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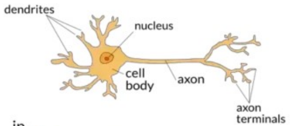

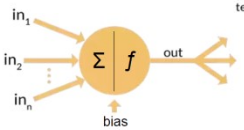

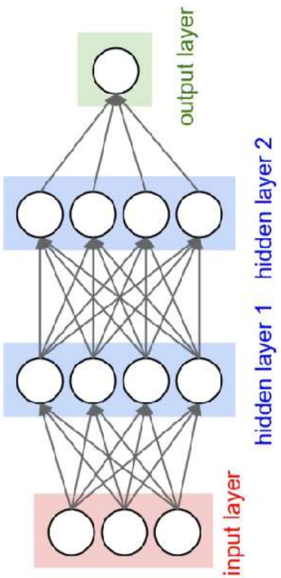

Computational Neuroscience, Theory of Control, and Networks

David Quesada – Saliba

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Abstract:

- The present communication is aimed at creating the biophysical and mathematical foundations for the understanding of the current trends theory of control and networks applied to Computational Neurosciences. There are many different models of interest on this area Hodgkin – Huxley model, Fitzhugh – Nagumo model, Morris – Lecar model, Hindmarsh – Rose model, Izhikievich model, Li – Rinzel model, Wilson – Cowan model, Kuramoto model, Hopfield and Spin Glass-like models, Cellular Automata models, etc. On this presentation the focus is on this class of models and their implications/relations to computational neurosciences.

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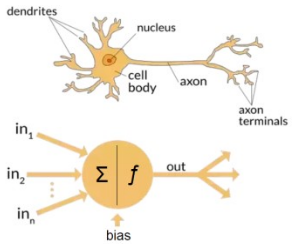
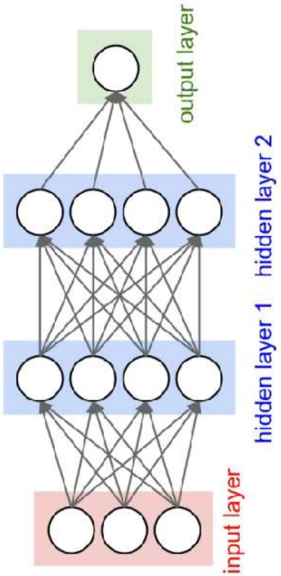
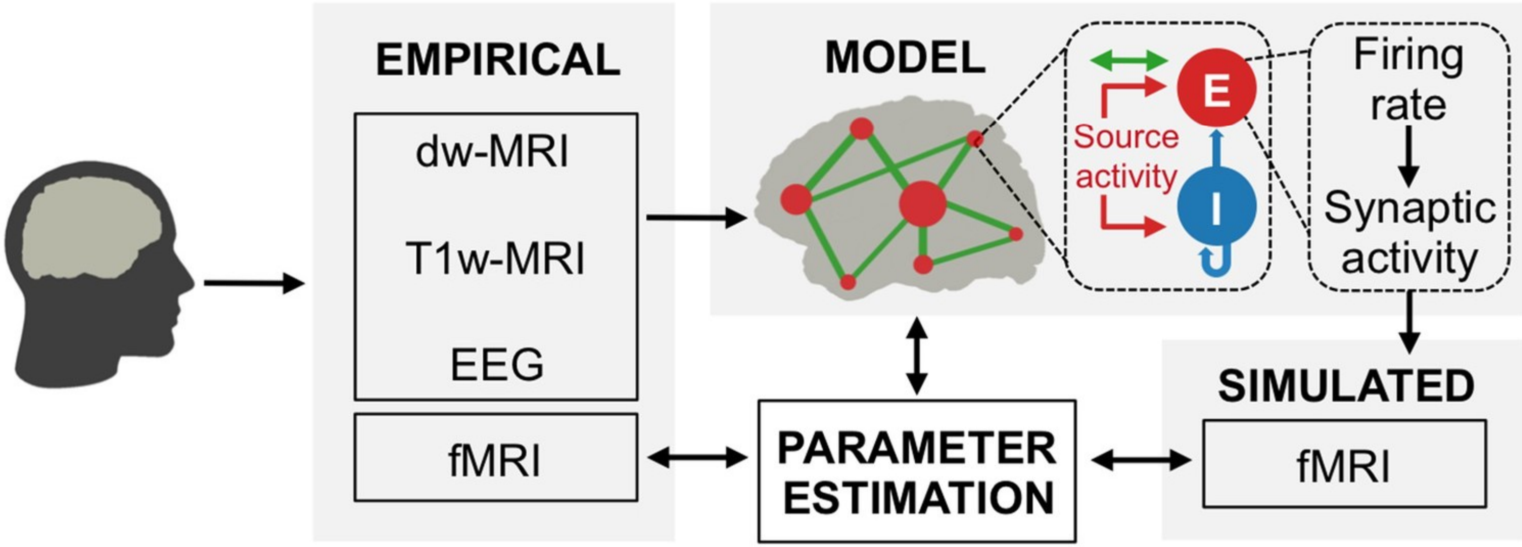




An important piece to understand, to integrate different data sets, and to predict future behaviors is the use of mathematical models, capturing specific situations and appealing to synthetic neural networks generated in advance.



Machine learning methods assisted by biophysical (mathematical) models will be capable to deeply understand brain activity.

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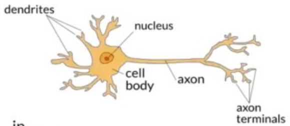
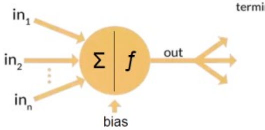
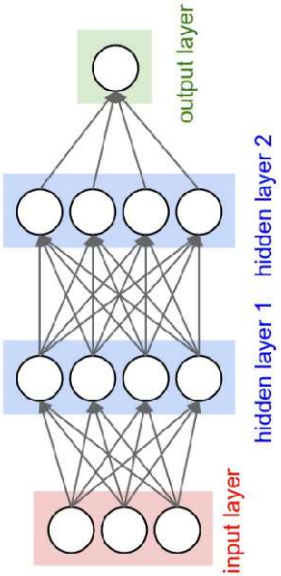
Computational Neuroscience, Theory of Control, and Networks

Most used Mathematical models of neuron dynamics and spike and busting activity







- **Hodgkin – Huxley model** – neurons are reduced to an equivalent electrical circuit with resistors and capacitors (RC circuits).
- **Fitzhugh – Nagumo model** – a simplified version of the Hodgkin – Huxley model, keeping the idea of the equivalent electrical circuit.
- **Morris – Lecar model** – a simplified version of the Hodgkin – Huxley model, with two phases of excitability.
- **Hindmarsh – Rose model** – nonlinear (polynomial) system of ODE aimed to model the spiking and bursting behavior of the membrane potential of a singe neuron.
- **Izhikievich model** – nonlinear (polynomial) system of ODE aimed at spiking behavior with post spike redefinition of the variables (2).
- **Li – Rinzel model** – modified Hodgkin – Huxley model to include the effect of the astrocytes in the firing of neurons. A model of a tripartite synapse.
- **Wilson – Cowan model** – consider a homogeneous network of excitatory neurons, and its used widely in modeling the triggering of epilepsy.
- **Kuramoto model** – neuronal network seen as a collection of linked oscillators.
- **Hopfield and Spin Glass-like models** – neurons treated as spins, described like the Ising model in magnetism. Spin glass version of the dynamics of neurons.
- **Cellular Automata models** – the neural network is represented as cells that update their states in connection to the states of surrounding cells and following specific rules set in advance.

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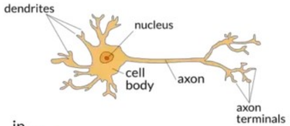
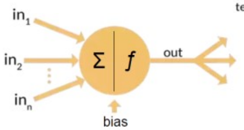
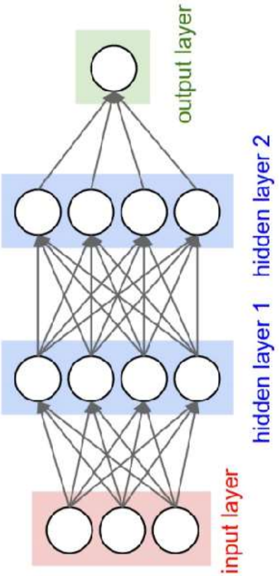
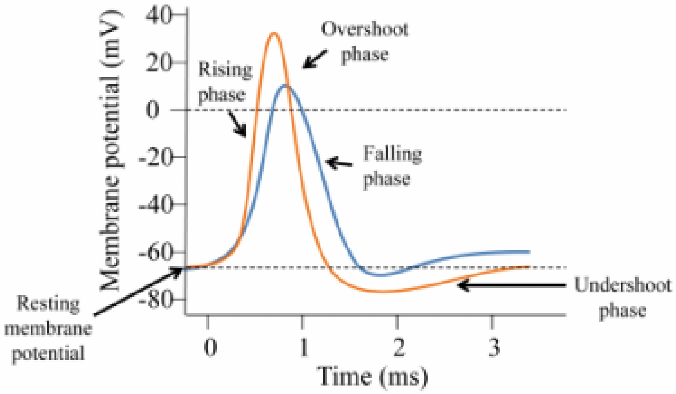
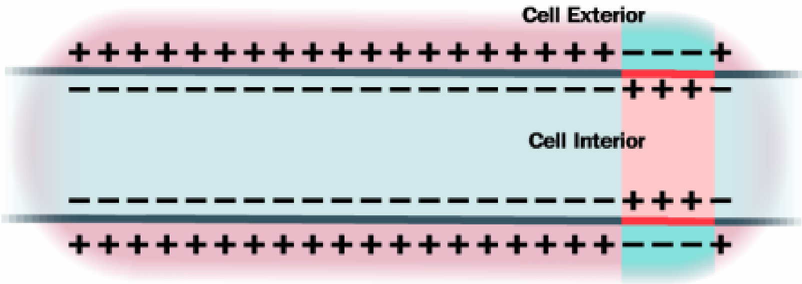
From Biophysics to Mathematical Models of Neurons







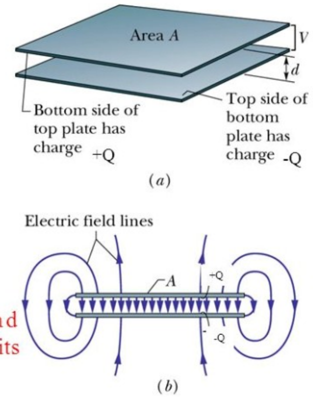


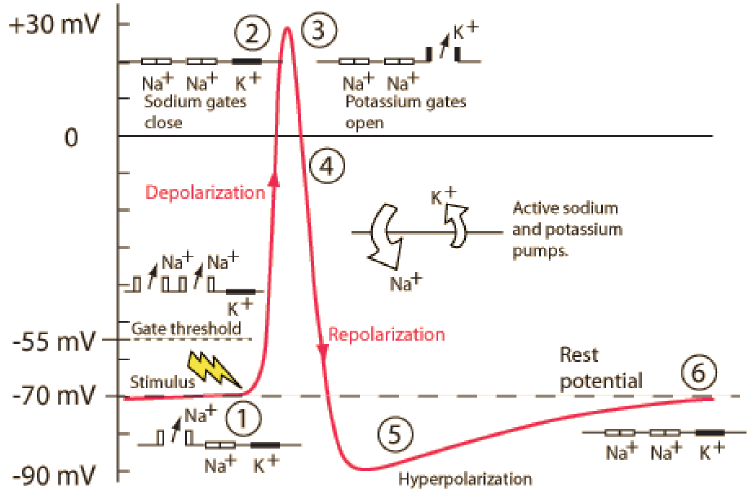


Electricity is created by a sudden reversal in charge. As you see here, an action potential is simply an electrical current that travels down an axon of a neuron.

Calculating Capacitance

- Parallel-plate capacitor in vacuum
  - Charge density: $\sigma = \frac{Q}{A}$
  - Electric field: $E = \frac{\sigma}{\varepsilon_0} = \frac{Q}{\varepsilon_0 A}$
  - Potential diff.: $V_{ab} = Ed = \frac{1}{\varepsilon_0}\frac{Qd}{A}$
    $V_{ab} = V_a - V_b = -\int_b^a \vec{E}\cdot d\vec{\ell} = \int_a^b \vec{E}\cdot d\vec{\ell} = Ed$
  - Capacitance: $C = \frac{Q}{V_{ab}} = \varepsilon_0 \frac{A}{d}$
- The capacitance depends only on the geometry of the capacitor.
- It is proportional to the area A.
- It is inversely proportional to the separation d
- When matter is present between the plates, its properties affect the capacitance.





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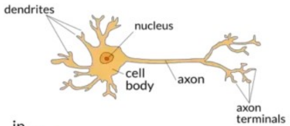

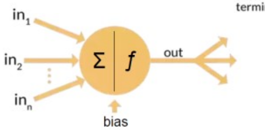

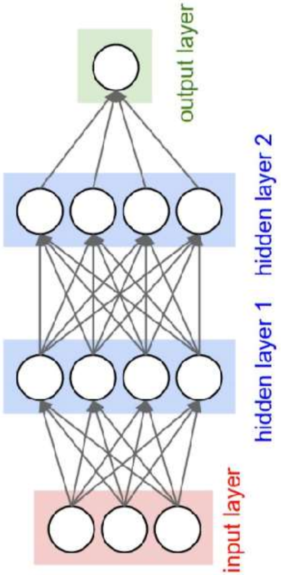

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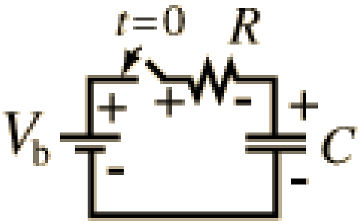
Moving to equivalent electrical circuits, RC circuits



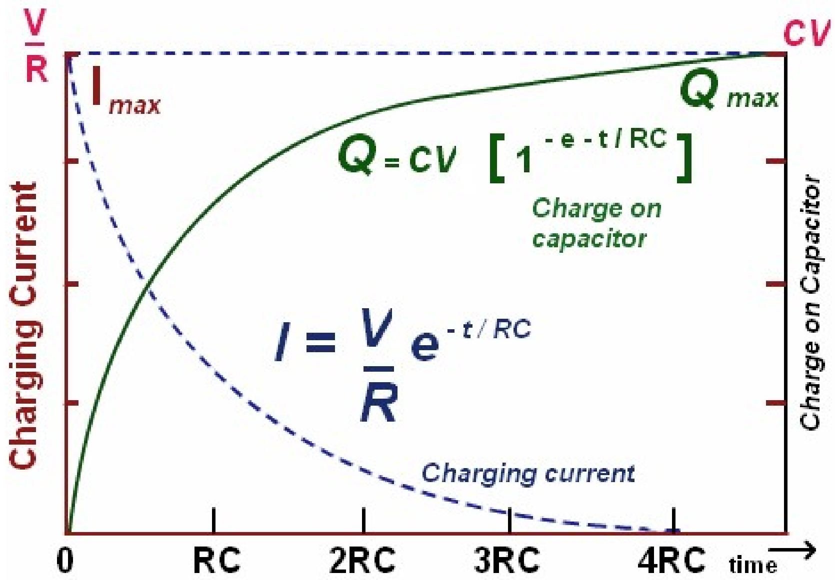
$$V_b = V_R + V_C$$

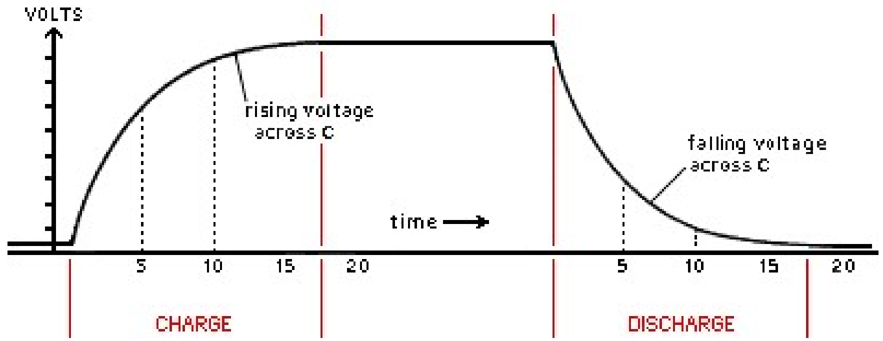
$$V_b = IR + \frac{Q}{C}$$

As charging progresses,

$$V_b = IR + \frac{Q}{C}$$

current decreases and charge increases.





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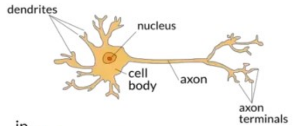

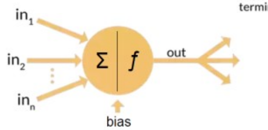

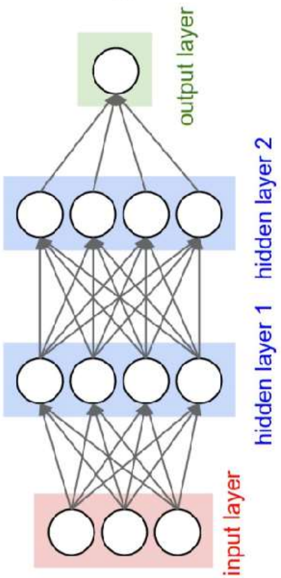

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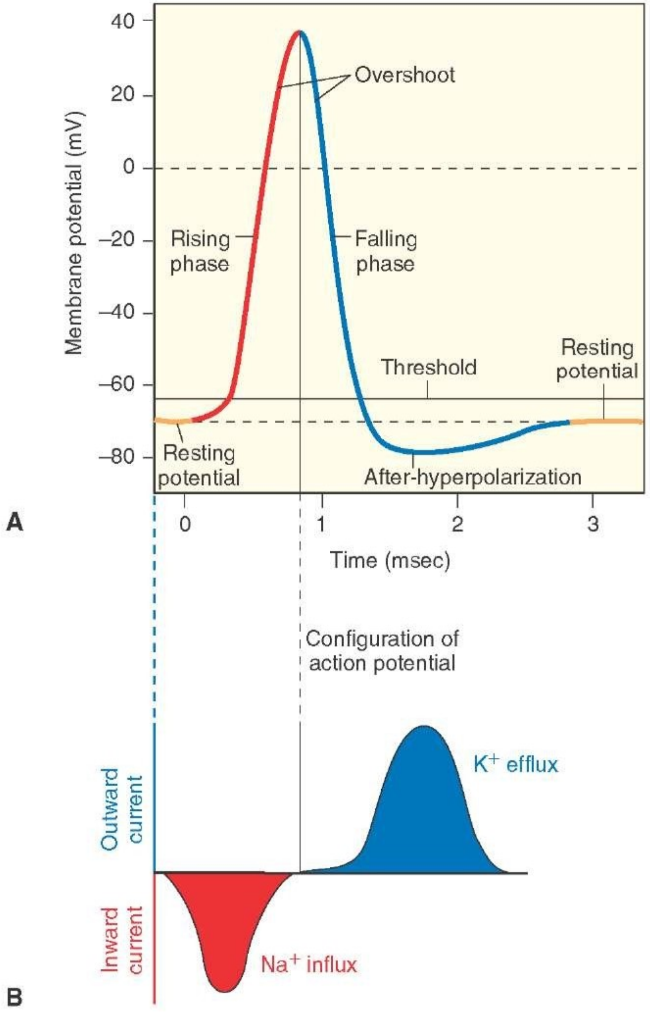
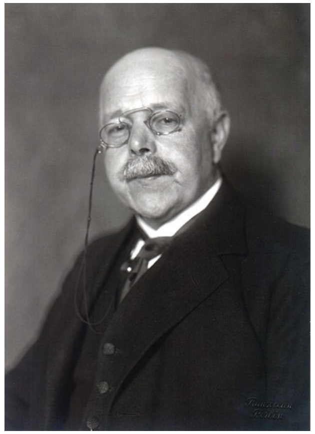




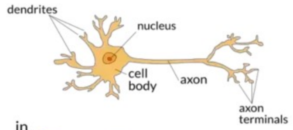
Walther Nernst

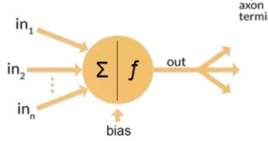
$$E = \frac{RT}{zF} \ln \frac{C_o}{C_i}$$

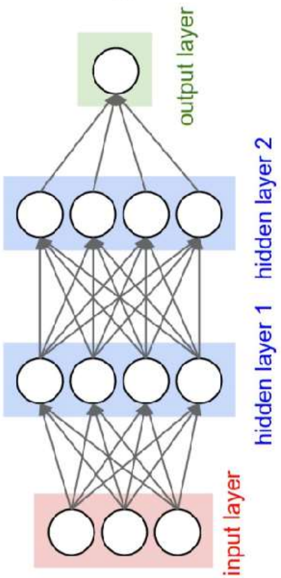
E = membrane potential (V is sometimes used as well)
R = Universal Gas constant (1.98 calories per mole Kelvin)
T = Temperature (Kelvin)
z = charge on the ion
F = Faraday's Constant (2.3x10⁴ calories per mole voltage)
ln = natural log base e
Co = Concentration of ion outside the cell
Ci = Concentration of ion inside the cell

E_{Na}= 55 mV; E_K= - 75 mV

Neuron membranes at rest are impermeable to Na^+. Therefore, the observed potential is near E_K = - 75 mV. When excited, Na^+ channels open rapidly and the membrane potential approaches E_{Na} = 55 mV. The K^+ channel opens more slowly, but eventually returns the membrane potential to near E_K.







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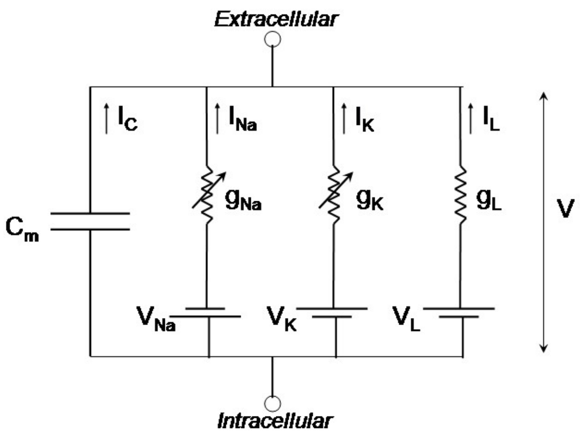
The Hodgkin – Huxley membrane model

- The Hodgkin – Huxley (HH) membrane model provides the analog circuit studied the most in neurophysiology. They formulated a membrane model that accounts for Na^+, K^+ and ion leakage channels.
- The membrane resting potential for each ion species is treated like a battery and the degree to which the channel is open is modeled by a variable resistor.
- There is a membrane capacitance. The resistances to Na^+ and K^+ ions change as does the membrane potential.



$$
\begin{aligned}
C\frac{dv}{dt} &= I - g_{Na}m^3h(V-V_{Na}) - g_K n^4(V-V_K) - g_L(V-V_L)\\
\frac{dm}{dt} &= a_m(V)(1-m) - b_m(V)m\\
\frac{dh}{dt} &= a_h(V)(1-h) - b_h(V)h\\
\frac{dn}{dt} &= a_n(V)(1-n) - b_n(V)n\\
a_m(V) &= .1(V+40)/(1-\exp(-(V+40)/10))\\
b_m(V) &= 4\exp(-(V+65)/18)\\
a_h(V) &= .07\exp(-(V+65)/20)\\
b_h(V) &= 1/(1+\exp(-(V+35)/10))\\
a_n(V) &= .01(V+55)/(1-\exp(-(V+55)/10))\\
b_n(V) &= .125\exp(-(V+65)/80)
\end{aligned}
$$

$$\nabla = \frac{\partial}{\partial x}\mathbf{i} + \frac{\partial}{\partial y}\mathbf{j} + \frac{\partial}{\partial z}\mathbf{k}$$

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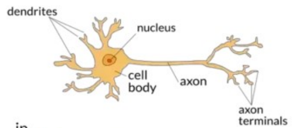
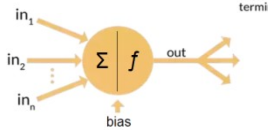
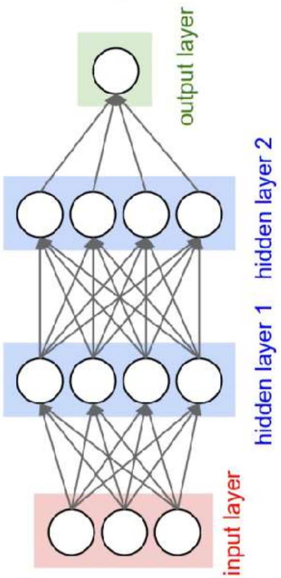
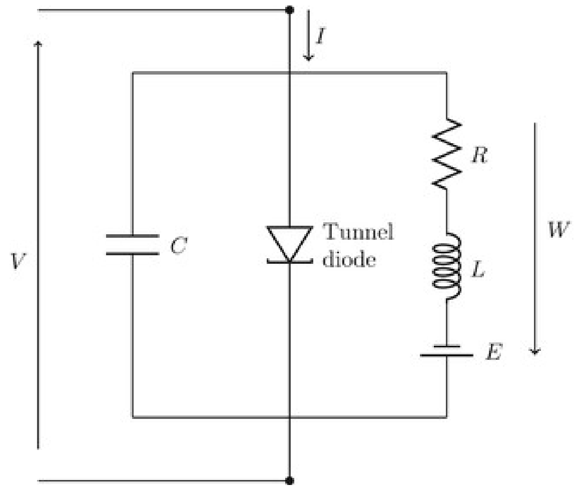
FitzHugh – Nagumo circuit model – introduced in 1960



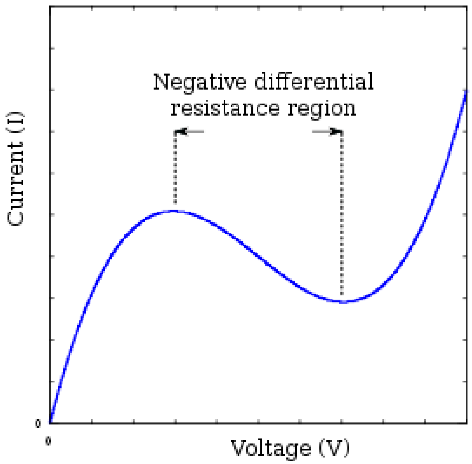


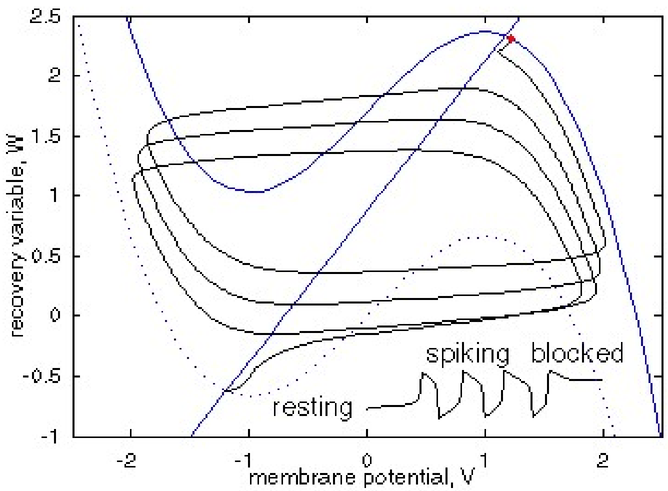




L dI/dt = E – V – RI
C dV/dt = I – g(V)

$$C\frac{dV}{dt} = I - \epsilon\left(\frac{V^3}{3} - V\right),$$





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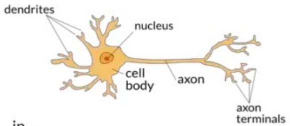
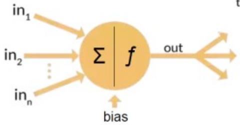
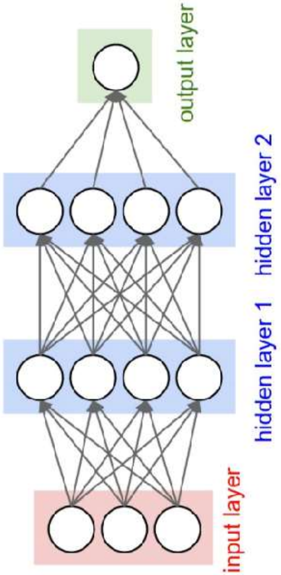
The Kuramoto model or Kuramoto Oscillators







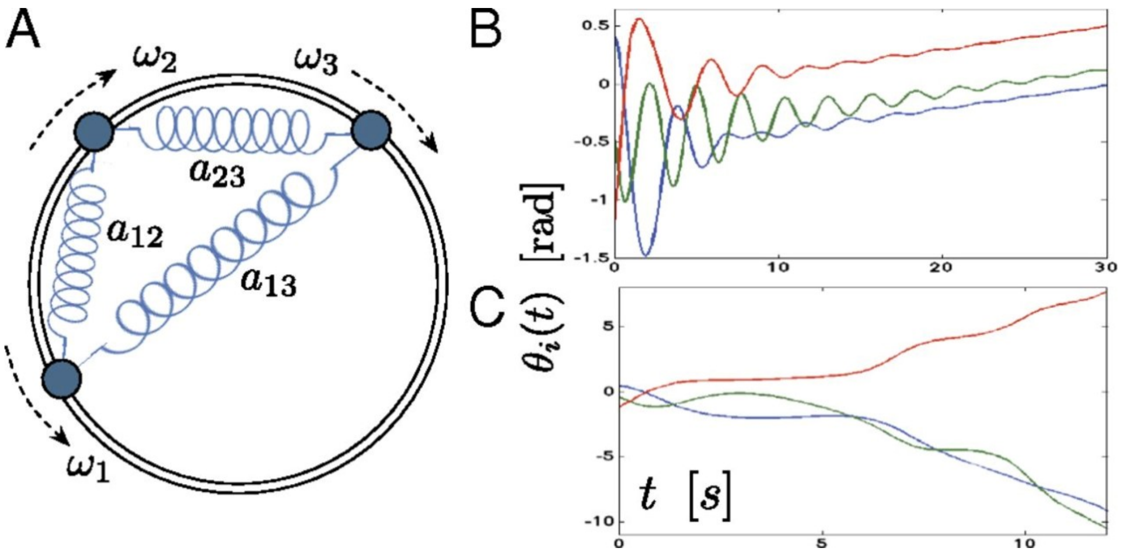
Kuramoto Dynamics

simple phase oscillator

other nodes

$$\frac{d\phi_k}{dt} = \omega_k + g\frac{1}{N}\sum_{j=1}^{N}\sin\left(\phi_j - \phi_k\right)$$

nonlinear coupling



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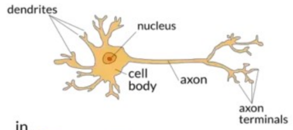

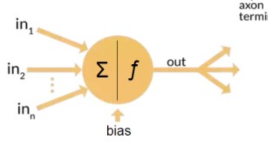

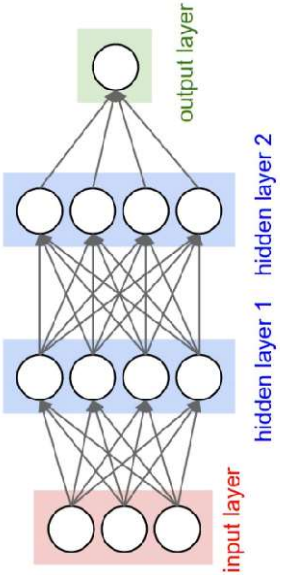

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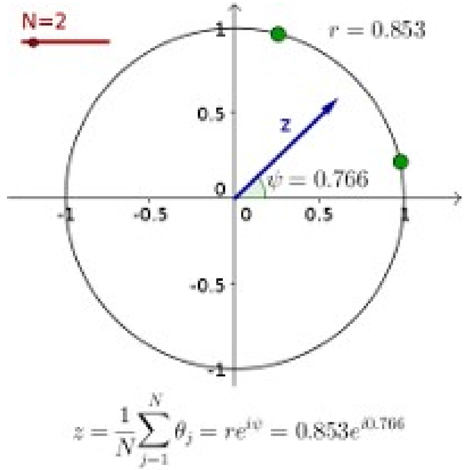
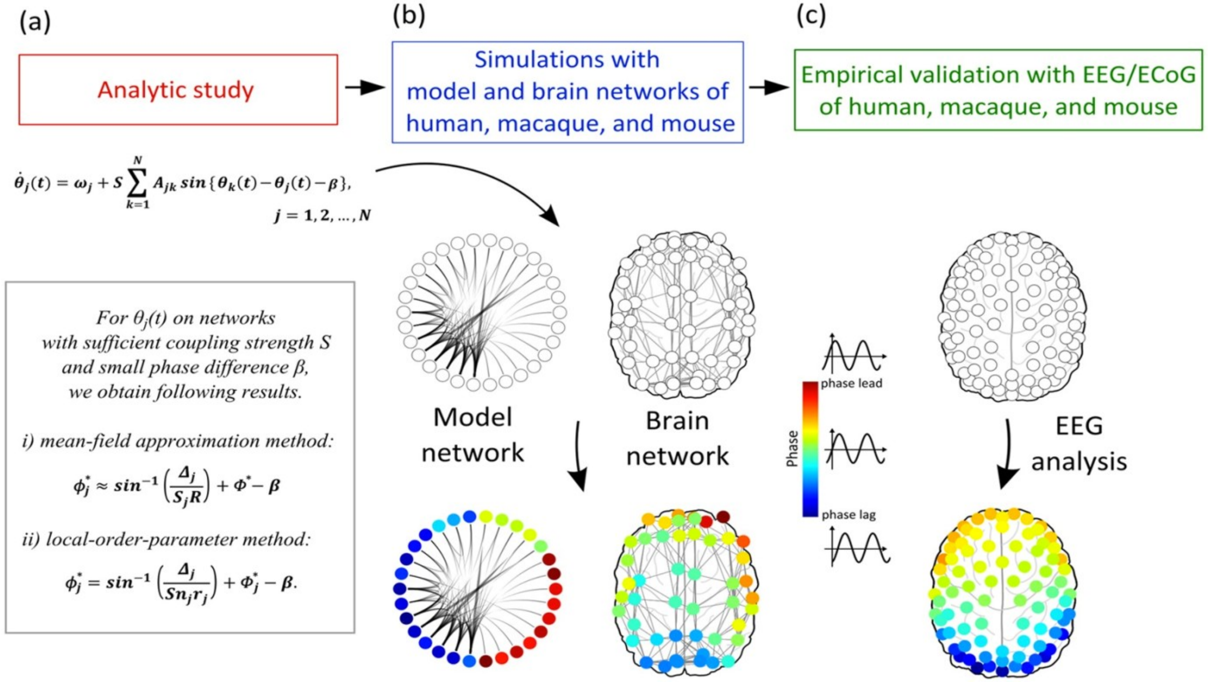
Structure Shapes Dynamics and Directionality in Diverse Brain Networks: Mathematical Principles and Empirical Confirmation in Three Species

Joon-Young Moon, Junhyeok Kim, Tae-Wook Ko, Minkyung Kim, Yasser Iturria-Medina, Jee-Hyun Choi, Joseph Lee, George A. Mashour & UnCheol Lee

Scientific Reports **7**, Article number: 46606 (2017) | Cite this article





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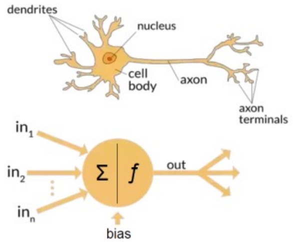
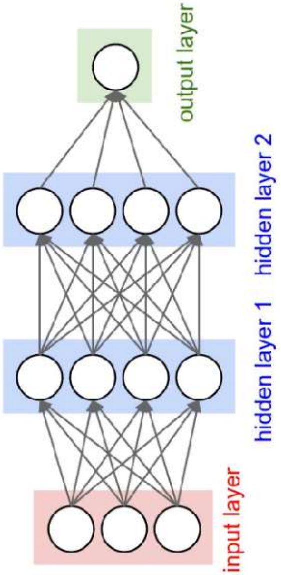
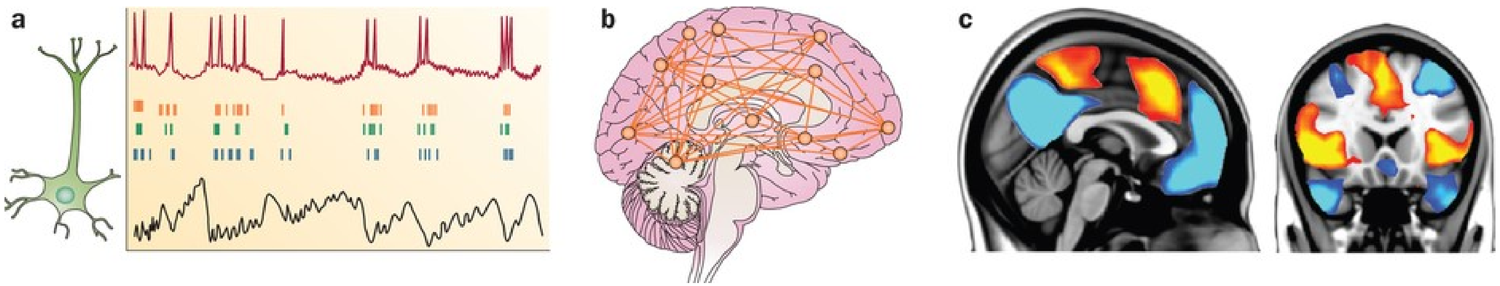
Modeling Philosophy – Bottom - Top





From a single neuron to a bundle of neurons with different **topologies of connectivity** and **interaction strengths**.

Cortical patches of neurons with different **topologies of connectivity** and **interaction strengths**.



Fitzhugh – Nagumo model (microscopic picture)

$$\frac{dv_i}{dt} = v_i - \frac{v_i^3}{3} - w_i + I_{inter} + I_{ext}$$

$$\varepsilon\frac{dw_i}{dt} = v_i + a - bw_i$$

$$I_{inter} = \sum_{j=1}^{N} G_{ij}a_{ij}(I_j - I_i)$$

Kuramoto model (mesoscopic picture)

$$\frac{d\theta_i}{dt} = \omega_i + \sum_{j=1}^{N} \sigma_{ij}a_{ij}\sin\left(\theta_j - \theta_i\right)$$

Neuronal Activity as a result of **synchronization** of either neural bundles or patches in the cortical area.

M. Rubinov and O. Sporns, "Complex networks measures of brain connectivity: Uses and interpretations", NeuroImage 52, 1059 – 1069 (2010).

P.N. Taylor, M. Kaiser, J. Dauwels, "Structural connectivity based whole brain modeling in epilepsy", J. Neuroscience Methods **236**, 51 – 57 (2014).

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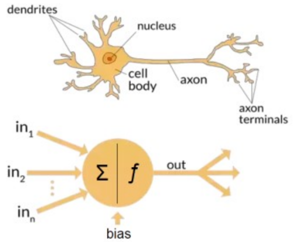
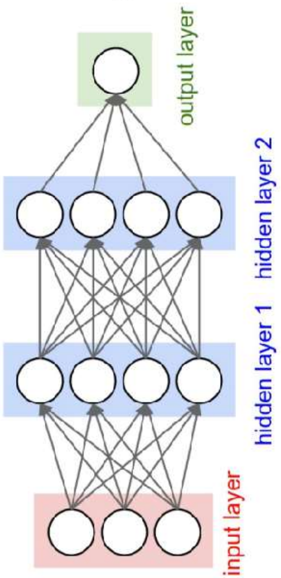
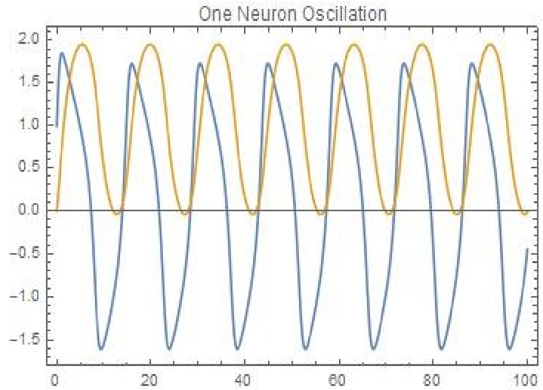
Solutions for the Fitzhugh – Nagumo Model

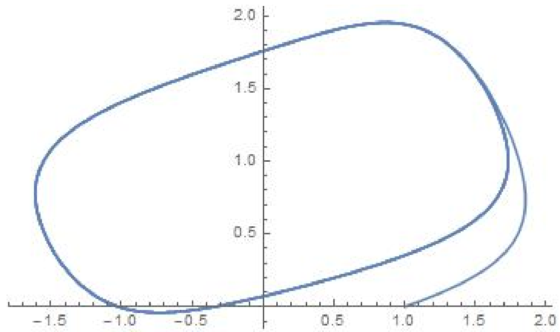




Effect of Brain network topologies on the synchronization of neuronal oscillations: Is this the gateway to the understanding of Central Nervous disorders? **Quesada, D.**; Astudillo, N.; Garcia-Russo, M. *In Proceedings of the MOL2NET, International Conference on Multidisciplinary Sciences*; Sciforum Electronic Conference Series, Vol. 2, 07004; http://doi:10.3390/mol2net-02-07004 , (2016).

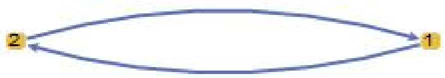
Single Neuron

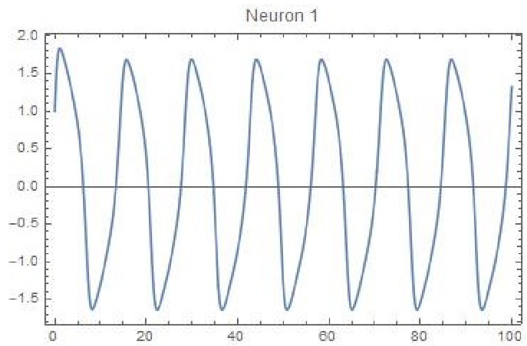


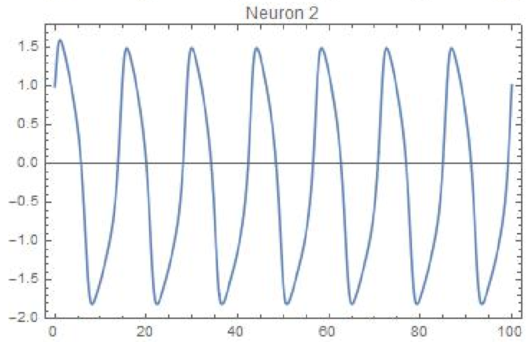


A phase portrait showing the existence of a **limit cycle**.

Two Neurons







$\nabla = \frac{\partial}{\partial x}\mathbf{i} + \frac{\partial}{\partial y}\mathbf{j} + \frac{\partial}{\partial z}\mathbf{k}$

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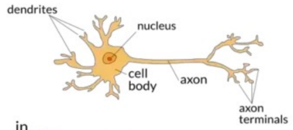

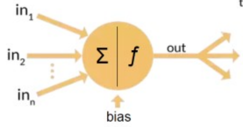

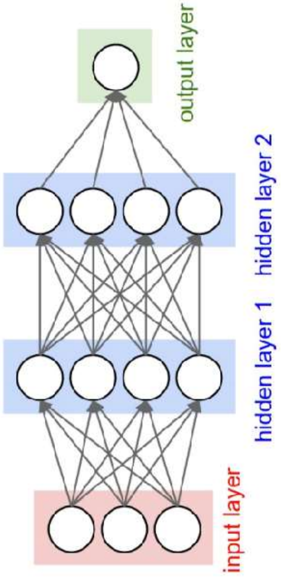

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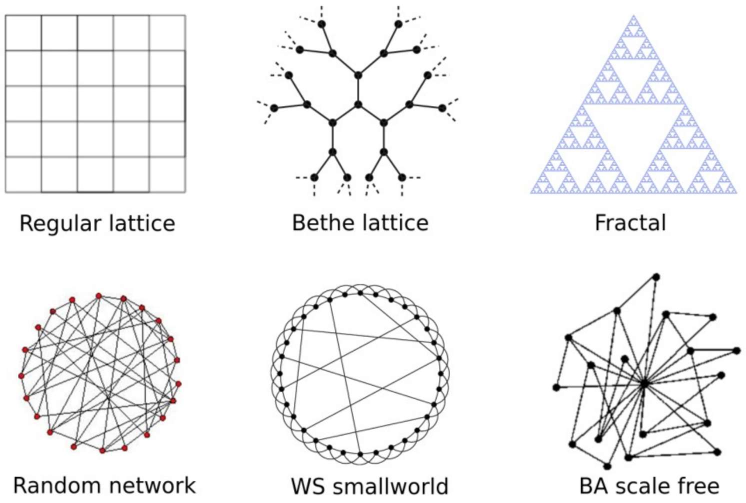
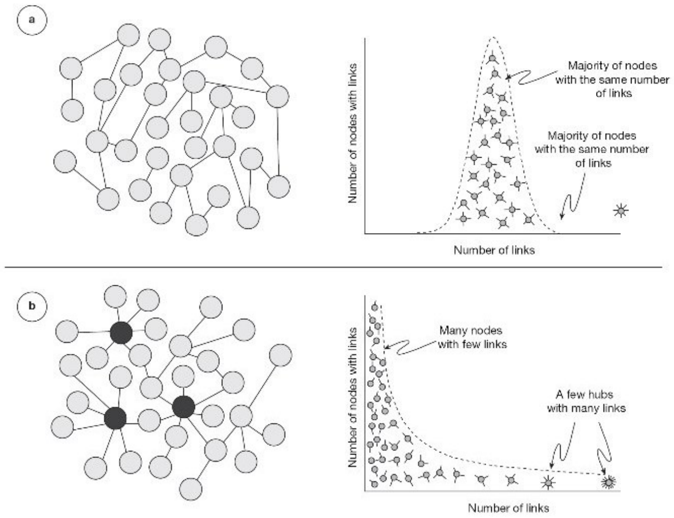
Computational Neuroscience, Theory of Control, and Networks

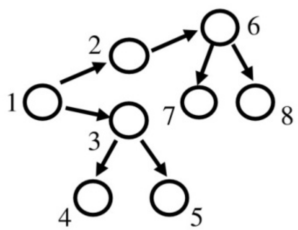
Types of Graphs often found in applications



Differences in Graphs with nodes distributed according to different Probability Distributions



One of the best methods to represent a graph it is by mean of an array of numbers, called a **matrix**; a matrix with m – rows and n – columns. These matrices are called **m x n matrices**.



$$\Rightarrow A = \begin{bmatrix} 0 & 1 & 1 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 1 & 0 & 0 \\ 0 & 0 & 0 & 1 & 1 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 1 & 1 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \end{bmatrix}$$

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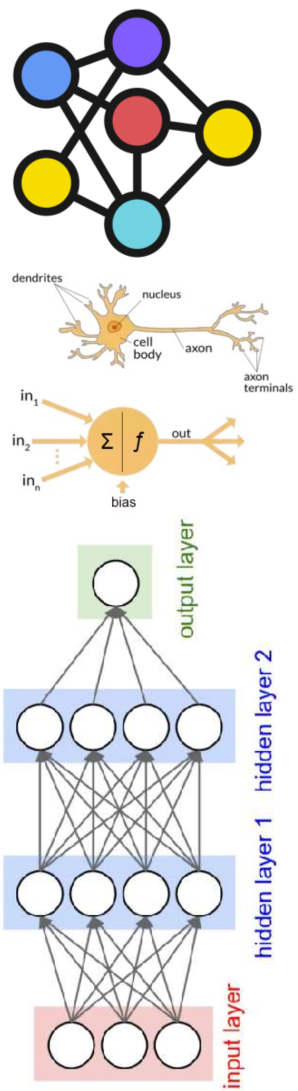
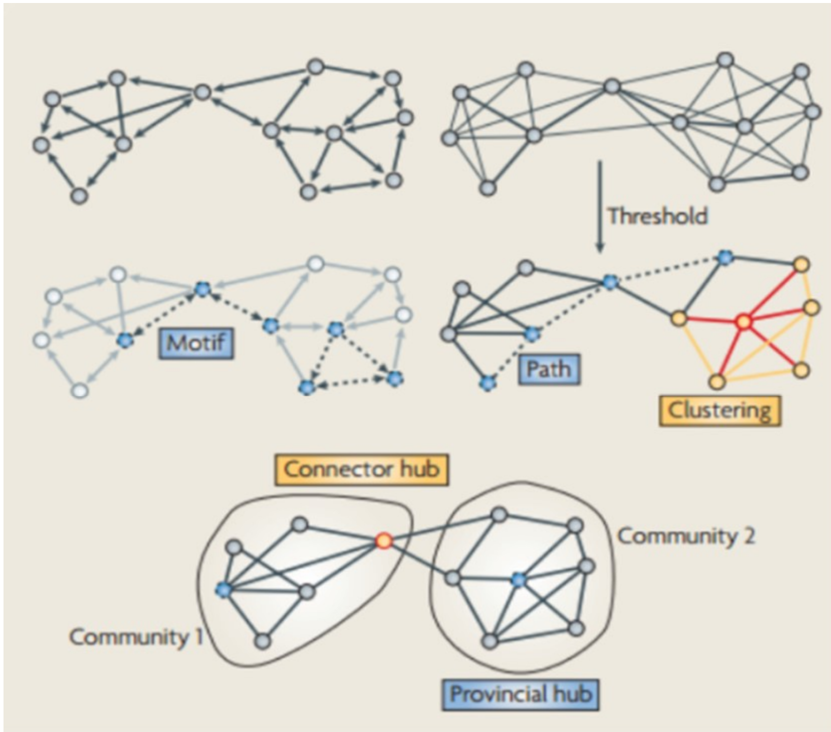


Steps for Modeling

1. Generate a model network
2. Compute the topological indices of the graph.
3. Save the information about the Adjacency matrix $A = ||a_{ij}||$ and the Weight-of-Connection matrix $G = ||g_{ij}||$.
4. Solve the system of ODE on the network.
5. Compute the synchronization properties for each of the two models: Fitzhugh-Nagumo and Kuramoto models.



H. Schmidt, G. Petkov, M. Richardson, J.R. Terry, “Dynamics on networks: The role of local dynamics and global networks on the emergence of hypersynchronous neural activity”, Plos Computational Biology **10**, 1 – 16 (2014).

E. Bullmore and O. Sporns, “Complex brain networks: graph theoretical analysis of structural and functional systems”, Nature Reviews Neuroscience **10**, 186 – 198 (2009).

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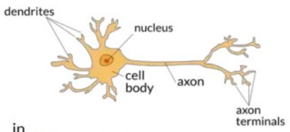
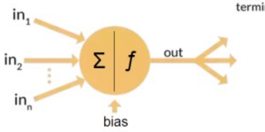
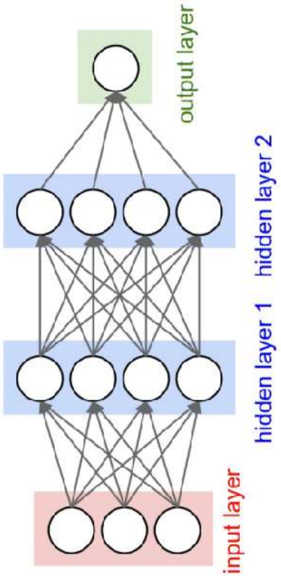
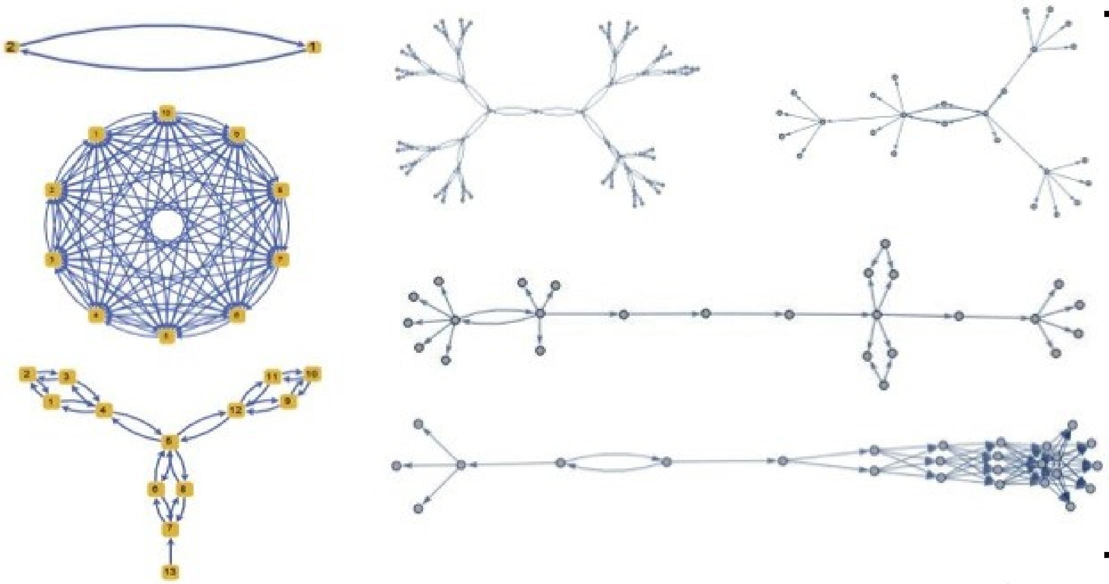
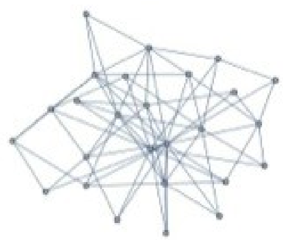




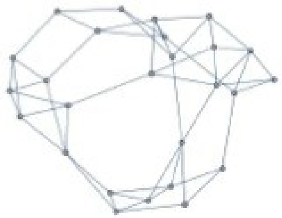




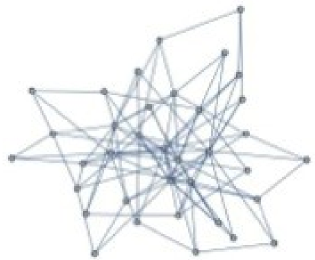
Networks created with **random tables** from 0 and 1, and used for the **Fitzhugh – Nagumo** model of neuron bundles



BarabasiAlbert[36,3]
Kuramoto Model



WattsStrogatz[36,0.2]
Kuramoto Model



BarabasiAlbert[36,4]
Kuramoto Model

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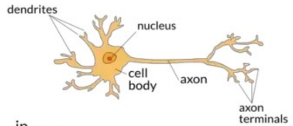
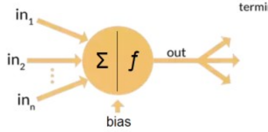
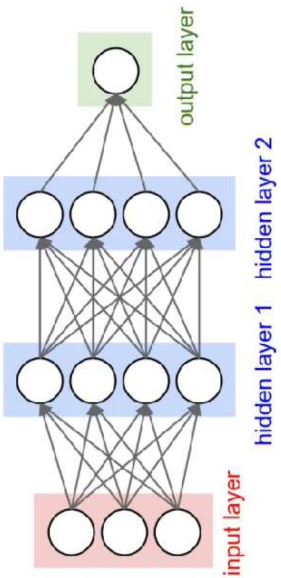
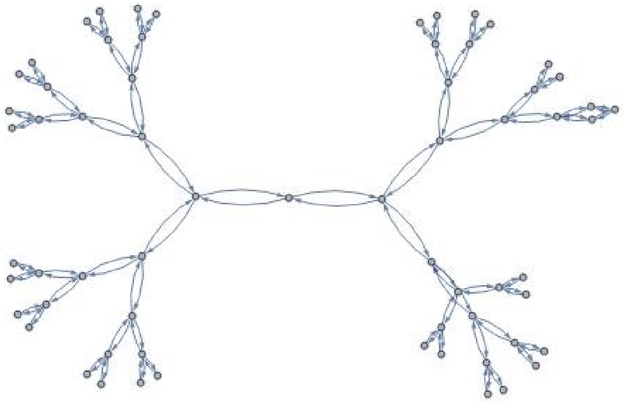
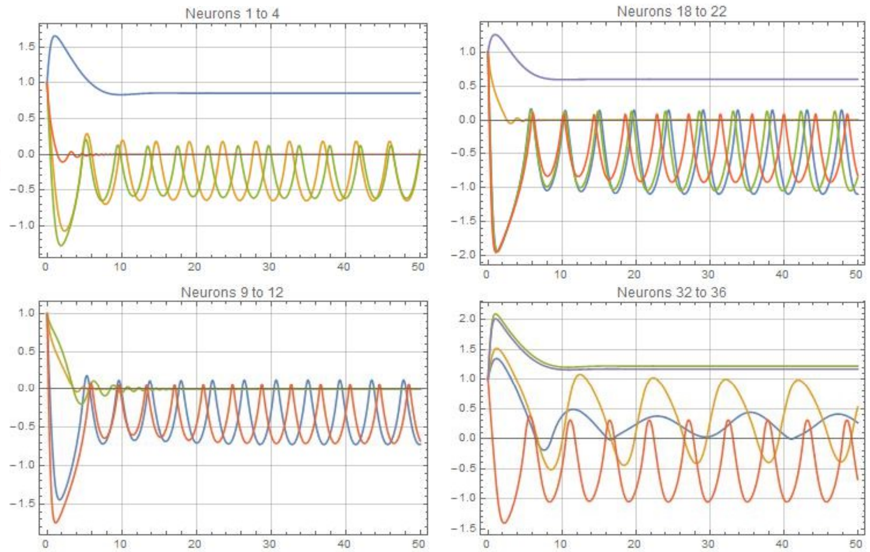




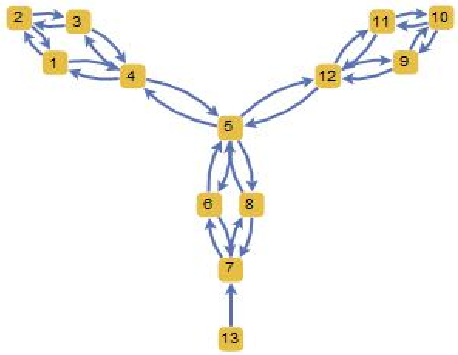


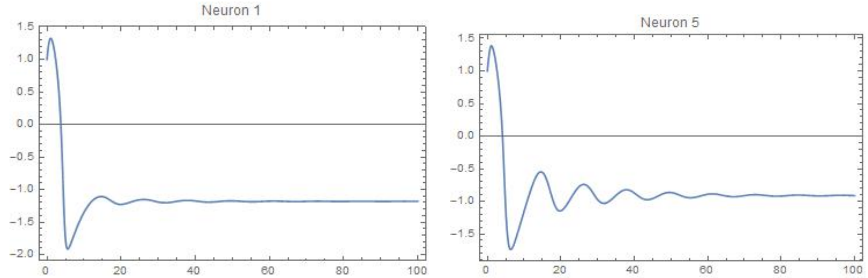
Sixty-four Neurons





The **lack of connectivity**, and the presence of **bridge points** in the neural network is extremely important when you are forced to do surgical interventions. It will determine the extension of the surgical removal and the concrete spot where the procedure should be done.





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$$\nabla = \frac{\partial}{\partial x}\mathbf{i} + \frac{\partial}{\partial y}\mathbf{j} + \frac{\partial}{\partial z}\mathbf{k}$$

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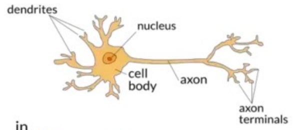
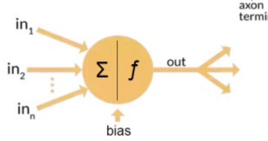
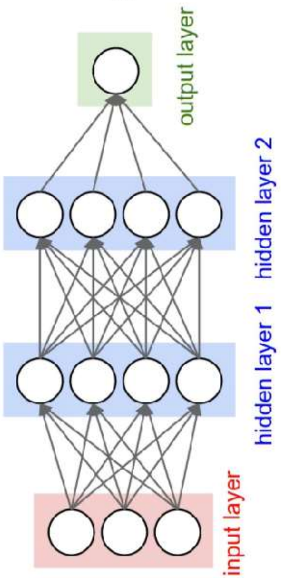


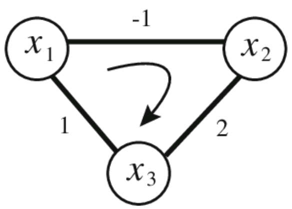




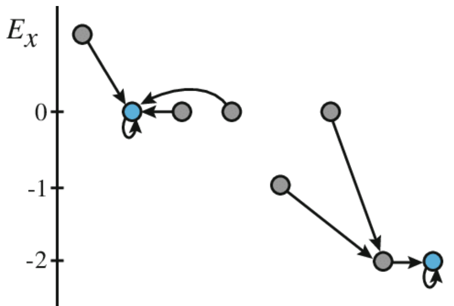
A Hopfield Network Model

$$E = -\frac{1}{2}\sum_i \sum_j w_{ij} V_i V_j$$

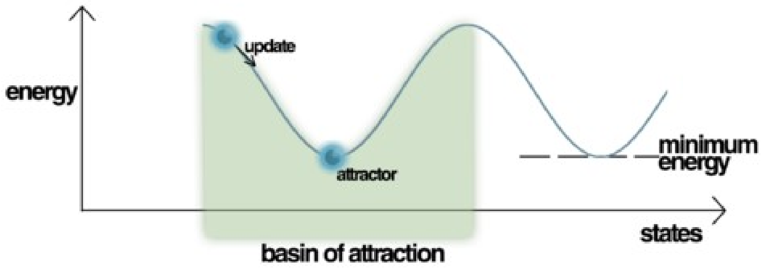
$$E = -\frac{1}{2} V^T W V$$



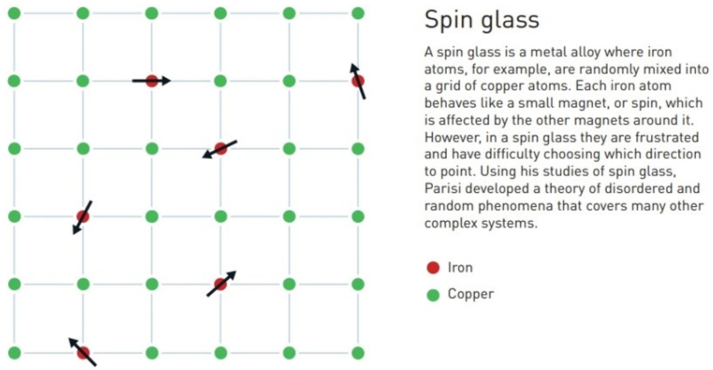
$$J = \begin{bmatrix} 0 & -1 & 1 \\ -1 & 0 & 2 \\ 1 & 2 & 0 \end{bmatrix}$$

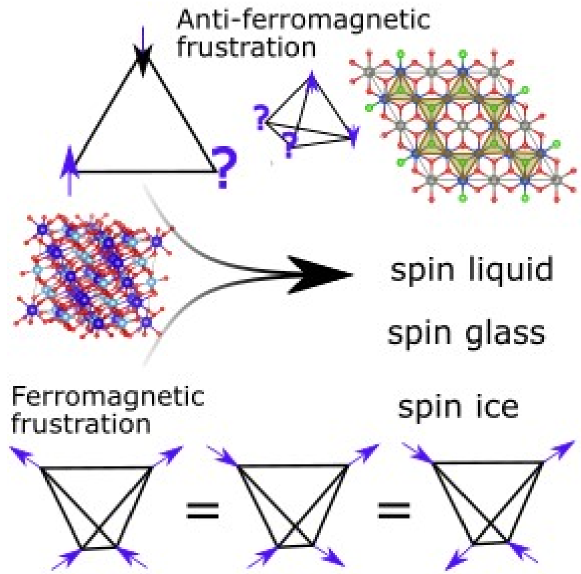


x_1	1	0	1	0	1	0	1	0
x_2	1	0	0	1	0	0	1	1
x_3	0	0	0	0	1	1	1	1



Spin Glass Model





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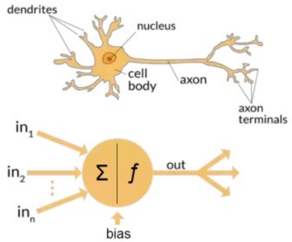

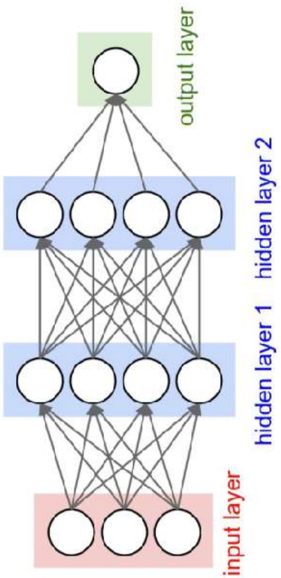

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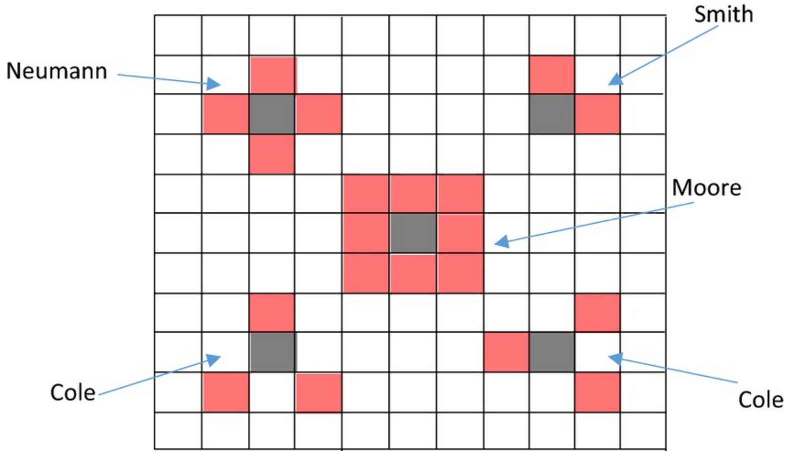
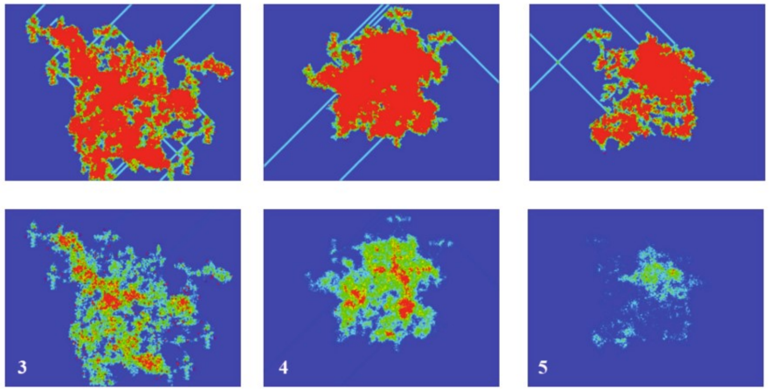
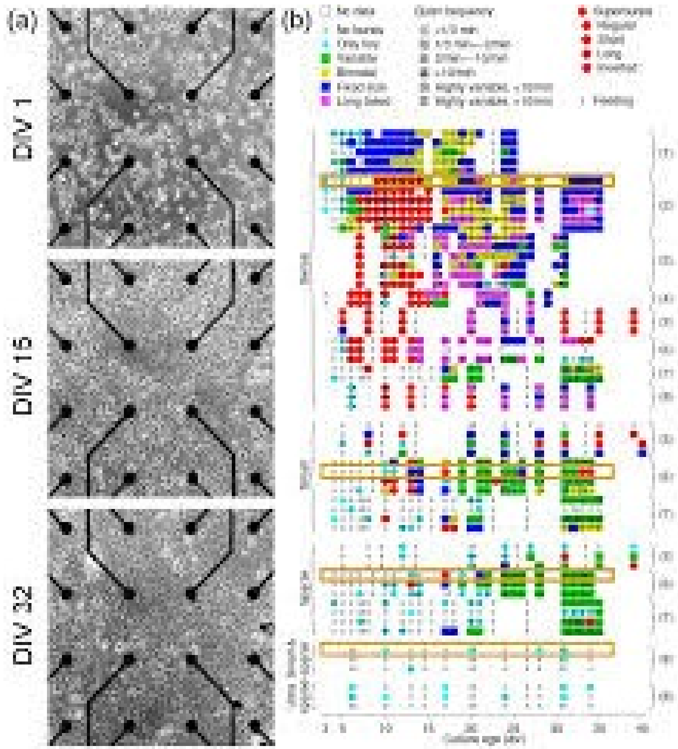
Computational Neuroscience, Theory of Control, and Networks

Cellular Automata in Brain Activity Modeling

Group of cells with a defined state for each one, which is updated in connection with the surrounding cells states. Rules are defined in advance, as well as the the type of neighborhood to be used.







Cellular Automata example of Brain activity (left) and neuron spikes (right)

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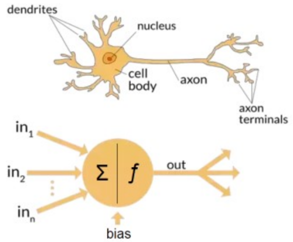

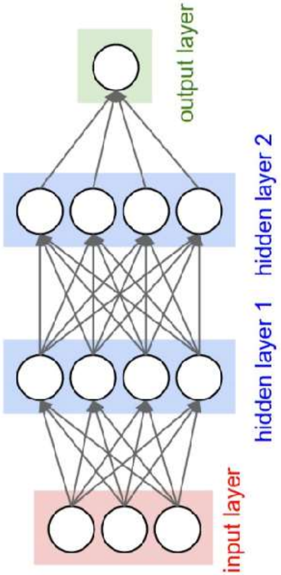

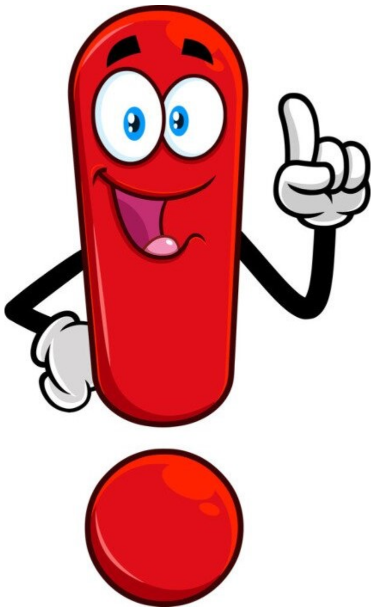

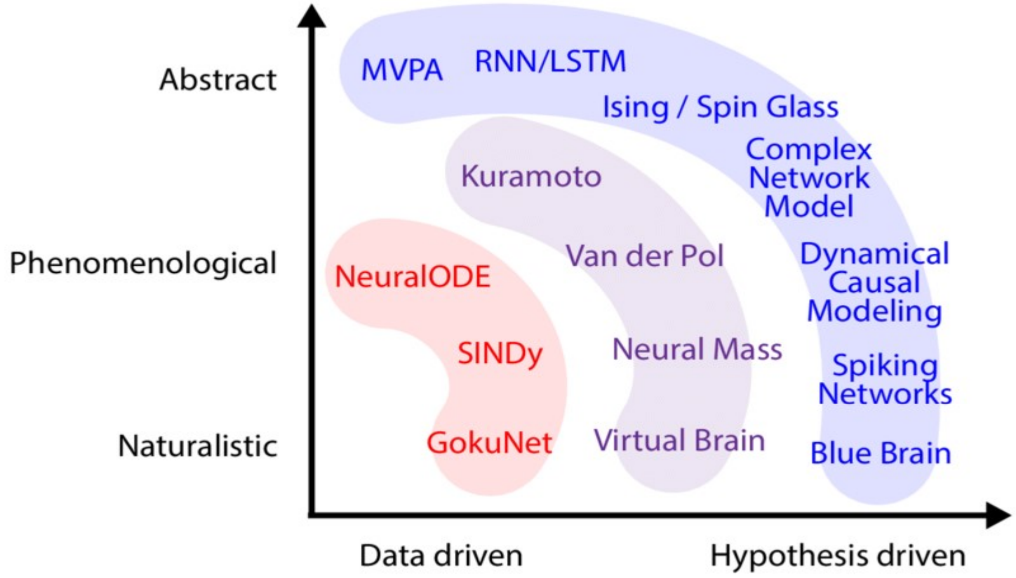

- Mathematical models used to simulate brain activity range from continuous non linear dynamical systems to network-based ones, where either you solve systems of ODE on the nodes or minimize energies in a neural network, as an optimization problem.

- Data-driven models, where information about anatomical networks are embedded into calculations (data assimilation) produce much better results