Activity-dependent synaptic plasticity is a characteristic of the nervous system that permits neurons to communicate and alter their connections in response to new experience. Rebuilding synaptic connections and changing their strength gives rise to long-lasting memories and provides the biological basis for mental functions. In contrast, impairments underline numerous neurological diseases, including depression, schizophrenia, and addiction. It should be emphasized that for a long time, most of the research was focused on the functional and structural aspects of the plasticity of glutamatergic (excitatory) transmission, creating the impression that inhibitory synapses are not plastic in the background. Numerous studies conducted in recent years have expanded our understanding of this topic and demonstrated that GABAergic synapses are plastic. The study concerning the mechanism of plastic changes is now a leading direction in neurobiology.

Although interneurons comprise only about 10—15% of all cells in the hippocampus, due to their great diversity, they play a key role in synchronizing the activity of the neural network. According to the latest research, at least 23 distinct inhibitory cell types have been identified in the CA1 area based on morphological features, firing patterns properties, or the expression of various molecular factors. Interneurons containing somatostatin (SST) and parvalbumin (PV) constitute two large groups that play an essential role in learning and memory consolidation. It has been observed that SST and PV cells innervate and regulate the activity of place cells, which encode and store spatial information. Additionally, it has been demonstrated that interneurons expressing vasoactive intestinal polypeptide (VIP) participate in the disinhibition of pyramidal cells by suppressing GABAergic interneurons. These findings confirm that the inhibitory network plays a key role in learning and memory. Previous research conducted under the supervision of Professor J. Mozrzymas indicated that matrix metalloproteinases (MMPs) and integrins have a crucial role in the induction and maintenance of inhibitory synaptic plasticity located on pyramidal cells in the hippocampus. However, the exact cellular and molecular process by which GABAergic plasticity is induced on particular interneurons is still largely unclear. The aim of this planned research in the frame of the "SONATINA 7" project is to increase the understanding of the plasticity of inhibitory transmission in PV and SST cells and to clarify the roles of several molecular factors, including adhesion proteins, MMPs, and dopamine receptors, in its molecular mechanism. The research will be carried out using an electrophysiological technique that allows recording a signal from a specific type of interneuron in brain slices. To induce plasticity, both chemical and electrical protocols will be used. In addition, the research will be extended to include GABAergic plasticity depending on the specific input of interneuron being studied. For this purpose, the optogenetics technique will be used, which controls the activity of selected interneurons containing photosensitive proteins-opsins.

It should be emphasized that for the proper functioning of the nervous system, it is vital to maintain a balance between inhibitory and excitatory transmission and that its imbalance is associated with many nervous system diseases. Therefore, research into elucidating the structural and functional GABAergic plasticity induced in interneurons is essential to better understand the mechanisms underlying normal development and behavior and identify the etiology of various psychiatric and neurological disorders to develop new therapies. As a result of the experiments conducted, our knowledge of the plasticity of inhibitory synapses and the functioning of the hippocampus network will be broadened.