IJCNN 2005 Tutorial - Biologically Plausible Artificial Neural Networks

Biologically Plausible Artificial Neural Networks

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Purpose of this Tutorial

- An attempt to restore biological inspiration of current connectionist systems;
- Two parts:
  - Part 1: History, artificial models, the brain, and Hebbian learning,
  - Part 2: Learning algorithms, connectionist representations, neuron signalling, and a model proposal.

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Part 1 Outline

- Artificial Neural Networks - History
- Simplified mathematical models for the neuron
- Human brain
- Hebbian learning

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Part 2 Outline

- Back-propagation
- Generalized recirculation
- Connectionist representations
- Intraneuron and interneuron signaling
- A biologically plausible ANN model proposal
- Conclusions

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Part 1

- **Artificial Neural Networks - History**
- Simplified mathematical models for the neuron
- Human brain
- Hebbian learning

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Artificial Neural Networks

- Based on an abstract and simplified view of the neuron;
- Artificial neurons connected and arranged in layers to form large networks;
- Learning and connections determine the network function;
- Connections can be formed through learning and do not need to be 'programmed'.

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Artificial Neural Networks

- Recent ANN models:
  - lack many physiological properties of the neuron;
  - more oriented to computational performance than to biological credibility;
- Purpose of this Tutorial:
  - an attempt to restore biological inspiration of current connectionist systems.

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Artificial Neural Networks

- A biologically inspired connectionist approach should present:
  - neurophysiologically motivated training algorithm;
  - bi-directional connectionist architecture;
  - several other features, e. g., distributed representations.

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History

– 1943: McCulloch and Pitts: first mathematical neuron model;

– 1948: Wiener's *Cybernetics* book;

– 1949: Von Neumann's speech at University of Illinois: McCulloch and Pitts theory propagation;

– 1949: Donald Hebb: *The Organization of Behavior*: learning rule for synaptic modification.

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History

– 1951: Minsky and Edmonds: first ANN computer:
  • *SNARC - Stochastic Neural-Analog Reinforcement Computer*;

– 1957: Rosenblatt: perceptron;

– 1960: Widrow and Hoff: adaline;

– 1969: Minsky and Papert: *Perceptrons*:
  • book with negative result on representation capability of 1-layer ANNs.

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History

– 1969: Bryson and Ho: discovery of Back-propagation learning algorithm;

– 1980: Grossberg: self-organization;

– 1982: Hopfield: recurrent networks with symmetrical synaptic connections;

– 1982: Kohonen: self-organizing maps;

– 1985: Ackley, Hinton, and Sejnowski: *Boltzmann Machine*: first succeeded multi-layer ANN.

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History

– 1986: McClelland and Rumelhart: *Parallel Distributed Processing: Explorations in the Microstructures of Cognition* (2 volumes):
  - Back-propagation reinvented;

– 1988: Broomhead and Lowe: radial basis functions (RBF): option to multi-layer perceptrons.

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History

– Fu (1991 and 1993), Towell and Shavlik (1993), Setiono and Liu (1996), Omlin and Giles (1996), d'Avila Garcez *et al.* (2001): symbolic knowledge extraction from ANNs → symbolic-connectionist approach → knowledge-based ANNs.

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Part 1

- Artificial Neural Networks - History
- **Simplified mathematical models for the neuron**
- Human brain
- Hebbian learning

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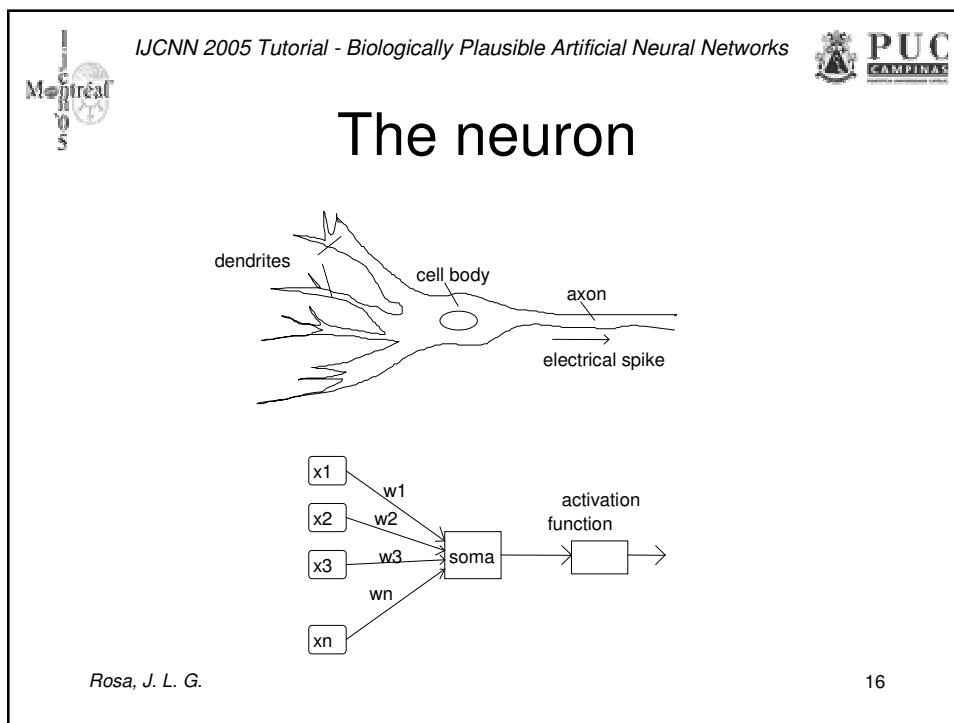*IJCNN 2005 Tutorial - Biologically Plausible Artificial Neural Networks*

The neuron

Soma (cell body)

Axon

Dendrite

10μm

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The neuron

dendrites

cell body

axon

electrical spike

x1

w1

x2

w2

activation
function

x3

w3

soma

wn

xn

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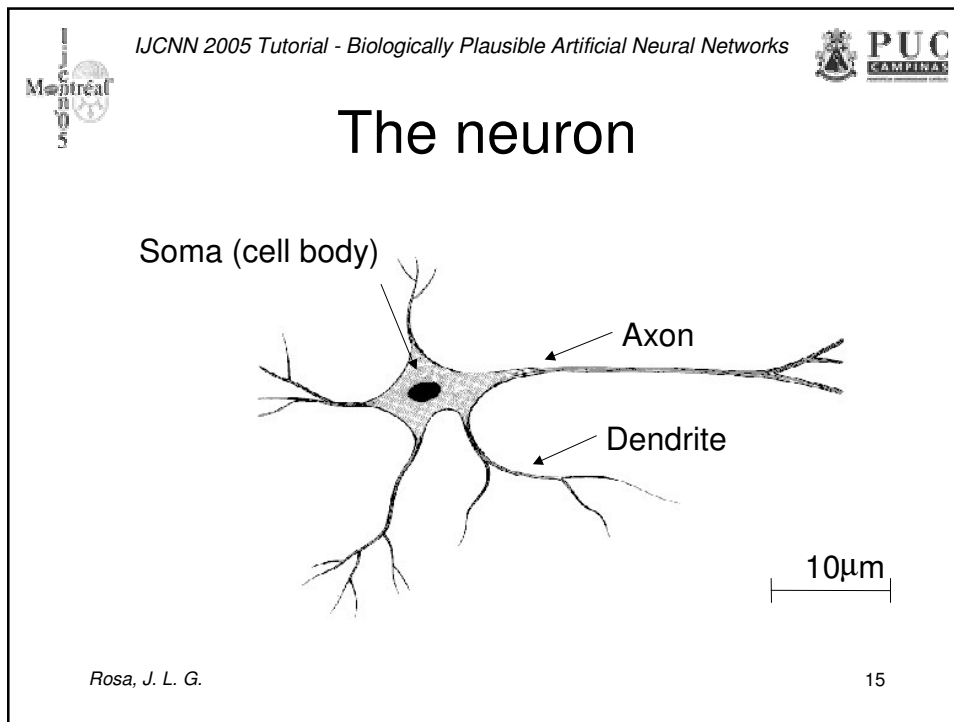
McCulloch-Pitts neuron (1943)

- First mathematical model:

   1. neuron activity → "all-or-none" process;

   2. a certain fixed number of synapses → excited within a latent addition period → to excite a neuron: independent of previous activity and of neuron position.

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McCulloch-Pitts neuron (1943)

   3. only significant delay in nervous system: synaptic delay;

   4. activity of any inhibitory synapse prevents neuron from firing;

   5. network structure does not change along time.

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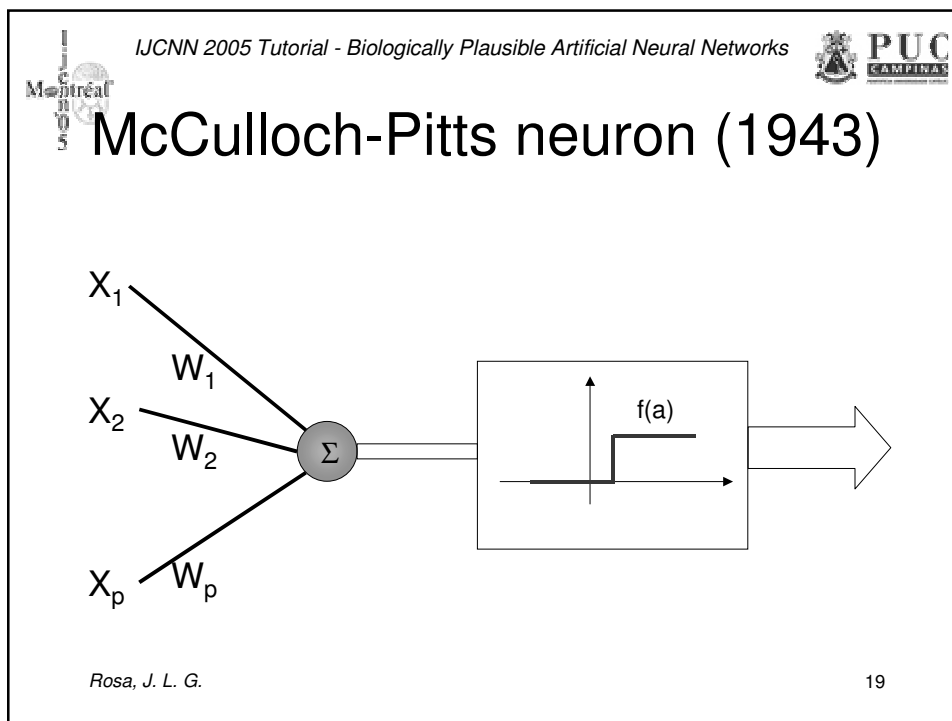
McCulloch-Pitts neuron (1943)

X_1

W_1

X_2

W_2

Σ

f(a)

X_p W_p

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Simplified mathematical model for the neuron

- x_i: inputs (binary);
- w_i : synaptic weights (real, because the synapses can inhibit (-) or excite (+) and have different intensities);
- computation occurs in soma:

$$\sum_{i=0}^{n} x_i \cdot w_i$$

$x_0 = 1$ and $w_0 = \beta = -\theta$

$\beta = bias$ and $\theta = $ activation threshold.

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Simplified mathematical model for the neuron

- The activation function can be:
  - hard limiter,
  - threshold logic,
  - sigmoid.
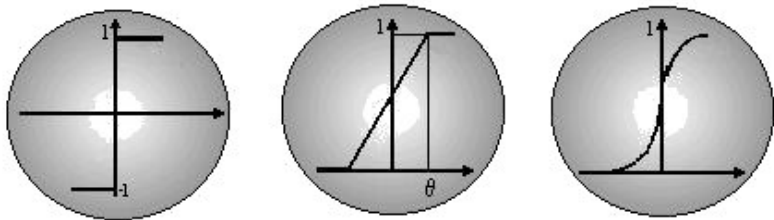
    - the biologically more plausible → sigmoid function.

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Activation functions

Hard Limiter   Threshold Logic   Sigmoid

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The perceptron

- Rosenblatt's perceptron (1957):
  - takes a weighted sum of neuron inputs,
  - sends output 1 (*spike*) if this sum > activation threshold.

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The perceptron

- A linear discriminator:
  - given 2 points, a straight line is able to discriminate them;
  - for some configurations of *m* points, a straight line is able to separate them in two classes.

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Set of linearly separable points

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Set of non-linearly separable points (by one straight line)

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Exclusive-OR

- {(0,0), 0; (0,1), 1; (1,0), 1; (1,1), 0}

- and by *n* straight lines?

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Perceptron limitations

- One-layer feed-forward network (non-recurrent);
- Only capable of learning solution of linearly separable problems;
- Its learning algorithm (delta rule) does not work with networks of more than one layer.

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Learning algorithm LMS

- Learning algorithm used by one-layer perceptrons:
  - LMS = Least-Mean-Square;
- Training set:
  - m input units x,
  - n desired output units t,
  - l patterns of the type $((x_1,...,x_m), (t_1,...,t_n))$.

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Learning algorithm LMS

- The l training patterns presented to the network;
- An error measurement produced by the network;
- The error is function of:
  - each pattern, and
  - error produced in each output unit, when each pattern is presented.

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Learning algorithm LMS

- If...
  - the network learns perfectly the training patterns, and
  - the training patterns reflect perfectly the task that is intended to learn;
- Then...
  - after training, the error will be zero.

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Learning algorithm LMS

- The error comes from the differences between real and desired outputs → consequence of the output produced by incorrect weights (and biases);

- To learn = to find the weights which make the error minimum.

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Learning algorithm LMS

- The total error *E* after training is:

$$E(w) = \sum_{p=1}^{l} E_p$$

  – where E_p is the error produced when the *p-th* training pattern is presented to the network;

- The most popular error measurement → mean quadratic error:

$$E_p(w) = \frac{1}{2} \sum_{k=1}^{n} (t_k - y_k)^2$$

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Neural network topology

- In cerebral cortex:
  – neurons disposed in columns,
  – most synapses occur between different columns;
- In the extremely simplified mathematical model:
  – neurons disposed in layers (representing columns),
  – there is communication between neurons in different layers.

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ANN topology

output units

$w2_{ij}$

1   h_1 h_2 h_3 . . . h_B hidden units

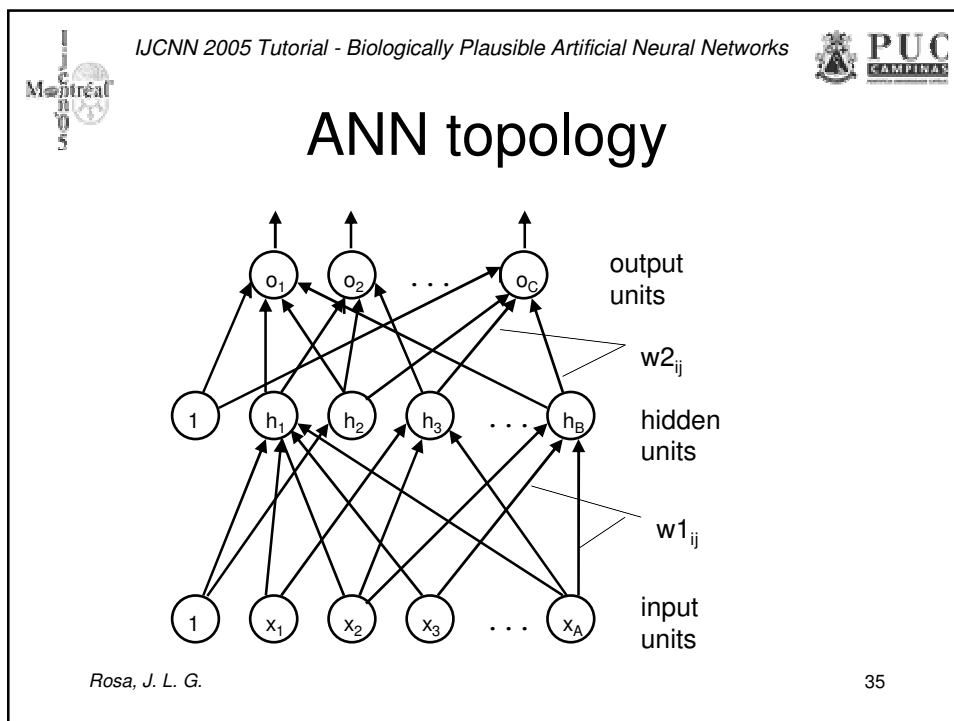$w1_{ij}$

1   x_1 x_2 x_3 . . . x_A input units
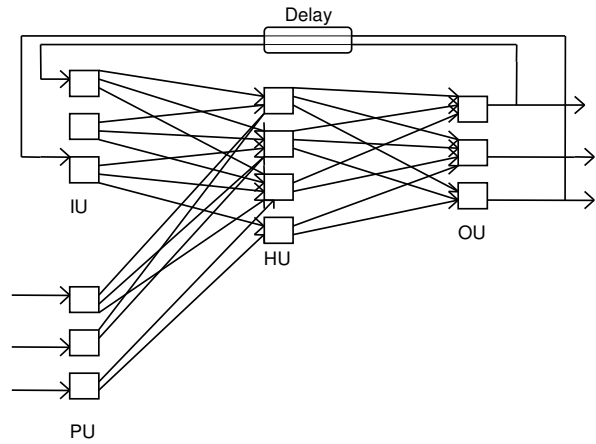
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ANN topology

- Types of connectionist networks:
  - feed forward multi-layer,
  - recurrent multi-layer:
    - at least one feedback loop, at the same unit, or between layers.

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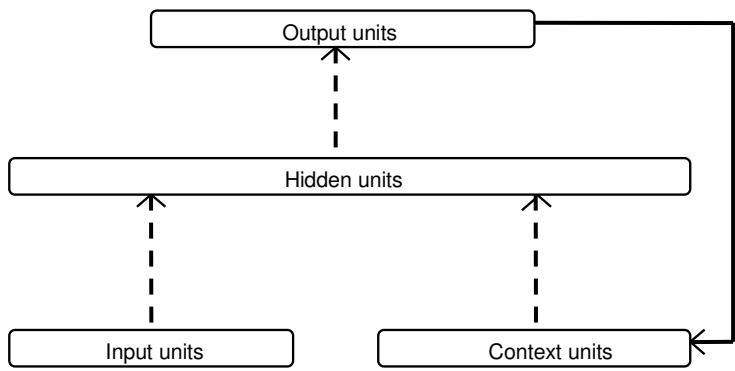# Jordan's recurrent network

(Source: Jordan, 1986)

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Elman's recurrent network

(Source: Elman, 1988)

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Classical ANN Models

- Based upon a simple description of the neuron, taking into account:
  - the presence of presynaptic cells and their synaptic potentials,
  - the activation threshold, and
  - the propagation of an action potential;
- Represent impoverished explanation of human brain characteristics.

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Advantages of ANNs

- Naturally parallel solution,
- Robust, fault tolerant,
- Allow integration of information from different sources or kinds,
- Adaptive systems, that is, capable of learning,
- Show a certain autonomy degree in learning,
- Display a very fast recognizing performance.

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Limitations of ANNs

- Still very hard to explain its behavior, because of lacking of transparency;
- Solutions do not scale well ...
  - computationally expensive for big problems;
- Yet very far from biological reality.

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Part 1

- Artificial Neural Networks - History
- Simplified mathematical models for the neuron
- **Human brain**
- Hebbian learning

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Inspiration

"Almost all aspects of life are engineered at the molecular level, and without understanding molecules we can only have a sketchy understanding of life itself."

Francis Crick, *What Mad Pursuit*, 1988.

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Human brain

- Natural evolution endowed human brain with many desirable characteristics, not present in von Neumann machine:
  – massive parallelism,
  – distributed representation and computation,
  – learning ability,
  – generalization ability,
  – adaptability,
  – inherent contextual information processing,
  – fault tolerance,
  – low energy consumption.

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Human brain

	Von Neumann computer	*Biological neural system*
Processor	Complex High speed One or few	Simple Low speed A large number
Memory	Separated from processor Localized Non-content addressable	Integrated with processor Distributed Content addressable
Computing	Centralized Sequential Stored programs	Distributed Parallel Self-learning
Reliability	Very vulnerable	Robust
Expertise	Numeric and symbolic manipulations	Perceptual problems
Operational environment	Well-defined, well-constrained	Poorly defined, unconstrained

(from Jain *et al.*, 1996)

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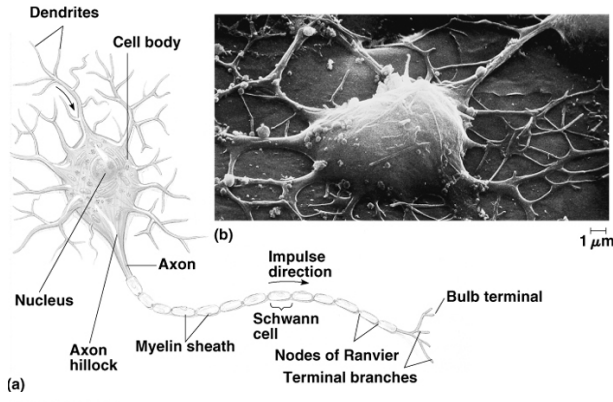
Human brain

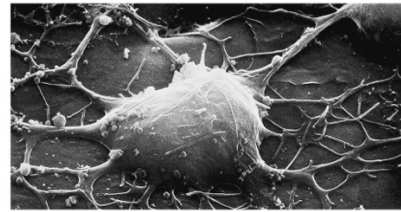|	*Computer*	*Human brain*
Computational units	1 CPU, 10 gates	10^{11} neurons
Storage units	10^9 bit RAM, 10^{10} bit disk	10^{11} neurons, 10^{14} synapses
Cycle time	10^{-8} sec.	10^{-3} sec.
Bandwidth	10^9 bits/sec.	10^{14} bits/sec.
Neuron updates /sec.	10^5	10^{14}

(from Russell and Norvig, 1995)

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The real neuron

Dendrites
Cell body
Axon
Nucleus
Axon hillock
Myelin sheath
Schwann cell
Impulse direction
Nodes of Ranvier
Terminal branches
Bulb terminal
1 μm
(a)
(b)
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(from MUN, 2005)

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The real neuron

- Axons:
  - 100 microns (typical granule cell);
  - 4.5 meters (giraffe primary afferent).

(source: Eliasmith and Anderson, 2002)

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Na-K pump

A

Stimulus

Na^+ Na^+ Na^+

B

K^+ K^+ K^+

Na^+ Na^+ Na^+

(from Biello, 2005)

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Measuring the potential

Measuring device

Axon

Recording electrodes

(from Biello, 2005)

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The action potential

(from MUN, 2005)

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The action potential

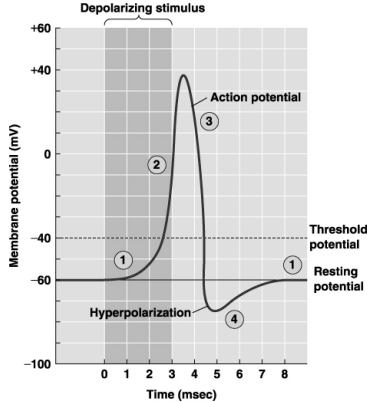(from Biello, 2005)

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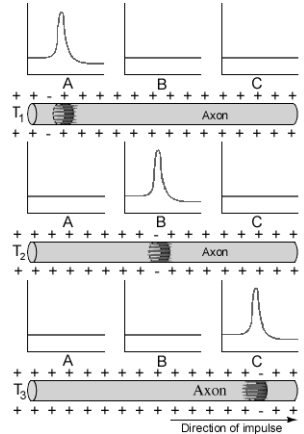
The action potential

- Communication through spikes:
  - stereotypical (common pyramidal cells),
  - none (retinal horizontal cells);
- Spike speed:
  - 2 km/h,
  - 400 km/h.

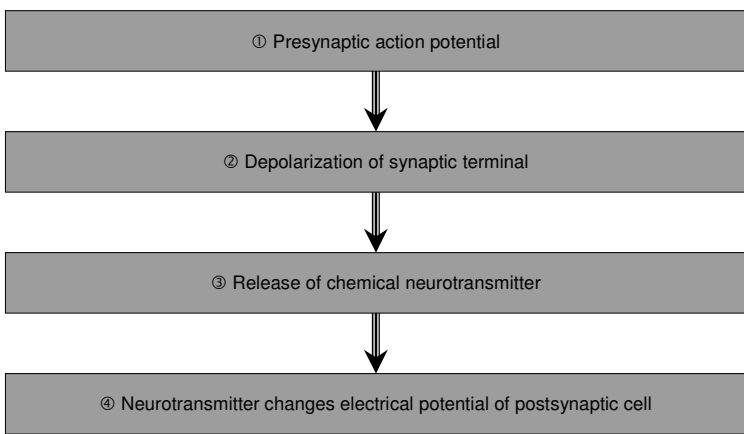(source: Eliasmith and Anderson, 2002)

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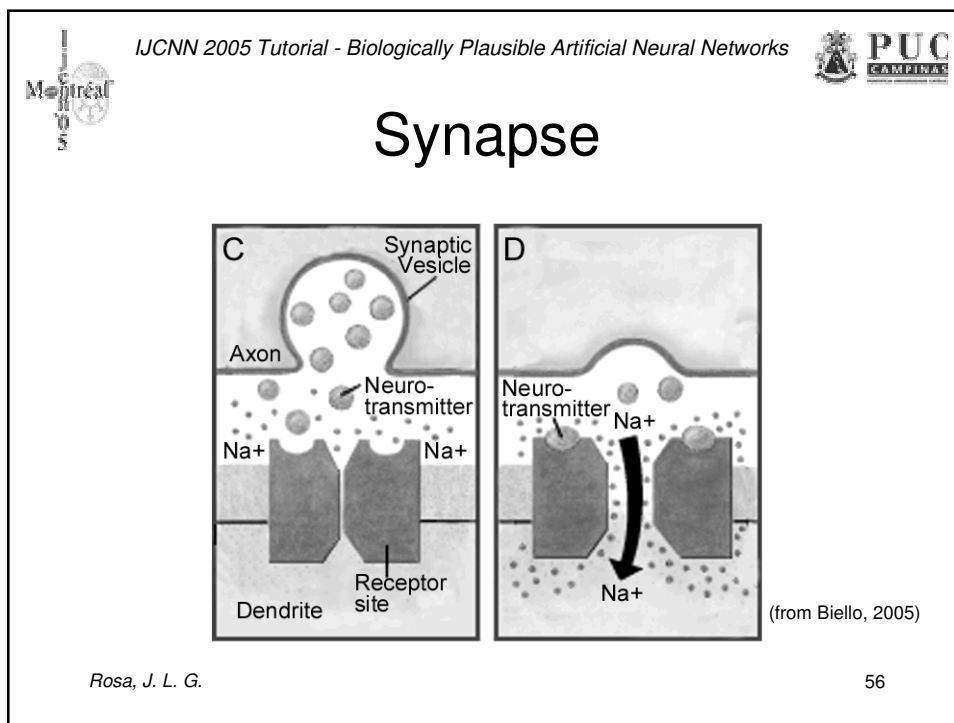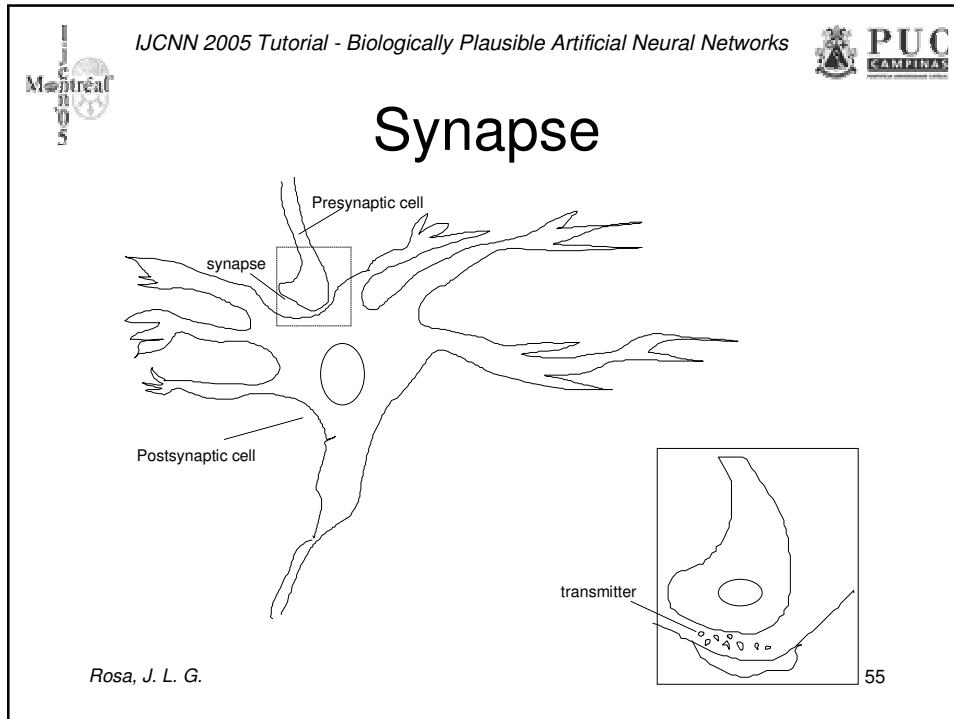*IJCNN 2005 Tutorial - Biologically Plausible Artificial Neural Networks*

The action potential

| ① Presynaptic action potential |

↓

| ② Depolarization of synaptic terminal |

↓

| ③ Release of chemical neurotransmitter |

↓

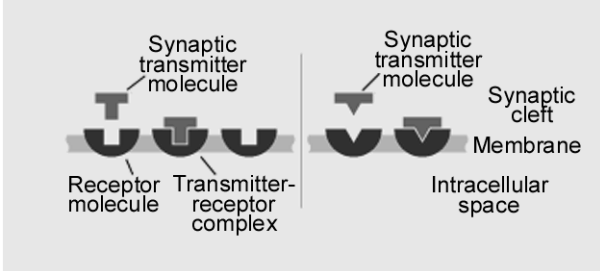| ④ Neurotransmitter changes electrical potential of postsynaptic cell |

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Synapse

Presynaptic cell

synapse

Postsynaptic cell

transmitter

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Synapse

C　Synaptic Vesicle

Axon

Neuro-transmitter

Na+　　Na+

Dendrite　Receptor site

D

Neuro-transmitter　Na+

Na+

(from Biello, 2005)

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Synapse

(from Biello, 2005)

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Synapse
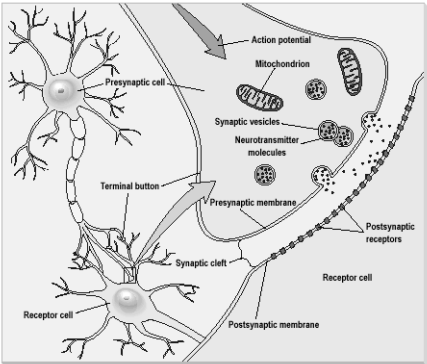

(from Biello, 2005)

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Synapse

- The synapse can be:
  - Type I – Excitatory (A): neurons contribute to produce impulses on other cells;
  - Type II – Inhibitory (B): neurons prevent the releasing of impulses on other cells.

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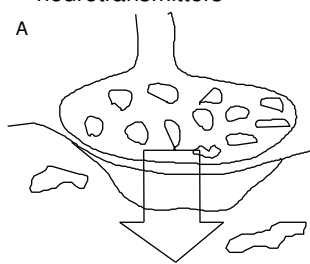
Synapse

EXCITATORY SYNAPSE:
- asymmetrical membrane specializations,
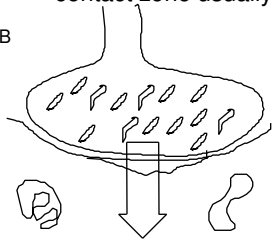- very large synaptic vesicles (50 nm) with packets of neurotransmitters

INHIBITORY SYNAPSE:
- symmetrical membrane specializations,
- synaptic vesicles are smaller and often ellipsoidal or flattened,
- contact zone usually smaller

A   B
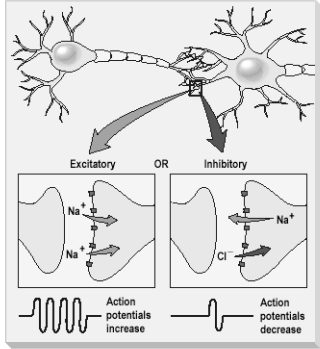
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Synapse

(from Biello, 2005)

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Synapse

(from Matthews, 2001)
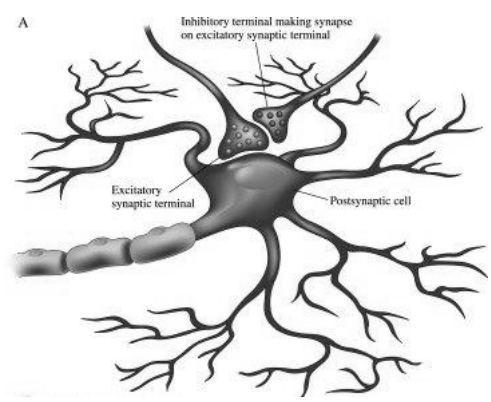
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Computation in CNS

- Input connections:
  - about 500 (retinal ganglion cells),
  - about 200,000 (purkinje cells);
- Hundreds or thousands of different kinds of neurons:
  - massively parallel,
  - very simple processing elements,
  - more than 100 billion neurons in human brain,
  - at least 100 trillion synapses,
  - 72 km of fiber,
  - 100 different kinds of neurotransmitters.
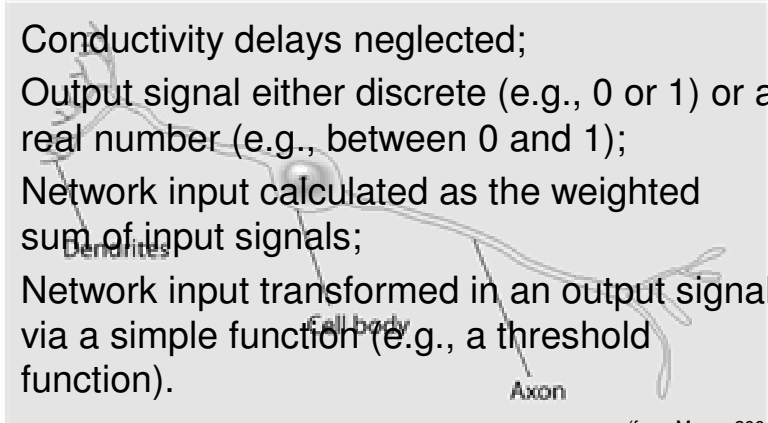
Rosa, J. L. G.   (source: Eliasmith and Anderson, 2002)

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ANNs do not focus on real neuron details

- Conductivity delays neglected;
- Output signal either discrete (e.g., 0 or 1) or a real number (e.g., between 0 and 1);
- Network input calculated as the weighted sum of input signals;
- Network input transformed in an output signal via a simple function (e.g., a threshold function).

Dendrites

Cell body

Axon

(from Murre, 2001)

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Part 1

- Artificial Neural Networks - History
- Simplified mathematical models for the neuron
- Human brain
- **Hebbian learning**

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Donald Olding Hebb

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Donald Hebb (1949)

"When an axon of cell A is near enough to excite a cell B and repeatedly or persistently takes part in firing it, some growth process or metabolic change takes place in one or both cells such that A's efficiency, as one of the cells firing B, is increased."

(source: Hebb, 1949)

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Donald Hebb (1949)

- Also, the word *connectionism* appeared for the first time:

"*The theory is evidently a form of connectionism, one of the switchboard variety, though it does not deal in direct connections between afferent and efferent pathways: not an 'S-R' psychology, if R means a muscular response. The connections server rather to establish autonomous central activities, which then are the basis of further learning*"

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Hebb hypothesis

- Knowledge: associations;
- Plasticity in CNS:
  - synapses: created and destroyed,
  - synaptic weights change values,
  - allow learning:
    - internal self-organizing:
      - encoding of new knowledge,
      - reinforcement of existent knowledge.

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Hebb hypothesis

- How to supply a neural substrate to association learning among world facts?
- Hebb hypothesis (1949):
  - connections between two nodes highly activated at the same time are reinforced,
  - this kind of rule is a formalization of the associationist psychology:
    - associations accumulated among things that happen together.

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Hebb hypothesis

- Permits to model the CNS plasticity, adapting it to environmental changes, through:
    - excitatory and inhibitory strength of existing synapses, and
    - its topology;
- Allows that a connectionist network learns correlation among facts.

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Connectionist systems are able to learn

- Connectionist networks learn:
    - through synaptic weight change:
        - statistical correlations from the environment;
    - through network topology change (in a few models);
- Probabilistic reasoning: without a statistical model of the problem.

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Two learning methods are possible with Hebbian learning

- Unsupervised learning,
- Supervised learning;

- In *unsupervised learning* there is no teacher:
  - the network tries to find regularities in the input patterns.

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Two learning methods are possible with Hebbian learning

- In *supervised learning*, input associated with output:
  - if input = output $\rightarrow$ *auto-associative* learning,
  - if input ≠ output $\rightarrow$ *hetero-associative* learning.

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Break

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Part 2

- **Back-propagation**
- Generalized recirculation
- Connectionist representations
- Intraneuron and interneuron signaling
- A biologically plausible ANN model proposal
- Conclusions

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Back-propagation

- Supervised algorithm for multilayer networks
  - applies the generalized delta rule;
  - requires two passes of computation:
    - activation propagation (forward pass),
    - error back propagation (backward pass).

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Back-propagation

1. Propagate the activation:
   - from input to hidden layer,
   - from hidden to output layer;
2. Calculate the error:
   - for output units,
   - back propagate the error to hidden units and then to input units;
     - passes 1 and 2 constitute a cycle.

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Back-propagation

- The problems with BP:
  - computationally expensive (slow),
  - does not solve satisfactorily big size problems,
  - sometimes, the solution found is a local minimum – a locally minimum value for the error function.

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Back-propagation

- Advantages of BP:
  - universal approximation power:
    - given a continuous function, there is a two-layer network (one hidden layer) that can be trained by Back-propagation in order to approximate as much as desired this function,
  - the most used algorithm.

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The biological implausibility of Back-propagation

- BP is considered biologically implausible:
  - the reason: based on the error back propagation:
    - stimulus propagates forwardly,
    - error (difference between the actual and the desired outputs) propagates backwardly.

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The biological implausibility of Back-propagation

Real outputs

C

B   Activation

A

Input presentation

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The biological implausibility of Back-propagation

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The biological implausibility of Back-propagation

- In the cerebral cortex:
  - stimulus generated when a neuron fires → crosses the axon towards its end → to make a synapse onto another neuron input.

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The biological implausibility of Back-propagation

– Suppose that BP occurs in the brain:
  • the error must have to propagate back:
    – from the dendrite of the postsynaptic neuron,
    – to the axon and
    – then to the dendrite of the presynaptic neuron;
  • It sounds unrealistic and improbable.

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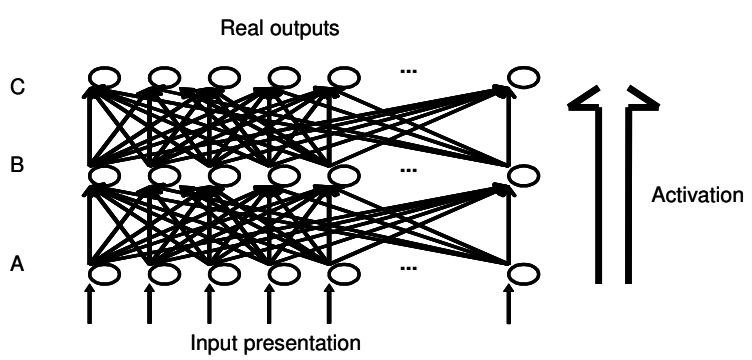
The biological implausibility of Back-propagation

• Synaptic "weights" have to be modified in order to make learning possible:

  – but certainly not in the way BP does,

  – weight change must use only local information in the synapse where it occurs,

  – that's why BP seems to be so biologically implausible.

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Part 2

- Back-propagation
- **Generalized recirculation**
- Connectionist representations
- Intraneuron and interneuron signaling
- A biologically plausible ANN model proposal
- Conclusions

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Generalized Recirculation algorithm[*]

- Supervised connectionist algorithm based on BP,
- Bi-directional architecture,
- Argued to be biologically more plausible,
- Consists of two phases: the *minus* and the *plus* phase.

(* Source: O'Reilly, 1996)

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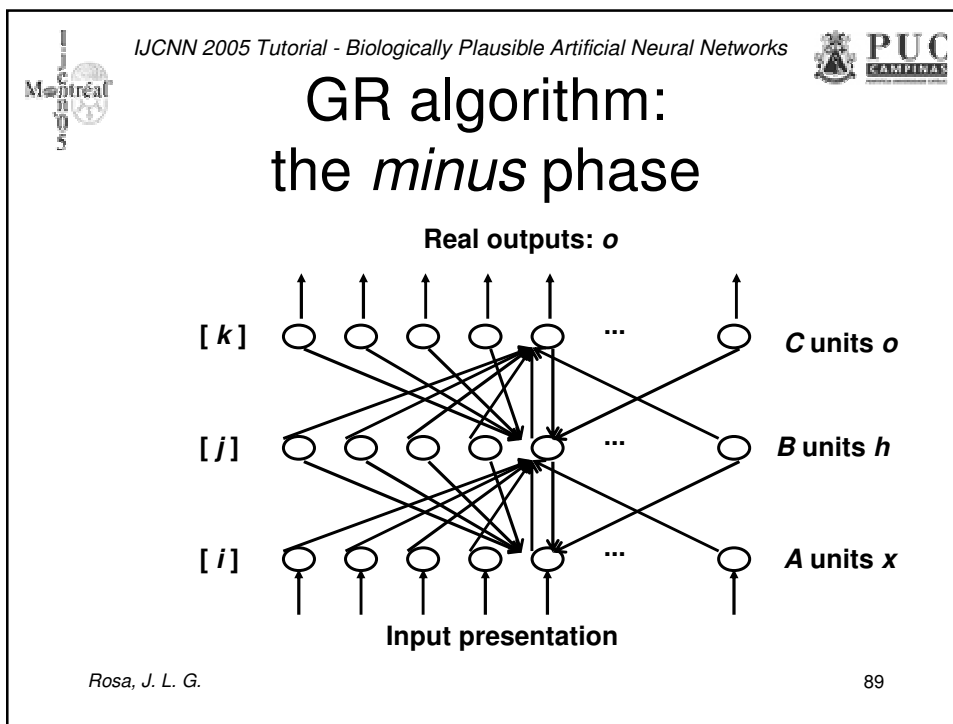
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GR algorithm:
the *minus* phase

Real outputs: *o*

[*k*] ◯ ◯ ◯ ◯ ◯ ⋯ ◯   ***C* units *o***

[*j*] ◯ ◯ ◯ ◯ ◯ ⋯ ◯   ***B* units *h***

[*i*] ◯ ◯ ◯ ◯ ◯ ⋯ ◯   ***A* units *x***

Input presentation

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GR algorithm:
the *minus* phase

$$h_j^{-} = \sigma(\sum_{i=0}^{A} w_{ij}.x_i(t) + \sum_{k=1}^{C} w_{jk}.o_k(t-1))$$

σ = sigmoid activation function

$$o_k(t) = \sigma(\sum_{j=1}^{B} w_{jk}.h_j^{-})$$
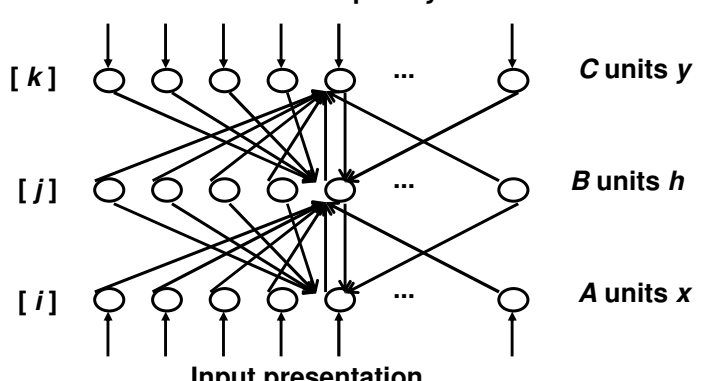
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GR algorithm:
the *plus* phase

Desired Outputs: *y*

[*k*] ... *C* units *y*

[*j*] ... *B* units *h*

[*i*] ... *A* units *x*

Input presentation

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GR algorithm:
the *plus* phase

$$h_j^+ = \sigma(\sum_{i=0}^{A} w_{ij}.x_i(t) + \sum_{k=1}^{C} w_{jk}.y_k(t))$$

$$\Delta w_{jk} = \eta.(y_k(t) - o_k(t)).h_j^-$$

$$\Delta w_{ij} = \eta.(h_j^+ - h_j^-).x_i(t)$$

η = learning rate

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A simple application

BP-GR comparison for digit learning

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Part 2

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Connectionist representation

- Need for representation → to receive information from and send information to the environment;
- Two types:
  - external representation:
    - used in input and output units,
  - internal representation:
    - associated to the hidden units.

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Connectionist representation

- External representation:
  - localist representation:
    - each entity or concept or feature → represented by only one processing unit,
    - the semantics of a unit → independent from the interpretations associated to other units,
    - less biologically plausible,
    - less economic in resources,
    - does not represent similarities between concepts.

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Connectionist representation

– distributed representation:
- each entity → represented by one activation pattern distributed among more than one unit,
- each unit → takes part in the representation of more than one entity,
- the interpretation of a given representation → obtained from the analysis of the activation global pattern of several units,
- the interpretation of the activation of one unit can make no sense.

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Connectionist representation

– distributed representation (continuation):
- the semantics associated is not compositional,
- more biologically plausible,
- more economic,
- much greater representational power → allows to represent similarities among concepts,
- radically different from symbolic representations.

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Connectionist representation

- The choice of external information representation is a key point in modeling:
    - connectionist networks capture statistical regularities present in the environment (training set),
    - the network only captures the information that the external representation can code,
    - the external representation can be the difference between learning or not.

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Part 2

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Santiago Ramón y Cajal

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Ramón y Cajal

- Principle of connectional specificity:
  - "nerve cells do not communicate indiscriminately with one another or form random networks";
- Principle of dynamic polarization:
  - "electric signals inside a nervous cell flow only in a direction: from neuron reception (often the dendrites and cell body) to the axon trigger zone".

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Intraneuron signaling

- Based on the principle of dynamic polarization, proposed by Ramón y Cajal;
- The signaling inside the neuron is performed by four basic elements:
  - receptive,
  - trigger,
  - signaling,
  - secretor.

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Intraneuron signaling

- *Receptive*: responsible for input signals → related to the dendritic region;
- *Trigger*: responsible for neuron activation threshold → related to the soma;
- *Signaling*: responsible for conducting and keeping the signal → related to the axon; and
- *Secretor*: responsible for signal releasing to another neuron → related to the presynaptic terminals of the biological neuron.

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The biological neuron and its morphological regions

(from Matthews, 2001)

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The biological neuron and its morphological regions

(from Matthews, 2001)

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Interneuron signaling

- Electrical and chemical synapses:
  - have completely different morphologies.

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The electrical synapse
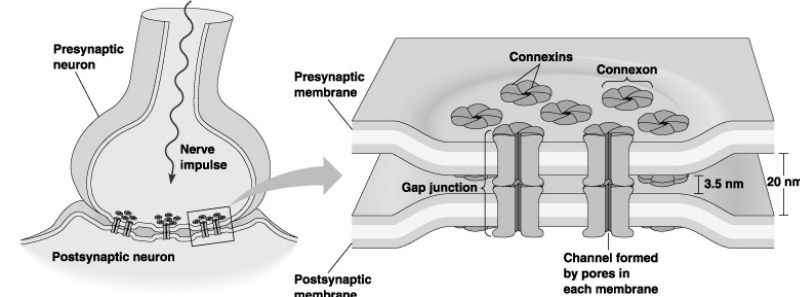
- At electrical synapses:
  - transmission occurs through gap junction channels (special ion channels),
    - located in the pre and postsynaptic cell membranes → cytoplasmatic connection between cells;
  - part of electric current injected in presynaptic cell escapes through resting channels;
  - remaining current driven to the inside of the postsynaptic cell through gap junction channels.

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The electrical synapse

(a) An electrical synapse

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(b) Gap junctions

(from MUN, 2005)

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The chemical synapse

- At chemical synapses:
  - synaptic cleft: small cellular separation between the cells;
  - vesicles containing neurotransmitter molecules in the presynaptic terminal;
  - when action potential reaches these synaptic vesicles → neurotransmitters released to the synaptic cleft.

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The chemical synapse

(from MUN, 2005)

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The chemical synapse

(from Matthews, 2001)

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Part 2

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A biologically plausible ANN model proposal

- Proposal for a biologically plausible model (Rosa, 2001):
  - to present a mechanism to generate a biologically plausible ANN model;
  - to redesign the classical framework to encompass:
    - the traditional features,
    - labels that model the binding affinities between transmitters and receptors.

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A biologically plausible ANN model proposal

- Departs from a classical connectionist model;
- Defined by a restricted data set, which explains the ANN behavior;
- Introduces T, R, and C variables → to account for the binding affinities between neurons (unlike other models).

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A biologically plausible ANN model proposal

- The following feature set defines the neurons:

$$N = \{\{w\}, \theta, g, T, R, C\}$$

  where:
  - *w* represents the connection weights,
  - θ is the neuron activation threshold,
  - *g* stands for the activation function,
  - T symbolizes the transmitter,
  - R the receptor, and
  - C the controller.
- θ, *g*, T, R, and C → genetic information,
- T, R, and C → labels, absent in other models.

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A biologically plausible ANN model proposal

- Follows Ramón y Cajal's *principle of connectional specificity*:
  - each neuron connected to another neuron:
    - not only in relation to $\{w\}$, θ, and *g*,
    - but also in relation to T, R, and C;
  - neuron *i* is only connected to neuron *j* → if there is binding affinity between the T of *i* and the R of *j*.

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A biologically plausible ANN model proposal

  - binding affinity means:
    - compatible types,
    - enough amount of substrate, and
    - compatible genes;
  - T + R = C;
  - C can act over other neuron connections.
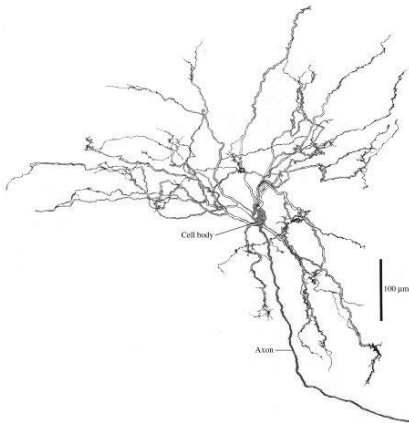
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The ordinary biological neuron

(from Matthews, 2001)

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The ordinary biological neuron

- Presents:
  - many dendrites usually branched → receive information from other neurons,
  - an axon → transmits the processed information → usually by propagation of an action potential;
  - the axon → divided into several branches → make synapses onto the dendrites and cell bodies of other neurons.

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Chemical synapse is predominant
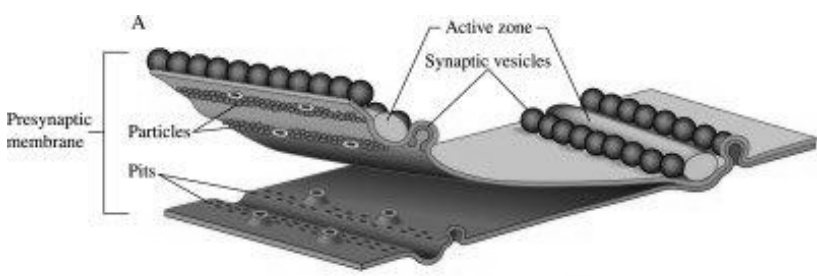
- Release of transmitter substance occurs in active zones, inside presynaptic terminals;
- Certain chemical synapses lack active zones → synaptic actions between cells are slower and more diffuse;
- neurotransmitter + receptor → makes the postsynaptic cell releases a protein.

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Active zone and synaptic vesicles

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Morphological synapses

- Although type I synapses seem to be excitatory and type II synapses inhibitory:
  - the action of a transmitter in the postsynaptic cell:
    - does not depend → chemical nature of the neurotransmitter,
    - depends → properties of the receptors with which the transmitter binds.

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Morphological synapses

  - in some cases, it is the receptor that determines whether:
    - a synapse is excitatory or inhibitory, and
    - an ion channel will be activated:
      - directly by the transmitter or
      - indirectly through a second messenger.

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Ion channels

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Second messengers

① Neurotransmitter is released by presynaptic neuron

② Neurotransmitter combines with specific receptor in membrane of postsynaptic neuron

③ Combination of neurotransmitter with receptor leads to intracellular release or production of a second messenger

④ Second messenger interacts (directly or indirectly) with ion channel, causing it to open or close

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Graded potentials

- 2 types of signaling:
  - propagation of action potential,
  - production of a graded potential by the axon;
- Graded potential signaling does not occur over long distances → because of attenuation.

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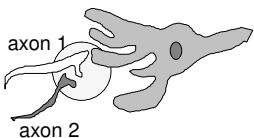
Graded potentials can occur in another level
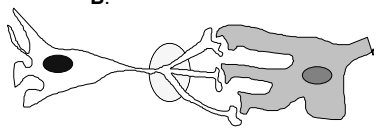
axon 1 makes synapse in a given cell can receive a synapse from axon 2

A.

axon 1

axon 2

B.

the presynaptic synapse can produce only a local potential change, which is then restricted to that axon terminal.

(from Crick and Asanuma, 1986)

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A biologically plausible ANN model proposal

- In view of these biological facts, it was decided to model through:
  - labels T and R:
    - the binding affinities between Ts and Rs;
  - label C:
    - the role of the "second messenger",
    - the effects of graded potential, and
    - the protein released by the coupling of T and R.

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The roles of the controller

- C can modify the binding affinities between neurons by modifying:
  - degrees of affinity of receptors,
  - amount of substrate (amount of transmitters and receptors),
  - gene expression, in case of mutation.

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Degrees of affinity modification

- Degrees of affinity are related to the way receptors gate ion channels at chemical synapses;
- Through ion channels transmitter material enters the postsynaptic cell:
  - in direct gating: receptors produce relatively fast synaptic actions,
  - in indirect gating: receptors produce slow synaptic actions:
    - these slower actions often serve to *modulate* behavior because they modify the degrees of affinity of receptors.

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Degrees of affinity modification

- In addition, modulation can be related to the action of peptides;
- Peptides: compound consisting of two or more amino acids, the building blocks of proteins;
- There are many distinct peptides, of several types and shapes, that can act as neurotransmitters.

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Degrees of affinity modification

- Peptides are different from many conventional transmitters:
  - "modulate" synaptic function instead of activating it,
  - spread slowly and persist for some time, much more than conventional transmitters, and
  - do not act where released, but at some distant site (in some cases).

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Degrees of affinity modification

- As transmitters, peptides:
  - act at very restricted places,
  - display a slow rate of conduction, and
  - do not sustain the high frequencies of impulses.

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Degrees of affinity modification

- As neuromodulators:
  - the excitatory effects of substance P (a peptide) $\rightarrow$ very slow in the beginning $\rightarrow$ longer in duration (more than one minute),
  - cannot cause enough depolarization to excite the cells;
  - the effect is to make neurons more readily excited by other excitatory inputs – "neuromodulation".

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Degrees of affinity modification

- In this model:
  - C explains this function by modifying the degrees of affinity of receptors.

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Amount of substrate modification

- In biological systems:
  - acetylcholine (a neurotransmitter):
    - spread over a short distance →
    - toward the postsynaptic membrane →
    - acting at receptor molecules in that membrane →
    - enzymatically divided →
    - part of it is taken up again for synthesis of a new transmitter →
    - an increase in the amount of substrate.

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Amount of substrate modification

- In this model:
  - C represents substrate increase by a variable acting over initial substrate amount.

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Gene expression modification

- Peptides are a second, slower, means of communication between neurons
  - more economical than using extra neurons;
- This second messenger:
  - besides altering the affinities between transmitters and receptors,
  - can regulate gene expression → synaptic transmission with long-lasting consequences.

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Gene expression modification

- In this model:
  - this is achieved by modification of a variable for gene expression:
    - mutation can be accounted for.

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The labels and their dynamic behaviors

A. For the network genesis:

– number of layers;

– number of neurons in each layer;

– initial amount of substrate (transmitters and receptors) in each layer; and

– genetics of each layer:

- type of transmitter and its degree of affinity,
- type of receptor and its degree of affinity, and
- genes (name and gene expression)).

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The labels and their dynamic behaviors

B. For the evaluation of controllers and how they act:

– Controllers can modify:

- the degree of affinity of receptors;
- the initial substrate storage; and
- the gene expression value (mutation).

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The labels and their dynamic behaviors

- Specifications lead to ANN with some distinctive characteristics:
  - each neuron has a genetic code:
    - a set of genes plus a gene expression controller;
  - controller can cause *mutation*:
    - because it can regulate gene expression.

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The labels and their dynamic behaviors

- substrate (amount of transmitter and receptor) defined by layer;
- substrate amounts limited:
  - some postsynaptic neurons not activated;
- network favors *clustering*.

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Part 2

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Conclusions

- Current models of ANN in debt with human brain physiology;
- Conventional ANN models are too simple:
  - because of mathematical simplicity,
  - lack several biological features of the cerebral cortex;
- Objective:
  - to present a biologically plausible ANN model, closer to human brain capacity.

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Conclusions

- In the model, the possibility of connections between neurons is related not only to:
  - synaptic weights,
  - activation threshold, and
  - activation function,
- but also to:
  - labels that embody the binding affinities between transmitters and receptors.

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Conclusions

- This type of ANN would be closer to human evolutionary capacity:
  - a genetically well-suited model of the brain;
- The hypothesis of the "dynamic core" (Edelman and Tononi, 2000) is also contemplated:
  - the model allows reentrancy in its architecture connections.

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Mercy beaucoup!

THANK YOU VERY MUCH FOR YOUR ATTENTION AND INTEREST

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