COMPARING SMALL WORLD NETWORK AND TRADITIONAL MODELS OF MARBURG VIRUS DISEASE

FARAJI MOHAMED TIRAGA[1,2,*], LORD CLIFFORD KAVI[2,3], NANCY ANN NEUDAUER[4]

[1]Department of Mathematics and Statistics, University of Dodoma, Dodoma, Tanzania

[2]African Institute for Mathematical Sciences, Summerhill Estates, East Legon Hills, Accra, Ghana

[3]Department of Mathematics and Statistics, University of Ottawa, Ottawa, Canada

[4]Department of Mathematics and Computer Science, Pacific University, Oregon, USA

Abstract. Like Ebola, Marburg Virus Disease (MVD), formerly known as Marburg hemorrhagic fever, belongs to the deadly Filovirus family of viruses. It is transmitted among humans when an individual comes into contact with the bodily fluids of an infected person. We model the spread of the Marburg virus disease using both the compartmental Susceptible-Exposed-Infectious-Recovered (SEIR) epidemic model and a small-world network model. The World Health Organization (WHO) recommends "community engagement as key to successfully controlling MVD outbreaks. Good outbreak control relies on applying a package of interventions, including infection prevention and control practices, surveillance and contact tracing and social mobilization". This paper highlights the need for a network based model over the traditional models to control the spread of MVD.

Keywords: epidemiology; infectious diseases; Marburg; spreading models; complex networks.

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*Corresponding author

E-mail address: faraji@aims.edu.gh

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1. INTRODUCTION

Mathematical modelling of infectious diseases plays a vital role in epidemiology as it helps in understanding the mechanism and processes that lead to the spread of diseases, and also suggests control strategies [1, 2, 3].

Modelling of the spread of disease can be done by using traditional models or small world network models. The traditional (or compartmental) models rely on differential equations, describing the dynamics of infection spreading within uniformly mixed populations [4, 5]. The basic premise of *uniform mixing* is that contacts between all individuals in the population are equally likely, and thus any infected person is likely to infect any other person.

The uniform mixing assumption does not depict reality well. Spatial effects and heterogeneity have been shown to mainly affect disease transmission and persistence [6]. Research has been done by applying the small-world network model to capture particular aspects of the population. In spatial structure, one might contrast settings in which mixing is mainly local in nature against settings in which population is substantially well-mixed. The network models, especially small-world networks, depict clearly local and global mixing of the population.

In the small-world networks introduced by *Watts* and *Strogatz* in 1998 [7], nodes correspond to individuals and edges to contacts, representing the interactions between people that could potentially lead to transmission of the disease. This network captures two important characteristics of social networks: the *small world effect* (when the distance between two randomly chosen nodes (individuals) in a network is short) and *high clustering* (when two nodes (individuals) having common neighbours have a tendency to be connected to each other). The network model counts each individual in the population as well as describing how all of these individuals interact with one other.

The genus Marburg includes a single species, *Marburg marburgvirus,* which is represented by two distinct viruses, Marburg Virus (MV) and Ravn Virus (RV) [8]. Both MV and RV cause severe hemorrhagic disease in humans and susceptible animals [10]. The first reported Marburg Virus Disease (MVD) outbreak took place in Marburg and Frankfurt, Germany and Belgrade, Serbia in 1967. There have been several outbreaks in sub-Saharan Africa. In $2004 - 2005$, the

largest MVD outbreak took place in Uige, Angola with 252 affected patients, many of them children, and a devastating case fatality rate of 90% [9, 11].

In this paper, we model the spread of MVD using both the compartmental model and the small-world network model. In the compartmental model, the spreading process itself is modelled using rate equations, describing population flows between epidemiological classes of individuals, such as susceptible (S), exposed (E), infected (I) and recovered (R). We adapt the SEIR dynamics to the *small-world effect*. This paper is organized in sections as follows: Section 2 deals with the traditional (compartmental) model of MVD, Section 3 covers the small-world network model of MVD, Section 4 deals with the simulated results. In Section 5 we discuss the control measures of the spread of MVD in small-world network model and finally Section 6 covers the conclusion.

2. TRADITIONAL MODEL

We consider a real world system consisting of a host population, $N(t)$, that is divided into four compartments $S(t)$, $E(t)$, $I(t)$ and $R(t)$ where $S(t)$ represents the number of susceptible individuals at time t, $E(t)$ represents the number of exposed individual at time t, $I(t)$ represents the number of infectious individuals at time t and $R(t)$ represents the number of recovered (or removed) individuals at time t. The exposed category refers to latent period (where all of the individuals have been infected but are not yet infectious). Therefore $N(t) = S(t) + E(t) + I(t) + R(t)$. These compartments form the model called SEIR.

2.1. Model Formulation.
In order to formulate the dynamics of the above system mathematically, the following assumptions are appropriate:

  a. There is a constant number entering the population due to birth or migration at a constant rate $b > 0$.

  b. The MVD is transmitted by contact between individuals in the $S-$compartment and the linear incidence rate for the $I-$compartment, given by βIS where $\beta > 0$ represents the infection rate.

  c. The individuals in the $I-$compartment are facing death due to the MVD with infection death rate $\alpha \geq 0$. Others recover from the MVD with recovery rate $\gamma > 0$.

d. There is a natural death rate $\mu > 0$ for the individuals in the host population.

The dynamics of the MVD can be represented in a compartmental model as shown in Fig. 1.
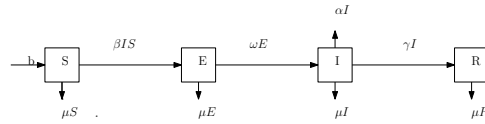

FIGURE 1. The Compartments of the SEIR model

Using these assumptions and the compartmental diagram in Fig. (1) the dynamics of Marburg Virus Disease (MVD) can be represented mathematically by using differential equations:

(1)
$$\frac{dS}{dt} = b - \beta IS - \mu S$$
$$\frac{dE}{dt} = \beta IS - (\omega + \mu)E$$
$$\frac{dI}{dt} = \omega E - (\alpha + \mu + \gamma)I$$
$$\frac{dR}{dt} = \gamma I - \mu R,$$

where b is recruitment rate, β is infection rate, ω is the transition rate from exposed to infectious, α is death rate induced by the disease, γ is the recovery rate and μ is the natural death rate.

We proceed to prove that S, E, I, and R are biologically true (non-negative) using Theorem 1.

Theorem 1. [25] The closed set $\Omega = \{(S(t), E(t), I(t), R(t)) \in \mathbb{R}_+^4 : N(t) \leq \frac{b}{\mu}\}$ is attracting and positively invariant with respect to the system (1).

Proof. We first assume that $\alpha > \mu$ during the modelling time. This assumption is sensible since the death rate of the MVD is higher than the natural death rate in the course of an MVD epidemic.

Let $(S(t), E(t), I(t), R(t))$ be any solution of the system with any given initial condition. Then

(2)
$$\frac{dN(t)}{dt} = \frac{dS(t)}{dt} + \frac{dE(t)}{dt} + \frac{dI(t)}{dt} + \frac{dR(t)}{dt}$$

Comparing the system (1) and (2) we get

$$(3) \qquad \frac{dN(t)}{dt} = b - \mu S - \mu E - (\alpha + \mu)I - \mu R.$$

$$(4) \qquad \frac{dN(t)}{dt} \leq b - \mu N.$$

Integrating Equation (4) gives the following solution

$$(5) \qquad 0 \leq N(t) \leq (N(0) - \frac{b}{\mu})e^{-\mu t} + \frac{b}{\mu}.$$

We have $\lim_{t \to \infty} N(t) \leq \frac{b}{\mu}$ when $N(0) \leq \frac{b}{\mu}$.

However, if $N(0) \geq \frac{b}{\mu}$, $N(t)$ will decrease to $\frac{b}{\mu}$. So Ω is positively invariant (all solutions in $\mathbb{R}_+^4$, eventually approach, enter or stay in Ω) [26]. □

Therefore, the system of equations given in model (1) is mathematically well-posed and epidemiologically reasonable.

We now proceed to find the constant solutions of the system which give an indication of the long term behaviour of the system.

2.2. The Equilibrium Points. We describe the disease free and endemic equilibrium points.

2.2.1. *The disease-free equilibrium point.* The disease-free equilibrium of the system (1) is obtained when $I = E = 0$. Thus the solution is given by

$$(6) \qquad P_0^*(S^*, E^*, I^*) = P_0(S(0), E(0), I(0)) = \left(\frac{b}{\mu}, 0, 0\right),$$

where S^* represents susceptible individuals at the equilibrium point, E^* represents exposed individuals at the equilibrium point and I^* represents infectious individuals at the equilibrium point. The disease-free equilibrium point implies that there exists no infection. Now, we need to find the equilibrium point where the infection exists.

2.2.2. *The endemic equilibrium point.* The endemic equilibrium point is obtained when $\dfrac{dS}{dt} = \dfrac{dE}{dt} = \dfrac{dI}{dt} = 0$ for $I > 0$ and $E > 0$. We solve to get the endemic equilibrium point as:

(7)

$$P^*(S^*, E^*, I^*) = \left(\frac{(\alpha + \mu + \gamma)(\mu + \omega)}{\beta \omega}, \frac{\beta \omega b - \mu(\alpha + \mu + \gamma)(\omega + \mu)}{\beta \omega(\mu + \omega)}, \frac{\beta \omega b - \mu(\alpha + \mu + \gamma)(\omega + \mu)}{\beta \omega(\mu + \omega)(\alpha + \mu + \gamma)} \right).$$

This is an endemic equilibrium point, where the disease persists in the community for a certain period of time.

We now find the average number of secondary infections that occur from one infected individual in contact with susceptible individuals. This helps us to identify the conditions under which the MVD will die out.

2.3. The Basic Reproduction Number. The basic reproduction number R_0 gives the expected number of infectious cases that are generated by one infectious individual in a totally susceptible population. To derive R_0, we use the next generation method introduced by [14]. Using the two infected compartments, that is, E and I, we get R_0 to be

$$R_0 = \frac{\beta \omega b}{\mu(\mu + \omega)(\alpha + \mu + \gamma)}.$$

Expressing the epidemic equilibrium point, P^*, in R_0 gives:

(8) $$P^* = \left(\frac{b}{uR_0}, \frac{\mu(\alpha + \mu + \gamma)}{\beta \omega}(R_0 - 1), \frac{\mu}{\beta \omega}(R_0 - 1) \right).$$

Therefore, Equation (8) shows that if $R_0 < 1$, then all partial populations are negative which is unrealistic biologically. This implies that an infected individual in the entire period of infection will produce less than one infected individual on average which shows that MVD will die out. However, Equation (8) shows that if $R_0 > 1$, then all partial populations are non-negative. Each infected individual in the entire infection period having contact with susceptible individual will produce more than one infected individual, which leads to an epidemic of MVD.

In the next section, we model the spread of the MVD using the small world network.

3. THE SMALL-WORLD (SW) NETWORK MODEL OF MVD

Using complex networks as spreading lattices in epidemic models has provided much insight in the context of human disease [7, 15].

In 1998, *Watts* and *Strogatz* proposed the small-world network model which aimed to address the limitations of the random graph of the Erdös-Rényi model [7, 23]. The small world network is constructed from a regular network that consists entirely of localized links, with edges connecting only nodes that are in close (nearest neighbour) proximity with each other up to some range k such that it has coordination number $k = 2q$ where $q \in \mathbb{N}$. This reflects that individuals are most likely to have contact with their neighbours. Nodes are then rewired randomly with a certain probability p. This reflects that an individual might travel to another neighbourhood, with some probability p.

A random rewiring process introduces connections known as *small world effect*, where any two nodes in a network are connected to each other through a small distance. Newman and Watts proved that when $p = 0$, the small world network reduces to a regular network and when $p = 1$, it reduces to a random (Erdös-Rényi) network by adding long-range shortcuts with a certain probability p [24]. Therefore a small-world network can be regarded as a way to interpolate between regular networks and random networks.

(A) Regural network
$p = 0$

(B) Small world network
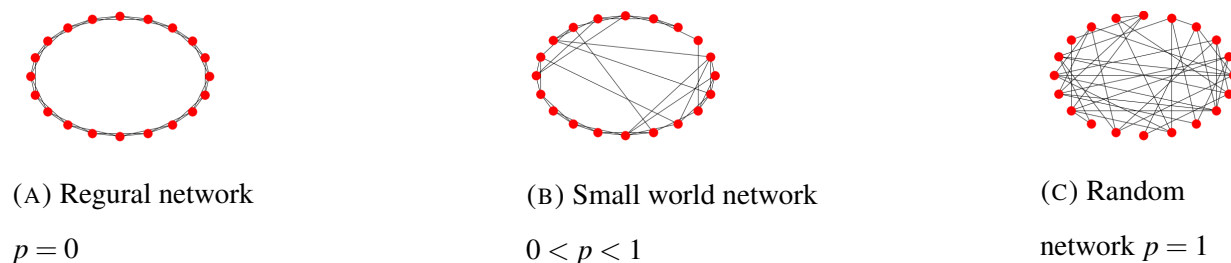$0 < p < 1$

(C) Random
network $p = 1$

FIGURE 2. Understanding small world networks

The spreading processes are very fast in complex networks, especially *small-world networks* due to very short average node-to-node distance. The SW network model eliminates the unrealistic uniform mixing assumption used in the compartmental model. One is more likely to spread the disease to someone in their house or workplace than an arbitrary person in the town. We are able to consider the case of an infectious individual having a higher probability of infecting his immediate neighbour while at the same time with a certain (lower) probability infecting some isolated neighbours. The effect of super spreaders is easily captured by the SW network model.

It also has the advantage of providing us with more realistic dynamics of the spread of the disease and therefore ensuring better control measures. For the main properties and further details of the SW network, one can refer to [7, 16, 17, 18].

3.1. Model Formulation. We adapt the SEIR dynamics to the *small world effects* situation where each individual in the population is in one of four states during the procession of the disease. The specification of the contact between people producing new exposures, plus the specification of the probability with which an individual moves between compartments after exposure, provides a full specification.

In our case the contact between people is modelled with a regular network with the lattice parameter k. This means every individual has k immediate neighbours.

The way individuals move between compartments is specified by an algorithm that involves two parameters of disease transmission p_s and p_j, where p_s is the probability of infecting nearest neighbours (family members or workmates) and p_j is the probability of infecting random individuals (long range spreading).

The infectious individuals are recovered/removed from the disease with parameter p_r (that is, p_r is the probability of recovering or dying from the disease). The algorithm [19] is described as follows. At every time step of duration $\triangle t$, every infectious individual in the network:

1. Infects its nearest susceptible neighbours with probability p_s per neighbour. When infected, the individual enters the exposed category and remains there for a period of time (specified by the mean and standard deviation of the length of time exposed). Once this time period is up, the individual enters the infectious compartment with parameter p_0;

2. With probability p_j, tries to infect one randomly chosen susceptible individual. When infected, the randomly chosen individual enters the exposed category and remains there for a period of time (specified by the mean and standard deviation of the length of time exposed). Once this time period is up, the individual enter the infectious category with parameter p_0;

3. With probability p_r, recovers and can no longer be infected or infect others.

This process can be readily iterated numerically until changes no longer take place. Step (1) corresponds to transmission of the infection along the regular underlying lattice (the short-range

spreading) and step (2) corresponds to transmission of the disease through randomly changing long-range connection (the long-range spreading).
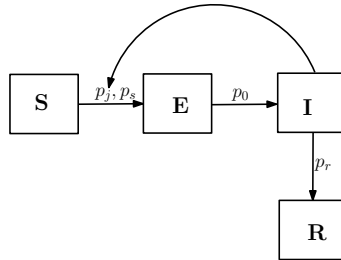

FIGURE 3. State transition flow graph

Fig. 3 shows the transmission state diagram: Transition from S to E is based on the small world network structure and the infection probabilities p_s and p_j, the transition from E to I with probability p_0 and while the transition from I to R is with probability p_r.

3.2. The Epidemic Threshold. Here, we need to derive the necessary condition for an epidemic to take off. That is, the epidemic threshold based on analysis of the rate equations describing the dynamics of the system. Suppose there is a population of N individuals, and each individual is connected directly to k nearest neighbours. An infected individual will infect its nearest neighbours with probability p_s provided that they are susceptible. Furthermore, each infectious individual is assumed to have an average of n susceptible distant individuals it can infect with probability p_j.

We denote the number of individuals that are susceptible by S, the number that has been exposed by E, the number of infectious by I and the number that have recovered by R. Since the epidemic occurs within a short period of time, we can neglect the demography (birth and death). Using Fig (3), the spread of MVD can be given by differential equations as follows:

(9)
$$\dot{S} = -(p_s kz + p_j n)I$$
$$\dot{E} = (p_s kz + p_j n)I - p_0 E$$
$$\dot{I} = p_0 E - p_r I$$
$$\dot{R} = p_r I$$

where n is the number of remote neighbors and z is the number of near neighbour links that support possible infection. In the case of transmission of the disease, $N \geq E + I + R$, and therefore $N \approx S$. So the system (9) is reduced to

$$\dot{E} = (p_s kz + p_j n)I - p_0 E$$

(10)
$$\dot{I} = p_0 E - p_r I$$

$$\dot{R} = p_r I.$$

We want to study the dynamics of the disease in terms of days, hence we write the system (10) as difference equations:

$$\triangle E_t = (p_s kz + p_j n)I_t - p_0 E_t$$

(11)
$$\triangle I_t = p_0 E_t - p_r I_t$$

$$\triangle R_t = p_r I_t$$

From the system (11), we can re-write $\triangle E_t$ as $\dfrac{E_{t+1} - E_t}{\triangle t}$. We assume the time step is 1 day, hence $\triangle t = 1$. Thus we have

(12)
$$E_{t+1} - E_t = (p_s kz + p_j n)I_t - p_0 E_t$$

$$E_{t+1} = (p_s kz + p_j n)I_t + (1 - p_0)E_t.$$

Similarly, we have $I_{t+1} = p_0 E_t + (1 - p_r)I_t$ and $R_{t+1} = p_r I_t + R_t$. So our new system of difference equations is

$$E_{t+1} = (p_s kz + p_j n)I_t + (1 - p_0)E_t$$

(13)
$$I_{t+1} = p_0 E_t + (1 - p_r)I_t$$

$$R_{t+1} = p_r I_t + R_t.$$

To test the stability of the system (13), we use its Jacobian matrix. Thus

(14)
$$J = \begin{bmatrix} 1 - p_0 & p_s kz + p_j n & 0 \\ p_0 & 1 - p_r & 0 \\ 0 & p_r & 1 \end{bmatrix}.$$

The corresponding eigenvalues of matrix (14) are

$$\lambda_1 = 1,$$

$$\lambda_2 = 1 - \frac{p_0 + p_r}{2} + \frac{1}{2}\sqrt{(p_0 - p_r)^2 + 4(p_s kz + p_j n)p_0},$$

and

$$\lambda_3 = 1 - \frac{p_0 + p_r}{2} - \frac{1}{2}\sqrt{(p_0 - p_r)^2 + 4(p_s kz + p_j n)p_0}.$$

Therefore it shows that the system is unstable if $\lambda_2\,\lambda_3 < 1$; otherwise it is stable. The epidemic will terminate if the rate of infection is smaller than the rate of recovery, that is, $p_s kz + p_j n < p_r$ and this implies that the condition for epidemic to terminate is

$$1 - \frac{p_0 + p_r}{2} - \frac{1}{2}\sqrt{(p_0 - p_r)^2 + 4(p_s kz + p_j n)p_0}.$$

And the disease will be endemic if the rate of infection is bigger than rate of recovery, that is, $p_s kz + p_j n > p_r$, and this implies that the condition for an endemic is

$$1 - \frac{p_0 + p_r}{2} + \frac{1}{2}\sqrt{(p_0 - p_r)^2 + 4(p_s kz + p_j n)p_0}.$$

The SW network model suggests that the spreading dynamics cannot be determined solely by a number of secondary infections caused by only one infected individual. In the small world network model, the effects of secondary infections caused by short-range transmission is different from ones caused by long-range spreading.

4. SIMULATED RESULTS

There is a paucity of available data for MVD outbreak. We perform numerical simulations, providing theoretical analysis of the SW model. Though it is difficult to get data on MVD, information from WHO [27], [20] and [12] provides us with parameter estimations. A typical small village in Angola, which had a devastating outbreak of Marburg, might have a population size of 253. In our simulation, we consider the population size of $N = 253$ with 2 initial infected individuals. The approximated size of each family is 4. We vary the values of the short-range and long-range spreading parameters to see their impact on the dynamics of the disease spread. We study the dynamics of the spread in intervals of three days. At the end of each time period,

we identify the number of individuals who recovered (or died) from the infection, the number of individuals still infectious or exposed and the number of individuals still at risk (susceptible).

In the following resulting networks, yellow nodes represent susceptible individuals, blue nodes represent infectious or exposed individuals and green nodes represent recovered/removed individuals.

(A) End of day 3:

$S = 204$, $I = 34$, $R = 15$

(B) End of day 6:

$S = 45$, $I = 71$, $R = 137$

(C) End of day 9:

$S = 18$, $I = 38$, $R = 197$

(D) End of day 12:

$S = 16$, $I = 7$, $R = 230$

(E) End of day 18:

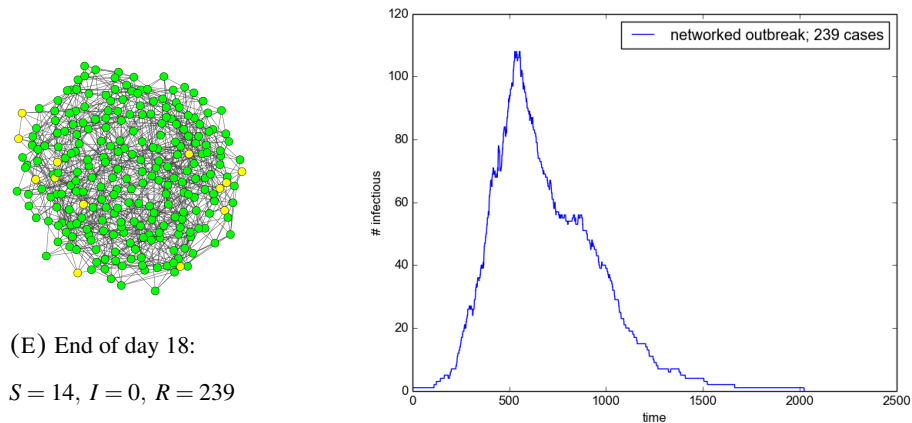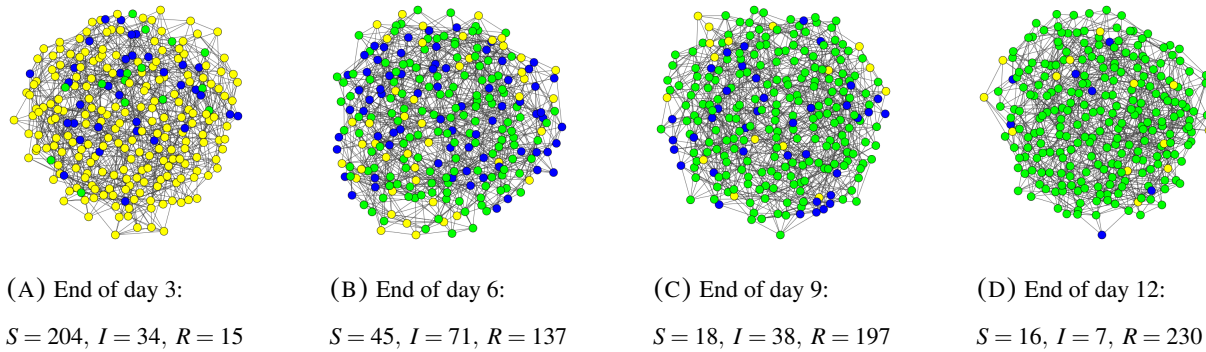$S = 14$, $I = 0$, $R = 239$

(F) Infection against time

FIGURE 4. Propagation of the spread of MVD for parameters $p_r = 0.1$, $p_s = 0.07$ and $p_j = 0.2$

Fig. 4a, 4b, 4c, 4d and 4e show the evolution of the disease spread in the network. We assume $p_r = 0.01$, $p_s = 0.2$ and $p_j = 0.02$. Fig. 4f shows the propagation of the spread of the disease throughout the period of the epidemic. It depicts that out of 253 individuals, there were 239 recorded cases, that is, 239 individuals were infected during the course of the outbreak, whereas 14 individuals were not infected.

(A) End of day 3:

$S = 0, I = 117, R = 137$

(B) End of day 6:

$S = 0, I = 47, R = 206$

(C) End of day 9:

$S = 0, I = 16, R = 237$

(D) End of day 12:

$S = 0, I = 2, R = 251$

(E) End of day 15:
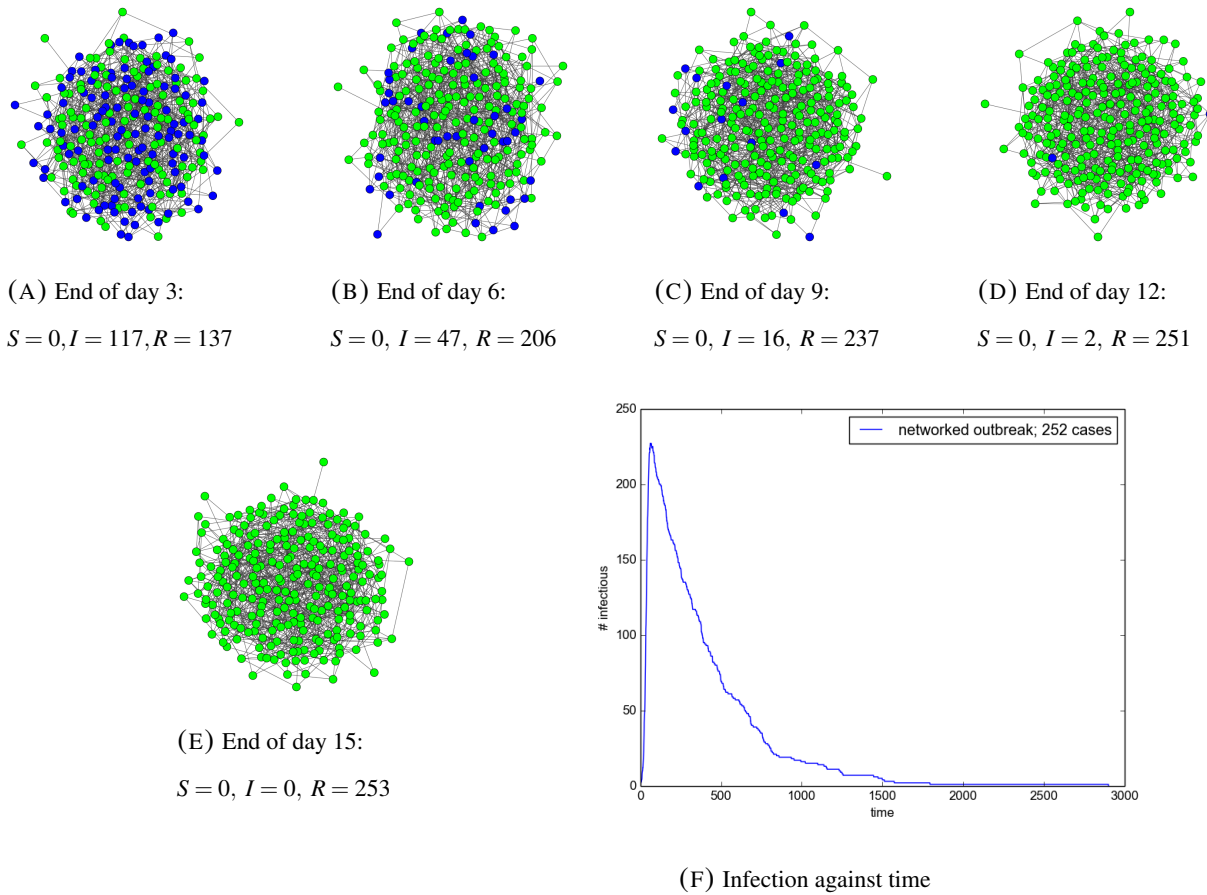
$S = 0, I = 0, R = 253$

(F) Infection against time

FIGURE 5. Propagation of the spread of MVD for parameters $p_r = 0.09$, $p_s = 0.5$ and $p_j = 0.07$

Fig. 5 depicts the effects of short-range spreading. The short-range parameter is greater than recovery rate. This leads to all individuals within the network being infected within a short period of time. It shows that by the end of the third day, all individuals were already infected. By the end of the fifteenth day, the spread is over. The upshot of this situation is that, since the spread is very fast within a short period of time, either the entire population is wiped off if there are no good control measures or the entire population is cured if there is a very effective medication for curing infected patients.

Fig. 6 depicts the spread of the MVD when the short-range spreading parameter and the long-range parameter are equal such that $p_j = p_s = 0.2$. All individuals are infected within 15 days. It also shows that the population is almost uniform in terms of contact although the *small world effect* is still visible. In other words, we don't only see what an SEIR model would normally

(A) End of day 3:

$S = 153$, $I = 82$, $R = 18$

(B) End of day 6:

$S = 1$, $I = 78$, $R = 174$

(C) End of day 9:

$S = 0$, $I = 20$, $R = 233$

(D) End of day 12:

$S = 0$, $I = 2$, $R = 251$

(E) End of day 15:

$S = 0$, $I = 0$, $R = 252$
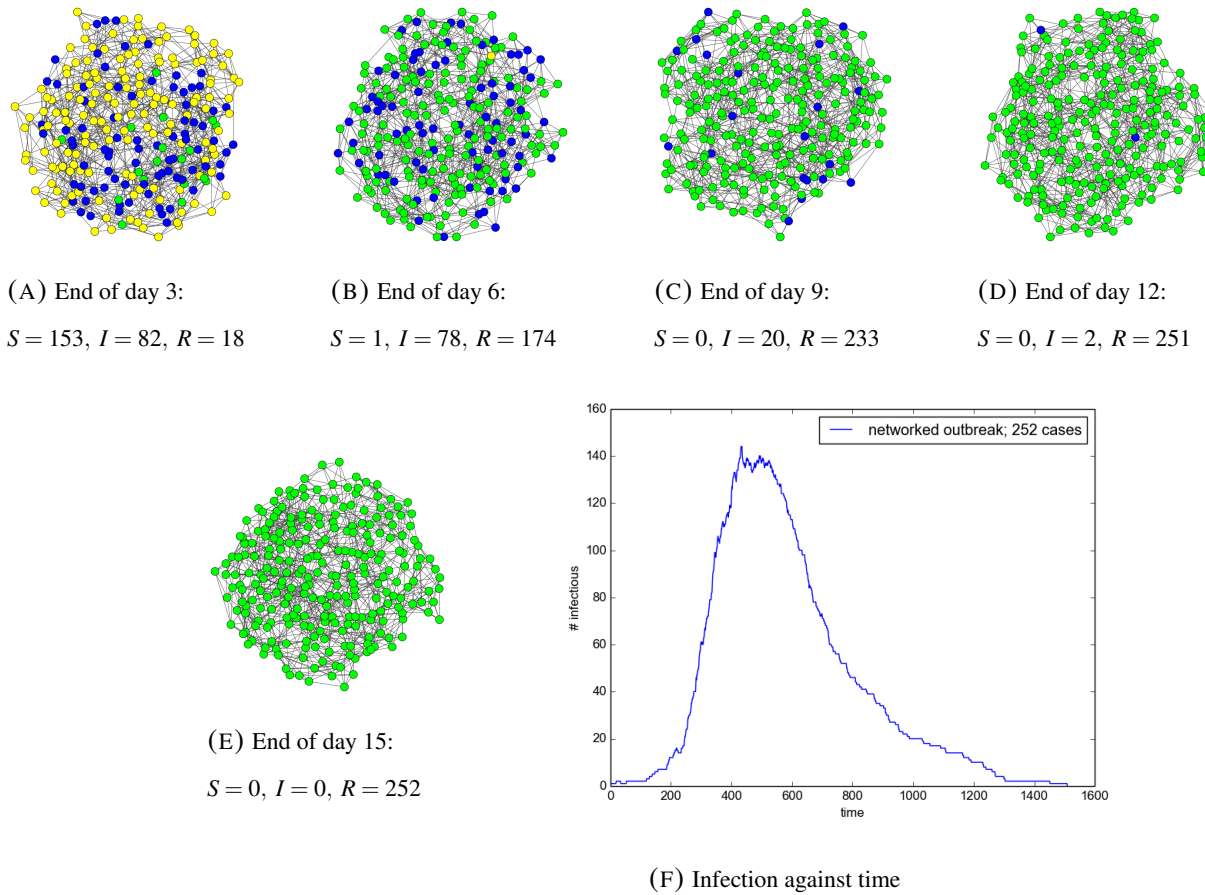

(F) Infection against time

FIGURE 6. Propagation of the spread of MVD for parameters $p_r = 0.1$, $p_s = 0.2$ and $p_j = 0.2$

tell us but also the small world effect, or clustering is seen. When we plot the graph of an SEIR model (infection against time), we expect to see a smooth curve, but using our SWN, we see the graph exhibits some level of randomness as well (see Fig. 6f). So the small world network model is better than the traditional model, since it takes into account the inherent randomness of spreading.

Fig. 7 depicts the evolution of MVD when the infectious rate is smaller than the recovery rate. The disease always dies out early when this condition holds. It shows that, within 9 days 182 individuals were infected and 73 individuals were still susceptible.
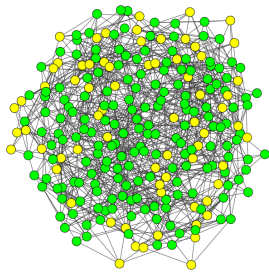
When we consider the case where p_r is low or high, the results from our simulations confirm the obvious conclusions; Low p_r means that, the infectious individuals stay longer in the population thereby infecting several people, whereas high p_r means that, the infectious individuals
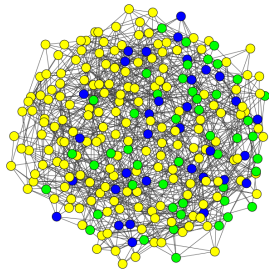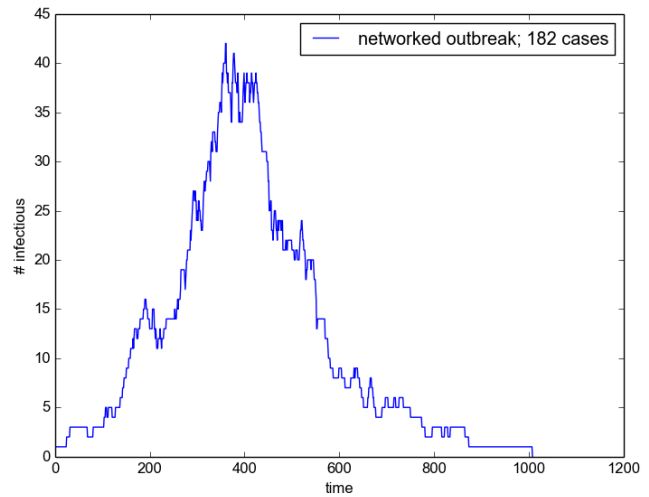
(A) End of day 3:

$S = 173$, $I = 33$, $R = 47$
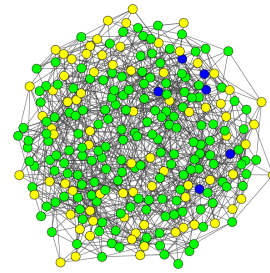
(B) End of day 6:

$S = 78$, $I = 6$, $R = 169$

(C) End of day 9:

$S = 73$, $I = 0$, $R = 182$

(D)

FIGURE 7. Propagation of the spread of MVD for parameters $p_r = 0.4$, $p_s = 0.1$ and $p_j = 0.2$

are quickly removed/recovered before they infect more people. Thus the value of p_r influences the duration of the outbreak. For instance, from Fig. 4 where p_r is low (0.1), the outbreak lasted for 18 days. On the other hand, from Fig. 7 where p_r is higher (0.4), the outbreak lasted for 6 days.

Our simulations also identify the impact of the short-range spreading and the long-range spreading parameters in the evolution of the disease. Fig. 4 and Fig. 6 depict the impact of short-range spreading rate over the long-range spreading rate. Although both parameters play a significant role, the short-range spreading rate has greater influence. The overall effect of

contacts to the spread of the disease is higher in the case where $p_s > p_j$. For instance, from Fig. 4 where $p_s < p_j$ the rate at which the susceptible individuals become infectious is lower than that of Fig. 6 where $p_s > p_j$.

5. CONTROL OF SPREAD OF MVD IN SMALL- WORLD NETWORK MODEL

Identification of each individual in the network and its importance is very crucial during the spread of the disease. Centrality is a way of measuring the importance of an individual in a network. It provides a good knowledge of how to control the spread of disease by breaking the transmission through an isolation process. There are various ways of measuring this property of a network. In this section, we discuss some centrality measures and how to apply them to control the disease spread.

5.1. Degree Centrality. Degree centrality assigns an importance score based purely on the number of links held by each individual in the network. It shows the direct connections each individual has with other individuals within the network. Degree centrality helps to find very connected individuals, popular individuals, individuals who are likely to hold most information and also individuals who can quickly connect with the entire network [21]. By cross-checking the values of degree centrality of each individual in a network we can identify the individuals who are very likely to spread the disease to several people. After identifying, those with high degree centrality can then be isolated to reduce the transmission.
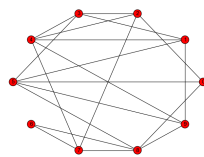

FIGURE 8. A network of 10 individuals with 20 connections

Example 2. The degree centrality of each individual in Fig. 8 is given in the table below.:

Table 1 shows that, individuals 4, 5 and 8 are more likely to hold the most information or have the highest connections within the network in Fig. 8 since they have the highest degree

TABLE 1. Degree centralities

Individual	0	1	2	3	4	5	6	7	8	9
Degree centrality	0.333	0.444	0.444	0.444	0.556	0.566	0.222	0.444	0.556	0.444

centrality. So in the case of disease spread, it is better to isolate these individuals in order to reduce transmission.

5.2. Betweenness Centrality. This measures the extent to which an individual in a network lies on the shortest path between other individuals. This measure shows which individual acts as a bridge between other individuals in a network. It identifies all shortest paths and counts how many times each individual lies between these shortest paths. Through this measure we can analyse the spread of the disease and then control the contact between distinct groups in a network by removing all individuals with the highest betweenness centrality [21].

The betweenness centrality of an individual i in a network is computed [22] as

$$BC(i) = \sum_{j \neq i} \sum_{k \neq i, j} \frac{\rho(j, i, k)}{\rho(j, k)}$$

where $\rho(j, k)$ is the number of shortest path from j to k and $\rho(j, i, k)$ is the number of those paths that pass through i.

Example 3. The betweenness centrality of each individual in Fig. 8 is given in the table below.

TABLE 2. Betweenness centralities

Individual	0	1	2	3	4	5	6	7	8	9
$BC(i)$	0.028	0.018	0.079	0.037	0.125	0.125	0.0	0.111	0.181	0.046

Table 2 shows that individual 8 is the most important bridge within the network in Fig. 8 since it has the highest betweenness centrality. So in case of disease spread, it is better to isolate that individual in order to divide the population into distinct groups hence reducing contacts.

5.3. Closeness Centrality. This measures the scores of each individual based on their closeness to all other individuals within the network. This measure calculates the shortest paths

between all individuals, then assigns each individual a score based on its sum of shortest paths. This measure helps to find the individuals who are best placed to influence the entire network quickly. During the spread of disease, this measure can help to identify the individuals who can pass information and create awareness to all individuals concerning the disease to the entire network [21].

The closeness centrality of individual i in a network G is defined as

$$CC(i) = \frac{n-1}{S(i)}$$

where $S(i)$ is the sum of distance calculated from the shortest path distance $d(i, j)$ as [22]

$$S(i) = \sum_{j \in V(G)} d(i, j)$$

Example 4. The closeness centrality of each individual in Fig. 8 is given in the table below.

TABLE 3. Closeness centralities

Individual	0	1	2	3	4	5	6	7	8	9
$BC(i)$	0.6	0.6	0.643	0.6	0.692	0.692	0.5	0.643	0.692	0.643

From Table 3, individuals 4, 5 and 8 have most influence within the network in Fig. 8 since they have highest closeness centrality. So in the case of disease spread, it is better to use those individuals to raise awareness in the entire network about the causes, symptoms and precautions of the disease for disease spread control. Also, in case they are infected, we isolate them in order to reduce or stop the transmission of the disease quickly.

6. CONCLUSIONS

Modelling a disease with small-world network model is preferred to the traditional model since it is possible to observe the topology of the spread of the disease over time within the population. While currently the MVD has no treatment, using this model, the measures of centrality can be used to control the spread. In this paper, we discussed some of the measures of centrality, which are degree centrality, betweenness centrality and closeness centrality. Through these measures of centrality we can identify the individuals who are likely to spread the disease

quicker to several people, then isolate them to stop or reduce the transmission. Finally, the small-world network model can be used to optimize the cost of control and treatment. For instance, instead of vaccinating the entire population as the traditional model may require, we trace only the susceptible individuals with high centrality and vaccinate them.

CONFLICT OF INTERESTS

The author(s) declare that there is no conflict of interests.

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