Synaptic plasticity is understood as a property, that functional and morphological features of the synapse change under the influence of neuronal activity. It is now believed that synaptic plasticity is an important substrate for memory processes, hence the great interest of researchers in this phenomenon. For more than 40 years, the plasticity of excitatory (glutamatergic) synapses has been extensively studied. Many forms of this plasticity have been revealed and some of its molecular mechanisms have been discovered. Much less is known about the plasticity of the inhibitory (GABAergic) transmission. GABAergic neurons are much less numerous in the brain than glutamatergic ones and they show a large diversity and for these reasons it is much more difficult to study their plastic properties than in the case of the excitatory neurons. Nevertheless, it is now known that inhibitory synapses are also plastic, but the mechanisms of this plasticity only start to be revealed. The aim of this project is to deepen our knowledge about the molecular mechanisms of plastic changes occurring in inhibitory synapses. Our research group investigates the plasticity model in which GABAergic synapses are strengthened (phenomenon referred to as iLTP), which is induced by a brief administration of a glutamate receptor agonist (NMDA). This is a heterosynaptic plasticity, which is initiated by the activation of NMDA type glutamate receptors (permeating calcium) followed by the mechanisms leading to plastic changes at the inhibitory synapses. Our research will concern three tasks, which were formulated on the basis of preliminary, unpublished experimental results. First of all, we noticed that the induction of iLTP is accompanied by an increase in the duration of synaptic currents in the inhibitory synapses. This is an important observation because the change in the time duration of the synaptic inhibitory signal can significantly affect the functioning of the entire neuronal network. We will check whether this effect is related to the accumulation in the synapse of $GABA_A$ receptors of $\alpha5\beta3\gamma2$ type, which show slow kinetics. Thanks to cooperation with prof. A. Barberis from IIT in Genoa, we will be able to verify this hypothesis using a technique that allows us to study the membrane mobility of a specific type of $GABA_A$ receptors. Secondly, we will examine the plasticity mechanisms of GABAergic synapses formed on GABAergic neurons. So far, this problem has been studied only on the so-called principal (pyramidal) neurons that are excitatory. Recently, we discovered that the RGD peptide (integrin ligand) induces the enhancement of GABAergic signals on inhibitory interneurons while in the case of inhibitory synapses on the principal cells, it leads to synaptic depression. In the frame of this project, we will try to describe the integrin-dependent plasticity mechanisms. The third research task will be to describe the modulation of the GABAergic plasticity by dopamine. Our preliminary results show that in the case of pyramidal cells, dopamine stably strengthens the signals mediated by inhibitory synapses, suggesting a modulation of the plasticity phenomenon of GABAergic synapses. We intend to investigate how the pharmacological activation or blocking of the activity of specific dopamine receptors affects the plasticity of inhibitory synapses on both inhibitory interneurons and on glutamatergic principal cells. This project concerns the fundamental problem of synaptic plasticity, which, as already underlined, is an important substrate for learning and memory processes. More and more premises indicate a prominent role of GABAergic transmission in creating the so-called memory engrams. However, the mechanisms whereby this type of plasticity contributes to its creation are just starting to be revealed and this project is meant to make a contribution in this field. In particular, the characterization of GABAergic plasticity mechanisms on various types of inhibitory interneurons seems to be necessary to understand its role in the functioning of neuronal networks. The study of the impact of dopamine on GABAergic plasticity is part of a wide and very promising area of research on the neuromodulation of the synaptic plasticity phenomenon. It should be mentioned that inhibitory transmission dysfunction is one of the basic pathomechanisms of many diseases including epilepsy, autism and schizophrenia. In this context, our contribution to understanding the mechanisms of the plasticity of inhibitory synapses may be important in the context of clinical investigations.