The 1st International Sport Neuroscience Conference 2019

Sep.18 WED 13:00-19:00
Sep.19 THU 9:00-13:00

TSUKUBA, JAPAN

IBRO Satellite Forum

International Congress Center
EPOCHAL TSUKUBA, Japan

IBRO 2019 Joint Meeting of IBRO & FAONS
SPORT NEURO SCIENCE
第74回 The 74th Japanese Society of Physical Fitness and Sports Medicine 日本体力医学会大会
WILLDYNAMICS

The 1st International Sport Neuroscience Conference 2019

Moderator: Masahiro Okamoto (University of Tsukuba, Japan)

DAY1 - September 18 Wed.		Speakers	Chairs	Venue
12:45-13:00	**Photo Session for Guest Speakers**			2F Convention Hall 200
13:00-13:05	**Opening Remarks**	Takeshi Nishijima (Tokyo Metropolitan University, Japan)		
13:05-14:00	**Prologue**	Hideaki Soya (University of Tsukuba, Japan)	Takeshi Nishijima (Tokyo Metropolitan University, Japan)	
14:00-14:10	Coffee Break			
14:10-14:55	**Session 1**	Pierre J. Magistretti (KAUST, Saudi Arabia and EPFL, Switzerland, IBRO's President)	Hideaki Soya (University of Tsukuba, Japan)	
14:55-15:05	Coffee Break			
15:05-15:25	**Session 2** **Co-sponsored by WILLDYNAMICS**	Takeshi Sakurai Shingo Soya (University of Tsukuba, Japan)	Kazuya Suwabe (University of Tsukuba, Japan)	
15:25-16:25	**Data Blitz for Poster Presentation**	All poster presenters	Shingo Soya (University of Tsukuba, Japan)	
16:25-17:15	**Poster Session** | Coffee Break			
17:15-18:45	**Session 3** **Exercise and Neurodegenerative Disease**	Ignacio Torres Aleman (Cajal Institute, Spain) Zsolt Radak (University of Physical Education, Hungary) Chia-Liang Tsai (National Cheng Kung University, Taiwan)	Tetsuya Shiuchi (Tokushima University, Japan)	
18:45-19:00	**Closing Remarks**	Junichi Nabekura (NIPS, Japan)		
	Photo Session			
19:30-	**Reception / Poster Award Ceremony**		Takashi Matsui (University of Tsukuba, Japan)	1F Restaurant

DAY2 - September 19 Thu.		Speakers	Chairs	Venue
9:00-9:50	**Session 4** **Sports Performance and Neurorehabilitation**	Naznin Virji-Babul (University of British Columbia, Canada) Kimitaka Nakazawa (University of Tokyo, Japan)	Ryoichi Nagatomi (Tohoku University, Japan) Hidefumi Waki (Juntendo University, Japan)	2F Convention Hall 200
9:50-9:55	Coffee Break			
9:55-10:50	**Session 5**	Henriette van Praag (Florida Atlantic University, USA)	Zsolt Radak (University of Physical Education, Hungary)	
10:50-10:55	Coffee Break			
10:55-11:50	**Session 6**	Art F. Kramer (Northeasten University, USA)	Hideaki Soya (University of Tsukuba, Japan)	
	Move to 1F Room 102			
12:00-13:00	**Luncheon Seminar**	Maria Llorens-Martin (Centro de Biología Molecular "Severo Ochoa", Spain)	Hideaki Soya (University of Tsukuba, Japan)	1F Conference Room 102

Welcome to the 1st International Sport Neuroscience Conference

Hideaki Soya

President of ISNC, University of Tsukuba

On behalf of the organizing committee, it is our great pleasure to welcome you to the 1st International Sport Neuroscience Conference and to also welcome you to the city of Tsukuba. This conference has been planned by the core members of the Japanese Society of Sport Neuroscience (JSSN), which was inaugurated in 2007 and belongs to the Japanese Society of Physical Fitness and Sports Medicine (JSPFSM), and approved as a satellite meeting of the 10th International Brain Organization (IBRO2019) (Daegu, Sep 21-25) with the strong recommendation of IBRO president Pierre Magistretti. Since the annual meeting of the JSPFSM will also be held in Tsukuba just before IBRO2019, this conference will be jointly hosted by JSSN, IBRO2019 and JSPFSM in Tsukuba.

Tsukuba plays an important role as an international science city with more than 60 national research institutes as well as a national university (University of Tsukuba). The University of Tsukuba was founded in 1872 and the faculty of physical education was opened in 1878, making it the oldest in Asia. Dr. Jigoro Kano, the founder of Judo, served for 25 years as a school principal before building a career at the University of Tsukuba. Dr. Kano's legacy to us is the educational philosophy that our minds are strengthened through proper physical training. Thus, the University of Tsukuba is a fitting place to assemble for this unique international conference.

While nascent, the field of Sport Neuroscience is rapidly growing worldwide and stands to make a substantial impact on human health. A growing number of studies have revealed that our brains are remodeled to become more powerful and efficient with regular exercise habits and sports challenges. Thus, the mission of the JSSN is to explore how the brain works and develops, and how it reaps the wide variety of exercise-induced benefits to cognition, motor control, stress resilience, and high-performance in various people, including vulnerable individuals such as the elderly, in order to design tailor-made exercise prescriptions and to help people develop and maintain healthy exercise habits.

The aim of the conference is to share current topics both with researchers who are engaged in work related to research into exercise and sports that is based on behavioral neuroscience, cognitive neuroscience and neuro-rehabilitation, and with those in fields of physiology, biochemistry, health fitness, and so on. Bringing together talented researchers in such a wide range of fields provides an opportunity to focus on many different aspects of the link between brain health and physical health; for example: How do exercise and sports impact brain functions and health? How does exercise contribute to reducing neurodegenerative diseases? What role do brain functions play in exerting human high performance?

In addition, we are proud to bring you a great synergy of expertise from internationally renowned neuroscientists with several interesting keynote lectures. Further, a datablitz session among keynote speakers at the midpoint of the first day of meetings will focus on addressing current achievements associated with future trends and challenges in scientific research, technology development, and clinical practice. Through this conference we will provide a forum for networking and information exchange that we hope will prove invaluable to current researchers. It is also an opportunity for young scientists and students to explore opportunities for future projects as well as potential for professional development.

Our committee is comprised of the following members: Prof. Pierre J. Magistretti (KAUST, EPFL, IBRO's President), Prof. Junichi Nabekura (NIPS, President of FAOPS2019), Prof. Art F. Kramer (Northeastern Univ.), Prof. Henriette van Praag (Florida Atlantic Univ.), Prof. Ryoichi Nagatomi (Tohoku Univ.), Prof. Hidefumi Waki (Juntendo Univ.), Prof. Ignacio Torres Aleman (Cajal Institute), Prof. Michael Yassa (UC-Irvine), Prof. Zsolt Radak (Univ. of Phys. Edu. Hungary), Dr. Maria Llorens-Martín (Univ. of Autonoma), Assoc. Prof. Takeshi Nishijima (Tokyo Metropolitan Univ.), Assoc. Prof. Tetsuya Shiuchi (Tokushima Univ.), Dr. Kazuya Suwabe, Dr. Genta Ochi, Assist. Prof. Masahiro Okamoto, Assist. Prof. Takashi Matsui and Prof. Hideaki Soya (Univ. of Tsukuba). We would like to thank our community supporters and advocates as well as our hard-working staff and students who have made this meeting possible. Welcome to the conference!

Hideaki Soya, Ph.D.
Director, Advanced Research Initiative for Human High Performance (ARIHHP)
Faculty of Health & Sport Sciences, University of Tsukuba, Japan

Promotion of Brain Fitness with Mild Enjoyable Exercise: Translational Research

Plato wrote, "In order for man to succeed in life, God provided him with two means, education and physical activity. Not separately one for the soul and the other for the body, but for the two together. With these two means, men can attain perfection" (Plato, 4th century B.C.).

Recent neuroscience research shows that for optimal physical and mental performance, exercise is essential. Since benefits induced by daily exercise habits are known to extend to the brain, where neurons and their networks can be remodeled, such habits result in improved mental health and physical performance.

Take, for example, the hippocampus, a region that is responsible for the formation of memories. Aerobic physical activity (APA) can enhance the formation of new neurons (neurogenesis), in the dentate gyrus in the hippocampus (van Praag, 2008 etc.). Thus, APA can lead to an increase in the size of the hippocampus, as it does with muscle tissue, and also to improved memory (Erickson and Kramer et al., 2011). This is also the case for most of the prefrontal cortex, which is responsible for executive functions. Furthermore, aerobic fitness is associated with memory and executive function (Erickson et al, 2009; Suwabe et al., 2017; Colcombe et al., 2004; Hyodo et al., 2016). The close relationship between cardiovascular functions and cognition is thought to be important in terms of evolutionary advantage of Homo sapiens (Lieberman, 2013). The reason for this remains uncertain, although there is a hypothetical relationship for an evolutionary model linking APA and brain size in human, where selection acting to improve APA performance through enhanced metabolic regulation and oxygen transport alters baseline neurotrophic and growth-factor signaling (Raichlen and Polk, 2013). Thus, Plato's words ring true: a healthy body and healthy mind are undeniably linked.

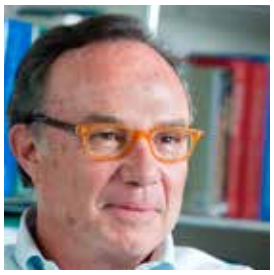
Mental health is a decisive factor in determining our quality of life, and exercise has a positive effect on mental health because it acts on the brain. Exercise impacts stress-related diseases such as depression, neurodegenerative disorders such as dementia, and inactivity-related diseases such as diabetes and obesity. Inactivity leads to an overall weakening of our bodies and minds: this is a global issue that must be addressed. In Japan, escalating healthcare costs recently reached a new high, and many people, including a large number of children, suffer from depression and obesity, often combined with reduced physical fitness. Finding ways to improve adherence to exercise is an urgent issue. But it's hard to maintain motivation with vague promises of future health benefits that are years away.

Fortunately, in many countries, several forms of bodywork such as yoga and tai chi, in which people meditate deeply and become aware of their posture and breathing, are used in daily life. However, there is still very little data for the effects of mild exercise and bodywork. While the ACSM (American College of Sports Medicine) guidelines for exercise prescription define both very light and light intensity exercise, empirical exploration of their beneficial effects is still limited, and most known exercise benefits result from moderate-intensity exercise.

To address this, we've been doing translational research, called the "Brain Fitness" project, which aims to explore exercise conditions that enhance endurance, cognition, and motivation. We developed animal and human exercise models based on lactate threshold level (a physiological measure) and found positive effects of acute mild exercise on brain loci including the prefrontal cortex in human (Byun et al., Neuroimage, 2014), and the hippocampus in human (Suwabe et al., PNAS, 2018) and animals (Soya et al., NRS, 2007). Chronic mild exercise regimens resulted in improved executive functions and spatial memory, respectively. Such beneficial effects were greater in subjects with higher endurance, which opened the possibility that endurance capacity is an important factor in improving cognitive function through exercise that might involve certain factors acting on brain and brawn simultaneously. Thus, we explored two types of exercise regimens, mild and high-intensity interval, which not only improved endurance, but also enhanced hippocampus-dependent neurogenesis and memory, warranting further mechanistic studies. Interestingly, brain dopaminergic regulation increased with both regimens. Furthermore, a whole-genome hippocampal array study revealed some crucial molecular alterations with both regimens (e.g., upregulated APOE and IGF2, and downregulated TNF) (Inoue et al., Plos One, 2015). Therefore, any regimen which is meant to induce cognitive functions may require some level of endurance, as well as anti-inflammatory benefits and motivation factors. Exploration of such factors shall provide new insight into mechanisms by which exercise enhances cognitive functions and mitigates cognitive decline.

During this conference, we will share some of our current studies showing the cognition-enhancing role of very mild exercise on the brain, especially on the hippocampus (animals and human) and the prefrontal cortex (human). Our current findings show that even mild exercise activates the lateral prefrontal cortex (Byun et al., Neuroimage, 2014.) and hippocampus (Soya, BBRC, 2007; Suwabe et al., PNAS, 2018), which in turn improves cognitive functions such as memory and executive functions. Brain-genic androgen (Okamoto et al., 2012, PNAS) and lipids and inflammatory factors (Inoue et al., Plos One, 2015), together with bloodborne insulin-like growth factor (IGF-I) (Nishijima et al., Neuron, 2010) are thought to play an important role in the development of enhanced cognition. One especially interesting finding is the synergistically beneficial effects associated with brain-genic leptin when mild exercise is combined with astaxanthin, a natural pigment used as a supplement (Yook et al, PNAS, 2019).

We would be delighted if these research were to give rise to a boom in mild exercise such as body work, and, thus, in some small way serve to improve overall health both in Japan and throughout the world.



Pierre J. Magistretti, Ph.D.
Division of Biological and Environmental Sciences and Engineering, KAUST, Saudi Arabia
Department of Psychiatry, University of Lausanne Medical School, Switzerland

Neuron-Glia metabolic coupling mediated by lactate: role in neuronal plasticity, neuroprotection and neuropsychiatric diseases

A tight metabolic coupling between astrocytes and neurons is a key feature of brain energy metabolism (Magistretti and Allaman, Neuron, 2015). Over the years we have described two basic mechanisms of neurometabolic coupling. First the glycogenolytic effect of VIP and of noradrenaline indicating a regulation of brain homeostasis by neurotransmitters acting on astrocytes, as glycogen is exclusively localized in these cells. Second, the glutamate-stimulated aerobic glycolysis in astrocytes. Both the VIP-and noradrenaline-induced glycogenolysis and the glutamate-stimulated aerobic glycolysis result in the release of lactate from astrocytes as an energy substrate for neurons (Magistretti and Allaman, Neuron, 2015; Magistretti and Allaman, Nat Neurosci Rev, 2018; Cali et al., Front Cell Neurosci, 2019)).

We have subsequently shown that lactate is necessary not only as an energy substrate but also as a signaling molecule for long-term memory consolidation and for maintenance of LTP (Suzuki et al, Cell, 2011). At the molecular level we have found that L-lactate stimulates the expression of synaptic plasticity-related genes such as Arc, Zif268 and BDNF through a mechanism involving NMDA receptor activity and its downstream signaling cascade Erk1/2 (Yang et al, PNAS, 2014). A transcriptome analysis in cortical neurons has shown that the expression of a total of 20 genes is modulated by L-Lactate; of these, 16 involved in plasticity and neuroprotection are upregulated and 4 involved in cell death are downregulated (Margineanu et al. Front. Mol Neurosci, 2018). This set of results reveal a novel action of L-lactate as a signaling molecule in addition to its role as an energy substrate (Magistretti and Allaman, Nat Neurosci Rev, 2018).

These actions of L-Lactate are also relevant for animal models of neuropsychiatric disorders. Indeed we have shown that peripheral administration of lactate exerts antidepressant-like effects in three animal models of depression, Forced Swim test, Open Space Forced Swim Test and chronic corticosterone administration. These behavioral effects of L-Lactate administration are accompanied by changes in the expression of genes that have been involved in mood disorders (Carrard et al, Mol.Psy., 2016).

Finally, we have also shown neuroprotective effects of L-Lactate in vivo in a model of stroke and in vitro on excitotoxicity(Berthet et al, Cerebrovasc. Dis, 2012; Jourdain et al, Sci Reports, 2018) .

Session 2: Co-sponsored by WILLDYNAMICS

Takeshi Sakurai, Ph.D. /Shingo Soya, Ph.D.
International Institute of Integrative Sleep Medicine, University of Tsukuba, Japan

Deciphering the neural mechanism of 'willpower' that motivates voluntary wheel running

To live a creative and active life, it is essential to keep high willpower: an ability to try to overcome difficulties and challenges to achieve goals. However, the biological mechanism Although the reward system, executive function controlled by the prefrontal cortex may be involved, the biological machinery to generate willpower, a unique function of human, has been largely unknown. This research area "Willdynamics" aims to uncover the mechanism of this mental function, and the impact of social/internal environment on willpower.

Orexins, a critical factor in stabilization of sleep/wakefulness state, has been involved in motivated behavior (Sakurai et al., 2014). However, it is still unknown about how the neural circuit controlling arousal is also involved in "willpower". We focused on the role of orexin 1 receptor (OX1R) in voluntary wheel running and found that OX1R deficient mice showed significant decrease of wheel running activity. Intraperitoneal injection of OX1R antagonist (SB334867) to the WT mice also showed significant decrease of voluntary wheel running for several hours. To identify the downstream effector to regulate voluntary wheel running via OX1R signaling, we used OX1R floxed (Ox1rloxp/loxp) mice mated with different Cre driver mice to generate cell-type selective knockout mice. Specific deletion of OX1R in the dopaminergic (DA) neurons using DAT-Cre mice (Ox1rloxp/loxp; DAT-Cre) showed significant decrease of voluntary wheel running, although these mice showed comparable basal locomotor activity with controls. Moreover, acute optogenetic stimulation of DA neurons in the VTA recovered the amount of wheel running activity in Ox1rloxp/loxp; DAT-Cre mice. These results suggest that the excitatory transmission via OX1R in the DA neurons might be important to generate "willpower" for voluntary wheel running. Selective deletion of OX1R in the noradrenergic (NA) neurons (Ox1rloxp/loxp; NAT-Cre mice), or serotonergic (5-HT) neurons (Ox1rloxp/loxp; ePET-Cre mice) also showed significant decrease of wheel running and basal locomotor activity. We are continuing to explore the mechanism by which these monoaminergic systems contribute to the voluntary wheel running activity.

Session 2: Data Blitz for Poster Presentation

No	Title	Presenter	Affiliation
01	Analysis of gait motion change by intervention using robot suit HAL in acute and chronic stage myelopathy patients decompression surgery	Seioh Ezaki	Department of Orthopaedic Surgery, Faculty of Medicine, University of Tsukuba
02	Modulated muscle control during walking with hybrid assistive limb (HAL) in a patient with severe spinal cord disorder	Hideki Kadone	Center for Innovative Medicine and Engineering, University of Tsukuba Hospital, Tsukuba, Japan
03	Robotic intervention alters muscle coordination in acute post-stroke patients	Chun Kwang Tan	Center for Innovative Medicine and Engineering, University of Tsukuba Hospital, Tsukuba, Japan
04	Effects of lumbar support exoskeleton on muscle coordination in healthy people	Chun Kwang Tan	Center for Innovative Medicine and Engineering, University of Tsukuba Hospital, Tsukuba, Japan
05	Upper Limb Triggered HAL method: Novel gait training methods for patients with complete Quadri/Paraplegia due to chronic spinal cord injuries	Yukiyo Shimizu	Department of Rehabititation Medicine, University of Tsukuba Hospital
06	Elbow extension-flexion training using single joint HAL for patients with spastic cerebral palsy	Yukiyo Shimizu	Department of Rehabititation Medicine, University of Tsukuba Hospital
07	The relationship between higher coincident-timing task performance and eye movement in baseball players	Tochikura I.	Field of Health and Sports, Niigata University of Health and Welfare, Japan
08	Effects of electrical muscle stimulation on cognitive performance and cerebral blood flow	Takagi Y. *Travel Award	The University of Electro-Communications
09	Sympathetic nervous system activity during electrical muscle stimulation and voluntary exercise	Kitajima D.	The University of Electro-Communications
10	Table Tennis Players Have Superior Motion Vision in Peripheral Vision	Ryoma Goya	Graduate School of Frontier of Bioscience, Osaka University
11	The functional roles of eye movement in the continuous visuomotor action	Chisa Aoyama	Grad. Sch. of Med. Osaka Univ., Osaka, Japan
12	The effects of acute exercise on the formation process of visual perception	Takaaki Komiyama	Center for Education in Liberal Arts and Science, Osaka University, Osaka, Japan
13	The effects of visual exposure during exercise on visual contrast sensitivity	Kurata Ryo	Graduate School of Frontier Biosciences, Osaka University
14	Effects of saccadic eye training on the continuous visuomotor action	Taiga Mizumori	Grad. Sch. of Frontier Biosci. Osaka Univ.
15	JUDO a Gentle-Way to Smarter Brain -The Effect of JUDO-based exercise program on physical fitness and cognitive function in older people (+65)	Sylwester Kujach *Travel Award	Department of Physiology, Gdansk University of Physical Education and Sport, Gdansk, Poland
16	The effects of long-term resistance and aerobic exercise interventions on neurocognition and neuroprotective growth factors in the elderly with memory problems	Yu-Ju Chen	Institute of Physical Education, Health and Leisure Studies, National Cheng Kung University, Taiwan
17	Purine nucleotide salvage pathway: the effect of short term high intensity interval training at the different times	Rouhollah Haghshenas Gatabi	Department of Exercise Physiology Faculty of Physical Education, University of Mazandaran
18	The effects of Virtual Reality Environment on Physiological and Behavioural Responses to Road Cycling	Nurul Farha Zainuddin	School of Biomedical Engineering and Health Sciences, Faculty of Engineering, Universiti Teknologi Malaysia, Johor, Malaysia
19	The effects of daily physical activity and acute moderate exercise on human dopaminergic system: A preliminary study with spontaneous eye blink rate	Ryuta Kuwamizu *Travel Award	Exercise Biochem. & Neuroendocrinol., Univ. Tsukuba, Ibaraki, Japan
20	Relationship between aerobic fitness and functional connectivity during working memory task in older adults: functional near infrared spectroscopy study	Kazuki Hyodo	Physical fitness institute, Meiji Yasuda Life Foundation of Health and Welfare, Tokyo, Japan
21	What is the factor which modulate the combined effect of upper body dance-like movement and groove rhythm on executive function?	T. Fukuie	Laboratory of Exercise Biochemistry and Neuroendocrinology, University of Tsukuba, Japan
22	Translating and verifying pediatric acute cognitive dysfunction syndrome “delirium” assessment scale	Yujiro Matsuishi	University of Tsukuba, Doctoral program in Clinical Sciences-Graduate School of Comprehensive Human Sciences, Tsukuba, Japan
23	Sustainable resistance exercise mode to improve cognitive function	Keigo Tomoo	Faculty of Sport and Health Science, Ritsumeikan University, Kusatsu, Shiga, Japan
24	Sustainable aerobic exercise mode to improve executive function	Takeshi Sugimoto	Faculty of Sport and Health Science, Ritsumeikan University, Kusatsu, Shiga, Japan
25	Science and culture: Krok-Kradeuang, a new exercise equipment for all	Akkaranee Timinkul	Bachelor of Thai Traditional Medicine Program, UdonThani Rajabhat University, Thailand

Session 2: Data Blitz for Poster Presentation

No	Title	Presenter	Affiliation
26	Roles played by midbrain neurons projecting to the ventral medulla in generating central command function	Satoshi Koba	Division of Integrative Physiology, Tottori University Faculty of Medicine
27	Neural maturation enhanced by exercise-induced extracellular vesicles	Hyo Youl Moon	Seoul National University
28	Exercise-induced increment of corticosterone is essential for exercise-enhanced adult hippocampal neurogenesis	Sheng-Feng Tsai *Travel Award	Department of Cell Biology and Anatomy, Institute of Basic Medical Sciences, College of Medicine, National Cheng Kung University, Tainan, Taiwan
29	Exercise inhibits microglial activation by increasing circulating extracellular vesicles	Sheng-Feng Tsai	Department of Cell Biology and Anatomy, Institute of Basic Medical Sciences, College of Medicine, National Cheng Kung University, Tainan, Taiwan
30	Exercise combined with low-level GABAA receptor inhibition modulates the expression of neurotrophinsin the cerebellum	Hiroshi Maejima	Department of Rehabilitation Science, Faculty of Health Sciences, Hokkaido University, Japan
31	Treadmill exercise beneficially contributes to gene expressions relating to synaptic and axonal plasticity in the motor cortex after ischemic stroke in rats	Takahiro Inoue *Travel Award	Graduate School of Health Sciences, Hokkaido University
32	The effects of treadmill exercise in acute stage after stroke on memory function in a novel rat model of vascular dementia	Naoyuki Himi	Dept Physiol2, Kawasaki Med Sch, Okayama, Japan
33	Modulatory effects of serotonin on the contrast sensitivity of rats and the visual responses of V1 neurons corresponding to the performance of a visual detection task	Akinori Sato	Laboratory of Brain Information Science in Sports, Grad Sch Frontier Biosci, Osaka Univ, Osaka, Japan
34	Effect of endurance training with and without Magnolia-officinalis extract on nesfatin-1, glucose, glycogen, and ATP levels of hypothalamus and plasma in male rats	Abbas Ghanbari Niaki	Department of Exercise Physiology Faculty of Physical Education, University of Mazandaran
35	Role of the amygdala and claustrum in cardiovascular control during high-intensity treadmill exercise in rats	Ko Yamanaka	Department of Physiology, Health and Sports Science, Juntendo University, Japan
36	Specific changing patterns of arterial pressure during high intensity of treadmill exercise in rats	Kei Tsukioka	Grduate School of Health and Sports Science, Juntendo University,
37	Vitamin B1 analog decreases sleep periods and increases physical activity in rats	François Grenier	Exercise Biochem. & Neuroendocrinol., Univ. Tsukuba, Ibaraki, Japan
38	Hippocampal leptin mediates synergistic benefits of mild exercise by an antioxidant on neurogenesis and memory	Jang Soo Yook *Travel Award	Center for Functional Connectomics, Korea Institute of Science and Technology, Seoul, Korea
39	Mild exercise activates astrocyte-neuron lactate shuttle in the hippocampus: A role of dopamine	Takashi Matsui *Travel Award	Sport Neuroscience Division, ARIHHP, University of Tsukuba, Japan
40	Mild exercise regimen from pre-diabetic stage prevents the onset of type 2 diabetes and its related hippocampal memory dysfunction	Subrina Jesmin	Laboratory of Exercise Biochemistry and Neuroendocrinology, Faculty of Health and Sport Sciences, University of Tsukuba, Japan
41	Mild, rather than intense, exercise during adolescence attenuates abnormal behavior in prenatal phencyclidine-treated mice	Hikaru Koizumi *Travel Award	Laboratory of Exercise Biochemistry and Neuroendocrinology, Faculty of Health and Sport Sciences, University of Tsukuba, Japan
42	Running exercise-induced stress response is cooperatively regulated by hypothalamus AVP and CRH: anatomical and pharmacological approaches	Kanako Takahashi *Travel Award	Faculty of Health and Sport Sciences, University of Tsukuba, Japan
43	The establishment of the rat model for investigating the roles of exercise-increased blood BDNF	Moe Yamashita	Faculty of Health & Sport Sciences, University of Tsukuba, Japan
44	Hippocampal glycogen loading with exhaustive exercise enhances hippocampus-dependent learning and memory: A short-term sports conditioning targeting memory function	Mariko Soya *Travel Award	Department of Psychiatry, Center for Psychiatric Neurosciences, Lausanne University Hospital, Lausanne, Switzerland
45	Withdrawn		
46	Effects of inosine monophosphate and exercise on nNOS-related protein expression in dorsal and ventral hippocampus	Yuki Tomiga	Fukuoka University
47	Enriched environment does not increase ambulatory physical activity in mice: involvement of social interaction	Daisuke Funabashi *Travel Award	Human Health Science, Tokyo Metropolitan University, Tokyo, Japan
48	Orexin receptor 1 is necessary to evoke voluntary wheel running behavior through regulating monoaminergic neurons	Shingo Soya	International Institute of Integrative Sleep Medicine (WPI-IIIS), Tsukuba university
49	Effects of Low Intensity Exercise on Amyloid Precursor Protein Production by Iron Metabolism in Alzheimer's Disease Mice	Dong Hun Choi *Travel Award	Exercise Biochemistry Laboratory, Korea National Sport University, Seoul, Korea

Ignacio Torres Aleman, Ph.D.
Cajal Institute, Madrid. Spain

Insulin peptides as mediators of neuroprotection by exercise

Beneficial actions of physical activity on brain function have been related to a variety of activity-dependent processes triggered by exercise such as enhanced blood perfusion, tissue remodeling (i.e.: neurogenesis and angiogenesis), improved oxidative defence, or even immune modulation. However, ever since the reported exercise-dependent increase in local growth factor production (i.e.: BDNF) more than 20 years ago, evidence of a key role of trophic factors in neuroprotection by exercise has become robust. The nature and sources (i.e.: local, blood, muscle) of trophic inputs to the brain during exercise are probably still not fully described, but an important origin for exercise-mediated trophic input are circulating hormones such as insulin-like growth factor I (IGF-I). During the last two decades we have been analyzing the role of IGF-I as a mediator of exercise neuroprotection and have found that participates in the striking variety of processes known to be positively affected by exercise. Stemming from the original observation that exercise increases brain uptake of serum IGF-I -resembling the capture by other target organs of IGF-I such as skeletal muscle, this growth factor has been shown to modulate new neuron formation, synaptic plasticity, anti-apoptotic and anti-oxidant defenses, inflammation, and even mood and reward. Due to its wide cytoprotective actions, the role of IGF-I in treatment and prevention of brain diseases by exercise seems logical, whereas its actions on higher brain functions (learning and memory, mood, attention..etc), already found in invertebrates, demands a better knowledge of the processes modulated by IGF-I at the molecular, cellular and system level. In this regard, our recent work in collaboration with other labs is showing an interaction of IGF-I with insulin in brain glucose handling, a cell-specific role of IGF-I in metaplasticity processes, and a direct modulatory role on orexinergic circuits impacting on mood and reward. Collectively, our observations provide molecular targets, cellular mediators, circuits, and specific brain skills affected by insulin-peptides. Hence, modulation of the activity of these hormones by exercise may explain part of its neuroprotective actions while its in-depth understanding will likely provide novel intervention targets in preventing and treating neurodegeneration.

Zsolt Radak, Ph.D.
University of Physical Education, Budapest, Hungary

The link between exercise, microbiome and Alzheimer Diseases

Exercise is documented to be beneficial to vertebrate organisms as it increases skeletal muscle, respiratory-cardiovascular fitness, cognitive functions, and extending health span. It is also known that exercise has powerful effects on microbiome. It has been reported that exercise-induced changes in microbiome has an impact on endurance capacity and neurogenesis. It is also known that regular physical exercise and nutritional intecrvention decreases both the incidence and symptoms intensity in Alzheimer Disease (AD) along with changes in microbiome including those in the gut, while direct link has not been established. We have examined if exercise-induced changes in gut microbiome have beneficial effect on cognitive functions using APP/PS1 mice. Results showed that when APP/PS1 mice subjected to exercise and probiotic treatments significantly over-performed controls in maze tests, while exercise, prebiotic alone and together decreases of beta-amyloid plaques, and increased microglia numbers around plaques. At molecular level improvement in cognitive functions was associated with increased expression 8-oxoguanine DNA glycosylase-1 (OOG1) in APP/PS1 mice. Microbiome data revealed that AD development is associated with leaky gut, which can be prevented by exercise training. Data also show that exercise training increases the levels of anti-inflammatory microorganism, such as bacteria that are involved in butyrogenesis. These data together show beneficial effects exercise and probiotic on cognitive functions in mouse model, which can be applied in benefit of human.

Chia-Liang Tsai, Ph.D.
National Cheng Kung University, Taiwan

Exercise types and neurocognitive performance in mild cognitive impairment

Alzheimer's disease (AD) is a common progressive neurodegenerative disorders. To date, no curative pharmacotherapy exists for AD. Therefore, strategies for prevention and/or delay AD disease onset or progression need to be examined and implemented. Better physical fitness levels induced by regular physical exercise has been recommended as an effective non-pharmacological intervention capable of modifing the neurocognitive disease. Since the individuals with family history of Alzheimer's disease and ApoE-4 (ADFH-ApoE-4) or mild cognitive impairment (MCI) have a higher risk of suffering dementia, the two groups will thus be discussed in this talk. Event-related potentials and event-related neural oscillatory are two electroencephalographic signals which are sensitive enough to identify elderly patients with early cognitive decline or disease progression to MCI and/or AD. Based on neuropsychological problems found in individuals with ADFH-ApoE-4/MCI and executive functioning deficits being associated with poorer physical fitness, the role of physical fitness in the relationship between MCI/ADFH-ApoE-4 and the neurophysiological performance explored using the ERPs and neural oscillatory will be introduced. Regular physical exercise is a promising nonpharmacological intervention to retard cognitive aging. Potential neurobiological mechanisms could be increased levels of peripheral exercise-induced exerkines, such as exercise-induced neuroprotective growth [e.g., brain-derived neurotrophic factor (BDNF) and insulin-like growth factor 1 (IGF-1)] and pro-angiogenic factors [e.g., vascular endothelial growth factor (VEGF) and fibroblast growth factor 2 (FGF-2)] and reduced levels of pro-inflammatory cytokines [e.g., tumor necrosis factor-α (TNFα), Interleukin-1-beta (IL-1β), IL-6, IL-8, and IL-15]. The effects of acute and chronic exercise (aerobic vs. resistance exercise modes) interventions on neurocognitive performance (i.e., behavior and ERPs) and neuroprotective (e.g., BDNF, IGF-1, VEGF, and FGF-2) and inflammatory (e.g., TNF-α, IL-1β, IL-6, IL-8, and IL-15) biomarkers in older individuals with MCI will be stated. In addition, whether the two exercise modes produce divergent effects on these molecular biomarkers and neurocognitive performance in them will also be explored.

Session 4: Sports Performance and Neurorehabilitation

Naznin Virji-Babul, Ph.D.
University of British Columbia, Canada

Brain recovery from sports related concussion: Is there a new normal?

Children and youth are at a greater risk of concussions than adults, and once injured, take longer to recover. The risk of a repeat concussion during recovery is also significantly higher, with subsequent injuries further complicating and prolonging recovery. The increased incidence of sports-related concussion in youth and the potentially serious long-term negative impact on their developing brains has enormous repercussions. Little is still known about the subtle and widespread changes in the brain and the effect of concussion on the developing brain.

My team has been collecting resting state EEG in adolescents with sports related concussion for the past few years. Probing the brain during the "resting state" has emerged as a powerful tool to map the functional organization of the brain. Analysis of resting state functional networks has emerged as a significant new way to investigate brain connectivity between regions that are functionally linked in both healthy and disease/injury states. We were the first to report significant differences in functional networks in the frontal regions of the brain in adolescents with concussion using resting state fMRI. Importantly, this work showed that functional connectivity measures derived from resting-state EEG signals support the fMRI findings, showing clear concussion related changes in local networks in the dorsolateral prefrontal cortex that subserve cognitive function in adolescents. This work established that resting state EEG signals provide a simple, low-cost, non-invasive and direct measure of brain connectivity that differentiates healthy controls from individuals with brain injury.

Our longitudinal data show that the functional connectivity of the concussed adolescents typically does not return to normal even after one-year post-injury. This may be due to continued neural trauma, reorganization of brain connectivity, or both. In this talk I will discuss our findings showing disturbed brain dynamics in adolescents who have experienced a sport related concussion. I will then present pilot data on interventions directed at facilitating brain recovery using several different methods such as physical exercise and transcranial direct current stimulation (tDCS).

Kimitake Nakazawa, Ph.D.
The University of Tokyo, Department of Integrated Sciences, Japan

Paralympic brain -compensation and reorganization in human brain-

One of the primary goals of basic neurorehabilitation studies is to figure out the underlying neural mechanisms in the reorganization of central nervous system after rehabilitation interventions. From this perspective the athletes with disabilities, such as Paralympic athletes would be the attractive research model, since they can show the use-dependent plastic changes in CNS after the long-term intervention of intensive physical training.

We have recently found that the brains of Paralympic athletes are reorganized uniquely in such a manner dependent on disability types and athletic-specific training. Factors playing the major roles in the reorganization are most probably use-dependent plasticity and disability-specific compensations.

In this presentation our current data obtained from the following Paralympic athletes with various types of disabilities who have been engaged in different sports will be introduced.

A) A long jumper and a high jumper with below knee amputee
Both para-athletes showed bilateral activation of the leg motor area when they were producing knee joint torque during fMRI recording, while no such bilateral motor area activation was observed for the non-amputated leg. The transcranial magnetic stimulation (TMS) study for the high jumper confirmed that the ipsilateral corticiospinal tract of the amputated side knee extensor has the higher excitability and is recruited when he produces knee extension torque to move the prosthesis.

B) The archer with congenital upper-limb loss
The highly skilled lower leg function and large expansion of toe motor area in the primary motor cortex were observed with both fMRI and TMS experiments.

C) Power lifters with spinal cord injury
The specifically improved upper-limb motor function assessed with the isometric grip force stability test after spinal cord injuries was found. The subsequent experiment showed that the higher stability in grip force was common in persons with complete spinal cord injury regardless of sports participation, although there is still possibility that the higher force stability would be further improved in power lifters.

D) A swimmer with cerebral palsy
Electromyographic (EMG) activity and motion recording were performed during swimming for the former Paralympic gold medalist. The EMG activities and motion analysis showed that she performed well-coordinated dynamic swimming movement without spasticity in water, whereas on land she showed a typical hemiplegic type of posture and gait pattern with spastic elbow flexor activity. Although the MRI image of her brain revealed a large lesion in her left sensory-motor area, the remarkable brain reorganization was found to occur with the TMS experiment. The neural mechanisms enabling dynamic swim motion in water will be presented with the current knowledge regarding interaction between autonomic nervous activity and motor system, which can be applied to neurorehabilitation for patients after damages to the CNS such as stroke and spinal cord injury.

In the overall discussion the hypothetical neural mechanisms inducing reorganization of their brains will be presented with clinical implication in neurorehabilitation.

Henriette van Praag, Ph.D.
Brain Institute and Charles E. Schmidt College of Medicine, Florida Atlantic University, USA

Physical activity and muscle-brain crosstalk

Most neurons in the adult central nervous system are terminally differentiated and cannot be replaced when they die. However, small populations of new neurons are generated in the mature olfactory bulb and the hippocampus. In the adult hippocampus, newly born neurons originate from putative stem cells that exist in the subgranular zone of the dentate gyrus. The production, survival and functional integration of newborn hippocampal cells is strongly upregulated by voluntary wheel running in rodents. Enhanced adult hippocampal neurogenesis is correlated with enhanced synaptic plasticity, spatial navigation and pattern separation in rodents. A recent focus of our research is to understand the functional contribution of the different structures that provide direct input to new neurons in the adult brain, as well as the reorganization of new neuron networks by short-term (one week) or long-term (at least one month) exercise. For these studies we combined retroviral labeling with rabies virus as a retrograde tracer to delineate the circuitry of new neurons during their development in the adult brain. There is substantial upregulation of innervation of new neurons by the entorhinal cortex supporting the proposed role of new neurons in spatial and contextual memory processes. These studies show that newly born neurons are an integral part of local intra-hippocampal circuits as well as more distal (sub)cortical networks, and that these connections are subject to exercise-induced plasticity. Another aspect of our work is to investigate the peripheral triggers that may mediate exercise induced changes in the brain. We have observed that compounds that activate energy metabolism pathways in muscle, such as AMP-kinase agonist AICAR, can also benefit adult neurogenesis and memory function. Next, we set out to identify factors that may be released into circulation from muscle (myokines) that could influence brain function. Using proteomic analyses, we found that conditioned medium derived from skeletal muscle cell cultures treated with AICAR contains factors that can promote differentiation of neural stem cells in vitro. In particular, we identified lysosomal enzyme Cathepsin B (Ctsb) as a novel myokine. Analyses across species in mice, monkeys and humans showed that this factor upregulated in plasma with exercise. In humans, these changes correlated with improved fitness and hippocampus-dependent memory function. A recent focus of work is to determine whether myokine levels may be upregulated in aging subjects following an exercise intervention. Overall, our research evaluating the relationship between myokines, adult hippocampal neurogenesis, neurotrophin levels and memory function aims to further our understanding of effects of exercise on the brain.

Art F. Kramer, Ph.D.
Northeastern University, USA

Walking Towards a Healthy Brain and Mind

The presentation will focus on recent research from our laboratories that has examined the effects of exercise training interventions and physical activity on cognitive and brain health. I will discuss research that has examined changes in brain structure and/or function along with behavioral measures of cognition in interventions lasting from several weeks to 1 year. Study populations will include children, young and middle-aged individuals, and the elderly in addition to a variety of patient groups. Although the focus will be on training to improve cardiorespiratory fitness I will also briefly cover resistance training and well as multi-modal cognitive and exercise training program. Finally, the presentation will identify gaps in the literature and potential solutions.

Maria Llorens-Martin, Ph.D.
Centro de Biología Molecular "Severo Ochoa", CSIC-UAM, Madrid, Spain

Human adult neurogenesis as a mechanism of brain plasticity in physiology and pathology

Memory impairment in Alzheimer's Disease (AD) can be attributed to a significant decline in the functioning of the hippocampal formation, a brain region crucial for learning and memory. Moreover, this structure hosts one of the most unique phenomena of the adult mammalian brain, namely the addition of new neurons throughout lifetime. This process, named adult hippocampal neurogenesis (AHN), confers an unparalleled degree of plasticity to the entire hippocampal circuitry. While synapse loss and consequent death of mature neurons may be responsible for much of the hippocampal malfunctioning in AD, studies in mice suggest that the disease could also target AHN. Nonetheless, direct evidence of AHN in humans has remained elusive. Thus, determining whether new neurons are continuously incorporated to the human dentate gyrus (DG) during physiological and pathological aging is a crucial question with outstanding therapeutic potential. This talk will present solid evidence supporting the occurrence of continued neurogenesis in the human hippocampus of aged healthy subjects and AD patients. By combining human brain samples obtained under tightly controlled conditions and state-of-the-art tissue processing methods, we have identified thousands of immature neurons in the DG of neurologically healthy human subjects up to the ninth decade of life. These neurons exhibited variable degrees of maturation along differentiation stages of AHN. In sharp contrast, the number and maturation of these neurons progressively declined as AD advanced. These results demonstrate the robust persistence of AHN during both physiological and pathological aging in humans, and evidence impaired neurogenesis as a potentially relevant mechanism underlying memory deficits in AD that might be amenable to novel therapeutic strategies. In this regard, our studies in murine models of AD point to neuroprotective effects of lifestyle modifying factors. Solid evidence of the positive effects exerted by physical exercise and environmental enrichment on animal models of neurodegenerative diseases will be presented.

References:

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イメージング質量顕微鏡

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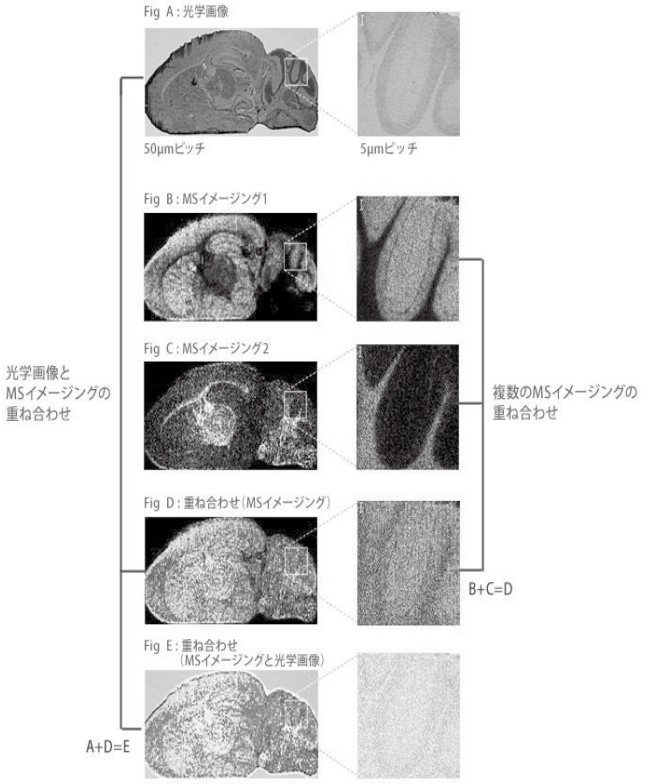
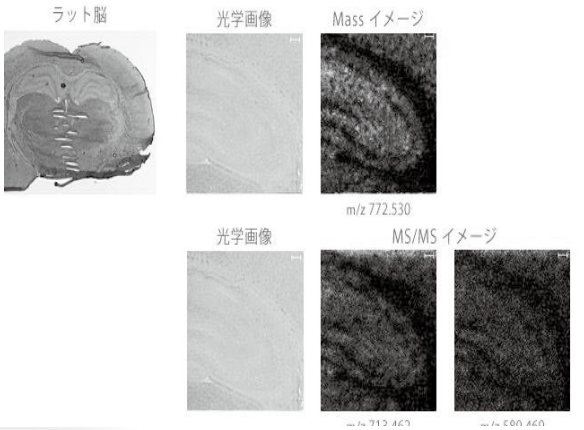
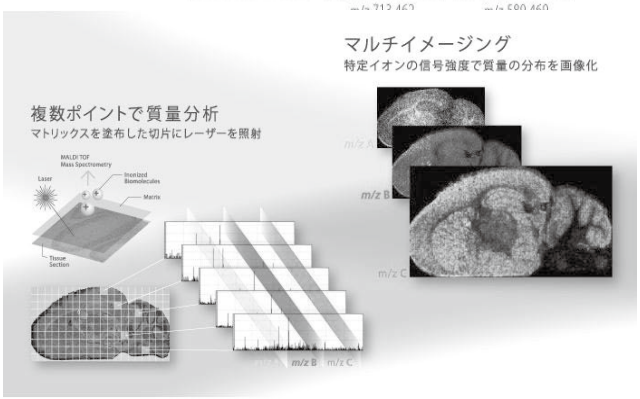
顕微鏡で観察された高解像度の形態画像と，質量分析計によって測定された分子の分布画像。異なる2つの原理から検出された画像を重ね合わせたイメージング質量分析画像を解析することで，研究はより速く，より正確になります。
最先端技術を凝縮したiMScopeが，ESI-MSとしても使用できる*iMScope TRIOとしてさらなる進化を遂げました。
*付け替えはフィールドエンジニアの作業となります。







イメージング質量分析では，試料の組織上の位置情報を残したまま，生体分子や代謝物を直接質量分析計で測定できます。測定から得られた位置情報と質量スペクトル中の特定イオンの信号強度によって，各種生体分子の二次元分布図を画像化します(MSイメージング)。このイメージング質量分析に特化した装置，これが，新たに開発された質量顕微鏡iMScopeです。光学顕微鏡と質量分析計がひとつになった，まったく新しいハイブリッド型の顕微鏡が，物質の構造解析を可能にし，あらゆる分野の研究の可能性を拡げます。

application

マウス小脳の脂質分布の可視化

疾患にはさまざまな物質が関与しています。イメージング質量分析では，指定した質量範囲で測定を行なうため，異なった質量を持つ物質それぞれの分布情報を一度のスキャニングで得ることができます。

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SLCの実験動物

マウス

●アウトブレッド
Slc：ddY
☆ IVCS
Slc：ICR
●インブレッド
DBA/1JJmsSlc(コラーゲン薬物誘導関節炎)
BALB/cCrSlc
C57BL/6NCrSlc・C57BL/6JJmsSlc
C3H/HeSlc
C3H/HeNSlc
C3H/HeJYokSlc
DBA/2CrSlc
NZW/NSlc
A/JJmsSlc
AKR/NSlc
NC/NgaSlc(薬物・アレルギー誘導アトピー性皮膚炎)
CBA/NSlc
129×1/SvJJmsSlc
●B10コンジェニック
C57BL/10SnSlc
B10.A/SgSnSlc・B10.BR/SgSnSlc
B10.D2/nSgSnSlc・B10.S/SgSlc
●ハイブリッド
B6D2F1/Slc(Slc:BDF1)
CB6F1/Slc(Slc:CBF1)
CD2F1/Slc(Slc:CDF1)
B6C3F1/Slc(Slc:B6C3F1)
※上記以外の系統については御相談ください。
●ヌードマウス(ミュータント系)
BALB/cSlc- *nu*(*Foxn1*^{nu})
KSN/Slc(*Foxn1*^{nu})
●疾患モデル
BXSB/MpJJmsSlc-*Yaa*（自己免疫疾患）
C3H/HeJJmsSlc-*lpr*（自己免疫疾患・*Fas*^{lpr}）
C57BL/6JSlc-*gld*（自己免疫疾患・*Fasl*^{gld}）
C57BL/6JJmsSlc-*lpr*（自己免疫疾患・*Fas*^{lpr}）
MRL/MpJmsSlc-*lpr*（自己免疫疾患・*Fas*^{lpr}）
NZB/NSlc（自己免疫疾患）
NZBWF1/Slc（自己免疫疾患）
WBB6F1/Kit-*Kit*^{W}/*Kit*$^{W-v}$/Slc(肥満細胞欠損貧血・*Kit*^{W}/*Kit*$^{W-v}$)
WBB6F1/Kit-*Kit*^{Sl}/*Kit*$^{Sl-d}$/Slc(肥満細胞欠損貧血・*Kit*^{Sl}/*Kit*$^{Sl-d}$)
NC/Nga(皮膚炎)
☆ Hos：HR-1（ヘアレスマウス）
☆ Hos：HRM-2(メラニン保有ヘアレスマウス)
★ SAMR1/TaSlc（非胸腺リンパ腫・SAM系対照動物）
★ SAMP1/SkuSlc（老化アミロイド症）
★ SAMP6/TaSlc（老年性骨粗鬆症）
★ SAMP8/TaSlc（学習・記憶障害）
★ SAMP10/TaSlc（脳萎縮を伴う学習・記憶障害）
AKITA/Slc（糖尿病）
☆ TSOD（2型糖尿病）
C57BL/6JHamSlc-*ob/ob*(肥満・2型糖尿病・*Lep*^{ob})
C57LKS/Jlar−+*Lepr*^{db}/+*Lepr*^{db}(肥満・2型糖尿病・*Lepr*^{db})
☆ NSY/Hos（2型糖尿病）
C57BL/6JHamSlc-*A*^{y}/＋(肥満)
HIGA/NscSlc（IgA腎症）
C.KOR/StmSlc-*Apoe*^{shl}(アポE欠損高脂血症・*Apoe*^{shl})
C.KOR/StmSlc-*Traf3ip2*^{adjm}(アトピー性皮膚炎マウス・*Traf3ip2*^{adjm})

ラット

●アウトブレッド
Slc：SD
Slc：Wistar
Slc：Wistar/ST
Hos: Donryu
☆ Iar：Wistar(Wistar-Imamichi)
☆ Iar：Long-Evans
☆ Iar:Copenhagen（前立腺腫瘍継代）
●インブレッド
F344/NSlc
WKAH/HkmSlc
BN/SsNSlc
DA/Slc(薬物誘導性関節炎)
LEW/SsNSlc(薬物誘導性関節炎)
●疾患モデル
★ SHR/Izm(高血圧)
★ SHRSP/Izm(脳卒中)
★ WKY/Izm(SHR/Izmのコントロール)
★ SHRSP5/Dmcr【NASHモデル【HFC飼料給餌】】
★ SHR/NDmcr-*cp/cp*（肥満・糖尿・高血圧・*Lepr*^{cp}）
★ SHRSP/Ezo(AD/HD)
★ SHRSP/IDmcr-*fa/fa*（肥満・高血圧・脳血管障害・*Lepr*^{fa}）
DIS/EisSlc(食塩感受性高血圧症)
DIR/EisSlc(食塩抵抗性)
Slc：Zucker-*fa/fa*（肥満・*Lepr*^{fa}）
☆ Hos：ZFDM-*Lepr*^{fa}(糖尿・肥満)
HWY/Slc（ヘアレスラット）
☆ Hos：OLETF(2型糖尿病)
☆ Hos：LETO(OLETFのコントロール)

モルモット

●アウトブレッド
Slc：Hartley

ウサギ

●アウトブレッド
Slc：JW/CSK
Slc：NZW

ハムスター

●アウトブレッド
Slc：Syrian
●疾患モデル
J2N-k（心筋症モデル）
J2N-n（J2N-kのコントロール）

スナネズミ

●インブレッド
MON/Jms/GbsSlc

無菌動物(ラット)

●ラット
●インブレッド
F344/NSlc(GF)

エンヴィーゴ(旧ハーランOEM生物動物)※維持

●アウトブレッドラット
RccHan®：WIST
※Hsd:Sprague Dawley®：SD®
●インブレッドマウス
CBA/CaOlaHsd
●免疫不全モデルマウス
C.B-17/IcrHsd-*Prkdc*^{scid}

遺伝子改変動物

●マウス
C57BL/6-Tg(CAG-EGFP)（グリーンマウス）
C57BL/6JJmsSlc-Tg(*gpt* delta)
●ヌードマウス
C57BL/6-BALB/*c-nu/nu*-EGFP(EGFP全身発現ヌードマウス)
●ラット
SD-Tg(CAG-EGFP)（グリーンラット）
F344/NSlc-Tg(*gpt* delta)
Slc:WistarHanover/Rcc-Tg(*gpt* delta)

その他(conventional動物)

●ビーグル犬
☆ 国内繁殖生産((一財)動物繁殖研究所)
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☆カニクイザル(輸入)
●ミニブタ
☆ 国内繁殖生産
((一財)日生研・NPO法人医用ミニブタ研究所)
●マイクロミニピッグ
☆国内繁殖生産(富士マイクラ(株))
●フェレット
自家繁殖生産(中伊豆支所)

★印は受託生産動物、☆印は仕入販売動物です。

受注生産動物

マウス

●疾患モデル
C3H/HeJJmsSlc-*gld*(自己免疫疾患動物・*Fasl*^{gld})
C57BL/6 JHamSlc-*bg/bg*(NK細胞活性低下)
CTS/Shi(免疫不全・白内障)
(NZW×BXSB)F1/Slc(紫斑症)

ラット

●インブレッド
ACI/NSlc
●疾患モデル
DahlS.Z-*Lepr*^{fa}/Slc
GK/Slc(2型糖尿病)
EHBR/EisSlc(高ビリルビン尿症)
PVG/SeaSlc
KDP(1型糖尿病・*Cblb*)
WBN/KobSlc(高血糖好発)
WBN/KobSlc-*fa/fa*(高血糖好発*Lepr*^{fa})
NAR/Slc(無アルブミン症)
NER(自発性強直・間代性けいれん発作発症)
DA/Slc-*bg/bg*(NK細胞機能低下)
SDR(矮小体駆症ラット)
OM/NSlc(栄養関係・腎障害)
FH/HamSlc(脳内セロトニン系の機能不全)
F344/NSlc-*Apc*^{m1Kyo}(大腸癌易誘発)
Gunn/Slc-*j/j*(高ビリルビン血症)
Slc：WsRC-*Ws/Ws*(肥満細胞欠損貧血-*c-kit*異常-*kit*$^{Ws/s}$)

モルモット

●アウトブレッド
Hos：Weiser-Meples(メラニン保有)
●インブレッド
Strain2/Slc
Strain13/Slc

ウサギ

●アウトブレッド
Slc：JWF-NIBS(ヘアレス)

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SLCの受託業務内容

受託試験

■医療機器の生物学的安全性試験(医療機器GLP省令適用)
○細胞毒性試験 ○細菌を用いる復帰突然変異試験 ○感作性試験
○刺激性／皮内反応試験
○急性全身毒性試験 ○発熱性物質試験 ○埋植試験
○血液適合性試験(溶血毒性試験) ○遺伝毒性試験(Ames試験)
■医薬品・医療機器の規格基準(GMP、QMS)に基づく試験
○細胞毒性試験 ○皮内反応試験 ○急性全身毒性試験
○発熱性物質試験 ○埋植試験 ○溶血性試験 ○抗原性試験
○局所刺激性試験(膝関節腔内)
■化合物等の安全性試験(非GLP)
○単回投与毒性試験 ○反復投与毒性試験 ○局所刺激性試験(皮膚・眼粘膜)
○抗原性試験(ASA、PCA) ○皮膚感作性試験
○細菌を用いる復帰突然変異試験 ○マウスを用いる小核試験
■再生医療用細胞・組織の安全性試験(非GLP)
○ヌードマウスあるいはSCIDマウスを用いた造腫瘍性試験
■マウス・ラット・ウサギ・フェレット・サルを用いた薬効薬理試験
○担がん動物を用いた薬効試験(抗がん剤スクーニングなど)
1)担がんヌードマウスあるいはSCIDマウスを用いた薬効試験
2)VX2担癌ウサギを用いた薬効試験
3)化学物質誘発大腸癌モデルマウスを用いた薬効試験
○自然発症モデル動物による試験
1)高血圧自然発症ラット(SHR・Dahl)を用いた試験
2)皮膚炎発症マウス(NC／Nga)を用いた試験
3)糖尿病モデルを用いた試験(マウス・ラット)
4)その他、自社自然発症モデルマウス・ラットを用いた試験
○外科的処置病態モデルによる試験(下記特殊動物供給を参照)
○薬物処置病態モデル動物を用いた試験
1)CCl_4急性肝炎ラット 2)STZ糖尿病マウス・ラット・サル
3)コラーゲン／プリスタン等誘発関節炎マウス・ラット
4)ブレオマイシン肺線維化マウス・ラット
○食餌性病態モデル動物による試験
1)高コレステロール食給与ウサギの試験
2)高脂肪食給与マウス、ラットの試験
3)その他、委託者様より提供される特殊飼料給与マウス・ラットの試験
○フェレットを用いた薬効薬理試験
1)嘔吐試験 2)感染試験
■薬物動態試験支援業務
○マウス、ラット、モルモット、ウサギ、サルへの化合物投与・経時的採血・検体採取業務
■抗体作製業務
○ポリクローナル抗体:モルモット、ウサギ ○モノクローナル抗体:マウス
■病理組織標本作製・鏡検

生殖・発生工学

■トランスジェニック動物(マウス、ラット)の作出
■ノックアウトマウス(キメラマウス)の作出
■ゲノム編集技術(CRISPR/Cas9,TALENなど)を用いた遺伝子改変動物(マウス、ラット)の作出
■マウス、ラットのSPF化(子宮切断術あるいは胚移殖)
■マウス、ラットの受託飼育・生産・供給
■凍結卵・凍結胚の供給(マウス・ラット・ウサギ)
その他SLCにて生産している動物の凍結胚(卵子)を供給いたします。
■受精卵(胚)の凍結・保存(マウス・ラット)
お客様より提供された動物から受精卵を採取し、凍結保存いたします。
■精子の凍結・保存(マウス)
お客様より提供された動物から精子を採取し、凍結保存いたします。
■凍結胚(マウス・ラット)・凍結精子(マウス)からの生体蘇生
■体外受精を用いたマウスの大量生産
■卵細胞質内精子注入法(ICSI)による受精卵・産子の作出
■スピードコンジェニックサービス

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特殊動物供給

■臓器摘出モデル(マウス、ラット、ハムスターに対応)
○下垂体 ○甲状腺(副甲状腺含む) ○副腎 ○精巣 ○卵巣 ○脾臓 ○胸腺 ○涙腺
○腎臓(1／2、5／6))(ラットのみ) ○肝臓部分
■痛覚過敏症モデル
○坐骨神経結紮による痛覚過敏症モデル(CCI)(ラット)
○脊髄神経結紮による痛覚過敏症モデル(SNL)(ラット)
○坐骨神経部分結紮による痛覚過敏症モデル(Seltzer)(マウス、ラット)
■カテーテル挿入動物
○頸静脈／動脈カニュレーション動物(マウス、ラット)
○大腿静脈／動脈カニュレーション(ラット)
○胆管カニュレーション動物(ラット)
○脊髄くも膜下カテーテル挿入動物(i.t)(ラット)
○脳室内投与ガイド装着動物(ラット)
○脳脊髄液採取用カテーテル挿入動物(ラット)
○門脈カニュレーション動物(ラット)
※2ヶ所同時カニュレーション動物の作製も承っております。
■特殊処置動物
○腎動脈7/8または6/8結紮モデル動物(ラット)
○頸部異所性心移植動物(ラット) ○坐骨神経切断動物(マウス、ラット、ハムスター)
○動脈内皮損傷動物(片側)(ラット) ○精管結紮動物(マウス、ラット)
○偽妊娠動物(マウス、ラット) ○テレメトリー移植(ラット)
○6-OHDA投与パーキンソン病モデル(マウス、ラット) ○心筋梗塞モデル(ラット)
○胆管結紮誘発肝線維症モデル(BDL)(マウス、ラット)
○中大脳動脈閉塞モデル(マウス、ラット)
○一側尿管結紮モデル(UUO)(マウス、ラット) ○協動筋切除(ラット)
■担癌動物の供給
○各種腫瘍移植(担癌)ヌードマウス・SCIDマウス
○VX2およびVX7担癌ウサギ ○大腸癌誘発KADラット
○化学物質誘発大腸癌モデルマウス
■薬物病態モデルの供給
○STZ糖尿病モデル(マウス、ラット、サル)
○コラーゲン／プリスタン等誘発関節炎マウス・ラット
○ブレオマイシン肺線維化モデル(マウス、ラット)
○CCl_4急性肝炎(ラット、マウス)
■食餌性病態モデルの供給
○高コレステロール食給与動物(マウス、ウサギ)
○高脂肪食給与動物(マウス、ラット)
○高脂肪・高コレステロール(HFC)飼料給与による
SHRSP5/Dmcrの非アルコール性脂肪性肝疾患モデル動物
○その他、委託者様より提供される特殊飼料給与
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ＭＩＣＥとは？

MICEとは、企業等の会議（Meeting）、企業等の行う報奨・研修旅行（インセンティブ旅行）（Incentive Travel）、国際機関・団体、学会等が行う国際会議（Convention）、展示会・見本市、イベント（Exhibition/Event）の頭文字のことであり、多くの集客交流が見込まれるビジネスイベントなどの総称です。MICEは、企業・産業活動や研究・学会活動等と関連している場合が多いため、一般的な観光とは性格を異にする部分が多いものです。このため、観光振興という文脈でのみ捉えるのではなく、MICEについて「人が集まる」という直接的な効果はもちろん、人の集積や交流から派生する付加価値や大局的な意義についての認識を高める必要があります。
具体的には、以下に掲げる３つの主要な効果が考えられます。

［1］ビジネス・イノベーションの機会の創造

MICE開催を通じて世界から企業や学会の主要メンバーが我が国に集うことは、我が国の関係者と海外の関係者のネットワークを構築し、新しいビジネスやイノベーションの機会を呼び込むことにつながります。

［2］地域への経済効果

MICE開催MICE開催を通じて世界から企業や学会の主要メンバーが我が国に集うことは、我が国の関係者と海外の関係者のネットワークを構築し、新しいビジネスやイノベーションの機会を呼び込むことにつながります。を通じた主催者、参加者、出展者等の消費支出や関連の事業支出は、MICE開催地域を中心に大きな経済波及効果を生み出します。MICEは会議開催、宿泊、飲食、観光等の経済・消費活動の裾野が広く、また滞在期間が比較的長いと言われており、一般的な観光客以上に周辺地域への経済効果を生み出すことが期待されます。

［3］国・都市の競争力向上

国際会議等のMICE開催を通じた国際・国内相互の人や情報の流通、ネットワークの構築、集客力などはビジネスや研究環境の向上につながり、都市の競争力、ひいては、国の競争力向上につながります。海外の多くの国・都市が、国・都市の経済戦略の中で、その達成手段の一つとし国際会議等のMICE開催を通じた国際・国内相互の人や情報の流通、ネットワークの構築、集客力などはビジネスや研究環境の向上につながり、都市の競争力、ひいては、国の競争力向上につながります。海外の多くの国・都市が、国・都市の経済戦略の中で、その達成手段の一つとしてMICEを位置付け、戦略分野／成長分野における産業振興、イノベーション創出のためのツールとして国際会議や見本市を活用しており、我が国においても、MICEを国・都市競争力向上のツールとして認識し、活用することが重要です。てMICEを位置付け、戦略分野／成長分野における産業振興、イノベーション創出のためのツールとして国際会議や見本国際会議等のMICE開催を通じた国際・国内相互の人や情報の流通、ネットワークの構築、集客力などはビジネスや研究環境の向上につながり、都市の競争力、ひいては、国の競争力向上につながります。海外の多くの国・都市が、国・都市の経済戦略の中で、その達成手段の一つとしてMICEを位置付け、戦略分野／成長分野における産業振興、イノベーション創出のためのツールとして国際会議や見本市を活用しており、我が国においても、MICEを国・都市競争力向上のツールとして認識し、活用することが重要です。市を活用しており、我が国においても、MICEを国・都市競争力向上のツールとして認識し、活用することが重要です。

一般社団法人 日本旅行業協会

観光庁長官登録旅行業第2032号
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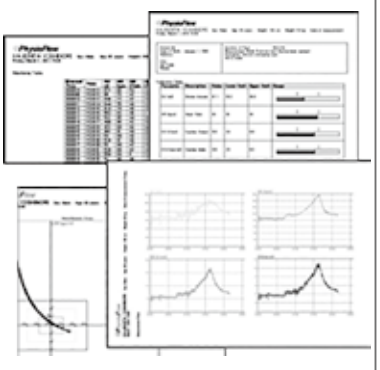

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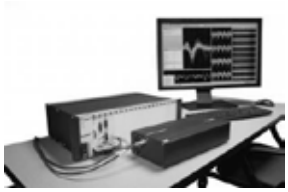

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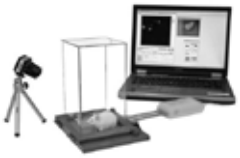

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～歩行再獲得のための理論と臨床～　第2版

著　中澤公孝（東京大学大学院総合文化研究科 教授）

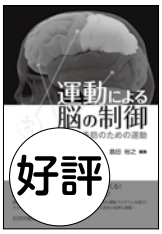
B5判・192頁/定価（本体2,700円+税）978-4-7644-0073-3

◆本書は，初版から継続して，歩行機能の再獲得をめざしたニューロリハビリテーションの理論的基盤，つまり中枢神経の可塑性とヒトの直立二足歩行に関する神経科学，その最新の知見をまとめています．

◆本改訂では，理論では，筋シナジーの詳細な解説，ニューロモジュレーションの今後の応用について，新たな章を設けて書き加えました．臨床では，従来のトレーニングに加え，ノルディックウォーク，水中ポールウォーキングの効果，外骨格型歩行装具についても言及しました．本書が歩行の再獲得とニューロリハビリテーションの研究・臨床の現場で，効果をあげるための論拠となることを意図しています．本書が読者の皆様の知的好奇心に応えるものであれば幸いです．

章目次

運動による脳の制御　認知症予防のための運動



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◆本書では，運動に着目した認知症予防の理解のため，「脳」「認知症」「運動」の関係を詳述するとともに，国内外の介入研究の結果を網羅し，わかりやすく解説しています．認知症予防の実践に役立つよう，具体的な評価方法や，認知機能を高める効果的な運動プログラムについても紹介しています．

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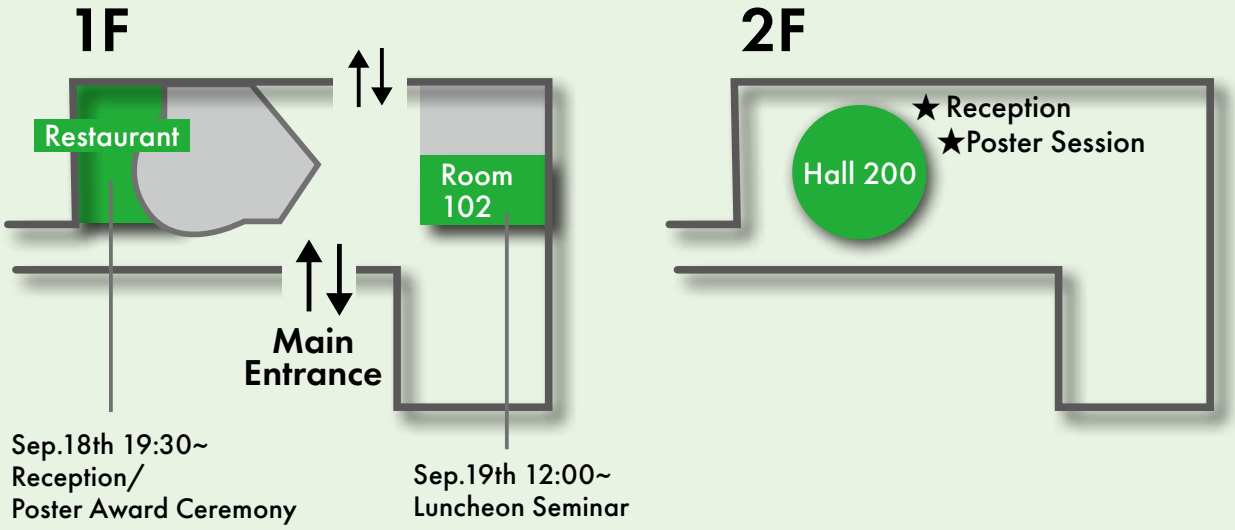

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