生物物理学 II　Handout No. 7

①

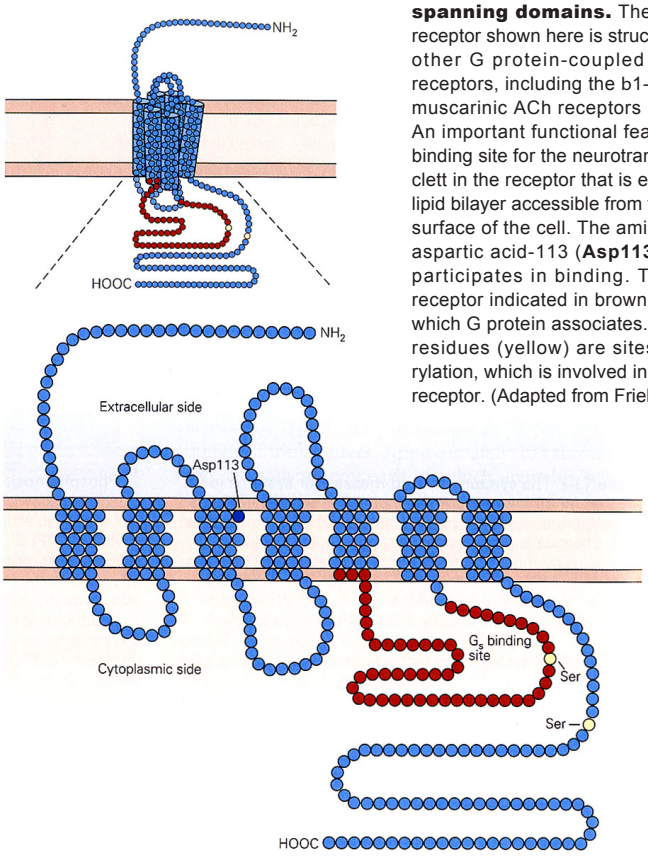

Figure 13-3　A G protein-coupled receptor contains seven membrane-spanning domains. The b2-adrenergic receptor shown here is structurally similar to other G protein-coupled metabotropic receptors, including the b1-adrenergic and muscarinic ACh receptors and rhodopsin. An important functional feature is that the binding site for the neurotransmitter lies in a clett in the receptor that is embedded in the lipid bilayer accessible from the extracellular surface of the cell. The amino acid residue aspartic acid-113 (**Asp113**, in dark blue) participates in binding. The part of the receptor indicated in brown is the part with which G protein associates. The two serine residues (yellow) are sites for phospho-rylation, which is involved in inactivating the receptor. (Adapted from Frielle et al. 1989.)

②

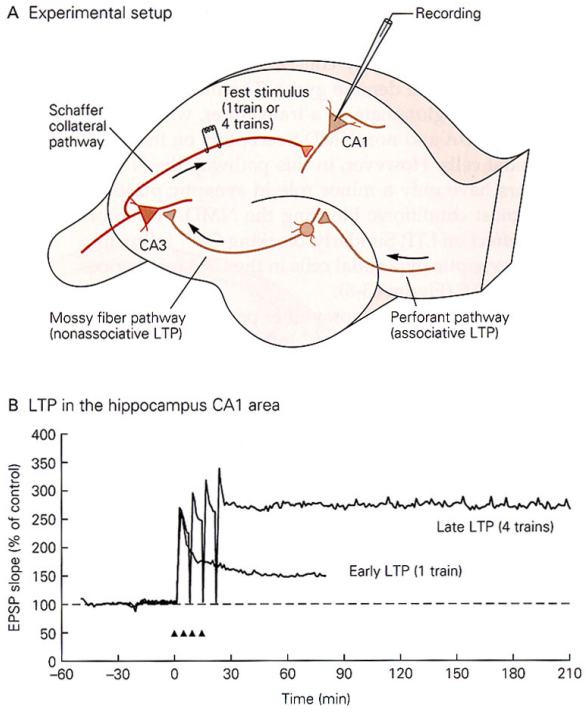

Figure 63-9　Long-term potentiation (LTP) in the Schaffer collateral pathway to the CA7 region of the hippocampus.
A. Experimental setup for studying LTP in the CA7 region of the hippocampus. The Schaffer collateral pathway is stimulated electrically and the response of the population of pyramidal neurons is recorded.
B. Comparison of early and late LTP in a cell in the CA1 region of the hippocampus. The graph is a plot of the slope (rate of rise) of the excitatory postsynaptic potentials (EPSP) in the cell as a function of time. The slope is a measure of synaptic efficacy. Excitatory postsynaptic potentials were recorded from outside the cell. A test stimulus was given every 60 s to the Schaffer collaterals. To elicit early LTP a single train of stimuli is given for 1 s at 100 Hz. To elicit the late phase of LTP four trains are given separated by 10 min. The resulting early LTP lasts 2-3 hours, whereas the late LTP lasts 24 or more hours.

③

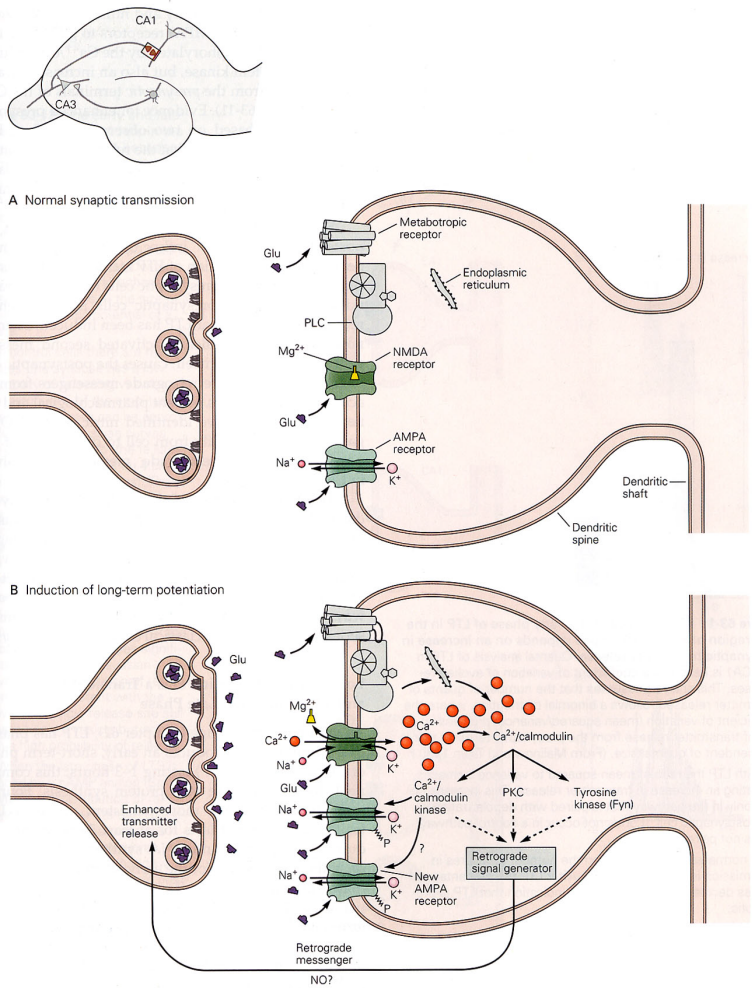

Figure 63-10　A model for the induction of the early phase of long-term poten- tiation. According to this model NMDA and non-NMDA receptor-channels are located near each other in dendritic spines.
A. During normal, low-frequency synaptic trans- mission glutamate (Glu) is released from the presynaptic terminal and acts on both the NMDA and non-NMDA receptors. The non-NMDA re- ceptors here are the AMPA type. Na^+ and K^+ flow through the non-NMDA channels but not through the NMDA channels, owing to Mg^{2+} blockage of this channel at the resting level of membrane potential.
B. When the postsynaptic membrane is depo- larized by the actions of the non-NMDA receptor- channels, as occurs during a high- frequency tetanus that induces LTP, the depolarization relieves the Mg^{2+} blockage of the NMDA channel. This allows Ca^{2+} to flow through the NMDA channel. The resulting rise in Ca^{2+} in the dendritic spine triggers calcium-dependent kinases (Ca^{2+}/calmodulin kinase and protein kinase C) and the tyrosine kinase Fyn that together induce LTP The Ca^{2+}/calmodulin kinase phosphorylates non-NMDA receptor-channels and increases their sensitivity to glumate thereby also activating some otherwise silent receptor channels. These changes give rise to a post-synaptic contribution for the maintenance of LTP. In addition, once LTP is induced, the postsynaptic cell is thought to release (in ways that are still not understood) a set of retrograde messengers, one of which is thought to be nitric oxide, that act on protein kinases in the presynaptic terminal to initiate an enhancement of transmitter release that contributes to LTP.