From epidemics to information propagation: Striking differences in structurally similar adaptive network models

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The continuous-time adaptive susceptible-infected-susceptible (ASIS) epidemic model and the adaptive information diffusion (AID) model are two adaptive spreading processes on networks, in which a link in the network changes depending on the infectious state of its end nodes, but in opposite ways: (i) In the ASIS model a link is removed between two nodes if exactly one of the nodes is infected to suppress the epidemic, while a link is created in the AID model to speed up the information diffusion; (ii) a link is created between two susceptible nodes in the ASIS model to strengthen the healthy part of the network, while a link is broken in the AID model due to the lack of interest in informationless nodes. The ASIS and AID models may be considered as first-order models for cascades in real-world networks. While the ASIS model has been exploited in the literature, we show that the AID model is realistic by obtaining a good fit with Facebook data. Contrary to the common belief and intuition for such similar models, we show that the ASIS and AID models exhibit different but not opposite properties. Most remarkably, a unique metastable state always exists in the ASIS model, while there an hourglass-shaped region of instability in the AID model. Moreover, the epidemic threshold is a linear function in the effective link-breaking rate in the AID model, while it is almost constant but noisy in the AID model.

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Over the past decade, many real-world networks have been characterized via graph metrics [1-3] such as clustering, assortativity, modularity, degree distribution, and spectral properties. Recently, robustness characteristics and complex dependences have been analyzed in networks of networks [4], while a parallel track in network science has focused on relatively simple dynamic processes on networks such as epidemics [5,6], synchronization [7], and opinion diffusion [8–10]. In most studies so far, the networks are considered fixed or independent of the dynamic process. After the seminal work of Gross *et al.* [11], the coupling between epidemic processes and the underlying network topology has been extensively studied [12–15].

The coupling between process and topology is natural in many cases. In epidemics [16], for example, after the observation of an infectious relative, one may either avoid him or her (by changing the social contact network) or increase the protection against the virus (without altering the topology). In human brain networks, Hebbian learning alters the connectivity between brain regions that are trained or neurally excited. Although self-adaptation naturally occurs in biology, adaptive networks, in which the process interacts with the topology, are unfortunately difficult to analyze and we barely understand the interplay between process and topology. Twitter measurements [17,18] show that the topology of the network adaptively changes connectivity towards users with high popularity and the ordinary users tend to directly follow the popular ones to get the information faster, instead of awaiting the retweets from their current friends.

Gross *et al.* [11] have spotted complex patterns during the evolution of the adaptive network through the healthy, the oscillatory, the bistable, and the endemic state. Extensions of

The epidemic (information) dynamic in the two models is based on the standard susceptible-infected-susceptible (SIS) epidemic process [26]. We describe the ASIS process, while the terminology for the AID model is given in angular brackets. The epidemic (information possession) state of node i in a network G with N nodes is specified by a Bernoulli random variable $X_i(t) \in \{0,1\}$: $X_i(t) = 1$ if node *i* is infected (possesses the information) and $X_i(t) = 0$ otherwise. At time t, a node i is infected (possesses the information) with probability $\Pr[X_i(t) = 1]$. The epidemic (information) spreading process from an infected (information possessing) node to its healthy (informationless) neighbors is a Poisson process with rate β . Only when node *i* is infected (has the information) it can infect (share the information) with the direct neighbors with rate β . In an online social network, a user can obtain the information via different sources (e.g., the social reinforcement effect [27,28]) that are not necessarily related to the network, which is modeled by the self-infection rate ε . An infected (information possessing) node is cured (releases the information) with a Poisson rate δ . The exact governing SIS

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the analysis of Gross *et al.* are presented in [19–25]. Instead of the discrete-time model with a unique rewiring rate in [11,22], here we present two continuous-time models, the continuoustime adaptive information diffusion (AID) model and the adaptive susceptible-infected-susceptible (ASIS) model, with separate link-breaking and -creating rates. The two seemingly similar epidemic models, both representing real-world appearances but with opposite topology dynamics, surprisingly exhibit a completely different stability of the metastable state. Moreover, the two models possess a different scaling of the epidemic threshold, while the properties of the metastable topologies show similar but phase rotated shapes. Interestingly, our analysis does not resort to mean-field approximations, taking into account the topological and epidemic variations of the nodes.

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equation for the infection (information possessing) probability of node *i* is

$$\frac{d}{dt}E[X_i] = E\left[(-\delta + \varepsilon)X_i + (1 - X_i)\beta \sum_{j=1}^N a_{ij}X_j\right].$$
 (1)

The topology dynamics in the ASIS and AID models are opposite. In both models, two Poisson processes, the link breaking and link creating with rates ζ and ξ , respectively, change the network's topology. In the AID model the linkcreating process establishes a link between a node pair (i, j)when exactly one node *i* or *j* has the information. The link-breaking process removes an existing link between a node pair (i, j) when both *i* and *j* do not possess the information and if there was no link in the original network $[a_{ij}(0) = 0]$. The AID governing equation for the link existence probability $E[a_{ij}(t)] = \Pr[a_{ij}(t) = 1]$ is, for $i \neq j$,

$$\frac{d}{dt}E[a_{ij}] = [1 - a_{ij}(0)]E[-\zeta a_{ij}(1 - X_i)(1 - X_j) + \xi(1 - a_{ij})(X_i - X_j)^2].$$
(2)

As an initial graph we confine ourselves to the empty graph with N nodes and no links $[a_{ii}(0) = 0 \text{ for } i \neq j]$. The righthand side of (2) consists of two opposing processes. (a) While either node *i* or *j* (but not both) possesses the information, the link between nodes i and j is created with rate ξ , in this way modeling the tendency for the informationless nodes to obtain the information faster. This link-creating process is applicable to information diffusion in online social networks, where friendship and follower links can be changed adaptively. (b) If two adjacent nodes i and j do not have the information, the link between them is broken with rate ζ , due to the absence of incentives of maintaining a link between the informationless nodes. In the case that both node *i* and *j* have the information (i.e., $X_i = X_j = 1$), the link is preserved, i.e., $\frac{dE[a_{ij}]}{dt} = 0$. Hence, the link dynamics in (2) tends to increase the degree of a node with information and to decrease the degree of a node without information. For convenience, we define the effective information expiring rate by $\delta^* = \delta - \varepsilon$. By expressing the time in units of δ^* , the model parameters in (1) and (2) can be reduced to the effective information spreading rate $\tau = \frac{\beta}{\delta^*}$, the effective link breaking rate $\omega = \frac{\zeta}{\xi}$, and a choice of either ξ , β , or ζ . Just like the SIS process on a fixed graph, the AID process is Markovian¹ with the overall-healthy state (or absorbing state) as the steady state. The relevant physical behavior happens in the metastable state in which the system (the SIS process and network) above the epidemic threshold² τ_c remains for a long time [26,29].

We verify that the AID model is realistic by using data from Facebook, the most well-known social network nowadays. Figure 1 shows that the AID model is realistic, by verifying Facebook wall posts [30] from the New Orleans area for the



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FIG. 1. (Color online) Prevalence of the AID model and the real Facebook data [30] from the New Orleans region (USA) for the last three months of 2008. A good fit is obtained.

last three months³ of 2008. The rates in the AID model are extracted [31] from the data and the process is detailed in [32]. Subsequently, the prevalences obtained from the AID model and from Facebook data are compared in Fig. 1, illustrating a good fit.

In the ASIS model [33], the topology changes in the opposite way: (a) While either node *i* or *j* (but not both) is infected, the link between node *i* and *j* is removed with rate ζ in order to protect the susceptible node from infection and (b) while both node *i* and *j* are susceptible, a link is created between them with rate ξ . For the ASIS model, in the case that both node *i* and *j* are infected (i.e., $X_i = X_j = 1$), the link is preserved, whose link dynamic, opposite to (2), is

$$\frac{d}{dt}E[a_{ij}] = a_{ij}(0)E[-\zeta a_{ij}(X_i - X_j)^2 + \xi(1 - a_{ij})(1 - X_i)(1 - X_j)].$$
 (3)

The aim of this paper is to report a striking difference that emerges from the two seemingly similar models, AID and ASIS, that both reflect realistic physical phenomena. Both models build upon the SIS epidemic model (1), but adaptively change the topology in opposite ways given in (2) and (3) for the AID and ASIS models, respectively. The factors $-\zeta a_{ij}$ and $\xi(1 - a_{ij})$ multiply $(1 - X_i)(1 - X_j)$ and $(X_i - X_j)^2$, respectively, for the AID model in (2), while the order of multiplication for the same factors is reverse in the ASIS model in (3). Most importantly, the AID model shows instability and nonexistence of the metastable state for some regions of τ and ω , which is not the case for the ASIS model. The characteristic differences between the ASIS and AID models are summarized in Table I. We further proceed to explain those differences.

Without resorting to any mean-field approximation, we provide exact expressions for the fraction of infected nodes and the epidemic threshold for both models. These relations, although not closed-form expressions, due to the existence of probabilistic and variance terms, provide exact solutions and, more interestingly, can explain the existence and stability of the number of infected nodes in the metastable state for both AID and ASIS models. The key observation that correlation terms the same as $E[a_{ij}X_iX_j]$ appearing in (1)–(3) led to the

¹We can enumerate the (huge) state space. From any state, there is a uniquely defined transition to another state with constant rate.

²For $\tau < \tau_c$, node *j* is almost always healthy, $X_j \rightarrow 0$, and the link dynamics (2) shows that the metastable topology is close to the original graph.

³After the major redesign of Facebook [32], allowing users to more easily see friends' posts (articles) on its own wall.

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TABLE I. Comparison of the adaptive models.

Model	Metastable state	Threshold $\tau_c(\omega)$	Topological metrics
ASIS AID	always stable unstable (τ, ω) regions	linear (mostly) constant	half elliptical rotated half elliptical

explicit relations of the prevalence, the fraction of information possessing or infected nodes. We define by $Z^* = \frac{1}{N} \sum_{j=1}^{N} X_j^*$ the prevalence and the average metastable state prevalence by $y = E[Z^*]$ in a graph with *N* nodes. Interestingly, $\operatorname{Var}[Z^*] \leq y \leq 1$, where $\operatorname{Var}[Z^*]$ is the variance of the prevalence holds.⁴ We also define $T(N) = \frac{E[\sum_{i=1}^{N} d_i^*(1-X_i^*)]}{N^2}$, where d_j denotes the degree of node *j*. T(N) is bounded by

$$0 \leq T(N) \leq \frac{E\left[\sum_{j=1}^{N} d_{j}^{*}\right]}{N^{2}} = \frac{E[2L^{*}]}{N^{2}} \leq \frac{N(N-1)}{N^{2}} < 1.$$

For the AID model we have [34]

$$y = \frac{1}{2} \left(1 + \frac{\omega - 2}{2\tau N} \right) \left(1 \pm \sqrt{1 - \frac{4 \operatorname{Var}[Z^*] + 2\omega T(N)}{[1 + (\omega - 2)/2N\tau]^2}} \right),$$
(4)

A key observation from (4), leading to the nonexistence of the metastable state for AID in some parameter regime, is the possibility that the argument under the square root is negative. Indeed, let us consider a large network size, where $N \rightarrow \infty$, so that (4) simplifies to

$$y = \frac{1}{2} \{ 1 \pm \sqrt{1 - (2\omega T_{\infty} + 4 \operatorname{Var}[Z^*])} \}.$$
 (5)

Equation (5) shows that the metastable state does not exist for AID if $4 \operatorname{Var}[Z^*] + 2\omega T_{\infty} > 1$ and hence

$$Var[Z^*] > \frac{1}{4}$$

is sufficient for the nonexistence of the metastable state in AID. Moreover, (5) leads to an upper bound for the link-breaking rate ω :

$$\omega \leqslant \frac{1 - 4\operatorname{Var}[Z^*]}{2T_{\infty}} \leqslant \frac{1}{2T_{\infty}};$$

otherwise there will not be a metastable state solution.

⁴Due to the Cauchy-Schwarz inequality, we have $(\sum_{j=1}^{N} X_j)^2 \leq N(\sum_{j=1}^{N} X_j^2) = N(\sum_{j=1}^{N} X_j)$ because $X_j \in \{0,1\}$. Now, we can apply this to $\operatorname{Var}[Z^*] = E[Z^2] - (E[Z])^2 \leq E[Z^2] = \frac{1}{N^2}E[(\sum_{j=1}^{N} X_j)^2] \leq \frac{1}{N^2}E[N\sum_{j=1}^{N} X_j] = \frac{1}{N}E[\sum_{j=1}^{N} X_j] = E[Z] \leq 1.$

The consequences of (5) are confirmed by extensive simulations. There are regions for (τ, ω) where the metastable state does not exist, as demonstrated in Figs. 2(a) and 2(b). The instability area exhibits an hourglass shape: it is wider close to the center of the coordinated system, further narrows, and then widens again for higher τ and ω . The instability area in AID finally vanishes for high enough τ and ω . Finally, as a side note, we find that there are regions of instability even in a model more general than ours, where the infection rates change over time.⁵

The ASIS metastable state prevalence is [34]

$$y = \left(1 - \frac{1}{2N} + \frac{\omega^* - 1/2}{\tau N}\right) \times \left(1 \pm \sqrt{1 - \frac{1 - 1/N + \operatorname{Var}[Z^*] - T(N)}{[1 - 1/2N + (\omega^* - 1/2)/\tau N]^2}}\right), \quad (6)$$

where the value under the square root in (6) is always positive. Hence, the metastable state always exists [33] and is given by (7) with a minus sign. The prevalence y as a function of τ is shown in Fig. 2(c). For $N \to \infty$, (6) can be reduced to

$$y = 1 \pm \sqrt{T_{\infty} - \operatorname{Var}[Z^*]}.$$
 (7)

In contrast to the AID model, there is in (7) no constraint on ω for the ASIS metastable state.

For a combination of $(\beta, \delta, \zeta, \xi)$ with a relatively higher link-breaking rate than the creating rate and small spreading rate in the AID model, a small portion of nodes obtains the information, which does not have the potential (the spreading rate is small and the breaking rate relatively big) to stay long nor can be considered as a metastable state. Consequently, in such a combination, multiple and sharp changes, both epidemically and topologically, are visualized in Fig. 3(a). In the other case of a stable combination, there is a critical mass of links and information-possessing nodes and although

⁵This is a case in the model of Huang *et al.* [35] for time-changing activity-dependent infection rates. To not break the flow of the paper, more details are given in [32].



FIG. 2. (Color online) (a) Instability region for the AID model. (b) Prevalence *y* in the AID model. (c) Prevalence *y* the ASIS model. (a) and (b) demonstrate the instability in the AID model. (c) demonstrates the stability in the ASIS model.



FIG. 3. (Color online) Numbers of links and infected nodes as functions of consecutive time moments in the AID model for N = 40, $\zeta = 0.32, \xi = 0.1, \delta = 1, \varepsilon = 10^{-3}$, and different spreading rates β : (a) $\beta = 0.152$ and (b) $\beta = 1.0$. The points of instability or stability are in accord with Fig. 2(a).

there are time changes, the forces of the epidemics reach an equilibrium (e.g., one link is broken, but another is created), which represents the metastable state as shown in Fig. 3(b).

The epidemic thresholds can be determined from the equations of both AID and ASIS models [34]. Surprisingly, the threshold is linear in ω only for the ASIS model,

$$\tau_c(\omega;\xi) = \frac{2\omega - 1}{N[h_{\text{ASIS}}(\omega;\xi) - 2 + 1/N]},\tag{8}$$

where $1 \leq h_{ASIS}(\omega;\xi) \leq 2 + \frac{1}{N}(\frac{1}{a}-1)$ and $a = \frac{\partial \tau_c(\omega;\xi)}{\partial \omega}|_{\omega \to \infty}$ is almost a constant. The function $h_{ASIS}(\omega;\xi)$ is positive, slowly varying, and obeys $h_{ASIS}(\frac{1}{2};\xi) = 2 - \frac{1}{N}$ for the ASIS model, for all $\omega > 0$.

For AID, on the other hand, the information threshold is the quotient of two linear functions, which approaches a constant for large ω ,

$$\tau_c(\omega;\xi) = \frac{\omega - 2}{2N[h_{\text{AID}}(\omega;\xi) - 1]},\tag{9}$$

where $h_{AID}(\omega;\xi) \leq 1 + \max\{1, 1 + \frac{\omega-2}{2Na}\}$ and $a = \lim_{\omega \to \infty} \frac{\partial h_{AID}(\omega;\xi)}{\partial \omega}$ is almost a constant. For $\omega > 2$, the function $h_{AID}(\omega;\xi)$ is almost linear in ω , obeying $h_{AID}(2;\xi) = 1$ for the AID model.

The simulations shown in Fig. 4(b) indicate that $\tau_c(\omega; \xi)$ increases almost linearly in ω , confirming (8) for the ASIS model, while Fig. 4(a), for the AID model, demonstrates that the epidemic threshold is the quotient of two linear functions in ω and is almost constant for large ω . Finally, a noisy epidemic



FIG. 4. (Color online) Threshold τ_c versus effective linkbreaking rate ω for N = 40 (the inset shows the large range of ω) for the (a) AID model and (b) ASIS model.

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FIG. 5. (Color online) Modularity in the (τ, ω) plane in the stable region for $\xi = 0.1$ for the (a) AID model and (b) ASIS model.

threshold in Fig. 4(a) is an identifying characteristic of the instability in the AID model.

Figure 5 shows, as a contour plot in the (τ, ω) plane, the modularity value of the networks in the metastable for both ASIS and AID (where stable) models. The contour lines resemble roughly concentric half ellipses for the effective infection rate τ well above the epidemic threshold where the epidemic is active in the metastable state. A remarkable observation is that, for a fixed effective infection rate τ , there are two different values for ω reaching the same value of the metric, each representing the regimes of very small or very high effective link-breaking rates ω . The half-elliptical contour lines of the modularity in the ASIS and AID models only differ in two aspects: (i) the order of the contour lines (the inner contour lines show higher modularity in the ASIS model and lower in AID) and (ii) they are rotated from one another, although the shape is surprisingly similar. In the (τ, ω) plane, the instability area, which has an hourglass shape [Fig. 2(a)], exists only for the AID model, is just below the half-ellipse extremal node, looks like their natural extension, and is close to the center of the coordinate system.

The metastable state (where it exists) of the AID model is a random graph, while the metastable state in the ASIS model is a graph with two separated components that are loosely connected, each representing the susceptible (close to a complete graph) and infected nodes (random graph). In the absolute stable state in both ASIS and AID models all nodes are susceptible. However, the final stable state topology in AID is an empty graph, while it is a complete graph in the ASIS model. The metastable states are physically more interesting and those states are the focus of this paper.

Summarizing, our analysis of the adaptive ASIS and AID epidemic models, with opposite topology dynamics, leads to the following contributions.

(i) In the metastable state, there is an hourglass-shape area of instability only for the AID model. The ASIS metastable state always exists.

(ii) The AID epidemic threshold τ_c is almost constant in the effective link-breaking rate ω , while $\tau_c(\omega)$ linearly increases with ω for the ASIS model.

(iii) In the (τ, ω) plane, topological metrics of both adaptive epidemic models exhibit concentric half ellipses. The ASIS and AID models differ in the order of the ellipses and the rotation.

(iv) By extracting the model rates (detailed in [32]), we validate the AID model with data [30] from Facebook, the most well-known social network nowadays.

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