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Inhibitory neurons play key role in mediating multiple phenotypes in a mouse model of inherited glycosylphosphatidylinositol deficiency disease

Qi Zhang

Laboratory of Social Neural Networks, University of Tsukuba, Japan

Posttranslational modification of a protein with glycosylphosphatidylinositol (GPI) is a conserved mechanism exists in all eukaryotes. Thus far, >150 human GPI-anchored proteins have been discovered and ~30 enzymes have been reported to be involved in the biosynthesis and maturation of mammalian GPI. Phosphatidylinositol glycan biosynthesis class A protein (PIGA) catalyzes the very first step of GPI anchor biosynthesis. Patients carrying a mutation of the PIGA gene usually suffer from inherited [glycosylphosphatidylinositol deficiency](#) (IGD) with intractable epilepsy and intellectual developmental disorder. We generated three mouse models with PIGA deficits specifically in telencephalon excitatory neurons (Ex-M-cko), inhibitory neurons (In-M-cko) or [thalamic neurons](#) (Th-H-cko), respectively. Both Ex-M-cko and In-M-cko mice showed impaired long-term fear memory and were more susceptible to kainic acid-induced seizures. In addition, In-M-cko demonstrated a severe limb-clasping phenotype. Hippocampal synapse changes were observed in Ex-M-cko mice. Our Piga conditional knockout mouse models provide powerful tools to understand the cell-type specific mechanisms underlying inherited GPI deficiency and to test different therapeutic modalities.

Biography

Qi Zhang is affiliated to University of Tsukuba, Japan. She is a recipient of many awards and grants for her valuable contributions and discoveries in major area of Human Sciences. Her international experience includes various programs, contributions and participation in different countries for diverse fields of study. Her research interests reflect in his wide range of Publications in various national and international journals. Research interested in [Neuroscience](#) social behaviour, emotion, behavioural genetics, synapse.

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