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***Plasticity in Single Axon Glutamatergic Connection to GABAergic Interneurons Regulates Complex Events in the Human Neocortex***

In the human neocortex, single excitatory pyramidal cells can elicit very large glutamatergic EPSPs (VLEs) in inhibitory GABAergic interneurons capable of triggering their firing with short (3-5 ms) delay. Similar strong excitatory connections between two individual neurons have not been found in nonhuman cortices, suggesting that these synapses are specific to human interneurons. The VLEs are crucial for generating neocortical complex events, observed as single pyramidal cell spike-evoked discharge of cell assemblies in the frontal and temporal cortices. However, long-term plasticity of the VLE connections and how the plasticity modulates neocortical complex events has not been studied. Using triple and dual whole-cell recordings from synaptically connected human neocortical layers 2-3 neurons, we show that VLEs in fast-spiking GABAergic interneurons exhibit robust activity-induced long-term depression (LTD). The LTD by single pyramidal cell 40 Hz spike bursts is specific to connections with VLEs, requires group I metabotropic glutamate receptors, and has a presynaptic mechanism. The LTD of VLE connections alters suprathreshold activation of interneurons in the complex events suppressing the discharge of fast-spiking GABAergic cells. The VLEs triggering the complex events may contribute to cognitive processes in the human neocortex, and their long-term plasticity can alter the discharging cortical cell assemblies by learning.