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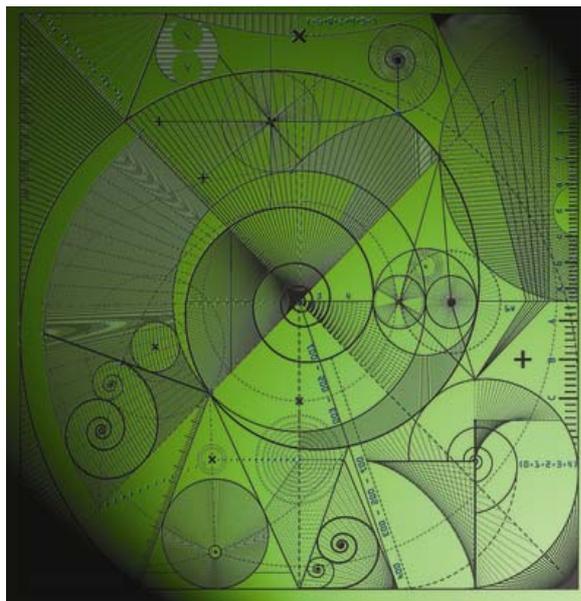
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Complex Dynamics in a Model of Social Norm

by

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Complex Dynamics in a Model of Social Norms

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Abstract

We propose a fully coupled model of social systems capable of exhibiting the emergent, dynamical, formation and dissolution of subgroups sharing social norms. It is “fully coupled” in that (i) pairwise connections are mediated by homophily effects, with connections being more likely to occur and endure between individuals sharing a common attitude (state); whilst (ii) the social evolving network itself allows connected neighbours to influence each other and align both their excitatory tendencies and their inhibitions, in parallel. Tension between the homophily effects and a possible Turing instability within the attitude dynamics produces both patterns and temporal dynamics, occurring in a pseudo-periodic fashion.

Keywords: Evolving networks, Full coupling, Social norms, Turing instability, Almost periodicity

1. Introduction

In recent years there has been a rapid development of research and understanding of dynamically evolving networks: time dependent pairwise interactions representing the existence, or the strength, of communication and mutual influence between vertices. In social applications the collection of vertices represents a population of individuals.

Besides the dynamics of such networks there is the problem of modelling the dynamics of properties transferred through, or on, the network. Statistical physics has had much to say about social dynamics and [1] reviews and summarizes a very wide range of problems and models employed to date. Most relevant to the work here are (a) models of cultural dynamics, attitudinal models going back to Axelrod [2], where each individual has a group of potential neighbours (to influence, and to be influenced by) and the weight of such interactions depends on their present cultural agreement/alignment; and (b) opinion dynamics, such as voter

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models on various types of networks, where a discrete state is selectively (probabilistically) influenced by that of its neighbours. The review in [3] shows how understanding of evolving networks across a range of applications may be organized within a common theoretical framework. Together [1, 3], and the references therein, provide context for this paper.

Here we consider a new type of related mathematical model for the unconscious development of social behaviors with subgroups of a population. Individuals are represented by vertices within an evolving undirected stochastic *influence* network, using a continuous-time extension of the algebraic framework introduced in [4]. We shall assume that some given element of the social behaviour of each individual is subject to both excitatory and inhibitory processes represented by real state variables, continuous in time, the subjects of a coupled dynamical system. Behavioral inhibition within subconscious decision-making is usually associated with activity in the prefrontal cortex, a relatively recently evolved part of the primate brain, whereas excitatory urges are associated with activity within much older parts of the brain, such as the limbic brain, see [6].

Excitatory urges and inhibitions can be influenced subconsciously by behaviour or cues observed in others with whom we seek to align ourselves. Indeed, mutual imitation and mirroring can take place subconsciously so that we take on similar social behaviours/personas. Such a process ensures individuals develop group behaviour and can change together. This effect may be blocked completely though if we do not currently identify with, or seek to become aligned with, the other person [6], because they are already giving cues that are much different from our own.

Pairwise interactions between any and all individuals are represented in our model by the evolving network's edges: each edge is a binary function of time. Although all pairwise interactions are possible, we shall assume that the principle mechanism of constraint on such interaction is that of homophily [5]. This is the process by which pairs of individuals exhibiting similar behaviours and attitudes may interact more frequently and influence each other more strongly. It models the subconscious need to become aligned with or else to block the cues given by another person's behaviour.

Within our model direct connections between more similar individuals are both more likely to occur and more likely to endure. Crucially the influence between presently connected individuals is assumed to be between the pair's corresponding state variables, so that excitatory and inhibitory variables influence their counterparts independently in parallel. This is reasonable since they are likely to be controlled by very different parts of the individuals' brains (the limbic brain and the prefrontal cortex respectively, for example).

When subgroups of the population develop a common behaviour, we can refer to this as a social norm: it is a property of the (sub)group. Within a population of students, for example, we may observe some subgroups whose members share and exhibit a relatively promiscuous norm, whilst other subgroups whose members exhibit a more prudish norm; we may observe some subgroups that are relatively hedonistic, and some subgroups who are much less so. Such social norms may be transient in the longer term, though individuals' journeys may

allow them to flip-flop between such alternative norm-based groupings. Hence we propose a fully coupled stochastic evolutionary model for individuals behavioural trajectories, within and across possible subgroup formation associated with alternative social norms. The novelty of this model lies in the tension created between the homophily within the evolving social network and the possibility of network-induced instabilities within the individuals' activator-inhibitor behavioural-state dynamics.

In section 2 we introduce the notion of activator inhibitor system that coexists within both the psychological and mathematical literatures. In section 3 we present the model and an analysis of its dynamics. We show that a combination of a Turing instability process, quite common in activator-inhibitor dynamics, and the homophilic effects, can condemn the systems to be pseudo-periodic: there can be no uniform stable state valid for all individuals, becoming ever better connected through their unanimity of behaviour. Instead, a constant *boiling of the pot* occurs with some (relatively well connected) subgroups becoming relatively excited or relatively inhibited compared to others. The macroscopic behaviour is predictably pseudo-periodic, while the individuals' behavioural trajectory is in a stochastic state of flux.

Such models are distinct from those considered previously since the network driven, Turing, instability competes with the homophily terms which force community strengthening and weakening, and consequently such fully coupled system never reach an equilibrium. In section 4 we present a specific example of such a system and a projection method for visualizing the pseudo-period behaviour within model simulations.

2. Activator-inhibitor processes

There is a large literature within psychology based on individuals being at a tensioned equilibrium between excitatory (activating) process and inhibiting process. Gray's [8] introduction of a model for behavior activation and behavior inhibition led to a large amount of research. More recently this has been applied to a range of social states, for example, the Dual Control Model summarized by [7], proposes that individuals' sexual responses involve an interaction between sexual excitatory and sexual inhibitory processes. It is now generally accepted that most brain functions are at a balance between excitory and inhibitory processes, and that across individuals this may vary naturally.

However variability across a population can be the result of dynamical processes resulting from the individual's internal tensioning (dynamic process) and peer to peer coupling. Typically the state is represented by an array of state variables, some measuring activating elements and some measuring the inhibiting elements. Most such mathematical models possess just two variables (as an exemplar) for more complicated models : one activator variable and one inhibitor variable. Both are time dependent.

Activator-inhibitor systems have had an impact within mathematical models where a uniformity equilibrium across a populations of individual systems becomes destabilized by the

very act of simple ‘passive’ coupling between those individual systems. Such Turing instabilities [9] naturally break the uniform symmetry, resulting in localized subsets of relatively increased activation and relatively increased inhibition. This occurs when the coupling between individuals’ is stronger for the inhibitor variables than for the activator variables. Such instabilities may seem counter intuitive at first sight since individuals are seeking pairwise alignment: yet, for example, the dissipation (dilution) of inhibitions allows extremes to develop. Thus any pairwise coupling of activator-inhibitor variables is a potential mechanism for creating variability across populations.

It is thus reasonable to postulate that any mathematical model of social behavioral norms emerging across a population of individuals should include a range of activator-inhibitor type systems within each individuals, while coupling these through communication or social influence (via processes of social awareness, exhibition).

3. Modelling

Consider a population of N identical individuals, each described by a set of m real attitude state variables, continuous functions of time t . Let $\mathbf{x}_i(t) \in \mathbb{R}^m$ denote the attitudinal state of the i th individual.

We suppose that the individuals are connected together by a dynamically evolving undirected network. Let $A(t)$ denote the $N \times N$ binary, symmetric, adjacency matrix for this network at time t , having a zero diagonal. We adopt a first order model for the coupled system:

$$\dot{\mathbf{x}}_i = \mathbf{f}(\mathbf{x}_i) + D \cdot \sum_{j=1}^N A_{ij} (\mathbf{x}_j - \mathbf{x}_i) \quad i = 1, \dots, N. \quad (1)$$

Here \mathbf{f} is a given smooth field over \mathbb{R}^m , such that $\mathbf{f}(\mathbf{x}^*) = 0$, for some, \mathbf{x}^* . We shall assume that the linearisation of \mathbf{f} at \mathbf{x}^* , denoted by $d\mathbf{f}(x^*)$, is an $m \times m$ stability matrix (all eigenvalues having negative real parts). In fact, as discussed in the introduction, we shall assume that \mathbf{f} is drawn from a class of activator-inhibitor systems. D is a real diagonal, nonnegative matrix containing the transmission coefficients for the corresponding attitudinal variables between adjacent neighbours. By construction there is a uniform population equilibrium $\mathbf{x}_i = \mathbf{x}^*$, for $i = 1, \dots, N$.

Let $\mathbf{X}(t)$ and $\mathbf{F}(\mathbf{X})$ denote $m \times N$ matrices with i th columns given by $\mathbf{x}_i(t)$, and $\mathbf{f}(\mathbf{x}_i(t))$ respectively. Then (1) may be written as

$$\dot{\mathbf{X}} = \mathbf{F}(\mathbf{X}) - D\mathbf{X}\Delta. \quad (2)$$

Here $\Delta(t)$ denotes the graph Laplacian for $A(t)$, given by $\Delta(t) = Dg(t) - A(t)$, where $Dg(t)$ is the diagonal matrix containing the degrees of the vertices ($Dg(t)_{ii} = \sum_{j=1}^N A(t)_{ij}$). It is positive semi-definite and symmetric. System (2) has an equilibria at $\mathbf{X} = \mathbf{X}^*$ say, where the i th column of \mathbf{X}^* is given by \mathbf{x}^* for all $i = 1, \dots, N$.

Now consider an evolution equation for $A(t)$. Each edge is assumed to evolve independently, though each is conditionally dependent on the current network (so edges conditional on related current sub-structures may well be highly correlated over time). So rather than model a full probability distribution for future network evolution, conditional on its current structure, say

$$\mathcal{P}_{\delta t}(A(t + \delta t)|A(t)),$$

it is enough to specify its expected value $E(A(t + \delta t)|A(t))$ (a matrix containing all edge probabilities, from which edges may be generated independently). Their equivalence is trivial, since $E(A(t + \delta t)|A(t)) = \sum_B B\mathcal{P}_{\delta t}(B|A(t))$, and

$$\mathcal{P}_{\delta t}(B|A(t)) = \prod_{i_1=1, i_2=i_1+1}^{N-1, N} (W)_{i_1, i_2}^{(B)_{i_1, i_2}} \cdot (1 - (W)_{i_1, i_2})^{1 - (B)_{i_1, i_2}},$$

where $W = E(A(t + \delta t)|A(t))$. Hence we shall specify our model for the stochastic network evolution via

$$E(A(t + \delta t)|A(t)) = A(t) + \delta t \mathcal{F}(A(t), \mathbf{X}(t)),$$

valid as $\delta t \rightarrow 0$. Here the real matrix valued function \mathcal{F} is symmetric; it has a zero diagonal, and such that all elements of right hand side are in $[0, 1]$. We write

$$\mathcal{F}(A(t), \mathbf{X}(t)) = -A(t) \circ \{\text{Death rates}\} + (\mathbf{1} - A(t)) \circ \{\text{Birth rates}\}.$$

Symmetry is a powerful consideration here. If there are no distinguished individuals then the dynamic must be invariant under any permutation of the vertices. So for example if Q is an $N \times N$ permutation matrix then we must have

$$\mathcal{F}(A, \mathbf{X}) = Q^T \cdot \mathcal{F}(Q \cdot A \cdot Q^T, Q \cdot \mathbf{X}) \cdot Q, \quad \text{for all } A, \mathbf{X},$$

This is not as restrictive as it appears. Forms involving polynomials, or Hadamard products of polynomials in A all satisfy this restriction. In [GHP] this symmetry suggested a raft of mean field approaches.

The dependence of \mathcal{F} upon \mathbf{X} is critical since it fully couples the system. We have in mind a homophily effect as mentioned in the introduction, where individuals having similar states are more likely to become directly connected and less likely to become unconnected. So if all the \mathbf{x}_i s were the same, then A will gain in edge density and approach the n -clique, $\mathbf{1}$. On the other hand if subgroups are relatively dissimilar in their states, then they should become disjoint.

We shall thus assume a simple form:

$$E(A(t + \delta t)|A(t)) = A(t) + \delta t \cdot (-A(t) \circ (\mathbf{1} - \Phi(\mathbf{X}(t))) \cdot \omega + (\mathbf{1} - A(t)) \circ \Phi(\mathbf{X}(t)) \cdot \delta). \quad (3)$$

Here δ and ω are positive constants representing the maximum birth rate and maximum death rate respectively; and the *pairwise similarity* matrix, $\Phi(\mathbf{X}(t))$ is such that each term, $\Phi(\mathbf{X}(t))_{i,j} \in [0, 1]$ is a monotonically decreasing function of a suitable semi-norm,

$$\|\mathbf{x}_j(t) - \mathbf{x}_i(t)\|,$$

measuring the similarity between the corresponding pairs of vertex-states. So edges between vertices with similar states have a relatively low death rate and a relatively high birth rate. For instance we might take $\Phi(\mathbf{X}(t))_{i,j} \sim 1$ for $\|\mathbf{x}_j(t) - \mathbf{x}_i(t)\| < \epsilon$, and $= 0$ otherwise, for some suitably chosen $\epsilon > 0$.

Now, there are equilibria at $\mathbf{X} = \mathbf{X}^*$ with either $A = 0$ or $A = \mathbf{1}$. There may well be others. To analyse their stability, let us assume that δ and $\omega \rightarrow 0$. Then $A(t)$ evolve slowly via (3) and we may consider the stability of the uniform population, X^* under the fast dynamic (1), for any fixed network, A .

Writing $\mathbf{X}(t) = \mathbf{X}^* + \tilde{\mathbf{X}}(t)$ and linearizing (2) at \mathbf{X}^* , we obtain

$$\dot{\tilde{\mathbf{X}}} = \mathbf{df}(\mathbf{x}^*)\tilde{\mathbf{X}} - D\tilde{\mathbf{X}}\Delta. \quad (4)$$

A stability analysis of (4) is straightforward. Let $(\lambda_i, \mathbf{w}_i) \in [0, \infty) \times \mathbb{R}^N$, $i = 1, \dots, N$ be the eigen-pairs for Δ , the Laplacian of A ; and employ the eigen-decomposition:

$$\tilde{\mathbf{X}}(t) = \sum_{i=1}^N \mathbf{u}_i(t) \cdot \mathbf{w}_i^T,$$

where each $\mathbf{u}_i(t) \in \mathbb{R}^m$. It follows, by similarly decomposing (4), that

$$\dot{\mathbf{u}}_i = (\mathbf{df}(\mathbf{x}^*) - D \cdot \lambda_i) \cdot \mathbf{u}_i.$$

Thus \mathbf{X}^* , the uniform equilibrium, is asymptotically stable only if all N matrices, $\mathbf{df}(\mathbf{x}^*) - D \cdot \lambda_i$, are simultaneously stability matrices; and conversely is it unstable in the i th mode of the graph Laplacian if $\mathbf{df}(\mathbf{x}^*) - D \cdot \lambda_i$ has an eigenvalue with positive real part.

The spectrum of $\mathbf{df}(\mathbf{x}^*) - D \cdot \lambda$ can be viewed as a function of real λ , a candidate eigenvalue of Δ . If λ is small then this is dominated by the stability of the autonomous system, $\mathbf{df}(\mathbf{x}^*)$. If λ is large then this is again a stability matrix, since D is positive definite. The situation, dependent on some interplay between choices of D and $\mathbf{df}(\mathbf{x}^*)$, where there is a *window of instability* for an intermediate range of λ , within which $\mathbf{df}(\mathbf{x}^*) - D \cdot \lambda$ has an eigenvalue with positive real part, is known as a Turing instability, see [9]). Note that as $A(t) \rightarrow \mathbf{1}$, we have $\lambda_i \rightarrow N$, for $i > 1$. So if N lies within the window then we are assured that the system can never reach a stable, consensual equilibrium. Turing instabilities can drive the breakup (weakening) of the network into relatively well-connected subnetworks. These in turn may re-stabilize the equilibrium dynamics (as the eigenvalues leave the window of instability), and then the whole process can begin again as homophily causes the absent edges to reappear. Thus we anticipate pseudo-cyclic emergence and diminution of patterns, representing transient variations in attitudes.

4. Schnackenberg dynamics

To be more concrete here we shall assume a specific form for the activator-inhibitor dynamics, though the process we have described can work for a general class of such systems. We

consider the Schnackenberg model, which is minimal in that $m = 2$, and $\mathbf{x} = (x_1, x_2)^T$, for each node. It is given by

$$\mathbf{f}(\mathbf{x}) = (p - x_1x_2^2, q - x_2 + x_1x_2^2)^T,$$

where p and q are positive constants. It has the the equilibrium

$$\mathbf{x}^* = \left(\frac{p}{(p+q)^2}, p+q \right).$$

Consider (1) with $D = \text{diag}(d_1, d_2)$. We have

$$\mathbf{df}(\mathbf{x}^*) = \begin{pmatrix} -(p+q)^2 & -\frac{2p}{p+q} \\ (p+q)^2 & \frac{p-q}{p+q} \end{pmatrix}.$$

This is a stability matrix if and only if

$$(p+q)^3 > (p-q). \quad (5)$$

Now consider $(\mathbf{df}(\mathbf{x}^*) - \lambda_i D)$. Its eigenvalues, say σ_{\pm} , satisfy

$$0 = \sigma^2 - \sigma(-(p+q)^2 + \frac{p-q}{p+q} - \lambda_i(d_1 + d_2)) + h(\lambda_i)$$

where $h(\lambda) = d_1d_2\lambda^2 - \lambda(d_1\frac{p-q}{p+q} - d_2(p+q)^2) + (p+q)^2$. Note, since $\lambda_i > 0$ we have $\sigma_+ + \sigma_- < 0$, and hence there is a loss in stability only when $h(\lambda_i) < 0$. This occurs if and only if $\lambda_i \in (\lambda_-, \lambda_+)$, say, where

$$\lambda_{\pm} = \frac{d_1\frac{p-q}{p+q} - d_2(p+q)^2 \pm \sqrt{(d_1\frac{p-q}{p+q} - d_2(p+q)^2)^2 - 4(p+q)^2d_1d_2}}{2d_1d_2}. \quad (6)$$

Now suppose $p > q > 0$, satisfying $(p+q)^3 > (p-q)$, and d_1 and d_2 are such that both

$$d_1 > \frac{(p+q)^3}{n(p-q)} \text{ and } d_2 < \frac{d_1n(p-q) - (p+q)^3}{n(p+q)(d_1n + (p+q)^2)},$$

then we will have $n \in (\lambda_-, \lambda_+)$, and consequently our system will reach this window of instability as the attributes approach their stable equilibrium and A approaches the clique $\mathbf{1}$, $\lambda_i = n$ for $i \neq 1$. Thus the uniform steady state for the fully connected connected clique is unstable in that case.

Under these conditions as A approaches $\mathbf{1}$, the fully coupled system must undergo a Turing instability in at least one eigenmode (of the associated Laplacian) and consequently the states of at least some of the vertices will begin to diverge. If $\epsilon > 0$ is so small that this will be enough to switch the homophily term then some edges will tend to expire once their end vertex states have diverged sufficiently. Eventually that instability will be reversed as all eigenvalues will leave the window (λ_-, λ_+) , and then all of the state start to converge again. So such a system is condemned to a such a pseudo periodic existence.

5. Projections

Here we shall present some simulations of the system. Since the output is a very long series of very large matrices, which themselves are difficult to visualize, we shall employ some different projections to capture the structure of our network at each time step, ultimately to show that it is pseudo periodic.

Our projections are of the form

$$A \rightarrow P_B(A) = (A : B)B \quad (7)$$

where $:$ denotes the Frobenius inner product, given by $A : B = \sum_{i,j} A_{ij}B_{ij}$, A is an adjacency matrix, at any time, and B is a given real symmetric matrix, normalized so that $B : B = 1$, spanning the projection space. We say $A : B$ is the amplitude of the projection P_B .

To be more specific we denote

$$\hat{\mathbf{1}} = \frac{1}{\sqrt{n(n-1)}}\mathbf{1},$$

the normalized clique matrix, and define a *clique projection*, denoted R , by (7) with

$$R(A) = P_{\hat{\mathbf{1}}}(A).$$

The amplitude of the projection, R , simply counts the edge density in the binary adjacency matrix A .

We generated an evolving network $A(t)$ and states $X(t)$ using small discrete time steps ($\delta t = 0.01$) and the parameter values $N = 40$, $p = 2$, $q = 1$, $d_1 = 2$, $d_2 = 0.005$, $\delta = 0.8$ and $\omega = 0.8$. In this case we started from an Erdos-Renyi network with 50% edge density at its initial timestep. We ran a significant burn-in period before we began taking projections at each time. Figure 1 shows the projection amplitude plotted against time and clearly illustrates the almost periodicity in this network's edge density.

Almost periodic behaviour can usually be observed in complex dynamical systems by projecting the dynamics into two dimensions (as in state space embedding). So we will look for a second projection that is orthogonal to the first.

Suppose that W is real, symmetric, and such that $W : W = 1$ and $W : \hat{\mathbf{1}} = 0$. We shall define our second projection, S , by (7) with $S(A) = P_W(A)$ so that $\hat{\mathbf{1}}$ and $\hat{\mathbf{1}} : W = 0$.

At any time t , $A(t)$, may be decomposed into a combination of our projections S and R , plus error terms, denoted $E(t)$, as follows

$$A(t) = R(A(t)) + S(A(t)) - E(t) \quad (8)$$

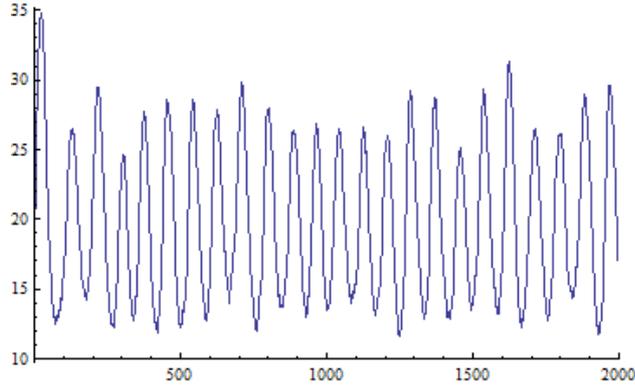


Figure 1: The amplitude of the projection R plotted against time across 2000 time steps, indicating the periodic nature of this network. The projection was applied to a generated evolving network, constructed with parameter values $N = 40$, $p = 2$, $q = 1$, $d_1 = 2$, $d_2 = 0.005$, $\delta = 0.8$ and $\omega = 0.8$.

We define the optimal choice of W as the matrix that represents most of the remaining behaviour of $A(t)$, achieved by minimizing the norm of our error term over a long time interval, $[0, T]$ say. This is given by

$$\int_0^T E(t) : E(t) dt.$$

But since the projections are orthogonal $E(t) : E(t) = (A(t) - R(A(t))) : (A(t) - R(A(t))) - (A(t) : W)^2$. Thus we may define W so as to maximise

$$\int_0^T (A(t) : W)^2 dt \quad \text{subject to } W : W = 1, \quad W : \hat{\mathbf{1}} = 0.$$

Again we are able to observe the almost periodicity of our network by plotting the amplitude of this projection against time, as seen in Figure 2. In Figure 3 we plot the projection amplitudes against each other and observe a circular, almost periodic evolutionary pattern.

In practice we calculate $A_k = A(k\delta t)$ for some small time steps δt and $k = 1, \dots, K$ where $T = \delta t K$. So the integral is replaced by a sum over time steps and this last problem may be solved by a constrained least squares (principal component) procedure.

The use of the projection R is related to the *mean field* approximation given in [4]. This approximates the expected value of the adjacency matrix, and allows us to replace the stochastic dynamics with a deterministic equation for the edge density, as follows. We employ the ansatz

$$E(A(t)) = \rho(t)\mathbf{1},$$

where ρ is some smooth function of time taking values in $[0,1]$. In that case the spectrum of the corresponding expected value for the Laplacian, Δ , consists of a single eigenvalue at

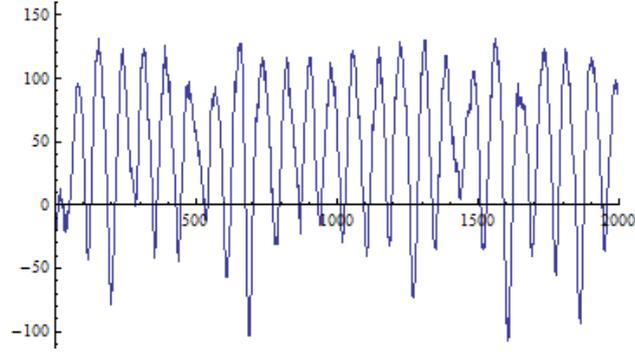


Figure 2: The amplitude of the projection S plotted against time across 2000 time steps, indicating the periodic nature of this network. The projection was applied to a generated evolving network, constructed with parameter values $N = 40$, $p = 2$, $q = 1$, $d_1 = 2$, $d_2 = 0.005$, $\delta = 0.8$ and $\omega = 0.8$.

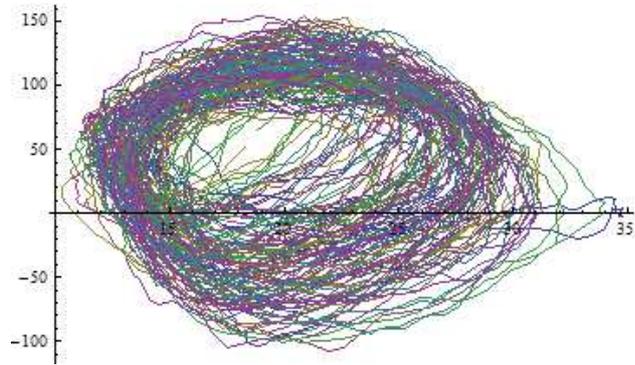


Figure 3: The projection amplitudes plotted against each other at each time step, with the amplitude for projection R along the x-axis and the amplitude for projection S along the y-axis. The plot consists of 2000 time step data point resulting in an anti-clockwise circular pattern that repeats throughout indicating the periodicity of the network's evolution.

0 and $N - 1$ eigenvalues at ρN . In these nontrivial eigenmodes, close to equilibrium, we have an m dimensional linear system $\dot{\mathbf{u}} = (\mathbf{d}\mathbf{f}(\mathbf{x}^*) - \rho ND)\mathbf{u}$. The stochastic equation can be approximated by

$$(\rho(t + \delta t) - \rho(t))\mathbf{1} = \delta t E(-A(t) \circ (\mathbf{1} - \Phi(\mathbf{X}(t))\omega + (\mathbf{1} - A(t)) \circ \Phi(\mathbf{X}(t))\delta)).$$

If the expectation on the right hand side is close to the sum of products of the expectations for each factor, then we obtain the approximation

$$(\rho(t + \delta t) - \rho(t))/\delta t = -\rho(t)(1 - \phi)\omega + (1 - \rho(t))\phi\delta.$$

Here ϕ must represent a mean-field approximation to the average of the terms held within $\Phi(\mathbf{X}(t))$. This must be some monotonically decreasing function of $\|\mathbf{u}\|$, that is equal to unity at zero (the equilibrium) and switches rapidly to zero at some threshold value $\hat{\epsilon} > 0$. The precise details must depend on the differences between pairs of elements drawn from the

Laplacian's normalized eigenvectors. Letting $\delta t \rightarrow 0$, we obtain the deterministic system

$$\begin{aligned}\dot{\rho} &= -\rho(1 - \phi(\|\mathbf{u}\|))\omega + (1 - \rho)\phi(\|\mathbf{u}\|), \\ \dot{\mathbf{u}} &= (\mathbf{d}\mathbf{f}(\mathbf{x}^*) - \rho ND).\mathbf{u}.\end{aligned}$$

For example we may set $\phi(\|\mathbf{u}\|) = (1 - \tanh(\frac{\|\mathbf{u}\| - \hat{\epsilon}}{\mu}))/2$, where $\hat{\epsilon}$, $\mu > 0$, chosen so that ϕ switches rapidly, as desired, as $\|\mathbf{u}\|$ increases through $\hat{\epsilon}$. Taking suitable values, numerical solution shows that this last systems possesses periodic orbits.

6. Conclusion

These models show that when individuals, who are each in a dynamic equilibrium between their activational and inhibitory tendencies, are coupled pairwise in a homophilic way, then we should expect a relative lack of global social convergence to be the norm. Radical and conservative behaviours can coexist across a population and are in a constant state of flux. While the macroscopic situation is predictable, the journeys for individuals are not, within both deterministic and stochastic versions of the model. There are some commentators in socio-economic fields who assert that divergent attitudes, beliefs and social norms require leaders and are imposed on populations; or else they are driven by partial experiences and events. But here we can see that the transient existence of locally clustered subgroups, holding diverse views, can be an emergent behaviour within fully coupled systems. This can be the normal state of affairs within societies, even without externalities and forcing terms.

Even if the stochastic dynamics for $A(t)$ are replaced by deterministic dynamics for a weighted communication adjacency matrix, one obtains a system that exhibits a-periodic, wandering and also sensitive dependence. This alternative model is discussed in [10]. In such cases the orbits are chaotic: we know that they will oscillate but we can not predict whether any specific individuals will become relatively inhibited or relatively activated within future cycles.

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