Neurons are only a portion of the brain cells. Depending on the area of the brain, they constitute from 10 to 50% of all cells. The rest are glial cells, especially astrocytes. In the gray mater, which is responsible for processes such as memory, thinking, and part of neurological diseases, the ratio of neurons to astrocytes is about 1: 1. The existence of glial cells, including astrocytes, have long been known, but the only functions attributed to these cells were the bonding of the brain tissue and participating in the nutrition of neurons. In contrast, recent research has shown that astrocytes perform active roles in functioning of synaptic connections, which are considered as the basic cellular units responsible for cognitive processes. It was found that astrocytes can modulate the structural remodeling of synapses, including even their elimination, particularly in pathological conditions. Epilepsy, which affects approx. 1% of the population, is an example of a disease process in which, as a result of repeated seizures, there is a loss of synapses. It has been shown that astrocytes are involved in the elimination of synaptic connections in the aftermath of seizures through their phagocytosis, i.e. absorption of synapses into their own cytoplasm for destruction. As part of our project we would like to understand the molecular mechanisms of these processes, in order to prevent the destruction of synapses by astrocytes following the seizures in people suffering from epilepsy or other neuropsychiatric diseases. Based on publications on organs other than the brain, it seems that the key role in the destruction of synapses by astrocytes can be mediated by a membrane protein called CD44. To verify this hypothesis we precisely designed our proposed research approach, i.e., a series of experiments using the cutting-edge research methods that could verify whether CD44 protein plays a supposed role in synapse elimination. If it turns out that, after all, this is not CD44, the grant funds will enable us to find other protein(s) on the surface of astrocyte that could be engaged in activities detrimental to the synapses.