



浙江大学医学院学术报告

NMDAR-controlled CCK switches LTP and memory



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报告人简介:

Professor Jufang He is Chair Professor of Neuroscience of the City University of Hong Kong. He received a Doctoral Degree in Medical Science at the University of Tokushima, and a second Doctoral Degree in Engineering from The University of Tokyo, Japan. Before joining the City University of Hong Kong in 2013, Prof. He had worked at RIKEN (Japan), University of Tokushima, and Advanced Research Laboratory of HITACHI Ltd and The Hong Kong Polytechnic University for 20 years. Jufang was the director of a Hong Kong-Chinese Academy of Science (CAS) Joint Laboratory funded by Croucher Foundation and CAS. Prof. He combines electrophysiological, anatomical, and engineering approaches to answer fundamental questions of hearing, and learning and memory. Prof. He was awarded the President's Award for Research and Scholarly Activities (The Hong Kong Polytechnic University) 2003/2004. Jufang was selected as one of Distinguished Young Scholars by NSFC in 2006 (自然科学基金委杰出青年基金,海外B类, Joint Research Fund for Hong Kong and Macau Young Scholars) and as a recipient of the Croucher Senior Research Fellowship in 2010. He was the inaugural chair of Gordon Research Conferences on Neuroplasticity of Sensory Systems in 2018.

报告简介:

Memory is stored in neural networks via changes in synaptic strength mediated in part by NMDA receptor (NMDAR) -dependent long-term potentiation (LTP). Here we show that a cholecystinin-(CCK)-B-receptor (CCKBR) antagonist blocks high-frequency stimulation (HFS)-induced neocortical LTP, whereas local infusion of CCK induces LTP. CCK^{-/-} mice lacked neocortical LTP and showed deficits in a cue-cue associative learning paradigm, and administration of CCK rescued associative learning deficits. HFS-induced neocortical LTP was completely blocked by either the NMDAR antagonist or the CCK_{BR} antagonist, while application of either NMDA or CCK induced LTP after low-frequency stimulation (LFS). In the presence of CCK, LTP was still induced even after blockade of NMDARs. Local application of NMDA induced the release of CCK in the neocortex. These novel findings suggest that NMDARs control the release of CCK, which enables neocortical LTP and the formation of cue-cue associative memory.

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