Epidemic Threshold in Dynamic Switching Networks

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Abstract—In this paper, we analyze dynamic switching networks where the networks switch arbitrarily among a set of topologies. For this class of dynamic networks, we derive an epidemic threshold considering the SIS epidemic model. First, an epidemic probabilistic model based on assumption of independence between states of nodes is developed. We identify conditions for the epidemic dying out by linearizing the underlying dynamical system and analyzing its asymptotic stability around the origin. The concept of joint spectral radius is then used to derive the epidemic threshold which we validate using a myriad of networks (Watts-Strogatz, Barabasi-Albert, MIT reality mining graphs, Regular, and Gilbert). A simplified version of epidemic threshold is proposed for undirected networks. Moreover, in the case of static networks, it is shown that the derived epidemic threshold matches with the conventional analytical results. Then, it is proved that analytical results for epidemic threshold of dynamic networks are applicable to periodic networks. We demonstrate for dynamic regular networks, the epidemic threshold is the same as the epidemic threshold for static regular networks. An upper bound for the probability of epidemic spread out in dynamic Gilbert networks is also derived and verified using simulation.

Index Terms—Dynamic Networks, Epidemic Threshold, Dynamical System.

I. INTRODUCTION

Epidemics typically start with some initial infected nodes. Infected nodes can cause their healthy neighbors to become infected with some probability. With time and in some cases with external intervention, infected nodes can be recovered and go back to a healthy state. The study of epidemic dispersals on networks aims at explaining how epidemics evolve and spread in networks. One of the most interesting questions regarding an epidemic spread in a network is whether the epidemic dies out or results in a massive outbreak. Epidemic threshold is a parameter that addresses this question by considering both the network topology and epidemic strength. Epidemic spread in static networks has been studied extensively [11, 2, 3, 4, 5]. In the recent years, dispersal of epidemics in dynamic networks has garnered a lot of attention and been studied considerably [8, 9, 10, 11, 12, 13, 14, 15, 16, 17, 18, 19]. It is remarkable that in most realistic cases of epidemic spread, the underlying networks are dynamic and links between nodes are functions of time. For example, in the area of human disease epidemics, contact networks between people are not fixed and change over time because people continuously move from one location to another location. Another example is malware propagation in mobile adhoc networks. The movement of users results in dynamic topologies. Many Bluetooth devices are becoming susceptible to viruses such as Cabir and Comm Warrior. Another classic example of dynamic networks is underlying networks for the spread of diseases between animals or plants where the factors that influence the spread of disease-carrying spores are typically dynamic.

In this paper, we consider the SIS (Susceptible-Infected-Susceptible) model for epidemic spread. In this model, healthy nodes can become infected through their infected neighbors and infected nodes have the probability to become recovered. We should notice that in the SIS model, an infected node’s state after being recovered is susceptible again. We assume that infected nodes have the same recovering probability of $\delta$ and every infected node can make its healthy neighbor infected with infection probability $\beta$. There are plenty of examples in the real world for which the SIS model is the right choice to model epidemics. For example, several diseases caused by bacteria do not produce an immune response in the body. So, the recovered individual return to the susceptible population. Also, the SIS model can be used for opinion analysis. In a contact network of people, every person can have either positive or negative attitude toward a subject and he can constantly change his attitude from being positive to negative and vice versa.

In this paper, we study dynamic switching networks where at each step the adjacency matrices of networks are randomly chosen from sets of matrices. It is notable that we do not consider any temporal correlation between subsequent adjacency matrices. In other words, we assume that the process of choosing the adjacency matrix at time index $t$ is independent from the chosen adjacency matrices at the previous time indices. This assumption is not realistic for the dynamic networks with strong temporal correlations between successive
adjacency matrices.

First, the nonlinear dynamical system of infection probabilities of nodes based on the assumption of independence among the state of nodes is developed. Then, we prove that the origin is always one equilibrium point of this time-varying dynamical system and its stability depends on the network topology and the values of $\delta$ and $\beta$. After that, the linearized version of the nonlinear epidemic system is derived to determine whether the origin is asymptotically stable or not. We show that if the origin is not a stable equilibrium of the system, the epidemic spreads out, otherwise it dies out. Then, the joint spectral radius of a set of matrices is defined. In Theorem 1 we employ the concept of the joint spectral radius to derive the analytical epidemic threshold for dynamic networks. In Theorem 2 the simplified version of epidemic threshold for undirected networks is derived. Since the epidemic threshold for undirected networks depends only on the largest spectral radius of a set of system matrices, evaluation of the epidemic threshold is computationally less expensive compare to directed networks. In corollary 1 it is shown that the derived epidemic threshold confirms the conventional analytical results for static networks. Then, the proposed epidemic threshold for dynamic networks is extended to the case of periodic networks. Moreover, we study epidemic spread in dynamic regular networks and show that the epidemic threshold for dynamic regular networks is the same as static regular networks. An upper bound for the probability of an epidemic spreading out in dynamic Gilbert networks is derived. Finally, we simulate epidemics in Watts-Strogatz, Barabasi-Albert, Regular, and dynamic Gilbert networks to validate our theoretical analysis.

In this section, we develop a dynamical system for epidemic spread based on the assumption of independence between nodes. In the case of a SIR model, the epidemic threshold in a network is determined based on the number of susceptible nodes and the infection probability. The SIS model, on the other hand, assumes that nodes can recover from infection and become susceptible again.

In Theorem 1 (Mobility model threshold) of their paper, they state that if the dynamic behavior of a time-varying network can be characterized by $T$ repeating alternating graphs, $L = \{A_1, A_2, \ldots, A_T\}$. Then, the system matrix, $S$, of this dynamical system can be expressed as

$$S = \prod_{i=1}^{T} [(1-\delta)I + \beta A_i],$$

where $A_i$’s dimension is $n \times n$, $n$ is the number of nodes, $I$ is an $n \times n$ identity matrix, and $\delta$ and $\beta$ denote, respectively, recovering probability and infection probability. They prove that if the spectral radius of the system matrix is less than one, the origin is an asymptotically stable equilibrium point of the system and the epidemic dies out. It is noticeable that this result holds for the cases where there are repeating patterns of adjacency matrices and the order of repetition is preserved. In [9], the same authors as [8] study malware propagation on mobile ad hoc networks. They extend their result for epidemic threshold of periodic networks in [8] to general cases in which repeating order of adjacency matrices can be arbitrary. In Theorem 1 (Mobility model threshold) of their paper, they state that if a mobility model can be represented as a sequence of connectivity graphs $L = \{A_1, A_2, \ldots, A_T\}$, one adjacency matrix $A_t$ for each index $t \in \{1, 2, \ldots, T\}$, then the epidemic threshold is

$$\tau = \lambda_S,$$

where $\lambda_S$ is the largest eigenvalue of the matrix $S$ defined in Eq. (1).

This Theorem claims that the condition for asymptotic stability of the origin for a given dynamic network whose adjacency matrices at each index can be arbitrarily chosen from a set of matrices is that the spectral radius of the matrix $S$ is less than one, which is different from our analytical results in Theorem 1 to be presented later, for the same assumptions.

III. ANALYTICAL RESULTS

In this section, we develop a dynamical system for epidemic spread based on the assumption of spatial independence be-
In addition, we show that the epidemic threshold for dynamic probability of each node in the network as spreading in dynamic Gilbert networks. We propose epidemic threshold to static and periodic networks. Next, employing the joint spectral radius, we quantify epidemic threshold for dynamic networks. Moreover, it is proved that the epidemic threshold in undirected networks only depends on the maximum spectral radius of the set of system matrices. Then, we extend the results of the proposed epidemic threshold to static and periodic networks. In addition, we show that the epidemic threshold for dynamic regular networks is the same as static regular networks. Finally, we calculate an upper bound for the probability of epidemic spreading in dynamic Gilbert networks.

Using the assumption of spatial independence between states of nodes in a given network, we can write the infection probability of each node in the network as

\[ p_i(t+1) = 1 - p_i(t)\delta - (1-p_i(t)) \prod_{j \in N_i(t)} [1 - p_j(t)\beta], \tag{3} \]

where \( N_i(t) \) denotes the set of neighbors of node \( i \) at index \( t \), which is a function of time. The infection probabilities of nodes can be interpreted as state variables of a dynamical system. Eq. \( (3) \) shows infection probabilities at a given index are nonlinear functions of infection probabilities of the previous index. Therefore, the epidemic dynamical system is nonlinear. The corresponding state space of this nonlinear system is the subspace \([0, 1]^n\) in \( R^n \), where \( n \) is the number of nodes in the network. For instance, when \( n = 2 \), the state space is a rectangle whose vertices are points of \((0, 0), (0, 1), (1, 0), \) and \((1, 1)\) in \( R^2 \). Given initial infection probabilities of nodes, we can calculate the evolving trajectory of infection probabilities in state space. The family of evolving trajectories of states in the state space is called a phase portrait. Studying the steady state behavior of dynamical systems requires finding the equilibrium points. If \( P^* \) is an equilibrium point, \( P^*(t+1) = P^*(t) = P^* \). So, we can write

\[ p_i^* = 1 - p_i^*\delta - (1-p_i^*) \prod_{j \in N_i(t)} [1 - p_j^*\beta], \tag{4} \]

where \( p_i^* \) is the infection probability of the \( i \)th node in steady state if there is an asymptotic equilibrium point. Eq. \( (4) \) is the equilibrium equation corresponding to node \( i \). To find the equilibrium points of a given epidemic system with \( n \) nodes, we need to solve a system of \( n \) equations with \( n \) unknowns. In the case of dynamic networks, this system of equations is changing with time and equilibrium points by definition are static points which satisfy this system of equations for all time. An epidemic dynamical system may have more than one equilibrium point. From Eq. \( (4) \), the origin is always an equilibrium point. It means that for all different values of \( \beta \) and \( \delta \) and for any arbitrary topologies of networks, the origin is always an equilibrium point. However, the values of \( \beta \) and \( \delta \) with topologies of networks determine the stability status of the origin. If the origin is an asymptotically stable equilibrium point, the epidemic dies out. On the other hand, the epidemic spreads out when the origin is an unstable equilibrium point.

We can rewrite \( (5) \) in the form of a matrix equation as

\[ P_{t+1} = [(1 - \delta)I + \beta A_t] P_t, \tag{6} \]

where \( P_t = [p_1(t), p_2(t), \ldots, p_n(t)]^T \) is the system state or, in the other words, the vector of infection probabilities of nodes at index \( t \), \( A_t \) is the adjacency matrix at index \( t \), and \( I \) denotes an \( n \times n \) identity matrix. From this point, we call \( M_t = [(1 - \delta)I + \beta A_t] \) the system matrix at index \( t \).

### A. Linearization of System Equations

One way to identify the stability status of an equilibrium point of a nonlinear system is to study the stability of the linearized system at that equilibrium point. In the case of epidemic networks, we are interested in determining the stability status of the origin. It is because if the origin is an asymptotically stable point, the epidemic dies out only if no other equilibrium points exist in the subspace \([0, 1]^n\), otherwise asymptotic stability is only local. Therefore, we linearize the epidemic nonlinear system at the origin. Neglecting nonlinear terms in Eq. \( (5) \), we can write

\[ p_i(t+1) = p_i(t)(1 - \delta) + \sum_{j \in N_i(t)} p_j(t)\beta. \tag{5} \]

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where \( P_t = [p_1(t), p_2(t), \ldots, p_n(t)]^T \) is the system state or, in the other words, the vector of infection probabilities of nodes at index \( t \), \( A_t \) is the adjacency matrix at index \( t \), and \( I \) denotes an \( n \times n \) identity matrix. From this point, we call \( M_t = [(1 - \delta)I + \beta A_t] \) the system matrix at index \( t \).
the three nodes over time. The dots with the $P_0$ label in state space represent the initial infection probabilities and the dot with the $P_{eq}$ represents the equilibrium point of the system. $\delta = .2$ and $\beta = .1$. We see that all trajectories reach the origin regardless of their initial states. It shows that in this case the origin is asymptotically stable and epidemics die out. In the case of spreading epidemics, the origin is not a stable equilibrium point and the state variables converge to a non-zero equilibrium point and remain in that point. This equilibrium point determines the final fraction of infected nodes. Fig. 2 shows the trajectories of state evolution of the same network as Fig. 1. The initial states are the same as the ones in Fig. 1 and the only difference is the value of $\beta$. In this case, $\beta = .6$. We see that the epidemic spreads out and for all different initial infection probability vectors, the system state reaches the equilibrium point $P_{eq} = [0.76791, 0.69731, 0.69731]$ whose elements represent, respectively, the infection probability of nodes 1, 2, and 3 in steady state. Before tackling the problem of stability of the origin, we need to present some definitions.

Definition 1. Given $M$ is a set of matrices, define

$$\hat{\rho}_k(M, ||.||) := \sup \left\{ \left\| \prod_{i=1}^{k} M_i \right\| : M_i \in M \text{ for } 1 \leq i \leq k \right\},$$

where $\hat{\rho}_k(M)$ is the largest possible norm of all products of $k$ matrices chosen in the set $M$. The joint spectral radius $\rho(M)$ is defined as [21]

$$\rho(M) := \lim_{k \to \infty} \hat{\rho}_k(M, ||.||)^\frac{1}{k}. \quad (7)$$

Therefore, the joint spectral radius of set $M$ is the maximum possible norm of products of matrices in the set $M$ when number of products $k$ goes to infinity.

Definition 2. Given $M$ is a set of matrices, define

$$\overline{\rho}_k(M) := \sup \left\{ \rho \left( \prod_{i=1}^{k} M_i \right) : M_i \in M \text{ for } 1 \leq i \leq k \right\},$$

where $\rho$ denoted the spectral radius and $\overline{\rho}_k(M)$ is the largest possible spectral radius of all products of $k$ matrices chosen in the set $M$. The generalized spectral radius $\overline{\rho}(M)$ is defined as [21]

$$\overline{\rho}(M) := \lim_{k \to \infty} \overline{\rho}_k(M)^\frac{1}{k}. \quad (8)$$

In [23], the authors proved that for a bounded set of matrices, the generalized spectral radius is equal to the joint spectral radius.

Lemma 1. Four member inequality [21] For a given arbitrary set of matrices $M$ and any $k \geq 1$

$$\rho_k(M)^\frac{1}{k} \leq \overline{\rho}(M) \leq \rho(M) \leq \rho_k(M)^\frac{1}{k},$$

independent of the induced norm used to define $\rho_k(M)$.

Let us consider a set $L$ of all possible adjacency matrices $A_i$ and at each time instant the adjacency matrix is randomly chosen from this set. $L$ is surely bounded and may be finite or infinite. We define $M$ as the set of system matrices corresponding to the adjacency matrices in $L$. $M_i$ is a member of the set $M$ and defined as $M_i = [(1 - \delta)I + \beta A_i]$. Therefore, $M$ is also bounded. If $L$ is finite, $M$ is finite too and if $M$ is infinite, $L$ is infinite.

Theorem 1. Consider a set $L$ of all possible adjacency matrices of a dynamic network, infection probability of $\beta$, and recovering probability of $\delta$. If the joint spectral radius of set $M$ of system matrices is less than one, the origin is an asymptotically stable equilibrium point and the epidemic dies out.

Proof: Assume $\hat{\rho}(M) = l < 1$. By definition there exists a value $k_0$ for each $\epsilon > 0$ with $\epsilon < 1 - l$ such that

$$1 - \epsilon < \hat{\rho}_k(M)^\frac{1}{k} < 1 + \epsilon, \forall k > k_0.$$ 

If we raise all sides of the above inequality to the power $k$, we can conclude that

$$\lim_{k \to \infty} \hat{\rho}_k(M) = 0.$$ 

Considering the formula of $\hat{\rho}_k(M)$ in Definition 1 for any product of matrices $M_i \in M$, we can write

$$0 \leq \left\| \prod_{i=1}^{k} M_i \right\| \leq \hat{\rho}_k(M).$$

We showed that the right hand side of the above inequality goes to zero when $k \to \infty$. Therefore for any product of $M_i$s, we can write
and respectively we can write symmetric matrices system matrix is also symmetric. We know that for a given probability vector is zero and the epidemic dies out. spectral radius is less than one. In this case, the final infection and any random sequence of adjacency matrices if the joint equilibrium point for any initial infection probability vector is \[ \lim_{k \to \infty} \prod_{i=1}^{k} M_i = 0. \]

If \[ \lim_{k \to \infty} \prod_{i=1}^{k} M_i = 0, \]
for any initial infection probability vector \( P_0 \) we can write

\[ \lim_{k \to \infty} \prod_{i=1}^{k} M_i \prod_{i=1}^{k} [I \cdot (1 - \delta) + \beta A_i] P_0 = 0, \]

which shows that the origin is an asymptotically stable equilibrium point for any initial infection probability vector and any random sequence of adjacency matrices if the joint spectral radius is less than one. In this case, the final infection probability vector is zero and the epidemic dies out.

\[ \text{Theorem 2. Consider a set } L \text{ of all possible adjacency matrices of a dynamic network with undirected graphs, set } M \text{ of the system matrices corresponding to set } L \text{ of the adjacency matrices, infection probability of } \beta, \text{ and recovering probability of } \delta. \text{ If the largest spectral radius of the matrices in set } M \text{ is less than one, the origin is an asymptotically stable equilibrium point and the epidemic dies out.} \]

\[ \text{Proof: If the network graph is undirected, its corresponding adjacency matrix is symmetric so that its corresponding system matrix is also symmetric. We know that for a given symmetric matrices } M_i, \text{ we can calculate the induced 2 norm of } M_i \text{ as follows} \]

\[ ||M_i||_2 = \sqrt{\rho(M_i^T M_i)} = \sqrt{\rho(M_i)^2} = \rho(M_i). \]

If we use the induced 2 norm to calculate \( \hat{\rho}_1(M) \) and \( \overline{\rho}_1(M) \), respectively we can write

\[ \hat{\rho}_1(M) = \sup \{ ||M_i||_2 : M_i \in M \} = \sup \{ \rho(M_i) : M_i \in M \} \]

and

\[ \overline{\rho}_1(M) = \sup \{ \rho(M_i) : M_i \in M \}. \]

Therefore, we can conclude that for a set of symmetric matrices

\[ \hat{\rho}_1(M) = \overline{\rho}_1(M) = \sup \{ \rho(M_i) : M_i \in M \}. \]

Moreover, we mentioned in Lemma 1 that the four member inequality holds for any \( k \geq 1 \). Therefore, we can write

\[ \overline{\rho}_1(M) \leq \overline{\rho}(M) \leq \hat{\rho}_1(M) \leq \hat{\rho}(M). \]

Considering Eq. (9) and Eq. (10), we can write

\[ \overline{\rho}(M) = \hat{\rho}(M) = \sup \{ \rho(M_i) ; M_i \in M \}. \]

Therefore the joint spectral radius of set \( M \) of symmetric matrices is equal to the largest spectral radius of matrices in the set. Based on Theorem 1 we can conclude that if the largest spectral radius of the system matrices of an undirected dynamic network is less than one, the origin is an asymptotically stable equilibrium point and the epidemic dies out.

\[ \text{Corollary 1. Consider a static epidemic network with adjacency matrix } A, \text{ infection probability of } \beta, \text{ and recovering probability of } \delta. \text{ The epidemic dies out if } s < \frac{\beta}{\delta}. \]

\[ \text{Proof: For a static network, } M, \text{ the set of system matrices, has only one element which is } (1 - \delta) I + \beta A. \text{ In this case, } \overline{\rho}_k(M), \text{ the largest possible spectral radius of all products of } k \text{ matrices chosen in the set } M, \text{ can be written as} \]

\[ \overline{\rho}_k(M) = \sup \{ \rho \left( \prod_{i=1}^{k} M_i \right) ; M_i \in M \} = \rho((1 - \delta) I + \beta A)^k. \]

\( M \) is a bounded set and it is proved in [23] that for a bounded set of matrices the joint spectral radius is equal to the generalized spectral radius. Hence, we can calculate the joint spectral radius as

\[ \hat{\rho}(M) = \lim_{k \to \infty} \overline{\rho}_k(M)^{\frac{1}{k}} = \rho((1 - \delta) I + \beta A). \]

Based on Theorem 1 the epidemic dies out if the joint spectral radius of the set of system matrices is less than one. For a static network, the joint spectral radius is equal to \( 1 - \delta + \beta \rho(A) \). Therefore, the epidemic dies out if

\[ \frac{\beta}{\delta} < \frac{1}{\rho(A)}. \]

The epidemic threshold for static networks in Eq. (11) is the same as analytical results for static networks’ epidemic threshold in [2].

\[ \text{Corollary 2. Consider a dynamic network with a fixed repetition pattern of } T \text{ adjacency matrices in a set } L = \{ A_1, A_2, \ldots, A_T \}, \text{ infection probability of } \beta, \text{ and recovering probability of } \delta. \text{ The epidemic dies out if} \]

\[ \rho(\prod_{i=1}^{T} [(1 - \delta) I + \beta A_i]) < 1. \]

\[ \text{Proof: Consider a dynamic network with a fixed repetition pattern of } T \text{ adjacency matrices and } k = mT \text{ where } m \text{ is a positive integer. For the case where } k = mT, \text{ } \hat{\rho}_k(M) \text{ can be written} \]

\[ \hat{\rho}_k(M) = \hat{\rho}_1(M)^k. \]
The epidemic in a dynamic regular network with undirected graphs and node degree of $k$ dies out if the largest spectral radius of the system matrices is less than one. Hence, the epidemic dies out if 

$$\rho(M_k) = 1 - \delta + \beta \rho(A_k) = 1 - \delta + \beta k.$$ 

Therefore, all system matrices have the same spectral radius of $1 - \delta + \beta k$ and the largest spectral radius of the system matrices is also equal to $1 - \delta + \beta k$. Based on Theorem 2, we can conclude that epidemics in dynamic networks with regular undirected graphs die out if the largest spectral radius of the system matrices is less than one. Hence, the epidemic dies out if $1 - \delta + \beta k < 1$ or equivalently if

$$\frac{\beta}{\delta} < \frac{1}{k}.$$ 

It is remarkable that Eq. (14) is the same as the epidemic threshold for static regular networks [2].

Up to this point, we studied the dynamic networks whose adjacency matrices are deterministic and given. However, for the cases that adjacency matrices are stochastic and not deterministic, the joint spectral radius of the set of the system matrices is a random variable. Therefore, the condition for the dying out of the epidemics turns out to be in terms of statistical characteristics of the joint spectral radius. Dynamic Gilbert networks are examples of such dynamic networks with stochastic adjacency matrices. In a Gilbert network with the parameter $P$, every link exists with probability $P$ [26]. In other words, the existence of a link is a Bernoulli random variable with the parameter $P$. For a dynamic Gilbert network, at every time instant, these binary random variables are redrawn according to the Bernoulli distribution. In the next corollary, we derive the upper bound for the probability of spreading out of epidemics for Gilbert dynamic networks in terms of expected value of the joint spectral radius.

**Corollary 4.** For a given dynamic Gilbert network with $N$ nodes and the probability $P$ of existence of links, the probability that epidemic spreads out is upper bounded by $1 - \delta + (N - 1)\beta P$.

**Proof:** We define $\tilde{M}$ as 

$$\tilde{M} = \prod_{i=1}^{k} M_i,$$ 

where $M_i$ denotes the system matrix corresponding to one realization of a Gilbert dynamic network’s adjacency matrix. $\tilde{m}_{q,n}$ denotes the element in $q^{th}$ row and $n^{th}$ column of matrix $\tilde{M}$. In the Appendix, we show that for all columns of $\tilde{M}$

$$E \left\{ \sum_{q=1}^{N} |\tilde{m}_{q,n}| \right\} = [1 - \delta + (N - 1)\beta P]^{k} \forall n = 1, 2, ..., N,$$

where $E$ denotes the expected value. Considering 

$$\|\tilde{M}\|_1 = \max_{n} \sum_{q=1}^{N} |\tilde{m}_{q,n}|$$

and (15), we can calculate $E \left\{ \|\tilde{M}\|_1 \right\}$ as

$$E \left\{ \|\tilde{M}\|_1 \right\} = [1 - \delta + (N - 1)\beta P]^{k}.$$ 

Because the above equality holds for any product of $M_i$s, $E \left\{ \hat{\rho}_k(M) \right\}$ can be written as

$$E \left\{ \hat{\rho}_k(M) \right\} = [1 - \delta + (N - 1)\beta P]^{k}.$$

And consequently, the expected value of the joint spectral radius can be calculated as

$$E \left\{ \hat{\rho}(M) \right\} = E \left\{ \lim_{k \to \infty} \hat{\rho}_k(M)^{k} \right\} = [1 - \delta + (N - 1)\beta P].$$

We employ the Markov inequality and the expected value of the joint spectral radius to compute the upper bound for the probability of the joint spectral radius to be more than one. Using the Markov inequality, we can write

$$Prob(\hat{\rho}(M) \geq 1) \leq E\{\hat{\rho}(M)\}. $$

(16)
Substituting the expected value of the joint spectral radius in Eq. \( (16) \), we can conclude

\[
\text{Prob}(\hat{\rho}(M) \geq 1) \leq 1 - \delta + (N - 1)\beta P. \tag{17}
\]

Based on Theorem \( \text{II} \), the epidemic dies out if the joint spectral radius is less than one. Therefore, the probability of the epidemic spreading out is equal to the probability of the joint spectral radius to be greater than one. Considering Eq. \( (17) \), we can conclude that the probability of epidemic to spread out is upper bounded by \( 1 - \delta + (N - 1)\beta P \).

We should notice that when \( 1 - \delta + (N - 1)\beta P \) is greater than one, it is not informative. Therefore, we consider the \( \min\{1, 1 - \delta + (N - 1)\beta P\} \) as the upper bound for the probability of spreading out.

IV. SIMULATION RESULTS

In this section, we validate our theoretical results via simulation an epidemic on synthetic and real dynamic networks. First, we simulate an epidemic on a dynamic Watts-Strogatz network and compare the derived epidemic threshold with the threshold proposed in [9]. Then, the simulation result of final fraction of infected nodes versus the joint spectral radius for a dynamic Barabasi-Albert network is presented. Moreover, we evaluate our analytical results in the case of real networks by simulating an epidemic on the set of extracted graphs from the MIT Reality Mining data set. Next, an epidemic on a dynamic regular network is simulated. Finally, we validate the derived upper bound for the probability of the epidemic spreading in a dynamic Gilbert network using simulation results.

In Fig. 3 we see the simulation results of epidemics on Watts-Strogatz and Barabasi-Albert dynamic networks. (A) Final fraction of infected nodes for dynamic Watts-Strogatz networks with 1000 nodes and rewiring probability of .5. (B) Comparison between the joint spectral radius and the spectral radius of the product of the system matrices for dynamic Watts-Strogatz networks. (C) Fraction of infected nodes over time for dynamic Barabasi-Albert network with 1000 nodes. (D) Final fraction of infected nodes for dynamic Barabasi-Albert network with 1000 nodes Vs. the joint spectral radius.
The joint spectral radius of the set of system matrices is equal to the spectral radius of the system matrix corresponding to the adjacency matrix with the largest spectral radius. We increase $\beta$ from 0.00052 to 0.86652 to generate different cases of epidemic strength. The number of iteration for each case is 20. We see that epidemics die out for all the cases that the value of the joint spectral radius is less than one. As soon as the value of the joint spectral radius increases beyond one, epidemics spread out, which confirms the analytical results of Theorems 1 and 2. The curve of the final fraction of infected nodes versus the spectral radius of the system matrices product shows that $\rho(\prod_{i=1}^{T} [(1 - \delta)I + \beta A_i])$ is not the accurate epidemic threshold. We see that epidemics spread out for some values of it which are less than one. This result is in contradiction with the analytical results of Theorem 1 in [9] which says that if the spectral radius of the product is less than one, the epidemic dies out.

Fig. 4 (A) Fraction number of infected nodes for MIT Reality Mining dynamic over time. (B) Final fraction of infected nodes for MIT Reality Mining dynamic Vs. the joint spectral radius. (C) Final fraction of infected nodes for a dynamic regular network with 1000 nodes and node degree of 8. (D) Final fraction of infected nodes for a dynamic Gilbert network with 1000 nodes and node degree of 8.

Strogatz, the adjacency matrix of the network at each index is chosen randomly from a set of four Watts-Strogatz graphs with average node degrees of 4, 8, 12, and 16 and, respectively, spectral radii of 4.46242, 8.41081, 12.40911, and 16.38739. The joint spectral radius of the set of system matrices is equal to the spectral radius of the system matrix corresponding to the adjacency matrix with the largest spectral radius. We increase $\beta$ from 0.00052 to 0.86652 to generate different cases of epidemic strength. The number of iteration for each case is 20. We see that epidemics die out for all the cases that the value of the joint spectral radius is less than one. As soon as the value of the joint spectral radius increases beyond one, epidemics spread out, which confirms the analytical results of Theorems 1 and 2. The curve of the final fraction of infected nodes versus the spectral radius of the system matrices product shows that $\rho(\prod_{i=1}^{T} [(1 - \delta)I + \beta A_i])$ is not the accurate epidemic threshold. We see that epidemics spread out for some values of it which are less than one. This simulation result contradicts the analytical results of Theorem 1 in [9] which says that if the spectral radius of the product is less than one, the epidemic dies out.

Fig. 3 (B) compares the spectral radius of the product of system matrices of the dynamic Watts-Strogatz network with the joint spectral radius of its set of system matrices. The curve of $Y = X$ helps us to determine for which values of the joint spectral radius, the spectral radius of the product is greater than the joint spectral radius or vice versa. We see that there are some points that the joint spectral radius is greater than one and the spectral radius of the product is less than one. This results in wrong predictions of dying out epidemics for those points if we choose the spectral radius of the product as the epidemic threshold.

Fig. 3 (C) shows the fraction of infected nodes over time for a dynamic Barabasi-Albert network. To realize a dynamic Barabasi-Albert network, we select four Barabasi-Albert graphs with average node degrees of 4, 8, 12, and 16 with, respectively, spectral radii of 12.66217, 17.36462, 22.36887, and 27.91071 as the set of adjacency matrices. During simulation, the adjacency matrix of this dynamic network at each index is randomly chosen from this set of
matrices with equal probability. The number of iteration for all cases is 20 and the initial fraction of infected nodes is 0.2. We see that increasing the value of β leads to an increase in the joint spectral radius and, eventually, the final fraction of infected nodes. Also, epidemics die out for the cases that the joint spectral radius is less than one. Fig. 3 (D) depicts the final fraction of infected nodes for the mentioned dynamic Barabasi-Albert network while β increases from 0.00038 to 0.63481. We see that epidemics spread out for the cases where the joint spectral radius is greater than one.

Fig. 4 (A) shows the fraction of infected nodes over time for a dynamic network whose time-varying adjacency matrices are extracted from the MIT Reality Mining data set [25]. This data set contains the adjacency connectivity matrix of 94 persons using mobile phones pre-installed with different special software including the logger of Bluetooth devices which was triggered when two mobile phones’ distance was approximately five meters or less. Bluetooth scans were carried out every 5 minutes. This data set contains the collected information from mobile phones from September 2004 to June 2005. We extract eight adjacency matrices for 8 consecutive hours from 8 o’clock in the morning to 4 o’clock in the afternoon of September 1, 2004. These eight adjacency matrices have, respectively, the spectral radii of 6.30117, 5.41546, 9.44439, 9.09696, 8.36535, 9.53451, 9.05251, and 7.41181. At each time index, the adjacency matrix was randomly chosen from the extracted matrices. For all four cases, δ = 0.2 and the initial fraction of infected nodes was 0.2. The number of iterations for each case is 50. We see that increasing β makes the joint spectral radius larger and the epidemic spreads for the values of the joint spectral radius larger than one.

Fig. 3 (B) depicts the final fraction of infected nodes for the dynamic network in Fig. 4 (A). We fix the value of δ = 0.2 while increasing the value of β from 0.00089 to 0.83142. The number of iteration for each case is equal to 50. We see that the epidemic spreads out when the joint spectral radius is greater than one. For the cases that the epidemic spreads out, the final fraction of infected nodes increases with the increase in the value of joint spectral radius.

Fig. 4 (C) shows the final fraction of infected nodes for a dynamic regular network with 1000 nodes and node degree of k = 8 versus the product of δ and node degree. We simulate epidemics for different values of β in the interval of [0.00106, 0.99089] while fixing the value of δ = 0.2. The number of iteration for each case is 100. We see that epidemics die out when β ≤ 1/4, which confirms the result of corollary 3.

Fig. 4 (D) depicts the final fraction of infected nodes for a dynamic Gilbert network with 1000 nodes and a probability of connection of P = 0.004 versus 1 − δ + (N − 1)βP, which is the upper bound for the probability of an epidemic spreading out derived in corollary 4. Table I shows the chosen values of δ and β in simulation as well as the values of 1 − δ + (N − 1)βP. We see that the epidemic dies out up to the point where the upper bound is less than one. When this upper bound reaches one, the epidemic starts to spread out, which confirms the result of corollary 4. We should note that although having an upper bound greater than one for a probability is not informative, it can be used as a measure of the epidemic strength. This is evident in Fig. 4 (D), as increasing value of upper bound leads to an increase in the final fraction of infected nodes.

V. CONCLUSION

In this paper, SIS epidemics spread in dynamic networks is studied. We propose an analytical way to derive the analytical epidemic threshold, which can be applied to any dynamic network whose adjacency matrix is randomly chosen from a set of matrices at any index. The linearized version of the nonlinear epidemic system is employed to derive the epidemic threshold. We show that an epidemic dies out if the origin is an asymptotically stable equilibrium point. We derive the epidemic threshold for dynamic networks using the joint spectral radius of the system matrices. We calculate the simplified version of epidemic threshold for undirected dynamic networks using the fact that the joint spectral radius of a set of symmetric matrices is equal to the largest spectral radius of matrices in that set. Then, the epidemic threshold for dynamic regular networks is derived. For dynamic Gilbert networks, we compute the upper bound of the probability of an epidemic spreading out in terms of the expected value of the joint spectral radius.

Our analytical results show that the epidemic thresholds of dynamic networks are determined by their dynamic topologies as well as the epidemic strengths on the networks. In particular, the joint spectral radius of the set of system matrices determines whether the epidemic dies out or not. In other words, the joint spectral radius characterizes the level of connectivity of the nodes in dynamic networks over time as well as epidemic strengths. In the case of undirected networks, the joint spectral radius

| δ     | β    | 1 − δ + (N − 1)βP |
|-------|------|--------------------|
| 0.01  | 0.01 | 0.09               |
| 0.05  | 0.01 | 0.19               |
| 0.10  | 0.01 | 0.24               |
| 0.15  | 0.01 | 0.34               |
| 0.20  | 0.01 | 0.44               |
| 0.25  | 0.01 | 0.54               |
| 0.30  | 0.01 | 0.64               |
| 0.35  | 0.01 | 0.74               |

TABLE I

δ AND β VALUES USED IN SIMULATION OF THE EPIDEMIC IN THE DYNAMIC GILBERT NETWORK
radius is only dependent on the adjacency matrix with the largest spectral radius. This implies that for the undirected networks the dynamics of epidemics mainly determined by the adjacency matrix with the largest spectral radius. The variance of topologies of dynamic networks impacts the spread of epidemics. In order to validate that our analytical results for dynamic networks with different dynamic topologies, we simulated epidemics on Watts-Strogatz, Barabasi-Albert, Regular, MIT Reality Mining, and Gilbert dynamic networks. The dynamic Watts-Strogatz network was used to model a dynamic network with small-world properties, including high clustering coefficient and small shortest path. On the other hand, the dynamic Gilbert network was employed to validate the derived epidemic threshold for the dynamic networks with random structure and small clustering coefficients. Many networks in the real world including e-mail networks, world wide web, and biological networks are considered to be in the category of scale-free networks which can be modeled by Barabasi-Albert networks [28], [29], [30], [31]. Our simulation result for the dynamic Barabasi-Albert validated the accuracy of the derived epidemic threshold for such scale-free networks. Also, we verified our theoretical result for a real-life network. We simulated the epidemic on the dynamic network with adjacency matrices extracted from MIT Reality Mining data set, which showed that the derived epidemic threshold holds for this real-life network as well.

The considered model of dynamic switching networks in this paper does not take into account any temporal correlation between consecutive adjacency matrices. This assumption is not realistic for the dynamic networks with strong correlation between consecutive adjacency matrices. For example, for the simulation of epidemics on MIT Reality Mining data set, the adjacency matrix at each times step was chosen at random and independently from the other time steps. However, in reality, the sequence of the adjacency matrices follows the temporal order, which implies the existence of a correlation among the adjacency matrices. Considering completely random and independent processes of choosing adjacency matrices in our framework accounts for the worst possible sequence of matrices in terms of epidemics. That is because the joint spectral radius of the set of system matrices is determined by the sequence of adjacency matrices which is the most vulnerable against epidemics. In conclusion, for dynamic networks with temporal correlation, if our framework predicts an epidemic dies out, the epidemic will die out even though this model neglects temporal correlations. On the other hand, its prediction can be too conservative. That is it predicts an epidemic spreads out but it dies out.

**APPENDIX**

**Theorem 3.** Consider \( \hat{M} = \prod_{i=1}^{k} M_i \) where \( M_i \) denotes the system matrix corresponding to one realization of a Gilbert dynamic network’s adjacency matrix. For the matrix \( \hat{M} \), the expected value of summation of each column’s elements is

\[
E \left( \sum_{q=1}^{N} |\hat{m}_{q,n}| \right) = \left( 1 - \delta + (N - 1)\beta P \right)^k \forall n = 1, 2, ..., N,
\]

where \( P \) is the probability of link existence and \( \hat{m}_{q,n} \) denotes the element in \( q^{th} \) row and \( n^{th} \) column of matrix \( \hat{M} \).

**Proof:** We prove this via induction. In the case of a Gilbert dynamic network, off-diagonal elements of adjacency matrix \( A \) are independent and identically distributed (iid) Bernoulli random variables with parameter \( P \). The first step is to show that Eq. (18) is correct when \( k = 1 \). Assume \( k = 1 \). In this case, \( \hat{M} = (1 - \delta)I + \beta A \) and \( E \left( \sum_{q=1}^{N} |\hat{m}_{q,n}| \right) \) can be written as

\[
E \left( \sum_{q=1}^{N} |\hat{m}_{q,n}| \right) = (1 - \delta) + \beta \sum_{i=1}^{N-1} E \{X_i\}, \quad (19)
\]

where the \( X_i \)‘s are iid random variables with parameter \( P \). \( E \{X_i\} = P \). Therefore, we can rewrite Eq. (19) as

\[
E \left( \sum_{q=1}^{N} |\hat{m}_{q,n}| \right) = (1 - \delta) + (N - 1)\beta P. \quad (20)
\]

Eq. (20) shows that Eq. (18) is correct for \( k = 1 \). The second step is to assume Eq. (18) is correct for \( k \) and prove it for \( k + 1 \). Assume \( \hat{M} = \prod_{i=1}^{k} M_i \). Considering the assumption of correctness of Eq. (18) for \( k \), \( E \left( \sum_{q=1}^{N} |\hat{m}_{q,n}| \right) = \left( 1 - \delta + (N - 1)\beta P \right)^k \). Suppose \( R = M_{k+1}\hat{M} \). \( r_{q,n} \), the element in the \( q^{th} \) row and \( n^{th} \) column of \( R \) can be written in terms of the elements of \( \hat{M} \) as

\[
r_{q,n} = (1 - \delta)\hat{m}_{q,n} + \beta \sum_{j=1,j\neq q}^{N} X_j\hat{m}_{j,n}, \quad (21)
\]

where the \( X_j \)‘s are iid Bernoulli random variables with parameter \( P \). Therefore, we can write \( \sum_{q=1}^{N} |r_{q,n}| \) as

\[
\sum_{q=1}^{N} \left| r_{q,n} \right| = (1 - \delta) \sum_{q=1}^{N} |\hat{m}_{q,n}| + \beta \sum_{q=1}^{N} \left| \sum_{j=1,j\neq q}^{N} X_j\hat{m}_{j,n} \right|, \quad (22)
\]

where \( X_j \)‘s and \( \hat{m}_{j,n} \) are independent. Hence, \( E \left( \sum_{q=1}^{N} |r_{q,n}| \right) \) can be written as

\[
E \left( \sum_{q=1}^{N} |r_{q,n}| \right) = (1 - \delta)E \left( \sum_{q=1}^{N} |\hat{m}_{q,n}| \right) + P\beta E \left( \sum_{q=1}^{N} \left| \sum_{j=1,j\neq q}^{N} \hat{m}_{j,n} \right| \right). \quad (23)
\]

On the other hand, \( E \left( \sum_{q=1}^{N} \left| \sum_{j=1,j\neq q}^{N} \hat{m}_{j,n} \right| \right) = (N - 1)E \left( \sum_{q=1}^{N} |\hat{m}_{q,n}| \right) \). Considering \( E \left( \sum_{q=1}^{N} |\hat{m}_{q,n}| \right) = [(1 - \delta) + \beta P(N - 1)]^k \), we can rewrite (23) as
\[
E \left\{ \sum_{q=1}^{N} [r_{q,n}] \right\} = \left[ (1 - \delta) + \beta P(N - 1) \right] E \left\{ \sum_{q=1}^{N} \hat{m}_{q,n} \right\} = \left[ (1 - \delta) + \beta P(N - 1) \right]^{k+1}.
\]

The result in Eq. (24) for \( k + 1 \) is the last step in the proof of this through induction.

### REFERENCES

[1] T. Kostova, “Interplay of node connectivity and epidemic rates in the dynamics of epidemic networks”, Journal of Difference Equations and its Applications, vol. 15, pp. 415-428, 2009.

[2] D. Chakrabarti, Y. Wang, C. Wang, J. Leskovec, and C. Faloutsos, “Epidemic thresholds in real networks”. ACM Trans. Inf. Syst. Secur. v. 10, pp. 1-26, 2008.

[3] Yang Wang, D. Chakrabarti, Chenxi Wang, and C. Faloutsos, “Epidemic spreading in real networks: an eigenvalue viewpoint,” In proceedings of the 22nd International Symposium on Reliable Distributed Systems, pp. 25-34, 2003.

[4] Z. Chen and C. Ji, “Spatial-temporal modeling of malware propagation in networks”, IEEE Transactions on Neural Networks, vol. 16, no. 5, pp. 1291-1303, 2005.

[5] P. V. Mieghem, J. Omic, and R. Kooij, “Virus Spread in Networks”, IEEE/ACM Transactions on Networking, vol. 17, no. 1, pp. 1-14, Feb. 2009.

[6] A. Ganesh, L. Massoulie, and D. Towsley, “The effect of network topology on the spread of epidemics,” In Proceedings IEEE INFOCOM, v. 2, pp. 1455-1466, 2005.

[7] J. Lindquist, J. Ma, P. V. D. Driessche, F. H. Willeboordse, “Effective degree network disease models”, Journal of Mathematical Biology, v. 62, no. 2, pp. 143-164, 2009.

[8] B. A. Prakash, H. Tong, N. Valler, M. Faloutsos, and C. Faloutsos, “Virus propagation on time-varying networks: theory and immunization algorithms,” In Proceedings of mMachine learning and knowledge discovery in databases, pp. 98-114, 2010.

[9] N. C. Valler, B. A. Prakash, H. Tong, M. Faloutsos, and C. Faloutsos, “Epidemic Spread in Mobile Ad Hoc Networks: Determining the Tipping Point,” In Proceedings of the 10th international IFIP TC 6 conference on Networking, Volume Part I, pp. 266-280, 2011.

[10] E. Volz and L. A. Meyers, “Epidemic thresholds in dynamic contact networks”, Journal of the Royal Society Interface, v. 6, pp. 233-241, 2009.

[11] M. Taylor, T. J. Taylor, and I. Z. Kiss, “Epidemic threshold and control in a dynamic network”, Phys. Rev. E., v. 85, pp. 016103, 2012.

[12] T. Gross, C. J. D. Lima Dommar, and B. Blasius, “Epidemic dynamics in an adaptive network”, Phys. Rev. Lett., v. 96, pp. 208701, 2006.

[13] L. E. C. Rocha and V. D. Blondel, “Epidemic spreading in complex networks with degree correlations,” PLoS Comput. Biol., v. 9, pp. e1002974, 2013.

[14] S. Bansal, J. Read, B. Pourbohloul, and L. A. Meyers, “The dynamic nature of contact networks in infectious disease epidemiology,” Journal of biological dynamics, 4 (5), pp. 478-489, 2010.

[15] T. Smieszek, L. Fiebig, and R. W. Scholz, “Models of epidemics: when contact repetition and clustering should be included,” Theoretical Biology and Medical Modeling, vol. 6, 2009.

[16] C. Kamp, “Untangling the Interplay between Epidemic Spread and Transmission Network Dynamics,” PLoS Comput. Biol., vol. 6, pp. e1000984, 2010.

[17] L. E. C. Rocha, F. Liljeros, and P. Holme, “Simulated Epidemics in an Empirical Spatiotemporal Network of 50,185 Sexual Contacts,” PLoS Comput. Biol., vol. 7, pp. e1001109, 2011.

[18] E. Volz, and L. A. Meyers, “Susceptible–infected–recovered epidemics in dynamic contact networks,” Proceedings of the Royal Society of London B: Biological Sciences, vol. 274, pp. 2925-2934, 2007.

[19] M. I. Chen, A. C. Ghani, and J. Edmunds, “Mind the Gap: The Role of Time Between Sex With Two Consecutive Partners on the Transmission Dynamics of Gonorrhea,” Sexually Transmitted Diseases, vol. 35, pp. 435-444, 2008.