

**An Investigation of Errors and of Quasi stationary  
behaviour for Pairwise Epidemic Models**

by

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**Thesis submitted in accordance with the requirements  
of the University of Liverpool for the  
degree of Doctor in Philosophy**

**March, 2016**

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# Dedication

This thesis is dedicated to the MIGHTY WARRIOR, THE ANCIENT OF DAYS  
from whom perfect gifts come.

## Acknowledgement

My profound gratitude goes to my supervisor, Doctor Kieran J. Sharkey, for his great support, guidance, time and patience throughout my doctoral studies at the University of Liverpool.

My undiluted gratitude also goes to the entire staff and students of the department of Mathematics especially, the HOD, Professor kurt Langfeld, the Departmental Director of Postgraduate Research, Dr. Vladimir Guletskii and to the Mathematical Biology group of the department.

I am indeed very grateful to my Uncle, Pastor Adebayo Israel for all his prayers and moral support and I owe much to my Mum, siblings, children and my wife, **Adetoun Mary Adeniji**, for believing in me and for the pains my absence caused them. I also appreciate my special friends, Suulola Akinyanju and brother Lanre Sangobunmi for their support.

Finally, I want to appreciate the entire staff of the Department of Mathematics and Management of Tai Solarin University of Education, Nigeria for their love and support.

Dapo LOYINMI  
Liverpool, 2016.

# Abstract

There are various modelling approaches designed to incorporate the complexities of the spread of infectious diseases in populations. At the level of individuals, the epidemic dynamics with Poisson transmission and removal processes can be described by a hierarchy of moment equations starting from the equations for the states of single individuals. In such equations, lower order quantities are expressed in terms of higher order quantities and for numerical feasibility the system is usually closed at some level through an approximate moment closure relation leading to lower-dimensional ordinary differential equations(ODE) models.

Here we focus our attention on pair-approximation models where hierarchy is closed at the level of pairs by approximating higher order terms. This method enables considerable heterogeneity that may be present in the network to be incorporated. It also provides insights into the connection between stochastic and deterministic models. Most of the work presented in this thesis is based around this type of model.

In chapter 2, we first develop a class of deterministic Susceptible-Exposed-Infectious-Removed (*SEIR*) epidemic models on time-independent weighted contact networks where the exposed, infection and removal processes are Poisson. Following earlier work on *SIR* dynamics, we then prove that a pair-level closed form of this model generates an exact representation of the expected infection time series for tree networks under the condition that the transmission rate across all links in the network is constant and the recovery rate for an infected individual is also constant.

It has been identified that there is a connection between the structure of networks and the validity of the type of moment closure that we consider here. In chapter 3, we make an investigation of the accuracy of moment closure relations. In particular, the presence of lower-order cycles such as triangles, is a major problem with network-based epidemic models that are approximated by second order moment closure equations. Cycles make it difficult to match a low-dimensional approximate model with its corresponding stochastic counterpart. Our study focusses on trying to understand and quantify the errors that emerge in *SIR* dynamics on networks comprising of a single cycle; triangle, square, pentagon and hexagon.

Finally, we look at *SIS* dynamics on networks. These dynamics eventually approach an absorbing state but nevertheless can appear to be stationary over a reasonable period of time. The time it takes for some absorbing stochastic process to

absorb can sometimes be long. The concept of quasi-stationary distribution(QSD) is used to represent this stationary-like behaviour. We employ the Master equation to calculate the average of the QSD on small networks and compare this with simulations. This approach is not feasible for larger networks due to the huge number of equations the method generated. We then develop a pair approximation method for accurately approximating the average of the QSD on large networks.

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# Chapter 1

## Introduction

### 1.1 Epidemic and modeling

Infectious diseases have remained the major cause of mortality in developing countries of the world despite the availability of effective antibiotic and vaccination programmes to fight and subsequently eliminate them [25, 27]. An infection outbreak may happen in one settlement or even extend to many countries [33]. It is also possible for the infection to persist from days to years and a single case of a new disease or an occurrence of a disease which has been away from a population for a long time may be considered an outbreak [1, 162]. An outbreak becomes epidemic when it spreads rapidly to many individuals.

Infectious diseases have inflicted terrible damage to mankind causing economic damage and social disruption throughout the centuries. Whole populations were almost completely wiped out, blood lines ended and more casualties have been claimed by epidemics than wars [25].

Early human beings came in contact with disease-causing microbes in drinking water, food and the environment. Once in a while an outbreak might considerably reduce the population of a small group but they never came close to experiencing the extensive diseases of more recent times [24, 25]. The fact that Ebola virus, among other deadly zoonists, is believed to be transmitted to people from wild animals is evidence that animal or human invasion of new ecosystems provides the opportunity for new and existing diseases and that humans opened themselves up to new and deadlier diseases by domesticating animals that have their own microbes [170, 171]. Human beings built wells and ditches to sustain their expanded population and by so doing they unconsciously provided more standing water to breed disease-carrying mosquitoes, hence as human expand their territory they came into close contact with microbes they might otherwise have never met so

communicable diseases had the opportunity to spread to epidemic proportions as human begin to come together in large number [25, 27].

New microbes could also easily find their way from a highly populated area to another as technology allows for wider travel and trade. Ironically, scientific invention in the transportation industry paves the way for one of the greatest threat to the modern human society. And as we continue to grow, so too microbes continue to change and transform to more lethal and drug-resistant forms [24, 26]. New diseases have emerged in recent times. Prominent epidemics include Smallpox, the Great flu, the Black Death, Malaria, Tuberculosis, Cholera, AIDS, Yellow fever and Typhus.

In recent times, we have witnessed at a frightening rate, the occurrence of new diseases and re-occurrence of old diseases in many parts of the world. An example of the coming back of an old menace is the Ebola disease that recently ravaged three West African countries-Liberia, Sierra Leone and Guinea [21, 48]. The virus which is particular to West Africa was first discovered nearly four decades ago in Congo in a village near the Ebola river and since then there have been random outbreaks. The recent outbreak, the largest in history, sickened and killed thousands of people since March 2014 [48].

Another threat to humanity is the recent emergence of ‘*MERS*’ infection in South Korean. In this era of ever growing population and national and international interactions, epidemic diseases are major threat. The occurrence and re occurrence of diseases encouraged interest in communicable diseases. Epidemics are dangerous occurrences, not only from a biological perspective, as infectious diseases, but also from a technological point of view, as malware propagation [92, 178]. Without a doubt, epidemics can cause great damage, and so developing the proper and useful models for epidemics is necessary to effectively use scarce control resources.

Epidemic models help us to understand how infectious diseases spread in a population and how various complexities of the pattern of interactions affect its propagation. It presents an epidemiologist with an ideal-world where different factors, such as number of partners [2, 17, 45, 49] and the effect of localized spread of infection [91, 92, 95] responsible for disease propagation can be isolated and examined in a fairly general framework, and where every stage of the disease is recorded in detail. Epidemic models as an approximation to reality are typically based on some simple assumptions, ignoring some individual differences such as the

susceptibility of individuals to infection, variation in the immunological response and transmissibility and so on. Biological characteristics such as the transmission rate are then translated into mathematical variables which leads to sets of equations that can be analysed to give meaningful mathematical conclusions from which meaningful biological interpretations can be derived [2, 74].

In epidemic models of communicable infections, individuals with some level of immunity which has not contracted a disease yet but could catch it if they come in contact with someone who does is referred to as **Susceptible**. When they become infected with a micro parasite, the pathogen begins to grow within the host and there is an interaction between the pathogen and the host's immune response [1, 75, 78]. The abundance of the pathogen grows over time and at this early stage, the host may show no symptoms of infection as the abundance of the pathogen may be too low to transmit infection to a susceptible individual. At this stage, the host is referred to as **Exposed**. As the growth rate of the pathogen increases and the level of parasite is large enough to transmit infection to a susceptible individual, the original host is referred to as being **Infectious**.

Finally, when the host's immune system has subdued the parasite and the host is no longer infectious, then the host is referred to as **Recovered**. It is worth mentioning that **removal** can happen either through quarantine (i.e. isolation of an infected individual from the rest of the population to prevent further spread of a contagious disease) or through immunization against infection or through death caused by the disease. These categorization are equivalent from a modelling point of view which only consider the infection status of individuals. For any model, the progression between these classes are translated into formal mathematical terms to make quantitative predictions.

## 1.2 Simple Epidemic models

In basic epidemic models, individuals in a population are grouped into different compartments according to their infection status. Notations such as  $\mathbf{S}(t)$ ,  $\mathbf{E}(t)$ ,  $\mathbf{I}(t)$  and  $\mathbf{R}(t)$  are often used to represent the number of individuals in the respective compartments of being susceptible, exposed, infectious and removed. The independent variable in this type of model is time,  $t$ , and the rate of change of this quantities from one compartment to the other are usually expressed as derivatives based on the assumption that the population size in each compartment is differentiable with respect to time. A differential equations based epidemic model is a

reasonable approximation provided there are many members in each compartment, and the epidemic process of such model is assumed *deterministic*.

Among notable early deterministic epidemic models was that formulated by Daniel Bernoulli in 1760 [41] to investigate the efficiency of variolation of healthy people with the smallpox virus. Another one was the discrete time model analysed by William Heaton Hamer in 1906 to understand the recurrence of measles epidemics [71]. Hamer's model was probably the first to assume that the number of cases per unit time depends on the product of the densities of the susceptible and infective [71].

Important properties, such as the final size of the epidemic and its duration can be predicted once an infectious disease is modelled by simple compartmental models [7, 41, 59, 62].

### 1.2.1 The *SI* model

Some infections, especially of plants, where the infected host remains infected forever, are better described by the **SI** (Susceptible-Infectious) model [2, 71].

Although this two-states categorisation of individuals as either being susceptible or infectious hides lots of biological information. It captures some of the many features of disease dynamics and simplifies our consideration of what goes on in the population rather than what goes on within the host [2, 8]. This model which can be modified in many ways to fit the description of specific infection dynamic applies to fatal infections such as HIV and the Highly Pathogenic Avian Influenza (H5N1) [1, 62] whereby the host remains infected and infectious forever.

The progression from state **S** to state **I** in such a closed population can be conceptually represented by a flow diagram of figure (1.1). It is essential to note that the choice of which compartments to include in a model depends on the characteristics of the particular infection under investigation and the purpose of the model. Acronyms for epidemic models such **SEIR**, **SIR**, **SI** and **SIS** are usually based on the flow pattern between the compartments.

Let us consider an infection spreading through a closed population of size **N** and denote the respective number of susceptible and infectious individuals at time  $t$  by **S** and **I**. We first note that 'effective contact' between a susceptible and infectious individuals transmits infection. The transition rate between susceptible and infectious individuals is  $\beta I$ , where  $\beta$  takes into consideration the probability that infection is contracted when susceptible and infectious individuals make contact [62, 114]. The total rate of new infection is  $\beta I \frac{S}{N}$  and the compartmental flow rate equations for a fixed population can then be written as

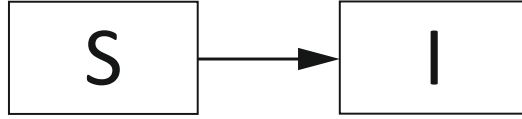


Figure 1.1: *Flow of  $S \rightarrow I$  in a well-mixed closed population.*

$$\begin{aligned}\frac{dS}{dt} &= -\beta I \frac{S}{N}, \\ \frac{dI}{dt} &= \beta I \frac{S}{N}.\end{aligned}\tag{1.1}$$

We can also use variables defined as fraction of the population:

$$x = \frac{S}{N} \quad \text{and} \quad y = \frac{I}{N}$$

In terms of these, equation (1.1) becomes

$$\begin{aligned}\frac{dx}{dt} &= -\beta xy, \\ \frac{dy}{dt} &= \beta xy.\end{aligned}\tag{1.2}$$

Since  $S + I = N$ , or equivalently  $x + y = 1$ , we can remove  $x$  from the equations by putting  $x = 1 - y$  as every individual is either susceptible or infectious, and from (1.2) we have

$$\frac{dy}{dt} = \beta(1 - y)y.\tag{1.3}$$

The solution of (1.3) by integration of its partial fractions is given as

$$y(t) = \frac{y_0 e^{\beta t}}{1 - y_0 + y_0 e^{\beta t}}.\tag{1.4}$$

where  $y_0$  is the initial fraction of infective in the population.

Equation (1.4) is referred to as *logistic growth equation* in biological studies, physics and many science fields [114].

For a typical population of 1,000,  $\beta = 1.5$  and with one initial infected individual, equation (1.4) produces an S-shaped curve, figure (1.2) showing an initial exponential growth of infection. As the figure illustrates, the whole population gets infected. This is possible because an infected individual in the population remains infected and can infect any other susceptible.

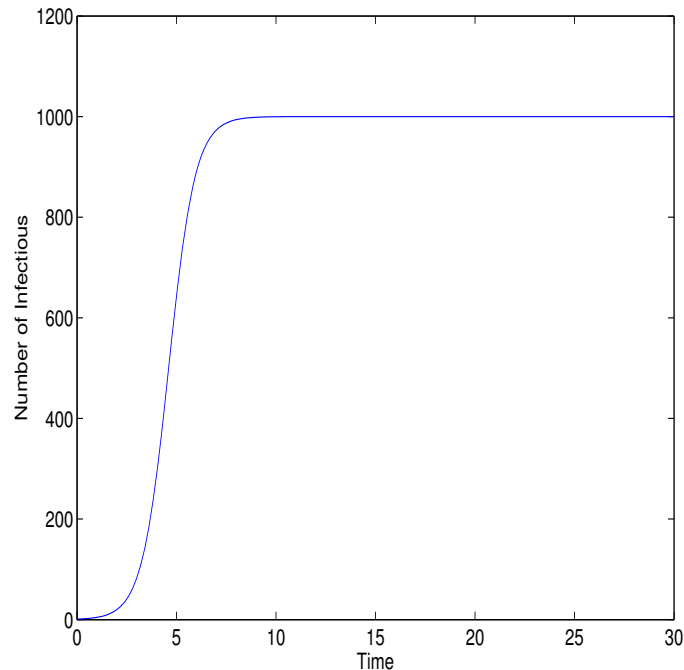


Figure 1.2: *The logistic growth curve of the SI model for a population with 1000 individuals, transmission rate,  $\beta = 1.5$  and a single initially infected individual.*

### 1.2.2 The *SIR* model

Some diseases such as measles give permanent immunity to the host after treatment and consequently their dynamics are best described by an **SIR** (Susceptible-Infectious-Recovered) model. This model which is an extension of the **SI** model is first formulated (though never published) by Lowell Reed and Wade Hampton Frost in the 1920s [68].

An infected individual is assumed to recover from an infection at a constant rate  $\gamma$  and the progression from state  $S$  to  $I$  and to  $R$  in a closed population can be conceptually represented by a flow diagram as shown in figure (1.3). In general, the rate at which a susceptible individual moves into the infected compartment and from the infected compartment to the recovery class yield the coupled non-linear differential equation (1.5).



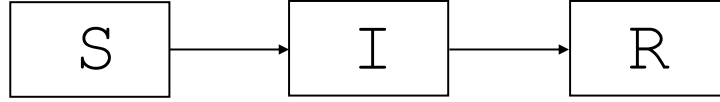


Figure 1.3: *Flow of  $S \rightarrow I$  and  $I \rightarrow R$  in a closed population.*

$$\begin{aligned}
 \frac{dx}{dt} &= -\beta xy, \\
 \frac{dy}{dt} &= \beta xy - \gamma y, \\
 \frac{dz}{dt} &= \gamma y.
 \end{aligned} \tag{1.5}$$

where  $x + y + z = 1$  is a constant, with  $x_0 > 0$ ,  $y_0 > 0$ . It is assumed  $z_0 = 0$  and so  $z_t > 0 \forall t > 0$ .  $z = \frac{R}{N}$ ,  $R$  is the number of removed or recovered individual(s).

Although these equations can not be solved explicitly but the second expression in (1.5), i.e.  $\frac{dy}{dt} = (\beta x - \gamma)y$  indicates that when  $\beta x \leq \gamma$ , the rate of change of the number of infected decreases with time. On the other hand when  $\beta x > \gamma$ , then  $\frac{dy}{dt} > 0$  at least initially, and the number of infected increases at the beginning. We just observed the threshold phenomena  $R_0 = \frac{\beta}{\gamma}$  where the infection spreads.

The critical quantity,  $\frac{\beta}{\gamma} = R_0$  is referred to as the basic reproductive ratio and it is defined as the average number of secondary cases arising from an average primary case when introduced into an entirely susceptible population[75, 78]. For an infection to invade a population,  $R_0$  must be greater than 1. Control measures such as vaccination can be applied to reduce  $R_0$  below this value and thus the infection from the population before invasion.

For a closed population of  $N=1,000$  with  $I_0 = 1$ ,  $\beta = 1.5$  and  $\gamma = 1$ , equation (1.5) gives the curves in figure (1.4)

### 1.2.3 The *SIS* model

This model is another extension of the **SI** model where recovery of an infected individual is temporary and recovery of host is immediately followed by the individuals becoming susceptible. Examples of human infections that give temporary immunity to victims are many of the sexually transmitted diseases (STDs) such as gonorrhoea and Syphilis [49, 52, 75]. The infected after recovery immediately replenishes the susceptible pool.

The dynamics of such infections are best described by an **SIS** (Susceptible-Infectious-Susceptible) model which was first introduced in 1971 by Weiss and

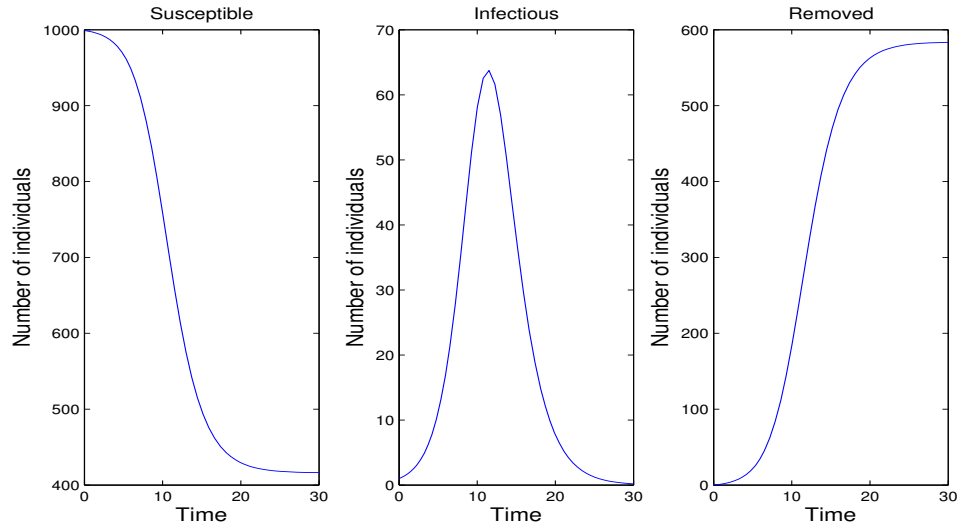


Figure 1.4: *The SIR epidemic dynamics: The trend of  $S$ ,  $I$  and  $R$  in a closed population of  $N = 1,000$  for  $I_0 = 1$ ,  $\beta = 1.5$  and  $\gamma = 1$ .*

Dishon [177]. The disease is able to remain in the population in the presence of an infected individual. With a constant recovery rate  $\gamma$  of an infected individual, the

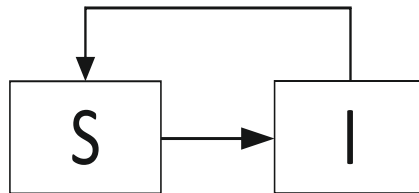


Figure 1.5: *Flow of  $S \rightarrow I$  and  $I \rightarrow S$  in a closed population.*

differential equations for this model are

$$\begin{aligned} \frac{dx}{dt} &= -\beta xy + \gamma y, \\ \frac{dy}{dt} &= \beta xy - \gamma y. \end{aligned} \tag{1.6}$$

And writing  $x = 1 - y$  and we have

$$\frac{dy}{dt} = (\beta - \gamma - \beta y)y \tag{1.7}$$

which after solving gives

$$y(t) = y_0 \frac{(\beta - \gamma)e^{(\beta - \gamma)t}}{\beta - \gamma + \beta y_0 e^{(\beta - \gamma)t}}. \quad (1.8)$$

For same parameter values;  $N = 1,000$ ,  $\beta = 1.5$ ,  $\gamma = 1$  and with one initially infected individual, equation (1.8) gives a logistic growth curve similar to that for **SI** model but different in the sense that the whole population is never infected with the disease.

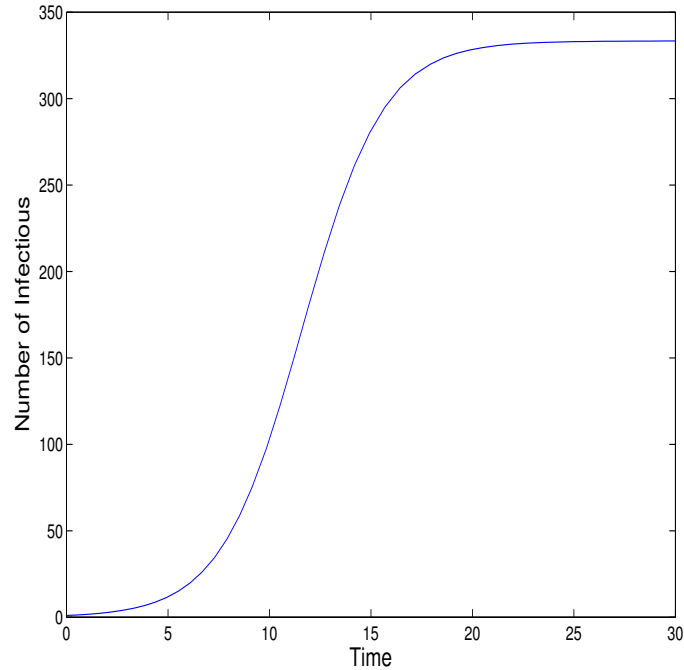


Figure 1.6: *The logistic growth curve of the SIS model for a 1,000 population, transmission rate,  $\beta = 1.5$ , recovery rate  $\gamma = 1$ , initiated with a single infected individual.*

### The endemic state

In the long run the **SIS** model reaches a stable state where the rate at which individual(s) are infected equals the rate at which people recover and a steady fraction of the population is always infected with the disease [111, 120]. The fraction infected at this stage can be obtained from and by setting (1.7) to zero. That is:

$$\frac{dy}{dt} = (\beta - \gamma - \beta y)y = 0 \quad \text{which gives} \quad y = \frac{\beta - \gamma}{\beta}$$

This steady state is an example of an **endemic disease state** [75, 114]. Provided  $R_0 > 1$ , the disease persists in the population. It is worth mentioning that

apart from the application of the *SIS* model to modelling the spread of diseases in humans and animals, it has a range of other applications such as modelling of the spread of viruses in computer networks [92, 127, 178].

The **SIS** and **SIR** models are two behavioural extremes where immunity simply does not occur or is lifelong [75, 78]. An intermediate assumption is that the host immunity after treatment or immune response lasts for a limited time before waning and thereby becoming susceptible again. The description of such infection dynamics can be represented by an **SIRS** model where removed individuals can move back to being susceptible. An example of an infection with such a profile is the 9-10 cycle observed in the Syphilis case in the United State case in 2005 [54].

Another variation of the **SIR** model is the inclusion of latent periods. This a period when the infected individual shows no symptoms of infection but carries the disease causing pathogens. At this stage the pathogens undergo unchallenged reproduction within the host. The host are referred to as being exposed and are duly represented by the variable **E** when the infection profile is being adequately captured by the **SEIR** model[75, 152].

### 1.3 Stochastic models

All the models described above can be studied in a more realistic stochastic framework by a class of stochastic models called Markov processes. ‘Stochastic’ indicates being or having a random variable [6, 11]. A stochastic model is a means of simulating or calculating probability distributions of possible outcomes by giving room for random variation in one or more inputs over time [37, 43]. Stochastic models are good tools when these fluctuations matter and are particularly useful when considering small populations [59, 76].

The so called Kolmogorov forward equation, also known as the master equation, permits one to examine the probability of occurrence of each state as a function of time. This equation which provides simple exact methods for evaluating population infection size is linear and has a natural matrix formulation of dynamics which absorbs the non-linearity associated with disease transmission processes, say for example, of systems (1.2), (1.5) and (1.6). Markov processes, using integer-based events (infection and recovery in this case) model stochastic population processes with the future state of the population determined by the current state- the system has no memory[76, 94, 146, 167]. The fundamental procedure allows the entire combination of stochastic activity to be predicted by a large set of deterministic equations. For example, the **SIS** dynamic will require  $N + 1$  differential equation

while **SIR** dynamic will require  $\frac{(N+1)(N+2)}{2}$  differential equation, where  $N$  is the number of individual in the population [76, 167].

Essentially, the Kolmogorov (or master equation) equation consists of a single matrix equation for the probability of being in each possible state, with the dynamics governed by the transition rates between states [76]. Hence, one can find a complete description of all possible behaviours of the stochastic system, solving one set of differential equations. For example, the  $N + 1$  differential equations for **SIS** dynamics correspond to the states  $I = \{0, 1, \dots, N\}$ . Letting  $P_I(t)$  be the probability that there are  $\mathbf{I}$  infectious individuals at a time  $t$  in the population and construct a set of differential equations for these probabilities, the so called Kolmogorov forward equations are:

$$\begin{aligned} \frac{d}{dt}P_I &= [\beta(N - I + 1)(N - 1)] P_{I-1} + [\gamma(I + 1)] P_{I+1} \\ &\quad - [\beta(N - 1)I + \gamma I] P_I, \end{aligned} \tag{1.9}$$

with  $P_{-1} = 0$  and  $P_{N+1} = 0$  because the population of infectious can not be negative and can also not be greater than  $N$ . The first term of (1.9) corresponds to increment in  $\mathbf{I}$  from  $\mathbf{I}+1$  by infection, the second term corresponds to reduction in  $\mathbf{I}$  from  $\mathbf{I}+1$  by recovery from infection and the third term corresponds to no recovery and no infection, and so the population of infection remains  $\mathbf{I}$ .

System (1.9) can be written in terms of simpler matrix and vector form, that is

$$\frac{dP}{dt} = P(t)Q, \tag{1.10}$$

where  $P$  is the row vector of the  $N + 1$  probabilities and  $Q$  is the transition rate matrix between states (see [76, 167] for more detail). The solution of (1.10) can be put in the form

$$P(t) = P(0)e^{Q(t)} = \sum_{n=1}^{N+1} q_n e^{\lambda_n t}, \tag{1.11}$$

where the eigenvectors,  $q_n$  and eigenvalues,  $\lambda_n$  are determined from the matrix  $Q$  and the initial distribution,  $P(0)$ .

### 1.3.1 The Quasi Stationary Distribution

One of the most important difference between the stochastic and the deterministic models is their asymptotic dynamics. For example, the **SIS** model for a closed population predicts infection growth and attains a lasting steady state as long as  $R_0 > 1$ , while its stochastic counterpart predicts ultimate extinction of infection

at  $\mathbf{I}=0$  irrespective of the initial condition(s). In such cases, we may be interested in the distribution of the dynamic conditioned on non absorption. This is the distribution, given that the disease is still in the population. This is described by the concept of **quasi stationary distribution** (QSD) which can be estimated adequately by forming a matrix  $Q^*$  from matrix  $Q$  by removing the first row and column which correspond to the transition process of the absorbing state,  $\mathbf{I}=0$ . The first normalized eigenvector  $\lambda^*$  of  $Q^*$  is the QSD of the **SIS** dynamics in a closed population.

This concept is used to model the behaviour of some stochastic systems which eventually approach an absorbing state but nevertheless appear stationary over a reasonable period of time. It is a powerful tool in many area of scientific studies. For example, in wild life management, it is used to predict persistence times, and the distribution of the number of individuals in animal populations that are subject to large-scale mortality or emigration [129, 133, 145]. It is also used to model the spread of a computer virus across a network with cure and reinfection [79, 105].

We consider this concept extensively in chapter 4 where we evaluate the average of the number of infectious individual in the quasi stationary state of the Markovian **SIS** epidemic dynamics on graphs.

## 1.4 The event driven approach - the SIR model

We outline a step by step pseudo code which can be used for implementing the Gillespie algorithm [53] to simulate a Markovia process (or model) for, say an *SIR* epidemic dynamics in a closed population. Transmission of infection is a **chance event**. The event-driven approach is one way of defining the fluctuation in population processes that happens as a result of the random nature of events in terms of individual. Here, the number of susceptible, infectious and recovery individual is treated as integer.

Consider an **SIR** model on a closed population of size  $N$  where infection and recovery are Poisson processes with respective constant rate of  $\tau$  and  $\gamma$ . Let  $\mathbf{S}$ ,  $\mathbf{I}$  and  $\mathbf{R}$  as the respective number of susceptible, infectious and removed in the population at any time  $t$ , written as  $(\mathbf{S}, \mathbf{I}, \mathbf{N}-\mathbf{S}-\mathbf{I})$ , there are two possible events in the population- either infection with rate  $\tau SI$  or recovery with rate  $\gamma I$ .

- The total event pressure on the process as a whole is  $\lambda = \tau SI + \gamma I$ .
- The time to next event is a Poisson process with rate,  $\lambda$ . That is,  $t \rightarrow t + \delta t$

where  $\delta t = -\frac{\ln(r)}{\lambda}$  is the waiting time for the next event and  $r$  is a uniformly generated random number between 0 and 1.

- The probability that the event is infection is  $\frac{\tau SI}{\lambda}$ , and the probability that the event is recovery is  $\frac{\gamma I}{\lambda}$ .
- After the waiting time, if the event on the process is infection then the population of infectious individuals increases by 1 while the population of susceptible is reduced by 1, that is,  $(\mathbf{S}, \mathbf{I}, \mathbf{N-S-I}) \rightarrow (\mathbf{S-1}, \mathbf{I+1}, \mathbf{N-S-I})$
- If the event is recovery then the population of recovered individuals increases by 1 while the population of infectious is reduced by 1, that is,  $(\mathbf{S}, \mathbf{I}, \mathbf{N-S-I}) \rightarrow (\mathbf{S}, \mathbf{I-1}, \mathbf{N-S-I+1})$

This code is run repeatedly, updating the various variables at each time step, until the number of infective goes to zero. Two realizations from implementing the pseudo code with the same set of parameter values of figure (1.4) is given in figure (1.7). For many epidemic models, averaging over such many realizations of the process converges to that of their deterministic counterparts in the limit of infinite population.

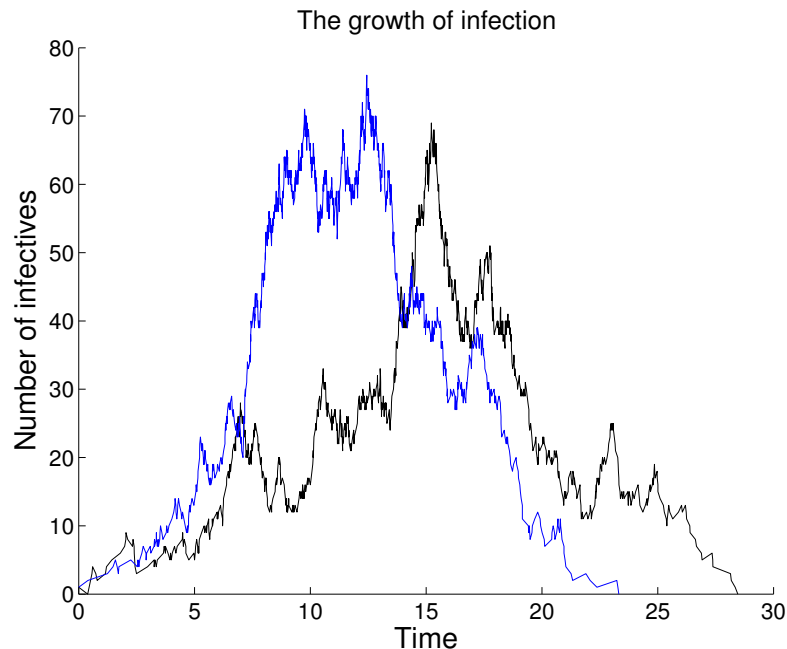


Figure 1.7: *Two different realizations of the stochastic SIR compartmental model for  $N = 1000$ ,  $\beta = 1.5$ , one initially infected individual and  $\gamma = 1$*

## 1.5 Networks

The assumption of a well mixed population is an inadequate one for the dynamics of many infectious diseases. Human interactions are limited by many factors, hence heterogeneous mixing is a fact for many epidemiological system as the infection status of an individual in a population depends on the interaction with limited neighbours. Among the potential limitations to possible contacts between individuals in the population are proximity, socio-economic status and culture [12, 46, 49]. Many social systems such as friendship networks, biological systems such as the food web and communication systems such as the internet can be adequately represented by complex networks whose nodes represent individuals or group of individuals, and connections represent the interactions among them [114]. These connections between individuals or group of individuals which ultimately allow infectious disease to spread naturally define a network. The wide applicability of networks as a modelling tool has revolutionized research into the dynamics of interacting units.

Modelling population growth in different biological systems is often very similar. For example, studying forest population growth with suitable and non suitable habitats (ecology) is mathematically similar to studying the growth of infection in a host population of susceptible and infectious individuals (epidemiology) [18, 88, 92, 127]. An important application of networks is epidemiology where mathematical models involving networks give fundamental insights into epidemic dynamics.

Networks can be defined as a collection of points (vertices/nodes) joined by lines(edges) (figure (1.8)). Networks can be represented mathematically by an

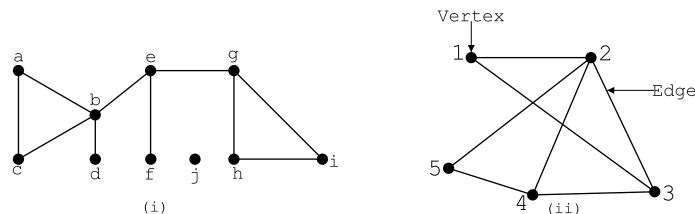


Figure 1.8: *Simple undirected networks*

adjacency matrix  $G$  where,

$$g_{ij} = \begin{cases} 1, & \text{if node } i \text{ is connected to node } j, \\ 0, & \text{otherwise.} \end{cases}$$



For the network in fig 1.8(ii);

$$G = \begin{bmatrix} g_{11} & g_{12} & g_{13} & g_{14} & g_{15} \\ g_{21} & g_{22} & g_{23} & g_{24} & g_{25} \\ g_{31} & g_{32} & g_{33} & g_{34} & g_{35} \\ g_{41} & g_{42} & g_{43} & g_{44} & g_{45} \\ g_{51} & g_{52} & g_{53} & g_{54} & g_{55} \end{bmatrix} = \begin{bmatrix} 0 & 1 & 1 & 0 & 0 \\ 1 & 0 & 1 & 1 & 1 \\ 1 & 1 & 0 & 1 & 0 \\ 0 & 1 & 1 & 0 & 1 \\ 0 & 1 & 0 & 1 & 0 \end{bmatrix}$$

A number of important quantities can be derived from the adjacency matrix. For a population of size  $N$ , the average number of neighbours per individual is

$$\langle k \rangle = \frac{1}{N} \sum_{ij=1}^N G_{ij} = \frac{1}{N} \|G\|$$

where  $\|G\|$  represents the number of ordered links in the network. The degree  $k$  of a node  $i$  in a network is the number of connections it has to other nodes. That is,

$$k_i = \sum_{j=1}^N G_{ij}$$

For example, node 2 in fig (1.8) has degree 4 which corresponds to the sum of the second row of matrix  $G$  above.

### 1.5.1 Structure Of Networks

Networks provide a rich framework for studying the propagation of infection in human and animal populations. However, modelling transmission through networks is mathematically and computationally daunting due to the high-dimensionality of networks themselves. Robust analytical results remain scarce though some progress has been made to improve the practical usefulness of network-based models by taking into consideration realistic features of mixing patterns. There is some structure behind social interaction; this structure, which can be represented by a contact network, determines the relationships that are permitted and the individuals capable of transmitting infection to each other [45, 46, 73, 99, 111, 112].

Good knowledge of this structure in epidemiology is essential to understanding routes through which diseases spread in a population and this leads to designing effective control measure. For example, contact tracing [75] which is a highly effective public health control identifies possible transmission network connections from known infected individuals and treats or regulates their contacts thereby reducing the spread of infection. A ready example is the first case of the dreaded Ebola

disease in Nigeria. In early April 2014, the Liberian Ebola infected air passenger (Mr. Sawyer) from Liberia reportedly showed no Ebola symptoms when boarding the plane to Nigeria but was vomiting and had diarrhoea by the time he arrived Nigeria's biggest city, Lagos. Although before then, there have not been case(s) of Ebola spreading through air travel, nearly 50 other passengers on the flight were quickly *traced* and monitored for signs of Ebola. With contact tracing combined with other robust control intervention, Ebola was successfully tamed and stamped out from the country within a relatively short period as World Health Organisation declared the country Ebola-free six months after the first case [48].

Important network properties which characterise the pattern of network structure and which can have profound effects on epidemic process on networks include the network link density, degree distribution, associativity of nodes, clustering coefficient, component size, path lengths between nodes, betweenness, and so on. The importance of some of these network properties are briefly explained below.

### Network link density

Let  $m$  be the number of links in a network, we have that  $2m = \sum_{ij=1}^N G_{ij}$ , i.e the sum of the degree of all the nodes in the network [114]. Therefore, the number of links,  $m$ , in a network is given by

$$m = \frac{1}{2} \sum_{ij=1}^N G_{ij}. \quad (1.12)$$

The average degree,  $c$  of a node in an undirected loopless network is given by

$$c = \frac{1}{N} \sum_{ij=1}^N G_{ij}, \quad (1.13)$$

where  $N$  is the number of nodes in the network. Equations (1.12) and (1.13) combine to give

$$c = \frac{2m}{N}.$$

The maximum possible number of links present in an undirected loopless network is

$$\binom{N}{2} = \frac{N}{2}(N - 1).$$

The fraction,  $\rho$  of these links that are actually present in the network is the density of the network [114]. That is

$$\rho = \frac{m}{\binom{N}{2}} = \frac{2m}{N(N-1)} = \frac{c}{N-1} \quad \text{with} \quad 0 \leq \rho \leq 1.$$

For large network,  $\rho$  is approximated as  $\rho = \frac{c}{N}$ . The network is said to be sparse

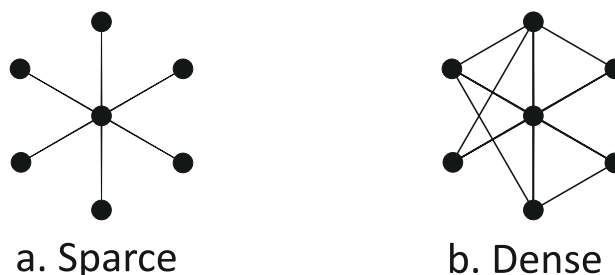


Figure 1.9: *Examples of sparse and dense networks*

if  $\rho \rightarrow \text{constant}$  as  $N \rightarrow \infty$  and dense if  $\rho \rightarrow 0$  as  $N \rightarrow \infty$ . The internet and the World Wide Web are examples of sparse networks [114]. Diverse mixing patterns support diverse epidemic dynamics, invariably influencing the effectiveness of control measure such as contact tracing, which attempts to detect and separate nodes with infectious contact [80]

### The Degree Distribution

The degree of a vertex is the number of edges (neighbours) attached to it [107]. It naturally captures the heterogeneity in individual's tendency to become infected as well as cause further infection by specifying the number of individuals connected to each individual in the population. For, example, the degree of vertices a, b and d in figure (1.8)(i) are respectively 2, 4 and 1.

For a network of  $N$  nodes, we write  $N_k$  for the number of nodes of degree  $k$ , and define  $P_k = \frac{N_k}{N}$  as the fraction of nodes in the network that have degree  $k$ . The network in figure (1.8(i)) has ten nodes, i.e.  $N = 10$  out of which 1 (i.e. only node  $j$ ) has degree 0 (i.e. not connected to any node), 2 (i.e. nodes  $d$  and  $f$ ) have degree 1, 4 (i.e. nodes  $a$ ,  $c$ ,  $i$  and  $h$ ) have degree 2, 2 (i.e. nodes  $e$  and  $f$ ) has degree 3, and 1 (i.e. only node  $b$ ) has degree 4, if chosen at random.

The values  $P_0 = \frac{1}{10}, P_1 = \frac{2}{10}, P_2 = \frac{4}{10}, P_3 = \frac{2}{10}$  and  $P_4 = \frac{1}{10}$  are the respective fraction or probability of a node in the network having degree 0, 1, 2, 3 and 4.

The quantities

$$P_k = \left\{ \frac{1}{10}, \frac{2}{10}, \frac{4}{10}, \frac{2}{10}, \frac{1}{10}, 0, 0, \dots \right\}$$

represent the degree distribution of the network.

Intuitively, the more neighbours an individual has, the more likely the individual is to cause more infection in the population, and also the higher the number of connection (edges) an individual has, more likely that they are connected to an already infected neighbour. All these depends on whether the network is directed or not.

**Note:**  $\sum_{k=0}^{\infty} P_k = 1$ .

### Assortativity

Assortative mixing is a bias of connection between network nodes with identical features. In social networks, for example, individuals usually prefer to associate with others of the same age, nationality, race, educational level, religion or language as themselves. The friendship paradox is a situation where most people have fewer friends than their friends have, on average. The tendency of higher-degree nodes connecting to other higher-degree nodes, that is assortative mixing by degree is of special interest to epidemiologists owing to its ability to determine direction of disease spread because many diseases are known to have different prevalence in different population groups. The rare case is the disassortative mixing where nodes attach preferentially to dissimilar nodes which is the case with partnerships between individuals of opposite sex. Part (a) of figure (1.10) is a network that is assortative by degree displaying the typical dense core of high-degree vertices surrounded by a periphery of lower degree ones. Part (b) is a disassortative network, displaying the star-like structure typical of this case [114]. These structural properties can help understand the spread of diseases or cure in the population. Diseases targeting high degree individuals in the assortative mixing are likely to spread to other high-degree nodes and the removal of a section of the network's node for example, may correspond to curing or quarantining individuals.

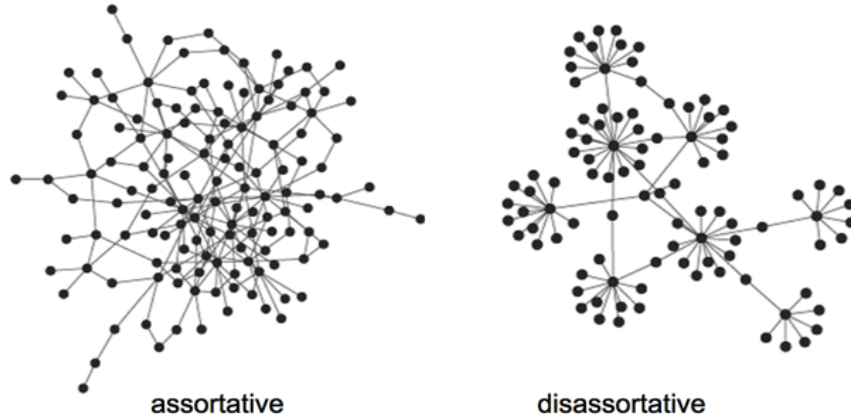


Figure 1.10: *Examples of assortative and disassortative networks (Adapted from Hao et al. (2011) PLOS ONE, DOI:10.1371/journal.pone.0028322)*

### Clustering Coefficient

Clustering coefficient ( $cc$ ) in the context of social network is an measure of the extent to which one's friends are also friends of each other. Study[176] indicates that in most real world networks, in particular social networks, nodes tend to form firmly bonded groups characterised by a relatively high density of clusters; this possibility have the tendency of being greater than the average probability of a connection randomly established between two nodes. In other word, Clustering coefficient is a measure that determine the frequency at which two neighbours of a node are also neighbours of each other.

Mathematically, the clustering coefficient ( $cc$ ),  $\phi$  is typically defined as the number of triangles divided by the number of triples (closed or unclosed) [100, 114, 126]. The powers of the adjacency matrix can be used to calculate this quantity:

$$\phi = \frac{\text{trace}(G^3)}{||G^2|| - \text{trace}(G^2)}, \quad \phi \in [0, 1].$$

One simple alternative way to calculate the  $cc$  of a network is to start off by talking about the  $cc(b)$  for example, of node  $b$  in the simple graph of figure (1.11). Let us define  $k_b$  as the degree of node  $b$ , and  $N_b$  the number of links between neighbours of  $b$ . Then,  $cc(b)$  is given as

$$cc(b) = \frac{2N_b}{k_b(k_b - 1)}.$$

The clustering coefficient  $\phi = cc(G)$  of the graph is the average of  $cc(b)$ ,  $b \in G$ . In the case of figure (1.11), node  $b$  is connected to nodes  $a$ ,  $c$  and  $d$  so the degree

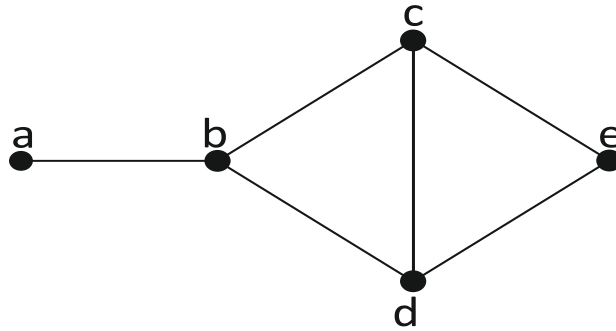


Figure 1.11: *A simple network to explain the concept of clustering coefficient*

$k_b = 3$ . Of these three neighbours of  $b$ , only nodes  $c$  and  $d$  are connected so  $N_b = 1$ , therefore,

$$cc(b) = \frac{2 \times 1}{3 \times 2} = \frac{1}{3}$$

And of course because  $cc(b)$  is a fraction of the possible interconnection between the neighbours of  $b$ ,  $0 \leq cc(b) \leq 1$ .

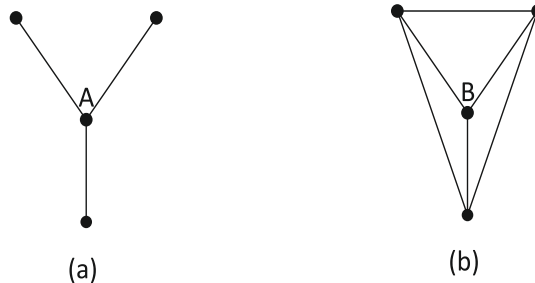


Figure 1.12: *Example of a node respectively forming a Star and Clique in a network*

If  $cc(b) = 0$ , then node  $b$  forms a star graph within the network as shown by node **A** in figure (1.12) where none of its neighbours are connected and if  $cc(b) = 1$ , node  $b$  forms a clique in the network as shown by node **B** in figure (1.12) where all its neighbours are connected. Clustering coefficient of a network is a relevant parameter that influences the speed of the spread of infection in the network.

### The Shortest Path

The **shortest path length**,  $d_{ij}$ , between two distinct nodes  $i$  and  $j$  specifies the distance between two nodes in terms of the minimum number of edges between them [114]. This idea in a network can be used to access how central an individual is in

the network. Intuitively, central nodes are more likely to be infected very quickly at the beginning of an epidemic, and also rapidly cause onward transmissions, and are hence essential targets when applying control measures to tackle infection in a population.

### Network Component

In most real-world undirected networks, there is a large component (giant component) that fills most of the network while the rest of the network is divided into a group of small components disconnected from the rest. That is, a network is said to have a giant component if a single component contains majority of the nodes in the network. Network representations of epidemic system will normally be useful

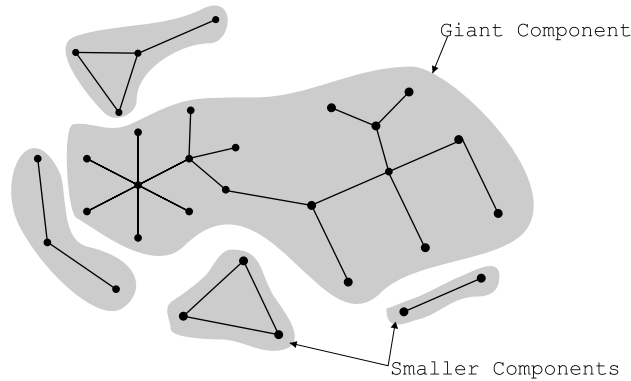


Figure 1.13: *Components of network*

if most of the network is connected together. If the network is sparsely connected as to be made of small components, then there is the possibility of a contagious infection being unable to spread to all the components parts, having been constrained to stay in the component of origin. In a strongly connected components, an infectious disease has the potential to reach any individual in the population.

## 1.6 Types of Networks

Infectious diseases motivate a significant proportion of network evaluation analysis [38, 82]. Mathematically, model networks can be configured following specific rules. For example, a random graph (i.e. network) is a model network in which some specific set of parameters take fixed values [113]. An example of a random graph is a network with a fixed number of vertices and edges [46]. That is, say  $n$  vertices and place  $m$  edges among them at random. One of the Erdos-Renyi graph, the

$G(n, p)$  model, is constructed by connecting nodes randomly, putting links in the network with probability  $p$  independently from every other link. Equivalently, all networks with  $n$  nodes and  $m$  links have equal probability of  $p^m(1-p)^{\binom{n}{2}-m}$ .  $p$  is assumed a weighting function that defines networks with varying structure as it increases from 0 to 1. Specifically,  $p = 0.5$  is the situation where all  $2^{\binom{n}{2}}$  networks on  $n$  nodes are constructed with equal probability.

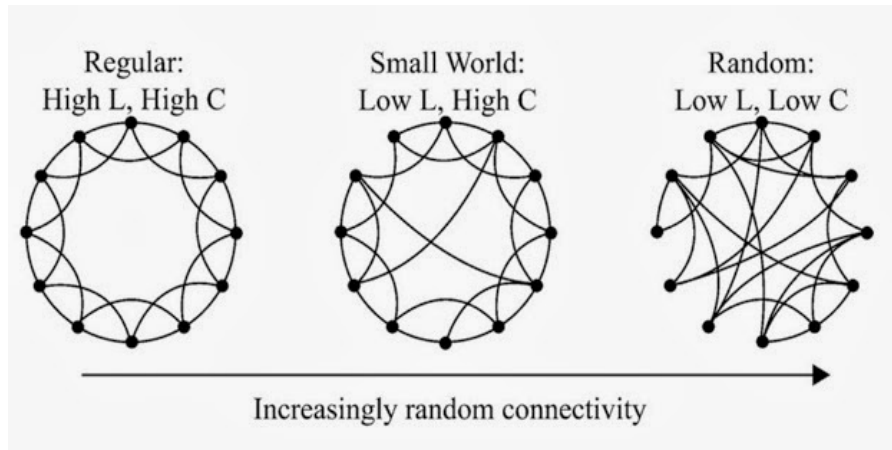


Figure 1.14: *Examples of random networks with increasing randomness from the left to the right. First on the left is a regular graph with low path length,  $L = d_{ij}$  and low clustering coefficient,  $C = \phi$ . The middle graph is an example of small world with low path length but high clustering coefficient. The graph to the right is another graph with both low path length and low clustering coefficient. Image copied from the internet-[http://images.slideplayer.com/24/7334623/slides/slides 8.jpg](http://images.slideplayer.com/24/7334623/slides/slides%208.jpg)*

### 1.6.1 Small world

The **small-world** network of contacts which typifies many biological settings and human social networks is known for being able to have a high level of clustering (i.e. significant value  $\phi$ ), while having relatively short path lengths (i.e. low integer value  $d_{ij}$ ). Exemplary biological insight into the “small-world” effect is provided by the Black Death that spread quickly through Europe which could be worse now as modern age transportation system mean epidemics can spread across continents in hours [34, 73, 99]. Epidemic spreading through small-world networks is usually rapid compared with that on sparse networks and is unlikely to be curbed in a small area of a population [114]. The mixing behaviour of a population can be regulated as a necessary mean of checking the spread of contagious disease. This measure is exemplified in the August 2015 flights cancellation from and to Liberia and Sierra Leone by British Airways and some other airline operators to prevent importation of the deadly Ebola virus into Europe.



### 1.6.2 Scale Free

The degree distribution of some highly structured networks such as the internet can be expressed as a function of  $k$ , the node degree. For example, studies [79, 122, 123, 178] have indicated, among other details, that the probability of a node in an internet network to be connected to  $k$  nodes follows a distribution  $P_k \sim k^{-\alpha}$ ,  $2 \leq \alpha \leq 3$  [122]. That is the distribution varies as a power of  $k$ . This distribution is referred to as power-law and networks with such distribution are called scale-free (SF) networks. In such a network most of the nodes have low degree but there are few nodes with substantially higher degree.

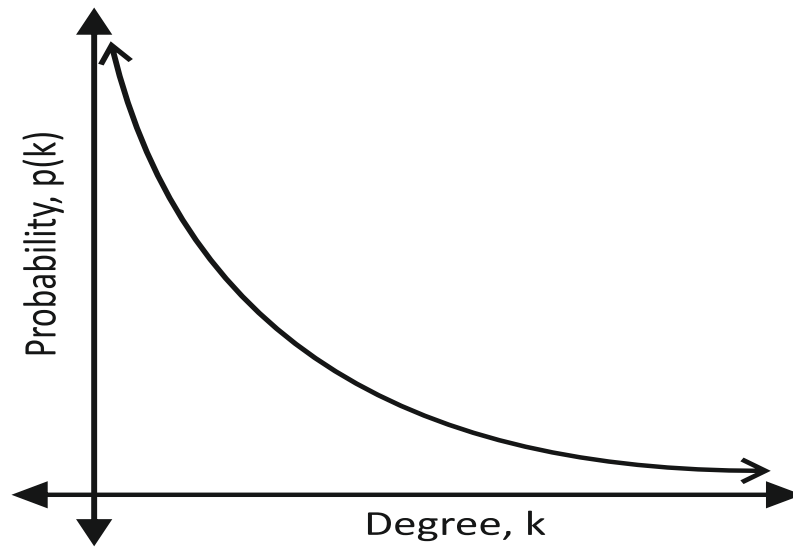


Figure 1.15: *The degree distribution of a typical Scale free network.*

These few ‘very connected’ nodes, hubs of connectivity influence the way the network behaves. SF networks has been used to describe among many behaviours, such as the spread of computer virus [87, 115] and the spread of contagious infection in a population [14, 116]. The existence of nodes with a very large number of connections is of major interest in the modelling of epidemic dynamics of networks[88]. These nodes are mostly targeted for vaccination in the population to control the spread of infection.

## 1.7 Network-based Modeling

There are several approaches to representing epidemic dynamics on networks. These include Degree-based [72, 74], Probability generating function [14, 15, 122, 123], edge-based [102, 103] and pair approximations [18, 40, 49] approaches, all differing in the choice of variables at which they are formulated [49, 85, 153], and whether averages are taken at the population level or a probabilistic approach is adopted where either infection starts at a node or the entire space of the possible system states is investigated.

### 1.7.1 Edge-based model

The edge-based compartmental modeling approach has at its root the recognition of the effect(s) of social heterogeneity and partnership between individuals in a population on the dynamics of infectious diseases. Miller, J.C et al [102] presented an edge-based compartmental modeling, motivating this approach using the standard mass action *SIR* epidemic model (1.14) of Kermack and McKendrick [78] which assumed individual's equal contact rate and short-lived partnership between individuals.

$$\begin{aligned}\dot{S}(t) &= -\beta IS, \\ \dot{I}(t) &= \beta IS - \gamma I, \\ \dot{R}(t) &= \gamma I.\end{aligned}\tag{1.14}$$

where  $S(t)$ ,  $I(t)$  and  $R(t)$  are the respective proportion of the population in susceptible, infectious and removed states at time  $t$ . He focused on evaluating these quantities using an alternative method by considering the probability that a randomly chosen node  $u$  is susceptible, infectious or recovered because if  $u$  is chosen randomly, then the probability that  $u$  is susceptible equals the proportion susceptible, and similarly for infection and removed [102]. The probability that a node  $u$  is susceptible depends on how many partner  $u$  has, the rate its partners change and that a random partner is likely infected at any given time.

He described the process of evaluating the probability that an individual  $u$  is susceptible as the probability that no partner of  $u$  has ever transmitted infection to  $u$  by applying system (1.14) to populations of different structure where partners were assigned to individual  $u$  according to a specific rule. He considered this for example, on a configuration model networks with  $N$  nodes and assigned each node  $u$  its degree  $k_u$  with probability  $P(k_u)$  and then give it  $k_u$  stubs (half-edge). Once all the nodes are allocated stubs, these stubs are then randomly paired into edges, representing partnership.

The probability that a randomly selected node  $u$  has degree  $k$  is  $P(k)$ , but in contrast, the probability a stub of  $u$  connects to some stubs  $v$  is proportional to  $k_v$ , so the probability a randomly chosen neighbour of  $u$  has degree  $k$  is

$$P_n(k) = \frac{kP(k)}{\langle K \rangle}$$

where  $\langle K \rangle$  is the average network degree.

Defining  $\theta(t)$  as the probability that a randomly chosen partner has not transmitted to  $u$ , where for large configuration network, neighbours of  $u$  are independent and so given its degree,  $u$  is susceptible at time  $t$  with probability

$$S(k, \theta(t)) = \theta(t)^k.$$

Thus

$$S(t) = \sum_k P(k) s(k, \theta(t)) = \psi(\theta(t))$$

where

$$\psi(x) = \sum_k P(k) x^k$$

is the probability generating function of the degree distribution [96].

This edge-based modeling approach allows more realistic effects and has helped to study, among many important variables, the spread of infection in a population for which some individual have different propensity to form partnerships (see [102, 103] for more detail).

### 1.7.2 Degree-based model

If networks are to be used for epidemiological intentions, then connections should only be assumed if they represent associations capable of allowing the transmission of infection [72]. However in many situations, it is not obvious how to illustrate such an interaction; how much contact is it required to have with somebody with influenza, say, prior to a significant risk? The problem is likely to be most severe where link representation should be more direct, such as for STDs, there are problems [72].

It is realised that because different infections are passed through different routes a mixing network is certainly disease specific [72, 114]. Consequently, a mixing network considered in the situation of HIV transmission would be different from one considered to study influenza; in such a situation, one might imagine the networks to be nested, that is, with the connections applicable for HIV spread to be a subset

of the ones important for influenza [74].

Several forms of computer-generated mixing networks have been studied in relationship to spread of infections. One of the most standard mixing network measure is of the degree-distribution. Networks are constructed dynamically by introducing new individual to a network one by one with a connection mechanism that imitates the natural evolution of social network[72]. An example of network constructed this way is the scale-free network where each new individual added to the population connects preferentially to people that already have a large number of connections, which amounts to individual desiring to be friends with the most popular individuals. This give rise to the number of connections per individual taking a power law distribution,  $P(k) \sim k^{-\gamma}$ , where an exponent  $\gamma$  ranges between 2 and 3. It is often the case that many individuals in the population have a small number of neighbours, while few have a lot more connections [13]. This extreme heterogeneity which is observable in real networks such as the World Wide Web and the internet, revealed that having too many contacts puts the individual at greater risk of infection and, once infected, can transmit the disease to many others [13, 122].

Pastor et al [ 122], among others, considered an *SIS* epidemic dynamic on complex networks of varying connectivity to determine and compare the behaviour of some fundamental epidemic variables. He specifically considered the Barabasi-Albert scale-free graph [13], constructing it by starting from a small number  $m_0$  of disconnected of nodes and then every time step a new vertex is added, with  $m$  links that are connected to an old node  $i$  with probability

$$\prod(k_i) = \frac{k_i}{\sum_j k_j},$$

where  $k_i$  is the connectivity of the *ith* node. After iterating this scheme a sufficient number of time, a network composed of  $N$  nodes was obtained with connectivity distribution  $P(k) \sim k^{-3}$  and average connectivity  $\langle k \rangle = 2m$ . This study was conducted for  $m_0 = 5$  and  $m_0 = 3$

He also considered the model on a generalised scale-free network with a normalised connectivity distribution

$$P(k) = (1 + \gamma)m^{1-\gamma}k^{-2-\gamma},$$

approximating the connectivity  $k$  as a continuous variable and assuming  $m$  the minimum connectivity of any node. In both cases, numerical analysis confirms the analytical results, pointing out an absence of any epidemic threshold or critical point for  $\gamma = 1$  but a non-trivial threshold,  $\frac{\gamma-1}{m\gamma}$  is observed for  $1 < \gamma < 2$  in the

second case (see [122] for more detail).

### 1.7.3 Pairwise model

In the pairwise method, the number of different types of pairs are included as variables rather than approximated in terms of individuals. It is vital to understand that the biological assumption that individuals interact in pairs is not just a matter of mathematics. The effects of neighbours on the infectious status of an individual is assumed to depend on pairs and the fate of a pair of individuals depends on triples [135]. The inclusion of the number of connected individuals as basic variables captures the mutual relationship between neighbouring individuals that emerge in the system.

Keeling, M.J [72] used an *SIR* epidemic model to investigate and emphasize the importance of individuals and spacial correlation on the capacity of infection to invade and persist in a population. Undirected network of different number of nodes,  $N$  with varying number of connections per nodes and clustering coefficient,  $\psi$  were formed by placing nodes randomly in two dimensions and weighting the probability of a connection between nodes by the distance. The dynamics of individual, pairs and triples were considered by defining the numbers of singles, pairs and triples of each type in the network. Number of pairs =  $\|G\| = nN$  and number of triples =  $\|G^2\| - \text{trace}(G^2)$ , where  $\|G\| = \sum_{i,j} G_{ij}$  is the sum of all element in the transmission matrix  $G$  and  $n$  is the average number of neighbours per node. A measure,  $\phi$  of how interconnected the local community is, is obtained as

$$\phi = \frac{\text{number of triangles}}{\text{number of triples}} = \frac{\text{trace}(G^3)}{\|G^2\| - \text{trace}(G^2)}.$$

Such correlation model showed the effects of the average numbers of neighbours per node and the interconnectedness of a contact network on the dynamics of infectious diseases, particularly sexually transmitted infections where most of the partners come from a small communal group (see [72] for more detail).

#### Pair Approximation

Pair approximation models present a simple way of extending the mean-field models, say for example of section (1.2.3), to accommodate observable connectivity of individuals in a population. Pair approximation has found applications in many areas of ecology, disease biology and evolutionary biology [40, 134, 135].

We explain the pair approximation process by considering *SIS* epidemic dynamics. In the mean field, the total rate of new infection is approximated as  $\beta \frac{S}{N} I$  (see section (1.2.1)) while in this model it is written as  $\tau[SI]$ ,  $\tau$  being the constant transmission rate across an *S-I* link and **SI** is interpreted as the expected number of partnership between susceptible and infected individuals [82, 84].

With infection and recovery both Poisson processes, the pairwise model is written as:

$$\begin{aligned} \frac{d}{dt}[S] &= -\tau[SI] + \gamma[I], \\ \frac{d}{dt}[I] &= \tau[SI] - \gamma[I]. \end{aligned} \tag{1.15}$$

where  $\gamma$  is the constant rate of recovery of an infected individual.

Equation (1.15) gives an exact infection dynamic of the model provided we know the exact number of *S-I* pairs in the network. One of the options open to us to close this system for mathematical solution is to approximate the term  $\tau[SI]$  as  $\tau[I] \frac{[S]}{N}$ ,  $N$  being the population size, which of course takes us back to the assumption of well-mixed population thereby leading us to the basic *SIS* model of (1.6). Note that  $\tau \times n = \beta$  where  $n$  is the number of links in the network [81]. The other option is to write an expression for the rate at which the number of the pair **[SI]** in the network evolve since it will not be difficult to identify the number of such pairs in a network given an initially infected node.

This number **SI** of *S-I* pairs changes over time by either external or internal infection or recovery of infected individual [49, 74, 84]. The rate at which this number of pairs changes is given as

$$\frac{d}{dt}[SI] = \tau[SSI] + \gamma[II] - \tau[ISI] - \tau[SI] - \gamma[SI]. \tag{1.16}$$

Systems (1.15) and (1.16) combine to give

$$\begin{aligned} \frac{d}{dt}[S] &= -\tau[SI] + \gamma[I], \\ \frac{d}{dt}[I] &= \tau[SI] - \gamma[I], \\ \frac{d}{dt}[SI] &= \tau[SSI] + \gamma[II] - \tau[ISI] - \tau[SI] - \gamma[SI]. \end{aligned} \tag{1.17}$$

where of course the initial numbers of possible pairs, *SI*, the triples, *SSI* and higher moments can be obtained from available network of contacts, given the initial condition of the system.

From (1.17), the system is not closed as the model expresses the number of pairs in the network in terms of the number of triples. Knowledge of the rate of change of higher order moments is required and this leads to a system with a large number of equations for large networks.

As the name pair approximation suggest, higher orders are ignored and the system is closed at the level of pairs by applying a moment closure. For example, a commonly used moment closure is

$$[ABC] \approx \frac{n-1}{n} \frac{[AB][BC]}{[B]}. \quad (1.18)$$

which reduces the system to a size that is mathematically solvable.  $A$ ,  $B$  and  $C$  represent state  $S$  or  $I$  in our case and  $n$  the degree of the middle susceptible node  $B$  in the triple,  $ABC$ .

This approach instead of modelling a complete network of contacts explicitly makes use of the various types of connected pairs that appear within a population. It has been used to approximate important epidemic quantifiers such as the final size of some childhood infection[70, 161], and the spread and control of some STDs in population with different connectivity [42, 49].

### Individual-level moment closure

While stochastic models allow for the inclusion of contact heterogeneity, it has been more difficult to construct network-based deterministic models. One method is to build up the entire epidemic system from the perspective of the infectious status of an individual to the population level leading to a set of differential equations [153, 154, 155]. We explain the nodal infection process by considering  $SIR$  epidemic dynamics on an arbitrary finite sized network which has an associated adjacency matrix  $G$ .

We assume that transmission and recovery from infection are Poisson processes where all rates are equal. We describe the infection status of an individual at any given time in the network by the components of the vectors  $I$  and  $S$  where  $I_i = 1$  if individual  $i$  is infected and  $I_i = 0$  otherwise,  $S_i = 1$  if individual  $i$  is susceptible and  $S_i = 0$  otherwise,  $i = \{1, 2, \dots, N\}$ . We also define a general contact network  $T = \tau G$  where  $T_{ij}$  represents the rate parameter of the Poisson process by which an infected individual  $j$  infects a susceptible individual  $i$ . Inherent in  $T$  thus defined are the individuals interacting rates and the probability that infection is transmitted from an infected individual to a susceptible individual when they come in contact.

Let  $\lambda_i$  be the infectious pressure on individual  $i$ . Similarly, let  $\mu_i$  be the recovery pressure on an infected individual  $i$ . The approximated underlying stochastic model [153] is given as

$$\begin{aligned}\lambda_i &= \sum_{j=1}^N T_{ij} S_i I_j, \\ \mu_i &= \gamma_i I_i.\end{aligned}\tag{1.19}$$

The total infection process rate  $\lambda_i$  on a susceptible individual  $i$  is by virtue of its links to neighbouring infected individual(s). Given an initial state of the system, direct simulation can be used to obtain statistically accurate realisations for any  $T$ . This is, by analogy, the Gillespie algorithm we explained in section (1.4) for the mean-field *SIR* epidemic dynamics.

Hierarchy of moment equations can be written for the time evolution of the probabilities of these infection states. Sharkey et al [153] presented a deterministic representation of the Markovian *SIR* epidemics on networks. For any pair of nodes  $i$  and  $j$  and any transmission matrix  $T$  for a contact network of finite size,  $N$ , the following differential equations are satisfied:

$$\begin{aligned}\langle \dot{S}_i \rangle &= - \sum_{j=1}^N T_{ij} \langle S_i I_j \rangle, \\ \langle \dot{I}_i \rangle &= \sum_{j=1}^N T_{ij} \langle S_i I_j \rangle - \gamma_i \langle I_i \rangle, \\ i, j &\in 1, 2, \dots, N.\end{aligned}\tag{1.20}$$

where notations  $\langle A_i \rangle$  represents the time dependent probability of individual  $i$  having infection status  $A$ ,  $\langle A_i B_j \rangle$  represents the time dependent probability of individual  $i$  being in state  $A$  and individual  $j$  being in state  $B$  in the pair  $A_i B_j$ . The dot notation here is the derivative of quantities with respect to time.

System (1.20) is not closed as the probabilities of for example, singles are expressed in terms of pairs. Using a moment closure relation of first order

$$\langle A_i B_j \rangle \approx \langle A_i \rangle \langle B_j \rangle$$

closes the system at the level of individual for mathematical feasibility, hence the name **individual-based model**. This closure expresses the probability of pairs as statistically independent of the probabilities of constituent individuals.

Including the rate of change of probabilities of pairs, equation (1.20) is extended



to

$$\begin{aligned}
\langle \dot{S}_i \rangle &= - \sum_{j=1}^N T_{ij} \langle S_i I_j \rangle, \\
\langle \dot{I}_i \rangle &= \sum_{j=1}^N T_{ij} \langle S_i I_j \rangle - \gamma_i \langle I_i \rangle, \\
\langle \dot{S}_i I_j \rangle &= \sum_{k=1, k \neq i}^N T_{jk} \langle S_i S_j I_k \rangle - \sum_{k=1, k \neq j}^N T_{ki} \langle I_k S_i I_j \rangle - (T_{ij} + \gamma_j) \langle S_i I_j \rangle, \\
\langle \dot{S}_i S_j \rangle &= - \sum_{k=1, k \neq j}^N T_{ik} \langle I_k S_i S_j \rangle - \sum_{k=1, k \neq i}^N T_{jk} \langle S_i S_j I_k \rangle.
\end{aligned} \tag{1.21}$$

with similar definition above for notation  $\langle A_i B_j C_k \rangle$ . The average number of infectious is generally given by

$$[I] = \sum_{i=1}^N \langle I_i \rangle.$$

System (1.21) is also not closed as the probabilities of pairs are expressed in that of triples. The probabilities of the triples in this case be can expressed in terms of the probabilities of pairs, using a moment closure relation of the form

$$\langle A_i B_j C_k \rangle \approx \frac{\langle A_i B_j \rangle \langle B_j C_k \rangle}{\langle B_j \rangle}. \tag{1.22}$$

Considering an open triple in figure (1.16) where nodes  $i$ ,  $j$  and  $k$  are in different

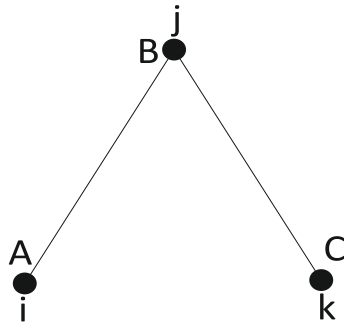


Figure 1.16: *An open triple*

states, A, B and C respectively, equation (1.22) basically implies that, conditioned on node  $j$  being in state B, the states of individuals  $i$  and  $k$  are assumed statistically

independent, that is;

$$\begin{aligned}\langle A_i|B_j\rangle \times \langle C_k|B_j\rangle &\approx \langle A_i C_k|B_j\rangle \\ \frac{\langle A_i B_j\rangle \times \langle C_k B_j\rangle}{\langle B_j\rangle^2} &\approx \frac{\langle A_i B_j C_k\rangle}{\langle B_j\rangle} \\ \frac{\langle A_i B_j\rangle \times \langle C_k B_j\rangle}{\langle B_j\rangle} &\approx \langle A_i B_j C_k\rangle,\end{aligned}$$

which is the same as (1.22).

Closing system (1.21) at the level of pair using equation (1.22) gives

$$\begin{aligned}\langle \dot{X}_i\rangle &= -\sum_{j=1}^N T_{ij}\langle X_i Y_j\rangle, \\ \langle \dot{Y}_i\rangle &= \sum_{j=1}^N T_{ij}\langle X_i Y_j\rangle - \gamma_i\langle Y_i\rangle, \\ \langle X_i \dot{Y}_j\rangle &= \sum_{k=1, k\neq i}^N T_{jk} \frac{\langle X_i X_j\rangle \langle X_j Y_k\rangle}{\langle X_j\rangle} - \sum_{k=1, k\neq j}^N T_{ik} \frac{\langle Y_k X_i\rangle \langle X_i Y_j\rangle}{\langle X_i\rangle} \\ &\quad - (T_{ij} + \gamma_j)\langle X_i Y_j\rangle, \\ \langle X_i \dot{X}_j\rangle &= -\sum_{k=1, k\neq j}^N T_{ik} \frac{\langle Y_k X_i\rangle \langle X_i X_j\rangle}{\langle X_i\rangle} - \sum_{k=1, k\neq i}^N T_{jk} \frac{\langle X_i X_j\rangle \langle X_j Y_k\rangle}{\langle X_j\rangle},\end{aligned}\tag{1.23}$$

hence the name **pair-based model** with  $X$  and  $Y$  emphasizing respective approximation for susceptible and infectious. The beauty of this approach is that it gives a unifying connection between the deterministic model and its stochastic counterpart [154, 155].

### Example of pair-based model on simple networks

We shall consider and investigate the validity of the first and second order moment closure on simple networks for the Markovian *SIR* infection dynamics [155]. We assume the same transmission rate  $\tau$  across all links and the same recovery rate  $\gamma$  for an infected individual.

For the open triple in figure (1.17), the transmission matrix via the adjacency matrix is:

$$T_{ij} = \begin{bmatrix} 0 & \tau & 0 \\ \tau & 0 & \tau \\ 0 & \tau & 0 \end{bmatrix}$$

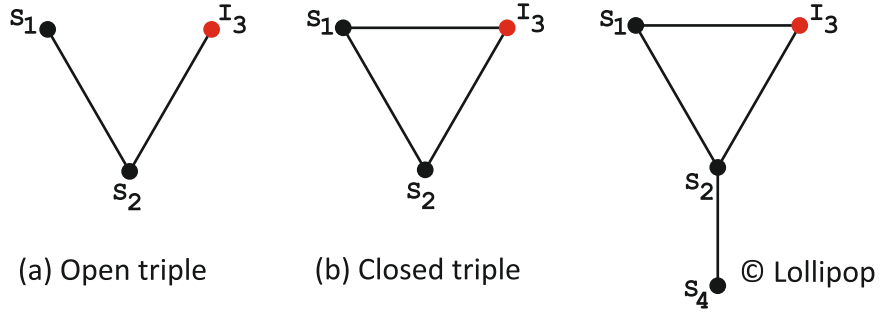


Figure 1.17: *Simple networks*

From (1.21) we have the following equations for the single nodes

$$\begin{aligned}
\langle \dot{S}_1 \rangle &= -\tau \langle S_1 I_2 \rangle, \\
\langle \dot{S}_2 \rangle &= -\tau \langle I_1 S_2 \rangle - \tau \langle S_2 I_3 \rangle, \\
\langle \dot{S}_3 \rangle &= -\tau \langle I_2 S_3 \rangle, \\
\langle \dot{I}_1 \rangle &= \tau \langle S_1 I_2 \rangle - \gamma \langle I_1 \rangle, \\
\langle \dot{I}_2 \rangle &= \tau \langle I_1 S_2 \rangle + \tau \langle S_2 I_3 \rangle - \gamma \langle I_2 \rangle, \\
\langle \dot{I}_3 \rangle &= \tau \langle I_2 S_3 \rangle - \gamma \langle I_3 \rangle.
\end{aligned} \tag{1.24}$$

And for the pairs:

$$\begin{aligned}
\langle \dot{S}_1 I_2 \rangle &= \tau \langle S_1 S_2 I_3 \rangle - (\tau + \gamma) \langle S_1 I_2 \rangle, \\
\langle \dot{I}_1 S_2 \rangle &= -\tau \langle I_1 S_2 I_3 \rangle - (\tau + \gamma) \langle I_1 S_2 \rangle, \\
\langle \dot{S}_2 I_3 \rangle &= -\tau \langle I_1 S_2 I_3 \rangle - (\tau + \gamma) \langle S_2 I_3 \rangle, \\
\langle \dot{I}_2 S_3 \rangle &= \tau \langle I_1 S_2 S_3 \rangle - (\tau + \gamma) \langle I_2 S_3 \rangle, \\
\langle \dot{S}_1 \dot{S}_2 \rangle &= -\tau \langle S_1 S_2 I_3 \rangle, \\
\langle \dot{S}_2 \dot{S}_3 \rangle &= -\tau \langle I_1 S_2 S_3 \rangle.
\end{aligned} \tag{1.25}$$

In similar manner, we can write equations for the triples because the graph in this case has three nodes:

$$\begin{aligned}
\langle \dot{S}_1 \dot{S}_2 I_3 \rangle &= -(\tau + \gamma) \langle S_1 S_2 I_3 \rangle, \\
\langle \dot{I}_1 \dot{S}_2 I_3 \rangle &= -2(\tau + \gamma) \langle I_1 S_2 I_3 \rangle, \\
\langle \dot{I}_1 \dot{S}_2 \dot{S}_3 \rangle &= -(\tau + \gamma) \langle I_1 S_2 S_3 \rangle.
\end{aligned} \tag{1.26}$$

For the individual based model, the set of Odes for the system is

$$\begin{aligned}
\langle \dot{S}_1 \rangle &= -\tau \langle S_1 \rangle \langle I_2 \rangle, \\
\langle \dot{S}_2 \rangle &= -\tau \langle I_1 \rangle \langle S_2 \rangle - \tau \langle S_2 \rangle \langle I_3 \rangle, \\
\langle \dot{S}_3 \rangle &= -\tau \langle I_2 \rangle \langle S_3 \rangle, \\
\langle \dot{I}_1 \rangle &= \tau \langle S_1 \rangle \langle I_2 \rangle - \gamma \langle I_1 \rangle, \\
\langle \dot{I}_2 \rangle &= \tau \langle I_1 \rangle \langle S_2 \rangle + \tau \langle S_2 \rangle \langle I_3 \rangle - \gamma \langle I_2 \rangle, \\
\langle \dot{I}_3 \rangle &= \tau \langle I_2 \rangle \langle S_3 \rangle - \gamma \langle I_3 \rangle.
\end{aligned} \tag{1.27}$$

Assuming  $\tau = 1$  across both links, a removal rate  $\gamma = 0.5$  for an infected individual and initiating the system with node 3 being infected, the numerical solution of system (1.27) is compared with the solution of the entire system (1.24) to (1.26) in figure (1.20).

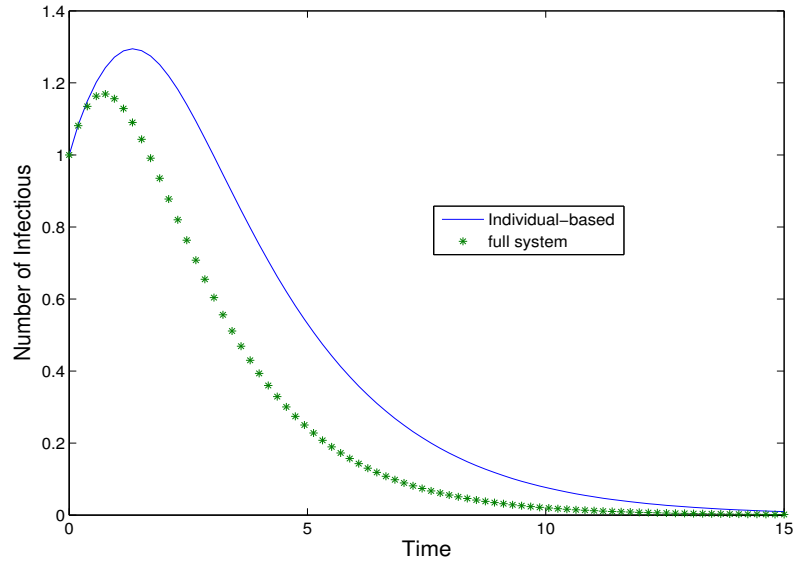


Figure 1.18: *The average infection time series of the individual-based SIR model compared with the full system for an open triple shows that the first order moment closure approximation is **not exact***

Although, model (1.20) which was closed with statistical independence between individuals to obtain equation (1.27) for the open triple is less computationally involved, it is significantly less accurate as depicted in figure (1.20).

Expressing a quantity such as  $\langle S_1 S_2 I_3 \rangle$  as

$$\langle S_1 S_2 I_3 \rangle \approx \frac{\langle S_1 S_2 \rangle \langle S_2 I_3 \rangle}{\langle S_2 \rangle}.$$

in the full system (1.24) to (1.26) reduces the number of equations to

$$\begin{aligned}
\langle \dot{S}_1 \rangle &= -\tau \langle S_1 I_2 \rangle, \\
\langle \dot{S}_2 \rangle &= -\tau \langle I_1 S_2 \rangle - \tau \langle S_2 I_3 \rangle, \\
\langle \dot{S}_3 \rangle &= -\tau \langle I_2 S_3 \rangle, \\
\langle \dot{I}_1 \rangle &= \tau \langle S_1 I_2 \rangle - \gamma \langle I_1 \rangle \\
\langle \dot{I}_2 \rangle &= \tau \langle I_1 S_2 \rangle + \tau \langle S_2 I_3 \rangle - \gamma \langle I_2 \rangle, \\
\langle \dot{I}_3 \rangle &= \tau \langle I_2 S_3 \rangle - \gamma \langle I_3 \rangle, \\
\langle \dot{S}_1 I_2 \rangle &= \tau \frac{\langle S_1 S_2 \rangle \langle S_2 I_3 \rangle}{\langle S_2 \rangle} - (\tau + \gamma) \langle S_1 I_2 \rangle, \\
\langle \dot{I}_1 S_2 \rangle &= -\tau \frac{\langle I_1 S_2 \rangle \langle S_2 I_3 \rangle}{\langle S_2 \rangle} - (\tau + \gamma) \langle I_1 S_2 \rangle, \\
\langle \dot{S}_2 I_3 \rangle &= -\tau \frac{\langle I_1 S_2 \rangle \langle S_2 I_3 \rangle}{\langle S_2 \rangle} - (\tau + \gamma) \langle S_2 I_3 \rangle, \\
\langle \dot{I}_2 S_3 \rangle &= \tau \frac{\langle I_1 S_2 \rangle \langle S_2 S_3 \rangle}{\langle S_2 \rangle} - (\tau + \gamma) \langle I_2 S_3 \rangle, \\
\langle \dot{S}_1 S_2 \rangle &= -\tau \frac{\langle S_1 S_2 \rangle \langle S_2 I_3 \rangle}{\langle S_2 \rangle}, \\
\langle \dot{S}_2 S_3 \rangle &= -\tau \frac{\langle I_1 S_2 \rangle \langle S_2 S_3 \rangle}{\langle S_2 \rangle}.
\end{aligned} \tag{1.28}$$

The numerical solution of the full system (i.e equations (1.24)-(1.26)) and the pair-based mode (1.28) are compared in figure (1.19).

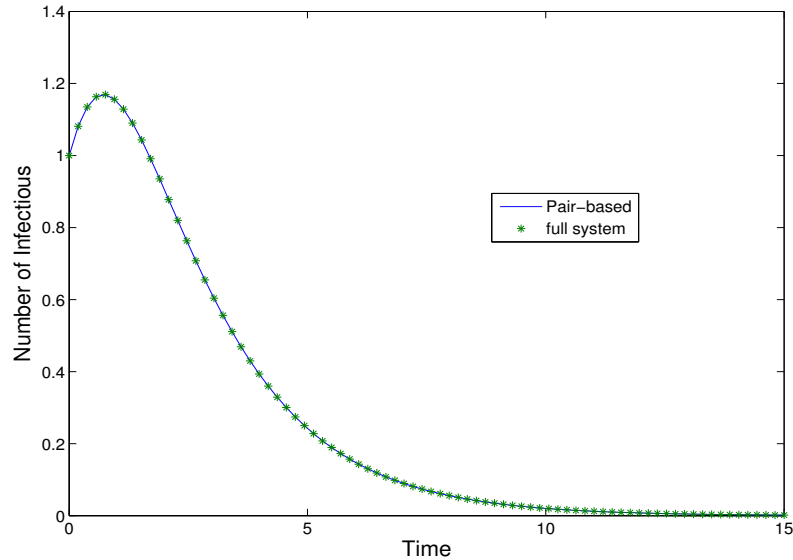


Figure 1.19: *The pair-based SIR model compared with the full system for an open triple shows the second order moment closure appears an exact approximation.*

We mention that system (1.21) can also be closed at the level of triples or higher terms but this will increase the number of equations and the system will become less practical for large networks.

### Exactness of the second order moment closure

From figure(1.19), the second order moment closure appears to be exact for the *SIR* model on open triple. For example if

$$\langle S_1 S_2 I_3 \rangle = \frac{\langle S_1 S_2 \rangle \langle S_2 I_3 \rangle}{S_2},$$

and we write  $\alpha(t) = \langle S_2 \rangle \langle S_1 S_2 I_3 \rangle - \langle S_1 S_2 \rangle \langle S_2 I_3 \rangle$ , then if the closure is exact,  $\alpha(t) = 0$  for all  $t \geq 0$ . In fact, this type of closure is true for any tree graph [155]. In the case we consider here, the argument presented in [155] works as follows.

The derivative of  $\alpha$  with respect to time yields:

$$\dot{\alpha}(t) = \langle \dot{S}_2 \rangle \langle S_1 S_2 I_3 \rangle + \langle S_2 \rangle \langle S_1 \dot{S}_2 I_3 \rangle - \langle \dot{S}_1 \dot{S}_2 \rangle \langle S_2 I_3 \rangle - \langle S_1 S_2 \rangle \langle \dot{S}_2 I_3 \rangle$$

Substituting the corresponding derivatives and quantities in from system (1.24) to (1.26) and cancelling all relevant terms gives:

$$\dot{\alpha}(t) = -(\tau + \gamma)\alpha(t)$$

which when solved directly gives:

$$\alpha(t) = \alpha(0)e^{-(\tau+\gamma)t}$$

Of course  $\alpha(0) = 0$  if the system is initiated in a pure state,  $S_1 S_2 I_3$ . So  $\alpha(t) = 0$  for all  $t \geq 0$ , hence the closure is exact.

The second order moment closure approximation is not exact for the *SIR* epidemic model on all kind of networks. There is always a connection between the topology of the network of contacts and the accuracy of closure. Earlier study [155] proved and established the exactness of the second order moment closure (i.e. the pair-based model) for an *SIR* infection dynamic on a finite undirected and static **tree** networks for constant transmission and recovery rates.

The presence of loops makes it difficult to match a low-dimensional approximate model with its corresponding stochastic model. For example, if we consider *SIR* epidemic dynamics for the closed triangle of figure (1.17)b, the corresponding

transmission matrix is

$$T_{ij} = \begin{bmatrix} 0 & \tau & \tau \\ \tau & 0 & \tau \\ \tau & \tau & 0 \end{bmatrix}$$

with the corresponding system of equations for the singles as:

$$\begin{aligned} \langle \dot{S}_1 \rangle &= -\tau \langle S_1 I_2 \rangle - \tau \langle S_1 I_3 \rangle, \\ \langle \dot{S}_2 \rangle &= -\tau \langle I_1 S_2 \rangle - \tau \langle S_2 I_3 \rangle, \\ \langle \dot{S}_3 \rangle &= -\tau \langle I_1 S_3 \rangle - \tau \langle I_2 S_3 \rangle \\ \langle \dot{I}_1 \rangle &= \tau \langle S_1 I_2 \rangle + \tau \langle S_1 I_3 \rangle - \gamma \langle I_1 \rangle, \\ \langle \dot{I}_2 \rangle &= \tau \langle I_1 S_2 \rangle + \tau \langle S_2 I_3 \rangle - \gamma \langle I_2 \rangle, \\ \langle \dot{I}_3 \rangle &= \tau \langle I_1 S_3 \rangle + \tau \langle I_2 S_3 \rangle - \gamma \langle I_3 \rangle. \end{aligned} \tag{1.29}$$

And for the pairs:

$$\begin{aligned} \langle \dot{S}_1 I_2 \rangle &= \tau \langle S_1 S_2 I_3 \rangle - (\tau + \gamma) \langle S_1 I_2 \rangle - \tau \langle S_1 I_2 I_3 \rangle, \\ \langle \dot{I}_1 S_2 \rangle &= \tau \langle S_1 S_2 I_3 \rangle - (\tau + \gamma) \langle I_1 S_2 \rangle - \tau \langle I_1 S_2 I_3 \rangle, \\ \langle \dot{S}_1 I_3 \rangle &= \tau \langle S_1 I_2 S_3 \rangle - (\tau + \gamma) \langle S_1 I_3 \rangle - \tau \langle S_1 I_2 I_3 \rangle, \\ \langle \dot{I}_2 S_3 \rangle &= \tau \langle I_1 S_2 S_3 \rangle - (\tau + \gamma) \langle I_2 S_3 \rangle - \tau \langle I_1 I_2 S_3 \rangle, \\ \langle \dot{S}_1 I_3 \rangle &= \tau \langle S_1 I_2 S_3 \rangle - (\tau + \gamma) \langle S_1 I_3 \rangle - \tau \langle S_1 I_2 I_3 \rangle, \\ \langle \dot{I}_1 S_3 \rangle &= \tau \langle S_1 I_2 S_3 \rangle - (\tau + \gamma) \langle I_1 S_3 \rangle - \tau \langle I_1 I_2 S_3 \rangle, \\ \langle \dot{S}_1 \dot{S}_2 \rangle &= -2\tau \langle S_1 S_2 I_3 \rangle, \\ \langle \dot{S}_2 \dot{S}_3 \rangle &= -2\tau \langle I_1 S_2 S_3 \rangle, \\ \langle \dot{S}_1 \dot{S}_3 \rangle &= -2\tau \langle S_1 I_2 S_3 \rangle. \end{aligned} \tag{1.30}$$

The equations for the triples are:

$$\begin{aligned} \langle \dot{S}_1 \dot{S}_2 I_3 \rangle &= -2(\tau + \gamma) \langle S_1 S_2 I_3 \rangle, \\ \langle \dot{I}_1 \dot{S}_2 S_3 \rangle &= -2(\tau + \gamma) \langle I_1 S_2 S_3 \rangle, \\ \langle \dot{S}_1 \dot{I}_2 S_3 \rangle &= -2(\tau + \gamma) \langle S_1 I_2 S_3 \rangle, \\ \langle \dot{I}_1 \dot{S}_2 I_3 \rangle &= -2(\tau + \gamma) \langle I_1 S_2 I_3 \rangle + \tau \langle S_1 S_2 I_3 \rangle + \tau \langle I_1 S_2 S_3 \rangle, \\ \langle \dot{S}_1 \dot{I}_2 I_3 \rangle &= -2(\tau + \gamma) \langle S_1 I_2 I_3 \rangle + \tau \langle S_1 S_2 I_3 \rangle + \tau \langle S_1 I_2 S_3 \rangle, \\ \langle \dot{I}_1 \dot{I}_2 S_3 \rangle &= -2(\tau + \gamma) \langle I_1 I_2 S_3 \rangle + \tau \langle I_1 S_2 S_3 \rangle + \tau \langle S_1 I_2 S_3 \rangle. \end{aligned} \tag{1.31}$$

The individual and pair approximation of system (1.29) to (1.31) are compared with the full system in figure (1.20) for the same parameter values of the open

triple. Results seems to show that none of these approximation is exact for this type of network.

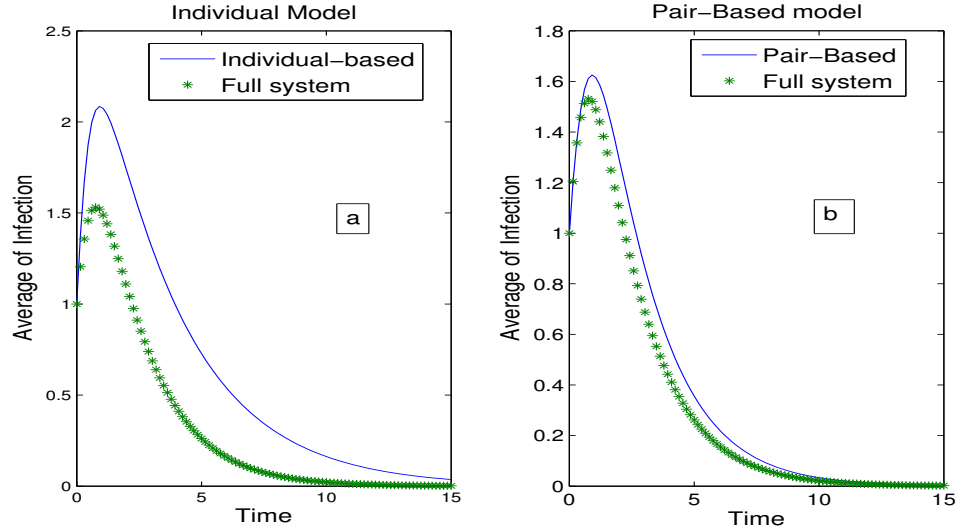


Figure 1.20: *Both the individual and pair approximation of **SIR** model compared with the full system for a closed triple. shows the second order moment closure appears an exact approximation*

Progress have been made to extend the loopless network results to networks with loop [55, 113]. Kiss et al [85] investigated the relationship between the structural property of network and the viability of the second order moment closure by considering the Markovian *SIR* epidemic dynamic for various networks with small cycles. The study revealed the types of closure that are feasible in the network. It was established that the closures where the loops are kept intact are exact. In other words, closure at the full system must be written in a way to preserve the loops. In figure 1.17(b) for example, exact closure can not be written for  $\langle S_1 S_2 I_3 \rangle$  in both the closed triangle the lollipop networks.

That is

$$\langle S_1 S_2 I_3 \rangle = \frac{\langle S_1 S_2 \rangle \langle S_2 I_3 \rangle}{\langle S_2 \rangle}$$

can not be exact but closure can be written for  $\langle S_1 S_2 I_3 S_4 \rangle$ , that is

$$\langle S_1 S_2 I_3 S_4 \rangle = \frac{\langle S_1 S_2 I_3 \rangle \langle S_2 S_4 \rangle}{\langle S_2 \rangle}$$

is exact. The preservation of the loop by  $S_1 S_2 I_3$  is evidence in the closure defined for  $S_1 S_2 I_3 S_4$ .



## 1.8 Summary

In this chapter, we have given a background to the research covered in this thesis. The underlying methodology of this thesis has been the pair approximation of the network-based epidemic models.

Earlier study [155] has established the exactness of the moment closure (1.22) for *SIR* on tree networks. In chapter two we develop an *SEIR* epidemic model and extend the above analysis of the pairwise moment closure (1.22) for *SEIR* epidemic models on tree network.

We mentioned earlier that there is a connection between the connectivity of networks and the accuracy of moment closure defined for epidemic models on networks. In fact, the presence of small cycles makes it difficult to establish a low-dimensional approximate model that will match the output of the corresponding stochastic model [85]. From the hindsight of this interdependency of the validity of moment closure and network topology, we consider and examine in chapter three, the deviation from being exact of the moment closure (1.22) for *SIR* epidemic model on **graphs with a single cycle**.

There are a lot of stochastic systems emerging in different areas such as chemical kinetics and wildlife management, which in the long run approach an absorbing state but nevertheless occur to be stationary over a sensible time scale. The time to absorption of some of these systems can be considerably large. The concept of a quasi-stationary distribution has been very helpful in modelling the behaviour of important quantifiers in this region of stationary equilibrium. Almost all the earlier study on the QSD of endemic infection is on the mean-field-especially *SIS* dynamics. In chapter four, we extend the idea of QSD to the model. We consider the Markovian *SIS* epidemic dynamic which is assumed suitable to describe the dynamic of endemic infections and we conceptually examine if the pair based *SIS* epidemic model without fadeout describes the quasi-stationary distribution of the number of infected individuals on graphs obtained from the method of the master equation. This will help us with the interpretation of the approximate equations.

# Chapter 2

## Exact Equations for the *SEIR* Epidemics on Tree Networks

### 2.1 Introduction

Factors such as proximity, culture, economic status of individual(s) are among the factors that determine whether or not individuals or group of individuals will relate with one another. Despite these numerous and obvious determining factors to possible interaction among individuals in a population, early epidemic models were based on the assumption of homogeneous mixing of individuals in a host population. There have been various modelling approaches to incorporate the observable complexities of interacting units for the best representation of the spread of infectious diseases.

One approach involves examining the various types of connected pairs found within the population. Such pairwise models capture the correlations between neighbouring individuals that are present in the network by including connected individuals as its basic variables. In such models, higher order quantities are usually expressed in terms of lower order quantities and for numerical tractability the system is usually closed at some level through a moment closure relation leading to low dimensional system models [40, 70, 85,154]. Pairwise models have proved very relevant in examining the evolution of pathogen virulence and in the spread and control of sexually transmitted diseases [17,40, 49, 84] where partnership between individuals plays a major role.

At the level of individuals, the subsystem method yields a unifying approach that gives a link between the deterministic and the stochastic model. The entire epidemic system is built up from the perspective of the probability of the infectious status of an individual to the population level leading to hierarchy of moment

equations. A special case of this approach is the pair-based **SIR** epidemic dynamic considered in [155] where a second order moment closure relation that was applied to close the system of equations at the level of pairs was proved to be exact for the model on tree networks.

An *SIR* model takes into account only those diseases which makes a susceptible individual immediately infectious upon making ‘effective’ contact. Here, we consider a modification of this model which describes diseases with an incubation period, during which the individual is infected but not contagious. It takes a while for infected individuals to show symptoms of infection from a disease with a long incubation period, allowing it to spread initially undetected.

An application of an *SIR* model to examine the initial phase of such an infectious disease could be misleading. This new category of individuals that are infected but not yet infectious is duly represented by the variable **E** in an **SEIR** model. **SEIR** is an important class of model that covers wide of diseases with latent states [2, 8, 47, 71, 75]. For example, Measles, Smallpox, Mumps and HIV. Such model becomes a valuable tool in understanding and predicting the delay prior to detecting outbreak and then proffer effective control strategy before outbreak.

In this study, we are interested in the development of an individual-level pairwise **SEIR** model and to determine the differences which the exposed state make while going through the same argument to prove exactness for the *SIR* model in [155]. It is not immediately obvious that the same argument can be applied. As in the *SIR* case, we consider the model on finite time-independent tree-networks of contacts with exponentially distributed transmission, exposed and removal processes. We apply a second order moment closure (1.22) to close the system at the level of pair and prove that the closed form describes an exact representation of the expected infection time series for tree networks. We document the differences with the *SIR* case as they arise.

## 2.2 The Model

We consider an *SEIR* compartmental model with  $N$  individuals whose infectious states are described at any point in time by vectors  $I$ ,  $E$  and  $S$  with respective component for  $i = \{1, 2, \dots, N\}$  as

$$I_i = \begin{cases} 1, & \text{if individual } i \text{ is infectious,} \\ 0, & \text{otherwise.} \end{cases}$$

$$E_i = \begin{cases} 1, & \text{if individual } i \text{ is exposed,} \\ 0, & \text{otherwise.} \end{cases}$$

and

$$S_i = \begin{cases} 1, & \text{if individual } i \text{ is susceptible,} \\ 0, & \text{otherwise.} \end{cases}$$

with  $\lambda_i$ ,  $\beta_i$  and  $\mu_i$  as the respective Poisson process rate parameters for transmission, becoming infectious and recovery where  $\lambda_i = \sum_{j=1}^N T_{ij} S_j I_j$ ,  $\beta_i = \sigma_i E_i$  and  $\mu_i = \gamma_i I_i$ .  $T$  and its elements  $T_{ij}$  are as defined in section (1.7.3),  $\sigma_i$  and  $\gamma_i$  represent the respective rate parameter for an infective individual  $i$  to become infectious and recover from infection.

We comment that a definition for  $R_i$  is not necessary as the definitions of  $I_i$ ,  $E_i$  and  $S_i$  define  $R_i$ .

Similar definitions in section (1.7.3) go for notations  $\langle A_i \rangle$  and  $\langle A_i B_j \rangle$  and then the following differential equations are satisfied for any transmission matrix  $T$  and any pair of nodes  $i$  and  $j$ ,

$$\begin{aligned} \langle \dot{S}_i \rangle &= - \sum_{j=1}^N T_{ij} \langle S_i I_j \rangle, \\ \langle \dot{E}_i \rangle &= \sum_{j=1}^N T_{ij} \langle S_i I_j \rangle - \sigma_i \langle E_i \rangle, \\ \langle \dot{I}_i \rangle &= \sigma_i \langle E_i \rangle - \gamma_i \langle I_i \rangle, \\ \langle \dot{S}_i I_j \rangle &= \sigma_j \langle S_i E_j \rangle - T_{ij} \langle S_i I_j \rangle \\ &\quad - \gamma_j \langle S_i I_j \rangle - \sum_{k=1, k \neq j}^N T_{ik} \langle I_k S_i I_j \rangle, \\ \langle \dot{S}_i E_j \rangle &= \sum_{k=1, k \neq i}^N T_{jk} \langle S_i S_j I_k \rangle - \sum_{k=1, k \neq j}^N T_{ik} \langle I_k S_i E_j \rangle \\ &\quad - \sigma_j \langle S_i E_j \rangle, \\ \langle \dot{S}_i S_j \rangle &= - \sum_{k=1, k \neq j}^N T_{ik} \langle I_k S_i S_j \rangle - \sum_{k=1, k \neq i}^N T_{jk} \langle S_i S_j I_k \rangle, \end{aligned} \tag{2.1}$$

where the dot notation denotes derivative with respect to time.

This system can also be continued up to the full system by writing differential

equations for triples, quadruples and so on. For example, equation for the triple  $\langle S_i S_j E_k \rangle$  will be written as

$$\begin{aligned} \langle S_i \dot{S}_j E_k \rangle &= \sum_{n=1, n \neq i, j}^N T_{kn} \langle S_i S_j S_k I_n \rangle - \sum_{n=1, n \neq j, k}^N T_{in} \langle I_n S_i S_j E_k \rangle \\ &- \sum_{n=1, n \neq i, k}^N T_{jn} \langle S_i I_n S_j I_k \rangle - \sigma_k \langle S_i S_j E_k \rangle. \end{aligned}$$

We then apply the second order moment closure (1.22) to obtain system (2.2)

$$\begin{aligned} \langle \dot{X}_i \rangle &= - \sum_{j=1}^N T_{ij} \langle X_i Z_j \rangle, \\ \langle \dot{Y}_i \rangle &= \sum_{j=1}^N T_{ij} \langle X_i Z_j \rangle - \sigma_i \langle Y_i \rangle, \\ \langle \dot{Z}_i \rangle &= \sigma_i \langle Y_i \rangle - \gamma_i \langle Z_i \rangle, \\ \langle X_i \dot{Z}_j \rangle &= \sigma_j \langle X_i Y_j \rangle - T_{ij} \langle X_i Z_j \rangle \\ &- \gamma_j \langle X_i Z_j \rangle - \sum_{k=1, k \neq j}^N T_{ik} \frac{\langle Z_k X_i \rangle \langle X_i Z_j \rangle}{\langle X_i \rangle}, \\ \langle X_i \dot{Y}_j \rangle &= \sum_{k=1, k \neq i}^N T_{jk} \frac{\langle X_i X_j \rangle \langle X_j Z_k \rangle}{\langle X_j \rangle} - \sigma_j \langle X_i Y_j \rangle \\ &- \sum_{k=1, k \neq j}^N T_{ik} \frac{\langle Z_k X_i \rangle \langle X_i Y_j \rangle}{\langle X_i \rangle}, \\ \langle X_i \dot{X}_j \rangle &= - \sum_{k=1, k \neq j}^N T_{ijk} \frac{\langle Z_k X_i \rangle \langle X_i X_j \rangle}{\langle X_i \rangle} \\ &- \sum_{k=1, k \neq i}^N T_{jk} \frac{\langle X_i X_j \rangle \langle X_j Z_k \rangle}{\langle X_j \rangle}. \end{aligned} \tag{2.2}$$

where  $X$ ,  $Y$  and  $Z$  are used for  $S$ ,  $E$  and  $I$  respectively to emphasize that they are approximations of their respective quantities based on the closure relation.

Our aim is to show that when matrix  $T$  has no cycles (that is, the network represents a tree) and the system is initiated in one of the  $4^N$  possible pure system states then  $\langle X_i \rangle = \langle S_i \rangle$ ,  $\langle Y_i \rangle = \langle E_i \rangle$  and  $\langle Z_i \rangle = \langle I_i \rangle$ . In other word, the closure (1.22) is exact.

## 2.3 The star graph

We shall consider the case of the undirected star graph of figure (2.1) ( where infection can be transmitted in either direction between connected nodes) to prove the case of an initially pure system state. We assume a constant transmission rate  $\tau$ , across all edges, the rate of an infected individual becoming infectious as  $\sigma$  and removal rate for an infectious individual as  $\gamma$ .

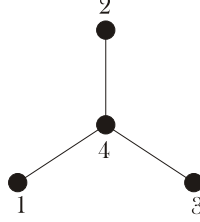


Figure 2.1: *A star graph*

The star graph graph in figure (2.1) has the transmission matrix

$$T = \begin{bmatrix} 0 & 0 & 0 & \tau \\ 0 & 0 & 0 & \tau \\ 0 & 0 & 0 & \tau \\ \tau & \tau & \tau & 0 \end{bmatrix}.$$

Following equation (2.1), the single node subsystem equations are

$$\begin{aligned}
 \langle \dot{S}_1 \rangle &= -\tau \langle S_1 I_4 \rangle, \\
 \langle \dot{S}_2 \rangle &= -\tau \langle S_2 I_4 \rangle, \\
 \langle \dot{S}_3 \rangle &= -\tau \langle S_3 I_4 \rangle, \\
 \langle \dot{S}_4 \rangle &= -\tau \langle I_1 S_4 \rangle - \tau \langle I_2 S_4 \rangle - \tau \langle I_3 S_4 \rangle, \\
 \langle \dot{E}_1 \rangle &= \tau \langle S_1 I_4 \rangle - \sigma \langle E_1 \rangle, \\
 \langle \dot{E}_2 \rangle &= \tau \langle S_2 I_4 \rangle - \sigma \langle E_2 \rangle, \\
 \langle \dot{E}_3 \rangle &= \tau \langle S_3 I_4 \rangle - \sigma \langle E_3 \rangle, \\
 \langle \dot{E}_4 \rangle &= \tau \langle I_1 S_4 \rangle + \tau \langle I_2 S_4 \rangle + \tau \langle I_3 S_4 \rangle - \sigma \langle E_4 \rangle, \\
 \langle \dot{I}_1 \rangle &= \sigma \langle E_1 \rangle - \gamma \langle I_1 \rangle, \\
 \langle \dot{I}_2 \rangle &= \sigma \langle E_2 \rangle - \gamma \langle I_2 \rangle, \\
 \langle \dot{I}_3 \rangle &= \sigma \langle E_3 \rangle - \gamma \langle I_3 \rangle, \\
 \langle \dot{I}_4 \rangle &= \sigma \langle E_4 \rangle - \gamma \langle I_4 \rangle.
 \end{aligned} \tag{2.3}$$

With the equations for pairs as

$$\begin{aligned}
\langle \dot{S}_1 I_4 \rangle &= \sigma \langle S_1 E_4 \rangle - (\tau + \gamma) \langle S_1 I_4 \rangle, \\
\langle \dot{S}_2 I_4 \rangle &= \sigma \langle S_2 E_4 \rangle - (\tau + \gamma) \langle S_2 I_4 \rangle, \\
\langle \dot{S}_3 I_4 \rangle &= \sigma \langle S_3 E_4 \rangle - (\tau + \gamma) \langle S_3 I_4 \rangle, \\
\langle \dot{I}_1 S_4 \rangle &= \sigma \langle E_1 S_4 \rangle - (\tau + \gamma) \langle I_1 S_4 \rangle - \tau \langle I_1 I_2 S_4 \rangle - \tau \langle I_1 I_3 S_4 \rangle, \\
\langle \dot{I}_2 S_4 \rangle &= \sigma \langle E_2 S_4 \rangle - (\tau + \gamma) \langle I_2 S_4 \rangle - \tau \langle I_1 I_2 S_4 \rangle - \tau \langle I_2 I_3 S_4 \rangle, \\
\langle \dot{I}_3 S_4 \rangle &= \sigma \langle E_3 S_4 \rangle - (\tau + \gamma) \langle I_3 S_4 \rangle - \tau \langle I_1 I_3 S_4 \rangle - \tau \langle I_2 I_3 S_4 \rangle, \\
\langle \dot{S}_1 E_4 \rangle &= -\sigma \langle S_1 E_4 \rangle + \tau \langle S_1 I_2 S_4 \rangle + \tau \langle S_1 I_3 S_4 \rangle, \\
\langle \dot{S}_2 E_4 \rangle &= -\sigma \langle S_2 E_4 \rangle + \tau \langle I_1 S_2 S_4 \rangle + \tau \langle S_2 I_3 S_4 \rangle, \\
\langle \dot{S}_3 E_4 \rangle &= -\sigma \langle S_3 E_4 \rangle + \tau \langle I_1 S_3 S_4 \rangle + \tau \langle I_2 S_3 S_4 \rangle, \\
\langle \dot{E}_1 S_4 \rangle &= -\sigma \langle E_1 S_4 \rangle - \tau \langle E_1 I_2 S_4 \rangle - \tau \langle E_1 I_3 S_4 \rangle, \\
\langle \dot{E}_2 S_4 \rangle &= -\sigma \langle E_2 S_4 \rangle - \tau \langle I_1 E_2 S_4 \rangle - \tau \langle E_2 I_3 S_4 \rangle, \\
\langle \dot{E}_3 S_4 \rangle &= -\sigma \langle E_3 S_4 \rangle - \tau \langle I_1 E_3 S_4 \rangle - \tau \langle I_2 E_3 S_4 \rangle, \\
\langle \dot{S}_1 S_4 \rangle &= -\tau \langle S_1 I_2 S_4 \rangle - \tau \langle S_1 I_3 S_4 \rangle, \\
\langle \dot{S}_2 S_4 \rangle &= -\tau \langle I_1 S_2 S_4 \rangle - \tau \langle S_2 I_3 S_4 \rangle, \\
\langle \dot{S}_3 S_4 \rangle &= -\tau \langle I_1 S_3 S_4 \rangle - \tau \langle I_2 S_3 S_4 \rangle.
\end{aligned} \tag{2.4}$$

Systems (2.3) to (2.4) are not closed as the probabilities of the pairs are expressed in terms of triples. We approximate the triples of the form:  $I - S - I$ ,  $S - S - I$  and  $E - S - I$  with the relation (1.22) to close the system. To do this, we need expressions for the triples which appear in system (2.3) to (2.4) and these are given as:

$$\begin{aligned}
\langle I_1 \dot{I}_2 S_4 \rangle &= \sigma \langle I_1 E_2 S_4 \rangle + \sigma \langle E_1 I_2 S_4 \rangle - 2(\tau + \gamma) \langle I_1 I_2 S_4 \rangle - \tau \langle I_1 I_2 I_3 S_4 \rangle, \\
\langle I_1 \dot{I}_3 S_4 \rangle &= \sigma \langle I_1 E_3 S_4 \rangle + \sigma \langle E_1 I_3 S_4 \rangle - 2(\tau + \gamma) \langle I_1 I_3 S_4 \rangle - \tau \langle I_1 I_2 I_3 S_4 \rangle, \\
\langle I_2 \dot{I}_3 S_4 \rangle &= \sigma \langle I_2 E_3 S_4 \rangle + \sigma \langle E_2 I_3 S_4 \rangle - 2(\tau + \gamma) \langle I_2 I_3 S_4 \rangle - \tau \langle I_1 I_2 I_3 S_4 \rangle, \\
\langle S_1 \dot{I}_2 S_4 \rangle &= \sigma \langle S_1 E_2 S_4 \rangle - (\tau + \gamma) \langle S_1 I_2 S_4 \rangle - \tau \langle S_1 I_2 I_3 S_4 \rangle, \\
\langle S_1 \dot{I}_3 S_4 \rangle &= \sigma \langle S_1 E_3 S_4 \rangle - (\tau + \gamma) \langle S_1 I_3 S_4 \rangle - \tau \langle S_1 I_2 I_3 S_4 \rangle, \\
\langle I_1 \dot{S}_2 S_4 \rangle &= \sigma \langle E_1 S_2 S_4 \rangle - (\tau + \gamma) \langle I_1 S_2 S_4 \rangle - \tau \langle I_1 S_2 I_3 S_4 \rangle, \\
\langle I_1 \dot{S}_3 S_4 \rangle &= \sigma \langle E_1 S_3 S_4 \rangle - (\tau + \gamma) \langle I_1 S_3 S_4 \rangle - \tau \langle I_1 I_2 S_3 S_4 \rangle, \\
\langle I_2 \dot{S}_3 S_4 \rangle &= \sigma \langle E_2 S_3 S_4 \rangle - (\tau + \gamma) \langle I_2 S_3 S_4 \rangle - \tau \langle I_1 I_2 S_3 S_4 \rangle, \\
\langle E_1 \dot{I}_2 S_4 \rangle &= \sigma \langle E_1 E_2 S_4 \rangle - (\sigma + \tau + \gamma) \langle E_1 I_2 S_4 \rangle - \tau \langle E_1 I_2 I_3 S_4 \rangle, \\
\langle E_1 \dot{I}_3 S_4 \rangle &= \sigma \langle E_1 E_3 S_4 \rangle - (\sigma + \tau + \gamma) \langle E_1 I_3 S_4 \rangle - \tau \langle E_1 I_2 I_3 S_4 \rangle,
\end{aligned} \tag{2.5}$$

$$\begin{aligned}
\langle I_1 \dot{E}_2 S_4 \rangle &= \sigma \langle E_1 E_2 S_4 \rangle - (\sigma + \tau + \gamma) \langle I_1 E_2 S_4 \rangle - \tau \langle I_1 E_2 I_3 S_4 \rangle, \\
\langle E_2 \dot{I}_3 S_4 \rangle &= \sigma \langle E_2 E_3 S_4 \rangle - (\sigma + \tau + \gamma) \langle E_2 I_3 S_4 \rangle - \tau \langle I_1 E_2 I_3 S_4 \rangle, \\
\langle I_1 \dot{E}_3 S_4 \rangle &= \sigma \langle E_1 E_3 S_4 \rangle - (\sigma + \tau + \gamma) \langle I_1 E_3 S_4 \rangle - \tau \langle I_1 I_2 E_3 S_4 \rangle, \\
\langle I_2 \dot{E}_3 S_4 \rangle &= \sigma \langle E_2 E_3 S_4 \rangle - (\sigma + \tau + \gamma) \langle I_2 E_3 S_4 \rangle - \tau \langle I_1 I_2 E_3 S_4 \rangle, \\
\langle S_2 \dot{I}_3 S_4 \rangle &= \sigma \langle S_2 E_3 S_4 \rangle - (\sigma + \tau + \gamma) \langle S_2 I_3 S_4 \rangle - \tau \langle I_1 S_2 I_3 S_4 \rangle.
\end{aligned}$$

Where

$$\begin{aligned}
\langle E_1 \dot{E}_2 S_4 \rangle &= -2\sigma \langle E_1 E_2 S_4 \rangle - \tau \langle E_1 E_2 I_3 S_4 \rangle, \\
\langle E_1 \dot{E}_3 S_4 \rangle &= -2\sigma \langle E_1 E_3 S_4 \rangle - \tau \langle E_1 I_2 E_3 S_4 \rangle, \\
\langle E_2 \dot{E}_3 S_4 \rangle &= -2\sigma \langle E_2 E_3 S_4 \rangle - \tau \langle I_1 E_2 E_3 S_4 \rangle, \\
\langle S_1 \dot{E}_2 S_4 \rangle &= -\sigma \langle S_1 E_2 S_4 \rangle - \tau \langle S_1 E_2 I_3 S_4 \rangle, \\
\langle S_1 \dot{E}_3 S_4 \rangle &= -\sigma \langle S_1 E_3 S_4 \rangle - \tau \langle S_1 I_2 E_3 S_4 \rangle, \\
\langle E_1 \dot{S}_2 S_4 \rangle &= -\sigma \langle E_1 S_2 S_4 \rangle - \tau \langle E_1 S_2 I_3 S_4 \rangle, \\
\langle E_1 \dot{S}_3 S_4 \rangle &= -\sigma \langle E_1 S_3 S_4 \rangle - \tau \langle E_1 I_2 S_3 S_4 \rangle, \\
\langle E_2 \dot{S}_3 S_4 \rangle &= -\sigma \langle E_2 S_3 S_4 \rangle - \tau \langle I_1 E_2 S_3 S_4 \rangle, \\
\langle S_2 \dot{E}_3 S_4 \rangle &= -\sigma \langle S_2 E_3 S_4 \rangle - \tau \langle I_1 S_2 E_3 S_4 \rangle.
\end{aligned} \tag{2.6}$$

For the quadruples that appear in systems (2.5) to (2.6) we have that:

$$\begin{aligned}
\langle I_1 I_2 \dot{I}_3 S_4 \rangle &= \sigma \langle E_1 I_2 I_3 S_4 \rangle + \sigma \langle I_1 E_2 I_3 S_4 \rangle + \sigma \langle I_1 I_2 E_3 S_4 \rangle - 3(\tau + \gamma) \langle I_1 I_2 I_3 S_4 \rangle, \\
\langle S_1 I_2 \dot{I}_3 S_4 \rangle &= \sigma \langle S_1 E_2 I_3 S_4 \rangle + \sigma \langle S_1 I_2 E_3 S_4 \rangle - 2(\tau + \gamma) \langle S_1 I_2 I_3 S_4 \rangle, \\
\langle I_1 S_2 \dot{I}_3 S_4 \rangle &= \sigma \langle E_1 S_2 I_3 S_4 \rangle + \sigma \langle I_1 S_2 E_3 S_4 \rangle - 2(\tau + \gamma) \langle I_1 S_2 I_3 S_4 \rangle, \\
\langle I_1 I_2 \dot{S}_3 S_4 \rangle &= \sigma \langle E_1 I_2 S_3 S_4 \rangle + \sigma \langle I_1 E_2 S_3 S_4 \rangle - 2(\tau + \gamma) \langle I_1 I_2 S_3 S_4 \rangle, \\
\langle E_1 I_2 \dot{I}_3 S_4 \rangle &= \sigma \langle E_1 E_2 I_3 S_4 \rangle + \sigma \langle E_1 I_2 E_3 S_4 \rangle - (\sigma + 2\tau + 2\gamma) \langle E_1 I_2 I_3 S_4 \rangle, \\
\langle I_1 E_2 \dot{I}_3 S_4 \rangle &= \sigma \langle E_1 E_2 I_3 S_4 \rangle + \sigma \langle I_1 E_2 E_3 S_4 \rangle - (\sigma + 2\tau + 2\gamma) \langle I_1 E_2 I_3 S_4 \rangle, \\
\langle I_1 I_2 \dot{E}_3 S_4 \rangle &= \sigma \langle E_1 I_2 E_3 S_4 \rangle + \sigma \langle I_1 E_2 E_3 S_4 \rangle - (\sigma + 2\tau + 2\gamma) \langle I_1 I_2 E_3 S_4 \rangle, \\
\langle E_1 E_2 \dot{I}_3 S_4 \rangle &= \sigma \langle E_1 E_2 E_3 S_4 \rangle - (2\sigma + \tau + \gamma) \langle E_1 E_2 I_3 S_4 \rangle, \\
\langle E_1 I_2 \dot{E}_3 S_4 \rangle &= \sigma \langle E_1 E_2 E_3 S_4 \rangle - (2\sigma + \tau + \gamma) \langle E_1 I_2 E_3 S_4 \rangle, \\
\langle I_1 E_2 \dot{E}_3 S_4 \rangle &= \sigma \langle E_1 E_2 E_3 S_4 \rangle - (2\sigma + \tau + \gamma) \langle I_1 E_2 E_3 S_4 \rangle, \\
\langle S_1 E_2 \dot{I}_3 S_4 \rangle &= \sigma \langle S_1 E_2 E_3 S_4 \rangle - (\sigma + \tau + \gamma) \langle S_1 E_2 I_3 S_4 \rangle, \\
\langle S_1 I_2 \dot{E}_3 S_4 \rangle &= \sigma \langle S_1 E_2 E_3 S_4 \rangle - (\sigma + \tau + \gamma) \langle S_1 I_2 E_3 S_4 \rangle, \\
\langle E_1 S_2 \dot{I}_3 S_4 \rangle &= \sigma \langle E_1 S_2 E_3 S_4 \rangle - (\sigma + \tau + \gamma) \langle E_1 S_2 I_3 S_4 \rangle.
\end{aligned} \tag{2.7}$$



Where

$$\begin{aligned}
\langle E_1 I_2 \dot{S}_3 S_4 \rangle &= \sigma \langle E_1 E_2 S_3 S_4 \rangle - (\sigma + \tau + \gamma) \langle E_1 I_2 S_3 S_4 \rangle, \\
\langle I_1 S_2 \dot{E}_3 S_4 \rangle &= \sigma \langle E_1 S_2 E_3 S_4 \rangle - (\sigma + \tau + \gamma) \langle I_1 S_2 E_3 S_4 \rangle, \\
\langle I_1 E_2 \dot{S}_3 S_4 \rangle &= \sigma \langle E_1 E_2 S_3 S_4 \rangle - (\sigma + \tau + \gamma) \langle I_1 E_2 S_3 S_4 \rangle, \\
\langle E_1 E_2 \dot{E}_3 S_4 \rangle &= -3\sigma \langle E_1 E_2 E_3 S_4 \rangle, \\
\langle S_1 E_2 \dot{E}_3 S_4 \rangle &= -2\sigma \langle S_1 E_2 E_3 S_4 \rangle, \\
\langle E_1 S_2 \dot{E}_3 S_4 \rangle &= -2\sigma \langle E_1 S_2 E_3 S_4 \rangle, \\
\langle E_1 E_2 \dot{S}_3 S_4 \rangle &= -2\sigma \langle E_1 E_2 S_3 S_4 \rangle.
\end{aligned} \tag{2.8}$$

We first consider closure for  $I_1 S_4 I_2$ , and following the notation in [155], we shall be using  $\psi_{142}^{ISI}$  to mean  $I_1 S_4 I_2$  and this applies to other states by extension.

We consider:

$$\langle \psi_{142}^{ISI} \rangle = \frac{\langle \psi_{14}^{IS} \rangle \langle \psi_{42}^{SI} \rangle}{\langle \psi_4^S \rangle}.$$

Then we define  $\alpha_1(t)$  as

$$\alpha_1(t) = \langle \psi_4^S \rangle \langle \psi_{142}^{ISI} \rangle - \langle \psi_{14}^{IS} \rangle \langle \psi_{42}^{SI} \rangle. \tag{2.9}$$

If the system is initiated in a definite state, then following the approach used in [155] as explained in section (1.7.3), the closure is exact if  $\alpha_1(t) = 0$  for all  $t \geq 0$ .

Differentiating  $\alpha_1(t)$  and substituting relevant derivatives and quantities from system (2.3)-(2.8) we have

$$\dot{\alpha}_1(t) = -(2\tau + 2\gamma)\alpha_1(t) - \tau\alpha_2(t) - \sigma\alpha_3(t) - \tau\alpha_4(t) - \sigma\alpha_5(t). \tag{2.10}$$

where:

$$\begin{aligned}
\alpha_2(t) &= \langle \psi_4^S \rangle \langle \psi_{1234}^{IIIS} \rangle - \langle \psi_{14}^{IS} \rangle \langle \psi_{234}^{IIS} \rangle, \\
\alpha_3(t) &= \langle \psi_{14}^{ES} \rangle \langle \psi_{24}^{IS} \rangle - \langle \psi_4^S \rangle \langle \psi_{124}^{EIS} \rangle, \\
\alpha_4(t) &= \langle \psi_{34}^{IS} \rangle \langle \psi_{124}^{IIS} \rangle - \langle \psi_{24}^{IS} \rangle \langle \psi_{134}^{IIS} \rangle, \\
\alpha_5(t) &= \langle \psi_{14}^{IS} \rangle \langle \psi_{24}^{ES} \rangle - \langle \psi_4^S \rangle \langle \psi_{124}^{IES} \rangle.
\end{aligned}$$

Differentiating  $\alpha_2(t)$  we have

$$\dot{\alpha}_2(t) = -(3\tau + 3\gamma)\alpha_2(t) - \sigma\alpha_6(t) - \sigma\alpha_7(t) - \tau\alpha_8(t) - \tau\alpha_9(t) - \sigma\alpha_{10}(t). \tag{2.11}$$

where

$$\begin{aligned}
\alpha_6(t) &= \langle \psi_{14}^{ES} \rangle \langle \psi_{234}^{IIS} \rangle - \langle \psi_4^S \rangle \langle \psi_{1234}^{EIIIS} \rangle, \\
\alpha_7(t) &= \langle \psi_{14}^{IS} \rangle \langle \psi_{234}^{EIS} \rangle - \langle \psi_4^S \rangle \langle \psi_{1234}^{IEIS} \rangle, \\
\alpha_8(t) &= \langle \psi_{24}^{IS} \rangle \langle \psi_{1234}^{IIS} \rangle - \langle \psi_{124}^{IIS} \rangle \langle \psi_{234}^{IIS} \rangle, \\
\alpha_9(t) &= \langle \psi_{34}^{IS} \rangle \langle \psi_{1234}^{IIS} \rangle - \langle \psi_{134}^{IIS} \rangle \langle \psi_{234}^{IIS} \rangle, \\
\alpha_{10}(t) &= \langle \psi_{14}^{IS} \rangle \langle \psi_{234}^{IEIS} \rangle - \langle \psi_4^S \rangle \langle \psi_{1234}^{IEIS} \rangle.
\end{aligned}$$

Differentiating  $\alpha_6(t)$  we have

$$\dot{\alpha}_6(t) = -(2\tau + 2\gamma + \sigma)\alpha_6(t) - \tau\alpha_{11}(t) - \tau\alpha_{12}(t) - \sigma\alpha_{13}(t) - \sigma\alpha_{14}(t), \quad (2.12)$$

where

$$\begin{aligned}
\alpha_{11}(t) &= \langle \psi_{124}^{EIS} \rangle \langle \psi_{234}^{IIS} \rangle - \langle \psi_{24}^{IS} \rangle \langle \psi_{1234}^{EIIIS} \rangle, \\
\alpha_{12}(t) &= \langle \psi_{134}^{EIS} \rangle \langle \psi_{234}^{IIS} \rangle - \langle \psi_{34}^{IS} \rangle \langle \psi_{1234}^{EIIIS} \rangle, \\
\alpha_{13}(t) &= \langle \psi_4^S \rangle \langle \psi_{1234}^{EEIS} \rangle - \langle \psi_{14}^{ES} \rangle \langle \psi_{234}^{EIS} \rangle, \\
\alpha_{14}(t) &= \langle \psi_4^S \rangle \langle \psi_{1234}^{EIEIS} \rangle - \langle \psi_{14}^{ES} \rangle \langle \psi_{234}^{IEIS} \rangle.
\end{aligned}$$

Differentiating  $\alpha_{11}(t)$  we have

$$\dot{\alpha}_{11}(t) = -(3\tau + 3\gamma + \sigma)\alpha_{11}(t) - \sigma\alpha_{15}(t), \quad (2.13)$$

where

$$\alpha_{15}(t) = \langle \psi_{24}^{IS} \rangle \langle \psi_{1234}^{EIEIS} \rangle - \langle \psi_{124}^{EIS} \rangle \langle \psi_{234}^{IEIS} \rangle.$$

Differentiating  $\alpha_{15}(t)$  we have

$$\dot{\alpha}_{15}(t) = -2(\tau + \gamma + \sigma)\alpha_{15}(t), \quad (2.14)$$

which implies

$$\alpha_{15}(t) = \alpha_{15}(0)e^{-2(\tau+\gamma+\sigma)t}.$$

Since the system is initiated in a pure state, we have that  $\alpha_{15}(0) = 0$ . Consequently,  $\alpha_{15}(t) = 0$  for all  $t \geq 0$ . Therefore, from (2.13),  $\alpha_{11}(t) = \alpha_{11}(0)e^{-(3\tau+3\gamma+\sigma)t}$  and by the same argument of starting the system in a pure state,  $\alpha_{11}(0) = 0$  and this implies  $\alpha_{11}(t) = 0$  for all  $t \geq 0$ .

Differentiating  $\alpha_{12}(t)$  we have

$$\dot{\alpha}_{12}(t) = -(3\tau + 3\gamma + \sigma)\alpha_{12}(t) - \sigma\alpha_{16}(t), \quad (2.15)$$

where

$$\alpha_{16}(t) = \langle \psi_{34}^{IS} \rangle \langle \psi_{1234}^{EES} \rangle - \langle \psi_{134}^{EIS} \rangle \langle \psi_{234}^{EIS} \rangle.$$

Differentiating  $\alpha_{16}(t)$  we have

$$\dot{\alpha}_{16}(t) = -2(\tau + \gamma + \sigma)\alpha_{16}(t). \quad (2.16)$$

That is,  $\alpha_{16}(t) = \alpha_{16}(0)e^{-2(\tau+\gamma+\sigma)t}$  and for the same reason above,  $\alpha_{16}(0) = 0$ . Consequently,  $\alpha_{16}(t) = 0$  for all  $t \geq 0$ . Hence from (2.15),  $\alpha_{12}(t) = \alpha_{12}(0)e^{-(3\tau+3\gamma+\sigma)t}$  and since  $\alpha_{12}(0) = 0$  for the same reason above, this also implies  $\alpha_{12}(t) = 0$  for all  $t \geq 0$ .

Differentiating  $\alpha_{13}(t)$  we get

$$\dot{\alpha}_{13}(t) = -(\tau + \gamma + 2\sigma)\alpha_{13} - \tau\alpha_{16} - \sigma\alpha_{17}, \quad (2.17)$$

where

$$\alpha_{17}(t) = \langle \psi_{14}^{ES} \rangle \langle \psi_{234}^{EES} \rangle - \langle \psi_4^S \rangle \langle \psi_{1234}^{EEES} \rangle.$$

Differentiating  $\alpha_{17}(t)$  we have

$$\dot{\alpha}_{17}(t) = -3\sigma\alpha_{17}(t). \quad (2.18)$$

We then also have that  $\alpha_{17}(t) = \alpha_{17}(0)e^{-3\sigma t}$ . For the same reason as above  $\alpha_{17}(0) = 0$  and we have that  $\alpha_{17}(t) = 0$  for all  $t \geq 0$ . Then from (2.17),  $\alpha_{13}(t) = \alpha_{13}(0)e^{-(\tau+\gamma+2\sigma)t}$  and because  $\alpha_{13}(0) = 0$  this also implies  $\alpha_{13}(t) = 0$  for all  $t \geq 0$ .

Differentiating  $\alpha_{14}(t)$  from (2.12) we have

$$\dot{\alpha}_{14}(t) = -(\tau + \gamma + 2\sigma)\alpha_{14} - \sigma\alpha_{16} - \tau\alpha_{17}. \quad (2.19)$$

where already  $\alpha_{16}(t) = 0$  for all  $t \geq 0$  from (2.16) and  $\alpha_{17}(t) = 0$  for all  $t \geq 0$  from (2.18). We then have that  $\alpha_{14}(t) = \alpha_{14}(0)e^{-(\tau+\gamma+2\sigma)t}$  and since  $\alpha_{14}(0) = 0$  for the same reason above, this means  $\alpha_{14}(t) = 0$  for all  $t \geq 0$ .

Since  $\alpha_{11}(t) = 0$ ,  $\alpha_{12}(t) = 0$ ,  $\alpha_{13}(t) = 0$  and  $\alpha_{14}(t) = 0$  for all  $t \geq 0$ , then by backward substitution into (2.12) we have that  $\alpha_6(t) = \alpha_6(0)e^{-(2\tau+2\gamma+\sigma)t}$  and from the initial condition that  $\alpha_6(0) = 0$ , we finally have that  $\alpha_6(t) = 0$  for all  $t \geq 0$ .

The derivatives for  $\alpha_2(t)$  to  $\alpha_{10}(t)$  and subsequent  $\alpha$ 's follow similar analysis as shown above. Results show that all the  $\alpha(t)$ , from  $\alpha_1(t)$  to  $\alpha_{17}(t)$  are all zero for all  $t \geq 0$ . Hence the closure is exact.

The proof for the closure of the other triples:  $S$ - $S$ - $I$  and  $E$ - $S$ - $I$  follows same procedure as above and we comment that the presence of an intermediate state

‘ $E$ ’ between a node being susceptible and infectious has resulted in more  $\alpha$ ’s to be differentiated and shown to be zero for all time,  $t \geq 0$ .

We observed that each of the closure relation proved here consists of two pairs of the form,  $\langle \psi_W^A \rangle \langle \psi_X^B \rangle$  and  $\langle \psi_Y^C \rangle \langle \psi_Z^D \rangle$ , which can be termed compatible pairs (CPs). The CPs that emerge in this process exhibited the following properties

- Any given node  $i$  appearing as  $S_i$ ,  $E_i$  or  $I_i$  appears equal number of times on both the left and right pair. For example, in  $\alpha_{12} = \langle E_1 I_3 S_4 \rangle \langle I_2 I_3 S_4 \rangle - \langle I_3 S_4 \rangle \langle E_1 I_2 I_3 S_4 \rangle$ , node 1 appears as  $E$  once in the left pair and also once in the right pair. For  $\alpha_{15}(t) = \langle I_2 S_4 \rangle \langle E_1 I_2 E_3 S_4 \rangle - \langle E_1 I_2 S_4 \rangle \langle I_2 E_3 S_4 \rangle$ , node 2 appears as  $I$  twice on the left pair  $\langle I_2 S_4 \rangle \langle E_1 I_2 E_3 S_4 \rangle$  and twice on the right pair  $\langle E_1 I_2 S_4 \rangle \langle I_2 E_3 S_4 \rangle$ .
- Any pair  $SI$  or  $SE$  on the left pair appears equal number of time on the right pair of the CPs. In the above example of  $\alpha_{12}$ , the pair  $I_3 S_4$  appears twice in each of the pairs while the pair  $E_1 S_4$  appears once in each of the pair. For  $\alpha_{15}$ , the pair  $I_2 S_4$  appears twice in each of the pair while the pair  $E_3 S_4$  appears once in each pair of the CPs.

Such pairs, for example, the  $\langle I_2 S_4 \rangle \langle E_1 I_2 E_3 S_4 \rangle$  and  $\langle E_1 I_2 S_4 \rangle \langle I_2 E_3 S_4 \rangle$  for  $\alpha_{15}$  of equation (2.13) are termed compatible pairs.

We shall quickly formalise our observation of the term ‘compatible pairs’ in definition 2.3.1 as we will need them in the general proof of the exactness of the closure relation (1.22):

**Definition 2.3.0:**  $\psi_i^a \subset \psi_W^A$  if  $\exists j$  such that  $W_j = i$  and  $A_j = a$ .  $\psi_{i_1, i_2}^{a_1 a_2} \subset \psi_W^A$  if  $\exists j_1, j_2$  such that  $W_{j_1} = i_1, W_{j_2} = i_2$  and  $A_{j_1} = a_1, A_{j_2} = a_2$

**Definition 2.3.1:** Two pairs of motif states  $\psi_W^A, \psi_X^B$  and  $\psi_Y^C, \psi_Z^D$  are called compatible pairs if

- CP(i)  $\psi_i^a \subset \psi_W^A$  or  $\psi_i^a \subset \psi_X^B$  implies  $\psi_i^a \subset \psi_Y^C$  or  $\psi_i^a \subset \psi_Z^D$
- CP(ii)  $\psi_i^a \subset \psi_W^A$  and  $\psi_i^a \subset \psi_X^B$  implies  $\psi_i^a \subset \psi_Y^C$  and  $\psi_i^a$  is in  $\psi_Z^D$
- CP(iii)  $\psi_{i_1, i_2}^{IS} \subset \psi_W^A$  or  $\psi_{i_1, i_2}^{IS} \subset \psi_X^B$  implies  $\psi_{i_1, i_2}^{IS} \subset \psi_Y^C$  or  $\psi_{i_1, i_2}^{IS} \subset \psi_Z^D$
- CP(iv)  $\psi_{i_1, i_2}^{IS} \subset \psi_W^A$  and  $\psi_{i_1, i_2}^{IS} \subset \psi_X^B$  implies  $\psi_{i_1, i_2}^{IS} \subset \psi_Y^C$  and  $\psi_{i_1, i_2}^{IS} \subset \psi_Z^D$
- CP(v)  $\psi_{i_1, i_2}^{ES} \subset \psi_W^A$  or  $\psi_{i_1, i_2}^{ES} \subset \psi_X^B$  implies  $\psi_{i_1, i_2}^{ES} \subset \psi_Y^C$  or  $\psi_{i_1, i_2}^{ES} \subset \psi_Z^D$
- CP(vi)  $\psi_{i_1, i_2}^{ES} \subset \psi_W^A$  and  $\psi_{i_1, i_2}^{ES} \subset \psi_X^B$  implies  $\psi_{i_1, i_2}^{ES} \subset \psi_Y^C$  and  $\psi_{i_1, i_2}^{ES} \subset \psi_Z^D$
- CP(v) and CP(vi) hold but with  $SS$  pairs.

We comment that CP(v) and CP(vi) are additional CPs when compared with those CPs observed for the *SIR* case of [155]. These extra CPs are due to the presence of the intermediate state, *E* between of a node being susceptible and being infectious.

## 2.4 Differential equations for general system and subsystem states

We prove the general case by formulating how the differential equations (2.1) of the subsystem states are generated. The stochastic system ( $\Gamma$ ) consists of  $N$  individuals, each of which can be in any of the *S*, *E*, *I* or *R* states at any time. This corresponds to  $4^N$  possible states. That is  $\Gamma^\alpha$ ,  $\alpha \in \{1, 2, \dots, 4^N\}$ . We want to comment that the master equation (2.20) completely describes the time-evolution of the probabilities of these states and that when  $T$  represents a tree, then system (2.20) implies system (2.1)

$$\langle \dot{\Gamma}^\alpha \rangle = \sum_{\beta=1}^{4^N} Q^{\alpha\beta} \langle \Gamma^\beta \rangle - \sum_{\beta=1}^{4^N} Q^{\beta\alpha} \langle \Gamma^\alpha \rangle. \quad (2.20)$$

where  $Q$  is the constant matrix of Poisson rate parameters.

We shall make use of some notations of [155] and define some function to help our derivation of the subsystem states:

Let  $A_i$  be in any of the infection states *S*, *E*, *I* and *R* for all  $i \in \{1, 2, \dots, r\}$ . We define  $A = (A_1, A_2, \dots, A_r)$  as a sequence of *S*, *E*, *I* and *R* symbols of length  $r$  such that the state of node  $W_i$  is  $A_i$  in  $\psi_w^A$ . That is  $\psi_i^S (= S_i)$  means node  $i$  is susceptible and the length (i.e. number of nodes in) of  $\psi_i^S$  is 1, and  $\psi_{ij}^{SE} = S_i E_j$  means node  $i$  is susceptible and node  $j$  is exposed in the pair  $S_i E_j$  and the length of  $\psi_{ij}^{SE}$  is 2.

The definitions for motif of in [155] also apply here. That is, for example,  $\psi_{12}^{SI}$  is a 2 – motif system if there is an edge between nodes 1 and 2. And the state of an  $r$  – motif is an  $r$  – state. For example,  $\psi_{12}^{SI}$  is a 2 – state system.

**Definition 2.4.0:** For the subsystem  $\psi_W^A$ , if node  $W_k$  is *I* (i.e. **infectious**) then;

$$h_{W_k}(\psi_W^A) = \psi_W^{A_1 \dots A_{k-1} E A_{k+1} \dots A_r}$$

If node  $W_k$  is  $S$  (i.e. **susceptible**) then;

$$h_{W_k}(\psi_W^A) = \psi_W^{A_1 \dots A_{k-1} S A_{k+1} \dots A_r}$$

If node  $W_k$  is  $E$  (i.e. **exposed**) then;

$$h_{W_k}(\psi_W^A) = \psi_W^{A_1 \dots A_{k-1} E A_{k+1} \dots A_r}$$

If node  $W_k$  is  $R$  (i.e. **removed**) then;

$$h_{W_k}(\psi_W^A) = \psi_W^{A_1 \dots A_{k-1} R A_{k+1} \dots A_r}$$

This means, if node  $W_k$  is infectious, this operator changes the state of the node to  $E$  but leaves the state unchanged if it is originally susceptible, exposed or removed. For example;

$$h_2(\psi_{12}^{SI}) = \psi_{12}^{SE}, \quad h_2(\psi_{12}^{SE}) = \psi_{12}^{SE} \quad \text{and} \quad h_2(\psi_{12}^{SR}) = \psi_{12}^{SR}.$$

We comment that for a network without cycles, this transformation function  $h_k$  on node  $k$  in any of the motifs that make up compatible pairs does not alter the compatibility of the pairs (see proposition 3.2 of [155]).

**Definition 2.4.1:** For an undirected network, we say node  $j$  is a neighbour to node  $i$  if there is an edge connecting  $i$  and  $j$ . Let  $N_i^*$  denote the set of neighbours of node  $i$ . From the transmission matrix, this implies  $T_{ij} = 1 \forall j \in N_i^*$ .

**Definition 2.4.2:** A subsystem of  $(r + 1)$  nodes can be generated from the subsystem state  $\psi_W^A$  of  $r$  node(s). Let  $k \in \{1, 2, \dots, r\}$  and consider a neighbour  $n$  of  $W_k$  outside of the subsystem with a network link towards  $W_k$ , i.e. let node  $n$  which is outside the subsystem  $\psi_W^A$  has a link towards its node  $k$ , that is,  $n \in N_{W_k}$ ,  $n \notin W$ . If  $A_k = S$ , then the generated  $(r + 1)$  state is given by the generating rule:

$$g_{W_k}^n(\psi_W^A) = \psi_{W_1, \dots, W_r, n}^{A_1 \dots A_r I}$$

In other words, If the node  $W_k$  is originally susceptible,  $S$ , then its state is not changed by the generating function while connecting an external infectious node,  $I_n$  to it. For example:

$$g_1^n(\psi_{12}^{SI}) = \psi_{n,12}^{ISI}$$

provided node  $I_n$  is connected to  $S_1$ .

If  $A_k$  is  $E$ , then the generated subsystem state is given by:

$$g_{W_k}^n(\psi_W^A) = \psi_{W_1, \dots, W_r, n}^{A_1 \dots A_{k-1}, S, A_{k+1}, \dots, A_r, I}.$$

That is, node  $W_k$  which is originally exposed,  $E$ , is first changed to susceptible,  $S$ , and then an external infectious node,  $I_n$  which has undirected link with  $W_k$  is connected to it. For example:

$$g_2^n(\psi_{12}^{SE}) = \psi_{12, n}^{SSI}$$

provided node  $I_n$  is external to the subsystem  $\psi_W^A$  and is linked to  $E_2$ .

If  $A_k = R$  i.e. removed or  $A_k = I$  i.e. infectious, then the generator  $g_{W_k}^n$  leaves the system unchanged. Generally, when there is no link between  $A_k$  and node  $n$  in matrix  $T$ , the subsystem is also left unchanged.

**LEMMA 2.4.3:** For a tree network, the transformation  $h_k$  acting on an infectious node  $k$  in any of the motifs of CPs generates CPs.

*PROOF:* The transformation  $h_k$  satisfies CP(i) and CP(ii) by changing an infectious node  $k$  to exposed without altering the form of the conditions of CP(i) and CP(ii).  $h_k$  also satisfies CP(iii) and CP(iv) by changing node  $I$  to  $E$  in an **SI** pair. The number(s) of **SI** pairs is reduced by this transformation and this reduction is reflected in the increased number of **SE** pairs. The transformation also leaves an existing **ES** pair as it is if node  $k$  is  $E$  thereby satisfying CP(v) and CP(vi) while a new pair of **SE** is formed from an **SI** pair by  $h_k$ . Also  $h_k$  satisfies CP(v) and CP(vi) leaving an existing **SS** pair in its original form.

As described by the generating rule,  $g_{W_k}^n(\psi_W^A)$ , it is observed from (2.1) that the infection process which starts and build up from the exposed states of the single nodes  $\psi_i^E$ ,  $i \in \{1, 2, \dots, N\}$  depends on the 2 – states  $\psi_{ij}^{SI}$ ,  $j \in N_i^*$ . The differential equation for the 2 – state  $\psi_{ij}^{SI}$  is also expressed in terms of the 3 – states  $\psi_{kij}^{ISI}$ ,  $k \in N_i^*$ . The differential equations for the 3 – states will contain the 4 – states and so on.

We comment here that similar transformation function for the  $(r + 1)$  motif in the **SIR** model in [155] replaces an infected node  $I$  with an **SI** edge while in this model an exposed node  $E$  is replaced with an **SI** edge.

**LEMMA 2.4.4:** The dependency of the  $r$  – states on the  $(r + 1)$  states for

$r \in \{1, 2, \dots, (N-1)\}$  implies that if the neighbours of  $E$  are susceptible in  $r$ -motif, their neighbours in  $(r+1)$  motif are also susceptible.

*PROOF:* According to the rules  $h_k$  and  $g_k^n$  of definitions 2.4.0 and 2.4.2, the process of building up the states never give rise to **removed**. That is, a subsystem such as  $\psi^A$  where  $A = S_1 R_2 R_3 I_4 \dots$  can not arise on tree network.

**Definition 2.4.5:** For any subsystem  $\psi_W$  of  $r$  node(s) in state  $\psi_W^A$  and a single node  $k \in \{1, 2, \dots, r\}$  in state  $\psi_{W_k}$ , we define according to [155]

$$D_k^{Aa} = \begin{cases} 1, & \text{if } A_k = a, \\ 0, & \text{otherwise.} \end{cases}$$

Then, provided lemma 2.4.4 holds, the rate of change of the probability of an  $r$ -state subsystem  $\psi_W^A$  is

$$\begin{aligned} \langle \dot{\psi}_W^A \rangle = & \sum_{k=1}^r [D_k^{AE} \sum_{n=1, n \neq w}^N T_{W_k n} \langle g_{W_k}^n(\psi_W^A) \rangle - D_k^{AE} \sigma_{W_k} \langle \psi_W^A \rangle \\ & - D_k^{AS} \sum_{n=1, n \neq w}^N T_{W_k n} \langle g_{W_k}^n(\psi_W^A) \rangle - D_k^{AS} \sum_{l=1}^N T_{W_k W_l} D_l^{AI} \langle \psi_{W_k}^A \rangle \\ & + D_k^{AI} \sigma_{W_k} h_{W_k} \langle \psi_W^A \rangle - D_k^{AI} \gamma_{W_k} \langle \psi_{W_k}^A \rangle]. \end{aligned} \quad (2.21)$$

The subsystem states that emerged in this study satisfy equation (2.21) and showed no **removed**, hence we can use equation (2.21) to obtain the set of equations in (2.1).

We want to state that system (2.21) is not valid for non-tree networks. Let us consider system (2.21) for, for example, the term  $\langle E_1 S_4 \rangle$  in figure (2.2). Equation (2.21) is reduced to:

$$\langle \dot{\psi}_{ij}^{SE} \rangle = - \sum_{n=1, n \neq j}^N T_{in} \langle \psi_{n,ij}^{ISE} \rangle + \sum_{n=1, n \neq i}^N T_{jn} \langle \psi_{ij,n}^{SSI} \rangle - \sigma_j \langle \psi_{ij}^{SE} \rangle,$$

which gives

$$\begin{aligned} \langle E_1 S_4 \rangle = & - \tau \langle E_1 I_2 S_4 \rangle - \tau \langle E_1 I_3 S_4 \rangle + \tau \langle I_5 S_1 S_4 \rangle \\ & + \tau \langle S_1 I_2 S_4 \rangle - \sigma \langle E_1 S_4 \rangle. \end{aligned} \quad (2.22)$$



Writing this equation directly from the graph gives

$$\begin{aligned} \langle E_1 S_4 \rangle &= -\tau \langle E_1 I_2 S_4 \rangle - \tau \langle E_1 I_3 S_4 \rangle + \tau \langle I_5 S_1 S_4 \rangle \\ &+ 2\tau \langle S_1 I_2 S_4 \rangle - \sigma \langle E_1 S_4 \rangle. \end{aligned} \quad (2.23)$$

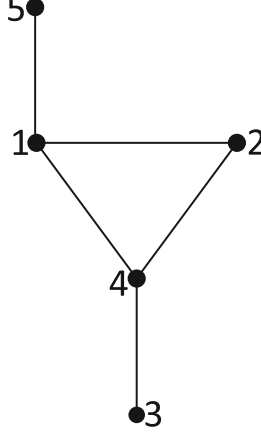


Figure 2.2: A simple graph to invalidate equation (2.21) for non-tree graphs.

The coefficients of the term  $\langle S_1 I_2 S_4 \rangle$  in the two equations are different. This difference is due to the presence of triangle in the network and of course these two equations can not define same distribution for the term  $\langle E_1 S_4 \rangle$ , hence equation (2.21) breaks down for non-tree networks.

We comment that while the generation of the subsystems  $\psi_i^S$  and  $\psi_{ij}^{SS}$  from (2.21) is similar to that for the *SIR* model of [155], (2.21) implies different equations for the subsystems  $\psi_i^E$ ,  $\psi_i^I$ ,  $\psi_{ij}^{SI}$  and  $\psi_{ij}^{SE}$ . For example,

- If the subsystem is a susceptible individual, say  $\psi_i^S$ , then  $r = 1$  so  $k$  can only take the value  $k = 1$  where  $W_1 = i$  and  $A_1 = S$ . The equation for this subsystem state comes from the third term of (2.21) which is:

$$\langle \dot{\psi}_i^S \rangle = -D_k^{AS} \sum_{n=1}^1 T_{in} \langle g_i^n(\psi_i^S) \rangle.$$

That is;

$$\langle \dot{\psi}_i^S \rangle = - \sum_{n=1, i \neq n}^N T_{in} \langle \psi_{i,n}^{SI} \rangle.$$

This term and that generated for subsystem  $\langle \psi_{ij}^{SS} \rangle$  have similar form of that in [155].

- For an exposed individual, say  $\psi_i^E$ , the first two terms of (2.21) generate the subsystem state:

$$\langle \dot{\psi}_i^E \rangle = D_k^{AE} \sum_{n=1}^N T_{in} \langle g_i^n(\psi_i^E) \rangle - D_k^{AE} \sigma_i \langle \psi_i^E \rangle.$$

That is;

$$\langle \dot{\psi}_i^E \rangle = \sum_{n=1, i \neq n}^N T_{in} \langle \psi_{i,n}^{SI} \rangle - \sigma_i \langle \psi_i^E \rangle.$$

This generator takes a different form compared to that of the *SIR* model in [155] for the same subsystem.

- For an infectious individual, say  $\psi_i^I$ , the last two terms of (2.21) combine to generate the subsystem state:

$$\langle \dot{\psi}_i^I \rangle = D_i^{AI} \sum_{i=1} \sigma_i h_i \langle \psi_i^I \rangle - \sum_{i=1} D_i^{AI} \gamma_i \langle \psi_i^I \rangle.$$

That is;

$$\langle \dot{\psi}_i^I \rangle = \sigma_i \langle \psi_i^E \rangle - \gamma_i \langle \psi_i^I \rangle.$$

Note the effect of the operator  $h_{W_k}$  which transforms  $I_i$  to  $E_i$ . This is also a new term when compared with those terms for the *SIR* model in [155].

- If the subsystem is the pair  $\psi_{ij}^{SI}$ ,  $r = 2$  and the sum over  $k$  is over  $k = 1$  and  $k = 2$ , and  $W_1 = i$  and  $W_2 = j$ ,  $A_1 = S$  and  $A_2 = I$ , the last four terms of (2.21) combine to generate the subsystem and so (2.21) reduces to

$$\begin{aligned} \langle \dot{\psi}_{ij}^{SI} \rangle &= -D_i^{AS} \sum_{n=1, n \neq j} T_{in} \langle g_i^n(\psi_{ij}^{SI}) \rangle - D_i^{AS} \sum_{i=1} T_{ij} D_j^{AI} \langle \psi_{ij}^{SI} \rangle \\ &+ D_j^{AI} \sigma_j h_j \langle \psi_{ij}^{SI} \rangle - D_j^{AI} \gamma_j \langle \psi_{ij}^{SI} \rangle. \end{aligned}$$

That is,

$$\langle \dot{\psi}_{ij}^{SI} \rangle = - \sum_{n=1, n \neq i}^N T_{in} \langle \psi_{n,ij}^{ISI} \rangle - T_{ij} \langle \psi_{ij}^{SI} \rangle + \sigma_j \langle \psi_{ij}^{SE} \rangle - \gamma_j \langle \psi_{ij}^{SI} \rangle.$$

where the first two terms correspond to  $k = 1$  and the last two terms correspond to  $k = 2$ . This is also a new term.

- If the subsystem is the pair  $\psi_{ij}^{SE}$ , then  $r = 2$ , the sum over  $k$  will be over  $k = 1$  and  $k = 2$ , so  $W_1 = i$ ,  $W_2 = j$ ,  $A_1 = S$  and  $A_2 = E$ , the 1st, 2nd and

fourth terms combine to generate the subsystem and this reduces (2.21) to

$$\begin{aligned}\langle \psi_{ij}^{SE} \rangle &= -D_i^{AS} \sum_{n=1, n \neq j} T_{in} \langle g_i^n(\psi_{ij}^{SE}) \rangle \\ &+ D_j^{AE} \sum_{n=1, n \neq i} T_{jn} \langle g_j^n(\psi_{ij}^{SE}) \rangle - D_j^{AE} \sigma_j \langle \psi_{ij}^{SE} \rangle.\end{aligned}$$

That is,

$$\langle \psi_{ij}^{SE} \rangle = - \sum_{n=1, n \neq j}^N T_{in} \langle \psi_{n,ij}^{ISE} \rangle + \sum_{n=1, n \neq i}^N T_{jn} \langle \psi_{ij,n}^{SSI} \rangle - \sigma_j \langle \psi_{ij}^{SE} \rangle,$$

where the first term corresponds to  $k = 1$  and the remaining two terms correspond to  $k = 2$ . This is also a new term.

## 2.5 The general closure relation: The main result

The symmetry we observed in the star graph is more general and so we want to show that the closure relation (1.22) is exact for tree graph. We specify in general that these closure relations are composed of two pairs of motif states  $(\psi_W^A, \psi_X^B)$  and  $(\psi_Y^C, \psi_Z^D)$  and that the closure is exact if

$$\alpha = \langle \psi_W^A \rangle \langle \psi_X^B \rangle - \langle \psi_Y^C \rangle \langle \psi_Z^D \rangle = 0 \quad \text{for all } t \geq 0.$$

As defined in [155], if  $\psi_W^A$  is an  $r$ -state motif and  $\psi_X^B$  is a  $q$ -state motif, then the order of the pair  $(\psi_W^A, \psi_X^B)$  is defined as  $r + q$ . Also, if  $(\psi_W^A, \psi_X^B)$  and  $(\psi_Y^C, \psi_Z^D)$  are compatible pairs as given by definition 3.1.1, then their order is equal. For example  $\alpha_{15}(t)$  of equation (2.13) has the compatible pairs  $\langle I_2 S_4 \rangle \langle E_1 I_2 E_3 S_4 \rangle$  and  $\langle E_1 I_2 S_4 \rangle \langle I_2 E_3 S_4 \rangle$  which are of equal order of 6.

**LEMMA 2.5.0.** Let  $(\psi_W^A, \psi_X^B)$  and  $(\psi_Y^C, \psi_Z^D)$  be compatible pairs of order  $R$  and  $\psi_W^A, \psi_X^B, \psi_Y^C$  and  $\psi_Z^D$  be in the extended state space. Let

$$\alpha_0 = \langle \psi_W^A \rangle \langle \psi_X^B \rangle - \langle \psi_Y^C \rangle \langle \psi_Z^D \rangle$$

Then

$$\dot{\alpha}_0 = \sum_{p=1}^m c_p \alpha_p + \sum_{r=1}^n \mu_r \alpha_r + c_0 \alpha_0, \quad (2.24)$$

where each  $\alpha_p$  can be expressed as:

$$\alpha_p = \langle \psi_{W^*}^{A^*} \rangle \langle \psi_{X^*}^{B^*} \rangle - \langle \psi_{Y^*}^{C^*} \rangle \langle \psi_{Z^*}^{D^*} \rangle,$$

where  $\psi_{W^*}^{A^*}$ ,  $\psi_{X^*}^{B^*}$ ,  $\psi_{Y^*}^{C^*}$  and  $\psi_{Z^*}^{D^*}$  are compatible pairs of order  $(R + 1)$  and each  $\alpha_r$  can be expressed as:

$$\alpha_r = \langle \psi_W^{A'} \rangle \langle \psi_X^{B'} \rangle - \langle \psi_Y^{C'} \rangle \langle \psi_Z^{D'} \rangle,$$

where  $\psi_W^{A'}$ ,  $\psi_X^{B'}$ ,  $\psi_Y^{C'}$  and  $\psi_Z^{D'}$  are compatible pairs of order  $R$ , here,  $c_0$ ,  $\mu_r$  and  $c_p$  are constants and  $m$  and  $n$  are integers denoting the number of terms in the summation.

We remark that (2.24) contains an extra term

$$\sum_{r=1}^n \mu_r \alpha_r,$$

when compared with its counterpart for the *SIR* model of [155] and it is also a general statement of equations (2.10) to (2.19) .

*PROOF.* The derivative of  $\alpha_0$  is

$$\dot{\alpha}_0 = \langle \dot{\psi}_W^A \rangle \langle \psi_X^B \rangle + \langle \psi_W^A \rangle \langle \dot{\psi}_X^B \rangle - \langle \dot{\psi}_Y^C \rangle \langle \psi_Z^D \rangle - \langle \psi_Y^C \rangle \langle \dot{\psi}_Z^D \rangle. \quad (2.25)$$

We shall consider the terms associated with removal, exposed, transmission terms of order  $R$  and  $R + 1$  separately.

Considering the removal term of (2.21) in (2.25) following the approach of [155], the removal term of order  $R$  in (2.21) is

$$- \sum_{k=1}^r D_k^{AI} \gamma_{W_k} \langle \psi_W^A \rangle.$$

Putting this in (2.25) we have

$$\begin{aligned} \dot{\alpha}_0 = & - \sum_{k_1} D_{k_1}^{AI} \gamma_{W_{k_1}} \langle \psi_W^A \rangle \langle \psi_X^B \rangle - \sum_{k_2} D_{k_2}^{BI} \gamma_{X_{k_2}} \langle \psi_X^B \rangle \langle \psi_W^A \rangle \\ & + \sum_{k_3} D_{k_3}^{CI} \gamma_{Y_{k_3}} \langle \psi_Y^C \rangle \langle \psi_Z^D \rangle + \sum_{k_4} D_{k_4}^{DI} \gamma_{Z_{k_4}} \langle \psi_Z^D \rangle \langle \psi_Y^C \rangle. \end{aligned}$$

The sums over  $k_1$ ,  $k_2$ ,  $k_3$  and  $k_4$  are over all nodes in the motifs  $\psi_W$ ,  $\psi_X$ ,  $\psi_Y$  and  $\psi_Z$  respectively. These sum total of these constants in the each pair of the

CPs are equal because they are equally matched, satisfying conditions(i) and (ii) of definition 3.1.1, so

$$\begin{aligned}
& \sum_{k_1} D_{k_1}^{AI} \gamma_{W_{k_1}} - \sum_{k_2} D_{k_2}^{BI} \gamma_{X_{k_2}} \\
&= \sum_{k_3} D_{k_3}^{CI} \gamma_{Y_{k_3}} + \sum_{k_4} D_{k_4}^{DI} \gamma_{Z_{k_4}} \\
&= \text{a constant, say } v.
\end{aligned}$$

That is

$$\dot{\alpha}_0 = -v[\langle \psi_W^A \rangle \langle \psi_X^B \rangle - \langle \psi_Y^C \rangle \langle \psi_Z^D \rangle] = -v\alpha_0,$$

with  $-v$  contributing to  $c_0$  of (2.24).

Considering the infection term of order  $R$  in (2.25), the infection term from (2.21) is

$$\sum_{k=1}^r -D_k^{AS} \sum_l T_{W_k W_l} D_l^{AI} \langle \psi_W^A \rangle.$$

Putting this into the RHS of (2.25) gives

$$\begin{aligned}
& - \sum_{k_1} D_{k_1}^{AS} \sum_{l_1} T_{W_{k_1} W_{l_1}} D_{l_1}^{AI} \langle \psi_W^A \rangle \langle \psi_X^B \rangle \\
& - \sum_{k_2} D_{k_2}^{BS} \sum_{l_2} T_{X_{k_2} X_{l_2}} D_{l_2}^{BI} \langle \psi_X^B \rangle \langle \psi_W^A \rangle \\
& + \sum_{k_3} D_{k_3}^{CS} \sum_{l_3} T_{Y_{k_3} Y_{l_3}} D_{l_3}^{CI} \langle \psi_Y^C \rangle \langle \psi_Z^D \rangle \\
& + \sum_{k_4} D_{k_4}^{DS} \sum_{l_4} T_{Z_{k_4} Z_{l_4}} D_{l_4}^{DI} \langle \psi_Z^D \rangle \langle \psi_Y^C \rangle
\end{aligned}$$

The sums over  $k_1, k_2, k_3, k_4, l_1, l_2, l_3$  and  $l_4$  are over all nodes in the relevant motifs. These CPs satisfy condition (iii) and (iv) of definition 3.1.1 and so

$$\begin{aligned}
\varpi &= \sum_{k_1} D_{k_1}^{AS} \sum_{l_1} T_{W_{k_1} W_{l_1}} D_{l_1}^{AI} + \sum_{k_2} D_{k_2}^{BS} \sum_{l_2} T_{X_{k_2} X_{l_2}} D_{l_2}^{BI} \\
&= \sum_{k_3} D_{k_3}^{CS} \sum_{l_3} T_{Y_{k_3} Y_{l_3}} D_{l_3}^{CI} + \sum_{k_4} D_{k_4}^{DS} \sum_{l_4} T_{Z_{k_4} Z_{l_4}} D_{l_4}^{DI} \\
&= \text{a constant, say } \varpi \text{ for same reason as above.}
\end{aligned}$$

That is;

$$-\varpi[\langle \psi_W^A \rangle \langle \psi_X^B \rangle - \langle \psi_Y^C \rangle \langle \psi_Z^D \rangle] = -\varpi\alpha_0,$$

with  $-\varpi$  also contributing to  $c_0$  of (2.24).

Considering the exposed term of order  $R$  in (2.25), this term consists of two

parts. one part is

$$\sum_{k=1}^r D_k^{AE} \sigma_{W_k} \langle \psi_W^A \rangle, \quad (2.26)$$

which is the rate of leaving the exposed state, and the other part

$$\sum_{k=1}^r D_k^{AI} \sigma_{W_k} h_{W_k} \langle \psi_W^A \rangle, \quad (2.27)$$

which is the rate of becoming infectious.

Putting (2.26) into the RHS of (2.25) gives

$$\begin{aligned} & - \sum_{k_1} D_{k_1}^{AE} \sigma_{W_{k_1}} \langle \psi_W^A \rangle \langle \psi_X^B \rangle - \sum_{k_2} D_{k_2}^{BE} \sigma_{X_{k_2}} \langle \psi_X^B \rangle \langle \psi_W^A \rangle \\ & + \sum_{k_3} D_{k_3}^{CE} \sigma_{Y_{k_3}} \langle \psi_Y^C \rangle \langle \psi_Z^D \rangle + \sum_{k_4} D_{k_4}^{DE} \sigma_{Z_{k_4}} \langle \psi_Z^D \rangle \langle \psi_Y^C \rangle. \end{aligned}$$

The sums over  $k_1$ ,  $k_2$ ,  $k_3$  and  $k_4$  are over all nodes in the relevant motifs. These CPs satisfy conditions CP(v) and CP(vi), so

$$\begin{aligned} v_\rho &= \sum_{k_1} D_{k_1}^{AE} \sigma_{W_{k_1}} + \sum_{k_2} D_{k_2}^{BE} \sigma_{X_{k_2}} \\ &= \sum_{k_3} D_{k_3}^{CE} \sigma_{Y_{k_3}} + \sum_{k_4} D_{k_4}^{DE} \sigma_{Z_{k_4}} \\ &= \text{a constant for same reason as above.} \end{aligned}$$

That is;

$$-v_\rho [\langle \psi_W^A \rangle \langle \psi_X^B \rangle - \langle \psi_Y^C \rangle \langle \psi_Z^D \rangle] = -v_\rho \alpha_r,$$

with  $-v_\rho$  also contributing to  $\mu_r$  of (2.24).

Putting (2.30) in the RHS of (2.25) gives

$$\begin{aligned} & \sum_{k_1} D_{k_1}^{AI} \sigma_{W_{k_1}} h_{W_{k_1}} \langle \psi_W^A \rangle \langle \psi_X^B \rangle + \sum_{k_2} D_{k_2}^{BI} \sigma_{X_{k_2}} h_{X_{k_2}} \langle \psi_X^B \rangle \langle \psi_W^A \rangle \\ & - \sum_{k_3} D_{k_3}^{CI} \sigma_{Y_{k_3}} h_{Y_{k_3}} \langle \psi_Y^C \rangle \langle \psi_Z^D \rangle - \sum_{k_4} D_{k_4}^{DI} \sigma_{Z_{k_4}} h_{Z_{k_4}} \langle \psi_Z^D \rangle \langle \psi_Y^C \rangle. \end{aligned}$$

**Note:** Here,  $h_i$  transforms node **I** to **E** in each pair of the CPs without increasing the motif of the CPs. The CPs thus satisfy conditions (iii) and (iv) of definition 3.1.1. The sum is over all nodes in the relevant motifs. We have that

$$-v_{\rho^*} [\langle \psi_W^A \rangle \langle \psi_X^B \rangle - \langle \psi_Y^C \rangle \langle \psi_Z^D \rangle] = -v_{\rho^*} \alpha_r,$$

with  $-v_{\rho^*}^*$  also contributing to  $\mu_r$  of (2.24).

The transmission terms with motifs of order  $R + 1$  in (2.21) which corresponds to the term  $\langle \psi_W^A \rangle \langle \psi_X^B \rangle$  in the derivative of  $\alpha_0$  in (2.25) is

$$\sum_{k_1}^r \left[ (D_{k_1}^{AE} - D_{k_1}^{AS}) \sum_{n=1, n \notin W} T_{W_{k_1 n}} \langle g_{W_{k_1}}^n(\psi_W^A) \rangle \right], \quad (2.28)$$

which can be considered for when a node is either susceptible,  $S$ , or exposed,  $E$ .

It is necessary to show that there is a unique one-to-one pairing of each term of order  $R + 1$  in all the four terms on the RHS of (2.25). To show this, we take an element from the sum and choose a node  $W_k$ ,  $k \in W = \{1, 2, \dots, r\}$  and consider a neighbouring node  $n$  which is connected to  $W_k$  but not in  $W$ , that is  $n \in N_{W_k}$ ,  $n \notin W$ . As earlier mentioned, two cases are possible from the sum either  $A_k = S$  or  $A_k = E$ .

*case 1:* Let  $A_k = S$  then (2.28) reduces to

$$-D_k^{AS} T_{W_{kn}} \langle g_{W_k}^n(\psi_W^A) \rangle = -T_{W_{kn}} \langle g_{W_k}^n(\psi_W^A) \rangle. \quad (2.29)$$

We desire to show that the term (2.28) can be matched with a term in  $\langle \psi_Y^C \rangle \langle \psi_Z^D \rangle$  or in  $\langle \psi_Y^C \rangle \langle \psi_Z^D \rangle$  to form a CP in such a way that  $-T_{W_{kn}}$  contributes to  $c_\rho$ .

Note that if we take an element in each of the relevant subsystem which is responsible for transmission term of order  $R + 1$  in (2.25) and input them into (2.24) we have the expression below for  $\dot{\alpha}_0$

$$\begin{aligned} \dot{\alpha}_0 = & -T_{W_{kn}} \langle g_{W_k}^n(\psi_W^A) \rangle \langle \psi_X^B \rangle - T_{X_{xn}} \langle g_{X_x}^n(\psi_X^B) \rangle \langle \psi_W^A \rangle \\ & + T_{Y_{yn}} \langle g_{Y_y}^n(\psi_Y^C) \rangle \langle \psi_X^B \rangle + T_{Z_{zn}} \langle g_{Z_z}^n(\psi_Z^D) \rangle \langle \psi_Y^C \rangle. \end{aligned} \quad (2.30)$$

Since  $A_k = S$ , conditions (i) and (ii) of definition 3.1.1 imply we can assume that  $A_k = E \in Y$ . In other word  $\exists y = k \in Y$  such that  $C_y = S$ . The neighbour node  $n$  of  $k$  which is considered not in  $W$  could either be or not in the subsystem  $\psi_Y^C$ , giving rise to two possible subcases.

- *Subcase 1:* Suppose the external node  $n$  is not  $Y$ , then by CP(i) node  $n$  is either in or not in  $Z$ . If  $n$  is in  $Z$ , CP(i) implies  $n$  is in  $X$  since  $n$  is not in  $W$ . Therefore the necessary corresponding term that pair with the first term of

(2.30) is

$$+T_{Y_{yn}} \langle g_{Y_y}^n(\psi_Y^C) \rangle \langle \psi_Z^D \rangle,$$

with  $T_{Y_{yn}}$  also contributing to  $c_p$ .

- *Subcase 2:* Suppose the external node  $n$  is in  $Y$ . Then the link  $n \rightarrow Y_y$  must be either an  $S$ - $S$  or  $I$ - $S$  link and it is the same edge in  $B$  by CP (iii) to (vii) because  $n$  is not in  $W$ , that is,  $\exists z = k \in Z$  such that  $D_z = S$ . The corresponding compatible term with (2.29) is therefore  $+T_{Z_{zn}} \langle \psi_Y^C \rangle g_{Z_z}^n(\psi_Z^D)$  where  $I_n$  is attached to  $Z_z$  in  $D$  by the generating function. That is

$$\alpha_p = -T_{W_{kn}} \langle g_{W_k}^n(\psi_W^A) \rangle + T_{Z_{zn}} g_{Z_z}^n(\psi_Z^D) \langle \psi_Y^C \rangle,$$

from where we can see  $T_{Z_{zn}}$  also contributing to  $c_p$ .

*case 2:* Let  $A_k = E$ , a term in (2.28) is reduced to

$$D_k^{AE} T_{W_{kn}} \langle g_{W_k}^n(\psi_W^A) \rangle = -T_{W_{kn}} \langle g_{W_k}^n(\psi_W^A) \rangle. \quad (2.31)$$

We have being able to show from above the transmission term of  $R+1$  motif when  $A_k$  is susceptible. The process of obtaining a corresponding compatible term to when  $A_k = E$  follows from above with node  $A_k = E$  being first transformed to  $S$ .

This process is different from that of the  $SIR$  case of [148] where an  $S$ - $I$  edge is replaced with a triple  $S$ - $S$ - $I$  with the  $I$  node being replaced by an  $S$  node with a connection to an external  $I$  node. Here, in an  $S$ - $E$  edge where  $A_k = E$ , node  $E$  is first changed to  $S$  before a neighbouring external node  $I_n$  is attached (see definition 2.4.2).

It is essential to state that equation (2.24) can be written in Matrix form

$$\dot{\alpha}^R(t) = A\alpha^{R+1}(t) + B\alpha^R(t). \quad (2.32)$$

where  $\alpha(t)$  is a vector of all the CPs and  $A$  and  $B$  are the respective coefficient matrices of CPs of order  $R+1$  and order  $R$ .

We give an example of  $B\alpha^R(t)$  from equations (2.10) to (2.19) for CPs of order 4 as Matrix  $A$  for CPs of order 5 from these equations is too big to be included. For CPs of order 4, we have from equations (2.10) to (2.19) that matrix  $B$  is multiplied by the column vector  $[\alpha_1(t) \quad \alpha_3(t) \quad \alpha_5(t) \quad \alpha_{18}(t)]^T$  where  $\alpha_{18}(t) = \langle \psi_4^S \rangle \langle \psi_{124}^{EES} \rangle - \langle \psi_{14}^{ES} \rangle \langle \psi_{24}^{ES} \rangle$  and



$$B = \begin{bmatrix} -2k_1 & -\sigma & -\sigma & 0 \\ 0 & -k_2 & 0 & \sigma \\ 0 & 0 & -k_2 & -\sigma \\ 0 & 0 & 0 & -2\sigma \end{bmatrix} \quad (2.33)$$

where  $k_1 = \tau + \gamma$  and  $k_2 = \tau + \gamma + \sigma$ .

**LEMMA 2.5.1:** Let us suppose that that an epidemic process is initiated in a definite state. In other word, there is a system state A with an initial probability 1. That is  $\exists A \in \{I, E, S\}^N$  such that  $\langle \psi_{1,2,\dots,N}^A \rangle = 1$ . If  $(\langle \psi_W^A \rangle, \langle \psi_X^B \rangle)$  and  $(\langle \psi_Y^C \rangle, \langle \psi_Z^D \rangle)$  are CPs, then for any network

$$\langle \psi_W^A \rangle \langle \psi_X^B \rangle - \langle \psi_Y^C \rangle \langle \psi_Z^D \rangle = 0 \quad \text{at } t = 0.$$

*PROOF:* The proof of this lemma is established in lemma 3.2 of [155] for the *SIR* model on graphs and by extension it applies to our study.

**THEOREM 2.5.2:** Let us assume that the contact network we consider represents a tree and epidemic is initiated in a pure state, and also  $(\psi_W^A, \psi_X^B)$  and  $(\psi_Y^C, \psi_Z^D)$  be compatible pairs. Then

$$\langle \psi_W^A \rangle \langle \psi_X^B \rangle - \langle \psi_Y^C \rangle \langle \psi_Z^D \rangle = 0 \quad \text{for all } t \geq 0.$$

*PROOF:* We prove this theorem by induction in line with the order of the closure following the process we used to arrive at equations (2.10) to (2.19) for the star graph.

If each of  $\langle \psi_W^A \rangle$ ,  $\langle \psi_X^B \rangle$ ,  $\langle \psi_Y^C \rangle$  and  $\langle \psi_Z^D \rangle$  is of order  $N$ , forming a CP of order  $2N$ , which is of course the highest possible order of any CPs in this study, then (2.32) which is equivalent to (2.24) becomes

$$\dot{\alpha}^R(t) = B\alpha^R(t), \quad (2.34)$$

since there can not be term(s) of order  $2N + 1$ .

The general solution of (2.34) is

$$\alpha_i^{(R)}(t) = \sum_{i=1}^n C_i e^{\lambda_i t} V_i, \quad (2.35)$$

where  $\lambda_i$  and  $V_i$  are the corresponding eigenvalues and eigenvectors of matrix  $B$ .

By lemma 2.5.1, the initial condition is  $\alpha_i^{(R)}(0) = 0$  for  $i = 1, 2, \dots, n$ .

From (2.35) the initial conditions  $\alpha_i(0) = 0$  implies

$$C_1V_1 + C_2V_2 + \dots + C_nV_n = 0.$$

That is,  $C_1 = C_2 = \dots = C_n = 0$  because  $V_i \neq 0$  for all  $i$  and assuming matrix  $B$  is invertible. Therefore,  $\alpha^R(t) = 0$  for all  $t \geq 0$ . Hence theorem 2.5.2 is true for  $2N$ .

If theorem 2.5.2 is true for  $R + 1$ , then we have from (2.32) that

$$\dot{\alpha}^R(t) = B\alpha^R(t).$$

This is the same form as equation (2.34). So, by the same argument, theorem 2.5.2 is true for  $R$ . Since is true for  $2N$ , then by induction, it is true for  $R \leq 2N$ .

## Conclusion

The study has developed an individual-level **SEIR** pairwise epidemic model and examined the validity of the exactness of a second order moment closure on the model on contact networks of a finite size, particularly on tree networks. This study is in line with similar result for the *SIR* epidemic model on tree networks where it was shown that the *SIR* epidemic dynamics of infection on some unclustered network can also be exactly defined by a pair-based model under some special initial conditions[154, 155]. The extension of this process to prove the validity of the second order moment closure for our model on trees is more complex because there are more compatible pairs to be validated. The extra compatible pairs are as a result of a new infectious status **E**, in our model.

The *S-E* link (as against the *S-I* link in the *SIR* model) is involved in the generation of higher motifs of the infection transmission process. The single equation (2.21) from which the rate of change of the probability of all motifs can be derived is more complex and contain extra terms compared similar result obtained from earlier of the *SIR* model. More steps are involved to validate the generation of a higher motif of the infection transmission process when compared with the process in [155].

We believe it is worthwhile to extend such earlier study of the *SIR* epidemic model to **SEIR** because the two models behave differently at invasion with the presence of an exposed class slowing down the infection dynamic of some infections, especially when there is a silent spread. The **SEIR** model is one of the simplest models commonly used for simulating measles. We note that, in the limit of the

incubation period going to zero, the **SEIR** model is equivalent to the *SIR* model.

Another approach, developed after this study is the network partitioning method[85, 156]. Using this method to obtain exact results for the **SEIR** model should be straightforward. However, the method presented here gives a complementary perspective with its own utility as seen in the next chapter.

# Chapter 3

## Approximating the Global Error for an *SIR* Epidemic model on Ring networks

### 3.1 Introduction

The main reason for modelling epidemics is to understand the way they spread and then give a sound basis for prescribing efficient control measures. Although the traditional homogeneous models describe the essential features of epidemic, helping us to extrapolate infection characteristics from the individual viewpoint to population level, studies [12, 13] show that, for most models, such mean field equations underestimate the true infection threshold, meaning the mean-field systems may hide away important details of infection process.

Epidemic dynamics are dominated by complementary interactions between discrete individuals and these dynamics are usually well captured by correlation equations with assumptions that can be experimentally investigated, so an improvement on the mean-field dynamics on network is achieved with the pairwise models to incorporate these interacting tendencies of constituent units. In this type of model the dynamics of individuals are usually expressed in terms of the number of pairs and that of the pairs expressed in terms of triples, and so on. Generally  $n$ th moment depends on  $(n + 1)$ th moment and this usually lead to unclosed system.

For numerical evaluation to be possible and in other to achieve analysis by standard process, these systems must be closed at some level. This challenge led to various closure methods [40, 49, 84, 163] and the evaluation and validity of these closures are usually done by numerically comparing the stochastic and deterministic dynamics which incorporate them.

Although there could be noticeable variations in higher order approximation when details of higher order interacting units are known [135], studies [40,49,52, 82] showed that the second-order approximation which closes the system by approximating triple as product of pairs gives remarkable result to the true dynamic of infection. An example of infection which is dominated by pairwise interaction and which is well captured by pair approximation model is the dynamics of sexually transmitted diseases (STDs).

Different network means different accuracy of a defined closure because the network structure has a profound effect on invasion and the establishment of infection. The exactness of the second order moment closure for the **SIR** model on tree network is one of many evidence of this [154, 155]. The presence of cycles of higher order, of which triangle is the smallest, is the major problem with network-based epidemic models which are based on second order moment closure. Cycles make it difficult to match a low - dimensional approximate model with its corresponding stochastic model.

In order to explain the relationship between the underlying topology of mixing networks and the feasibility of the second order moment closure, Kiss et al [85] considered the Markovian **SIR** epidemic dynamic for some simple networks with lower order cycles-triangles and showed that the closures where the triangles are kept intact are exact. Although progress have been made towards finding solution to this challenge [15, 55, 100], pairwise models are generally less accurate when connections are strongly localized, that is when the presence of lower order cycles is relatively high [49, 81].

Pair approximation models on contact networks of varying structures have been investigated. Particularly, the effects of triangles in networks on the propagation of infection has greatly being exploited [55, 73, 81, 100]. The common approach has been to investigate infection features from simulations but not providing a particular analytic bound for when the closures work. Our motivation for the present study comes from the existing gap between simulations and possible analytical bound for when a second order moment closure works.

Our interest in this study also stem from the hindsight that the underlying topology determines the type of closure that are feasible. Earlier study [155] proved that the second order moment closure of the **SIR** epidemic model is exact on **tree networks** under certain conditions. Following the process of earlier study, we want to try to understand and quantify, if possible, the error in second order

moment closure of **SIR** epidemic dynamics on time-independent **cycle** with a constant transmission rate across all links and a constant recovery rate for an infected individual. We simply want to investigate if the error from the deviation from being exact of a second order moment closure of **SIR** epidemic model on networks of contacts containing a single cycle can be quantified and then write a general representation for cycle of any size.

### 3.2 Statement of the Problem and Procedure

We consider an **SIR** epidemic model on ring network of  $N$  individuals whose infections status are as described in section (1.7.3). The transmission and recovery are both Poisson processes with respective constant rates  $\tau$  and  $\gamma$  and system (1.21) holds for any pair of nodes  $i$  and  $j$  and transmission matrix  $T$ .

We begin our investigation with the smallest loop, the triangle and assume an initial pure system state-  $S_a S_b I_c$  (see figure (3.1)). We then introduce a susceptible node at a time, creating an additional  $S$ - $S$  link to define a sequence of cycles:- square, pentagon and hexagon respectively. We maintain node  $c$  as the only initially infected node in all the networks and apply the second order moment closure (1.22) to the two possible triples that appear in the hierarchy of moment equations generated from (1.21) for each network.

To test the validity of the moment closure, we follow the usual approach of the previous chapter by differentiating the respective  $\alpha^{SSI}(t)$  and  $\alpha^{ISI}(t)$  in each network and observe if the deviation of the closure for these triples from being exact follows an obvious pattern from which a general representation can be written for a ring network.

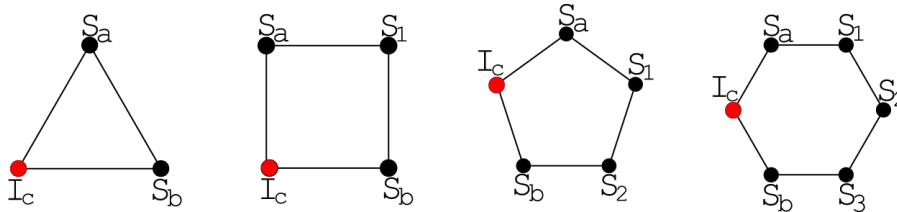


Figure 3.1: Cycles of three, four, five and six nodes respectively where the node marked as red is the only initially infectious node in the networks.

The labelling of nodes with a specific node  $c$  been marked as the only initially infectious node in each network is to simplify our notation and ease representation and has no effect whatsoever on the dynamic of infection. We consider closure for two possible forms of triples, the  $S$ - $S$ - $I$  and the  $I$ - $S$ - $I$ .

### 3.3 The Triangular Network

In this section we shall consider the second order moment closure for both the  $S$ - $S$ - $I$  and  $I$ - $S$ - $I$  for the triangular network of figure (3.1) whose transmission matrix is

$$T = \begin{bmatrix} 0 & \tau & \tau \\ \tau & 0 & \tau \\ \tau & \tau & 0 \end{bmatrix}.$$

The single node equations from (1.21) for this network are

$$\begin{aligned} \langle \dot{S}_a \rangle &= -\tau \langle S_a I_b \rangle - \tau \langle S_a I_c \rangle, \\ \langle \dot{S}_b \rangle &= -\tau \langle I_a S_b \rangle - \tau \langle S_b I_c \rangle, \\ \langle \dot{S}_c \rangle &= -\tau \langle I_a S_c \rangle - \tau \langle I_b S_c \rangle, \\ \langle \dot{I}_a \rangle &= \tau \langle S_a I_b \rangle + \tau \langle S_a I_c \rangle - \gamma \langle I_a \rangle, \\ \langle \dot{I}_b \rangle &= \tau \langle I_a S_b \rangle + \tau \langle S_b I_c \rangle - \gamma \langle I_b \rangle, \\ \langle \dot{I}_c \rangle &= \tau \langle I_a S_c \rangle + \tau \langle I_b S_c \rangle - \gamma \langle I_c \rangle. \end{aligned} \tag{3.1}$$

The following are for pairs

$$\begin{aligned} \langle \dot{S}_a I_b \rangle &= \tau \langle S_a S_b I_c \rangle - \tau \langle S_a I_b I_c \rangle - (\tau + \gamma) \langle S_a I_b \rangle, \\ \langle \dot{I}_a S_b \rangle &= \tau \langle S_a S_b I_c \rangle - \tau \langle I_a S_b I_c \rangle - (\tau + \gamma) \langle I_a S_b \rangle, \\ \langle \dot{S}_a I_c \rangle &= \tau \langle S_a I_b S_c \rangle - \tau \langle S_a I_b I_c \rangle - (\tau + \gamma) \langle S_a I_c \rangle, \\ \langle \dot{I}_a S_c \rangle &= \tau \langle S_a I_b S_c \rangle - \tau \langle I_a I_b S_c \rangle - (\tau + \gamma) \langle I_a S_c \rangle, \\ \langle \dot{S}_b I_c \rangle &= \tau \langle I_a S_b S_c \rangle - \tau \langle I_a S_b I_c \rangle - (\tau + \gamma) \langle S_b I_c \rangle, \\ \langle \dot{I}_b S_c \rangle &= \tau \langle I_a S_b S_c \rangle - \tau \langle I_a I_b S_c \rangle - (\tau + \gamma) \langle I_b S_c \rangle, \\ \langle \dot{S}_a S_b \rangle &= -2\tau \langle S_a S_b I_c \rangle, \\ \langle \dot{S}_a S_c \rangle &= -2\tau \langle S_a I_b S_c \rangle, \\ \langle \dot{S}_b S_c \rangle &= -2\tau \langle I_a S_b S_c \rangle. \end{aligned} \tag{3.2}$$

Finally for the triples we have;

$$\begin{aligned}
\langle I_a \dot{S}_b S_c \rangle &= -(\tau + \gamma) \langle I_a S_b S_c \rangle, \\
\langle S_a \dot{I}_b S_c \rangle &= -(\tau + \gamma) \langle S_a I_b S_c \rangle, \\
\langle S_a \dot{S}_b I_c \rangle &= -(\tau + \gamma) \langle S_a S_b I_c \rangle, \\
\langle I_a \dot{S}_b I_c \rangle &= -2(\tau + \gamma) \langle I_a S_b I_c \rangle + \tau \langle I_a S_b S_c \rangle + \tau \langle S_a S_b I_c \rangle, \\
\langle S_a \dot{I}_b I_c \rangle &= -2(\tau + \gamma) \langle S_a I_b I_c \rangle + \tau \langle S_a I_b S_c \rangle + \tau \langle S_a S_b I_c \rangle, \\
\langle I_a \dot{I}_b S_c \rangle &= -2(\tau + \gamma) \langle I_a I_b S_c \rangle + \tau \langle I_a S_b S_c \rangle + \tau \langle S_a I_b S_c \rangle.
\end{aligned} \tag{3.3}$$

### 3.3.1 Closure for $S$ - $S$ - $I$

We shall first apply the closure relation to  $\langle S_a S_b I_c \rangle$  and examine the accuracy of

$$\langle S_a S_b I_c \rangle \approx \frac{\langle S_a S_b \rangle \langle S_b I_c \rangle}{\langle S_b \rangle}.$$

We write  $\alpha^{SSI}(t)$  as

$$\alpha^{SSI}(t) = \langle S_b \rangle \langle S_a S_b I_c \rangle - \langle S_a S_b \rangle \langle S_b I_c \rangle. \tag{3.4}$$

We take the derivative of  $\alpha^{SSI}(t)$  with respect to time and substitute the terms in from (3.1)-(3.3) as follows

$$\begin{aligned}
\dot{\alpha}^{SSI}(t) &= \langle \dot{S}_b \rangle \langle S_a S_b I_c \rangle + \langle S_b \rangle \langle S_a \dot{S}_b I_c \rangle - \langle \dot{S}_a S_b \rangle \langle S_b I_c \rangle - \langle S_a S_b \rangle \langle \dot{S}_b I_c \rangle \\
&= [-\tau \langle I_a S_b \rangle - \tau \langle S_b I_c \rangle] \times \langle S_a S_b I_c \rangle + \langle S_b \rangle \times [-(2\tau + \gamma) \langle S_a S_b I_c \rangle] \\
&\quad - [-2\tau \langle S_a S_b I_c \rangle] \times \langle S_b I_c \rangle \\
&\quad - \langle S_a S_b \rangle \times [\tau \langle I_a S_b S_c \rangle - \tau \langle I_a S_b I_c \rangle - (\tau + \gamma) \langle S_b I_c \rangle].
\end{aligned} \tag{3.5}$$

When expanded we have

$$\begin{aligned}
\dot{\alpha}^{SSI}(t) &= -\tau \langle I_a S_b \rangle \langle S_a S_b I_c \rangle - \tau \langle S_b I_c \rangle \langle S_a S_b I_c \rangle \\
&\quad - (2\tau + \gamma) \langle S_b \rangle \langle S_a S_b I_c \rangle + 2\tau \langle S_a S_b I_c \rangle \langle S_b I_c \rangle \\
&\quad - \tau \langle S_a S_b \rangle \langle I_a S_b S_c \rangle + \tau \langle S_a S_b \rangle \langle I_a S_b I_c \rangle \\
&\quad + (\tau + \gamma) \langle S_a S_b \rangle \langle S_b I_c \rangle.
\end{aligned} \tag{3.6}$$



Rewriting this in order to collect compatible pairs, we have

$$\begin{aligned}
\dot{\alpha}^{SSI}(t) = & -(\tau + \gamma)\langle S_b \rangle \langle S_a S_b I_c \rangle + (\tau + \gamma)\langle S_a S_b \rangle \langle S_b I_c \rangle \\
& + \tau \langle S_b I_c \rangle \langle S_a S_b I_c \rangle - \tau \langle S_b \rangle \langle S_a S_b I_c \rangle \\
& - \tau \langle I_a S_b \rangle \langle S_a S_b I_c \rangle - \tau \langle S_a S_b \rangle \langle I_a S_b S_c \rangle \\
& + \tau \langle S_a S_b \rangle \langle I_a S_b I_c \rangle.
\end{aligned}$$

Then we finally have

$$\begin{aligned}
\dot{\alpha}^{SSI}(t) = & -(\tau + \gamma)\alpha^{SSI}(t) + \tau\beta_1(t) - \tau\beta_2(t) \\
& - \tau\beta_3(t) - \tau\beta_4(t) + \tau\beta_5(t).
\end{aligned} \tag{3.7}$$

where

$$\begin{aligned}
\beta_1 &= \langle S_b I_c \rangle \langle S_a S_b I_c \rangle, & \beta_2 &= \langle S_b \rangle \langle S_a S_b I_c \rangle, \\
\beta_3 &= \langle I_a S_b \rangle \langle S_a S_b I_c \rangle, & \beta_4 &= \langle S_a S_b \rangle \langle I_a S_b S_c \rangle, \\
\beta_5 &= \langle S_a S_b \rangle \langle I_a S_b I_c \rangle.
\end{aligned} \tag{3.8}$$

Fundamentally and from the previous chapter, the closure is exact if and only if  $\dot{\alpha}^{SSI}(t)$  is expressed solely in terms of ‘compatible pairs’ and by so doing the solution for  $\alpha^{SSI}(t)$  must be zero at all time given a pure initial state. Of course  $\alpha^{SSI}(0) = 0$  but  $\dot{\alpha}^{SSI}(t)$  in equation (3.7) contains some terms which are not all initially zero.

The initial configuration of the triangular network means that  $\beta_1(0) = 1$ ,  $\beta_2(0) = 1$ ,  $\beta_3(0) = 0$ ,  $\beta_4(0) = 0$  and  $\beta_5(0) = 0$  and this might lead us to conclude that their total effect cancels out by merely looking at equation (3.7). Such assumption will be erroneous because the behaviour of the  $\beta$ s is not that obvious so the solution of  $\alpha^{SSI}(t)$  cannot necessarily be zero at all time.

If the solution for  $\alpha^{SSI}(t) \neq 0 \forall t \geq 0$ , then we want to conclude these  $\beta$ s represent cause of the deviation of the approximation from exactness and we refer to them here as ‘**error terms**’ in  $\alpha(t)$ .

By simple differentiation and substitution where necessary, we also have that;

$$\begin{aligned}
\dot{\beta}_1(t) &= -(3\tau + 2\gamma)\beta_1(t) + \tau\beta_6(t) - \tau\beta_7(t), \\
\dot{\beta}_2(t) &= -(2\tau + \gamma)\beta_2(t) - \tau\beta_1(t) - \tau\beta_3(t), \\
\dot{\beta}_3(t) &= -(3\tau + 2\gamma)\beta_3(t) - \tau\beta_7(t) + \tau\beta_8(t), \\
\dot{\beta}_4(t) &= -(2\tau + \gamma)\beta_4(t) - 2\tau\beta_6(t), \\
\dot{\beta}_5(t) &= -2(\tau + \gamma)\beta_5(t) + \tau\beta_4(t) - 2\tau\beta_7(t) + \tau\beta_9(t),
\end{aligned} \tag{3.9}$$

$$\begin{aligned}
\dot{\beta}_6(t) &= -2(2\tau + \gamma)\beta_6(t), \\
\dot{\beta}_7(t) &= -(4\tau + 3\gamma)\beta_7(t) + \tau\beta_6(t) + \tau\beta_8(t), \\
\dot{\beta}_8(t) &= -2(2\tau + \gamma)\beta_8(t), \\
\dot{\beta}_9(t) &= -(2\tau + \gamma)\beta_9(t) - 2\tau\beta_8(t).
\end{aligned}$$

where

$$\begin{aligned}
\beta_6 &= \langle I_a S_b S_c \rangle \langle S_a S_b I_c \rangle, & \beta_7 &= \langle I_a S_b I_c \rangle \langle S_a S_b I_c \rangle, \\
\beta_8 &= \langle S_a S_b I_c \rangle \langle S_a S_b I_c \rangle, & \beta_9 &= \langle S_a S_b \rangle \langle S_a S_b I_c \rangle.
\end{aligned} \tag{3.10}$$

From the initial configuration of the system we have that  $\beta_1(0) = 1$ ,  $\beta_2(0) = 1$ ,  $\beta_8(0) = 1$  and  $\beta_9(0) = 1$  with all other  $\beta$ 's being zero at  $t = 0$ . From these, the solution of the  $\beta$ s can be obtained by direct integration and then by backward substitution. We have from system (3.9) that  $\beta_6(0) = 0 \implies \beta_6(t) = 0$  for all  $t \geq 0 \implies \beta_4(t) = 0$  for all  $t \geq 0$ . Following this process lead us to obtain;

$$\begin{aligned}
\beta_8(t) &= e^{-2(2\tau+\gamma)t}, \\
\beta_9(t) &= \frac{2\tau}{2\tau + \gamma} e^{-2(2\tau+\gamma)t} + \frac{\gamma}{2\tau + \gamma} e^{-(2\tau+\gamma)t}, \\
\beta_7(t) &= \frac{\tau}{\gamma} e^{-2(2\tau+\gamma)t} - \frac{\tau}{\gamma} e^{-(4\tau+3\gamma)t}, \\
\beta_5(t) &= \frac{2\tau^2}{\gamma(2\tau + \gamma)} e^{-2(2\tau+\gamma)t} + \frac{\tau}{2\tau + \gamma} e^{-(2\tau+\gamma)t} \\
&\quad - \frac{2\tau^2}{\gamma(2\tau + \gamma)} e^{-(4\tau+3\gamma)t} - \frac{\tau}{2\tau + \gamma} e^{-2(\tau+\gamma)t} \\
\beta_3(t) &= -\frac{\gamma - \tau}{\gamma} e^{-2(2\tau+\gamma)t} - \frac{\tau^2}{\gamma(\tau + \gamma)} e^{-(4\tau+3\gamma)t} \\
&\quad + \frac{\gamma}{\tau + \gamma} e^{-(3\tau+2\gamma)t}, \\
\beta_2(t) &= \frac{\tau}{\gamma} e^{-2(2\tau+\gamma)t} - \frac{\tau^2}{\gamma(\tau + \gamma)} e^{-(4\tau+3\gamma)t} \\
&\quad + \frac{\gamma}{\tau + \gamma} e^{-(3\tau+2\gamma)t}, \\
\beta_1(t) &= \frac{2\tau^2 - \tau\gamma}{\gamma(2\tau + \gamma)} e^{-2(2\tau+\gamma)t} - \frac{\tau^3}{\gamma(\tau + \gamma)^2} e^{-(4\tau+3\gamma)t}, \\
&\quad + \frac{2\tau\gamma}{(\tau + \gamma)^2} e^{-(3\tau+2\gamma)t} + K_6 e^{-(2\tau+\gamma)t}.
\end{aligned} \tag{3.11}$$

where

$$K_6 = \frac{3\tau\gamma + \gamma^2 - 2\tau^2}{\gamma(2\tau + \gamma)} + \frac{\tau^3 - 2\tau\gamma^2}{\gamma(\tau + \gamma)^2}.$$

Since  $\beta_4(0) = 0$  for all  $t \geq 0$ , equation (3.7) is reduced to

$$\dot{\alpha}^{SSI}(t) = -(\tau + \gamma)\alpha^{SSI}(t) + \tau\beta_1(t) - \tau\beta_2(t) - \tau\beta_3(t) + \tau\beta_5(t). \tag{3.12}$$

Plugging in the solutions of the  $\beta$ s from (3.11), the solution of (3.12) is obtained by method of integrating, and it yields

$$\begin{aligned} \alpha^{SSI}(t) = & -\frac{\tau}{2\tau + \gamma}e^{-2(2\tau+\gamma)t} - \frac{C_1}{3\tau + 2\gamma}e^{-(4\tau+3\gamma)t} \\ & + \frac{2\tau^2\gamma}{(2\tau + \gamma)(\tau + \gamma)^2}e^{-(3\tau+2\gamma)t} - \frac{C_2}{\tau}e^{-(2\tau+\gamma)t} \\ & + \frac{\tau^2}{(2\tau + \gamma)(\tau + \gamma)}e^{-2(\tau+\gamma)t} + K_7e^{-(\tau+\gamma)t}. \end{aligned} \quad (3.13)$$

where

$$C_1 = \frac{\tau^4(2\tau + \gamma) - 2\tau^3(\tau + \gamma)^2}{\gamma(\tau + \gamma)^2(2\tau + \gamma)}, \quad C_2 = \frac{\tau^2}{2\tau + \gamma} - \tau K_6$$

And

$$\begin{aligned} K_7 = & \frac{\tau}{2\tau + \gamma} + \frac{C_1}{3\tau + 2\gamma} + \frac{C_2}{\tau} \\ & - \frac{2\tau^2\gamma}{(2\tau + \gamma)(\tau + \gamma)^2} - \frac{\tau^2}{(2\tau + \gamma)(\tau + \gamma)}. \end{aligned}$$

In figure (3.2), we verify that equation (3.13) corresponds with the solution of  $\alpha^{SSI}(t)$  from the hierarchy of moment equation (3.1) to (3.3).

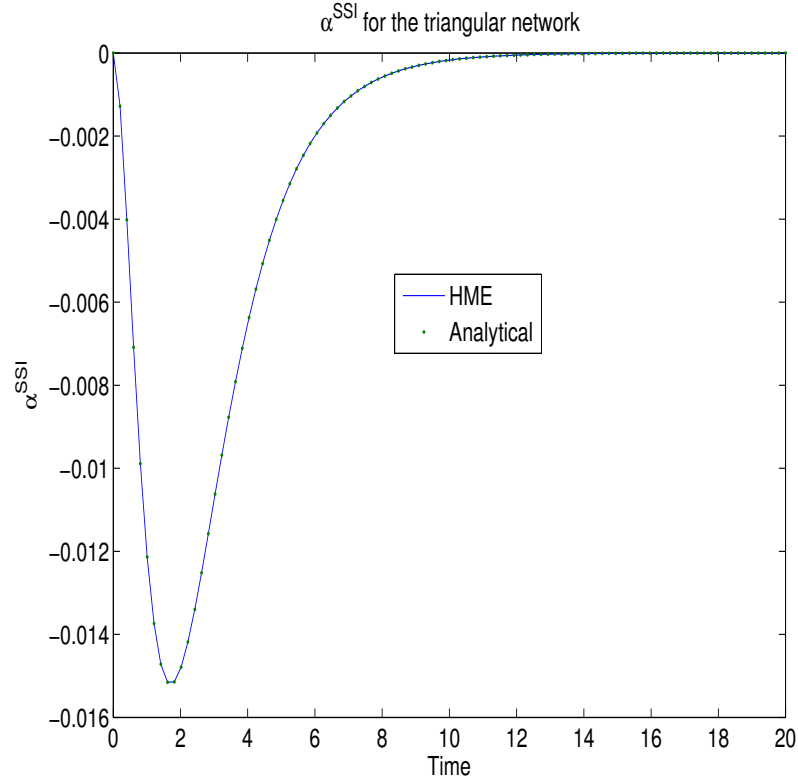


Figure 3.2: For the triangular network, with  $\tau = 0.4$  and  $\gamma = 0.2$ , we evaluate  $\alpha^{SSI}(t)$  from the hierarchy moment equations of system (3.1) to (3.3) and compare with that derived analytically in equation (3.13)

### 3.3.2 Closure for $I-S-I$

In this second part, we follow the procedure above to consider the closure for the triple of the form  $I-S-I$  for the same triangular network. We investigate the closure for  $\langle I_a S_b I_c \rangle$ . Of course

$$\langle I_a S_b I_c \rangle \approx \frac{\langle I_a S_b \rangle \langle S_b I_c \rangle}{\langle S_b \rangle}.$$

so we write

$$\alpha^{ISI}(t) = \langle S_b \rangle \langle I_a S_b I_c \rangle - \langle I_a S_b \rangle \langle S_b I_c \rangle$$

We find the derivative of  $\alpha^{ISI}(t)$ , substitute for terms in the same sets of system of equations (3.1) to (3.3) to obtain

$$\dot{\alpha}^{ISI}(t) = -2(\tau + \gamma)\alpha^{ISI}(t) - \tau\beta_1(t) + \tau\beta_2(t) - \tau\beta_{10}(t) + \tau\beta_{11}(t). \quad (3.14)$$

where

$$\begin{aligned} \beta_{10}(t) &= \langle I_a S_b \rangle \langle I_a S_b S_c \rangle, & \beta_{10}(0) &= 0, \\ \beta_{11}(t) &= \langle S_b \rangle \langle I_a S_b S_c \rangle, & \beta_{11}(0) &= 0. \end{aligned} \quad (3.15)$$

With  $\beta_1$  and  $\beta_2$  retaining the form in the first part of this section.

In similar manner from the first part, simple differentiation of the  $\beta$ s leads us to obtain

$$\begin{aligned} \dot{\beta}_{10}(t) &= -(3\tau + 2\gamma)\beta_{10}(t) + \tau\beta_6(t) - \tau\beta_{12}(t), \\ \dot{\beta}_{11}(t) &= -(2\tau + \gamma)\beta_{11}(t) - \tau\beta_{10}(t) - \tau\beta_{14}(t), \\ \dot{\beta}_{12}(t) &= -(4\tau + 3\gamma)\beta_{12}(t) + \tau\beta_6(t) + \tau\beta_{13}(t), \\ \dot{\beta}_{13}(t) &= -2(2\tau + \gamma)\beta_{13}(t), \\ \dot{\beta}_{14}(t) &= -(3\tau + 2\gamma)\beta_{14}(t) - \tau\beta_{12}(t) + \tau\beta_{13}(t). \end{aligned} \quad (3.16)$$

where

$$\begin{aligned} \beta_{12} &= \langle I_a S_b I_c \rangle \langle I_a S_b S_c \rangle, & \beta_{12}(0) &= 0, \\ \beta_{13} &= \langle I_a S_b S_c \rangle \langle I_a S_b S_c \rangle, & \beta_{13}(0) &= 0, \\ \beta_{14} &= \langle S_b I_c \rangle \langle I_a S_b S_c \rangle, & \beta_{14}(0) &= 0. \end{aligned} \quad (3.17)$$

Except for  $\beta_1(t)$  and  $\beta_2(t)$  which are initially unity, all the extra  $\beta$ s in this part are zero at  $t = 0$  due to the initial pure state of the network. Hence from (3.16), we have that  $\beta_{10}(t) = \beta_{11}(t) = \beta_{12}(t) = \beta_{13}(t) = \beta_{14}(t) = 0$  for all  $t \geq 0$ . Then, equation (3.14) is reduced to a simple form;

$$\dot{\alpha}^{ISI}(t) = -2(\tau + \gamma)\alpha^{ISI}(t) - \tau\beta_1(t) + \tau\beta_2(t). \quad (3.18)$$

which is also solved by method of integrating and it is;

$$\begin{aligned}
\alpha^{ISI}(t) = & \frac{\tau}{2\tau + \gamma} e^{-2(\tau+\gamma)t} - \frac{\tau^3}{(2\tau + \gamma)(\tau + \gamma)^2} e^{-(4\tau+3\gamma)t} \\
& - \frac{\gamma(\tau - \gamma)}{(\tau + \gamma)^2} e^{-(3\tau+2\gamma)t} + \frac{\tau K_6}{\gamma} e^{-(2\tau+\gamma)t} \\
& + K_7 e^{-2(\tau+\gamma)t},
\end{aligned} \tag{3.19}$$

with  $K_6$  as defined for (3.11) and

$$K_7 = \frac{\tau^3}{(2\tau + \gamma)(\tau + \gamma)^2} + \frac{\gamma(\tau - \gamma)}{(\tau + \gamma)^2} - \frac{\tau}{2\tau + \gamma} - \frac{\tau K_6}{\gamma}.$$

We also validate in figure (3.3) that computation of equation (3.19) corresponds with its solution from the moment equations (3.1) to (3.3) for the same parameters values.

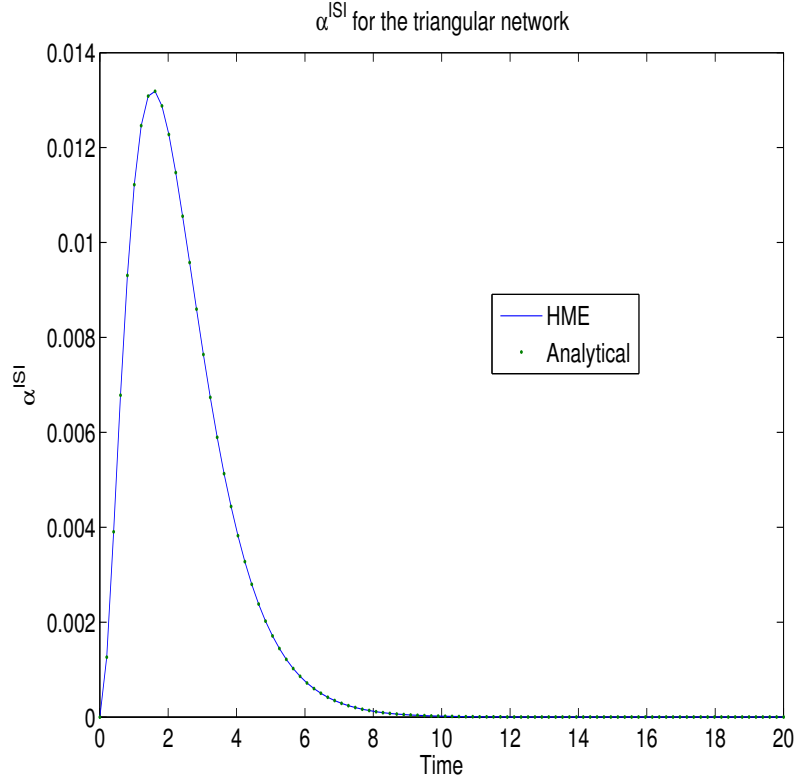


Figure 3.3: For the triangular network, with  $\tau = 0.4$  and  $\gamma = 0.2$ , we also evaluate  $\alpha^{ISI}(t)$  from the hierarchy moment equations and compare with that derived analytically in equation (3.19)

To go beyond the triangular network with this approach, computation for equation of the forms of (3.13) and (3.19) for higher order cycles becomes infeasible due to the large number of  $\beta$ s involved and the complexity of the interdependency of their derivatives. In that case, we take a different approach to approximate the

respective  $\alpha(t)$  by a zero polynomial of a finite order and invariably bound the error. That is, we will show that the truncating error for doing this is an upper bound for this approximation and that this truncating error degenerates for a finite time.

### 3.3.3 Taylor Polynomial

We shall find an approximating zero polynomial of degree  $n$  for the two forms of  $\alpha(t)$  about  $t = 0$  and show that the truncating error converges to zero for a finite  $t$ .

Generally, Taylor series for  $\alpha(t)$  about  $t = 0$  is

$$\begin{aligned}\alpha(t) &= \alpha(0) + \alpha^{(1)}(0)\frac{t}{1!} + \alpha^{(2)}(0)\frac{t^2}{2!} \\ &+ \alpha^{(3)}(0)\frac{t^3}{3!} + \dots + \alpha^{(n)}(0)\frac{t^n}{n!} + E_n \\ &= P_n(t) + E_n.\end{aligned}\tag{3.20}$$

where

$$\alpha^{(n)}(t) = \frac{d^n}{dt^n}\alpha(t).$$

$P_n(t)$  is the approximating or Taylor polynomial of degree  $n$  and  $E_n$  is the truncating error. The Lagrange form of  $E_n$  is

$$|E_n| \leq M \frac{|t|^{n+1}}{(n+1)!} \quad \text{where} \quad |\alpha^{(n+1)}| \leq M, \quad 0 \leq t < \infty.$$

In spite that we got result for the triangular network, we discuss the Taylor's polynomial for it to explain what we actually intend to do.

#### Taylor Polynomial for $\alpha^{SSI}(t)$

To consider the Taylor polynomial for  $\alpha^{SSI}(t)$  in the triangular network, we need to recall the initial condition for  $\alpha^{SSI}(0)$ . That is

$$\alpha^{SSI}(0) = \langle S_b \rangle \langle S_a S_b I_c \rangle - \langle S_a S_b \rangle \langle S_b I_c \rangle = 0.$$

From the first derivative of  $\alpha(t)$  in equation (3.12) and the initial condition of  $\beta_1(0) = 1$ ,  $\beta_2(0) = 1$ ,  $\beta_3(0) = 0$  and  $\beta_5(0) = 0$  we also have that

$$\dot{\alpha}^{(SSI)}(0) = -(\tau + \gamma) \times 0 + \tau \times 1 - \tau \times 1 - \tau \times 0 + \tau \times 0 = 0.$$

To avoid clumsy representation, we shall use  $\alpha$  and  $\alpha^{SSI}$  interchangeably in this section and thereafter. Recalling that  $\beta_4(t) = 0$  for all  $t \geq 0$  and also  $\beta_6(t) = 0$  for all  $t \geq 0$ , then plugging in the various derivatives of the respective  $\beta$ 's from (3.9) into the derivative of (3.12), the second derivative of  $\alpha(t)$  at  $t = 0$  is obtained as follows

$$\begin{aligned}
\alpha^{(2)}(t) &= -(\tau + \gamma)\dot{\alpha}(t) + \tau\dot{\beta}_1(t) - \tau\dot{\beta}_2(t) - \tau\dot{\beta}_3(t) + \tau\dot{\beta}_5(t) \\
&= -(\tau + \gamma)\alpha(t) + \tau\beta_1(t) - \tau\beta_2(t) - \tau\beta_3(t) + \tau\beta_5(t) \\
&\quad + \tau[-(3\tau + 2\gamma)\beta_1(t) - \tau\beta_6(t) - \tau\beta_7(t)] \\
&\quad - \tau[-(2\tau + \gamma)\beta_2(t) - \tau\beta_1(t) - \tau\beta_3(t)] \\
&\quad - \tau[-(3\tau + 2\gamma)\beta_3(t) - \tau\beta_7(t) + \tau\beta_8(t)] \\
&\quad - \tau[-2(\tau + \gamma)\beta_5(t) - 2\tau\beta_7(t) + \tau\beta_9(t)], \\
\alpha^{(2)}(0) &= [\tau - 2\tau\gamma - 2\tau^2] + [2\tau^2 + \tau\gamma - \tau] \\
&= -\tau\gamma.
\end{aligned}$$

Summarily, for the triangle  $\alpha^{SSI}(0) = 0$ ,  $\dot{\alpha}^{SSI}(0) = 0$  and  $\ddot{\alpha}^{SSI}(0) = -\tau\gamma$ . The Taylor series for  $\alpha(t)$  can therefore be written as

$$\begin{aligned}
\alpha(t) &= 0 \times \frac{t^0}{0!} - 0 \times \frac{t^1}{1!} - \tau\gamma \frac{t^2}{2!} - \dots - \alpha^{(n)}(0) \frac{t^n}{n!} \\
&= P_1(t) + E_1.
\end{aligned} \tag{3.21}$$

where  $P_1(t) = 0 - 0 = 0$  is a zero polynomial of order 1 and the truncation error  $E_1$  is

$$|E_1| \leq |-\tau\gamma| \frac{t^2}{2!}. \tag{3.22}$$

### Taylor Polynomial for $\alpha^{ISI}(t)$

Equation (3.18) is differentiated to obtain the coefficients of the approximating polynomial that we are about to obtain. From the initial conditions of  $\alpha^{ISI}(0) = 0$ ,  $\beta_1(0) = 1$  and  $\beta_2(0) = 1$  we have from (3.18) that  $\dot{\alpha}(0) = 0$ . The second derivative of  $\alpha^{ISI}(t)$  is direct from (3.18). That is

$$\begin{aligned}
\alpha^{(2)}(t) &= -2(\tau + \gamma)\dot{\alpha}(t) - \tau\dot{\beta}_1(t) + \tau\dot{\beta}_2(t) \\
&= -2(\tau + \gamma)\alpha(t) - \tau\beta_1(t) + \tau\beta_2(t) \\
&\quad - \tau[-(3\tau + 2\gamma)\beta_1(t) - \tau\beta_6(t) - \tau\beta_7(t)] \\
&\quad + \tau[-(2\tau + \gamma)\beta_2(t) - \tau\beta_1(t) - \tau\beta_3(t)], \\
\alpha^{(2)}(0) &= [2\tau^2 + 2\tau\gamma - 2\tau] + [2\tau - 2\tau^2 - \tau\gamma] \\
&= \tau\gamma.
\end{aligned}$$

That is, in this case,  $\alpha^{ISI}(0) = 0$ ,  $\dot{\alpha}^{ISI}(0) = 0$  and  $\ddot{\alpha}^{ISI}(0) = \tau\gamma$ . The Taylor series for  $\alpha(t)$  can therefore be written as

$$\begin{aligned}\alpha^{ISI}(t) &= 0 \times \frac{t^0}{0!} + 0 \times \frac{t^1}{1!} + \tau\gamma \frac{t^2}{2!} + \dots + \alpha^{(n)}(0) \frac{t^n}{n!} \\ &= P_1(t) + E_1.\end{aligned}$$

where  $P_1(t) = 0 + 0 = 0$  is also a zero polynomial of order 1 and the truncation error  $E_1$  is

$$|E_1| \leq \tau\gamma \frac{t^2}{2!}. \quad (3.23)$$

We proceed to do similar analysis for the two forms of triple for the square network.

### 3.4 The Square Network

We shall also consider the closure for both the  $S$ - $S$ - $I$  and  $I$ - $S$ - $I$  triple in the square network of figure (3.1) whose transmission matrix is

$$T = \begin{bmatrix} 0 & \tau & 0 & \tau \\ \tau & 0 & \tau & 0 \\ 0 & \tau & 0 & \tau \\ \tau & 0 & \tau & 0 \end{bmatrix}.$$

With the single node equations from (1.21) as

$$\begin{aligned}\langle \dot{S}_a \rangle &= -\tau \langle S_a I_1 \rangle - \tau \langle S_a I_c \rangle, \\ \langle \dot{S}_1 \rangle &= -\tau \langle I_a S_1 \rangle - \tau \langle S_1 I_b \rangle, \\ \langle \dot{S}_b \rangle &= -\tau \langle I_1 S_b \rangle - \tau \langle S_b I_c \rangle, \\ \langle \dot{S}_c \rangle &= -\tau \langle I_a S_c \rangle - \tau \langle I_b S_c \rangle, \\ \langle \dot{I}_a \rangle &= \tau \langle S_a I_1 \rangle + \tau \langle S_a I_c \rangle - \gamma \langle I_a \rangle, \\ \langle \dot{I}_1 \rangle &= \tau \langle I_a S_1 \rangle + \tau \langle S_1 I_b \rangle - \gamma \langle I_1 \rangle, \\ \langle \dot{I}_b \rangle &= \tau \langle I_1 S_b \rangle + \tau \langle S_b I_c \rangle - \gamma \langle I_b \rangle, \\ \langle \dot{I}_c \rangle &= \tau \langle I_a S_c \rangle + \tau \langle I_b S_c \rangle - \gamma \langle I_c \rangle.\end{aligned} \quad (3.24)$$



For the pairs we have;

$$\begin{aligned}
\langle \dot{S}_a I_1 \rangle &= \tau \langle S_a S_1 I_b \rangle - \tau \langle S_a I_1 I_c \rangle - (\tau + \gamma) \langle S_a I_1 \rangle, \\
\langle \dot{I}_a \dot{S}_1 \rangle &= \tau \langle S_a S_1 I_c \rangle - \tau \langle I_a S_1 I_b \rangle - (\tau + \gamma) \langle I_a S_1 \rangle, \\
\langle \dot{S}_1 I_b \rangle &= \tau \langle S_1 S_b I_c \rangle - \tau \langle I_a S_1 I_b \rangle - (\tau + \gamma) \langle S_1 I_b \rangle, \\
\langle \dot{I}_1 \dot{S}_b \rangle &= \tau \langle I_a S_1 S_b \rangle - \tau \langle I_1 S_b I_c \rangle - (\tau + \gamma) \langle I_1 S_b \rangle, \\
\langle \dot{S}_b I_c \rangle &= \tau \langle I_1 S_b S_c \rangle - \tau \langle I_a S_b I_c \rangle - (\tau + \gamma) \langle S_b I_c \rangle, \\
\langle \dot{I}_b \dot{S}_c \rangle &= \tau \langle I_1 S_b S_c \rangle - \tau \langle I_a I_b S_c \rangle - (\tau + \gamma) \langle I_b S_c \rangle, \\
\langle \dot{S}_a I_c \rangle &= \tau \langle S_a I_b S_c \rangle - \tau \langle S_a I_1 I_c \rangle - (\tau + \gamma) \langle S_a I_c \rangle, \\
\langle \dot{I}_a \dot{S}_c \rangle &= \tau \langle S_a I_1 S_c \rangle - \tau \langle I_a I_b S_c \rangle - (\tau + \gamma) \langle I_a S_c \rangle, \\
\langle \dot{S}_a \dot{S}_1 \rangle &= -\tau \langle S_a S_1 I_c \rangle - \tau \langle S_a S_1 I_b \rangle, \\
\langle \dot{S}_1 \dot{S}_b \rangle &= -\tau \langle I_a S_1 S_b \rangle - \tau \langle S_1 S_b I_c \rangle, \\
\langle \dot{S}_b \dot{S}_c \rangle &= -\tau \langle I_1 S_b S_c \rangle - \tau \langle I_a S_b S_c \rangle, \\
\langle \dot{S}_a \dot{S}_c \rangle &= -\tau \langle S_a I_1 S_c \rangle - \tau \langle S_a I_b S_c \rangle.
\end{aligned} \tag{3.25}$$

The triples are expressed in quadruples, some of which are

$$\begin{aligned}
\langle \dot{S}_a \dot{S}_1 I_b \rangle &= -(\tau + \gamma) \langle S_a S_1 I_b \rangle + \tau \langle S_a S_1 S_b I_c \rangle - \tau \langle S_a S_1 I_b I_c \rangle, \\
\langle \dot{S}_1 \dot{S}_b I_c \rangle &= -(\tau + \gamma) \langle S_1 S_b I_c \rangle + \tau \langle I_a S_1 S_b S_c \rangle - \tau \langle I_a S_1 S_b I_c \rangle, \\
\langle \dot{I}_a \dot{S}_1 I_b \rangle &= -2(\tau + \gamma) \langle I_a S_1 I_b \rangle + \tau \langle S_a S_1 I_b I_c \rangle + \tau \langle I_a S_1 S_b I_c \rangle, \\
\langle \dot{S}_a \dot{I}_1 I_c \rangle &= -2(\tau + \gamma) \langle S_a I_1 I_c \rangle + \tau \langle S_a S_1 I_b I_c \rangle + \tau \langle S_a I_1 I_b S_c \rangle.
\end{aligned} \tag{3.26}$$

### 3.4.1 Closure for $S$ - $S$ - $I$

We first examine the closure for the triple  $\langle S_1 S_b I_c \rangle$  by writing

$$\alpha^{SSI}(t) = \langle S_b \rangle \langle S_1 S_b I_c \rangle - \langle S_1 S_b \rangle \langle S_b I_c \rangle.$$

We find its derivative with respect to time, substitute the relevant terms from (3.24) to (3.26) to obtain;

$$\begin{aligned}
\dot{\alpha}^{SSI}(t) &= -(\tau + \gamma)\alpha^{SSI}(t) - \tau\alpha_2(t) - \tau\alpha_3(t) \\
&\quad - \tau\beta_1(t) + \tau\beta_2(t), \\
\dot{\alpha}_2(t) &= -(\tau + \gamma)\alpha_2(t) + \tau\beta_3(t) - \tau\beta_4(t) \\
&\quad + \tau\beta_5(t) - \tau\beta_6(t) - \tau\beta_7(t) \\
&\quad - \tau\beta_8(t) + \tau\beta_9(t), \\
\dot{\alpha}_3(t) &= -2(\tau + \gamma)\alpha_3(t) - \tau\beta_8(t) + \tau\beta_9(t) \\
&\quad + \tau\beta_{10}(t) - \tau\beta_{11}(t) + \tau\beta_{12}(t) - \tau\beta_{13}(t).
\end{aligned} \tag{3.27}$$

where

$$\begin{aligned}
\alpha_2 &= \langle S_1 S_b \rangle \langle I_a S_b S_c \rangle - \langle S_b \rangle \langle I_a S_1 S_b S_c \rangle, \\
\alpha_3 &= \langle S_b \rangle \langle I_a S_1 S_b I_c \rangle - \langle S_b I_c \rangle \langle I_a S_1 S_b \rangle,
\end{aligned}$$

With

$$\begin{aligned}
\beta_1 &= \langle I_1 S_b \rangle \langle S_1 S_b I_c \rangle, & \beta_2 &= \langle S_1 S_b \rangle \langle I_1 S_b I_c \rangle, \\
\beta_3 &= \langle S_1 S_b \rangle \langle S_a I_1 S_b S_c \rangle, & \beta_4 &= \langle S_1 S_b \rangle \langle I_a I_1 S_b S_c \rangle, \\
\beta_5 &= \langle I_1 S_b \rangle \langle I_a S_1 S_b S_c \rangle, & \beta_6 &= \langle S_b S_c \rangle \langle I_a S_1 S_b S_c \rangle, \\
\beta_7 &= \langle I_a S_b S_c \rangle \langle S_1 S_b I_c \rangle, & \beta_8 &= \langle I_a S_b S_c \rangle \langle I_a S_1 S_b \rangle, \\
\beta_9 &= \langle S_b \rangle \langle I_a S_1 S_b S_c \rangle, & \beta_{10} &= \langle S_b \rangle \langle S_a S_1 S_b I_c \rangle, \\
\beta_{11} &= \langle I_1 S_b \rangle \langle I_a S_1 S_b I_c \rangle, & \beta_{12} &= \langle I_1 S_b I_c \rangle \langle I_a S_1 S_b \rangle, \\
\beta_{13} &= \langle S_b I_c \rangle \langle S_a S_1 S_b I_c \rangle.
\end{aligned} \tag{3.28}$$

The derivatives of the  $\beta$ s are expressed in terms of other form of  $\beta$ s, for example

$$\begin{aligned}
\dot{\beta}_1(t) &= -2(\tau + \gamma)\beta_1(t) + \tau\beta_5(t) - \tau\beta_{12}(t) - \beta_{14}(t) - \beta_{15}(t), \\
\dot{\beta}_2(t) &= -2(\tau + \gamma)\beta_2(t) + \tau\beta_4(t) - \tau\beta_{12}(t) - \beta_{14}(t) + \beta_{16}(t), \\
\dot{\beta}_{47}(t) &= -2(2\tau + 2\gamma)\beta_{47}(t).
\end{aligned} \tag{3.29}$$

where

$$\begin{aligned}
\beta_{14}(t) &= \langle I_1 S_b I_c \rangle \langle S_1 S_b I_c \rangle, \\
\beta_{15}(t) &= \langle I_a S_1 S_b \rangle \langle S_1 S_b I_c \rangle, \\
\beta_{47}(t) &= \langle S_a S_1 S_b I_c \rangle \langle S_a S_1 S_b I_c \rangle.
\end{aligned} \tag{3.30}$$

The interdependency of the derivatives of these  $\beta$ s on one another resulted in forty seven (47)  $\beta$ s for this first part for the square network (see Appendix for list of  $\beta$ s), and as mention earlier, it is not feasible to obtain the solution of (3.27) due to the huge number of the solutions of  $\beta$ s to be substituted into it before integration.

We proceed in the same manner above to derive the Taylor's polynomials for the two forms of  $\alpha(t)$  for this square network.

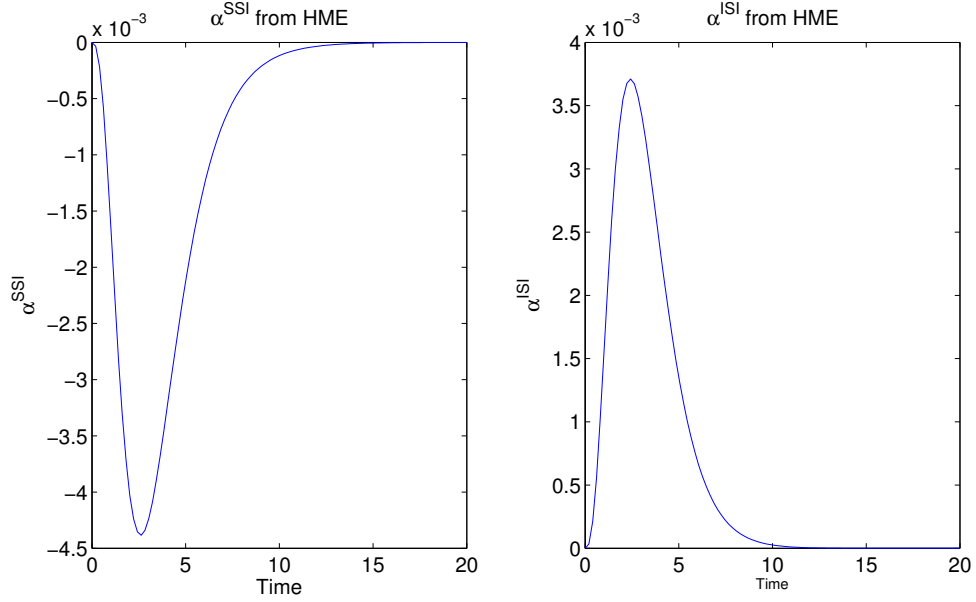


Figure 3.4: For the square network, with  $\tau = .04$  and  $\gamma = 0.2$ , we evaluate both  $\alpha^{SSI}(t)$  and  $\alpha^{ISI}(t)$  from the corresponding hierarchy moment equations (3.24) to (3.26). We want to extrapolate that the respective analytical representations of these quantities will correspond with them if all the  $\beta$ s involved in their derivation can completely be evaluated.

### Taylor Polynomial for $\alpha^{SSI}(t)$

The process of computing the coefficients of the Taylor polynomial for the case of triangular network yield a value different from zero at the second derivative. Computation of similar coefficients for the square network is a step further to obtain a value different from zero, i.e. the third derivative of (3.27) gives a value different from zero at  $t = 0$ . We can not write down all these equations due to their enormity.

That is, we obtain  $\alpha^{SSI}(0) = 0$ ,  $\dot{\alpha}^{SSI}(0) = 0$ ,  $\ddot{\alpha}^{SSI}(0) = 0$  and  $\alpha^{(3)}(0) = -\tau^2\gamma$  and so the Taylor polynomial for  $\alpha^{SSI}(t)$  can therefore be written as

$$\begin{aligned} \alpha^{SSI}(t) &= 0 \times \frac{t^0}{0!} - 0 \times \frac{t^1}{1!} - 0 \times \frac{t^2}{2!} - \tau^2\gamma \frac{t^3}{3!} - \dots - \alpha^{(n)}(0) \frac{t^n}{n!} \\ &= P_2(t) + E_2. \end{aligned}$$

where  $P_2(t) = 0 - 0 - 0 = 0$  is a zero polynomial of order 2 and the truncation error  $E_2$  is;

$$|E_2| \leq |-\tau^2\gamma| \frac{t^3}{3!}. \quad (3.31)$$

### 3.4.2 Closure for $I$ - $S$ - $I$

In this part, we follow the above procedure and write

$$\alpha^{ISI}(t) = \langle S_b \rangle \langle I_1 S_b I_c \rangle - \langle I_1 S_b \rangle \langle S_b I_c \rangle.$$

to examine the pair closure for  $\langle I_1 S_b I_c \rangle$ . We take its derivative with respect to time as usual and substitute the relevant derivatives. We then have

$$\begin{aligned} \dot{\alpha}^{ISI}(t) &= -2(\tau + \gamma)\alpha^{ISI}(t) - \tau\alpha_2(t) - \tau\alpha_3(t), \\ \dot{\alpha}_2(t) &= -2(\tau + \gamma)\alpha_2(t) + \tau\beta_8(t) - \tau\beta_9(t) \\ &\quad + \tau\beta_{48}(t) + \tau\beta_{49}(t) + \tau\beta_{50}(t) - \tau\beta_{51}(t), \\ \dot{\alpha}_3(t) &= -2(\tau + \gamma)\alpha_3(t) + \tau\beta_8(t) - \tau\beta_9(t) \\ &\quad - \tau\beta_{11}(t) + \tau\beta_{12}(t) + \tau\beta_{13}(t) - \tau\beta_{52}(t). \end{aligned} \tag{3.32}$$

where

$$\begin{aligned} \alpha_2 &= \langle I_1 S_b \rangle \langle I_a S_b S_c \rangle - \langle S_b \rangle \langle I_a I_1 S_b S_c \rangle, \\ \alpha_3 &= \langle S_b I_c \rangle \langle I_a S_1 S_b \rangle - \langle S_b \rangle \langle I_a S_1 S_b I_c \rangle, \end{aligned}$$

with

$$\begin{aligned} \beta_{48} &= \langle I_1 S_b \rangle \langle S_1 I_1 S_b S_c \rangle, & \beta_{49} &= \langle S_b \rangle \langle S_a I_1 S_b S_c \rangle, \\ \beta_{50} &= \langle S_b I_c \rangle \langle I_a I_1 S_b S_c \rangle, & \beta_{51} &= \langle I_1 S_b I_c \rangle \langle I_a S_b S_c \rangle, \\ \beta_{52} &= \langle S_b I_c \rangle \langle S_a I_1 S_b S_c \rangle. \end{aligned} \tag{3.33}$$

The derivatives of these extra  $\beta$ s are similarly expressed in terms of other  $\beta$ s and we have a total of fifty nine  $\beta$ s to deal with while examining closure for both the  $S$ - $S$ - $I$  and  $I$ - $S$ - $I$  triples for the square network. The solution of (3.32) is also not feasible for same reason above and we proceed to obtain a Taylor polynomial for  $\alpha^{ISI}(t)$ .

#### Taylor Polynomial for $\alpha^{ISI}(t)$

From the subsequent derivative of system (3.32) at  $t = 0$ , we have that  $\alpha^{(3)} = \tau^2\gamma$  and with  $\ddot{\alpha}(t) = 0$ ,  $\dot{\alpha}(t) = 0$  and  $\alpha(0) = 0$  we can then write

$$\begin{aligned} \alpha^{ISI}(t) &= 0 \times \frac{t^0}{0!} + 0 \times \frac{t^1}{1!} + 0 \times \frac{t^2}{2!} + \tau^2\gamma \frac{t^3}{3!} + \dots + \alpha^{(n)}(0) \frac{t^n}{n!} \\ &= P_2(t) + E_2. \end{aligned}$$

where  $P_2(t) = 0 + 0 + 0 = 0$  is a zero polynomial of order 2 and the truncation error  $E_2$  is;

$$|E_2| \leq \tau^2 \gamma \frac{t^3}{3!}. \quad (3.34)$$

We follow the above process to investigate the closure of the two forms of triples for the pentagon.

## 3.5 The Pentagon

The transmission matrix for this network in figure (3.1) is

$$T = \begin{bmatrix} 0 & \tau & 0 & 0 & \tau \\ \tau & 0 & \tau & 0 & 0 \\ 0 & \tau & 0 & \tau & 0 \\ 0 & 0 & \tau & 0 & \tau \\ \tau & 0 & 0 & \tau & 0 \end{bmatrix}.$$

### 3.5.1 Closure for $S$ - $S$ - $I$

Writing out the sets of differential equations from (1.21), we also consider the accuracy of the second order moment closure for the triple  $\langle S_2 S_b I_c \rangle$  following the same procedure of writing

$$\alpha^{SSI}(t) = \langle S_b \rangle \langle S_2 S_b I_c \rangle - \langle S_2 S_b \rangle \langle S_b I_c \rangle.$$

We take the derivative of  $\alpha^{SSI}(t)$  and substitute the relevant terms from the corresponding hierarchy of moment equations to get

$$\begin{aligned} \dot{\alpha}^{SSI}(t) &= -(\tau + \gamma)\alpha^{SSI}(t) - \tau\alpha_2(t) - \tau\alpha_3(t) - \tau\beta_1(t) - \tau\beta_2(t), \\ \dot{\alpha}_2(t) &= -(\tau + \gamma)\alpha_2(t) - \tau\alpha_4(t) - \tau\alpha_5(t) - \tau\beta_3(t) + \tau\beta_4(t) \\ &\quad + \tau\beta_5(t) - \tau\beta_6(t), \\ \dot{\alpha}_4(t) &= -(\tau + \gamma)\alpha_4(t) - \tau\beta_7(t) - \tau\beta_8(t) - \tau\beta_9(t) + \tau\beta_{10}(t) \\ &\quad + \tau\beta_{11}(t) - \tau\beta_{12}(t) + \tau\beta_{13}(t), \\ \dot{\alpha}_5(t) &= -2(\tau + \gamma)\alpha_5(t) - \tau\beta_8(t) + \tau\beta_{10}(t) + \tau\beta_{14}(t) - \tau\beta_{15}(t) \\ &\quad - \tau\beta_{16}(t) + \tau\beta_{17}(t) + \tau\beta_{18}(t) - \tau\beta_{19}(t), \\ \dot{\alpha}_3(t) &= -2(\tau + \gamma)\alpha_3(t) - \tau\alpha_5(t) - \tau\alpha_6(t) + \tau\beta_{20}(t) - \tau\beta_{21}(t), \\ \dot{\alpha}_6(t) &= -2(\tau + \gamma)\alpha_6(t) + \tau\beta_{14}(t) - \tau\beta_{19}(t) - \tau\beta_{22}(t) + \tau\beta_{23}(t) \\ &\quad + \tau\beta_{24}(t) - \tau\beta_{25}(t). \end{aligned} \quad (3.35)$$

where

$$\begin{aligned}
\alpha_2 &= \langle S_b S_2 \rangle \langle I_a S_b S_c \rangle - \langle S_b \rangle \langle I_a S_2 S_b S_c \rangle, \\
\alpha_3 &= \langle S_b \rangle \langle I_1 S_2 S_b I_c \rangle - \langle S_b I_c \rangle \langle I_1 S_2 S_b \rangle, \\
\alpha_4 &= \langle S_b \rangle \langle S_a I_1 S_2 S_b S_c \rangle - \langle S_2 S_b \rangle \langle S_a I_1 S_b S_c \rangle, \\
\alpha_5 &= \langle I_1 S_2 S_b \rangle \langle I_a S_b S_c \rangle - \langle S_b \rangle \langle I_a I_1 S_2 S_b S_c \rangle, \\
\alpha_6 &= \langle S_b I_c \rangle \langle I_a S_1 S_2 S_b \rangle - \langle S_b \rangle \langle I_a S_1 S_2 S_b I_c \rangle.
\end{aligned}$$

and where the  $\beta$ s are

$$\begin{aligned}
\beta_1(t) &= \langle I_2 S_b \rangle \langle S_2 S_b I_c \rangle, & \beta_2(t) &= \langle S_2 S_b \rangle \langle I_2 S_b I_c \rangle, \\
\beta_3(t) &= \langle S_2 S_b I_c \rangle \langle I_a S_b S_c \rangle, & \beta_4(t) &= \langle I_2 S_b \rangle \langle I_a S_2 S_b S_c \rangle, \\
\beta_5(t) &= \langle S_b I_c \rangle \langle I_a S_2 S_b S_c \rangle, & \beta_6(t) &= \langle S_2 S_b \rangle \langle I_a I_2 S_b S_c \rangle, \\
\beta_7(t) &= \langle I_2 S_b \rangle \langle S_a I_1 S_2 S_b S_c \rangle, & \beta_8(t) &= \langle S_b I_c \rangle \langle S_a I_1 S_2 S_b S_c \rangle, \\
\beta_9(t) &= \langle S_b \rangle \langle S_a I_1 S_2 S_b S_c \rangle, & \beta_{10}(t) &= \langle I_1 S_2 S_b \rangle \langle S_a I_1 S_b S_c \rangle, \\
\beta_{11}(t) &= \langle S_2 S_b I_c \rangle \langle S_a I_1 S_b S_c \rangle, & \beta_{12}(t) &= \langle S_2 S_b \rangle \langle S_a S_1 I_2 S_b S_c \rangle, \\
\beta_{13}(t) &= \langle S_2 S_b \rangle \langle S_a I_1 I_2 S_b S_c \rangle, & \beta_{14}(t) &= \langle I_a S_1 S_2 S_b \rangle \langle I_a S_b S_c \rangle, \\
\beta_{15}(t) &= \langle I_1 S_2 S_b I_c \rangle \langle I_a S_b S_c \rangle, & \beta_{16}(t) &= \langle I_1 S_2 S_b \rangle \langle I_a I_2 S_b S_c \rangle, \\
\beta_{17}(t) &= \langle I_2 S_b \rangle \langle I_a I_1 S_2 S_b S_c \rangle, & \beta_{18}(t) &= \langle S_b I_c \rangle \langle I_a I_1 S_2 S_b S_c \rangle, \\
\beta_{19}(t) &= \langle S_b \rangle \langle I_a S_1 S_2 S_b S_c \rangle, & \beta_{20}(t) &= \langle I_2 S_b I_c \rangle \langle I_1 S_2 S_b \rangle, \\
\beta_{21}(t) &= \langle I_2 S_b \rangle \langle I_1 S_2 S_b I_c \rangle, & \beta_{22}(t) &= \langle I_2 S_b I_c \rangle \langle I_a S_1 S_2 S_b \rangle, \\
\beta_{23}(t) &= \langle S_b I_c \rangle \langle S_a S_1 S_2 S_b I_c \rangle, & \beta_{24}(t) &= \langle I_2 S_b \rangle \langle I_a S_1 S_2 S_b I_c \rangle, \\
\beta_{25}(t) &= \langle S_b \rangle \langle S_a S_1 S_2 S_b I_c \rangle.
\end{aligned} \tag{3.36}$$

For this network, the number of  $\beta$ s involved is large and the interdependency of their derivatives show no sign of stopping. To go beyond this level to obtain results for *cycles* of higher order, we want to state a relevant hypothesis.

### 3.5.2 Taylor Expansion Hypothesis

We hypothesise that throwing away the  $\beta$ s that are initially zero makes no difference in obtaining the coefficients for the Taylor's polynomials of both the  $\alpha^{SSI}(t)$  and  $\alpha^{ISI}(t)$  for *cycle*.

As noted and stated earlier, exact analysis is prohibited for higher order cycle due to the massive  $\beta$ s involved. Applying this hypothesis considerably reduces the number of  $\beta$ s in the derivatives of  $\alpha^{SSI}(t)$  and  $\alpha^{ISI}(t)$  in all the networks and the process still lead to obtaining the same coefficients for the Taylor's polynomial of  $\alpha^{SSI}(t)$  and  $\alpha^{ISI}(t)$  in all the networks.

Although we already have results for the two forms of  $\alpha$  for both the triangle and square networks, we shall validate this hypothesis by applying it to these networks.

### Triangle - $\alpha^{SSI}(t)$ and $\alpha^{ISI}(t)$

Invoking the Taylor's expansion hypothesis (*TEH*), the initial configuration of the triangular network in figure (3.1) means  $\beta_3(0) = 0$ ,  $\beta_4(0) = 0$  and  $\beta_5(0) = 0$ . This reduces (3.7) to

$$\dot{\alpha}^{SSI}(t) = -(\tau + \gamma)\alpha^{SSI} + \tau\beta_1(t) - \tau\beta_2(t). \quad (3.37)$$

with the relevant  $\beta$ s as

$$\begin{aligned} \beta_1(t) &= \langle S_b I_c \rangle \langle S_a S_b I_c \rangle, & \beta_1(0) &= 1 \\ \beta_2(t) &= \langle S_b \rangle \langle S_a S_b I_c \rangle, & \beta_2(0) &= 1. \end{aligned} \quad (3.38)$$

and their derivatives as

$$\begin{aligned} \dot{\beta}_1(t) &= -(3\tau + 2\gamma)\beta_1(t), \\ \dot{\beta}_2(t) &= -(2\tau + \gamma)\beta_2(t) - \tau\beta_1(t). \end{aligned} \quad (3.39)$$

Of course, from the initial conditions and equation (3.37) we have that

$$\alpha^{SSI}(0) = 0, \quad \dot{\alpha}^{SSI}(0) = -(\tau + \gamma) \times 0 + \tau \times 1 - \tau \times 1 = 0.$$

By differentiating (3.37), the second derivative of  $\alpha^{SSI}(t)$  at  $t = 0$  is obtained as

$$\begin{aligned} \ddot{\alpha}(t) &= -(\tau + \gamma)\dot{\alpha}(t) + \tau\dot{\beta}_1(t) - \tau\dot{\beta}_2(t) \\ &= -(\tau + \gamma)[-(\tau + \gamma)\alpha(t) + \tau\beta_1(t) - \tau\beta_2(t)] \\ &\quad + \tau[-(3\tau + 2\gamma)\beta_1(t)] - \tau[-(2\tau + \gamma)\beta_2(t) - \tau\beta_1(t)] \\ &= (\tau + \gamma)^2\alpha^{SSI} - \tau(\tau + \gamma)\beta_1(t) + \tau(\tau + \gamma)\beta_2(t) \\ &\quad - \tau(3\tau + 2\gamma)\beta_1(t) + \tau(2\tau + \gamma)\beta_2(t) + \tau^2\beta_1(t), \\ \ddot{\alpha}(0) &= (\tau + \gamma)^2 \times 0 - \tau(\tau + \gamma) + \tau(\tau + \gamma) \\ &\quad - \tau(3\tau + 2\gamma) + \tau(2\tau + \gamma) + \tau^2 \\ &= -3\tau^2 - 2\tau\gamma + 2\tau^2 + \tau\gamma + \tau^2 \\ &= -\tau\gamma. \end{aligned}$$

That is  $\alpha^{SSI}(0) = 0$ ,  $\dot{\alpha}^{SSI}(0) = 0$  and  $\ddot{\alpha}^{SSI}(0) = -\tau\gamma$  which are the same coefficients obtained above from the first method and these results validate the

Taylor polynomial (3.21) for the triangular network.

By invoking the *TEH*, the derivative of  $\alpha^{ISI}(t)$  in equation (3.14) is already in its simplest form in equation (3.18) with  $\beta_{10}(0) = 0$  and  $\beta_{11}(0) = 0$  and the process of finding the coefficients for the Taylor polynomial of  $\alpha^{ISI}(t)$  follows directly from section (3.3.3), giving us the same values and subsequently the same Taylor's polynomial and finally leading us to compute the same truncating error,  $E_1(t)$  of (3.23). We validate our hypothesis for the triangular network.

### Square - $\alpha^{SSI}(t)$ and $\alpha^{ISI}(t)$

Considering first for  $\alpha^{SSI}(t)$  in this network, invoking the *TEH* reduces equation (3.27) to

$$\begin{aligned}\dot{\alpha}^{SSI}(t) &= -(\tau + \gamma)\alpha^{SSI}(t) - \tau\alpha_2(t) - \tau\alpha_3(t), \\ \dot{\alpha}_2(t) &= -(\tau + \gamma)\alpha_2(t), \\ \dot{\alpha}_3(t) &= -2(\tau + \gamma)\alpha_3(t) - \tau\beta_1(t) + \tau\beta_2(t).\end{aligned}\tag{3.40}$$

where

$$\begin{aligned}\beta_1(t) &= \langle S_b I_c \rangle \langle S_a S_1 S_b I_c \rangle, & \beta_1(0) &= 1 \\ \beta_2(t) &= \langle S_b \rangle \langle S_a S_1 S_b I_c \rangle, & \beta_2(0) &= 1.\end{aligned}\tag{3.41}$$

which correspond to  $\beta_{13}(t)$  and  $\beta_{10}(t)$  respectively in equation (3.28). We want to state that the derivatives of these two  $\beta$ s are always of the form in (3.39).

With the initial conditions that  $\alpha^{SSI}(0) = 0$ ,  $\alpha_2(0) = 0$  and  $\alpha_3(0) = 0$  we have that

$$\begin{aligned}\dot{\alpha}^{SSI}(0) &= -(\tau + \gamma)\alpha^{SSI}(0) - \tau\alpha_2(0) - \tau\alpha_3(0) = 0, \\ \ddot{\alpha}^{SSI}(0) &= -(\tau + \gamma)\dot{\alpha}_1(0) - \tau\dot{\alpha}_2(0) - \tau\dot{\alpha}_3(0) \\ &= -(\tau + \gamma) \times 0 - \tau[-(\tau + \gamma)\alpha_2(0)] \\ &\quad - \tau[-2(\tau + \gamma)\alpha_3(0) - \tau\beta_1(0) + \tau\beta_2(0)] \\ &= 0 - \tau \times 0 + \tau \times 0 + \tau[0 - \tau + \tau] = 0, \\ \alpha^{(3)}(0) &= -(\tau + \gamma)\ddot{\alpha}_1(0) - \tau\ddot{\alpha}_2(0) - \tau\ddot{\alpha}_3(0) \\ &= 0 - \tau \times 0 - \tau[-2(\tau + \gamma)\dot{\alpha}_3(0) - \tau\dot{\beta}_1(0) + \tau\dot{\beta}_2(0)] \\ &= -\tau[-2(\tau + \gamma)[-2(\tau + \gamma)\alpha_3 - \tau\beta_1 + \tau\beta_2]] \\ &\quad + \tau^2[-(3\tau + 2\gamma)\beta_1] - \tau^2[-(2\tau + \gamma)\beta_2 - \tau\beta_1] \\ &= 0 - 3\tau^3 - 2\tau^2\gamma + 3\tau^3 + \tau^2\gamma = -\tau^2\gamma.\end{aligned}\tag{3.42}$$

That is,  $\alpha^{SSI}(0) = 0$ ,  $\dot{\alpha}^{SSI}(0) = 0$ ,  $\ddot{\alpha}^{SSI}(0) = 0$  and the third derivative  $\alpha^{(3)}(0) = -\tau^2\gamma$ . These are the same as the coefficients we obtained for  $\alpha^{SSI}(t)$  of this network from the first approach which lead us to compute the correspond-



ing polynomial and ultimately the truncation error  $E_3(t)$  of equation (3.31) for  $\alpha^{SSI}(t)$ .

We go through the above process by invoking the *TEH* on (3.32) and this reduces equation (3.32) to

$$\begin{aligned}\dot{\alpha}^{ISI}(t) &= -2(\tau + \gamma)\alpha^{ISI}(t) - \tau\alpha_2(t) - \tau\alpha_3(t), \\ \dot{\alpha}_2(t) &= -2(\tau + \gamma)\alpha_2(t), \\ \dot{\alpha}_3(t) &= -2(\tau + \gamma)\alpha_3(t) + \tau\beta_1(t) - \tau\beta_2(t).\end{aligned}\tag{3.43}$$

with the relevant  $\beta$ s and their derivatives as given in equation (3.39). The progressive differentiation of  $\alpha^{ISI}(t)$  at  $t = 0$  gives  $\alpha^{ISI}(0) = 0$ ,  $\dot{\alpha}^{ISI}(0) = 0$ ,  $\ddot{\alpha}^{ISI}(0) = 0$  and  $\alpha^{(3)}(0) = \tau^2\gamma$  leading to the corresponding Taylor's polynomial and finally the truncation error  $E_3$  in (3.34).

From here, we want to take a leap of faith based on the validity of the hypothesis for both the triangular and square networks and extend it to find the Taylor polynomials of the respective quantities for the pentagon and hexagon.

### The Pentagon-Taylor Polynomial for $\alpha^{SSI}(t)$

From above, equation (3.35) is completely reduced to

$$\begin{aligned}\dot{\alpha}^{SSI}(t) &= -(\tau + \gamma)\alpha^{SSI}(t) - \tau\alpha_2(t) - \tau\alpha_3(t), \\ \dot{\alpha}_2(t) &= -(\tau + \gamma)\alpha_2(t) - \tau\alpha_4(t) - \tau\alpha_5(t), \\ \dot{\alpha}_4(t) &= -(\tau + \gamma)\alpha_4(t), \\ \dot{\alpha}_5(t) &= -2(\tau + \gamma)\alpha_5(t), \\ \dot{\alpha}_3(t) &= -2(\tau + \gamma)\alpha_3(t) - \tau\alpha_5(t) - \tau\alpha_6(t), \\ \dot{\alpha}_6(t) &= -2(\tau + \gamma)\alpha_6(t) + \tau\beta_1(t) - \tau\beta_2(t).\end{aligned}\tag{3.44}$$

where the relevant  $\beta$ s are

$$\begin{aligned}\beta_1(t) &= \langle S_b I_c \rangle \langle S_a S_1 S_2 S_b I_c \rangle, & \beta_1(0) &= 1 \\ \beta_2(t) &= \langle S_b \rangle \langle S_a S_1 S_2 S_b I_c \rangle, & \beta_2(0) &= 1.\end{aligned}\tag{3.45}$$

which were originally labelled as  $\beta_{23}$  and  $\beta_{25}$  respectively in equation (3.36).

Differentiating  $\alpha^{SSI}(t)$  at  $t = 0$ , we have that  $\alpha^{SSI}(0) = 0$ ,  $\dot{\alpha}^{SSI}(0) = 0$ ,  $\ddot{\alpha}^{SSI}(0) = 0$ ,  $\alpha^{(3)}(0) = 0$  and  $\alpha^{(4)}(0) = -\tau^3\gamma$ . The Taylor polynomial for  $\alpha^{SSI}(t)$  is therefore;

$$\begin{aligned}
\alpha^{SSI}(t) &= 0 \times \frac{t^0}{0!} + 0 \times \frac{t^1}{1!} + 0 \times \frac{t^2}{2!} + 0 \times \frac{t^3}{3!} \\
&\quad - \tau^3 \gamma \frac{t^4}{4!} + \dots - \alpha^{(n)}(0) \frac{t^n}{n!} \\
&= P_3(t) + E_3.
\end{aligned}$$

where  $P_1(t) = 0 + 0 + 0 + 0 = 0$  is a zero polynomial of order 3 and the truncation error  $E_3$  is

$$|E_3| \leq |-\tau^3 \gamma| \frac{t^4}{4!}. \quad (3.46)$$

### The Pentagon-Taylor Polynomial for $\alpha^{ISI}(t)$

We write

$$\alpha^{ISI}(t) = \langle S_b \rangle \langle I_2 S_b I_c \rangle - \langle I_2 S_b \rangle \langle S_b I_c \rangle,$$

and following the process of differentiation as above, we have that

$$\begin{aligned}
\dot{\alpha}^{ISI}(t) &= -(\tau + \gamma)\alpha^{ISI}(t) - \tau\alpha_2(t) - \tau\alpha_3(t), \\
\dot{\alpha}_2(t) &= -(\tau + \gamma)\alpha_2(t) - \tau\alpha_4(t) - \tau\alpha_5(t), \\
\dot{\alpha}_4(t) &= -(\tau + \gamma)\alpha_4(t), \\
\dot{\alpha}_5(t) &= -2(\tau + \gamma)\alpha_5(t) - \tau\beta_1(t) + \tau\beta_2(t), \\
\dot{\alpha}_3(t) &= -2(\tau + \gamma)\alpha_3(t) - \tau\alpha_4(t) - \tau\alpha_6(t), \\
\dot{\alpha}_6(t) &= -2(\tau + \gamma)\alpha_6(t).
\end{aligned} \quad (3.47)$$

Our usual process of evaluating the derivative of  $\alpha^{ISI}(t)$  at  $t = 0$  gives  $\alpha^{ISI}(0) = 0$ ,  $\dot{\alpha}^{ISI}(0) = 0$ ,  $\ddot{\alpha}^{ISI}(0) = 0$ ,  $\alpha^{(3)}(0) = 0$  and  $\alpha^{(4)}(0) = \tau^3 \gamma$ . Therefore, the Taylor polynomial for  $\alpha^{ISI}(t)$  is

$$\begin{aligned}
\alpha^{SSI}(t) &= 0 \times \frac{t^0}{0!} + 0 \times \frac{t^1}{1!} + 0 \times \frac{t^2}{2!} + 0 \times \frac{t^3}{3!} \\
&\quad + \tau^3 \gamma \frac{t^4}{4!} + \dots + \alpha^{(n)}(0) \frac{t^n}{n!} \\
&= P_3(t) + E_3.
\end{aligned}$$

where  $P_1(t) = 0 + 0 + 0 + 0 = 0$  is a zero polynomial of order 3 and the truncation error  $E_3$  is;

$$|E_3| \leq \tau^3 \gamma \frac{t^4}{4!}. \quad (3.48)$$

## 3.6 The Hexagonal Network

The transmission matrix for this network (see figure (3.1)) is

$$T = \begin{bmatrix} 0 & \tau & 0 & 0 & 0 & \tau \\ \tau & 0 & \tau & 0 & 0 & 0 \\ 0 & \tau & 0 & \tau & 0 & 0 \\ 0 & 0 & \tau & 0 & \tau & 0 \\ 0 & 0 & 0 & \tau & 0 & \tau \\ \tau & 0 & 0 & 0 & \tau & 0 \end{bmatrix}.$$

### 3.6.1 Closure for S-S-I

We follow similar procedure as above and consider the closure for the triple  $\langle S_3 S_b I_c \rangle$  by writing

$$\alpha^{SSI}(t) = \langle S_b \rangle \langle S_3 S_b I_c \rangle - \langle S_3 S_b \rangle \langle S_b I_c \rangle.$$

We take the derivative of  $\alpha^{SSI}(t)$  with respect to time, substitute the relevant derivatives from its hierarchy of moment equations to obtain

$$\begin{aligned} \dot{\alpha}^{SSI}(t) &= -(\tau + \gamma)\alpha^{SSI}(t) - \tau\alpha_2(t) - \tau\alpha_3(t), \\ \dot{\alpha}_2(t) &= -(\tau + \gamma)\alpha_2(t) - \tau\alpha_4(t) - \tau\alpha_5(t), \\ \dot{\alpha}_4(t) &= -(\tau + \gamma)\alpha_4(t) - \tau\alpha_6(t) - \tau\alpha_7(t), \\ \dot{\alpha}_6(t) &= -2(\tau + \gamma)\alpha_6(t), \\ \dot{\alpha}_7(t) &= -(\tau + \gamma)\alpha_7(t), \\ \dot{\alpha}_5(t) &= -2(\tau + \gamma)\alpha_5(t) - \tau\alpha_6(t) - \tau\alpha_8(t), \\ \dot{\alpha}_8(t) &= -2(\tau + \gamma)\alpha_8(t), \\ \dot{\alpha}_3(t) &= -2(\tau + \gamma)\alpha_3(t) - \tau\alpha_5(t) - \tau\alpha_9, \\ \dot{\alpha}_9(t) &= -2(\tau + \gamma)\alpha_9(t) - \tau\alpha_8(t) - \tau\alpha_{10}(t), \\ \dot{\alpha}_{10}(t) &= -2(\tau + \gamma)\alpha_{10}(t) - \tau\beta_1(t) + \tau\beta_2(t). \end{aligned} \tag{3.49}$$

where  $\alpha_2(t)$  to  $\alpha_{10}(t)$  are some compatible pairs and after throwing away all the  $\beta$ s that are zero at  $t = 0$  we have

$$\begin{aligned} \beta_1 &= \langle S_b I_c \rangle \langle S_a S_1 S_2 S_3 S_b I_c \rangle, & \beta_1(0) &= 1 \\ \beta_2 &= \langle S_b \rangle \langle S_a S_1 S_2 S_3 S_b I_c \rangle, & \beta_2(0) &= 1. \end{aligned} \tag{3.50}$$

### Taylor Polynomial for $\alpha^{SSI}(t)$

We have  $\alpha(0) = 0$ ,  $\alpha^{(1)}(0) = 0$ ,  $\alpha^{(2)}(0) = 0$ ,  $\alpha^{(3)}(0) = 0$ ,  $\alpha^{(4)}(0) = 0$  and  $\alpha^{(5)}(0) = -\tau^4\gamma$ . The Taylor polynomial for  $\alpha^{SSI}(t)$  in this network is therefore

$$\begin{aligned}\alpha^{SSI}(t) &= 0 \times \frac{t^0}{0!} + 0 \times \frac{t^1}{1!} + 0 \times \frac{t^2}{2!} + 0 \times \frac{t^3}{3!} \\ &\quad + 0 \times \frac{t^4}{4!} - \tau^4\gamma \frac{t^5}{5!} + \dots - \alpha^{(n)}(0) \frac{t^n}{n!} \\ &= P_4(t) + E_4.\end{aligned}$$

where  $P_1(t) = 0+0+0+0+0 = 0$  is a zero polynomial of order 4 and the truncation error  $E_4$  is;

$$|E_4| \leq |-\tau^4\gamma| \frac{t^5}{5!}. \quad (3.51)$$

### 3.6.2 Closure for I-S-I

We write

$$\alpha^{ISI}(t) = \langle S_b \rangle \langle I_3 S_b I_c \rangle - \langle I_3 S_b \rangle \langle S_b I_c \rangle$$

for the triple  $\langle I_3 S_b I_c \rangle$ . We differentiate it and substitute relevant terms from its hierarchy of moment equations to obtain

$$\begin{aligned}\dot{\alpha}^{ISI}(t) &= -2(\tau + \gamma)\alpha^{ISI}(t) - \tau\alpha_2(t) - \tau\alpha_3(t), \\ \dot{\alpha}_2(t) &= -2(\tau + \gamma)\alpha_2(t) - \tau\alpha_4(t) - \tau\alpha_5(t), \\ \dot{\alpha}_4(t) &= -2(\tau + \gamma)\alpha_4(t), \\ \dot{\alpha}_5(t) &= -2(\tau + \gamma)\alpha_5(t) - \tau\alpha_6(t) - \tau\alpha_7(t), \\ \dot{\alpha}_6(t) &= -2(\tau + \gamma)\alpha_6(t) + \tau\beta_1(t) - \tau\beta_2(t), \\ \dot{\alpha}_7(t) &= -2(\tau + \gamma)\alpha_7(t), \\ \dot{\alpha}_3(t) &= -2(\tau + \gamma)\alpha_3(t) - \tau\alpha_4(t) - \tau\alpha_9(t), \\ \dot{\alpha}_8(t) &= -2(\tau + \gamma)\alpha_8(t) - \tau\alpha_9(t) - \tau\alpha_{10}(t), \\ \dot{\alpha}_9(t) &= -2(\tau + \gamma)\alpha_9(t) - \tau\alpha_{10}(t), \\ \dot{\alpha}_{10}(t) &= -2(\tau + \gamma)\alpha_{10}(t).\end{aligned} \quad (3.52)$$

### Taylor Polynomial for $\alpha^{ISI}(t)$

We also have that  $\alpha(0) = 0$ ,  $\alpha^{(1)}(0) = 0$ ,  $\alpha^{(2)}(0) = 0$ ,  $\alpha^{(3)}(0) = 0$ ,  $\alpha^{(4)}(0) = 0$  and  $\alpha^{(5)}(0) = \tau^4\gamma$ . The Taylor expansion for  $\alpha(t)$  in this network is therefore;

$$\begin{aligned}\alpha(t) &= 0 \times \frac{t^0}{0!} + 0 \times \frac{t^1}{1!} + 0 \times \frac{t^2}{2!} + 0 \times \frac{t^3}{3!} \\ &+ 0 \times \frac{t^4}{4!} + \tau^4\gamma \frac{t^5}{5!} + \dots + \alpha^{(n)}(0) \frac{t^n}{n!} \\ &= P_4(t) + E_4.\end{aligned}$$

where  $P_1(t) = 0+0+0+0+0 = 0$  is a zero polynomial of order 4 and the truncation error  $E_4$  is;

$$|E_4| \leq \tau^4\gamma \frac{t^5}{5!}. \quad (3.53)$$

We believe that using the first method without the hypothesis, we will obtain the same Taylor's polynomials of (3.46), (3.48), (3.51) and (3.53) for the respective quantities provided the derivatives of the enormous  $\beta$ s involved in these networks can be entirely carried out.

## 3.7 The Trend of $E_n$ and $\beta$

Equations (3.6), (3.31), (3.46) and (3.51) show that the truncation error for approximating  $\alpha^{SSI}(t)$  with zero polynomial in all the network follows a trend that indicate a general representation of

$$E_k \leq -\tau^k\gamma \frac{t^{k+1}}{(k+1)!}$$

where  $k = N - 2$  and  $N$  the number of nodes in the ring. Of course the truncation error  $E_k \rightarrow 0$  for a finite  $t$ , hence  $\alpha^{SSI}(t) \rightarrow 0$  for a finite  $t$ .

Similar observation for  $\alpha^{ISI}(t)$  from equations (3.23), (3.34), (3.48) and (3.53) indicate that a general representation can also be written for the degenerating and converging truncation error  $E_k$  as

$$E_k \leq \tau^k\gamma \frac{t^{k+1}}{(k+1)!}$$

where  $k$  is also as defined above. In figures (3.5) and (3.6),  $E_1$  is a bound for both  $\alpha^{SSI}(t)$  and  $\alpha^{ISI}(t)$  in the triangular network.

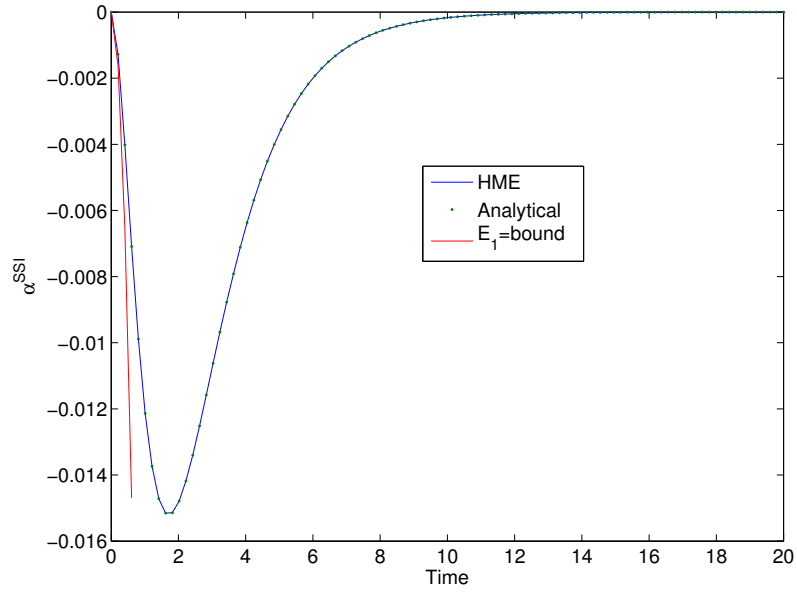


Figure 3.5: For the square network, with  $\tau = 0.4$  and  $\gamma = 0.2$ , we evaluate both  $\alpha^{SSI}(t)$  and  $\alpha^{ISI}(t)$  from the corresponding hierarchy.

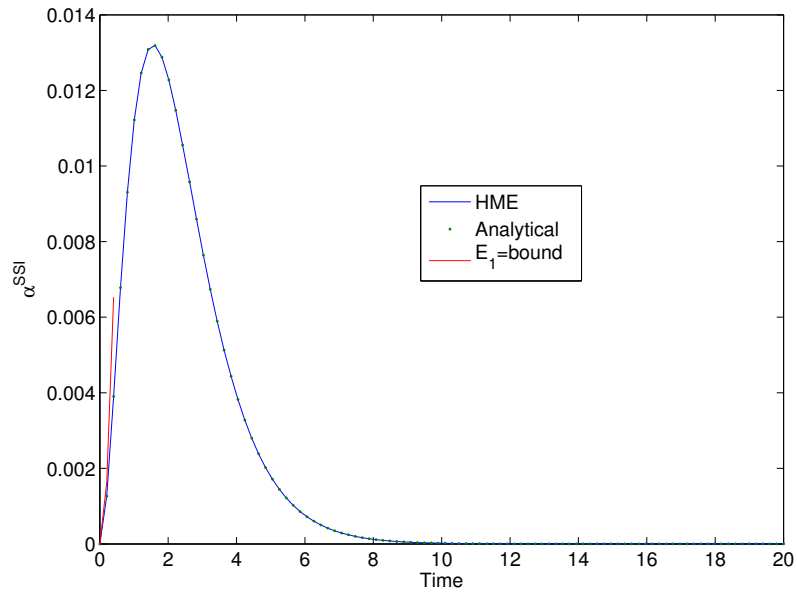


Figure 3.6: For the square network, with  $\tau = 0.4$  and  $\gamma = 0.2$ , we evaluate both  $\alpha^{SSI}(t)$  and  $\alpha^{ISI}(t)$  from the corresponding hierarchy.

Our next intention will be to show that  $\alpha^{SSI}$  and  $\alpha^{ISI}$  can be obtained for any ring of size  $N$  by averaging thousands of simulations and that they are actually bounded by their corresponding  $E_n$ , since the HME for large  $N$  is not feasible.. While numerical issues prevented us from obtaining results for rings of higher order, we can only ascertain the robustness of our simulation process by comparing the values of  $\alpha^{SSI}$  and  $\alpha^{ISI}$  from simulations with that already obtained from the HME for smaller networks - triangle and square. In figures (3.7) and (3.8), the respective average of 200,000 simulations of  $\alpha^{SSI}$  and  $\alpha^{ISI}$  for both graphs seems to compare well with that obtained from their corresponding HMEs.

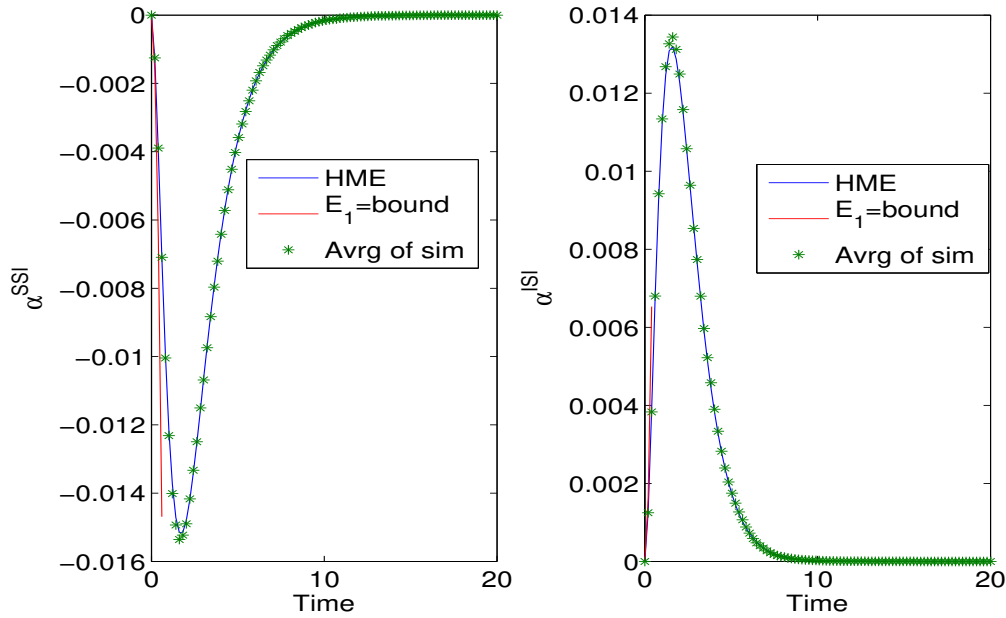


Figure 3.7: The plot of  $\alpha^{SSI}$  and  $\alpha^{ISI}$  from HME and average of 200,000 simulation for the triangular network, with  $\tau = 0.4$  and  $\gamma = 0.2$ .

If we concentrate on the application and result of the hypothesis for approximating the respective  $\alpha^{SSI}(t)$  and  $\alpha^{ISI}(t)$  in the various networks and given the initial state of the system with node  $c$  being infectious we can see a general trend in the evolution of  $\beta_1(t)$  and  $\beta_2(t)$  in all the network. The formation of the  $\beta$ s follows a systematic pattern. From equations (3.38),(3.41), (3.45) and (3.50), the two  $\beta$ 's have the general form

$$\beta_1(t) = \langle S_b I_c \rangle \langle S_a S_1 S_2 S_3 \dots S_m S_b I_c \rangle,$$

$$\beta_2(t) = \langle S_b \rangle \langle S_a S_1 S_2 S_3 \dots S_m S_b I_c \rangle,$$

where  $m$  is the number of susceptible nodes introduced between node  $a$  and  $b$  in the basic triangular network. One of the two terms that make up each of the  $\beta$ s

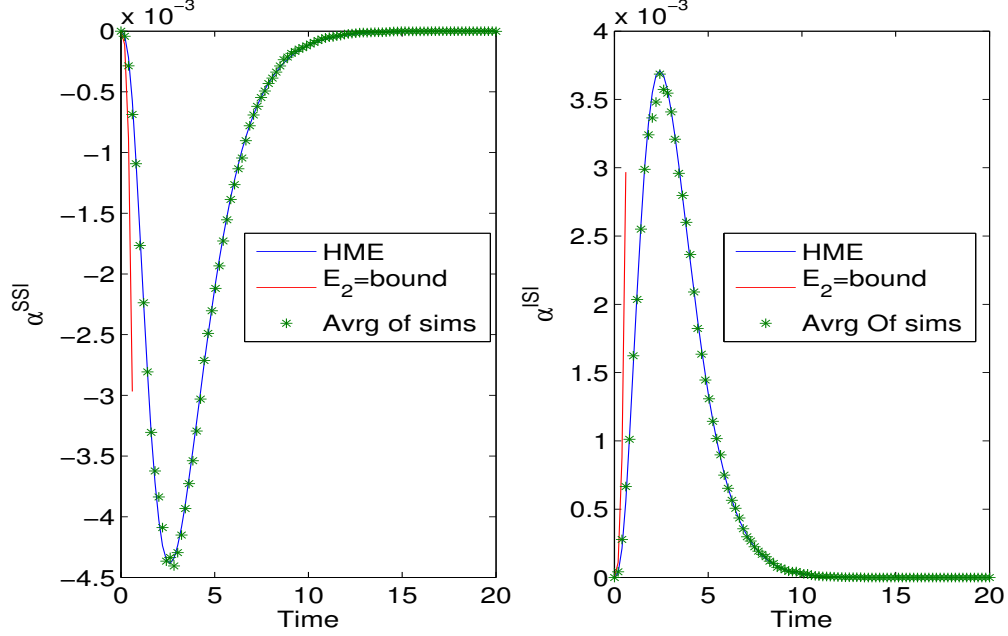


Figure 3.8: The plot of  $\alpha^{SSI}$  and  $\alpha^{ISI}$  from HME and average of 200,000 simulation for the square network, with  $\tau = 0.4$  and  $\gamma = 0.2$ .

have similar form ( $\langle S_b \rangle$  and  $\langle S_b I_c \rangle$ ) in all the network considered with the other term ‘increasing in length’ according to the number of susceptible nodes introduced into the basic triangle.

Fundamentally and from (1.21) we have that for a finite size contact network;

$$\langle S_i I_j \rangle = \sum_k T_{jk} \langle S_i S_j I_k \rangle - \sum_k T_{ik} \langle I_i S_j I_k \rangle - T_{ij} \langle S_i I_j \rangle - \gamma \langle S_i I_j \rangle \quad (3.54)$$

We recall the respective general second order moment closure for the possible triples of  $S$ - $S$ - $I$  and  $I$ - $S$ - $I$  as

$$\alpha^{S_i S_j I_k}(t) = \langle S_j \rangle \langle S_i S_j I_k \rangle - \langle S_i S_j \rangle \langle S_j I_k \rangle,$$

$$\alpha^{I_k S_i I_j}(t) = \langle S_i \rangle \langle I_k S_i I_j \rangle - \langle I_k S_i \rangle \langle S_i I_j \rangle,$$

with node  $S_j$  as the middle term in the triple  $S_i S_j I_k$  and node  $S_i$  as the middle term in the triple  $I_k S_i I_j$ . Expressing these two terms in term of the original triples in (3.54) gives

$$\langle S_i S_j I_k \rangle = \left[ \frac{\langle S_i S_j \rangle \langle S_j I_k \rangle}{\langle S_j \rangle} + \frac{\alpha^{S_i S_j I_k}}{\langle S_j \rangle} \right] \quad \text{and} \quad \langle I_k S_i I_j \rangle = \left[ \frac{\langle I_k S_i \rangle \langle S_i I_j \rangle}{\langle S_i \rangle} + \frac{\alpha^{I_k S_i I_j}}{\langle S_i \rangle} \right]$$



Plugging these terms into (3.54) gives;

$$\begin{aligned}
\langle S_i I_j \rangle &= \sum_k T_{jk} \langle S_i S_j I_k \rangle - \sum_k T_{ik} \langle I_i S_j I_k \rangle - T_{ij} \langle S_i I_j \rangle - \gamma \langle S_i I_j \rangle \\
&= \sum_k T_{jk} \left[ \frac{\langle S_i S_j \rangle \langle S_j I_k \rangle}{\langle S_j \rangle} + \frac{\alpha^{SSI}}{\langle S_j \rangle} \right] - \sum_k T_{ik} \left[ \frac{\langle I_k S_i \rangle \langle S_i I_j \rangle}{\langle S_i \rangle} + \frac{\alpha^{ISI}}{\langle S_i \rangle} \right] \\
&\quad - T_{ij} \langle S_i I_j \rangle - \gamma \langle S_i I_j \rangle
\end{aligned}$$

By collecting like terms we have

$$\begin{aligned}
\langle S_i I_j \rangle &= \underbrace{\sum_k T_{jk} \frac{\langle S_i S_j \rangle \langle S_j I_k \rangle}{\langle S_j \rangle} - \sum_k T_{ik} \frac{\langle I_k S_i \rangle \langle S_i I_j \rangle}{\langle S_i \rangle} - T_{ij} \langle S_i I_j \rangle - \gamma \langle S_i I_j \rangle}_{\text{familiar pair approximation of equation (3.54)}} \\
&\quad + \underbrace{\left[ \sum_k T_{jk} \frac{\alpha^{S_i S_j I_k}}{\langle S_j \rangle} - \sum_k T_{ik} \frac{\alpha^{I_k S_i I_j}}{\langle S_i \rangle} \right]}_{\text{terms representing the error}}
\end{aligned} \tag{3.55}$$

where

$$\sum_k T_{jk} \frac{\alpha^{S_i S_j I_k}}{\langle S_j \rangle} - \sum_k T_{ik} \frac{\alpha^{I_k S_i I_j}}{\langle S_i \rangle},$$

can be described as term quantifying the error in  $\langle S_i I_j \rangle$ .

## 3.8 Conclusion

The presence of loops is a major challenge against defining suitable low dimensional approximate epidemic model that readily matches the results of the corresponding stochastic model. Here, we have investigated and attempted to quantify the error in approximating triples by a second order moment closure for an *SIR* epidemic model on a network with a single *cycle* where the clustering coefficient is zero (that is, no triangle is present) and the average degree distribution is 2.

Proving the exactness of such closure can involve differentiation and integration of the difference of some form of ‘compatible pair(s)’ to show that such difference is consistently zero at all time. We believe the two unusual terms that appeared in this process for ring networks are responsible for the deviation of the closure from being exact and we refer to them as ‘error terms’.

We were able to write a representation for the error terms for cycle with 3-nodes because the sets of differential equations here is of lower order. We validated this representation with that computed from the numerical solution of the hierarchy of moment equations. To go forward beyond the challenge posed by the large number

of ‘error terms’ involved for cycles of higher order and the interdependency of their derivatives, we stated an hypothesis which, although it is not proved in this study, helped us to extrapolate results from cycles of lower to those of higher orders.

We were also able to show that the representation of the error term can be approximated by a zero polynomial for *cycle* of finite size with the truncating error for this approximation as the upper bound. We observe that while network size increases, the error rapidly decreases which is exactly what we are predicting and this is consistent with what we observe numerically. A reason why we were struggling going beyond  $N = 4$ .

Unfortunately, we can not say much about the error quantity of equation (3.55) at the moment and we also hope that the Taylor’s expansion hypothesis (**TEH**) will be rigorously proved presumably by induction method in future studies. We recall **TEH** implies that all  $\beta_i(0) = 0$ ,  $i = 1, 2, 3, \dots, N$  where

$$\beta_i \neq \langle S_b I_c \rangle \langle S_a S_1 S_2 S_3 \dots S_m S_b I_c \rangle \quad \text{and} \quad \langle S_b \rangle \langle S_a S_1 S_2 S_3 \dots S_m S_b I_c \rangle.$$

Using the formalism of chapter 2, we have shown in both the triangular and square networks that  $\dot{\beta}_i(t) = 0$  and that  $\beta_i(t) = 0$  for all  $t \geq 0$ . If this is true for the square network where  $m = 1$  and it is true for the pentagon network by extension of **TEH** where  $m = 2$ , then it will be nice to show, possibly by induction, that it is true for all  $N$ .

# Chapter 4

## Quasi-Stationary Distribution of the pair-based *SIS* Epidemic model on line graphs

### 4.1 Introduction

The concept of quasi-stationary distributions (QSD) is used to model the behaviour of some stochastic systems which eventually approach an absorbing state but nevertheless appear to be stationary over a reasonable period of time.

For example, in modelling the spread of a computer virus across a network with cure and reinfection, the number of computers infected attains some stationary-like behaviour prior to the total cure of computers in the network [79, 105, 122]. The idea of QSD has been used in this regard to describe the behaviour of the region of this temporary equilibrium for computer virus spread[178].

Another example is provided by chemical reactions in which materials or catalysts can be exhausted (corresponding to an absorbing state) yet the reactions appear to attain a temporary equilibrium. QSDs have been applied in this context to describe and understand the concentration of catalyst in this region of temporary equilibrium [35, 36, 116 121 , 130].

QSDs are also powerful tools in area of wildlife management. They are useful in predicting persistence times, and the distribution of the number of individuals, in animal populations that are subject to large-scale mortality or emigration [133]. Despite the fact that the usual stochastic models predict ultimate extinction, these populations can be amazingly resilient. The application of QSDs in wildlife management has been investigated in several papers [63, 86, 98, 118, 129, 145].

The time it takes for some absorbing stochastic process to absorb can sometimes be long. The time evolutions of the probability of the states in an absorbing Markovian process during this temporary equilibrium state can be approximated by the probability distribution under the condition that absorption has not taken place. The limit of the distribution of the probabilities of states conditioned on non-absorption is the QSD of the system.

Using the Frobenius theorem, Mandl (1960) [94] proved that for all initial distributions, excluding the absorbing state, a unique and positive stationary distributions exists for a discrete time Markov Chain that has a finite state space. Based on this fact, Cavender [23] amongst many researchers [19, 87, 115, 117, 167] on the QSD, used the Birth-and-Death process to model a finite biological populations in order to investigate and then establish the existence and uniqueness of stationary distributions of, among many variables, the population size(s) at time  $t$  conditioned on non-extinction.

Darroch and Seneta [39] among others [107, 115, 117], considered the QSD of absorbing discrete-time Markov chains with discrete finite state space and extended this concept to the continuous-time Markov chain, giving a precise relationship between the quasi-stationary distributions and the transition matrix.

The QSD does not necessarily exist for infinite state systems [115]. On the assumption that absorption is certain, Seneta and Vere-Jones [148], using some matrix properties and invariant distributions of transition matrix [128, 131, 132, 149, 150], extended Mandl proof by developing infinite-dimensional Frobenius theorems to find a large class of initial conditions under which a positive QSD exists for discrete time discrete *infinite* states space system. By this, the QSD of a number of stochastic process with infinite state space, such as the branching process have been studied.

The **SIS** model is considered as one of the simplest stochastic models for endemic infections [107]. This model allows an individual to fluctuate between two infection states: Susceptible and Infectious. Recovery from infection is immediately followed by replenishing the susceptible pool. The deterministic model which was introduced by Ross [141] leads to a logistic curve with the value of  $R_0$  determining the persistence or extinction of the disease from a population. There is an equilibrium state of infection if  $R_0 > 1$  and extinction if  $R_0 < 1$  for all positive initial fraction of infected individuals.

The Stochastic **SIS** model which was introduced by Weiss and Dishon [177] is a continuous time Markov birth-and-death process with an absorbing state. Studies

[6, 7, 115] established that for  $R_0$  greater than unity, time to extinction of the stochastic *SIS* epidemic dynamics increases exponentially with population size and can be extraordinarily large for small population size. The time to absorption can also be very small and is independent on population size for small value of  $R_0$ [107]. In the end, extinction of infection from the population is certain, regardless of the value of  $R_0$ .

Earlier studies of the QSD of endemic infections have concentrated on Markovian mean-field models-especially **SIS** dynamics. Kriscio and Lefevre[87] used a conditional birth-and-death process of a fixed population to approximate the QSD of Markovian **SIS** epidemic dynamics in a closed population. Norden[115] also considered Markovian **SIS** epidemic dynamics in a closed population to investigate the distribution of the extinction times from both the numerical and the theoretical standpoint. Nassel[109] in the same manner considered the **SIS** epidemic dynamics, using the concept of QSD, to account for the influence of epidemic and demographic forces on the time to extinction of recurrent epidemics in a population.

Here, we investigate the average of the QSD of Markovian **SIS** epidemic dynamics on networks. The study is first approached by considering line graphs with few nodes where the master equation can feasibly be written for the dynamics of the epidemic system. The solutions of these systems can readily be compared with simulation, but then, we have the problem where bigger networks cannot be analysed due to the sheer size of the state spaces and subsequently the higher dimensional sets of equations that are not numerically feasible.

We progress to adopt the ideas of the earlier chapters by considering the pair-approximation of the **SIS** epidemic model conditioned on non-extinction to approximate the process of the Master equation dynamics and compare the results with the QSD from averages of many simulations to validate its accuracy with the results from the master equation for smaller networks.

We then advance to consider this pair-approximation model for larger networks and compare the results with average of simulations for considerably bigger networks to see if this model can approximately describe the average of the QSDs for more complex networks. This also enable us to gain better understanding of the pair-approximation of the **SIS** dynamics.

## 4.2 Defining the QSD for SIS dynamics on a Network

For stochastic *SIS* epidemic dynamics on an arbitrary graph of finite size  $N$ , an individual fluctuates between two infection status, i.e susceptible and infectious. We suppose that an individual  $i$  is susceptible with probability  $\langle S_i \rangle$  or infectious with probability  $\langle I_i \rangle$ ,  $i \in \{1, 2, \dots, N\}$ . Infection and subsequent recovery are both Poisson processes with respective rates

$$\lambda_i = \sum_{j=1}^N T_{ij} S_j I_i \quad \text{and} \quad \mu_j = \gamma_j I_j,$$

where  $T_{ij}$ ,  $\lambda_i$  and  $\mu_i$  are as defined in section (1.7.3). Here, an infected individual after recovery immediately become susceptible.

Let  $\Gamma$  be the state space of the system and let  $P_\alpha(t)$  denote the probability that the system is in state  $\alpha$  at time  $t$ , assuming an initial distribution  $\{P(0)\}$ . Let  $P_\sigma(t)$  be the probability that the system is in the absorbing state,  $\sigma = S_1 S_2 \dots S_N$ , representing the all-susceptible state which is the only absorbing state for the system.

Let  $P^*(t)$  represent the vector which contain all the component of  $P(t)$  except the first element,  $P_\sigma(t)$ , which corresponds to the probability distribution of the ground state,  $\sigma$ . The QSD is defined as the probability of states conditioned on non absorption. Let us assume  $P_\sigma(0) < 1$ ; that is the probability of the system being initiated at the ground state is not ‘certain’ and define that for any state  $\alpha$

$$q_\alpha(t) = \frac{P_\alpha^*(t)}{1 - P_\sigma(t)}. \quad (4.1)$$

Thus,  $q_\alpha(t)$  will be used to describe the distributions of the conditioned system and

$$q_x = \lim_{t \rightarrow \infty} q_\alpha(t),$$

is the QSD of the system. This is unique for any transient initial condition as shown by Darroch and Seneta [39].

Since we are dealing with a continuous time Markov chain, the time evolution of the probabilities of the system states can be represented by the Master equation, a gain-loss equation

$$\dot{P}_\alpha(t) = \sum_{\beta} [Q_{\alpha\beta} P_\beta(t) - Q_{\beta\alpha} P_\alpha(t)],$$

or

$$\dot{P}_\alpha(t) = QP_\alpha(t). \quad (4.2)$$

where  $Q_{\alpha\beta}P_\beta(t)$  is the gain due to transmission from other states  $\beta$  to state  $\alpha$ , and  $Q_{\beta\alpha}P_\alpha(t)$  is the loss due to transmission from  $\alpha$  into other states  $\beta$ .

We emphasize  $q_\alpha(t)$  is the probability that at time  $t$  the system is in state  $\alpha$ , conditional on it not being in state  $\sigma$  and that  $q_\alpha(t \rightarrow \infty) = q$  is the QSD. It is essential to note that the system of differential equations for  $P^*(t)$  is closely related to the system in equation (4.2) [107]. That is

$$\dot{P}_\alpha^*(t) = AP_\alpha^*(t), \quad (4.3)$$

where  $A$  is the submatrix obtained from matrix  $Q$  by deleting the first row and the first column which respectively correspond to the transmission rates into and out of the ground state,  $\sigma$ .

The QSD can be evaluated by looking at the stationary behaviour of equation (4.1). Differentiating equation (4.1) using product rule gives

$$\begin{aligned} \dot{q}_\alpha(t) &= \frac{d}{dt} \left\{ \frac{P_\alpha^*(t)}{1 - P_\sigma(t)} \right\} \\ &= \frac{\dot{P}_\alpha^*(t)}{1 - P_\sigma(t)} + \frac{P_\alpha^*(t)\dot{P}_\sigma(t)}{(1 - P_\sigma(t))^2} \\ &= \frac{AP_\alpha^*(t)}{1 - P_\sigma(t)} + \left[ \frac{P_\alpha^*(t)}{1 - P_\sigma(t)} \right] \left[ \frac{\dot{P}_\sigma(t)}{1 - P_\sigma(t)} \right] \\ &= Aq_\alpha(t) + q_\alpha(t) \frac{\dot{P}_\sigma(t)}{1 - P_\sigma(t)}. \end{aligned} \quad (4.4)$$

That is;

$$\dot{q}_\alpha(t) = q_\alpha(t)A + \frac{\dot{P}_\sigma(t)}{1 - P_\sigma(t)}q_\alpha(t). \quad (4.5)$$

For stationarity,  $\dot{q}_\alpha(t) = 0$  so we therefore have

$$q_x A = \lambda q_x. \quad (4.6)$$

where, at stationarity we have:

$$\lambda = \lim_{t \rightarrow \infty} \frac{\dot{P}_\sigma(t)}{P_\sigma(t) - 1} = \lim_{t \rightarrow \infty} \frac{\sum_{j=1}^N Q_{1,j} P_j(t)}{P_\sigma(t) - 1}.$$

The process above is a general case for an arbitrary contact network.

From established results [39, 107, 130, 133] for the existence of a limiting proba-

bility distribution for discrete state space models, we know that  $q_\alpha(t) \rightarrow q_x$  for each  $\alpha \in \Gamma$ , regardless of the state  $\alpha$  in which the system is initiated. Moreover, each  $q_\alpha$  is positive. From (4.6), the QSD is a left eigenvector of the matrix  $A$  corresponding to the eigenvalue  $\lambda$ . In this case,  $q_x$  is the normalized eigenvector corresponding to the maximum (or the leading) eigenvalue of  $A$ . An example of the QSD evaluated in this manner is in Markovian reliability models where the stationary distribution of the number of functioning units (conditioned on the system not having failed) is obtained as the dominant left eigenvector of the transition-rate matrix restricted to the transient states [115, 117]. This value can be evaluated numerically for a system with reasonably small number of states such as the ones considered in the next section.

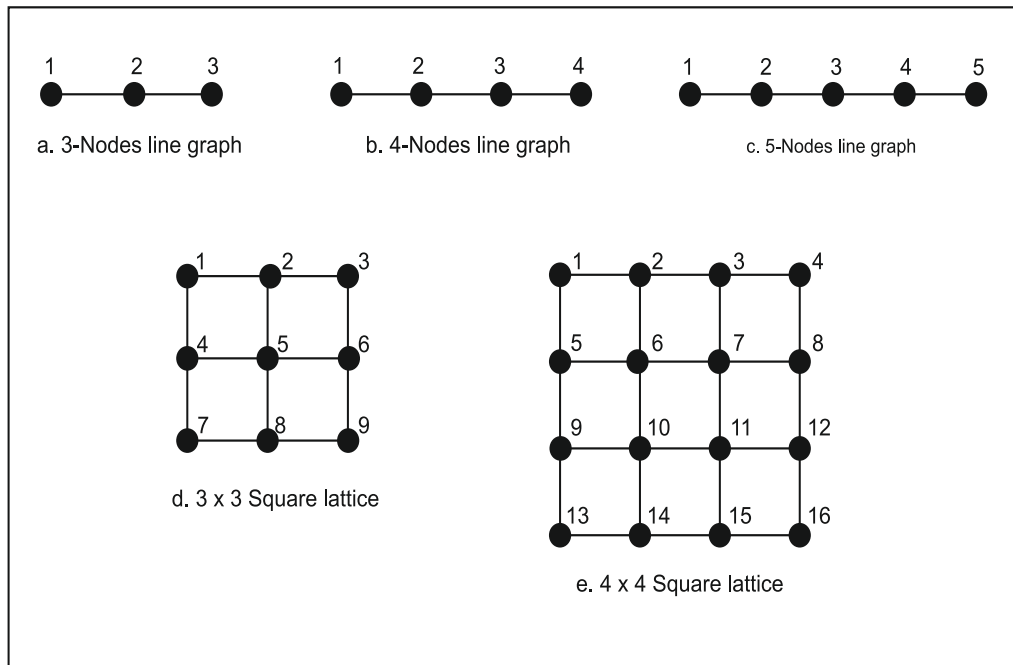


Figure 4.1: *Simple networks considered in this study*

### 4.3 Calculating the QSD on a lattice

We begin our study by giving a step-by-step analysis of the methodology by treating examples of **SIS** epidemic dynamics on line graphs with few nodes. This enables us to write down low-dimensional set of equations which are mathematically tractable. We assume a constant transmission rate,  $\tau$  across all links and a constant recovery



rate  $\gamma$  for an infected individual. We start by writing down the corresponding transition rates matrix for each of the simple networks considered and then evaluate the QSD using the master equation, following the process of equation (4.2) through equation (4.6) as explained in the preceding section.

We start with the 3-nodes line graph of figure 4.1(a) with the state space given as

$$\Gamma = \{S_1S_2S_3, S_1S_2I_3, S_1I_2I_3, S_1I_2S_3, I_1S_2S_3, I_1I_2I_3, I_1S_2I_3, I_1I_2S_3\}.$$

Note that the number of states for each network in our case is  $2^k$  where  $k$  is the number of nodes in the network. In the case of the 3-nodes graph presently considered, the number of states is  $2^k = 8$ , where  $k = 3$ .

The corresponding matrix of transition rates  $Q$  for this system in (4.2) is

$$Q = \begin{bmatrix} 0 & \gamma & \gamma & \gamma & 0 & 0 & 0 & 0 \\ 0 & a & 0 & 0 & \gamma & \gamma & 0 & 0 \\ 0 & 0 & b & 0 & \gamma & 0 & \gamma & 0 \\ 0 & 0 & 0 & a & 0 & \gamma & \gamma & 0 \\ 0 & \tau & \tau & 0 & c & 0 & 0 & \gamma \\ 0 & 0 & 0 & 0 & 0 & 2a & 0 & \gamma \\ 0 & 0 & \tau & \tau & 0 & 0 & c & \gamma \\ 0 & 0 & 0 & 0 & \tau & 2\tau & \tau & -3\gamma \end{bmatrix}$$

where  $a = -(\tau + \gamma)$ ,  $b = -(2\tau + \gamma)$  and  $c = -(\tau + 2\gamma)$ .

We numerically solve equation (4.2) for this system by starting the system in a pure state,  $S_1S_2I_3$  with  $\tau = 0.3$  and  $\gamma = 0.1$ . Figure 4.2(a) shows the distributions of states with the probabilities of the ground state  $S_1S_2S_3$  increasing from 0 to 1, indicating final absorption of the system to the ground state while the distributions of other states initially increase and then decrease to zero in the long run. The expected number of infective of the system which can be calculated as

$$[I](t) = \sum_{\alpha} I(\alpha)P_{\alpha}(t), \quad (4.7)$$

is shown in figure 4.2(b), where  $I(\alpha)$  is the number of infected individuals in state  $\alpha \in \Gamma$  and  $P_{\alpha}(t)$  is the corresponding probability distribution for state  $\alpha$ . Having initiated the system in state,  $S_1S_2I_3$ , the number of infective as shown in figure 4.2(b) initially increases from 1, peaks and then decreases to zero indicating extinction of infection in the system.

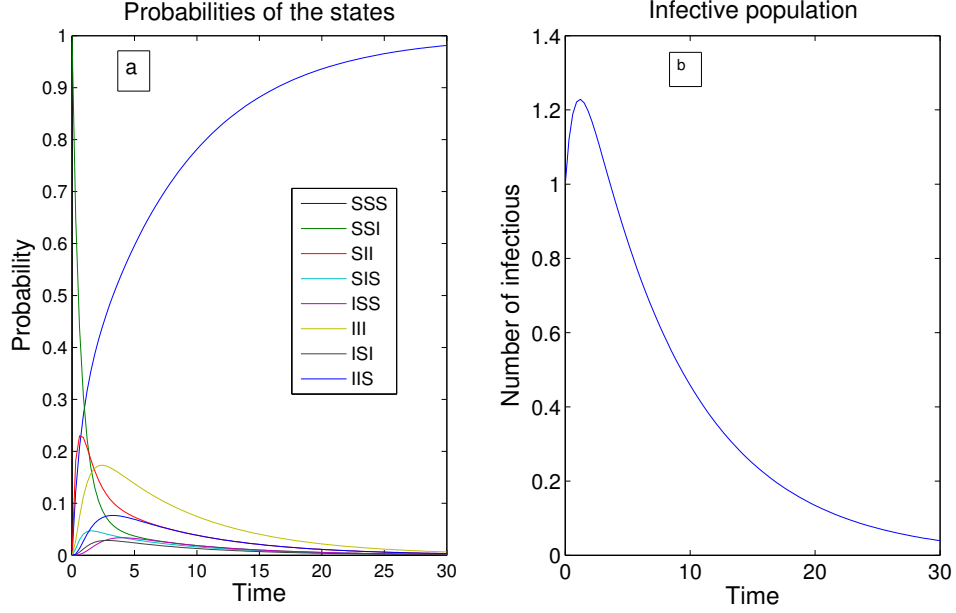


Figure 4.2: (a) The Probability distribution of states and (b) the corresponding infection time series of **SIS** epidemic dynamics on a 3-node line graph with  $\tau = 0.3$ ,  $\gamma = 0.1$  and initiating the system in state  $S_1S_2I_3$

We now proceed to evaluate the distributions across states of this same system in the QSD. We start by partitioning  $\Gamma$ , writing  $\Gamma$  as  $\Gamma = \{S_1S_2S_3\} \cup \{\Gamma'\}$  where

$$\Gamma' = \{S_1S_2I_3, S_1I_2I_3, S_1I_2S_3, I_1S_2S_3, I_1I_2I_3, I_1S_2I_3, I_1I_2S_3\}. \quad (4.8)$$

We comment here that the corresponding submatrix  $A$ , which appears in equations (4.3) to (4.6) for this system is a 7 by 7 matrix which is obtained by deleting the first row and column of matrix  $Q$  above. These entries correspond to the flow rates of states into and out of the ground state,  $\sigma = S_1S_2S_3$ .

The values of  $q_\alpha(t)$  for this system which are derived from equation (4.1) are plotted in figure 4.3(a). Here, state  $I_1I_2I_3$  is the most likely in the QSD. The expected number of infective for this system which can be written as

$$[I^*](t) = \sum_{\alpha \neq \sigma} I(\alpha)q_\alpha(t). \quad (4.9)$$

can be seen in the long-term behaviour of figure 4.3(b).  $I(\alpha)$  is the number of infectious individuals in state  $\alpha \in \Gamma'$  and where  $q_\alpha(t)$  the corresponding probability distributions.

The stationary behaviour can also be determined by equation (4.6). Specifically, the maximum eigenvalue for this truncated matrix is  $-0.016$  and its scaled

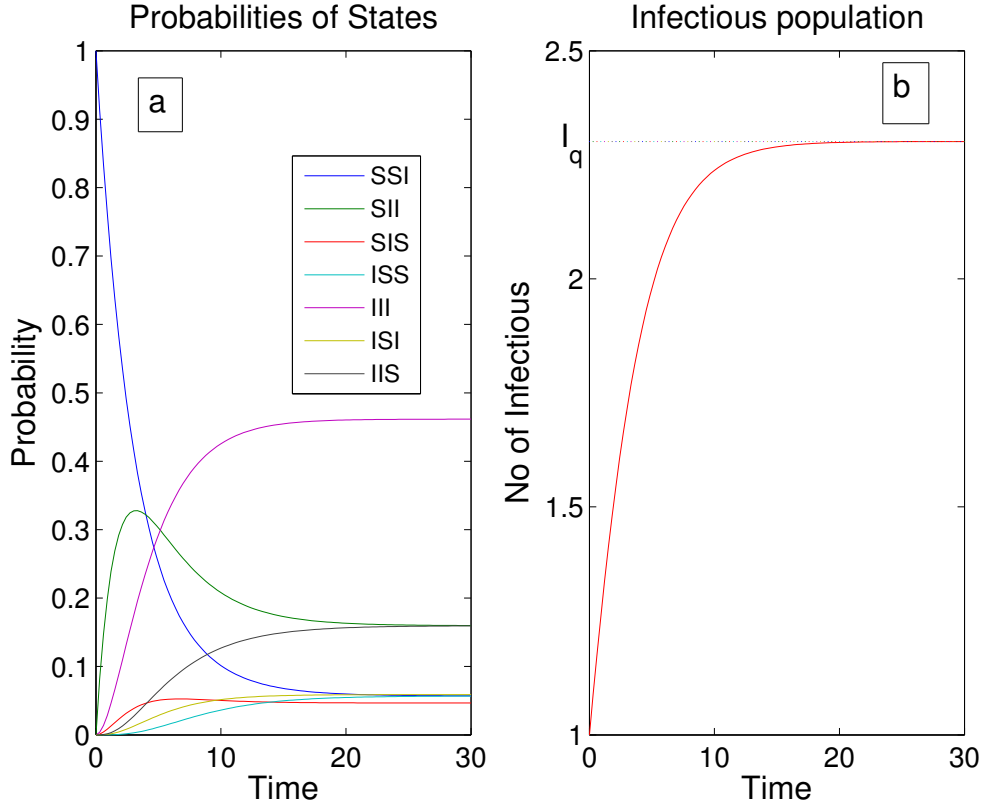


Figure 4.3: (a) The quasi stationary distribution comprising of the seven transient states and (b) the expected number of infectious individuals in the conditioned **SIS** epidemic dynamics on a 3-nodes line graph with  $\tau = 0.3$ ,  $\gamma = 0.1$  and initiating the system in state  $S_1S_2I_3$ .

eigenvector  $q_x$ , is

$$q_x = \begin{bmatrix} 0.0569 \\ 0.1596 \\ 0.0467 \\ 0.0560 \\ 0.4616 \\ 0.0589 \\ 0.1596 \end{bmatrix}$$

in the order given in equation (4.8). This coincides with the long-term behaviour in figure 4.3(a). The expected number of infective  $I_q$  (marked in figure 4.3(b)), in this stationary state can alternatively be estimated from the eigenvector as  $I_q = \sum_{\alpha \neq \sigma} I(\alpha)q_x = 2.3011$  where  $I(\alpha) = [1 \ 2 \ 1 \ 1 \ 3 \ 2 \ 2]$  for this case.

We can also compute the QSD directly from stochastic simulation. To do this, we run many simulations from the same initial state (any state except  $\sigma$ ). The mean of these simulations is taken over time, but when fade-out occurs for a given

simulation at time  $t_f$ , it is not included in the average after  $t_f$ . The plots for the average of the QSD from 1,000,000 simulations is identical to the Master equation results in figure 4.3(b) within the resolution of the figure. We compare extensively with this simulation method in the following two sections.

## 4.4 Approximations for the QSD

While it is feasible to use the Master equation to determine the QSD for small networks, for larger networks this is not feasible. Here we investigate the possibility of using either individual or pair-level approximation method to approximate the expected infection time series in the QSD.

Firstly note that from equations (4.1), (4.7) and (4.9)

$$\begin{aligned} [I^*](t) &= \sum_{\alpha \neq \sigma} I(\alpha) q_\alpha(t) \\ &= \frac{\sum_{\alpha \neq \sigma} I(\alpha) P_\alpha^*(t)}{1 - P_\sigma(t)} \\ &= \frac{\sum_{\alpha} I(\alpha) P_\alpha(t)}{1 - P_\sigma(t)} \\ &= \frac{I(t)}{1 - P_\sigma(t)} \end{aligned}$$

That is

$$[I^*](t) = \frac{[I](t)}{1 - P_\sigma(t)}, \quad (4.10)$$

consequently

$$I_q = \lim_{t \rightarrow \infty} \frac{[I](t)}{1 - P_\sigma(t)}. \quad (4.11)$$

Our objective is therefore to approximate  $[I](t)$  and  $P_\sigma(t)$  by investigating the individual and pair-approximations of the Markovian **SIS** epidemic dynamics.

### 4.4.1 Individual-approximation

For any transmission matrix  $T$  as defined in section (1.7.3) and any pair of nodes  $i$  and  $j$ , the stochastic individual based **SIS** epidemic dynamic[60] is consistent with system (4.12)

$$\begin{aligned} \langle \dot{S}_i \rangle &= - \sum_{j=1}^N T_{ij} \langle S_i I_j \rangle + \gamma_i \langle I_i \rangle, \\ \langle \dot{I}_i \rangle &= \sum_{j=1}^N T_{ij} \langle S_i I_j \rangle - \gamma_i \langle I_i \rangle. \end{aligned} \quad (4.12)$$

We consider its individual approximation by approximating the probabilities of pairs as being statistically independent of the probabilities of constituent individuals; that is,

$$\langle S_i I_j \rangle = \langle S_i \rangle \langle I_j \rangle,$$

and we write system (4.12) as

$$\begin{aligned} \langle \dot{X}_i \rangle &= - \sum_{j=1}^N T_{ij} \langle X_i \rangle \langle Y_j \rangle + \gamma_i \langle Y_i \rangle, \\ \langle \dot{Y}_i \rangle &= \sum_{j=1}^N T_{ij} \langle X_i \rangle \langle Y_j \rangle - \gamma_i \langle Y_i \rangle, \end{aligned} \tag{4.13}$$

with  $\langle X_i \rangle$  and  $\langle Y_i \rangle$  as the respective approximations for  $\langle S_i \rangle$  and  $\langle I_i \rangle$ , and where as in earlier chapters, notations such as  $\langle A_i B_j \rangle$  represents the time dependent probability of individual  $i$  being in state  $A$  and individual  $j$  being in state  $B$  in the pair  $A_i B_j$ . The dot notation here is the derivative of quantity with respect to time.

We define the infection time series of system (4.13) as

$$[I](t) \approx [X_a](t) = \sum_{i=1}^N \langle X_i \rangle,$$

and approximate the probability,  $P_\sigma(t)$  of the ground state  $\langle S_1 S_2 \dots S_N \rangle$  as

$$P_\sigma(t) \approx \mathbb{S}(t) = \prod_{i=1}^N \langle S_i \rangle.$$

The average number of infectious individuals conditioned on non-extinction is therefore approximated as

$$[I_*](t) \approx [X_*](t) = \frac{[X_a](t)}{1 - \mathbb{S}(t)}. \tag{4.14}$$

And the corresponding average of the QSD is obtained as

$$[I_q] = \lim_{t \rightarrow \infty} \frac{[X_a](t)}{1 - \mathbb{S}(t)}. \tag{4.15}$$

The infection time series of equation (4.14) for all the line graphs considered appear not consistent with the estimate from the corresponding Master equation and averages of simulations in figure (4.4).

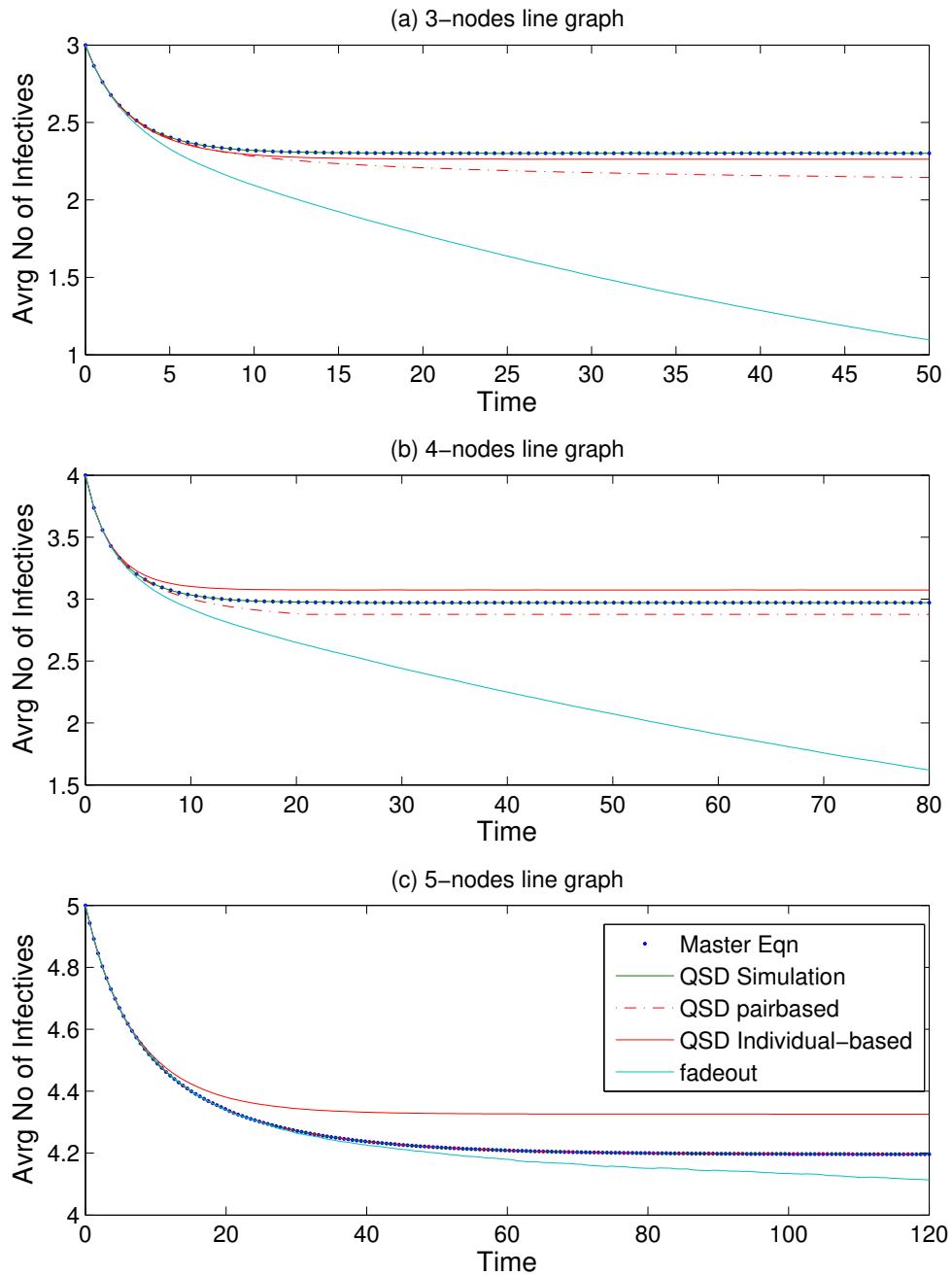


Figure 4.4: The average number of infectious individuals as obtained from the Master equation, the conditional individual and pair based model and the average of 1,000,000 simulations for the **SIS** epidemic dynamics on (a) 3-nodes, (b) 4-nodes and (c) 5-nodes line graphs with  $\tau = 0.3$  and  $\gamma = 0.1$ .

## 4.4.2 Pair-approximation

For the pair-based model, stochastic **SIS** epidemic dynamics are consistent with system (4.16), for any transmission matrix  $T$  and any pair of nodes  $i$  and  $j$  [60];

$$\begin{aligned}
\langle \dot{S}_i \rangle &= - \sum_{j=1}^N T_{ij} \langle S_i I_j \rangle + \gamma_i \langle I_i \rangle, \\
\langle \dot{I}_i \rangle &= \sum_{j=1}^N T_{ij} \langle S_i I_j \rangle - \gamma_i \langle I_i \rangle, \\
\langle \dot{S}_i I_j \rangle &= \sum_{k=1, k \neq i}^N T_{jk} \langle S_i S_j I_k \rangle - \sum_{k=1, k \neq j}^N T_{ki} \langle I_k S_i I_j \rangle - (T_{ij} + \gamma_j) \langle S_i I_j \rangle, \\
\langle \dot{S}_i S_j \rangle &= - \sum_{k=1, k \neq j}^N T_{ik} \langle I_k S_i S_j \rangle - \sum_{k=1, k \neq i}^N T_{jk} \langle S_i S_j I_k \rangle.
\end{aligned} \tag{4.16}$$

Following the process of pair-approximation adopted in earlier chapters, we close the system at the level of pairs with a second order moment closure of equation (1.22), that is

$$\langle A_i B_j C_k \rangle \approx \frac{\langle A_i B_j \rangle \langle B_j C_k \rangle}{\langle B_j \rangle}.$$

which expresses the probabilities of triples in terms of that of pairs to obtain a lower-dimensional system of differential equations (4.17).

$$\begin{aligned}
\langle \dot{X}_i \rangle &= - \sum_{j=1}^N T_{ij} \langle X_i Y_j \rangle + \gamma_i \langle Y_i \rangle, \\
\langle \dot{Y}_i \rangle &= \sum_{j=1}^N T_{ij} \langle X_i Y_j \rangle - \gamma_i \langle Y_i \rangle, \\
\langle \dot{X}_i Y_j \rangle &= \sum_{k=1, k \neq i}^N T_{jk} \frac{\langle X_i X_j \rangle \langle X_j Y_k \rangle}{\langle X_j \rangle} - \sum_{k=1, k \neq j}^N T_{ik} \frac{\langle Y_k X_i \rangle \langle X_i Y_j \rangle}{\langle X_i \rangle} \\
&\quad - (T_{ij} + \gamma_j) \langle X_i Y_j \rangle, \\
\langle \dot{X}_i X_j \rangle &= - \sum_{k=1, k \neq j}^N T_{ik} \frac{\langle Y_k X_i \rangle \langle X_i X_j \rangle}{\langle X_i \rangle} - \sum_{k=1, k \neq i}^N T_{jk} \frac{\langle X_i X_j \rangle \langle X_j Y_k \rangle}{\langle X_j \rangle}.
\end{aligned} \tag{4.17}$$

where  $\langle X_i \rangle$  and  $\langle Y_i \rangle$  also represent approximations of the respective probabilities,  $\langle S_i \rangle$  and  $\langle I_i \rangle$ , etc.

The approximate expected number of infectious individuals for system (4.17) is

given by

$$[I](t) \approx [X](t) = \sum_{i=1}^N \langle X_i \rangle.$$

This system has been tested and established accurate for quantifying the apparent stationary-like behaviour when the rate of absorption is small [60]. However, when fade-out is rapid, equation (4.17) can still exhibit a genuine stationary state and no longer matches numerical simulation. The main reason for this is that equation (4.17) does not properly represent the absorbing state since this would represent a correlation between the states of all individuals in the system. Here we will be conditioning against the absorbing state and so hope to obtain the average of the QSD when absorbing is rapid.

We need to approximate  $P_\sigma(t)$  from our pair-approximation model (4.17). To approximate the all-susceptible state, we make use of a pair-level closure proposed in [149]:

$$P_\sigma(t) \approx \mathbb{S}(t) = \frac{\prod_{j<i}^N G_{ij} \langle X_i X_j \rangle}{\prod_i^N \langle X_i \rangle^{n_i-1}}. \quad (4.18)$$

where  $G$  is the adjacency matrix of the underlying graph ( $G_{ij} = 1$  if  $T_{ij} > 0$  or  $T_{ji} > 0$ , and is zero otherwise) and  $n_i = \sum_j G_{ij}$ , (i.e. the degree of susceptible node  $X_i$ ). Note that notation  $\langle \cdot \rangle$  denotes probability and that  $\langle \sigma \rangle(t)$  is the same as  $P_\sigma(t)$ .

As we have approximated  $[I](t)$  for the Master equation in (4.10), we also need to approximate  $[I](t)$  from our pair-approximation model (4.17). We have

$$[I](t) \approx [X^*](t) = \frac{[X](t)}{1 - \mathbb{S}(t)}. \quad (4.19)$$

And

$$[I_q^*] = \lim_{t \rightarrow \infty} \frac{[X](t)}{1 - \mathbb{S}(t)}. \quad (4.20)$$

We proceed to numerically investigate the accuracy of  $[I_q^*]$  for quantifying the average of the expected number of infectious in the QSD. We first compare (4.20) with the average of the QSD derived from the Master equation and numerical simulation for 3-node, 4-node and 5-node line graphs. These results are shown in figure 4.4 where the same  $\tau$  and  $\gamma$  as in figure 4.2 are used in each and where stochastic simulation results are based on 1,000,000 simulations with an initial condition of all infected.

Firstly we observe no difference at the scale of the graph between numerical result for the pair-approximation and the Master equation results. The infection time series shows that the pair based model seems to perform better with increased



network size. The plots on the line graphs illustrate the problem of fade-out and hence we obtain results much closer to the master equation by conditioning against extinction for both the individual-based and pair-based model. Meanwhile, the pair-approximation seems to adequately capture the solution of the various original systems for the 5-nodes line graph where fade-out is not more pronounced compared to the 3-nodes and 4-nodes line graphs. Inaccuracy is expected to arise from low-order cycles(not relevant in the examples) and the error due to approximation (4.20).

## 4.5 QSD on larger networks

While we have determined the QSD from the Master equation for a line graph of order 5, this already entails a state space of size  $2^5 = 32$ . However, the numerical simulation for pair-approximation has proved to be a robust method for determining the QSD, we therefore make comparison on larger networks between stochastic simulation and pair-approximation, and also obtaining results for the corresponding conditional individual-approximations..

We now consider graphs where it is not visible to compute the QSD from the method of the master equation and compare the infection time series solution of the pair-approximation with simulations. We solved for the pair based infection time series for the 3by3 and 4by4 square lattices of figure (4.1), starting the systems with all initially infected individuals with  $\tau = 0.5$  and  $\gamma = 0.2$  (chosen to result in a significant probability of fade out over the simulation time period) and compare with the corresponding infection time series of the average of 1,000,000 simulations. Figure (4.5) shows the two results from simulations and pair-approximation seem to be on top of each other within the resolution of the graph.

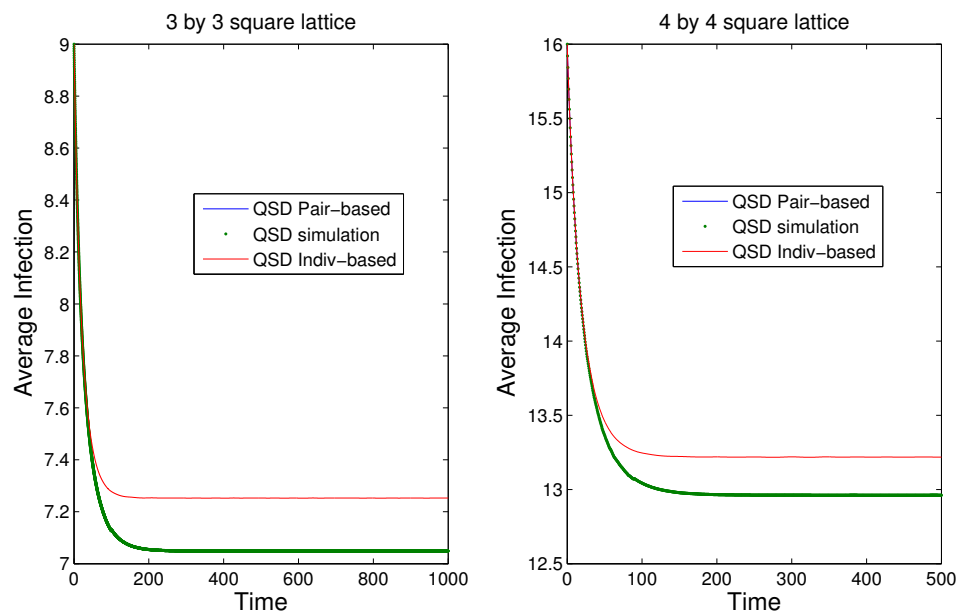


Figure 4.5: The average number of infectious individuals in the quasi stationary distribution of the conditional individual based model, the conditional pair based model and the average of 1,000,000 simulation of the **SIS** epidemic dynamics on 3by3 and 4by4 square lattices. The blue lines, the plots for QSD-pairbased look obscure because it is completely overshadowed by the green dots, the plots for QSD-simulation. The average of the QSD for the pair based model seems to coincide with that obtained from the average of simulations. For the 3by3 lattice, 80 thousand simulations of 1,000,000 simulations (which is 8%) went extinct over the time period shown in the graph while about 6% went extinct for the 4by4 square lattice over the time period shown in the graph.

## 4.6 Conclusion

We have studied, for the first time, the stationary distribution of the average number of infective of Markovian **SIS** epidemic dynamics on networks. We numerically show that the dynamics of an endemic infection can be approximated by the pair-based **SIS** epidemic model conditioned on non-extinction of infection.

We started our analysis by considering **SIS** epidemic dynamics on graphs with few nodes, particularly 3-nodes, 4-nodes and 5-nodes line graphs. These are of particular interest because we saw in the **SIR** case [155], the lack of cycles generate exact model. For each of the line graph, we wrote down the Master equation leading us to compute the QSDs. From the eigenvector perspective, we were able to compute the QSDs and the average number of infective in the QSD for the various line graphs. The results seem identical with simulations.

This process of finding the QSDs from the Master equation and the eigenvector approach is not feasible for bigger networks due to the high dimensionality of equations involved. To go beyond the line graphs, we advanced to approximate the Master equation by investigating the possibility of using either the individual approximation or pair approximation of the **SIS** epidemic dynamics.

For the line graphs earlier considered, we attempted to compare the infection time series from the Master equation with that from both the individual and pair approximation processes. Results showed that there was divergent between the QSD simulations and QSD pair based for the 3-nodes line graph. This is as a result of the error due to the approximation of the probability of the ground state for small populations. The pair-approximation seems to become more accurate with network size while the individual approximation is not giving correct estimate for all the graphs considered.

However, the numerical simulation has proved to be a robust method for determining the QSD, we then make comparison between stochastic simulation and pair-approximation on larger networks. Here, we considered the 3by3 and 4by4 square lattices and results show good performance of the pair-approximation in computing the QSD. While plots on the line graphs explain the issue of fade-out, leading to getting results much similar to the master equation by conditioning for both the individual and pair-based models, the plots on the lattices show the advantage of the pair-based model in describing the average of the QSD over the individual-based model.

Earlier studies of QSD have been on Markovian mean-field, particularly **SIS** epidemic dynamics but we have given the first study of the quasi stationary distribution of the number of infectious individuals on contact networks. It would be of

future interest to also consider the QSD of the **SIS** epidemic dynamics on adaptive networks, although the transmission matrix will no longer be constant.

# Chapter 5

## Discussion and Conclusion

Mean-field epidemic models typically neglect heterogeneity in the interactions among individuals [8, 12, 13, 155]. It is increasingly clear that the assumption of a well-mixed population can be a poor one for the dynamics of many diseases and that connectivity between individuals significantly affects the dynamics of many of infectious diseases. Integrating these interactions into models is therefore important [82, 153].

In this thesis, we considered Markovian epidemic models propagated on contact networks. In particular, we focused on moment closure models evaluated at the level of individual. An entire epidemic system can be built from the perspective of the probability of the infectious status of an individual. The probability of an individual being in an infectious state is expressed in terms of the probability of infectious status of pairs in which the individual is a constituent unit and the probability of infectious status of pair is expressed in terms of the probability of triples in which the pair is a constituent unit, and so on. This process leads to a hierarchy of moment equations which are not closed unless we continue up until the size of the system because lower order quantities are expressed in higher order quantities [64, 153, 154, 155].

For mathematical feasibility, these equations are closed at some level by adopting some form of moment closure relation which approximates the probabilities of higher order quantities in terms of the probabilities of constituent lower order quantities. The systems, so obtained, are usually of lower-dimension compared to a complete Master equation description. While they represent an approximation, in some cases, they can be exact [85, 155, 156].

The moment closure approximation models have proved reliable and helpful for calculating important parameters such as the epidemic peak and duration in epidemics propagated on general networks without relying on full numerical sim-

ulation [18, 22, 40, 46]. In our case, we have concentrated on pair-approximation models. Pairwise models in general have proved very relevant, for example, in investigating the propagation of sexually transmitted diseases where partnership between individuals plays an important role [17, 45, 49, 52].

In chapter 2, we developed at the level of individuals, an *SEIR* epidemic model and investigated the exactness of a second order moment closure of the infection dynamics on tree network, where the transmission and recovery processes are Poisson and with respective constant rates. We considered in particular, a star graph and initiated the epidemic process in a pure state to observe the evolution of the various compatible pairs earlier defined, following the approach of an earlier study [155]. We were able to write and prove a general equation from which the probability of all forms of system states can be obtained for tree graphs and we were also able to establish the exactness of the second order moment closure of the model on trees.

As the structure of contact networks has profound effects on the dynamics of the infectious diseases that are transmitted through it, so also the exactness of the moment closure of epidemic models largely depends on the underlying topology of the contact network [85, 155]. The presence of lower order cycles in networks is often highlighted as a major cause of inaccuracy in these models. In chapter 3, we considered *SIR* epidemic model on contact networks containing a single cycle thereby directly considering the simplest form of these problematic structure. We applied a second order moment closure and set out to quantify a general representation for the error responsible for the deviation of moment closure models from being exact. Considering the approximations for *S-S-I* and *I-S-I* triples, it will be nice to develop expressions to bound the error in the infectious time series. With an hypothesis which was not proved but reserved for further study, we were able to define and obtain expressions for the error terms for large cycles and then show that these error terms are bounded by the approximating Taylor's polynomials.

Earlier studies of the quasi-stationary distributions (QSD) of, especially *SIS* epidemic dynamics, and its approximation models has been on Markovian mean-field models [19, 23, 29, 30, 39, 107, 115, 129]. In chapter 4, we set out to investigate the number of infected individuals in the stationary distribution of infection of *SIS* epidemic dynamics on graphs. We investigated and numerically showed that the number of infected individuals at the stationary stage of the *SIS* epidemic dynamics on networks can approximately be computed by the pair based Markovian *SIS* epidemic dynamics for constant transmission rate across all links and a constant recovery rate for infected individuals.

Having investigated and proved the exactness of a second order moment closure for **SEIR** epidemic dynamics on tree networks, it will be of future interest to extend this study to **SIRS** epidemic model where an infected individual maintain a temporary recovery and then becomes susceptible once again. Probably the flipping of a recovered individual back to the susceptible pool which of course affects the dynamics of infection could also affect the exactness of the second order moment closure on trees.

While investigating errors involved in pair-approximation for **SIR** epidemic dynamics on cycles, we were able to established numerically that the errors involved decrease with network size but needed to propose an hypothesis, the Taylor Expansion Hypothesis (TEH), so that we can write a general approximate representation for the error, given cycle of any size. This hypothesis is not proved here, so it will make sense to consider establishing whether the hypothesis is true. It will also be of interest to extend this study to embedded cycles within graphs.

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