1-LBS-06 Late-Breaking Session

Oxytocin recovers Aβ-induced impairment of hippocampal synaptic plasticity in mice

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Oxytocin (OXT) is a peptide hormone synthesized in the hypothalamic paraventricular nucleus. OXT has been reported to be involved in regulation of learning and memory performance. However, there is no report that shows the effect of OXT on the amyloid-beta (Aβ)-induced impairment of synaptic plasticity. Here, we examined whether OXT have effects on the Ab-induced impairment of synaptic plasticity in mice.

Methods: Male ddY mice were used. To investigate the effect of OXT on synaptic plasticity, we prepared acute hippocampal slice for extracellular recording, and assessed long-term potentiation (LTP) with A$\beta_{25\text{-}35}$ perfusion in the absence and presence of OXT.

Results: In the present study, we found that OXT recovered the LTP impaired by perfusion of A$\beta_{25\text{-}35}$ in the mouse hippocampus. These effects were blocked by the pretreatment with a selective OXT receptor antagonist L-368,899. Further, the pretreatment with an ERK inhibitor U0126 and a selective Ca^{2+}-permeable AMPA receptor antagonist NASPM were completely antagonized the effects of OXT, respectively.

Conclusion: These results suggested that OXT recovered Aβ–induced impairment of hippocampal synaptic plasticity through the OXT receptors in the mice. We proposed that ERK phosphorylation and Ca^{2+}-permeable AMPA receptors are involved in these effects of OXT.