

How the brain stays receptive: Channel protein Pannexin1 is critical for memory and orientation

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The channel protein Pannexin1 keeps nerve cells flexible and thus the brain receptive for new knowledge. Together with colleagues from Canada and the U.S., researchers at the Ruhr-Universität Bochum led by the junior professor Dr. Nora Prochnow from the Department of Molecular Brain Research describe these results in *PLoS ONE*. In the study, mice comprising no Pannexin1 in memory-related brain structures displayed symptoms similar to autism. Their nerve cells lacked synaptic plasticity, i.e. the ability to form new synaptic contacts or give up old contacts based on the level of usage.

Pannexins traverse the cell membrane of [vertebrate animals](#) and form large pored channels. They are permeable for certain signalling molecules, such as the energy storage molecule ATP (adenosine triphosphate). The best known representative is Pannexin1, which occurs in abundance in the brain and spinal cord and among others in the hippocampus - a [brain structure](#) that is critical for long-term memory. Malfunctions of the pannexins play a role in the development of epilepsy and strokes.

The research team studied mice in which the gene for Pannexin1 was lacking. Using cell recordings carried out on isolated brain sections, they analysed the long-term potentiation in the hippocampus. Long-term potentiation usually occurs when new memory content is built - the contacts between [nerve cells](#) are strengthened; they communicate more

effectively with each other. In mice without Pannexin1, the long-term potentiation occurred earlier and was more prolonged than in mice with Pannexin1. "It looks at first glance like a gain in long-term memory", says Nora Prochnow. "But [precise analysis](#) shows that there was no more scope for upward development." Due to the lack of Pannexin1, the cell communication in general was increased to such an extent that a further increase through the learning of new knowledge was no longer possible. The [synaptic plasticity](#) was thus extremely restricted. "The plasticity is essential for learning processes in the brain", Nora Prochnow explains. "It helps you to organise, keep or even to forget contents in a positive sense, to gain room for new inputs."

The absence of Pannexin1 also had an impact on behaviour: when solving simple problems, the animals were quickly overwhelmed in terms of content. Their spatial orientation was limited, their attention impaired and an increased probability for seizure generation occurred. "The behavioural patterns are reminiscent of autism. We should therefore consider the Pannexin1 channel more closely with regard to the treatment of such diseases", says the neurobiologist from Bochum.

According to the scientists' theory, nerve cells lack a feedback mechanism without Pannexin1. Normally the [channel protein](#) releases ATP, which binds to specific receptors and thus reduces the release of the neurotransmitter glutamate. Without Pannexin1 more glutamate is released, which leads to increased long-term potentiation. This causes the cell to lose its dynamic equilibrium, which is needed for an efficient learning process.

More information: N. Prochnow, A. Abdulazim, S. Kurtenbach, V. Wildförster, G. Dvorianchikova, J. Hanske, E. Petrasch-Parwez, V.I. Shestopalov, R. Dermietzel, D. Manahan-Vaughan, G. Zoidl (2012): Pannexin1 stabilizes synaptic plasticity and is needed for learning, *PLoS ONE*, [DOI: 10.1371/journal.pone.0051767](https://doi.org/10.1371/journal.pone.0051767)

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