2D pattern evolution constrained by complex network dynamics

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Abstract. Complex networks have established themselves in recent years as being particularly suitable and flexible for representing and modelling several complex natural and artificial systems. In the same time in which the structural intricacies of such networks are being revealed and understood, efforts have also been directed at investigating how such connectivity properties define and constrain the dynamics of systems unfolding on such structures. However, less attention has been focused on hybrid systems, i.e. involving more than one type of network and/or dynamics. Several real systems present such an organization, e.g. the dynamics of a disease coexisting with the dynamics of the immune system. The current paper investigates a specific system involving diffusive (linear and nonlinear) dynamics taking place in a regular network while interacting with a complex network of defensive agents following Erdös–Rényi (ER) and Barabási–Albert (BA) graph models with moveable nodes. More specifically, the complex network is expected to control, and if possible, to extinguish the diffusion of some given unwanted process (e.g. fire, oil spilling, pest dissemination, and virus or bacteria reproduction during an infection). Two types of pattern evolution are considered: Fick and Gray–Scott. The nodes of the defensive network then interact with the diffusing patterns and communicate between themselves in order to control the diffusion. The main findings include the identification of higher efficiency for the BA control networks and the presence of relapses in the case of the ER model.

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1. Introduction

Complex systems have continuously motivated intense scientific research. In recent decades, much attention has been focused on systems involving strongly interacting agents. More recently, tools provided by the theory of complex networks have been successfully applied in order to characterize the structure of many of such systems [1]–[4]. Once the system of interest is properly translated into a network, its structural properties [1], [3]–[6] can be calculated and used to characterize and analyse the system as well as dynamical processes being underlined by the network [1, 4], [7]–[10]. However, many dynamics are often related to processes taking place outside the network, possibly also over some network (the same or a different network). Such systems have received little attention from the complex network community.

The current paper investigates the evolution of dynamical systems underlined by two distinct (but coexisting) networks. The first system (which represents a defensive system), involving a complex network of Erdös–Rényi (ER) [11] or Barabási–Albert (BA) [12] type, senses and interacts with the other system, here represented by a regular network over which linear (Fick [13]) and nonlinear (Gray–Scott [14]) pattern formation (henceforth called disease) is allowed to evolve. The Fick diffusion model provides a linear, homogeneous and isotropic flow of mass from a fixed and infinite source. The Gray–Scott reaction–diffusion dynamics produces non-static, growing patterns without well-defined sources. Examples of such complex systems include forest fires, where the nodes of the complex networks represent firemen, organized into communicating groups, trying to stop the spreading of the fire, represented by a diffusive process in the regular network. Other similar situations include oil spills (oil diffusing along the regular network, while a complex network of cleaners try to control the process) and the evolution of a disease along healthy tissue, with the nodes representing the defensive cells trying to self-organize in order to control and stop the disease. Observe that the connections established by the agents of the system are not necessarily physical. In fact, these connections may correspond to wireless communication, bio-chemical signalling or even intermediate agents (as modelled in bi-partite graphs), e.g. enzymes in biological networks.

The current paper starts by presenting the pattern formation models (Fick and Gray–Scott) and proceeds by describing the interaction between the two involved networks (i.e. complex and...
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The results and discussion follow, and the paper is concluded by emphasizing the main contributions and perspectives for future developments.

2. Diffusion models

Over the twentieth century, a number of natural phenomena were modelled by diffusion and pattern formation processes. The former type of dynamics includes the established topics of atom and molecule diffusion [15] as well as heat diffusion through different materials [16]. In addition, econometricians have developed diffusion models to forecast the acceptance of new products and to understand their life-cycle [17]. Migration of animals and spreading of organisms and chemical substances are often investigated in terms of biological diffusion models [18]. More recently, complex biological and chemical patterns have been reproduced by systems of equations with diffusive and reactive terms [19]. These models range from simple diffusion equations (e.g. heat diffusion in a rod) to more sophisticated advection–diffusion (e.g. chemical oceanography) and reaction–diffusion equations (e.g. chemical and biological patterns). Two such models are considered in the present paper in order to represent a reasonable range of natural and artificial phenomena: Fick diffusion and the Gray–Scott reaction–diffusion models.

The Fick diffusion model of an entity \( U \) (1) can be derived from the continuity equation [13]. The concentration \( u \) of \( U \) evolves in time proportionally to the difference between the average value of \( u \) around a given point and the value of \( u \) at that point. The proportionality constant is given by the diffusion coefficient \( D_u \).

\[
\frac{\partial u}{\partial t} = D_u \nabla^2 u.
\]

The Gray–Scott model includes the following two irreversible reactions [14]:

\[
U + 2V \rightarrow 3V, \quad V \rightarrow P,
\]

where \( U \) and \( V \) are two reacting specimens and \( P \) an inert precipitate. Considering the concentrations of specimens \( U \) and \( V \), respectively, as \( u \) and \( v \), these reactions can be expressed by the following pair of nonlinear partial differential equations (3) with diffusive and reactive terms:

\[
\frac{\partial u}{\partial t} = D_u \nabla^2 u - uv^2 + f(1-u), \quad \frac{\partial v}{\partial t} = D_v \nabla^2 v + uv^2 - (f + k)v,
\]

where \( D_u \) and \( D_v \) are the diffusion coefficients. The dimensionless feed rate of the first reaction is \( f \); \( k \) is the dimensionless rate constant of the second reaction.

3. Diffusion and defence dynamics

Both diffusion models were evaluated on a spatial mesh (i.e. a regular network) of 256 by 256 points with periodic boundary conditions. The system size was 3.0 in both directions. Numerical integrations were carried out by the forward Euler method of the finite-difference equations resulting from discretization of the diffusion operator. The time step was 1 time unit.
Two configurations of initial conditions for the Fick diffusion model: (a) one-source and (b) two-sources. Similar initial conditions were used for the Gray–Scott model, except for the source value.

diffusion coefficients were set as $D_u = 0.00002$ (to both diffusion models) and $D_v = 0.00001$. A complex network was used to represent the interaction between agents (i.e. nodes) susceptible to being activated by the regular network. There were two states associated with each node: susceptible or activated. All the nodes began in the susceptible state. As soon as the disease overcomes a threshold at the node spatial position $(x, y)$, or if the node is requested to help its neighbours, the node is switched to the activated state. If a node is requested simultaneously as a consequence of high activity in the regular network as well as by one of its neighbours in the complex network, priority is given to the former request. After a while, the node returns to the susceptible state.

Two configurations of initial conditions were investigated. In the first configuration (figure 1(a)), the entire system was placed in the uninfected state: $u_{x,y} = 0$ (Fick model), and $u_{x,y} = 1$ and $v_{x,y} = 0$ (Gray–Scott model). The source of the disease, an 11 by 11 square of mesh points, was centred in the middle of the board and set as $u_{x,y} = 1$ (Fick model), and $u_{x,y} = 0.5$ and $v_{x,y} = 0.25$ (Gray–Scott model). In the latter model, the source was perturbed by adding random values of $\pm 0.01$, in order to break the square symmetry. The nodes were randomly distributed inside a rectangular area (one third of the board area, with 256 by 85 points) on the left side of the mesh, at 38 mesh points away from the disease source. This simple arrangement was chosen to create a ‘wall’ of nodes and contributed to the vertical symmetry of the configuration, reducing the number of parameters to be considered during simulation.

In the second configuration (figure 1(b)), the nodes were distributed inside the same rectangular region as before, but the area was centred in the middle of the mesh. The source was broken into two (11 by 6 rectangular mesh points each piece), in order to correspond to about the same amount of initial disease. Both sources were symmetrically placed at the same distance (i.e. 38 mesh points) and opposite sides from the nodes ‘wall’. This assembly induced a competition for neighbours of activated nodes.

In the Fick model, a node became activated when the disease overcame a threshold $T_{u_{x,y}} = 0.4$ at the respective node position, i.e. $x$ and $y$. In the Gray–Scott model, the disease must fall below a threshold $T_{u_{x,y}} = 0.6$ in order to activate the node. Remember that absence of disease was represented by $u_{x,y} = 0$ in the Fick model and by $u_{x,y} = 1$ in the Gray–Scott model. As soon as a node has been activated, all its topological neighbours are requested to help (figure 2). The engaged neighbours were randomly distributed at distance $R = 5$ from the...
Figure 2. Diagram illustrating a sub-network (a) before and (b) after the node activation. The dark-grey node was spatially activated by the disease and the four light-grey nodes were the topologically activated helpers.

activated node. In order to avoid overlapping in the liberation of antidote, a circular area of influence (with radius $R_i = 5$) was defined around every node, so that no other activated node was included within this area. In fact, we guaranteed a minimum distance ($R = R_i = 5$) between any two activated nodes while ensuring a compact distribution of the nodes. Once this circle was filled, the remaining nodes were assembled at double the initial radius (for example, the node with a star in figure 2), and so on. The antidote liberation consisted of keeping for 50 time units an opposite and negative Fick diffusion from all activated nodes with $D_a = 0.00003$, and fixed intensity $a_{x,y} = -1$ (Fick model) and $a_{x,y} = -10$ (Gray–Scott model). The higher intensity was necessary in the latter model because of the fast moving characteristic of this reaction–diffusion. The resulting pattern intensity $u^{t+1}_{x,y}$ was given by the positive values obtained from the addition of the disease intensity $u^t_{x,y}$ and the antidote intensity $a^t_{x,y}$ (4). Similarly, the new antidote intensity $a^{t+1}_{x,y}$ was given by the negative values obtained from the same equation (4).

\[
\begin{align*}
    &u^{t+1}_{x,y} = u^t_{x,y} + a^t_{x,y} & \text{and} & a^{t+1}_{x,y} = 0 & \text{if} & u^t_{x,y} + a^t_{x,y} \geq 0, \\
    &a^{t+1}_{x,y} = u^t_{x,y} + a^t_{x,y} & \text{and} & u^{t+1}_{x,y} = 0 & \text{if} & u^t_{x,y} + a^t_{x,y} \leq 0.
\end{align*}
\]

Observe that the activation time is calculated so as to liberate enough antidote within the circular area of influence of the node, reducing the overlap between different nodes. Afterwards, the node ceased its activity and returned to the susceptible state. If two nodes requested help from the same neighbour, the latter chose one of them with equal probability.

4. Results

The suggested dynamics involving the interaction between two networks always resulted in competition between the disease and the antidote, where the winner was ultimately defined by the values chosen for the diffusion and defence parameters. Some parameter configurations have been observed to lead to a situation where a great part of the effort to control the disease was
Figure 3. Snapshots of the pattern for the four cases: (a) Fick diffusion and ER network, (b) Fick diffusion and BA network, (c) Gray–Scott reaction–diffusion and ER network and (d) Gray–Scott reaction–diffusion and BA network. Red represents maximum disease intensity and cyan no disease. The nodes are represented by the black dots. Both networks have 300 nodes and $\langle k \rangle \approx 4$.

wasted. On the other hand, it was possible to find parameter configurations where the defence always succeed, i.e the disease vanished. Once such parameters were identified and adopted, we compared the role of the network structure in the proposed defence dynamics.

4.1. Pattern evolution

Figure 3 presents snapshots of the evolution of the disease for four cases assuming the one-source configuration. A set of movies with all the configurations discussed in this paper is also available. The first two rows show the evolution of the Fick diffusion controlled by (a) ER and (b) BA defensive networks, both with 300 nodes and $\langle k \rangle \approx 4$. The activation of the first node only
took place after a relatively long period of time, about 8200 time units before the first snapshot in the ER case and about 3600 time units in the BA case. Although these early activations had little effect on disease control, they were important because they brought some nodes closer to the source of the disease. As soon as some nodes were close enough to the source, they were activated, triggering a chain activation effect (first snapshot in figure 3(a)). The latter effect occurred because some of the activated nodes fell at positions where the disease had already overcome the threshold (second snapshot in figure 3(a)). Hence, the spatial activation of the nodes resulted in requests to their own neighbours and so on. The hub activation in the BA case (first snapshot in figure 3(b)) requested many nodes at once and, consequently, some of them fell very near the source. As a result, their own neighbours, i.e. the neighbours of the hub’s neighbours, were activated, consequently populating the area around the source and enclosing it with a considerable amount of antidote (second snapshot in figure 3(b)). The ER network nodes took a longer time to achieve control of the source (third snapshot in figure 3(a)). After this stage of the chain reaction, the source became enclosed and the nodes kept on changing their states. Each new spatial activation redistributed the helpers around the source and even requested nodes which had never been activated before. The activation of the hubs implied the fastest decrease in the total quantity of the disease considering the BA network (fourth snapshot in figure 3(b)). In the last snapshot considered, the mesh was found to be more free of disease in the BA case (fifth snapshot in figure 3(b)), while a substantially more infected configuration was observed in the ER cases (fifth snapshot in figure 3(a)). After very long times, the nodes ceased to converge around the source.

The chosen configuration of the Gray–Scott reaction–diffusion ($f = 0.04$ and $k = 0.064$) generated non-static patterns whose spots and stripes tended to quickly reach the complex network. Figures 3(c) and 3(d), represent the reaction–diffusion evolution constrained by the ER and BA defensive networks, respectively, with 300 nodes and $\langle k \rangle \approx 4$ each. After the first node activation (first snapshot in figures 3(c), and (d)), a chain reaction was triggered as in the Fick diffusion model. The nodes were activated from the centre to the boundary of each case (second snapshot in figures 3(c), and (d)), a natural consequence of the dynamics rules. Once again, the hub-based characteristic of the BA network resulted in massive attack against the disease. This type of attack can be identified by the great amount of eliminated disease in the reaction-diffusion constrained by the BA network (second snapshot in figure 3(b)) in contrast to the ER network (second snapshot in figure 3(c)). Because of the finite-size and sparse connectivity of both types of networks, not enough neighbour nodes were requested, allowing leakage and subsequent relapsing of the disease. Due to the antidote liberation, the disease grew in the direction contrary to where the nodes were placed. Even the small disease sources of the BA case produced much infection in the mesh after about 3000 time units from the first activation (third snapshot in figure 3(d)). However, the non-massive attacks of ER network nodes resulted in more isolated patterns and in faster increase of the disease quantity (third snapshot in figure 3(c)). After the interval of increase (fourth snapshot in figures 3(c) and (d)), the nodes retook control, eliminating many isolated patterns (fifth snapshot in figures 3(c) and (d)). While the nodes were eliminating these isolated patterns (fifth snapshot in figure 3(c)), a uniform spatial node distribution emerged in the mesh. Conversely, in the presence of few infected areas, the nodes joined efforts to eliminate them and concentrated themselves in the highest infected regions of the mesh (fifth snapshot in figure 3(d)). After the complete elimination of the disease, the nodes remained in their last respective positions. Observe that the original pattern was modified at the places where the antidote acted, especially near the activated nodes.
The amount of disease $I$ in the mesh (y-axis) over time (x-axis). (a) Fick diffusion model and (b) Gray–Scott model. One source, $N = 300$ and $\langle k \rangle \approx 4$ configuration. The standard deviation is (a) one fifth and (b) one tenth of the real value.

The ability of the defensive network to control and stop the disease was verified to be directly related to the number of nodes and to the connectivity of the network. We expected that with more nodes being activated, they would more readily gather control and completely eliminate the disease spreading. A larger and completely connected network would activate all the neighbours at once and hence fully populate the mesh. Consequently, the disease would fade down quickly until complete elimination. Such a network would imply high maintenance costs if adopted by natural (or artificial) systems. In fact, it is often mandatory to achieve maximum efficiency by using the minimum amount of energy. In practice, many of the networks which have been investigated in complex networks research are characterized by low connectivity among their nodes [1]–[3]. Therefore, it is interesting to investigate the efficiency of ER and BA networks with small numbers of nodes (relative to the mesh size) and low connectivity.

4.2. One-source configuration

Figures 4(a), 5(a) and 6(a) show the evolution of the amount of disease $I(t) = \sum_{x,y}^{256,256} u_{x,y}^I$ for the Fick diffusion model using the one-source configuration. A total of 100 realizations was considered for each parameter configuration. The function $I(t)$ had a nearly constant growth rate up to a maximum, when the first nodes were activated. These nodes triggered a chain reaction, but on average both networks had similar efficiency in controlling the diffusion up to about 60 000 time units. The time spent to enclose the source was relatively short and, on average, no difference could be observed between both types of networks. The importance of hub-activation, implying liberation of more antidote, showed up after 60 000 time units, when the diffusion constrained by the BA network clearly decreased faster than the diffusion observed for the ER network. By comparing figures 4(a), 5(a) and 6(a), it is clear, for both types of networks, that the diffusion dropped and reached minimum levels faster when the number of defensive nodes and the connectivity of them were increased, with better performance for the BA network. Interestingly, the minimal level of diffusion was reached at nearly the same time in both types of networks in the first configuration ($N = 300$ and $\langle k \rangle \approx 4$) and about 20 000 time units earlier in the BA case than in the ER case for the other two configurations ($N = 500$, $\langle k \rangle \approx 4$ and $\langle k \rangle \approx 6$), a consequence of the increased amount of antidote liberated in the first stages of the defence.
Figure 5. The amount of disease $I$ in the mesh (y-axis) in terms of time (x-axis). (a) Fick diffusion model and (b) Gray–Scott model. One source, $N = 500$ and $\langle k \rangle \approx 4$ configuration. The standard deviations in this figure correspond to one fifth of their real values.

Figure 6. The amount of disease $I$ in the mesh (y-axis) over time (x-axis). (a) Fick diffusion model and (b) Gray–Scott model. One source, $N = 500$ and $\langle k \rangle \approx 6$ configuration. The shown standard deviation is one fifth of its real value.

The non-uniform patterns generated by the Gray–Scott reaction–diffusion implied richer dynamics (figures 3(c) and (d)). Starting from the initial source, non-localized patterns emerged over time, creating fast moving spots and stripes, so that the nodes had to actively move through the regular network in order to eliminate the disease. The amount of disease $I(t)$ increased in nearly quadratic fashion with time up to a maximum when the first nodes were activated. Depending on the connectivity of the defensive network, different evolutions were clearly obtained after the first activation. The reaction–diffusion constrained by networks with $\langle k \rangle \approx 4$ (figures 4(b) and 5(b)), implied the following three stages: (i) a decrease down to a minimum level, (ii) a relapse up to a local maximum level and (iii) resumption of the decrease until the disease was eliminated. On the other hand, the network with $\langle k \rangle \approx 6$ (figure 6(b)) exhibited two stages: (i) fast and (ii) slow elimination of the disease. This phenomenon also depends on the number of nodes: a higher quantity of simultaneously activated nodes resulted in more antidote and, consequently, reduction of $I(t)$. 

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The first stage of defense (between 4000 and 6000 time units) was a consequence of *hierarchical neighbours* activation [20]. Once the disease had considerably diffused along the space, every new request contributed to the distribution of more activated nodes radially to the boundary of the disease. It was also possible to have some nodes requested by their own requested neighbours. Because of the finite size of the network, on average the hierarchical number of neighbours has a peak $n_{\text{max}}$ whose value depends on the number of nodes and on the connectivity of the network [21]. The presence of hubs implies that $n_{\text{max}}$ is reached faster (in terms of *hierarchical levels*) in the BA than in the ER network. In other words, BA nodes activate more neighbours at once than the ER nodes in the first *hierarchical levels*. Therefore, in this stage the disease decreased faster in the BA than in the ER case as shown in figures 4(b), 5(b) and 6(b). As expected, more nodes and higher connectivity provided more effective decrease in the disease intensity.

The second stage of defence (between 6000 and about 8000 time units) was characterized by leakage of disease from the first massive attack (i.e. chain reaction). The requested neighbours in the first stage were not enough to control the disease. Although they broke the pattern, some isolated regions of disease concentration remained which resumed progression. ER networks tended to engage less nodes than BA networks, allowing the creation of a larger number of isolated patterns. The latter effect implied more competition for nodes, postponing the control of the disease. Figures 4(b) and 5(b) show that as the number of network nodes was increased, the relapse peak tended to diminish. The relapse peak depended considerably on the height of the disease intensity $I(t)$ at the turning point. More distributed patterns implied more intense relapse and increased difficulty of respective control. The nodes had to swap their places constantly, following the requests which depended on the connectivity of the defensive network and not on the nodes distance in the regular network. Consequently, these movements of nodes resulted in vacancies in the regular network, which allowed the local development of disease. The effective elimination of disease by the network configured with $N = 500$ and $\langle k \rangle \approx 6$ (figure 6(b)) resulted in few remaining sources. Consequently, the defensive network was able to control the disease and maintain a low level of disease prior to its complete elimination.

The third stage (between 8000 time units and the complete disease elimination—figures 4(b) and 5(b)—absent in the third network configuration (figure 6(b)) was a consequence of the recovery of control by the activated nodes. Recall that due to the initial conditions, much antidote was liberated in the central area of the board in the first stage of defence. Naturally, the disease grew faster in the antidote-free regions, e.g. opposite to the central area. However, much antidote was also concentrated in other regions over time, which contributed to slowing down the growing rate of the disease and to faster elimination of the infection. Interestingly, the long tail in the graphics in figures 4(b), 5(b) and 6(b) was a result of some small steady focuses enclosed by the antidote, these focuses were not eliminated but could not grow. Under this situation, an equilibrium was ultimately established where any growth of the disease was promptly eliminated by antidote being liberated by the surrounding nodes.

### 4.3. Two-sources configuration

The competition for nodes played a fundamental role in the proposed dynamics since help requests implied depletion of nodes which were previously activated. If a neighbour $j$ was

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2 The turning point corresponds to the abscissae of the relative minimum of the disease intensity $I(t)$, which tended to occur after nearly 6000 steps.
helping a node $k$ and another node $w$ requested help from $j$, the node $j$ changes its position with 0.5 probability. Recall that a node request as a consequence of high activity in the regular network has higher priority than solicitations by neighbouring nodes. Therefore, only regular-network-activated nodes did not change their positions while in this state. Given the degree distribution of ER and BA networks, we expected improvement in the ability of disease control to be observed for the ER network. The more uniform distribution of degrees in the former type of network resulted in a better management of the distribution of nodes among many disease focuses. Conversely, the request of many nodes by hubs tended to unbalance the number of nodes at each infected area.

We also investigated the evolution of the disease when two sources were considered as initial conditions. Again, a total of 100 realizations was considered for each parameter configuration. The resulting shape of the curves was similar to that observed for the one-source configuration. Naturally, the Fick diffusion with two symmetrically displaced sources resulted in faster increase in the total amount of disease, so that the threshold was quickly overcome (about 20 000 time units before the one-source case—figures 7(a), 8(a) and 9(a). The main strategy against Fick diffusion is to enclose the sources, which was achieved as soon as the chain effect was triggered. Afterwards, the nodes only had to keep generating antidote in order to completely eliminate the already spread disease. A disease decrease rate similar to the one-source configuration was also expected. Once many nodes were still in their original positions, there were many susceptible nodes to be shared between the sources.

The configuration with higher number of nodes and connectivity (figures 8(a) and 9(a)) resulted in decrease of the efficiency in the BA network during the last stage of the defence dynamics. The uniform distribution of ER connections resulted, on average, in a higher efficiency in the surrounding of both sources. Over time, ER better managed the swapping of nodes between both sources. On the other hand, hubs requests resulted in a higher concentration of nodes around one of the sources (e.g. source 1). Consequently, the nodes were hardly activated due to the disease generated by the other source (source 2). The latter effect diminished the elimination rate of the disease because one of the sources (source 2) turned out to be indirectly controlled, i.e. through the antidote generated only by the nodes which were activated by the first source (source 1).
Figure 8. The amount of disease $I$ in the mesh (y-axis) over time (x-axis). (a) Fick diffusion model and (b) Gray–Scott model. Two-sources, $N = 500$ and $\langle k \rangle \approx 4$ configuration. The standard deviation is shown at one fifth of its real value.

Figure 9. The amount of disease $I$ in the mesh (y-axis) over time (x-axis). (a) Fick diffusion model and (b) Gray–Scott model. Two-sources, $N = 500$ and $\langle k \rangle \approx 6$ configuration. The standard deviation is shown at one fifth of its real value.

It also implied higher standard deviation of the disease intensity $I(t)$, especially at its minimal levels.

The initial effect of the two sources in the Gray–Scott model was the creation of two large infected areas in both sides of the wall of nodes. Each of them had approximately the same size as the area generated by the one-source configuration. The total amount of disease before the first activation was nearly twice as much in the two-sources configuration than that observed for the one-source case. Consequently, when the patterns reached the nodes, they initially had a larger amount of disease to eliminate. The same three stages were identified (figure 7(b)) as in the one-source case. However, the uniform distribution of neighbours in the ER network favoured a better distribution of the nodes among the many infected areas. This effect contributed to improving the defence ability of the ER network and to the enhancement of its efficiency. On the other hand, hubs made massive attacks against large infected areas. However, they requested many nodes which were defending other areas. The increase of network nodes (figure 8(b)) resulted
in better control of the disease constrained by the ER network. In fact, the second peak (relapse) was practically absent in this case when compared to the one-source configuration (figure 5(b)). Finally, the increase in the connectivity of the network resulted in even faster elimination of the disease (figure 9(b)). On average, each request engaged more nodes, which contributed to the steady reduction of the amount of disease.

5. Conclusions

Many natural phenomena involve interactions between two or more independent sub-systems with specific space–time properties (e.g. firemen combating forest fire, infection spreading into healthy tissue while interacting with defensive cells, cleaners controlling oil spills, pest control, etc). The structure of each sub-system can be modelled in terms of a network while the dynamics is represented by processes occurring in each network (e.g. the movement of agents or pattern formation). An interaction rule couples both sub-systems in such a way that the evolution of one sub-system depends on the other one and vice versa. Since the connections are responsible for the way the defensive agents communicate, they play a fundamental role in the behaviour of such complex systems, i.e. controlling the dynamical evolution of the agents (i.e. nodes) which, in turn, constrains the evolution of the dynamic patterns. For example, the specific way in which groups of firemen are organized determines whether or not they will control the spread of the fire. Similarly, the signal connectivity of antibodies is crucial to efficiently activate them to stop an infection diffusing through healthy tissue.

In order to investigate such phenomena, we proposed a dynamical hybrid system composed of a complex and a regular network. The complex network represented connected defensive agents (i.e. nodes) self-organizing to eliminate patterns evolving in the regular network which, in turn, represented the unwanted process. According to the local pattern intensity, the nodes were activated to liberate an opposite Fick diffusion aiming to eliminate the pattern. Two pattern growth models were considered: Fick diffusion and Gray–Scott reaction–diffusion. The defensive agents were connected following ER and BA models. Two types of initial conditions were investigated: one-source and two-sources. The role of the network structure was investigated by using three network configurations: (i) $N = 300$ and $\langle k \rangle \approx 4$ (ii) $N = 500$ and $\langle k \rangle \approx 4$ (iii) $N = 500$ and $\langle k \rangle \approx 6$.

The main results included the better performance obtained by the BA comparatively to the ER network for any chosen configuration. The hub-based characteristic of the BA network provided massive attacks against the disease. Heavy defence was crucial in the beginning in order to quickly accelerate the ratio of decrease of the amount of disease in the regular network. These massive attacks avoided much leakage and the emergence of isolated patterns which were present at higher rates in the ER case. Isolated patterns were responsible for the relapse of the disease. The increase in the number of network nodes and in their connectivity contributed significantly to faster elimination of the disease. These results have shown the importance of hubs in defensive networks. Hubs contribute to diminishing the average path length in the network. Consequently, on average the hierarchical level with maximum number of nodes can be reached earlier in the BA than in the ER network. As a result, a more effective defence can be evaluated when the disease is concentrated in a large area. On the other side, despite the better efficiency of the BA network, the uniform distribution of nodes in the ER network contributed to efficient defence strategies when many isolated patterns emerged at different places in the regular network.

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The reported work has paved the way to many future developments, which include: (i) investigation of optimal network structure to efficiently eliminate the pattern, (ii) analysis of how the system properties scale with its size, (iii) study of the pattern evolution under network perturbations (e.g. node attack or edge rewiring), and (iv) improvement of the model by inclusion of other communication protocols taking place in the defensive network, such as broadcasting.

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