

Topological resilience in non-normal networked systems

Malbor Asllani¹ and Timoteo Carletti¹

¹Department of Mathematics & naXys, Namur Institute for Complex Systems
University of Namur, Rue Rempart de la Vierge, 8, B-5000 Namur - Belgium
timoteo.carletti@unamur.be

Abstract

The network of interactions in complex systems, strongly influences their resilience, the system capability to resist to external perturbations or structural damages and to promptly recover thereafter. Understanding the topological features of the networks that affect the resilience phenomenon remains a challenging goal for the design of robust complex systems. We hereby introduce the concept of non-normal networks, namely networks whose adjacency matrices are non-normal and we show that such feature can drastically change the global dynamics through an amplification of the system response to exogenous disturbances and eventually impact the system resilience. This early stage transient period can induce the formation of inhomogeneous patterns, even in systems involving a single diffusing agent, providing thus a new kind of dynamical instabilities complementary to the Turing one. We provide an illustrative application of this result to ecology by proposing a mechanism to mute the Allee effect.

Introduction

The ecological resilience (Gunderson, 2000) is the ability of ecosystems to respond to challenges such as fires, windstorms, deforestation, flooding or the presence of invasive species, and their aptitude to return close to the initial state. This general idea has been rapidly extended beyond the initial framework, e.g. the impact of climate changes on the natural habitats (Bachelet et al., 2001) and the responsibility of the human activity on environmental disasters (Adger et al., 2005). Resilience is also encountered in human-made systems such as power grids or communications systems where a failure of a component of interconnected elements can trigger a cascade of failures of successive components (Motter and Lai, 2002). In this case, the response of the system is directly correlated to the structural changes in the networked support where the dynamics occurs (Gao et al., 2016). Efforts have been made to understand how complex interactions influence the systems resilience (Buldirev et al., 2010) in order to optimise the design that enhances their robustness and reduce their vulnerability (Asha and Newth, 2007).

Our goal is to show that the resilience of networked dynamical systems is highly determined by the degree of non-

normality characterising the underlying complex network, more precisely we define a network to be non-normal if its adjacency matrix does (Asllani and Carletti, 2018). This technical definition (Trefethen and Embree, 2005), based on the non-existence of a unitary matrix which diagonalises it, will be proved to determine an unexpected system response to small disturbances; this abnormal behaviour follows a transient amplification process during the initial linear regime which, if sufficiently large, subsequently leads the system to another state. The latter being possibly characterised by spatial inhomogeneities and potentially far from the initial one, reducing thus the system resilience.

The non-normality has been previously considered in different domains e.g. hydrodynamic stability (Trefethen et al., 1993), non-Hermitian quantum-mechanics (Hatano and Nelson, 1996), synchronisation of networked optoelectronic devices (Ravoori et al., 2011), ecology (Neubert and Caswell, 1997), population dynamics (Neubert et al., 2002; Ridolfi et al., 2011) or taking into account the stochastic dynamics (Biancalani et al., 2017). Let us stress that the proposed framework is different from the latter ones. Indeed the effect of the non-normality, which enters through the network structure, allows us to potentially consider applications to complex systems where the geometry of the spatial interactions play a crucial role. The goal of this paper is thus to bring to the fore a general framework, to accommodate for a better understanding of the impact of the non-normality assumption on the resilience of networked systems.

As previously stated, the transient amplification due to the network non-normality can push the system into a new state, possibly far from the initial one, and usually exhibiting spatial inhomogeneities (patterns). For this reason the proposed mechanism (see panel d of Fig. 3 (Asllani and Carletti, 2018)) could be an alternative pathway to the emergence of spatially self-organised heterogeneous patterns, complementary to the one introduced by Turing, for which a minimal system of two species, activator-inhibitor, is needed to generate complex patterns following a diffusion-induced instability (Turing, 1952; Murray, 2001). Remarkably enough, we can assume symmetric reaction terms and even equal dif-

fusion coefficients for both species, and still obtain patterns. In particular a single inhibitor species allowed to freely diffuse on a non-normal network, can experience a patchy solution because of the transient instability induced by the non-normal topology.

Results

The Allee effect (Allee et al., 1949) can explain the community cooperation or facilitation phenomena in natural ecosystems; it is often introduced by modifying some generic model of population growth, e.g. the logistic equation Murray (2001) (see Fig. 3 (Asllani and Carletti, 2018) panel (a)). To go one step further let us assume the population of a single species to live in a patchy environment where animals can move across the niches; the dynamics can thus be described by the following diffusively coupled equations:

$$\frac{dx_i}{dt} = rx_i(1 - x_i) \left(\frac{x_i}{A} - 1 \right) + D \sum_{j=1}^M L_{ij} x_j, \forall i, \quad (1)$$

where x_i denotes the species density in the i -th patch, r the reproduction rate, A the Allee coefficient and D the diffusion coefficient, all assumed for simplicity to be the same for all patches. Given the network adjacency matrix $A_{ij} = 1$ if the node j is directly connected to node i and zero otherwise, we define the out-degree of node i as $k_i^{out} = \sum_j A_{ij}$, namely the number of outgoing links from node i , and the (network) Laplacian matrix, $L_{ij} = A_{ij} - k_i^{out} \delta_{ij}$.

If species x diffuses using a symmetric network and the initial conditions do not exceed the Allee threshold, $x_i(0) < A \forall i$, then the species goes extinct and diffusion cannot prevent it. Conversely if the underlying network is non-normal, the system fate turns upside down and the population will survive reducing thus the system resilience. Indeed it can happen that the transient amplification induced by the non-normality is strong enough to surpass the Allee threshold, at least in some of the patches, and consequently the system saturates avoiding the extinction. The non-normal spatial support makes the stable undesired equilibrium $x_1^* = 0$ less robust against perturbations and thus a larger set of initial conditions close to the latter can in fact escape and end up in a new survival state \mathbf{x}^* , close to the second stable equilibrium $x_2^* = 1$ (see panels (b) and (c) Fig. 3 (Asllani and Carletti, 2018) where β denotes the size of the perturbation with respect to the trivial equilibrium, i.e. the size of the initial condition).

Conclusions

The previously result holds generically for non-normal networked dynamical systems. This topological feature of the network of interactions will force the inhibitors in some of the nodes to initially increase their concentration until they saturate in the non-linear phase. What is remarkable is that the species which apparently tends to go extinct because of the negative growth rate, exploit a faster diffusion process that makes the species to spread before the individuals counteract, and eventually lead to self-organisation. This is thus

a new mechanism, different from the Turing one, capable to explain the pattern formation process.

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