流程。

參考文獻

1. Barabási A.L. and Bonabeau, E., Scale-Free Network, *Scientific American* (*Chinese*), No.**16**, 83-89 (2003).
2. Albert, R. and Barabási, A.L., Statistical Mechanics of Complex Networks, *Reviews of Modem Physics,* Vol.**74**, 47-97 (January 2002).
3. Dorogovtesev, S.N. and Mendes, J.F.F., Evolution of Network, *cond-mat*/0106144 v2, 7 September 2001.
4. Watts, D.J. and Strogatz, S.H., Collective Dynamics of Small-Word Networks, *Nature*, **393**, 440-445 (1998).
5. Erdös, P. and Rényi, A., On Random Graphs, *Publications Mathematic*, **6**, 290-295 (1959).
6. Barabási, A.L. and Albert, R. Emergence of Scaling in Random Networks, *Science*, **286**, 509-512 (October 1999).
7. Cohen, R., Erez, K., ben-Avraham, D., and Havlin, S., Resilience of the Internet to Random Breakdowns, *cond-mat/*0007048 v2, 19 October 2001.
8. Dorogovtesev, S.N., Mendes, J.F.F., and Samukhin, A.N., Structure of Growing Networks: Exact Solution of the Barabási-Albert's Model, *cond-mat/*0004434 v1, 26 April 2000.

9. Dorogovtesev, S.N., Mendes, J.F.F., and Samukhin, A.N., Generic Scale of the "Scale-Free" Growing Networks, *cond-mat/*0011151 v1, 7 November 2000.

10. Klemm, K. and Eguíluz, V.M., Highly Cluster Scale-Free Networks, *cond-mat/*0107606 v1, 30 July 2001.

11. Klemm, K. and Eguíluz, V.M., Growing Scal-Free Networks with Small World Behavior, *cond-mat/*0107607 v1, 30 July 2001.

12. Albert, R and Barabási, A.L., Topology of Evolving Networks: LOcal Events and Universality, *Physical Review Letters*, **85**(24), 5234-5237.

13. Chen, C.J., 2004, Epidemiology: Principle and Method (*Chinese*), 2nd, Linking Inc., 73-159.

14. Gerstman, B.B., 2003, Epidemiology Kept Simple: An Introduction to Traditional and Modern Epidemiology, 2nd, *Wiley-Liss Inc.*, 338-347.

15. Gordis, L., 2004, Epidemiology, 3rd, *Elsevier Inc.*, 15-68.

16. Uen, K.L., Hwang, Y.F., Chen, F.S., Lee, Y.B., Lian, J.F., and Lay, J.R., 2002, Grey Predication: Principle and Application (*Chinese*), Chwa Taiwan Inc.

17. Uen, K.L., Hwang, Y.F., Chang, W.J., Cheng, T.J., You, M.L., and Lay, J.R., 2003, Grey Relational: Model and Application (*Chinese*), Gau Lih Book Co., Ltd.

18. Su, I.J., September 2003, Memoir of Severe Acute Respiratory Syndrome Control in Taiwan, *Center for Disease Control, Department of Health, Taiwan*, GPN: 1009202897.

19. Su, I.J., October 2003, Memoir of Severe Acute Respiratory Syndrome Control in Taiwan, *Center for Disease Control, Department of Health, Taiwan*, GPN: 1009203400.

20. Su, I.J., 2004, SARS and Flu Prevention: Taiwan Experience, *Center for Disease Control, Department of Health, Taiwan*, GPN: 1009301256.

21. Su, I.J., 2004, Statistics of Communicable Diseases and Surveillance Report in Taiwan Area (2002), *Center for Disease Control, Department of Health, Taiwan*, GPN: 2009300465.

22. Department of health, Taiwan, Web, http://www.doh.grv.tw/newverprog/proclaim/sars_list.asp

23. Union Daily News, Taiwan, March 20-June 15, 2003.20

24. Center for Disease Control, Taiwan, Web, http://www.cdc.grv.tw

25. Liljeros, F., Edling, C.R., Åmaral, L.A.N., Stanley, H.E., and Aberg, Y., 2001, The Web of Human Sexual Contacts, *Nature*, **411**, 907-908.

1. Introduction

1.1 Motivation

A scale-free network paper from Scientific American [1], talking about Internet and human societal behavior generalized to a kind of scale-free network, really attracts me. From other papers [2,3], I found that the power law distributions of scale-free networks could be applied to other field studying cellules, computers, linguistics, society, operation, medicine and business. Applying this model and method to diseases for prevention and suppression is my concern.

1.2 Purposes

The ways of epidemic infections resemble a scale-free network. But the differences in epidemic infections are loop-less, single-incoming, non-self-organization, non-linear growing, multiply-outgoing, with nodes of isolation or death, intra-individual variability, racial differences and so on. So the specific characters or parameters should be collected and new model be developed.

A new virus epidemic disease always causes lots of death and horror in the pre-epidemic period (Sever Acute Respiratory Syndrome, SARS, in 2003). There are few conditions controlled by the CDC (Center for Disease Control) in Taiwan. So it was hard to make an effective policy to control and reduce the death and damage. Construction of a new model, The Scale-Free Epidemic Model, may help control infections.

2. Methodology

2.1 Study process

Research of collection data and papers on Epidemic disease was the first and continuous process. A new Scale-Free Epidemic Model was constructed after studying the Epidemiology's Reed-Frost Model and scale-free networks' Barabási-Albert Model. Epidemic data from Taiwan CDC reports was input into this model. Grey theory GM(1,N) and GM(1,1) models for relational analysis and prediction was use to check and modify the model. The results of different Grey decision marking to find the optimum prevention and evaluate the effectiveness of this model were estimated. Two case studies were used to evaluate the accuracy and usefulness of the model. Finally, a new method was proposed to fight against scale-free epidemic. Study procedure is shown in Fig. 1.

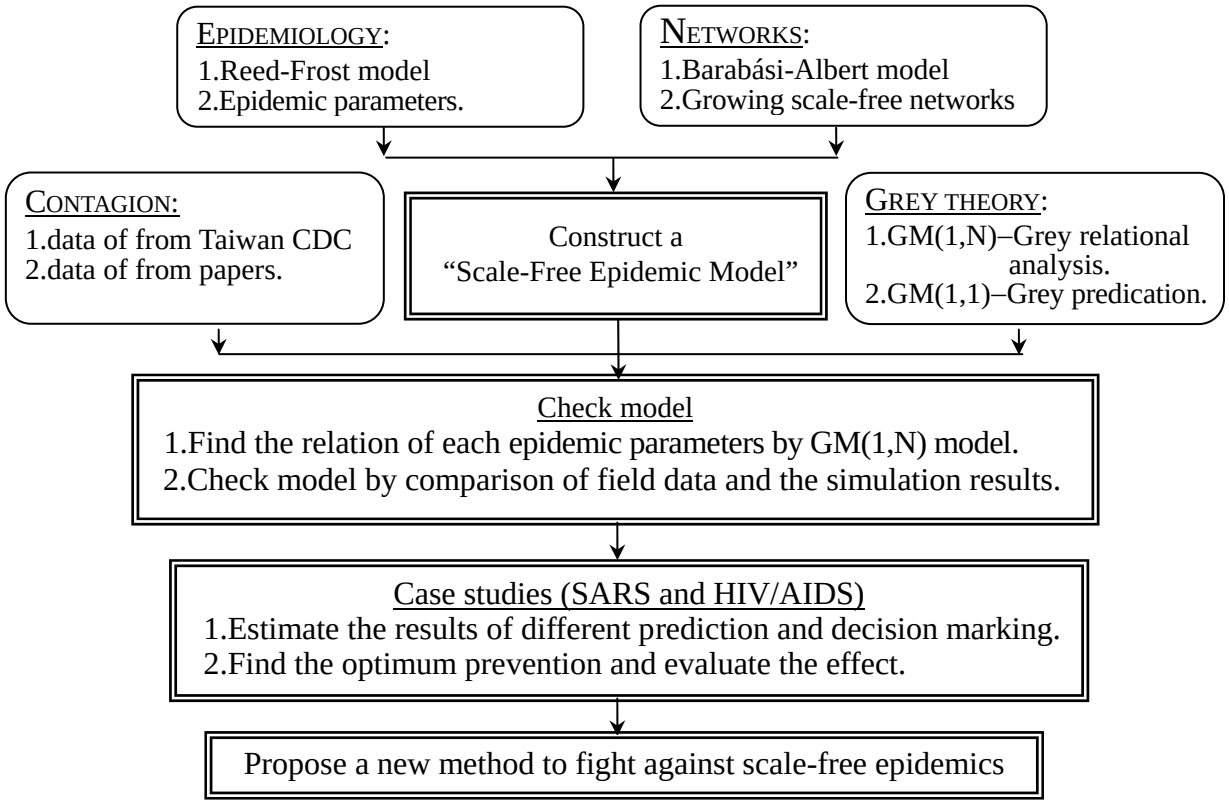

Fig. 1. Study procedures

2.2 Networks

In general there are three kinds of the representative and well-documented networks; they are small-world networks, random networks and scale-free networks [2,3]. The real "small-world" network, unsociable inhabitants live in a village, usually, contact with limited to their neighbors but some of them attends the church. The network is small and the links are simple. The degree distribution of a small-world network follows the Watts-Strogatz model, (WS-model) [2,4] (Eq. (1)).

$$P(k) = \sum_{n=0}^{f(k,K)} C_{K/2}^{n} (1-p)^{n} p^{K/2-n} \frac{(p\,K/2)}{(k-K/2-n)!} e^{-p\,K/2}, \text{ for } k \geq K/2 \qquad (1)$$

where k is degree; K is number of neighbors; $K/2$ is neighbors on either side; p is the opportunity of two nodes to connect; C is clustering coefficient.

The classic random networks can be considered a traffic system, the connectivity distribution of node following belled Poisson distribution, all nodes having approximately the same number of edges. Eq. (2) is the Erdös-Rényi model (ER-model) of the degree distribution in random network [2,3,5].

$$P(k) = \begin{cases} C_{N-1}^{k} p^{k} (1-p)^{N-1-k} \\ P(k) = \dfrac{e^{-\bar{k}} \bar{k}}{k!} \qquad \text{for large N} \end{cases} \qquad (2)$$

where N is total number of nodes; k is degree; and $\bar{k} = p(N-1)$ is average degree.

There are hubs in scale-free networks, with connectivity distribution between nodes following power law distribution, most of nodes have few connections, but some of them

have high numbers of connections. Eqs. (3) to (6) are the Barabási-Albert model, (BA-model) [2,3,6,7], the probability and connection degree are

$$P(k) \propto k^{-\gamma} \tag{3}$$

$$P(k) = \begin{cases} \dfrac{k-1}{k+2} P(k-1) & \text{for } k \geq m+1 \\ 2/(m+2) & \text{for } k = m \end{cases} \tag{4}$$

$$P(k) = \frac{2m(m+1)}{k(k+1)(k+2)} \tag{5}$$

$$k_i(t) = m\left(\frac{t}{t_i}\right)^{1/2} \tag{6}$$

where γ is the exponent; m is connection degree of new and old nodes; t and t_i are the time and the time of introduced node-i.

There are some papers and models related to my study where the epidemic infection is a scale-free growing network. Dorogovtesev et al. [7] generalize the B-A model of growing networks accounting for initial properties of sites and final exactly the distribution of connectivity of the network $P(k)$ and the averaged connectivity $\bar{k}(s,t)$ of a site s in the instant t (one site is added per unit of time). Dorogovtesev et al. [8] show that the connectivity distribution $P(k,t)$ of scale-free growing networks (t is the network size) have the generic scale the with cut-off at $k_{cut} \sim t^{\beta}$. The scaling exponent β is related to the exponent γ of the connectivity distribution and same as exponent β in $P(k) \sim k^{\beta}$ [6]. The scaling relation form and the equation of degree distribution were proposed. Klemm et al. [10] proposed the growth and deactivation model of scale-free network nodes. At any step of the time-discrete dynamics m nodes in the network are active, others are inactive. Introduction of a new node i to the network, the probability ($D(k_j)$) that the node j is deactivated. Klemm et al. [11] introduce a simple dynamical model that unifies the generic feature of real networks: scale-free distribution of degree and small world effect.

In addition, Albert and Barabási [12] investigated the networks growth and evolution from a local event. They proposed a continuum theory that predicts these two regimes as well as the scaling function and the exponents. The connectivity ($k_i(t)$) of a node added at time t_i has the form

$$k_i(t) = \left[A(p,q,m) + m + 1\right]\left(\frac{t}{t_i}\right)^{1/B(p,q,m)} - A(p,q,m) - 1 \tag{7}$$

$$\text{where} \quad A(p,q,m) = (p-q)\left(\frac{2m(1-q)}{1-p-q} + 1\right) \quad \text{and} \quad B(p,q,m) = \frac{2m(1-q)+1-p-q}{m} \tag{8}$$

The ways of epidemic infection resemble a scale-free network, with growth, deactivation, crashes and high clustering, and small-word effect. But they are different due to more variables in an epidemic infection. Therefore, the model of scale-free networks is not enough to satisfy the epidemic infection data. A new scale-free epidemic model is proposed in this study.

2.3 The Epidemiology

There are basically two kinds infections, common source infection and propagated infection.

2.3.1 *Common source infections*

The characteristics of common source infections are the epidemic curve peaks toward to right, with a short infection period, one or two incubation periods that belong to the outbreak (for example food-borne). Both the incubation and induction period, are short so the causes of common source infection is easy to research. The common source infection is a random occurrence and belongs to Poisson distribution, the probability Pr($A=a$) can be found from Eq. (9).

$$\Pr(A = a) = \frac{e^{-\mu}\mu^{a}}{a!} \tag{9}$$

where A is cases in certain person-time and variable number, μ is expected number. Comparing Eqs. (9) with (2), we can find that common source infection resembles random networks.

2.3.2 *Propagated infection*

The characteristics of propagated infection are the epidemic curve peaks toward to left; the infection period time always includes two or more incubation periods. The contagions are immediate or mediate from sickness and carrier to susceptible host for example passing to another via insect or body fluids. The propagated infection will be affected by generation time, herd immunity and secondary attack rate. The behavior of propagated infection also resembles scale-free networks, in which this study is interested.

If propagated infection is limited to person-to-person infection, the behavior resembles the Reed-Frost model. The conditions are: (1) disease is infectious susceptible host; (2) the susceptible host is infectious only to next generation who is susceptible, but the grand generation is not infectious; (3) the effective contact rate of each person and the next generation with others is fixed; (4) each person should be isolated from the population; (5) above conditions are tight in epidemic stage. The Reed-Frost model [13,14]

$$H_{t+1} = S_t\left(1 - q^{H_t}\right) \tag{10}$$

where H_t and H_{t+1} are numbers of incident cases of t and t+1 generation, S_t is number of susceptible host in t generation, the function q=1-C_R is contact rate of unfound cases,

C_R is contact rate, and the contact rate can be found by initial susceptible host (S_0) and residual susceptible host (S_m):

$$C_R = \frac{\ln(S_m / S_o)}{S_m - S_o} \tag{11}$$

Some factors of Epidemiology will be used to set up scale-free epidemic networks, they are: (1) attack rate, (2) incident rate, (3) mortality, (4) fatality, and (5) herd immunity.

2.4. Grey model

Studying the strength and relational analysis of epidemic factors in a Scale-Free Epidemic Model, to develop GM (1, N) model based on Grey Theory. The flow chart is:

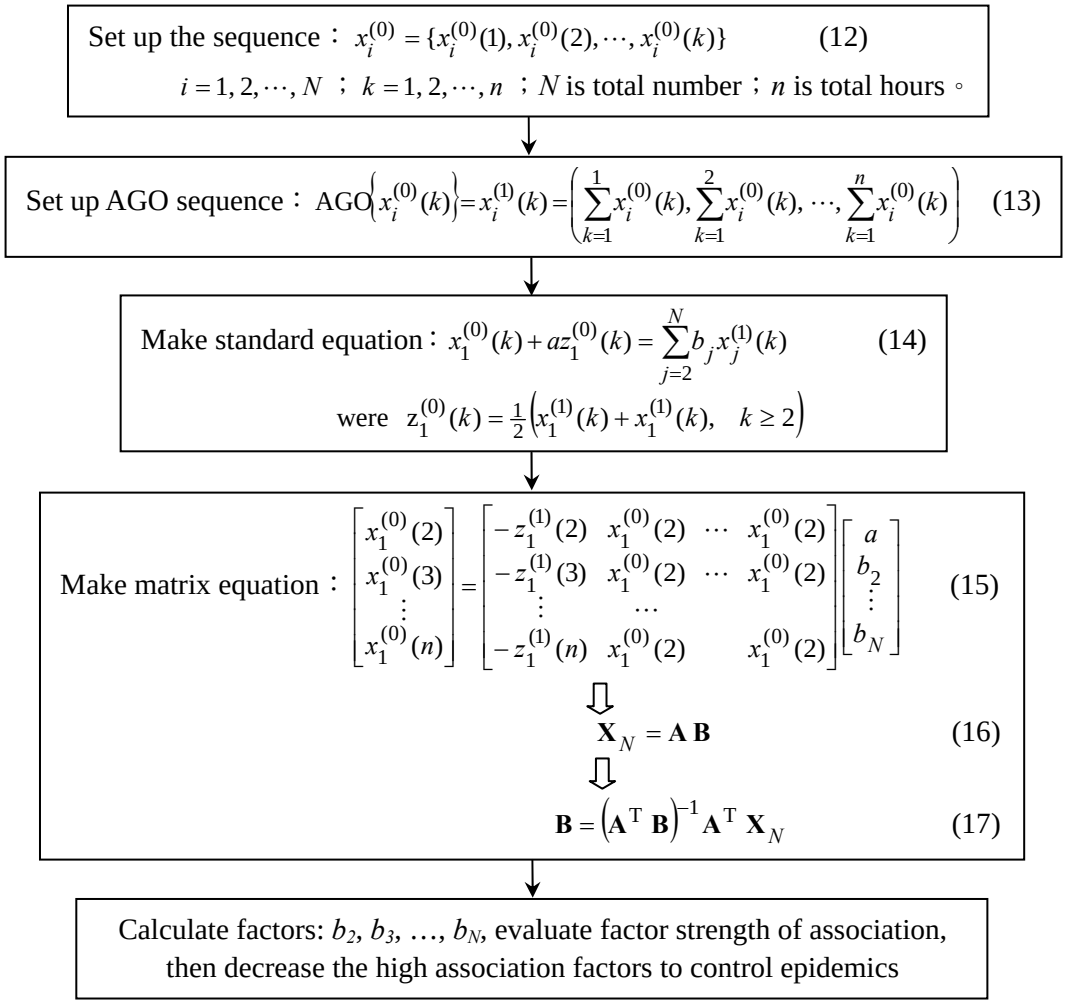

Fig. 2. GM(1,N) model operation procedures of this study.

GM(1,N) model is multi factor input for relational analysis, and GM(1,1) is a one factor input for prediction of that factor's future dynamic state. Decision marking based on above predictions is called gray decision marking. Evaluate the factor's strength of association and find the high association factors, fitting the factor's values then input them to the Scale-Free Epidemic model, finally calculate the number of probable cases. Resetting the original sequence procedure after rearranging the factor's value is called gray model construction. Using the model predicts future possibilities. If the possibility is satisfactory, the data can be used to make prevention decisions. Continue this process until everything is

perfect. The GM (1,N) and GM(1,1) software is with the Grey Perdition and Grey Relation books [16,17].

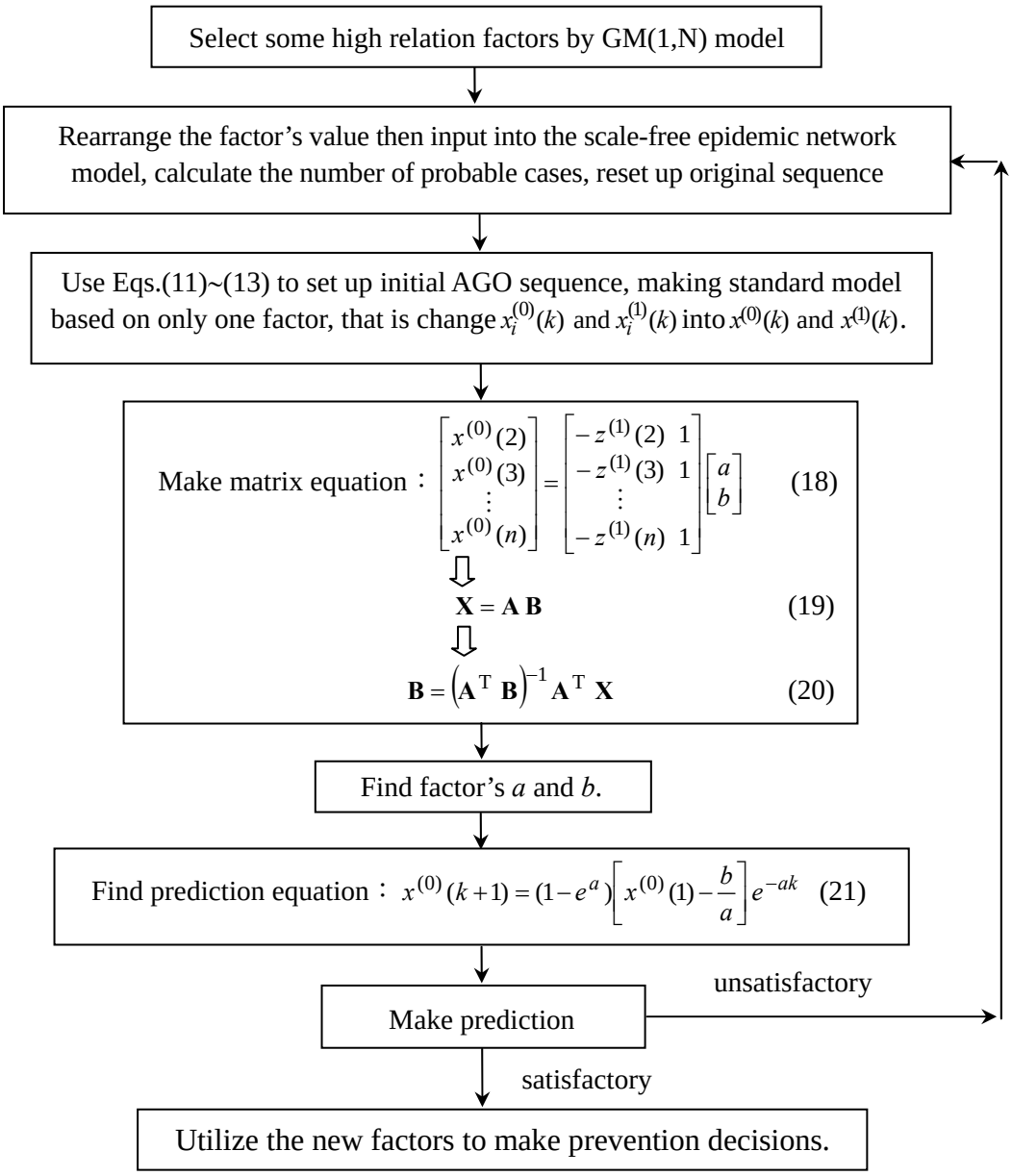

Fig. 3. GM(1,1) model and gray decision marking operation procedures of this study.

3. Results and Discussion

3.1 Epidemic scale-free network model construction

The behavior of epidemics very similar to scale-free networks but has some specificity of association: loop-less, single-incoming, multiple outgoing, non-self-organization, non-linear growing, nodes of isolation or death, intra-individual variability, racial differences and so on. Because of the specificity of association, normal math in scale-free network can't be satisfied; new math and models including special characters or parameters are needed. Fig. 4 illustrates the schematic structure of an epidemic scale-free network. There are two stages: pre-epidemic stage (endemic) and epidemic stage (pandemic). Multiple stratum clusters and sudden change cases

will appear during the course of the infection, its progress is quite different from small-word networks, random networks and scale-free networks.

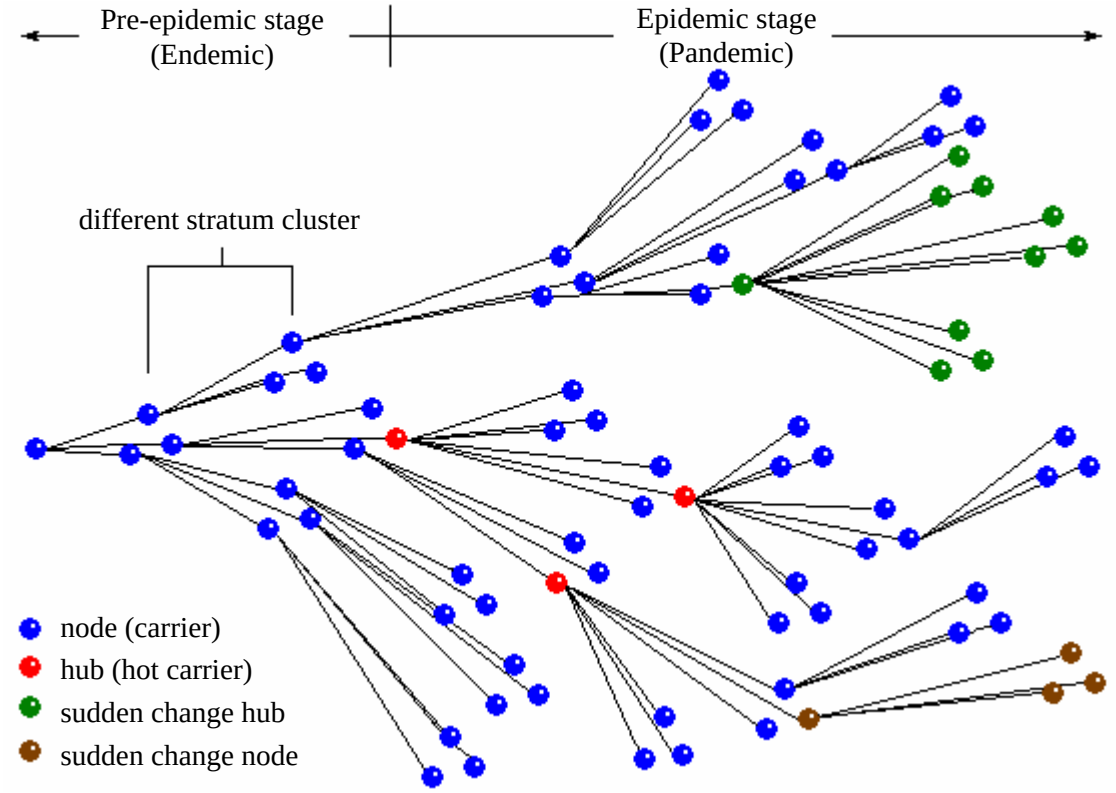

Fig. 4. The schematic structure of a epidemic scale-free network

Some special characters or parameters should be considered, such as: (1) the attack rate increases at the beginning then decreases with the time of incident and carrier, (2) the attack rate will decrease with the sickness isolation, cures and death, (3) the virus can mutate suddenly and the mutation is passed on but has the same infection ways, and (4) the attack rate will decrease as the herd immunity increases. Considering the above characteristics, I propose a new model, the Scale-Free Epidemic Model, that is

$$k_i(t) = (m+1)\left(t - t_i + 1\right)^{m/(2m+\gamma)} - (m+1), \qquad i = 1, 2, 3, \cdots, n \tag{22}$$

$$\text{where } m = A_t S_t - A_{t-1} S_{t-1} \quad \text{and} \quad \begin{cases} \gamma = 2 & \text{at high attack rate} \\ 2 < \gamma < 4 & \text{at middle attack rate} \\ \gamma = 4 & \text{at low attack rate} \end{cases} \tag{22}$$

$$N_n = N_0 + \sum_{i=1}^{n-1} \sum_{t=t_i}^{n-1} \psi \left[(m+1)\left(t - t_i + 1\right)^{m/(2m+\gamma)} - (m+1) \right] \tag{23}$$

$$\text{where } \psi = \begin{cases} 0 & \text{if the case is death, recoved, or in quarantine.} \\ \text{variable} & \text{if it is function of infection.} \\ 1 & \text{elsewhere} \end{cases} \tag{24}$$

where N_o is the cumulative numbers of cases; $k_i(t)$ is the increasing numbers at time t, γ is the exponent of scale-free networks, and $2 \le \gamma \le 4$ [6]; ψ is degree of isolation, the value can be constant or variable; $(t - t_i + 1)$ is the days after incident; A_t and S_t are attack rate and isolation number at time t. Comparing the Scale-Free Epidemic model and Reed-Frost model, the

Scale-Free Epidemic model can evaluate the time dynamic cumulative case number ($k_i(t)$), but Reed-Frost can not.

3.2 Case study-I: SARS

3.2.1. *SARS epidemics in Taiwan in 2003*

Since the first imported case of Sever Acute Respiratory Syndrome (SARS) was identified in Taiwan in March 2003, two distinct stages have been defined for the SARS outbreak in Taiwan [18]. The time during early March to early April 2003 was the first stage. There were fewer than five cases a day and all cases had either distinct history of contact with index cases or history of traveling to affected areas. On the other hand, the time during mid-April to mid-June was the second stage. During the second stage, the infection in the Taipei Municipal Hoping Hospital spread out to the northern part of Taiwan and later to the southern part. It was finally contained by mid-June.

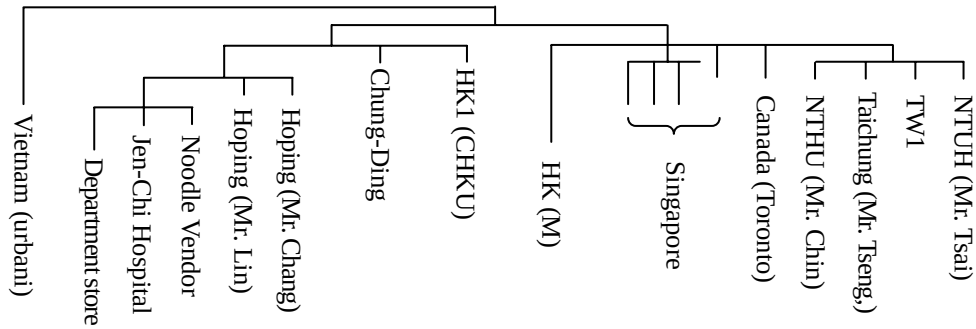

Fig. 5. Phylogenic tree analysis of SARS CoVs from three SARS Clusters in Taipei [18].
(*Source*: Memoir of Severe Acute Respiratory Syndrome Control in Taiwan, September 2003)

Collection of important SARS virus transmission information from Taiwan CDC.

(1) About 90% of SARS cases in Taiwan are the result of hospital transmission. In late April, the SARS infection rate for the medical personnel reached 32%.
(2) The incubation period of SARS virus infection is 3 to 10 days. There is no evidence showing that the infected patients can pass the disease to other people during the incubation period.
(3) SARS virus replication stars 7 to 10 days after developing the symptoms. The virus load will reach its peak and the SARS clinical symptoms are the most sever at this time.
(4) 90% 0f the infected patients will no longer carry the SARS corona virus in their bodies by the third week of the symptoms development.
(5) SARS patients do not need to be quarantined ten days after they are recovered from their fever because they are no longer capable of infecting other people.
(6) It taken an extra week to two detect the SARS antibody in the patients compared to that for the traditional virus infection.
(7) Among the high risk groups or Level-A contacts, about 0.22% of them would be suspected infection and 2% of the sub-clinical infection might be tested positive for antibody but do not develop any clinical symptoms.
(8) Among the reported SARS cases, 58% of the probable cases are related to SARS virus.

Epidemic SARS in Taiwan in 2003, the numbers of cumulative probable cases, cumulative confirmed cases, cumulative death cases, death and survival, (data provided by CDC), are illustrated in Fig 6. Fig. 6 shows the numbers of people quarantined and reasons between March 27 and June 15 in Taiwan in 2003. There is a statistical method change in April 13 so the rate of quarantines appeared to slow down. Statistical method based on Level-A Quarantine was only

used before May 21, Level-A Quarantine plus Level-B Quarantine were used later, so the peaks show an obvious change after that day (Data from Union Daily [23], Taiwan).

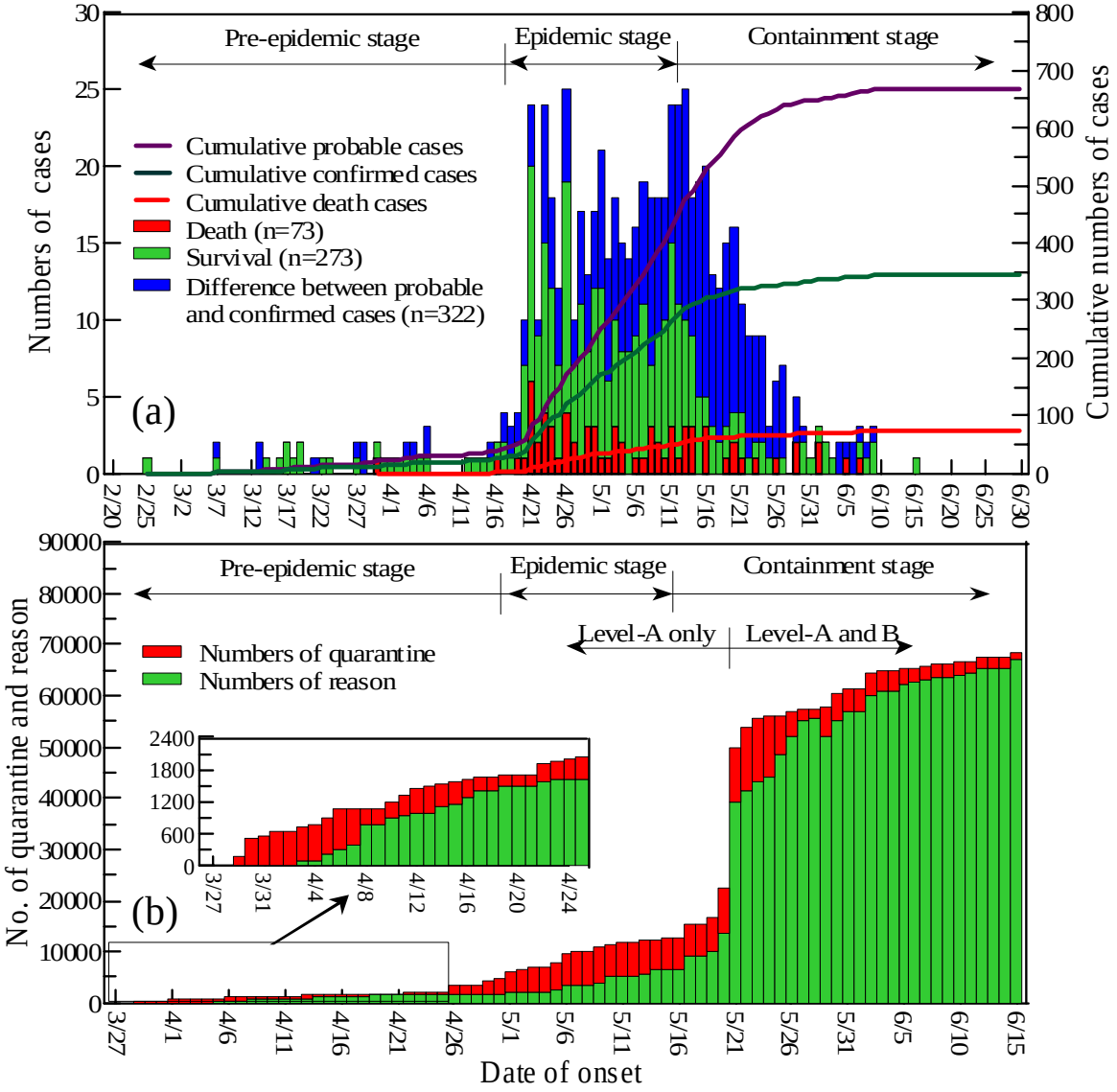

Fig. 6. (a) Epidemiological curves of SARS cases and (b) numbers of quarantine and reason for SARS in Taiwan in 2003. (*Data from*: The publications and the reports of Center for Disease Control [18-21], Department of Health Web [22] and Union Daily [23], Taiwan)

The ratio of cumulative confirmed cases with cumulative quarantines is the attack rate. The ratio of cumulative death cases with cumulative quarantines is mortality. The ratio of cumulative death cases with cumulative confirmed cases is the fatality rate. Fig. 7 (a) and (b) shows that the variation behavior between attack rate and mortality was very close, both had a steep increase from March 27 to April 7, that means there was a high infection rate and the mortality rate during this period and decent prevention was needed. Finally the outbreak happened in the Hoping hospital in late April. After April 8 the serious isolation and quarantine were conducted, so the attack rate and mortality rate decreased.

Otherwise, the incubation period of SARS virus infection is 3 to 10 days and onset of the disease within two weeks, so the increasing rate of fatality was two weeks delayed behind the attack rate and mortality rate. Because the incident of clustering infections (in Hoping

Hospital, Kaohsiung Chang Gung Memorial Hospital, Jenchi Hospital, etc) the fatality rate maintained a high of about 21% on April 24. On Jun 10 SARS was being controlled sufficiently, so the attack rate and the mortality rate slowed down to about 0.5% and 0.1%. Fig.7 (d) shows the ratio of the number of confirmed cases and the number of probable cases. There are two dramatic increases from March 28 to April 2 and from April 17 to April 22. The first one reflects the attack rate and the mortality rate the second reflects the confirmed cases with clustering infection in Hoping Hospital.

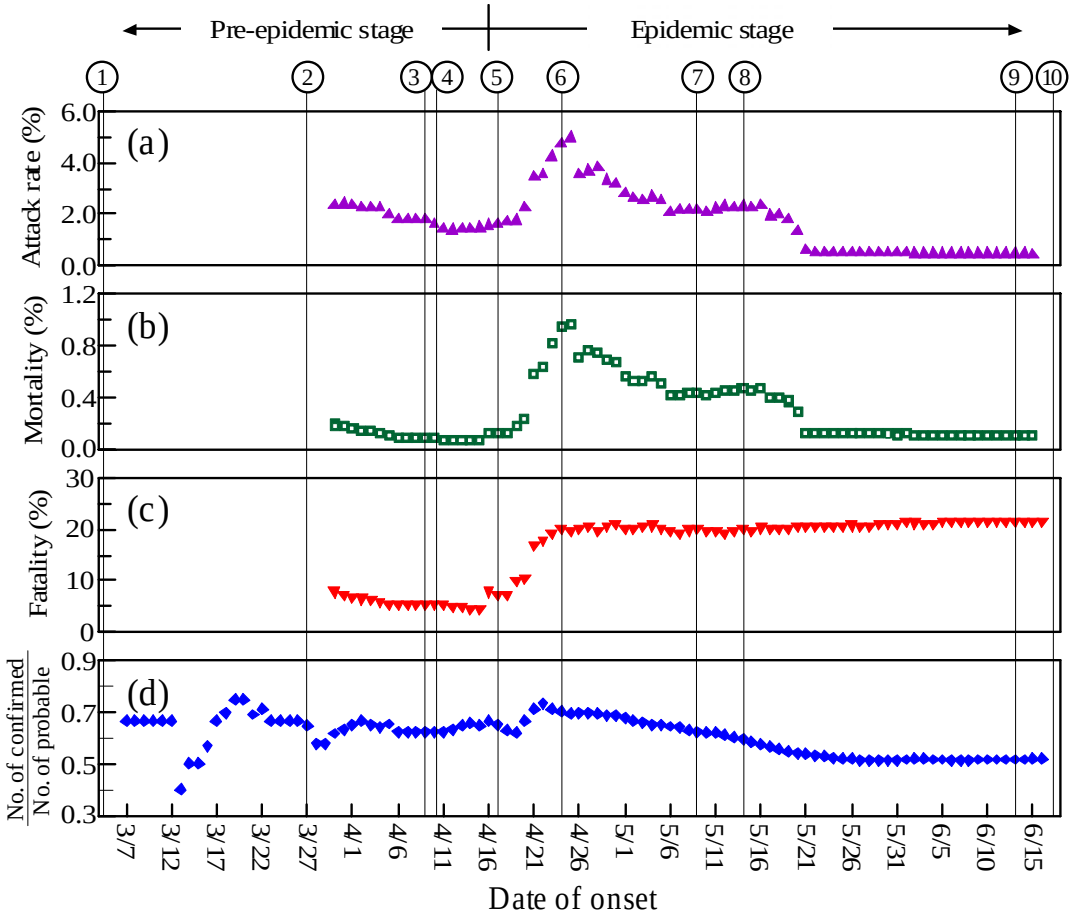

Fig. 7. SARS attack, mortality and fatality rates and the ratio of numbers of confirmed cases and numbers of probable cases in Taiwan in 2003. ① First suspect SARS case was reported by CDC employee Ming-Chu Kuo; ② Home quarantine measures were announced; ③ The incubation period in the Hoping Hospital (April 9-16); ④ Inbound travelers to Taiwan were requested to have their body temperature taken; ⑤ The nosocomial period in the Hoping Hospital (17-23 April); ⑥ The hospital closure period (24 April to 8 May); ⑦ The isolation/containment period (from 9 May) and WHO website listed Taipei as the area with recent local transmission; ⑧ Clustering infections in the NTUH and the Chang-Gung Hospitals. ⑨ WHO raised Taiwan travel advisory to level B; ⑩ Taiwan was lifted from WHO's travel advisory list.

3.2.2. *Check model*

The Scale-Free Epidemic Model was checked using real data of SARS in Taiwan in 2003. There are three periods: pre-epidemic period (March 30–April 16, 2003); epidemic period (April 17–May 8, 2003); containment period (May 8–June 15, 2003.)

Daily fatality is the sequence referenced. Daily dynamic mortality, dynamic attack rate, and the ratio of number of confirmed cases with number of probable cases are comparative sequences. The

Grey relation analysis in Table 1 estimates the relational strength of epidemic factors. Dynamic attack rate has the highest relational strength in three periods; especially in pre-epidemic and containment period where it was always above 0.9. The dynamic mortality is the worst. So the attack rate was chosen as the main factor.

Table 1. Summary of the results of Grey relational analysis of four epidemic parameters.

Period	Dynamic mortality	Dynamic attack rate	$\frac{\text{No. of confirmed cases}}{\text{No. of probable cases}}$
Pre-epidemic (March 30~April 16)	0.907	0.945	0.918
Epidemic (April 17~May 14)	0.580	0.639	0.585
Containment (May 15~June 15)	0.885	0.913	0.894
Full epidemic (March 30~June 15)	0.555	0.601	0.565

In this model simulation, the scale-free exponent γ and degree of the isolation function ψ as Eq. (25).

$$\psi = \begin{cases} 1 & \text{if } t - t_i + 1 \le 3 \\ 0 & \text{if } t - t_i + 1 > 3 \end{cases} \text{ and } \gamma = \begin{cases} 3.5 & \text{in the pre-epidemic and containment periods} \\ 3.0 & \text{in the epidemic period} \end{cases} \quad (25)$$

Therefore, ψ is a step function where each case has a three-day isolation but the attack rate increases everyday. This condition is identical to SARS in Taiwan in 2003. SARS virus infection during pre-epidemic and containment as not so relatively serious and dynamic mortality was below 5 cases, so assume $\gamma = 3.5$; SARS virus infection during epidemic had a higher confirmed case number but below was 20, so assume $\gamma = 3$.

Fig. 8 illustrates the simulation results of the scale-free epidemic model. The results show that the confirmed case numbers from the Scale-Free Epidemic Model (Eqs. (21) – (24)) CDC reports are identical. The confirmed case numbers from this model during the epidemic period, April 20–25, are slightly less than the data reported by CDC, the possibility is $\gamma > 3$ during epidemic period. Even the clustering infections in Hoping Hospital (April 24) suddenly increasing unconfirmed isolation number and confirmed cases didn't affect these results.

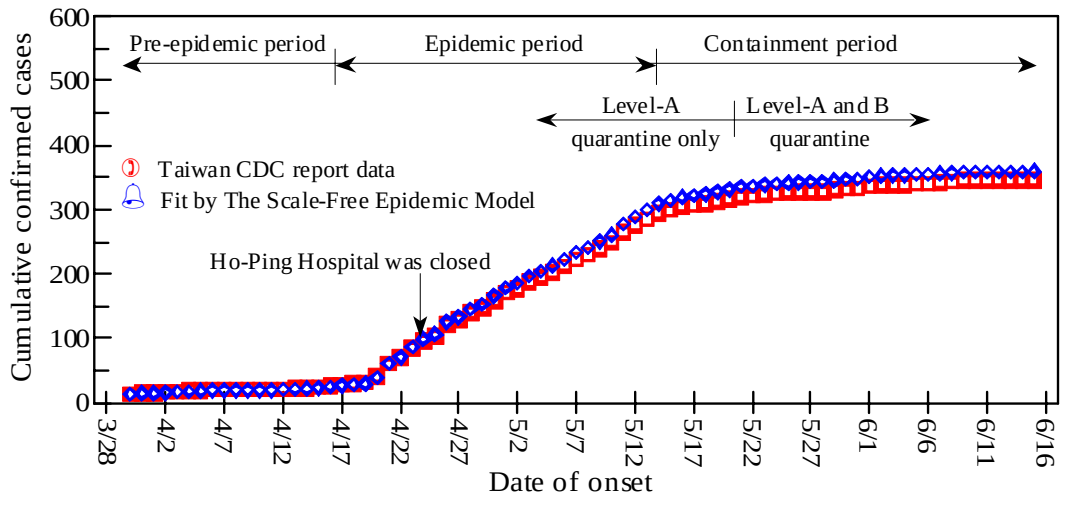

Fig.8. Comparison the simulation results by Eq. (23) with Taiwan CDC report data for cumulative

confirmed cases. The values were used in the simulation that $\psi = 1$ at $t - t_i + 1 \leq 3$ and $\psi = 0$ at $t - t_i + 1 > 3$. The values of γ in pre-epidemic, epidemic, and containment periods are 3.5, 3, and 3.5, respectively.

The results of the Grey Relational Analysis (sec Table 1) and the simulation results of the Scale-Free Epidemic Model (sec Fig. 8) all show that using the exponent γ and the value of ψ to fit the Scale-Free Epidemic Model is accurate and useful.

3.2.3 *Predication and decision marking*

Fig. 9 shows that the simulation results of the Scale-Free Epidemic Model with changing ψ, γ, and m factor's values to demonstrate the influence on SARS infection.

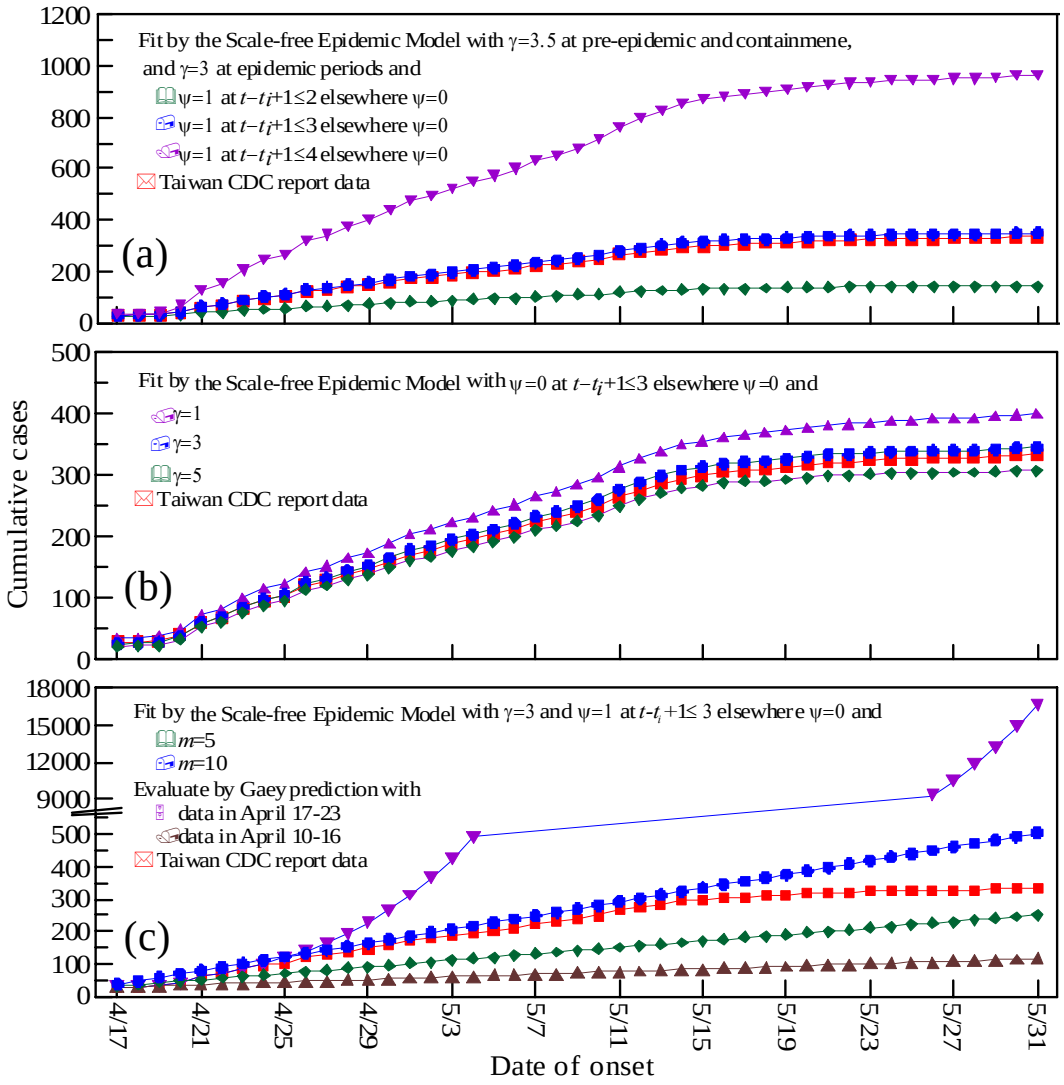

Fig. 9. The effects of ψ, γ, and m on reducing the numbers of SARS confirmed cases. (a) Changed ψ, (b) changed γ, and (c) changed m. Condition-1, 2, and 3 are $\psi = 1$, at $t - t_i + 1 \leq 2$, ≤ 3, and ≤ 4, respectively, elsewhere $\psi = 0$.

1. Effect of ψ

SARS confirmed cases in three ψ conditions are shown in Fig. 9 (see Eq. (26)). If a strict isolation policy is enforced infectious days would decrease to 2 days as shown in the green curve. The confirmed case number would be reduced to 143 (–190) calculated to May

31. If there is a loose isolation policy infectious day would increase to 4 days and the confirmed case numbers would raise to 963 (+630). The difference between the two cases is 820 lives. So a strict and efficient isolation policy is very important.

$$\left.\begin{array}{l}\text{Condition-1}: \quad \psi = 1 \text{ at } t - t_i + 1 \le 2, \text{ elsewhere } \psi = 0 \\ \text{Condition-2}: \quad \psi = 1 \text{ at } t - t_i + 1 \le 3, \text{ elsewhere } \psi = 0 \\ \text{Condition-3}: \quad \psi = 1 \text{ at } t - t_i + 1 \le 4, \text{ elsewhere } \psi = 0\end{array}\right\} \tag{26}$$

2. Effect of γ

Referring back to Eq. (2) we find that the larger value of γ, the smaller the connectivity distribution $P(k)$ therefore the infection rate and attack rate decreases, where $\gamma = 1, 3, \text{or } 5$ is used to calculate the variation of case number with results show in Fig. 9 (b). If $\gamma = 1$, the confirmed case would increase to 399 (+66); and if $\gamma = 5$, the confirmed cases would reduce to 307 (–26). Because the inverse of γ value and the attack rate, avoid contact with SARS patients can decrease the attack rate.

3. Effect of *m*

Fig. 9(c) indicates that using serious epidemic period data (April 17-26) in the Grey Prediction, and loose prevention policy, the confirmed case numbers would increase to 16,649 (+16,316) up to May 31; using serious epidemic period data (April 10-16) in the Grey Prediction, calculated to May 31, the confirmed case numbers would be 116 (–217).

In the Scale-Free Epidemic Model prediction, if the daily new confirmed case number were maintained at 10 people ($m = 10$) the case numbers would increase to 503 (+270); if $m = 5$, the case number would reduce to 250 (–83).

Therefore the strength of the epidemic infection factor is:

$$\psi > m > \gamma$$

In fact for high infection epidemic SARS, isolation quarantine and medical treatment can decrease the ψ value, which would decrease the new case numbers (*m*) and the attack rate. So strict and efficient isolation quarantine policy to reduce infectious time is important and would limit the attack rate, which would control the increase of case numbers. Method, to decrease infectious time and details about more effective prevention, needs further investigation.

3.3 Case study- II: HIV/AIDS

3.3.1. *HIV/AIDS epidemics in Taiwan in 1984~2004*

There were 9 human immunodeficiency virus (HIV(+)) infectors in Taiwan in 1984 and 2 acquired immunodeficiency syndrome(AIDS) patients in 1986. The cumulative death total is 1025, HIV(+) case are 6762 and AIDS case are 1880 as of 2004. The death ratio of HIV(+) and AIDS are 15.16% and 54.52%, The percentage of HIV(+) patients 6.94% are female, 93.06% are male; the percentage of AIDS patients 7.55% are female, 92.45% are male; he percentage of death patients 7.80% are female, 92.20% are male [24].

Fig. 10 (a) shows that cumulative HIV(+) and AIDS cases and annual HIV(+) and AIDS cases from 1984 to 2004. The results (Fig. 10(b)) show the cumulative HIV(+) and AIDS cases and annual HIV(+) and AIDS cases in the last three years (January 2002-December 2004). HIV(+) and AIDS case numbers increase every year. Prevention and effective policy are needed.

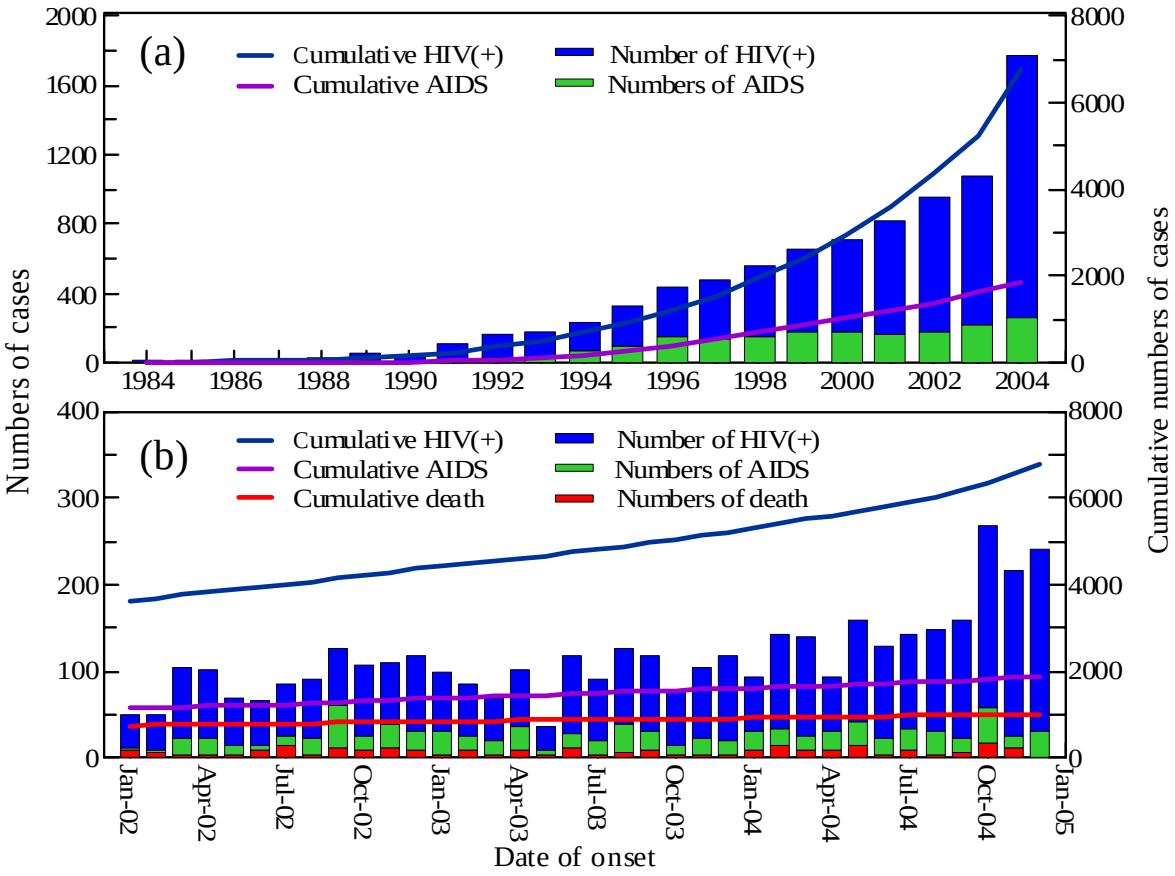

Fig. 10. Epidemiological curves of HIV(+) and AIDS in Taiwan. (a). From 1984 to 2004 and (b) from January 2002 to December 2004. (*Data from*: Center for Disease Control, Taiwan, web site [24])

3.3.2. *Model simulation*

The main infection of HIV virus is from human sexual contact. Liljeros et al. [25] proposed the exponent of the web of human sexual contacts for females and males are $\gamma = 2.1 \pm 0.3$ and $\gamma = 1.6 \pm 0.3$ when the numbers of sexual partners is greater than 20, respectively. In Taiwan, 93% of HIV(+) and AIDS patients are male. Therefore, $\gamma = 1.6$ was used in the simulation of the Scale-Free Epidemic Model.

The simulation results are shown in Fig. 11. The variable ψ is a step function and is listed in Fig. 11. The time scale for the monthly and annual simulations are t = 3 months and t = 3 years, respectively. In each time period, the infection rate decreases progressively due to the worsening health of HIV(+) and AIDS patients (sec Fig. 11(a) and (b)). This condition is identical to the progress of HIV virus infection in Taiwan.

Comparison of the simulation results with Taiwan CDC report data for HIV/AIDS cases shows that the Scale-Free Epidemic Model is accurate. The error of the monthly and annual

simulations results are < 0.41% and <0.17%, respectively. This indicates that the value of ψ is almost exact in the simulation. The Scale-Free Epidemic Model is accurate and useful again.

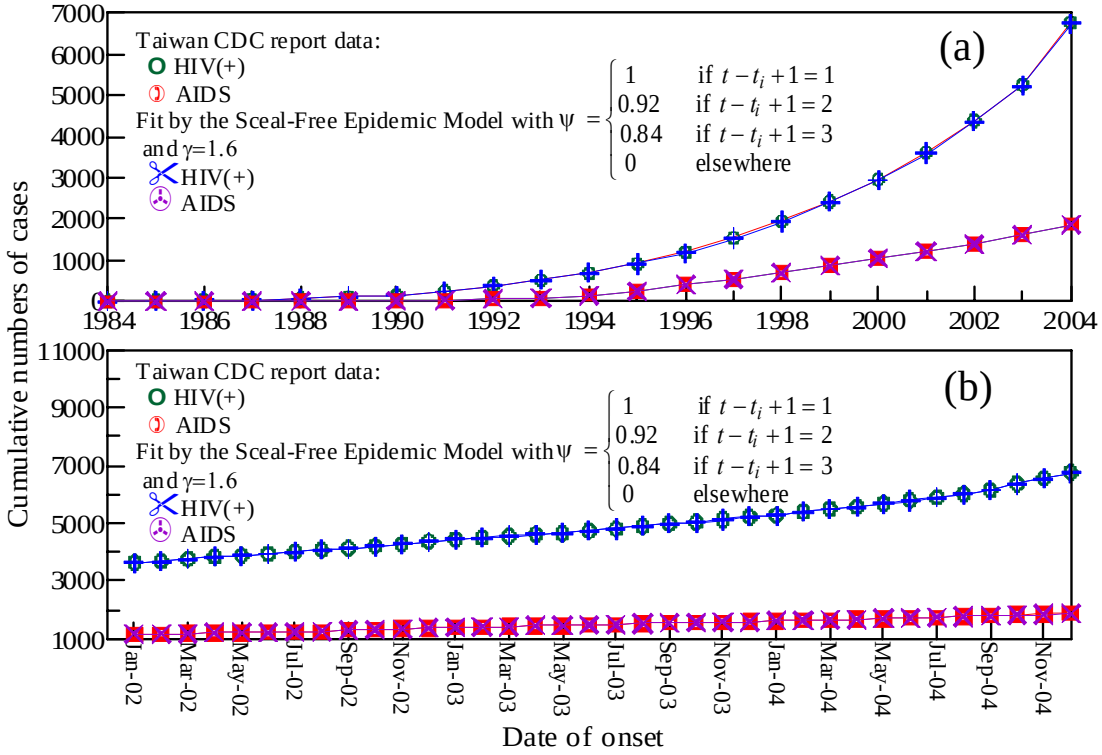

Fig. 11. Comparison the simulation results from the Scale-Free Epidemic Model with CDC reported data for HIV (+) and AIDS. (a) Annual simulation and (b) monthly simulation.

3.3.3 *Prediction and decision marking*

The Scale-Free Epidemic Model with a variable ψ and the Grey Prediction were used to investigate the effect of HIV(+) and AIDS on the Scale-Free Epidemic Model. The results are shown in Fig. 12. In the simulation, the value of ψ are listed in Eq. (27) and $\gamma = 1.6$.

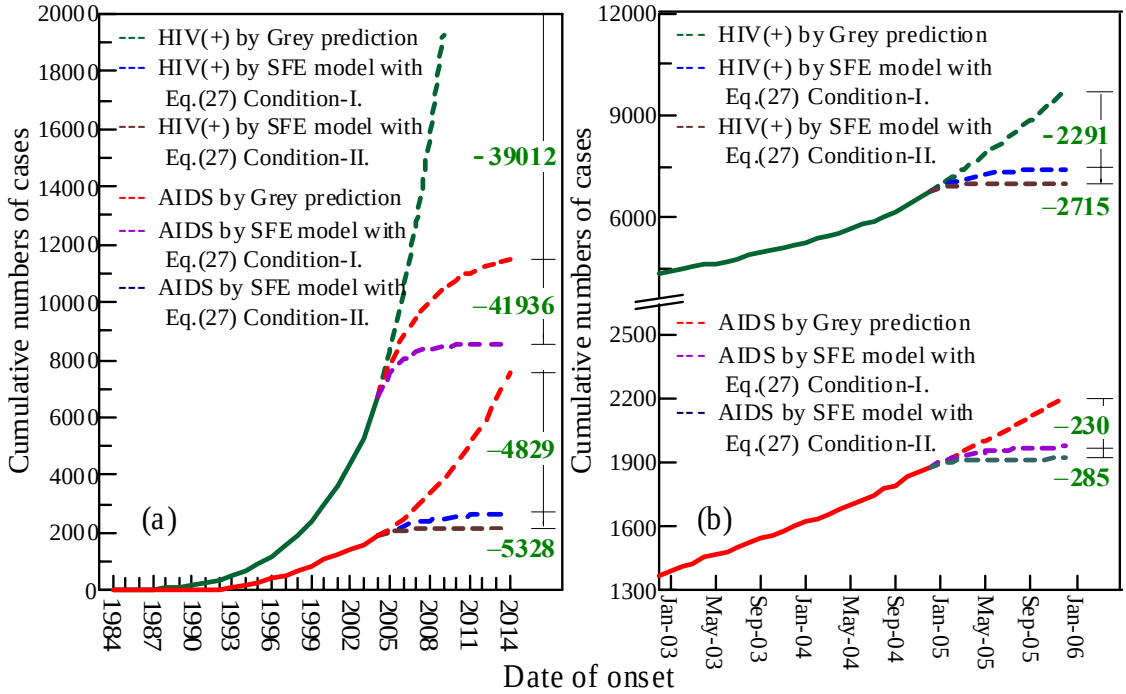

Fig. 12. The effects of ψ on reducing the numbers of HIV(+) and AIDS. SFE Model is the Scale-Free Epidemic Model. (a). Annual simulation and prediction and (b) monthly simulation and prediction

$$\text{Condition-I}: \ \psi = \begin{cases} 1 & \text{if } t - t_i + 1 = 1 \\ 0.8 & \text{if } t - t_i + 1 = 2 \\ 0.6 & \text{if } t - t_i + 1 = 3 \\ 0 & \text{elsewhere} \end{cases} \quad \text{and} \quad \text{Condition-II}: \ \psi = \begin{cases} 1 & \text{if } t - t_i + 1 = 1 \\ 0.6 & \text{if } t - t_i + 1 = 2 \\ 0.4 & \text{if } t - t_i + 1 = 3 \\ 0 & \text{elsewhere} \end{cases} \tag{27}$$

The simulation results of the Scale-Free Epidemic Model indicates with Eq. (27) Condition-I that the reduced numbers of HIV(+) and AIDS in the monthly simulation calculated to December 2005 are 2,291 and 230 (sec Fig. 12(a)) and the annual simulation calculated to December 2014 are 39,012 and 4,829 (see Fig. 12(b)). The simulation results of the Scale-Free Epidemic Model indicates with Eq. (27) Condition-II that the reduced numbers of HIV(+) and AIDS in the monthly simulation calculated to December 2005 are 2,715 and 285 and the annual simulation calculated to December 2014 are 41,936 and 5,328. However, the process to reduce ψ value to satisfy Eq.(27) conditions need deeper investigation.

3.4 A new method to fight against scale-free epidemics

Comparison of the simulation results with Taiwan CDC report data for SARS and HIV/AIDS cases show that the scale-free epidemic model is accurate and useful. However, the fight against a new epidemic and how to reduce the number of deaths is the main purpose of this study. Therefore, a new method of combining the Scale-Free Epidemic Model and the Grey Prediction to fight against epidemics is proposed. Detail of the procedure of this method is explained in Fig. 13.

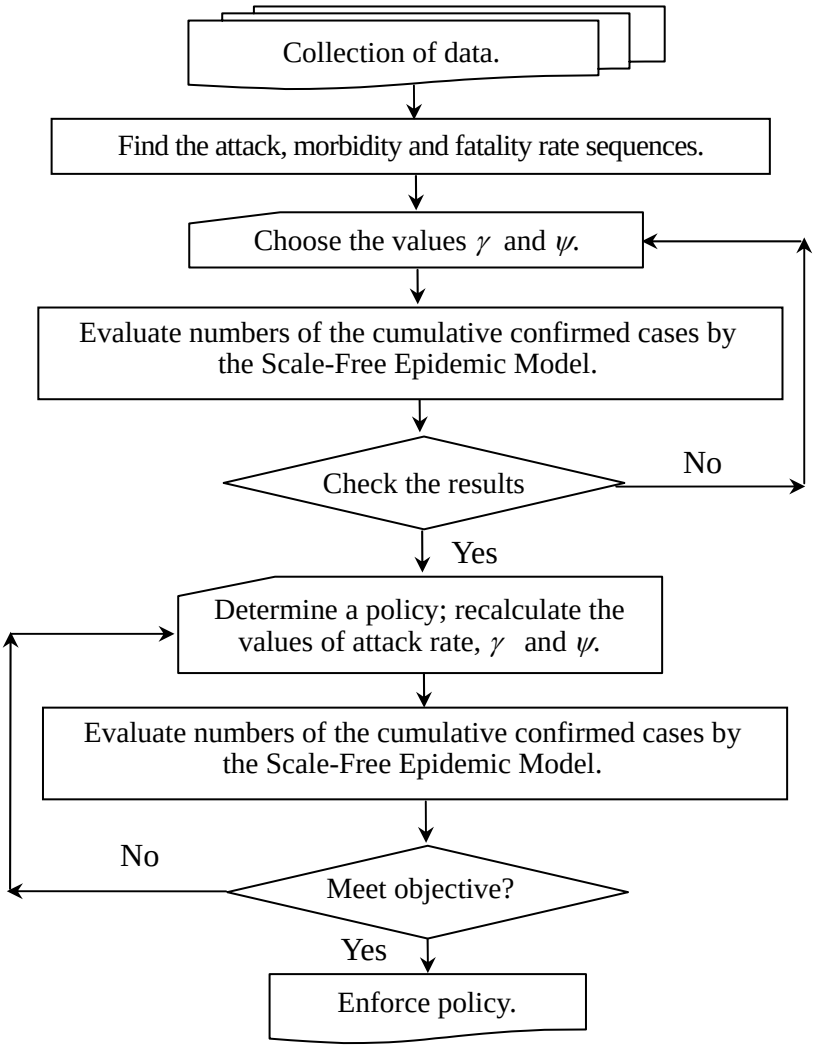

Fig.13. A new method to fight against scale-free epidemics

4. Conclusion

The course of epidemic infections resembles a scale-free network. However, they are different due to more variables in the epidemic infection. Therefore, the model of scale-free networks is not enough to satisfy the reality epidemic infections. In this study, I propose a new the Scale-Free Epidemic Model.

In the SARS case study, the results show that the sequence of effect of the epidemic factors was: $\psi > m > \gamma$. The SARS confirmed cases would decrease to 143 cases (reduced 190 confirmed cases or 3 death cases) calculated to May 31, 2003, if the average infection time was reduced to two days (an optimum value of ψ). Therefore, vigorous action in isolation quarantine and treatment for SARS cases is most effective policy; the number of new cases and the attack rate would also decrease.

In the HIV/AIDS case study, the simulation results of the Scale-Free Model indicates that the reduced numbers of HIV(+) and AIDS in the monthly simulation calculated to December 2005 are 2,715 and 285 and the annual simulation by December 2014 are 41,936 and 5,328.

Comparison of the simulation results with Taiwan CDC report data for SARS and HIV/AIDS cases show that the Scale-Free Epidemic Model is accurate and useful. This model can help determine the level of caution needed and the projected results of policy decisions. Therefore it is a useful tool in assisting the government to balance socio-economic and health concerns.

The fight against a new epidemic and how to reduce the number of deaths is the main purpose of this study. So, a new method to fight against epidemics is proposed. Detailed procedures of this method are explained.

Reference

1. Barabási A.L. and Bonabeau, E., Scale-Free Network, *Scientific American* (*Chinese*), No.**16**, 83-89 (2003).
2. Albert, R. and Barabási, A.L., Statistical Mechanics of Complex Networks, *Reviews of Modem Physics,* Vol.**74**, 47-97 (January 2002).
3. Dorogovtesev, S.N. and Mendes, J.F.F., Evolution of Network, *cond-mat*/0106144 v2, 7 September 2001.
4. Watts, D.J. and Strogatz, S.H., Collective Dynamics of Small-Word Networks, *Nature*, **393**, 440-445 (1998).
5. Erdös, P. and Rényi, A., On Random Graphs, *Publications Mathematic*, **6**, 290-295 (1959).
6. Barabási, A.L. and Albert, R. Emergence of Scaling in Random Networks, *Science*, **286**, 509-512 (October 1999).
7. Cohen, R., Erez, K., ben-Avraham, D., and Havlin, S., Resilience of the Internet to Random

Breakdowns, *cond-mat/*0007048 v2, 19 October 2001.

8. Dorogovtesev, S.N., Mendes, J.F.F., and Samukhin, A.N., Structure of Growing Networks: Exact Solution of the Barabási-Albert's Model, *cond-mat/*0004434 v1, 26 April 2000.
9. Dorogovtesev, S.N., Mendes, J.F.F., and Samukhin, A.N., Generic Scale of the "Scale-Free" Growing Networks, *cond-mat/*0011151 v1, 7 November 2000.
10. Klemm, K. and Eguíluz, V.M., Highly Cluster Scale-Free Networks, *cond-mat/*0107606 v1, 30 July 2001.
11. Klemm, K. and Eguíluz, V.M., Growing Scal-Free Networks with Small World Behavior, *cond-mat/*0107607 v1, 30 July 2001.
12. Albert, R and Barabási, A.L., Topology of Evolving Networks: LOcal Events and Universality, *Physical Review Letters*, **85**(24), 5234-5237.
13. Chen, C.J., 2004, Epidemiology: Principle and Method (*Chinese*), 2nd, Linking Inc., 73-159.
14. Gerstman, B.B., 2003, Epidemiology Kept Simple: An Introduction to Traditional and Modern Epidemiology, 2nd, *Wiley-Liss Inc.*, 338-347.
15. Gordis, L., 2004, Epidemiology, 3rd, *Elsevier Inc.*, 15-68.
16. Uen, K.L., Hwang, Y.F., Chen, F.S., Lee, Y.B., Lian, J.F., and Lay, J.R., 2002, Grey Predication: Principle and Application (*Chinese*), Chwa Taiwan Inc.
17. Uen, K.L., Hwang, Y.F., Chang, W.J., Cheng, T.J., You, M.L., and Lay, J.R., 2003, Grey Relational: Model and Application (*Chinese*), Gau Lih Book Co., Ltd.
18. Su, I.J., September 2003, Memoir of Severe Acute Respiratory Syndrome Control in Taiwan, *Center for Disease Control, Department of Health, Taiwan*, GPN: 1009202897.
19. Su, I.J., October 2003, Memoir of Severe Acute Respiratory Syndrome Control in Taiwan, *Center for Disease Control, Department of Health, Taiwan*, GPN: 1009203400.
20. Su, I.J., 2004, SARS and Flu Prevention: Taiwan Experience, *Center for Disease Control, Department of Health, Taiwan*, GPN: 1009301256.
21. Su, I.J., 2004, Statistics of Communicable Diseases and Surveillance Report in Taiwan Area (2002), *Center for Disease Control, Department of Health, Taiwan*, GPN: 2009300465.
22. Department of health, Taiwan, Web, http://www.doh.grv.tw/newverprog/proclaim/sars_list.asp
23. Union Daily News, Taiwan, March 20-June 15, 2003.20
24. Center for Disease Control, Taiwan, Web, http://www.cdc.grv.tw
25. Liljeros, F., Edling, C.R., Åmaral, L.A.N., Stanley, H.E., and Aberg, Y., 2001, The Web of Human Sexual Contacts, *Nature*, **411**, 907-908.