

A dynamical model for SIS epidemic propagation on adaptive networks

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Summary. The pairwise ODE model for SIS epidemic propagation on adaptive networks with link number preserving rewiring is studied. The model is a system of four ordinary differential equations with three parameters, the infection rate τ , the recovery rate γ and the rewiring rate w . The model exhibits transcritical, saddle-node and Andronov-Hopf bifurcations. Using the parametric representation method, these bifurcation curves are determined analytically in the (τ, w) parameter plane that is divided into four regions. Numerical evidence shows that homoclinic and cycle-fold bifurcations occur.

Model formulation

Spreading processes on adaptive networks have been widely studied, since it is natural to take into account that the network itself is changing during the process [2]. Among many other processes, SIS (susceptible-infected-susceptible) epidemic propagation on adaptive networks has been well-studied [1, 5, 6], motivated by the fact that susceptible nodes try to cut their links to infected ones and they create new links to susceptible nodes, in order not to be separated from the network. Consider an undirected simple graph with N nodes, where each node can be either susceptible or infected. A susceptible node can become infected when contacted with an infected one, and an infected one can recover and become susceptible again. The mathematical model is a system of ordinary differential equations, which describes the pairwise approximation of SIS epidemic spread on an adaptive network with link preserving rewiring. In this model, susceptible nodes break their links to infected nodes and reconnect to a randomly chosen susceptible node from the population. The model is written in the following form in [1]:

$$\dot{[I]} = \tau[SI] - \gamma[I], \quad (1)$$

$$\dot{[SI]} = -(\tau + \gamma)[SI] + \tau \left(\frac{[SS][SI]}{[S]} - \frac{[SI][SI]}{[S]} \right) - w[SI] + \gamma[II], \quad (2)$$

$$\dot{[SS]} = 2\gamma[SI] - 2\tau \frac{[SS][SI]}{[S]} + 2w[SI], \quad (3)$$

$$\dot{[II]} = -2\gamma[II] + 2\tau \left(\frac{[SI][SI]}{[S]} + [SI] \right), \quad (4)$$

where the time-dependent variables are: $[I](t)$ the expected number of the infected nodes at time t and $[SI](t)$, $[II](t)$, $[SS](t)$ the expected number of SI , II and SS edges. The model contains three parameters, the per contact transmission rate τ , the recovery rate γ , and the rewiring rate w . It can be easily seen that the differential equations for the edges are not independent, because the total number of edges is constant in time. Thus the average degree of the network n is given by the initial condition for the number of edges, namely $n = ([SS](0) + 2[SI](0) + [II](0))/N$, where N denotes the number of nodes. The edge conservation relation

$$nN = [SS](t) + 2[SI](t) + [II](t) \quad \text{for all } t$$

enables us to reduce system (1)-(4) to three equations by eliminating one of the variables corresponding to the number of edges. The aim of our investigation is to describe the local and global qualitative behaviour of the dynamical system (1)-(4) in the domain given by $0 \leq [I] \leq N$, $0 \leq [SI], [SS], [II] \leq Nn$.

Analytic and simulation results

Number of steady states

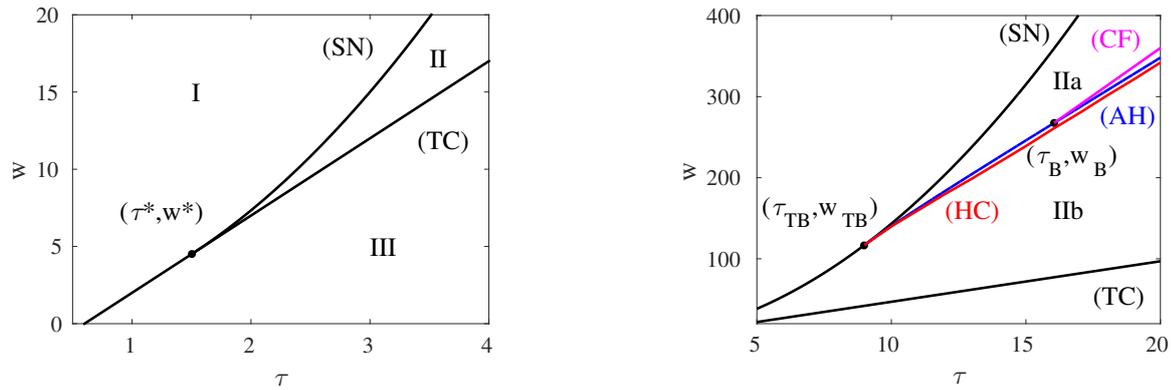
It is easy to show that there is a trivial (disease-free) equilibrium. Besides the trivial solution there may occur endemic equilibria. According to the number of steady states we can divide the (τ, w) parameter plane, see Figure 1a. In domain I, which is above the parabola

$$(2\gamma + \tau - \tau n)^2 = 4\gamma(\gamma + w - \tau n) \quad (\text{SN})$$

and $\tau > \tau^* := 2\gamma/(n-1)$ or above the line

$$\gamma + w = \tau n \quad (\text{TC})$$

and $\tau \leq \tau^*$ there is no endemic steady state and there is the trivial equilibrium. In domain II, between (SN) and (TC) the number of endemic steady states is 2 and there is the trivial steady state as well. In domain III, below (TC) there is only one endemic steady state besides the trivial steady state. Based on the above investigation saddle-node bifurcation occurs along (SN), since the number of steady states changes by 2 as the parabola is crossed. The common point of the curves (TC) and (SN) is at $(\tau^*, w^*) = (2\gamma/(n-1), \gamma(n+1)/(n-1))$.



(a) The saddle-node (SN) and transcritical (TC) bifurcation curves divide the (τ, w) parameter plane into three domains according to the number of endemic steady states. The parameter values are $\gamma = 1$, $N = 100$, $n = 5$.

(b) The bifurcation diagram with the transcritical (TC), saddle-node (SN), Andronov-Hopf (AH), cycle-fold (CF) and homoclinic (HC) bifurcation curves. The parameter values are $\gamma = 1$, $N = 100$, $n = 5$.

Figure 1

Stability analysis

It can be seen that if (τ, w) is in domain I or II, then the trivial steady state is asymptotically stable, while for (τ, w) parameter pairs in domain III, it is unstable. Thus the disease-free steady state loses its stability along the line (TC) and the number of endemic steady states changes by 1 when this line is crossed. Using a general transcritical bifurcation theorem proved in [3] we have verified that in system (1)-(4) transcritical bifurcation occurs along the line (TC). The bifurcation is of forward type when the line (TC) is crossed from domain I to domain III and it is of backward type when the line (TC) is crossed from domain II to domain III.

As the stability of the disease-free steady state is fully characterized, we turn to the stability of the endemic steady states. We have proved that for (τ, w) parameter pairs in domain III the unique endemic steady state is asymptotically stable. As the direct investigation of the eigenvalues is not feasible, we used a different way to prove the stability. We turn our attention to domain II and show that Andronov-Hopf bifurcation may occur there. In order to determine the Andronov-Hopf bifurcation curve (AH), we have applied the parametric representation method. Furthermore, we have determined the Takens-Bogdanov bifurcation point (τ_{TB}, w_{TB}) , where the Andronov-Hopf bifurcation curve (AH) branches out of the saddle-node curve (SN). Extensive numerical study, for several values of γ and n showed that the Andronov-Hopf bifurcation curve divides the parameter domain II into two parts according to the stability of the endemic steady states. Further numerical studies yield that there is a Bautin bifurcation point (τ_B, w_B) on the Andronov-Hopf bifurcation curve, which separates the subcritical and supercritical branches of the bifurcation curve.

A cycle-fold bifurcation curve (CF) emerges from the Bautin bifurcation point due to the Bautin bifurcation theorem (see Section 8.3 in [4]). This curve divides domain IIa to two parts: the oscillatory and non-oscillatory regimes. A stable and an unstable limit cycle collide and disappear as we increase the value of w . This implies the appearance of an unstable periodic orbit in domain IIb via homoclinic bifurcation (HC). This can be shown using numerical simulations. According to the Takens-Bogdanov bifurcation theorem (see Section 8.4 in [4]) a homoclinic bifurcation curve starts from the Takens-Bogdanov bifurcation point in domain IIb.

Discussion

The full characterization of the system (1)-(4) describing SIS epidemic propagation on adaptive networks has been presented. The model exhibits transcritical, saddle-node and Andronov-Hopf bifurcations. Homoclinic bifurcation giving rise to an unstable periodic orbit and cycle-fold bifurcation also occur. The full bifurcation diagram of the (τ, w) parameter plane is shown in Figure 1b.

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