Self-adapting infectious dynamics on random networks

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Self-adaptive dynamics occurs in many physical systems such as socio-economics, neuroscience, or biophysics. We formalize a self-adaptive modeling approach, where adaptation takes place within a set of strategies based on the history of the state of the system. This leads to piecewise deterministic Markovian dynamics coupled to a non-Markovian adaptive mechanism. We apply this framework to basic epidemic models (SIS, SIR) on random networks. We consider a co-evolutionary dynamical network where node-states change through the epidemics and network topology changes through creation and deletion of edges. For a simple threshold base application of lockdown measures we observe large regions in parameter space with oscillatory behavior. For the SIS epidemic model, we derive analytic expressions for the oscillation period from a pairwise closed model. Furthermore, we show that there is a link to self-organized criticality as the basic reproduction number fluctuates around one. We also study the dependence of our results on the underlying network structure.

I. INTRODUCTION

Complex adaptive systems often arise when interacting nodes (or agents) adapt their dynamics (or strategies) due to external influences. This occurs for example in socio-economic systems, such as epidemic and information spreading, markets, evolution of languages, or evolutionary games with environmental feedback, but also in automation problems including machine learning or control theory. In standard network models adaptive dynamics is often imposed by creation, deletion or rewiring of edges between pairs of nodes, some aspects of which have been extended to higher-order networks. From a more general perspective adaptivity is characterized by a set of strategies which are selected either by the system itself or by individual agents in the system, each leading to potentially different dynamical rules and future evolution.

The adaption of strategies may be characterized by a change of parameters. This occurs for example in piecewise deterministic Markov processes (PDMPs), where some parameter of the system evolves according to an additional, independent stochastic process. Assuming that the parameter space represents a possible set of strategies, this is equivalent to a system where a decision maker (randomly) changes the strategy. If this parameter crosses a bifurcation the stability of the system changes abruptly, typically leading to an entirely different dynamical evolution. On the other hand, if parameter changes are influenced by the history of the system and occur with some temporal delay usually delay equations are considered. Examples are biomedical models of cancer evolution, population dynamics or machine learning. It is reasonable to assume that in real world systems both effects occur: delayed adaptation depending on the history of the state and piece-wise deterministic evolution coupled to sudden strategy changes. Still, these approaches have so far not been connected.

Particularly interesting are systems with policy makers who adapt either the rules of the system or their own behavior according to some, usually complex, evaluation mechanism. For example, restrictions and lockdown laws have been enforced and lifted during the Sars-Cov-2 pandemics depending on the recent course of the pandemic, and are regularly changed, e.g., due to the emergence of new variants or available vaccinations. Therefore it is reasonable to ask how basic epidemic models behave in the context of adaptive policies. This is related to recent models for the influence of individual risk perception on the epidemic spreading of Sars-Cov-2, where direct feedback mechanisms are considered.

Epidemic modeling has a long history, the most famous examples being the SIS and SIR models. In these compartmental models the population is divided into susceptible (S) and infected individuals (I), while after recovery become either susceptible again or recovered (R), for SIS and SIR systems, respectively. These models are often considered on contact networks, where existing links determine possible infections between individuals, see Ref. and references therein. Many extensions have been studied, from additional compartments, to adaptive rewiring of edges, adaptive force of infection, and including vaccination. Depending on the specific model and parameters, the system state typically either approaches a stable disease-free or endemic equilibrium, oscillates, or shows more complex dynamics. Apparently such adaptive models often become analytically unfeasible, even if the high-dimensional network dynamics is reduced to the level of differential equations by appropriate closure techniques.

In this paper we propose a framework for history dependent strategy adaption with piece-wise deterministic dynamics. This is applied to simple models of adaptive epidemics for which an analytical treatment is possible. In particular we consider a threshold-based activation and deactivation of edges within a fixed contact network for the examples of SIS and SIR systems. We observe and describe a large parameter region with stable oscillations, as well as bifurcations towards stable disease-free and stable endemic states for the case of SIS. We derive
analytic expressions for the period and observe that the network topology significantly influences the agreement by comparing different types of random networks.

The paper is structured as follows. In Sec. II we propose a model of strategy adaption. Section III recapitulates SIS and SIR models on networks. In Sec. IV we apply the adaptive framework to SIS and SIR epidemics on networks. A summary and outlook is presented in Sec. V.

II. DYNAMICS WITH STRATEGY ADAPTION

There are many ways to include adaptivity into dynamical systems, e.g., by rewiring rules \cite{27} on the network level or by state-dependent parameter changes \cite{30} on the level of differential equations. In contrast, here we extend a system with a strategy space $S$ such that the dynamics depends on the currently active strategy $S_t \in S$. For example, in a system with infectious dynamics, the strategies $S_t$ could refer to different combinations of reduced numbers of average contacts $k_t < k_0$, and/or increased measures against transmission, $\tau_t < \tau$. Alternatively, a strategy could impose the emergence (or deletion) of additional compartments $X \in \{Q,E,I,\ldots\}$ into the model, e.g., enforcing quarantine, separately counting exposed but not infectious agents, or introducing new variants of the disease.

It is natural to assume that the chosen strategy depends on some observable function $g : \Gamma \to \mathbb{R}^n$, which evaluates the state $x \in \Gamma$ of the system. This is in particular relevant if the strategies are chosen by some decision maker(s) based on their evaluation metric. Note that in real social systems often many different metrics are used to evaluate the state, e.g., incidence, hospitalization rate, and vaccination rate are all possible options to evaluate the state of an epidemic. For the sake of simplicity we consider a one-dimensional metric, $n = 1$, $g(x) \in \mathbb{R}$, in the following.

In general, the history and the current state are important for the evaluation of future strategies. In order to include the recent history of the state we average the observable $g$ over time \cite{30},

$$J(t) = \int_{0}^{\infty} \rho(t') g(x(t-t')) dt' = \int_{-\infty}^{t} \rho(t-t') g(x(t')) dt',$$

with a suitable integration kernel $\rho$, also known as delay kernel, satisfying $\int_{0}^{\infty} \rho(t) dt = 1$. The function $J$ at the time $t$ acts as a measure on how the strategy of the system is adapted due to the history of the state. We call $J$ strategy function in the following. Additionally let us define the adaption function $A$ which specifies which strategy $S \in S$ is currently chosen. For observable $g$ and strategy function $J$ defined as above, the adaption function is defined as a mapping from $A : T \times \mathbb{R} \times S \to S$, where $T$ is the time domain and $A(t,J(t),S) = S' \in S$.

We define an adaptative dynamical system with discrete strategies as follows. Let $\Phi$ be a map from the strategy space $S$ to a flow on $\Gamma$, i.e., $\Phi_S := \Phi(S)$ is a mapping $\Phi_S : T \times \Gamma \to \Gamma$ for all $S \in S$ with $\Phi_S(0,x) = x$ and $\Phi_S(t_2,\Phi_S(t_1,x)) = \Phi_S(t_1 + t_2,x)$ for all $t_1, t_2 \in T$ and $x \in \Gamma$. An adaptive dynamical system is then defined as the tuple $(T,\Gamma,\Phi,\Phi_S,\rho)$, such that for each $S \in S$ the triple $(T,\Gamma,\Phi(S))$ is a dynamical system and where the strategy $S_t$ evolves according to the adaption function $A$ as defined above.

We emphasize that the time evolution of the system depends on the current strategy $S_t$, and simultaneously the current strategy depends on the time evolution of the system. This leads to a nontrivial feedback mechanism and possibly complex and interesting dynamics. In order to introduce such an adaptive mechanism to some dynamical system, it is therefore necessary to specify a set of strategies $S$ and their implications on the internal dynamics, some observable $g$ and strategy function $J$, and how the system adapts its dynamics to changes in $J$ through the function $A$.

This framework connects piecewise deterministic Markov processes and delay equations: If the switching of strategies $A$ is governed by a Markov process the full system is equivalent to PDMPs, see e.g. \cite{13,16}. On the other hand, if the selected strategy depends on some parameter evaluated at a delayed time $\tau$ in the past, i.e., the integration kernel is $\rho(t) = \delta(t-\tau)$, the resulting system can be reduced to delay equations \cite{36}. In the following we apply this adaptive framework to epidemiological models on networks.

III. EPIDEMICS ON NETWORKS

One of the simplest epidemiological models on networks is the SIS model, see \cite{3}. Let $G = (\mathcal{N},\mathcal{E})$ be a graph consisting of nodes $\mathcal{N}$ and edges $\mathcal{E}$. The state of each node $n \in \mathcal{N}$ is either susceptible ($X(n) = S$) or infected ($X(n) = I$). The epidemic is transmitted through the edges $(i,j) \in \mathcal{E}$ from infected nodes to adjacent susceptible nodes with a fixed transmission rate $\gamma$, see Fig. 1. Recovery of infected nodes occurs with rate $\gamma$. For SIS recovered nodes become susceptible again. The full SIS dynamics on a network is described by the unclosed differential equations for the number of infected $n_I$ and the number of susceptible $n_S$ individuals \cite{37}.

$$n_I' = -\gamma n_I + \gamma n_S,$$

$$n_S' = \gamma n_I - \gamma n_S.$$

(a) $\begin{array}{c}
\text{I} \\
\tau \\
\text{S} \\
\end{array}$

(b) $\begin{array}{c}
\gamma_{\text{sim}} \\
\text{I} \\
\gamma_{\text{sim}} \\
\end{array}$

Figure 1. Evolution of SIS and SIR epidemics. (a) Infection along SI-edges with rate $\tau$. (b) Recovery of infected nodes with rate $\gamma$ for SIS (left) and SIR (right).
where \( n_{SI} \) denotes the number of SI-edges. The differential equation for \( n_{SI} \) depends on the higher-order moments \( n_{abc} \) with \( a, b, c \in \{S, I\} \), which in general leads to an infinite series of differential equations. Only for specific network types, such as the complete graph, or by applying closure relations to Eq. (2) one obtains a finite system of differential equations, which in general only approximate the full network dynamics, see e.g. \([37, 35]\).

In the following we focus on the simplest closure relation, given by the pairwise approximation \( n_{SI} \approx \frac{k_0}{N} n_{IS} \), which is based on the assumption that connections between \( S \) and \( I \) individuals are spread homogeneously through the network. Here, \( k_0 \) denotes the average degree of nodes in the network and \( N = |\mathcal{N}| \) is the number of nodes. Without birth and death-processes the number of infected individuals \( x_1 = n_I / N \in [0, 1] \) the dynamics reduces to the one-dimensional ODE

\[
x_1' = -\gamma x_1 + \tau k_0 (1 - x_1) x_1.
\]

Stability analysis reveals two different regimes \([37]\): For \( \beta := \tau k_0 / \gamma < 1 \) the disease free state \( x_0 = 0 \) is stable and it is the only equilibrium within the reasonable interval \( x_1 \in [0, 1] \). For \( \beta > 1 \) a transcritical bifurcation occurs and \( x_0 \) becomes unstable, while the endemic state \( x_e := 1 - 1/\beta \in [0, 1] \) emerges as a stable equilibrium.

Another simple model for the spreading of a single epidemic wave through a population is the SIR model, see e.g. \([37]\). Here, infected individuals which recover are completely removed (R) from the system, and thereby determines the current strategy

\[
\begin{align*}
n_I' &= -\gamma n_I + \tau n_{SI}, \\
n_S' &= -\tau n_{SI}, \\
n_R' &= \gamma n_I.
\end{align*}
\]

The corresponding pairwise closed moment equations reduce to the two-dimensional system

\[
\begin{align*}
x_I' &= -\gamma x_I + \tau k_0 x_I x_S \\
x_S' &= -\tau k_0 x_I x_S.
\end{align*}
\]

The fraction of recovered individuals follows from \( x_R = 1 - x_I - x_S \) for all times.

IV. EPIDEMICS WITH STRATEGY ADAPTATION

We consider an epidemic system on a network \( \mathcal{G} = (\mathcal{N}, \mathcal{E}) \) with transmission rate \( \tau \) and recovery rate \( \gamma \). The basic network structure of \( \mathcal{G} \) is assumed to be fixed, i.e., the set of edges \( \mathcal{E} \) does not change. Adaptive activation and deactivation of edges occurs within this framework by assigning a weight \( w : \mathcal{E} \to [0, 1] \) to each edge \( e \in \mathcal{E} \), which determines if the edge is active, \( w(e) = 1 \), or not \( w(e) = 0 \). More generally, one could assume transmission rates \( \tau(e) = w(e) \tau \), which are relevant in the context of different preventive measures, such as quarantine \([39, 40]\) or social distancing \([41]\). In order to make this system adaptive we consider the simplest case with only two strategies, \( \mathcal{S} = \{S_0, S_\gamma\} \), which determine the number of active edges. Here, the null-strategy \( S_0 \) corresponds to case where all (existing) edges in the network are active, \( w(e) = 1 \forall e \in \mathcal{E} \). The strategy \( S_\gamma \) corresponds to a lockdown strategy, where each edge is deactivated with probability \( p_{cut} \), i.e., \( F[w(e) \to 0] = p_{cut} \). This means that the average degree in the adaptive network becomes time dependent with \( k_\gamma = k_0 (1 - p_{cut}) \).

For the adaptive mechanism \( A \) consider prevalence and incidence as suitable observable functions, i.e., \( g_1(t, x_1) = x_1 \) and \( g_2(t, x_1) = \tau n_{SI} / N \approx \tau k_0 (t) x_1 x_S \). Furthermore, the kernel in Eq. (1) is chosen such that \( g \) is averaged over fixed time intervals \([t - \Delta t, t]\) for some time span \( \Delta t \), i.e., \( \rho(t) = \frac{1}{\Delta t} \Theta(t) - \Theta(t + \Delta t) \) with Heaviside function \( \Theta \). This leads to \( J_{1,2}(t) = \int_{t-\Delta t}^{t} g_{1,2}[t', x_1(t')] \, dt' \). We emphasize that such an average has been a commonly used measure during the Corona pandemic, e.g., in terms of the so-called 7-day incidence, see e.g., \([22]\). Let us emphasize that \( J \) measures the seriousness of the current pandemic situation, where more infections or larger prevalence manifest in larger values of \( J \).

The simplest possible adaptive function \( A \) for two levels is based on fixed thresholds. For this, we define two thresholds \( \xi_+ > \xi_- \) and the following strategy adoptions,

\[
A(t, J(t), S_\gamma) =
\begin{cases}
S_\gamma \text{ for } J(t) \geq \xi_+, \\
S_0 \text{ else,}
\end{cases}
\]

\[
A(t, J(t), S_-) =
\begin{cases}
S_0 \text{ for } J(t) \leq \xi_- , \\
S_- \text{ else.}
\end{cases}
\]

This means, whenever the strategy function hits the predefined thresholds from below or from above, respectively, the system automatically enters the state of social distancing or goes back to the usual state. Let us emphasize that the memory dependence is hidden in the strategy function \( J \). This function is continuously evaluated for all \( t \) and thereby determines the current strategy \( S \in \mathcal{S} \) of the system.

Note, that there are numerous possibilities for defining the adaption function, which can either deterministically or also stochastically select the future strategy. In the latter case the system contains two distinct sources of stochasticity, one from the dynamics and one from the adaption mechanism, which adds another layer of complexity. Here we focus on the deterministic case.

We emphasize that our proposed model differs quite substantially from recent stochastic SIRS models \([14, 16]\), which are piecewise deterministic Markov processes (PDMPs). In these systems, the switching of parameters is guided by an additional random stochastic process, which is independent of the (deterministic or stochastic) dynamics. Here, in contrast, the dynamical evolution of the system feeds back into the adaptive mechanism. If the underlying dynamics is stochastic, the adaption of
strategies is guided by the (stochastic) feedback process implemented with the adaptive function. On the other hand, in a purely deterministic setting the proposed system can be seen as an extension of the phase-space with the set of strategies, $\mathcal{X} \times S$, where switching between different branches occurs deterministically according to the adaptive function $A$.

A. Results for SIS

For the SIS epidemics on a network one expects an initial growth of the prevalence $x_I$ until $J$ exceeds $\xi_+$. At this point the social distancing strategy $S_-$ is applied by removing a proportion of $p_{\text{cut}}$ edges from the system, see illustration in Fig. 2(a). If sufficiently many edges are removed, the prevalence decreases and $J$ becomes smaller than $\xi_-$, such that the null-strategy $S_0$ is reapplied. Within this regime we expect periodic behaviour.

In the following we first derive different dynamical regimes in the adaptive pairwise closed SIS model and secondly compare this to numerical results obtained from simulation on networks. Recall that for each strategy, $S_0$ and $S_-$, there is one stable equilibrium $x_I(\beta)$ with $x_I(\beta) = x_0 = 0$ for $\beta = k\tau/\gamma < 1$ and $x_I(\beta) = x_{\text{en}}(\beta) = 1 - \beta^{-1}$ for $\beta > 1$, see red curve in Fig. 2(a). In the following we assume arbitrary but fixed thresholds $0 < \xi_- < \xi_+ < 1$ and specify how the dynamics depends on the choice of $(\beta_0, \beta_-)$. For an illustration see Fig. 2(b). For simplicity, we also assume that $\Delta t = 0$, such that $J(t) = g(x_I(t))$. First, if $\beta_0 < 1$ the state of the system converges to the stable equilibrium $x_0 = 0$ and the strategy remains in $S_0$ for all times. Secondly, for $\beta_0 > 1$ the state of the system approaches the endemic state $x_{\text{en}}(\beta_0)$. The strategy switches, if $g(x_I) \geq \xi_+$, which implies the limit $x_{\text{en}}(\beta_0) = 1 - \beta_0^{-1} = g^{-1}(\xi_+)$. In particular, for $1 < \beta_0 < 1 - g^{-1}(\xi_+)$ the threshold $\xi_+$ is too large and the state of the system converges to $x_{\text{en}}(\beta_0)$. On the other hand, for $\beta_0 > 1 - g^{-1}(\xi_+)$ the strategy will eventually switch to $S_-$ for some $t > 0$. After switching, the state converges to the stable equilibrium $x_0(\beta_-)$. Similar considerations as above lead to the following limits depending on the threshold $\xi_-$. If $\beta_- > 1 - g^{-1}(\xi_-)$ the state approaches the stable endemic equilibrium $x_{\text{en}}(\beta_-)$, without reaching the lower threshold, thus remaining in strategy $S_-$ for all times. If $\beta_- < 1 - g^{-1}(\xi_-)$ the stable equilibrium is below the threshold, such that after some finite time the system switches back to $S_0$ again. Hence, in this regime the dynamics is periodic.

Conversely, if $\beta_0 > 1$ and $\beta_- \text{ are fixed}$, it is possible to specify limits for the thresholds $\xi_\pm$, within which we expect periodic dynamics. In particular, one obtains $\xi_+ \leq g(1 - 1/\beta_0)$ and $\xi_- \geq g(x_0(\beta_-))$. With $\beta_- = (1 - p_{\text{cut}})k\tau/g^{-1}(\xi_-)$ these conditions similarly imply a lower bound for the cutting probability $p_{\text{cut}}$ at given $\xi_-$, given by $p_{\text{cut}} \geq 1 - (1 - g^{-1}(\xi_-))k\tau$.

In Figure 3 we illustrate the time dependence of the relative prevalence $x_I$ for the adaptive SIS epidemics with strategy function $J_2$ on two different network types, networks form the random Erdős-Rényi (ER) ensemble $G(N, p)$ [42,43] and scale-free Barabasi-Albert (BA) networks [44]. The parameters are $\gamma = 0.25$, $k_0 = 50$, $\beta_0 = 2$ and $p_{\text{cut}} = 0.8$ with thresholds $\xi_+ = 0.025$ and $\xi_- = 0.005$, i.e., the lockdown strategy is enforced when each infected individual infects on average 2.5% of the population (over the past time-window $\Delta t = 5$) and it ends below 0.5%. These parameters correspond to the periodic regime in Fig. 2(b).

For both network types the fraction of infected individuals $x_I$ oscillates see top panels in Fig. 3(a) and (b). The corresponding incidence function also oscillates between the predefined thresholds $\xi_\pm$, shown as blue curve and red dotted lines, respectively, in the bottom panels. Note that the discontinuities of the incidence function are caused by the sudden (de)activation of edges, which immediately changes the number of SI-edges and thereby the possible number of new infections. For comparison the analytic result of the pairwise closed moment system is shown as a dashed black curve, see App. A. This agrees very well with the simulation for the ER-network. In contrast, the initial spreading of the epidemics in the BA network is much steeper, showing also larger maximal values of $x_I$. Consequently, the frequency of fluctuation for these scale-free networks is increased compared to the pairwise closed system.

a. Period of fluctuations $T$. We further investigate how the period of fluctuations $T$ depends on the parameters of the system for the prevalence strategy function $J_1$. The period $T$ is the sum of the expected time $T_0$ to switch from $S_0$ to $S_-$ and $T_{-0}$ to switch back. For $J_1$
a derivation of $T$ is possible in the pairwise closed SIS model, see App. A, which leads to

$$T = T_{0-} + T_{00}$$

$$= \frac{1}{\gamma} \log \left[ \frac{\xi_+ (\beta_0 - 1 - \beta_0 \xi_-)}{(\beta_0 - 1) \xi_- - \xi_+ \beta_0 \xi_-} \right]^{\beta_0 - 1}$$

$$+ \frac{1}{\gamma} \log \left[ \frac{\xi_- (\beta_- - 1 - \beta_- \xi_+)}{(\beta_- - 1) \xi_+ - \xi_- \beta_- \xi_+} \right]^{\beta_- - 1}.$$  \hspace{1cm} (8)

In Figure 3 we show $T$ versus the upper threshold $\xi_+$ for two different values of $\beta_0 \in \{1.6, 2\}$ and compare network simulations (green errorbars) to the analytic result (black curve). The upper threshold is chosen in the interval $\xi_- \leq \xi_+ < x_{\text{crit}}(\beta_0)$. Note that the expected period becomes zero at some $\xi_+$ smaller than $\xi_-$, see Fig. 2 e.g., for $\Delta t = 0$ this occurs at $\xi_+ = \xi_-$. On the other hand, if $\xi_+ \to x_{\text{crit}}(\beta_0)$ the expected period goes to infinity for the analytic model. This is also observed in the network simulations: For random ER networks the analytic expression of the pairwise closed model fits very well for both $\beta_0$, see (a) and (c). For the scale-free BA network and $\beta_0 = 1.6$ the observed period is systematically too large for smaller $\xi_+$ and too small for larger $\xi_+$.

Intuitively, this is explained as follows. The prevalence increases initially much faster in scale-free networks due to nodes with very large degrees (superspreaders), thereby overshooting the threshold significantly stronger, see Fig. 3(c). Thus, the initial prevalence in the second half of the cycle is increased compared to the random networks, thereby increasing the period significantly. This effect becomes negligible, if the upper threshold gets closer to the endemic equilibrium, since here the prevalence typically flattens for all network types and the overshooting effect vanishes. Instead, the initially much faster speed of infection leads to shorter periods in this regime.

The strong dependence on the topology is revealed when considering larger values of $\beta_0 = 2$ and leaving the other parameters unchanged. Note that with $p_{\text{cut}} = 0.6$ this still implies $\beta_- = 0.8$ significantly smaller than one. In the scale-free network the observed periods are up to four times longer than predicted by the moment equations, Fig. 4(d). This means that with the same number of deleted edges in scale-free networks the progression of the epidemic spreading is slowed down more efficiently than in random networks. Note that these longer periods are mainly caused by a much flatter slope during

Figure 3. Relative prevalence $x_1 = I/N$ and incidence $\tau_{SI}/N$ for SIS epidemics on adaptive network with $N = 2000$, $k_0 = 50$, $\gamma = 0.25$, $\beta_0 = 2$, and threshold adaption as in Eq. 1 using $g_2$ (incidence thresholds) and $\Delta t = 5$, $\xi_+ = 0.025$, $\xi_- = 0.005$. The lockdown strategy is given by $p_{\text{cut}}(S_-) = 0.8$ ($\beta_-$ = 0.4). Considered are (a) Erdős–Rényi networks and (b) Barabasi–Albert networks.
the lockdown phase $S_{-}$. This corresponds to an effective cutting probability of $p_{\text{cut}} \approx 0.49$ (gray line).

This shows that the network topology leads to observable differences in adaptive network systems. We believe that such topology induced effects on oscillations can also be observed in more complex models.

b. Basic reproductive number $R_0$. The basic reproductive number $R_0$ is commonly defined as the expected number of new infections caused by one infected individual in a fully susceptible community [37, 47]. For the pairwise closed SIS model this gives the ratio of total rate of infection to total rate of recovery, $R_0 = \beta = k\tau/\gamma$ [37]. If for infectious disease $R_0 > 1$ an epidemic outbreak occurs, while for $R_0 < 1$ the disease vanishes.

For the SIS epidemics with adaption the average degree on the network depends on the currently active strategy. Hence, the simplest approximation for $R_0$ is given by $\beta \in \{\beta_0, \beta_-\}$ with $\beta_0 = k\tau/\gamma$ and $\beta_- = \beta_0(1 - p_{\text{cut}})$. Numerically, the reproductive number is obtained by averaging the number of infections caused by each infected node over time. In particular, let $t_{\text{inf}}$ be the time of the $j$-th infection during the simulation and let $N_{\text{inf}}^j$ be the number of infections caused by the infected $j$-th node.

With this, we estimate $R_0$ as

$$R_0(t) \approx \langle \{ N_{\text{inf}}^j : t - \delta t < t_{\text{inf}}^j \leq t \} \rangle,$$

where $\langle \cdot \rangle$ denotes the average and we choose the time interval according to the recovery time $\delta t = 1/\gamma$.

In Fig. 5 we illustrate the reproductive ratio $R_0$ as a function of time, comparing numerical values (blue) to the simplest estimation based on $\beta$ (black). The numerically observed $R_0$ oscillates in a similar pattern around $R_0 \approx 1$, however, fluctuating on faster scales and typically below $R_0 = \beta_0 = 2$. We assume that the latter is caused by infectious clusters with a small number of susceptibles, leading to much fewer infections for large prevalence.

We emphasize that such fluctuations of the reproductive number around $R_0 \approx 1$ are also observed in many different countries during the Corona pandemic, e.g., for evolution in Germany see Fig. 6. This indicates that in real epidemics there is some form of self-organized criticality around $R_0 = 1$ due to the conflicting goals of reducing the number of infections and increasing the freedom of individuals. In particular, our simple model with adaptive strategies already mimics the complex adaptive dynamics due to stricter lockdown measures and more general cautious behavior at higher incidence levels. It is therefore reasonable to expect similar observations in more sophisticated models, e.g., when each agent chooses from a set of strategies and adapts in a more complex manner to the development of an epidemic. Furthermore, our results strongly support the claim that epidemic models with self-limiting feedback mechanisms

![Figure 5. Reproductive number $R_0$ as a function of $t$ for (a) Erdős-Rényi and (b) Barabasi-Albert networks, each with $N = 1000$ nodes and $k_0 \approx 50$. Other parameters are $\gamma = 0.25$, $\beta_0 = 2$, $k = 50$, $\xi_+ = 0.025$, $\xi_- = 0.005$, $p_{\text{cut}}(S_{-}) = 0.6$, $\Delta t = 5$, and the prevalence strategy function $J_1$ is considered. The reproductive ratio is numerically determined from the simulation (blue), compared to the prediction of the pairwise model (black dashed).](image)

![Figure 6. Estimation for daily new infections $\Delta n_I$ and $R_0$ based on 7-day nowcast for Sars-Cov-2 epidemics in Germany between 12.03.2020 and 06.03.2022 [45, 46]. The nowcast estimation for $R_0$ is determined by the quotient of new infections for one time-interval and those of the preceding interval, assuming that each interval corresponds to one generation of the virus.](image)
should be viewed through the lens of self-organized criticality [48].

B. Results for SIR

We further apply the proposed adaptive strategy approach to the SIR epidemics on networks [37]. In the SIR model recovered individuals do not contribute to the epidemic spreading anymore. Similar to the SIS model there is a regime where initial transient oscillations are expected. When a significant proportion of individuals is recovered, R, the epidemic dies out after a certain number of cycles.

This is illustrated in Fig. 4 showing the time dependence of relative prevalence and incidence for the SIR dynamics on two different network types, similar to Fig. 3. The considered thresholds are $\xi_+ = 0.01$ and $\xi_- = 0.002$. Note that for real epidemics these thresholds should be chosen according to the capacity of the health system, which typically can sustain only a very small fraction of infected individuals. Furthermore, choosing larger thresholds in the simulations leads to many infected and recovered individuals already before strategy adaption takes place. For both network types we observe the expected initial oscillatory dynamics in Fig. 7 similar to the SIS system. We observe that Erdős-Rényi networks show comparable results to the pairwise closed solution (black dashed line), even though individual simulations differ substantially. In contrast, the epidemic spreading in the scale-free network takes place much faster. This is intuitively explained with the power-law degree distribution and the existence of a small number of highly connected nodes. First, if one of these nodes is infected it triggers a lot of subsequent infections. Secondly, after its recovery it efficiently blocks the epidemic spreading.

a. Adaption times. We investigate numerically how the times of strategy adaption depend on the probability to cut edges during the lockdown phase $p_{\text{cut}}(S_-)$. This is illustrated in Fig. 8 for random ER and scale-free BA networks and compared to numerical solutions of the first
order moment SIR system with strategy adaption (black lines). For both network types and small \( p_{\text{cut}} \leq 0.5 \) the strategies switch only twice, from \( S_0 \) to \( S_- \) (violet colors) and back (green colors). In this regime the corresponding \( \beta_- \geq 1 \), such that the epidemic spreading slows down, but does not revert. Note that for \( p_{\text{cut}} = 0 \) no edges are removed and time evolution is equivalent to the SIR dynamics on the original networks. For increasing \( p_{\text{cut}} \) the number of adaptations increases and we find very good agreement between simulations on the ER network and the solution of the closed moment equation, see Fig. \( \text{Fig. 3a} \). The regime with \( p_{\text{cut}} \gtrsim 0.8 \) is characterized by initial transient oscillations similar to SIS epidemics. We observe for the scale-free network that the time of the first strategy adaption is much smaller than for the closed moment equation. This is caused by the initial faster epidemic spreading observed in these networks.

V. CONCLUSION AND OUTLOOK

In this paper a self adaptive mechanism with a finite set of strategies is proposed, which leads to a coupled system of piecewise deterministic dynamics with history dependent strategy switching. Such a modelling approach will be helpful in the description of systems, where the dynamics depends strongly on the currently active rules, e.g., social systems and opinion formatting, epidemics. It also may be applied to systems, where the full history of the current state changes its evolution, like weaker regeneration of skin cells after multiple exposure with sun light leads to increased probability of cancer.

This adaptive framework is applied to epidemic models on networks. For the SIS system we observe a stable regime of strategy induced oscillations. Their period depends on the network type and the degree distribution. Based on the pairwise closed model an analytic prediction for the period is derived, which fits very well for Erdős-Rényi and random regular networks and also approximates the period observed in scale free networks. We emphasize that the proposed adaptive mechanism deterministically depends on the state of the system and its history. One promising generalization is to consider a stochastic process for the adaptive mechanism, with state (and history) dependent transition rates between the strategies. This adaptive mechanism could be coupled with deterministic ODE models (like the pair-wise closed SIS), or with stochastic models (like the SIS network model). The latter implies two distinct sources of random behavior, which could lead to interesting phenomena.

It would be further interesting to investigate in more detail the observation of the fluctuating reproductive rate around its critical value \( R_0 = 1 \). There is a clear connection of this observation to self-organized criticality, which has been observed in a wide variety of adaptive/coevolutionary network models \[49, 51\]. We conjecture that having an observable controlling a switching mechanism that entails lowering or increasing the infection numbers is the simplest mechanism to obtain self-organized criticality in epidemic dynamics. One could now also investigate power law distributions of epidemic event sizes in various models that could further confirm this conjecture. Yet, for our SIS-based model the underlying bifurcation-theoretic mechanism is the switching around a transcritical bifurcation, where one can calculate mathematically that power laws emerge close to the bifurcation point \[16, 52\]. Hence, finding evidence for self-organized criticality in very complex epidemic models and/or long-time data sets with multiple outbreaks are the most challenging aspects.

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Appendix A: Period of threshold adaption for SIS

In the following we derive an analytic expression for the expected period in the oscillatory regime for the SIS-model with threshold adaption of strategies. For a simplified analysis the pairwise closure, of the system is considered, Eq. \( \text{[3]} \), \( x'_1 = -\gamma x_1 + \tau k(1-x_1)x_1 \). The average node degree takes one of two values, \( k \in \{ k_0, k_- \} \), referring to the strategies \( S_0 \) and \( S_- \), respectively. If in \( S_- \) the probability to cut links is \( p_{\text{cut}} \), then \( k_- = k_0(1-p_{\text{cut}}) \). The ratio of the expected rate of infection to the rate of recovery, \( \beta(k) = \tau k/\gamma \), also takes one of two values \( \beta \in \{ \beta_0, \beta_- \} \) with \( \beta_- = \beta(k_-) = \beta_0(1-p_{\text{cut}}) \).

Solving Eq. \( \text{[3]} \) explicitly for initial condition \( x(t_0) = 0 \) one obtains

\[
x(t) = \frac{(\beta - 1)x_0}{\beta x_0 + (\beta - 1 - \beta x_0)e^{-\gamma(\beta-1)t}},
\]

where \( \beta \) depends on the current strategy and is considered fix between \( t_0 \) and \( t \). The threshold criterion for switching from \( S_0 \) to \( S_- \) is \( J(t_1) = \xi_+ \). First, we consider \( J \) to be the prevalence averaged over the past \( \Delta t \), which leads to

\[
J(t_1) = \xi_+ = \frac{1}{\Delta t} \int_{t_1 - \Delta t}^{t_1} x(t') dt'
\]

with \( \beta = \beta_+ \). This integral is solved using the indefinite integral \( \int \frac{1}{a + be^{-ct}} dt' = \frac{\log(ac + be^{-ct})}{ac} \). With \( a = \beta x_0, b = \beta - 1 - \beta x_0 \) and \( c = \gamma(\beta - 1) \) we obtain

\[
\xi_+ \Delta t = \frac{(\beta - 1)x_0}{\beta x_0(\beta - 1)} \frac{\beta x_0 e^{ct_1} + b}{\beta x_0 e^{c(t_1 - \Delta t)} + b}.
\]
The period depends implicitly on the considered network degree $k$ and the cutting probability $p_{\text{cut}}$, the thresholds $\xi_{\pm}$, and the evaluation time-span $\Delta t$. Taking the limit $\Delta t \to 0$ significantly simplifies these equations. For the prevalence strategy function $J_1$ we trivially get the limits $x_+ = x(\xi_+, k_0) = \xi_+$ and $x_- = x(\xi_-, k_-) = \xi_-$, i.e., the strategy adaption takes place precisely at the threshold levels. Equation (A6) becomes

$$t_s(x_0, \xi) = \frac{1}{\gamma(\beta - 1)} \log \left( \frac{\xi - \beta x_0}{\beta - 1 - \beta x_0} \right) \gamma^\beta \log \left( \frac{\xi - \beta x_0}{\beta - 1 - \beta x_0} \right)$$

(A8)

with $\beta(\gamma, r, k)$. Altogether, the period is calculated as

$$T = \frac{1}{\gamma} \log \left( \frac{\xi_+ (\beta_0 - 1 - \beta_0 \xi_+) \cdots (\beta_0 - 1 - \beta_0 0)}{\beta_0 - 1 - \beta_0 \xi_+} \right) \beta_0 - 1$$

(A9)

Reordering and applying $\beta_- = \beta_0(1 - p_{\text{cut}})$ one gets

$$T = \frac{1}{\gamma} \log \left( \frac{\xi_+}{\xi_-} \right)^{p_{\text{cut}} \beta_0} \left( \frac{\beta_0 - 1 - \beta_0 \xi_+}{\beta_0 - 1 - \beta_0 \xi_-} \right)^{\beta_0 - 1}$$

(A10)

Note that a similar calculation for $J$ being the average incidence over past $\Delta t$ leads to the condition $\xi_+ = \frac{x_{\xi} k}{\beta} \int_{\xi_+}^{\xi_-} x(t') [1 - x(t')] dt'$ which is becomes difficult to solve analytically.

In Fig. 9 we illustrate the period $T$ as a function of $\xi_+$ for different values of $p_{\text{cut}}$ within the periodic regime. For smaller $p_{\text{cut}}$ the system converges towards an endemic state, for larger one to the disease-free state, see Fig. 2. Increasing (a) $\Delta t = 0$ to (b) $\Delta t = 5$ mainly influences the regime with small $\xi_+$. For $\Delta t = 0$ lower and upper boundaries of the periodic regime in terms of $\xi_+$ are given by $\max(\xi_+, x_{\text{en}}(\beta_-))$ and $x_{\text{en}}(\beta_0)$, respectively, indicated by gray dashed lines.

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