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A note on global stability of the virose equilibrium for network-based computer viruses epidemics

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ABSTRACT

It has been recently conjectured in Yang et al. (2013) [27] that the unique virose equilibrium of a network-based model is globally asymptotically stable in the feasible region. The aim of this paper is to investigate the global stability of the virose equilibrium by using the direct Lyapunov method and appropriate grouping techniques for Lyapunov function derivatives. Some sufficient conditions are established, which improves the relevant results. Moreover, we provide a new brief proof for global stability of the virus-free equilibrium by using comparison method, and discuss quantitatively the effect of various immunization schemes, including uniform immunization, targeted immunization and high-risk immunization, on the viruses spread, which suggests promising strategy to halt viruses spread in computer networks.

1. Introduction

The novel network-based susceptible-infectious-susceptible (SIS) model (i.e., infected nodes go back to the susceptible state once recovered, without any immunity), pioneered by the seminal papers of Pastor-Satorras and Vespignani [1,2] in 2001, describes the transmission of infectious diseases between susceptible and infectious individuals constrained by network links and provides a basic framework for almost all later network epidemic models, including stochastic epidemic models using Monte Carlo approach or agent-based methods (ABM). Since then, lots of extensions and variants to the Pastor-Satorras and Vespignani model [3–15] are emerging.

The intimate relationship between epidemiology and network theory dates back to the mid-1980s [16,17] and it might be argued that epidemiology has embraced the potential of network theory more than any other discipline. Traditional compartmental epidemic models usually assume that agents are homogeneously distributed, that is, each agent has the equal chance of contacting any other agent in the group. This is an overly simplified assumption, and is generally unrealistic. In fact, the spread of infectious diseases depends not only on biological characteristics of the disease, but also on contact patterns in a population. Contacts and interactions among the whole population naturally form a network, characterized by the local structures of the population, which are neglected by classical compartmental models. Hence, one important effort along this direction is the usage of network-based models. Under such a framework, each agent corresponds to a node of the net-

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work, and interaction relation between two agents corresponds to a link between two nodes; these two nodes are neighbors of each other.

Computer viruses are defined as small malicious programs developed to damage computer systems via modifying data, stealing information, encrypting files, formatting disks, etc. Nowadays, these virus codes become more and more complex, being able to produce mutations of themselves, and their detection and removal by anti-virus programs is more difficult [18]. In addition, they can steal personal data from computer users, such as passwords and bank accounts, causing severe damages to individuals and corporations [19]. Inspired by classical epidemiological models for disease propagation [20], Kephart et al. [21] published a seminal paper triggering the efforts on the development of anti-virus programs, responsible for the detection and removal of viruses. Although these programs have a great upgrading power, they only provide us with simple vaccines against diseases, and cannot predict the long-term behavior of computer viruses when an infection is established in a machine. Furthermore, Kephart et al. [22] established an epidemiological counterparts from macroscopic approach to show the long-term behavior of a virus propagation by investigating the network connections graph. From then on, a vast amount of modeling work has been proposed to investigate their complex dynamical properties [23–27], to name but a few.

Most of the previous models assume that all computers are linked uniformly. However, the finding at the end of last century shows that the Internet topology asymptotically follows a power-law degree distribution [28,29], which arouses the interest in understanding the effect of network topology on virus spreading, leading to a surprising result that the absence of epidemic threshold in infinite size scale free network [1]. A little attention has been paid to the network-based computer viruses models. Recently, Yang et al. [27] formulated a network epidemic model to understand the way that computer viruses spread across the Internet. They mainly focused on determining the epidemic threshold and global stability of the virus-free equilibrium, and conjectured that the unique positive equilibrium is globally asymptotically stable in the feasible region. However, they did not investigate the global stability of the positive equilibrium. As is pointed out in [30] that "A major project in deterministic epidemiological modeling of heterogeneous populations is to find conditions for local and global stability of the equilibria and to work out the relations among these stability conditions, the threshold of epidemic takeoff, and endemicity, and the basic reproduction number".

Indeed, determining the conditions under which a disease, irrespective of the initial infections, either remain endemic or become extinct is probably the most important issue. In mathematical epidemiology, the vast majority of efforts have been conducted to the investigation of global stability. For 2-dimensional systems, phase methods, such as Poincaré-Bendixson theorem, Dulac criteria or condition of Busenberg and van den Driessche [31], could be used to prove global stability by ruling out periodic orbits, while global stability for the endemic equilibrium of SEIR models with constant size has long been conjectured but only proven in 1995 [32], whose proof relies heavily on the competitive structure of the system. Actually, there is no general approach to the global stability problem, especially for high-dimensional nonlinear systems [33,34]. For arbitrary dimensional system, the most promising method may be that of Lyapunov. The systematic use of Lyapunov function in studying stability problem is relatively recent. The exception is the result of Lajmanovitch and Yorke [35] evoked before. On the other hand, another key problem in mathematical epidemiology is whether the virus propagation can be effectively controlled through immunization scheme aiming at a portion of the population. Hence, we also discuss the effect of different immunization strategies on viruses spread.

The aim of this paper, on the contrary, is to investigate the global stability of the positive equilibrium of the network-based viruses spread model, and discuss the effect of various immunization schemes. The demonstrations will be straightforwardly performed by using the direct Lyapunov method and appropriate group techniques for Lyapunov function derivatives. Moreover, we give a new brief proof for the global asymptotic stability of the virus-free equilibrium through standard comparison principle.

The paper is organized as follows. The model is described and parameters of the model are interpreted in Section 2. For completeness, we discuss the existence of the equilibria and give a new brief proof for the global asymptotic stability of the virus-free equilibrium in Section 3. In Section 4, we investigate global stability of the positive equilibrium by using the direct Lyapunov method and appropriate group techniques for Lyapunov function derivatives. In Section 5, various immunization strategies are discussed and compared. Section 6 ends with some final remarks on the problem and with some considerations on the future work.

2. The model description

Under a network framework, the nodes of network represent computers and communication links between them present possible connections. All nodes can only exist in one of the three discrete states, susceptible, exposed (infected but not infectious), and infectious, with size denoted by $X(t), Y(t), Z(t)$ at time t, respectively. The total size is $N(t) = X(t) + Y(t) + Z(t)$. Typically after the initial infection, a computer stays in a latent period before becoming infectious. The paper mainly focus on the SEIS model in which both the latent period and the infected period have infectious force.

It is well known that the degree distribution of a internet asymptotically follows a power-law form [28,29], i.e., $P(k) \propto k^{-\gamma}$ $(2 < \gamma \leq 3)$, where $P(k)$ is the probability that a node chosen randomly from the internet is of degree k. To account for this heterogeneous degree of nodes, we denote by $X_k(t), Y_k(t), Z_k(t)$ the number of susceptible, exposed, and infectious nodes of degree k at time t, respectively. Thus, $X(t) = \sum_k X_k(t)$, $Y(t) = \sum_k Y_k(t), Z(t) = \sum_k Z_k(t)$, $N_k(t) = X_k(t) + Y_k(t) + Z_k(t)$ and