Email Virus Propagation Modeling and Analysis

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Abstract

Email viruses constitute one of the major Internet security problems. In this paper we present an email virus model that accounts for the behaviors of email users, such as email checking frequency and the probability of opening an email attachment. Email viruses spread over a logical network defined by email address books. The topology of email network plays an important role in determining the behavior of an email virus spreading. Our observations suggest that the node degrees in an email network are heavy-tailed distributed and we model it as a power law network. We compare email virus propagation on three topologies: power law, small world and random graph topologies. The impact of the power law topology on the spread of email viruses is mixed: email viruses spread more quickly than on a small world or a random graph topology but immunization defense against viruses is more effective on a power law topology.

Methods keywords: Simulations, Graph theory, Statistics.

I. Introduction

Computer viruses have been studied for a long time both by the research and by the application communities. Cohen's work [13] formed the theoretical basis for this field. In the early 1980s, viruses mainly spread through the exchange of floppy disks. At that time, only a small number of computer viruses existed and virus infection was usually restricted to a local area. As computer networks and the Internet became more popular from the late 1980s on, viruses quickly evolved to be able to spread through the Internet by various means such as file downloading, email, exploiting security holes in software, etc.

Currently, email viruses constitute one of the major Internet security problems. For example, the *Melissa* virus in 1999, "*Love Letter*" in 2000 and "*W32/Sircam*" in 2001 widely spread throughout the Internet and caused millions or even billions of dollars in damage [19][20][22]. There is, however, no formal definition of "email virus" in the virus research area — any computer program can be called an *email virus* as long as it can replicate and propagate by sending copies of itself through email messages. While *Melissa* is an email virus that only uses email to propagate [21], most email viruses can also use other mechanisms to propagate in order to increase their spreading speed on the Internet. For example, "*W32/Sircam*" can spread through unprotected network shares — the shared resources that others can access through network [23]; "*Love Letter*" can propagate through Internet Relay Chat (IRC) or network shares [25]; *Nimda* can use four other mechanisms besides email to propagate [27].

Though virus spreading through email is an old technique, it is still effective and is widely used by current viruses and worms. Sending viruses through email has some advantages that are attractive to virus writers:

- Sending viruses through email does not require any security holes in computer operating systems or software.
- Almost everyone who uses computers uses email service.
- A large number of users have little knowledge of email viruses and trust most email they receive, especially email from their friends [28][29].
- Email are private properties like post office letters. Thus correspondent laws or policies are required to permit checking email content for detecting viruses before end users receive email [18].

In order to understand how viruses propagate through email, in this paper we focus exclusively on those email viruses that propagate solely through email, such as *Melissa* virus [21] (if we overlook its slow spreading via file exchange). Thus "*email virus*" used in this paper is defined as a virus that only spreads through email by including a copy of itself in the email attachment — an email user will be infected only if he/she opens the virus email attachment. If the email user opens the attachment, the virus program will infect the user's computer and send itself as an attachment to all email addresses in the user's email address book.

A. Prior and related work

Considerable research has focused on detection and defense against email viruses. Anti-virus software companies continuously add new techniques into their products and provide email virus defense software such as SMTP gateway anti-virus system [17]. But little research has been pursued on modeling viruses and worms propagation, not even to mention email viruses propagation.

Kephart, White and Chess of IBM published a series of papers from 1991 to 1993 on viral infection based on epidemiology models [6][7][8]. [6][7] were based on a birth-death model in which viruses were spread via activities mostly confined to local interactions. They further improved their model by adding the "Kill signal" process and also considered the special model of viral spread in organizations [8]. Though at that time the assumption of local interaction was accurate because of sharing disks, today it's no longer valid when most viruses and worms propagate through the Internet. In 2000 Wang et al. studied a simple virus propagation model based on a clustered topology and a tree-like hierarchic topology [9]. In their model, copies of the virus would activate at a constant rate without accounting for any user interactions. The lack of a user model coupled with the clustered and tree-like topologies make it unsuitable for modeling the propagation of email viruses over the Internet. Recently, Staniford et al. studied *Code Red* worm propagation and presented several new techniques to improve the spreading speed of worms [10]. The worm model considered in their paper assumes that a worm can directly reach and infect any other computers, which is suitable for worms but not the case for email viruses — email viruses must pass through an email network hop-by-hop.

Some researchers have studied immunization defense against virus propagation. Immunization means that some nodes in a network are immunized and can not be infected by the virus or worm. Wang et al. showed that selective immunization can significantly slow down virus propagation for tree-like hierarchic topology [9]. From an email virus point of view, the connectivity of a partly immunized email network is a percolation problem. Newman et al. derived the analytical solution of the percolation threshold of small world topology [15][16]: if nodes are removed randomly from a small world network and the fraction of these nodes is higher than the percolation threshold, the network will be broken into pieces. Albert et al. were the first to explain the vulnerability of power law networks under attacks: by selectively attacking

the most connected nodes, a power law network tends to be broken into many isolated fragments [4]. The authors concluded that the power law topology was vulnerable under deliberate attack.

B. Our contributions

We present an email virus model that accounts for the behaviors of email users, such as email checking frequency and the probability of opening an email attachment.

Our observation shows that the size of email groups follows a heavy-tailed distribution. Since email network contains email groups, we believe an email network is also heavy-tailed distributed and we model it as a power law network.

We carry out extensive simulation studies of email virus propagation. From these experiments we derive a better understanding of the dynamics of an email virus propagation, how the degree of initially infected nodes affects virus propagation, how the network parameters such as the power law exponent affect virus behavior, etc.

For simplified email virus models, we mathematically prove that an email virus propagates faster as the email checking time becomes more variable although the average email checking time does not change.

We know better of the differences among power law, small world and random graph topologies by simulate email virus propagation on them.

We derive by simulations the selective percolation curves and thresholds for the power law, small world and random graph topologies. These selective percolation curves can explain why selective immunization defense against virus spreading is quite effective for a power law topology but not so good for the other two topologies.

C. Organization of the paper

The rest of the paper is organized as follows: We present the email virus model in Section II. In Section III we discuss the email network topology and model it as a power law topology. In Section IV, we present simulation studies of email virus propagation without considering immunization. We also compare virus spreading among power law, small world and random graph topologies. In Section V, we study the immunization defense against email viruses and the corresponding percolation problem. Section VI concludes this paper with some discussions.

II. Email virus propagation model

Because of the complexity of an email network and the randomness of email users' behaviors, it's difficult to mathematically analyze email virus propagation. Thus in this paper we will rely primarily on simulation rather than mathematical analysis. In this way we can focus on realistic scenarios of email virus propagation.

In this paper, we consider email viruses that only transfer through users' email address books. Thus email address relationship between users' address books forms a logical network for email viruses. Strictly speaking, the email logical network is a directed graph: each vertex in the graph represents an email user while a directed edge from node A to node B means that user B's email address is in user A's address book. Email address book of a user usually contains the user's friends' or business partners' email addresses. Thus if user A has user B's address, user B probably also has user A's address in his own address book, which means that many of the directed edges on the email network point to both direction. Although this may not always be true, we model the email network as an undirected graph in this paper.

We represent the topology of the logical email network by an undirected graph $G =< V, E >$, $\forall v \in V$, v denotes an email user and $\forall e = (u, v) \in E$, $u, v \in V$, represents two users u and v that have the email address of each other in their own address books. $|V|$ is the total number of email users.

Let's first describe the email virus propagation scenario captured by our model: users check their email from time to time. When a user checks his email and encounters a message with a virus attachment, he may discard a message with a viral attachment (if he suspects the email or detects the email virus by using anti-virus software) or open the virus attachment if unaware of it. When the virus attachment is opened, the virus immediately infects the user and sends out virus email to all email addresses on this user's email address book. The infected user will not send out virus email again unless the user receives another copy of the virus email and opens the attachment again.

From the above description, we see that email viruses, not like worms, depend on email users' interaction to propagate. There are primarily two human behaviors affecting email viruses: one is the *email checking time*, denoted by T_i, $i = 1, 2, \cdots, |V|$, the time interval when user i checks email; another is the *opening probability*, denoted by P_i, $i = 1, 2, \cdots, |V|$, the probability with which user i opens a virus attachment.

The email checking time T_i of user i, $i = 1, 2, \cdots, |V|$, is a random variable with an average email checking time, $E[T_i]$, determined by user i's habits. We assume that when a user checks his email, he checks all new email in his mailbox. The opening probability P_i of user i is determined by the user's awareness and knowledge of email viruses.

We assume that each user's behaviors are independent of each other. We model T_i and P_i, $i = 1, 2, \cdots, |V|$, as follows:

- Email checking time T_i of user i, $i = 1, 2, \cdots, |V|$, is exponentially distributed with the mean $E[T_i]$. We assume that $E[T_i]$ is itself a random variable, which we denote as T.
- User i opens a virus attachment with probability P_i when he checks any virus email. Let P denote the random variable that generates P_i, $i = 1, 2, \cdots, |V|$.
- Since the number of email users, $|V|$, is very large and a user's behaviors are independent of others, we assume that T and P are independent Gaussian random variables, i.e., $T \sim N(\mu_T, \sigma_T^2)$, $P \sim N(\mu_P, \sigma_P^2)$, $i = 1, 2, \cdots, N$.

An email user is called *infected* once the user opens a virus email attachment. Let N_0 denote the number of initially infected users that send out virus email to all their neighbors at the beginning of a virus propagation. Let random variable N_t denote the number of infected users at time t during email virus propagation, $N_0 \leq N_t \leq |V|$, $\forall t > 0$.

It takes time before a recipient receives a virus email sent out by an infected user. But the email transmission time is usually much smaller comparing to a user's email checking time (the time interval between a user's two consecutive email checking). Thus in our model we ignore the email transmission time.

Table. I lists most of the notations used in this paper.

III. Email network topology discussion

The email network is determined by users' email address books. The size of a user's email address book is the degree of the corresponding node in the network graph. Since email address books are private property, we have no such data to tell us what the email topology is. We have, however, examined the sizes of the more than 800,000 email groups in *Yahoo!* [11]. Thus we can use it to figure out what the topology might be like although the topology of email groups is not the complete email network topology.

TABLE I

NOTATIONS USED IN THIS PAPER

Notation	Explanation
$G =< V, E >$	Undirected graph representing the email network. $v \in V$ denotes an email user, $\|V\|$ is user population.
T_i	Email checking time of user i — the time interval between user i's two consecutive email checking, $i = 1, 2, \cdots, \|V\|$. T_i is exponentially distributed with mean value $E[T_i]$.
$E[T_i]$	Average email checking time of user i, $i = 1, 2, \cdots, \|V\|$.
P_i	Opening probability of user i — the probability with which user i opens a virus attachment, $i = 1, 2, \cdots, \|V\|$.
T	Gaussian-distributed random variable that generates $E[T_i]$, $i = 1, 2, \cdots, \|V\|$. $T \sim N(\mu_T, \sigma_T^2)$.
P	Gaussian-distributed random variable that generates P_i, $i = 1, 2, \cdots, \|V\|$. $P \sim N(\mu_P, \sigma_P^2)$.
N_0	Number of initially infected users at the beginning of virus propagation.
N_t	Number of infected users at time t, $\forall t > 0$.
$E[N_t]$	Average number of infected users at time t, $\forall t > 0$.
V_t	Number of virus email in the system at time t, $\forall t > 0$.
α	Power law exponent of a power law topology that has complementary cumulative degree distribution $F(d) \propto d^{-\alpha}$.
N_∞^h	Number of users that are not infected when virus propagation is over.
D_t	Average degree of nodes that are healthy before time t but are infected at time t, $\forall t > 0$.
$C(p)$	Connection ratio — the percentage of remaining nodes still connected after removal of the top p percent of most connected nodes from a network.
$L(p)$	Remaining link ratio — fraction of links remaining after removal of the top p percent of most connected nodes.

As mentioned in Section II, we model the email network as an undirected graph $G =< V, E >$. Let $f(d)$ be the fraction of nodes with degree d in G. The complementary cumulative distribution function (ccdf) is denoted by $F(d) = \sum_{i=d}^{\infty} f(i)$, i.e., the fraction of nodes with degree greater than or equal to d. We present the *Yahoo!* empirical ccdf of the group sizes for May 2002 in the log-log format Fig. 1.

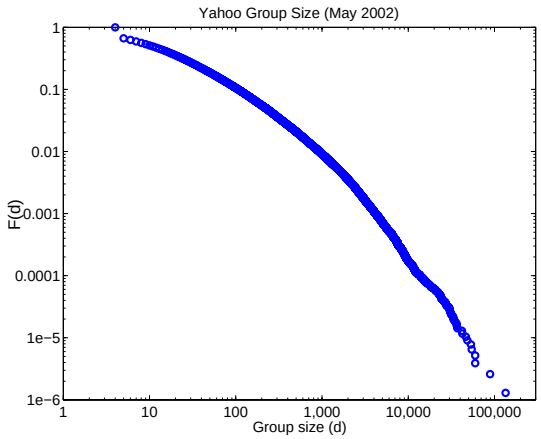


Fig. 1. Complementary cumulative distr. of *Yahoo!* group size

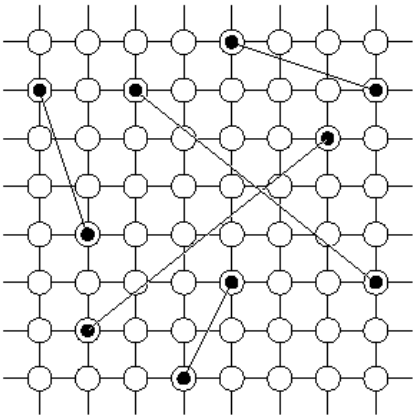


Fig. 2. Illustration of a two-dimensional small world network

The size of *Yahoo!* groups varies from as low as 4 to more than 100,000. From Fig. 1 we can see that the size of Yahoo groups is *heavy-tailed distributed*, i.e., the ccdf $F(d)$ decays slower than exponentially [12].

Currently, email lists, or called email groups, have become very popular. Once a user puts the address of an email list in his address book, from the virus point of view, the address book virtually has all the addresses contained in the email list. Since email groups are heavy-tailed distributed as shown in Fig. 1, it is reasonable to believe that email network is also heavy-tailed distributed.

In order to study email virus propagation, we need to use a network generator to create the email networks. In this paper we use the GLP power law topology generator [1] to generate power law topologies to represent email networks with a heavy-tailed node degree distribution. A power law topology is heavy-tailed distributed and has the power law ccdf $F(d) \propto d^{-\alpha}$ [1], which is linear on a log-log plot [3]. Except power law topology generators, there is no other network generator available to create a heavy-tailed distributed topology. Thus a power law topology generator is the best candidate to generate the email network although the size of *Yahoo!* groups and the degree of a real email network may not be strictly power law distributed.

There are some other popular topologies such as random graph topology [14] and small world topology [2]. They are not suitable for the email network because they do not provide heavy-tailed node degree distributions. However, we study email virus propagation on these two topologies in order to understand how different topologies affect email virus propagation.

In this paper, a random graph network of n vertices and average degree $k \geq 2$ is constructed as follows. Begin with n vertices and no edges. In order to produce a connected network, we first add n edges one by one: edge i, $i = 1, 2, \cdots, n$, connects vertex i to another randomly chosen vertex. Then at each step two randomly chosen vertices are connected with an edge until the total number of edges reaches $nk/2$.

We generate the small world network by using the model in [16], which is depicted in Fig. 2: on two-dimensional lattice each one of additional links randomly selects two nodes on the lattice to connect.

IV. Email virus simulation studies

We are interested in $E[N_t]$ — the average number of infected users at any time t. We derive $E[N_t]$ by averaging the results of N_t from many simulation runs that have the same inputs but different random seeds.

Discrete-time simulation has been used in many viruses and worms modeling papers [8][9][10]. Thus we simulate our email virus model in discrete time, too. Before the start of virus propagation, we first assign P_i and $E[T_i]$ for each user i, $i = 1, 2, \cdots, |V|$ as follows:

$$P_i = \begin{cases} P & 0 \leq P \leq 1 \\ 0 & P < 0 \\ 1 & P > 1 \end{cases} \tag{1}$$

$$E[T_i] = \max\{T, 1\} \tag{2}$$

where both random variables T and P are Gaussian-distributed and are defined in Section II (see Table. I).

At each discrete time clock t, $t = 1, 2, 3, \cdots$, if user i opens an email virus attachment, the virus will send out virus email to all user i's neighbors. These in turn will affect the activities of user i's neighbors at or after the subsequent time $t + 1$. After user i checks email at time t, a new email checking time T_i is assigned to this user in order to determine when he will check email again. In the discrete-time simulation model, T_i is a positive integer derived by:

$$T_i = \max\{\lfloor X \rfloor, 1\} \tag{3}$$

where random variable X is exponentially distributed with mean value $E[T_i]$ derived from (2).

For most experiments in the following, we perform 100 simulation runs to derive the average value $E[N_t]$. The underlying power law network has 100,000 nodes, average degree 8 and power law exponent $\alpha = 1.7$. Other simulation parameters are: $T \sim N(40, 400)$, $P \sim N(0.5, 0.09)$ and $N_0 = 2$. Initially infected nodes are randomly chosen in each simulation run. We use the same power law network and the same parameters in all simulations if not mentioned explicitly.

A. Reinfection vs. Non-reinfection

First we consider two cases under different infection assumptions: the *reinfection* case versus the *non-reinfection* case. *Reinfection* means that a user will send out email virus copies whenever he opens a virus email attachment. Thus a recipient can repeatedly receive virus email from the same infected user. *Non-reinfection* means that each infected user sends out virus copies only once, after which he will not send out any virus email even if he opens virus attachment again. Some email viruses belong to the non-reinfection case, such as *Melissa* and "*Love Letter*" [21][26]. Others belong to reinfection case, such as "*W32/Sircam*" [23][24].

Fig. 3 illustrates the behavior of $E[N_t]$ as a function of time t for both the reinfection case and non-reinfection case.

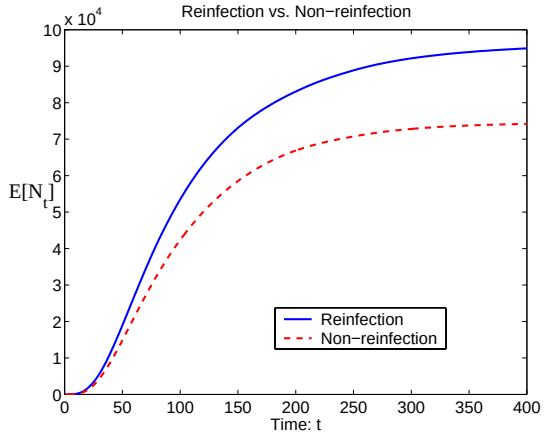


Fig. 3. Reinfection vs. non-reinfection

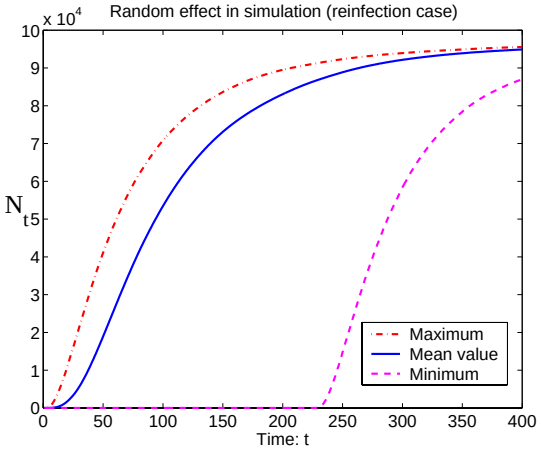


Fig. 4. Random effect in simulation (reinfection case)

In our email virus model, each user i opens an email attachment with probability P_i when he checks any virus email. Thus if user i receives m virus email, he has the probability $1-(1-P_i)^m$ to be infected — his chance of being infected increases as a function of the amount of virus email that he receives. For this reason more users are infected in the reinfection case than in the non-reinfection case as shown in Fig. 3.

Since some users open email attachment with very low probability, in both cases a certain number of users will not be infected when the virus propagation is over. Let N_∞^h denote the number of users that are not infected when virus propagation is over. We can derive some analytical results for N_∞^h for the non-reinfection case because the virus propagation of this case is relatively simple.

In the non-reinfection case, each infected user sends out only once the virus copies to all of his neighbors. Thus user i who has m_i links will receive at most m_i copies of virus email — the probability that user i is not infected is at least $(1-P_i)^{m_i}$. For the non-reinfection case, we can derive a lower bound for $E[N_\infty^h]$ if we know the network degree distribution and assume that P_i is the same for all users.

Let $G(x)$ denote the probability generating function of the degree of the email network:

$$G(x) = \sum_{k=1}^{\infty} P(d=k)x^k \tag{4}$$

where $P(d=k)$ is the probability a node has degree k. In the case that all users are equally likely to

open virus attachments, i.e., $P_i = p, \forall i \in \{1, 2, \cdots, |V|\}$, we can get a lower bound for $E[N_\infty^h]$:

$$E[N_\infty^h] \geq |V| \sum_{k=1}^{\infty} P(d = k)(1-p)^k = |V|G(1-p) \tag{5}$$

where $|V|$ is the user population.

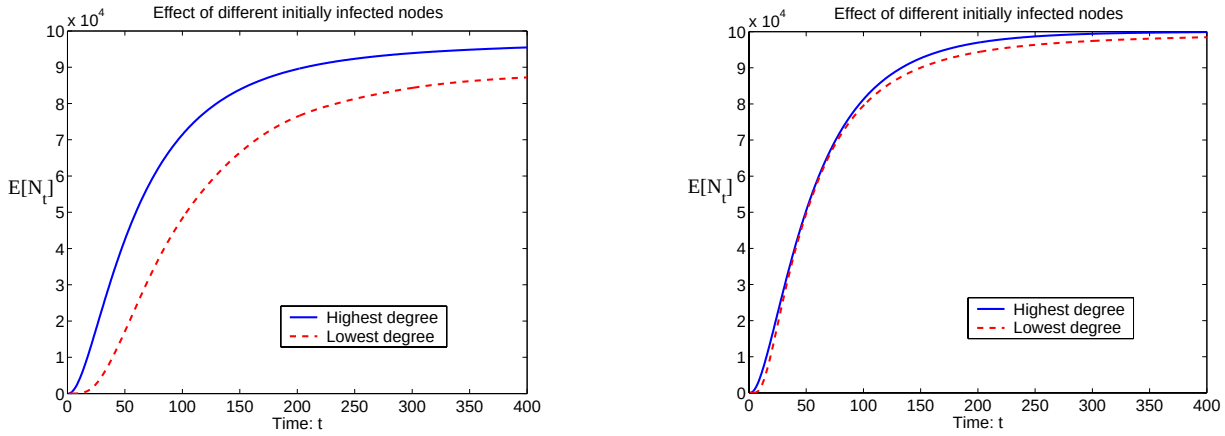
Fig. 4 shows how variable each simulation run can be. For each time t we get the maximum, average and minimum values among the previous 100 simulation runs and plot the three maximum, average and minimum simulation curves, respectively (reinfection case). We observe that the initial phase of virus spreading determines the overall propagation speed.

The email virus has successfully spread out in all 100 simulation runs in Fig. 4. In fact, the email virus has a small chance to die before it spreads out. For example, in the beginning those initially infected users send out virus copies to their neighbors. If all their neighbors decide not to open email attachment for the first round, then no virus email exists in the network after those neighbors finish checking email for the first time. If we assume that all users open virus attachments with the same probability p and the number of virus copies sent out by those initially infected users is m, then the email virus has the probability $(1-p)^m$ to die before it infects any users besides those initially infected ones.

B. Initially infected users with highest degree vs. lowest degree

In our previous experiment, the degree of the power law network varies from 3 to 1833. It seems that the degree of initially infected nodes, i.e., the size of email address books of initially infected users, is important to email virus propagation. We consider two cases: in the first case the initially infected nodes have the highest degree while in the second case the initially infected nodes have the lowest degree. Both cases have the same number of initially infected nodes, i.e., $N_0 = 2$. Fig. 5 shows the behavior of $E[N_t]$ as a function of time t of these two cases on two power law networks, respectively. Both power law networks have the same $100,000$ nodes and power law exponent $\alpha = 1.7$ but different connection density — one has average degree 8 while the other one has average degree 20.

All simulations here belong to the reinfection scenario. In the following all email virus propagation simulations belong to the reinfection scenario if not mentioned explicitly.



a. Network with average degree 8 b. Network with average degree 20

Fig. 5. Effect of different degrees of initially infected nodes

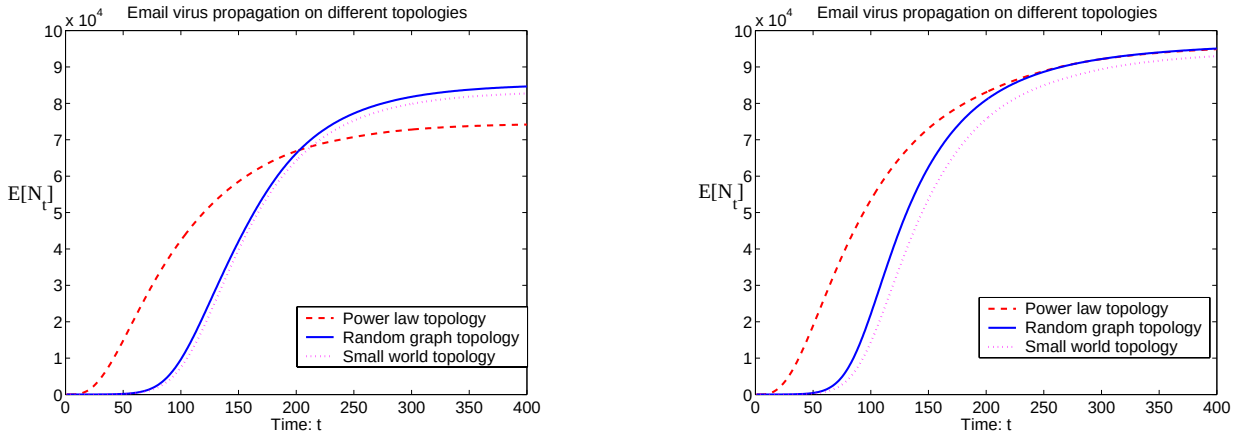
Fig. 5 shows that the identities of the initially infected nodes are more important in a sparsely connected network than a densely connected network. From a virus writer's point of view, it's important to let his

virus spread as widely as possible before people become aware of the virus or have anti-virus patches available. It will help the virus to propagate by choosing the right initial launching points such that those initially infected users have large email address books.

C. Topology effect: Power law, Small world and Random graph

In Section III, we discussed why we believe the email logical network is a heavy-tailed network. In this section we examine how topology affects email virus propagation.

We run our email virus simulation on a power law network, a small world network and a random graph network, respectively. All three networks have the same average degree 8 and $100,000$ nodes. Fig. 6 shows the $E[N_t]$ as a function of time t of these three topologies for non-reinfection case and reinfection case, respectively.



a. Non-reinfection case b. Reinfection case

Fig. 6. Effect of topology on email virus propagation

Fig. 6 shows that virus propagation on the small world network is very similar to the one on the random graph network. It is not a surprising result if we think about the relationship between a small world and a random graph topology: small world topology and random graph topology have the similar characteristic path length while small world topology has much larger clustering coefficient than random graph topology has[2].

Characteristic path length is defined as the number of edges in the shortest path between two vertices, averaged over all pairs of vertices [2]. For the email network, characteristic path length corresponds to the average number of friendships in the shortest chain connecting two users. The definition of *clustering coefficient* is complex but for the email network it measures the cliquishness of a typical friendship circle [2] — what matters to the speed of email virus propagation is the characteristic path length, not the clustering coefficient.

From Fig. 6 we observe that the virus infection speed on power law topology is much greater than the speed on either a small world or random graph topology. One reason is that the characteristic path length of a power law network is smaller than the one of a small world or a random graph network [1][5], the virus can reach and infect a node earlier by passing through a shorter path on a power law network than on a small world or a random graph network.

Another reason is that an email virus exhibits more "firing power" on a power law network at the early stage of virus propagation. On a power law network, the degree of different nodes varies significantly [3]. Once the virus infects some highly connected nodes, a much larger number of virus copies will be

sent out from these nodes. Thus the infection speed will be "amplified" by these highly connected nodes. Neither a small world nor a random graph network exhibits such amplification because all nodes on these networks have a similar degree.

Let D_t denote the average degree of nodes that are healthy before time t but are infected at time t. D_t tells us what kind of node is being infected at each time t, $t = 1, 2, 3, \cdots$. We repeat the experiment in Fig. 6(b) and derive D_t for each topology by averaging the results of $1,000$ simulation runs. We plot each D_t of these three networks as a function of time t in Fig. 7. Note that D_t is very similar in small world and random graph networks.

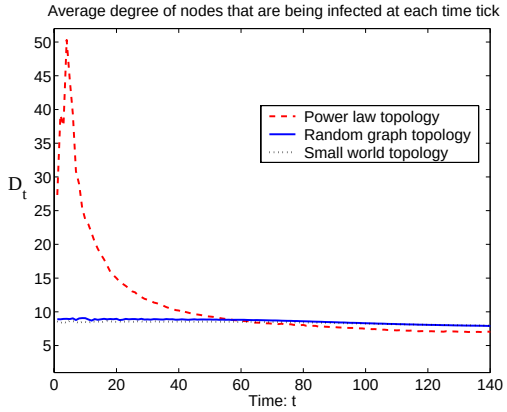


Fig. 7. Average degree of nodes that are being infected at each time tick

Fig. 7 clearly shows that email virus propagation on power law network has two phases. In the beginning, virus will infect the most highly connected nodes. Later on it will primarily attempt to infect nodes that have small degree. These two phases are not exhibited by either a small world network or a random graph network.

In the non-reinfection case, once the virus propagation is over, more nodes remain healthy on a power law topology than on a small world or a random graph topology (see Fig. 6(a)). As explained in the first experiment, the probability that a node will be infected decreases as the degree does. Since these three topologies have the same average degree, compared to the small world and random graph topologies, more nodes in the power law topology have degree less than the average degree. Thus more nodes in the power law topology will remain healthy at the end of the virus propagation than in the other two topologies.

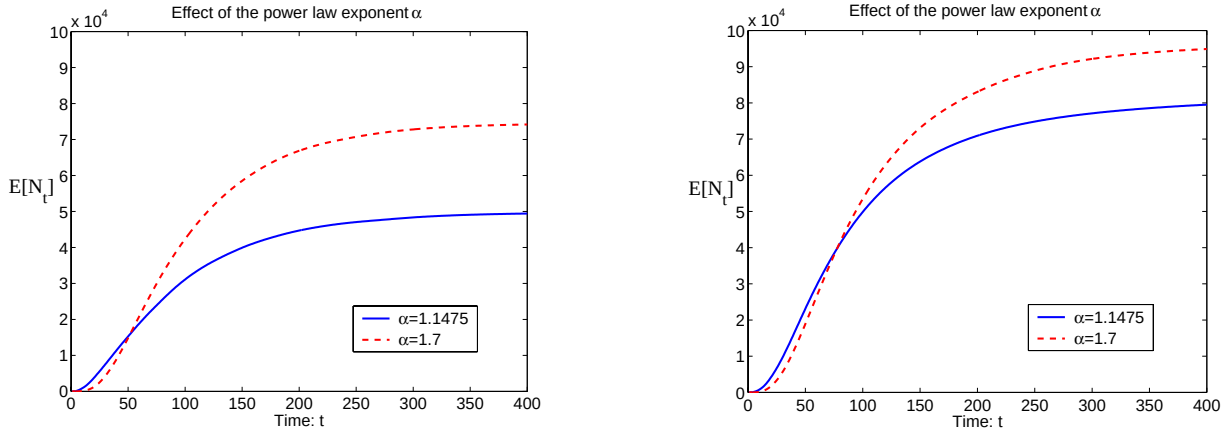
We also investigate the sensitivity of our results to tenfold increase in the number of users by running the same simulations as in Fig. 6 where the number of nodes is $1,000,000$ and average degree remains 8. we observe the same behaviors of virus propagation on tenfold larger networks for all three topologies. The results show that the behavior of virus propagation doesn't change when the network scale changes.

D. Effect of the power law exponent α

The power law exponent α is an important parameter for a power law topology. It is the slope of the curve of the complementary cumulative degree distribution in a log-log graph [1] — the smaller α is, the more variable is the degree of the topology. In our previous simulations, we use $\alpha = 1.7$ to generate the power law network with $100,000$ nodes and average degree 8. This power law network has the highest degree 1833 and the lowest degree 3. Since the degree corresponds to the size of email address book, we think $\alpha = 1.7$ will give us a reasonable power law email network that has $100,000$ nodes.

The Internet AS level power law topology has the power law exponent $\alpha = 1.1475$ [1]. Using $\alpha = 1.1475$ for a $100,000$ nodes power law network with average degree 8 will produce a network with the highest degree up to $28,000$ and lowest degree of 1. Thus we think $\alpha = 1.1475$ is too small for modeling the email network.

However, we don't know the value of α for the real email network. In order to see how the power law exponent α affects virus propagation, we compare virus propagation on the following two power law networks: one has $\alpha = 1.7$ and the other one has $\alpha = 1.1475$. Both networks have $100,000$ nodes and average degree 8. We denote the network with $\alpha = 1.7$ as the power law network Net_a and the network with $\alpha = 1.1475$ as the power law network Net_b. $E[N_t]$ is plotted for both networks as functions of time t in Fig. 8 for the non-reinfection and reinfection cases, respectively.



a. Non-reinfection case

b. Reinfection case

Fig. 8. Effect of power law exponent α on email virus propagation

For both cases, Fig. 8 shows that an email virus initially propagates faster on power law network Net_b than on Net_a. Later, however, the virus spreads more quickly on Net_a than on Net_b.

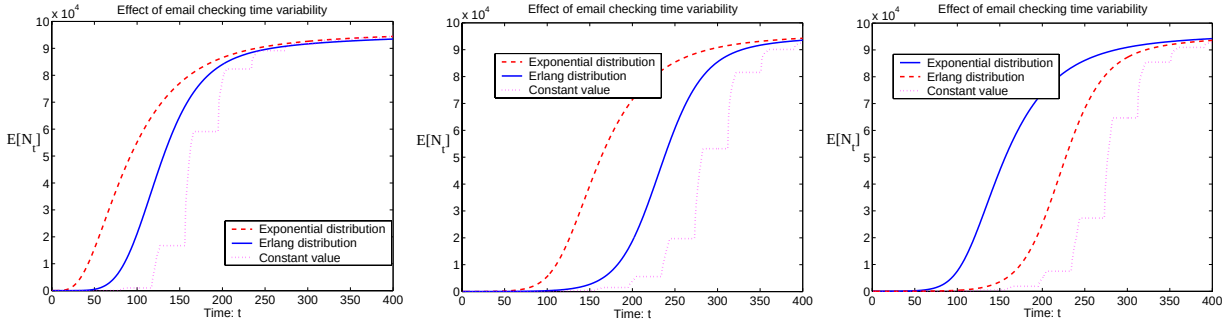
Power law network Net_b concentrates a large number of links on a small number of nodes. Once some of these have been infected, there will be more copies of virus sent out than in network Net_a. Those highly connected nodes are like "virus amplifiers" in email virus propagation. Thus the initial virus infection speed is larger on Net_b than on Net_a.

After having infected most highly connected nodes, the email virus enters the second phase, mainly trying to infect the nodes that have small degree. In power law network Net_b, more nodes have smaller degree than in network Net_a — the smallest degree in Net_b is one while in Net_a it is three. A node with a smaller number of links than another node is less likely to receive virus email. Thus during the second phase of virus propagation, the virus infection speed in Net_b is smaller than the speed in Net_a.

E. Effect of email checking time variability

In our email virus model, we assume that user i's email checking time, T_i, is exponentially distributed with mean $E[T_i]$, $i = 1, 2, \cdots, |V|$. What if the email checking time is drawn from some other distributions, like Erlang distribution, or is simply a fixed value? What effect will the variability of email checking time have on the email virus propagation?

Fig. 9 shows the average number of infected users, $E[N_t]$, under three different simulation cases (on a power law network, a small world network and a random graph network, respectively). In order to study the effect of the variability of email checking time without other disturbance, we let all users have



a. Power law topology b. Small world topology c. Random graph topology

Fig. 9. Effect of the variability of email checking time on virus propagation

the same average email checking time $E[T_i] = L$, $\forall i \in \{1, 2, \cdots, |V|\}$. In the first case we let email checking time be exponentially distributed with mean value L. In the second case we use a 3rd-order Erlang distribution with mean value L. In the third case all users have a constant email checking time L. In the simulations of Fig. 9, we let $L = 40$. For each simulation runs we randomly select 10 nodes as initially infected nodes, i.e., $N_0 = 10$.

Given the same mean value L the exponential distribution is more *stochastically variable* [30] than the *k*th-order Erlang distribution where $k > 1$. Both of them are more stochastically variable than the constant value L. Fig. 9 shows the same relationship among these three cases on all topologies: virus propagation speed is not only determined by the average spreading time, but also is affected by the variability of the spreading time — an email virus propagates faster as the email checking time becomes more variable.

Unfortunately, email virus propagation is a very complex process that we can't strictly prove the above conclusion. But for certain simplified email virus models, we can derive the formula of $E[N_t]$ and show why the variability of email checking time counts. The intuitive answer is what we refer to as the so called *snowball* effect: before virus copies in the system with less variable checking time give birth to the next generation — infecting some new users, virus copies in another system with more variable checking time have already given birth to *several* generations although each generation's population is relatively small.

We introduce the simplified model in the following: suppose the email virus propagation is a discrete-time process as we have used in our simulations. A user always opens an email virus attachment when he checks email. Once an email virus attachment is opened, it sends virus email to C vulnerable users. Last, we assume the user population is infinite. Each vulnerable user that receives virus email only receives one copy — no virus email will be wasted trying to infect an infected user again. When an email virus copy is activated by a user, it infects the user, generates C new copies of virus email, and then disappears. Suppose at the beginning, $t = 0$, all users are vulnerable but not infected, i.e. $N_0 = 0$, and V_0 users have one copy of virus email on each of them. Let V_t denote the number of virus copies existing in the system at time t. Note that $V_t \neq N_t$, $t > 0$ and C is a positive integer.

Let's compare the following two cases: In the first case every user has a constant email checking time denoted by L (L is a positive integer) while in the second case every user has an exponentially distributed email checking time with mean L. Thus all users have the same average email checking time in these two cases.

For the first case, the virus propagation is a deterministic process. N_t and V_t are updated only when

$t = kL$, $k = 1, 2, 3, \cdots$:

$$\begin{cases} V_{(k+1)L} &= CV_{kL} \\ N_{(k+1)L} &= V_{kL} + N_{kL} \\ N_0 = 0 & k = 0, 1, 2, 3, \cdots \end{cases} \tag{6}$$

Note that L is the mean email checking time for all users, C is the number of virus email sent out by an infected user. From the iterative equation (6), we get:

$$N_t = \begin{cases} V_0 \lfloor t/L \rfloor & \text{if} \quad C = 1 \\ V_0 (C^{\lfloor t/L \rfloor} - 1)/(C-1) & \text{if} \quad C > 1 \end{cases} \tag{7}$$
$$t = 1, 2, 3, \cdots$$

For the second case where email checking time is a random variable, we can use the mean value analysis to get the average value $E[N_t]$ and $E[V_t]$. Since the email checking time is exponentially distributed with mean L, at any time $t+1$, each one of the existing V_t virus copies has probability $(1 - e^{-1/L})$ of being opened by the user who receives it regardless of what the time t is and how long the virus copy has been received by the user. Thus we can derive the following recursive equation describing $E[N_t]$ and $E[V_t]$:

$$\begin{cases} E[V_{t+1}] &= e^{-1/L} E[V_t] + C(1 - e^{-1/L}) E[V_t] \\ E[N_{t+1}] &= E[N_t] + (1 - e^{-1/L}) E[V_t] \\ E[N_0] = 0 & E[V_0] = V_0, t = 0, 1, 2, 3, \cdots \end{cases} \tag{8}$$

From the equation (8), we get:

$$E[N_t] = \begin{cases} V_0 (1 - e^{-1/L}) t & \text{if} \quad C = 1 \\ \frac{V_0 [C(1 - e^{-1/L}) + e^{-1/L}]^t}{C-1} & \text{if} \quad C > 1 \end{cases} \tag{9}$$
$$t = 1, 2, 3, \cdots$$

Comparing equation (7) and (9), if $C = 1$, it's hard to tell in which case an email virus propagates faster. But what we are interested is the normal situation where $C \geq 2$ and $L \geq 2$. When $L \geq 3$ and $C = 2$, or $L \geq 2$ and $C \geq 3$, $E[N_t]$ in the equation (9) is always larger than N_t in the equation (7), $\forall t \geq 1$. This means that a more variable checking email time can help the virus to propagate faster.

Here we rely heavily on the memoryless property of the exponential distribution in our analysis. Hence we have been unsuccessful in obtaining an expression for $E[N_t]$ for other distributions.

V. Immunization, Percolation for Email Virus Defense

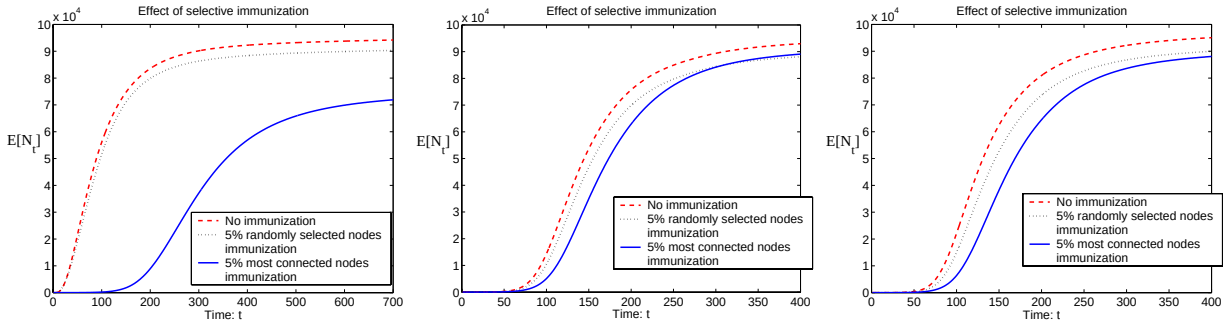
In this section, we consider immunization defense against email virus. For the email network, immunizing a node means that the node can't be infected by the virus. In this paper we consider a *static* immunization defense. By this we mean that before an email virus starts to propagate, a small number of nodes in the network have already been immunized. If some email users are well educated and they never open suspicious email attachment, they can be treated as immunized nodes in the email network.

A. Effect of selective immunization

It's unfeasible for us to immunize all email users, i.e., immunize all nodes in the email network. A realistic approach is to immunize a subset of nodes. Thus we need to know how to choose the appropriate size and the membership of this subset in order to slow down or constrain the email virus spreading as best as we could.

Wang et al. explained that selective immunization could significantly slow down virus propagation for tree-like hierarchic topology [9]. We find that for power law email network, selecting highly connected nodes to immunize is also quite effective against virus propagation.

We simulate virus propagation under two different immunization defense methods: in the first case we randomly choose 5% nodes to immunize while in the second case we choose 5% most connected nodes to immunize. We plot $E[N_t]$ as a function of time t for these two immunization methods in Fig. 10 (on a power law network, a small world network and a random graph network, respectively). In order to see the effect of immunization, we also plot $E[N_t]$ for the original case where there is no immunization.



a. Power law topology b. Small world topology c. Random graph topology

Fig. 10. Effect of selective immunization on email virus propagation (reinfection case)

We observe from Fig. 10 that selective immunization plays an important role for a power law topology while it has little effect for a small world topology or a random graph topology. On a power law email network, we can significantly slow down email virus propagation by selecting those most connected nodes to immunize.

The results here are consistent with the conclusions in [4]. The authors in [4] showed that selectively attacking the most connected nodes rapidly increases the diameter of a power law network. Since an email virus depends on the connectivity of the email network to spread, immunizing the most connected nodes has the effect of rapidly increasing the network diameter. This in turn significantly slows down virus propagation.

B. Selective percolation and email virus prevention

We have observed that selective immunization is quite effective for a power law email network. Then what is the appropriate size of the subset to immunize? How many nodes do we need to immunize in order to prevent an outbreak of an email virus?

From an email virus point of view, the connectivity of a partly immunized email network is a percolation problem. Newman et al. derived the analytical solutions of percolation on small world networks [15][16]. The "percolation" in these paper means removing some nodes from the networks *uniformly*. Since we want to study the effect of selective immunization, we introduce the corresponding concept *selective percolation*. For example, a selective percolation value is p means to remove the top p percent of the most connected nodes from the network.

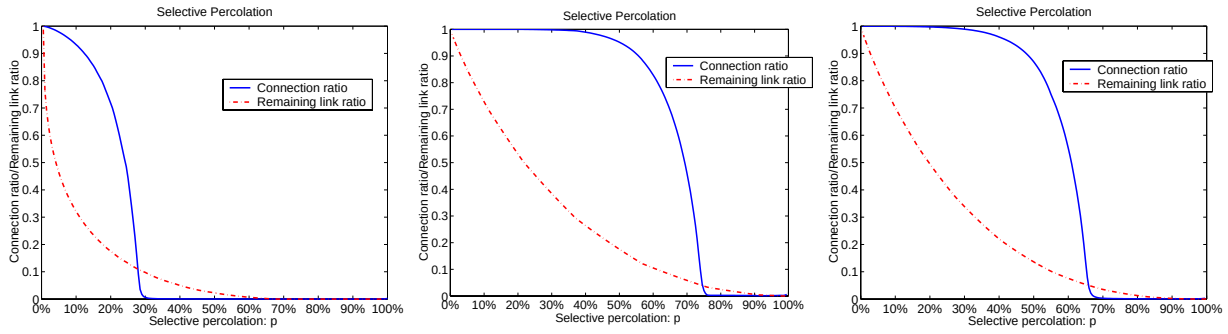
Suppose the email graph $G =< V, E >$ has $|V|$ nodes and $|E|$ edges. For a selective percolation value p, $0 < p < 1$, let $C(p)$ denote the *connection ratio*, the percentage of how many remaining nodes still connected after removing the top p percent of the most connected nodes from the network. Let $L(p)$ denote the *remaining link ratio*, the fraction of links remaining after removal the top p percent most connected nodes from the network.

$$\begin{cases} C(p) & = c_p/(|V| - |V|p) \\ L(p) & = (|E| - e_p)/|E| \end{cases} \quad 0 < p < 1 \tag{10}$$

where e_p is the number of removed edges and c_p is the size of the largest cluster in the remaining network when we remove the top p percent most connected nodes.

We generate 100 networks for each type of the three topologies, power law, small world and random graph topologies. Each network has average degree 8 and $100,000$ nodes. For every selective percolation value p we calculate $C(p)$ and $L(p)$ by averaging those 100 numbers derived by equation (10) from each of these 100 networks. Thus $C(p)$ and $L(p)$ here are properties of the corresponding topology, not of one single network.

For each of the three topologies, we plot $C(p)$ and $L(p)$ as functions of the selective percolation value p in Fig. 11.



a. Power law topology b. Small world topology b. Random graph topology

Fig. 11. Selective percolation on three topologies

Fig. 11(a) shows that a power law topology has a selective percolation threshold (the threshold here is about 0.29). If the fraction of users selectively immunized exceeds this threshold, the email network will be broken into separated fragments and no virus outbreak will occur.

The selective percolation threshold of a power law topology is much smaller than that for either a small world topology or a random graph topology. Although a power law topology is more vulnerable under deliberate attacks [4], it benefits more from a selective immunization defense.

From Fig. 11(a) we can see that when we immunize the top 5% of most connected nodes in a power law network, though 97.5% of remaining nodes in the network are still connected, 55.5% of the network edges have been removed. Thus an email virus has fewer and longer paths to reach and infect nodes in the remaining network. Fig. 11(b)(c) show that this is not the case for a small world topology or a random graph topology, a 5% selective immunization removes fewer than 20% edges.

VI. Conclusion

In this paper we presented an email virus model by considering email users' behaviors such as email checking frequency and the probability to open an email attachment. We explained why we believe email network can and should be modeled by a power law topology. We carried out extensive simulations of email virus propagation and mathematically proved that the variability of email checking time affects virus spreading speed. We also studied the effect of topology on virus propagation on power law, small world and random graph topologies. From these simulation studies, we have derived a better understanding of email virus's behaviors and also the differences among power law, small world and random graph topologies.

Compared to small world and random graph topologies, the impact of power law topology on email virus is mixed: on the one hand, an email virus will spread faster on a power law topology than on a

small world or a random graph topology; on the other hand, it is more effective to carry out selective immunization on a power law topology than on the other two topologies.

In this paper we mainly use simulation to study email virus propagation. The next step is to mathematically analyze email virus spreading. We also only considered a static immunization defense. In real email virus propagation, some infected users will recover and develop an immunity to the email virus during virus propagation. Thus in the real world, the immunization defense is a dynamic process. Considering this dynamic process will give us more accurate modeling and prediction of email virus propagation. In this paper we assume that the relationship of all email addresses is bi-directional, which may not be true for some email users. In the future we need to consider directed graph for email network in order to get a better picture of email virus propagation.

VII. Acknowledgement

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