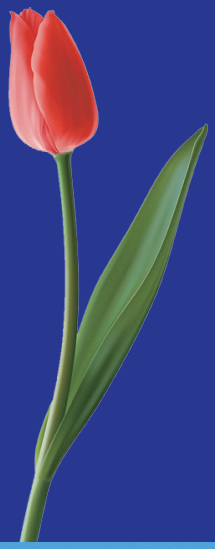
The 24th “Takamatsu” International Symposium for PD & MD in TOKYO



Dates : February 1 (Sat)・2 (Sun), 2025

Venue : Tokyo International Exchange Center
Tokyo Academic Park, 2-2-1 Aomi, Koto-ku, Tokyo, Japan

The 24th “Takamatsu” International Symposium for PD & MD in TOKYO

Date

2025. 2. 1（Sat）・ 2. 2（Sun）

Venue

Tokyo International Exchange Center
Tokyo Academic Park, 2-2-1 Aomi, Koto-ku, Tokyo, Japan

Secretariat

Department of Neurology, Juntendo University School of Medicine

Venue：
Tokyo International Exchange Center
Tokyo Academic Park, 2-2-1 Aomi, Koto-ku, Tokyo 135-8630 Japan

会場：
東京国際交流館内　国際交流会議場
〒135-8630 東京都江東区青海2-2-1

Secretariat:
Department of Neurology, Juntendo University School of Medicine
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E-mail：ipdstjimu@gmail.com

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順天堂大学医学部神経学講座
〒113-8421 東京都文京区本郷2-1-1
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Welcome to The 24th “Takamatsu” International Symposium for PD and MD in TOKYO

Welcome message from Chairpersons.

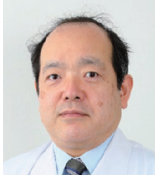
The 24th “Takamatsu” International Symposium for PD & MD in TOKYO will be held from Saturday, February 1st to Sunday, February 2nd, 2025, with the theme of “A New Era of Movement Disorder Treatment.” The event will be held at the Tokyo International Exchange Center（Koto-ku, Tokyo）.

We are once again dedicating ourselves to organizing a highly meaningful symposium for all participants, drawing upon the most up-to-date advancements in the research and treatment of Parkinson's disease. This year, we are honored to welcome distinguished researchers from both Japan and around the globe, whose contributions continue to shape the future of this field.

As this marks my final year as an active professor, I sincerely hope that this symposium will serve as a platform for vibrant presentations and stimulating discussions, fostering new insights and collaborations.

We eagerly anticipate your participation and look forward to welcoming you to the venue.

Nobutaka Hattori, MD, PhD, FANA
Chair



Atsushi Takeda, MD, PhD
Co-Chair

The Organizing Committee

Chair

Nobutaka Hattori, MD, PhD
Department of Neurology, Juntendo University School of Medicine, Tokyo, Japan

Co-Chair

Atsushi Takeda, MD, PhD
National Hospital Organization, Sendai Nishitaga Hospital, Miyagi, Japan

Advisor

Mitsutoshi Yamamoto, MD, PhD
Takamatsu Neurology Clinic, Kagawa, Japan

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Neurological Center, Asanogawa General Hospital, Ishikawa, Japan

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Taku Hatano, MD, PhD
Department of Neurology, Juntendo University School of Medicine, Tokyo, Japan

Program

Theme: The new era of treatment and diagnosis for Movement Disorders

1st Day：1 Feb 2025 (Saturday)

9：20-9：30

Opening Remarks Nobutaka Hattori, Mitsutoshi Yamamoto

9：30-11：00

Symposium 1 Pathophysiology of Parkinson's disease and related disorders

Chair：Genjiro Hirose, Mitsutoshi Yamamoto

1. Disease modification in Parkinson disease: new developments
   Speaker：Eduardo Tolosa
2. What is Parkinson's Disease – are we ready for a Biological Definition
   Speaker：Werner Poewe
3. Proposed Biological Definitions of Parkinson's Disease Confuse Understanding Without Delivering Meaningful Advances
   Speaker：Francisco Cardoso

11：00-11：10 **Short Break**

11：10-12：10

Symposium 2 Management of advanced Parkinson's disease Eisai Co., Ltd.

Chair：Tatsushi Toda, Tadashi Ichikawa

1. Cognitive and psychiatric issue
   Speaker：Hirohisa Watanabe
2. Motor Fluctuations and Continuous Dopaminergic Stimulation
   Speaker：Wataru Sako

12：10-12：50 **Lunch Break**

12：50-13：50

Premium Seminar Treating and diagnosing for atipical Parkinsonism

Chair：Kazunori Ito, Satoshi Orimo

1. Exploring Lewy body pathology from genetic Parkinson's disease
   Speaker：Daisuke Taniguchi
2. Is neuropathology the gold standard for MSA diagnosis ?
   Speaker：Yasuo Miki

13：50-14：50

Symposium 3 Development of new treatment for Parkinson's disease

Chair：Masahiko Tomiyama, Masahisa Katsuno

1. Therapeutic strategies based on antisence oligonucleotides
   Speaker：Yasuyoshi Kimura
2. Cell therapy for Parkinson's disease with pluripotent stem cells
   Speaker：Asuka Morizane

14：50-15：00 **Coffee Break**

15：00-16：00

Symposium 4 Investigating Parkinson's disease and related disorders

Chair：Ritsuko Hanajima, Noriko Nishikawa

1. Mutimodal imaging in Movement disorders
   Speaker：Hironobu Endo

2. Advantages and Drawbacks in Animal Models of Prodromal PD
Speaker：Hodaka Yamakado

16：00-17：00
Evening Seminar　New technologies for Movement disorders
EA Pharma Co., Ltd.
Chair：Yoshikazu Ugawa, Shinji Saiki
1. New Technologies for Movement Disorders; Adaptive Deep Brain Stimulation
Speaker：Genko Oyama
2. Artificial Intelligence and Machine Learning for Movement Disorders
Speaker：Shunsuke Kobayashi

17：00-17：10　**Short Break**

17：10-17：50
Special Seminar　ONO PHARMACEUTICAL CO., LTD.
Chair：Nobutaka Hattori
1. Therapies for Parkinson's disease – Present Status and Future Perspectives –
Speaker：Ryosuke Takahashi

17：50-18：20
Video Presentation Lecture
Chair：Nobutaka Hattori
Speaker：Eduardo Tolosa, Werner Poewe, Francisco Cardoso

18：20-18：30
Closing Remark　Nobutaka Hattori

18：30-19：30
Social Gathering

2nd Day：2 Feb 2025 (Sunday)　Japanese Session

10：00-10：40
パーキンソン病および関連運動障害疾患と正常圧水頭症　アッヴィ合同会社
Chair：大熊 泰之　Speaker：常深 泰司

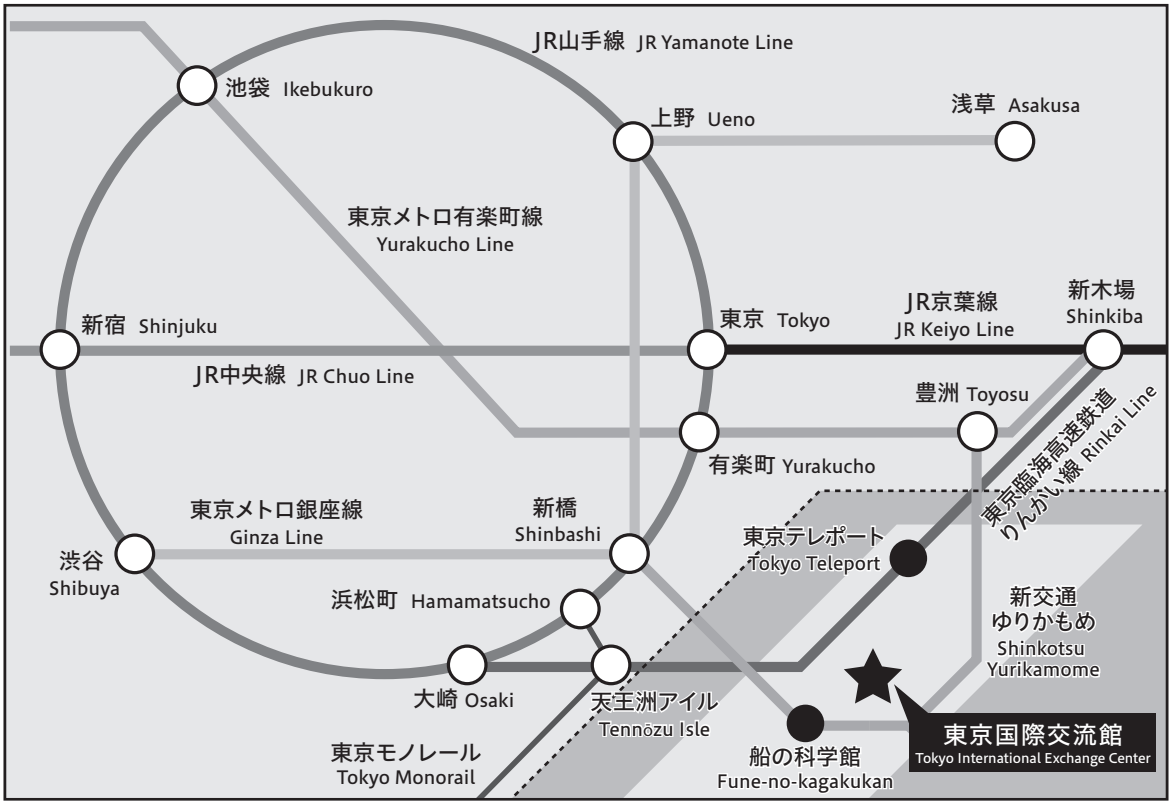
10：40-11：20
早期パーキンソン病の治療～再考～　武田薬品工業株式会社
Chair：坪井 義夫　Speaker：高橋 一司

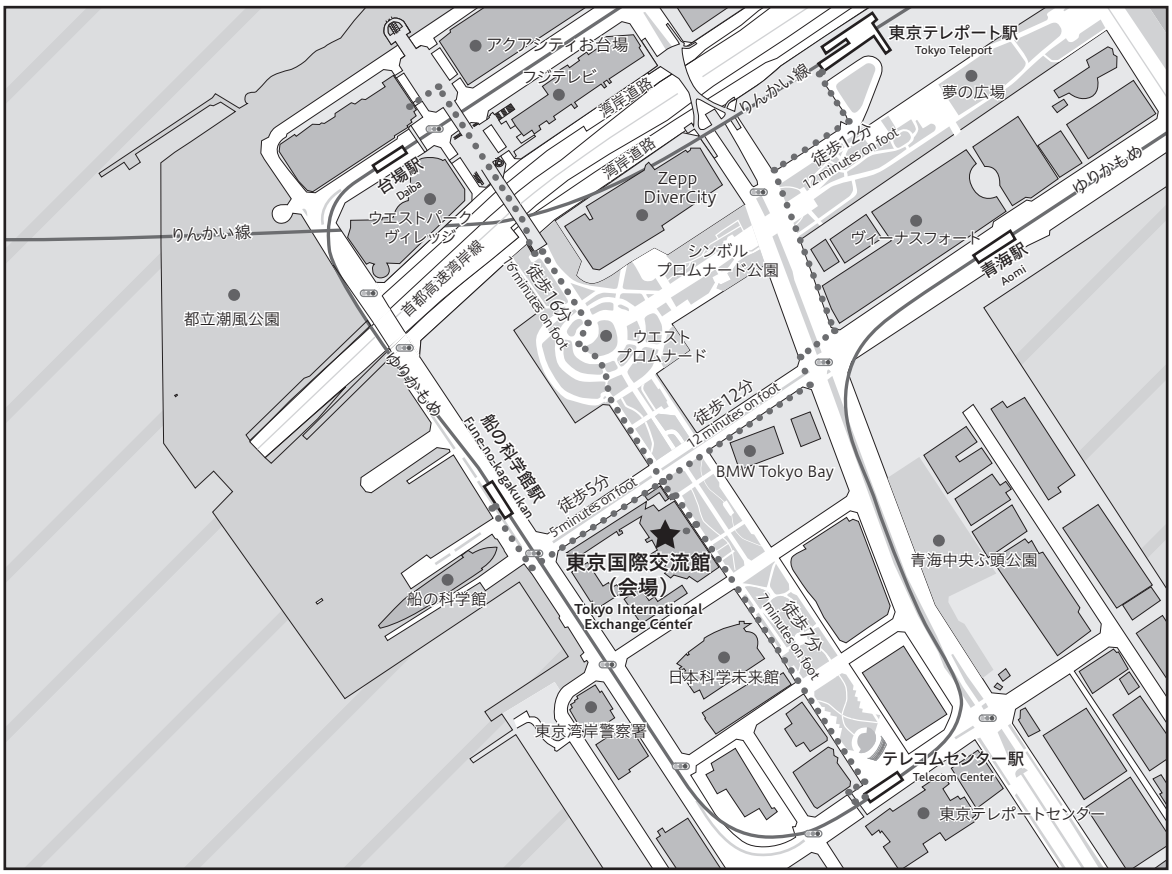
11：20-12：00
パーキンソン病の非運動症状アップデート　大塚製薬株式会社
Chair：下 泰司　Speaker：永山 寛

12：00-12：40
超超高齢社会におけるパーキンソン病診療　協和キリン株式会社
Chair：武田 篤　Speaker：関 守信

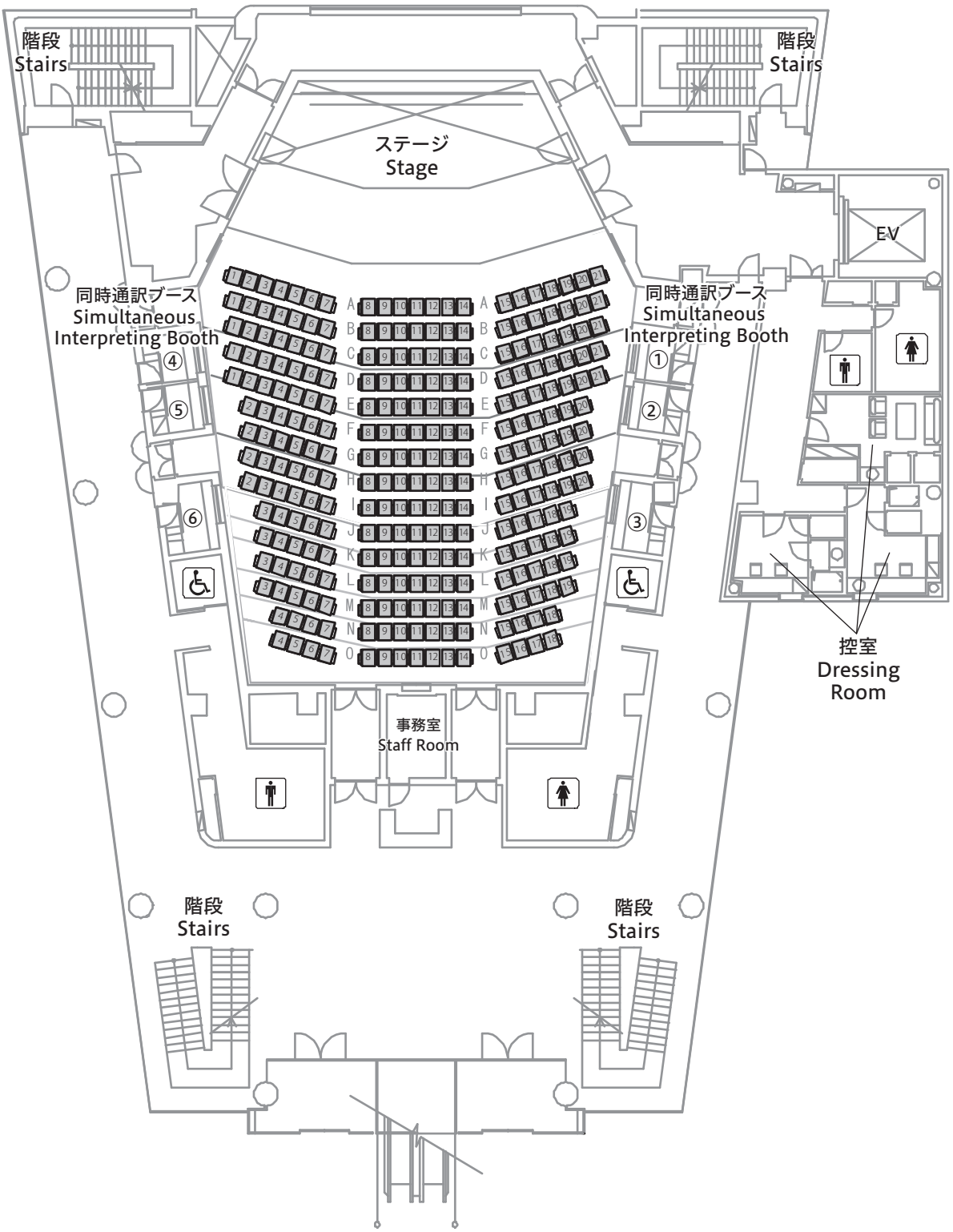
12：40-12：50
Closing Remark　服部信孝、山本光利

Access, Floor Map





1F　国際交流会議場（Room A）
International Conference Hall



Abstracts

1st Day
1 Feb 2025 (Saturday)

Symposium 1 Pathophysiology of Parkinson's disease and related disorders

Chair:
Genjiro Hirose
Neurological Center, Asanogawa General Hospital

Mitsutoshi Yamamoto
Takamatsu Neurology Clinic

1. Disease modification in Parkinson disease: new developments

Speaker:
Eduardo Tolosa, MD
University of Barcelona Emeritus Professor

Abstracts:

Slowing down disease progression is the biggest unmet need in PD. Disease modification trials have failed consistently in the last decades, despite preclinical studies that suggested the contrary. In my presentation I will highlight results on the latest clinical trials in disease modification and will review recent developments that strongly suggest that we can now identify subjects at high risk for developing PD and that successful trials in such premotor subjects may soon be possible. These include: 1. development of synuclein seeding amplification assays (SAA's); 2. proposal of a biological definition and staging of neuronal synuclein disease and 3. implementation of studies to recruit subjects at high risk to develop PD in the general population.

Biosketch:

Eduardo Tolosa obtained his MD degree from the University of Barcelona and received his neurological training at the University of Minnesota in the United States. He is a founding member and past President of the Movement Disorder Society and past President of the Spanish and of the European Neurological Society. In 1993 he founded the University of Barcelona Brain Bank, which he directed for 15 years, and in 2014 he received the American Academy of Neurology Movement Disorders Research Award.
Prof. Tolosa is the past Chairman of the Department of Neurology at the University of Barcelona Hospital and is currently Emeritus Professor at the University of Barcelona and Neurology Consultant at the Hospital Clinic Universitary.
Professor Tolosa's research interests include experimental therapeutics, etiology and pathophysiology of various Parkinson syndromes. He was involved in pioneering studies defining the mechanisms underlying levodopa-related motor fluctuations and the role of DAT SPECT in the diagnosis of Parkinson disease and his team was among the first in Europe to evaluate the efficacy of novel therapeutic strategies for movement disorders, such as botulinum toxin, subthalamic nucleus stimulation, subcutaneous dopamine agonist infusions and intraduodenal infusions of levodopa for Parkinson disease.
His research is currently focused on the prodromal phase of Parkinson disease and the study of tissue biomarkers in these preclinical stages.
He is involved in the Parkinson Progression Markers Initiative (PPMI)-a study on biomarkers and on the preclinical phase of Parkinson disease - supported by the Michael Fox Foundation and the Healthy Brain Aging project.

Key articles:

1. Lang AE, Siderowf AD, Macklin EA, Poewe W, Brooks DJ, Fernandez HH, Rascol O, Giladi N, Stocchi F, Tanner CM, Postuma RB, Simon DK, **Tolosa E**, Mollenhauer B, Cedarbaum JM, Fraser K, Xiao J, Evans KC, Graham DL, Sapir I, Inra J, Hutchison RM, Yang M, Fox T, Budd Haeberlein S, Dam T; SPARK Investigators.Trial of Cinpanemab in Early Parkinson's Disease. N Engl J Med. 2022 Aug 4; 387 (5): 408-420. doi: 10.1056/NEJMoa2203395

2. Garrido A, Fairfoul G, **Tolosa E**, Marti MJ, Ezquerra M, Green AJE. Brain and Cerebrospinal Fluid α-Synuclein Real-Time Quaking-Induced Conversion Identifies Lewy Body Pathology in LRRK2-PD. Mov Disord. 2023 Feb; 38(2): 333-338. doi: 10.1002/mds.29284. Epub 2022 Dec 5.
3. Lees A, **Tolosa E**, Stocchi F, Ferreira JJ, Rascol O, Antonini A, Poewe W. Optimizing levodopa therapy, when and how ? Perspectives on the importance of delivery and the potential for an early combination approach. Expert Rev Neurother. 2023 Jan; 23 (1): 15-24. doi: 10.1080/14737175.2023.2176220. Epub 2023 Feb 10.
4. Simuni T, Chahine LM, Poston K, Brumm M, Buracchio T, Campbell M, Chowdhury S, Coffey C, Concha-Marambio L, Dam T, DiBiaso P, Foroud T, Frasier M, Gochanour C, Jennings D, Kieburtz K, Kopil CM, Merchant K, Mollenhauer B, Montine T, Nudelman K, Pagano G, Seibyl J, Sherer T, Singleton A, Stephenson D, Stern M, Soto C, Tanner CM, **Tolosa E**, Weintraub D, Xiao Y, Siderowf A, Dunn B, Marek K. A biological definition of neuronal alpha-synuclein disease: towards an integrated staging system for research. Lancet Neurol. 2024 Feb; 23(2): 178-190. doi: 10.1016/S1474-4422(23)00405-2.
5. Cardoso F, **Tolosa E**. Lancet Neurol. Fluctuations in Parkinson's disease: progress and challenges. 2024 May; 23(5): 448-449. doi: 10.1016/S1474-4422(24)00116-9. Epub 2024 Mar 15.
6. **Tolosa E**, Vila M, Klein C, Rascol O. LRRK2 in Parkinson disease: challenges of clinical trials. Nat Rev Neurol. 2020 Feb; 16 (2): 97-107. doi: 10.1038/s41582-019-0301-2. Epub 2020 Jan 24. PMID: 31980808 Review.
7. **Tolosa E**, Garrido A, Scholz SW, Poewe W. Challenges in the diagnosis of Parkinson's disease. Lancet Neurol. 2021 May; 20(5): 385-397. doi: 10.1016/S1474-4422(21)00030-2.

2. What is Parkinson's Disease – are we ready for a Biological Definition

Speaker:
Werner Poewe, em. Prof. Dr.
Dept of Neurology, Medical University Innsbruck, Austria

Abstracts:

Current clinical diagnostic criteria for PD require the presence of cardinal motor features of the disease but there is ample evidence to suggest that the underlying pathological events may start many years prior to the full expression of PD motor symptoms. There is justifiable concern that the start of disease-modifying interventions after PD motor symptoms are fully established may correspond to a relatively late time point on the trajectory of progressive PD pathology and thus may have a reduced likelihood of success. Intervening at the earliest stages of the biological processes driving PD pathology would require diagnostic criteria that are anchored on reliable biomarkers of disease –potentially even enabling identification of pre-clinical disease in asymptomatic subjects. The concept of a 'biological' definition of disease independent of the presence of defining clinical features has been pioneered by the Alzheimer field by developing a framework of biomarkers for Abeta- and Tau-pathology, and imaging evidence for neurodegenerative brain changes. Similar efforts are now underway also for Parkinson's disease and may have far-reaching implications not only for the planning of clinical trials but also for future implementation of PD risk screening programmes and ultimately efforts aimed at disease prevention.

Two recent proposals have for the first time conceptualized biological disease anchors that would enable a 'preclinical' diagnosis in asymptomatic individuals. Despite important differences, both the 'SynNeurGe' and the 'Neuronal Synuclein Disease Integrated Staging System (NSD-ISS)' use a framework of positive α-synuclein SAA's, imaging evidence for neurodegeneration and certain genetic mutations for their diagnostic classification. These developments have far-reaching implications for clinical research and drug development and ultimately population-based risk-screening, but also pose important ethical and research challenges.

Both frameworks have not yet been validated in prospective studies regarding their predictive value for symptomatic disease in biologically defined sibjects without clinical symptoms and thus are only appropriate for research purposes at present. Once validated, however, a framework based on a biological definition of PD that enables early diagnosis would be invaluable in supporting R & D and improving the design of clinical trials in a number of ways – including pathogenic subtype-specific drug targeting and patient stratification.

Biosketch:

Professor Werner Poewe is emeritus Professor of Neurology in the Department of Neurology at the Medical University of Innsbruck in Austria. He completed a residency in clinical neurology and psychiatry at the University of Innsbruck and then a British Council research fellowship at University College and Middlesex Hospital Medical School in London. He previously served as a senior lecturer in the Department of Neurology at the University of Innsbruck and as a professor of neurology and acting director of the Department of Neurology at the Virchow Hospital of the Free University of Berlin in Germany before becoming director of the Department of Neurology at the Medical University of Innsbruck in 1995 – a position he held until 2019.

Professor Poewe has served as president of the International Parkinson and Movement Disorder Society (MDS), chair of the MDS European Section, president of the Austrian Society of Neurology, and president of the Austrian Parkinson's Disease Society. He is a corresponding member of the American Neurological Association and French Neurological Society and an honorary member of the German Neurological Society, Japanese Neurological Society, and MDS. He has received the Walther Birkmayer Prize from the Austrian Parkinson's Disease Society, the Dingebauer Prize from the German Neurological Society, and the Research Excellence Award from the Medical University and Leopold-Franzens University of Innsbruck.
Professor Poewe's main research interests are in the field of Parkinson's disease and movement disorders, with particular emphasis on the diagnosis, natural history and clinical trials in the fields of Parkinson's disease and atypical parkinsonism. He has authored or co-authored more than 800 original articles and reviews in the field of movement disorders with a total of more than 139.000 citations and an h-index of 172 (Google Scholar).
Professor Poewe is listed among top 1% of, highly cited researchers' in neuroscience (Clarivate Web of Science 2024).

Key articles:

1. **Poewe W**, Seppi K, Tanner CM, Halliday GM, Brundin P, Volkmann J, Schrag AE, Lang AE. Parkinson disease. Nat Rev Dis Primers. 2017 Mar 23; 3: 17013. doi: 10.1038/nrdp.2017.13. PMID: 28332488
2. Tolosa E, Garrido A, Scholz SW, **Poewe W**. Challenges in the diagnosis of Parkinson's disease. Lancet Neurol. 2021 May; 20 (5): 385-397. doi: 10.1016/S1474-4422 (21)00030-2. PMID: 33894193; PMCID: PMC8185633.
3. Höglinger GU, Adler CH, Berg D, Klein C, Outeiro TF, **Poewe W**, Postuma R, Stoessl AJ, Lang AE. A biological classification of Parkinson's disease: th SynNeurGe research diagnostic criteria. Lancet Neurol 2024; 23: 191-204
4. Mahlknecht, P, **Poewe, W**. Pharmacotherapy for Disease Modification in Early Parkinson's Disease: How Early Should We Be? J Parkinsons Dis. 2024

3. Proposed Biological Definitions of Parkinson's Disease Confuse Understanding Without Delivering Meaningful Advances

Speaker:
Francisco Cardoso, MD, PhD
Neurology Service, Internal Medicine Department, The Federal University of Minas Gerais, Brazil

Abstracts:

Classification of diseases based on proper biological markers leads to significant progress in their diagnosis and management. The best example of this is in oncology. This talk reflects on two proposals to classify and stage Parkinson's disease (PD) and related conditions based on the presence of aggregated alpha-synuclein (ASN) detected by alpha-synuclein aggregation assay (SAA). The authors highlight the significant shortcomings of the proposals: detection of ASN by SAA is not specific of PD or other well-established clinical conditions; they do not allow prediction of clinical course and prognosis; and they are not suitable as an endpoint for clinical trials. To move forward with proposals reflecting so many uncertainties and unknowns will just sow confusion and misunderstanding within the PD community.

Biosketch:

Francisco Cardoso MD PhD is a Professor at the Internal Medicine Department (Neurology Service) of the Federal University of Minas Gerais (UFMG) in Belo Horizonte, Brazil. He is the founder and current Director of the UFMG Movement Disorders Clinic. He did a Neurology Residency at his current institution and a Movement Disorders Fellowship at the Baylor College of Medicine under the supervision of Joseph Jankovic MD. He is the immediate Past-President of the International Parkinson's Disease and Movement Disorders Society (MDS). His main areas of research are choreas, particularly those of auto-immune origin; epidemiology of parkinsonism (he and his associates performed the first population-based study of prevalence of parkinsonism in Brazil); genetics of dystonia (one of the studies of his group led to the discovery of the DYT16 gene). He has authored more than 272 peer-reviewed papers and 120 chapters of books. He is honorary member of the Japanese Neurological Society and the Association of British Neurology. During the 5th Pan American Parkinson's Disease and Movement Disorders Congress held in Cartagena, Colombia (February 9-11, 2024), he received the MDS-PAS Leadership Award.

Key articles:

1. Cardoso F, Schmidt P. Proposed Biological Definitions of Parkinson's Disease Confuse Understanding Without Delivering Meaningful Advances. J Park Dis 2025 (in press)
2. Cardoso F, Goetz CG, Mestre TA, Sampaio C, Adler CH, Berg D, Bloem BR, Burn DJ, Fitts MS, Gasser T, Klein C, de Tijssen MAJ, Lang AE, Lim SY, Litvan I, Meissner WG, Mollenhauer B, Okubadejo N, Okun MS, Postuma RB, Svenningsson P, Tan LCS, Tsunemi T, Wahlstrom-Helgren S, Gershanik OS, Fung VSC, Trenkwalder C. A Statement of the MDS on Biological Definition, Staging, and Classification of Parkinson's Disease. Mov Disord. 2024; 39: 259-66.
3. Camargos S, Scholz S, Simón-Sánchez J, Paisán-Ruiz C, Lewis P, Hernandez D, Ding J, Gibbs JR, Cookson MR, Bras J, Guerreiro R, Oliveira CR, Lees A, Hardy J, Cardoso F, Singleton AB. DYT16, a novel young-onset dystonia-parkinsonism disorder: identification of a segregating mutation in the stress-response protein PRKRA. Lancet Neurol. 2008; 7(3): 207-15.
4. Barbosa MT, Caramelli P, Maia DP, Cunningham MC, Guerra HL, Lima-Costa MF, Cardoso F. Parkinsonism and Parkinson's disease in the elderly: a community-based survey in Brazil (the Bambuí study). Mov Disord. 2006 Jun; 21(6): 800-8.
5. Cardoso F, Seppi K, Mair KJ, Wenning GK, Poewe W. Seminar on choreas. Lancet Neurol. 2006; 5(7): 589-602.

Symposium 2 Management of advanced Parkinson's disease

(Eisai Co., Ltd.)

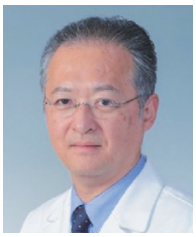
Chair:

Tatsushi Toda

Department of Neurology, University of Tokyo

Tadashi Ichikawa

Saitama Prefectural Rehabilitation Center



1. Cognitive and psychiatric issue

Speaker:

Hirohisa Watanabe, M.D., Ph.D.

Department of Neurology, Fujita Health University, School of Medicine

Abstracts:

Parkinson's disease (PD) is characterized not only by motor impairments but also by significant cognitive and psychiatric complications that severely affect patients' quality of life, especially in advanced stages. These non-motor symptoms are heterogeneous and dynamic: some patients maintain stable cognitive function, while others progress from mild cognitive impairment (PD-MCI) to dementia (PDD), influenced by factors such as age, education, and REM sleep behavior disorder. Recent multimodal imaging and neurobiological studies have revealed that cognitive dysfunction in PD involves multiple neurotransmitter systems and structural networks, including the medial temporal lobe and basal forebrain. Psychiatric symptoms, such as hallucinations and depression, stem from complex alterations in dopaminergic, serotonergic, and cholinergic pathways. Understanding these pathophysiological underpinnings is crucial for developing comprehensive management strategies. This lecture will highlight cutting-edge approaches to monitoring, diagnosing, and managing cognitive and psychiatric issues in advanced PD. We will discuss biomarkers, novel imaging techniques, and emerging interventions—ranging from pharmacotherapy to behavioral and technological solutions—aimed at personalizing care. By focusing on the interplay between cognitive decline and psychiatric symptoms, we seek to inform more targeted and integrative treatments that ultimately preserve patients' cognitive capacities, improve mental health, and enhance overall quality of life.

Biosketch:

Dr. Hirohisa Watanabe is currently a Professor and Chairperson of the Department of Neurology, Fujita Health University school of medicine. He graduated from Mie University School of Medicine in 1993, received his neurological training at Nagoya Daini Red Cross Hospital and Nagoya University Hospital, and was certified as a neurologist by the Japanese Society of Neurology. As a designated professor, he intensively investigated the relationship between brain network changes and aging and neurodegenerative diseases at the Brain and Mind Research Center, Nagoya University. He is currently a Delegate of the Japanese Society of Neurology, and Director of the Movement Disorders Society Japan, Japan Society of Neurovegetative Research, Japanese Society for Dementia Research, Japanese Society of Neurological Therapeutics, and Japanese Neuropsychiatric Association. His research aims to develop biomarkers for early diagnosis and treatment of Parkinson's disease, multiple system atrophy, and neurodegenerative dementia. His citation index is 8326 and h-index is 48 (Scopus, December 14, 2024).

Key articles:

1. Kawabata K, et al. Cognitive dysfunction in de novo Parkinson disease: Remitting vs. progressive cognitive impairment. ***Parkinsonism Relat Disord***. 2024; 120: 105984.
2. Mizutani Y, et al. Associations of Alzheimer's-related plasma biomarkers with cognitive decline in Parkinson's disease. ***J Neurol***. 2023; 270: 5461-5474.
3. Nishikawa N, et al. Idiopathic rapid eye movement sleep behavior disorder in Japan: An observational study. ***Parkinsonism Relat Disord***. 2022; 103: 129-135.
4. Kawabata K, et al. Cerebello-basal ganglia connectivity fingerprints related to motor/cognitive performance in Parkinson's disease. ***Parkinsonism Relat Disord***. 2020: 80; 21-27.
5. Ohdake R, et al. Individual changes in visual performance in non-demented Parkinson's disease patients: a 1-year follow-up study. ***J Neural Transm*** (Vienna). 2020; 127: 1387-1397.

(Eisai Co., Ltd.)

2. Motor Fluctuations and Continuous Dopaminergic Stimulation

Speaker:
Wataru Sako, MD, PhD
Department of Neurology, Juntendo University School of Medicine

Abstracts:

Motor fluctuations are a part of motor complications at the advanced stage of Parkinson's disease due to L-Dopa therapy. As the name indicated, motor fluctuations are comprised of two distinct components: on and off time including end of dose off, sudden off, delayed on, and dose failure, and these were troublesome symptoms for patients with advanced Parkinson's disease. High dose of L-Dopa is a risk factor for motor fluctuations, which is recommended to be avoided at an early stage; however, a sufficient dose of L-Dopa should be administered to maintain social position. The abnormalities in pre- and post-synapse cause motor complications in the striatum. That said, dopaminergic neuronal loss at the presynapse induced pulsatile dopaminergic stimulation, resulting in alteration in the activity of the postsynaptic neurons. To overcome this problem, we seek to have a therapy that produces continuous dopaminergic stimulation to resemble natural steady state even at the advanced stage of Parkinson's disease. There are several therapeutic options that can achieve continuous dopaminergic stimulation, including adjunctive and device aided therapies. In particular, device aided therapy is remarkably developing and a novel method has been devised to provide continuous dopaminergic stimulation one after another.

Biosketch:

Professional Training

1997-2003	M.D., Tokushima University
2003-2006	Residency: Internal Medicine and Neurology, Kitano Hospital
2006-2011	Ph.D. and Fellowship: Molecular biology and Movement Disorders, Tokushima University (Prof. Ryuji Kaji)
2011-2014	Postdoctoral Research Trainee: Neuroimaging, Center for Neurosciences, The Feinstein Institutes for Medical Research (Prof. David Eidelberg)

Academic Position

2014-2021	Assistant Professor, Tokushima University Graduate School of Biomedical Sciences
2021-present	Associate Professor, Juntendo University School of Medicine

Key articles:

1. Olanow et al., Mov Disord 2024
2. Sako et al., NPJ Parkinsons Dis 2023
3. Chou et al., Parkinsonism Relat Disord 2018
4. Aquino and Fox, Mov Disord 2015

Premium Seminar Treating and diagnosing for atipical Parkinsonism

Chair:
Kazunori Ito
IWAMIZAWA NEUROLOGY CLINIC

Satoshi Orimo
Kamiyoga Setagaya Street Clinic

1. Exploring Lewy body pathology from genetic Parkinson's disease

Speaker:
Daisuke Taniguchi, MD, PhD
Department of Neurology, Juntendo University Graduate School of Medicine

Abstracts:

It is well known that Parkinson's disease is a Lewy body disease in which aggregates of α-synuclein accumulate in both the central and peripheral nervous systems. On the other hand, while the pathology of genetic Parkinson's disease has been summarized in the literature, it is rare to observe the detailed pathological findings. Among genetic Parkinson's diseases, the type in which α-synuclein accumulates is particularly important for understanding the pathophysiology of Parkinson's disease. Our institute has been screening Parkinson's disease causative genes and risk genes. Therefore, we have a collection of postmortem brains from genetic Parkinson's disease. In this session, I will provide a detailed presentation of the pathological features of familial Parkinson's disease in which α-synuclein accumulates, such as PARK4 (SNCA duplication), PARK22 (CHCHD2 mutation), the GBA L444P variant, and the LRRK2 G2385R variant. I hope that presenting the differences in pathology between sporadic and genetic Parkinson's disease will enhance understanding of Lewy body disease.

Biosketch:

Daisuke Taniguchi is a neuropathologist and the person in charge of the Neuropathology Department at the Department of Neurology, Juntendo University. He specializes in Parkinson's disease and Parkinson's disease-related disorders, with additional expertise in muscle and nerve biopsies. His research focuses on elucidating the pathophysiology of 4-repeat tauopathies, and recently, he has reported on the pathological and biochemical differentiation of corticobasal degeneration and progressive supranuclear palsy.

Key articles:

1. Taniguchi D. et al. Legumain/asparaginyl endopeptidase-resistant tau fibril fold produces corticobasal degeneration-specific C-terminal tau fragment. Neurobiol Dis. 2024 Oct 15; 201: 106686.
2. Taniguchi D. et al. Neuromelanin imaging and midbrain volumetry in progressive supranuclear palsy and Parkinson's disease. Mov Disord. 2018 Sep; 33(9): 1488-1492.

2. Is neuropathology the gold standard for MSA diagnosis ?

Speaker:
Yasuo Miki, MD, PhD
Department of Neuropathology, Biomedical Research Center,
Hirosaki University Graduate School of Medicine

Abstracts:

Multiple system atrophy (MSA) is characterised by the presence of α-Synuclein-positive glial cytoplasmic inclusions (GCIs) and, less frequently, neuronal cytoplasmic inclusions (NCIs) within the central nervous system. Although the striatonigral and olivopontocerebellar regions are primarily affected in the brains of individuals with MSA, there is considerable variation in the distribution of the inclusions (GCIs and NCIs) and associated neuronal loss. This leads to a wide spectrum of clinical manifestations of MSA, with atypical MSA cases not encompassed by the second consensus diagnostic criteria for MSA (Gilman et al., Neurology 2008; 71: 670-9). Furthermore, individuals with other Parkinsonian disorders, such as Parkinson's disease and progressive supranuclear palsy, may present with clinical features suggestive of MSA. The considerable overlap in clinical symptoms among Parkinsonian disorders can make a clinical diagnosis of MSA challenging.

In this seminar, I will provide an overview of the clinical manifestations of MSA, and other Parkinsonian disorders as well as immune-mediated neurological disorders that may masquerade as MSA (MSA mimics). Furthermore, I will briefly review the potential of classical and novel biomarkers to facilitate an ante-mortem diagnosis of MSA.

Biosketch:

Dr Miki is a Japanese neuropathologist at the Department of Neuropathology, Hirosaki University Graduate School of Medicine, Japan.
Following his graduation from Hirosaki University School of Medicine in 2005, he held the position of registrar at Yodogawa Christian Hospital from 2005 to 2007. He then undertook neurological training at Hirosaki University Hospital and Aomori Prefectural Central Hospital until 2013. In 2011, Dr Miki also completed a PhD course under the supervision of Prof Koichi Wakabayashi. During the PhD course, he studied the formation processes of Lewy bodies and GCIs, the pathological hallmarks of Lewy body diseases and MSA, respectively; his doctoral thesis is 'Accumulation of histone deacetylase 6, an aggresome-related protein, is specific to Lewy bodies and glial cytoplasmic inclusions' (Neuropathology 2011; 31: 561-80). In 2013, Dr Miki was appointed as an assistant professor at the Department of Neuropathology, Hirosaki University Graduate School of Medicine. His research focused on the formation process of inclusion bodies in neurodegenerative diseases. However, he gradually shifted his research focus towards MSA. This led him to relocate to the University College London, Queen Square Brain Bank in the UK (2018 – 2020). He conducted several clinicopathological studies of MSA (Key articles 1-3) under the supervision of Prof Janice Holton and Prof Niall Quinn. He also contributed to the revision of the 3rd consensus criteria for the diagnosis of MSA as a member of the revision task force (Key article 4). Currently, Dr Miki is investigating how α-synuclein conformers affect neuronal functions in MSA, using human MSA cases and an adult-onset MSA mouse model (Key article 5 and further articles).

Key articles:

1. Miki Y, Foti SC, Asi YT et al., Improving diagnostic accuracy of multiple system atrophy: a clinicopathological study, Brain 2019; 142: 2813-27.
2. Miki Y, Foti SC, Hansen D et al., Hippocampal α-synuclein pathology correlates with memory impairment in multiple system atrophy, Brain 2020; 143: 1798-820.
3. Miki Y, Tsushima E, Foti SC et al., Identification of multiple system atrophy mimicking Parkinson's disease or progressive supranuclear palys, Brain 2021; 144: 1138-51.
4. Wenning GK, Stankovic I, Vignatelli L et al., The Movement Disorder Society Criteria for the Diagnosis of Multiple System Atrophy, Mov Disord 2022; 37: 1131-48.
5. Miki Y, Tanji K, Shinnai K et al., Pathological substrate of memory impairment in multiple system atrophy, Neuropathol Appl Neurobiol 2022; 48: e12844.

Symposium 3 Development of new treatment for Parkinson's disease

Chair:

Masahiko Tomiyama
Department of Neurology, Hirosaki University Graduate School of Medicine

Masahisa Katsuno
Department of Neurology, Nagoya University Graduate School of Medicine

1. Therapeutic strategies based on antisence oligonucleotides

Speaker:

Yasuyoshi Kimura, MD, PhD
Department of Neurology, Osaka University Graduate School of Medicine

Abstracts:

Antisense oligonucleotides (ASOs) are synthetic single-stranded short nucleic acids that bind complementary to target mRNAs and/or pre-mRNAs. Upon binding, they degrade the target mRNA by recruiting RNase-H or modulate splicing or stability of the target pre-mRNA. While the capacity of ASOs to suppress gene and protein expression was confirmed in the 1970s, recent years have witnessed remarkable advances in basic science and clinical applications. In the field of neurology, ASOs that modulate splicing of SMN2 and Dystrophin pre-mRNA were approved for spinal muscular atrophy and Duchenne muscular dystrophy, respectively. Gapmer-ASOs, which degrade transthyretin and SOD1 mRNA, were approved in the United State and Europa for familial amyloid neuropathy and familial amyotrophic lateral sclerosis caused by *SOD1* mutation, respectively. Many others are under development targeting neurodegenerative diseases, including alpha-synucleinopathies. Preclinical studies have shown beneficial effects of gapmer-ASOs targeting SNCA mRNA on animal models of Parkinson's disease (PD), and a phase 1/2 clinical trial of alpha-synulein suppressing ASO for multiple system atrophy is ongoing. ASOs targeting LRRK2 mRNA are also being developed for PD.

In this symposium, I will review therapeutic strategies based on ASOs for PD and alpha-synucleinopathies. We will also discuss new technologies that may improve the safety and stability/efficacy of ASOs, as well as drug delivery systems that can bring ASOs through the blood-brain barrier to the brain.

Biosketch:

Dr. Yasuyoshi Kimura is an assistant professor in the Department of Neurology, Osaka University Graduate School of Medicine. He received his M.D. from Osaka University in 2008. He completed a residency in clinical neurology at Higashiosaka City Medical Center and Osaka University Hospital. After obtaining his Ph.D. degree from Osaka University in 2016, he worked as an assistant professor in the Department of Pathology, Osaka University Graduate School of Medicine. From 2017 to 2019, he worked as a postdoctoral research fellow at the Institute for Cell Engineering, Johns Hopkins University School of Medicine, USA. He became a specially appointed assistant professor (from 2020) and as assistan professor (from 2021) in the Department of Neurology, Osaka University Graduate School of Medicine. His research interests include neurodegenerative disorders especially alpha-synucleinopathy and development of treatments.

Key articles:

1. Uehara T et al. Amido-bridged nucleic acid (AmNA)-modified antisense oligonucleotides targeting α-synuclein as a novel therapy for Parkinson's disease. Sci Rep 2019; 9: 7567
2. Alarcón-Arís, D et al. Anti-α-synuclein ASO delivered to monoamine neurons prevents α-synuclein accumulation in a Parkinson's disease-like mouse model and in monkeys. eBioMedicine 2020; 59: 102944
3. Cole TA et al. α-Synuclein antisense oligonucleotides as a disease-modifying therapy for Parkinson's disease. JCI insight 2021; 6: e135633
4. Sano T et al. Effects of local reduction of endogenous α-synuclein using antisense oligonucleotides on the fibril-induced propagation of pathology through the neural network in wild-type mice. Acta Neuropathol Commun 2024; 12: 75

Symposium 3 Development of new treatment for Parkinson's disease

2. Cell therapy for Parkinson's disease with pluripotent stem cells

Speaker:
Asuka Morizane, MD, PhD
Department of Regenerative Medicine, Kobe City Medical Center General Hospital

Abstracts:

Cell therapy for Parkinson's disease (PD) has been applied to several clinical trials in the world using human pluripotent stem cells (PSCs), namely embryonic stem cells (ESCs) or induced pluripotent stem cells (iPSCs). In Japan, a clinical trial with allogeneic iPSCs was conducted with seven subjects since 2018, Kyoto Trial (UMIN. 000033564). Before designing the clinical trials, various pre-clinical studies reported that the graft of PSC-derived dopaminergic progenitors into PD animal models survived and worked functionally.

The key elements for optimal results in cell therapy for PD can be divided into components called 'DISH': donor cells (D), immune control (I), surgery (S) and host (H). The donor cells need to be optimized in the context of purity, maturation stage, and viability. Immune rejection should be controlled. Proper surgery is required with minimal damage to the donor cells and the host's brain. Factors of the host, such as age, clinical stages of PD, genetic background, and chronic inflammation, will affect the results.

The first-generation stem cell therapy for PD has just started. Several issues should be further improved for efficacy and safety. It is expected that cell therapy will become a standard treatment option for PD.

Biosketch:

Education

2000-2004	Ph.D. Kyoto University Graduate School of Medicine
1990-1996	M.D. Tokyo Medical and Dental University

Positions

2021-	Director, Dep. of Regenerative Medicine, Kobe City Medical Center General Hospital
2019-2021	Junior Associate Professor, Dep of Clinical Application, CiRA, Kyoto University
2012-2021	Assistant Professor, Dep of Clinical Application, CiRA, Kyoto University
2008-2012	Postdoctoral researcher, CiRA and Institute for Frontier Medical Sciences, Kyoto Univ.
2006-2008	Postdoctoral fellow, Biomedical Center, Lund University, Sweden
2003-2006	Clinical staff, Department of Neurosurgery, Kobe City General Hospital

Key articles:

1. Morizane, A. Cell therapy for Parkinson's disease with induced pluripotent stem cells. Inflamm Regen 43, 16 (2023).
2. Morizane, A. & Takahashi, J. Evading the Immune System: Immune Modulation and Immune Matching in Cell Replacement Therapies for Parkinson's Disease. J Parkinsons Dis 11, S167–S172 (2021).
3. Doi, D. et al. Pre-clinical study of induced pluripotent stem cell-derived dopaminergic progenitor cells for Parkinson's disease. Nat Commun 11, 3369 (2020).
4. Morizane, A. et al. MHC matching improves engraftment of iPSC-derived neurons in non-human primates. Nat Commun 8, 385 (2017).
5. Kikuchi, T. et al. Human iPS cell-derived dopaminergic neurons function in a primate Parkinson's disease model. Nature 548, 592–596 (2017).

Symposium 4 Investigating Parkinson's disease and related disorders

Chair:

Ritsuko Hanajima
Department of Neurology, Tottori University School of Medicine

Noriko Nishikawa
Department of Neurology, Juntendo University School of Medicine

1. Mutimodal imaging in Movement disorders

Speaker:

Hironobu Endo, Ph.D., M.D.
National Institutes for Quantum Science and Technology

Abstracts:

Pathological protein aggregations, including tau and α-synuclein depositions, are hallmarks of neurodegenerative diseases, and recent neuroimaging advances have improved our understanding of their relation to movement disorders. We developed PET tracers to detect these protein pathologies *in vivo*, aiding clinical diagnostic workups (Tagai K, et al. *Neuron* 2021; Endo H, et al. *Neuron* 2024). However, localizing these pathologies alone is not necessarily sufficient to explain or predict clinical symptoms, highlighting the need for incorporating other imaging modalities to assess tau-triggered network disruptions in relation to individual manifestations.

We accordingly implemented a multimodal imaging strategy integrating PET imaging of pathological proteins, structural MRI, and diffusion tensor imaging for tissue connectivity. Specifically, comparisons of brain structures showing associations of tau depositions, atrophy, and tract disruptions with symptoms revealed that tau-induced neuronal deterioration arises from both local toxicity and network deficits. Furthermore, core neural circuits linking tau pathologies and specific symptoms were identified by combining tau PET imaging and standard human connectome data in the lesion network mapping paradigm.

Our research explores how diverse PET findings correlate with clinical symptoms while investigating tau-provoked structural and functional alterations in the brains of patients with movement disorders.

Biosketch:

Current Appointments

2023-present Senior Researcher, Sector for Neurodegenerative Disease Research, Brain Disorder Translational Research Group, Advanced Neuroimaging Center, Institute for Quantum Medical Science, National Institutes for Quantum Science and Technology

Education and Training (in chronological order):

Undergraduate/Doctoral

2003 B.S. in Laboratory Medicine, School of Allied Health Sciences, Kitasato University, Kanagawa, Japan
2008 M.D. in Medicine "Transfer (Bachelor's Program) in 2003", School of Medicine, Oita University, Oita, Japan
2018 Ph.D. in Neurology, Graduate School of Medicine, Kobe University, Kobe, Japan

Postdoctoral

2019-2020 Postdoctoral fellow, The Russell H. Morgan Department of Radiology and Radiological Science MR Research Division, Johns Hopkins University, Baltimore, MD, USA
2020-2023 Researcher (including postdoctoral fellow), Brain Disorder Translational Research Group, Department of Functional Brain Imaging Research, Institute for Quantum Medical Science, National Institutes for Quantum Science and Technology, Chiba, Japan

Awards, Honors

62nd Annual Meeting of the Japanese Society of Neurology, Best Free Paper Oral Presentation (Clinical Research) for JSN members, 2021
The 40th Annual Meeting of Japan Society for Dementia Research, Society Encouragement Award (Clinical Research), 2021

Key articles:

1. Endo H, et al. Imaging α-synuclein pathologies in animal models and patients with Parkinson's and related diseases. Neuron. 2024 Aug 7; 112(15): 2540-2557.e8. doi: 10.1016/j.neuron.2024.05.006.
2. Tagai K, et al. An optimized reference tissue method for quantification of tau protein depositions in diverse neurodegenerative disorders by PET with 18F-PM-PBB3 (18F-APN-1607). Neuroimage. 2022 Dec 1; 264: 119763. doi: 10.1016/j.neuroimage.2022.119763.
3. Endo H, et al. A Machine Learning-Based Approach to Discrimination of Tauopathies Using [18 F] PM-PBB3 PET Images. Mov Disord. 2022 Nov; 37(11): 2236-2246. doi: 10.1002/mds.29173.
4. Tagai K, et al. High-Contrast In Vivo Imaging of Tau Pathologies in Alzheimer's and Non-Alzheimer's Disease Tauopathies. Neuron. 2021 Jan 6; 109(1): 42-58.e8. doi: 10.1016/j.neuron.2020.09.042.
5. Endo H, et al. In vivo binding of a tau imaging probe, [11 C] PBB3, in patients with progressive supranuclear palsy. Mov Disord. 2019 May; 34(5): 744-754. doi: 10.1002/mds.27643.

2. Advantages and Drawbacks in Animal Models of Prodromal PD

Speaker:
Hodaka Yamakado, M.D., Ph.D.
Department of Therapeutics for Multiple System Atrophy, Kyoto University Graduate School of Medicine

Abstracts:

Recent large-scale clinical trials of disease-modifying therapies (DMT) suggest the necessity of targeting patients at earlier stages of the disease. From this perspective, the prodromal phase of PD is currently the focus of attention, emphasizing the need for a prodromal mouse model that accurately reflects the pathophysiology, along with early biomarkers. The model must possess high construct validity that accurately incorporates clinical and pathological features in the prodromal phase and high predictive validity to accurately evaluate the response to intervention.

In this talk, I will review animal models that reflect the characteristics of prodromal PD such as α-synuclein (aS) accumulation and associated early non-motor symptoms, with a focus on aS propagation models and genetic models. Among these, our BAC transgenic (Tg) mice exhibit neutrophilia, lymphopenia, and T-cell activation, reflecting peripheral pathology, as well as multiple prodromal symptoms. However, genetic models, including ours, often fail to induce motor symptoms, whereas aS propagation models bypass the critical early stage of aS aggregate formation. Consequently, neither model currently fully captures the entire natural course of the disease. Identifying factors that drive the transition from the prodromal to the symptomatic phase is crucial for developing preclinical models of disease-modifying therapies (DMTs) aimed at preventing or delaying disease onset.

Biosketch:

Dr. Hodaka Yamakado graduated from Kyoto University in 2000 and currently serves as a research associate professor in the Department of Therapeutics for MSA at Kyoto University. He specializes in the pathophysiology of PD and MSA, focusing on α-synuclein aggregation, prodromal biomarkers, and the development of novel animal models to study disease-modifying therapies.

Key articles:

1. Yamakado H, Takahashi R. Experimental Animal Models of Prodromal Parkinson's Disease. J Parkinsons Dis. 2024; 14(s2): S369-S379.
2. Sawamura M, Onoe H, Tsukada H, Isa K, Yamakado H, Okuda S, Ikuno M, Hatanaka Y, Murayama S, Uemura N, Isa T, Takahashi R. Lewy Body Disease Primate Model with α-Synuclein Propagation from the Olfactory Bulb. Mov Disord. 2022 Oct; 37(10): 2033-2044.
3. Okuda S, Uemura N, Sawamura M, Taguchi T, Ikuno M, Uemura MT, Yamakado H, Takahashi R. Rapid Induction of Dopaminergic Neuron Loss Accompanied by Lewy Body-Like Inclusions in A53T BAC-SNCA Transgenic Mice. Neurotherapeutics. 2022 Jan; 19(1): 289-304.
4. Taguchi T, Ikuno M, Hondo M, Parajuli LK, Taguchi K, Ueda J, Sawamura M, Okuda S, Nakanishi E, Hara J, Uemura N, Hatanaka Y, Ayaki T, Matsuzawa S, Tanaka M, El-Agnaf OMA, Koike M, Yanagisawa M, Uemura MT, Yamakado H, Takahashi R. α-Synuclein BAC transgenic mice exhibit RBD-like behaviour and hyposmia: a prodromal Parkinson's disease model. Brain. 2020 Jan 1; 143(1): 249-265.
5. Ikuno M, Yamakado H, Akiyama H, Parajuli LK, Taguchi K, Hara J, Uemura N, Hatanaka Y, Higaki K, Ohno K, Tanaka M, Koike M, Hirabayashi Y, Takahashi R. GBA haploinsufficiency accelerates alpha-synuclein pathology with altered lipid metabolism in a prodromal model of Parkinson's disease. Hum Mol Genet. 2019 Jun 1; 28(11): 1894-1904

Evening Seminar New technologies for Movement disorders

(EA Pharma Co., Ltd.)

Chair:

Yoshikazu Ugawa
Department of Human Neurophysiology, Fukushima Medical University

Shinji Saiki
Department of Neurology, Institute for Medicine, University of Tsukuba

1. New Technologies for Movement Disorders; Adaptive Deep Brain Stimulation

Speaker:

Genko Oyama, MD, PhD, FAAN
Department of Neurology and Stroke Medicine, Saitama Medical University

Abstracts:

Deep Brain Stimulation (DBS) has transformed the treatment of movement disorders such as Parkinson's disease, dystonia, and essential tremor. However, conventional DBS operates in an open-loop mode, delivering continuous stimulation irrespective of symptom fluctuations. To address these limitations, adaptive DBS (aDBS) has emerged as a novel approach that tailors stimulation parameters in real time based on physiological feedback.

A cornerstone of aDBS technology is the utilization of local field potentials (LFPs), which are neural signals recorded from implanted electrodes in the basal ganglia. LFPs reflect the oscillatory activity associated with motor symptoms such as bradykinesia and rigidity in Parkinson's disease. By using LFPs as biomarkers, aDBS systems can dynamically adjust stimulation, optimizing symptom control while minimizing unnecessary energy consumption and adverse effects.

This seminar will review the evidence supporting aDBS, including clinical trials and real-world applications. We will also discuss our experiences in implementing aDBS, addressing both its successes and challenges.

Biosketch:

1996-2002: Saitama Medical University, Saitama, Japan (M.D)
2002-2006: Residency in Neurology at Juntendo University Hospitals, Tokyo, Japan
2006-2010: Juntendo University Faculty of Medicine, Tokyo, Japan (Ph.D)
2009-2011: Research fellow, Center for Movement Disorders & Neurorestoration, University of Florida, FL, USA
2011-2014: Assistant professor, Department of Neurology, Juntendo University School of Medicine, Tokyo, Japan
2014-2023: Associate professor, Department of Neurology, Juntendo University School of Medicine, Tokyo, Japan
2024-current: Professor, Department of Neurology and Stroke Medicine, Saitama Medical University

Key articles:

1. Fasano A, Mure H, Bick SK, Schiess M, Witt T, Kimura K, Singer A, Sannelli C, Morelli N, Oyama G; PSR Study Group. Real-world local field potential recordings in patients with deep brain stimulation for Parkinson's disease. Parkinsonism Relat Disord. 2024; 125: 106090.
2. Sekimoto S, Oyama G, Bito K, Tsuchiya M, Kikuchi S, Takimoto B, Ichihashi T, Bautista JMP, Nuermaimaiti M, Sasaki F, Nakamura R, Iwamuro H, Ito M, Umemura A, Hattori N. Three-dimensional gait analysis of the effect of directional steering on gait in patients with Parkinson's disease. Parkinsonism Relat Disord. 2023; 114: 105770.

3. Oyama G, Burq M, Hatano T, Marks WJ Jr, Kapur R, Fernandez J, Fujikawa K, Furusawa Y, Nakatome K, Rainaldi E, Chen C, Ho KC, Ogawa T, Kamo H, Oji Y, Takeshige-Amano H, Taniguchi D, Nakamura R, Sasaki F, Ueno S, Shiina K, Hattori A, Nishikawa N, Ishiguro M, Saiki S, Hayashi A, Motohashi M, Hattori N. Analytical and clinical validity of wearable, multi-sensor technology for assessment of motor function in patients with Parkinson's disease in Japan. Sci Rep. 2023; 13(1): 3600.
4. Sasaki F, Oyama G, Sekimoto S, Nuermaimaiti M, Iwamuro H, Shimo Y, Umemura A, Hattori N. Closed-loop programming using external responses for deep brain stimulation in Parkinson's disease.. Parkinsonism Relat Disord. 2021. doi: 10.1016/j.parkreldis.2021.01.023.
5. Kasemsuk C, Oyama G, Hattori N. Management of impulse control disorders with deep brain stimulation: A double-edged sword. J Neurol Sci. 2017; 374: 63-68. doi: 10.1016/j.jns.2017.01.019.

2. Artificial Intelligence and Machine Learning for Movement Disorders

Speaker:
Shunsuke Kobayashi, MD, PhD
Department of Neurology, Teikyo University

Abstracts:

Recent advancements in artificial intelligence (AI) and machine learning (ML) have revolutionized the analysis of movement disorders by utilizing data from videos and sensors. This talk reviews AI and ML applications in evaluating motor symptoms, focusing on their potential and challenges. Key areas include hand movement, gait, handwriting, and speech, where high-resolution video recordings and wearable sensors provide quantitative data beyond traditional clinical assessments. AI enables early diagnosis, disease monitoring, and personalized treatment strategies. ML algorithms, particularly deep learning, excel in classifying subtypes, predicting disease progression, and identifying subtle symptom patterns often missed by clinicians. Despite these advancements, challenges such as data heterogeneity, privacy concerns, regulatory constraints, and the need for interpretable models limit generalizability and integration into clinical workflows. Addressing these barriers is crucial for maximizing the impact of AI and ML in improving the management of movement disorders.

Biosketch:

Dr. Kobayashi graduated from the Faculty of Medicine, The University of Tokyo in 1995 and earned his PhD for research on the frontal lobe in macaque monkeys. From 2005 to 2010, he was a research fellow at the University of Cambridge, where he studied the roles of dopamine neurons. Upon returning to Japan, he joined Fukushima Medical University. Since 2020, he has held key academic positions at Teikyo University, becoming Chair in 2023. Recently, he has been collaborating with experts in engineering and mathematics to analyze motor data in Parkinson's disease and related disorders using AI, furthering the integration of technology in neurological research.

Key articles:

1. Wiratman W, Kobayashi S, Chang FY, Asano K, Ugawa Y. Assessment of cognitive and motor skills in Parkinson's disease by a robotic object hitting game. *Front Neurol*, 2019.
2. Chang FY, Wiratman W, Ugawa Y, Kobayashi S. Event-related potentials during decision-making in a mixed-strategy game. *Front Neurosci* 15: 552750. 2021.
3. Tokushige S, Kobayashi S, et al. Roles of the cerebellum and basal ganglia in temporal integration: insights from a synchronized tapping task. *Clin Neurophysiol*. 158: 1-15, 2023.
4. Shin J, Kobayashi S. et al. Classification of hand-movement disabilities in Parkinson's disease using a motion-capture device and machine learning. *IEEE Access*. 1-14, 2024.

Special Seminar

(ONO PHARMACEUTICAL CO., LTD.)

Chair:
Nobutaka Hattori
Department of Neurology, Juntendo University School of Medicine

1. Therapies for Parkinson's disease – Present Status and Future Perspectives –

Speaker:
Ryosuke Takahashi, MD, PhD
Kyoto University Research Administration Center(KURA)

Abstracts:

I will talked about two different topics in this lecuture as follows.

1. L-dopa represents the mainstay in the currect anti-Parkinson's disease (PD) therapy. Motor complications including wearing-off and dyskinesia, however, accompany the long-tern use of L-dopa and reduces the quality of life of PD patients. Opicapone, a second generation catechol-o-methyltransferas (COMT) inhibitor, prolongs the plasma half-life of L-dopa and significantly decreases the predictable off time with a good safety profile. I would like to show the effects of opicapone on the pharmacokinetics of L-dopa and on early wearing-off in PD patients based on 203 and Korean Adoption trials, respectively.
2. Alpha-synuclein propagation plays a crucial role in the progression of Parkinson's disease (PD). We have shown that an α-amino-3-hydroxy-5-methyl-4-isoxazolepropionic acid (AMPA) receptor antagonist, perampanel (PER), blocks neuronal uptake of α-synuclein preformed fibrils (PFFs). However, the effect of PER on transsynaptic α-synuclein propagation remain elusive. Here, we report that dynamin-dependent, clathrin-independent endocytosis plays a role in neuronal uptake of PFFs *in vitro*. Oral administration of PER significantly increased the expression level of phosphorylated dynamin 1 in mouse brains, suggesting that PER suppresses dynamin-dependent endocytosis *in vivo*. To evaluate the efficacy of PER against transsynaptic α-synuclein propagation *in vivo*, we started PER treatment in PFFs-injected A53T BAC-SNCA transgenic mice 1 week after the injection, the timepoint when injected PFFs was cleared. PER treatment reduced the amount of α-synuclein pathology in second- or higher-order regions, but not in first-order regions, from the injection site and ameliorated neurodegeneration. Thus, PER could be a disease-modifying drug that modulates α-synuclein propagation in PD.

Biosketch:

Ryosuke Takahashi, MD, PhD graduated from Kyoto University, Japan in 1983. He received his training of neurology in Kyoto University Hospital. In 1989, he started basic researches as a staff scientist at Tokyo Metropolitan Institute for Neurosciences, then he worked as a postdoctoral fellow with Prof. Jon C. Reed at the Sanford-Burnham Institute, California, USA. He became Laboratory Head at RIKEN Brain Science Institute, Japan, in 1999. In 2005, he was appointed Professor and Chair of the Department of Neurology, Kyoto University Graduate School of Medicine. From 2014 to 2018, he served as the President of Japanese Society of Neurology. He also served as the Vice President of Japanese Society for Neuroscience. Currently he is the Program-specific Professor of Kyoto University Research Administration Center (KURA). His major research interests are in the molecular pathogenesis of Parkinson's disease and its related disorders.

Key articles:

1. Taguchi T, Ikuno M, Uemura MT, Yamakado H, Takahashi R, et al. (2000) α-Synuclein BAC transgenic mice exhibit RBD-like behaviour and hyposmia: a prodromal Parkinson's disease model. ***Brain*** 143: 249-265.
2. Ueda J, Uemura N, Takahashi R et al. (2021) Perampanel Inhibits α-synuclein transmission in Parkinson's disease models. ***Mov Disord*** 36:1554-1564.
3. Uemura N, Ueda J, Takahashi R, et al. (2021) α-Synuclein Spread from Olfactory Bulb Causes Hyposmia, Anxiety, and Memory Loss in BAC-SNCA Mice. ***Mov Disord*** 36:2036-2047.
4. Ueda J, Uemura N, Takahashi R et al. (2023) Ca^{2+} -Calmodulin-Calcineurin signaling modulates α-synuclein transmission. ***Mov Disord*** 38: 1056-1067.

Abstracts

2nd Day
2 Feb 2025（Sunday）

Japanese Session

（アッヴィ合同会社）

Chair:

大熊 泰之
順天堂大学医学部附属静岡病院

パーキンソン病および関連運動障害疾患と正常圧水頭症

Speaker:

常深 泰司
順天堂大学医学部附属順天堂医院

Abstracts:

正常圧水頭症（NPH）は、くも膜下出血など原因が明らかな二次性正常圧水頭症と、原因不明の特発性正常圧水頭症（iNPH）に大別される。iNPHの三主徴は認知機能障害、歩行障害、尿失禁であるが、これらは神経変性疾患にも一般的な症状であり、特異的な臨床症状や病理所見に乏しいため、さまざまな神経変性疾患が鑑別疾患として挙げられる。しかし、最近の報告では、これらの鑑別すべき疾患がiNPHの合併疾患として報告されるケースも増えている。

我々は後ろ向き研究において、iNPHにパーキンソン病（PD）が高頻度に合併する（29%）ことを報告した（Sakurai, 2022）。PDの合併は臨床症状に大きな影響を与え、DaTscan、MIBG心筋シンチグラフィー、髄液αシヌクレインRT-QuICがPD併存の診断に有用であることを示した。さらに、合併例であってもシャント術が短期的および長期的に予後を改善することを示した（Sakurai, 2022）。シャント術を行った15例の術前の臨床的および画像的特徴から手術予後予測因子を検討したところ、術前のMIBG心筋シンチの早期相が高値であり、淡蒼球の体積が大きいほうがシャント術の予後が良好であることが示された。これらの結果は、PD病理が全身に進行しておらず、脳萎縮が進行していない場合、シャント術の効果が得られやすいことを意味する。つまり、PDおよびiNPHのいずれも病初期のほうが適応が高い可能性を示唆している。

また、進行性核上性麻痺（PSP）とiNPHの合併も稀ではないことを確認した。さらに、シャント術が短期的な予後を改善するものの、長期的予後は大きな差がないことを明らかにした。

以上から、iNPHの併存疾患を正確に診断することが、シャント手術の適応可否を決定する上で重要であることが示唆される。

略歴:

1994年　東京医科歯科大学（現：東京科学大学）医学部卒業
2002年　東京医科歯科大学（現：東京科学大学）大学院脳神経機能病態学卒業
2016年　順天堂大学医学部附属順天堂医院脳神経内科准教授
2024年　順天堂大学医学部附属順天堂医院脳神経内科先任准教授

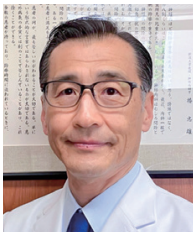
主要論文：

1. Sakurai A, **Tsunemi T***, Ishiguro Y, Okuzumi A, Hatano T, and Hattori N. (2021) Comorbid alpha synucleinopathies in idiopathic normal pressure hydrocephalus. **J Neurol** 269, 2022-2029.
2. Sakurai A, **Tsunemi T***, Shimada T, Kawamura K, Nakajiima M, Miyajima M, and Hattori N (2022) The effectiveness of cerebrospinal fluid shunt surgery for idiopathic normal pressure hydrocephalus with comorbid Parkinson's disease/PD dementia. **J Neurosurg** 11: 1-8.
3. Ishiguro Y, **Tsunemi T***, Shimada T, Yoroisaka A, Ueno S, Takeshige-Amanno H, Hatano T, Inoue Y, Saiki S, Hattori N. (2024) Extracellular vesicles contain filamentous alpha-synuclein and facilitate the propagation of Parkinson's pathology. **Biochem Biophys Res Commun**. Apr 9: 703: 149620. doi: 10.1016/j.bbrc.2024.149620.

Japanese Session

（武田薬品工業株式会社）

Chair:
坪井 義夫
順天堂大学大学院医学研究科 PD長期観察研究講座



早期パーキンソン病の治療～再考～

Speaker:
高橋 一司
東京都立神経病院 脳神経内科

Abstracts:

初期治療の選択と時期は、患者の背景因子（年齢、症状、基礎疾患、生活状況など）を考慮し、初期治療の診断的意義についても十分に話し合い、決定する。治療の目的は自立の保持、QoLの維持であり、運動、食事や水分摂取などの生活指導も重要である。特に運動療法の重要性に関してはエビデンスが蓄積されている。薬物治療の選択は、医師の臨床経験に加え、臨床試験のエビデンスに関する知識と理解に依存する。臨床試験データは、治療薬がプラセボに比べ有効であることを示すが、個々の患者にとって最適な薬剤を決定する上ではほとんど役に立たない。

非麦角系ドパミンアゴニスト、MAO-B阻害薬の単剤療法が推奨された時期を経て、現在ではRCTのデータからL-ドパが他のすべての治療薬と比べ、運動症状を最も改善すること、副作用が最も少ないことが確認されている。L-ドパのdelayed start studyでは、40週早期導入の有効性も問題点も示されず、ジスキネジアの早期発現や疾患修飾作用のエビデンスもない。米国神経学会、英国NICEのガイドラインはともに、その有効性から初期治療としてL-ドパを推奨している。患者には、低～中等量のL-ドパ（400 mg/日未満）で治療を開始しても、ジスキネジアなどの運動合併症の出現を促進する可能性が低いことを十分説明する。

L-ドパに対する初期反応が良好な患者でも進行に伴い、運動合併症はいまだ発症していないが、単剤療法にて十分な症状コントロールが得られない病状がしばしば経験される。この場合、増量による単剤療法の継続か、L-ドパ補助薬の導入かを選択する必要が生じる。多くの場合、単一のドパミン補充療法を高用量にて使用するより、複数の薬剤を低～中等量にて使用するほうが副作用が少なく、症状を適切にコントロールできることが多い。L-ドパ補助薬の選択の際には、各薬剤の非運動症状に対する効果にも注意を払う。

略歴:

1987年　慶應義塾大学医学部 卒業
1991年　慶應義塾大学大学院医学研究科博士課程 修了
1992年　浦和市立病院(現：さいたま市立病院)神経内科
1995年　米国ペンシルバニア大学 留学
1999年　国立病院東京医療センター 神経内科
2005年　慶應義塾大学医学部 神経内科 専任講師
2012年　東京都立神経病院 脳神経内科 部長
2014年　埼玉医科大学 脳神経内科 教授
2020年　東京都立神経病院 院長､現在にいたる

主要論文：

1. Seki M, Takahashi K et al, Clinical features and varieties of non-motor fluctuations in Parkinson's disease: A Japanese multicenter study. *PRD* **19** (1): 104-108, 2013
2. Nihei Y, Takahashi K et al, REM Sleep Behavior Disorder in Japanese Patients with Parkinson's Disease: A Multicenter Study Using the REM Sleep Behavior Disorder Screening Questionnaire. *J Neurol* **259** (8): 1606-1612, 2012

Japanese Session

（大塚製薬株式会社）

Chair:
下 泰司
順天堂大学医学部附属練馬病院

パーキンソン病の非運動症状アップデート

Speaker:
永山 寛
日本医科大学脳神経機能解析学講座

Abstracts:

近年、パーキンソン病（PD）では運動障害以外にも多くの症候が存在することが指摘されてきている。これらは非運動症状としてまとめられ、自律神経症状、睡眠症状、痛み、精神症状などが含まれることが知られている。

非運動症状の多くは、PDの病態に関連して出現している。これらが特に重要とされる点としては、第一に運動障害の発現に先立って認められることである。例えばPD発症前に認められるレム睡眠行動障害の存在は、将来のPD発症を強く予測させることが指摘されている。言い換えれば、個々の非運動症状のみをもってPDと診断することはできないものの、発症前の非運動症状の発現はPDの病態自体と関連する可能性があり、その把握はパーキンソン病患者にとっては非常に重要となりえる可能性がある。

またPDでは、夜間の睡眠障害の原因になりえる過活動膀胱などの排尿障害や、日常の活動へ多大な影響を及ぼす種々の痛みといった非運動症状もよく見られる。血圧調節などの自律神経症状も非常に大きな問題の一つであり、起立性低血圧や食事性低血圧もよく認められ、治療に奏効したとしても臥位高血圧など、対応で難渋することも少なくない。

うつ症状などの一部の非運動症状もPDではよく認められる。PDのうつは運動症状発症前から存在するものや、経過とともに出現するものなど多彩であり、生活の質（QOL）に最も大きく影響することも指摘されている。しかもその多くはPD 罹患による反応性の発症ではなくPDの病態に伴っての発症のため、運動症状の改善があってもうつ症状は改善せず、QOLは低いままとなる。

このような観点から、非運動症状はPDでは非常に重要視されている。ここでは非運動症状に関する最近の知見を紹介し、個々の部分症としての視点だけではなくPD全体にどのように関連しているのかも考えていきたい。

略歴:

学歴
平成5年3月　日本医科大学卒業
平成7年4月　日本医科大学大学院入学
平成12年3月　学位（医学博士）取得
職歴
平成5年5月～　日本医科大学付属病院 神経精神科、日本医科大学付属第一病院第二内科で研修
平成7年2月　東京都多摩老人医療センター 神経内科
平成11年7月　日本医科大学付属病院 第二内科医員（神経内科）
平成12年4月　日本医科大学 内科学第二 助手
平成20年4月　日本医科大学付属病院 病院講師
平成22年4月　日本医科大学 内科 神経・腎臓・膠原病リウマチ部門 講師
平成25年4月　日本医科大学大学院 医学研究科 神経内科学分野 講師（組織名称変更）
平成27年10月　日本医科大学大学院 医学研究科 神経内科学分野 准教授
平成29年4月　東京大学医学部老年病科 非常勤講師兼任
令和3年4月　日本医科大学 脳神経機能解析学講座 教授、現在に至る

主要論文：

1. Nagayama H, et al. Effect of istradefylline on mood disorders in Parkinson's disease. J Neurol Sci 2019; 396: 78-83.
2. Nagayama H, et al. Abnormal cardiac [^{123}I] -meta-iodobenzylguanidine uptake in multiple system atrophy. Mov Disord 2010; 25: 1744-7.
3. Nagayama H, et al. Influence of ageing on the pharmacokinetics of levodopa in elderly patients with Parkinson's disease. Parkinsonism Relat Disord 2011; 17: 150-2.
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5. Nagayama H, et al. Reliability of MIBG myocardial scintigraphy in the diagnosis of Parkinson's disease. J Neurol Neurosurg Psychiatry 2005. 76: 249-51.

Japanese Session

（協和キリン株式会社）

Chair:
武田 篤
国立病院機構仙台西多賀病院

超超高齢社会におけるパーキンソン病診療

Speaker:
関 守信
慶應義塾大学医学部神経内科，慶應義塾大学病院パーキンソン病センター，
慶應義塾大学パーキンソン病研究センター

Abstracts:

令和5年10月現在、日本の高齢化率は29.1％に達し、「超超高齢社会」に直面しています。そのなかで、日本では世界に先駆けて高齢パーキンソン病（PD）患者が急増しており、その診療が重要な課題となっています。日本では“65歳”以上を高齢者と定義し、ガイドラインにおいても運動合併症発現リスクの観点から“65歳”発症で治療方針が分けられていますが、実臨床で重要なのはより高齢の患者の診療です。しかし、超高齢PD患者は症状が重症、併存疾患が多いなどの理由で治験や臨床研究には含まれずOrphan Populationと称されます。

超高齢発症PD患者は初期から症状が重症で進行が速く、運動合併症の発現頻度は低い一方、複数の背景病理があることが多いのが特徴です。また、晩期高齢PD患者は運動緩慢が高度で、レボドパ抵抗性の体軸症状、非運動症状が主症状となり、運動合併症の影響は小さくなります。超高齢PD患者に対しては有効性、安全性の観点からレボドパ製剤が治療の中心となりますが、その効果は併存病理や薬剤の溶解・吸収の問題から中年PD患者に比べ減弱することが報告されています。また、超高齢の女性患者が急増する中で、レボドパの生体利用能の性差などを考慮した治療戦略も重要となります。日本のレセプトデータの解析から、超高齢PD患者ではレボドパ製剤が治療の主役であり、年齢ごとにレボドパ製剤の使われ方に違いがあることがわかりました。レボドパ製剤以外の薬剤については、国内の臨床試験の平均年齢は65歳前後であり、その知見を超高齢PD患者に適用できるかは疑問が残ります。一方、市販後の使用成績調査には75歳以上が多く含まれ、超高齢PD患者の治療実態の把握に役立ちます。薬物療法とともに、超高齢PD患者に対してはリハビリが重要なことはいうまでもありません。超高齢PD患者が抱える課題は非常に多岐にわたり、多職種による包括的アプローチが不可欠であり、患者、家族を支える体制作りが喫緊の課題です。

略歴:

2003年3月　慶應義塾大学医学部卒業
2007年4月　慶應義塾大学医学部内科学（神経内科）助教
2013年3月　医学博士
2014年11月　Innsbruck Medical University, Department of Neurology, Research Fellow（Prof. Werner Poewe）
2017年7月　慶應義塾大学医学部内科学（神経内科）助教
2018年10月　慶應義塾大学医学部内科学（神経内科）専任講師
2022年10月　慶應義塾大学医学部内科学（神経内科）准教授
慶應義塾大学病院パーキンソン病センター実務責任者（兼務）
慶應義塾大学パーキンソン病研究センター実務責任者
2024年4月　慶應義塾大学パーキンソン病研究センターセンター長（兼務）

主要論文:

1. Seki M, Takahashi K, Koto A, et al. Camptocormia in Japanese patients with Parkinson's disease: a multicenter study. *Mov Disord*. 26(14): 2567-71, 2011.
2. Seki M, Takahashi K, Uematsu D, et al. Clinical features and varieties of non-motor fluctuations in Parkinson's disease: a Japanese multicenter study. *Parkinsonism Relat Disord*. 19(1): 104-8, 2013.
3. Seki M, Seppi K, Mueller C, et al. Diagnostic potential of dentatorubrothalamic tract analysis in progressive supranuclear palsy. *Parkinsonism Relat Disord*. 49: 81-7. 2018.
4. Seki M, Seppi K, Mueller C, et al. Diagnostic Potential of Multimodal MRI Markers in Atypical Parkinsonian Disorders. *J Parkinsons Dis*. 9(4): 681-91. 2019.
5. Seki M, Kawata Y, Hayashi A, et al. Prescribing patterns and determinants for elderly patients with Parkinson's disease in Japan: a retrospective observational study using insurance claims databases. *Front Neurol*. 14: 1162016. 2023

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OPJ-COMH0004-1.0　2024年12月 作成

パーキンソン病専門ホーム

PDハウス

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患者様の日々に役立つ情報まで配信中！

SUNWELS

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見学のご予約・お問い合わせは下記へご連絡ください

0120-540-367［入居相談室］9:00-17:00

ご予約状況などによりご希望のお日にちにご見学を承れない場合がございます。あらかじめご了承ください。

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