Recent studies showed a crucial importance of communication between two major types of brain cells: neurons transmitting electrical signals and glial cells which maintain the wellbeing and function of neurons. Still, the study of age-related changes in the neuron-glia signaling is far from complete. We have shown previously that cortical astrocytes are capable to release ATP and glutamate by vesicular SNARE complex-dependent mechanism. Furthermore, we showed that synergistic action of astrocyte-derived ATP and glutamate is important for the synaptic plasticity in the neocortex. Induction of long-term potentiation by strong stimulation was impaired in the dn-SNARE mice but was rescued by application of exogenous non-hydrolysable ATP analogs. Secondly, weak sub-threshold stimulation became able to induce LTP when astrocytes were additionally activated. Facilitatory effect of astrocyte activation was abolished in the dn-SNARE mice suggesting the involvement of exocytosis of ATP and glutamate. Our recent experiments, carried out in both wild-type and dnSNARE transgenic mice, have shown that age-related decline in the astroglial Ca$^{2+}$ signalling can cause substantial decrease in the exocytosis of gliotransmitters, in particular ATP, glutamate and D-Serine. This in turn can cause the decrease in the extent of astroglial modulation of synaptic transmission in the neocortex and therefore can contribute to the age-related impairment of synaptic plasticity and cognitive decline. Combined, our results strongly support the physiological relevance of glial exocytosis for glia neuron-communications and brain function.