Modeling the Transmission of Infectious Disease in a Dynamic Network

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Abstract. The transmission of infectious disease in epidemiological models usually is based on the assumption that population within random-mixing. Although medical developments can reduce the consequences of the spread of infectious diseases, prevention of plague remains a major toehold. After a model is formulated containing the main fitur the development and transmission of infectious disease, onward to the model can be used to predict, making eradication strategies, control or prevent the spread. Modeling the spread of the disease has the potential to improve the quality of human life. The social life of humans far more complex exceed a diverse population. The transmission dynamics of infectious diseases is sensitive to patterns the interaction between the individual vulnerable (susceptible) and contracted (infectious). Human social contact very heterogeneous group. To predict the impact of this pattern against the transmission of infectious diseases, the use of epidemiological random network model, where the nodes serves individuals exposed, contracting or cured and connectedness presents contact transmission. Type the model spread (epidemic) that examined the model type is exposed, tetular exposed, and cured, or better known as a type of SIRS.

1. Introduction
Outbreaks of infectious diseases such as SARS, avian flu disease, flu pigs occur consecutively in the year 2002, 2003 and 2006 that worry and takes a lot of sacrifice and gives rise to a variety of psychological as well as material losses, making researchers think about the importance of understanding and prediction of the dynamics of the spread of infectious diseases, so that the impact of the spread of the disease can be diminamalisir. Experts and scientists feel has the challenge and the opportunity to continuously explore and discover new knowledge in order to solve this problem. Mathematical scientists including contributed and would like to contribute and donations of thought break down existing problems.

Mathematical model is expected to provide insight into the dynamics of the epidemic and can also be used as a basis for making decisions on policies related to public health, either to reduce the chance of spread of the outbreak as well as stopping the infection. Mathematical models have been used since ages to predict the dynamics of the epidemic spread of infectious diseases as well as to test the proposed control strategy.

Epidemic models can be a simple model consisting of some equation or can also be shaped in a complex model in such case is the model needs to be simulated on a computer. Lately, it appears the debate warmly about the epidemiology mathematic with regard to how detailed the factors that need to be taken include in model epidemics (Smieszek, 2009). A simple model is built based on assumptions and therefore a little more transparent, so that these models can provide a clear
understanding of the factors that may cause against the epidemic. However, if a model represents a very simple fact, likely that these models are less useful when used as a tool to give understanding and forecasting the epidemic. The complex model requires a lot of assumptions and because it looks more realistic and accurate compared to a simple model.

Traditional compartmental SIRS models developed by Kenmack and McKendrick (1927) to describe endemic infections. Let \( N \) be the population size parameter. For \( t \geq 0 \), let \( S(t) \) be the number of susceptible individuals at time \( t \), \( I(t) \) the number of infected individuals at time \( t \), \( R(t) \) the number of recovered individuals until time \( t \), and let \( s(t) = \frac{S(t)}{N} \), \( i(t) = \frac{I(t)}{N} \) and \( r(t) = \frac{R(t)}{N} \). In any time interval \([t, t + \Delta t]\) a susceptible individual is equally likely to come into contact with any of the currently infected individuals In the same time interval, any infected individual may recover and become temporarily immune and an immune individual may lose his immunity and become susceptible. Since \( S(t) + I(t) + R(t) = N \), the pair \((I(t), R(t))\) or \((i(t), r(t))\) completely describes the state of the system at any time \( t \).

Biological systems are likely to be breaking some assumptions which aspects influenced the establishment of the model of the classic disease epidemic compartment, among other things, for example a population consisting of individuals. Therefore the population size can only be changed in discrete (integer with a scale) and on discrete events (birth, death, and others), the initial terms cannot be perfectly didefinisikan, last, not all individuals can interact one with the other. A more complex model can address these deficiencies. A simple stochastic model can cope with discrete scale (with respect to time and population size) and can form a range of different results by implementing the influence of stochastic (random). However, this model still looked all individuals equally and interchangeable. Resulted in two individuals can interact with each other with the same opportunities. On the system of real things like this do not correspond with reality. Most of the interaction, and the transmission of the disease, wants the proximity or at least become more tenuous with the growing distance between two individuals. Within populations, individuals who are sedentary have proximity pattern that can be traced by means of monitor networks of contacts between individuals, called social networks.

Nowadays, mathematical models have been developed to examine the influence of heterogeneity among individuals in the mixing patterns of spread of infectious diseases (Hethcote, 2000). On the various techniques that exist, most network model proposed, especially with regard to social contact. This mendefinisikan interaction between a couple or a group of individuals and memperhitungkannya as the route of transmission of the disease (Meyers et al., 2005; Eubank, 2006; Newman, 2002).

A contact network is increasingly known as the central point for the dynamics of infectious diseases and other transmission phenomena (Lloyd and May, 2001; Barabasi, 2002; Newman et al., 2006). As a result of the structure of a network of contacts, most of the population is fully incorporated in heterogeneous which led to the assumption of action-mass (mass-action) can not be used to describe the spread of epidemic diseases. The results of the development of the spread of the disease in the network has provided a challenge to the formalism is still used extensively about epidemic models based on equation Kermack and McKendrik (1927) (see for example, Morris, 1995; May and Lloyd, 2001); Eames and Keeling, 2002; Newman, 2002). Deterministic model introduced by Ermack and McKendrik, along with most of its derivatives is known as a model of mean-field, or compartment, or mass-action. Because these models use assumptions is mixing a homogenous individuals (Anderson and May, 1992). For a large population, every individual makes contact with the small subpopulations and voices. Then local correlation resulting from transmission within the network is structured so it can not be accommodated by the raw model of mean-field perfectly (Keeling, 1999. a, b).

Mathematical models can estimate the likelihood of a disease outbreak based on the basic reproduction number. The basic reproduction number \( R_0 \) is the average number of secondary cases produced by a “typical” infectious individual during its infectious period [20]. The rate at which infectious individuals spread the disease depends on the number of adequate contacts (the contacts that will result in infection) between infected and susceptible. Several studies have shown that averaging the mixing patterns of heterogeneous populations can cause \( R_0 \) to decrease or remain unaltered [28].
Thus, if we determine the mixing patterns in the population, we can obtain better estimates of $R_0$. This result can help modelers predict the severity of an outbreak and the best means of containing it.

Deterministic model, also known as the Kompartmental model categorizes individuals into different subgroups (Compartment). For example, individuals categorized into three subgroups are mutually exclusive; vulnerable subgroups (Susceptibles), subgroup of infection (Contracting (infektives) and subgroup are moved (removed) that represent individuals who died of the disease, recover from infection, and immunity were fixed or individuals who have been set apart from the rest of the population. Most of the models that describe the behavior of the infectious disease, which has been used up to now, is deterministic. Because this model requires only a little data, and relatively easier to implement it.

This paper shows that it is possible to approximate the main features of disease spread in networks with simple mean-field compartmental models by constructing the transmission rate from individual level parameters to implicitly include some effects of network structure on disease dynamics. Specifically, we propose that the basic reproductive number $R_0$, which is typically derived from the model, be considered instead as a fundamental parameter to construct the equations from.

2. Basic Theory of Epidemic Dynamics

2.1. Reproductive Number and Time Evolution

Epidemiologic modeling over the transmission of infectious diseases are increasingly influential on the theory and practice of the handling and control of the disease. Mathematical modeling on the spread of infectious diseases has become part of the epidemiological policy decision-making in many developed countries, including the United Kingdom, the Netherlands, Canada, and the United States. The modeling approach thus becomes very important for decision making about program control of infectious diseases, in this form of public health interventions. A basic parameter in epidemiology is the basic reproductive number $R_0$, which counts the number of secondary infected cases generated by one primary infected individual. Under the assumption of the homogeneous mixing of the population if an infected individual is in contact with another individual, the basic reproductive number is defined as

$$R_0 = \frac{\lambda \langle k \rangle}{\mu}$$

where $\lambda$ is the spreading rate, defined as the probability rate that a susceptible individual in contact with an infected individual will contract the disease, and $\mu$ is the recovery rate of infected individuals, either to the susceptible or the recovered states. It is easy to understand that any epidemic will spread across a non-zero fraction of the population only for $R_0 > 1$. In this case the epidemic is able to generate a number of infected individuals larger than those which are recovered, leading to an increase of the infected individuals $i(t)$ at time $t$ following the exponential form

$$i(t) = i_0 e^{\lambda t}$$

Here $i_0$ is the initial density of infected individuals and $\tau_d$ is the typical outbreak time that in general reads as ([2])

$$\tau_d^{-1} = \mu (R_0 - 1)$$

The previous considerations lead to the definition of a crucial epidemiological concept, namely the epidemic threshold. Indeed, if the spreading rate is not large enough to allow a reproductive number larger than one ($\lambda > \mu \langle k \rangle$), the epidemic outbreak will not affect a finite portion of the population and dies out in a finite time. In epidemiological studies for which the two important assumptions of “homogeneous mixing” and constant infectiousness (constant $\lambda$) are made, the spreading pattern of the epidemics is therefore controlled by the generation time scale $1/\lambda$ and $R_0$ and there are roughly three different stages in an epidemics [29]. More precisely, when infectious individuals are introduced in a network, one observes a first noisy phase followed in general by an exponential outbreak of the
epidemics. Depending on the long term behavior of individuals against the disease we will observe at large times a different behavior described by the specific epidemic model used.

The above considerations and parameters are at the core of several epidemic models based on the compartmentalization of the population. In other words, each individual of the population can only exist in a certain number of discrete states such as susceptible, infected or permanently recovered. The latter state is equivalent to the removal of the individual from the population since it is supposed that it cannot get the infection anymore. The total population $N$ is assumed to be constant and if $S(t)$, $I(t)$ and $R(t)$ are the number of susceptible, infected and removed individuals at time $t$, respectively, then $N = S(t) + I(t) + R(t)$. The simplest epidemiological model one can consider is the susceptible-infected susceptible (SIS) model. The SIRS model is mainly used as a paradigmatic model for the study of infectious diseases leading to an endemic state with a stationary and constant value for the prevalence of infected individuals, i.e. the degree to which the infection is widespread in the population. In the SIS model, individuals can only exist in two discrete states, namely, susceptible and infected. The disease transmission is described in an effective way. The probability that a susceptible vertex acquires the infection from any given neighbor in an infinitesimal time interval $dt$ is $\lambda dt$, where $\lambda$ defines the virus spreading rate. At the same time, infected vertices are cured and become again susceptible with probability $\mu dt$. Individuals thus run stochastically through the cycle susceptible $\rightarrow$ infected $\rightarrow$ susceptible, hence the name of the model. The SIRS model does not take into account the possibility of individual’s removal due to death or acquired immunization, which would lead to the so-called susceptible-infected-removed (SIR) model [5] and [30]. The SIR model, in fact, assumes that infected individuals disappear permanently from the network with rate $\mu$. In models such as the SIRS, the number of infected individuals increases up to a stationary constant value which is non zero if $R_0 > 1$. On the contrary, in models such as the SIR, the number of infected individuals tends toward zero since all infected will sooner or later become removed from the population. Also in this case, however, a finite fraction of the population is affected by the epidemic outbreak only if $R_0 > 1$. It should be noted that it is also possible to induct a steady state in the SIRS model, by introducing new susceptible individuals at a constant rate. This new parameter constitutes a new time scale that gives rise to oscillations in the endemic phase [18].

2.2. Networks Models

In the network model, each individual $N$ in the population represented by vertices. Edge connects a pair of vertices representing the contact between the individu. The number of arcs vertexs given known as degree. The structure of the network is explained by adjacency matrix $A_{ij}$ the entry point is 1 if the individu i is the individual contact with j and 0 otherwise. Here, consider the edge bi-directional and therefore a symmetric adjacency matrix.

$$A_{ij} = \begin{cases} 1, & \text{jika individu i kontak dengan } j \\ 0, & \text{jika sebaliknya} \end{cases}$$

Simulation of stochastic epidemic SIRS in static networks. Note the two structures network: Poisson (random) and small-world networks. The probability of transmission of the disease on an edge during the interval $\delta t$ is:

$$P(S \rightarrow I : \delta t) = 1 - \frac{1}{e^{r\delta t}}$$

where $\tau$ is the transmission probability per contact per unit of time. To put it demografi and maintain a constant population size and structure of the network, the individual is ’dead’ at the level of $\mu$ and was soon replaced by a vulnerable individual with the same contact. Transmission period is assumed exponential Gaussian with mean $1/\gamma$. The study also supposes that the disease did not increase mortality and $r = \gamma - \mu$, where $r$ is the rate of recovery. All the size of the rate used in this research is measured in units of inverse of rate of healing (i.e., taken $r - 1 = 1$) and because it means the non-dimensional. The population is composed of subpopulations of S, I and R from individuals who are vulnerable, infected and recovered.

3. Mean-Field Models for Random Networks: Beyond Homogeneous Mixing
3.1. Standard Mean Field Models

In a network of size $N$ with homogeneous mixing, all the individuals are in ‘weak’ contact with each other. Thus, the degree of each individual is $N - 1$. On an average, infections are produced at the rate $\varepsilon S I$ and therefore, the mean of the infected population evolves according to the ordinary differential equation $\frac{dS}{dt} = (\varepsilon S - \gamma)I$. For large values of $N$, the threshold parameter $N\varepsilon\gamma$ (derived from the equations of the model) coincides with the basic reproductive number (see electronic supplementary material) and at equilibrium the average susceptible proportion satisfies $\langle S / N \rangle = 1 / R_0$, as expected. The mean-field model for networks with homogeneous mixing is therefore a mass-action model. However, homogeneous mixing may be realistic only for small populations. More typically, an average individual is in contact only with a small fraction of the population, and the mean of the degree distribution, $\langle n \rangle$, is much smaller than the population size $N$ in [27], [9], and [7].

In random networks [31] e.g. [32], the degree distribution is Poisson with mean $n$. Since an average infectious individual is in contact with $n$ individuals (a random sample from the total population; see electronic supplementary material) but only the fraction $S/N$ is susceptible, the average rate of infection becomes $n\varepsilon S(N-I)$ and the mean-field model is given by

$$\frac{dI}{dt} = \left(\frac{n}{N} \frac{S}{N} - \gamma\right)I,$$  \hfill (4)

which, by construction, reproduces the initial rate of disease spread [27]. However, the threshold parameter derived from this model is $nt/g$ which does not coincide with the basic reproductive number $R_0 = n\varepsilon(\tau + \gamma)$ [33], [34] and [35]. In fact, according to this model, an average infectious individual may produce as many as $n\varepsilon\gamma$ secondary infections which may exceed the maximum number of available susceptible contacts, $n$. On the other hand, owing to the random nature of the network, it is expected that at equilibrium $\langle S / N \rangle = 1 / R_0$, a fact that is confirmed by the stochastic simulations. Thus, this standard mean-field model fails to reproduce equilibrium values and the epidemic threshold parameter derived from its equations is not the basic reproductive number corresponding to the epidemiological setting. These failures are a direct consequence of the fact that $n \geq N$.

3.2. A Modified Mean-Field Model Constructed from Individual-Level Parameter

Although the standard mean-field model fails to capture the course of epidemics in random networks, we expect given this randomness that some mean-field model may successfully describe the average epidemic evolution. Next, we present a derivation of a mean-field model that takes into account implicitly the influence of network contact structure on disease spread. Key information on this influence is contained in the basic reproductive number.

The basic reproductive number is usually defined as the number of secondary cases produced by an average infectious individual placed in a completely susceptible population. This definition implicitly assumes homogeneously mixed populations. However, for other contact network structures, it does not incorporate the effect of local contact structure on the initial spread of the disease. An alternative definition is to consider the number of cases produced by an average infective at the beginning of the epidemic, i.e. when the depletion of susceptible is negligible, but many generations of infectives have occurred, in order to ‘wash out’ the effect of initial conditions e.g. [35]. For the cases considered in this work, this last definition leads to rather complex computations for the basic reproductive number. As a useful alternative, we considered here an intermediate situation: the basic reproductive number was defined as the number of secondary cases produced by an average infectious individual in the second generation [33] and [34]. This quantity can differ significantly from the first generation calculation for contact networks, particularly for small $n$ and for networks with high clustering. The value of $R_0$ estimated in the second generation better approximates the number of new cases produced per case at the beginning of an epidemic. This basic reproductive number can be computed from individual level considerations e.g. [34], [35], and [27] and its expression will depend on the local structure of contacts and other individual level parameters, such as infectiousness and the distribution of the infectious period.
For example, in Poisson random networks with exponentially distributed infectious periods, the basic reproductive number is \( R_0 = n \tau / (\tau + \gamma) \) [33] and [35]. We propose a mean-field model whose central feature is a transmission rate built from this basic reproductive number instead of the phenomenological parameter \( \beta = n \tau \).

At the beginning of the epidemic, an average infectious individual will produce \( R_0 \) new infections during a mean effective infectious period \( t_e \), which is shorter than the mean infectious period, \( 1/\gamma \). To approximate this pattern, we split the mean infectious period into two contributions \( 1/\gamma = 1/\gamma_e + 1/g \) where \( t_e = 1/\gamma_e \). Infectious individuals produce infections only during the effective infectious period, \( 1/g \), remaining the rest of their infectious life \((1/g)\) ‘inactive’. These individuals are still infected but owing to the stochasticity of the transmission process, the last infection they produce always occurs before complete recovery. In addition, infected individuals can deplete their local pool of susceptible and no longer be able to transmit the disease. As the epidemic progresses, the number of infections caused by each infectious individual is reduced by the susceptible fraction because the cluster of contacts of any individual is a random sample of the population. Thus, an average infectious individual will produce \( R_0 (S/N) \) infections during a period \( 1/\gamma_e \) and will remain ‘inactive’ during a period \( 1/g \).

The evolution of the active infectious population \( I \) is now given by
\[
\frac{dI}{dt} = \gamma_e R_0 \left( \frac{S}{N} - 1 \right) I - \gamma_e I = \gamma_e (R_0 (S/N - 1) - 1) I.
\]

Individuals leaving the active class \( I \) are still infected and may be moved to a new class \( Y \) defined as containing infected but ‘no longer infectious’ individuals. The mean-field model becomes

\[
\frac{dS}{dt} = \mu N - \gamma_e R_0 \left( \frac{S}{N} - 1 \right) I - (\mu + \lambda) S
\]

\[
\frac{dI}{dt} = \gamma_e R_0 \left( \frac{S}{N} - 1 \right) I
\]

\[
\frac{dY}{dt} = (\gamma_e - \mu) I - (g + \mu + \lambda) Y
\]

\[
\frac{dR}{dt} = g Y - (\mu + \lambda) R.
\]

The threshold parameter of this model is \( R_0 \) and at equilibrium \( \langle S/N \rangle = 1/R_0 \). Neither of these two key properties depends on the value of \( \gamma_e \) which only affects the transients. Here, we choose \( \gamma_e = \tau + \gamma \) in order to reproduce the initial rate of disease spread. Owing to the random structure of these networks, we expected an appropriately defined mean-field model to work well. A more challenging case is considered next using small-world networks, for which local structure exists and individual contacts are no longer a random sample of the population.

4. Networks with Clustering

A drawback of Poisson random networks as models of social networks is their low clustering coefficient. Small world networks [36] have become a popular toy model for social networks because they exhibit a high clustering coefficient, \( C \), and a short mean path length, \( L \). Thus, \( C \) corresponds to the probability that two neighbours of a node are themselves connected, while \( L \) is the average shortest distance between the two nodes in the network. We specifically consider small-world networks of mean degree \( n = 8 \). The clustering coefficient and mean path length are determined by the disorder parameter \( (\phi) \). When \( \phi = 0 \), the ordered network is a regular network where each individual is in contact with its eight nearest neighbours. The disorder parameter specifies the probability that an individual has a long-distance contact, i.e. a contact which is not among its local neighbours. We constructed the small-world networks \((0 < \phi < 1)\) using the algorithm of [36]. In our implementation, the regular network \( (\phi = 0) \) is a two-dimensional lattice on a torus (see [37] for details), a choice motivated by the spatial nature of local interactions in epidemiology. In the stochastic simulations, we
consider a disorder parameter $\phi = 0.1$ for which the network is in the small-world regime (with a high $C$ of approximately 0.75 but a short $L$ of 0.08, both normalized by their respective values for $\phi = 0$ when the network is regular).

Next, we derive an expression for $R_0$ for small-world networks (see further details in the electronic supplementary material) and examine whether the resulting mean-field system approximates the stochastic dynamics of the disease. For $\phi > 0$, an average individual has $n$ contacts of which $i \leq n$ may be long-distance contacts with probability approximately given by

$$P(i, \phi) = \binom{n}{i} (1-\phi)^{-1} \phi^i$$

(9)

At the beginning of the epidemic, infected long-distance contacts may themselves produce $R_{0 \text{ordm}}$ infections on average (and we make the approximation that $R_{0 \text{ordm}} = n \rho$).

Consider now a source case with exactly $i$ long-distance contacts that produce a secondary case. This secondary case may be one of its $i$ long-distance contacts with probability $i/n$ or one of its local contacts with probability $(n-i)/n$. In the first case, the secondary infection will produce, on average, $R_{0 \text{ordm}}$ ternary cases. In the second, the secondary infection may have $j$ long-distance contacts with probability approximately given by $P(j, \phi)$. This secondary case will therefore produce, on an average, $(n-j)R_{0 \text{ordm}}/n$ infections among its local contacts and $jR_{0 \text{ordm}}/n$ among its long-distance contacts, where $R_{0 \text{sp}}$ denotes the basic reproductive number of the regular ordered network. By averaging the overall values of $j$, we obtain the expected number of infections produced by a secondary case which is itself a local contact of a source case with exactly $i$ long-distance contacts: $R_{0 \text{sp}}(\phi) = \sum_{j=0}^{\infty} P(j, \phi) ((n-j)R_{0 \text{ordm}} + jR_{0 \text{ordm}})/n$. Finally, averaging for all the values of $i$, we obtain the following expression for the number of infections caused by an infected in the second generation

$$R_{0 \text{sp}}(\phi) = \sum_{j=0}^{\infty} P(j, \phi) ((n-j)R_{0 \text{ordm}} + jR_{0 \text{ordm}})/n$$

(10)

where $\rho = \sigma(\tau + \gamma)$ is the probability of transmission per case and the expression of $R_{0 \text{sp}}$ used to calculated equation (10) is:

$$R_{0 \text{sp}} = \frac{1}{2} \rho \left( 7 - 6 \rho + \frac{4}{1+\rho} + \frac{3}{1+2\rho} \right)$$

(11)

The values obtained with equation (10) are in excellent agreement with empirical estimates of the basic reproductive number obtained from simulations. A high clustering coefficient greatly decreases the initial rate of disease spread but has a much less noticeable effect on equilibrium values. For both the cases, model (5-8) performs much better than the standard mean-field model (4) which greatly overestimates the initial rate of disease spread because it ignores the local structure of the contacts. This last deficiency could be corrected by defining an effective neighbourhood as $n_e = R_{0 \text{sp}}/\rho$.

However, even with this correction, the threshold for this model would always overestimate the basic reproductive number, because $n_e/\gamma = R_{0 \text{sp}}(\gamma + 1)/\gamma > R_{0 \text{sp}}$. The modified mean-field model approximates the initial growth, the turning point and the trough of the first epidemic, as well as the equilibrium level of susceptible. These results are robust across a range of parameters of the stochastic simulations. The model cannot, however, completely reproduce the exact phase and amplitude of the decaying oscillations to equilibrium.

5. Conclusions
Desertasi this file a communicable disease epidemic model in the network SIRS, for type a dynamic model of mean-field baku was used as a framework basic work. SIRS epidemic model in this, the very basic parameters specific interventions in epidemic disease is caraan-R0 (basic reproductive number). R have a major role as the threshold of the aba aba-the existence of an outbreak, against the relevansi people to test the size of the control. The Value Of R0 so this can be generated from the model. This dissertation research also has filed to reverse perspective towards the key epidemic quantity. (especially, when the basic assumptions from the model, mixing a homogeneous population, does not apply). In other words, basic reproductive number becomes the main parameters so that a model of the
A type of mean-field can be formulated. Model modifikasi mean-field generated on the substance contains implied some important effects of pencampu-heterogeneous network of contacts in the ran, which can provide more approach both to the direction of time population levels of disease than the formulation mean-field baku. As such a simple model can be used unknowing the temporal direction Tuk epidemic, although transmission between individuals occur in a complex network of contacts.

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