Special Lectures for Kagoshima Meeting

Educational Lecture

1SL02D1 March 16, 9:00–10:00, Room D1

Physiology as a science for elucidating the mechanism of behavior

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Behavior is composed of bodily and mental activities occurring in an organism for a certain purpose. While physiological study can help reveal the underlying mechanisms of a variety of behaviors, the basis of many daily behaviors remains poorly understood. Owing to the influence by the late Professor Yojiro Kawamura, as a founder of Oral Physiology, I have been working in the field of ingestive behavior with special reference to chemical senses such as taste and smell by seeking reasons for simple questions, such as why sugars are sweet and sodium chloride is salty? Why some foods are palatable and others are aversive? Why some people eat fast and others slow? Why some people are fat and others are lean, etc. I would like to show some data addressing these questions, namely, the importance of the chemotopic organization in the cortex for taste quality recognition, the basolateral nucleus of amygdala and the reward system for the hedonic shift of taste palatability in the conditioned taste aversion paradigm and the involvement of brain substances such as beta-endorphin, dopamine and orexin for palatability-induced feeding behavior. In addition, a certain odor, Osmanthus fragrance, attenuates food intake by influencing the expression of feeding-related neuropeptides and one of the spices, wasabi, may be effective in preventing obesity and diabetes mellitus. Such physiological studies as answering the popular questions regarding common human behaviors are not only scientifically important to solve but also would attract young people to physiology as well as science itself. No COI.

3SL13E March 18, 11:05–12:05, Room E

Adaptation of neuromuscular properties to microgravity environment

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Chronic exposure to space environment causes some serious responses in physiological properties, due to gravitational unloading, radiation, mental stress, etc. Removal of weight-bearing activity induces remarkable effects on the skeletal muscles and bones responsible for maintaining posture and ground support. For example, chronic unloading of muscles, such as bedrest in humans and hindlimb suspension and actual spaceflight in animals, results in muscle fiber atrophy and a shift toward a faster myosin heavy chain profile, particularly in muscles composed predominantly of slow fibers such as soleus and adductor longus. These responses are closely related to a decrease in the neuromuscular activity levels, i.e., mechanical loading, neural activation, and/or metabolic factors. Inhibition of tension development caused by passive shortening of muscle fibers or sarcomeres plays one of the major roles in advanced breakdown and/or shift of the distribution of proteins in skeletal muscles. Further, unloading-related changes in muscular properties, including mobilization patterns, are influenced by inhibition of afferent input. However, activation of afferent input also stimulates the invasion of T helper 17 cells at L5 segmental level of spinal cord, which causes neuroimmune disease. I would like to discuss about the neuromuscular responses and roles of afferent input on the physiological adaptation to gravitational unloading. No COI.

2SL09F March 17, 11:05–12:05, Room F

Computer simulation of basic cardiac cell functions for learning Cardiac Physiology at levels of cell and tissue

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To greatly facilitate understanding of dynamic properties of cardiac cells, we are aiming at preparing a computational package of educational simulation softwares, which are largely based on detailed biophysical cardiac myocyte models. The cell models include the membrane excitation, intracellular ion concentrations, Ca2+ dynamics at the sarcoplasmic reticulum, the contraction of myofilaments, and the oxidative phosphorylation in mitochondria. The cell models are for the sinoatrial node pacemaker, atrial, and ventricular cells. The ion channel as well as ion transporters, such as Na/K pump and the Na/Ca exchanger, incorpolated in the cell models, can be examined by reconstructing the voltage clamp records, single channel recordings, or by applying different ion concentrations, to see their time- and voltage-dependent activation as well as inactivation if any. The cell models can be connected to form a one-dimensional array of cells connected with the gap junctional channels to simulate the AP propagation under various physiological and pathophysiological conditions. The relationship between the gap junction channel conductance and the AP propagation velocity can be visualized on the graphics. Principal mechanisms underlying the pacemaker shift, the one directional conduction as well as the re-entry can be demonstrated in appropriate cell arrays. The abnormal membrane excitation, such as the delayed after depolarization or the early after-depolarization can be demonstrated by applying various interventions to the ventricular cell models. The ventricular cell models can also be used to simulate the function at the tissue and organ levels. The abnormal excitation patterns can be triggered in a two dimensional array of model cells. The cardiac cycle can be simulated by assuming a simple Laplace heart, after-load and pre-load. The pressure-volume curve or trajectry can be examined at various pre-load or after-load by which maximum elastance (Emax) can also be evaluated. The relation between the conduction velocity and the force length relation of cell can be examined by using the one dimensional fiber model combined with the circulation model. If ectopic focuses of membrane excitation were assumed in a heart model of a ring shape, modifications of cardiac output were examined. No COI.