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Sommario/riassunto	Learning and memory are believed to depend on plastic changes of neuronal circuits due to activity-dependent potentiation or depression of specific synapses. During the last two decades, plasticity of brain circuits was hypothesized to mainly rely on the flexibility of glutamatergic excitatory synapses, whereas inhibitory synapses were assumed relatively invariant, to ensure stable and reliable control of the neuronal network. As a consequence, while considerable efforts were made to clarify the main mechanisms underlying plasticity at excitatory synapses, the study of the cellular/molecular mechanisms of inhibitory plasticity has received much less attention. Nevertheless, an increasing body of evidence has revealed that inhibitory synapses undergo several types of plasticity at both pre- and postsynaptic levels. Given the crucial role of inhibitory interneurons in shaping network activities, such as generation of oscillations, selection of cell assemblies and signal integration, modifications of the inhibitory synaptic strength

represents an extraordinary source of versatility for the fine control of brain states. This versatility also results from the rich diversity of GABAergic neurons in several brain areas, the specific role played by each inhibitory neuron subtype within a given circuit, and the heterogeneity of the properties and modulation of GABAergic synapses formed by specific interneuron classes. The molecular mechanisms underlying the potentiation or depression of inhibitory synapses are now beginning to be unraveled. At the presynaptic level, retrograde synaptic signaling was demonstrated to modulate GABA release, whereas postsynaptic forms of plasticity involve changes in the number/gating properties of GABAA receptors and/or shifts of chloride gradients. In addition, recent research indicates that GABAergic tonic inhibition can also be plastic, adding a further level of complexity to the control of the excitatory/inhibitory balance in the brain. The present Topic will focus on plasticity of GABAergic synapses, with special emphasis on the molecular mechanisms of plasticity induction and/or expression.
