Names (Presenter in bold font)	**Xuan Ling Hilary Yong**[1], Lingrui Zhang[1], Liming Yang[1], Xiumin Chen[2], Jing Zhi Anson Tan[1], Xiaojun Yu[1], Mintu Chandra[3], Emma Livingstone[3], Jocelyn Widagdo[1], Marta M Vieira[4], Katherine W Roche[4], Joseph W Lynch[2], Angelo Keramidas[1,3], Brett M Collins[3], Victor Anggono[1]
Affiliations	1 Clem Jones Centre for Ageing Dementia Research, Queensland Brain Institute, The University of Queensland, St Lucia, QLD, Australia 2 Queensland Brain Institute, The University of Queensland, Brisbane, QLD, Australia 3 Institute for Molecular Bioscience, The University of Queensland, Brisbane, QLD, Australia 4 Receptor Biology Section, National Institute of Neurological Disorders and Stroke, National Institutes of Health, Bethesda, MD, USA
Title	Regulation of NMDA receptor trafficking and gating by activity-dependent CaMKIIα phosphorylation of the GluN2A subunit
Abstract (max 300w)	NMDA receptor (NMDAR)-dependent Ca^{2+} influx underpins multiple forms of synaptic plasticity. Most synaptic NMDAR currents in the adult forebrain are mediated by GluN2A-containing receptors, which are rapidly inserted into synapses during long-term potentiation (LTP); however, the underlying molecular mechanisms remain poorly understood. In this study, we show that GluN2A is phosphorylated at Ser-1459 by Ca^{2+}/calmodulin-dependent kinase IIα (CaMKIIα) in response to glycine stimulation that mimics LTP in primary neurons. Phosphorylation of Ser-1459 promotes GluN2A interaction with the sorting nexin 27 (SNX27)-retromer complex, thereby enhancing the endosomal recycling of NMDARs. Loss of SNX27 or CaMKIIα function blocks the glycine-induced increase in GluN2A-NMDARs on the neuronal membrane. Interestingly, mutations of Ser-1459, including the rare S1459G human epilepsy variant, prolong the decay times of NMDAR-mediated synaptic currents in heterosynapses by increasing the duration of channel opening. These findings not only identify a critical role of Ser-1459 phosphorylation in regulating the function of NMDARs, but they also explain how the S1459G variant dysregulates NMDAR function.

Abstract deadline: Jan 20 (Please send to cjcadradmin@qbi.uq.edu.au)