The emergence of connectivity in neuronal networks: from bootstrap percolation to auto-associative memory

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Neural Network

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- $X(t) = (X_i(t),\ i \in \Lambda)$ is a stochastic process describing some characteristic (e.g., membrane potential) of the ith neuron,

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- $W(t) = \{w_t(u, v), (u, v) \in L(t)\}$ is the set of synaptic strengths.

Neural Network as Interacting Particle System

System with interactions in a discrete time:

$$X_i(t+1) = F(\, X_j(t),\ j \in \mathcal{N}(i,t)\,).$$

The interacting neighbourhood
$\mathcal{N}(i,t) = \{(i,j) : \ (i,j) \in L(t)\}$ may be

- fixed $\mathcal{N}(i,t) = \{j \in \Lambda : |i-j| \le R\}$
(implying local interactions if $R < \infty$, or global if
$\mathcal{N}(i,t) = \Lambda$),

- or randomly chosen in Λ at each time t.

Connections in a Neural Network

$$\mathcal{L}(t) = \{(j,i),\ j \in \mathcal{N}(i,t),\ i \in \Lambda\}$$

- form A RANDOM DYNAMICAL GRAPH

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- WHAT IS A "PROPER" ARCHITECTURE OF A NEURAL NETWORK?

- "Modeling Neural Development", ed. by A. van Ooyen, The MIT Press, 2003.

  - *onset of the connections between the neurons.*

Main features of Neural Networks

- Activity-dependent connections:

widely accepted Hebb principle of modification, which says that

if one neuron caused a firing of another one then the strength of the corresponding connection between them is increasing,

while if the firing of one neuron does not affect firing of another one, the strength of the corresponding connection between them is decreasing.

What is Random in the structure of a Neural Network?

There are evidences (E. Rolls "Memory, Attention and Decision Making", Oxford University Press, 2008) that in some areas of brain, as hippocampus, each neuron within a network of N neurons receives on $average$ inputs from $C = C(N)$ neurons, chosen uniformly among $N - 1$ neurons.

Plausible assumptions:
the underlying (sub-) graph is a C-out random graph.

Death of some connections or neurons also contributes to the network formation (e.g., van Ooyen (2003)) .

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- Given a set of local updates in the network describe the resulting network after a long-time evolution.
- How to obtain a given (assumed) architecture in a course of some biologically plausible evolution of a large network of initially homogeneous units?
- WHAT YOUR NETWORK IS GOOD FOR?
- Hopfield model for associative memory.

Can we get a desired architecture?

- Is it possible to get a network with almost constant finite in- and out- degrees as a result of some natural evolution of a neural network, which initially admits all-to-all random connections?

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- Iglesias, J., Eriksson, J., Grize, F., Tomassini, M., Villa, A. E. P. Dynamics of pruning in simulated large-scale spiking neural networks. BioSystems 79, 11–20.

Model

TT and A. Villa ['07], TT ['11]

$$\Lambda = \Lambda^+ \cup \Lambda^- \subset Z^3$$

2 types of neurons:
at every vertex of Λ^+ there is an excitatory neuron,
at every vertex of Λ^- there is an inhibitory neuron.

$$n^\pm = |\Lambda^\pm|, \qquad |\Lambda| = n = n^+ + n^-, \qquad n^+/n^- = 4.$$

Initial graph: $G_{n,p}$ graph with a probability of connection between any pair

$$p = \frac{c}{n}.$$

Potential

- $X_v(t)$ denotes potential of a neuron at point $v \in \Lambda$ at time $t \in \{0, 1, \ldots\}$.
  $X_v(t) \geq 0$ is a random process.
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- No interactions: $X_v(t)$ are $i.i.d.$ $U[0, 1]$ for different v (hence, no firing).

Propagation of External Activation in a Network

- Initial Condition:
  $\mathcal{A}(0) = \mathcal{A}_-(0) \cup \mathcal{A}_+(0)$ is a set of active neurons,
  $\mathcal{A}_-(0)$ are inhibitory, $\mathcal{A}_+(0)$ are excitatory:

  $X_v(0) = 1$ for all $v \in \mathcal{A}(0)$.

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- Propagation
  Given $\mathcal{A}_+(t)$, $\mathcal{A}_-(t)$, and independent $X_v(t)$, $v \notin \mathcal{A}(t)$, define

$$\mathcal{A}(t+1) = \left\{ i : X_i(t+1) = \frac{X_i(t) + \sum_{j \in \mathcal{A}_+(t)} \omega_{ij}(t)\varepsilon_{ij}}{1 + \sum_{j \in \mathcal{A}_-(t)} \omega_{ij}(t)\varepsilon_{ij}} \geq 1 \right\},$$

$$\varepsilon_{ij} = \begin{cases} 1, & \text{if there is an edge } (i,j), \\ 0, & \text{otherwise.} \end{cases}$$

Dynamics of Activation with fixed $\omega_{ij}(t) = \omega$

- Neuron i fires at time $t+1$ if

$$X_i(t+1) = \frac{X_i(t) + \sum_{j \in \mathcal{A}_+(t)} \varepsilon_{ij}\omega}{1 + \sum_{j \in \mathcal{A}_-(t)} \varepsilon_{ij}\omega} \geq 1,$$

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- Rewrite as $X_i(t) + w\left(N_i^+(t) - N_i^-(t)\right) \geq 1,$

  where $N_i^+(t)$ and $N_i^-(t)$ are the numbers of excitatory and inhibitory connections to i from the firing set at time t.

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- Markov chain on $[0,1] \times [0,1]$

$$\left\{ \left(\frac{|\mathcal{A}_+(t)|}{n_+}, \frac{|\mathcal{A}_-(t)|}{n_-} \right), \; t = 0, 1, 2, \dots \right\}$$

Mean drifts

- $$D_+(q_+, q_-) := \lim_{n\to\infty} \frac{1}{n_+} \mathbf{E} \{ |\mathcal{A}_+(t+1)| - |\mathcal{A}_+(t)|$$

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- where $q_+, q_- \in [0, 1]$

  $$n_+ = 4n/5, \qquad n_- = n/5.$$

Stability conditions

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- in which case $(q_+, q_-) = (q, q)$.
  q is the unique solution to

$$q = \mathbf{P}\left\{Y(4qc/5) - Y'(qc/5) \geq \frac{X}{\omega}\right\},$$

  where $Y(a)$ and $Y'(a)$ denote independent Poission distributions with mean a, X is uniform on $[0, 1]$.

Numbers of in- and out-coming firing impulses per neuron

q is the unique solution to

$$q = \sum_{n=0}^{\frac{1}{\omega}-1} \omega \sum_{k=0}^{\infty} \sum_{l=k+n+1}^{\infty} e^{-cq} \frac{(4qc/5)^l}{l!} \frac{(qc/5)^k}{k!} =: p(q),$$

it is a stable point for the system $q_{i+1} = p(q_i)$.

- Let $N_n^{in,v}(q)$ and $N_n^{out,v}(q)$ denote random numbers of incoming and outcoming excitatory impulses for a firing neuron v in a network with excitation $qn_{\pm}$.

$$N_n^{in,v}(q), \quad N_n^{out,v}(q) \quad \to_P \quad 4cq/5 + p'(q)$$

as $n \to \infty$.

Propagation of Activation on $G_{n,p}$

$X_i(t) \in \{0,1\}$, $t = 0,1,\dots,$ $i \in \{1,\dots,n\}$.

- Let $\mathcal{A}(t)$ be the set of neurons activated by time t:

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- – *Bootstrap percolation on Random Graph* $G_{n,p}$.

Question of Interest: the Size of Total Activation

Let $A(t) = |\mathcal{A}(t)|$. Propagation of activation stops when

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- The size of the total activation is

$$A^* := \lim_{t\to\infty} A(t) = A(n).$$

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- Parameters of the model: $n, \quad r, \quad p(n), \quad A(0)$, where typically $r > 1$ and

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- Is it possible to have $A^* \sim qn, \quad q < 1$?

Dynamics of $A(t)$.

- Dynamics of $A(t)$ follows

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- The activation stops at t_c if $t_c < n$.
- Exists unique critical value a_c, such that if $A(0) = a_c$ there is a unique solution t_c to

$$S(t) = t$$

Sharp Phase Transition (r>1) along initial condition

THEOREM (Janson S., Łuczak T., TT, and Vallier T. ('10))
Define

$$a_c = \left(1 - \frac{1}{r}\right)\left(\frac{(r-1)!}{np^r}\right)^{1/(1-r)} \quad \ll n,$$

(i) If $\frac{A(0)}{a_c} \to \alpha < 1$, then

$$A^* = \phi(\alpha, r)a_c + o_P(a_c) \ll n,$$

where $\phi(\alpha, r)$ is the unique root in $[0, 1]$ of

$$r\phi - \phi^r = (r-1)\alpha.$$

(ii) If $\frac{A(0)}{a_c} \geq 1 + \delta$, $\delta > 0$, then $A^* = n - o_P(n)$.

Critical phase (r>1)

If $\frac{A(0)}{a_c} \to 1$, then A^* is distributed on $n - o(1)$ and $t_c = o(n)$.

Heuristic Argument

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Critical probabilities of edges

THEOREM (Janson S., Łuczak T., TT, and Vallier T. ('10))
Suppose that $r \geq 2$ and that $a \to \infty$ with $a = o(n)$. Then the threshold for p for almost percolation is

$$p_c = \left(\frac{(r-1)^{r-1}(r-1)!}{r^{r-1}} \right)^{1/r} \frac{n^{-1/r}}{a^{1-1/r}}$$

i.e., if, for some $\delta > 0$, $p \leq (1-\delta)p_c$ then

$$A^* \leq 2a = o(n) \quad w.h.p.,$$

while if $p \geq (1+\delta)p_c$ then $A^* = n - o_P(n)$.

Hopfield model

Hopfield (1982): Model for autoassiciative memory.
N neurons, each neuron i is characterized by the state $\sigma_i(t) \in \{-1, 1\}$ ("spins"), at time t.
The state space S_N is the set of configurations of spins:

$$S_N = \{\sigma = (\sigma_1, \ldots, \sigma_N) : \sigma_i \in \{-1, 1\}\}.$$

Given M numbers of patterns $\xi^1, \ldots, \xi^M$ in S_N define
Hopfield Hamiltonian

$$H_N(\sigma) = -\sum_{i,j=1}^{N} J_{ij}\sigma_i\sigma_j,$$

where $J_{ij} = \frac{1}{N}\sum_{\mu=1}^{M} \xi_i^\mu \xi_j^\mu$.

Storage capacity

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- The storage capacity is the greatest number $M(N)$ such that the patterns ξ^μ are stable (modification: close to stable in some precise sense.)
- When ξ_i^μ are $i.i.d.$ then the storage capacity is αN.
  Hopfield (1982), numerics, $\alpha \geq 0.14$
  Neuman (1988): $\alpha \geq 0.055$

Diluted Hopfield Model

- $$H_N(\sigma) = -\frac{1}{N}\sum_{i,j=1}^{N}\sum_{\mu=1}^{M} \varepsilon_{ij}\sigma_i\sigma_j\xi_i^\mu\xi_j^\mu,$$

  where ε_{ij} are $i.i.d.$ $Be(p)$ for different pairs (i,j).

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- Theorem (Bovier and Gayrard (1992)). If

  $$p > c \left(\frac{\log N}{N} \right)^{1/2}$$

  then the storage capacity of the diluted network is at least

  $$\alpha(c) p N.$$

Discussion

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  What they are good for?
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- carefully specify which part of brain and which brain function the model concerns.
- study the data and at best ask the experts on the related known models:
  What they are good for?
  What are the functions of the model?
- Roughly, one can say that there are models on the collective result of network performance as, e.g., memory (macro-level),
  and the models of dynamics of chemical/electrical currents between the neurons (micro-level).

There are many very recent experimental studies of the brain where the neurobiologists are making link with known theoretical results on random graphs and networks.