

3rd International Conference on

Epilepsy and Treatment

August 31 - September 01, 2017 Brussels, Belgium

Palmitoylation of neural proteins might be a link between lipid metabolism and anti-convulsant action

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We recently found that valproic acid suppresses palmitoylation of neural proteins including AKAP5 (AKAP79/150). Reduced AKAP5 palmitoylation disrupted regulation of the M-current, which is generated by neural Kv7 channel family. Various neurotransmitters that activate Gq-coupled receptors suppress M-current and increase neuronal excitability. We show that palmitoylation is required for receptor-induced M-current suppression. Similar disruption of M-current suppression was observed by inhibition of acyl-CoA synthetase. These results might fill in a gap between lipid metabolism and its anti-convulsant action.

Biography

Naoto Hoshi has completed his MD/PhD at Kanazawa University (Japan) and became a junior faculty there. He moved to the lab of Dr. John D. Scott, (Howard Hughes Medical Institute/Oregon Health Sciences University at the time). He now is in University of California, Irvine. His primary interest is physiological and pathological relevance of neuronal K7.2 channel modulation.

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