College of Engineering

Department of Biomedical Sciences

Seminar

NMDAR-controlled CCK switches neocortical LTP and associative memory

Chair Prof. Jufang He

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Date: 26 February 2019 (Tuesday)

Time: 12:00 nn – 1:30 pm (Reception with light sandwiches at 11:45am, talks start at 12nn. To facilitate the order of sandwiches, please register through email chchung33@cityu.edu.hk.)

Venue: R5004, Bank of China Building, City University of Hong Kong (Please note the new location)

Abstract

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 cholecystokinin-(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 CCKBR 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.

Besides research presentation, there will be discussions on research collaborations for major grants.

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