Hierarchical coarse-grained approach to the duration-dependent spreading dynamics on complex networks

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Abstract
Various coarse-grained models have been proposed to study the spreading dynamics on complex networks. A microscopic theory is needed to connect the spreading dynamics with individual behaviors. In this letter, we unify the description of different spreading dynamics by decomposing the microscopic dynamics into two basic processes, the aging process and the contact process. A hierarchical duration coarse-grained (DCG) approach is proposed to study the duration-dependent processes. Applied to the epidemic spreading, such formalism is feasible to reproduce different epidemic models, e.g., the SIS and the SIR models, and to associate the macroscopic spreading parameters with the microscopic mechanism. The DCG approach enables us to study the steady state of the duration-dependent SIS model. The current hierarchical formalism can also be used to describe the spreading of information and public opinions, or to model a reliability theory on networks.

1. Introduction
The epidemics [1–6], rumors or information [7–11], and public opinions [12–15], etc., usually spread on complex networks with predefined structures. The spreading dynamics is strongly affected by the characteristic of the structural networks [16, 17]. The utilization of the susceptible-infected-susceptible (SIS) and the susceptible-infected-recovered (SIR) models initiated the study of the epidemic spreading on networks [2–4]. The network structure is much more than the degree distribution. Many network features other than the degree distribution affect the epidemic threshold [17–29], which is an index to determine whether the disease spreads over society. The spreading dynamics is also affected by the microscopic mechanism, namely, the rules of the state change and the transition rates of the basic processes. Currently, a unified spreading model combining both the network structure and microscopic mechanism remains missing. In this Letter, we propose a unified formalism to describe the spreading dynamics on the network with general microscopic mechanism.

For the Markovian spreading models with constant transition rates, serial mean-field theories have been applied to the spreading dynamics with neglecting the correlation between nodes [30–32]. For instance, the epidemic threshold of the standard SIS model on networks was obtained via the heterogeneous-mean-field approach with the degree distribution [2–4, 17], and was later refined via the quenched-mean-field approach by including the details of the network topology [18–22]. For a real-world epidemic, the transmissibility varies in different disease stages [33–44], where the infection rate generally depends on the infection duration. Such non-Markovian property was proposed to dramatically affect the spreading dynamics and alter the epidemic threshold [38–43]. Here we extend the mean-field theories to the duration-dependent spreading models. By introducing the probability density function (PDF) of the duration, we can adopt the time-varying transition rates from the reliability theory [45–47]. In our formalism, the spreading dynamics are decomposed into two basic processes, the aging process and the contact process. The former describes the self-evolution of one node.
(single-body process), and the latter describes the state change of two connected nodes (two-body process). The two processes are modeled here as a continuous-time stochastic process among a set of discrete states.

Inspired by the coarse-grained approaches of the complex networks [48–51], the duration-dependent spreading models are presented at three levels, hierarchically organized as the microscopic, the mesoscopic, and the macroscopic models. In the microscopic model, we derive the basic equations of the PDF of each node with neglecting the correlation between nodes. In the microscopic model, a duration coarse-grained (DCG) approach is proposed to obtain the coarse-grained PDF of the nodes with the same degree. Applied to the epidemic spreading, we obtain a refined spreading rate for the duration-dependent SIS model. The microscopic (mesoscopic) model extends the quenched (heterogeneous) mean-field approaches to the duration-dependent spreading models. The macroscopic model describes the spreading dynamics by assuming the identical PDF of all nodes, and recovers to the compartmental epidemic model [52–54]. The macroscopic model is quantitatively applicable for a homogeneous network with a narrow degree distribution, but gives qualitative predictions about the spreading dynamics.

2. Two basic processes

We consider an undirected network with $N_T$ nodes represented by an adjacency matrix $A_{lm}$. The node state is picked from the state set $i \in \{0, 1, 2, \ldots\}$. The state evolution is governed by two basic processes, the aging process and the contact process, as shown in figures 1(a) and (b), respectively.

The aging process describes the state change $i \xrightarrow{\alpha_{i'}} i'$ of one node. The transition rate $\alpha_{i'}(\tau_i)$ generally relates to its duration $\tau_i$ on the state $i$ [46]. The maximum entropy principle can be used to estimate the most probable transition rate [54, 55], when limited information, e.g., the mean infection time, is known about the process.

The contact process describes the correlated state change $i+j \xrightarrow{\beta_{ij}} i'+j'$ of two connected nodes. The transition rate $\beta_{i'j'}(\tau_i, \tau_j)$ relates to the duration $\tau_i$ and $\tau_j$ of the two nodes in the states $i$ and $j$. Different patterns exist for the contact process, e.g., the exchange process $i+j \xrightarrow{\beta_{ij}} j+i$ and the infection process $i+j \xrightarrow{\beta_{ij}} j+j$.

Most spreading models can be constructed with the above two processes. For example, the state 0 (1) is the susceptible (infected) state in the SIS model. The basic processes are an aging process $1 \xrightarrow{\alpha_{1'}} 0$ with the recovery rate $\alpha_{1'}$, and a contact process $0+1 \xrightarrow{\beta(\tau_0, \tau_1)} 1+1$ with the infection rate $\beta(\tau_0, \tau_1)$. With different duration on the susceptible (infected) state, the vulnerability (transmissibility) of the node changes, and is reflected by the duration-dependent infection rate $\beta(\tau_0, \tau_1)$. A typical evolution of one node is shown in figure 1(c). At the initial time $t = t_0$, the node stays in the state 0 with $\tau = 0$. At the time $t = t_1$ ($t = t_2$),
The conventional spreading models [2, 17] with only recording the node states are infeasible to describe the spreading, a typical model [8, 9] is constructed with three states 0, 1 and 2 representing ignorant, spreading, and stifling states. The basic processes are $0 \rightarrow 1 + 1, 1 \rightarrow 1 + 1$, and $1 \rightarrow 2 + 1$, and $1 \rightarrow 2 + 2$. The transition rates generally depend on the duration, but this effect was rarely considered in the current studies.

3. Duration-dependent spreading models

The conventional spreading models [2, 17] with only recording the node states are infeasible to describe the spreading dynamics with the duration-dependent transition rates. In figure 2, we introduce the PDF $\rho_{l,i}(\tau_i, t)$ of the duration for the node $l$ in the microscopic model. The probability of the node $l$ in the state $i$ follows as 

$$
\frac{\partial p_{l,i}(\tau_i, t)}{\partial \tau_i} + \frac{\partial p_{l,i}(\tau_i, t)}{\partial t} = -\Gamma_{l,i}(\tau_i, t) p_{l,i}(\tau_i, t).
$$

(1)

The total transformation rate for the node $l$ of leaving the state $i$ is $\Gamma_{l,i}(\tau_i, t) = \sum_{j} \gamma_{l,i,j}(\tau_i, t)$, with the transformation rate $\gamma_{l,i,j}(\tau_i, t)$ from the state $i$ to the state $j$ explicitly as

$$
\gamma_{l,i,j}(\tau_i, t) = \alpha_{l,j} + \sum_{m,j} A_{lm} \int_{0}^{\infty} \beta_{l,j,m} \rho_{l,m}(\tau_m, t) d\tau_m.
$$

(2)

The connecting condition for the PDF at the boundary $\tau_i = 0$ is determined by the flux to the state $i$ as $\rho_{l,i}(0, t) = \Phi_{l,i}(t) = \sum_{j} \phi_{l,j}(t)$, where $\phi_{l,j}(t) = \int_{0}^{\infty} \gamma_{l,j}(\tau_i, t) \rho_{l,j}(\tau_i, t) d\tau_i$ is the probability of the node $l$ transforming from the state $j$ to the state $i$ in unit time.

To effectively describe the spreading dynamics, we propose a DCG approach to study the duration-dependent effect. In the mesoscopic model, the nodes are sorted according to the degree $k$, as shown in figure 2. The states of the network are described by the coarse-grained PDF $\rho_{k,i}(\tau_i, t) = \sum_{l} a_{k,i} \rho_{l,i}(\tau_i, t) / n_k$ of the $k$-degree nodes, where $n_k$ is the number of the $k$-degree nodes. The population of the $k$-degree nodes in the state $i$ follows as $n_k i(t) = n_k \int_{0}^{\infty} \rho_{k,i}(\tau_i, t) d\tau_i$. The PDFs of the nodes with the same degree are assumed identical $\rho_{l,i}(\tau_i, t) = \rho_{k,i}(\tau_i, t)$. The equation of the coarse-grained PDF is obtained from equation (1) as

$$
\frac{\partial \rho_{k,i}(\tau_i, t)}{\partial \tau_i} + \frac{\partial \rho_{k,i}(\tau_i, t)}{\partial t} = -\Gamma_{k,i}(\tau_i, t) \rho_{k,i}(\tau_i, t).
$$

(3)
The total transformation rate is \( \Gamma_i(t, t) \), giving the rate of change of the process from one state to another, with transformation rates \( \Gamma_k f_i(t, t) \) with the transformation rate [56]

\[
\gamma_{k, f_i} = \alpha_{f_j} + k \sum_{j, f, k} P(k')P(k) \int_0^\infty \beta_{f_j, f_i} \rho_{k, i} \mathrm{d}\tau.
\]

(4)

The degree correlation \( P(k') | k \) is determined by the adjacency matrix \( A_{km} \) as \( P(k') | k = \sum_{m, f} \delta_{k, k} \delta_{l, k} A_{km} / (kt) \) [56]. The connecting condition for the coarse-grained PDF is \( \rho_{ki}(t) = \Phi_{f_i}(t) = \sum_{f} \phi_{k, f}(t) \), where \( \phi_{k, f}(t) = \int_0^\infty \gamma_{k, f_i}(t, t) \rho_{k, f}(t, t) \mathrm{d}\tau \) is the flux of one k-degree node transforming from the state \( i \) to the state \( i \). An example with explicit equations of PDFs in the duration-dependent SIS model can be found in supplementary materials (https://stacks.iop.org/JPCOMPLEX/2/02LT01/mmedia) [56] or in reference [40].

At the macroscopic level, a further coarse-grained procedure introduces the gross PDF \( \rho_i(t, t) = \rho_{k, f}(t, t) / N_T \), of all nodes, as shown in figure 2. The dynamics is then regarded to be homogeneous for all nodes. The population of the nodes in the state \( i \) follows as \( N_i(t) = N_T \int_0^\infty \rho_i(t, t) \mathrm{d}\tau \). This approximation is suitable for the homogeneous network with similar degrees for all nodes. The equation of the gross PDF is obtained from equation (3) as

\[
\frac{\partial \rho_i(t, t)}{\partial \tau} + \frac{\partial \rho_i(t, t)}{\partial t} = -\Gamma_i(t, t) \rho_i(t, t).
\]

(5)

The total transformation rate is \( \Gamma_i(t, t) = \sum_{j, f} \gamma_{f_i, f_j}(t, t) \) with the transformation rate

\[
\gamma_{f_i, f_j} = \alpha_{f_j} + \langle k \rangle \sum_{j, f} \int_0^\infty \beta_{f_j, f_i} \rho_{i, f} \mathrm{d}\tau.
\]

(6)

The effect of the network structure on the spreading dynamics is reflected by the average degree \( \langle k \rangle = \sum_{k=1}^\infty kP(k) \). For the gross PDF, the connecting condition is \( \rho_i(0, t) = \Phi_i(t) = \sum_{f} \phi_{f_i, f}(t) \) with the gross flux \( \phi_{f_i, f}(t) = \int_0^\infty \gamma_{f_i, f}(t, t) \rho_i(f, t) \mathrm{d}\tau \). Details of the coarse-grained procedures are shown in the supplementary materials [56].

With different interpretations of states and nodes, the proposed spreading models can be widely used in different problems. For example, the node states describe disease of individuals in an epidemic model [2], or performance of components in a reliability model [47]. The transformation rates and the connecting conditions are given accordingly from the specific microscopic mechanism. For the constant transition rates, our models retain the conventional models describing the spreading dynamics with the probabilities \( P_{i, f}(t) \) or the populations \( n_{i, f}(t) \) and \( N_i(t) \). The detailed derivation is given in the supplementary materials [56].

As follows, the proposed spreading models are applied to the epidemic spreading. In table 1, we list the dictionary for constructing the duration-dependent SIS and SIR models with the transformation rates, the fluxes and the connecting conditions. The two models share the same partial differential equations, but have different coupling forms of the connecting conditions.

| Node states | 0, 1 | 0, 1, 2 |
|-------------|------|---------|
| Rules       | \( \frac{\alpha(n)}{n} \) = 0 | \( \frac{\alpha(n)}{n} \) = 2 |
| Transformation rates | \( \Gamma_{ki}(t, t) = \Gamma_{f_k f_i}(t, t) \) | \( \Gamma_{ki}(t, t) = \Gamma_{f_k f_i}(t, t) \) |
| Fluxes      | \( \Phi_{f_i}(t) = \int_0^\infty \alpha_{f_i} \rho_{f_i}(t, t) \mathrm{d}\tau \) | \( \Phi_{f_i}(t) = \int_0^\infty \alpha_{f_i} \rho_{f_i}(t, t) \mathrm{d}\tau \) |
| Connecting conditions | \( \rho_i(0, t) = \Phi_{f_i}(t) \) | \( \rho_i(0, t) = \Phi_{f_i}(t) \) |

Table 1. The dictionary for constructing the duration-dependent SIS and SIR model.

With the constant recovery and infection rate, the macroscopic model recovers to the standard compartmental epidemic model [52, 53, 57]. The susceptible and the infected populations satisfy \( N_0(t) = \alpha N_i(t) - \beta (k) N_0(t) N_i(t) / N_T \) and \( N_i(t) = N_T - N_0(t) \). In reference [55], the effect of the duration-dependent recovery rate \( \alpha(n) \) has been studied in an extended compartmental model with the integro-differential equations. In supplementary materials [56], we derive both the standard and the extended compartmental model from the macroscopic model.
4. SIS model in a network

As an example, we study the spreading dynamics of the duration-dependent SIS model on an uncorrelated network at the mesoscopic level. The degree correlation of an uncorrelated network is simplified as $P(k'|k) = k' P(k') / \langle k' \rangle$ [17]. The DCG approach enables us to obtain the steady state with arbitrary duration-dependent recovery and infection rates by solving a self-consistent equation.

In the duration-dependent SIS model, the DDFs $\rho_{k,0}(\tau_0, t)$ and $\rho_{k,1}(\tau_1, t)$ obey equation (3). The transformation rates and the connecting conditions are listed in Table 1. The epidemic spreading is typically assessed by the fraction $r_1(t) = \sum_{k=1}^{\infty} n_{k,1}(t) / \langle n_k \rangle$ of the infected nodes. For the infection rate $\beta(\tau_0, \tau_1)$, its dependence on the susceptible (infection) duration describes the vulnerability (transmissibility) of a susceptible (infected) node. For simplicity, we assume the vulnerability does not depend on the susceptible duration [58]. Thus, the spreading dynamics is independent of the susceptible duration $\tau_0$, with the infection rate reduced to $\beta(\tau_0, \tau_1) = \beta(\tau_1)$.

On the uncorrelated network, the transformation rate of the contact process is simplified as $\Gamma_{k,0}(t) = k \Theta(t)$ with

$$\Theta(t) = \sum_{k=1}^{\infty} k P(k) \int_0^\infty \beta(\tau_1) \rho_{k,1}(\tau_1, t) d\tau_1. \quad (7)$$

We focus on the steady state $\partial \rho_{k,1}(\tau_1, t) / \partial t = 0$. The DDFs of the steady state are solved from equation (3) as

$$\rho_{k,0}(\tau_0) = \Phi_1 \exp[-k \Theta \tau_0], \quad (8)$$

and

$$\rho_{k,1}(\tau_1) = \Phi_2 \exp\left[-\int_0^{\tau_1} \alpha(\tau) d\tau\right]. \quad (9)$$

where $\Phi_1 = n_k k \Theta / (1 + k \Theta \bar{\tau}_1)$ is the steady-state flux, and $\bar{\tau}_1 = \int_0^\infty \exp[-\int_0^{\tau_1} \alpha(\tau) d\tau] d\tau_1$ is the average infection duration, i.e., the average time to recover from the disease. It follows from equation (7) that

$$\Theta = \frac{\Upsilon \Theta}{\langle k \rangle} \sum_{k=1}^{\infty} \frac{k^2 P(k)}{1 + k \Theta \bar{\tau}_1}, \quad (10)$$

which is the self-consistent equation to determine the quantity $\Theta$ of the steady state. Here, $\Upsilon$ is the refined spreading rate for the duration-dependent SIS model, explicitly as

$$\Upsilon = \int_0^\infty \beta(\tau_1) \exp\left[-\int_0^{\tau_1} \alpha(\tau) d\tau\right] d\tau_1. \quad (11)$$

The steady-state fraction of the infected nodes is

$$r_1 = \sum_{k=1}^{\infty} \frac{k \Theta \bar{\tau}_1}{1 + k \Theta \bar{\tau}_1} P(k), \quad (12)$$

which is determined by the refined spreading rate $\Upsilon$ via the quantity $\Theta$ and the average infection duration $\bar{\tau}_1$ [59]. The effect of network structure is explicitly reflected via the degree distribution $P(k)$. For the constant recovery and infection rates, the refined spreading rate $\Upsilon$ returns to the effective spreading rate $\Upsilon = \beta / \alpha$ used in the duration-independent SIS model [2].

The existence of the non-zero solution $\Theta$ requires the refined spreading rate $\Upsilon$ to exceed a critical value $\Upsilon_c = \langle k \rangle / \langle k^2 \rangle$, which is defined as the epidemic threshold solely determined by the network structure. When the refined spreading rate exceeds the epidemic threshold $\Upsilon > \Upsilon_c$, the system reaches the epidemic steady state with existing infected nodes. At the situation $\Upsilon < \Upsilon_c$, the system reaches the disease-free steady state. A necessary condition to ensure a disease-free steady state is $\langle k \rangle / \Upsilon < 1 / \bar{\tau}_1$, which implies the contacts of people need to be controlled according to the spreading ability of the epidemic.

To validate the current coarse-grained model, we simulate the duration-dependent SIS model on an uncorrelated scale-free network with the continuous-time Monte Carlo method [23, 60]. Details of the simulation are illustrated in supplementary materials [56]. The uncorrelated scale-free network with $N_T = 2500$ is generated via the configuration model [61]. The degree sequence $\{k_i\}$ is generated according to the degree distribution $P(k) = c/k^3$, where $k$ ranges from the minimal degree $k_{\text{min}} = 10$ to the maximal degree $k_{\text{max}} = 50$ with the normalized constant $c = 1/\sum_{k=k_{\text{min}}}^{k_{\text{max}}} 1/k^3$ of the degree distribution. The minimal degree $k_{\text{min}}$ is chosen not so small to avoid large fluctuations of the infected neighbors for low-degree nodes, since the mean-field approach assumes the static PDF for the steady state. The maximal degree $k_{\text{max}}$ fulfills the condition $k_{\text{max}} \leq \sqrt{N_T}$ to
ensure an uncorrelated network [61]. All nodes are randomly connected respecting the assigned degrees without multiple and self-connection.

We carry out the simulation with the Weibull distribution of the recovery and the infection time $\psi_R(\tau_I) = a_{\alpha}/b_{\alpha}(\tau_I/b_{\alpha})^{a_{\alpha}-1} \exp[-(\tau_I/b_{\alpha})^{a_{\alpha}}]$ and $\psi_T(T_I) = a_{\beta}/b_{\beta}(T_I/b_{\beta})^{a_{\beta}-1} \exp[-(T_I/b_{\beta})^{a_{\beta}}]$. The corresponding transition rates are $\alpha(\tau) = a_{\alpha}/b_{\alpha}(\tau/b_{\alpha})^{a_{\alpha}-1}$ and $\beta(\tau) = a_{\beta}/b_{\beta}(\tau/b_{\beta})^{a_{\beta}-1}$. In each simulation, the evolution is first run for 500,000 events to ensure reaching the steady state. Then, the steady-state fraction $r_1$ is obtained as the average with the later 200,000 events.

In figure 3, the steady-state fraction $r_1$ of the infected nodes is plotted as the function of the refined spreading rate $\Upsilon$ for the DCG approach (solid curve) and the continuous-time Monte Carlo simulation results (dots). In the simulation, the effects of duration-dependent recovery and the infection rates are considered by choosing different sets of parameters. The agreement between the analytical and the simulation results validates that the steady-state fraction $r_1$ can be effectively obtained by equation (11). The existence of the epidemic threshold matches with the theoretical prediction $\Upsilon_c = (k)/\langle k^2 \rangle = 0.051$ (gray grid-line). The current model shows the availability of the refined spreading rate $\Upsilon$ for justifying the spreading ability of an epidemic.

5. Conclusion

In this letter, we generalize the mean-field theories for the spreading dynamics with duration-dependent mechanism by superseding the probability distribution of states with the PDF of the duration, and show the hierarchical emergence of the widely-used coarse-grained spreading models. The unified formalism enables us to rebuild different epidemic models, e.g., the SIS and the SIR model.

Previous studies, for example references [38, 39, 42], studied the non-Markovian property via introducing non-exponential distributions of the recovery and the infection time. Regarded as renewal processes, the recovery (infection) time in each event satisfies an independently identical distribution. In our formalism, the non-Markovian property is understood as the duration dependence of the infection and the recovery rates. For the duration-dependent SIS model, we obtain the refined spreading rate $\Upsilon$ as a coarse-grained parameter of the microscopic mechanism details. This suggests the duration-dependent SIS model can be mapped to the standard one at least in the meaning of the steady states [44]. With the refined spreading rate $\Upsilon$, the epidemic threshold $\Upsilon_c = (k)/\langle k^2 \rangle$ is still applicable for the duration-dependent SIS model, and determine the fate of the epidemic spreading.

Limited by the mean-field approach, the current formalism has neglected correlations and fluctuations between nodes, and therefore cannot accurately predict the critical point of the epidemic phase transition, i.e., the epidemic threshold. In the standard SIS model, the correlations and fluctuations affect the epidemic threshold through the mutual reinfection of the high-degree nodes [24–26, 29], and was recently understood through the cumulative merging percolation process [62]. It is still an open question to describe such correlation effect in a duration-dependent model, which is beyond the scope of the current work and worth for further investigation.
Data availability statement
The data that support the findings of this study are available upon reasonable request from the authors.

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