

Proyectos de Sistemas Complejos

RUBÉN CALVO IBÁÑEZ

CARLES MANUEL MARTORELL ARGEMÍ

Universidad de Granada

February 25, 2023

CONTENTS

I Pattern formation in ecological systems	1
II Pedestrian synchronization and the wobbling of the Millenium Bridge	3
III Stem Cell differentiation: multi-lineage priming and the Waddington Landscape	4
i The 2-dimensional switch-like model	5
ii The Waddington Landscape:	6
IV Game Theory	9
i Nowak's article: evolution of cooperation	10
ii Stochastic evolutionary Game Theory	11
V Viczek's system: Studying the dynamics of flocks	13
VI Neural network: a firing-rate model	16

I. PATTERN FORMATION IN ECOLOGICAL SYSTEMS

In this project, we focus on ecological systems, and in particular, in vegetation dynamics. The starting point is to understand the mechanisms behind the formation of spatial patterns in the vegetation (Figure 1). The spatial distribution of the vegetation is the consequence of a complex interaction mechanism between the flora and the resources of the environment, in such a way that the vegetation is organized to make the most of the resources without depleting them (to go deeper into this, see the Rietkerk article [16]). If the resources are abundant there is no limitation in the system, and therefore, the vegetation is distributed uniformly in space; however, when resources are limited, the ecological system has to self-organized leading to the emergence of spatial patterns. Examples of vegetation patterns are found in arid climates, displaying circular structures, stripes, labyrinths, etc.

From the point of view of Dynamical Systems, the resource-vegetation interaction can be "encoded" in the form of non-linear differential equations that describes the evolution, both in space and in time, of two fields: vegetation density and resource density. From this perspective, the states of the system (homogeneous vegetation, system without vegetation, or even the appearance of patterns) are encoded as stationary solutions of the dynamical system.

The system we propose is based on the seminal Klausmeier's article [11] in which the interaction of a single species of vegetation and a resource (water) is studied. The vegetation density is described by the field $n(t, x)$ and the water density by $w(t, x)$; are subject to the following equations:

$$\begin{cases} \frac{\partial w}{\partial t} = a - w - wn^2 + v\frac{\partial w}{\partial x} \\ \frac{\partial n}{\partial t} = wn^2 - mn + \left(\frac{\partial^2}{\partial x^2} + \frac{\partial^2}{\partial y^2}\right)n \end{cases} \quad (1)$$

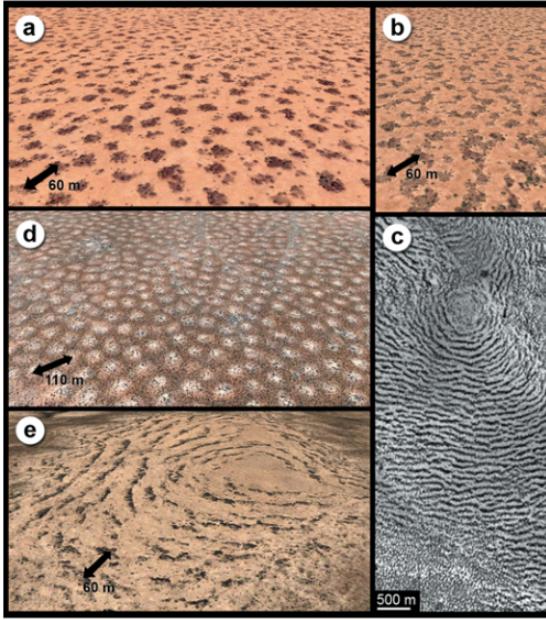


Figure 1: Left figure: Pictures of spatial patterns produced by vegetation systems. Image taken from Deblauwe's article [7]. Right figure: Bifurcation diagram for an ecological system. Obtained from the Rietkerk article [16].

The water emerges uniformly at rate a and flows out of the system at rate $-w$. Vegetation absorbs water at a rate wn^2 , and it grows with the same rate. In turn, the vegetation disappears under a linear rule, mn , where m is the rate of death. Finally, a spatial displacement operator is added to the system: the water flows with velocity v , produced by the groundwater currents; while the vegetation propagates by means of a Laplacian operator (diffusive process).

The objective is to study the previous system of differential equations under the tools of Dynamical Systems, characterizing the fixed points and their stability, and obtaining some condition for the emergence of spatial patterns (Turing patterns). Ultimately, the goal is to obtain simulations of these patterns. Here is a list of some tasks.

1. **Research question 1:** (Easy) Analyze the homogeneous dynamical system (1), that is, without considering the spatial component (spatial derivatives vanish). It is necessary to characterize the fixed points of the system and its stability, and plot the bifurcation diagram of the system. Strogatz's book is a good reference for this part [19].
2. **Research question 2:** (Intermediate) Consider the system spatial-extended system (with spatial components). For this system, characterize the Turing patterns around the unstable points.
3. **Research question 3:** (Intermediate) Finally, we propose the numerical resolution of the system of differential equations (1). There are different ways to do it, however, the easiest is through the *finite differences method* (see, for instance, Toral and Colet's book [21, Ch. 11.]). Once the numerical algorithm is obtained, we can reproduce some Turing patterns for the system (Figure 3 of the article [11]).
4. **Research question 4:** (Hard) An extension of this work is based on the article by Fernandez-Oto [9], in which they consider the intervention of cattle in the Klausmeier equations (1), obtaining a Lotka-Volterra system. This research question proposes reproducing the results from the cited article.

II. PEDESTRIAN SYNCHRONIZATION AND THE WOBBLING OF THE MILLENIUM BRIDGE

After its opening in June 10, 2000, the famous Millenium bridge in London exhibited some lateral vibration or ‘wobbling’ when a critical number of pedestrians stepped on it [6]. The bridge is essentially a three span shallow suspension bridge, with horizontally suspension cables supporting a 4m wide, lightweight, steel and aluminum deck. When the bridge was crowded, movement of the south and center spans became big enough that pedestrians had to hold onto the balustrades, or stop walking to retain balance. Observations on that day and studies of video footage revealed that the oscillations came to have a maximum amplitude of around 5 cm when occupied by around 150-200 people. The first scientific papers aiming at reproducing and understanding the underlying mechanism of this wobbling proposed a Kuramoto-based model [20] in which pedestrians are represented by harmonic oscillators fulfilling the Kuramoto equations [1]. The schematics of the model are shown in Figure 2.

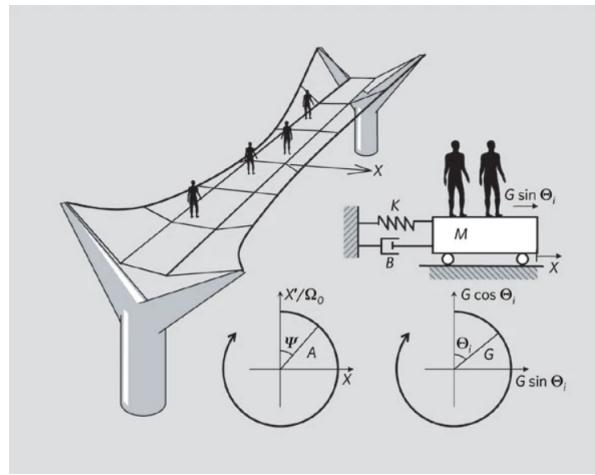


Figure 2: Schematics of the Millenium suspension bridge. On the right, we see a pair of pedestrians exerting force on the deck of the bridge when walking.

We suggest the following lines of research:

1. **Research question 1:** (**Easy**) Can we explain the wobbling of the bridge in terms of a Kuramoto model of coupled oscillators? Can we use this model to calculate the critical number of pedestrians that forced these oscillations (approximately 150-200 people)? In order to solve this, study the article by Strogatz et al [20] (more information in the Supplementary material). Solve the Kuramoto differential equations numerically in order to reproduce the main Figure of the article (see Figure 3), that shows how the amplitude of lateral oscillations starts to raise when a critical number of pedestrians are walking on the bridge.
2. **Research question 2:** (**Intermediate**) Can we understand analytically the synchronization mechanism using averaging theory? In particular, can we calculate the critical number of pedestrians inducing wobbling into the bridge? These are the calculations that Strogatz and his colleges carry out in the Supplementary Information. You can also read a bit about averaging theory in the book by Strogatz [19].
3. **Research question 3:** (**Hard**) In a more recent article, Belykh et al proposed a different model of pedestrian synchronization based on inverted pendulums [5]. This model is later simplified into a Van-der Pol-type walker model, which is the one used to study lateral oscillations. In this paradigm, are lateral oscillations exhibited? Do phase-locking solutions exist? (Careful with this article, it might be tricky to set the initial conditions of the model. If you cannot reproduce the results, contact with us).

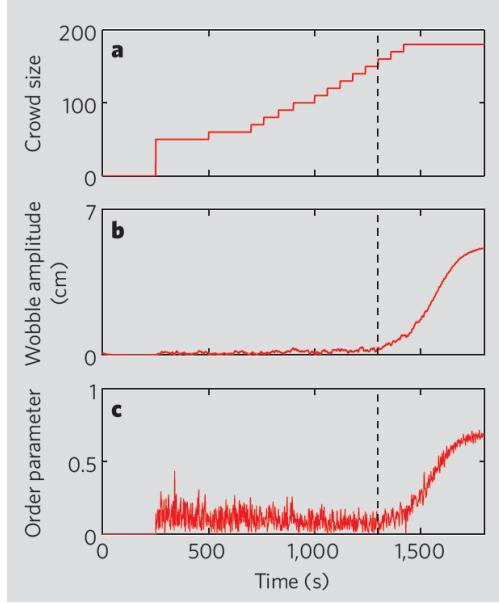


Figure 3: In this figure, we represent: in the upper panel, the crowd size as a function of time (the amount of people in the bridge: they are added periodically); in the intermediate panel, the wobble amplitude, in cm, showing how it grows after a critical number of pedestrians are added; in the lowest panel, the order parameter of the Kuramoto model, showing how pedestrians spontaneously synchronize.

III. STEM CELL DIFFERENTIATION: MULTI-LINEAGE PRIMING AND THE WADDINGTON LANDSCAPE

Pluricellular organisms come from a unique cell called zygote, that proliferates to create a mass of stem cells that organize, diversify and specialize to generate the different tissues conforming the body. During this process of development, stem cells start to differentiate, slowly acquiring characteristics that can eventually allow them to fulfill a specific function on the pluricellular organism. During the 50s, the biologist Conrad Waddington developed a popular metaphor to illustrate the process of cell differentiation: a cell experiences a series of changes as a result of its irreversible movement along a static landscape, that drives it to stable valleys that can be thought of as differentiated states, much like a marble rolling through an irregular terrain. This metaphor is still used to explain the differentiation process, but with new insights that have been derived by applying the standard techniques of dynamical systems and stochastic processes. Now, this static landscape has been replaced by a dynamical landscape, that usually changes as a result of an external parameter that fluctuates, inducing a bifurcation. This line of thought is schematized in Figure 4.

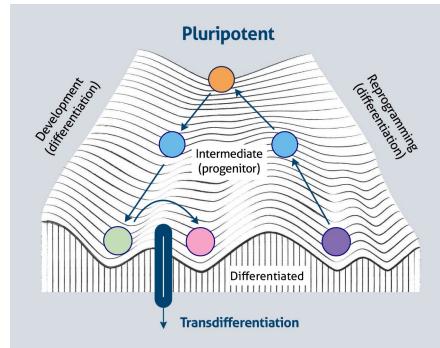


Figure 4: Drawing of the differentiation process in the Waddington picture. The pluripotent cell differentiates in its way down the epigenetic landscape.

i. The 2-dimensional switch-like model

A very simple model for stem cell differentiation is proposed in [3]. Here, we have a very simple Gene Regulatory Network with two transcriptor factors, X and Y, and two promoters, A and B. The promoter binding and unbinding, subsequent self-activation, inhibition, dissociation and the degradation reactions for X and Y are summarized on the following table:

Table 1: Reactions for the Switch-Like model

$A + R \xrightarrow{k_{AR}} A + R + X$	Generation of X
$B + R \xrightleftharpoons{k_{BR}} B + R + Y$	Generation of Y
$X \xrightarrow{k_X^-} \emptyset, Y \xrightarrow{k_Y^-} \emptyset$	Degradation of X,Y
$A + X \xrightleftharpoons{k_{AX}^{+-}} AX$	Binding k_{AX}^+ , unbinding k_{AX}^-
$A + Y \xrightleftharpoons{k_{AY}^{+-}} AY$	Binding k_{AY}^+ , unbinding k_{AY}^-
$AX + R \xrightarrow{k_X^+} AX + R + X$	Activation of X
$BY + R \xrightarrow{k_Y^+} BY + R + Y$	Activation of Y

We suggest the following research lines related with the model of [3]:

1. **First task:** (Easy) Using the Gillespie algorithm [10], generate trajectories in the (X, Y) space (the space of concentrations of each transcription factor) by simulating the reactions of table 1. Use the parameters in 2 and study these trajectories in terms of the parameter $\rho = (K_{AX}^-/K_{AX}^+)(K_{AY}^+/K_{AY}^-)$, as in the article.

Table 2: Parameters for the Switch-Like model

$R = 100$	$A_0 = B_0 = 1, X_0 = Y_0 = 0$
$k_X^- = k_Y^- = 0.01$	$k_{AR} = k_{BR} = 0.01$
$k_X^+ = k_Y^+ = 0.01$	$k_{AX}^+ = k_{BY}^+ = 1$
$k_{AY}^+ = k_{BX}^+ = 1$	

Question: What can you say about **transdifferentiation** in the case in which $\rho > 1$?

2. **Second task:** (Intermediate) If you are here, you were able to simulate the reactions of the Gene Regulatory Network correctly. I congratulate you personally. Now, let's do a bit of statistical physics. First, compute the stationary probability by letting the system evolve over long times and then calculating the likeliness that the system states in each particular point of the phase space. This is easy by just counting the time (the time between reactions in the Gillespie algorithm) the system spends at a particular configuration of (X, Y) before changing. Use this to calculate the potential energy (similarly to a Boltzmann distribution): $V(\mathbf{X}) = -\log(P_{st}(\mathbf{X}))$ (see Figure 5).

Question: What can you say about **multi-lineage priming** in the case in which $\rho = 1$?

3. **Third task:** (Intermediate-Theoretical) Use a mean-field approach to compute the differential equations that give the deterministic behavior of the gene regulatory network. Calculate its fixed points and study the stability. Compare with the results of the last task.

Question: Can you relate these mean-field equations with the famous Hill-equations, that describe promoter and transcriptor factor binding-unbinding process?

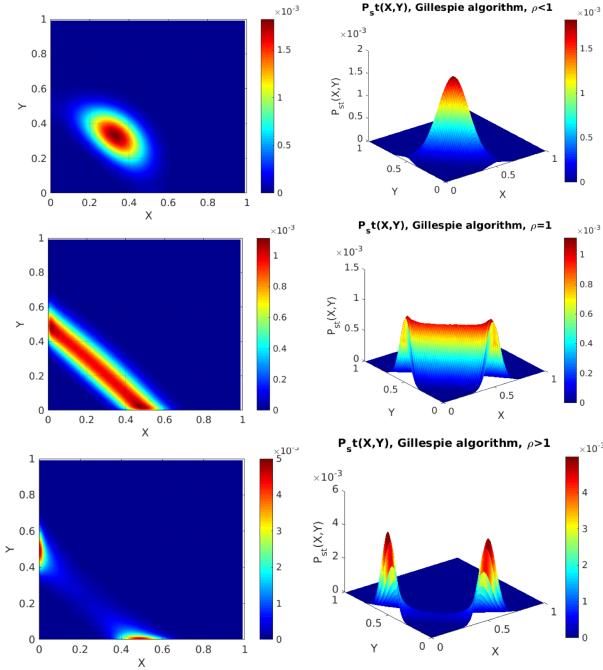


Figure 5: Probability landscape of the differentiation process in terms of the parameter ρ .

ii. The Waddington Landscape:

Another simple model of stem cell differentiation is the one in [25]. In this case, the process of differentiation is already described with a mean-field deterministic approach using the Hill equations:

$$\begin{cases} \frac{dx_1}{dt} = \frac{a_1 x_1^n}{S^n + x_1^n} + \frac{b_1 S^n}{S^n + x_2^n} - k_1 x_1 \\ \frac{dx_2}{dt} = \frac{a_2 x_2^n}{S^n + x_2^n} + \frac{b_2 S^n}{S^n + x_1^n} - k_2 x_2 \end{cases} \quad (2)$$

where x_1, x_2 are the expression of each gene, a_i are the auto-activation parameters, b_i are the mutual-inhibition parameters and k_i are degradation rates. In vector form, this can be rewritten as: $\dot{\mathbf{x}}(t) = \mathbf{F}(\mathbf{x})$. It is usual to consider the symmetric situation in which $a = a_1 = a_2$, $b = b_1 = b_2$, and $k = k_1 = k_2$. In the case of stem-cell differentiation, it is typical to set $n = 4$, since the dynamical system then exhibits three fixed points, that can be understood as the stem-cell (non-differentiated) state when $x_1 = x_2$, and the differentiated states corresponding to the case in which one gene has null expression.

We suggest the following research lines related with the model of [25]:

1. **First task: (Easy)** Study the system of differential equations and find the bifurcation diagram in terms of a . Find the critical value of a at which the non differentiated state becomes unstable (see Figure 6). If you value your time in this Earth, don't try to do this analytically.
2. **Second task: (Intermediate)** Study the stationary probability of this dynamical system using the Fokker-Planck equation.

$$\frac{\partial P(\mathbf{x}, t)}{\partial t} = -\frac{\partial}{\partial x_1} (F_1(\mathbf{x}) P) - \frac{\partial}{\partial x_2} (F_2(\mathbf{x}) P) + D \frac{\partial^2}{\partial x_1^2} (P) + D \frac{\partial^2}{\partial x_2^2} (P) \quad (3)$$

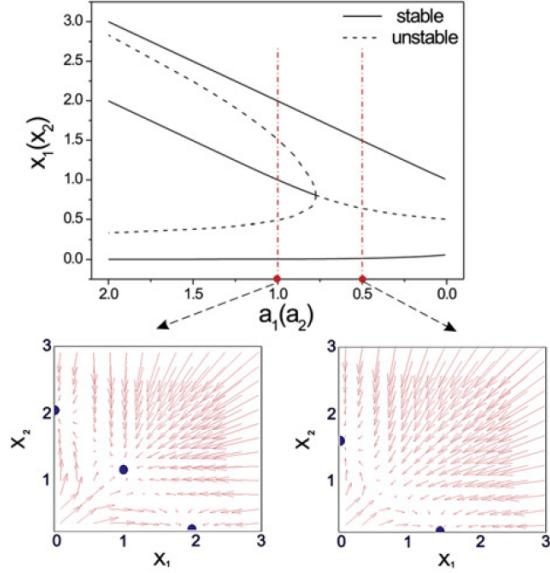


Figure 6: Bifurcation diagram for the Hill equations in terms of the parameter a .

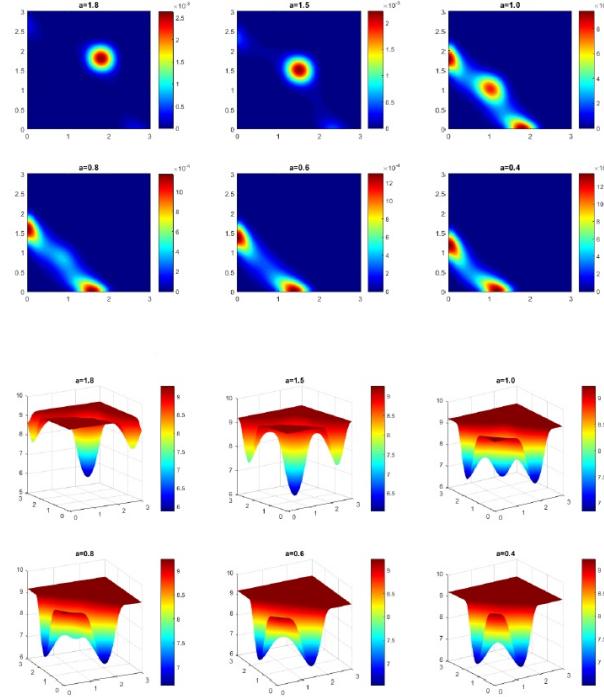


Figure 7: Probability landscape derived from the Fokker-Planck equation as a function of the external parameter a .

This PDE gives the evolution of the probability in terms of the force $F(x)$ of the dynamical system and the diffusion parameter D . Discretize this equation using a finite difference approach and solve it using Dirichlet boundary conditions. Evolve the solution for large times to get the asymptotic behavior or stationary solution when $t \rightarrow \infty$ (take a look at the Supplementary information for this article). You might have to tune the initial condition to get to the desired result (see Figure 7).

3. **Third task:** (Intermediate) Slice the probability landscape appropriately by the line joining the

differentiated state and the non-differentiated state to get a local coordinate x that varies in this hyperplane (see Figure 8). Put together a lot of these to get the dynamical Waddington landscape of Figure 9.

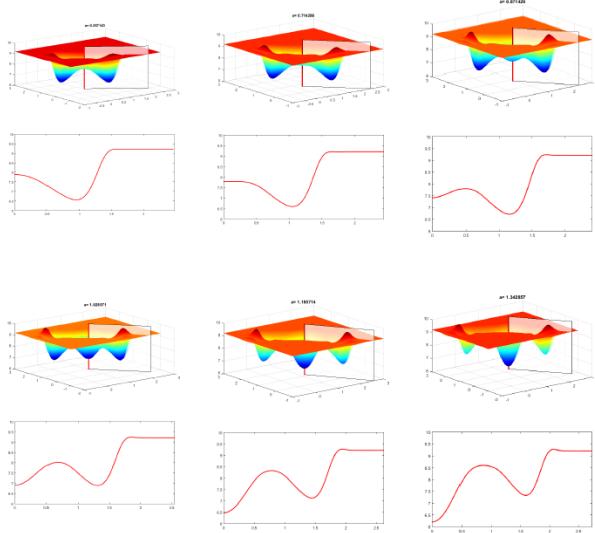


Figure 8: Slicing the probability landscape derived from the Fokker-Planck equation by the line joining the differentiated state and the non-differentiated state. This gives a local coordinate x and a projection of the landscape into this hyperplane.

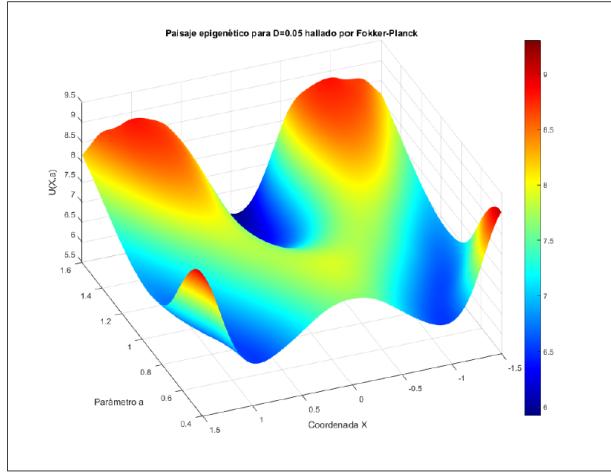


Figure 9: Waddington Landscape obtained as a function of the external parameter a and the local coordinate x of the slicing done in Figure 8

Question: How does the obtained landscape relate with the Waddington original landscape?

4. **Fourth task:** (Hard: only for the brave) Use the path integral approach described in [24] to get to the same result for the epigenetic landscape. In order to find the path that minimizes the action, we suggest you use an annealing method, or a graph-based method (discretize the space and create a graph connecting close regions. Put weights into each edge according to the differential contribution of the path integral. Minimize using Bellman-Ford algorithm or similar).

IV. GAME THEORY

The framework of the following projects is Game Theory. The traditional Theory of Games was developed during the first half of the 20th century, principally by John Von Neumann. In its beginnings, it was devised as a theory to understand human competition in a social background. There, agents were supposed to be **rational**. That is, they took decisions according to a given *plan* or **strategy** in order to maximize their utility function, also called **payoff function**. One example can be a system of individuals competing for some resource, such as money in a gambling game.

Here, we do not focus on traditional Game Theory, but rather on Evolutionary Game Theory (EGT), that was first developed by John Maynard Smith and George Price in the 70s. At first, this theory was mainly applied to biology: usually, animal and plants contests. It has only been recently that this theory has been applied to other fields, like microbiology or economics. A crucial part of EGT is that it does not need any notion of rationality. In this theory, strategies spread because they increase the **fitness** of those phenotypes that play according to it.

The mathematical model of Game Theory (and EGT) is defined according to an interaction rule, which specifies the form of the interplay between different agents. When a pair of individuals interact, each one chooses a strategy to compete; after that, each individual gains (or loses) a resource. For this reason, the interaction is called *game*, as each individual plays with other agents in order to win the resource involved (*pay-off*). For example, for the first project proposed, we focus on a simple model or *game*: the **Prisoner's Dilemma** (PD). It is a "1 vs 1" game on which each player chooses one of both strategies: cooperation or defection. If both players cooperate, they obtain the same pay-off –they distribute the benefits of a cooperative activity; on the contrary, if one of them is a defector, she obtains all the benefits –the defection strategy takes advantage of cooperation. The best way to study the interactions is through the following Table 3. It can be proved that any PD game can be reduced to an equivalent game described by Table 3 –characterized by only one parameter, $b > 0$ [17].

	Cooperation	Defection
Cooperation	1	0
Defection	b	0

Table 3: Pay-offs for the Prisoner's Dilemma. Agent A chooses one strategy on the first column (left) and competes against Agent B, who plays a strategy of the upper row. The values of the Table are the pay-offs obtained by Agent A after playing the game.

Once the interaction rule is defined, we construct the model by defining when the interaction happens between agents. We are interested on the Evolutionary Game Theory, on which the interaction process follows a time-dependent dynamics: at time t , each player interacts with another player; after that, each player updates its strategy depending on the result of the previous interaction. Following this rule, the system of agents evolves reaching a non-trivial state –full-cooperation, full-defection, spatio-temporal patterns, etc., can emerge depending on the network of interactions and the update rule.

A first approach in order to determine the stationary state of the evolutionary PD game is given by means of the *replicator dynamics*. Assuming an infinite number of individuals such that all the players meet randomly, a differential dynamical expression can be obtained in order to characterize the system. This expression is called replicator equation, and it delineates the "averaged" behaviour of the system –as a mean-field approach. For the PD, the replicator dynamics leads to a state of **fully defectors** (indeed, Defection is *Nash equilibrium* –see, for instance, the article of Roca et. al. [17]). Cooperation, under the replicator's assumption, is simply not possible. At this point, we deal with an interesting problem – the *problem of cooperation*: Can we observe cooperation in dynamical Game Theory? The following project is devoted to solve this problem.

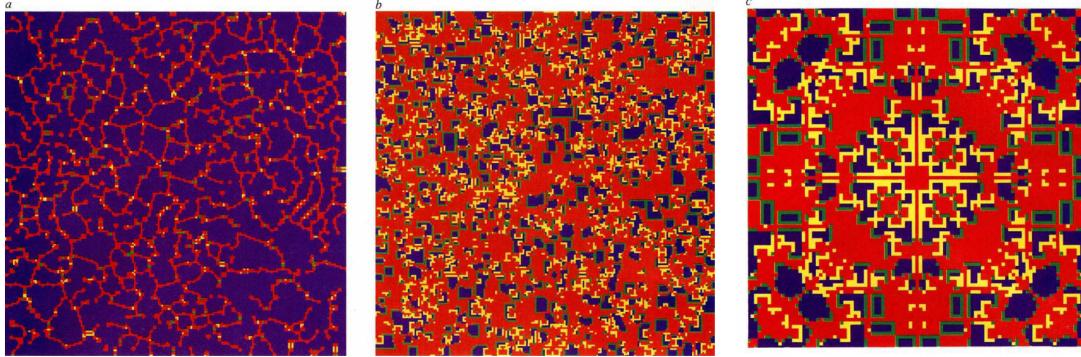


Figure 10: The spatial PD can generate different qualitative patterns depending on the parameter b , which represents the advantage for defectors. This Figure is obtained from [14], with $N = 200 \times 200$ individuals playing the evolutionary PD game in a regular lattice of $k = 8$. We show three snapshots of simulations for different b values. Each image shows the players on the lattice: in blue, cooperators; red, defectors; and yellow/green represents those players who have changed its strategy in the last round. **Image a)**: regime of cooperation, with small b . **Image b)**: Regime of defection. **Image c)**: For larger values of b the stationary pattern loses its stability and the system reaches a chaotic spatio-temporal pattern.

i. Nowak's article: evolution of cooperation

We follow the Nowak's article [14], a seminal work on the investigation of cooperation in networks. The idea is to study the evolutionary Prisoner's Dilemma (PD) in a spatial structure (players are located in a network) such that each individual can only interact with its neighbours. We use the *Unconditional Imitation* (UI) update rule.

We begin with a network with N players whose strategies at initial time $t = 0$ are selected randomly. At time t each player interacts with all its neighbours following the PD game (see Table 3), for a fixed value b . Each interaction is defined as a *simple game*, and the total of games played at time t is called a *round*. We define the total score at time t as the sum of the pay-off obtained at each simple game of this round. At the end of the round, each player compares the total score with its neighbours; hence, the player updates its strategy choosing the strategy of the neighbour who has obtained the best score – unconditional imitation. In this way, the system evolves until it reaches a stationary (or chaotic) spatial pattern (see Figure 10). In general, there are two different phases depending on the value of b : for small values of b , the regime is cooperator-dominated; if b is larger enough, a regime of defector-domination emerges. In order to characterize both regimes we define a phase parameter, the **density of cooperators**,

$$\rho(b) = \frac{N_C(b)}{N} \quad (4)$$

being N_C the number of cooperators at stationarity. We note that N_C depends on the value of b .

- **First task: (Easy):** Write down the *replicator equation* for the PD game [17]. As a 2-dimensional system of differential equations, using the tools of dynamical systems [19], obtain the fixed-point solutions and its stability as a function of b . Demonstrate that only two solutions exist: full-cooperation or full-defection.
- **Second task: (Intermediate):** As a second exercise we propose to reproduce the main results of Nowak's article [14]. In this article, Nowak used a regular lattice of connections, with periodic conditions and $k = 8$ neighbours (each individual is linked with its 8 nearest neighbours). The idea is to characterize both phases. To do that, use the phase parameter (4) to determine the phase transition (Figure 10 in [17]). Why does a chaotic pattern emerge when defectors dominate? We note that the difficulty of this part is to obtain the evolutionary algorithm. Once it is obtained, simulations are straightforward –we encourage you to simulate the chaotic pattern of Figure 10 c). To add more complexity to this part, consider the substitution of the UI update rule [17, Section 4] and study the changes introduced in this way.

- **Third task: (Hard):** For the UI update rule the variance of the network's degree k and the clustering coefficient are the most important parameters to determine the evolution of the system. For this reason we want to analyse different network structures in order to characterize such dependency. We propose two attack lines: (i) consider heterogeneous networks, such as Erdős-Renyi or Barabasi-Albert networks (see, for instance, [2]), and compute the phase-transition for such network architecture; (ii) consider a network of communities (a network divided into different sub-networks with a given structure), hence study the role played by the mesoscopic structure in the context of the evolutionary PD game [13].

ii. Stochastic evolutionary Game Theory

We have already said that EGT deals with the evolution of strategies throughout time. The way that strategies evolve is given by a certain update rule. In the previous example, we worked with a very particular update rule, called unconditional imitation. Here, we take a different road and work in the lines of **Stochastic Evolutionary Games**. To understand this theory, we recommend the excellent review by Traulsen and Hauert [22]. In general, these games are usually defined in terms of **birth-death processes** with a certain transition rate, such that the probability for a certain strategy to reproduce increases with its payoff (payoff translates to fitness). The games are usually assumed to be played on a **Fully connected network**, meaning all individuals interact with each other (this hypothesis is also referred to as the **well-mixed hypothesis**). Suppose that the payoff matrix for the game is:

$$\Pi = \begin{pmatrix} a & b \\ c & d \end{pmatrix} \quad (5)$$

Now, suppose we have a population of N individuals, such that i of them are playing the first strategy (A), $N - i$ are playing the second one (B). The total payoff that an individual playing the first strategy receives, under the assumption of a well-mixed population, is:

$$\pi_A(i) = \frac{i-1}{N}a + \frac{N-i}{N}b \quad (6)$$

While the total payoff for players that adopt the second strategy is:

$$\pi_B(i) = \frac{i}{N}c + \frac{N-i-1}{N}d \quad (7)$$

In this project, we will work with the Fermi game, in which the transition probabilities for the birth-death process are given by the Fermi distribution:

$$T_i^\pm = \frac{i}{N} \frac{N-i}{N} \frac{1}{1 + \exp(\pm\beta\Delta\pi)} \quad (8)$$

where T_i^\pm is the probability of the reaction $i \rightarrow i \pm 1$, β is the intensity of selection (analogous to the inverse temperature) and $\Delta\pi = \pi_A - \pi_B$. You are going to work with stochastic evolutionary Fermi-games, in which the payoff matrix will fluctuate somehow. Your task is to:

- **First task: (Easy):** Using the master equation for the birth-death process and the Kramers-Moyal expansion, derive the following Langevin equation for the frequency $x(t) = i(t)/N$ of the strategy A as a function of time:

$$\dot{x}(t) = (T^+(x) - T^-(x)) + \sqrt{\frac{1}{N}(T^+(x) + T^-(x))}\xi(t) \quad (9)$$

where $T^\pm(x)$ are the transition rates, that now depend on x instead of i . Take the limit $N \rightarrow \infty$ to get the **adjusted replicator dynamics**:

$$\dot{x}(t) = x(t)(1-x(t)) \tanh\left(\frac{\beta}{2}(\pi_A(x) - \pi_B(x))\right) \quad (10)$$

Now, classify the stability of the fixed points of this deterministic equation and check that you obtain the same conditions in the elements of the payoff matrix that you would get using the usual replicator equation [22].

- **Second task:** (Intermediate): Use the theory of stochastic processes to calculate the probability of fixation, which is the probability that a single individual playing strategy A takes over a whole population of individuals playing B [22]. Prove that the expression for this probability is nothing but:

$$\phi_1 = \frac{1}{1 + \sum_{k=1}^{N-1} \prod_{j=1}^k \gamma_j} \quad (11)$$

where $\gamma_j = \frac{T_j^-}{T_j^+}$. Also, prove that the time at which this fixation happens is:

$$t_1 = \phi_1 \sum_{k=1}^{N-1} \sum_{l=1}^k \frac{1}{T_l^+} \prod_{m=l+1}^k \gamma_m \quad (12)$$

- **Third task:** (Intermediate): Follow the article by Aschcroft et al [4] about fixation in fluctuating environments. Using the Gillespie algorithm, generate trajectories of the birth-death process and study the fixation probability and fixation time in terms of the probabilities p_{\pm} to jump from coexistence to coordination. Use the linear theory they propose to calculate the theoretical curves of fixation probability and fixation times (that is, reproduce Figure 3 of the article).
- **Fourth task:** (Hard) Study games in the limit of fast switching, when the elements of the matrix are drawn at random at each generation of the game out of some probability distribution. Use the simple game:

$$\Pi = \begin{pmatrix} b & b \\ 1 & 1 \end{pmatrix} \quad (13)$$

Do the following:

1. First, analyze what is the behavior of this game deterministically in terms of parameter b (use the replicator dynamics).
2. Now, suppose that, at each step, we draw the values of b out of some probability distribution $f(b)$. Define the effective transition rates:

$$T_{i,eff}^{\pm} = E[T_i^{\pm}] = \frac{i}{N} \frac{N-i}{N} \int \frac{1}{1 + \exp(\pm \beta \Delta \pi(b))} f(b) db = \frac{i}{N} \frac{N-i}{N} I^{\pm} \quad (14)$$

We are going to study this game in the space (μ, σ) , where $\mu = E[b]$ and $\sigma^2 = Var(b)$. Define the **line of neutral selection** as the set of points in this space such that the fixation probability is $1/N$ (Why do we say that, at this line, the selection is neutral?). Prove that, for this game, the **line of neutral selection** is found by solving $I^- = 1/2$.

3. Suppose that the elements of b are drawn out of a discrete distribution that can only take two values: b_1 and b_2 with probabilities p_1 and p_2 respectively. Prove that, in this case, the divisor line of neutral selection can be written as:

$$\mu = 1 - \sigma \sqrt{\frac{p_1}{1-p_1}} - \frac{1}{\beta} \ln(f(\sigma)) \quad (15)$$

where $f(\sigma)$ is:

$$f(\sigma) = \left(p_1 - \frac{1}{2} \right) \left(\frac{1}{\gamma(\sigma)} - 1 \right) + \frac{1}{2\gamma(\sigma)} \sqrt{4(\gamma(\sigma) - 1)^2 p_1(p_1 - 1) + (\gamma(\sigma) + 1)^2} \quad (16)$$

and calculate $\gamma(\sigma)$.

4. Simulate the dynamics and obtain Figure 11. Is there bet-hedging in general? Why? Connect this with the shape of the divisor line of neutral selection that you obtained in last section.

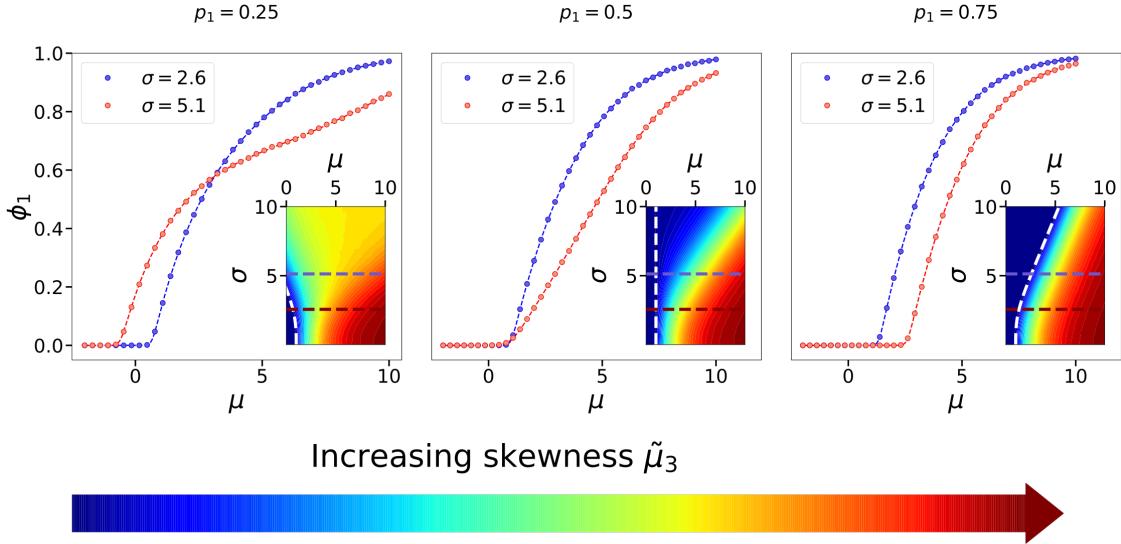


Figure 11: Fixation probability as a function of (μ, σ) for the discrete distribution with only two possible values. Each panel shows the case of a certain probability p_1 . The inset shows the heat map in the (μ, σ) space, while the main plots are sections of this heat map. The dots are from the simulations, the lines are from theory.

V. VICZEK'S SYSTEM: STUDYING THE DYNAMICS OF FLOCKS

In this project we study systems of self-propelled particles, a particular type of non-equilibrium systems on which its constitutive particles move following a dynamical rule. Under certain conditions, the system can display extraordinary collective dynamics, such as highly cooperative motion (*flocking*, in this context) and complex dynamical patterns. The initial idea is to start with a simple model on which we can characterize the different collective behaviour from the point of view of Statistical Physics. For this reason, we focus on the seminal Vicsek's article [23].

We consider a system of N particles that moves on a 2-dimensional space, let's say on a square of size L . Each particle moves with constant velocity and interacts with neighbouring particles to align the direction of motion to the mean direction of the local flock. There exist multiple *rules* in order to define the interaction between particles and to establish the criterion to update the direction of the particle. Nevertheless, for different rules, the main ingredient is given by an additive noise introduced on the direction of the particle (which can be thought as an error on the estimation of the new direction). Depending on the level of this noise, and the density of particles, two different phases appear: (i) a disordered phase, given by a large noise term, on which each particle moves independently; (ii) an ordered phase, for small noise term, on which some clusters or flocks of particles appear, moving as a collective system.

In the following, we will define the dynamical rule followed in the Vicsek's article. Particles are represented by points moving continuously (off lattice) on the square of size L . We define $r = 1$ as the unit measure of distance, and $\Delta t = 1$ the time unit (which describes the time between two updates of the system). The position of i -th particle at time $t = 1, 2, 3, \dots$ is described by the vector $\mathbf{x}_i(t)$; in addition, its velocity is given by vector $\mathbf{v}_i(t)$. At time $t + 1$, the position of its particle is updated according to the following rule. The position of i -th particle is transformed by equation

$$\mathbf{x}_i(t+1) = \mathbf{x}_i(t) + \mathbf{v}_i(t)\Delta t$$

The new velocity $\mathbf{v}_i(t+1)$ is given by a constant absolute value v , and a direction given by the angle $\theta_i(t+1)$. This angle is obtained by the equation

$$\theta_i(t+1) = \langle \theta \rangle_r + \Delta\theta_i$$

where $\langle \cdot \rangle_r$ denotes the average of the velocities of particles (including particle i) being within a circle

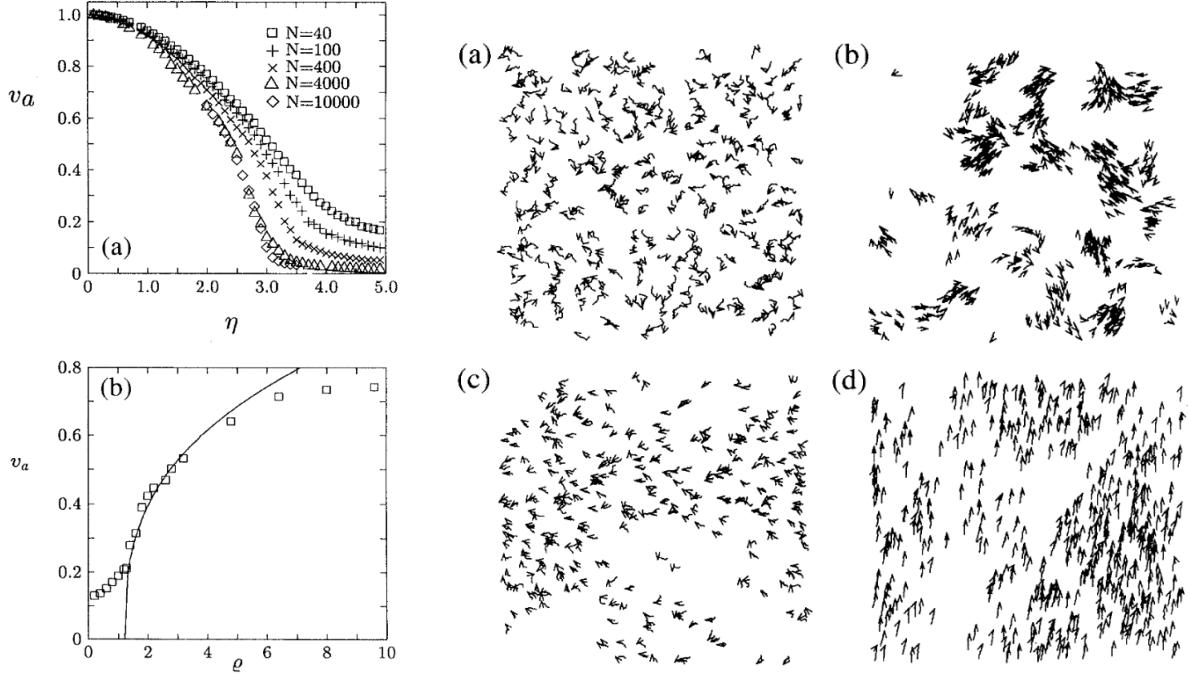


Figure 12: Figures form the article [23]. **First column:** Phase transition for parameters η and ρ for the Vicsek's model. **Second and third column:** different snapshots of a given realization of the model. Particles are depicted as arrows, indicating the direction of the motion. At some time, particles creates a flock leading to a collective pattern.

of radius r surrounding the given particle. The term $\Delta\theta_i$ is a random number chosen with a uniform probability from the interval $[-\eta/2, \eta/2]$.

This simple model is defined by three parameters: the noise value η , the density of particles $\rho = N/L^2$, and the absolute value of the velocity v . Using these values as control parameters, we can characterize the phase transitions of the system from the disordered phase to the ordered phase. To do that, we construct an order parameter v_a defined as the mean value of the global velocity,

$$v_a = \frac{1}{Nv} \left| \sum_{i=1}^N \mathbf{v}_i \right|$$

The v_a value of the system depends only on the three parameter η , ρ and v ; hence, it can be used to characterize the state of the system. In the ordered phase, where all the particles move together, $v_a = 1$; on the other hand, in the disordered phase, $v_a = 0$ (See Figure 13).

For this model, we propose the following tasks.

- **First task: (Easy)** The idea is to obtain all the figures which appear in the Vicsek's article [23]. In particular, we expect to obtain the phase-transition varying the noise parameter η and the density of the system, ρ (Figure 13). The difficulty of this part is given in the update algorithm; but once the algorithm is obtained, simulations are straightforward.
- **Second task: (Intermediate)** In the thermodynamic limit, Vicsek's model exhibits a phase transition analogous to the continuous phase transition in equilibrium system (in particular, the Ising model), hence close to criticality the order parameter behaves as a power-law with an universal coefficient, i.e.

$$v_a \sim [\eta_c - \eta]^\beta \text{ and } v_a \sim [\rho - \rho_c]^\delta.$$

- We propose to obtain the critical exponents β and δ for the Vicsek's model (Figure 3 in article [23]).
- **Third task: (Hard)** As a final task, it is interesting to modify the previous model adding a new rule. Indeed, there exist many articles dedicated to the *improvement* of the Vicsek's model. However, we

propose one attack line: to add the effect of visual interaction. Following this rule, each particle interacts only with the particles that are in its *cone of vision*. This cone of vision is determined by a visual angle, which adds a new control parameter to be considered. The article of Durve and Sayeed [8] analyzes this kind of models.

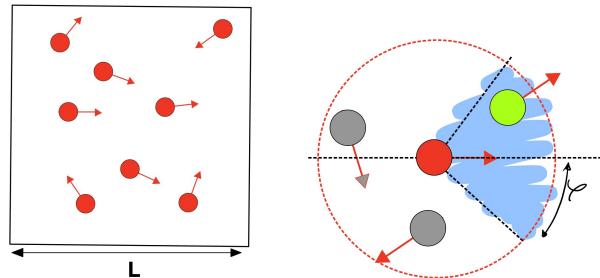


Figure 13: Characterization of the Cisek's model with visual interaction. Particles are confined in a square box of size L . Each particle has a visual cone given by the visual angle φ , in such a way that it only interacts with particles inside of the visual cone –as it is depicted in the image, the red particle only interacts with the green particle.

VI. NEURAL NETWORK: A FIRING-RATE MODEL

In these lines we study a simple dynamical model which tries to simulate neural activity. We consider a set of N neurons (or nodes) connected by an adjacency matrix W , such that the connection between nodes i -th and j -th is given by a link with weight $W_{ij} \in \mathbb{R}$. In this way, the set of nodes and the adjacency matrix W defines our system: the neural network. For each neuron, we define a firing-rate $r_i(t)$, for $i = 1, \dots, N$, which establishes the spiking neuron ratio at time t . We use a simple dynamical equation to determine the firing rate of each neuron,

$$\frac{dr_i(t)}{dt} = -r_i(t) + \phi \left(g \sum_{j=1}^N W_{ij} r_j(t) + I \right) + \xi_i(t) \quad (17)$$

This system is called *firing-rate model*, and it is the simplest neural model. We note that the firing-rate of neuron i -th depends on the interaction with other neurons by means of the coupling term $W_{ij} r_j(t)$. The parameter g measures the coupling strength, and I is an external input ($I \in \mathbb{R}$). The values of the links W_{ij} will be obtained from a random distribution. The variable $\xi(t)$ is, indeed, a stochastic process introducing random fluctuations on the dynamics – we will set to 0 this term to simplify the calculations. Finally, the function $\phi(x)$ is known as *saturation function*, which adds a non-linearity on the differential equation in order to bound the trajectories on a limited area. In this project, we define it as a hyperbolic function $\phi(x) = \tanh(x)$.

On the following, we set $I = 0$ and $\xi = 0$ in order to simplify our model. A firing-rate model, under these assumptions and due to its simplicity, displays only two different regimes of activity: (i) one called *quiescent regime*, on which the activity of each neuron is 0; (ii) and an *active regime*, on which each neuron takes a non-trivial value (and depending on the shape of matrix W it can be chaotic [18]). The phase transition is controlled by parameter g , the coupling strength; and, the critical value can be obtained by linear stability analysis. Indeed, the critical value depends on the larger eigenvalue of matrix W , such that $g_c = 1/\lambda_M$ –where λ_M is the larger eigenvalue. Therefore, we can conclude that the dynamics of this firing-rate model is governed by the shape of matrix W , and in particular, by its distribution of eigenvalues. We are interested in studying the phase transition depending on the form of the matrix W . The most simple model used in neuroscience to represent synaptic strength variability is described by a fully-connected matrix of independent, identically distributed random variables (usually Gaussian). In this case, the distribution of eigenvalues is known to obey the celebrated circle's law [15], which states that, in the limit $N \rightarrow \infty$, the eigenvalues are uniformly distributed in a circle that has a radius proportional to the standard deviation of the synaptic weight distribution: $R = \sqrt{N\sigma^2(W_{ij})}$. Usually, one takes $\sigma(W_{ij}) = J/\sqrt{N}$ (J is a free parameter) in order to have a radius that does not scale with system size: $R = J$.

When studying the phase transition, it is commonly used in the literature the classification into two *types of criticality* depending the distribution of eigenvalues of the adjacency matrix W . As we have pointed out, the transition between the quiescent and active phase depends on the larger eigenvalue of W ; nevertheless, the other eigenvalues can play an important dynamical role. If the larger eigenvalue is isolated, and a considerable distance separates this eigenvalue (known as *outlier*) and the rest of the eigenvalues (which forms a "bulk"), then we say that the system displays *type I criticality*. It is characterized by a phase transition to a synchronous regime. On the other hand, if the larger eigenvalue is not isolated and it belongs to the "bulk" of eigenvalues, we say that the system displays *type II criticality*. It is characterized by a transition to a non-synchronous regime (see Figure 14).

For this project, we propose to study the system (17) for different shapes of W . To do that, we follow the article of Li and Shew [12] (note that they used a discrete-time model rather than our system (17)). We construct the adjacency matrix W following a random distribution of weights W_{ij} that obeys the *Dale's law*¹. The protocol is as follows: for a network of N neurons, each neuron connects to each other neuron with a probability $p = 0.2$ (a disconnected link, say the i - j connection, implies $W_{ij} = 0$). A fraction $\alpha = 0.2$ of the neurons are inhibitory with outgoing synapse strength drawn from a uniform distribution $[-w, 0]$,

¹There are two types of neurons, excitatory (E) and inhibitory (I). The Dale's rule states that all the outgoing links from an excitatory neuron are excitatory (positive), and all the outgoing links from an inhibitory neuron are inhibitory (negative). Therefore, for i -th neuron, the link $i \rightarrow j$ is described by the matrix element $W_{ij} > 0$, if it is excitatory, or $W_{ij} < 0$, inhibitory.

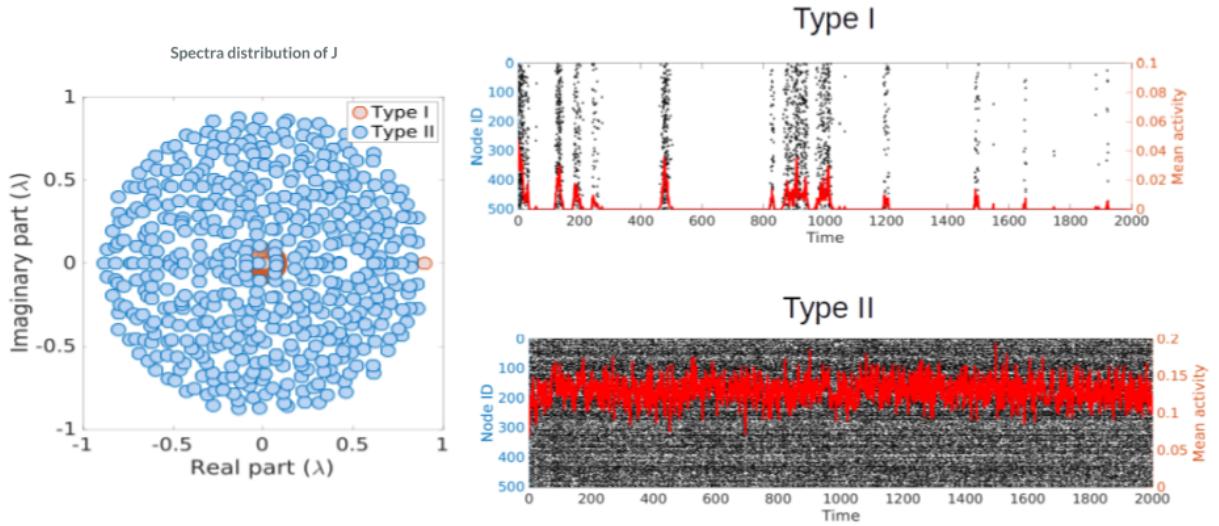


Figure 14: Phenomenology of Type I and Type II criticality. For the firing-rate model, and close to criticality, Type I has an active phase dominated by the homogeneous state (the outlier). It displays synchronous states and avalanches. On the other hand, Type II has an active phase described by non-trivial combination of unstable eigenmodes. It is an asynchronous phase, with long-time scales. **Left panel:** The spectra distribution for the adjacency matrix W for a random Gaussian matrix. **Right panel:** raster plot for Type I and Type II model. A black dot represents a spike of a particular neuron at a given time t ; in red, we plot the ratio of spikes on a given time (the mean value of the firing rate value).

for $w > 0$. The rest of the neurons are excitatory, with outgoing synapse strength drawn from a uniform distribution in $[0, 1]^2$. Hence, the connectivity matrix gW is governed by two parameters, the coupling strength g and the fraction-strength w , which measures the fraction between the strength of inhibitory links and excitatory links.

- **First task: (Easy)** Consider the 1-dimensional firing-rate model (17), with $N = 1$ (and $I = 0$, $\xi = 0$). Characterize the fixed point solutions as a function of g and its stability. Plot the bifurcation diagram. The book of Strogatz is a good reference [19].
- **Second task: (Intermediate)**: Study the distribution of eigenvalues for the random matrix W proposed on this project. Characterize Type I and Type II criticality by means of parameters g and w . To do that, consider the analysis on Methods in the article of Li and Shew [12] –it is convenient to study the Circle law, also called Girko’s law, for random matrices; see for instance [15].
- **Third task: (Intermediate)**: In this part, consider the network with $N = 1000$ neurons. The idea is to obtain an equivalent Figure 15 for our model. To do that, we define the average firing-rate (i.e. the average spike rate on the Figure) as

$$S = \frac{1}{N} \sum_{i=1}^N \langle r_i^2 \rangle$$

where $\langle \cdot \rangle$ means average over time. This order parameter depends on parameters g and w . Hence, averaging over M simulations, we can obtain all these figures.

- **Fourth task: (Hard)**: Repeat the previous points but considering a different saturation function. In this case, we propose to use a rectified linear function, defined as

$$\phi(x) = \begin{cases} 0, & \text{if } x < 0 \\ x, & \text{if } 0 < x < 1 \\ 1, & \text{if } x > 1 \end{cases}$$

²For this kind of adjacency matrix, that obeys the Dale’s law, there is an additional row-wise condition that has to be imposed for the eigenvalue distribution to converge into the Circle Law, see Rajan and Abbott [15].

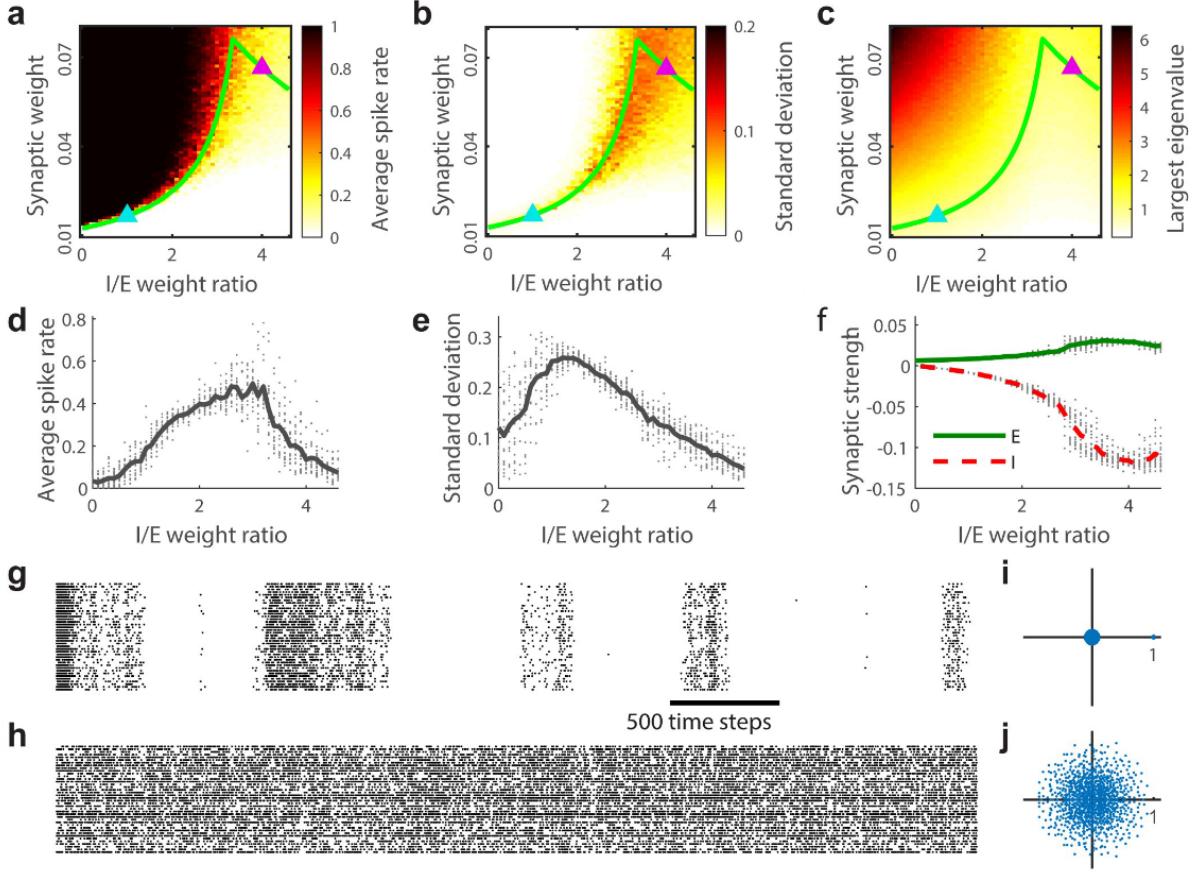


Figure 15: Synchronous and asynchronous dynamical regimes (Type I and Type II) along the critical boundary. Figure from the article of Li and Shew [12].

This case is interesting because of the "rectification" on negative values, but the difficulty appears as a consequence of the non-differentiable shape. In this case, an external input should be considered ($I \neq 0$) in order to obtain non-zero activity. Despite of this difficulty, we expect to obtain something similar to the article of Li and Shew [12] as they used an equivalent saturation function for the probability.

REFERENCES

- [1] Juan A. Acebrón, L. L. Bonilla, Conrad J. Pérez Vicente, Félix Ritort, and Renato Spigler. The Kuramoto model: A simple paradigm for synchronization phenomena. *Reviews of Modern Physics*, 77(1):137–185, April 2005.
- [2] Reka Albert and Albert-Laszlo Barabasi. Statistical mechanics of complex networks. *arXiv:cond-mat/0106096*, June 2001. arXiv: cond-mat/0106096.
- [3] M. Andrecut. Monte-Carlo Simulation of a Multi-Dimensional Switch-Like Model of Stem Cell Differentiation. *arXiv:1304.2054 [q-bio]*, February 2011. arXiv: 1304.2054.
- [4] Peter Ashcroft, Philipp M. Altrock, and Tobias Galla. Fixation in finite populations evolving in fluctuating environments. *Journal of The Royal Society Interface*, 11(100):20140663, November 2014.
- [5] Igor Belykh, Russell Jeter, and Vladimir Belykh. Foot force models of crowd dynamics on a wobbly bridge. *Science Advances*, 3(11):e1701512, November 2017.
- [6] Pat Dallard, Tony Fitzpatrick, Anthony Flint, Angus Low, Roger Ridsdill Smith, Michael Willford, and Mark Roche. London Millennium Bridge: Pedestrian-Induced Lateral Vibration. *Journal of Bridge Engineering*, 6(6):412–417, December 2001.
- [7] Vincent Deblauwe, Nicolas Barbier, Pierre Couteron, Olivier Lejeune, and Jan Bogaert. The global biogeography of semi-arid periodic vegetation patterns. *Global Ecology and Biogeography*, 17(6):715–723, November 2008. Number: 6.
- [8] Mihir Durve and Ahmed Sayeed. First-order phase transition in a model of self-propelled particles with variable angular range of interaction. *Physical Review E*, 93(5):052115, May 2016.
- [9] C. Fernandez-Oto, D. Escaff, and J. Cisternas. Spiral vegetation patterns in high-altitude wetlands. *Ecological Complexity*, 37:38–46, January 2019.
- [10] Daniel T Gillespie. A general method for numerically simulating the stochastic time evolution of coupled chemical reactions. *Journal of Computational Physics*, 22(4):403–434, December 1976.
- [11] C. A. Klausmeier. Regular and Irregular Patterns in Semiarid Vegetation. *Science*, 284(5421):1826–1828, June 1999. Number: 5421.
- [12] Jingwen Li and Woodrow L. Shew. Tuning network dynamics from criticality to an asynchronous state. *PLOS Computational Biology*, 16(9):e1008268, September 2020. Number: 9.
- [13] Sergi Lozano, Alex Arenas, and Angel Sánchez. Mesoscopic Structure Conditions the Emergence of Cooperation on Social Networks. *PLoS ONE*, 3(4):e1892, April 2008. Number: 4.
- [14] Martin A. Nowak and Robert M. May. Evolutionary games and spatial chaos. *Nature*, 359(6398):826–829, October 1992. Number: 6398.
- [15] Kanaka Rajan and L. F. Abbott. Eigenvalue Spectra of Random Matrices for Neural Networks. *Physical Review Letters*, 97(18):188104, November 2006. Number: 18.
- [16] M. Rietkerk, S. C. Dekker, P. C. de Ruiter, and J. van de Koppel. Self-Organized Patchiness and Catastrophic Shifts in Ecosystems. *Science*, 305(5692):1926–1929, September 2004. Number: 5692.
- [17] Carlos P. Roca, José A. Cuesta, and Angel Sánchez. Evolutionary game theory: Temporal and spatial effects beyond replicator dynamics. *Physics of Life Reviews*, 6(4):208–249, December 2009. Number: 4.
- [18] H. Sompolinsky, A. Crisanti, and H. J. Sommers. Chaos in Random Neural Networks. *Physical Review Letters*, 61(3):259–262, July 1988.

-
- [19] Steven H. Strogatz. *Nonlinear dynamics and Chaos: with applications to physics, biology, chemistry, and engineering*. Studies in nonlinearity. Addison-Wesley Pub, Reading, Mass, 1994.
 - [20] Steven H. Strogatz, Daniel M. Abrams, Allan McRobie, Bruno Eckhardt, and Edward Ott. Crowd synchrony on the Millennium Bridge. *Nature*, 438(7064):43–44, November 2005. Number: 7064 Publisher: Nature Publishing Group.
 - [21] R. Toral and P. Colet. *Stochastic Numerical Methods: An Introduction for Students and Scientists* | Wiley. Wiley, June 2014.
 - [22] Arne Traulsen and Christoph Hauert. Stochastic evolutionary game dynamics. *arXiv:0811.3538 [q-bio]*, November 2008. arXiv: 0811.3538.
 - [23] Tamás Vicsek, András Czirók, Eshel Ben-Jacob, Inon Cohen, and Ofer Shochet. Novel Type of Phase Transition in a System of Self-Driven Particles. *Physical Review Letters*, 75(6):1226–1229, August 1995.
 - [24] J. Wang, K. Zhang, L. Xu, and E. Wang. Quantifying the Waddington landscape and biological paths for development and differentiation. *Proceedings of the National Academy of Sciences*, 108(20):8257–8262, May 2011.
 - [25] Jin Wang, Li Xu, Erkang Wang, and Sui Huang. The Potential Landscape of Genetic Circuits Imposes the Arrow of Time in Stem Cell Differentiation. *Biophysical Journal*, 99(1):29–39, July 2010.