Seminar 1

Wednesday, 24 June 15:00 – 15:50

Title: How do we define the reward in reinforcement learning?
Speaker: Doctor Eiji Uchibe, OIST

Abstract:

In application of reinforcement learning algorithms to real world problems, the design of reward functions is critical for successful achievement of a task. We may have only a very rough idea of the reward function whose optimization would generate desirable behavior, so straightforward reinforcement learning may not be usable. To find a good reward function, two approaches are considered. One is inverse reinforcement learning which infer the reward function from observed behaviors which are usually assumed to be optimal. The other approach is so-called intrinsically motivated reinforcement learning, in which the agent learns behaviors from extrinsic rewards from the environment and intrinsic rewards calculated by the agent based on information theory, emotion, task complexity, and so on. This talk briefly introduces inverse reinforcement learning and intrinsically motivated reinforcement learning. Next, we will explain our methods for those problems: inverse reinforcement learning with density ratio estimation and constrained policy gradient for intrinsic and extrinsic rewards.

Seminar 2

Wednesday, 24 June 16:00 – 16:50

Title: Deep Convolutional Neural Network Neocognitron and its Advances
Speaker: Doctor Kunihiko Fukushima, Fuzzy Logic Systems Institute

Abstract:

The "neocognitron" is a multi-layered convolutional network that can be trained to recognize visual patterns robustly. In lower layers of the network, local visual features are extracted from input patterns. Extraction and integration of visual features are repeated in the intermediate layers, and higher-order features are gradually extracted. In the highest layer, input patterns are classified based on the features extracted by the intermediate layers. Although the neocognitron has a long history, modifications of the network to improve its performance are still going on. The neocognitron can be classified to a so-called deep convolutional neural network, but there are several differences in detail. Focusing on these differences, this paper discusses neocognitron of a recent version.

Seminar 3

Wednesday, 24 June 17:00-17:50

Title: Exploring the mechanisms which control emotions and their memories by phosphoproteomic analysis

Speaker: Doctor Kozo Kaibuchi,
Department of Cell Pharmacology, Nagoya University Graduate School of Medicine

Abstract:

It is well known that dopamine (DA) is necessary for motor function, motivation, working memory and the reward system. DA signaling dysfunction has been implicated in various neuropsychological diseases, including Parkinson's disease, drug addiction, compulsive behavior, attention-deficit/hyperactivity disorder, autism spectrum disorders and schizophrenia. The principal target of DA is medium spiny neurons (MSNs), which are a special type of GABAergic inhibitory cell that represents 95% of the neurons within the striatum, including the nucleus accumbens (NAc). There is a distinct class of spatially intermixed MSNs that express DA type 1 or 2 receptors (D1R-MSNs or D2R-MSNs, respectively). D1R signaling mediates direct pathways projecting to the pars reticulata, whereas D2R signaling mediates indirect pathway projecting to the external globus pallidus. D1R signaling in the NAc has been implicated in reward-related behaviors such as appetitive reward learning and adaptive responses to cocaine. DA appears to modulate the excitability of MSNs via D1R stimulation for controlling the behaviors presumably acting through protein kinase A (PKA) because PKA plays pivotal roles in DA signaling. However, whether and how PKA regulates the excitability of MSNs, and the reward-related behaviors remains largely unknown. Although major efforts have been made to identify the target substrates of PKA to understand the modes of action of DA, a few substrates including DARPP-32, GluR1 and NR1 have been reported and these substrates do not explain how PKA regulates the neuronal excitability.

To this end, we here developed a kinase-oriented phospho-proteomic analysis method that uses affinity beads coated with 14-3-3 proteins to enrich the phosphorylated proteins. By using this method, we comprehensively identified the PKA substrates downstream of D1Rs in the striatum to elucidate the PKA-mediated signal pathways. We found more than 100 candidate substrates of PKA, including Rap1 GEF (Rasgrp2). We also found that Rap1 activated by PKA and Rasgrp2 regulated the neuronal excitability and cocaine-induced reward responses.