

# Modeling Biological Neural Networks

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## 1 INTRODUCTION

### 1.1 BACKGROUND CONTEXT

#### 1.1.1 ANATOMY OF A NEURON

BIOLOGICAL NEURAL NETWORKS are complex networks composed of nerve cells (neurons) in organisms (especially human brains and animal brains) and the connections between them. This network is the basis of the biological nervous system and is responsible for processing and transmitting information, allowing organisms to perceive the environment, make decisions, control movement and perform various complex behaviors.

Neurons receive signals from other neurons through synapses located – not exclusively – on their dendritic tree, which is a complex, branching, sprawling structure. If you are wondering what the king of dendritic complexity is, that would be the Purkinje cell, which may receive up to 100,000 other connections. Dendrites are studded with dendritic spines – little bumps where other neurons make contact with the dendrite.

Signals from the dendrites propagate to and converge at the soma – the cell's body where nucleus and other typical cell organelles live.

Coming off the soma is the axon hillock which turns into the axon. The axon meets other neurons at synapses. It allows a neuron to communicate rapidly over long distances without losing signal integrity. To allow signals to travel rapidly, the axon is myelinated – it is covered with interspersed insulators which allows the neuron's signal to jump between insulated sections. To allow the signal to maintain integrity, the neuron signal in the axon is 'all-or-nothing' – it is a rather bit-like impulse, which we will discuss next.

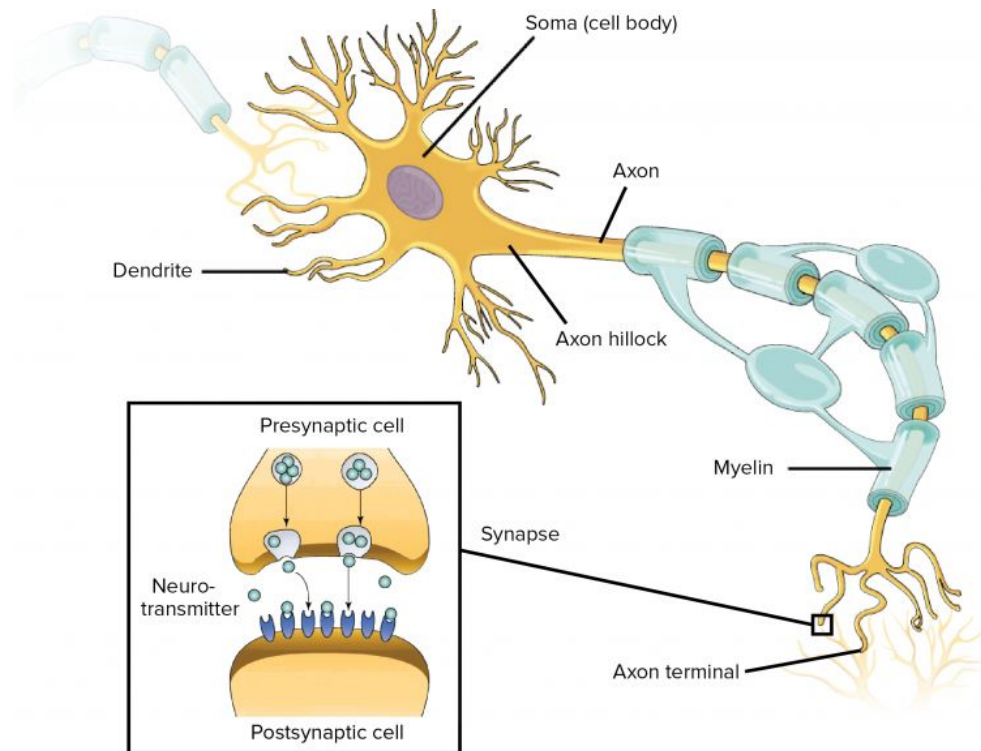


Figure 1.1: Anatomy of the neuron

Source: Source:“Neurons and glial cells” by OpenStax College, Biology CC BY-NC-SA 3.0 License.

### 1.1.2 PHYSIOLOGY OF A NEURON

THE second thing to appreciate about neurons is their specialized physiology — that is the cellular functions of neurons. The most striking feature of neural cellular function is the action potential. This is the mechanism which allows neurons to transmit information reliably over long distances without the transmission attenuating.

It is important to remember that neurons bathe in an extracellular solution of mostly water, salts and proteins. The forces caused by the movement of salts into and out of the cell and the different concentrations of these salts is the physical basis of the neuron’s remarkable behavior. There are sodium-potassium pumps which move sodium out of the cell and potassium in, so that the concentration of sodium outside the cell is higher than inside and the concentration of potassium outside the cell is lower than inside.

An action potential is a discrete event in which the membrane potential rapidly rises (depolarizes) and then falls (polarizes). This discrete event is all-or-nothing, meaning that if an action potential occurs at one part of the neuron’s membrane, it will also occur in the neighboring part, and so on until it reaches the axon terminal. Action potentials do not tend to travel backwards, because, once a section of the membrane has fired an action poten-

tial, electrochemical-forces hyper-polarize the region of membrane while the channels which were previously open close and become inactive for some duration of time.

The action potential is the result of different species of ions traveling across the cell membrane through channels and the activation and inactivation of those channels on different time scales. A stereotypical action potential occurs as follows:

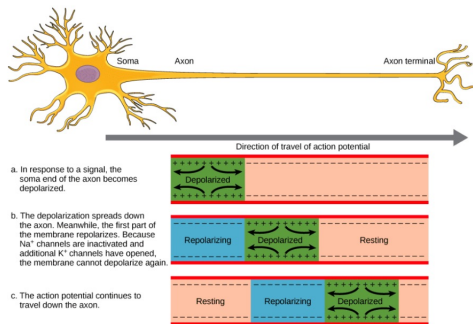


Figure 1.2: Propagation of nerve impluse

Source: [Opentext](#)

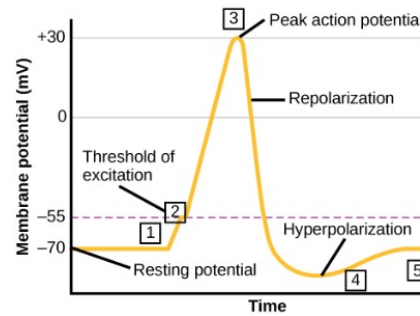


Figure 1.3: Neuronal action potential

Source: [Opentext](#)

- **Equilibrium:** The neuron's equilibrium membrane potential is near  $-70$  mV — roughly the Nernst Equilibrium of  $E_{K^+} \approx -75$ . At equilibrium, the net current is 0 — inward and outward currents are balanced.
- **Depolarization:** Incoming excitatory signals depolarize the membrane. Quick-to-respond voltage gated  $Na^+$  channels are activated, and  $Na^+$  rushes in, pushing the membrane potential higher. Slower-to-respond  $K^+$  channels open, and  $K^+$  rushes out, pushing the membrane potential lower.
- **Amplification:** If the neuron becomes more stimulated or is stimulated rapidly, many more  $Na^+$  channels are activated than  $K^+$  channels. This causes a feedback loop where the influx of  $Na^+$  causes more  $Na^+$  channels to activate.
- **Repolarization:** Eventually the membrane potential is near the Nernst Equilibrium of  $Na^+$  as the sodium channels are maximally open. The slower  $K^+$  channels catch up to  $Na^+$ , which repolarizes the membrane potential. Meanwhile, the  $Na^+$  channels become inactive.
- **Hyper-polarization:**  $K^+$  channels are open while  $Na^+$  channels are inactive, causing the membrane potential to dip below its typical equilibrium point, near the  $K^+$  Nernst equilibrium.
- **Refractory Period:** The  $Na^+$  channels, take a while to become deinactivated, meaning after an action potential, they remain incapable of opening again for a period of time. The period in which most  $Na^+$  channels are called the absolute refractory period (the

neuron cannot spike no matter the strength of the stimulus) while the period in which many  $Na^+$  channels are inactivated is called the relative refractory period (the neuron can spike given a sufficiently strong stimulus).

### 1.1.3 SPIKE TRAIN

Spike trains are the language of neurons. People tend to think of spikes as point-events and spike trains as point-processes. We describe these with the neural response function:

$$\rho(t) = \sum_{i=1}^k \delta(t - t_i) \quad (1.1)$$

where an impulse is defined as the dirac delta function (which is convenient for counting things):

$$\delta(t) = \begin{cases} 1 & \text{if } t = 0, \\ 0 & \text{otherwise} \end{cases} \quad (1.2)$$

Often, it is useful for analysis to assume spike trains are generated by random processes. Assuming spikes are independent of each other, we can model this point process as a Poisson process, in which we know the probability  $n$  spikes occur in the interval  $\Delta T$ :

$$P\{n \text{ spikes occur in } \Delta t\} = \frac{(r \Delta t)^n}{n!} \exp(-r \Delta t) \quad (1.3)$$

To generate spikes according to a Poisson point process, generate a random number  $r$  in a sufficiently small time interval, such that only 1 spike should occur, and check whether  $r < \text{firingRate} \Delta T$ . However, make sure that  $\text{firingRate} \Delta T < 1$ .

## 1.2 SIGNIFICANCE OF MODELING

Modeling serves as a pivotal tool in neuroscience, providing insight into the functionality of the brain and the intricate mechanisms of neural processes. It offers a window into the otherwise inaccessible workings of neural communications.

## 1.3 HISTORICAL OVERVIEW

### 1.3.1 STAGE 1 – THE M-P MODEL

Since the Spanish anatomist Cajal founded the neuron theory at the end of the 19th century, the biological characteristics and related electrical properties of neurons have been discovered one after another. In 1943, the M-P model of neurons (shown in Figure 1.4) was first proposed in the paper "Logical Activities of Thoughts Contained in Neural Activities" [1]. The model was created by psychologists W.S. McCulloch and W.S. McCulloch from the United States. Another mathematician, W.A. Pitts.

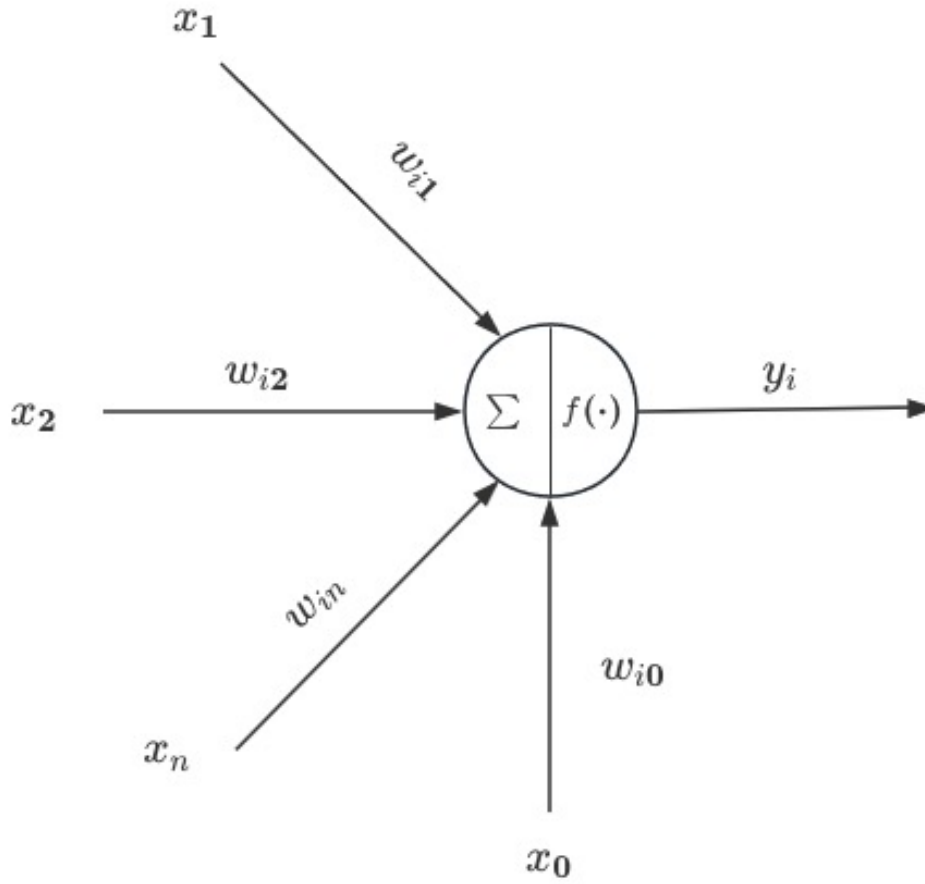


Figure 1.4: Schematic diagram of the M-P model of neurons

Source: Draw by Kai

In the figure,  $x_i$ , 1, 2, ..., represents the input signal transmitted from other neurons connected to the current neuron,  $w_{ij}$  represents the connection strength or weight from neuron  $j$  to neuron  $i$ ,  $\theta_i$  is the neuron The activation threshold or bias of the element,  $f$  is called the activation function or transfer function. The output of the neuron  $y_i$  can be expressed as follows:

$$y_i = f\left(\sum_{j=1}^n w_{ij} x_j - \theta_i\right) \quad (1.4)$$

This model describes neurons from the perspective of logical functional devices, opening up a path for theoretical research on neural networks. The M-P model is a mathematical simplification of the biological neuron information processing model, and subsequent neural network research work is based on it.

### 1.3.2 STAGE 2 – HEBB LEARNING RULES

In 1949, in the book "The Organization of Behavior", the psychologist Donald O. Hebb analyzed the change rules of the connection strength between neurons, and based on this, he proposed the famous Hebb learning rule [2]. Inspired by Pavlov's conditioned reflex experiments, Hebb believed that if two neurons are fired at the same moment, the connection between them should be strengthened. The Hebb learning rule defined based on this is as follows:

$$w_{ij}(t+1) = w_{ij}(t) + \alpha y_j(t) y_i \quad (1.5)$$

Among them,  $w_{ij}(t+1)$  and  $w_{ij}(t)$  represent the connection strength between neuron  $j$  and neuron  $i$  at time  $t$  and  $t+1$  respectively, while  $y_i$  and  $y_j$  are the outputs of neurons  $i$  and  $j$ . Hebb's rule belongs to the category of unsupervised learning algorithms. Its main idea is to adjust the connection relationship between two neurons according to their excitation state, so as to realize the simulation of simple neural activities. Following the Hebb learning rule, the supervised delta learning rule of neurons was proposed to solve the learning problem of neuron weights when the input and output are known. This algorithm continuously adjusts the connection weights to make the actual output of the neuron consistent with the expected output. Its learning correction formula is as follows

$$w_{ij}(t+1) = w_{ij}(t) + \alpha(d_i - y_i)x_j(t) \quad (1.6)$$

Among them,  $\alpha$  is the learning rate of the algorithm,  $d_i$  and  $y_i$  are the expected output and actual output of neuron  $i$ ,  $x_j(t)$  represents the state (activation or inhibition) of neuron  $j$  at time  $t$ .

Intuitively speaking, when the actual output of neuron  $i$  is greater than the expected output, the connection weight with the activated neuron is reduced, and the connection weight with the inhibited neuron is increased; when the actual output of neuron  $i$  is smaller than the expected output, the connection weight with activated neurons will be increased, while the connection weight with inhibited neurons will be reduced. Through such an adjustment process, neurons will store the correct mapping relationship between input and output in the weight, thus possessing the ability to represent data. The Hebb learning rule and the Delta learning rule are both proposed for a single neuron. The learning rules for parameters in a network composed of neurons will be discussed later. The research work done by the above pioneers paved the way for the emergence of neural computing and inspired many scholars to continue exploring and researching this field.

### 1.4 CHALLENGES AND LIMITATIONS

Accurately modeling biological neural networks presents numerous challenges. These include the complexity of neuronal dynamics, the nonlinear nature of neural responses, and the vast interconnectivity within the neural network.

### 1.5 RECENT ADVANCES

Recent advancements in computational power and mathematical methodologies have allowed for the creation of more sophisticated and detailed models of neural networks, pushing the boundaries of what was previously possible.

### 1.6 APPLICATIONS

The application of neural network models is vast, ranging from the exploration of neurological disorders, such as epilepsy and Alzheimer's disease, to the investigation of cognitive processes.

### 1.7 OBJECTIVES OF YOUR WORK

The objectives of the presented work are to ... (here, you would specify the goals of your own research or modeling approach).

## REFERENCES

- [1] Warren S Mcculloch and Walter Pitts. A LOGICAL CALCULUS OF THE IDEAS IMMANENT IN NERVOUS ACTIVITY. September 1854.
- [2] D. O. Hebb. *The Organization of Behavior: A Neuropsychological Theory*. L. Erlbaum Associates, Mahwah, N.J, 2002.