



## 演者:Dr. Ryohei Yasuda

Assistant Professor

Department of Neurobiology, Duke University Medical Center

## 演題:樹状突起スパインの構造的機能的変化の分子メカニズム

Signaling mechanisms underlying structural and functional plasticity of dendritic spines

## 要旨:

In the central nervous system, most excitatory synapses terminate on dendritic spines, tiny (~0.1 femtoliter) protrusions emanating from the dendritic surface. Calcium-dependent signaling in dendritic spines underlies many forms of synaptic plasticity. To further our understanding of signal transduction in spines, we have developed a technique to measure signaling activity in individual spines in light scattering tissue using FRET signaling sensor in combination with 2-photon fluorescence lifetime imaging microscopy. Using this technique, we imaged activity of signaling proteins important for synaptic plasticity, Ras, RhoA, Cdc42 and CaMKII, during synaptic potentiation and associated spine enlargement induced at a single spine. These signaling proteins have distinct spatiotemporal patterns: Ras and RhoA activation spread out of the stimulated spine and diffuse along the parent dendrite over  $\sim 5$ micrometers. This diffusive signaling may play roles in dendritic signaling required for synaptic potentiation such as receptor exocytosis in dendrites or endosome trafficking from the dendrites to spines, as well as cooperative synaptic plasticity. In contrast, CaMKII and Cdc42 activations are restricted to the stimulated spines. CaMKII activity lasts only  $\sim 1 \text{ min}$ , while Cdc42 activity lasts more than 30 min, acting downstream of CaMKII. Thus, CaMKII-Cdc42 signaling is presumably important for maintaining synapse-specificity of synaptic potentiation for long term.

(講演は日本語で行われます)

日時:平成22年10月18日(月)16:30~18:00

場所:東京大学医学部教育研究棟2階第1・第2セミナー室



多数の皆様のご来聴を お待ちしております

主催:大学院医学系研究科 神経生理学教室

03-5802-3314 内線23536