Project

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1 Introduction

2 Neural networks

Neural networks are comprised of nodes called neurons and connections between the neurons called synapses. Before we can discuss neural networks, we need to know more about the basic building blocks, the neuron and the synapse.

The arcitecture of the neuron is important to understand the directionality of of the graph. The mechanisms of the synapse is important later when we will discuss stability of learning. Finally, important aspects of the network that cannot be un derstood from a single unit, will be described.

2.1 The neuron

In mathematical terms from graph theory, a biological version of a neural network can be called a directed syclic graph. In terms from neuroscience this meens that the network og neurons is recurrent (with feedback) and that the synapses (the connections between neurons) are directional (information flows in one direction). More on this later.

The neuron is surrounded by the cell membrane. This membrane has low permeability to ions from the fluid surrounding the neuron to the intracellular fluid of the neuron. In addition we have different ionic pumps that pumps different ions from one side of the membrane to the other. This creates an ionic difference over the membrane, also called an electric potential. The membrane potential typically is around $-70mV$ at rest.

When the neuron is exited (gets a exitatory transmission through one of its synapses), the neuron is said to become *depolarized*.

When a synaptic transmission arrives at a synapse, neurotransmitters are released from the axon terminal of the presynaptic neuron into the synaptic cleft. On the postsynapsic membrane in the synapse are different receptors for different neurotransmitters[2]. Describing the different known kinds of receptors will be outside the scope of this report. We will limit the description to one class of receptors, called "ligand–gated receptors".

Ligand–gated receptors are direcly connected to ion channels in the membrane. Opening of these ion specific channels enables some ions to flow throught (depending on what kind of channel the receptor is connected to). Depending on which ions are let through, the neuron is either depolarized (towards zero polarization, less than its resting potential) of hyberpolarized (getting a more negative membrane potential) by the transmission.

Then the polarization (also called the value of the neuron) becomes more positive than the firing threshold of the neuron, an action potential is initiated at the axon hillock near the cell body of the cell. This is often referred to as "firing an action potential".

Since depolarizing the neuron causes firing, the neurons potential is often referred to as the neurons depolarization in the litterature.

2.2 The axon

In order to give (approximately) the same output to different output sites (axon terminals) along the axon, the action potential is a self carrying–signal based on different voltage gated channels along the axon membrane. Since the ion channels are voltage gated, and cause larger depolarization when open, the membrane potential diverges, and the channels are open until some timing mechanism causes them to close. The exact mechanisms are not important in this context, but the boolean carracteristics of the action potential is.

To summarize the layout of the neuron related to information processing, on one side of the neuron we have the output. Boolean output signal from the neuron is mediated through the axon of the neuron that splits into collaterals along the axon with axon terminals at the end. The axon terminal has mechanisms for releasing neurotransmittors intro the synaptic cleft(the area between the presynaptic membrane and the membrane of the postsynaptic cell). In the membrane on the oposite side of the synaptic cleft are neurotransmittor-receptors that alters the postsynaptic neurons depolarization.

Most, but not all incoming synapses are located in the dendrite, a specialized part of the neuron for recieving information. At the postsynaptic membrane are receptors. Most of these receptors are ligand-gated receptors, either exitatory (causes more depolarization of the neuron) or inhibitory (hyperpolarizes the neuron). The combination of neurotransmitters released from the presynaptic terminal and postsynaptic receptors in one synapse typically makes the synapse either exitatory or inhibitory.

2.3 The synapse

When the action potential reaches the axon terminal, the size of the signal is the same as when it first was generated at the axon hillock. This enshures that the distance the action potential has to travel does not affect the transmission at the synapses of the different axon terminals. [**?**]

This does not mean that the transmission for different synapses is the same. At each synapse the connection to the postsynaptic neuron is different. There are many mechanisms behind this, but I will focus on the mechanisms within the neuron:

- Presynaptically, different amount of neurotransmittors are released from the axon terminal following an action potential.
- Postsynaptically the amount of receptors varies between different synapses. The amount also varies with time.

When the action potential reaches the axon terminal, it will open voltage–gated Ca^{2+} channels in the active zone of the terminal, and Ca^{2+} enters the

cytosol of the axon terminal of the presynaptic neuron[3].

2.3.1 Presynaptic mechanisms behind synaptic plasticity

Ca^{2+} causes release of neurotransmittors from the presynaptic axon terminal into the synaptic cleft[3]. Long–term potentiation (LTP) causes a lasting change of the tranmission through the synapse.

The amount of Ca^{2+} inflow, and thus the amount of neurotransmitter release can be modulated by socalled axoaxonic synapses[1], synapses that is connected directly to the presynaptic axon terminal. A transmission here will cause a small increase in the axon terminals amount of Ca^{2+} and "prime" the synapse for a transmission. Multiple incoming action potentials in fast succession will have the same effect on the following action potentials and causes what is called *potentiation*[4].

2.3.2 Postsynaptic mechanisms behind synaptic plasticity

Glutamate is the main exitatory neurotransmittor in the CNS[7]. There are two main groups of ligand–gated glutamate receptors, the N-methyl D-aspartate (NMDA) receptors and the non-NMDA receptors. The non-NMDA receptors mainly consists of the α-amino-3-hydroxy-5-methyl-4-isoxazolepropionic acid (AMPA) receptor.

Most non-NMDA receptors are permeable to ions that changes the postsynaptic potensial without having lasting changes on the synaptic strength.The NMDA receptor is permeable to Ca^{2+}, which is important for lasting changes of the synaptic strength.

An other important difference between the NMDA-R and the AMPA-R is that NMDA receptors have an additional condition for opening of its ion channel. In the NMDA receptor there is a Mg^{2+} ion blocking the channel. When the potential across the membrane is sufficiently depolarized, the Mg^{2+} will float more freely and the block is removed from the NMDA receptor. Ca^{2+} diffuses into the cell following an action potential[5].

Also on the postsynaptic part of the synapse ca^{2+} has an important role in synaptic plasticity. Ca^{2+} activates production of more non-NMDA receptors for the postsynaptic membrane, resulting in LTP[**?**].

The NMDA-related synaptic plasticity is the background for what is called "Spike Timing Dependent Plasticity" that will be important in the rest of this text.

References

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