Form 1-5-2

Japan-U.S. Brain Research Cooperation Program
Researchers Dispatched to the U.S. Program FY2015: Report

Field:_ Cellular/Molecular___

1. Researcher

Name: Tomohiko Irie
Title: Senior Researcher
Affiliation: Division of Pharmacology, National Institute of Health Sciences

2. Research Title:
How do Kv1.4 channels on dendrites and spines control spike-timing dependent plasticity? -A physiological study using 2-photon microscopy imaging and patch-clamp recordings-

3. U.S. Joint Researchers/Institutes
Please give the name, title and affiliation.
Laurence O. Trussell, Professor, Oregon Hearing Research Center & Vollum Institute, Oregon Health & Science University,

4. Research Period, from/to (mm/dd/yyyy):
From 06/06/2015 to 02/20/2016

5. Abstract, Results, and Research Significance (300 Words):

Spontaneous firings underlie a variety of neuronal functions, including breathing and sleep rhythms. However, the cellular mechanisms that modulate the spontaneous firings are poorly understood.

It is known that auditory midbrain nuclei of the mammalian auditory system play roles in sound localizations. When the neurons in the nucleus exhibit hyper-excitability, this causes tinnitus. The interneurons and principal cells in the nucleus can express spike-timing synaptic plasticity, which is thought to cause the tinnitus. The interneurons show spontaneous firing even in slice preparations, and therefore we considered that the neurons could be the ideal neuronal model for the study of modulations of neuronal spontaneous firings.

In this experiment, we conducted in vitro patch-clamp recording and simultaneous calcium imaging using two-photon microscopy Mice brainstem slice preparations were used. Bath application of ryanodine induced the change of spontaneous firing pattern. The similar changes were observed in the presence of fast synaptic transmitter blockers. In order to determine the subcellular regions in which the Ca^{2+}-induced-Ca^{2+} release mediated by ryanodine receptors occurs, intracellular Ca^{2+} imaging was performed. When action potentials were evoked, calcium transients were observed in the cell body, axon initial segment, and dendrite. The transients evoked at cell body were ryanodine-sensitive, indicating that the Ca^{2+}-induced-Ca^{2+} release in the interneurons can be induced at the cell body, not axon initial segment nor dendrite

These results would contribute to understand the cellular mechanisms of the modulations of spontaneous firings in the central nervous systems.

6. Other (Research concerns, particular points of note):

*Please attach any reference materials as necessary.