Eur. Phys. J. B **50**, 513–520 (2006)
DOI: 10.1140/epjb/e2006-00148-3

THE EUROPEAN PHYSICAL JOURNAL B

Dynamic domain networks

M.N. Kuperman[1,2,a], M. Ballard[1], and F. Laguna[1]

[1] Centro Atómico Bariloche and Instituto Balseiro, 8400 S. C. de Bariloche, Argentina
[2] Consejo Nacional de Investigaciones Científicas y Técnicas, Argentina

Received 17 March 2005 / Received in final form 18 October 2005
Published online 5 May 2006 –

Abstract. A model for a dynamic network consisting of changing local interactions is presented in this work. While the network maintains solely local connections, certain properties known only to Small World Networks may be extracted due to the dynamic nature of the model. At each time step the individuals are grouped into clusters creating neighborhoods or domains of fully connected agents. The boundaries of these domains change in time, corresponding to a situation where the links between individuals are dynamic only throughout the history of the network. A question that we pose is whether our model, which maintains a local structure such that diffusion calculations are possible, might lead to analytic or conceptual advances for the much more complicated case of diffusion on a static disordered network that exhibits the *same* macroscopic properties as our dynamic ordered network. To answer this, we compare certain properties which characterize the dynamic domain network to those of a Small World Network, and then analyze the diffusion coefficients for three possible domain mutations. We close with a comparison and confirmation of previous epidemiological work carried out on networks.

PACS. 89.75.Hc Networks and genealogical trees – 89.65.-s Social and economic systems

1 Introduction

Diffusion through various types of media, be they disordered, dynamic, or otherwise, offers a perplexing problem present in many physical systems. The canonical example of diffusion through an ordered homogenous lattice is the process by which a particle randomly walks through a certain viscous media. In such ordered media, random walkers are considered to move probabilistically in discrete steps from neighboring site to neighboring site. Given this general abstraction, the idea may be applied to many seemingly unrelated systems, such as the spread of disease within a population, the step by step transfer of an idea or *meme* amongst the members of a given society, or the evolution of tradition through successive generations. The study of diffusion in different media (where transitions are not necessarily limited to local sites, but may include jumps to distant sites), is thus highly applicable to certain social phenomena, among other applications within computational complexity, neurobiology, and economic exchange. An obvious and problematic difference between real social systems and the idealized diffusive model, is that the structure of society is hardly an ordered lattice. Not only do individuals interact with an immediate neighborhood as well as distant nodes, but for real systems, the structure of these connected clusters changes throughout time. Diffusion in complex networks thus becomes quite complicated, as a diffusing state now moves through a dynamic network which maintains certain structural properties. We thus begin by considering a network topology already composed of clusters of completely connected individuals. Only through time, do these clusters overlap and interact, linking the clustered neighborhoods among themselves locally as the system evolves.

A common aim of contemporary network studies is to propose a model based on the local interactions among nodes that compose a given network, which gives rise to the global properties observed in the real systems to be considered. The study of Small World Networks [1], among other pioneering networks such as scale-free networks [2] and community networks [3], have done precisely this. Two properties proposed in [4] have proved to be useful within social systems; 1) a measure of clustering among groups of connected nodes, and 2) a measure of distance between any two given nodes on a graph. Real social systems naturally give rise to closely knit clusters of connected individuals, as a result of shared acquaintances. Furthermore, at least one member within a given neighborhood is likely to be connected to a member within another neighborhood. The effect of a few long range connections within an ordered nearest neighbor network, is that any two nodes are now connected by a surprisingly small number of links. Other pertinent global properties may of course be conceived. However, here we ask whether the same two pervading properties may be found in

[a] e-mail: kuperman@cab.cnea.gov.ar

different network models. Perhaps the Small World effect is not unique to the Small World network as defined by [1], and is in fact present in some form within other models that retain a certain local structure.

In this work, we present a network model with a particular topology that effectively links entire neighborhoods through local dynamic connections. The network structure may be considered to be composed of many disconnected subnetworks, or domains, each of which is completely connected among the individual components within each domain. At every time step the domain boundaries are redefined to encompass a different set of nodes. One may thus consider an overlap of domains between successive time steps, through which an active state, idea, or infection is passed from one neighborhood to another. The restriction to local interactions, via mutations of the domains between two time steps, is rather relative. That is, it is the relation between the entire system size to the local interaction range, which permits one to declare local or nonlocal interactions. Here we say *local* in that, all nodes are connected up to a certain domain size. The maximum domain size that we consider is relatively small with respect to the system size, thus validating the claim that though an active node spontaneously activates all nodes within the domain, interactions are local.

As the domains change, new disconnected subnetworks are created. It is through this *redivision* of subnetworks, and subsequent interaction among connected neighbors, and again redivision, that diffusion occurs. Curiously, the dynamic domain networks (DDN) exhibit certain properties also present in other disordered networks, such as Small World Networks where diffusion calculations are much more complicated if not altogether impossible. With new dynamic definitions, designed to be analogous to those of SWNs, the dynamic domain model demonstrates a sort of high clustering and a short average path length combination, as a parameter which governs the domain mutation process increases. Since the DDN exhibits features of a disordered network, it might be a useful tool with which to probe long range diffusion phenomena within disordered media. Rather than try to characterize diffusion through a complex disordered and dynamic network, we propose to study some diffusive processes in a network model that *exhibits* the macroscopic properties of a disordered, dynamic network yet maintains a simple, locally mutating, structure. The primary aspects that will be examined in this work are,

- The validity of an alternative dynamic model that is computationally simple, yet retains disordered properties of the well-established SWN. We demonstrate that long range links are not necessary to minimize the dynamically adapted definition of path length, and maximize the dynamically adapted clustering coefficient.
- The underlying diffusive processes that occur in the proposed model as the domains change according to three different types of mutation (a domain shift, a change in shape, or a change in size). The manner in which the domains change their overlap with each other between time steps, dictates a propagation velocity and thus a separate diffusion coefficient for each.
- The DDN model is rather unique in that it examines the transmission of an active state not exclusively on the level of the *individual agents*, but on the scale of *local domains*. Domains may migrate through the displacement probability, and/or fluctuate in size and shape. These two fluctuations are naturally correlated, as larger domains are more malleable in a discrete system. Essentially in order to transmit an active state, a domain of size one must be displaced. Whereas domains which are larger may change shape and thus transmit an active state solely through fluctuations of the domain shape and size, though the domain remains anchored spatially. This is an important aspect of the DDN model and its applications to modelling social systems. By dividing the dynamic into three separate, though not entirely uncorrelated dynamics, we are able to analyze the propagation of an infection as it is transmitted through groups of individuals. Furthermore, the groups have a different dynamic mechanism than does a single individual. In the limit that a group is composed of a single individual, we recover the solely migratory dynamic behavior. Otherwise, one must consider the fluctuations in group size and shape which are necessarily correlated.
- A transition that is witnessed in epidemiological studies on SWNs [13], is observed here in the DDN model. This comparison with real world observations suggests a clear utility of the DDN model as a dynamic model, simple in concept and computational design.

In the following sections, we outline the DDN model and all definitions necessary to understand the underlying network, imposed dynamic, and method of diffusion between domains. Though the model may further be generalized to include a sort of long range link, in section three we find that this is not necessary to reproduce the Small World effect. Since the propagation occurs through a specialized local interaction, the diffusion is then analyzed by considering a random walk with different jump probabilities in section four. Finally, we examine the validity of the DDN by observing certain transitions in the infection rate of the propagation of a disease, as the disorder (given by the domain mutation probability) increases. The model has been based on a two-dimensional projection of local social interactions. This aspect may suggest an immediate applicability to certain real systems composed of spatially dispersed groups of fully connected individuals. We thus aim to examine the utility of such a computationally feasible dynamic model, and the diffusive processes that occur upon the substrate of the network.

2 The model

The model is based on a two-dimensional matrix of static individuals, each one situated on a node of a $n \times n$ lattice. The entire lattice is divided into separate subnetworks, or domains with rectangular boundaries. Each of these

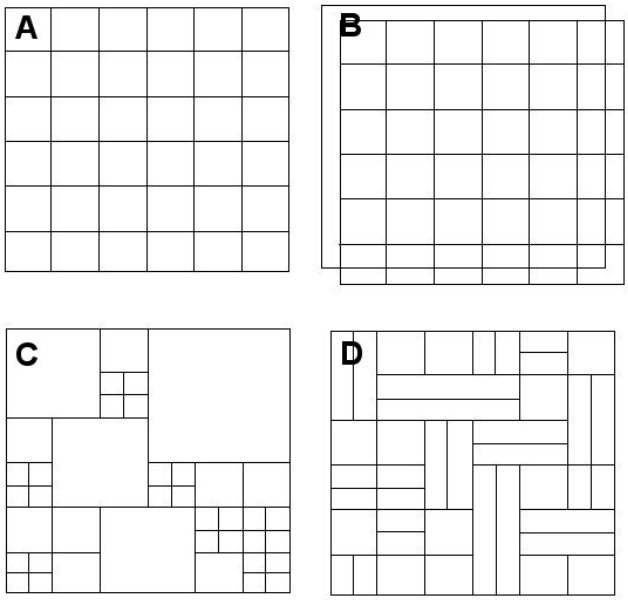

Fig. 1. Schematic representation of tessellations of the system for the, (a) regular case, (b) overall displaced configuration, (c) change in size, and (d) change in form.

N domains into which the individuals are grouped, composing a fully connected subnetwork, is not connected to any of the other subnetworks during each single time step. That is, these disconnected subnetworks are only interconnected when one observes the history of the entire network and notes the overlap that occurs between domains in two successive time steps. The initial configuration of the system is what we call the *regular* case, in which all domains have the same shape and size. The boundaries that demarcate the neighborhoods of the individuals, change at every time step according to the rules yet to be explained. As a consequence of the redivision of domains, a different configuration of domains will be compared at every time step. The original tessellation of the system will thus be exchanged for a new one, and an overlap of domains between two consecutive time steps is measured, see Figure 1 for examples of possible domain tessellations. Though the agents are static, the dynamic neighborhoods incorporate those individuals sharing the same domain at a given time step. The dynamic boundaries of the domains thus translate into the dynamic connections between static individuals. That is, the set of all individuals linked to a given individual changes at every time step according to the dynamic domain boundaries. We consider rigid walls along the borders of the entire $n \times n$ system, though this is fairly insignificant since we measure the mean square displacement and activation before an active state reaches the boundaries.

As an example, let's now consider the evolution of the neighborhood of the node j. At time t_i, some of the nodes belong to the neighborhood Λ_i^j. They may still belong to the same neighborhood at time t_{i+1}, Λ_{i+1}^j, although some may now pertain to a neighboring domain. While others which were not linked to j at time t_i, may now be within Λ_{i+1}^j, the neighborhood of j at time t_{i+1}. A simplified version of this system was also examined in [9] to model minority opinion spreading. In this context, one might build an interpretation of a population divided into separate forums of discussion. Forums, or domains, reach a consensus or equilibrium, and then transform and interact with other forums as the domains between two successive configurations overlap and allow interaction between certain members of the previously separated forums. As the configuration of the forums changes locally from t_i to t_{i+1}, the recorded history of domain boundary mutation determines the architecture of the underlying dynamic network.

A set of three processes govern the mutations of the domain walls. At every time step, the entire set of domains may be, 1) shifted from their former positions with a probability p_d; 2) undergo a change in size with a probability p_s and/or; 3) change shapes with a probability p_f, though they are required to remain convex (domains are always squares or rectangles). The probabilities *only* give rise to the possibility of change and do not dictate how much change occurs in each process. Because of this, the probability is defined to elect a tessellation out of all possible tessellations that have domains with a displacement, different sizes, or different shapes. Each $\boldsymbol{p} = p_d, p_s, p_f$ vector characterizes a set of possible tessellations from which one is elected. Refer to Figure 1 as an example of possible tessellations from each of the three sets of tessellations corresponding to $\boldsymbol{p} = 1, 0, 0$, $\boldsymbol{p} = 0, 1, 0$, and $\boldsymbol{p} = 0, 0, 1$. The new tessellation is the new configuration of divided domains with which to evaluate the overlap and to propagate and to activate states. Each of the three processes may act separately or together (where each combination $\boldsymbol{p}$, defines a separate set of configurations from which to choose), however there is some correlation between changes in shape and size. The larger a domain is, the more shapes it may take on. This is a natural consequence of having a discrete system. By reducing the domain size to one, only one shape is permitted, and we recover the traditional random walker that can only move through domain shifts. This limit is the same as considering diffusing particles which activate each other through diffusive interaction. However, we are considering a more general model where domains may be composed of a single, diffusively migrating random walker and/or discrete neighborhoods that not only migrate, but also fluctuate in size and shape. This is an important aspect of the DDN model and its applications to modelling social systems. This approach looks at the transmission of an active state not exclusively on the level of the interactions between individuals, but on the scale of interacting neighborhoods which are recognized to fluctuate in shape and size when there is more than one node contained within. We observe that the probability defined here is not the probability for each domain to change placement, size, or shape, but rather, it is a probability to elect or not a tessellation (a configuration with full coverage) from a set of all possible tessellations that are composed of uniformly displaced

domains, different sized domains, or different shaped domains.

Here we consider the extreme cases of domain change. That is, we vary one mutation probability while holding the other two constant at one or zero, and thereby look at all tessellations where the domains have either shifted, changed size, or changed shape. When all three take place at the same time, a superposition of their effects occur. The changes in *size* are limited to a magnitude no greater than twice the regular domain size. Possible *shapes* are limited to the given size, meaning that each size has a limited and different number of accessible rectangular shapes. (This is a consequence of requiring convex domain forms). Finally, a domain may not be *displaced* by a size more than an other domain size per time step. These restrictions thus preserve a varying *local range* of interaction throughout the entire network. Naturally, for small system sizes, these local interactions may become global. However, the regular configuration and associated mutations are very small compared to the system size (with domain size between $N^2 = 1$ to $N^2 = 6^2$, in a system of size $n^2 = 10^4$ to $n^2 = 10^7$). In Figure 1 we show a schematic realization of these three different processes.

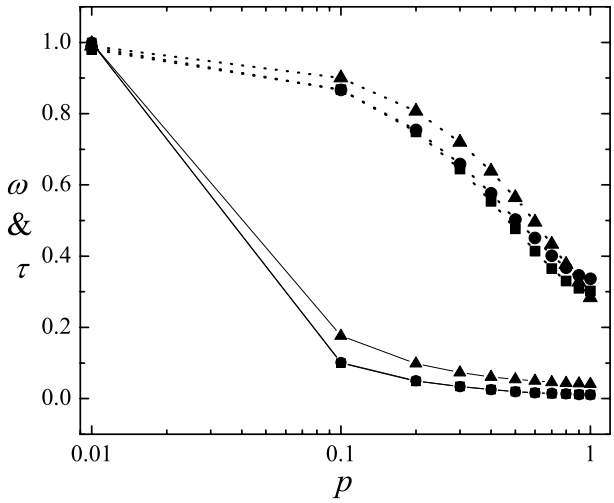

Fig. 2. The bold line τ, and the dotted line ω, as functions of p (the compact expression of a single varying mutation probability), where $p = p_d$(squares), $p = p_s$ (triangles), and $p = p_f$ (circles). Non-varying p values are set equal to zero and τ is normalized to the total activation time for the case when $p = 10^{-2}$.

3 Dynamic properties

To characterize certain global properties of the dynamic network, we define quantities similar to those suggested in [8]. A macroscopic variable analogous to the average path length in SWNs is measured by considering a propagation process throughout the system. We first distinguish between *active* and *inactive* nodes. Initially, a single node is chosen at random to be activated, which in turn, immediately activates its entire domain. That is, any inactive individual in a domain is immediately activated by the presence of at least one active individual in the same domain. Once activated, the individual remains as such for all time. The time necessary for an active state to propagate across the system is a measure of the path length of the entire dynamic network, in that counting the number of iterations necessary to activate the entire system is a measure of distance as well. Naturally, this activation time is affected by the mutation probabilities of displacement p_d, size change p_s, and form change p_f. These probabilities give rise to the amount of overlap between domains over two successive time steps, and thus determine how far the activation front extends and how quickly. For system sizes of $n^2 = 10^4$ to $n^2 = 10^7$ nodes, we numerically measure the *activation time* τ_i, as defined in [8], necessary to activate the node i within the system,

$$\tau = \frac{1}{n^2} \sum_{i=1}^{n^2} \tau_i. \tag{1}$$

Next we define an average overlap between the domains over two successive time steps as a parameter analogous to the SWN clustering coefficient. Naturally, as the mutation probabilities increase, the domains tend to have less overlap with the previous configuration. This is evident for the case of domain displacement, however this is not so obvious for changes in shape or form since this is only true for a statistically large number of events. Again, numerical calculation of the mean domain overlap is performed using

$$\omega = \frac{1}{\tau_a n^2} \sum_{t}^{\tau_a} \sum_{i}^{n^2} \omega_i(t), \tag{2}$$

where the summation over time is performed over all the time steps until total activation at time τ_a is achieved. In Figure 2 we plot the values of τ and ω for the particular cases in which two of the three mutation probabilities are zero and the remaining one varies from zero to one.

The mean overlap when displacement is not considered, may be probabilistically calculated with a binomial distribution over all possible situations of overlapping domains. For the moment, this calculation is simply intended to encourage an interest in the possible resultant behaviors due to the different types of domain mutations. The analytic calculations of the overlap ω for a varying p_s or p_f, and their numerical comparisons are displayed in Figure 3. The lower curves correspond to changing either p_s or p_f, while p_d and the remaining p value are both zero. The upper curves depict a varying p_s or p_f, while p_d is again equal to zero, but the remaining term is now constant with a value of one. The continuous line is obtained by analytic calculation on top of which we have plotted the numerical results.

4 Propagation on the dynamic substrate

The model depicted thus far may be visualized as a two-dimensional configuration of rectangular domains, changing at every time step with a probability associated with

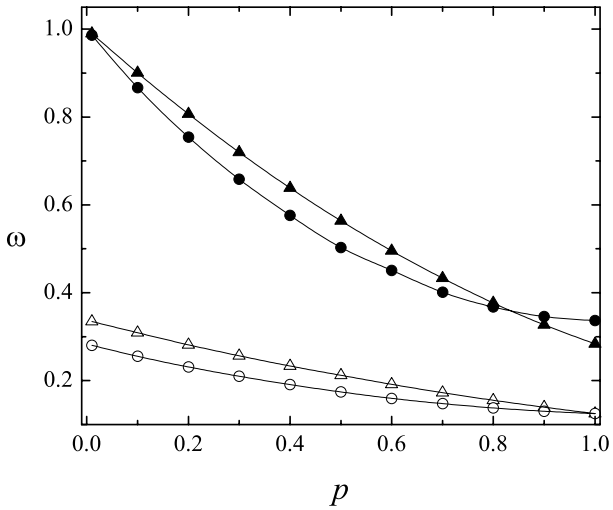

Fig. 3. The average overlap ω, as a function of p, where $p = p_s$ (triangles), $p = p_f$ (circles), and $p_d = 0$ in all cases plotted here. Solid symbols indicate that the non-varying p parameter is zero, whereas open symbols indicate the non-varying p parameter is one.

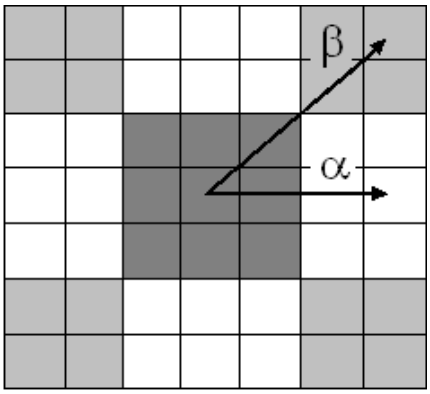

Fig. 4. The external square corresponds to the range of movement of the initial domain (internal square). Darker grey areas represent the region denoted by β and lighter grey areas represent regions denoted by α. Diffusive movement can thus be decomposed into these directions.

each type of domain mutation. Between any two time steps, there are different amounts of overlap between a given initial domain, and its previous domain location, shape, and size. A statistical sampling of overlaps throughout the system gives one an idea of how much interaction occurs between domains (also considered to be subnetworks), for each mutation probability throughout time. Overlap naturally only occurs between a domain, and a mutation of the same domain. A given domain remains in the *local* neighborhood between two successive time steps, unless zero overlap occur, which is a situation that is not permitted due to the mutation restrictions outlined previously. Since domains evolve through local probabilistic mutations, the underlying process is actually diffusive. We may thus count all possible domain shifts to calculate the probability associated for a random walker to jump left, right, up, down, or diagonal, on a two-dimensional lattice. A random walker is placed in a given domain on the lattice. As the initial domain mutates and shares overlap with the neighboring local domains of a regular configuration, the contained random walker jumps from the original domain center to the center of the overlapped domain. The changing domain walls thus permit the random walker to either remain in the initial domain, or move to neighboring domains with probabilities that are related to the mutation probabilities.

Indeed, the process may be considered as a diffusive random walk, characterized by a probability distribution particular to each dynamic process. Some work has previously been done with diffusion on top of SWNs [10,11]. In this system however, the diffusion is inherently part of the model. The random walker is not a process that occurs *on top* of a network, but a process that naturally occurs as the domain walls change. The dynamic of the network thus forces the diffusion of an active state, disease, or rumor. This aspect may play an important role in the real world counterpart of propagation phenomena. Propagation is thus here woven into the topology of the network.

An analytic understanding of the diffusive movement may be developed by considering the possible domain mutations and the directional jump probabilities α and β, to be calculated. Consider the case of a dynamic domain originally composed of nine sites. Two different jumps of a centered random walker should be considered; those to neighboring domains that share a side with the original one with probability α, and those to any of the other four domains along the diagonals with probability β, see Figure 4. The α and β probabilities for each of the three processes are calculated by counting all of the possible overlap situations that lead to a given jump, either to a side or along the diagonal. By anchoring the center of a domain, then considering all possible domain changes for each mutation, and finally using symmetry arguments for the remaining three possible anchored corners, we have calculated the jump probabilities for each dynamic mutation process. This is simply a counting method for all possible configuration mutations. For displacement $\alpha_d = .1200$, $\beta_d = .0400$, for size change $\alpha_s = .0240$, $\beta_s = .0039$, and for changes in form $\alpha_f = .1111$, $\beta_f = 0$. With the jump probabilities α and β, a prepared standard binomial distribution may be used to evaluate the random walk process and the associated diffusion coefficients of an active state diffusing throughout the dynamic model.

Since we will later measure the mean square displacement as a projection onto the x-axis, here we consider the movement of a random walker only to the left and right, and along the diagonals. The total probability to move to the left is given by the sum of the probabilities to move along either the left-up or left-down diagonal, and the probability to move directly to the left, $q_L = p(\alpha + 2\beta)$. The sum of the jump probabilities is weighted by the probability that the domain mutates and permits any movement at all: p, which again is the mutation probability, a compact notation for the individual probabilities to change location, size, or form. Due to the

symmetry of the model $q_L = q_R$, the probability to jump to the left is equal to that of the right. There are also probabilities to move directly up and down (2α), or stay in the same site, $\gamma = 1-2p(\alpha+2\beta)$. By performing a standard random walker calculation, we may derive a binomial probability distribution of the displacement of a random walker from the initial location. The following expression, which depends only on the jump probabilities, has been derived by first considering the characteristic function of the sum of t statistically independent random variables. This sum constitutes a random walk.

$$T(k) = (q_L \exp(-3ik) + q_R \exp(+3ik) + \gamma). \qquad (3)$$

The final location of the random walker as projected onto the x-axis is given by the random variable x of the Fourier transform of the characteristic function, which is the probability distribution of x by definition. The central limit theorem, and appropriate Stirling approximations (for $t \longrightarrow \infty$), may be used to derive a Gaussian which characterizes the mean and variance of the spreading active state. However, a more direct approach is to recognize that the second moment is also defined by the second derivative of the generating function with respect to k and evaluating at $k = 0$ [12].

$$\langle x(t)^2 \rangle = \frac{d^2}{dk^2}(T(k))^t \Big|_{k=0}. \qquad (4)$$

From this, the mean square displacement $\langle x(t)^2 \rangle = 2Dt$ is easily calculated and found to be $2Dt = 18p(\alpha + 2\beta)t$. By considering the appropriate jump probabilities for each dynamic process as listed previously, we find that the diffusion coefficients D, for each type of movement are given by $D_d = 1.80p_d$, $D_s = 0.28p_s$ and $D_f = 1.00p_f$.

In conjunction with the analytical interpretation of this particular diffusive process, we have performed some numerical simulations. The mean square displacement $\langle X^2 \rangle$, of the growing cluster of active individuals is evaluated. From the analysis of this quantity throughout time, we may numerically obtain a value for the diffusion coefficient. Figure 5 displays the diffusion coefficient as a function of each of the three processes defined by p_s, p_d, and p_f and the corresponding analytical calculations as solid lines. A fitting by minimum squares method gives us the following numeric values of the diffusion coefficient: $D_d = 1.60p_d$, $D_s = 0.27p_s$ and $D_f = 1.00p_f$, which are in good agreement with analytic predictions.

A further measurable property of diffusion through the system is the evolution of the size of the cluster of active individuals. For this we have evaluated the velocity of the mean radius of the growing nucleus. The radius evolves linearly with time and thus the velocity of propagation v of the active state is easily calculated. In Figure 6, v is depicted as a function of p for each dynamic process. The inset shows v as a function of the mean overlap. It is apparent from the figure that the velocity adopts a different behavior for each of the three dynamic processes. This value is studied for different sets of p_d, p_s, and p_f.

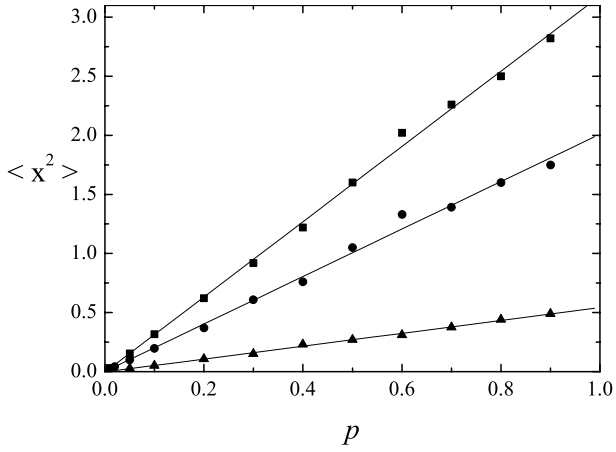

Fig. 5. Diffusion coefficients as p varies, where $p = p_d$(squares), $p = p_s$ (triangles), and $p = p_f$ (circles). Solid lines indicate the analytic calculation on which the numerical results are plotted as solid shapes.

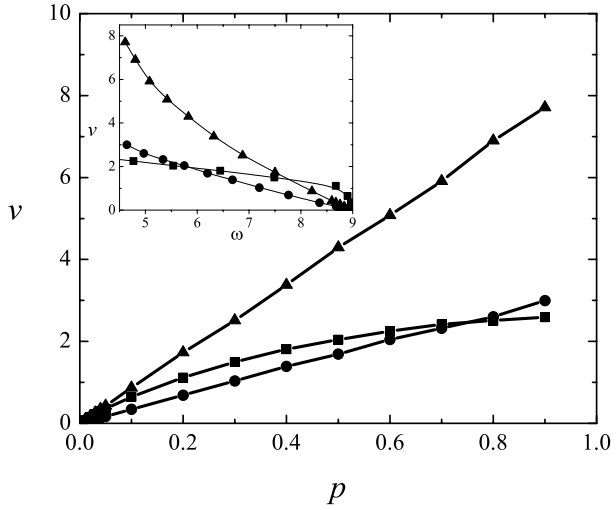

Fig. 6. The average velocity of propagation v as a function of p, where $p = p_d$(squares), $p = p_s$ (triangles), and $p = p_f$ (circles). All mutation probabilities, aside from the one which varies, are zero. The inset shows v as a function of the mean overlap ω.

5 Disease propagation

To compare the results of the DDN model with those of a SWN, we propose here an epidemiological model as a different type of propagative phenomena on top of the network. This model introduces a threshold phenomena that is not present in the previous activation process. The results of disease propagation within Dynamic Small World Networks [13] is obtained in the following way. We consider a standard model [14] for an infectious disease with three stages: susceptible (S), infectious (I), and removed (R). Any susceptible individual can become infected with a given probability by an infected individual within the same domain or neighborhood. A third epidemiological state (R) and the probability to become infected are what

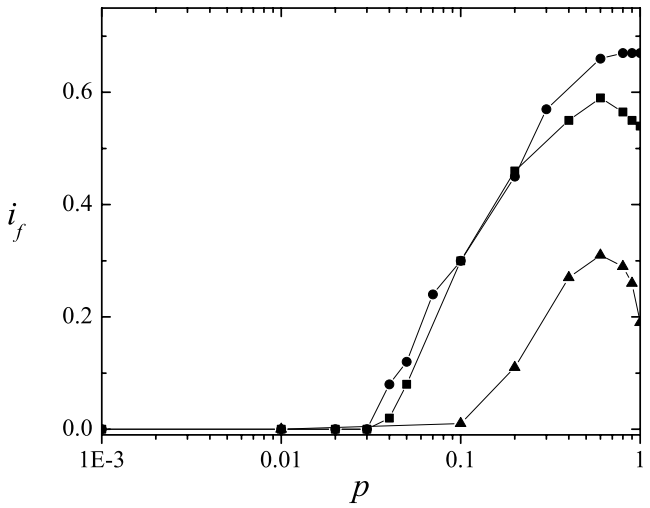

Fig. 7. The fraction of infected individuals with respect to the total population i_f, as a function of p, with $p = p_d$(squares), $p = p_s$ (triangles), and $p = p_f$ (circles).

distinguish the activation process on the DDN model and the new propagative process. The infection cycle ends when the element reaches the removed state after τ_r time steps. The removed state is a permanent condition and thus the individual cannot be infected again. The algorithm goes as follows. At each step an infected element i is chosen at random. If the time elapsed from the moment t_i when it entered the infection cycle up to the current time t is larger than the infection time τ_r, the element i becomes removed. Otherwise, one of its neighbors j of i is randomly selected. If j is in the susceptible state, contagion occurs. Element j becomes infected and its infection time $t_j = t$ is recorded. If on the other hand, j is already infected or removed, it preserves its state. Since each time step corresponds to the choice of an infected individual, the update of the time variable depends on the number $N_I(t)$ of infected individuals at each step, $t \to t+1/N_I(t)$. Once there is no change in the number of infected individuals for the duration of time necessary for all infected individuals to transform into the removed state R, the process stops. Here we set $\tau_r = 3$ for the infection time which insures that for intermediate values of the $\overline{p}$ parameters, the disease spreads over a finite fraction of the population. Moreover, we consider an initial condition where there is only one infected element; all the other elements being susceptible. The considered initial condition thus represents an initially localized disease. The infection will remain localized during the initial stages and will propagate according to the behavior of the domains. In Figure 7 we plot the proportion of total infected individuals i_f as it varies with different values of p. Two of the p values are maintained fixed while the remaining one varies between zero and one.

In comparison with the results obtained in [13], we find that the threshold values of p for disease propagation are of the same order. The sharp transition from the non-propagative regime to the propagative one as a function of the structure of the network is one of the most interesting aspects presented by epidemiological models on networks. The maximum proportion of infected individuals shows that a fraction of individuals remain not infected. Similar values of final infected individuals have been obtained in [13]. The non-monotonic behavior of this quantity has also been observed in other epidemiological models based on Small World Networks [15]. A possible explanation for the decrease of infection for large values of p_s involves the fact that in the DDN system, there is a bias towards decreasing the size of a domain. A high level of dynamics will temporally isolate infected individuals. It is because of this limitation of the model that the infection cannot propagate as efficiently for large p_s and a smaller number of infected individuals is detected.

6 Conclusions

Though the DDN model is only constructed with local connections, we aim to compare the system presented here with previous work on dynamic Small Worlds with long range connections. The local nature of the connections in the present model is due to the fact that new links are only to be established with nodes occupying neighboring domains. It is interesting that we still observe prominent properties analogous to those found in SWN models without local restrictions. The parameters describing neighborhood overlap ω and mean persistence time τ indicate behavior synonymous with the SWN clustering and average path length, respectively. That is, there exists a region in the p parameter space when the system displays a mixture of the two limiting cases, namely relatively high overlap and relatively low activation time. Furthermore, epidemiological threshold phenomena is reproduced in the DDN model without the necessity of SWN short cuts. Though no analysis of how the domain size scales with these quantities was done, qualitative similarities ought to be noted. While some SWN results may be reproduced in the DDN model, the latter may provide a more realistic description of social and cultural behavior. Domains mutate and migrate as entire clusters on a global level, rather that local or individual level as occurs in SWNs. Interesting results of the spread of minority opinion have been obtained in [9] by considering a particular case of the model presented here.

We have assumed that underlying diffusion is actually built into the model, as an inherent aspect of the imposed dynamic. Three types of diffusive movement may be analyzed. A diffusion coefficient was numerically as well as analytically calculated for the three dynamic processes, p_s, p_f, and p_d. This demonstrated the diffusive contribution of each type of domain mutation. As a final application and point of comparison with previous work, we analyzed the propagation velocity and outbreak transition of an infection. For small p values, an initial infection cannot propagate. At a given p value, propagation is possible and grows rapidly to a maximum infected fraction of the entire population as p increases. The threshold values are

similar to those previously observed for a dynamic SWN. In this case, we show that despite the fact that there is a transition around the same value of p, the behavior of the system strongly depends on which dynamic domain process governs the dynamics of the system. We are thus permitted to observe the different effects and relative importance of the three separate means of diffusion for each type of mutation, on the spread of a disease, rumor, or *meme*.

M.B. would like to thank the OAS for partial support.

Appendix

In our model for the random walker, we consider two stages. The first one takes into account the probability of the movement or jump to be accepted p. This parameter denotes the mutation probability, a compact notation for the probabilities to change location, size, or form. The second stage accounts for the movement of the individual to any of the allowed directions. Since the random walk is isotropic and symmetric, lets consider the projection of the movement of a 2D random walker onto the x-axis. As mentioned before, given that a movement of the walker is accepted with probability p, the conditional probability to move to the left is given by the sum of the probabilities to move along either the left-up or left-down diagonal, and the probability to move directly to the left. Finally we have $q_L = p(\alpha + 2\beta)$ according to Figure 4. Accordingly, by symmetry, the probability to move directly to the right is $q_R = q_L = q$. There are also probabilities to move directly up and down (2α), that when projected onto the x-axis, are equivalent to a situation when the walker remains static with respect to the x-axis. The probability to remain static is thus $q_S = \gamma = 1 - 2p(\alpha + 2\beta)$.

With a standard homogenous 1D random walker calculation, we can extract the diffusion coefficient from the second moment of the displacement, or the mean square displacement $\langle x^2 \rangle$,

$$D = \frac{\langle x(t)^2 \rangle}{2t},$$

where t is time or the number of time steps. The mean square displacement can be calculated from the hopping matrix, which is composed of all transition probabilities between random walker sites. In our case, due to spatial invariance, the probability of transition from a site x to another x' can be written as

$$\begin{aligned} T(x, x') &= T(x - x') \\ &= q_L \delta_{x,x'-3} + q_R \delta_{x,x'+3} + q_S \delta_{x,x'} \\ &= p(\alpha + 2\beta)(\delta_{x,x'-3} + \delta_{x,x'+3}) + \gamma \delta_{x,x'}. \end{aligned}$$

The corresponding characteristic function is the discrete Fourier transform of the former expression,

$$\begin{aligned} T(k) &= p(\alpha + 2\beta)(exp(-3ik) + exp(+3ik) + \gamma) \\ &= (2q\cos(3k) + \gamma). \end{aligned} \quad (5)$$

The mean square displacement can be now calculated as,

$$\langle x(t)^2 \rangle = \frac{\partial^2}{\partial (k)^2} (T(k)^t) P(k, 0) \Big|_{k=0}$$

where $P(k, 0)$ is the Fourier transform of the initial condition $P(x, 0)$, the probability that the walker is at x at time $t = 0$. We consider the initial condition to be $P(x, 0) = \delta_{x,0}$. We must thus solve,

$$\begin{aligned} \langle x(t)^2 \rangle &= \frac{\partial^2}{\partial (k)^2} (2q\cos(3k) + \gamma)^t \Big|_{k=0} \\ &= 18qt(2q + \gamma)^(t - 1). \end{aligned}$$

Since $(2q + \gamma) = 1$ due to normalization, we finally recover

$$\langle x(t)^2 \rangle = 18p(\alpha + 2\beta)t.$$

and thus $D = 9p(\alpha + 2\beta)$.

References

1. D.J. Watts, S.H. Strogatz, Nature **393**, 440 (1998)
2. R. Albert, A.-L. Barabasi, Rev. Mod. Phys. **74**, 47 (2002)
3. A. Arenas, L. Danon, A. Díaz-Guilera, P.M. Gleiser, R. Guimera. Europ. Phys. J. B **38**, 373 (2004)
4. D.J. Watts, *Small Worlds* (Princeton University Press, 1999)
5. L.A.N. Amaral, A. Scala, M. Barthélemy, H.E. Stanley, Proc. Natl. Acad. Sci. U.S.A. **97**, 11149 (2000)
6. S.H. Strogatz, Nature **410**, 268 (2001)
7. R. Albert, A.L. Barabási, Phys. Rev. Lett. **85**, 5234 (2000)
8. S.C.Manrubia, J. Delgado, B. Luque, Europhys. Lett. **53**, 693 (2001)
9. C. Tessone, R. Toral, P. Amengual, H. Wio, M. San Miguel, Eur. Phys. J. B. **39**, 535 (2004)
10. R. Monassen. Eur. Phys. J.B. **12**, 555 (1999)
11. S. Jespersen, M. Sokolov, A. Blumen. Phys. Rev. E **62**, 4405 (2000)
12. M. Cáceres, *Elementos de estadística de no equilibrio y sus aplicaciones al transporte en medios desordenados* (Reverté, 2003)
13. D. Zanette, Phys. Rev. E **65**, 041908 (2002)
14. J.D. Murray, *Mathematical Biology* (Springer, Berlin, 1993)
15. D. Zanette, M. Kuperman, Physica A **309**, 445 (2002)