

# Why nerve cells work faster than the theory allows

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With accuracy unknown until now, researchers from the Max Planck Institute for Dynamics and Self-Organization and the Bernstein Center for Computational Neuroscience in Göttingen together with the neurophysiologist Maxim Volgushev from the Ruhr-Universität Bochum have analyzed, by which rules, the nerve cells in the cerebral cortex decide to send out impulses. They surprisingly found, that the high flexibility and speed with which these cells work cannot be explained using the present, central model of neurophysiology, the Hodgkin-Huxley model.

Their findings suggest that the sodium channels, which open in the cell membranes during a nerve impulse, do not work independently of each other, as assumed so far, but support each other during the opening process. This new type of mechanism appears to help the cells transmit fast changing signals and suppress slow signals.

Every living cell maintains a voltage difference across its cell membrane. Nerve cells distinguish themselves from other cells in that they use this voltage difference to process and transmit messages. When a nerve cell receives an impulse, the voltage across the cell membrane is reversed. This "action potential" spreads out through the long appendages of the cell with high speed. At the end of the appendages it is transmitted to other cells. In 1952, Alan Lloyd Hodgkin and Andrew Fielding Huxley described in a mathematical model how such an action potential originates on the basis of measurements on neurons of the squid. The Hodgkin-Huxley model, for which the scientists later received the Nobel

Prize, has since then served to explain the signal processes in all neurons.

According to the Hodgkin-Huxley model, an action potential is initiated when the voltage across the membrane of the nerve cell reaches to a certain threshold value. Voltage gated sodium channels react to this voltage change by opening up and triggering an avalanche-like reaction. Positively charged sodium ions flow through the open channels into the cell, which leads to a further increase of the membrane potential and the opening of additional sodium channels. The threshold and the speed with which the action potential originates vary from cell to cell – for any individual cell however, these parameters are specified for the most part by the characteristics of its sodium channels.

An interdisciplinary team of physicists and neurophysiologists from the Max Planck Institute for Dynamics and Self-Organization in Göttingen and the Ruhr-Universität Bochum has now examined more closely the speed and threshold of action potentials in nerve cells of the cerebral cortex of the mammal brain. They were able to show that action potentials are initiated extremely rapid here. Although a single action potential lasts a millisecond, a stronger influx of sodium already sets in during the first 200 microseconds. The sodium channels appear to open almost simultaneously, so that sodium ions can flow into the cells very quickly and in large amounts. At the same time, however, the researchers found in their measurements that the threshold values at which the action potentials were initiated were very variable.

In order to understand what causes this unusual behavior, the scientists tried to recreate the behavior of the cells in computer simulations of Hodgkin-Huxley-type models. To their surprise, it turned out that a high variability of the threshold value and a rapid onset of the action potential cannot be unified in this model. Both characteristics behave like both sides of a seesaw. To obtain a high variability of the threshold value, the model requires a low speed of initiation of the action potential. A rapid

onset is only obtained, when the variability of the threshold value is low.

In order to recreate the observed behavior of the nerve cells in computer simulations, Wolf and his colleagues postulated a new mechanism, which explains how the sodium channels not always open at the same threshold value, but nevertheless open almost simultaneously. When a sodium channel opens, it influences, according to the new model, other sodium channels in the immediate neighborhood – the channels open "cooperatively" and not – as according to Hodgkin-Huxley – independently of each other and only dependent on the voltage across the membrane. To test this hypothesis, the scientists used a trick: If it would be possible to measurably stop the cooperative mechanism, then that would be a good argument for its existence. They achieved this by blocking a part of the sodium channels with the nerve poison tetrodotoxin, so that the channels that still functioned lay so scattered in the membrane, that they were not able to cooperate.

Furthermore, the researchers were able to show that the cells probably used this novel mechanism to differentiate between the received signals and only answer to certain ones. Bjoern Naundorf summarizes these results, „The cells function like a high-pass filter; fast signals are transmitted well, slow signals are suppressed". Both aspects of the initiation of the action potential play different roles. The large variability of the threshold potentials allows the cells to ignore slowly varying stimuli. The cells continuously increase their threshold so that in many cases no impulse is initiated at all. The fast activation of action potentials, on the other hand, helps the cells to transmit fast changing signals, even with high precision. According to the Hodgkin-Huxley model, the cells would lack the ability to do this.

"Many scientists – including us – saw the Hodgkin-Huxley model up to now no longer as a hypothesis, but believed that it was principally applicable to all neurons", says Fred Wolf, who led the study at the Max

Planck Institute for Dynamics and Self-Organization in Göttingen. He and his colleagues have now shown that this is not so. The better cognitive ability of higher animals, such as cats or humans compared to squid or snails, is not only attributed to the higher number of neurons in the brains of these animals, but also to the manner in which the neurons process signals. To do so, these higher animals presumably use molecular mechanisms which the lower animals do not possess.

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