SY1-F1-5 Adaptability of the biped robot

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Body compliance of organims is supposed to play a crucial role for adaptive behavior. If robots are designed to have rigid bodies, their upper limitation of controllability is the control bandwidth. On the other hand, if the robots are compliant, it can react to environmental changes with no time-delay. In the talk, we will pick up biped locomotion driven by pneumatic artifitial muscles which is essentially compliant, and discuss on the adaptability.

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SY1-F2-1 Neuronal development and neurodegeneration on Cdk5 and related signaling

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Cdk5, a member of cyclin-dependent kinases, is a Ser/Thr protein kinase activated by binding a neuron specific activator p35 or p39. Cdk5/p35 functions in both brain formation and neurodegeneration. These functions are closely related to the kinase activity and cellular localization of Cdk5. In healthy neurons, Cdk5 associates with membranes via myristoylation of p35, and membrane association maintains the kinase activity to moderate levels. The kinase activity of Cdk5 is also regulated by degradation and cleavage of p35. p35 is a short life protein degraded by proteasome on membranes and cleaved to p25 by calpain in neurons undergoing cell death. The cleavage induces abnormal activation, resulting in neuronal cell death of neurodegenerative diseases. Degradation of p35 is regulated developmentally by phosphorylation. The phosphorylated form in embryonic or juvenile brains is easily degraded and the dephosphorylated form in matured neurons has a propensity to be cleaved. How Cdk5 kinase activity is regulated and involved in brain development and pathology will be further discussed.

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SY1-F2-2 The role of Dock family Rho-GEFs in dendrite morphogenesis

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Rho family small GTPases play important roles in various aspects of neuronal development including dendritic growth and spine formation, although the molecular mechanisms that control their activities are not fully understood. Recent studies have identified an evolutionarily conserved superfamily of Dock180-related proteins that function as novel types of guanine nucleotide exchange factors (GEFs) for Rho family GTPases, and disruption of genes encoding members of this family has been associated with human neurological diseases such as mental retardation. We found that Dock4, a Rac-specific GEF, regulates dendrite branching, whereas Dock9/Zizimin1, a Cdc42-specific GEF, promotes dendrite growth in hippocampal neurons. In addition, Dock4 localizes to dendritic spines at late developmental stages in hippocampal neurons and regulates spine formation. We are also studying the molecular mechanisms of the regulation of dendrite development by Dock proteins. I will discuss the diverse roles of Dock family GEFs in the regulation of dendrite morphogenesis and neuronal development.

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SY1-F2-3 Homeostasis of nigro-striatal dopaminergic projection – compensatory regulation of tyrosine hydroxylase and dopamine content

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Nigro-striatal dopaminergic transmission is important for regulation of voluntary movement. Homeostatic change of nigro-striatal dopaminergic projection has been suggested from studies on Parkinson's disease and its models. However, it is still unclear how dopaminergic neurons are homeostatically regulated. To further investigate this issue, we generated floxed tyrosine hydroxylase (TH) mice and used an adeno-associated viral vector expressing Cre recombinase (AAV-Cre). Stereotaxic microinjection of AAV-Cre into the substantia nigra pars compacta (SNc) of adult floxed TH mice efficiently ablated the TH gene. We found that, the rate of decrease in TH protein level is slower in the striatum (axon terminals) than in SNc (cell bodies). Moreover, in the striatum, while TH protein level decreased to about 20% eight weeks after injection, dopamine contents decreased to only about 50%. These results suggest that both TH protein level and activity are compensatory regulated in response to TH gene inactivation.

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SY1-F2-4 Control of myelination by oligodendrocytes through the novel phosphorylation mechanism of cytoskeletal proteins

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Oligodendrocyte precursor cells (OPCs) differentiate into oligodendrocytes (OLs) in order to form myelin, which is required for the rapid propagation of action potentials. During the development, precursors of OPCs are generated in germinal areas of the central nervous system (CNS), then proliferate and migrate into subcortical white matter, where they extend complex branched processes and initiate myelination. Since the molecular mechanism(s) that control OL development and function are still largely unknown, it is important to identify the signaling molecule and the pathway leading to each of these developmental stages. Here, we identify that the unexpected new role of Cdk5 in the early developmental stages of OLs. This will provide not only new insights into OL biology but also possible therapeutic approaches for CNS diseases such as demyelination and damaged myelin.

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SY1-F2-5 Role of Hes family molecules in the therapeutic appoach for spinal cord injury

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In the rodent moldels of spinal cord injury, the insults induce proliferation of endogenous oligodendrocyte progenitor cells (OPCs). Since those OPCs have potency for tissue repair, the manipulation of their proliferation and differentiation can be a therapeutic target for the disorder. To understand the mechanisms which govern the maturation of oligodendrocytes, we established murine OPC primary culture in which basic fibroblast growth factor (bFGF) promotes proliferation and maintains the cells in an immature state. From the molecular screening using DNA microarray, we identified Hes1 as a crucial molecule in bFGF-mediated expansion of OPCs in vitro. On the other hand, we could not detect Hes1 expression in OPCs located in injured spinal cords, which may explain the limited expansion of those cells after traumatic insults and the poor recovery of the impaired locomotor functions.

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SY1-F3-1 Development of a retinal circuitry coding for motion directions

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Selective responses of retinal ganglion cells (RGCs) to motion directions have been recorded extracellularly from the rabbit and the mouse retina at eye-opening. It has been shown that the development of this circuitry is light independent. With whole-cell patch clamp recording, we found that early postnatal direction selective ganglion cells (DSGCs) showed many difference in physiological properties, yet the same degree of DS measured by the DS index and the width of directional tuning curve to that of adult DSGCs. The DSGCs exhibited a clear selectivity for motion directions at the onset of light sensitivity. Furthermore, the degree of DS is not affected by dark-rearing from birth to postnatal day 11 (P11) or to P30. The formation of the retinal neurocircuitry for coding motion direction is completely independent of light. The contribution of different neurotransmitters to the excitatory input and the neuronal activity to the circuitry formation are also investigated.

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