

5th International Conference of
Korean Movement Disorder Society



ICKMDS 2025

Bridging from the Origin to Clinical Practice: A Beginning Has an Ending

October 31 (Fri) – November 1 (Sat), 2025 | Lotte Hotel Jeju, Jeju, Korea

ABSTRACT BOOK



International Parkinson and
Movement Disorder Society
MDS-Endorsed Event

Endorsed by the International Parkinson
and Movement Disorder Society

 **KMDS**
The Korean Movement
Disorder Society



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SCIENTIFIC PROGRAM

DAY 1_ October 31 (Fri), 2025

Room A

09:00-09:30 **Opening Ceremony**

09:30-11:00 **Plenary Session 1**
What Triggers Synucleinopathy?

Chairs Caroline TANNER (USA), Jin Whan CHO (Korea)

09:30-09:55 **P1-1. Genetic Architecture of Synucleinopathy** p. 34
Yun Joong KIM (Korea)

09:55-10:20 **P1-2. Environmental and Related Factors as a Trigger for Synucleinopathy** p. 44
Caroline TANNER (USA)

10:20-10:45 **P1-3. Metabolic Factors Contributing to Risk and Progression of Parkinson's Disease** p. 47
Ai Huey TAN (Malaysia)

10:45-11:00 **Q&A**

11:00-11:20 **Break**

11:20-12:50 **Plenary Session 2**
When and Where Does Synucleinopathy Begin? (Parkinson's Disease Perspective)

Chairs Nobutaka HATTORI (Japan), Tae-Beom AHN (Korea)

11:20-11:45 **P2-1. Neuroanatomical Origins and Clinical Landscape of Parkinson's Disease** p. 52
Chaewon SHIN (Korea)

11:45-12:10 **P2-2. Unraveling the Clinical Origins of Parkinson's Disease: Insights into Idiopathic RBD as the Prodromal Stage** p. 55
Yun Kwok WING (Hong Kong)

12:10-12:35 **P2-3. Cellular Origins of Parkinson's Disease and Emerging Therapeutic Strategies** p. 58
Lorraine KALIA (Canada)

12:35-12:50 **Q&A**

12:50-14:00 **Lunch**

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17:00-18:10 **Parallel Session 5**

Non-Motor Symptoms: From Prodromal Indicators to Advanced Manifestations

Chairs Ai Huey TAN (Malaysia), Mee Young PARK (Korea)

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Chairs Jeffrey HAUSDORFF (Israel), Seong-Beom KOH (Korea)

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14:40-15:00 **S2-3. Practical Management of Freezing of Gait Based on Pathomechanism** p. 100
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Chairs Raymond ROSALES (Philippines), Young H. SOHN (Korea)

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Young H. SOHN (Korea)

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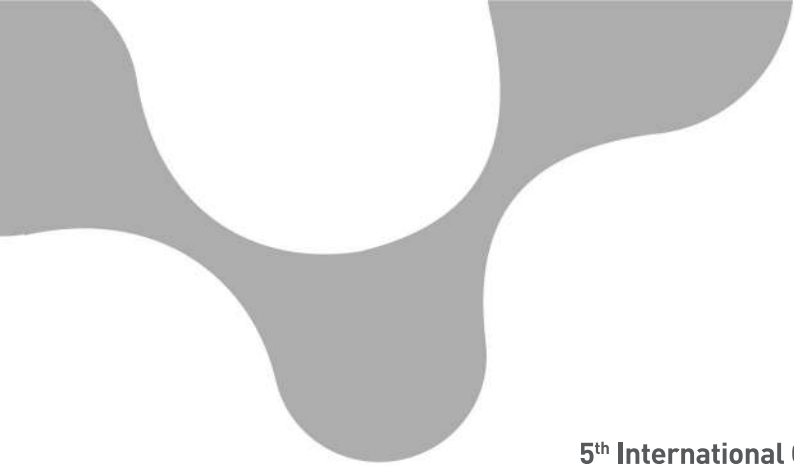
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ICKMDS 2025

5th International Conference of Korean Movement Disorder Society

**Bridging from the Origin to Clinical Practice:
A Beginning Has an Ending**



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A Beginning Has an Ending

DAY 1_ October 31 (Fri), 2025

Room A

Plenary Session 1

What Triggers Synucleinopathy?

Chairs Caroline TANNER (USA)
Jin Whan CHO (Korea)



P1-1

Genetic Architecture of Synucleinopathy

Yun Joong KIM

Yongin Severance Hospital, Yonsei University College of Medicine, Korea

Genetic studies have greatly advanced our understanding of Parkinson's disease (PD). This lecture will highlight key findings from genome-wide association studies (GWAS) in PD and discuss how these findings intersect with emerging insights into synucleinopathy.

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Education / Training

Institution and Location	Degree	Year(s)	Field of Study
Yonsei University, Seoul, Korea	M.D.	1983-1989	Medicine
Yonsei University, Seoul, Korea	M.S.	1991-1993	Medicine
Yonsei University, Seoul, Korea	Ph.D.	1994-2004	Neurology
Dept of Neurology, Yonsei Medical Center	Resident	1990-1994	Neurology
Dept of Neurology, The Toronto Hospital	Fellow	1997-1999	Movement disorders
Dept of Neurology, The Massachusetts General Hospital	Fellow	1999-2002	Cellular neurobiology

Positions and Honors

Professional Experience

RESEARCH AND PROFESSIONAL EXPERIENCE:

2003.04 – 2019.12 Professor, ILSONG Institute of Life Science, Hallym University
Department of Neurology, Hallym university college of medicine
2012.03 – 2019.12 Director, Hallym Institute of Translational Genomics & Bioinformatics
2014.01 – 2017.12 Editor-in-Chief, Journal of Movement Disorders
2018.01 – 2019.12 President, The Korean Movement Disorders Society
2020.01 – present Professor, Department of Neurology,
Director, Center for Neurodegenerative Disorders, Yongin Severance Hospital
Yonsei University College of Medicine, Yonsei University

PROFESSIONAL MEMBERSHIPS:

- 1990-present The Korean Neurological Association
- 1997-present International Parkinson's and Movement Disorder Society
- 2006-present The Korean Movement Disorders Society

AWARD

- 2018.10 2017-2018 The Best Original Article of the Year Award, Movement Disorders

Executive summary of Professor Yun Joong Kim

Yun Joong Kim, MD, PhD, is a Professor of the Department of Neurology at Yonsei University College of Medicine. He graduated with a medical degree from Yonsei University College of Medicine in Seoul, Korea, and currently works at the Center for Neurodegenerative Disorders in Yongin Severance Hospital of Yonsei University College of Medicine. Dr. Kim completed his movement disorders fellowship under the mentorship of Dr. A.E. Lang at the University of Toronto and postdoctoral training in Dr. M. DiFiglia's laboratory at the Massachusetts General Hospital of Harvard Medical School. He is a founding member of the Korean Movement Disorders Society (KMDS) and served as the Editor-in-Chief of the Journal of Movement Disorders published by KMDS, as well as the president of KMDS in 2018-2019.

Dr. Kim's main research interests are genetics/genomics of Parkinson's disease (PD) and other hereditary movement disorders. He focuses on the discovery of new genes/alleles for PD, deep phenotyping, and genotype-phenotype correlation of hereditary movement disorders. He is also interested in systems biology research to understand the pathogenesis of PD by integrating diverse Mendelian genes for hereditary parkinsonism in the multiple layers of genomic, transcriptomic, and proteomic data. Dr. Kim has published a series of original articles demonstrating the contribution of hereditary parkinsonism genes (PARK and non-PARK genes) to the pathogenesis of PD in gene expression layers and genomic layers. He was awarded the 2017-2018 Original Research Article of the Year Award from the International Parkinson and Movement Disorder Society for his work on this topic.

Peer Reviewed Publications (* corresponding author):

1. Chun MY, Chung SJ, Kim SH, Park CW, Jeong SH, Lee HS, Lee PH, Sohn YH, Jeong Y, Kim YJ*. Hippocampal Perfusion Affects Motor and Cognitive Functions in Parkinson Disease: An Early Phase 18F-FP-CIT Positron Emission Tomography Study. *Ann Neurol*. 2024 Feb;95(2):388-399. doi: 10.1002/ana.26827. Epub 2023 Nov 28. PMID: 37962393.
2. Baek K, Kim YM, Na HK, Lee J, Shin DH, Heo SJ, Chung SJ, Kim K, Lee PH, Sohn YH, Yoon J, Kim YJ*. Comparing MoCA Performance in Parkinson's disease: Age and Education-Adjusted Cutoffs vs. Machine Learning. *J Mov Disord*. 2024 Feb 13. doi:10.14802/jmd.23271. Epub ahead of print. PMID: 38346940.
3. Thi Lai T, Kim YE, Nguyen LTN, Thi Nguyen T, Kwak IH, Richter F, Kim YJ, Ma HI. Microglial inhibition alleviates alpha-synuclein propagation and neurodegeneration in Parkinson's disease mouse model. *NPJ Parkinsons Dis*. 2024 Feb 2;10(1):32. doi: 10.1038/s41531-024-00640-2. PMID: 38302446; PMCID:PMC10834509.
4. Jeong SH, Park CJ, Cha J, Kim SY, Lee SK, Kim YJ, Sohn YH, Chung SJ, Lee PH. Choroid Plexus Volume, Amyloid Burden, and Cognition in the Alzheimer's Disease Continuum. *Aging Dis*. 2024 Jan 23. doi: 10.14336/AD.2024.0118. Epub ahead of print. PMID: 38300638.
5. Park CJ, Eom J, Park KS, Park YW, Chung SJ, Kim YJ, Ahn SS, Kim J, Lee PH, Sohn YH, Lee SK. An interpretable multiparametric radiomics model of basal ganglia to predict dementia conversion in Parkinson's disease. *NPJ Parkinsons Dis*. 2023 Aug 30;9(1):127. doi: 10.1038/s41531-023-00566-1. PMID: 37648733; PMCID: PMC10468504.

6. Nguyen HD, Kim YE, Nhat Nguyen LT, Kwak IH, Lee YK, Kim YJ, Hai Nguyen TT, Pham HN, Ma HI. Upregulation of immuno-proteasome PSMB8 is associated with Parkinson's disease. *Parkinsonism Relat Disord*. 2023 Sep;114:105797. doi:10.1016/j.parkreldis.2023.105797. Epub 2023 Aug 5. PMID: 37562243.
7. Chung SJ, Kim SH, Park CW, Lee HS, Kim YJ, Lee PH, Jeong Y, Sohn YH. Is the cingulate island sign a marker for early dementia conversion in Parkinson's disease? *Eur J Neurol*. 2023 Dec;30(12):3732-3740. doi: 10.1111/ene.16007. Epub2023 Aug 15. PMID: 37505994.
8. Jeong SH, Jeong HJ, Sunwoo MK, Ahn SS, Lee SK, Lee PH, Kim YJ, Sohn YH, Park CJ, Chung SJ. Association between choroid plexus volume and cognition in Parkinson disease. *Eur J Neurol*. 2023 Oct;30(10):3114-3123. doi:10.1111/ene.15999. Epub 2023 Aug 6. PMID: 37498202.
9. Jeong SH, Kim SH, Park CW, Lee HS, Lee PH, Kim YJ, Sohn YH, Jeong Y, Chung SJ. Differential Implications of Cerebral Hypoperfusion and Hyperperfusion in Parkinson's Disease. *Mov Disord*. 2023 Oct;38(10):1881-1890. doi: 10.1002/mds.29565. Epub 2023 Jul 25. PMID: 37489576.
10. Jeong SH, Park CJ, Jeong HJ, Sunwoo MK, Ahn SS, Lee SK, Lee PH, Kim YJ, Sohn YH, Chung SJ. Association of choroid plexus volume with motor symptoms and dopaminergic degeneration in Parkinson's disease. *J Neurol Neurosurg Psychiatry*. 2023 Dec;94(12):1047-1055. doi: 10.1136/jnnp-2023-331170. Epub 2023 Jun 30. PMID: 37399288.
11. Lee H, Jeon J, Jung D, Won JI, Kim K, Kim YJ, Yoon J. RelCurator: a text mining-based curation system for extracting gene-phenotype relationships specific to neurodegenerative disorders. *Genes Genomics*. 2023 Aug;45(8):1025-1036. doi: 10.1007/s13258-023-01405-6. Epub 2023 Jun 10. PMID: 37300788.
12. Kwak IH, Lee YK, Ma HI, Lee S, Yun M, Kim YJ, Hwang HS, Kim YE. Striatal Subregion Analysis Associated with REM Sleep Behavior Disorder in Parkinson's Disease. *J Integr Neurosci*. 2023 Jan 16;22(1):18. doi: 10.31083/j.jin2201018. PMID: 36722243.
13. Jeong SH, Park CW, Lee HS, Kim YJ, Yun M, Lee PH, Sohn YH, Chung SJ. Patterns of striatal dopamine depletion and motor deficits in de novo Parkinson's disease. *J Neural Transm (Vienna)*. 2023 Jan;130(1):19-28. doi: 10.1007/s00702-022-02571-9.
14. Vollstedt EJ, Schaake S, Lohmann K,, Kim YJ,, Klein C; MJFF Global Genetic Parkinson's Disease Study Group. Embracing Monogenic Parkinson's Disease: The MJFF Global Genetic PD Cohort. *Mov Disord*. 2023 Jan 24. doi: 10.1002/mds.29288.
15. Chung SJ, Kim YJ, Kim YJ, Lee HS, Jeong SH, Hong JM, Sohn YH, Yun M, Jeong Y, Lee PH. Association Between White Matter Networks and the Pattern of Striatal Dopamine Depletion in Patients With Parkinson Disease. *Neurology*. 2022 Dec 12;99(24):e2672-e2682. doi: 10.1212/WNL.0000000000201269. PMID: 36195451
16. Chung SJ, Kim YJ, Kim YJ, Lee HS, Yun M, Lee PH, Jeong Y, Sohn YH. Potential Link Between Cognition and Motor Reserve in Patients With Parkinson's Disease. *J Mov Disord*. 2022 Sep;15(3):249-257. doi: 10.14802/jmd.22063. PMID: 36065615
17. Kim YJ*, Kim K, Lee H, Jeon J, Lee J, Yoon J. The Protein-Protein Interaction Network of Hereditary Parkinsonism Genes Is a Hierarchical Scale-Free Network. *Yonsei Med J*. 2022 Aug;63(8):724-734. doi: 10.3349/ymj.2022.63.8.724.
18. Jeon J, Kim K, Baek K, Chung SJ, Yoon J, Kim YJ*. Accuracy of Machine Learning Using the Montreal Cognitive Assessment for the Diagnosis of Cognitive Impairment in Parkinson's Disease. *J Mov Disord*. 2022 May;15(2):132-139. doi:10.14802/jmd.22012.
19. Shin J, Jeon J, Jung D, Kim K, Kim YJ, Jeong DH, Yoon J. PhenGenVar: A User-Friendly Genetic Variant Detection and Visualization Tool for Precision Medicine. *J Pers Med*. 2022 Jun 12;12(6):959. doi: 10.3390/jpm12060959. PMID:35743744
20. Kim YJ, Park CW, Shin HW, Lee HS, Kim YJ, Yun M, Lee PH, Sohn YH, Jeong Y, Chung SJ. Identifying the white matter structural network of motor reserve in early Parkinson's disease. *Parkinsonism Relat Disord*. 2022 Sep;102:108-114. doi:10.1016/j.parkreldis.2022.08.005

21. Nguyen LTN, Nguyen HD, **Kim YJ**, Nguyen TT, Lai TT, Lee YK, Ma HI, Kim YE. Role of NLRP3 Inflammasome in Parkinson's Disease and Therapeutic Considerations. *J Parkinsons Dis.* 2022;12(7):2117-2133. doi: 10.3233/JPD-223290.
22. Grover S, Kumar Sreelatha AA, Pihlstrom L, . . . , **Kim YJ**, . . . , Sharma M; and the Comprehensive Unbiased Risk Factor Assessment for Genetics and Environment in Parkinson's Disease (COURAGE-PD) Consortium. Genome-wide Association and Meta-analysis of Age at Onset in Parkinson Disease: Evidence From the COURAGE-PD Consortium. *Neurology.* 2022 Aug 16;99(7):e698-e710. doi: 10.1212/WNL.0000000000200699.
23. Domenighetti C, Douillard V, Sugier PE, Sreelatha AAK, . . . , **Kim YJ**, . . . , Elbaz A. Comprehensive Unbiased Risk Factor Assessment for Genetics and Environment in Parkinson's Disease (Courage-PD) Consortium. The Interaction between HLA-DRB1 and Smoking in Parkinson's Disease Revisited. *Mov Disord.* 2022 Sep;37(9):1929-1937. doi: 10.1002/mds.29133
24. Nguyen TT, **Kim YJ**, Lai TT, Nguyen PT, Koh YH, Nguyen LTN, Ma HI, Kim YE. PTEN-Induced Putative Kinase 1 Dysfunction Accelerates Synucleinopathy. *J Parkinsons Dis.* 2022;12(4):1201-1217. doi: 10.3233/JPD-213065.
25. Lai TT, **Kim YJ**, Ma HI, Kim YE. Evidence of Inflammation in Parkinson's Disease and Its Contribution to Synucleinopathy. *J Mov Disord.* 2022 Jan;15(1):1-14. doi: 10.14802/jmd.21078
26. Sung YH, Kim JS, Yoo SW, . . . , **Kim YJ**, Kim HS, Woo MH, Choi HJ, Kim EY. A prospective multi-centre study of susceptibility map-weighted MRI for the diagnosis of neurodegenerative parkinsonism. *Eur Radiol.* 2022 May;32(5):3597-3608. doi: 10.1007/s00330-021-08454-z
27. Domenighetti C, Sugier PE, Ashok Kumar Sreelatha A, . . . , **Kim YJ**, Kolber P, . . . , Elbaz A. Comprehensive Unbiased Risk Factor Assessment for Genetics and Environment in Parkinson's Disease (Courage-PD) Consortium. Dairy Intake and Parkinson's Disease: A Mendelian Randomization Study. *Mov Disord.* 2022 Apr;37(4):857-864. doi: 10.1002/mds.28902.
28. Lai TT, **Kim YJ**, Nguyen PT, Koh YH, Nguyen TT, Ma HI, Kim YE. Temporal Evolution of Inflammation and Neurodegeneration With Alpha-Synuclein Propagation in Parkinson's Disease Mouse Model. *Front Integr Neurosci.* 2021 Oct 5;15:715190. doi: 10.3389/fnint.2021.715190
29. Domenighetti C, Sugier PE, Sreelatha AAK, Schulte C, . . . , **Kim YJ**, . . . , Elbaz A. Comprehensive Unbiased Risk Factor Assessment for Genetics and Environment in Parkinson's Disease (Courage-PD) consortium. Mendelian Randomisation Study of Smoking, Alcohol, and Coffee Drinking in Relation to Parkinson's Disease. *J Parkinsons Dis.* 2022;12(1):267-282. doi: 10.3233/JPD-212851.
30. Chung SJ, **Kim YJ**, Yoo HS, Jung JH, Baik K, Lee HS, Lee YH, Hong JM, Sohn YH, Lee PH. Temporalis Muscle Thickness as an Indicator of Sarcopenia Is Associated With Long-term Motor Outcomes in Parkinson's Disease. *J Gerontol A Biol Sci Med Sci.* 2021 Nov 15;76(12):2242-2248. doi: 10.1093/gerona/glab082. PMID: 33754634.
31. Chung SJ, Yoo HS, Shin NY, Park YW, Lee HS, Hong JM, **Kim YJ**, Lee SK, Lee PH, Sohn YH. Perivascular Spaces in the Basal Ganglia and Long-term Motor Prognosis in Newly Diagnosed Parkinson Disease. *Neurology.* 2021 Apr 20;96(16):e2121-e2131. doi: 10.1212/WNL.0000000000011797.
32. Chung SJ, Lee PH, Sohn YH, **Kim YJ***. Glucocerebrosidase Mutations and Motor Reserve in Parkinson's Disease. *J Parkinsons Dis.* 2021 Aug 26; 11(4):1715-1724. doi:10.3233/JPD-212758. Epub ahead of print. PMID: 34459414.
33. **Kim YJ***, Lee JW, Kim NY, Hong SK, Cho YS, Yoon J*. The burden of rare damaging variants in hereditary atypical parkinsonism genes is increased in patients with Parkinson's disease. *Neurobiology of Aging.* 2021 Apr;100:118.e5-118.e13 doi:10.1016/j.neurobiolaging.2020.11.011
34. Chung SJ, Lee S, Yoo HS, Baik KW, Lee HS, Jung JH, Choi Y, Hong JM, **Kim YJ**, Ye BS, Sohn YH, Yun M, Lee PH. Different patterns of β amyloid deposition in patients with Alzheimer's disease according to the presence of mild parkinsonism. *Neurobiol Aging* 2021 101 199-206. <https://doi.org/10.1016/j.neurobiolaging.2021.01.022>

35. Phung DM, Lee JW, Hong SK, Kim YE, Yoon J, **Kim YJ***. Meta-analysis of differentially expressed genes in the substantia nigra in Parkinson's disease supports phenotype-specific transcriptome changes. *Frontiers in Neuroscience* <https://doi.org/10.3389/fnins.2020.596105> Dec 18 2020
36. Kim YE, **Kim YJ**, Hwang HS, Ma HI. REM sleep behavior disorder in early Parkinson's disease predicts the rapid dopaminergic denervation. *Parkinsonism Relat Disord*. 2020 Sep 21;80:120-126. doi: 10.1016/j.parkreldis.2020.09.032.
37. Kim YE, Lai TT, **Kim YJ**, Jeon B. Preferential microglial activation associated with pathological alpha synuclein transmission. *J Clin Neurosci* 2020 81 469-476
38. Kim YE, Ma HI, Seong GH, Huh JY, Park JS, **Kim YJ***. Early Impairment of Chopsticks Skill in Parkinsonism Suggests Progressive Supranuclear Palsy. *J Clin Neurol*, 2020;16:254-260
39. Park I, Kim YJ, **Kim YJ**, Lee U. Automatic, Qualitative Scoring of the Interlocking Pentagon Drawing Test (PDT) based on U-Net and Mobile Sensor Data. *Sensors* 2020 27;20(5):1283. doi: 10.3390/s20051283
40. Choi JH, Lee JY, Cho JW, Ko SB, Ahn TB, Kim SJ, Cheon SM, Kim JS, **Kim YJ**, Ma HI, Baik JS, Lee PH, Chung SJ, Kim JM, Song IU, Kim HJ, Sung YH, Kwon DY, Lee JH, Kim JY, Kim JS, Yun JY, Kim HJ, Hong JY, Kim MJ, Youn J, Kim JS, Oh ES, Yang HJ, Yoon WT, You S, Kwon KY, Park HE, Lee SY, Kim Y, Kim HT, Park MY. Validation of the Korean Version of the Questionnaire for Impulsive-Compulsive Disorders in Parkinson's Disease Rating Scale. *J Clin Neurol* 2020;16: 254-260
41. Yang HJ, Kim HJ, Koh SB, Kim JS, Ahn TB, Cheon SM, Cho JW, **Kim YJ**, Ma HI, Park MY, Baik JS, Lee PH, Chung SJ, Kim JM, Song IU, Kim JY, Sung YH, Kwon DY, Lee JH, Lee JY, Kim JS, Yun JY, Kim HJ, Hong JY, Kim MJ, Youn J, Kim JS, Oh ES, Yoon WT, You S, Kwon KY, Park HE, Lee SY, Kim Y, Kim HT, Kim SJ. Subtypes of Sleep Disturbance in Parkinson's Disease Based on the Cross-Culturally Validated Korean Version of Parkinson's Disease Sleep Scale-2. *J Clin Neurol*. 2020 Jan;16(1):66-74.
42. Kwak YH, Kwak DK, Kim NY, **Kim YJ**, Lim JS, Yoo JH. Significant changes in synovial fluid microRNAs after high tibial osteotomy in medial compartmental knee osteoarthritis: Identification of potential prognostic biomarkers. *PLoS One*. 2020 Jan 10;15(1):e0227596.
43. Lee MJ, Pak K, Kim JH, **Kim YJ**, Yoon J, Lee J, Lyoo CH, Park HJ, Lee JH, Jung NY. Effect of polygenic load on striatal dopaminergic deterioration in Parkinson disease. *Neurology*. 2019 Aug 13;93(7):e665-e674.
44. Byun JH, Cho H, **Kim YJ**, Kim JS, Baik JS, Jang S, Ma HI. Trends in the Prevalence of Drug-Induced Parkinsonism in Korea. *Yonsei Med J*. 2019 Aug;60(8):760-767.
45. Kim SI, Jeon B, Bae J, Won JK, Kim HJ, Yim J, **Kim YJ**, Park SH. An Autopsy Proven Case of CSF1R-mutant Adult-onset Leukoencephalopathy with Axonal Spheroids and Pigmented Glia (ALSP) with Premature Ovarian Failure. *Exp Neurobiol*. 2019 Feb;28(1):119-129
46. Yoon SH, Kim NY, **Kim YJ**, Lyoo CH. Novel Ferritin Light Chain Gene Mutation in a Korean Patient with Neuroferritinopathy. *J Mov Disord*. 2019 Jan;12(1):63-65
47. Park HY, Kwon MJ, Kang HS, **Kim YJ**, Kim NY, Kim MJ, Min KW, Choi KC, Nam ES, Cho SJ, Park HR, Min SK, Seo J, Choe JY, Lee HK. Targeted next-generation sequencing of well-differentiated rectal, gastric, and appendiceal neuroendocrine tumors to identify potential targets. *Hum Pathol*. 2019 May;87:83-94.
48. Min KW, Choe JY, Kwon MJ, Lee HK, Kang HS, Nam ES, Cho SJ, Park HR, Min SK, Seo J, **Kim YJ**, Kim NY, Kim HY. BRAF and NRAS mutations and antitumor immunity in Korean malignant melanomas and their prognostic relevance: Gene set enrichment analysis and CIBERSORT analysis. *Pathol Res Pract*. 2019 Dec;215(12):152671. PMID: 31630873.
49. Lee EJ, Oh MS, Kim JS, Chang DI, Park JH, Cha JK, Heo JH, Sohn SI, Kim DE, Kim HY, Kim J, Seo WK, Lee J, Park SW, **Kim YJ**, Lee BC; EMOTION investigators. Serotonin transporter gene polymorphisms may be associated with poststroke neurological recovery after escitalopram use. *J Neurol Neurosurg Psychiatry*. 2018 Mar;89(3):271-276.

50. Sung YH, Kim HJ, Koh SB, Kim JS, Kim SJ, Cheon SM, Cho JW, **Kim YJ**,, Ahn TB. Validation of the Korean Version of the Scales for Outcomes in Parkinson's Disease-Sleep. *J Korean Med Sci*. 2018 Jan 8;33(2):e14. doi: 10.3346/jkms.2018.33.e14
51. Yoo HS, Lee H, Chung SJ, Lee JS, Hong SK, Lee PH, **Kim YJ**, Sohn YH, Shin HW. A novel heterozygous ANO3 mutation with basal ganglia dysfunction in a patient with adult onset isolated segmental dystonia. *J Clin Neurol* 2018 14 596
52. **Kim YJ***, Jeon J, Shin J, Kim NY, Hong JH, Oh JM, Hong S, Kim YJ, Kim YE, Kang SY, Ma HI, Lee U, Yoon J*. Dysregulation of the causative genes for hereditary parkinsonism in the midbrain in Parkinson's disease. *Mov Disord*. 2017 Aug;32:1211-1220
53. Kim HK, Ki CS, **Kim YJ**, Lee MS. Radiological Findings of Two Sisters with Aceruloplasminemia Presenting with Chorea. *Clin Neuroradiol*. 2017 Sep;27(3):385-388
54. Hong JH, Kim YK, Park JS, Lee JE, Oh MS, Chung EJ, Kim JY, Sung YH, **Kim YJ***. Lack of association between LRRK2 G2385R and cognitive dysfunction in Korean patients with Parkinson's disease. *J Clin Neurosci*. 2017; 36:108-113.
55. Kim MK, Kim NY, Hong S, Ma HI, **Kim YJ***. Presynaptic Dopaminergic Degeneration in a Patient with Beta-Propeller Protein-Associated Neurodegeneration Documented by Dopamine Transporter Positron Emission Tomography Images: A Case Report. *J Mov Disord*. 2017 Sep;10(3):161-163.
56. Wang L, Heckman MG, Aasly JO, Annesi G, Bozi M, Chung SJ, Clarke C, Crosiers D, Eckstein G, Garraux G, Hadjigeorgiou GM, Hattori N, Jeon B, **Kim YJ**, Kubo M, Lesage S, Lin JJ, Lynch T, Lichtner P, Mellick GD, Mok V, Morrison KE, Quattrone A, Satake W, Silburn PA, Stefanis L, Stockton JD, Tan EK, Toda T, Brice A, Van Broeckhoven C, Uitti RJ, Wirdefeldt K, Wszolek Z, Xiromerisiou G, Maraganore DM, Gasser T, Krüger R, Farrer MJ, Ross OA, Sharma M; GEOPD Consortium. Evaluation of the interaction between LRRK2 and PARK16 loci in determining risk of Parkinson's disease: analysis of a large multicenter study. *Neurobiol Aging*. 2017 Jan;49:217.e1-217.e4. doi: 10.1016/j.neurobiolaging.2016.09.022
57. Kim JY, Song IU, Koh SB, Ahn TB, Kim SJ, Cheon SM, Cho JW, **Kim YJ**, Ma HI, Park MY, Baik JS, Lee PH, Chung SJ, Kim JM, Kim HJ, Sung YH, Kwon DY, Lee JH, Lee JY, Kim JS, Yun JY, Kim HJ, Hong JY, Kim MJ, Youn J, Kim JS, Oh ES, Yang HJ, Yoon WT, You S, Kwon KY, Park HE, Lee SY, Kim Y, Kim HT, Kim JS. Validation of the Korean Version of the Scale for Outcomes in Parkinson's Disease-Autonomic. *J Mov Disord*. 2017 Jan;10(1):29-34
58. Kim YJ, Kim HJ, **Kim YJ**, Seo SW. Synergistic effects of longitudinal amyloid and vascular changes on lobar microbleeds. *Neurology*. 2016; 87: 1575-82.
59. Lee CY, Kang SY, Hong SK, Ma HI*, Lee UJ*, **Kim YJ***. A validation study of a smartphone-based finger tapping application for the quantitative assessment of bradykinesia in Parkinson's disease. *PLoS ONE* 2016;11(7): e0158852.
60. Lee MW, Kim NY, Huh JY, Kim YE, **Kim YJ***,. Ataxia with oculomotor apraxia type I without oculomotor apraxia: A case report *J Clin Neurol*. 2016; 12: 126-8
61. Kim YJ. **Kim YJ**. . . . Na DL, Seo SW. Gray and white matter changes linking cerebral small vessel disease to gait disturbances. *Neurology* 2016 Mar 29;86(13):1199-207.
62. Jeon I, Cicchetti F, Cisbani G, Lee S, Li E, Bae J, Lee N, Li L, Im W, Kim M, Kim HS, Oh SH, Kim TA, Ko JJ, Aubé B, Oueslati A, **Kim YJ**, Song J. Human-to-mouse prion-like propagation of mutant huntingtin protein. *Acta Neuropathol*. 2016; 132:577-92
63. Lyoo CH, Cho H, Choi JY, Hwang MS, Hong SK, **Kim YJ**, Ryu YH, Lee MS. Tau Accumulation in Primary Motor Cortex of Variant Alzheimer's Disease with Spastic Paraparesis. *J Alzheimers Dis*. 2016 Feb 16;51(3):671-7.
64. Lee JH,, **Kim YJ**,, Lee MS. Clinical Heterogeneity of Atypical Pantothenate Kinase-Associated Neurodegeneration in Koreans. *J Mov Disord*. 2016 Jan;9(1):20-7.
65. Kim YE, Kim YH, Ma HY, **Kim YJ***. A Visual Rating Scale for the Hummingbird Sign with Adjustable Diagnostic Validity. *J Parkinsons Dis*. 2015;5(3):605-12. doi: 10.3233/JPD-150537.

66. Kim YH, Ma HY, **Kim YJ***. Utility of the diameter of the midbrain tegmentum in differential diagnosis of progressive supranuclear palsy from idiopathic Parkinson's disease. *J Clin Neurol*. 2015 Jul;11(3):268-74. doi: 10.3988/jcn.2015.11.3.268.
67. **Kim YJ***, Lyoo CH, Hong S, Kim NY, Lee MS. Neuroimaging studies and whole exome sequencing of PLA2G6-associated neurodegeneration in a family with intrafamilial phenotypic heterogeneity. *Parkinsonism Relat Disord*. 2015 Apr 21(4):402-406
68. Kim JH, Park J, Kim YH, Ma HI, **Kim YJ***. Characterization of cerebral microbleeds in idiopathic Parkinson's disease. *Eur J Neurol* 2015 Feb 22(2):377-83
69. Lisa Wang, Jan O. Aasly, Grazia Annesi, **Kim YJ**, et al. Large-scale assessment of polyglutamine repeat expansions in Parkinson disease. *Neurology* 2015 13;85(15):1283-92.
70. Ahn HJ, Yoo WK, Park J, Ma HI, **Kim YJ**. Cognitive Dysfunction in Drug-induced Parkinsonism Caused by Prokinetics and Antiemetics. *J Korean Med Sci* 2015 Sep;30(9):1328-33.
71. Lee MS, **Kim YJ**, Kim EJ, Lee MJ. Overlap of autism spectrum disorder and glucose transporter 1 deficiency syndrome associated with a heterozygous deletion at the 1p34.2 region.. *J Neurol Sci*. 2015 Sep 15;356(1-2):212-4.
72. Kim YJ, Kwon HK, Lee JM, Kim YJ, **Kim YJ**, Jung NY, Kim ST, Lee KH, Na DL, Seo SW. White matter microstructural changes in pure Alzheimer's disease and subcortical vascular dementia. *Eur J Neurol*. 2015 Apr;22(4):709-16.
73. Kim YJ, Cho H, **Kim YJ**, Ki CS, Chung SJ, Ye BS, Kim HJ, Kim JH, Kim ST, Lee KH, Jeon S1, Lee JM, Chin J, Kim JH, Na DL, Seong JK, Seo SW. Apolipoprotein E4 Affects Topographical Changes in Hippocampal and Cortical Atrophy in Alzheimer's Disease Dementia: A Five-Year Longitudinal Study. *J Alzheimers Dis* 2015 Jan 1;44(4): 1075-85
74. Choi HK, Choi Y, Kang H, Lim EJ, Park SY, Lee HS, Park JM, Moon J, **Kim YJ**, Choi I, Joe EH, Choi KC, Yoon HG. PINK1 positively regulates HDAC3 to suppress dopaminergic neuronal cell death. *Hum Mol Genet*. 2015 Feb 24(4):1127-41
75. Chu MK, Kim WC, Choi JM, Hong JH, Kang SY, Ma HI, **Kim YJ***. Analysis of dosage mutation in PARK2 among Korean patients with early-onset or familial Parkinson disease. *J Clin Neurol* 2014 Jul;10(3):244-8.
76. Chung EJ, Hong JH, Lee CS, Lee MS, **Kim YJ***. Expansion of the clinicopathological and mutational spectrum of Perry syndrome. *Parkinsonism Relat Disord*. 2014 20:388-93.
77. Lee DW, Eum SW, Moon CO, Ma HI, **Kim YJ***. Corticobasal syndrome associated with antiphospholipid syndrome without cerebral infarction. *Neurology*. 2014 Feb 25;82(8):730-1
78. **Kim YJ**, Ma HI, Lee EJ, Yoo WK. Asymmetrical changes of the pedunculopontine nucleus in a case of freezing of gait after carbon monoxide intoxication. *Clin Neurol Neurosurg* 2014 Oct;125:15-8
79. Chung SJ, Biernacka JM, **Kim YJ**, Wszolek ZK, Elbaz A, Maraganore DM et al., on behalf of the Genetic Epidemiology of Parkinson's Disease Consortium. Alpha-synuclein repeat variants and survival in Parkinson's disease. *Mov Disord*. 2014 Feb 29: 1053-7
80. Heckman MG, Elbaz A, Soto-Ortolaza, ...**Kim YJ**, ...Farrer MJ, Ross OA. Genetic Epidemiology Of Parkinson's Disease (GEO-PD) Consortium. The protective effect of LRRK2 p.R1398H on risk of Parkinson's disease is independent of MAPT and SNCA variants. *Neurobiol Aging*. 2014 Jan ;35(1):266.e5-14.
81. Heckman MG, Soto-Ortolaza AI, Aasly JO MJ, **Kim YJ** Ross OA; Genetic Epidemiology of Parkinson's Disease (GEO-PD) Consortium. Population-specific frequencies for LRRK2 susceptibility variants in the Genetic Epidemiology of Parkinson's Disease (GEO-PD) Consortium. *Mov Disord*. 2013 Oct;28(12):1740-4.
82. Kang SY, Ma HI, Lim YM, Hwang SH, **Kim YJ**. Fatigue in drug-naive Parkinson's disease. *Eur Neurol*. 2013;70(1-2):59-64.

83. Choi JM, Kim WC, Lyoo CH, Kang SY, Lee PH, Baik JS, Koh SB, Ma HI, Sohn YH, Lee MS, **Kim YJ ***. Association of mutations in the glucocerebrosidase gene with Parkinson disease in a Korean population. **Neurosci Lett** 2012 514:12-15
84. Kim JH, Park J, Yu KH, Ma HI, Lee BC, Kim I, **Kim YJ ***. Prodromal dementia with Lewy bodies manifesting as sertraline-induced parkinsonism: A case report. **Alzheimer disease & associated disorders** 2012; 26: 191-3
85. Kang SY, Ma HI, Lim YM, Jeong MG, **Kim YJ**, Kwon SB, Hwang SH. Normal diffusion-weighted MRI during the acute stage of central pontine myelinolysis. *Int J Neurosci*. 2012 Aug;122(8):477-9
86. Oh YS, Ma HI, **Kim YJ**, Kim JS. Task-specific tremor with use of scissors. *Mov Disord*. 2012 Jun;27(7):921-2.
87. Choi JM, Hong JH, Chae MJ, Hung NP, Kang HS, Ma HI, **Kim YJ ***. Analysis of mutations and the association between polymorphisms in the cerebral dopamine neurotrophic factor (CDNF) gene and Parkinson disease. **Neurosci Lett**. 2011 Apr 493(3):97-101
88. Kim HL, Do JY, Cho HJ, Jeon YC, Park SJ, Ma HI, Song JH, Lee Y, Choi H, Choi KC, Kim YS, Zerr I, Kallenberg K, **Kim YJ ***. Dura mater-associated Creutzfeldt-Jakob disease: The first case in Korea. **J Kor Med Sci** 2011 Nov; 26: 1515-17
89. Association of LRRK2 exonic variants with susceptibility to Parkinson's disease: a case-control study. Ross OA, Sato-Otolaza AI, Heckman MG,**Kim YJ**,.....Wszolek ZK, Wu RM, Farrer M. **Lancet Neurol** 2011 Oct;10:898-908
90. Ipsilateral tilt and contralateral sensory change of neck in cortical infarction. Kang SY, Ma HI, Kwon SB, Jung S, **Kim YJ**, Hwang SH. **J Clin Neurol** 2011; 7: 156-158
91. Long-term prognosis of symptomatic isolated middle cerebral artery disease in Korean stroke patients. Oh MS, Yu KH, Chu MK, Ma HI, Kim YJ, **Kim JY**, Lee BC. **BMC Neurology** 2011; 11:138
92. Kim HJ,... **Kim YJ**, .. Kim HJ.SCA in Korea and its regional distribution: a multicenter analysis. *PRD*. 2011 Jan;17(1):72-5. doi: 10.1016/j.parkreldis.2010.09.006.
93. Sung YH, Park KH, Lee YB, Park HM, Shin DJ, Park JS, Kang SY, Oh MS, Ma HI, Yu KH, **Kim YJ***, Lee BC. Midbrain atrophy in subcortical ischemic vascular dementia. **J Neurol** 2009 256 1997
94. Ma HI, Kim JH, Chu MK, Oh MS, Yu KH, Kim J, Hahn W, **Kim YJ***, Lee BC. Diabetes mellitus and drug-induced parkinsonism: a case-control study. **J Neurol Sci** 2009;284:140-3
95. Choi JM, Woo MS, Ma HI, Kang SY, Sung YH, Yong SW,, Sohn YH, Kim JH, Kim JW, Lee MS, Lee MC, Kim DH, Kim YJ*. Analysis of PARK genes in a Korean cohort of early-onset Parkinson disease. **Neurogenetics** 2008 9 263-9
96. Lee PH, Yeo SH, Yong SW, **Kim YJ**. Odor identification test and its relation with cardiac 123I-MIBG in patients with drug-induced parkinsonism. **J Neurol Neurosurg Psychiatry**. 2007 78 1250
97. **Kim YJ**, Sapp E, Cuiffo B, Sobin L, Yoder J, Kegel KB, Qin ZH, Detloff P, Aronin N, DiFiglia M. Lysosomal proteases are involved in generation of N-terminal huntingtin fragments. **Neurobiol Dis** 2006 22 346-356
98. Kim Y, Kim JH, Nam YJ, Kong M, **Kim YJ**, Yu KH, Lee BC, Lee C. Klotho is a genetic risk factor for ischemic stroke caused by cardioembolism in Korean females. **Neurosci Lett**. 2006 407(3) 189-94
99. Kim Y, Kim JH, Nam YJ, **Kim YJ**, Yu KH, Lee BC, Lee C. Sequence variants of ACE, AGT, AT1R, and PAI-1 as genetic risk factors for vascular dementia. **Neurosci lett** 2006 401 276-9
100. Kegel KB, Sapp E, Yoder J, Cuiffo B, Sobin L, **Kim YJ**, Qin ZH, Hayden MR, Aronin N, Scott D, Isenberg G, Goldmann WH, DiFiglia M. Huntingtin associates with acidic phospholipids at the plasma membrane. **J Biol Chem**. 2005 Oct 280 36464-36473
101. Tatsuro T, Ballinger JR, Vines D, **Kim YJ**, Utsunomiya K, Lang AE, Ichise M. Reproducibility of dopamine transporter density measured with 123I-FPCIT SPECT in normal control and Parkinson's disease patients. **Ann Nucl Med** 2004;18: 609-616

102. Kim M, Roh H-K, Yoon BW, Kang L, Kim YJ, Aronin N, DiFiglia M. Huntingtin is degraded to small fragments by calpain after ischemic injury. 2003 **Exp Neurol** ; 183:109-115
103. Kegel KB, Meloni AR, Yi Y, Kim YJ, Doyle E, Cuiffo BG, Sapp E, Wang Y, Qin ZH, Chen JD, Nevins JR, Aronin N, DiFiglia M. Huntingtin is present in the nucleus, interacts with the transcriptional corepressor C-terminal binding protein, and represses transcription. **J Biol Chem**. 2002;277(9):7466-76.
104. Kim YJ, Ichise M, Ballinger JR, Vines D, Erami SS, Tatschida T, Lang AE. Combination of dopamine transporter and D2 receptor SPECT in the diagnostic evaluation of PD, MSA, and PSP. **Mov Disord**. 2002;17(2):303-12.
105. Kim YJ, Yi Y, Sapp E, Wang Y, Cuiffo B, Kegel KB, Qin ZH, Aronin N, DiFiglia M. Caspase 3-cleaved N-terminal fragments of wild-type and mutant huntingtin are present in normal and Huntington's disease brains, associate with membranes, and undergo calpain-dependent proteolysis. **Proc Natl Acad Sci U S A**. 2001;98(22):12784-9.
106. Ashby P, Kim YJ, Kumar R, Lang AE, Lozano AM. Neurophysiological effects of stimulation through electrodes in the human subthalamic nucleus. **Brain**. 1999;122:1919-31.
107. Kim YJ, Pakiam AS, Lang AE. Historical and clinical features of psychogenic tremor: a review of 70 cases. **Can J Neurol Sci**. 1999;26(3):190-5.
108. Ichise M, Kim YJ, Ballinger JR, Vines D, Erami SS, Tanaka F, Lang AE. SPECT imaging of pre- and postsynaptic dopaminergic alterations in L-dopa-untreated PD. **Neurology**. 1999;52(6):1206-14.
109. Ichise M, Kim YJ, Erami SS, Ballinger JR, Vines D, Tanaka F, Lang AE. Functional morphometry of the striatum in Parkinson's disease on three-dimensional surface display of 123I-beta-CIT SPECT data. **J Nucl Med**. 1999;40(4):530-8.
110. Kumar R, Lozano AM, Kim YJ, Hutchison WD, Sime E, Halket E, Lang AE. Double-blind evaluation of subthalamic nucleus deep brain stimulation in advanced Parkinson's disease. **Neurology**. 1998;51(3):850-5.

Environmental and Related Factors as a Trigger for Synucleinopathy

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In human studies, clinically diagnosed Parkinson's disease (PD) has been associated with exposure to environmental toxicants including certain pesticides, chlorinated solvents and air pollution as well as with life style factors such as physical inactivity. Specific pesticides implicated include paraquat, rotenone, 2,4-D, organochlorine pesticides and glyphosate. Residential as well as occupational exposure has been implicated, including living near to areas where pesticides have been applied. In a few human studies, alpha-synuclein abnormality has been confirmed by biomarker or postmortem exam, but in most cases synuclein pathology has been assumed based on clinical features. In the laboratory, many of these toxicants have been shown to cause alpha-synuclein pathology. Occupational exposure and exposure due to residence near to areas where pesticides have been applied (agriculture, golf courses) are associated with greater risk of PD. Pesticides associated with PD, including paraquat, rotenone, 2,4-D and several organochlorines, cause experimental parkinsonism in laboratory studies, including alpha-synuclein-specific models. Genetically determined characteristics, such as impairment in detoxification, can increase the effect of pesticide exposure, an example of gene-environment interaction. In contrast, behaviors such as use of personal protective equipment when mixing and applying pesticides or eating a healthy diet may reduce the risk of PD associated with pesticide exposure. Exposure to chlorinated solvents (trichloroethylene, perchloroethylene, carbon tetrachloride), commonly used industrial solvents used in dry cleaning, in many industrial processes as degreasers, has been associated with increased risk of PD in humans and cause parkinsonism-associated toxicity in animal models. Importantly, these chemicals are environmentally persistent, and can continue to contaminate soil and water for decades after use. In the US, trichloroethylene is in continued use, and can be found in nearly one third of US drinking water supplies, as well as in air, soil, food and human breast milk. Trichloroethylene (TCE) is a volatile organic compound, and can concentrate in the air in buildings near to contaminated sites. Clusters of PD have been associated with environmental TCE exposure. Occupational exposure to these chemicals is also associated with greater risk of PD. Welders exposed to manganese in welding fumes may be at greater risk of PD and asymptomatic reductions in dopamine function in imaging studies. Air pollution, and most specifically PM 2.5 has been associated with greater risk of Parkinson's disease, and alpha-synuclein abnormalities in animal models and in humans at post mortem. This lecture will provide an overview of environmental factors associated with synucleinopathies in human populations and explore studies of the combined effects of environmental factors, as well as the effects of environmental plus genetic factors, as triggers for synucleinopathy.

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Education / Training

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Rush-Presbyterian-St. Luke's Medical Center, Chicago, Illinois	Residency & Fellowship	1976-1982	Neurology, Movement Disorders, Clinical Neuropharmacology
University of California, Berkeley School of Public Health	PhD	1990-1998	Environmental Health Sciences, Epidemiology

Positions and Honors

Professional Experience

Current

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Previous

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2013 - 2020 Director, Parkinson's Disease Research Education and Clinical Center, San Francisco Veterans Affairs Medical Center

1990 - 2013 Director of Clinical Research, Senior Scientist, The Parkinson's Institute & Clinical Center, Sunnyvale, CA

1988 - 1992 Associate Professor, Rush University, Chicago, IL

1982 - 1988 Assistant Professor, Rush University, Chicago, IL

Honors

2012	Movement Disorders Research Award, American Academy of Neurology
2015	White House Champions of Change for Parkinson's Disease
2020	International Parkinson's Disease and Movement Disorders Society Honorary Membership Award
2020	Tom Isaacs Award, Cure Parkinson's Trust & Van Andel Institute
2020	Robert A. Pritzker Prize, Michael J Fox Foundation
2021	Robert Wartenberg Lecture Award, American Academy of Neurology
2021	Advocacy Award, Parkinson's Policy Forum
2023	Stanley Fahn Lecture Award, International Parkinson's Disease and Movement Disorders Society
2025	Melvin Yahr Lecture Award, International Association of Parkinsonism and Related Disorders

Selected Peer Reviewed Publications

1. **Tanner CM**, Ottman R, Goldman SM, Ellenberg J, Chan P, Mayeux R, Langston JW. Parkinson's disease in twins: an etiologic study. *JAMA* 1999;281(4):341-46. PMID:9929087
2. Ross GW, Abbott RD, Petrovitch H, **Tanner CM**, Davis DG, Nelson J, Markesbery WR, Masaki K, Launer L, White LR. Association of olfactory dysfunction with incidental Lewy bodies. *Movement Disorders*. *Movement Disorders* 2006;21(12):2062-2067. PMID:16991138

Shin C, Han JY, Kim SI, Park SH, Yang HK, Lee HJ, et al. In vivo and autopsy validation of alpha-synuclein seeding activity using RT-QulC assay in the gastrointestinal tract of patients with Parkinson's disease. *Parkinsonism Relat Disord*. 2022 Oct;103:23-28.
3. **Tanner CM**, Kamel F, Ross GW, Hoppin JA, Goldman SM, Korell M, Marras C, Bhudhikanok GS, Kasten M, Chade AR, Comyns K, Richards M, Meng C, Priestley B, Fernandez HH, Cambi F, Umbach DM, Blair A, Sandler DP, Langston JW. Rotenone, Paraquat and Parkinson's Disease. *Environ Health Perspect* 2011 Jan 26. PMID:21269927; PMCID: PMC3114824

Goldman SM, Marek K, Ottman R, Meng C, Comyns K, Chan P, Ma J, Marras C, Langston JW, Ross GW, **Tanner CM**. Concordance for Parkinson's disease in twins: A 20-year update. *Ann Neurol*. 2019 Feb 20. PMID: 30786044
5. **Tanner CM**, Cummings SR, Schwarzschild MA, Brown EG, Dorsey ER, Espay AJ, Galifianakis NB, Goldman SM, Litvan I, Luthra N, McFarland NR, Mitchell KT, Standaert DG, Bauer DC, Greenspan SL, Beck JC, Lyles KW. The TOPAZ study: a home-based trial of zoledronic acid to prevent fractures in neurodegenerative parkinsonism. *NPJ Parkinsons Dis*. 2021 Mar 01; 7(1):16. PMID: 33649343. PMCID: PMC7921548
6. Goldman SM, Weaver FM, Stroupe KT, Cao L, Gonzalez B, Colletta K, Brown EG, **Tanner CM**. Risk of Parkinson Disease Among Service Members at Marine Corps Base Camp Lejeune. *JAMA Neurol*. 2023 May 15. PMID: 37184848. PMCID: PMC10186205
7. Brown EG, Chahine LM, Siderowf A, Gocharanour C, Kurth R, Marshall MJ, Caspell-Garcia C, Brumm MC, Stanley CE, Korell M, McMahon B, Kuhl M, Fabrizio K, Heathers L, Concha-Marambio L, Soto C, Chowdhury S, Coffey CS, Foroud TM, Simuni T, Marek K, **Tanner CM**, Parkinson Progression Marker Initiative. Staged Screening Identifies People with Biomarkers Related to Neuronal Alpha-Synuclein Disease. *Ann Neurol*. 2024 Dec 24. PMID: 39719857

Metabolic Factors Contributing to Risk and Progression of Parkinson's Disease

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Parkinson's disease (PD) is a progressive neurodegenerative disorder driven by a complex interplay of aging, genetic susceptibility, and environmental exposures. Mounting evidence now implicates metabolic dysfunction as an additional, modifiable contributor to both PD risk and progression. This is of particular importance in Asia, where the prevalence of metabolic diseases—including diabetes, obesity, dyslipidemia, and hypertension—is rising rapidly, offering potential opportunities for earlier intervention.

Epidemiological studies have demonstrated consistent associations between metabolic syndrome and increased PD risk. Individuals with metabolic syndrome (defined by ≥ 3 metabolic risk factors) face approximately a 40% higher risk of developing PD compared to those without the syndrome. Even individual components such as hypertension, central obesity, and impaired fasting glucose are independently associated with elevated PD risk in meta-analyses. Among these factors, type 2 diabetes stands out as a particularly strong and independent risk factor. Importantly, both diabetes and metabolic syndrome have been linked to faster motor and cognitive decline in patients with PD. Notably, in advanced stages of the disease, unintentional weight loss, sarcopenia, orthostatic hypotension, and frailty frequently emerge, contributing to falls, hospitalization, and increased mortality.

Mechanistic insights from basic and translational studies provide biological plausibility for these associations. Metabolic disturbances such as insulin resistance (a hallmark of type 2 diabetes), mitochondrial dysfunction, chronic neuroinflammation, and altered lipid metabolism converge to amplify PD pathogenesis. Insulin resistance impairs cellular energy signaling and promotes oxidative stress and α -synuclein aggregation, while metabolic inflammation may exacerbate microglial activation. In parallel, altered lipid metabolism can disrupt membrane homeostasis, lipid-protein interactions, and lysosomal pathways relevant to neurodegeneration. Metabolic comorbidities are also associated with cerebral small vessel disease, which may compound neurodegeneration through chronic hypoperfusion and disruption of dopaminergic and non-dopaminergic networks.

This integrated view of systemic and brain health opens promising translational opportunities. If metabolic dysfunction contributes to PD pathophysiology, targeting metabolic pathways could delay disease onset or modify its course. Clinical and experimental data support further exploration of GLP-1 receptor agonists (e.g., exenatide, lixisenatide), metformin, and other metabolic therapies for their potential neuroprotective effects. In parallel, lifestyle interventions—including structured exercise programs, weight management, and dietary modification—represent low-cost, broadly applicable strategies that may improve metabolic resilience and brain health.

In conclusion, addressing metabolic dysfunction may offer a powerful adjunctive strategy in PD management. By targeting modifiable systemic risk factors alongside dopaminergic therapies, clinicians and researchers may not only improve patient outcomes but also help reshape the disease trajectory. This emerging intersection between metabolic and neurodegenerative pathways underscores the importance of treating the whole patient—not just the brain.

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Education / Training

Institution and Location	Degree	Year(s)	Field of Study
Universiti Kebangsaan Malaysia	MD		Medicine
University of Malaya	PhD		Neuroscience/Parkinson
Royal College of Physician Edinburgh	FRCP (Edin)		Medicine

Positions and Honors

Professional Experience

Current

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Selected Peer Reviewed Publications

1. Tan AH, Lim SY, Lang AE. The microbiome–gut–brain axis in Parkinson disease - from basic research to the clinic. *Nature Reviews Neurology*. 2022; 18:476-495
2. Tan AH, Chong CW, Lim SY, et al. Gut microbial ecosystem in Parkinson s disease: New clinico-biological insights from multi-omics. *Annals of Neurology*. 2021; 89:546-559
3. Tan AH, Lim SY, Chong KK, et al. Probiotics for constipation in Parkinson s disease: A randomized placebo-controlled study. *Neurology*. 2021;96: e772-e782
4. Tan AH, Lim SY, Mahadeva S, et al. Helicobacter pylori eradication in Parkinson's disease: a randomized placebo-controlled trial. *Movement Disorders*. 2020;35(12):2250-2260
5. Tan AH, Hew YC, Lim SY, et al. Altered body composition, sarcopenia, frailty, and their clinico-biological correlates in Parkinson's disease. *Parkinsonism & Related Disorders*. 2018; 56:58-64

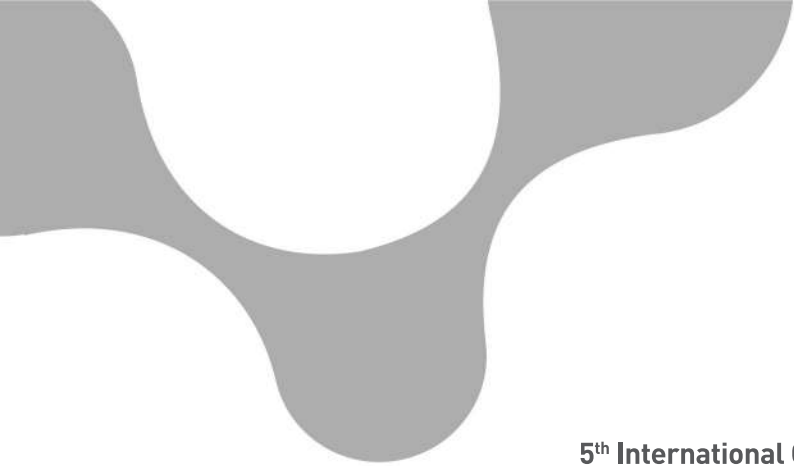
6. Tan AH, Saffie-Awad P, Schumacher Schuh AF, et al. Global Parkinson's Genetic Program (GP2). Global Perspectives on Returning Genetic Research Results in Parkinson Disease. *Neurol Genet*. 2024 Dec 5;10(6):e200213.
7. Tan AH, Cornejo-Olivas M, Okubadejo N, et al. Genetic testing for Parkinson's Disease and Movement Disorders in Less Privileged Areas: Barriers and Opportunities. *Movement Disorder Clinical Practice*. 2023; 11: 14-20



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5th International Conference of Korean Movement Disorder Society

**Bridging from the Origin to Clinical Practice:
A Beginning Has an Ending**



ICKMDS 2025

5th International Conference of Korean Movement Disorder Society

Bridging from the Origin to Clinical Practice:
A Beginning Has an Ending

DAY 1_ October 31 (Fri), 2025

Room A

Plenary Session 2

When and Where Does Synucleinopathy Begin?
(Parkinson's Disease Perspective)

Chairs Nobutaka HATTORI (Japan)
Tae-Beom AHN (Korea)



Neuroanatomical Origins and Clinical Landscape of Parkinson's Disease

Chaewon SHIN

Department of Neurology, Seoul National University Bundang Hospital, Republic of Korea

The neuroanatomical origin of Parkinson's disease (PD) refers to the initial site of Lewy pathology (LP), the pathognomonic hallmark of PD, and the subsequent propagation pattern of this pathology. Traditionally, the brainstem nuclei, as described in the Braak staging system proposed in 2003, were considered the initial sites within the brain. However, accumulating evidence has since pointed to alternative origins, including the olfactory bulb and other regions of the brain. In parallel, growing attention has been directed toward the peripheral nervous system, particularly the gastrointestinal tract, as a possible starting point of LP in PD. This has fueled ongoing debate over whether LP progresses from the gut to the brain or vice versa. This lecture aims to provide a comprehensive overview of the current evidence regarding the neuroanatomical origin of PD from a pathological perspective. By integrating recent advances and diverse findings, this presentation highlights the heterogeneity of PD pathogenesis and offers insights that may guide future diagnostic and therapeutic approaches.

Personal Information

Full Name Chaewon SHIN
Current Position Associate Professor
Department Department of Neurology
Organization Seoul National University Bundang Hospital
Country Republic of Korea



Education / Training

Institution and Location	Degree	Year(s)	Field of Study
Seoul National University, Republic of Korea	B.Sc	2006	
Seoul National University, Republic of Korea	M.Sc	2015	Neuroscience, Movement Disorders
Seoul National University, Republic of Korea	Ph.D	2018	Translational Medicine, Movement Disorders

Positions and Honors

Professional Experience

Current

2025- Clinical Associate Professor, Department of Neurology, Seoul National University Bundang Hospital, Seongnam-si, Republic of Korea

Previous

2019-2024 Assistant/Associate Professor, Department of Neurology, College of Medicine, Chungnam National University; Chungnam National University Sejong Hospital, Sejong-si, Republic of Korea

2017-2019 Clinical Assistant Professor, Department of Neurology, Kyung Hee University Hospital, Seoul, Republic of Korea

Honors

2023 Travel Grant Award (Complimentary Registration), International Congress of Parkinson's Disease and Movement Disorders, Copenhagen, Denmark

2023 Outstanding Oral Presentation Award, the Korean Movement Disorder Society Annual Symposium, Seoul, Korea

Selected Peer Reviewed Publications

1. Shin C, Kim SI, Park SH, Kim JM, Lee JY, Chung SJ, et al. Diagnostic accuracy and predictors of alpha-synuclein accumulation in the gastrointestinal tract of Parkinson's disease. *NPJ Parkinsons Dis.* 2024;10(1):155.
2. Shin C, Kim SI, Park SH, Shin JH, Lee CY, et al. Absence of alpha-synuclein pathology in the stomach of a patient with prodromal dementia with Lewy bodies. *J Mov Disord.* 2023 May;16(2):213-216.
3. Shin C, Han JY, Kim SI, Park SH, Yang HK, Lee HJ, et al. In vivo and autopsy validation of alpha-synuclein seeding activity using RT-QulC assay in the gastrointestinal tract of patients with Parkinson's disease. *Parkinsonism Relat Disord.* 2022 Oct;103:23-28.
4. Shin C, Lim Y, Lim H, Ahn T-B. Plasma Short-Chain Fatty Acids in Patients With Parkinson's Disease. *Movement Disorders.* 2020 Jun;35(6):1021-1027
5. Shin C, Park SH, Yun JY, Shin JH, Yang HK, Lee HJ, et al. Gastric Synucleinopathy as Prodromal Pathologic Biomarker in idiopathic REM Sleep Behavior Disorder. *J Neurol Neurosurg Psychiatry.* 2020 Nov 13;jnnp-2020-324743.
6. Shin C, Yang H-K, Park S, Lee H-J, Kong S-H, Suh Y-S, et al. Prospective cohort study of patients with early gastric cancer to detect prodromal Parkinson disease (EGC-PPD): A study protocol and baseline characteristics. *Journal of Clinical Neuroscience.* 2019 Nov;26(11):1417-1420.
7. Shin C, Park SH, Yun JY, Shin JH, Yang HK, Lee HJ, et al. Fundamental limit of alpha-synuclein pathology in gastrointestinal biopsy as a pathologic biomarker of Parkinson's disease: Comparison with surgical specimens. *Parkinsonism & related disorders.* 2017;44:73-7.

Unraveling the Clinical Origins of Parkinson's Disease: Insights into IRBD as the Prodromal Stage

Yun Kwok WING

Department of Psychiatry, The Chinese University of Hong Kong, Hong Kong

As global populations age, neurodegenerative diseases, particularly synucleinopathies, have garnered significant attention regarding early diagnosis, intervention, and prevention during the prodromal stages. The complex multifactorial etiology, heterogeneous neuropathology, and unclear progression trajectories of synucleinopathies pose considerable challenges to effective early intervention.

This talk will explore current knowledge on identifying at-risk and prodromal populations for synucleinopathies, focusing specifically on REM sleep behavior disorder (RBD) as a critical clinical prodromal marker. Innovative study designs, such as familial cohort studies of RBD, alongside novel technologies like digital-based screening and emerging biomarkers such as gut microbiota, hold promise for cost-effective community screening of at-risk individuals. Additionally, a deeper understanding of long-term disease trajectories and clinically relevant heterogeneities of RBD may enhance personalized prevention and intervention strategies. Despite recent advances in elucidating the pathophysiology of RBD and synucleinopathies, the efficacy of existing disease-modifying therapies remains limited. There is a timely need for the development of novel screening, monitoring, and interventional therapies and active clinical trials targeting high-risk populations, especially RBD and prodromal RBD, who are at the early and potentially preventable stages of the synucleinopathy neurodegeneration.

Personal Information

Full Name Yun Kwok WING
Current Position Chairman & Choh Ming Li Professor of Psychiatry
Department Department of Psychiatry
Organization The Chinese University of Hong Kong
Country Hong Kong



Education / Training

Institution and Location	Degree	Year(s)	Field of Study
CUHK & Hong Kong	MB.ChB (Hons)	1986	Medicine
United Kingdom	MRCPsych (UK)	1992	Psychiatry
Hong Kong	FKCPSych	1993	Psychiatry
United Kingdom	FRCPSych (UK)	2007	Psychiatry

Positions and Honors

Professional Experience

Current

2017 - now Chairman & Professor, Department of Psychiatry, CUHK
2020 - now Direct of the Li Chiu Kong Family Sleep Assessment Unit, CUHK

Previous

2023 - 2025 Honorary Professor, Department of Psychology, HKU
2018 - 2021 Associate head, New Asia College, CUHK
2011 - 2019 Associate Dean (Student affairs), Faculty of Medicine, CUHK
2004 - 2011 Assistant Dean (Student affairs), Faculty of Medicine, CUHK

Honors

2023 - now Choh-Ming Li Professor of Psychiatry, CUHK
2016 Awarded by Chinese Sleep Research Society for distinguished contributions to the development of sleep medicine and sleep research
2010 Awarded the distinguished national award for Sleep Medicine Scientific Technological Advance in China by the Chinese Medical Doctor Association

Selected Peer Reviewed Publications

1. Tang S, Huang B, Wang YL, Liu YP, Wang J, Zhou L, Gong SY, Yang YH, Chan JWY, Chau SWH, Chu WCW, Abrigo J, Gagnon JF, **Wing YK**. Brain-clinical biotyping in patients with idiopathic REM sleep behavior disorder. *NPJ Parkinsons Disease*. 2025 Jun 7; 11(1):156.
2. Yang Y, Wang J, Zhou L, Liu Y, Tang S, Gong S, Li N, He Z, Ni J, Chan JWY, Chau SWH, **Wing YK**, Huang B. Sleep related injury and its correlates in isolated rapid eye movement sleep behavior disorder. *Sleep Med*. 2025 Feb;126:9-18.
3. Wang J, Huang B, Zhou L, Tang S, Feng H, Chan JWY, Chau SWH, Zhang J, Li SX, Mok V, **Wing YK**, Liu Y. Visuospatial dysfunction predicts dementia-first phenocconversion in isolated REM sleep behaviour disorder. *J Neurol Neurosurg Psychiatry*. 2024 Dec 16;96(1):76-84.
4. Yau CN, Hung JTS, Campbell RAA, Wong TCY, Huang B, Wong BTY, Chow NKN, Zhang L, Tsoi EPL, Tan Y, Li JJX, **Wing YK**, Lai HM. INSIHGT: an accessible multi-scale, multi-modal 3D spatial biology platform. *Nat Commun*. 2024 Dec 30;15(1):10888.
5. Huang B, Chau SWH, Liu Y, Chan JWY, Wang J, Zhang J, Ma SL, Chan PKS, Yeoh, YK, Chen Z, Wong, SH, Mok VCT, To KF, Lai HM, Ng S, Trenkwalder C, Chan FKL, **Wing YK**. Gut microbiome dysbiosis across early Parkinson's disease, REM sleep behavior disorder and their first-degree relatives. *Nature Communications*. 2023;14(1): 2501.
6. Feng H, Yang L, Liang YY, Ai S, Liu Y, Liu Y, Jin X, Lei B, Wang J, Zheng N, Chen X, Chan JWY, Sum RKW, Chan NY, Tan X, Benedict C, **Wing YK**, Zhang J. Associations of timing of physical activity with all-cause and cause-specific mortality in a prospective cohort study. *Nat Commun*. 2023 Feb 18;14(1):930.
7. Wang J, Lam SP, Huang B, Liu Y, Zhang J, Yu MWM, Tsang JCC, Zhou L, Chau SWH, Chan NY, Chan JWY, Schenck CH, Li SX, Mok VCT, Ma KKY, Chan AYY, **Wing YK**. Familial α -synucleinopathy spectrum features in patients with psychiatric REM sleep behaviour disorder. *J Neurol Neurosurg Psychiatry*. 2023 Nov;94(11):893-903.
8. Lee WJ, Baek SH, Im HJ, Lee SK, Yoon JE, Thomas RJ, **Wing YK**, Shin C, Yun CH. REM Sleep Behavior Disorder and Its Possible Prodromes in General Population: Prevalence, Polysomnography Findings, and Associated Factors. *Neurology*. 2023 Dec 4;101(23):e2364-e2375.
9. Liu Y, Zhang J, Chau SWH, Man Yu MW, Chan NY, Chan JWY, Li SX, Huang B, Wang J, Feng H, Zhou L, Mok V, **Wing YK**. Evolution of Prodromal REM Sleep Behavior Disorder to Neurodegeneration: A Retrospective Longitudinal Case-Control Study. *Neurology*. 2022 Aug 9;99(6):e627-e637.
10. Liu Y, Zhang J, Lam SP, Zhou J, Yu MWM, Li SX, Chan JWY, Postuma RB, Mok VCT, **Wing YK**. A case-control-family study of idiopathic rapid eye movement sleep behavior disorder. *Ann Neurol*. 2019 Apr;85(4):582-592.

P2-3

Cellular Origins of Parkinson's Disease and Emerging Therapeutic Strategies

Lorraine KALIA

Department of Medicine (Neurology), University of Toronto, Canada

Parkinson's disease is a complex and heterogeneous neurodegenerative disorder, with emerging evidence pointing to diverse cellular origins and disease mechanisms. These include distinct contributions from dopaminergic neurons, glial cells, and peripheral systems, such as the gut and immune system. This plenary lecture will explore how advances in understanding the cellular and molecular underpinnings of Parkinson's disease are reshaping the landscape of disease-modifying therapy development. A focus will be placed on key cellular pathways implicated in the pathobiology of Parkinson's disease, including protein aggregation, lysosomal dysfunction, mitochondrial stress, and neuroinflammation, and how they inform current therapeutic strategies. The lecture will also provide an overview of recently completed and ongoing clinical trials targeting these mechanisms, particularly those in phase 2 and 3 development. The objective is for attendees to gain a clearer understanding of how evolving insights into the cellular origins of Parkinson's disease are guiding translational efforts and what this may mean for clinical practice in the coming years.

Personal Information

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Current Position Associate Professor
Department Medicine (Neurology)
Organization University of Toronto
Country Canada



Education / Training

Institution and Location	Degree	Year(s)	Field of Study
University of Toronto (Canada)	Postdoctoral fellowship	2011-2013	Movement disorders
Harvard University (United States)	Postdoctoral fellowship	2009-2010	Parkinson's disease
University of Toronto (Canada)	FRCPC	2006-2011	Neurology
University of Toronto (Canada)	MD/PhD	1997-2006	Neuroscience
Queen's University (Canada)	BSc Honours	1993-1997	Biology

Positions and Honors

Professional Experience

Current

2021-Present Senior Scientist, Krembil Research Institute, University Health Network (Canada)
2020-Present Associate Professor, Department of Medicine (Neurology), University of Toronto (Canada)
2013-Present Staff Neurologist, Department of Medicine (Neurology), University Health Network & Sinai Health System (Canada)
2013-Present Movement Disorders Neurologist, Edmond J. Safra Program in Parkinson's Disease & Morton and Gloria Shulman Movement Disorders Clinic, Toronto Western Hospital, University Health Network (Canada)

Previous

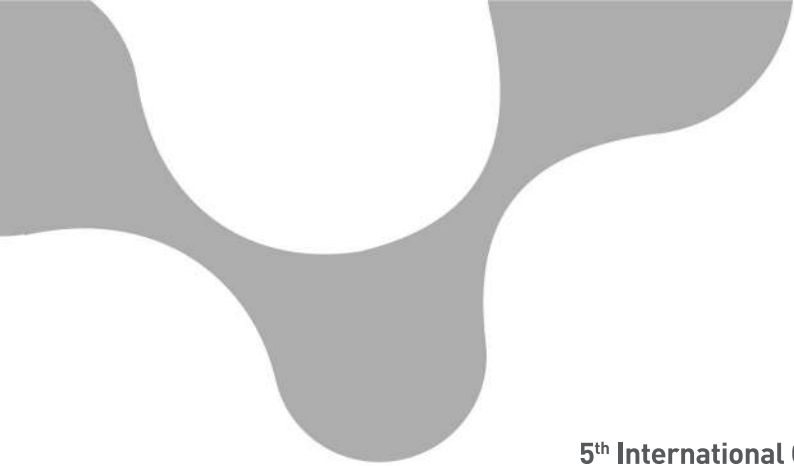
2013-2021 Scientist, Krembil Research Institute, University Health Network (Canada)
2013-2020 Assistant Professor, Department of Medicine (Neurology), University of Toronto (Canada)

Honors

2023-Present	Wolfond-Krembil Chair in Parkinson's Disease Research, University of Toronto
2023-Present	Vice Chair, Basic Science Special Interest Group (SIG) Steering Committee, International Parkinson and Movement Disorder Society (MDS)
2021-Present	Vice Chair, Scientific Issues Committee, International Parkinson and Movement Disorder Society (MDS)
2020-Present	Treasurer, Canadian Movement Disorder Society (CMDS)

Selected Peer Reviewed Publications

1. Kalia L. V., Arbour N., Bar-Or A., Bove R., Di Luca D. G., Fon E. A., Fox S., Gan-Or Z., Gommerman J. L., Kang U. J., Klawiter E. C., Koch M., Kolind S., Lang A. E., Lincoln M. R., MacDonald P. A., McKeown M. J., Mestre T. A., Miron V. E., Ontaneda D., Rousseaux M. W. C., Schlossmacher M. G., Schneider R., Stoessl A. J., Oh J. Disease-modifying therapies for Parkinson disease: lessons from multiple sclerosis. *Nature Reviews Neurology*, 2024.
2. Kalia L. V., Berg D., Kordower J. H., Shannon K. M., Taylor J. P., Cardoso F., Goldman J. G., Jeon B., Meissner W. G., Tijssen M. A. J., Burn D. J., Fung V. S. C. MDS viewpoint on biological frameworks of Parkinson's disease: current status and future directions. *Movement Disorders*, 2024.
3. Burre J., Edwards R., Halliday G., Lang A. E., Lashuel H., Melki R., Murayama S., Outeiro T. F., Papa S. M., Stefanis L., Woerman A. L., Surmeier D. J., Kalia L. V., Takahashi R. & the MDS Scientific Issues Committee. Discussion of research priorities on the role of alpha-synuclein in Parkinson's disease. *Movement Disorders*, 2024.
4. Nim S. *, O'Hara D. M. *, Corbi-Verge C., Perez-Riba A., Fujisawa K., Kapadia M., Chau H., Albanese F., Pawar G., De Snoo M. L., Ngana S. G., Kim J., El-Agnaf O. M. A., Rennella E., Kay L. E., Kalia S. K. *, Kalia L. V. *, Kim P. M. * Disrupting the α -synuclein-ESCRT interaction with a peptide inhibitor mitigates neurodegeneration. *Nature Communications*, 2023.
5. Meng Y., People C. B., Huang Y., Jones R. M., Ottoy J., Goubran M., Oliveira L. M., Davidson B., Lawrence L. S. P., Lau A. Z., Bethune A., Maralani P., Abrahao A., Hamani C., Hynynen K., Kalia S. K. *, Lipsman N. *, Kalia L. V. * Unilateral putaminal delivery of recombinant glucocerebrosidase using transcranial low-intensity MR-guided focused ultrasound in Parkinson's disease. *Movement Disorders*, 2022.
6. Chen K. S. *, Menezes K. *, Rodgers J. B., O'Hara D., Tran N., Fujisawa K., Ishikura S., Khodaei S., Chau H., Lacoste A., Spangler S., Visanji N., Marras C., Majbour N. K., El-Agnaf O. M. A., Lozano A. M., Culotti J., Suo S., Ryu W. S., Kalia S. K. *, Kalia L. V. * Small molecule inhibitors of α -synuclein oligomers identified by targeting early dopamine-mediated motor impairment in *C. elegans*. *Molecular Neurodegeneration*, 2021.



ICKMDS 2025

5th International Conference of Korean Movement Disorder Society

Bridging from the Origin to Clinical Practice:
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DAY 1_ October 31 (Fri), 2025

Room A

Parallel Session 1

When and Where Does Synucleinopathy Begin?
(Multiple System Atrophy Perspective)

Chairs Hirohisa WATANABE (Japan)
Jongsam BAIK (Korea)



Neuroanatomical Origins and Clinical Landscape of Multiple System Atrophy

Woong-Woo LEE

Department of Neurology, Nowon Eulji Medical Center, Eulji University, Seoul, South Korea

MSA presents with two primary motor phenotypes. MSA-P (Parkinsonian type) manifests with classic parkinsonian signs, including bradykinesia, rigidity, and postural instability. A key differentiator from Parkinson's disease (PD) is the poor or absent response to levodopa treatment. This subtype is caused by pathological changes and neuronal loss in the striatonigral system, particularly in the putamen and caudate nucleus, leading to motor deficits. MSA-C (Cerebellar type) presents with cerebellar ataxia, which causes uncoordinated movements, unsteady gait, and slurred or scanning dysarthria. These symptoms result from degeneration of the olivopontocerebellar system, including the pons, inferior olivary nucleus, and cerebellar cortex, which control coordination and balance.

Autonomic dysfunction is a hallmark of MSA and often precedes motor symptoms. It is an important sign for diagnosis and is associated with a poor prognosis. Orthostatic hypotension (OH) is a classic finding, causing a significant drop in blood pressure when standing, affecting about 75% of patients. Supine hypertension can also occur. OH may be caused by degeneration of the hypothalamus, periaqueductal gray (PAG), locus coeruleus (LC), rostral ventrolateral medulla (RVLM), caudal ventrolateral medulla (CVLM), and the intermediolateral cell column (IML) of the thoracolumbar spinal cord. Another important autonomic dysfunction is urinary dysfunction. In addition, patients commonly experience urgency, frequency, difficulty in emptying their bladder, and incontinence. A post-void residual volume (PVR) over 100 mL is highly specific for MSA. The relevant pathological changes are found in the hypothalamus, substantia nigra (SN), LC, raphe nuclei, cerebellar vermis, basal ganglia, PAG, pontine micturition center (PMC), pontine continence center (PCC), IML, and Onuf's nucleus. Erectile dysfunction is often an early symptom in men, and gastrointestinal symptoms such as constipation, delayed gastric emptying, and dysphagia may occur. Other non-motor symptoms may include REM sleep behavior disorder (RBD), laryngeal stridor, and mild cognitive impairment.

MSA is a highly heterogeneous disease, and its symptoms often overlap with other neurodegenerative conditions, such as PD, progressive supranuclear palsy (PSP), and certain ataxias. This makes early and accurate diagnosis difficult. Early MSA-P can be easily misdiagnosed as PD. Key distinguishing features of MSA-P include poor response to levodopa, early and severe autonomic failure, and preserved olfaction and cognition in the initial stages. There have been rare cases of "minimal change," where pathology is restricted to only a few brain regions. Some patients with pathologically confirmed MSA may not show typical motor symptoms at all. In contrast, some autopsy-confirmed cases have shown extensive cognitive impairment, while significant dementia is an exclusion criterion for MSA. Coexisting pathologies such as Lewy body disease or Alzheimer's-like changes were found in those cases.

While advances have clarified many aspects of the neuroanatomical origins and clinical landscape of MSA, the precise pathogenesis remains incompletely understood. Clinical manifestations often reflect underlying pathological changes, yet interindividual

variabilities in pathological patterns and coexisting pathologies contribute to diverse clinical trajectories. To overcome these challenges, future studies will need to establish robust in vivo biomarkers that can provide not only pathological confirmation during life but also a detailed mapping of disease distribution, thereby enhancing diagnostic accuracy and enabling more targeted therapeutic strategies.

Personal Information

Full Name **Woong-Woo LEE**
 Current Position **Associate professor**
 Department **Department of Neurology**
 Organization **Nowon Eulji Medical Center, Eulji University**
 Country **Republic of Korea**



Education / Training

Institution and Location	Degree	Year(s)	Field of Study
Seoul National University, Seoul, Republic of Korea	Bachelor	2005	Medicine
Seoul National University, Seoul, Republic of Korea	Master	2013	Neuroscience
Seoul National University, Seoul, Republic of Korea	Ph.D.	2022	Neuroscience
Seoul National University Hospital, Seoul, Republic of Korea	Internship /Residency	2005-2010	Neurology
Seoul National University Hospital, Seoul, Republic of Korea	Fellowship	2013-2015	Movement disorders

Positions and Honors

Professional Experience

Current

2023/09 – Associate professor, Department of Neurology, Nowon Eulji Medical Center, Eulji University, Seoul, Republic of Korea

Previous

2017/03 – 2023/08 Assistant professor, Department of Neurology, Nowon Eulji Medical Center, Eulji University, Seoul, Republic of Korea

2015/03 – 2017/02 Clinical professor, Department of Neurology, Nowon Eulji Medical Center, Eulji University, Seoul, Republic of Korea

Honors

2009	Best Resident Award, Department of Neurology, Seoul National University Hospital
2010	Best Paper Award, 29th Annual Meeting of the Korean Neurological Association
2017	Travel grant award, 2017 International Congress of Parkinson's disease and Movement disorders
2019	LEAP Program Class of 2019, MDS

Selected Peer Reviewed Publications

1. Koo Y, Kim M, **Lee WW**. Predicting Parkinson's Disease Using a Deep-Learning Algorithm to Analyze Prodromal Medical and Prescription Data. *J Clin Neurol* 2025 Jan.
2. **Lee WW**, Lee CG, Ki CS. KCNJ3 is a novel candidate gene for autosomal dominant pure hereditary spastic paraplegia identified using whole genome sequencing. *Am J Med Genet B Neuropsychiatr Genet*. 2024 Apr.
3. Yoon S, Kim M, **Lee WW**. Long Short-Term Memory-Based Deep Learning Models for Screening Parkinson's Disease Using Sequential Diagnostic Codes. *J Clin Neurol*. 2023 May.
4. **Lee WW**, Kim HJ, Lee HJ, Kim HB, Park KS, Sohn CH, et al. Semiautomated Algorithm for the Diagnosis of Multiple System Atrophy With Predominant Parkinsonism. *J Mov Disord*. 2022 Sep.
5. Park HR, Lim YH, Song EJ, Lee JM, Park K, Park KH, **Lee WW**, Kim HJ, Jeon B, Paek SH. Bilateral Subthalamic Nucleus Deep Brain Stimulation under General Anesthesia: Literature Review and Single Center Experience. *J Clin Med*. 2020 Sep.
6. **Lee WW**, Jeon B, Kim R. Expanding the Spectrum of Dopa-Responsive Dystonia (DRD) and Proposal for New Definition: DRD, DRD-plus, and DRD Look-alike. *J Korean Med Sci*. 2018 May.
7. **Lee WW**, Yoon EJ, Lee JY, Park SW, Kim YK. Visual Hallucination and Pattern of Brain Degeneration in Parkinson's Disease, Neurodegener Dis. 2017.

Unveiling Prodromal and Early Multiple System Atrophy: From Mono-System Signs to Biomarker-Based Insights

Hirohisa WATANABE

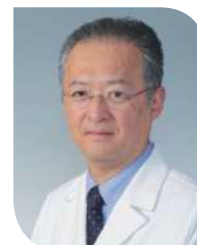
Department of Neurology, Fujita Health University, School of Medicine

Multiple system atrophy (MSA) is a rapidly progressive α -synucleinopathy with poor prognosis. The 2022 MDS criteria newly define possible prodromal MSA as a multisystem constellation, requiring combinations such as REM sleep behavior disorder and/or early autonomic and subtle motor signs. However, some patients initially show isolated dysfunction in a single domain. We therefore propose the concept of a mono-system phase MSA, in which early MRI abnormalities or isolated clinical features represent the first detectable stage. MRI remains a cornerstone but shows conflicting results: autopsy-based studies indicate that making MRI markers mandatory reduces sensitivity, whereas combined clinical and MRI assessment—particularly when abnormalities are evident early—increases diagnostic accuracy. This likely reflects biological heterogeneity, with some MSA cases showing infratentorial-predominant changes and others striatonigral-predominant involvement. Importantly, when characteristic MRI abnormalities are present from the outset, such patients might reasonably be diagnosed as mono-system phase MSA. Furthermore, as demonstrated by Kawabata et al. (2025), documenting progressive MRI changes over six months may substantially enhance diagnostic confidence. Beyond structure, molecular and fluid biomarkers are advancing. Neurofilament light chain and GFAP correlate with severity but lack specificity. α -synuclein seed amplification assays and skin biopsy provide disease-specific signatures. Moreover, α -synuclein PET enables in vivo visualization of pathology and may distinguish MSA from PD in early stages. Early abnormalities of tryptophan metabolism also support prodromal detection. In conclusion, defining mono-system phase MSA and combining longitudinal MRI, α -synuclein PET, and multimodal biomarkers may enable ultra-early diagnosis, improved stratification, and future disease-modifying trials.

Keywords: Multiple system atrophy; Prodromal stage; Mono-system phase; Biomarkers; Neuroimaging

Personal Information

Full Name Hirohisa WATANABE
Current Position Professor and Chairman
Department Department of Neurology
Organization Fujita Health University, School of Medicine
Country Japan



Education / Training

Institution and Location	Degree	Year(s)	Field of Study
Nagoya Daini Red Cross Hospital, Japan	Residency	1993-1995	Neurology
Nagoya Daini Red Cross Hospital, Japan	Doctor	1995-1999	Neurology
Nagoya University, Japan	Graduate Student	1999-2003	Neurology

Positions and Honors

Professional Experience

Current

2019 - present Professor, Department of Neurology, Fujita Health University, Japan

Previous

2013 - 2019 Specially Appointed Professor, Brain and Mind Research Center, Graduate School of Medicine, Nagoya University, Japan

2009-2013 Associate Professor, Department of Neurology, Nagoya University Hospital, Japan

2007-2008 Assistant Professor, Department of Neurology, Nagoya University Hospital, Japan

2006-2007 Research Associate, Department of Neurology, Nagoya University Hospital, Japan

Honors

2004/11 Aichi Prefecture Award for Rare Disease Researchers

2016/6 The 9th International Congress of the Asian Society Against Dementia, Oral Presented to Award

2017/6 International Congress of Parkinson's Disease and Movement Disorders. Blue Ribbon Award

2014-2025 Best Doctors in Japan

Selected Peer Reviewed Publications

1. Kawabata K, Krismer F, Ito M, Hara K, Bagarinao E, Beliveau V, Péran P, Arribarat G, Traon AP, Meissner WG, Foubert-Samier A, Fabbri M, Gordon MF, Ogura A, Katsuno M, Rascol O, Scherfler C, Seppi K, **Watanabe H**, Poewe W. Trajectories of Pontine Volume in Patients with Multiple System Atrophy. *Mov Disord*. 2025.
2. Nagao R, Mizutani Y, Kawabata K, Yoshimoto J, Inamoto Y, Shibata S, Ito M, Otaka Y, **Watanabe H**. Correlations between Dysphagia Severity Scale Scores and Clinical Indices in Individuals with Multiple System Atrophy. *Mov Disord Clin Pract*. 2025
3. Nagao R, Mizutani Y, Shima S, Ueda A, Ito M, Yoshimoto J, **Watanabe H**. Correlations between serotonin impairments and clinical indices in multiple system atrophy. *Eur J Neurol*. 2024
4. **Watanabe H**, Shima S, Mizutani Y, Ueda A, Ito M. Multiple System Atrophy: Advances in Diagnosis and Therapy. *J Mov Disord*. 2023
5. Ebina J, Hara K, **Watanabe H**, Kawabata K, Yamashita F, Kawaguchi A, Yoshida Y, Kato T, Ogura A, Masuda M, Ohdake R, Mori D, Maesawa S, Katsuno M, Kano O, Sobue G. Individual voxel-based morphometry adjusting covariates in multiple system atrophy. *Parkinsonism Relat Disord*. 2021
6. Lee YH, Ando T, Lee JJ, Baek MS, Lyoo CH, Kim SJ, Kim M, Cho JW, Sohn YH, Katsuno M, **Watanabe H**, Yoshida M, Lee PH. Later-Onset Multiple System Atrophy: A Multicenter Asian Study. *Mov Disord*. 2020
7. **Watanabe H**, Riku Y, Hara K, Kawabata K, Nakamura T, Ito M, Hirayama M, Yoshida M, Katsuno M, Sobue G. Clinical and Imaging Features of Multiple System Atrophy: Challenges for an Early and Clinically Definitive Diagnosis. *J Mov Disord*. 2018

Cellular and Molecular Mechanisms of Multiple System Atrophy

Nadia STEFANOVA

Department of Neurology, Medical University of Innsbruck, Austria

Multiple system atrophy (MSA) is a rare α -synucleinopathy characterized by neurodegeneration in striatonigral, olivopontocerebellar regions and autonomic brain centers. There is still a hiatus in our understanding on the pathogenesis of the disease. This lecture will address the complexity of MSA pathogenic mechanisms. It will discuss the current views on α -synuclein misfolding, aggregation, spreading and strain specificity. The hypothesis of primary oligodendrogliopathy will be introduced and discussed in view of the recent genetic, neuropathological and experimental studies. Examples on the role of iron dyshomeostasis, neuroinflammation, and mitochondrial dysfunction with relevance to the progression of MSA will be presented. The lecture will emphasize on the complexity of the multifactorial pathogenic cascade of MSA and its relevance to identifying biomarkers and efficient therapies for this devastating disorder.

Personal Information

Full Name **Nadia STEFANOVA**
Current Position **Professor**
Department **Department of Neurology**
Organization **Medical University of Innsbruck**
Country **Austria**



Education / Training

Institution and Location	Degree	Year(s)	Field of Study
Medical University of Sofia, Bulgaria and University of Innsbruck, Austria	MD	1988-1994	medicine
Medical University of Sofia, Bulgaria	PhD	1995-1999	neuroscience
Medical University of Innsbruck, Austria	DSc	2008	neuroscience

Positions and Honors

Professional Experience

Current

2024- Director, Division of Neurobiology, Department of Neurology, Medical University of Innsbruck
2020- Professor of Translational Neurodegeneration Research, Department of Neurology, Medical University of Innsbruck, Austria

Previous

2020-2023 Coordinator of the PhD Neuroscience Program at the Medical University of Innsbruck, Austria
2012-2019 Associate Professor, Department of Neurology, Medical University of Innsbruck, Austria
2007-2011 Assistant Professor, Department of Neurology, Medical University of Innsbruck, Austria
1997-2000 Assistant Professor, Medical University of Sofia, Bulgaria

Honors

2018 JP Award for MSA Research
2007 Science Award of Innsbruck

Selected Peer Reviewed Publications

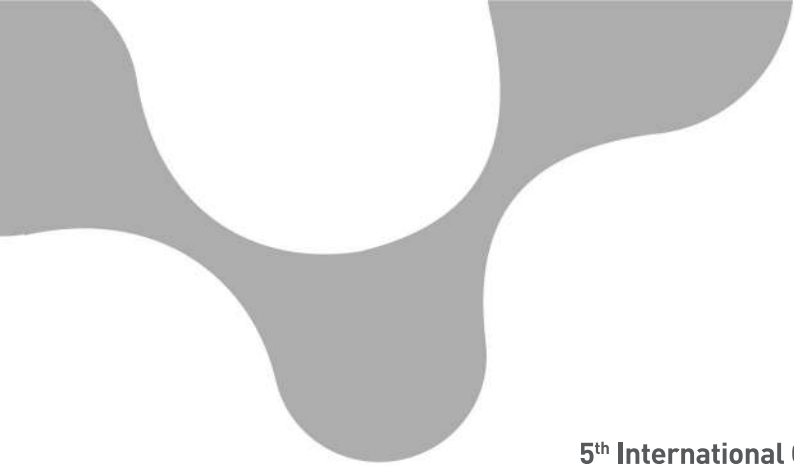
1. Chia R, Ray A, Shah Z, Ding J, Ruffo P, Fujita M, Menon V, Saez-Atienzar S, Reho P, Kaivola K, Walton RL, Reynolds RH, Karra R, Sait S, Akcimen F, Diez-Fairen M, Alvarez I, Fanciulli A, **Stefanova N**, Seppi K, Duerr S, Leys F, Krismer F, Sidoroff V, Zimprich A, Pirker W, Rascol O, Foubert-Samier A, Meissner WG, Tison F, Pavy-Le Traon A, Pellecchia MT, Barone P, Russillo MC, Marín-Lahoz J, Kulisevsky J, Torres S, Mir P, Perinán MT, Proukakis C, Chelban V, Wu L, Goh YY, Parkkinen L, Hu MT, Kobylecki C, Saxon JA, Rollinson S, Garland E, Biaggioni I, Litvan I, Rubio I, Alcalay RN, Kwei KT, Lubbe SJ, Mao Q, Flanagan ME, Castellani RJ, Khurana V, Ndayisaba A, Calvo A, Mora G, Canosa A, Floris G, Bohannon RC, Moore A, Norcliffe-Kaufmann L, Palma JA, Kaufmann H, Kim C, Iba M, Masliah E, Dawson TM, Rosenthal LS, Pantelyat A, Albert MS, Pletnikova O, Troncoso JC, Infante J, Lage C, Sánchez-Juan P, Serrano GE, Beach TG, Pastor P, Morris HR, Albani D, Clarimon J, Wenning GK, Hardy JA, Rytten M, Topol E, Torkamani A, Chiò A, Bennett DA, De Jager PL, Low PA, Singer W, Cheshire WP, Wszolek ZK, Dickson DW, Traynor BJ, Gibbs JR, Dalgard CL, Ross OA, Houlden H, Scholz SW. Genome sequence analyses identify novel risk loci for multiple system atrophy. *Neuron*. 2024 Jul 3;112(13):2142-2156.e5.
2. **Stefanova N**, Wenning GK. Multiple system atrophy: at the crossroads of cellular, molecular and genetic mechanisms. *Nat Rev Neurosci*. 2023 Jun;24(6):334-346.
3. Torre-Muruzabal T, Van der Perren A, Coens A, Gelders G, Janer AB, Camacho-Garcia S, Klingstedt T, Nilsson P, Stefanova N, Melki R, Baekelandt V, Peelaerts W. Host oligodendrogliopathy and α -synuclein strains dictate disease severity in multiple system atrophy. *Brain*. 2023 Jan 5;146(1):237-251.
4. **Stefanova N**. A Mouse Model of Multiple System Atrophy: Bench to Bedside. *Neurotherapeutics*. 2023 Jan;20(1):117-126.
5. Refolo V, Bez F, Polissidis A, Kuzdas-Wood D, Sturm E, Kamaratou M, Poewe W, Stefanis L, Angela Cenci M, Romero-Ramos M, Wenning GK, **Stefanova N**. Progressive striatonigral degeneration in a transgenic mouse model of multiple system atrophy: translational implications for interventional therapies. *Acta Neuropathol Commun*. 2018 Jan 3;6(1):2.
6. Fellner L, Jellinger KA, Wenning GK, **Stefanova N**. Glial dysfunction in the pathogenesis of α -synucleinopathies: emerging concepts. *Acta Neuropathol*. 2011 Jun;121(6):675-93.
7. Wenning GK, **Stefanova N**, Jellinger KA, Poewe W, Schlossmacher MG. Multiple system atrophy: a primary oligodendrogliopathy. *Ann Neurol*. 2008 Sep;64(3):239-46.



ICKMDS 2025

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**Bridging from the Origin to Clinical Practice:
A Beginning Has an Ending**



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Bridging from the Origin to Clinical Practice:
A Beginning Has an Ending

DAY 1_ October 31 (Fri), 2025

Room A

Parallel Session 3

Advancing Biomarker Horizons in Parkinson's Disease

Chairs Lorraine KALIA (Canada)
Joong-Seok KIM (Korea)



Neuroinflammation and Immune Biomarkers: Insights into Parkinson's Pathogenesis and Progression

Young Eun KIM

Department of Neurology, Hallym University Sacred Heart Hospital, Hallym University, South Korea

Parkinson's disease (PD) is a progressive neurodegenerative disorder marked by the accumulation of misfolded α -synuclein and selective loss of dopaminergic neurons. Accumulating evidence highlights the pivotal role of immune mechanisms—both innate and adaptive—in modulating disease onset and progression. Rather than being a downstream response, immune activation is increasingly recognized as an active participant in the pathological cascade of PD.

In the brain, α -synuclein aggregates activate microglia and trigger the NLRP3 inflammasome, leading to the secretion of IL-1 β , IL-18, and other inflammatory mediators. In α -synuclein preformed fibril (PFF)-injected animal models, microglial activation precedes pathological protein spread and exacerbates neurodegeneration. Astrocyte reactivity and peripheral infiltration of T cells further amplify neuroinflammation. Suppressing microglial activation mitigates α -synuclein pathology, supporting the notion that inflammation directly contributes to disease propagation.

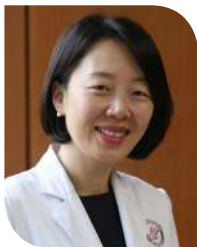
Peripheral immune profiles—such as elevated monocyte-to-HDL (MHR) and neutrophil-to-HDL (NHR) ratios—and proteomics correlate with disease severity or diagnosis in early PD and reflect systemic inflammatory priming. These findings bridge central and peripheral immune responses and suggest the possibility of immune-based biomarkers for disease monitoring.

The immunoproteasome represents a critical point of convergence between protein homeostasis and immune signaling. Up-regulation of its catalytic subunit, PSMB8, has been observed in PD patient-derived blood cells, neuronal extracellular vesicles, and postmortem brain tissue, often colocalizing with Lewy bodies. Its inhibition reduces α -synuclein levels and modulates T cell activity, illustrating the immunoproteasome's dual role in proteostasis and immune regulation.

Taken together, current evidence supports a model in which α -synuclein pathology, immune activation, and proteostatic dysfunction interact in a self-amplifying loop. Understanding the bidirectional crosstalk between immunity and Parkinson's disease not only elucidates disease pathogenesis but also opens new avenues for biomarker development and therapeutic intervention targeting the immune system at multiple levels.

Personal Information

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Country South Korea



Education / Training

Institution and Location	Degree	Year(s)	Field of Study
Hallym University College of Medicine	MD	2005	Medicine
Hallym University	MS	2010	Neurology
Seoul National University	PhD	2014	Neurology
Columbia University College of Physicians and Surgeons Department of Pathology & Cell Biology	Visiting Researcher	2015-2017	Basic neuroscience

Positions and Honors

Professional Experience

Current

2021-Current Associate Professor, Hallym University

Previous

2014-2021 Assistant Professor, Chung-Ang University

2010-2014 Fellowship, Seoul National University

Selected Peer Reviewed Publications

1. Peripheral Inflammatory Markers and Clinical Phenotypes Reflecting the Impact of Diabetes on Parkinson's Disease Kwak IH, Ma H., Kim YJ, Noh HM, Lee J, Yu JK, Kim YE*, Journal of Parkinson's disease, 2025, accepted
2. Prevalence and clinical impact of alpha-synuclein pathology in idiopathic normal pressure hydrocephalus: Insights from RT-QuIC assay. Yu JK, Choi HJ, Kim D, Ngoc PH, Kwak IH, Nguyen HD, Thanh TN, Song SJ, Lee J, Ma HI, Kim YE*.Parkinsonism Relat Disord. 2025 May 8;136:107857. doi: 10.1016/j.parkreldis.2025.107857. Online ahead of print.
3. Monocyte to High-density Lipoprotein Cholesterol Ratio Reflects the Peripheral Inflammatory State in Parkinsonian Disorders Kwak IH, Kim YE*, Kim YJ, Noh HM, Lee J, Yu JK, Ma HI. Parkinsonism Relat Disord. 2024 Dec;129:107155.

4. Olfactory and Gustatory Function in Early-stage Parkinson's Disease: Implication for Cognitive Association. Lee J, **Kim YE***, Lee JS, Kang SY, Kim MS, Kwak IH, Park J, Nam JY, Ma HI. *J Mov Disord*. 2024 Jun 27. doi: 10.14802/jmd.23243. Online ahead of print.
5. Thi Lai T, **Kim YE***, Nguyen LTN, Thi Nguyen T, Kwak IH, Richter F, Kim YJ, Ma HI. Microglial inhibition alleviates alpha-synuclein propagation and neurodegeneration in Parkinson's disease mouse model. *npj Parkinsons Dis*. 2024 Feb 2;10(1):32. doi: 10.1038/s41531-024-00640-2. PMID: 38302446
6. Kwak IH, **Kim YE***, Kang SY, Lee JS, Lee J, Kim MS, Yea DA, Ma HI. Comparative Olfactory Profiles in Parkinson's Disease and Drug-Induced Parkinsonism. *J Mov Disord*. 2024 Jan;17(1):64-70. doi: 10.14802/jmd.23105. Epub 2023 Oct 6.
7. Nguyen HD, **Kim YE***, Nhat Nguyen LT, Kwak IH, Lee YK, Kim YJ, Hai Nguyen TT, Pham HN, Ma HI. Upregulation of immunoproteasome PSMB8 is associated with Parkinson's disease. *Parkinsonism Relat Disord*. 2023 Sep;114:105797. doi: 10.1016/j.parkreldis.2023.105797. Epub 2023 Aug 5.
8. Kwak IH, Lee YK, Ma HI, Lee S, Yun M, Kim YJ, Hwang HS, **Kim YE***. Striatal Subregion Analysis Associated with REM Sleep Behavior Disorder in Parkinson's Disease. *J Integr Neurosci*. 2023 Jan 16;22(1):18. doi: 10.31083/j.jin2201018.
9. Linh Thi Nhat Nguyen , Huu Dat Nguyen , Yun Joong Kim , Tinh Thi Nguyen , Thuy Thi Lai , Yoon Kyoung Lee , Hyeo-Il Ma , Young Eun Kim Yoo D, Bang JI, Ahn C, Nyaga VN, **Kim YE***, Role of NLRP3 Inflammasome in Parkinson's Disease and Therapeutic Considerations *J Parkinsons Dis* 2022 Aug 17. DOI: 10.3233/JPD-223290
10. Nguyen TT, Kim YJ, Lai TT, Nguyen PT, Koh YH, Nguyen LTN, Ma HI, **Kim YE***. PTEN-Induced Putative Kinase 1 Dysfunction Accelerates Synucleinopathy. *J Parkinsons Dis*. 2022 Feb 28. doi: 10.3233/JPD-213065. Online ahead of print. PMID: 35253778
11. Lai TT, Kim YJ, Ma HI, **Kim YE***. Evidence of Inflammation in Parkinson's Disease and Its Contribution to Synucleinopathy. *Journal of movement disorder*, 2022, Jan;15(1):1-14. doi: 10.14802/jmd.21078. Epub 2021 Nov 3.
12. Lai TT, Kim YJ, Nguyen PT, Koh YH, Nguyen TT, Ma HI, **Kim YE***. Temporal Evolution of Inflammation and Neurodegeneration With Alpha-Synuclein Propagation in Parkinson's Disease Mouse Model. *Front Integr Neurosci*. 2021 Oct 5;15:715190.
13. **Kim YE**, Lai Thi Thuy, Jeon BS, Kim YJ Preferential microglial activation associated with pathological alpha synuclein transmission. *Journal of Clinical Neuroscience* 2020 Nov;81:469-476
14. **Kim YE**, Ma HI, Seong GH, et al. Early Impairment of Chopsticks Skills in Parkinsonism Suggests Progressive Supranuclear Palsy. *J Clin Neurol*. 2020;16(2):254-260.

Neuroimaging biomarkers for Cognitive decline in Parkinson's Disease

Seok-Jong CHUNG

Department of Neurology, Yonsei University College of Medicine, South Korea

Cognitive impairment is commonly observed in patients with Parkinson's disease, and approximately 80% of patients eventually develop dementia within 20 years. Dementia conversion is a key milestone of the disease progression in Parkinson's disease, greatly affecting the morbidity and mortality. Therefore, early detection of patients at a high risk of dementia conversion is important for close monitoring and appropriate implementation of personalized treatment strategies. To date, several studies have been reported to propose neuroimaging biomarkers for ongoing cognitive decline in Parkinson's disease. Using T1-weighted images, some stressed the importance of frontal atrophy in dementia conversion, while others demonstrated that atrophy in the posterior cortical regions, basal forebrain, or hippocampus was more relevant to dementia in Parkinson's disease. Studies using diffusion tensor images reported that increased mean diffusivity values in the nucleus basalis of Meynert can predict the onset of cognitive impairment. Functional MRI studies have shown that alterations within the neurocognitive networks, such as default mode network, central executive network, and salience network, are associated with cognitive prognosis in Parkinson's disease. Changes in cerebral glucose metabolism or perfusion on functional neuroimaging studies (e.g., Parkinson's disease-related cognitive pattern) also can be a promising biomarker for ongoing cognitive decline. In summary, a number of studies attempted to find out which neuroimaging biomarker can be a useful marker for future cognitive decline in Parkinson's disease. Each neuroimaging modality may have its own advantage, and a multimodal combination of imaging modalities would provide an ideal biomarker for cognitive prognosis in Parkinson's disease.

Personal Information

Full Name Seok-Jong CHUNG
Current Position Associate Professor
Department Department of Neurology
Organization Yonsei University College of Medicine
Country South Korea



Education / Training

Institution and Location	Degree	Year(s)	Field of Study
Yonsei University College of Medicine, Seoul	MD	2009	
Yonsei University Graduate School, Seoul	MS	2014	Neurology
Yonsei University Graduate School, Seoul	PhD	2021	Neurology

Positions and Honors

Professional Experience

Current

2025.09~ Associate Professor, Department of Neurology, Severance Hospital, Yonsei University College of Medicine, Seoul, South Korea

Previous

2017.05~2019.12 Clinical Fellowship, Department of Neurology, Severance Hospital, Yonsei University College of Medicine, Seoul, South Korea

2020.01~2022.02 Clinical Assistant Professor, Department of Neurology, Yongin Severance Hospital, Yonsei University College of Medicine, Yongin, South Korea

2022.03~2024.02 Assistant Professor, Department of Neurology, Yongin Severance Hospital, Yonsei University College of Medicine, Yongin, South Korea

2024.02~2025.08 Associate Professor, Department of Neurology, Yongin Severance Hospital, Yonsei University College of Medicine, Yongin, South Korea

Honors

2019 Korean Movement Disorder Society Fellowship Academic Award

2019 LG Chemical Future Medical Scientist Award

2020 Korean Neurological Association Hyang-Seol Young Investigator Award

2021	Yonsei University College of Medicine Academic Award for Outstanding Research Activity (Junior Researcher Category)
2022	Severance Young Medical Scientist Award
2023	Korean Movement Disorder Society Investigator Award
2024	Ministry of Health and Welfare Award for Meritorious Service in Health and Medical Technology Promotion (Junior Researcher Category)

Selected Peer Reviewed Publications

1. Chun MY, Lee T, Kim SH, Lee HS, Kim YJ, Lee PH, Sohn YH, Jeong Y,* **Chung SJ**.* Hypoperfusion in Alzheimer's disease-prone regions and dementia conversion in Parkinson's disease. Clin Nucl Med 2024.
2. Chun MY,* **Chung SJ**.* Kim SH, Park CW, Jeong SH, Lee HS, Lee PH, Sohn YH, Jeong Y, Kim YJ. Hippocampal Perfusion Affects Motor and Cognitive Functions in Parkinson Disease: An Early Phase (18) F-FP-CIT Positron Emission Tomography Study. Ann Neurol 2024.
3. Park CJ, Eom J, Park KS, Park YW,* **Chung SJ**.* Kim YJ, Ahn SS, Kim J, Lee PH, Sohn YH, Lee SK. An interpretable multi-parametric radiomics model of basal ganglia to predict dementia conversion in Parkinson's disease. NPJ Parkinsons Dis 2023
4. **Chung SJ**, Kim SH, Park CW, Lee HS, Kim YJ, Lee PH, Jeong Y, Sohn YH. Is the cingulate island sign a marker for early dementia conversion in Parkinson's disease? Eur J Neurol 2023
5. Jeong SH, Jeong HJ, Sunwoo MK, Ahn SS, Lee SK, Lee PH, Kim YJ, Sohn YH, Park CJ,* **Chung SJ**.* Association between choroid plexus volume and cognition in Parkinson disease. Eur J Neurol 2023.
6. Jeong SH, Kim SH, Park CW, Lee HS, Lee PH, Kim YJ, Sohn YH, Jeong Y, **Chung SJ**. Differential Implications of Cerebral Hypoperfusion and Hyperperfusion in Parkinson's Disease. Mov Disord 2023.
7. **Chung SJ**,* Kim YJ,* Kim YJ, Lee HS, Jeong SH, Hong JM, Sohn YH, Yun M, Jeong Y, Lee PH. Association between white matter networks and the patterns of striatal dopamine depletion in patients with Parkinson's disease. Neurology 2022
8. **Chung SJ**,* Kim YJ,* Jung JH, Lee HS, Ye BS, Sohn YH, Jeong Y, Lee PH. Association between white matter connectivity and early dementia in patients with Parkinson disease. Neurology 2022
9. **Chung SJ**, Lee HS, Kim HR, Yoo HS, Lee YH, Jung JH, Baik K, Ye BS, Sohn YH, Lee PH. Factor analysis-derived cognitive profile predicting early dementia conversion in PD. Neurology 2020
10. **Chung SJ**, Lee HS, Yoo HS, Lee YH, Lee PH, Sohn YH. Patterns of striatal dopamine depletion in early Parkinson disease: Prognostic relevance. Neurology 2020
11. **Chung SJ**,* Jeon S,* Yoo HS, Lee YH, Yun M, Lee SK, Lee PH, Sohn YH, Evans AC, Ye BS. Neural Correlates of Cognitive Performance in Alzheimer's Disease- and Lewy Bodies-Related Cognitive Impairment. J Alzheimers Dis 2020
12. **Chung SJ**, Yoo HS, Lee YH, Lee HS, Ye BS, Sohn YH, Kwon HK, Lee PH. Frontal atrophy as a marker for dementia conversion in Parkinson's disease with mild cognitive impairment. Hum Brain Mapp 2019
13. **Chung SJ**,* Park YH,* Yoo HS, Lee YH, Ye BS, Sohn YH, Lee JM, Lee PH. Mild cognitive impairment reverts have a favorable cognitive prognosis and cortical integrity in Parkinson's disease. Neurobiol Aging 2019
14. **Chung SJ**, Park YH, Yun HJ, Kwon H, Yoo HS, Sohn YH, Lee JM, Lee PH. Clinical relevance of amnesic versus non-amnesic mild cognitive impairment subtyping in Parkinson's disease. Eur J Neurol 2019
15. **Chung SJ**, Yoo HS, Oh JS, Kim JS, Ye BS, Sohn YH, Lee PH. Effect of striatal dopamine depletion on cognition in de novo Parkinson's disease. Parkinsonism Relat Disord 2018

Cutting-Edge Biomarkers Research and Future Directions in Parkinson's Disease

Nobutaka HATTORI^{1,2}

¹Department of Neurology, Juntendo University, Neuron-Glia Crosstalk Center, Juntendo University, Tokyo, Japan

²Neurodegenerative Disorders Collaboration Laboratory, RIKEN Center for Brain Science, Saitama, Japan

Parkinson's disease (PD) is a single disease name but represents a diverse group of disorders. The clinical presentation varies among patients, with some showing resting tremor and others not, and the progression of symptoms and cognitive dysfunction also differ. Pathologically, many cases are characterized by Lewy bodies, which are inclusion bodies. The main component of these bodies, α -synuclein, is presumed to aggregate and propagate, and it is believed that the progression and severity of clinical symptoms are determined by this "propagation ability," or seed activity. The pathogenic β -sheet seed conformation of α -synuclein is found in various tissues, suggesting potential as a biomarker. Still, few studies have been able to detect these seeds in serum samples reliably. Recently, we developed a modified assay system, immunoprecipitation-based real-time quaking-induced conversion (IP/RT-QuIC), which enables the detection of pathogenic α -synuclein seeds in the serum of individuals with synucleinopathies such as PD, Dementia with Lewy bodies (DLB), and Multiple system atrophy (MSA). Currently, PD cannot be cured entirely, but symptomatic treatments have significantly improved life expectancy. However, the necessity of disease-modifying therapies (DMTs) to suppress symptom progression is being advocated. DMTs targeting α -synuclein are adequate for sporadic and hereditary PD for types where α -synuclein aggregates and propagates. In this lecture, I would like to explain the status and future of disease-modifying therapies for PD.

Personal Information

Full Name Nobutaka HATTORI
Current Position Project Professor
Department Department of Neurology
Organization Juntendo University
Country Japan



Education / Training

Institution and Location	Degree	Year(s)	Field of Study
Juntendo University Faculty of Medicine	M.D.	1985	Medicine
Juntendo University Graduate School of Medicine	Ph.D.	1994	Neurology

Positions and Honors

Professional Experience

Current

2025- Director, Neuron-Glia Crosstalk Center (NGCC) Juntendo, Juntendo University
2025- Project Professor, Dept. of Neurology, Juntendo University Faculty of Medicine
2024- Advisor to the University President, Juntendo University School of Medicine
2025- Team Director, Neurodegenerative Disorders Collaborative Laboratory, RIKEN Center for Brain Science (CBS)

Previous

2006-2025 Chairman and Professor, Department of Neurology, Juntendo University Faculty of Medicine
2020-2025 Team Leader, Neurodegenerative Disorders Collaborative Laboratory, RIKEN Center for Brain Science (CBS)
2019-2024 Dean, Faculty of Medicine and Graduate School of Medicine, Juntendo University

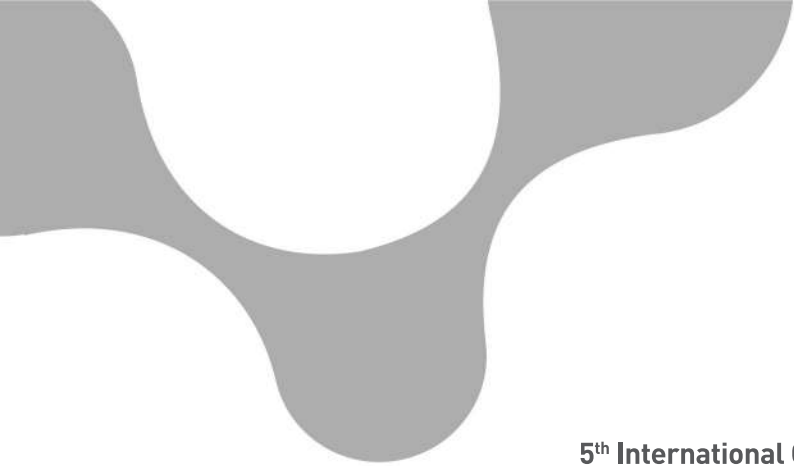
Honors

2025 The Japan Academy Award
2024 The Takeda Prize for Medical Science
2024 Tokizane Toshihiko Prize (Official prize of Japan Neuroscience Society)

2023	The Honorary Membership Award, the MDS Congress 2023, Copenhagen
2022	C. David Marsden Lecture Award 2022, the MDS Congress 2022, Madrid
2012	The Commendation for Science and Technology by the Minister of Education, Culture, Sports, Science and Technology
2002	The 39th Baelz Prize

Selected Peer Reviewed Publications

1. Okuzumi A, Hatano T, Matsumoto G, Nojiri S, Ueno SI, Imamichi-Tatano Y, Kimura H, Kakuta S, Kondo A, Fukuhara T, Li Y, Funayama M, Saiki S, Taniguchi D, Tsunemi T, . . . , Nukina N, and **Hattori N**. Propagative α -synuclein seeds as serum biomarkers for synucleinopathies. *Nat Med*. 2023
2. Oji Y, Hatano T, Ueno SI, Funayama M, Ishikawa KI, Okuzumi A, Noda S, Sato S, Satake W, Toda T, Li Y, Hino-Takai T, Kakuta S, Tsunemi T, Yoshino H, Nishioka K, Hattori T, Mizutani Y, Mutoh T, Yokochi F,, Matsuda J, and **Hattori N**. Variants in saposin D domain of prosaposin gene linked to Parkinson's disease. *Brain*. 2020
3. Mori A, Hatano T, Inoshita T, Shiba-Fukushima K, Koinuma T, Meng H, Kubo SI, Spratt S, Cui C, , Imai Y, and **Hattori N**. Parkinson's disease-associated iPLA2-VIA/PLA2G6 regulates neuronal functions and α -synuclein stability through membrane remodeling. *Proc Natl Acad Sci U S A*. 2019
4. Funayama M, Ohe K, Amo T, Furuya N, Yamaguchi J, Saiki S, Li Y, Ogaki K, Ando M, Yoshino H, Tomiyama H, Nishioka K, Hasegawa K, . . . , Ohno K, and **Hattori N**. CHCHD2 mutations in autosomal dominant late-onset Parkinson's disease: a genome-wide linkage and sequencing study. *Lancet Neurol*, 2015
5. Matsuda N, Sato S, Shiba K, Okatsu K, Saisho K, Gautier CA, Sou YS, Saiki S, Kawajiri S, Sato F, Kimura M, Komatsu M, **Hattori N**, Tanaka K. PINK1 stabilized by mitochondrial depolarization recruits Parkin to damaged mitochondria and activates latent Parkin for mitophagy. *J Cell Biol*, 2010
6. Shimura H, **Hattori N**, Kubo S, Mizuno Y, Asakawa S, Minoshima S, Shimizu N, Iwai K, Chiba T, Tanaka K, Suzuki T. Familial Parkinson disease gene product, parkin, is a ubiquitin-protein ligase. *Nat Genet*, 2000
7. Kitada T, Asakawa S, **Hattori N**, Matsumine H, Yamamura Y, Minoshima S, Yokochi M, Mizuno Y, Shimizu N. Mutations in the parkin gene cause autosomal recessive juvenile parkinsonism. *Nature*, 1998



ICKMDS 2025

5th International Conference of Korean Movement Disorder Society

Bridging from the Origin to Clinical Practice:
A Beginning Has an Ending

DAY 1_ October 31 (Fri), 2025

Room A

Parallel Session 5

Non-Motor Symptoms: From Prodromal Indicators to
Advanced manifestations

Chairs Ai Huey TAN (Malaysia)
Mee Young PARK (Korea)



Visual Dysfunction Throughout the Progression of Parkinson's Disease

Jee-Young LEE

Department of Neurology, Seoul National University College of Medicine

Parkinson and Rare disease Center, Seoul Metropolitan Government-Seoul National University Boramae Medical Center, Seoul, Korea

Visual dysfunction is commonly observed in patients with Parkinson's disease (PD). The true prevalence is unknown, but data of our center shows that about 30% of PD patients have symptoms relating to afferent visual dysfunction at the time of PD diagnosis, and some of the symptoms are improved with levodopa therapy, and others progressed. Common visual dysfunctions can be categorized into pupillary dysfunction, macular thinning, ocular motor dysfunction, and contrast/color discriminating functions. Retina and optic nerve pathology have been identified in autopsy tissues of PD patients, which are highly specific to PD(&LBD), correlating with brain Lewy body pathology. Studies have shown visual dysfunctions are linked to retinal thinning detected by optical coherence tomography and functionally linked to visual perceptual and constructional dysfunctions in PD patients. Longitudinal studies revealed that presence of afferent dysfunctions are associated with higher risk of visual hallucinations and dementia in the future. It is noteworthy that visual illusion and hallucinations are very specific nonmotor symptom of PD which discriminates from other parkinsonian syndromes. This talk will present typical features of visual dysfunctions in PD and propose possible mechanism of the pathophysiology and up to date knowledge about its clinical implications.

Personal Information

Full Name Jee-Young LEE
Current Position Professor
Department Neurology
Organization Seoul National University College of Medicine,
 SMG-SNU Boramae Medical center
Country Seoul



Education / Training

Institution and Location	Degree	Year(s)	Field of Study
Graduate School of Medicine, Seoul National University, Seoul	PhD	2014	Neuroscience
Graduate School of Medicine, Seoul National University, Seoul	MSc	2006	Neuroscience
College of Medicine, Seoul National University, Seoul	MD	2002	Medicine
Seoul National University Hospital	Residency	2003-2007	Neurology
Seoul National University Hospital	Fellow	2007-2009	Movement Disorders

Positions and Honors

Professional Experience

Current

2020 - Professor in Department of Neurology, SMG-SNU Boramae Medical Center, and
 SNU College of Medicine
2025 - Director of Parkinson and Rare disease Center, SMG-SNU Boramae Medical Center

Previous

2014 - 2020 Associate Professor, SMG-SNU Boramae Medical Center
2010 - 2014 Assistant Professor, SMG-SNU Boramae Medical Center
2009 - 2010 Instructor, Inje University College of Medicine
2016 - 2017 Honorary Researcher, Division of Brain Science, Imperial College of London

Honors

2022	KMDS Great Academic Awards
2013	MDS Junior Awards

Selected Peer Reviewed Publications

1. Kim R, Jin BR, Kim H, Woo KA, Yoon EJ, Kim S, Shin JH, Nam H, Kim YK, Jeon B, **Lee JY** Cholinergic basal forebrain degeneration in isolated REM sleep behavior disorder. *Brain* 2025 May 26. Epub ahead of print
2. Woo KA, Kim H, Kim R, Jin B, Shin JH, Kim S, Kim YK, Nam H, Jeon B, **Lee JY**. Cholinergic degeneration and early cognitive signs in prodromal Lewy body dementia. *Alzheimers Dement*. 2025 Feb;21(2):e14584.
3. Woo KA, Yoon EJ, Kim HJ, Kim SY, Jin BR, Lee SM, Kim R, Park HW, Kim YK, Nam H, **Lee JY**. Cognitive impact of β -amyloid load across the REM sleep behavior disorder-Lewy body disease Continuum. *Mov Disord* 2024 Dec;39(23): 2259-2270.
4. Woo KA, Joun JH, Yoon EJ, Kim YK, Jang HJ, **Lee JY**. Monoaminergic degeneration and ocular motor abnormalities in de novo Parkinson's disease. *Mov Disord* 2023 Dec; 38(12): 2291-2301.
5. Pérez-Acuña D, Rhee KH, Shin SJ, Ahn J, Lee JY, **Lee SJ**. Retina-to-brain spreading of α -synuclein after intravitreal injection of preformed fibrils. *Acta Neuropathol Commun*. 2023 May 20;11(1):83.
6. **Lee JY**, Martin-Bastida A, Murueta-Goyena A, Gabilondo I, Cuenca N, Piccini P, Jeon B. Multimodal brain and retina imaging of dopaminergic degeneration in Parkinson disease. *Nat Rev Neurol*. 2022 Apr;18(4):203-220.
7. **Lee JY**, Ahn J, Oh S, Shin JY, Kim YK, Nam H, Jeon B. Retina thickness as a marker of neurodegeneration in prodromal Lewy body disease. *Mov Disord* 2020 Feb;35(2):349-354.
8. Ahn J, **Lee JY**, Kim TW, Yoon EJ, Oh Sohee, Kim YK, Kim JM, Woo SJ, Kim KW, Jeon BS. Retinal thinning and nigral dopaminergic loss in de novo Parkinson disease. *Neurology* 2018 Sep 11;91(11):e1003-e1012
9. **Lee JY**, Yoon EJ, Lee WW, Kim YK, Lee JunY, Jeon BS. Lateral geniculate atrophy in parkinsonian patients with visual hallucination: a trans-synaptic degeneration? *Mov Disord*. 2016 Apr;31(4):547-54.
10. **Lee JY**, Kim JM, Ahn Y, Kim HJ, Jeon BS, Kim TW. Retinal Nerve Fiber Layer Thinning and Visual Hallucinations in Parkinson's disease. *Mov Disord* 2014 Jan: 29(1):61-67

Mind Matters: Addressing the Psychiatric Symptoms in Parkinson's Disease

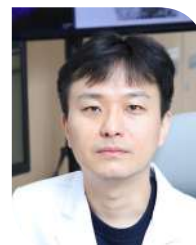
Han Soo YOO

Department of Neurology, Gangnam Severance Hospital, Yonsei University College of Medicine, South Korea

Psychiatric and behavioral symptoms are now recognized as core features of Parkinson's disease (PD), transforming the concept of PD from a purely motor disorder to a complex neuropsychiatric condition. Depression, anxiety, apathy, psychosis, impulse control disorders, and cognitive impairment occur across all disease stages, often preceding motor onset, and are major drivers of disability, caregiver burden, institutionalization, and mortality. Epidemiological studies report depression in nearly half of patients, anxiety in up to sixty percent, psychosis in up to forty percent, and impulse control disorders in nearly one-fifth, with higher rates in long-term follow-up. The mechanisms underlying these manifestations reflect both disease-related degeneration and treatment effects. Beyond nigrostriatal dopamine loss, disturbances in serotonergic, noradrenergic, and cholinergic circuits contribute. Depression and anxiety arise from mesolimbic and serotonergic dysfunction, apathy relates to impaired frontostriatal and cholinergic tone, psychosis emerges from combined limbic pathology and dopaminergic therapy, and impulse control disorders exemplify therapy-induced morbidity through mesolimbic overstimulation. These symptoms predict poorer outcomes: depression and psychosis accelerate cognitive decline, apathy increases dementia risk, and all psychiatric comorbidities amplify caregiver stress and healthcare costs. Despite this, recognition remains limited due to symptom overlap with motor features and inconsistent use of validated scales. Treatment options are modest. Antidepressants show limited efficacy, cholinesterase inhibitors help psychosis in dementia, and pimavanserin offers targeted benefit without worsening motor function. Reducing dopamine agonists remains key for impulse control disorders. Non-pharmacological strategies, including cognitive behavioral therapy, structured exercise, and caregiver support, are increasingly recognized as essential components of care. Psychiatric symptoms in PD constitute a "second burden" equal to motor impairment. Proactive screening, multidisciplinary management, and research prioritization are critical to improving patient outcomes and reducing the societal impact of Parkinson's disease.

Personal Information

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Current Position Associate Professor
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Country South Korea



Education / Training

Institution and Location	Degree	Year(s)	Field of Study
Yonsei University College of Medicine	M.D.	2009	Medicine
Yonsei University College of Medicine	Ph.D.	2025	Medicine

Positions and Honors

Professional Experience

Current

2025 - Present Associate Professor, Department of Neurology, Gangnam Severance Hospital, Yonsei University College of Medicine

Previous

2022 - 2025 Assistant Professor, Department of Neurology, Gangnam Severance Hospital, Yonsei University College of Medicine

2020 – 2022 Researcher, Department of Pathology and Laboratory Medicine, Center for Neurodegenerative Disease Research, University of Pennsylvania Perelman School of Medicine

2018 – 2020 Clinical Research Assistant Professor, Department of Neurology, Shinchon Severance Hospital, Yonsei University College of Medicine

2017 – 2018 Fellowship, Movement Disorder and Dementia Section, Department of Neurology, Shinchon Severance Hospital, Yonsei University College of Medicine

2010 – 2014 Resident, Department of Neurology, Severance Hospital, Yonsei University Health System

Honors

2024	Excellent Presentation Award, 2024 the Korean Neurological Association
2024	Best Presentation Award, 2024 Korea Movement Disorder Society Autumn Symposium
2023	Journal of Movement Disorders Best Paper Award

Selected Peer Reviewed Publications

1. Song Y, Lee JH, Kim HK, Lee JH, Ryu YH*, **Yoo HS***, Lyoo CH. Longitudinal trajectory of dopamine and serotonin transporters in Parkinson disease. *J Nucl Med* 2025 Feb 3;66(2):286-292.
2. **Yoo HS**, Kim HK, Na HK, Kang SW, Park M, Ahn SJ, Lee JH, Ryu YH*, Lyoo CH*. Association of striatal dopamine depletion and brain metabolism changes with motor and cognitive deficits in patients with Parkinson disease. *Neurology* 2024 Dec 24;103(12):e210105.
3. **Yoo HS**, Lee YG, Sohn YH, Yun M, Cha J*, Lee PH*. Association of relative brain hyperperfusion independent of dopamine depletion with motor dysfunction in patients with Parkinson's disease. *Neurology* 2024 Dec 24;103(12):e210077.
4. **Yoo HS***, Jeong SH*, Oh KT, Lee S, Sohn YH, Ye BS, Yun M, Lee PH (co-first author). Interrelation of striatal dopamine, brain metabolism and cognition in dementia with Lewy bodies. *Brain* 2022 Dec;145(12):4448-4458.
5. **Yoo HS***, Jeon S*, Cavedo E, Ko MJ, Yun M, Lee PH, Sohn YH, Grothe MJ, Teipel S, Hampel H, Evans AC, Ye BS (co-first author). Association of β -amyloid and basal forebrain with cortical thickness and cognition in Alzheimer and Lewy body disease spectra. *Neurology* 2022 Mar;98(9):e947-e957.
6. **Yoo HS**, Lee S, Chung SJ, Lee YH, Lee PH, Sohn YH, Lee S, Yun M, Ye BS. Dopaminergic depletion, β -amyloid burden, and cognition in Lewy body disease. *Ann Neurol* 2020 May;87(5):739-750.
7. **Yoo HS**, Lee S, Chung SJ, Lee YH, Ye BS, Sohn YH, Yun M, Lee PH. Clinical and striatal dopamine transporter predictors of β -amyloid in dementia with Lewy bodies. *Neurology* 2020 Mar;94(13):e1344-e1352.

Beyond the Motor: Unmasking Non-Motor Fluctuations in Parkinson's Treatment

Eungseok OH

Department of Neurology, Chungnam National University Hospital, Republic of Korea

Non-motor fluctuations (NMFs) in Parkinson's disease (PD) are relatively under-estimated than the motor fluctuations, however it could significantly impact a quality of life of PD patients and may sometimes be more debilitating than motor fluctuations. NMF includes variation of frequency and severity of non-movement symptoms, such as mood, cognitive, or autonomic issues, which coincide with "medication ON" and "medication off" periods of dopaminergic therapy. Therefore, NMFs are often responsive to dopaminergic treatment otherwise, non-dopaminergic therapy, deep brain stimulation and non-pharmacological approaches are also useful. We can discuss the type of NMF and impact of NMF and treatment of NMF for PD patients.

Personal Information

Full Name Eungseok OH
Current Position Professor
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Country Republic of Korea



Education / Training

Institution and Location	Degree	Year(s)	Field of Study
Chungnam National University College of Medicine	Bachelor	2003	Medicine
Chungnam National University College of Medicine	Master	2009	Neurology
Chungnam National University College of Medicine	Ph.D	2014	Parkinson's disease

Positions and Honors

Professional Experience

Current

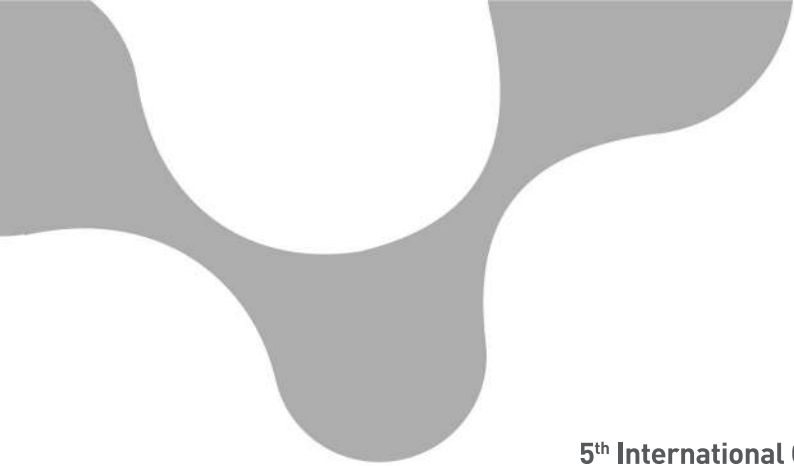
2025 ~ Professor, Chungnam National University Hospital
2024 ~ Managing Director, KMDS

Previous

2011 ~ 2013 Clinical Fellow, Bundang Seoul National University Hospital
2013 ~ 2016 Clinical professor, Chungnam National University Hospital
2016 ~ 2021 Assistant professor, Chungnam National University Hospital
2020 ~ 2021 Research fellow, Toronto Western Hospital
2021 ~ 2025 Associate professor, Chungnam National University Hospital

Selected Peer Reviewed Publications

1. Moon J, Oh E, Kim M, Kim R, Yoo D, Shin C, Lee JY, Kim JM, Koh SB, Kim M, Jeon B. A Practical Guide for Diagnostic Investigations and Special Considerations in Patients With Huntington's Disease in Korea. *J Mov Disord*. 2025 Jan;18(1):17-30
2. Oh E, Cheon SM, Cho JW, Sung YH, Kim JS, Shin HW, Kim JM, Park MY, Kwon DY, Ma H, Park JH, Koh SB, Choi SM, Park J, Lee PH, Ahn TB, Kim SJ, Lyoo CH, Lee HW, Kim J, Lee Y, Baik JS. Efficacy and safety of safinamide in Parkinson's disease patients with motor fluctuations without levodopa dosage escalation over 18 weeks: KEEP study. *J Neural Transm (Vienna)*. 2025 Mar;132(3):431-441.
3. Hwang YS, Oh E. Solute Carrier Family 2 Member 1 Gene Mutation Presenting as Adult-Onset Paroxysmal Exercise-Induced Dyskinesia Without Epilepsy. *J Clin Neurol*. 2024 Nov;20(6):627-629
4. Park JM, Moon CW, Lee BC, Oh E, Lee J, Jang WJ, Cho KH, Lee SH. Detection of freezing of gait in Parkinson's disease from foot-pressure sensing insoles using a temporal convolutional neural network. *Front Aging Neurosci*. 2024 Jul 18;16:1437707.
5. Kim D, Kim S, Seok JM, Shin KJ, Oh E, Jeon MY, Park J, Chang HJ, Youn J, Oh J, Sohn E, Park J, Cho JW, Kim BJ. Establishment of a registry of clinical data and bioresources for rare nervous system diseases. *Osong Public Health Res Perspect*. 2024 Apr;15(2):174-181.
6. Amartumur S, Nguyen H, Huynh T, Kim TS, Woo RS, Oh E, Kim KK, Lee LP, Heo C. Neuropathogenesis-on-chips for neurodegenerative diseases. *Nat Commun*. 2024 Mar 12;15(1):2219



ICKMDS 2025

5th International Conference of Korean Movement Disorder Society

Bridging from the Origin to Clinical Practice:
A Beginning Has an Ending

DAY 1_ October 31 (Fri), 2025

Room B

Parallel Session 2

Freezing of Gait: Subtypes, Prediction, and Management

Chairs Jeffrey HAUSDORFF (Israel)
Seong-Beom KOH (Korea)



FOG: Heterogeneity of the Frustrating Disorder

Dallah YOO

Department of Neurology, Kyung Hee University Hospital, Republic of Korea

Freezing of gait (FOG) is one of the most disabling features of Parkinson's disease (PD), marked by brief, episodic failures to initiate or continue stepping—patients describe it as feet “glued to the floor.” Its prevalence rises with disease progression and is tightly linked to falls, loss of independence, and reduced quality of life. FOG is “frustrating” not only because episodes are unpredictable and context-sensitive, but because treatments work inconsistently across patients. The key to clinical progress is recognizing that FOG is not a single entity but a heterogeneous syndrome with different mechanisms and therapeutic levers.

Phenomenology and clinical heterogeneity

- **Phenotypes:** Three common expressions are trembling-in-place, akinetic (no visible leg movement), and shuffling-forward FOG. These patterns differ in triggers, fall risk, and responses to therapy. Recent work suggests phenotypes cluster differently by medication state and task demands.
- **Triggers and contexts:** Turning-in-place, narrow passages/doorways, gait initiation, time pressure, dual-tasking, and anxiety are potent precipitants. FOG during turning tends to be more severe and more likely to cause falls.
- **State dependence:** OFF-FOG often improves with levodopa optimization; ON-FOG and levodopa-unresponsive FOG reflect broader network dysfunction beyond dopaminergic pathways. Cognitive and visuospatial deficits are more prevalent in unresponsive subtypes, implicating frontostriatal circuits.

Pathophysiological heterogeneity: a network disorder

FOG arises from dysfunctional interactions across a distributed locomotor network: cortical (SMA, prefrontal executive control, visuospatial processing), basal ganglia (STN/GPi), and brainstem locomotor regions (pedunculopontine and cuneiform nuclei). Imaging studies point to cortico-striatal decoupling and dorsal visuomotor stream abnormalities in freezers. Structural connectivity analyses suggest that STN stimulation sites linked to motor/premotor and dorsolateral prefrontal areas may better alleviate FOG, underscoring the circuit-level basis of heterogeneity.

Personal Information

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Country Republic of Korea



Education / Training

Institution and Location	Degree	Year(s)	Field of Study
Pohang University of Science and Technology (POSTECH), ROK	BS	2009	Life Science
Seoul National University College of Medicine, ROK	MD	2013	Medicine
Seoul National University College of Medicine, ROK	PhD	2022	Translational Medicine

Positions and Honors

Professional Experience

Current

2020- Assistant Professor, Kyung Hee University Hospital (Neurology)

Previous

2019 Fellow (Movement disorders), Kyung Hee University Hospital (Neurology)
2018 Fellow (Movement disorders), Seoul National University Hospital (Neurology)
2014-2017 Resident, Seoul National University Hospital (Neurology)
2013 Intern, Seoul National University Hospital

Selected Peer Reviewed Publications

1. Jung D, Yoo D, Kim J, Ahn TB, Mun KR. Identifying Parkinson's disease and its stages using static standing balance. NPJ Digit Med. 2024 Nov 30;7(1):347.
2. Chung SH, Yoo D, Ahn TB, Lee W, Hong J. Profiling Analysis of Tryptophan Metabolites in the Urine of Patients with Parkinson's Disease Using LC-MS/MS. Pharmaceuticals (Basel). 2023 Oct 20;16(10):1495.

3. Kim T, Ha SH, **Yoo D**, Park KS, Ahn TB. A Novel Variant of GCH1 in Dopa-Responsive Dystonia With Oculogyric Crises and Intrafamilial Phenotypic Heterogeneity. *J Mov Disord*. 2023 Sep;16(3):339-342.
4. **Yoo D**, Bang JI, Ahn C, Nyaga VN, Kim YE, Kang MJ, Ahn TB. Diagnostic value of α -synuclein seeding amplification assays in α -synucleinopathies: A systematic review and meta-analysis. *Parkinsonism Relat Disord*. 2022 Nov;104:99-109.
5. **Yoo D**, Lee W, Lee SJ, Sung JJ, Jeon GS, Ban JJ, Shin C, Kim J, Kim HS, Ahn TB. A Novel TFG Mutation in a Korean Family with α -Synucleinopathy and Amyotrophic Lateral Sclerosis. *Mov Disord*. 2022 Feb;37(2):384-391.
6. **Yoo D**, Lim Y, Son Y, Rho H, Shin C, Ahn TB. Dietary intake and plasma levels of polyunsaturated fatty acids in early-stage Parkinson's disease. *Sci Rep*. 2021 Jun 14;11(1):12489.
7. **Yoo D**, Lee JY, Kim YK, Yoon EJ, Kim H, Kim R, Nam H, Jeon B. Mild cognitive impairment and abnormal brain metabolic expression in idiopathic REM sleep behavior disorder. *Parkinsonism Relat Disord*. 2021 Sep;90:1-7.

Prediction and Detection of FOG: From Clinical Assessment to Emerging Technologies

Jeffrey M. HAUSDORFF

Center for the Study of Movement, Cognition and Mobility, Neurological Institute, Tel Aviv Sourasky Medical Center; Gray Faculty of Medical & Health Sciences & Sagol School of Neuroscience, Tel Aviv University Tel Aviv, Israel.

Freezing of gait (FOG) is a debilitating and unpredictable phenomenon that affects many, but not all, people with Parkinson's disease (PD). Its mysterious and episodic nature, appearing and disappearing apparently without rhyme or reason, makes it difficult to study, evaluate, and treat. Here, we outline approaches to FOG on two timescales: (1) assessing FOG severity in patients who already exhibit FOG, and (2) predicting the risk of developing FOG among those with PD who have not yet developed it.

For patients with established FOG, assessment methods include self-report and standardized clinical tests, with refined approaches evolving. The proportion of gait time affected by freezing—as rated by expert annotators—remains the gold standard. Emerging technologies based on wearable sensors and computer vision show promise for automated and scalable detection, as well as for at-home testing. We also summarize exciting evidence that suggests there may be a pre-clinical stage of FOG, and discuss how FOG may be best provoked in patients who they and their clinicians may not yet be aware of its presence.

For patients who do not yet have FOG, recent longitudinal studies point to identifiable risk factors. Our meta-analysis of 35 studies ($n=8,973$; mean follow-up 4.1 ± 2.7 years) revealed that higher age at onset, greater motor symptom severity, depression, anxiety, poorer cognition, and use of levodopa or COMT inhibitors significantly increase the risk of developing FOG. Importantly, such changes can sometimes be detected more than a decade before FOG onset. These findings support the feasibility of predicting FOG incidence and suggest that modifiable factors may provide opportunities to reduce risk, delay onset, or even prevent the future development of FOG.

Personal Information

Full Name Jeffrey M. HAUSDORFF

Current Position Professor, Tel Aviv University; Director, Center for the Study of Movement Cognition and Mobility

Department Neurological Institute

Organization Tel Aviv Sourasky Medical Center

Country Israel



Education / Training

Institution and Location	Degree	Year(s)	Field of Study
The Cooper Union, NY	BSE	1985	Engineering
Massachusetts Institute of Technology	MSME	1988	Biomechanics
Boston University	PhD	1995	Biomedical Engineering
Harvard Medical School	Post Doc	1997	Gerontology

Positions and Honors

Professional Experience

Current

2011- Full Professor, Sagol School of Neuroscience, Tel Aviv University

2011- Full Professor, Sagol School of Neuroscience, Tel Aviv University

2019- Professor, Rush Alzheimer's Disease Center and Department of Orthopedic Surgery, Rush University Medical Center

2025- Advisor to the Board of Directors, International Society for the Measurement of Physical Behaviour

2024- Board member, International Consortium on Freezing of Gait (ICFOG)

2020- Working Group Leader and Member of international task force to develop guidelines for the prevention and management of falls in older adults

Previous

1999-2002 Associate Director, Institute for Nonlinear Dynamics in Physiology and Medicine, Boston, MA, USA

1999-2002 Assistant Professor of Medicine, Harvard Medical School

2015-2025 Movement Disorders Society Task Force on Technology

2016-2025 Gait Advisory Committee for the Michael J. Fox Foundation

Honors

2013	Gerontology Society of America's Excellence in Rehabilitation of Aging Persons Award
2016	Fellow of the Gerontological Society of America
2019-2022	Listed among the top 0.01% of scientists based on impact of publications
2023	Aufzien Family Center for the Prevention and Treatment of Parkinson's Disease APPD Established Scientist Prize 2023
2024	The Igor Ornstein Chair for the Study of Geriatrics, Tel Aviv University
2025	Over 100,000 citations (Google Scholar, May, 2025)

Selected Peer Reviewed Publications

1. Hausdorff JM, Schaafsma JD, Balash Y, Bartels AL, Gurevich T, Giladi N. Impaired regulation of stride variability in Parkinson's disease subjects with freezing of gait. *Exp Brain Res* 2003;149:187-194.
2. Bloem BR, Hausdorff JM, Visser JE, Giladi N. Falls and freezing of gait in Parkinson's disease: a review of two interconnected, episodic phenomena. *Mov Disord* 2004;19:871-884.
3. Giladi N, Hausdorff JM. The role of mental function in the pathogenesis of freezing of gait in Parkinson's disease. *J Neurol Sci* 2006;248:173-176.
4. Maidan I, Plotnik M, Mirelman A, Weiss A, Giladi N, Hausdorff JM. Heart rate changes during freezing of gait in patients with Parkinson's disease. *Mov Disord* 2010;25:2346-2354.
5. Manor B, Dagan M, Herman T, Gouskova NA, VanderHorst VG, Giladi N, Trivison TG, Pascual-Leone A, Lipsitz LA, Hausdorff JM. Multitarget Transcranial Electrical Stimulation for Freezing of Gait: A Randomized Controlled Trial. *Mov Disord* 2021;36:2693-2698.
6. Denk D, Herman T, Zoetewei D, Ginis P, Brozgov M, Cornejo TP, Decaluwe E, Ganz N, Palmerini L, Giladi N, Nieuwboer A, Hausdorff JM. Daily-Living Freezing of Gait as Quantified Using Wearables in People With Parkinson Disease: Comparison With Self-Report and Provocation Tests. *Phys Ther* 2022;102.
7. Salomon A, Gazit E, Ginis P, Urazalinov B, Takoi H, Yamaguchi T, Goda S, Lander D, Lacombe J, Sinha AK, Nieuwboer A, Kirsch LC, Holbrook R, Manor B, Hausdorff JM. A machine learning contest enhances automated freezing of gait detection and reveals time-of-day effects. *Nat Commun* 2024;15:4853.
8. Mancini M, McKay JL, Cockx H, D'Cruz N, Esper CD, Filtjens B, Heimler B, MacKinnon CD, Palmerini L, Roerdink M, Young WR, Hausdorff JM. Technology for measuring freezing of gait: Current state of the art and recommendations. *J Parkinsons Dis* 2025;15:19-40.

Practical Management of Freezing of Gait Based on Pathomechanism

Jeong-Ho PARK

Department of Neurology, College of Medicine, Soonchunhyang University Bucheon Hospital, Republic of Korea

Freezing of gait (FOG) is one of the most challenging and disabling motor symptoms in Parkinson's disease and related disorders. Despite its prevalence, effective management remains complex due to its heterogeneous clinical presentations and underlying mechanisms. This lecture offers a practical, phenotype-based approach to FOG treatment. We will review key clinical patterns and provide guidance on tailored interventions, including medication adjustments, cueing techniques, and rehabilitative strategies. Join us to enhance your diagnostic precision and therapeutic effectiveness in managing this complex and treatment-resistant symptom.

Personal Information

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Country Republic of Korea



Education / Training

Institution and Location	Degree	Year(s)	Field of Study
Soonchunhyang Univ. Asan, Chungchungnamdo, Republic of Korea	MD	1991-1997	Medicine
Soonchunhyang Univ. Hospital	Internship/Residency	1997-2002	Neurology
Soonchunhyang Univ. Asan, Chungchungnamdo, Republic of Korea	PhD	2011	Eye movements in Parkinson's disease
Balance lab., Parkinson Center of Oregon, OHSU, Portland, OR	Post-doctoral fellowship	2013	Gait and Balance disorders in PD

Positions and Honors

Professional Experience

Current

2018 - Professor, SoonChunHyang Univ. Bucheon Hospital

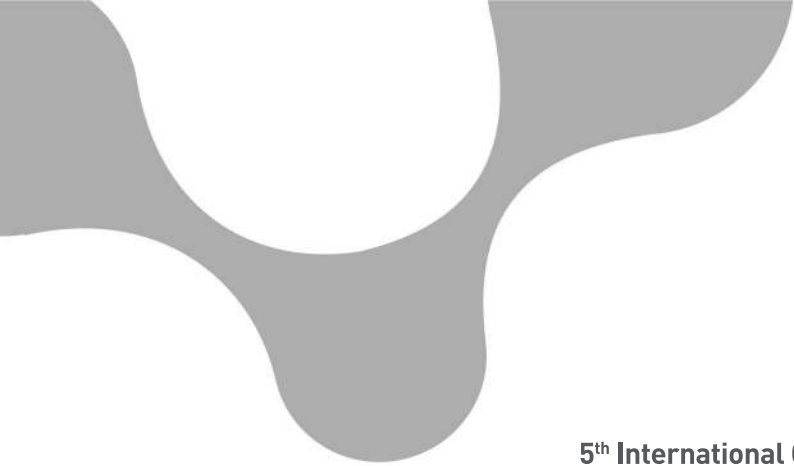
Previous

2012 - 2018 Associate Professor, SoonChunHyang Univ. Bucheon Hospital

Selected Peer Reviewed Publications

1. Park, JH, et al. "Initial Vestibular Function May Be Associated with Future Postural Instability in Parkinson's Disease." J Clin Med 2022; 11(19).
2. Park JH, Kang SY. Dizziness in Parkinson's disease patients is associated with vestibular function. Sci Rep 2021;11:18976.
3. Park SA, Jung JM, Park JH, et al. SWATH-MS analysis of cerebrospinal fluid to generate a robust battery of biomarkers for Alzheimer's disease. Sci Rep 2020;10:7423.

4. Lee J, Lee AL, **Park JH**, et al. The clinical application of nigrosome 1 detection on high-resolution susceptibility-weighted imaging in the evaluation of suspected Parkinsonism: The real-world performance and pitfalls. PLoS One 2020;15:e0231010.
5. **Park JH**, Kang YJ, Horak FB. What Is Wrong with Balance in Parkinson's Disease? J Mov Disord 2015;8:109-114
6. **Park JH**, Mancini M, Carlson-Kuhta P, Nutt JG, Horak FB. Quantifying effects of age on balance and gait with inertial sensors in community-dwelling healthy adults. Exp Gerontol 2016;85:48-58.
7. Park JE, Hallett M, **Park JH**, et al. Effects of anodal stimulation and motor practice on limb-kinetic apraxia in Parkinson's disease. Exp Brain Res 2022.



ICKMDS 2025

5th International Conference of Korean Movement Disorder Society

Bridging from the Origin to Clinical Practice:
A Beginning Has an Ending

DAY 1_ October 31 (Fri), 2025

Room B

Parallel Session 4

Classification of Dystonia—Pathophysiological Perspectives

Chairs Raymond ROSALES (Philippines)
Young H. SOHN (Korea)



Overview of Pathophysiology of Dystonia: A Decade Since the New Classification of Dystonia in 2013

Young H. SOHN

Neurology, Yonsei University College of Medicine, Seoul, Korea

Dystonia is defined as a movement disorder characterized by sustained or intermittent muscle contractions causing abnormal, often repetitive, movements or postures, or both. Dystonic movements and postures are typically patterned and repetitive, and may be tremulous and jerky. The clinical features of dystonia belong to a varied phenomenological spectrum. This clinical complexity comes in parallel with marked heterogeneity of etiologies and biological mechanisms. This heterogeneity has led to different classification approaches that have been evolved over the years. In 1976, Fahn and Eldridge first distinguished primary dystonia (with or without a hereditary pattern) from secondary dystonia (with other hereditary neurological conditions or due to a known environmental cause), and psychological forms of dystonia. Subsequently, Fahn, Marsden, and Calne proposed a classification of dystonia based on three axes: age at onset, distribution, and etiology. Later, the etiological classification was expanded to include four subgroups of dystonia syndromes: primary, dystonia-plus, secondary, and heredo-degenerative. The changing system of classification for dystonia may reflect an increased understanding of the various clinical manifestations and etiologies. Due in part to increased understanding of etiology, in 2013, an innovative classification system was proposed to divide dystonia into two axes: axis 1 - clinical characteristics, including age at onset, body distribution, temporal pattern and associated features; and axis 2 – etiology, which includes nervous system pathology and inheritance. Although this two-axis plan has solved many issues related to prior classifications, some areas where varied interpretations lead to inconsistent applications still exists. Here this session reviews the change of concepts related to dystonia classification based on historical background, and discusses emerging controversies related to current concept and classification of dystonia.

Personal Information

Full Name Young H. SOHN
Current Position Professor
Department Neurology
Organization Yonsei University Health System
Country Korea



Education / Training

Institution and Location	Degree	Year(s)	Field of Study
Yonsei University, Korea	MD	1986	Medicine
Yonsei University, Korea	PhD	1995	Neuroscience
Yonsei University Medical Center, Korea	Korean National Board	1990	Neurology
NINDS, NIH, USA	Clinical Associate	2000-2002	Human Motor Control

Positions and Honors

Professional Experience

Current

2008 - 2025 Professor of Neurology, Yonsei University Health System
2010 - 2025 Chief, Yonsei Parkinson Center, Yonsei University

Previous

2005 - 2011 Member of Executive Committee, AOS-MDS
2007 - 2012 Editor, Journal of Movement Disorders
2011 - 2014 Editorial Board Member, Current Treatment Options in Neurology
2014 - 2015 President, Korean Movement Disorder Society

Honors

2005 The Superior Research Professor Award, Yonsei University
2012 The Academic Award, Korean Movement Disorder Society

Selected Peer Reviewed Publications

1. Sohn YH, Hallett M. Disturbed surround inhibition in focal hand dystonia. *Ann Neurol* 2004;56:595-599.
2. Sohn YH, Lee PH. Paroxysmal choreodystonic disorders. *Handb Clin Neurol* 2011; 100:367-373.
3. Lee PH, Lee JE, Kim H-S, Song SK, Lee HS, Nam HS, Cheong J-W, Jeong Y, Park H-J, Kim DJ, Nam CM, Lee JD, Kim HO, **Sohn YH**. A randomized trial of mesenchymal stem cells in multiple system atrophy. *Ann Neurol* 2012; 72:32-40.
4. Kang SY, Hallett M, **Sohn YH**. Exercise-induced strengthening of inter-digital connections in musicians. *Clin Neurophysiol* 2013; 124:1622-1627.
5. Lee DH, Oh JS, Ham JH, Lee JJ, Lee I, Lee PH, Kim JS, Sohn YH. Is normosmic Parkinson's disease a unique clinical phenotype? *Neurology* 2015; 85:1270-1275.
6. Lee Y, Oh JS, Chung SJ, Chung SJ, Kim SJ, Nam CM, Lee PH, Kim JS, **Sohn YH**. Does smoking impact dopamine neuronal loss in de novo Parkinson's disease? *Ann Neurol* 2017; 82:850-854.
7. Chung SJ, Lee HS, Yoo HS, Lee YH, Lee PH, **Sohn YH**. Patterns of striatal dopamine depletion in early Parkinson's disease: Prognostic relevance. *Neurology* 2020; 95:e280-e290.

Genetic Causes of Dystonia and the Associated Pathogenesis

Katja LOHMANN

Institute of Neurogenetics and Institute of Human Genetics, University of Lübeck, Germany

Dystonia is a clinically and genetically highly heterogeneous neurological disorder. Presently, the Movement Disorder Society Task Force for the Nomenclature of Genetic Movement Disorders (MDS Nomenclature Task Force) recognizes >50 established dystonia genes. With ongoing sequencing efforts, the list of dystonia candidate genes continues to grow, reaching over 400 potential dystonia-linked genes by now. Although the molecular characterization of novel dystonia genes lags behind, several meaningful molecular insights have recently been gained. While these discoveries improve genetic testing and counseling, their translation into new (molecular) therapies is still limited. However, we are beginning to understand shared pathophysiological pathways and molecular mechanisms. It has become clear that dystonia results from a dysfunctional network involving the basal ganglia, cerebellum, thalamus, and cortex. On the molecular level, more than a handful of, often intertwined, pathways have been linked to pathogenic variants in dystonia genes, including gene transcription during neurodevelopment (e.g., KMT2B, THAP1), calcium homeostasis (e.g., ANO3, HPCA), striatal dopamine signaling (e.g., GCH1, GNAL), endoplasmic reticulum stress response (e.g., EIF2AK2, PRKRA, TOR1A), autophagy (e.g., VPS16), and others. Thus, different forms of dystonia can be molecularly grouped, which may facilitate treatment development in the future.

Personal Information

Full Name **Katja LOHMANN**
 Current Position **Associate Professor**
 Department **Institute of Neurogenetics AND Institute of Human Genetics**
 Organization **University of Luebeck**
 Country **Germany**



Education / Training

Institution and Location	Degree	Year(s)	Field of Study
University of Dresden, Germany	B.Sc	2	Biology
University of Halle, Germany	M.Sc	3	Genetics
University of Luebeck, Germany AND Massachusetts General Hospital, Boston, MA	PhD	3	Neurogenetics
Oregon Health & Science University, Portland, OR	Postdoc	0.25	Statistical Genetics
University of Lübeck	Postdoc, Assistant Prof., Associate Prof.	20	Neurogenetics and Human Genetics

Positions and Honors

Professional Experience

Current

Since 2017 Associate Professor, University of Luebeck

Previous

2010-2017 Assistant Professor, University of Luebeck

Honors

2017 David Marsden Award

Selected Peer Reviewed Publications

1. Laurie S, Steyaert W, de Boer E, Polavarapu K, Schuermans N, Sommer AK, [85 authors], Solve-RD DITF-GENTURIS, Solve-RD DITF-ITHACA, Solve-RD DITF-EURO-NMD, Solve-RD DITF-RND, Solve-RD consortium, Brookes AJ, Evangelista T, Gilissen C, Graessner H, Hoogerbrugge N, Ossowski S, Riess O, Schüle R, Synofzik M, Verloes A, Matalonga L, Brunner HG, **Lohmann K (shared last author)**, de Voer RM, Töpf A, Vissers L.E.L.M., Beltran S, Hoischen A. Genomic Reanalysis of a Pan-European Rare Disease Resource Yields >500 New Diagnoses. *Nat Med* 2025. doi: 10.1038/s41591-024-03420-w. Online ahead of print. PMID: 39825153
2. Thomsen M, Lange LM, Zech M, **Lohmann K**. Genetics and Pathogenesis of Dystonia. *Annu Rev Pathol* 2024;19:99-131. doi: 10.1146/annurev-pathmechdis-051122-110756. PMID: 37738511
3. Rafehi H, Read J, Szmulewicz DJ, Davies KC, Snell P, Fearnley LG, Scott L, Thomsen M, Gillies G, Pope K, Bennett MF, Munro JE, Ngo KJ, Chen L, Wallis MJ, Butler EG, Kumar KR, Wu KH, Tomlinson SE, Tisch S, Malhotra A, Lee-Archer M, Dolzhenko E, Eberle MA, Roberts LJ, Fogel BL, Brüggemann N, **Lohmann K**, Delatycki MB, Bahlo M, Lockhart PJ. An intronic GAA repeat expansion in FGF14 causes the autosomal-dominant adult-onset ataxia SCA27B/ATX-FGF14. *Am J Hum Genet* 2023;110:105-119. doi: 10.1016/j.ajhg.2022.11.015. PMID: 36493768
4. Lange LM, Junker J, Loens S, Baumann H, Olschewski L, Schaake S, Madoev H, Petkovic S, Kuhnke N, Kasten M, Westerberger A, Domingo A, Marras C, König IR, Camargos S, Ozelius LJ, Klein C, **Lohmann K**. Genotype-Phenotype Relations for Isolated Dystonia Genes: MDSGene Systematic Review. *Mov Disord* 2021;36:1086-1103. doi: 10.1002/mds.28485. PMID: 33502045
5. Baumann H, Ott F, Weber J, Trilck-Winkler M, Münchau A, Zittel S, Kostić VS, Kaiser FJ, Klein C, Busch H, Seibler P, **Lohmann K**. Linking Penetrance and Transcription in DYT-THAP1: Insights From a Human iPSC-Derived Cortical Model. *Mov Disord* 2021, doi: 10.1002/mds.28506. PMID: 33547842.
6. Marras C, Lang A, van de Warrenburg BP, Sue CM, Tabrizi SJ, Bertram L, Mercimek-Mahmutoglu S, Ebrahimi-Fakhari D, Warner TT, Durr A, Assmann B, **Lohmann K**, Kostic V, Klein C. Nomenclature of genetic movement disorders: Recommendations of the international Parkinson and movement disorder society task force. *Mov Disord* 2016;31:436-457.
7. **Lohmann K**, Wilcox RA, Winkler S, Ramirez A, Rakovic A, Park J-S, Arns B, Lohnau T, Groen JL, Kasten M, Brüggemann N, Hagenah J, Schmidt A, Kaiser FJ, Kumar KR, Zschiedrich K, Alvarez-Fischer D, Altenmüller E, Ferbert A, Lang AE, Münchau A, Kostic V, Simonyan K, Agzarian M, Ozelius LJ, Langeveld APM, Sue CM, Tijssen MAJ, Klein C. Whispering dysphonia (DYT4 dystonia) is caused by a mutation in the TUBB4 gene. *Ann Neurol* 2013;73:537-545.

Task-Specific vs. Non-Task-Specific Focal Dystonia: Shared Pathophysiology or Distinct Mechanisms?

Hae-Won SHIN

Department of Neurology, Chung-Ang University College of Medicine, Korea

Focal dystonia is a disabling movement disorder defined by sustained or intermittent muscle contractions that cause abnormal postures or repetitive movements. Two broad subtypes can be identified. Task-specific focal dystonia (TSFD) emerges only during highly skilled actions such as handwriting or musical performance, whereas non-task-specific focal dystonia (NTSFD) manifests across a wider range of everyday postures and movements, such as cervical dystonia and blepharospasm. Although these clinical entities share fundamental physiological abnormalities, recent advances in neurophysiological and imaging studies have highlighted important differences in their underlying mechanisms.

Across focal dystonias, several abnormalities are consistently observed. These include loss of intracortical inhibition, impaired surround inhibition, aberrant and poorly regulated cortical plasticity, and dysfunction of sensorimotor integration. TMS studies reveal that patients with dystonia have difficulty selectively activating relevant muscles while suppressing unwanted activity, resulting in overflow and co-contraction. PAS protocols show exaggerated and spatially diffuse plastic changes, indicating impaired homeostatic control of synaptic efficacy. Sensorimotor integration deficits are demonstrated by reduced short-latency afferent inhibition and abnormal temporal discrimination thresholds.

Within this shared background, TSFD and NTSFD diverge in several ways. TSFD appears to preferentially affect circuits specialized for precise motor skills. TMS motor mapping, EEG/MEG, and task-based fMRI reveal abnormalities in somatotopic organization and task-triggered cortical disinhibition, implicating dysfunction of premotor–parietal–M1 loops. In writer’s cramp, ccPAS has demonstrated hyper-responsive parietal-to-motor plasticity, suggesting abnormal sensory–state estimation during overlearned actions. These findings support the concept of maladaptive, use-dependent reorganization as a central mechanism of TSFD.

NTSFD reflects more widespread network instability. Resting-state fMRI and structural connectivity analyses consistently demonstrate altered functional coupling within the cerebello-thalamo-cortical and striato-pallidal loops, even outside of task performance. Lesion-network mapping has further shown that anatomically diverse lesions leading to dystonia converge on circuits linked to the cerebellum and basal ganglia. Moreover, experimental studies combining cerebellar stimulation with PAS indicate impaired cerebellar control over cortical plasticity in NTSFD, underscoring the contribution of intrinsic cerebellar dysfunction in this form.

Taken together, the current evidence suggests that TSFD and NTSFD share partially overlapping but circuit-biased pathophysiology. TSFD is characterized by maladaptive plasticity within skill-specific parieto-premotor–M1 pathways, whereas NTSFD reflects baseline instability of core motor output loops integrating the basal ganglia and cerebellum. This framework emphasizes the importance of considering phenotypic classification not only for mechanistic understanding but also as the foundation for future development of targeted, phenotype-specific therapies.

Personal Information

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Education / Training

Institution and Location	Degree	Year(s)	Field of Study
Yonsei University College of Medicine	MD	2000	Medicine
Yonsei University	MS	2004	Neurology
Yonsei University	PhD	2009	Neurology
NINDS, National Institute of Health, USA	Visiting Researcher	2017	Human Motor Control

Positions and Honors

Professional Experience

Current

2020-Current Professor, Chung-Ang University

Previous

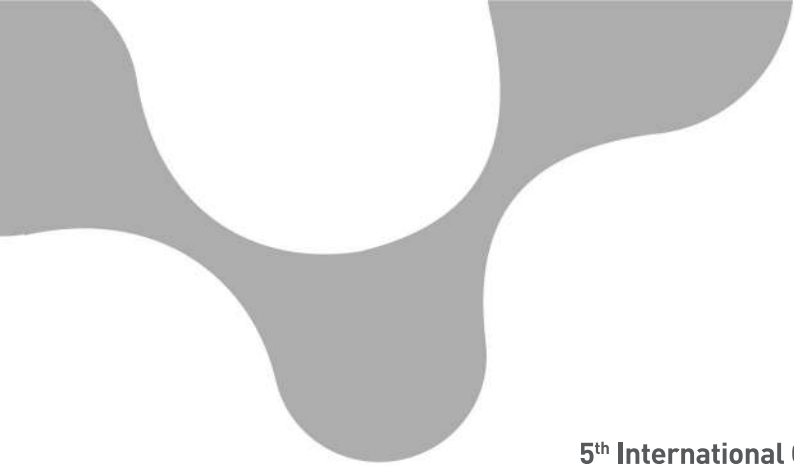
2013-2020 Associate Professor, Chung-Ang University

2010-2013 Assistant professor, Chung—Ang University

Selected Peer Reviewed Publications

1. Cho HJ, Shin H-W, Panyakaew P, Kassavetis P, Popa T, Wu T, et al. Differential induction of Parieto-motor plasticity in writer's cramp and cervical dystonia. *Neurobiology of disease*. 2024
2. Shin H-W, Hong S-W, Youn YC. Clinical aspects of the differential diagnosis of Parkinson's disease and parkinsonism. *Journal of Clinical Neurology (Seoul, Korea)*. 2022;18(3):259.
3. Shin H-W, Cho HJ, Lee SW, Shitara H, Hallett M. Sensory tricks in cervical dystonia correlate with enhanced brain activity during motor preparation. *Parkinsonism & related disorders*. 2021;84:135-8.

4. **Shin H-W**, Hallett M. Low-frequency transcranial magnetic stimulation of the left dorsal premotor cortex in patients with cervical dystonia. *Parkinsonism & Related Disorders*. 2020;76:13-5.
5. Lee S, Chung SJ, **Shin H-W**. Neuropsychiatric symptoms and quality of life in patients with adult-onset idiopathic focal dystonia and essential tremor. *Frontiers in Neurology*. 2020;11:1030.
6. **Shin H-W**, Ryu H-S, Chung SJ. Long-term Effects of Bilateral Subthalamic Deep Brain Stimulation on Postural Instability and Gait Disturbance in Parkinson's Disease (P1. 8-033). *Neurology*. 2019;92(15_supplement):P1. 8-033.
7. **Shin H-W**, Hallett M, Sohn YH. Cerebellar repetitive transcranial magnetic stimulation for patients with essential tremor. *Parkinsonism & Related Disorders*. 2019;64:304-7.



ICKMDS 2025

5th International Conference of Korean Movement Disorder Society

Bridging from the Origin to Clinical Practice:
A Beginning Has an Ending

DAY 1_ October 31 (Fri), 2025

Room B

Oral Presentation Session 1

Chairs Wooyoung JANG (Korea)
Seung-Hwan LEE (Korea)



Early Fall Risk Prediction in Parkinson's Disease Through Feature Importance and Statistical Methods Based on Machine Learning

Sumin KIM¹, Hakje YOO^{2,3}, Jiwon YOO⁴, Jin Whan CHO^{4,5}, Soonjung OH², Myung Jin CHUNG^{2,6,7}, Jinyoung YOUN^{4,5}

¹Neurology, Kangbuk Samsung Hospital, Sungkyunkwan University School of Medicine, Seoul, Republic of Korea

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⁷Data Convergence and Future Medicine, Sungkyunkwan University School of Medicine, Seoul, Republic of Korea

Background & Objective:

Falls can occur early in Parkinson's disease (PD) and lead to morbidity and mortality, but early prediction is challenging due to heterogeneous motor and non-motor features. We aimed to identify fall-related biomarkers and develop a machine learning model by integrating clinical and gait data from two medical centers.

Methods:

Clinical and gait data were collected from 450 subjects, with a larger training dataset from Samsung Medical Center (SMC, n=342) and a smaller validation dataset from Korea University Medical Center (KUMC, n=108). Assessments included demographics, K-ABC, TUG, Tinetti test, PHQ, HADS, K-MoCA, SCOPA-AUT, RBDQ-KR, and fall status from the Gait and Falls Questionnaire (GFQ). Gait parameters were measured with GAITRite. Fall history determines group allocation (controls, PD fallers, PD non-fallers). Data were preprocessed by scaling and oversampling (SMOTE/Borderline SMOTE). Features were selected using statistics-based (correlation, chi-square, mutual information) and importance-based (Extra Trees Classifier) methods. Seven algorithms were compared with Optuna-based hyperparameter optimization, and models were validated internally on SMC and externally on KUMC (Figure 1)

Results:

Ultimately 273 subjects were analyzed: 33 PD fallers, 171 non-fallers, and 69 controls. The best performing machine learning model (Extra Trees Classifier) achieved 88–89% accuracy in classifying the three groups. The importance-based method showed 85% internal accuracy but dropped to 73% externally, while the statistics-based method maintained 89% internal and 87% external accuracy (Table 1). Key biomarkers were fear of falling, impaired balance, and reduced gait velocity/stride length (Figure 2). ROC analysis showed excellent discrimination (AUC: controls 0.98, PD fallers 0.99, non-fallers 0.96).

Conclusions:

Our machine learning model integrating clinical and gait data predicted fall risk in PD, with fear of falling, balance, and gait stability emerging as key biomarkers for early intervention.

Figure

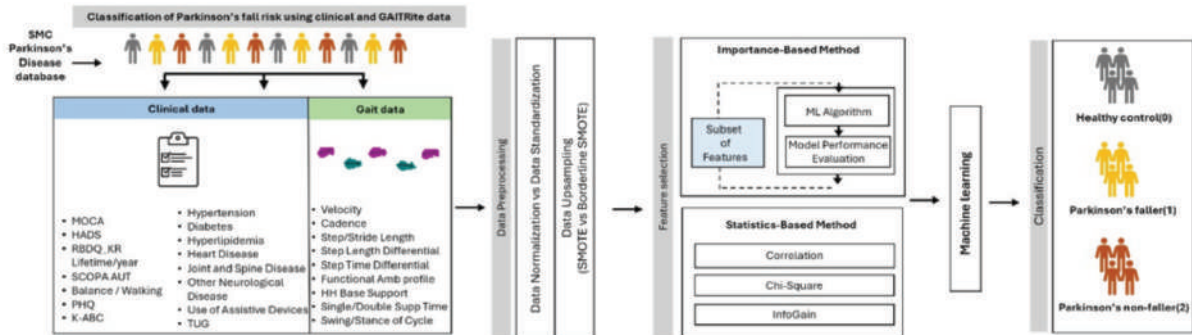


Figure 1. Study Flowchart of Fall Risk Prediction in Parkinson's Disease

Figure

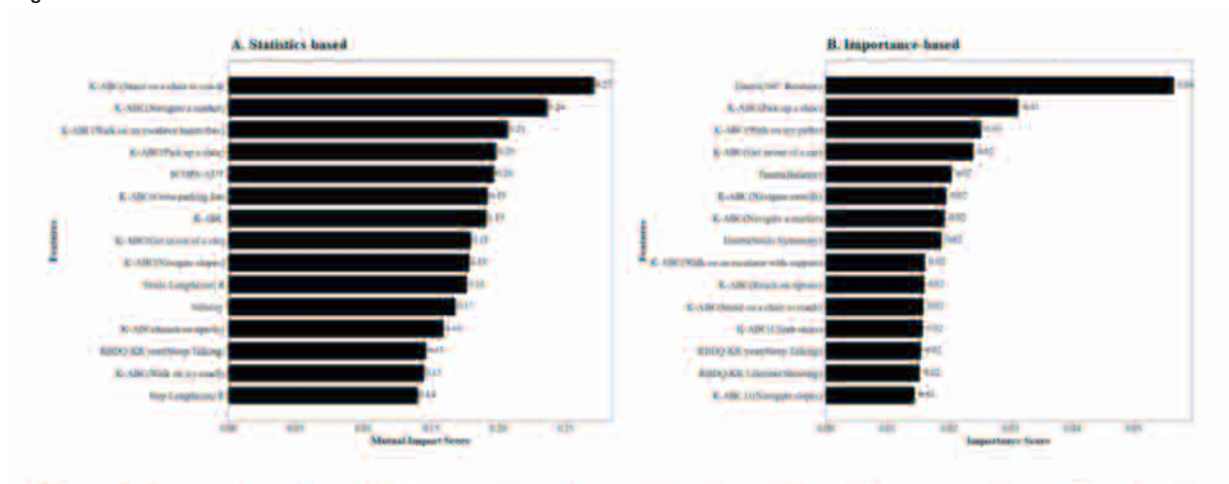


Figure 2. Comparison of top 15 features selected by statistics-based (A) and importance-based (B) methods.

Table

A. Internal validation						
Methods	Dataset		Accuracy (%)	Precision	Recall	F1-score
Statistics-Based Method	SMC Data set	Minmax scaler + BorderlineSMOTE	83	0.83	0.83	0.83
		Standard scaler + BorderlineSMOTE	84	0.84	0.84	0.84
		Minmax scaler + SMOTE	89	0.89	0.89	0.89
		Standard scaler + SMOTE	84	0.84	0.84	0.84
		Skewness+ Minmax scaler + BorderlineSMOTE	84	0.86	0.86	0.85
		Skewness+ Standard scaler + Borderline SMOTE	86	0.86	0.86	0.86
Importance-Based Method	SMC Data set	Minmax scaler + BorderlineSMOTE	84	0.85	0.84	0.83
		Standard scaler + BorderlineSMOTE	86	0.86	0.86	0.85
		Minmax scaler + SMOTE	85	0.85	0.85	0.84
		Standard scaler + SMOTE	85	0.85	0.85	0.84
		Skewness+ Minmax scaler + BorderlineSMOTE	85	0.85	0.85	0.84
		Skewness+ Standard scaler + Borderline SMOTE	85	0.85	0.85	0.84

B. External validation							
Methods	Model	Dataset		Accuracy (%)	Precision	Recall	F1-score
Statistics-Based Method	Extra Tree Classifier	KUMC Dataset	Minmax scaler + SMOTE	87	0.88	0.87	0.87

Table 1. Validation results of the best-performing model: internal validation (A) and external validation (B)

Keywords

Parkinson's disease, Fall, Machine learning, Gait analysis, Biomarkers

Personal Information

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Education / Training

Institution and Location	Degree	Year(s)	Field of Study
Sungkyunkwan University, Seoul	Master Student	2	Movement disorder
Inje University, Pusan	M.D.	6	Medicine

Positions and Honors

Professional Experience

Current

2021-2025 Resident, Kangbuk samsung hospital

Previous

2019-2020 Intern, Inje University Sanggye Paik Hospital

Selected Peer Reviewed Publications

1. Sumin Kim, Sorae Lee, Jun-Sang Sunwoo, Acute Ischemic Stroke in a Patient with Metastatic Gastric Cancer after Ramucirumab Chemotherapy. Journal of the Korean Neurological Association. 2024.
2. Sumin Kim, Sorae Lee, Jun-sang Sunwoo, Unilateral Retro-orbital Headache Secondary to Septic Cavernous Sinus Thrombosis: A Case Report. Headache and Pain Research. 2023.
3. Sumin Kim, Jun-Sang Sunwoo, Acute Necrotizing Encephalopathy Associated with COVID-19. Journal of the Korean Neurological Association. 2024.
4. Sorae Lee, Sumin Kim, Sanggyu Han, So-Ei Ann, Pil-Wook Chung, Bum Chun Suh, Jun-Sang Sunwoo, Invasive Aspergillosis Manifesting as Painful Ophthalmoplegia. Korean Journal of Headache. 2023.
5. Sorae Lee, Sumin Kim, Jun-Sang Sunwoo, Otogenic enterobacter cloacae meningitis complicated with pneumocephalus. Encephalitis. 2023.
6. So-Ei Ann, Suho Ro, Yun Hyeong Jeong, Sumin Kim, Pil-Wook Chung, Hemichorea associated with subcortical border-zone infarction and middle cerebral artery stenosis. Journal of geriatric neurology. 2021.

Repetitive Transcranial Magnetic Stimulation Improves Static Balance Control in Cerebellar Ataxia: A Pilot Study

Ho-Won LEE¹, Sumin LEE², Minyoung JUNG²

¹Neurology, Kyungpook National University Chilgok Hospital, Daegu, Republic of Korea

²Cognitive Science Research Group, Korea Brain Research Institute, Daegu, Republic of Korea

Background & Objective:

Cerebellar ataxia (CA) patients suffer from motor dysfunctions, including impaired balance. Repetitive transcranial magnetic stimulation (rTMS) has shown promise in improving motor functions in other movement disorders. This pilot study aimed to investigate the effects of cerebellar rTMS on balance in CA patients.

Methods:

Ten CA patients (4 males, mean age = 58 ± 9.07 years) received 50 Hz rTMS over the cerebellum for 7 minutes, for five consecutive days. Static balance was assessed using a balance trainer (BT4) at baseline (V0), immediately after 5 days of rTMS (V1), and at a 6-week follow-up (V2).

Results:

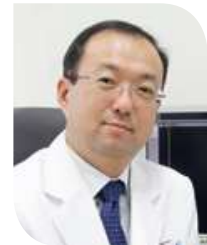
The Friedman test revealed significant improvements in static balance at V1 compared to V0. Specifically, significant changes were observed in medial-lateral and anterior-posterior sway, particularly in anterior-posterior displacement (ΔY) and total displacement (Δd) during the eyes-open condition, and medial-lateral displacement (ΔX) during the eyes-closed condition. These improvements were followed by a return to baseline at the 6-week follow-up.

Conclusions:

High-frequency rTMS applied to the cerebellum can temporarily improve static balance control in patients with CA. These findings suggest rTMS may be a potential therapeutic tool, although further research is needed to optimize stimulation protocols for sustained effects.

Personal Information

Full Name Ho-Won LEE
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Country Korea



Education / Training

Institution and Location	Degree	Year(s)	Field of Study
Kyungpook National University, Korea	Ph.D.	2008	Neuroscience
Center for Movement Disorders & Neurorestoration, University of Florida, USA	Fellowship	2013	Neurology
Department of Neurology and Alzheimer Disease Center, Baylor College of Medicine, USA	Fellowship	2003	Neurology
Kyungpook National University Hospital, Korea	Residency	1999 - 2002	Neurology
Kyungpook National University, Korea	MD	1996	Medicine

Positions and Honors

Professional Experience

Current

2017~ Full Professor, Department of Neurology, School of Medicine, Kyungpook National University

Previous

2010~2011 Associate Dean, Research Program, School of Medicine, Kyungpook National University

2011~2020 Head, Neurology Center, Kyungpook National University Medical Center

2011~2024 Director, Geriatric Health Care Center, Kyungpook National University Medical Center

2022~2024 Director, Brain Science & Engineering Institute, Kyungpook National University

Selected Peer Reviewed Publications

1. Kim S, Sharma C, Hong J, Kim JH, Nam Y, Kim MS, Lee TY, Kim KS, Suk K, **Lee HW**, Kim SR. Post-symptomatic administration of hMSCs exerts therapeutic effects in SCA2 mice Stem Cell Res Ther. 2024
2. Park N, Sharma C, Jung UJ, Kim S, Nam Y, Kim KS, Suk K, Lee HW, Kim SR. Mesenchymal Stem Cell Transplantation Ameliorates Ara-C-Induced Motor Deficits in a Mouse Model of Cerebellar Ataxia. J Clin Med. 2023
3. Kang K, **Lee HW**, Lee DH. Plasma exchange: An effective method to eliminate neurotoxic lipophilic chemicals from blood. Alzheimers Dement. 2022
4. Kim NG, Effken JA, **Lee HW**. Impaired Affordance Perception as the Basis of Tool Use Deficiency in Alzheimer's Disease. Healthcare (Basel). 2022
5. Lim YH, Baek Y, Kang SJ, Kang K, **Lee HW**. Clinical application of the experimental ADL test for patients with cognitive impairment: pilot study. Sci Rep. 2021
6. Ko PW, Park S, Kang K, Lim YH, Kim SR, Suk K, Kim KS, **Lee HW**. Human Allogeneic Bone Marrow-Derived Mesenchymal Stem Cell Therapy for Cerebellar Ataxia: A Case Report. Medicina (Kaunas). 2021
7. **Lee HW**, Lim YH, Kim SH. Dizziness in patients with cognitive impairment. J Vestib Res. 2020

Machine Learning Derived Probabilistic Scoring of Spatiotemporal Gait Patterns as a Digital Biomarker of Parkinsonian Gait Severity Based on Real World Data

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¹Department of Biomedical Informatics, Ajou University School of Medicine, Suwon, Republic of Korea

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Background & Objective:

Gait disturbance is a major manifestation of parkinsonism, yet its assessment remains subjective. Conventional scales such as the Unified Parkinson's Disease Rating Scale (UPDRS) provide only coarse quantification, while heterogeneous gait patterns across patients and healthy individuals complicate normative definitions. Machine learning-based approaches may offer objective metrics of gait severity.

Methods:

We retrospectively analyzed GAITrite® spatiotemporal gait assessments from 1,602 patients with gait complaints between February 2023 and March 2025. Diagnostic labels for parkinsonism (n=1,005) and without parkinsonism (n=597) were obtained from electronic medical records. Subsets included patients with motor UPDRS part 3 scores within 90 days of gait measurement (n=340) and those with prior striatal standardized uptake value ratios (SUVRs) from F-18 FP-CIT PET (n=171). A LightGBM classifier was trained with an 85:15 patient-level stratified split and five-fold cross-validation for hyperparameter tuning. Out-of-fold probabilistic scores, termed the Parkinsonism Gait Probability Score (P-GPS), were generated across the dataset. Generalized additive models (GAMs) tested associations of P-GPS with mUPDRS and DAT SUVR, adjusting for age, sex, levodopa timing, and acquisition intervals.

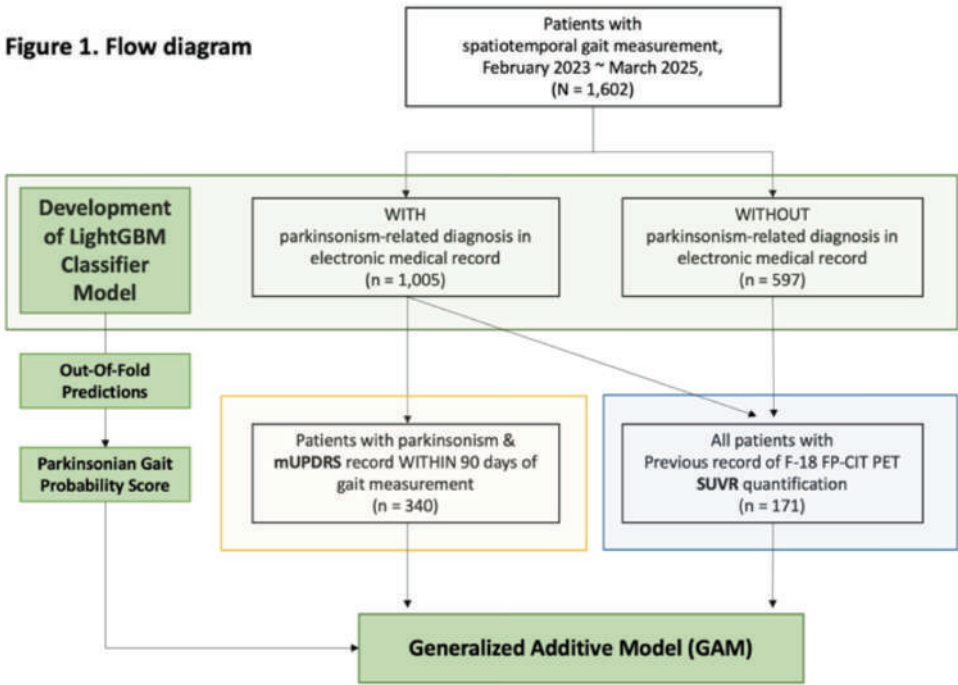
Results:

The model achieved an AUROC of 0.780, with PPV 0.815 and NPV 0.611, indicating robust phenotypic classification despite clinical heterogeneity. P-GPS was significantly associated with higher UPDRS part 3 severity (p=0.001), reflecting disease burden. Higher probabilities were also linked to reduced putaminal DAT binding (p=0.006), consistent with nigrostriatal degeneration.

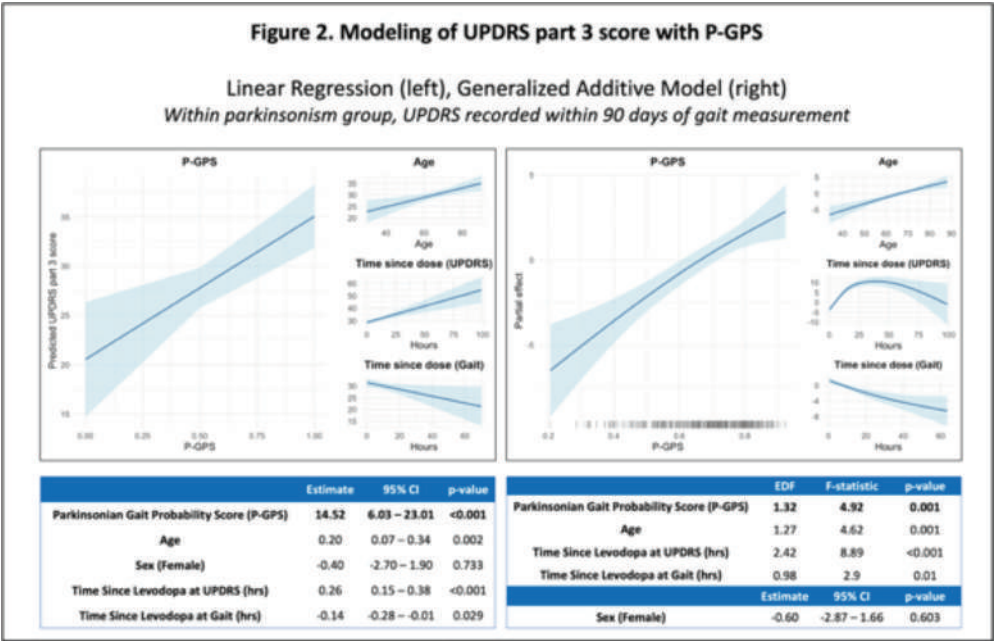
Conclusions:

A LightGBM model trained on spatiotemporal gait features produced P-GPS, a probabilistic score that discriminates parkinsonian gait and aligns with established clinical and imaging markers. P-GPS represents a promising objective digital biomarker for quantifying parkinsonian gait severity.

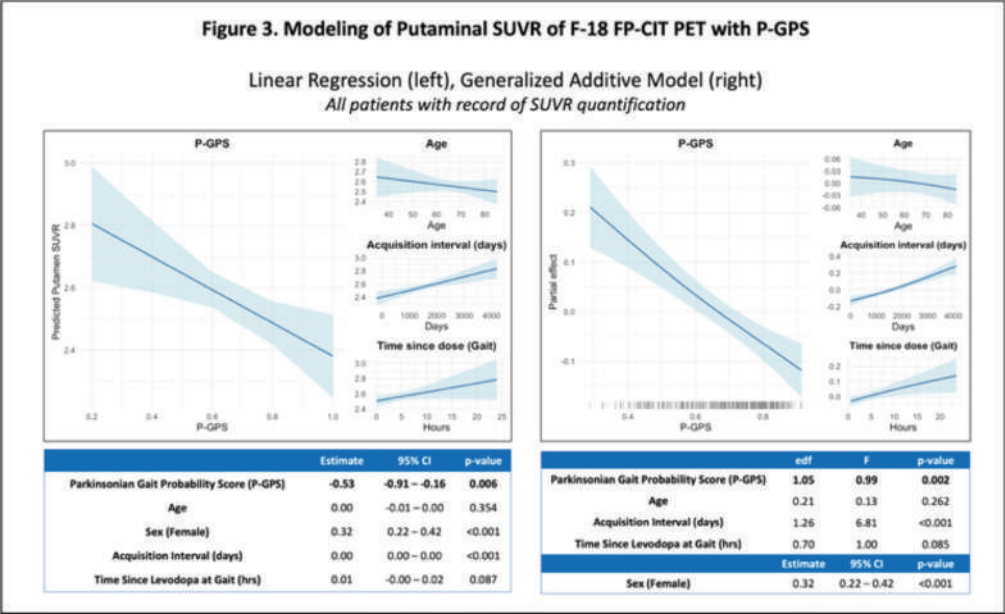
Figure



Figure



Figure



Keywords
Digital Biomarker, Parkinson's Disease, Gait Analysis, Machine Learning

Personal Information

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Current Position PhD Student
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Education / Training

Institution and Location	Degree	Year(s)	Field of Study
Ajou University	M.D.	2014.03 – 2020.02	Medicine
Ajou University Medical Center	Intern	2020.03-2021.02	
Ajou University Medical Center	Resident	2021.03-2025.02	Neurology
Ajou University		2025.03 -	Biomedical Informatics

Positions and Honors

Professional Experience

Current

2025 - PhD Student, Ajou University

Previous

2021.03 -2025.02 Neurology Resident, Ajou University Medical Center

Selected Peer Reviewed Publications

1. YS Kim, IJ Shin, DG Park, JH Yoon. Hashimoto's encephalopathy with diffuse leukoencephalopathy: serial MRI and gait analysis. Journal of Neurology. 2025
2. YS Kim, D Yoon, JH Byun, H Park, A Lee, IH Kim, S Lee, HS Lim, RW Park. Extracting information from free-text electronic patient records to identify practice-based evidence of the performance of coronary stents. Plos one. 2017
3. YS Kim, DG Park, MS Kim, JH Yoon. Deep brain stimulation in Parkinson disease with valosin-containing protein gene mutation. European Journal of Neurology. 2023
4. YS Kim, TJ Kim, JH Yoon, DG Park. Misleading EEG in CACNA1A mutation: A case of late-onset episodic ataxia type 2. Parkinsonism & Related Disorders. 2025

5. **YS Kim**, DG Park, IJ Shin, YS An, JH Yoon. Striatal Hyperperfusion Observed in Dual-Phase 18F-FP-CIT PET Imaging of Hyperglycemic Chorea. Clinical Nuclear Medicine. 2023

Essential Tremor: Novel neuroimaging insights from a Singaporean Imaging-Genetics Cohort

Thomas WJ TEO¹, Celeste CHEN¹, Ling Ling CHAN^{2,3}, Louis CS TAN^{1,2}, Eng King TAN^{1,2,4}, Thomas WELTON^{1,2}

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³Neuroradiology, Singapore General Hospital, Singapore, Singapore

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Background & Objective:

Essential tremor (ET) is a common movement disorder in the elderly. Despite ET incidence and progression being related to chronological age, no studies have investigated MRI-derived brain ageing in ET. We sought to compare neuroimaging, cognitive, and clinical data among ET patients and healthy controls.

Methods:

Brain MRI, clinical and cognitive data were gathered from ET patients and healthy controls. Brain age gap (difference between predicted brain age and chronological age) was estimated from MRI images using a deep-learning model trained on n=17296 individuals. Statistical analyses were controlled for age, gender, education, and total intracranial volume. Neuroimaging analyses such as Voxel-Based Morphometry (VBM) and Tract-Based Spatial Statistics (TBSS) were used to interrogate T1 and diffusion-tensor images.

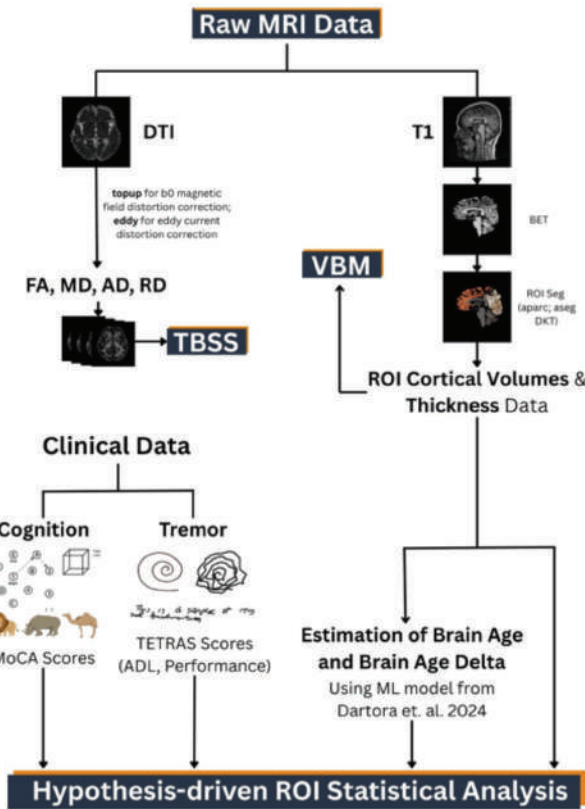
Results:

We gathered data for 31 ET (aged 67±6 years) and 31 controls (70±8 years). The ET group had a significant TETRAS-ADL deficit (10 pts, p<.0001) and motor impairment (14 pts, p<.0001) compared to controls. Brain age and brain age gap did not differ between ET and controls, and were not correlated with ET onset or duration. However, brain age gap was significantly correlated with the TETRAS ADL Total and several of the ADL subscores (voice, keys, pouring, speaking), as well as the TETRAS spiral-drawing test. The brain age gap effects were mostly related to volume and thickness reduction in the postcentral (thickness -.36, p=.002; right vol -.43, p=.017) and paracentral gyri (left thick .35, p=.033). Neither TBSS nor VBM analyses revealed significant differences in brain morphology or microstructure between ET and controls.

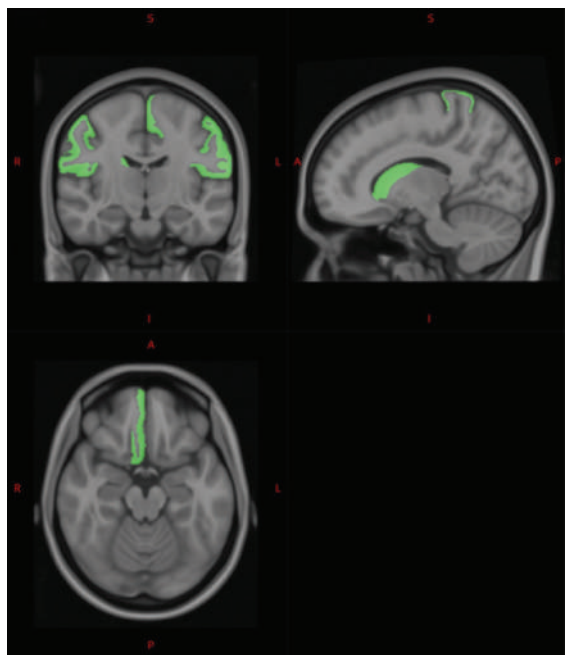
Conclusions:

Our data suggests that while brain ageing may not be significantly accelerated in ET, it may underlie susceptibility to functional deficits that affect daily life. These effects were driven by morphological changes in the sensorimotor-related brain network. We are gathering additional data on biological ageing in ET to investigate other systemic ageing-related changes, and to inform the ageing-related tremor hypothesis.

Figure



Figure



Figure

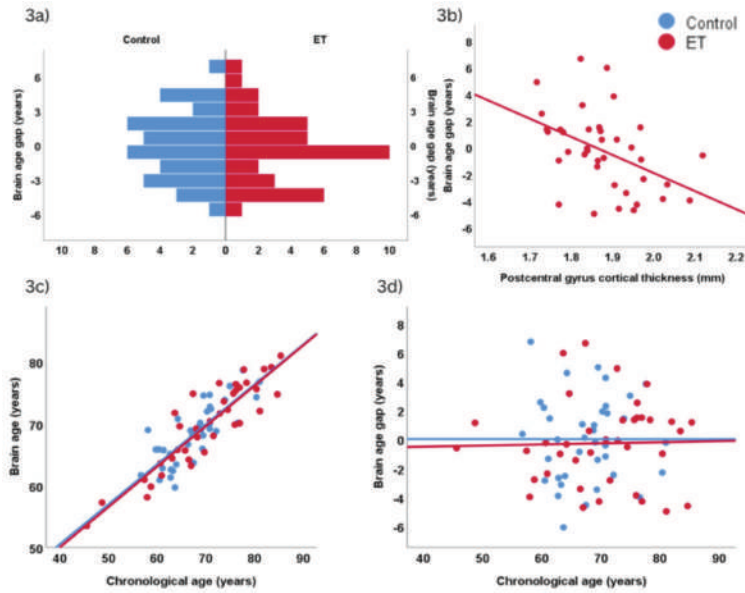


Fig 3a. Distribution of brain age gap (BAG) between ET and control groups.
Fig 3b. Correlation between BAG and bilateral postcentral gyrus thickness.
Fig 3c. Correlation of model estimated brain age and chronological age.
Fig 3d. Correlation of BAG to chronological age.

Keywords

Essential Tremor, Neuroimaging, Magnetic Resonance Imaging, Brain age, Cortical thickness

Personal Information

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Country Singapore



Education / Training

Institution and Location	Degree	Year(s)	Field of Study
Sir Peter Mansfield Imaging Centre, Division of Clinical Neuroscience, University of Nottingham, UK	PhD	2017	Radiological Science
Sir Peter Mansfield Imaging Centre, Division of Clinical Neuroscience, University of Nottingham, UK	MSc	2013	Translational Neuroimaging
Nottingham Trent University, UK	BSc (Hons)	2012	Computer Science

Positions and Honors

Professional Experience

Current

2025 - Principal Investigator, National Neuroscience Institute, Singapore
2022 - Assistant Professor, Neuroscience Academic Clinical Programme, Duke-NUS Medical School, Singapore

Previous

2020 – 2025 Research Fellow, National Neuroscience Institute, Singapore
2017 – 2020 Postdoctoral Scientist, Translational Imaging Lab, University of Sydney, Australia
2016 – 2017 Research Associate, Radiological Sciences, University of Nottingham, UK

Honors

2021 Movement Disorders Society, AOPMC Junior Award
2021 ISMRM Junior Fellowship

Selected Peer Reviewed Publications

1. **Welton T**, Chew G, Mai AS, Ng JH, Chan LL & Tan EK, Association of gene expression and tremor network structure. *Movement Disorders*. 2024.
2. **Welton T**, Hartono S, Lee W, Teh PY, Hou W, Chen R, Chen C, Lim EW, Kumar P, Tan LCS, Tan EK & Chan LL, Classification of Parkinson's Disease by Deep Learning on Midbrain MRI. *Frontiers in Ageing Neuroscience*. 2024.
3. **Welton T**, Teo TWJ, Chan LL, Tan EK & Tan LCS, Parkinson's Disease Risk Variant rs9638616 is Non-Specifically Associated with Altered Brain Structure and Function. *Journal of Parkinson's Disease*. 2024.
4. **Welton T**, Xing Y, Shih YC, Hartono S, Schwarz S, Auer DP, Tan EK, Tan LCS & Chan LL, Ultra-High-Field MRI in Parkinson's Disease: Ready for Clinical Use? A Narrative Review. *Quantitative imaging in medicine and surgery*. 2023.
5. **Welton T**, Hartono S, Shih YC, Lee W, Chong SL, Ng SYE, Chia NSY, Tan EK, Tan LCS & Chan LL, Microstructural changes in grey matter nuclei in early Parkinson's disease: a longitudinal diffusion kurtosis imaging study. *Journal of Parkinson's Disease*. 2023.
6. **Welton T**, Chan L, Carr J, Cardoso F, Deutchl G, Jankovic J & Tan EK, Essential Tremor. *Nature Reviews Disease Primers*. 2021.
7. **Welton T** & Tan EK, Applying artificial intelligence to multi-omic data: new functional variants in Parkinson's disease. *Movement Disorders*. 2021.

Distinct spatiotemporal progression patterns of brain metabolism in de novo Parkinson's disease

Jung Hyun LEE¹, Da Eun JUNG¹, Mina PARK², Sung Jun AHN², Jae Hoon LEE³, Young Hoon RYU³, Chul Hyung LYOO¹, Han Soo YOO¹

¹Neurology, Gangnam Severance Hospital, Seoul, Republic of Korea

²Radiology, Gangnam Severance Hospital, Seoul, Republic of Korea

³Nuclear medicine, Gangnam Severance Hospital, Seoul, Republic of Korea

Background & Objective:

Understanding the heterogeneity in Parkinson's disease (PD) is crucial for patient care, prognosis prediction, and treatment development. We used 18F-FDG PET and Subtype and Stage Interference (SuStaln) to define metabolic subtypes of drug-naïve patients with PD.

Methods:

We enrolled 147 de novo PD patients and 38 healthy individuals who underwent 18F-FDG PET, 18F-FP-CIT PET, and brain MRI. The SuStaln algorithm was employed to identify FDG-based subtypes. Motor and nonmotor features were compared between subtypes. Correlation analysis was performed to assess associations between progression stage and motor or cognitive scores within each subtype.

Results:

Two distinct subtypes were identified: subtype 1 (fronto-striatal) and subtype 2 (posterior cortical). In subtype 1, caudate DAT uptake reduction was accompanied with an early decrease in frontal and caudate metabolism, followed by hypometabolism in the posterior cortical and limbic regions. Subtype 2 was characterized by putaminal DAT loss, along with an early metabolic reduction in the lateral occipital cortex, progressing to the parieto-temporal, frontal, and limbic regions. Clinically, the two subtypes exhibited comparable motor deficits. Regarding non-motor symptoms, subtype 2 showed lower visuospatial and memory scores, lower CCSIT total scores, and higher anxiety score compared with subtype 1. Moreover, subtype 2 showed a more pronounced reduction in putaminal DAT uptake and less asymmetry. The correlation between stage and Montreal Cognitive Assessment score was not significant in subtype 1 but was significantly negative in subtype 2, with a significant stage-by-group interaction.

Conclusions:

De novo PD can be classified into two distinct metabolic subtypes: the fronto-striatal subtype and the posterior cortical subtype, with the latter exhibiting more severe non-motor manifestations, including cognitive impairment, olfactory dysfunction, and neuropsychiatric burden.

Keywords

Parkinson's disease, Metabolism, SuStaln

Personal Information

Full Name Jung Hyun LEE
Current Position Resident
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Organization Gangnam Severance, Yonsei University College of Medicine
Country South Korea



Education / Training

Institution and Location	Degree	Year(s)	Field of Study
Dankook University, Korea	M.D.	Mar.2014 – Feb.2020	Medicine
Yonsei University, Korea	Integrated PhD program	Mar.2023 – Current	Medicine

Positions and Honors

Professional Experience

Current

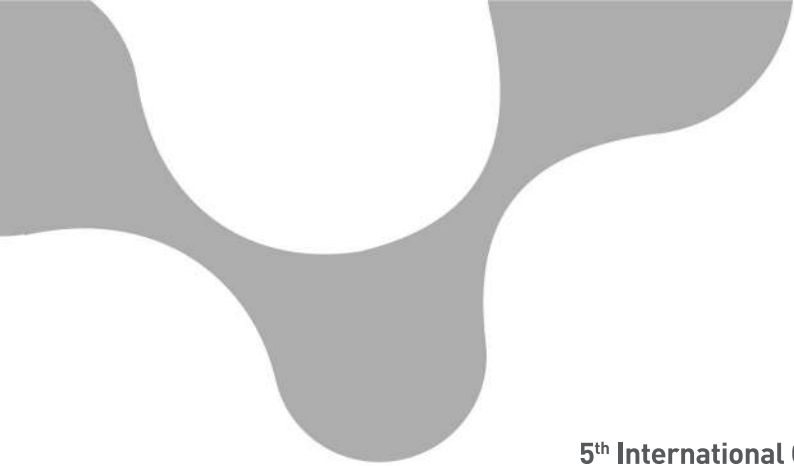
2021 - Resident, Severance Hospital, Yonsei University College of Medicine

Previous

2020 – 2021 Internship, Severance Hospital, Yonsei University College of Medicine

Selected Peer Reviewed Publications

1. [Lee JH](#), Kim HK, Ahn SJ, Park M, Yoo HS, Lyoo CH. Subregion-specific associations of the basal forebrain with sleep and cognition in Parkinson's disease. NPJ Parkinsons Dis. 2025;11(1):33. Published 2025 Feb 20. doi:10.1038/s41531-025-00880-w
2. [Lee JH](#), Park Mina, Ahn SJ, Lee JH, Ryu YH, Lyoo CH, Yoo HS. Association of dopamine depletion and cholinergic basal forebrain atrophy with brain metabolism and cognition in Parkinson's disease. J Parkinsons Dis. 2025 (Accepted)
3. [Kook D](#), Joe S, [Lee JH](#), Yoo HS. Chikungunya Encephalitis Presenting as Rhombencephalitis. J Clin Neurol. 2024;20(1):106-108. doi:10.3988/jcn.2023.0151



ICKMDS 2025

5th International Conference of Korean Movement Disorder Society

Bridging from the Origin to Clinical Practice:
A Beginning Has an Ending

DAY 2_ November 1 (Sat), 2025

Room A

Plenary Session 3

When and Where Does Tauopathy Begin?

Chairs Shen-Yang LIM (Malaysia)
Jae Woo KIM (Korea)



Progressive Supranuclear Palsy: Symptoms and signs of prodromal phase to full-blown phase

Jin Whan CHO
Sungkyunkwan University, Korea

Progressive supranuclear palsy (PSP) is a devastating and rapidly progressive neurodegenerative disorder characterized by ocular motor dysfunction, postural instability, akinesia, and cognitive impairment. Although it can be easily distinguished from other types of parkinsonism in the fully developed stage, making a correct diagnosis in the early stage is extremely difficult. Diagnosis is often delayed by 3–4 years after symptom onset, until the cardinal features of recurrent falls and supranuclear gaze palsy become unequivocally apparent. Thus, early and reliable diagnosis of PSP remains a major clinical challenge. Early diagnosis is critical for understanding prognosis and for enrolling patients in clinical trials of potential neuroprotective therapies.

The pre-diagnostic period includes both a pre-symptomatic and a prodromal phase. In contrast to Parkinson's disease, relatively little is known about this period in PSP due to the paucity of genetic predisposition and the lack of well-defined prodromal symptoms. Prodromal PSP is characterized by subtle yet distinctive clinical features that precede the full expression of the disorder. Early manifestations often include unexplained dizziness, visual symptoms, apathy, anxiety, and depression. Cognitive and behavioral disturbances—particularly executive dysfunction, apathy, and impaired social cognition—reflect early fronto-striatal involvement and may be under-recognized. As the disease progresses, postural instability with recurrent backward falls, axial rigidity, and bradykinesia that is typically unresponsive to dopaminergic therapy emerge. Vertical saccadic slowing, especially in downgaze, represents a hallmark ocular motor abnormality and may precede overt supranuclear gaze palsy.

Understanding the sequence of changes that occur during disease progression is crucial for characterizing the prodromal phase of PSP. Identifying early symptoms and signs, along with developing reliable early biomarkers, is essential for timely diagnosis, differentiation from other parkinsonian syndromes, and inclusion in disease-modifying clinical trials.

Personal Information

Full Name Jin Whan CHO
Current Position Professor
Department Neurology
Organization Sungkwunkwan University/Samsung Seoul Hospital
Country Korea



Education / Training

Institution and Location	Degree	Year(s)	Field of Study
Seoul National University, College of Medicine, Seoul, KOREA	M.D.	1992	Medicine
College of Medicine, Kangwon University, Korea	PhD	2006	Neurology
Seoul National University Hospital, Seoul, KOREA	Intern/Resident	1997	Neurology
Seoul National University Hospital, Seoul, KOREA	Fellow	2001	Movement Disorders
Columbia University Hospital, NY, USA	Research Fellow	2004	Movement Disorders

Positions and Honors

Professional Experience

Current

2010~current Professor, Samsung Seoul Hospital/Sungkwunkwan University

2023~current Vice-President, KMDS

Previous

2004~2010 Assistant/Associate Professor, Seoul National University/Boramae Hospital

Honors

2020 Best Academic Achievement of KMDS

Selected Peer Reviewed Publications

1. Ahn JH, Lee S, Han Y, Chang H, Youn J, Jang JH, **Cho JW**. Another common genetic ataxia in South Korea: Spinocerebellar ataxia 36. *Eur J Hum Genet*. 2025. Doi:10.1038/s41431-024-01783-9.
2. **Cho JW**, Lee JY, Kim HJ, Kim JS, Park KW, Choi SM, Lyoo CH, Koh SB. Efficacy and Safety of Taltirelin Hydrate in Patients With Ataxia Due to Spinocerebellar Degeneration. *JMD* 2025;18(1):35-44
3. Ahn JH, Yoon J, Cho J, Lee S, Kim S, Kim MJ, Lee S, Chu K, Lee SK, Kim HJ, Youn J, Jang JH, Chae J, Moon J, **Cho JW**. Implementing genomic medicine in clinical practice for adults with undiagnosed rare diseases. *NPJ Genom Med*. 2024 Nov 28;9(1):63. doi: 10.1038/s41525-024-00449-1.
4. Ha J, Park JH, Lee SK, Kim HY, Song JO, Yoo J, Ahn JH, Youn J, **Cho JW**. Effectiveness of live-streaming tele-exercise intervention in patients with Parkinson's disease: A pilot study. *J Mov Disord*. 2024 17(2):189-197
5. Lee JS, Ahn JH, Ha JM, Youn J, **Cho JW**. Subjective cognitive complaints in patients with progressive supranuclear palsy. *Front Neurol*. 2023 Dec 14;14:1326571.
6. Ahn JH, Kwon J, Won JH, Byeon K, Youn J, Park H, **Cho JW**. Waiting impulsivity in progressive supranuclear palsy-Richardson's syndrome. *Front Neurosci*. 2023 Sep 25;17:1240709
7. Ahn JH, Kim M, Kim JS, Youn J, Jang W, Oh E, Lee PH, Koh SB, Ahn TB, **Cho JW**. Midbrain atrophy in patients with presymptomatic progressive supranuclear palsy-Richardson's syndrome. *Parkinsonism Relat Disord*. 2019 Sep;66:80-86

Cellular Origin of Tauopathy

Yun Kyung KIM

Brain science institute, KIST, Korea

Tauopathies encompass a spectrum of neurodegenerative disorders unified by the abnormal aggregation of the microtubule-associated protein tau, yet their anatomical patterns and cellular substrates vary strikingly across diseases. Recent advances in single-cell transcriptomics, spatial proteomics, and cryo-EM have begun to redefine the concept of tau pathology not as a uniform biochemical event, but as a process rooted in cellular identity and network context. Emerging evidence indicates that neurons are the principal source of pathogenic tau, with MAPT expression peaking in long-range excitatory projection neurons of the entorhinal and frontotemporal cortices. These neurons, characterized by extensive axonal arbors, high metabolic demand, and intricate cytoskeletal architecture, constitute intrinsic sites of vulnerability where physiological tau becomes mislocalized, modified, and aggregation-prone. In parallel, glial cells—particularly astrocytes and oligodendrocytes—serve as secondary amplifiers, acquiring and reshaping neuronal tau into disease-specific conformers such as astrocytic plaques, tufted astrocytes, or coiled bodies. The interplay between neuronal stress, glial reprocessing, and regional circuitry underlies the diverse topography of tauopathies: from the limbic confinement of PART, to the neocortical transmission of AD, the layer V cortical origin of CBD, the subcortical motor network involvement of PSP, and the perivascular pattern of CTE.

Having developed experimental platforms and therapeutic candidates targeting tau pathology, I have come to recognize that a true leap in tau-directed therapy will arise from understanding its cellular origin—the precise molecular and anatomical contexts in which tau first diverges from its physiological role. Elucidating these origins will enable a new generation of precision interventions designed not merely to remove tau aggregates, but to prevent their birth.

Personal Information

Full Name Yun Kyung KIM
Current Position Principal Scientist, KIST
Department Brain Science Institute
Organization KIST, Korea Institute of Science and Technology
Country South Korea



Education / Training

Institution and Location	Degree	Year(s)	Field of Study
New York University, New York.	Ph.D	2009	Chemistry
KAIST, Daejeon	M.S	2005	Biological Science
KAIST, Daejeon	B.S.	2002	Biological Science

Positions and Honors

Professional Experience

Current

2018 ~present Principal Scientist, KIST
2021~ present Professor, KIST School UST

Previous

2010 -2017 Senior Research Scientist, KIST
2012 -2020 Associate Professor, KIST School UST

Honors

2025 KIST Best Research Team 2025
2024 Prime Minister's Prizes for Science Excellence, Ministry of Science and ICT
2024 KIST Fellow
2022 Best Lecture Award, KIST school UST
2021 KIST-Grand Prize 2021
2020 KIST Best Research Team 2020

2018 KIST Young-Fellow

2018 Prime Minister's Prizes for Science Excellence, Ministry of Science and ICT

Selected Peer Reviewed Publications

1. Yoonsik Sung, Lizaveta Gotina, Kyu Hyeon Kim, Jung Yeol Lee, Seulgi Shin, Hira Aziz, Dong Min Kang, Xiao Liu, Na-Kyeong Hong, Hong-Guen Lee, Jun-Seok Lee, Hyeyeong Ku, Cherlhyun Jeong, Ae Nim Pae, Sungsu Lim, Young-Tae Chang, and **Yun Kyung Kim***, NeuM: A Neuron-Selective Probe Incorporates into Live Neuronal Membranes via Enhanced Clathrin-Mediated Endocytosis in Primary Neurons, *Angewandte. Chemie. Int. Ed.* 2024, [IF:16.60]
2. Sungsu Lim, Seulgi Shin, Yoonsik Sung, Ha Eun Lee, Kyu Hyeon Kim, Ji Yeon Song, Gwan-Ho Lee, Hira Aziz, Nataliia Lukianenko, Dong Min Kang, Nicolette Boesen, Hyeonjeong Jeong, Aizhan Abdildinova, Junghee Lee, Byung-Yong Yu, Sang Min Lim, Jun-Seok Lee, Hoon Ryu, Ae Nim Pae, **Yun Kyung Kim***, Levosimendan inhibits disulfide tau oligomerization ameliorating tau pathology in TauP301L-BiFC mice. *Experimental & Molecular Medicine*, 2023 [IF:12.53]
3. Seulgi Shin, Dohee Kim, Ae Nim Pae, Sungsu Lim, YunKyung Kim*, Visualization of soluble tau oligomers in TauP301L-BiFC transgenic mice demonstrates the progression of tauopathy **Progress in Neurobiology**, 2020 [IF: 14.16]
4. Jusung An, Peter Verwilt, Hira Aziz, Jinwoo Shin, Sungsu Lim, Ilwha Kim, **YunKyung Kim***, Jong Seung Kim* Picomolar-sensitive β -amyloid fibril fluorophores by tailoring the hydrophobicity of biannulated π -elongated dioxaborine-dyes, *Bioactive Material*, 2022 [IF: 16.44]
5. Chang Hoon Ji, Hee Yeon Kim, Min Ju Lee, Ah Jung Heo, Daniel Park, Sungsu Lim, Seulgi Shin, Woo Seung Yang, Chang An Jung, Kun Young Kim, Eun Hye Jeong, Sun Ho Park, Su Bin Kim, Su Jin Lee, Jeong Eun Na, Ji In Kang, Hyung Min Chi, Hyun Tae Kim, **Yun Kyung Kim***, Bo Yeon Kim*, and Yong Tae Kwon* The AUTOTAC chemical biology platform for targeted protein degradation via the autophagy-lysosome system **Nature Communication**. 2022, [IF: 17.69].
6. Kyuhyeon Kim, Geon Lee, Jinwoo Lee, Taeyeon Kim, Beomju Shin, Dong Min Kang, **Yun Kyung Kim***, Minah Seo* Meta-surface-enhanced terahertz imaging for glioblastoma in orthotopic xenograft mouse model combined with neural network decision making **Biosensors and Bioelectronics**, 2025 [IF: 12.51]

Genetics and Biomarkers for Early Detection of Tauopathies

Huw MORRIS

Department of Clinical and Movement Neuroscience, UCL Queen Square Institute of Neurology, UK

Developments in genetics have provided insights into the biology and early diagnosis of PSP and other tauopathies. Efforts to understand these disorders are progressing on a global basis. New developments in high throughput biomarker assays and in the development of protein / aggregation assays are also showing promise in early diagnosis but also showing the extent of overlap and co-pathology across disease. Integrating genetics, biomarkers, clinical data and ultimately pathology will provide new insights into disease heterogeneity and improve diagnosis and enable future clinical trials. In this talk I will cover the heterogeneity of Parkinson's syndrome and study design to explore disease heterogeneity, I will cover new development in genome wide association studies, high throughput proteomic studies and seed amplification assays.

Personal Information

Full Name Huw MORRIS
Current Position Professor of Neurology and Neurogenetics
Department Clinical and Movement Neuroscience
Organization UCL Queen Square Institute of Neurology
Country UK



Education / Training

Institution and Location	Degree	Year(s)	Field of Study
Guy's Hospital, London	MBBS	06/1992	Medicine
Royal College of Physicians, London	MRCP	06/1995	MRCP
Guy's Hospital, London	BSc	06/1989	Neuroscience (First)
UCL, London	PHD	06/2002	Neuroscience
Royal College of Physicians, London	FRCP	06/2009	FRCP

Positions and Honors

Professional Experience

Current

2013-Present Professor of Neurology and Neurogenetics, UCL, London, UK
2013-Present Honorary Consultant in Neurology, Royal Free Hospital, London
2013-Present Honorary Consultant in Neurology, National Hospital, London
2009-Present Medical Advisory Board National Tremor Foundation
2013-Present Scientific Advisory Board, CurePSP
2020-Present Toronto Rossy PSP Scientific Advisory Committee
2021-Present Editorial Board Practical Neurology
2019-Present College of Experts, Parkinson's UK
2021-Present Editorial Board, Neurology and Therapy
2023-Present Operations Committee Member for Global Parkinson's Genetics Program (www.gp2.org)
2020-Present Cohort Lead for Global Parkinson's Genetics Program (www.gp2.org)

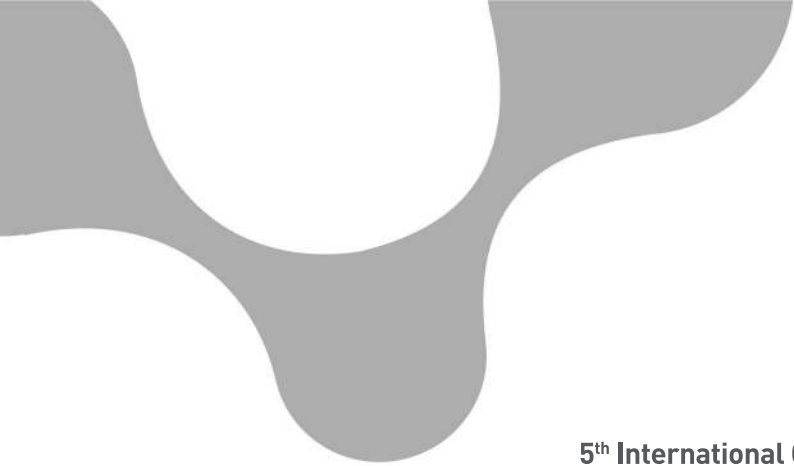
2011-2024	Advisor, Queen Square Brain Bank Management Board
2017-2020	PASSPORT Study (BII092) Steering Committee
2012-2013	Professor of Neurology and Neurogenetics, Cardiff University, Cardiff, UK
2012-2018	Editorial Board, Journal of Neurology
2006-2013	Executive Group Member, Wales Dementia and Neurodegenerative Diseases Clinical Research Network (Neurodem Cymru)
2006- 2012	Parkinson's Disease Clinical Studies Group Member, Dementia and Neurodegenerative Diseases Network UK (DENDRON UK)
2003-2012	Senior Lecturer in Neurology and Neurogenetics, Cardiff University, Cardiff, UK
2001-2003	Specialist Registrar Neurology, South London Rotation
1999-2001	MRC Clinical Training Fellow, Institute of Neurology, Queen Square, London, UK
1999-2007	Associate Editor, Postgraduate Medical Journal
1997-1999	PSP Association Clinical Training Fellow, Institute of Neurology, Queen Square, London, UK

Honors

1992	Charles Symonds Prize in Neurology
1992	Distinctions in Medicine and Obstetrics/Gynecology, University of London

Selected Peer Reviewed Publications

- Jabbari E, Holland N, Chelban V, Jones PS, Lamb R, Rawlinson C, et al. Diagnosis Across the Spectrum of Progressive Supranuclear Palsy and Corticobasal Syndrome. *Jama Neurol.* 2020;77(3):377–87.
 - Jabbari E, Koga S, Valentino RR, Reynolds RH, Ferrari R, Tan MMX, et al. Genetic determinants of survival in progressive supranuclear palsy: a genome-wide association study. *Lancet Neurology.* 2021;20(2):107–16.
 - Jabbari E, Woodside J, Tan MMX, Shuai M, Pittman A, Ferrari R, et al. Variation at the TRIM11 locus modifies progressive supranuclear palsy phenotype. *Ann Neurol.* 2018;84(4):485–96.
 - Herbst S, Lewis PA, Morris HR. The emerging role of LRRK2 in tauopathies. *Clin Sci.* 2022;136(13):1071–9.
 - Langworth-Green C, Patel S, Jaunmuktane Z, Jabbari E, Morris H, Thom M, et al. Chronic effects of inflammation on tauopathies. *Lancet Neurology.* 2023;22(5):430–42.
 - Morris HR, Khan MN, Janssen JC, Brown JM, Perez-Tur J, Baker M, et al. The Genetic and Pathological Classification of Familial Frontotemporal Dementia. *Arch Neurol-chicago.* 2001;58(11):1813–6.
 - Majounie E, Renton AE, Mok K, Doppler EGP, Waite A, Rollinson S, et al. Frequency of the C9orf72 hexanucleotide repeat expansion in patients with amyotrophic lateral sclerosis and frontotemporal dementia: a cross-sectional study. *Lancet Neurology.* 2012;11(4):323–30.
- Renton AE, Majounie E, Waite A, Simón-Sánchez J, Rollinson S, Gibbs JR, et al. A hexanucleotide repeat expansion in C9ORF72 is the cause of chromosome 9p21-linked ALS-FTD. *Neuron.* 2011;72(2):257–68



ICKMDS 2025

5th International Conference of Korean Movement Disorder Society

Bridging from the Origin to Clinical Practice:
A Beginning Has an Ending

DAY 2_ November 1 (Sat), 2025

Room A

Plenary Session 4

New Innovative Treatments in Parkinson's Disease

Chairs Hubert FERNANDEZ (USA)
Sun Ju CHUNG (Korea)



Promising Therapeutic Approaches Targeting Alpha-Synuclein

Tae-In KAM

Department of Brain and Cognitive Sciences, KAIST, Republic of Korea

Parkinson's disease (PD) is characterized by disorders of movement, which are caused by the progressive loss of dopamine neurons in the substantia nigra pars compacta (SNpc). To date there are no disease modifying treatments that prevent the relentless neurodegeneration. The pathologic accumulation and aggregation of α -synuclein (α -syn) underlies PD through loss of substantia nigra pars compacta dopamine neurons. However, what drives the abnormal assembly of pathologic α -syn and death mechanisms that are activated by pathologic α -syn are not known. In this lecture, comprehensive overview of pivotal advances in α -synuclein research is provided. Structural features and physiological functions of α -synuclein is summarized. I also highlight the existence of disease-specific α -synuclein strains and their mechanisms of pathological spread, and discuss seed amplification assays and PET tracers as emerging diagnostic tools for detecting pathological α -synuclein in clinical settings. α -Synuclein aggregation, clearance mechanisms, and cell-autonomous and non-cell-autonomous processes that contribute to neuronal death is reviewed. Finally, promising therapeutic approaches that target pathological α -synuclein and insights into emerging areas of research is provided.

Personal Information

Full Name Tae-In KAM
Current Position Assistant Professor
Department Department of Brain and Cognitive Sciences
Organization KAIST
Country Korea



Education / Training

Institution and Location	Degree	Year(s)	Field of Study
Johns Hopkins University / USA	Postdoctoral Fellow	2014-2019	Neurodegenerative Disease
Seoul National University / Korea	Ph.D.	2006-2013	Molecular Neuroscience
Seoul National University / Korea	B.S.	2002-2006	Biology

Positions and Honors

Professional Experience

Current

2023-current Assistant Professor, KAIST
2024-current Associate Professor, Johns Hopkins University

Previous

2019-2024 Assistant Professor, Johns Hopkins University

Honors

2023 Excellence in Junior Faculty Award,
Association of Korean Neuroscientists (AKN), USA
2022 Stanley Fahn Junior Faculty Award, Parkinson's Foundation, USA
2020 Launch Award, Maryland Stem Cell Research Fund (MSCRF), USA
2019 Excellence in Scientific Research Award, Johns Hopkins University, USA

Selected Peer Reviewed Publications

1. Park H*, Kam TI*, Dawson VL, Dawson TM. α -Synuclein pathology as a target in neurodegenerative diseases. *Nature Review Neurology*. 21(1):32-47. 2025.
2. Kang H, Park S, Jo A, Mao X, Kumar M, Park CH, Ahn JY, Lee Y, Choi JY, Lee YS, Dawson VL, Dawson TM, Kam TI#, Shin JH#. Parkin-interacting substrate, ZNF746 undergoes liquid-liquid phase separation and poly(ADP-ribose)-mediated solidification. *EMBO Reports*. 24(11):e56166. 2023. (#Co-corresponding)
3. Chun YS, Kim MY, Lee SY, Kim MJ, Hong TJ, Jeon JK, Ganbat D, Kim HT, Kim SS, Kam TI#, Han S#. MEK1/2 inhibition rescues neurodegeneration by TFEB-mediated activation of autophagic lysosomal function in a model of Alzheimer's disease. *Molecular Psychiatry*. 27(11):4770-4780. 2022. (#Co-corresponding)
4. Park HJ*, Kam TI*, Peng H, Chou SC, Mehrabani-Tabar AA, Song JJ, Yin X, Karuppagounder SS, Umanah GK, Rao S, Choi Y, Aggarwal A, Chang S, Kim H, Byun J, Liu JO, Dawson TM, Dawson VL. PAAN/MIF Nuclease Inhibition Prevents Neurodegeneration in Parkinson's Disease. *Cell*. 185(11):1943-1959.e21. 2022 (*Co-first)
5. Kam TI*, Mao X*, Park H*, Chou SC, Karuppagounder SS, Umanah GE, Yun SP, Brahmachari S, Panicker N, Chen R, Andrabi SA, Qi C, Poirier GG, Pletnikova O, Troncoso JC, Bekris LM, Leverenz JB, Pantelyat A, Ko HS, Rosenthal LS, Dawson TM, Dawson VL. Poly (ADP-ribose) drives pathologic α -synuclein neurodegeneration in Parkinson's disease. *Science* 362(6414), doi: 10.1126/science. aat8407. 2018.
6. Yun SP*, Kam TI*, Panicker N, Kim S, Oh Y, Park JS, Kwon SH, Park YJ, Karuppagounder SS, Park H, Kim S, Oh N, Kim NA, Lee S, Brahmachari S, Mao X, Lee JH, Kumar M, An D, Kang SU, Lee Y, Lee KC, Na DH, Kim D, Lee SH, Roschke V, Liddel SA, Mari Z, Barres BA, Dawson VL, Lee S, Dawson TM, Ko HS. Block of A1 astrocyte conversion by microglia is neuroprotective in models of Parkinson's disease. *Nature Medicine* 24(7), 931-938. 2018.

Stem Cell Therapy for Parkinson's Disease: Phase 1/2a Clinical Trial with hESC-derived Dopamine Progenitors

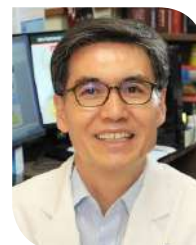
Dong-Wook KIM

Department of Physiology, Yonsei University College of Medicine, Seoul, Korea

Parkinson's disease (PD) is caused by the progressive loss of dopaminergic neurons in the substantia nigra pars compacta and has long been considered an appropriate candidate for cell replacement therapy. We generated high-purity dopaminergic progenitors (A9-DPC, also called TED-A9) from human embryonic stem cells (hESCs) (**Cell Stem Cell**, 2024). The therapeutic potential and safety of A9-DPC were evaluated in rodent and primate models. Transplantation into PD rats significantly improved dopaminergic function, while PD monkeys demonstrated behavioral improvements for up to 28 weeks with increased dopamine activity on PET following transplantation. Comprehensive safety assessments conducted in GLP-compliant facilities showed no toxicity or tumorigenicity. A9-DPC received IND approval from the Korean MFDS. The phase 1/2a clinical trial (NCT05887466) was conducted on 12 participants who had been diagnosed with PD for more than 5 years and exhibited motor complications such as wearing-off, freezing of gait, or dyskinesia. Participants were aged 50 to 75 years. A9-DPC was administered to 6 participants in the low-dose group (3.15 million cells) and to another 6 participants in the high-dose group (6.30 million cells) using a 3+3 rule-based design with immunosuppression. One-year follow-up data revealed no significant adverse events, confirming the safety of A9-DPC. Off-medication MDS-UPDRS Part III scores and Hoehn and Yahr stage improved, with greater motor improvements in the high-dose group. Dopamine transporter PET imaging showed increased posterior putamen uptake with greater uptake in the high-dose group after transplantation, supporting graft survival. These findings indicate that bilateral transplantation of A9-DPC is safe and may improve parkinsonian motor symptoms in patients with PD (**Cell**, in press).

Personal Information

Full Name Dong-Wook KIM
Current Position Professor
Department Physiology
Organization Yonsei University College of Medicine
Country Korea



Education / Training

Institution and Location	Degree	Year(s)	Field of Study
Korea University, Korea	B.S.	1986	Life Science/ Genetic Engineering
The University of Tokyo, Japan	Ph.D.	1996	Biotechnology
Yale Univ. School of Medicine, USA	Research Scientist	1997-2001	Cell Biology
Harvard Medical School, USA	Junior Faculty	2001-2004	Stem Cell Biology
USC Keck School of Medicine, USA	Visiting Faculty	2006-2007	Stem Cell Biology

Positions and Honors

Professional Experience

Current

2003~ Faculty, Dept of Physiology, Yonsei Univ. College of Medicine, Korea
2015~ CTO, S.BIOMEDICS

Previous

2020 Chairman of the Board of Directors, Korean Society for Stem Cell Research (KSSCR)
2019 President, Korean Society for Stem Cell Research (KSSCR)
2018-2019 Program Committee (Korea Representative), International Society for Stem Cell Research (ISSCR)
2015- Editorial Board, JBC (Journal of Biological Chemistry)
2012-2017 Director, Stem Cell Research Center for Drug Development, Korea
2011-2015 Special Advisory Board, Korean FDA (Cell-based Therapeutics)
2011- Editorial Board, Inflammation and Regeneration

2009-	Editorial Board, Stem Cell Reviews and Reports
2008-2012	Editorial Board, Stem Cell Research
2009	Vice President, TERMIS World Congress
2008-2009	Government Affairs Committee Member, ISSCR (Korea Representative)
2007-2012	Steering Committee Member, Stem Cell Network for the Asia Pacific Region
2007-2012	Steering Committee Member, International Stem Cell Forum (Korea Representative)
2007-2012	Co-chair, International Stem Cell Proteomics Initiative
2007-	Board of Directors, Korean Society for Stem Cell Research (KSSCR)
2006-2008	Editor-in-Chief, Tissue Engineering and Regenerative Medicine
2006-2010	International Committee, ISSCR (International Society for Stem Cell Research) (Korea Representative)
2006	Organizing Committee, TERMIS World Congress
2006-2012	Director, Korean Stem Cell Research Center, Korea
2006	Director, Task Force of 10-Year National Plan for Stem Cell Research in Korea

Honors

2022	Selected for Excellence in Advanced Medical Technology Development (Ministry of Health and Welfare / Korea Health Industry Development Institute)
2020	Recognized for Contributions to the Advancement of the Biofield (Ministry of Science and ICT)
2019	Selected for Top 30 Healthcare R&D Achievements (Ministry of Health and Welfare / Korea Health Industry Development Institute)
2019	Selected for Top 100 National R&D Achievements (Ministry of Science and ICT)
2018	Selected for Top 20 Healthcare R&D Achievements (Ministry of Health and Welfare / Korea Health Industry Development Institute)
2018	Outstanding Researcher Award in Industry-Academia Collaboration (Yonsei University)
2015	Seongjae Medical Award (Yonsei University)
2014	Innovator Leader of Korea 2014
2013	Selected as a Key Player in the 100 Future Technologies Leading Korea's Industry in 2020 (Korean Academy of Engineering)
2013	2013 Power Brain Award of Korea
2013	Outstanding Professor Award for Research Activities (Yonsei University)
2012	Minister of Education, Science, and Technology's Letter of Appreciation
2010	Outstanding Professor Award for Research Activities (Yonsei University)
2008	Yudang Science Award of the Korean Physiological Society

Selected Peer Reviewed Publications

- Jin Woo Chang, Han Kyu Na, Kyung Won Chang, Chan Wook Park, Do-Hun Kim, Sanghyun Park, Chul-Yong Park, Jang Hyeon Eom, Seung Taek Nam, Ki-Sang Jo, Mi-Young Jo, Sung Kyoung Choi, Hye-Jin Hur, Sarang Kim, Minseok Kim, Dae-Sung Kim, Dong-Youn Hwang, Myoung Soo Kim, Inkyung Jung, Jongwan Kim, Myung Soo Cho, **Phil Hyu Lee***, and **Dong-Wook Kim*** (* Co-corresponding authors), Phase 1/2a clinical trial of hESC-derived dopamine progenitors in Parkinson's disease. **Cell**. 2025 (in press).
- Do-Hun Kim, Sang-Hwi Choi, Jin Jea Sung, Sieun Kim, Hanui Yi, Sanghyun Park, Chan Wook Park, Young Woo Oh, Jungil Lee, Dae-Sung Kim, Jong-Hoon Kim, Chul-Yong Park, **Dong-Wook Kim**, Long-term correction of hemophilia A via integration of a functionally enhanced FVIII gene into the AAVS1 locus by nickase in patient-derived iPSCs. **Experimental and Molecular Medicine (EMM)**. 2025.
- Sanghyun Park, Chan Wook Park, Jang Hyeon Eom, Mi-Young Jo, Hye-Jin Hur, Sung Kyoung Choi, Jae Souk Lee, Seung Taek Nam, Ki-Sang Jo, Young Woo Oh, Jungil Lee, Sieun Kim, Do-Hun Kim, Chul-Yong Park, Su Jin Kim, Ho-Young Lee, Myung Soo Cho, Dae-Sung Kim, **Dong-Wook Kim**, Preclinical and dose-ranging assessment of hESC-derived dopaminergic progenitors for a clinical trial on Parkinson's disease. **Cell Stem Cell**. 2024.
- Gyu-Bum Yeon, Byeong-Min Jeon, Seo Hyun Yoo, Dongyun Kim, Seung Soo Oh, Sanghyun Park, Won-Ho Shin, Hyung Wook Kim, Dokyun Na, **Dong-Wook Kim***, Dae-Sung Kim* (* Co-corresponding authors), Differentiation of astrocytes with characteristics of ventral midbrain from human embryonic stem cells. **Stem Cell Reviews and Reports**. 2023.
- Hye-Jin Hur, Ji Yong Lee, Do-Hun Kim, Myung Soo Cho, Sangsik Lee, Han-Soo Kim, **Dong-Wook Kim**, Conditioned Medium of Human Pluripotent Stem Cell-Derived Neural Precursor Cells Exerts Neurorestorative Effects against Ischemic Stroke Model. **International Journal of Molecular Sciences**. 2022.
- Do-Hun Kim, Hyun-Ju Cho, Chul-Yong Park, Myung Soo Cho, **Dong-Wook Kim**, Transplantation of PSA-NCAM-Positive Neural Precursors from Human Embryonic Stem Cells Promotes Functional Recovery in an Animal Model of Spinal Cord Injury. **Tissue Engineering and Regenerative Medicine**. 2022.
- Jeong Sang Son, Chul-Yong Park, Gyunggyu Lee, Ji Young Park, Hyo Jin Kim, Gyeongmin Kim, Kyun Yoo Chi, Dong-Hun Woo, Choongseong Han, Sang Kyum Kim, Han-Jin Park, **Dong-Wook Kim***, Jong-Hoon Kim* (* Co-corresponding authors), Therapeutic correction of hemophilia A using 2D endothelial cells and multicellular 3D organoids derived from CRISPR/Cas9-engineered patient iPSCs. **Biomaterials**. 2022.
- Sanghyun Park, Jeong-Eun Yoo, Gyu-Bum Yeon, Jin Hee Kim, Jae Souk Lee, Sung Kyoung Choi, Young-Gi Hwang, Chan Wook Park, Myung Soo Cho, Jongwan Kim, Dokyun Na, Hyung Wook Kim, Dae-Sung Kim and **Dong-Wook Kim**, Trophoblast glycoprotein is a new candidate gene for Parkinson's disease. **npj Parkinson's Disease**. 2021.
- Jeong-Eun Yoo, Dongjin R. Lee, Sanghyun Park, Hye-Rim Shin, Kun Gu Lee, Dae-Sung Kim, Mi-Young Jo, Jang-Hyeon Eom, Myung Soo Cho, Dong-Youn Hwang, **Dong-Wook Kim**, Trophoblast glycoprotein is a marker for efficient sorting of ventral mesencephalic dopaminergic precursors derived from human pluripotent stem cells. **npj Parkinson's Disease**. 2021.
- Juwon Jung, Sunsook Hwang, Hyewon Seol, A-Hyeon Kim, Ha Myoung Lee, Jin Jea Sung, Seung Min Jeong, Young Min Choi, Jong Kwan Jun, **Dong-Wook Kim***, Jiho Jang* (* Co-corresponding authors), Generation of Brachyury-mCherry knock-in reporter human pluripotent stem cell line (SNUe003-A-2) using CRISPR/CAS9 nuclease. **Stem Cell Research**. 2021.
- Do-Hun Kim, Chul-Yong Park, Sung-Rae Cho, **Dong-Wook Kim**, Generation of a human induced pluripotent stem cell line, YCMi002-A, from a Factor VII deficiency patient carrying F7 mutations. **Stem Cell Research**. 2020.
- Jin Jea Sung, Sanghyun Park, Sang-Hwi Choi, Jongwan Kim, Myung Soo Cho, **Dong-Wook Kim**, Generation of a gene edited hemophilia A patient-derived iPSC cell line, YCMi001-B-1, by targeted insertion of coagulation factor FVIII using CRISPR/Cas9. **Stem Cell Research**. 2020.

- Sanghyun Park, Jeong-Eun Yoo, Dongjin R. Lee, Jiho Jang, Myung Soo Cho, Dae-Sung Kim, **Dong-Wook Kim**, Establishment of PITX3-mCherry knock-in reporter human embryonic stem cell line (WAe009-A-23). **Stem Cell Research**. 2019.
- Chul-Yong Park, Jin Jea Sung, Sung-Rae Cho, Jongwan Kim, **Dong-Wook Kim**, Universal Correction of Blood Coagulation Factor VIII in Patient-Derived Induced Pluripotent Stem Cells Using CRISPR/Cas9. **Stem Cell Reports**. 2019.
- Jin Jea Sung, Chul-Yong Park, Joong Woo Leem, Myung Soo Cho, **Dong-Wook Kim**, Restoration of FVIII expression by targeted gene insertion in the FVIII locus in hemophilia A patient-derived iPSCs. **Experimental and Molecular Medicine (EMM)**. 2019.
- Junwon Lee, Sang-Hwi Choi, Young-Beom Kim, Ikhyun Jun, Jin Jea Sung, Dongjin R. Lee, Yang In Kim, Myung Soo Cho, Suk Ho Byeon, Dae-Sung Kim, **Dong-Wook Kim**, Defined Conditions for Differentiation of Functional Retinal Ganglion Cells From Human Pluripotent Stem Cells. **Investigative ophthalmology & visual science (IOVS)**. 2018.
- Ji Young Kim, Jae Souk Lee, Hyun Sub Hwang, Dongjin R. Lee, Chul-Yong Park, Sung Jun Jung, Young Rang You, Dae-Sung Kim, **Dong-Wook Kim**, Wnt signal activation induces midbrain specification through direct binding of the beta-catenin/TCF4 complex to the EN1 promoter in human pluripotent stem cells. **Experimental and Molecular Medicine (EMM)**. 2018.
- Junwon Lee, Sang-Hwi Choi, Dongjin R. Lee, Dae-Sung Kim, **Dong-Wook Kim**, Generation of Isthmic Organizer-Like Cells from Human Embryonic Stem Cells. **Molecules and Cells**. 2018.
- Dae-Sung Kim, Se Jung Jung, Jae Souk Lee, Bo Young Lim, Hyun Ah Kim, Jeong-Eun Yoo, **Dong-Wook Kim***, Joong Woo Leem* (* Co-corresponding authors), Rapid generation of OPC-like cells from human pluripotent stem cells for treating spinal cord injury. **Experimental and Molecular Medicine (EMM)**. 2017.
- Jiho Jang, Yong Jun Huh, Hyun-Ju Cho, Boram Lee, Jaepil Park, Dong-Youn Hwang and **Dong-Wook Kim**, SIRT1 Enhances the Survival of Human Embryonic Stem Cells by Promoting DNA Repair. **Stem Cell Reports**. 2017.
- Jongman Yoo, Han-Soo Kim, Jin-Ju Seo, Jang-Hyoun Eom, Sung-Mi Choi, Sanghyun Park, **Dong-Wook Kim***, Dong-Youn Hwang* (* Co-corresponding authors), Therapeutic effects of umbilical cord blood plasma in a rat model of acute ischemic stroke. **Oncotarget**. 2016.
- Jiho Jang, Sangjun Park, Hye Jin Hur, Hyun-Ju Cho, Inhwa Hwang, Yun Pyo Kang, Isak Im, Hyunji Lee, Eunju Lee, Wonsuk Yang, Hoon-Chul Kang, Sung Won Kwon, Je-Wook Yu, **Dong-Wook Kim**, 25-hydroxycholesterol contributes to cerebral inflammation of X-linked adrenoleukodystrophy through activation of the NLRP3 inflammasome. **Nature Communications**. 2016.
- Chul-Yong Park, Jin Jea Sung, Sang-Hwi Choi, Dongjin R. Lee, In-Hyun Park, **Dong-Wook Kim**, Modeling and correction of structural variations in patient-derived iPSCs using CRISPR/Cas9. **Nature Protocols**. 2016.
- Chul-Yong Park, Dongjin R. Lee, Jin Jea Sung, **Dong-Wook Kim**, Genome-editing technologies for gene correction of hemophilia. **Human Genetics**. 2016.
- Chul-Yong Park, Jin Jea Sung, **Dong-Wook Kim**, Genome Editing of Structural Variations: Modeling and Gene Correction. **Trends in Biotechnology**. 2016.
- Chul-Yong Park, Tomer Halevy, Dongjin R. Lee, Jin Jea Sung, Jae Souk Lee, Ofra Yanuka, Nissim Benvenisty, **Dong-Wook Kim**, Reversion of FMR1 methylation and silencing by editing the triplet repeats in Fragile-X iPSC-derived neurons. **Cell Reports**. 2015.
- Dongjin R. Lee, Jeong-Eun Yoo, Jae Souk Lee, Sanghyun Park, Junwon Lee, Chul-Yong Park, Eunhyun Ji, Han-Soo Kim, Dong-Youn Hwang, Dae-Sung Kim, and **Dong-Wook Kim**, PSA-NCAM-Negative Neural Crest Cells Emerging during Neural Induction of Pluripotent Stem Cells Cause Mesodermal Tumors and Unwanted Grafts. **Stem Cell Reports**. 2015.

- Chul-Yong Park, Duk Hyoung Kim, Jeong Sang Son, Jin Jea Sung, Jaehun Lee, Sangsu Bae, Jong-Hoon Kim, **Dong-Wook Kim***, Jin-Soo Kim* (* Co-corresponding authors), Functional Correction of Large Factor VIII Gene Chromosomal Inversions in Hemophilia A Patient-Derived iPSCs Using CRISPR-Cas9. **Cell Stem Cell**. 2015.

P4-3

Repurposing DBS - A Disease-Modifying Therapy?

Suneil KALIA

Neurosurgery, Dept of Surgery, University of Toronto

Deep Brain Stimulation (DBS) is a standard symptomatic therapy for Parkinson's disease (PD), but it does not stop or slow the underlying neurodegeneration. The potential to repurpose DBS as a disease-modifying therapy offers significant promise but remains controversial. The core pathology of PD is driven by alpha-synuclein oligomers and fibrils, which cause cytotoxicity and potential spread from cell to cell, leading to disease progression.

A novel approach may involve repurposing DBS as a disease-modifying therapy however clinical data is limited and complicated by a lack of definitive biomarkers for disease progression and by biologically heterogeneous patient populations. Realizing the neuroprotective potential of DBS requires a fundamental re-evaluation of our approach. Further work is needed to understand the mechanisms by which DBS might regulate alpha-synuclein aggregation, possibly via cellular pathways like the proteasome or lysosome. Moreover, it will be necessary to refine stimulation parameters, reconsider surgical targets, personalize patient selection, and explore intervention earlier in the disease course to transform this promising concept into a clinical reality for the treatment of Parkinson's disease and other neurodegenerative movement disorders.

Personal Information

Full Name **Suneil KALIA**
Current Position **Neurosurgeon, Senior Scientist, and Associate Professor**
Department **Neurosurgery**
Organization **University of Toronto**
Country **Canada**



Education / Training

Institution and Location	Degree	Year(s)	Field of Study
McGill University	BSc	1993-1997	Immunology
University of Toronto	MD. PhD.	1997-2006	PhD in Molecular Neuroscience
University of Toronto	FRCS	2006-2012	Neurosurgery
Harvard University	Post-doctoral	2009-2010	Molecular Neuroscience
University of Toronto	Fellowship in Stereotactic and Functional Neurosurgery	2012-2013	Functional Neurosurgery

Positions and Honors

Professional Experience

Current

2021 - present R.R. Tasker Chair in Stereotactic and Functional Neurosurgery
2021 - present Senior Scientist, Krembil Research Institute
2020 - present: Associate Professor, Department of Surgery, University of Toronto

Selected Peer Reviewed Publications

1. Lee EJ, Aguirre-Padilla DH, Fomenko A, Pawar G, Kapadia M, George J, Lozano AM, Hamani C, Kalia LV, Kalia SK. Reduction of alpha-synuclein oligomers in preclinical models of Parkinson's disease by electrical stimulation in vitro and deep brain stimulation in vivo. Brain Stimul. 2024

2. George J, Shafiq K, Kapadia M, Kalia LV, **Kalia SK**. High frequency electrical stimulation reduces α -synuclein levels and α -synuclein-mediated autophagy dysfunction. *Sci Rep*. 2024 Jul 12;14(1):16091.
3. Nim S, O'Hara DM, Corbi-Verge C, Perez-Riba A, Fujisawa K, Kapadia M, Chau H, Albanese F, Pawar G, De Snoo ML, Ngana SG, Kim J, El-Agnaf OMA, Rennella E, Kay LE, **Kalia SK**, Kalia LV, Kim PM. Disrupting the α -synuclein-ESCRT interaction with a peptide inhibitor mitigates neurodegeneration in preclinical models of Parkinson's disease. *Nat Commun*. 2023 Apr 19;14(1):2150.
4. Meng Y, Pople CB, Huang Y, Jones RM, Ottoy J, Goubran M, Oliveira LM, Davidson B, Lawrence LSP, Lau AZ, Bethune A, Maralani P, Abrahao A, Hamani C, Hynynen K, **Kalia SK**, Lipsman N, Kalia LV. Putaminal Recombinant Glucocerebrosidase Delivery with Magnetic Resonance-Guided Focused Ultrasound in Parkinson's Disease: A Phase I Study. *Mov Disord*. 2022 Oct;37(10):2134-2139.
5. Boutet A, Madhavan R, Elias GJB, Joel SE, Gramer R, Ranjan M, Paramanandam V, Xu D, Germann J, Loh A, **Kalia SK**, Hodaie M, Li B, Prasad S, Coblenz A, Munhoz RP, Ashe J, Kucharczyk W, Fasano A, Lozano AM. Predicting optimal deep brain stimulation parameters for Parkinson's disease using functional MRI and machine learning. *Nat Commun*. 2021 May 24;12(1):3043
6. Fomenko A, Lee DJ, McKinnon C, Lee EJ, de Snoo ML, Gondard E, Neudorfer C, Hamani C, Lozano AM, Kalia LV, **Kalia SK**. Deep Brain Stimulation of the Medial Septal Nucleus Induces Expression of a Virally Delivered Reporter Gene in Dentate Gyrus. *Front Neurosci*. 2020 May 12;14:463.
7. De Snoo ML, Friesen EL, Zhang YT, Earnshaw R, Dorval G, Kapadia M, O'Hara DM, Agapova V, Chau H, Pellerito O, Tang MY, Wang X, Schmitt-Ulms G, Durcan TM, Fon EA, Kalia LV, **Kalia SK**. Bcl-2-associated athanogene 5 (BAG5) regulates Parkin-dependent mitophagy and cell death. *Cell Death Dis*. 2019 Dec 2;10(12):907

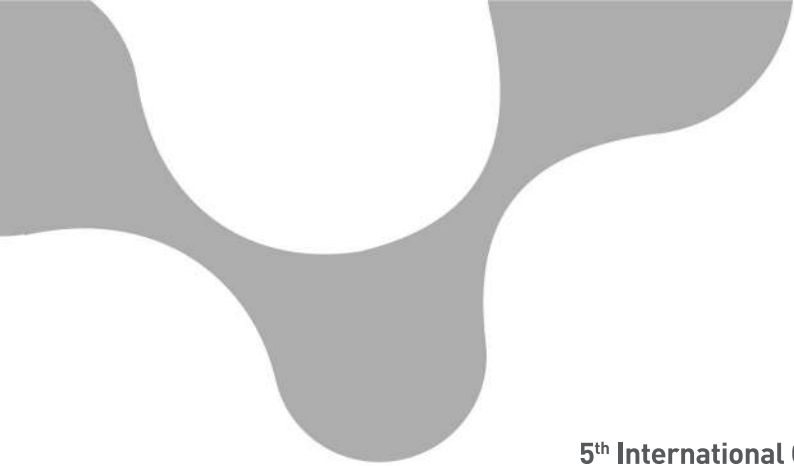
Full list of publications available at kalialabs.org



ICKMDS 2025

5th International Conference of Korean Movement Disorder Society

**Bridging from the Origin to Clinical Practice:
A Beginning Has an Ending**



ICKMDS 2025

5th International Conference of Korean Movement Disorder Society

Bridging from the Origin to Clinical Practice:
A Beginning Has an Ending

DAY 2_ November 1 (Sat), 2025

Room A

Parallel Session 6

Digital Technology in Movement Disorders

Chairs Roongroj BHIDAYASIRI (Thailand)
Sang-Myung CHEON (Korea)



Current and Future Landscape of Digital Clinic in Movement Disorders

Hubert FERNANDEZ

James and Constance Brown Endowed Chair in Movement Disorders
Chairperson and Director, Center for Neurological Restoration, Cleveland Clinic, USA
Professor of Neurology, Cleveland Clinic Lerner College of Medicine

Technology, and its use and integration in clinic, is a powerful tool and an inevitable feature in the daily assessment of any movement disorder patient. While developed countries are first to initiate and integrate technology in their clinics, it will eventually be used, even selectively, in developing countries. The huge advantages that technology offers are efficiency in evaluation, consistency and accuracy, and deeper granularity of assessment and outcomes data. This should translate into a more comprehensive evaluation and the implementation of precision medicine. The challenges of technology, on the other hand, include the difficulty of selecting which system to adopt given the endless options, the short lifecycle of any current technology given the remarkably fast pace of development, the initial and ongoing maintenance cost, and the buy-in from traditional/technologically-challenged members of the team. To provide a concrete example of how technology is currently integrated into clinical practice, this lecture will highlight the efforts of one Center (i.e. Cleveland Clinic's Movement Disorders Program under the Center for Neurological Restoration) to incorporate digitization in the way patients are evaluated and treated; including the use of big data to perform outcome measures, enhance assessment and management, and assist in clinical trials.

Bottomline, with the myriad of technological enhancements available, it can be challenging to select which ones to invest in. Practical recommendations include: a) selecting technology, not for the bells and whistles it offers, but because it is the best fit in fulfilling the program's goals and objectives; b) selecting technology that serves more than one purpose (e.g. a system that eases assessment, improves efficiency, but also collects outcomes data that can be published, or be utilized to further improve care); being mindful of the maintenance cost/resources needed to keep the digital infrastructure; and, d) the lifespan of the purchased system.

Personal Information

Full Name Hubert FERNANDEZ
Current Position Director and Chair
Department Center for Neurological Restoration
Organization Cleveland Clinic Neurological Institute
Country USA



Education / Training

Institution and Location	Degree	Year(s)	Field of Study
Brown University	Fellowship	1998-1999	Movement Disorders
Boston University	Residency	1995-1998	Neurology
University of Pennsylvania	Internship	1994-1995	Medicine
University of Santo Tomas/ Virgen Milagrosa Univ.	Doctor of Medicine	1988-1992	Medicine
University of Santo Tomas	Bachelor of Science	1985-1988	Biology, Accelerated Program

Positions and Honors

Professional Experience

Current

2016-Present Director and Chair, Center for Neurological Restoration, Cleveland Clinic
2011-Present Professor of Neurology, Cleveland Clinic Lerner College of Medicine
2011-Present Program Director, Fellowship in Movement Disorders, Cleveland Clinic

Previous

2011-2015 Section Head for Movement Disorders, Center for Neurological Restoration, Cleveland Clinic
2003-2010 Vice-Chair of Neurology, University of Florida
2003-2010 Co-Director, Movement Disorders Division, University of Florida
1999-2003 Associate Program Director, Movement Disorders, Brown University

Honors

- 2016-Present Inaugural Holder of James & Constance Brown Endowed Chair for Movement Disorders, Cleveland Clinic
- 2012 Presidential Distinguished Service Award, Movement Disorders Society

Selected Peer Reviewed Publications

1. **Fernandez HH**, Macklin EA, Hauser RA, Chou KL, Cakmak YO, Ozsoy B, Fahn S; Parkinson Study Group EARSTIM-PD Investigators. Intrinsic auricular muscle zone stimulation for Parkinson disease: The EARSTIM-PD Phase II multi-center pilot study results. *Parkinsonism Relat Disord*. 2024 Apr
2. **Fernandez HH**, Odin P, Standaert DG, Henriksen T, Jimenez-Shahed J, Metz S, Alobaidi A, Yan CH, Kukreja P, Parra JC, Zamudio J, Onuk K, Wright J, Antonini A. Healthcare resource utilization and device-aided therapy discussions with eligible patients across the Parkinson's disease continuum: Revelations from the MANAGE-PD validation cohort. *Parkinsonism Relat Disord*. 2023 Jul 6
3. **Fernandez HH**, Weintraub D, Macklin E, Litvan I, Schwarzschild MA, Eberling J, Videnovic A, Kenney CJ; Parkinson Study Group SYNAPSE Investigators. Safety, tolerability, and preliminary efficacy of SYN120, a dual 5-HT₆/5-HT_{2A} antagonist, for the treatment of Parkinson disease dementia: A randomized, controlled, proof-of-concept trial. *Parkinsonism Relat Disord*. 2023 Jul
4. Antonini A, Odin P, Schmidt P, Cubillos F, Standaert DG, Henriksen T, Jimenez-Shahed J, Alobaidi A, Jalundhwala YJ, Bao Y, Zamudio J, Parra JC, Kukreja P, Onuk K, Skalicky AM, Kleinman L, Jones E, Metz S, **Fernandez HH**. Validation and clinical value of the MANAGEPD tool: A clinician-reported tool to identify Parkinson's disease patients inadequately controlled on oral medications. *Parkinsonism Relat Disord*. 2021 Nov
5. Salles PA, Mata IF, **Fernandez HH**. Should we start integrating genetic data in decisionmaking on device-aided therapies in Parkinson disease? *Parkinsonism Relat Disord* 2021 Jul; 18 (88): 51-57.
6. **Fernandez HH**, Boyd JT, Fung VSC, Lew MF, Rodriguez RL, Slevin JT, Standaert DG, Zadikoff C, Vanagunas AD, Chatamra K, Eaton S, Facheris MF, Hall C, Robieson WZ, Benesh J, Espay AJ. Long-term safety and efficacy of levodopa-carbidopa intestinal gel in advanced Parkinson's disease. *Mov Disorder* 2018 March
7. **Fernandez HH**, Factor SA, Hauser RA, Jimenez-Shahed J, Ondo WG, Jarskog LF, Meltzer HY, Woods SW, Bega D, LeDoux MS, Sphrechter DR, Davis C, Davis MD, Stamler D, Anderson KE. Randomized controlled trial of deutetrabenazine for tardive dyskinesia: The ARM-TD Study. *Neurology* 2017 May;

Digital and/or Wearable Technology for Motor Symptoms of Parkinson's Disease

Jung Hwan SHIN

Department of Neurology, Seoul National University, Korea

This lecture will provide a comprehensive review of recent applications of digital technology in Parkinson's disease and movement disorders, with a particular focus on motor aspects. Advances in sensors, video-based assessments, and smartphone applications have enabled more detailed evaluation of motor symptoms. These technologies facilitate early detection and assessment in the prodromal and early stages of disease, as well as longitudinal monitoring of symptom changes in real-world settings. Furthermore, they are increasingly being adopted as outcome measures in clinical trials. This presentation will highlight these developments and discuss their implications for both clinical practice and research.

Personal Information

Full Name Jung Hwan SHIN
Current Position Assistant Professor
Department Department of Neurology
Organization Seoul National University Hospital
Country South Korea



Education / Training

Institution and Location	Degree	Year(s)	Field of Study
Seoul National University College of Medicine, Seoul		2010	Medicine
Seoul National University College of Medicine, Seoul	M.S.	2015	Clinical neuroscience
Korea Advanced Institute of Science and Technology	Ph.D.	2019	Systems neuroscience

Positions and Honors

Professional Experience

Current

2023.3- Assistant Professor, Seoul National University Hospital

Previous

2019-2022 Clinical Fellow, Seoul National University Hospital

2015-2019 Researcher and Ph.D candidate, Korea Advanced Institute of Science and Technology

2011-2015 Resident, Seoul National University Hospital

2010-2011 Intern, Seoul National University Hospital

Honors

2025 Junior award, Movement Disorder Society

2025 Best oral presentation award, KMDS

2022 Best researcher award, Seoul National University Hospital

2022	Best oral presentation award, Korean Neurology association
2021	Best oral presentation award, IC-KMDS
2021	Young Investigator award, Korean College of Geriatric Psychoneuropharmacology
2021	Postdoctoral Research Award, Association of Korean Neuroscientists
2021	SK Young investigator award, Korean Neurology association
2021	Best Researcher Award, Korea Movement Disorder Society
2020	Research Award for Clinical Fellow, Korea Movement Disorder Society
2019	Best presentation award, SYNERGIES meeting, MDS-AOS
2018	Research Award, Association of Korean Neuroscientists
2017	Best oral presentation award, 20th annual meeting of the Korean society for brain and neural science
2016	Research award for Resident, Korea Movement Disorder Society

Selected Peer Reviewed Publications

1. JH Shin, S Lee, KA Woo, HA Jinnah, A Shaikh, M Hallett, S Pandey, RC Helmich, M Vidailhet, V Fung, A Fasano, HJ Kim, B Jeon. Waveform-based analysis of head tremor using a marker-less tracking algorithm with 2D-video: evaluation of sinusoidality and rhythmicity, *Movement Disorders*, 2025
 2. JH Shin, YK Kang, Choi HY, Kim HJ, Cheon GJ, B Jeon. Whole brain dopamine transporter binding pattern predicts survival in multiple system atrophy. *Translational Neurodegeneration*. 2024
 3. JH Shin, HJ Kim, YK Kim, EJ Yoon, HW Nam, B Jeon, JY Lee. Longitudinal evolution of cortical thickness signature reflecting Lewy body dementia in isolated REM sleep behavior disorder: a prospective cohort study. *Translational neurodegeneration*. 2023
 4. JH Shin, JY Lee, YK Kim, EJ Yoon, HJ Kim, HW Nam, B Jeon. Parkinson's disease-related brain metabolic patterns and neurodegeneration in isolated REM sleep behavior disorder, *Neurology*. 2021
 5. JH Shin*, RR Lee*, HY Choi, HJ Kim, GJ Cheon, B Jeon. Variability of FP-CIT PET Patterns Associated with Clinical Features of Multiple System Atrophy. *Neurology*. 2021 (*Co-first author)
 6. JH Shin, M Song, SB Paik, MW Jung. Spatial organization of functional clusters representing reward and movement information in the striatal direct and indirect pathways. *Proceedings of the National Academy of Sciences of the United States of America*. 2020
 7. JH Shin, JY Lee, YK Kim, SA Shin, HW Nam, HJ Kim, B Jeon. Longitudinal change in dopamine transporter availability in idiopathic REM sleep behavior disorder. *Neurology*. 2020
- JH Shin, DH Kim, MW Jung. Differential coding of reward and movement information in the dorsomedial striatal direct and indirect pathways. *Nature Communications*. 2018

Digital and/or Wearable Technology for Non-Motor Symptoms of Parkinson's Disease

Kye Won PARK

Department of Neurology, Gangneung Asan Hospital, University of Ulsan College of Medicine, Republic of Korea

Digital biomarkers enable objective, remote, and repeated assessments of Parkinson's disease (PD) in patients' free-living environments, tackling the limitations of episodic, semi-subjective in-clinic evaluations. Although most progress has centered on motor features, extending digital technologies to non-motor symptoms (NMS) is imperative given their critical impact on quality of life. However, their heterogeneity and dynamic fluctuations across disease stages are challenging to address. This lecture will discuss the state of digital measures for key NMS domains and their technology readiness toward clinical utility and trial endpoints.

In sleep, wrist-worn actigraphy, multimodal wearables, and ambient/bed-integrated sensors have advanced practical monitoring. Actigraphy-derived features of nocturnal movement distribution, immobile bouts, and fragmentation showed meaningful associations with gold-standard sleep parameters. Home-based classifiers demonstrate promising accuracy for detecting rapid eye movement sleep behavior disorder in free-living conditions. Key limitations include limited validation against full polysomnography in PD cohorts, suboptimal specificity for differentiating sleep disorder subtypes, and incomplete PD-specific validation.

Autonomic dysfunction spans orthostatic hypotension, heart-rate variability (HRV) abnormalities, sudomotor alterations, and skin temperature changes, with broader links to gastrointestinal, urinary, and sexual function. Multimodal sensing—rings, wrist-watches, and chest straps, electrodermal activity (EDA)-capable wearables, etc.—enables continuous characterization of sympathetic–parasympathetic dynamics. Consistent findings include reduced HRV in PD and EDA-derived analytics can indicate non-motor fluctuations.

For mood and emotion, active voice-based biomarkers, digital patient-reported outcomes, and passive phenotyping show promise for screening depressive burden and apathy. However, critical challenges include confounding from motor speech impairment, the need for cross-cultural and multilingual validation, and sustaining long-term adherence.

Cognitive impairment is being captured through smartphone micro-tasks, sensor-rich smart-home measures (activity/movement patterns), and ePRO-based cognitive snapshots. Early data indicate strong correlations with traditional neuropsychological tests, but much validation derives from general populations and PD-specific longitudinal datasets are maturing.

Future efforts should prioritize multicenter, PD-specific validation with standardized protocols, the development of composite digital endpoints aligned with existing non-motor clinical scales, and privacy-preserving integration of personalized multimodal monitoring into clinical trials and routine care.

Personal Information

Full Name Kye Won PARK
Current Position Assistant Professor
Department Neurology
Organization Gangneung Asan Hospital
Country South Korea



Education / Training

Institution and Location	Degree	Year(s)	Field of Study
University of Ulsan, College of Medicine, Seoul, Korea	Ph.D.	2021	Medicine (Neurology)
University of Ulsan, College of Medicine, Seoul, Korea	M.Sc.	2017	Medicine (Neurology)
Catholic University of Korea College of Medicine, Seoul, Korea	M.D.	2012	Medicine

Positions and Honors

Professional Experience

Current

2024- Assistant Professor, Department of Neurology, Gangneung Asan Hospital, University of Ulsan College of Medicine, Gangneung, Korea

Previous

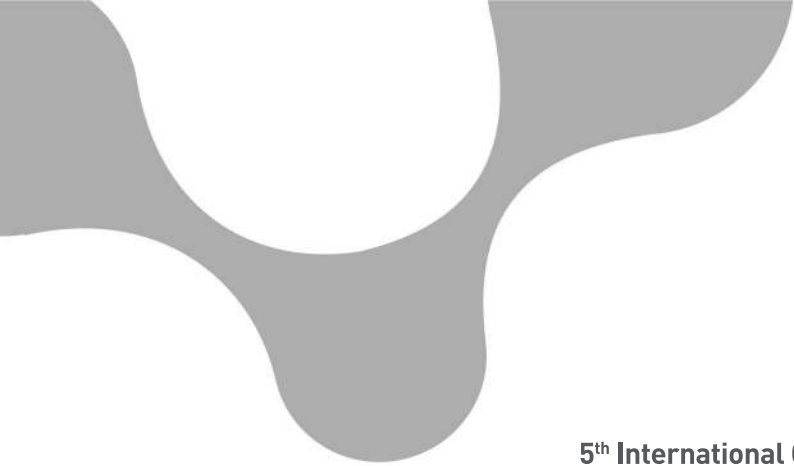
2022-2024 Postdoctoral research fellow, Pacific Parkinson Research Centre, University of British Columbia, BC, Canada

2021-2022 Assistant Professor, Department of Neurology, University of Eulji College of Medicine, Uijeongbu Eulji Medical Centre, Uijeongbu, Korea

2018-2021 Clinical Fellow, Neurology (Movement Disorders), Asan Medical Centre, Seoul, Korea

Selected Peer Reviewed Publications

1. **Park KW**, Mirian MS, McKeown MJ. Artificial intelligence-based video monitoring of movement disorders in the elderly: a review on current and future landscapes. *Singapore Med J*. 2024 Mar 1;65(3):141-149.
2. **Park KW**, Woo HT, Hwang YS, Lee SH, Chung SJ. Appendectomy and the Risk of Parkinson's Disease: A Korean Nationwide Study. *Mov Disord Clin Pract*. 2024 Jun;11(6):704-707.
3. Yu T, **Park KW**, McKeown MJ, Wang ZJ. Clinically Informed Automated Assessment of Finger Tapping Videos in Parkinson's Disease. *Sensors (Basel)*. 2023 Nov 13;23(22):9149.
4. Gholami M, Ward R, Mahal R, Mirian M, Yen K, **Park KW**, McKeown MJ, Wang ZJ. Automatic labeling of Parkinson's Disease gait videos with weak supervision. *Med Image Anal*. 2023 Oct;89:102871.
5. **Park KW**, Ryu HS, Shin E, Park Y, Jeon SR, Kim SY, Kim JS, Koh SB, Chung SJ. Ethnicity- and sex-specific genome wide association study on Parkinson's disease. *NPJ Parkinsons Dis*. 2023 Oct 7;9(1):141.
6. **Park KW**, Hwang YS, Lee SH, Jo S, Chung SJ. The Effect of Blood Lipids, Type 2 Diabetes, and Body Mass Index on Parkinson's Disease: A Korean Mendelian Randomization Study. *J Mov Disord*. 2023 Jan;16(1):79-85.
7. **Park KW**, Wu HJ, Yu T, Mahal R, Mirian MS, McKeown MJ. Potential Pitfalls of Remote and Automated Video Assessments of Movements Disorders. *Mov Disord*. 2023 Mar;38(3):504-506.



ICKMDS 2025

5th International Conference of Korean Movement Disorder Society

Bridging from the Origin to Clinical Practice:
A Beginning Has an Ending

DAY 2_ November 1 (Sat), 2025

Room A

Parallel Session 7

Is Normal Pressure Hydrocephalus a Movement Disorder?

Chairs Jinyoung YOUN (Korea)
Hee-Tae KIM (Korea)



Epidemiology, Natural History, and Genetic Factors of Idiopathic Normal Pressure Hydrocephalus

Chifumi ISEKI

Department of Behavioral Neurology and Cognitive Neuroscience, Tohoku University, Japan

We had initiated a cohort study of residents in Takahata Town and Sagae City, Yamagata Prefecture, in 2000. From the cohort, the prevalence of iNPH was 0.5% for residents aged 61 years and older, and 7.7% for those aged 86, 27.4% in aged 92, with the prevalence increasing with age, and the incidence was 15 times higher (Iseki et al. 2015) than previous hospital-based study in Netherland. We also found that there are cases suspected to be complicated with Alzheimer's disease and other forms of dementia. It is challenging to differentiate iNPH from age-related changes, other diseases, especially in those over 80. We were also the first familial normal pressure hydrocephalus (fNPH), which was indistinguishable from iNPH both in symptoms and brain imaging (Takahashi et al. 2011). Furthermore, genetic analysis found that a copy number loss in the *SFMBT1* gene was identified as a risk gene for iNPH (Sato et al. 2016). It was hoped that the pathogenesis of iNPH, which has been "pathogenesis unknown," could be elucidated by the combination and accumulation of multifactorial risks.

Personal Information

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Education / Training

Institution and Location	Degree	Year(s)	Field of Study
Fukushima Medical University	Medical Doctor	2003	Medicine
Yamagata University	Ph.D.	2007	Neuroepidemiology

Positions and Honors

Professional Experience

Current

2023- Lecturer, Tohoku University

Previous

2016- Lecturer, Yamagata University

2015- Assistant Professor, Yamagata University

Honors

2017 Yamagata Soroptimist Scientist Award

Selected Peer Reviewed Publications

1. Fasano A, Iseki C, Yamada S, Miyajima M. What is idiopathic in normal pressure hydrocephalus? J Neurosurg Sci. 2025 Feb;69(1):20-36.
2. Iseki C, Ishii K, Pozzi NG, Todisco M, Pacchetti C. Instrumental assessment of INPH: structural and functional neuroimaging. J Neurosurg Sci. 2025 Feb;69(1):64-78.
3. Yamada S, Ito H, Iseki C, Otani T, Ii S, Watanabe Y, Wada S, Oshima M. Regional Brain Volume Changes in Hakim's Disease vs. Alzheimer's and Mild Cognitive Impairment. Brain Commun. 2025 Mar 26;7(2):fcaf122.

4. Sugai Y, Hiraka T, Shibata A, Taketa A, Tanae T, Moriya Y, Ohara S, Iseki C, Ohta Y, Kanoto M. Augmentation of perivascular space visualization in basal ganglia and white matter hyperintensity lesion is a meaningful finding for subsequent cognitive decline. *Acta Radiol.* 2024 Jul;65(7):792-799.
5. Iseki C, Hayasaka T, Yanagawa H, Komoriya Y, Kondo T, Hoshi M, Fukami T, Kobayashi Y, Ueda S, Kawamae K, Ishikawa M, Yamada S, Aoyagi Y, Ohta Y. Artificial Intelligence Distinguishes Pathological Gait: The Analysis of Markerless Motion Capture Gait Data Acquired by an iOS Application (TDPT-GT). *Sensors (Basel).* 2023 Jul 7;23(13):6217.
6. Iseki C, Takahashi Y, Adachi M, Igari R, Sato H, Koyama S, Ishizawa K, Ohta Y, Kato T. Prevalence and development of idiopathic normal pressure hydrocephalus: A 16-year longitudinal study in Japan. *Acta Neurol Scand.* 2022 Nov;146:680-689.
7. Suzuki Y, Iseki C, Igari R, Sato H, Koyama S, Kawahara H, Itagaki H, Sonoda Y, Ohta Y. Reduced cerebral blood flow of lingual gyrus associated with both cognitive impairment and gait disturbance in patients with idiopathic normal pressure hydrocephalus. *J Neurol Sci.* 2022;437:120266.

Parkinsonism in Idiopathic Normal Pressure Hydrocephalus

Jinyoung YOUN

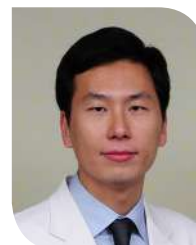
Department of Neurology, Samsung Medical Center, Sungkyunkwan University School of Medicine, Seoul, Republic of Korea

Normal pressure hydrocephalus (NPH) is a communicating hydrocephalus with normal pressure and the triad of gait disturbance, cognitive decline and urinary symptoms. Among these characteristic symptoms, gait disturbance is most frequent and responsive symptom. However, gait disturbance itself can be common in various neurodegenerative disorders, and various gait patterns can be seen in NPH. Therefore, the differential diagnosis can be difficult in clinical practice.

Typically, lower body parkinsonism, such as short and symmetrical steps, freezing of gait, festination and magnetic gait, is regarded as clinical presentation in NPH. However, gait patterns in NPH can be heterogenous, and NPH patients with locomotor subtype of gait disturbance demonstrated the correlation between dopamine transporter uptake and parkinsonism in lower body. NPH patients revealed different response of dopamine transporter uptake to shunt surgery.

Personal Information

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Education / Training

Institution and Location	Degree	Year(s)	Field of Study
Sungkyunkwan university, school of medicine, Korea	MD	2003	Medicine
Sungkyunkwan university, school of medicine, Korea	MS	2008	Neurology
Sungkyunkwan university, school of medicine, Korea	PhD	2016	Neurology

Positions and Honors

Professional Experience

Current

2025- Professor, Department of Neurology, Samsung Medical Center, Sungkyunkwan University School of Medicine, Seoul, Korea

Previous

2018-2025 Assistant Professor, Department of Neurology, Samsung Medical Center, Sungkyunkwan University School of Medicine, Seoul, Korea

2015-2018 Clinical Assistant Professor, Department of Neurology, Samsung Medical Center, Sungkyunkwan University School of Medicine, Seoul, Korea

2014-2015 Instructor, Department of Neurology, Samsung Medical Center, Sungkyunkwan University School of Medicine, Seoul, Korea

2020-2021 Clinical fellow in Movement Disorder Surgical program, University of Toronto, Toronto, Ontario, Canada

Selected Peer Reviewed Publications

1. Ha J, Lee S, Kim S, Lee JS, Ahn JH, Cho JW, Fasano A, **Youn J**. The "Hedgehog-Halo Sign" Is Associated with Gait Symptom Severity and Tap Response in Normal Pressure Hydrocephalus. *Mov Disord Clin Pract*. 2025. PMID: 39503269
2. **Youn J**, Todisco M, Zappia M, Pacchetti C, Fasano A. Parkinsonism and cerebrospinal fluid disorders. *J Neurol Sci*. 2022 Feb 15;433:120019. doi: 10.1016/j.jns.2021.120019. PMID: 34674853
3. Bluett B, Ash E, Farheen A, Fasano A, Krauss JK, Maranzano A, Passaretti M, Tang-Wai DF, Van Gerpen J, Alonso-Canovas A, **Youn J**, Malm J, Martino D; International Parkinson and Movement Disorder Society Normal Pressure Hydrocephalus Study Group. Clinical Features of Idiopathic Normal Pressure Hydrocephalus: Critical Review of Objective Findings. *Mov Disord Clin Pract*. 2022 Nov 23;10(1):9-16. doi: 10.1002/mdc3.13608. PMID: 36704075
4. **Youn J**, Kovacs GG, Kongkham P, Fasano A. Corpus Callosum Hyperintensity in Normal Pressure Hydrocephalus After Ventriculoperitoneal Shunt. *Neurology*. 2021 Jun 8;96(23):1096-1097. doi: 10.1212/WNL.0000000000012041. PMID: 33893207
5. Park G, Ha J, Lee JS, Ahn JH, Cho JW, Seo SW, **Youn J**, Kim H. Data-driven, cross-sectional image-based subtyping and staging of brain volumetric changes in Parkinson's disease. *Neurobiol Dis*. 2025 Aug;212:106970. doi: 10.1016/j.nbd.2025.106970. PMID: 40418995
6. **Youn J**, Won JH, Kim M, Kwon J, Moon SH, Kim M, Ahn JH, Mun JK, Park H, Cho JW. Extra-Basal Ganglia Brain Structures Are Related to Motor Reserve in Parkinson's Disease. *J Parkinsons Dis*. 2023;13(1):39-48. doi: 10.3233/JPD-223542. PMID: 36565134
7. **Youn J**, Kim M, Park S, Kim JS, Park H, Cho JW. Pallidal Structural Changes Related to Levodopa-induced Dyskinesia in Parkinson's Disease. *Front Aging Neurosci*. 2022 May 6;14:781883. doi: 10.3389/fnagi.2022.781883. eCollection 2022. PMID: 35601615

Idiopathic Normal Pressure Hydrocephalus and Neurodegenerative Comorbidities

Kyunghun KANG

Department of Neurology, School of Medicine, Kyungpook National University, South Korea

Idiopathic normal pressure hydrocephalus (INPH) is defined by non-obstructive ventricular enlargement accompanied by the clinical triad of gait disturbance, cognitive impairment, and urinary dysfunction. Although INPH is one of the few treatable neurological conditions, its diagnosis is often challenging because of variability in symptom expression and frequent overlap with neurodegenerative diseases such as Alzheimer's disease (AD) and Parkinson's disease (PD). Among neurodegenerative disorders in the elderly, AD is the most common and PD is the second most common, and both are prevalent in the same age group as INPH, making comorbidity an important consideration. Neuroimaging biomarkers provide essential tools for distinguishing INPH from these conditions. Fluorine-18 N-3-fluoropropyl-2 β -carbomethoxy-3 β -(4-iodophenyl)-nortropane (F-18 FP-CIT) positron emission tomography (PET), which assesses dopamine transporter (DAT) availability, has been widely used in the evaluation of parkinsonian syndromes. Pathological studies demonstrate that reduced DAT binding reflects nigrostriatal terminal loss, a hallmark of PD and atypical parkinsonian syndromes. In INPH, presynaptic nigrostriatal degeneration is generally absent or minimal, and most patients exhibit normal DAT binding, which enables differentiation from neurodegenerative parkinsonism. Amyloid PET, on the other hand, provides sensitive and specific detection of neuritic amyloid plaque accumulation, a pathological feature of AD, and is increasingly applied to assess possible AD comorbidity in INPH patients. Because amyloid deposition occurs silently over years, amyloid PET helps identify INPH patients who harbor concurrent AD pathology and allows more precise stratification. Clinical observations indicate that some patients present with definite INPH but also have a high likelihood of comorbid PD, AD, or even both, which complicates diagnostic judgment and treatment decisions. Traditionally, the presence of comorbid AD or PD has been regarded as a predictor of poor response to cerebrospinal fluid shunt surgery, leading many clinicians to hesitate in offering surgical treatment when neurodegenerative pathology is suspected. However, this conventional view warrants reassessment. The coexistence of AD or PD does not necessarily preclude shunt responsiveness. If additional clinical and imaging assessments suggest that the patient has shunt-responsive INPH, surgical intervention should still be considered. Evidence indicates that even in the presence of comorbidity, certain patients achieve meaningful symptomatic improvement, particularly in gait function, following shunt surgery. Therefore, suspicion of comorbid neurodegeneration should not automatically exclude patients from surgical treatment, and careful patient selection based on multimodal diagnostic approaches is essential. In conclusion, INPH frequently coexists with AD and PD, and awareness of such comorbidities is crucial for clinical management. The integration of imaging biomarkers such as F-18 FP-CIT PET and amyloid PET provides valuable insight into underlying pathology and refines diagnostic and therapeutic strategies. Revisiting the traditional assumption that comorbidity precludes successful surgical outcomes may broaden treatment opportunities and ultimately improve quality of life for patients affected by this complex condition.

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Education / Training

Institution and Location	Degree	Year(s)	Field of Study
Kyungpook National University	M.D.	2003	Medicine
Kyungpook National University Hospital	Internship	2004	Medicine
Kyungpook National University Hospital	Residency	2008	Neurology
Kyungpook National University	M.M.Sc.	2006	Neurology
Kyungpook National University Hospital	Fellowship	2013	Neurology
Hanyang University	Ph.D.	2020	Biomedical Engineering

Positions and Honors

Professional Experience

Current

2024 - Professor
Department of Neurology, School of Medicine, Kyungpook National University
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2025 - Director of Education and Research
Kyungpook National University Chilgok Hospital

Previous

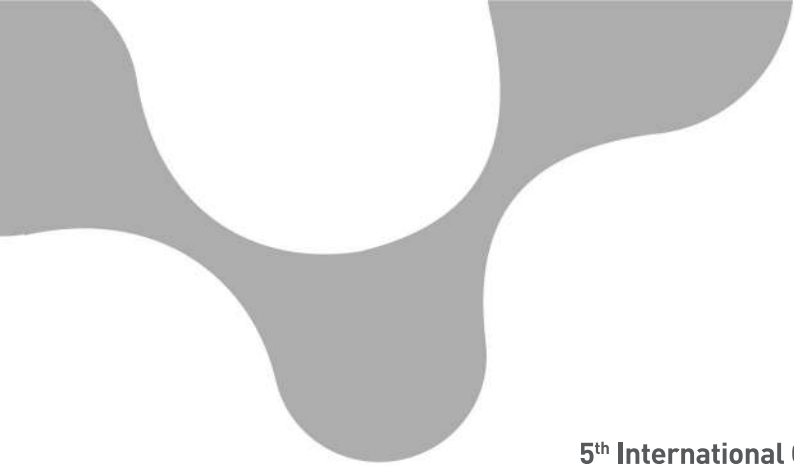
2019 – 2024 Associate Professor
Department of Neurology, School of Medicine, Kyungpook National University
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2014 – 2019 Assistant Professor
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Department of Neurology, Kyungpook National University Chilgok Hospital

2017 – 2018 International Visiting Scholar
Department of Neurology, Memory and Aging Center, University of California, San Francisco

Selected Peer Reviewed Publications

1. Minju Kim, Nayoung Ryoo, Sang-Won Han, So Young Ji, Kihwan Hwang, Jung Ho Han, Chae-Yong Kim, Eugene Lee, Eunjeong Ji, Ki-Su Park, **Kyunghun Kang**, Young Ho Park, SangYun Kim. Long-term gait improvement following a CSF tap test in idiopathic normal pressure hydrocephalus patients: an analysis of clinical outcomes. *Neurol Sci*. 2025.
2. Eunhee Park, **Kyunghun Kang**, Janghoon Lee, Jong Taek Lee, Ki-Su Park, Chul-Hyun Kim, Yu-Sun Min. Characteristics of Postural Instability in Idiopathic Normal Pressure Hydrocephalus. *Neurol Ther*. 2025.
3. Man-Kyu Park, **Kyunghun Kang**, Sang-Kyu Son, Eunhee Park, Ki-Su Park. Combining Unilateral Biportal Endoscopic Surgery with Lumboperitoneal Shunt Surgery for Patients with Coexisting Normal-Pressure Hydrocephalus and Lumbar Spinal Stenosis: A Technical Note. *World Neurosurg*. 2024.
4. Eunhee Park, Sanghyeon Lee, Tae-Du Jung, Ki-Su Park, Jong Taek Lee, **Kyunghun Kang**. Changes in postural stability after cerebrospinal fluid tap test in patients with idiopathic normal pressure hydrocephalus. *Front Neurol*. 2024.
5. **Kyunghun Kang**, Shin Young Jeong, Ki-Su Park, Myong Hun Hahm, Jaeil Kim, Ho-Won Lee, Chi-Hun Kim, Eunkyeong Yun, Jaehwan Han, Uicheul Yoon, Sang-Woo Lee. Distinct cerebral cortical perfusion patterns in idiopathic normal-pressure hydrocephalus. *Hum Brain Mapp*. 2023.
6. **Kyunghun Kang**, Ho-Won Lee, Duk-Hee Lee. Plasma exchange: An effective method to eliminate neurotoxic lipophilic chemicals from blood. *Alzheimers Dement*. 2022.
7. Jaehwan Han, Myoung Nam Kim, Ho-Won Lee, Shin Young Jeong, Sang-Woo Lee, Uicheul Yoon, **Kyunghun Kang**. Distinct volumetric features of cerebrospinal fluid distribution in idiopathic normal-pressure hydrocephalus and Alzheimer's disease. *Fluids Barriers CNS*. 2022.



ICKMDS 2025

5th International Conference of Korean Movement Disorder Society

Bridging from the Origin to Clinical Practice:
A Beginning Has an Ending

DAY 2_ November 1 (Sat), 2025

Room A

Parallel Session 9

Update on Non-Invasive Brain Stimulation in Movement Disorders

Chairs Yoshikazu UGAWA (Japan)
Sang Jin KIM (Korea)



Recent Advances in Transcranial Ultrasound Stimulation for Neurodegenerative Disorders

Hyungmin KIM

Bionics Research Center, Korea Institute of Science and Technology, Republic of Korea

Transcranial ultrasound stimulation (TUS) is emerging as a promising noninvasive neuromodulation technique with unique advantages over existing brain stimulation modalities. Conventional approaches such as transcranial magnetic stimulation (TMS) or transcranial direct current stimulation (tDCS) are limited in their ability to engage deep circuits, whereas TUS can target both cortical and subcortical regions with millimeter-scale precision. Moreover, its bidirectional capacity to facilitate or suppress neural activity provides a versatile platform adaptable to diverse neurological and neurodegenerative disorders.

Technological progress has accelerated the translational potential of TUS. Computational simulations and subject-specific targeting strategies help overcome skull-induced distortions and ensure precise energy delivery, while portable and wearable stimulation systems allow for repeated or even ambulatory application. These advances are transforming TUS from a conceptual tool into a clinically viable therapeutic option.

Preclinical and translational research underscores the wide range of conditions in which TUS may be beneficial. In stroke, ultrasound stimulation has been shown to promote neuroplasticity, restore interhemispheric balance, and enhance functional recovery, positioning it as a potential adjunct to rehabilitation. In epilepsy, TUS has demonstrated the ability to suppress abnormal activity with high spatial selectivity, raising the possibility of noninvasive seizure control. In essential tremor (ET), studies using transcranial focused ultrasound (tsFUS) have shown that ultrasound can modulate thalamic networks implicated in tremor generation, suggesting a noninvasive alternative or complement to invasive neuromodulation approaches. In normal-pressure hydrocephalus (NPH), ongoing work is investigating whether ultrasound can improve cerebrospinal fluid (CSF) circulation and relieve pressure-related dysfunction, offering a potential alternative to shunt surgery. In Alzheimer's disease (AD), repeated low-intensity stimulation is being evaluated for its effects on amyloid-beta aggregation, neurovascular coupling, and cognitive outcomes, reflecting its potential role as a disease-modifying therapy.

The convergence of these applications highlights TUS as a platform technology rather than a disorder-specific intervention. Across stroke, epilepsy, ET, NPH, and AD, ultrasound targets key pathophysiological features such as disrupted network dynamics, abnormal oscillatory activity, impaired CSF flow, and pathological protein accumulation. This broad applicability emphasizes its value as a versatile and adaptable therapeutic approach.

For clinicians, several aspects of TUS are particularly compelling. It enables noninvasive access to deep and functionally critical brain circuits, offers flexibility through excitatory or inhibitory modulation, and carries a favorable safety profile that supports repeated or long-term use. These characteristics make TUS suitable not only for acute interventions but also for chronic disease management across a variety of neurological conditions.

In summary, TUS is rapidly evolving into a clinically relevant neuromodulation strategy with broad therapeutic promise. By combining engineering innovations, preclinical validation in multiple disease models, and growing translational research, TUS is positioned to expand treatment options for stroke, epilepsy, essential tremor, NPH, Alzheimer's disease, and beyond. For movement disorder specialists and neurologists, the central message is that ultrasound offers a noninvasive yet precise means of modulating dysfunctional brain circuits, with the potential to reshape therapeutic strategies in neurology.

Personal Information

Full Name Hyungmin KIM
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Country South Korea



Education / Training

Institution and Location	Degree	Year(s)	Field of Study
Harvard Medical School, USA	PostDoc	2013	Radiology
University of Bern, Switzerland	Ph.D.	2011	Cellular and Biomedical Science
Seoul National University, South Korea	M.S.	2001	Mechanical Engineering
Seoul National University, South Korea	B.S.	1999	Mechanical Engineering
Kyungpook National University Hospital	Fellowship	2013	Neurology

Positions and Honors

Professional Experience

Current

2021 - Professor, KIST School, University of Science & Technology (UST)
2019 - Principal Research Scientist, Korea Institute of Science and Technology (KIST)

Previous

2014 - 2021 Associate Professor, KIST School, University of Science & Technology (UST)
2013 - 2019 Senior Research Scientist, Korea Institute of Science and Technology (KIST)
2007 - 2011 PhD Student, Institute for Surgical Technology and Biomechanics, Switzerland
2002 - 2007 Technical Research Personnel / Director of Research Center, Cybermed Inc.

Honors

2025 KIST Person of the Month, Korea Institute of Science and Technology (KIST)
2021 Academic Award, The Korean Society for Therapeutic Ultrasound (KSTU)

- 2019 Outstanding Professor Award, University of Science & Technology (UST)
- 2018 Excellent Paper Award, National Research Foundation of Korea

Selected Peer Reviewed Publications

1. Sungjun Lee, Jeungeun Kum, Sumin Kim, Hyunjin Jung, Soojung An, Soon Jin Choi, Jae Hyuk Choi, Jinseok Kim, Ki Jun Yu, Wonhye Lee, Hyeok Kim, Hyung-Seop Han, Mikyung Shin, **Hyungmin Kim**, Donghee Son, A shape-morphing cortex-adhesive sensor for closed-loop transcranial ultrasound neurostimulation, *Nature Electronics*, 2024
2. Tae Young Park, Heekyung Koh, Wonhye Lee, So Hee Park, Won Seok Chang, **Hyungmin Kim**, Real-Time Acoustic Simulation Framework for tFUS: A Feasibility Study Using Navigation System, *NeuroImage*, 2023
3. Tae Young Park, Ji Hyeok Jeong, Yong An Chung, Sang Hoon Yeo, **Hyungmin Kim**, Application of subject-specific helmets for the study of human visuomotor behavior using transcranial focused ultrasound: a pilot study, *Computer Methods and Programs in Biomedicine*, 2022
4. Heekyung Koh, Tae Young Park, Yong An Chung, Jong-Hwan Lee, **Hyungmin Kim**, Acoustic simulation for transcranial focused ultrasound using GAN-based synthetic CT, *IEEE Journal of Biomedical and Health Informatics*, 2021
5. Hongchae Baek, Anvar Sariev, Seul Lee, Suh-Yeon Dong, Sebastien Royer, **Hyungmin Kim**, Deep Cerebellar Low-Intensity Focused Ultrasound Stimulation Restores Interhemispheric Balance after Ischemic Stroke in Mice, *IEEE Transactions on Neural Systems and Rehabilitation Engineering*, 2020
6. Ki Joo Pahk, Matheus Oliveira de Andrade, Pierre G  lat, **Hyungmin Kim**, Nader Saffari, Mechanical damage induced by the appearance of rectified bubble growth in a viscoelastic medium during boiling histotripsy exposure, *Ultrasonics Sonochemistry*, 2019
7. Hongchae Baek, Ki Joo Pahk, Min-Ju Kim, Inchan Youn, **Hyungmin Kim**, Modulation of cerebellar cortical plasticity using low-intensity focused ultrasound for poststroke sensorimotor function recovery, *Neurorehabilitation and neural repair*, 2018

Utility of Transcranial Magnetic Stimulation to Understand Pathophysiology: Lessons on Dementia

Takenobu MURAKAMI

Division of Neurology, Department of Brain and Neurosciences, Faculty of Medicine, Tottori University, Japan

Transcranial magnetic stimulation (TMS) is a noninvasive brain stimulation technique extensively used in neuroscience and neuropsychiatry. Here I would like to update TMS research focusing on dementing disorders.

Short-latency afferent inhibition (SAI), which can evaluate the central cholinergic function, decreases in Alzheimer's disease (AD) and dementia with Lewy bodies (DLB). Short-interval intracortical inhibition and intracortical facilitation measured by paired-pulse paradigms, which show the functions of central GABAergic and glutamatergic neuronal circuits respectively, are reduced in patients with frontotemporal dementia and DLB. Combination of these parameters can successfully differentiate dementing disorders. A novel technique combining TMS with electroencephalogram allows the evaluation of functional brain networks. Neural activity propagation from precuneus to frontal lobe is disrupted in patients with AD. The long-term potentiation (LTP)-like synaptic plasticity induced by repetitive TMS mode is impaired in patients having AD pathology, and its severity correlates with degrees of cognitive declines, AD-related biomarkers including amyloid-beta 42 in the cerebrospinal fluid and amyloid accumulation in precuneus detected by amyloid positron emission tomography.

In conclusion, TMS holds significant promise as a tool for understanding the pathophysiology of dementia.

Personal Information

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Country Japan



Education / Training

Institution and Location	Degree	Year(s)	Field of Study
Graduate School, Tottori University, Japan	Ph.D.	2008	Medicine
Faculty of Medicine, Tottori University, Japan	M.D.	2002	Medicine

Positions and Honors

Professional Experience

Current

2025 Lecturer, Division of Neurology, Department of Brain and Neurosciences, Faculty of Medicine, Tottori University

Previous

2021-2024 Assistant Professor, Division of Neurology, Department of Brain and Neurosciences, Faculty of Medicine, Tottori University

2019-2020 Director of Neurology, Tottori Prefectural Kousei Hospital

2012-2019 Assistant Professor, Department of Neurology, Faculty of Medicine, Fukushima Medical University

2009-2011 Postdoctoral Research Fellow, Department of Neurology, J.W. Goethe-University, Frankfurt am Main, Germany

2005-2008 Staff Physician, Department of Neurology, Institute of Neurological Sciences, Faculty of Medicine, Tottori University

2004-2005 Staff Physician, Tottori Prefectural Kousei Hospital

2002-2004 Resident, Tottori Prefectural Central Hospital

2002 Resident, Department of Neurology, Institute of Neurological Sciences, Faculty of Medicine, Tottori University

Honors

2024	Teraoka Award, Japan Brain Foundation
2024	The Editor's Choice Award, International Federation of Clinical Neurophysiology
2021	Best Oral Presentation Award, International Conference of Korean Movement Disorder Society
2017	Encouragement Prize, Japanese Society of Clinical Neurophysiology
2015	Mentor Award, Japanese Society of Internal Medicine
2010	Best Investigator Award, 29 th International Congress of Clinical Neurophysiology
2010	Poster Prize, 2010 TMS Summer School, University of Oxford, UK
2010	Best Poster Prize, 54. Jahrestagung der DGKN, Germany
2008	Best Paper Award, The YONAGO Medical Association, Tottori University

Selected Peer Reviewed Publications

1. **Murakami T.**, Abe M., Tiksnadi A., Nemoto A., Futamura M., Yamakuni R., Kubo H., Kobayashi N., Ito H., Hanajima R., Hashimoto Y., Ugawa Y. Abnormal motor cortical plasticity as a useful neurophysiological biomarker for Alzheimer's disease pathology. *Clinical Neurophysiology* 2024; 158: 170-179.
2. Wiratman W., **Murakami T.**, Tiksnadi A., Kobayashi S., Hanajima R., Ugawa Y. Enhancement of LTD-like aftereffects by combining QPSLTD with paired-associative peripheral nerve stimulation (PA_{LTD}-QPS_{LTD}). *Clinical Neurophysiology* 2022; 138: 9-17.
3. Tiksnadi A., **Murakami T.**, Wiratman W., Matsumoto H., Ugawa Y. Direct Comparison of Efficacy of the Motor Cortical Plasticity Induction and the Interindividual Variability between TBS and QPS. *Brain Stimulation* 2020; 13: 1824-1833.
4. **Murakami T.**, Abe M., Wiratman W., Fujiwara J., Okamoto M., Mizuuchi-Endo T., Iwabuchi T., Makuuchi M., Yamashita A., Tiksnadi A., Chang F-Y., Kubo H., Matsuda N., Kobayashi S., Eifuku S., Ugawa Y. The motor network reduces multisensory illusory perception. *The Journal of Neuroscience* 2018; 38: 9679-9688.
5. **Murakami T.**, Kell C., Restle J., Ugawa Y., Ziemann U. Left dorsal speech stream components and their contribution to phonological processing. *The Journal of Neuroscience* 2015; 35: 1411-1422.
6. **Murakami T.**, Müller-Dahlhaus F., Lu M., Ziemann U. Homeostatic metaplasticity of corticospinal excitatory and intracortical inhibitory neural circuits in human motor cortex. *Journal of Physiology* 2012; 590: 5765-5781.
7. **Murakami T.**, Restle J., Ziemann U. Observation-execution matching and action inhibition in human primary motor cortex during viewing of speech-related lip movements or listening to speech. *Neuropsychologia* 2011; 49: 2045-2054.

S9-3

Update of Transcranial Direct Current Stimulation: Challenges in the Clinical Trials and Application

Wooyoung JANG

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Transcranial direct current stimulation (tDCS) is a non-invasive neuromodulation technique that delivers low-intensity direct current through the scalp to modulate cortical excitability. According to ClinicalTrials.gov, there are now several hundred registered trials investigating tDCS across a wide range of conditions. Despite this large body of work, results remain highly heterogeneous. Variability in stimulation parameters (intensity, electrode placement, session number) and patient characteristics has led to inconsistent outcomes, and meta-analyses often conclude that robust, reproducible evidence is still limited. In this lecture, I will first highlight recent **mechanistic insights**, including evidence that tDCS induces metabolomic changes, biological modulation, and alters network connectivity. Next, I will review **updates of tDCS studies in movement disorders field**, with a focus on new approaches such as **home-based protocols**, **high-definition tDCS**, and **network-targeted stimulation**. Finally, I will discuss recent clinical findings in Movement disorders field. Together, these findings position tDCS as “promising but not definitive,” especially in movement disorders.

Personal Information

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Education / Training

Institution and Location	Degree	Year(s)	Field of Study
Hanyang University College of Medicine	Medical Doctor	1997~2003	
Postgraduate School of Medicine, Hanyang University	Ph.D	2015~2017	Neurology
Department of neurology, Hanyang medical hospital	Trainee	2004~2008	Neurology
Department of neurology, Hanyang medical hospital	Clinical Fellow	2011~2013	Neurology

Positions and Honors

Professional Experience

Current

2025 - Professor, Department of Neurology, Gangneung Asan Hospital, University of Ulsan College of Medicine

Previous

2018~2024 Associate professor, Department of Neurology, Gangneung Asan Hospital, University of Ulsan College of Medicine

Selected Peer Reviewed Publications

1. Kim JE, Lee HS, Jang W. Serum zinc deficiency is a potential risk factor for the occurrence of levodopa-induced dyskinesia in drug-naïve Parkinson's disease. *Frontiers in Aging Neuroscience*. 2023;Volume 15 - 2023.
2. Oh E, Park J, Youn J, Jang W. Anodal Transcranial Direct Current Stimulation Could Modulate Cortical Excitability and the Central Cholinergic System in Akinetic Rigid-Type Parkinson's Disease: Pilot Study. *Frontiers in Neurology*. 2022;Volume 13 - 2022.

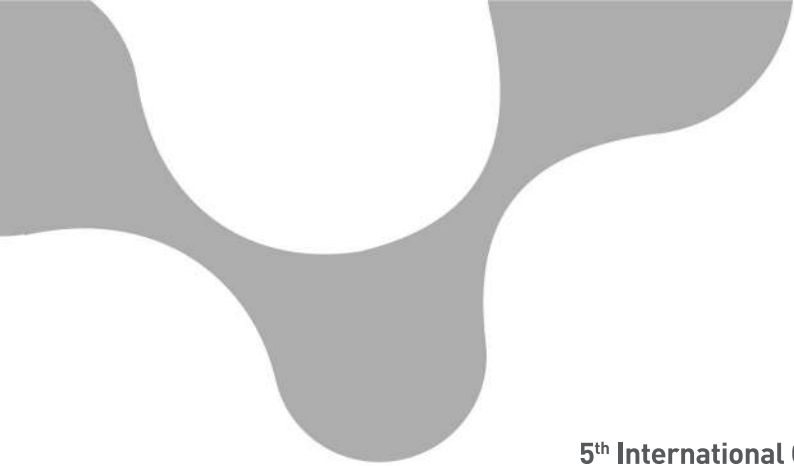
3. Choi, Y. J., I. Y. Choi, **W. Jang**, S.-M. Jeong, S. Park, K. Han, Y. Lee, D. H. Lee and D. W. Shin (2021). "Gastrectomy, vitamin B12 supplementation and the risk of Parkinson's disease: A nationwide cohort study." Parkinsonism & Related Disorders **83**: 15-21.
4. Yoo, J.E., **Jang, W.**, Shin, D.W., Jeong, S.-M., Jung, H.-W., Youn, J., Han, K. and Kim, B. (2020), Timed Up and Go Test and the Risk of Parkinson's Disease: A Nation-wide Retrospective Cohort Study. *Mov Disord*, 35: 1263-1267. <https://doi.org/10.1002/mds.28055>
5. Lee, S.-B., J. Youn, **W. Jang** and H. O. Yang (2019). "Neuroprotective effect of anodal transcranial direct current stimulation on 1-methyl-4-phenyl-1,2,3,6-tetrahydropyridine (MPTP)-induced neurotoxicity in mice through modulating mitochondrial dynamics." Neurochemistry International **129**: 104491.
6. Lee, S.-B., H.-T. Kim, H. O. Yang and **W. Jang** (2018). "Anodal transcranial direct current stimulation prevents methyl-4-phenyl-1,2,3,6-tetrahydropyridine (MPTP)-induced neurotoxicity by modulating autophagy in an in vivo mouse model of Parkinson's disease." Scientific Reports **8**(1): 15165.



ICKMDS 2025

5th International Conference of Korean Movement Disorder Society

**Bridging from the Origin to Clinical Practice:
A Beginning Has an Ending**



ICKMDS 2025

5th International Conference of Korean Movement Disorder Society

Bridging from the Origin to Clinical Practice:
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DAY 2_ November 1 (Sat), 2025

Room B

Oral Presentation Session 2

Chairs Yun Kyung KIM (Korea)
Jae-Hyeok LEE (Korea)



Presynaptic Serotonergic Autoreceptors as a Therapeutic Target for Levodopa-Induced Dyskinesia

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¹Pharmacology, Punjabi University, Patiala, India

²Medicine, All India Institute of Medical Sciences, Punjab, India

Background & Objective:

Levodopa-induced dyskinesia (LID) is a disabling complication of chronic dopaminergic therapy in Parkinson's disease (PD), driven in part by unregulated dopamine release from serotonergic terminals. Presynaptic 5-HT_{1A} autoreceptor activation reduces aberrant serotonin-driven dopamine release and may mitigate dyskinesia without compromising motor benefit. S-15535(1-(2,3-dihydro-1,4-benzodioxin-8-yl)-4-(2,3-dihydro-1H-inden-2-yl)piperazine), a high-affinity 5-HT_{1A} ligand, acts as a potent agonist at presynaptic auto receptors and a weak partial agonist/functional antagonist at postsynaptic sites, offering a unique pharmacological profile that could optimize antidyskinetic efficacy while limiting adverse serotonergic effects. The objective of this study is to evaluate the antidyskinetic potential of S-15535 in a mouse model of LID.

Methods:

Male albino mice were subjected to unilateral 6-hydroxydopamine (6-OHDA) lesions and received chronic levodopa (20 mg/kg/day, i.p.) for 6 weeks to induce stable dyskinesia, quantified via abnormal involuntary movement (AIM) scores. Once LID was established, S-15535 (1, 2, 4 mg/kg, i.p.) was administered 30 min before levodopa for 7 consecutive days. Motor function was assessed using rotarod and cylinder tests. Striatal serotonin, dopamine, and metabolites were measured using HPLC-ECD detector.

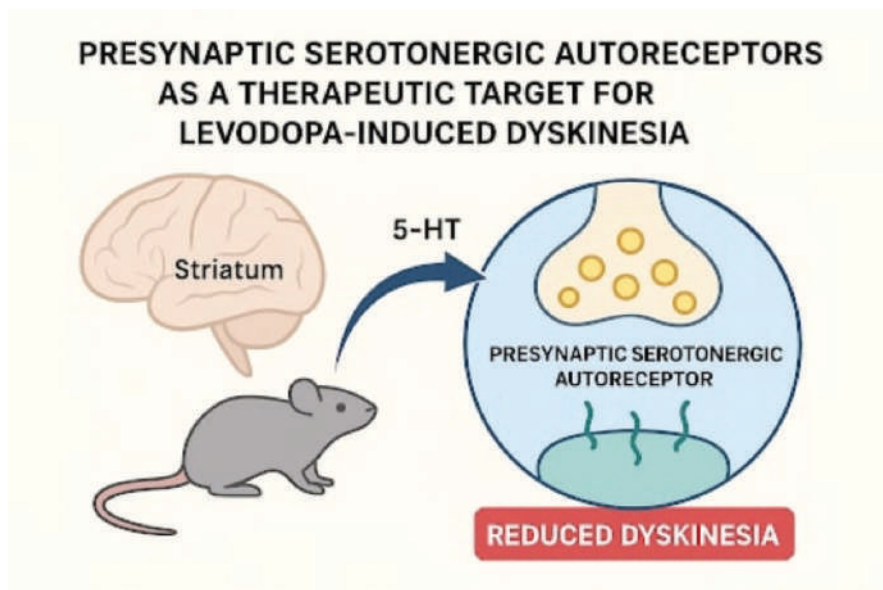
Results:

S-15535 produced a dose-dependent reduction in AIM scores without diminishing levodopa-induced motor improvements. Neurochemical assays showed reduced striatal serotonin overflow and stabilization of dopamine tone, consistent with presynaptic autoreceptor engagement.

Conclusions:

S-15535 represent the first demonstration of a presynaptic-selective 5-HT_{1A} agonist with dual receptor activity reducing LID in a PD model. These findings could open new avenues for adjunctive therapy targeting serotonergic autoreceptors to optimize levodopa efficacy while minimizing dyskinesia.

Figure



Keywords

5HT1A receptors, Neurochemistry, Parkinson's disease, LID

Personal Information

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Department **PHARMACEUTICAL SCIENCES AND DRUG RESEARCH**
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Country **INDIA**



Education / Training

Institution and Location	Degree	Year(s)	Field of Study
PANJAB UNIVERSITY	Ph.D	2004	Pharmacology
Panjab University	M.Pharm	1995	Pharmacology

Positions and Honors

Professional Experience

Current

2013-2025 Professor, Punjabi University, Patiala

Previous

2007-2013 Associate Professor Punjabi University Patiala

Honors

2023 Fellow of Indian Pharmacological Society
2024 Honored to deliver Prestigious Prof. S C Lahiri Oration during annual meeting of IPS at AIIMs New Delhi
2025 Selected as DHR_ International fellow to visit Neuropharmacology Lab., as visiting prof in Texas university Texas, USA.

Selected Peer Reviewed Publications

- Samriti, Kaur, Arvinder; Kaur, Arshbir; Goel, RK;
Ameliorative effect of diclofenac in rotenone corneal kindling model of drug-resistant epilepsy: Edge of dual COX and KMO inhibition
Brain Research 2025

- 2 Kaur, Arvinder; **Goel, Rajesh Kumar**;
Modelling Epilepsy Associated Alzheimer's Disease Through Mitochondrial Complex-I Inhibition: Neurochemical and Therapeutic Perspectives
Neurochemical Research 2025
3. Kaur, A., Kaur, A., & **Goel, R. K.** (2024). Attenuation of mitochondrial refractory epilepsy in rotenone corneal kindling model of drug resistance by idebenone: An approach to bypass mitochondrial complex I. *Epilepsy Research*, 107458. **Impact factor (2023): 2.2**
4. Kaur, A., Kaur, A., & **Goel, R. K.** (2024). Ameliorative effect of diclofenac in rotenone corneal kindling model of drug-resistant epilepsy: Edge of dual COX and KMO inhibition. *Brain Research*, 149246. **Impact factor (2023): 2.**
5. Kaur, A., Raji, Verma, V., & **Goel, R. K.** (2024). Strategic pathway analysis for dual management of epilepsy and comorbid depression: a systems biology perspective. *In Silico Pharmacology*, 12(1), 36. **Impact factor (2023): 4.0**
6. Kaur, B., Kumar, S., Kaur, A., & **Goel, R. K.** (2023). A network pharmacology approach to explore pharmacological mechanisms of *Asparagus racemosus* for ameliorative effect in epilepsy and comorbid progressive memory dysfunction. *In Silico Pharmacology*, 11(1), 29. **Impact factor (2023): 4.0**
7. Kaur, A., Kumar, S., & **Goel, R. K.** (2023). Adjunct antiseizure effect of clotrimazole in a rotenone corneal kindling mouse model of mitochondrial drug-resistant epilepsy. *Epilepsy Research*, 198, 107246. **Impact factor (2023): 2.2**
8. Goyal, H., Gupta, R. C., Pradhan, S. K., & **Goel, R.** (2024). Antimicrobial and Hepatoprotective Activity of Different Cytotypes of *Taraxacum officinale* from the Indian Himalayas. *Journal of Herbs, Spices & Medicinal Plants*, 30(1), 12-25. **Impact factor (2023): 1.2**
- 9 Dhillon HK, Singh T, **Goel RK.** Ferulic acid inhibits catamenial epilepsy through modulation of female hormones. *Metabolic Brain Disease*. 2022 Aug 6:1-2. **Impact factor (2021): 0.76**

Pathological role of immunoproteasome PSMB8 in Parkinson's disease: A link between α -synuclein aggregation and immune activation

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²Neurology, Hallym University Sacred Heart Hospital, Anyang, Republic of Korea

Background & Objective:

Parkinson's disease (PD) is marked by α -synuclein accumulation, neurodegeneration, and immune activation. The immunoproteasome (IP), especially PSMB8, regulates proteostasis and immunity, but its role in synucleinopathies is unclear.

Methods:

We examined 288 blood samples from PD, MSA, RBD, and controls for IP expression/activity in PBMCs and neuronal EVs, and assessed effects of IP inhibition using flow cytometry. PSMB8 localization was analyzed in post-mortem substantia nigra. PD mouse and human dopaminergic neuron models, along with transcriptomic profiling, were used to investigate IP dynamics and inhibition outcomes. CD8 T cell derived from people with PD was co-cultured to PD dopaminergic neurons to examine PSMB8 inhibition effect through intrinsic neuron mechanism and cytotoxicity of CD8 T cells.

Results:

PSMB8 mRNA and protein were consistently upregulated in PBMCs and neuronal EVs from people with PD and MSA, and across multiple PD models including rotenone exposure, SNCA-A53T mutation, α -synuclein overexpression, PFF treatment, and PFF-injected mice. In PD substantia nigra, PSMB8 was enriched in mature Lewy body cores. Pharmacological PSMB8 inhibition reduced α -synuclein accumulation, apoptosis in dopaminergic neurons, and percentage of CD8⁺ T cells in PD PBMCs. Notably, α -synuclein clearance was independent of proteasomal or autophagy-lysosomal pathways, instead correlating with non-proteasomal trypsin-like activity. In CD8⁺ T cell-dopaminergic neuron co-cultures, PSMB8 inhibition improved neuronal survival, lowered α -synuclein, and attenuated CD8⁺ T cell degranulation, linking neuronal α -synuclein burden to immune-mediated toxicity.

Conclusions:

PSMB8 is persistently upregulated in synucleinopathies and mediates both α -synuclein pathology and immune dysregulation. Targeting the immunoproteasome may offer therapeutic potential by reducing neuronal toxicity and aberrant immune responses in PD and related disorders.

Personal Information

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Department Department of Neurology
Organization Hallym University Sacred Heart Hospital
Country Republic of Korea



Education / Training

Institution and Location	Degree	Year(s)	Field of Study
Hue University College of Sciences	Engineer (Summa Cum Laude)	2021	Biotechnology (Human Physiology and Cell therapy)
Hallym University College of Medicine	MMSc	2023	Biomedical Science (Neuroscience)
Hallym University College of Medicine	PhD Candidate (Diss. sched.) in Medical Science	2025	Biomedical Science (Neuroscience)

Positions and Honors

Professional Experience

Current

2023 - Present Doctoral Researcher, Department of Neurology, Hallym University Sacred Heart Hospital, Korea

Previous

2021-2023 Research Assistant, Department of Neurology, Hallym University Sacred Heart Hospital, Korea

2018-2021 Research Assistant, Stem Cell Unit, Hematology – Transfusing Center, Hue Central Hospital, Vietnam

2019-2021 Researcher - Laboratory manager, Laboratory of Human Physiology, College of Sciences, Hue University, Vietnam

Honors

2022 Honda - YES Award (Japan)

2021 Odon Vallet Scholarship (Paris, France)

2021 Panasonic Scholarship (Japan)

2021	Third prize, the 11th Thua Thien Hue Technical Innovation Contest
2020	Consolation prize, Vietnam's National Scientific Research Contest
2019	Second prize, Stem Cell Innovation Contest
2017- 2021	Academic Encouragement Scholarships (ranked in the top 1% of students), awarded by Hue University of Sciences (4-time recipient)

Selected Peer Reviewed Publications

1. **Nguyen, H. D.**, Tran, H. B. T., Nguyen, T. T., Kwak, I. H., Kim, Y. J., Ma, H., Kim, H.-J., & Kim, Y. E.* (in press, 2025). Pathological role of immunoproteasome PSMB8 in Parkinson's disease: A link between α -synuclein aggregation and immune activation. *eBioMedicine*.
2. **Nguyen, H. D.**, Kim, Y. E*., Nguyen, L. T. N., Kwak, I. H., Lee, Y. K., Kim, Y. J., ... & Ma, H. I. (2023). Upregulation of immunoproteasome PSMB8 is associated with Parkinson's disease. *Parkinsonism & Related Disorders*, 114, 105797. DOI: 10.1016/j.parkreldis.2023.105797
3. Nguyen, L. T. N., **Nguyen, H. D.**, Kim, Y. J., Nguyen, T. T., Lai, T. T., Lee, Y. K., ... & Kim, Y. E*. (2022). Role of NLRP3 inflammasome in Parkinson's disease and therapeutic considerations. *Journal of Parkinson's Disease*, 1-17. DOI: 10.3233/JPD-223290
4. **Nguyen, H. D***, Thi, L. H., Ho, X. B., Cao, V. A., & Le Hoang, D. M. (2022). Effect of *Rehmannia glutinosa* Libosch extract on proliferation and cardiogenic pre-differentiation of human mesenchymal stem cells. *BioMedicine*, 12(1), 39. DOI: 10.37796/2211-8039.1243
5. Thi, C.* , **Nguyen, H. D** & Hoang, D. M. (2021). Influence of Piper betle L. extract on umbilical cord cells in vitro and potential treating cutaneous wound. *Heliyon*. 7: e06248. DOI: 10.1016/j.heliyon.2021.e06248
6. kook Yu, J., Choi, H. J., Kim, D., Ngoc, P. H., Kwak, I. H., **Nguyen, H. D.**, ... & Kim, Y. E*. (2025). Prevalence and clinical impact of alpha-synuclein pathology in idiopathic normal pressure hydrocephalus: Insights from RT-QulC assay. *Parkinsonism & Related Disorders*, 136, 107857. DOI: 10.1016/j.parkreldis.2025.107857
7. Lee, J., Kim, H. J., **Nguyen, H. D.**, Song, S. J., Thanh, T. N., Kwak, I. H., ... & Kim, Y. E*. Differential Peripheral NLRP3 Inflammasome Expression in Parkinson's Disease and Multiple System Atrophy. *Journal of movement disorders*. DOI: 10.14802/jmd.25124

Blood-based targeted proteomics of metabolic pathways in the isolated REM sleep behavior disorder–Lewy body disease continuum

Ryul KIM¹, Seohee CHOI¹, Heejeung KIM², Eun Jin YOON², Yu Kyeong KIM², Jee-Young LEE¹

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²Department of Nuclear Medicine, Seoul Metropolitan Government - Seoul National University Boramae Medical Center, Seoul, Republic of Korea

Background & Objective:

Metabolic dysfunction has been increasingly recognized as a contributor to neurodegeneration, but its role in Lewy body disease (LBD), particularly in prodromal stages, remains poorly understood. Identifying metabolism-related biomarkers in prodromal LBD may provide mechanistic insights and support early intervention. This study investigated blood-based targeted proteomic profiles related to metabolic pathways across the REM sleep behavior disorder (RBD)–LBD continuum and explore their potential as biomarkers linked to clinical severity and brain metabolic patterns.

Methods:

Plasma samples were obtained from 116 patients (47 isolated RBD [iRBD], 45 Parkinson's disease with probable RBD [PDRBD], 20 dementia with Lewy bodies with probable RBD) and 30 healthy controls. Clinical assessments included the MDS-UPDRS and comprehensive neuropsychological testing. 18F-fluorodeoxyglucose PET quantified PD-related brain metabolic patterns. Protein levels were measured with the Olink Target 96 Metabolism panel.

Results:

Sixteen proteins, mainly involved in dopamine biosynthesis, oxidative stress, and vesicle-mediated transport, were differentially expressed in the RBD–LBD continuum compared to controls. Changes were most prominent in clinically manifest LBD, with DOPA decarboxylase (DDC) showing the strongest signal. While DDC levels did not differ significantly between PD and DLB, both were higher than in iRBD. Notably, NT-proBNP was the only protein upregulated in iRBD versus controls. Within iRBD, higher NT-proBNP correlated with greater motor severity, poorer visuospatial performance, and higher PDRBD-related metabolic pattern z scores.

Conclusions:

Targeted proteomics revealed stage-specific metabolism-related protein changes in LBD. NT-proBNP may serve as a novel biomarker of prodromal neurodegeneration, associated with both clinical severity and brain metabolic alterations. Longitudinal validation is required to determine its predictive value for phenoconversion in iRBD.

Personal Information

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Current Position Clinical Assistant professor
Department Department of Neurology
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Country South Korea



Education / Training

Institution and Location	Degree	Year(s)	Field of Study
Seoul National University	PhD	2023	Translational Research
Seoul National University	MS	2020	Translational Research
Jeju National University	MD	2011	College of Medicine

Positions and Honors

Professional Experience

Current

2024- Clinical Assistant professor, Department of Neurology, Seoul Metropolitan Government - Seoul National University Boramae Medical Center

Previous

2022-2024 Assistant professor, Department of Neurology, Inha University Hospital
2020-2022 Clinical assistant professor, Department of Neurology, Inha University Hospital
2019-2020 Fellowship, Movement disorders division, Department of Neurology, Seoul National University Hospital
2017-2019 Flight surgeon, Department of Neurology, Aerospace Medical Center
2016-2017 Chief of Neurology, Aerospace Medical Group, Air Force Education and Training Command
2012-2016 Residency, Department of Neurology, Seoul National University Hospital
2011-2012 Internship, Seoul National University Hospital

Honors

2024 인지중재치료학회 학술상
2023 보건의료기술진흥 유공자 정부포상 보건복지부장관상 신진연구부문

2023	대한파킨슨병및이상운동질환학회 연구자상
2021	유당명호진선생기념사업회 유당학술상
2020	한국노년신경정신약물학회 젊은연구자상
2020	대한파킨슨병및이상운동질환학회 전임의상
2019	대한신경과학회 SK젊은연구자상

Selected Peer Reviewed Publications

1. Choi S, Kim R¶, Kwon S, Jun JS, Byun K, Kang N, Park K, Lee JY, Jeon B. Prevalence of muscle mass loss and its association with orthostatic hypotension and related symptoms in Parkinson's disease. *npj Parkinson's disease*. 2025;[Epub ahead of print].
2. Kim R, Jin B, Kim H, Woo KA, Yoon EJ, Kim S, Shin JH, Nam H, Kim YK, Jeon B, Lee JY. Cholinergic basal forebrain degeneration in isolated REM sleep behavior disorder. *Brain*. 2025;[Epub ahead of print].
3. Kim R*, Oh S*, Woo KA, Shin CM, Park KU, Lee JY. Blood microbiome signatures in the REM sleep behavior disorder–Lewy body disease continuum. *J Neural Transm (Vienna)*. 2025 Aug;132(8):1143-1154.
4. Kim R, Kang N, Byun K, Park K, Jun JS, Jeon B. Association of long-term glycemic variability with longitudinal motor and nonmotor progression in patients with Parkinson's disease: an 8-year follow-up. *Age Ageing*. 2025 Aug;54(9):afaf220.
5. Lee JH, Jun JS, Kang N¶, Kim R¶, Choi BJ, Byun K, Park K, Lee JY, Jeon B. Transcranial direct current stimulation combined with motor training for motor symptoms in Parkinson's disease: a systematic review and meta-analysis. *Ageing Research Reviews*. 2025 May;109:102781.

Drug-Target Mendelian Randomization Analysis of Dipeptidyl Peptidase-4 Inhibitors for Parkinson's Disease Prevention

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²Neurology, Ajou University School of Medicine, Suwon, Republic of Korea

³Graduate School of Public Health, Seoul National University, Seoul, Republic of Korea

Background & Objective:

Parkinson's disease (PD) lacks disease-modifying treatments due to insufficient mechanistic understanding. Drug-target Mendelian randomization (MR) leverages genetic variants affecting drug targets to assess therapeutic potential while minimizing confounding. Recent evidence suggests that dipeptidyl peptidase-4 (DPP4) inhibitors, a class of antidiabetic drugs, may have neuroprotective properties. We employed drug-target MR to investigate whether DPP4-Is could reduce PD risk, focusing on sex-specific effects.

Methods:

We conducted two-sample MR using instrumental variables (IVs) representing DPP4 inhibition derived from expression quantitative trait loci (IV_{eQTL}; eQTLGen consortium) and protein QTL (IV_{pQTL}; UK Biobank). PD associations were examined using the largest available genome-wide association study from the International Parkinson's Disease Genomics Consortium, including sex-stratified analyses. Primary analysis utilized inverse variance weighted MR, supplemented by sensitivity analyses, mediation analysis via diabetes, and colocalization assessment.

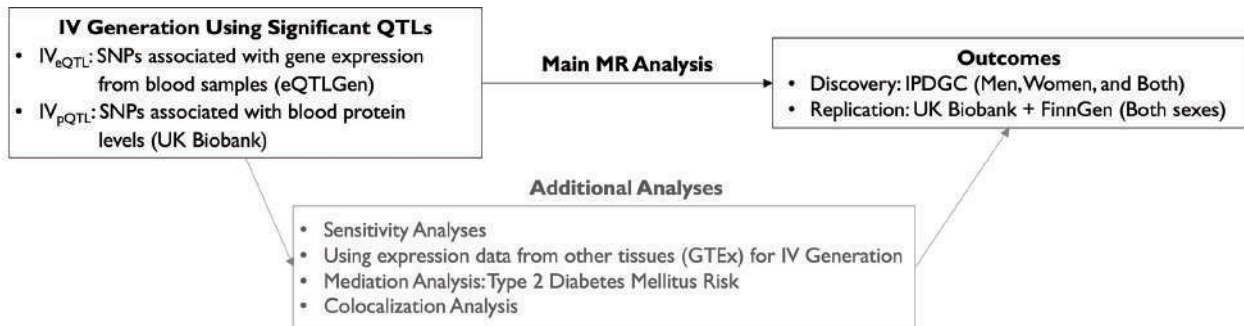
Results:

Genetically proxied DPP4 inhibition associated with decreased PD risk: OR (95% CI) = 1.78 (1.21-2.61) for IV_{eQTL} and 1.21 (1.02-1.45) for IV_{pQTL}. In sex-stratified analysis, the protective effects were more pronounced in males—IV_{eQTL}: 2.25 (1.55-3.28); IV_{pQTL}: 1.35 (1.15-1.57)—versus non-significant effects in females, indicating sex-specific mechanisms. Results remained robust across sensitivity analyses and replicated in independent PD GWAS. Diabetes did not mediate this association, and colocalization provided modest evidence for shared causal variants only in males.

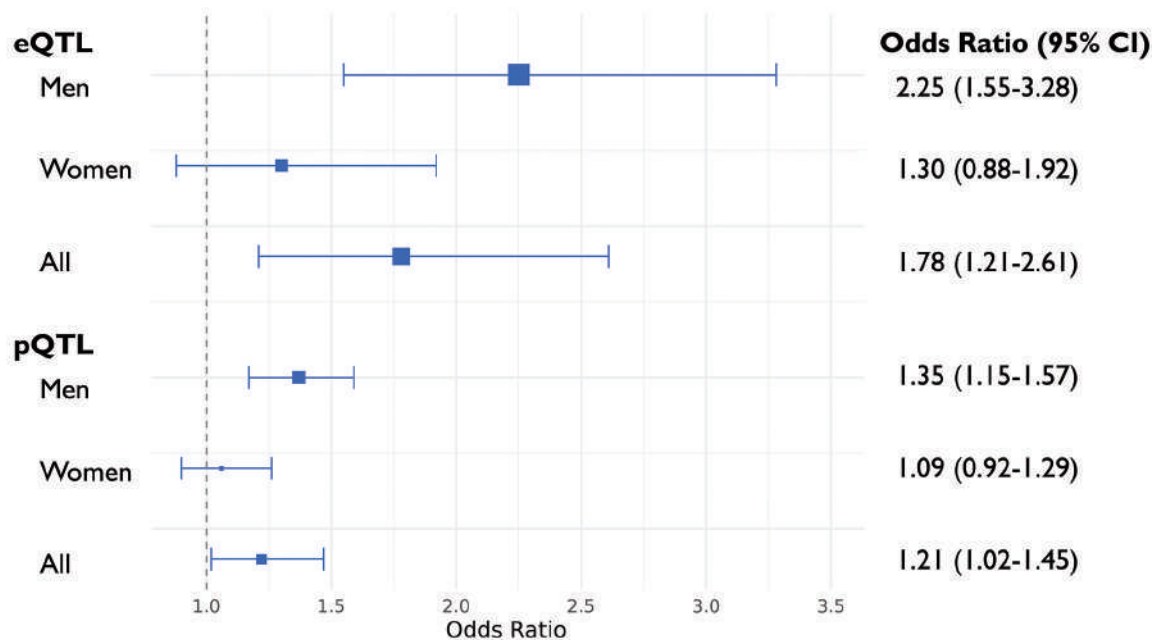
Conclusions:

This drug-target MR approach reveals that DPP4 inhibitors may offer protective effects against PD, particularly in males. The observed sex-specific effects underscore the importance of considering biological sex in therapeutic development, though these findings require careful interpretation given the inherent limitations of drug-target MR methodology.

Figure



Figure



Table

Table 1. MR Analysis of Genetically Proxied DPP4 Inhibitors on the Risk of Parkinson's Disease (PD)

IV	Method	Men (n=12,054 + 11,999)*			Women (n=7,384 + 12,389)*			All (N=37,688 + 981,372)*		
		No. of SNPs	OR (95% CI)	P-value	No. of SNPs	OR (95% CI)	P-value	No. of SNPs	OR (95% CI)	P-value
IV _{eQTL}	IVW		2.25 (1.55-3.28)	2.06E-05		1.3 (0.88-1.92)	0.195		1.78 (1.21-2.61)	0.003
	Weighted Median		2.09 (1.4-3.11)	0.003		1.1 (0.66-1.83)	0.713		1.82 (1.09-3.04)	0.022
	Weighted Mode	9	1.92 (1.28-2.89)	0.022	10	1.04 (0.58-1.86)	0.887	10	1.68 (0.91-3.11)	0.131
	MR Egger		1.6 (0.71-3.59)	0.14		1.37 (0.6-3.13)	0.483		1.27 (0.56-2.88)	0.586
	MR-PRESSO		2.07 (1.45-2.95)	0.001		1.3 (0.98-1.71)	0.101		1.78 (1.21-2.61)	0.016
IV _{pQTL}	IVW		1.36 (1.19-1.56)	5.56E-05		1.06 (0.9-1.26)	0.465		1.22 (1.02-1.47)	0.034
	Weighted Median		1.37 (1.14-1.65)	0.002		1.02 (0.81-1.28)	0.883		1.25 (0.98-1.58)	0.068
	Weighted Mode	33	1.39 (1.16-1.65)	0.003	33	1.06 (0.86-1.31)	0.573	39	1.26 (1.01-1.57)	0.051
	MR Egger		1.43 (1.12-1.83)	0.252		1.01 (0.72-1.42)	0.941		1.15 (0.78-1.69)	0.480
	MR-PRESSO		1.36 (1.2-1.54)	1.00E-04		1.06 (0.93-1.22)	0.382		1.22 (1.02-1.47)	0.040

* Numbers of cases and controls, respectively.

Abbreviations: MR, Mendelian randomization; DPP4, dipeptidyl peptidase; IV: instrumental variable, eQTL: expression quantitative trait loci, pQTL: protein QTL, OR: odds ratio, CI: confidence interval, IVW: inverse variance weighted.

Keywords

Parkinson's disease, DPP-4 inhibitor, Mendelian randomization, Drug repurposing, Genome Epidemiology

Personal Information

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Education / Training

Institution and Location	Degree	Year(s)	Field of Study
Seoul National University Graduate School of Public Health	PhD	2019	Genome Epidemiology
Seoul National University Graduate School of Public Health	MPH	2011	Genome Epidemiology
Sungkyunkwan University School of Pharmacy	BS	2009	Pharmacy

Positions and Honors

Professional Experience

Current

2019- Post-doc Researcher, Seoul National University Institute of Health and Environments

Previous

2016-2017 Researcher, Seoul National University Institute of Health and Environments

2011-2012 Medical Information Associate, Pfizer Pharmaceuticals Korea

Selected Peer Reviewed Publications

1. Sangwon Han, Jiyun Hwang, Tamina Park, Jung-Min Pyun, **Joo-Yeon Lee**,, Young Ho Park. Transcriptome analysis of early- and late-onset Alzheimer's disease in Korean cohorts. ALZHEIMERS & DEMENTIA. 2025

2. Jong Hyun Park, Kyu-Taek Lim, **Jooyeon Lee**, Yongjin Gil, Joohon Sung. The Diabetogenic Effect of Statin Use May Interact With Polygenic Risk Scores for Type 2 Diabetes: Evidence From the UK Biobank. Journal of Preventive Medicine and Public Health. 2025.

3. Shin In Ja, Kim Yoon Seob, **Joo-Yeon Lee**, Min Seung Kim, Jung Han Yoon, Don Gueu Park. Adult-onset YY1-associated combined dystonia syndrome with infantile nystagmus as a diagnostic clue. PARKINSONISM & RELATED DISORDERS. 2024.

4. Karri Silventoinen, ..., **Jooyeon Lee**, ..., Jaakko Kaprio. Smoking remains associated with education after controlling for social background and genetic factors in a study of 18 twin cohorts. *SCIENTIFIC REPORTS*. 2022
5. Martina Meszaros, ..., **Jooyeon Lee**, ..., Andras Bikov. Obstructive sleep apnea and hypertriglyceridaemia share common genetic background: Results of a twin study. *JOURNAL OF SLEEP RESEARCH*. 2020.
6. Soo Ji Lee, **Joo-Yeon Lee**, Joonhon Sung. Obesity and Bone Health Revisited: A Mendelian Randomization Study for Koreans. *JOURNAL OF BONE AND MINERAL RESEARCH*. 2019.
7. **Joo-Yeon Lee**, et al. BRCA1/2-negative, high-risk breast cancers (BRCAx) for Asian women: genetic susceptibility loci and their potential impacts. *SCIENTIFIC REPORTS*. 2018.

Phase 1/2a clinical trial of hESC-derived dopaminergic progenitors in Parkinson's disease

Han Kyu NA¹, Kyung Won CHANG², Chan Wook PARK^{1,3}, Do-Hun KIM^{3,4}, Sanghyun PARK³, Chul-Yong PARK^{3,4}, Jang Hyeon EOM⁴, Seung Taek NAM⁴, Ki-Sang JO⁴, Mi-Young JO⁴, Sung Kyoung CHOI⁴, Hye-Jin HUR⁴, Sarang KIM⁴, Minseok KIM⁵, Dae-Sung KIM⁶, Dong-Youn HWANG⁷, Myoung Soo KIM⁸, Inkyung JUNG⁵, Jongwan KIM⁴, Myung Soo CHO⁴, Jin Woo CHANG⁹, Phil Hyu LEE¹, Dong-Wook KIM^{3,4}

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⁴Biomedics, S. Biomedics Co., Ltd, Seoul, South Korea, Republic of Korea

⁵Biomedical Systems Informatics, Division of Biostatistics, Department of Biomedical Systems Informatics, Yonsei University College of Medicine, Seoul, South Korea, Republic of Korea

⁶Biotechnology, Department of Biotechnology, College of Life Sciences and Biotechnology, Korea University, Seoul, South Korea, Republic of Korea

⁷Biomedical Science, Department of Biomedical Science, CHA University, Gyeonggi-do, Republic of Korea

⁸Surgery, Department of Surgery, Yonsei University College of Medicine, Seoul, South Korea, Republic of Korea

⁹Neurosurgery, Department of Neurosurgery, Korea University Anam Hospital, Seoul, South Korea, Republic of Korea

Background & Objective:

To evaluate the safety and exploratory efficacy of intraputamenal engraftment of human embryonic stem cell (hESC)-derived dopaminergic progenitors (A9-DPCs) in patients with Parkinson's disease (PD).

While PD has long been considered as a potential candidate for cell replacement therapy, the application has been limited by inconsistent results, restricted tissue availability, and quality issues. Recent studies suggest that hESCs may provide a scalable source for generating dopaminergic (DA) progenitors with functional properties suitable for therapeutic application in PD.

Methods:

This single-center, open-label, dose-escalation Phase 1/2a trial evaluated the safety and exploratory efficacy of A9-DPC, an allogenic hESC-derived DA progenitor product. Twelve patients with moderate-to-severe PD were enrolled received bilateral transplantation into the putamen of either a low-dose (3.15 million cells, n=6) or a high-dose (6.3 million cells, n=6). The primary endpoint was safety and tolerability. Exploratory efficacy endpoints included changes in the MDS-UPDRS score, Hoehn and Yahr (H&Y) stage, and ¹⁸F-FP-CIT PET imaging at 12 months.

Results:

No dose-limiting toxicities or graft-related adverse events were observed in either group. At 12 months, compared with baseline, off-medication MDS-UPDRS III scores (low dose: -12.7 ± 8.2 ; high dose: -15.5 ± 3.6) and H&Y stage (low dose: -1.0 ± 0.6 ; high dose: -1.7 ± 0.5) improved. Motor function improved more in the high-dose group, consistent with a dose-dependent treatment effect. These clinical improvements were accompanied by increased ¹⁸F-FP-CIT uptake at the graft sites, supporting graft survival and functional DA activity.

Conclusions:

Intraputaminaal transplantation of A9-DPC was safe and demonstrated clinical benefit over 12 months, providing preliminary evidence that this approach may offer a promising therapeutic strategy for improving motor symptoms in PD (ClinicalTrials.gov number, NCT05887466).

Personal Information

Full Name Han Kyu NA
Current Position Fellowship Trainee
Department Neurology
Organization Yonsei University College of Medicine
Country South Korea



Education / Training

Institution and Location	Degree	Year(s)	Field of Study
Yonsei University, Korea	B.S.	2013	Chemical Engineering
Yonsei University, Korea	M.D.	2017	Medicine
Yonsei University, Korea	Ph.D.	2020-(Current)	Neurology

Positions and Honors

Professional Experience

Current

2022 - Fellowship trainee, Severance Hospital, Yonsei University College of Medicine

Previous

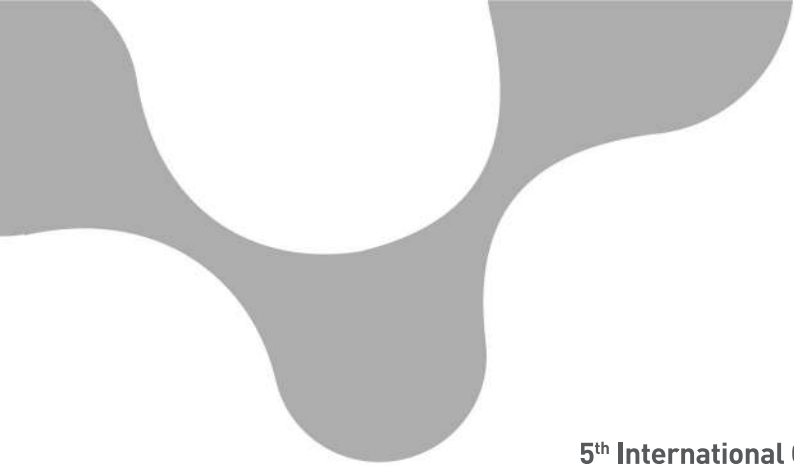
2018 – 2022 Residency, Severance Hospital, Yonsei University College of Medicine

2017 – 2018 Internship, Severance Hospital, Yonsei University College of Medicine

Selected Peer Reviewed Publications

1. Chang JW, Na HK, Chang KW, Park CW (*Co-first author*), Kim DH, Park S, Park CY, Eom JH, Nam ST, Jo KS, Jo MY, Choi SK, Hur HJ, Kim S, Kim M, Kim DS, Hwang DY, Kim MS, Jung I, Kim J, Cho MS, Lee PH, Kim DW. Phase 1/2a clinical trial of hESC-derived dopamine progenitors in Parkinson's disease. *Cell*. 2025;S0092-8674(25)01041-4
2. Na HK, Cho H, Lee HS, Kim HK, Yoon S, Lee JH, Ryu YH, Yoo HS, Lyoo CH. Neural basis of motor symptoms in Alzheimer's disease: role of regional tau burden and cognition. *Alzheimers Dement*. 2025;21(8):e70598
3. Cho S, Song S, Yum J, Kim EH, Roh YH, Kim WJ, Heo K, Na HK*, Kim KM* (*Co-corresponding author*). Enlarged perivascular space in the temporal lobe as a prognostic marker in temporal lobe epilepsy with hippocampal sclerosis. *Epilepsia*. 2025;66(5):1665-1676

4. Na HK, Shin JH, Kim SW, Seo S, Kim WR, Kang JM, Lee SY, Cho J, Byun J, Okamura N, Seong JK, Young Noh. Diverging Relationships among Amyloid, Tau, and Brain Atrophy in Early-Onset and Late-Onset Alzheimer's Disease. *Yonsei Med J* 2024;65(8):434-447
5. Kim S, Na HK (Co-first author), Sun Y, Yoon YJ, Chung SJ, Sohn YH, Lyoo CH, Lee PH. Regional Burden of Enlarged Perivascular Spaces and Cognition and Neuropsychiatric Symptoms in Drug-Naive Patients With Parkinson Disease. *Neurology*. 2024 25;102(12):e209483.
6. Na HK, Kim HK, Lee HS, Park M, Lee JH, Ryu YH, Cho H, Lyoo CH. Role of Enlarged Perivascular Space in the Temporal Lobe in Cerebral Amyloidosis. *Ann Neurol*. 2023; 93(5):965-978
7. Jung JH, Na HK (Co-first author), Jeong SH, Chung SJ, Yoo HS, Lee YH, Baik K, Kim SJ, Sohn YH, Lee PH. Effects of Dihydropyridines on the Motor and Cognitive Outcomes of Patients with Parkinson's Disease. *Mov Disord*. 2023;38(5):843-853



ICKMDS 2025

5th International Conference of Korean Movement Disorder Society

Bridging from the Origin to Clinical Practice:
A Beginning Has an Ending

DAY 2_ November 1 (Sat), 2025

Room B

Parallel Session 8

Palliative Care in Parkinson's Disease:
Balancing Treatment and Support

Chairs Yih-Ru Wu (Taiwan)
Seongmin CHOI (Korea)



Current Status of Neuropalliative Care for Parkinsonism: For Successful Integration in Korea

Sooyeoun YOU

Department of Neurology, Seoul Medical Center, Republic of Korea

Neuropalliative care (NPC) has become an important component in the management of Parkinson's disease (PD) and related parkinsonian disorders. Traditionally linked to end-of-life settings, NPC is now understood as an early, integrated approach that addresses both motor and non-motor symptoms. These include tremor, rigidity, bradykinesia, pain, psychiatric and autonomic disturbances, sleep problems, communication difficulties, and progressive functional decline. Early application of NPC improves quality of life, enhances symptom control, facilitates advance care planning, and reduces caregiver burden. Effective delivery requires collaboration among neurologists, palliative specialists, rehabilitation professionals, and psychosocial support teams.

The long disease course of PD, often exceeding 10–20 years, highlights the need for supportive care elements in addition to traditional palliative interventions. Supportive care includes ongoing psychological support, caregiver education, social resource navigation, and measures to preserve autonomy and dignity. These dimensions ensure continuity of care throughout the chronic trajectory of PD.

Internationally, NPC models are being implemented in movement disorder clinics, with benefits for patient satisfaction and caregiver outcomes. However, adoption remains variable due to healthcare infrastructure, workforce training, reimbursement, and cultural views. In Korea, awareness is growing, but barriers include fragmented services, insufficient specialist education, and the lack of standardized pathways. The government-led insurance system, while equitable, creates rigid regulations that slow the adoption of new care models. Moreover, cultural traditions such as *hyo* (filial piety) and family-centered decision-making influence how advance care planning is approached.

Integration of NPC in Korea will require multidisciplinary training, coordinated care networks, culturally adapted guidelines, and reimbursement reform. Combining palliative and supportive elements is essential to achieve patient-centered care and improve outcomes for both patients and caregivers.

Personal Information

Full Name Sooyeoun YOU
Current Position Neurologist (Movement Disorders Specialist)
Department Neurology
Organization Seoul Medical Center
Country Republic of Korea



Education / Training

Institution and Location	Degree	Year(s)	Field of Study
School of Medicine, Ewha Womans University, Seoul, South Korea	Graduate	2005	Medicine
Graduate School of Medicine, Ewha Womans University	Master's degree	2009	Neurology
Graduate School of Medicine, Ewha Womans University	Ph.D.	2020	Neurology

Positions and Honors

Professional Experience

Current

2025- Neurologist, Seoul Medical Center, Department of Neurology

Previous

2010-2012 Clinical Fellow, Asan Medical Center, Department of Neurology, Seoul, South Korea

2012-2013 Research Fellow, Dongsan Hospital, Department of Neurology, Keimyung University School of Medicine, Daegu, South Korea

2013-2020 Assistant Professor, Dongsan Hospital, Department of Neurology, Keimyung University School of Medicine, Daegu, South Korea

2020-2025 Associate Professor, Dongsan Hospital, Department of Neurology, Keimyung University School of Medicine, Daegu, South Korea

Selected Peer Reviewed Publications

1. Jinse Park, Eungseok Oh, Seong-Beom Koh, In-Uk Song, Tae-Beom Ahn, Sang Jin Kim, Sang-Myung Cheon, Yoon-Joong Kim, Jin Whan Cho, Hyeo-Il Ma, Mee Young Park, Jong Sam Baik, Phil Hyu Lee, Sun Ju Chung, Jong-Min Kim, Han-Joon Kim, Young-Hee Sung, Do Young Kwon, Jae-Hyeok Lee, Jee-Young Lee, Ji Seon Kim, Ji Young Yun, Hee Jin Kim, Jin Yong Hong, Mi-Jung Kim, Jinyoung Youn 9, Hui-Jun Yang 26, Won Tae Yoon, **Sooyeoun You**, Kyum-Yil Kwon, Su-Yun Lee, Younsoo Kim, Hee-Tae Kim, Joong-Seok Kim, Ji-Young Kim, Evaluating the Validity and Reliability of the Korean Version of the Scales for Outcomes in Parkinson's Disease-Cognition. *J Mov Disord*. 2024 Jul;17(3):328-332.
2. Park KW, Choi N, Oh E, Lyoo CH, Baek MS, Kim HJ, Yoo D, Lee JY, Choi JH, Lee JH, Koh SB, Sung YH, Cho JW, Yang HJ, Park J, Shin HW, Ahn TB, Ryu HS, **You S**, Choi SM, Kim BJ, Lee SH, Chung SJ. Movement Disorders Associated With Cerebral Artery Stenosis: A Nationwide Study. *Front Neurol*. 2022 Jul 14;13:939823.
3. **You S**, Hong JH, Yoo J. Analysis of pupillometer results according to disease stage in patients with Parkinson's disease. *Sci Rep*. 2021 Sep 9;11(1):17880
4. Ryu HS, Kim HJ, **You S**, Kim MJ, Kim YJ, Kim J, Kim K, Lee SA, Chung SJ. Nocturnal stridor in multiple system atrophy: Video-polysomnography and clinical features. *Parkinsonism Relat Disord*. 2021 Aug;89:48-53.
5. **You S**, Kim HA, Lee H. Association of Postural Instability with Autonomic Dysfunction in Early Parkinson's Disease. *J Clin Med*. 2020 Nov 23;9(11):3786.
6. Kim KT, **You S**. Against COVID-19: Inspired by Greek Myth. *Occup Med (Lond)*. 2020 Dec 12;70(8):612-613.
7. Choi JH, Lee JY, Cho JW, Ko SB, Ahn TB, Kim SJ, Cheon SM, Kim JS, Kim YJ, Ma HI, Baik JS, Lee PH, Chung SJ, Kim JM, Song IU, Kim HJ, Sung YH, Kwon DY, Lee JH, Kim JY, Kim JS, Yun JY, Kim HJ, Hong JY, Kim MJ, Youn J, Kim JS, Oh ES, Yang HJ, Yoon WT, **You S**, Kwon KY, Park HE, Lee SY, Kim Y, Kim HT, Park MY. Validation of the Korean Version of the Questionnaire for Impulsive-Compulsive Disorders in Parkinson's Disease Rating Scale. *J Clin Neurol*. 2020 Apr;16(2):245-253.

S8-2

Exploring Unmet Needs in Neuropalliative-Supportive Care for Parkinson's Disease: Perspectives from Patients, Caregivers, and Physicians in Korea

Chan Young LEE

Department of Neurology, Ewha Womans University Mokdong Hospital, Republic of Korea

Parkinsonism, especially Parkinson's disease (PD), is a progressive neurodegenerative disorder whose prevalence continues to rise globally with population aging. In addition to the cardinal motor manifestations, patients experience a wide range of non-motor symptoms that exert a profound negative impact on quality of life and substantially contribute to caregiver burden. Palliative care, originally developed in oncology, has expanded into chronic neurological conditions. However, recent international surveys, such as one conducted among Movement Disorder Society (MDS) members in 2021, illustrate both the growing recognition of palliative care and persistent barriers to implementation in movement disorders. Although 89% of respondents reported access to multidisciplinary teams, major barriers included an inadequate workforce (62%), the oncology-centric model of palliative care (56%), and limited familiarity with neurological conditions among palliative specialists. Only 12% of respondents had formal training in palliative care, reflecting both opportunities and challenges for global implementation. These findings provide the international background for our Korean study.

To assess perceptions in the Korean context, a multicenter cross-sectional survey was conducted with 112 patients, 110 caregivers, and 61 physicians. Structured questionnaires assessed participants' prior awareness of palliative care and their attitudes toward neuropalliative approaches. Caregiver burden was evaluated using the validated Korean version of the Parkinson's Disease Questionnaire-Carer (PDQ-Carer), which was specifically developed to assess the multidimensional burden of caregivers of patients with parkinsonism.

Awareness of palliative care varied markedly among groups: only 31.3% of patients and 27.5% of caregivers were aware of the concept, compared with 90.2% of physicians, highlighting a marked awareness gap. Despite this awareness gap, attitudes towards neuropalliative care were favorable. Caregivers were stratified into two groups based on the mean PDQ-Carer score: a low-burden group and a high-burden group. Patients in the high-burden group had longer disease duration, poorer cognitive function, more advanced Hoehn and Yahr stages, and higher levodopa equivalent daily doses (LEDD). In addition, caregiving duration was longer among caregivers with higher burden. To identify determinants of caregiver burden, linear regression analysis was conducted. Caregiver demographics (age, sex, education level) were not significantly associated with PDQ-Carer scores. In contrast, caregiving duration, patient cognitive decline, Hoehn and Yahr stage, LEDD, and disease duration were correlated with caregiver burden. However, in multivariable analysis, only patient disease duration remained a significant determinant ($B=2.048$, 95% CI 0.083-4.012, $p=0.041$). These findings highlight that caregiver stress is driven primarily by disease-related and contextual factors rather than demographic characteristics.

Beyond traditional surveys, innovative approaches are needed to address unmet needs. Our Special Interest Group (SIG) developed a study to explore the role of generative artificial intelligence (AI) in supporting patient counseling. A set of 100 commonly asked patient and caregiver questions was compiled and entered into leading generative AI platforms. Responses were evaluated by neurologist with expertise in Parkinsonism. This study, demonstrated both the promise and limitations of AI tools. While they may supplement patient education and support, ongoing expert oversight remains essential to ensure accuracy and safety.

In conclusion, neuropalliative-supportive care in Parkinsonism is urgently needed but remains under-implemented. Both international and Korean data reveal an substantial awareness gap between physicians and patients/ caregivers, while caregiver burden is primarily influenced by disease progression, particularly disease duration. Importantly, although palliative care has historically been oncology-based, our findings emphasize that neuropalliative care should be introduced much earlier in the disease course, rather than being reserved solely for the terminal stage. By integrating structured education, systematic counseling, and innovative tools such as AI, neuropalliative-supportive care can evolve into a proactive model that addresses unmet needs from the time of diagnosis, ultimately improving quality of life for patients and their caregivers.

Personal Information

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Current Position Clinical Assistant Professor
Department Neurology
Organization Ewha Womans University Mokdong Hospital
Country Korea



Education / Training

Institution and Location	Degree	Year(s)	Field of Study
College of Medicine, Ewha Womans University	M.D.	2011-2015	Medicine
Ewha Womans University Mokdong Hospital	Internship	2015-2016	Medicine
Ewha Womans University Mokdong Hospital	Residency	2016-2020	Neurology
Seoul National University Hospital	Fellowship	2020-2022	Movement Disorder

Positions and Honors

Professional Experience

Current

2023 Clinical Assistant Professor

Selected Peer Reviewed Publications

1. Lee CY, Ro JS, Jung H, Kim M, Jeon B, Lee JY. Increased 10-Year Prevalence of Huntington's Disease in South Korea: An Analysis of Medical Expenditure Through the National Healthcare System. J Clin Neurol. 2023 Mar;19(2):147-155. doi: 10.3988/jcn.2022.0212. Epub 2023 Jan 17. PMID: 36700478; PMCID: PMC9982180.
2. Lee CY, Kim H, Kim HJ, Shin JH, Chang HJ, Woo KA, Jung KY, Kwon O, Jeon B. Diet quality and prodromal Parkinson's disease probability in isolated REM sleep behavior disorder. Parkinsonism Relat Disord. 2023 Sep;114:105794. doi: 10.1016/j.parkreldis.2023.105794. Epub 2023 Aug 3. PMID: 37549588.
3. Lee CY, Shin C, Hwang YS, Oh E, Kim M, Kim HS, Chung SJ, Sung YH, Yoon WT, Cho JW, Lee JH, Kim HJ, Chang HJ, Jeon B, Woo KA, Koh SB, Kwon KY, Moon J, Kim YE, Lee JY. Caregiver Burden of Patients With Huntington's Disease in South Korea. J Mov Disord. 2024 Jan;17(1):30-37. doi: 10.14802/jmd.23134. Epub 2023 Sep 11. PMID: 37691330; PMCID: PMC10846961.
4. Lee SH, Lee CY, Park HS, Park J, Yun JY. Anti-N-Methyl-D-Aspartate Receptor (NMDAR) Encephalitis Associated With Mediastinal and Ovarian Teratomas: A Case Report. J Korean Med Sci. 2023 Feb 13;38(6):e31. doi: 10.3346/jkms.2023.38.e31. PMID: 36786082; PMCID: PMC9925329.

5. Shin JH, Yu R, Ong JN, **Lee CY**, Jeon SH, Park H, Kim HJ, Lee J, Jeon B. Quantitative Gait Analysis Using a Pose-Estimation Algorithm with a Single 2D-Video of Parkinson's Disease Patients. *J Parkinsons Dis.* 2021;11(3):1271-1283. doi: 10.3233/JPD-212544. PMID: 33935106.
6. Kim R, Kim HJ, Shin JH, **Lee CY**, Jeon SH, Jeon B. Serum Inflammatory Markers and Progression of Nonmotor Symptoms in Early Parkinson's Disease. *Mov Disord.* 2022 Jul;37(7):1535-1541. doi: 10.1002/mds.29056. Epub 2022 May 21. PMID: 35596676.
7. Park S, Shin JH, Jeon SH, **Lee CY**, Kim HJ, Jeon B. Investigation of the Long-Term Effects of Amantadine Use in Parkinson's Disease. *J Mov Disord.* 2023 May;16(2):224-226. doi: 10.14802/jmd.23037. Epub 2023 May 24. PMID: 37258283; PMCID: PMC10236022.

Home-Based Care for Advanced Parkinsonism

Sun Young LEE

Public Healthcare Center, Seoul National University Hospital, Republic of Korea

With an aging population and advancements in medical technology, the number of people living with mobility impairments from neurodegenerative diseases, such as Parkinson's disease, is increasing. Patients with advanced neurodegenerative diseases spend a significant portion of their illness trajectory at home, facing numerous challenges including high symptom burden, functional decline, and limited access to timely medical service. These difficulties can lead to increased suffering for patients and their caregivers, as well as increased preventable hospital visits due to condition exacerbation.

To address the growing population of patients with mobility impairments, various home-based medical care (HBMC) models are emerging. In South Korea, a Home-Based Primary Care pilot program for patients with chronic conditions treating in primary care clinic as launched in 2018, followed by the Long-Term Care Home Medical Center pilot program in 2023. However, services specifically for advanced neurodegenerative diseases remain insufficient.

Patients with advanced neurodegenerative diseases require a HBMC model that can provide not only appropriate symptom management but also timely advance care planning for patients and caregivers as the disease progresses. A targeted HBMC model for patients with advanced neurodegenerative diseases should aim to provide individualized care through close collaboration with a medical team, enhance continuity of care, reduce avoidable hospital utilization, and improve the experience of both patients and caregivers.

In this lecture, based on our experience with a hospital-based HBMC model at a tertiary hospital in Korea, we aim to explore an HBMC model that can provide appropriate neuropalliative care for patients with advanced neurodegenerative diseases.

Personal Information

Full Name Sun Young LEE
Current Position Clinical Assistant Professor
Department Public Healthcare Center
Organization Seoul National University Hospital
Country Korea



Education / Training

Institution and Location	Degree	Year(s)	Field of Study
Seoul National University College of Medicine, Seoul, South Korea	B.A	2006-2012	Medicine
Seoul National University College of Medicine, Seoul, South Korea	M.A	2015-2017	Medicine: Health policy and management
Seoul National University College of Medicine, Seoul, South Korea	Ph.D	2017-2020	Medicine: Health policy and management

Positions and Honors

Professional Experience

Current

2020~ Clinical assistant professor, Public healthcare center, Seoul National University Hospital
2023~ Clinical assistant professor, Department of Human System Medicine, Seoul National University College of Medicine

Previous

2018~2020 Clinical assistant professor, Department of Emergency Medicine, Seoul National University Hospital
2017~2018 Fellow, Department of Emergency Medicine, Seoul National University Hospital
2013~2017 Resident, Department of Emergency Medicine, Seoul National University Hospital

Honors

2024 Commendation from the Director of the Korea Disease Control and Prevention Agency
2020 Commendation from the Minister of Public Administration and Security

Selected Peer Reviewed Publications

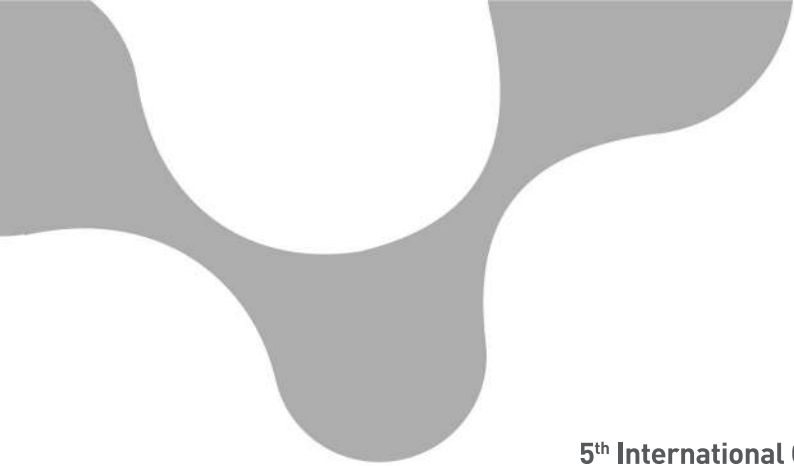
1. Min Sun Kim, Shin Hye Yoo, Kyae Hyung Kim, Belong Cho, **Sun Young Lee**, Telemedicine Experiences of People Living with Amyotrophic Lateral Sclerosis at Home in South Korea, *Yonsei Medical Journal*, 2025
2. **Sun Young Lee**, Shin Hye Yoo, Belong Cho, Kye Hyung Kim, Min Seoul Jang, Jeongmi Shin, Inyoung Hwang, Seok-Jin Choi, Jung-Joon Sung, Min Sun Kim, Burden and preparedness of care partners of people living with amyotrophic lateral sclerosis at home in Korea: a care partner survey, *Muscle&Nerve*, 2024
3. **Sun Young Lee**, Hyein Chu, Yu-Mi Kim, Characteristics of Homebound and Patients with Disability Who Use Home-Based Primary Care in Korea: A Retrospective Study for 2018–2022, *Journal of Clinical Medicine*, 2024
4. Min Seol Jang, Shin Hye Yoo, Min Sun Kim, Belong Cho, Kyae Hyung Kim, Jeongmi Shin, Inyoung Hwang, Seok-Jin Choi, Jung-Joon Sung, **Sun Young Lee**, Healthcare utilization and timing of supportive care equipment application of patients with Amyotrophic lateral sclerosis in Korea: Single center retrospective cohort study, *Journal of Clinical Neurology*, 2024
5. Jung-In Ko, Seok-Jin Choi, Shin Hye Yoo, Belong Cho, Min Sun Kim, Kyae Hyung Kim, **Sun Young Lee**, Epidemiology and characteristics of emergency department utilization by patients with amyotrophic lateral sclerosis in Korea from 2016 to 2020: A nationwide study, *Muscle&Nerve*, 2023
6. In Young Hwang, Yohan Han, Min Sun Kim, Kyae Hyung Kim, Belong Cho, Wonho Choi, Yejin Kim, Shin Hye Yoo, Sun Young Lee, Preferred versus actual place of care and factors associated with home discharge among Korean patients with advanced cancer: a retrospective cohort study, *Healthcare*, 2023
7. Yo Han Han MD, MS, Shin Hye Yoo, **Sun Young Lee**, In Young Hwang, Kyae Hyung Kim, Belong Cho, Min Sun Kim, Wonho Choi, Yejin Kim, The Medical Needs and Characteristics of Cancer and Progressive Neurologic Disease Patients Who Use Home-Based Medical Care in Korea: A Retrospective Study for 2011-2020, *Journal of the American Medical Directors Association*, 2022



ICKMDS 2025

5th International Conference of Korean Movement Disorder Society

**Bridging from the Origin to Clinical Practice:
A Beginning Has an Ending**



ICKMDS 2025

5th International Conference of Korean Movement Disorder Society

Bridging from the Origin to Clinical Practice:
A Beginning Has an Ending

DAY 2_ November 1 (Sat), 2025

Room B

Parallel Session 10

Tremor: From Pathophysiology to Clinical Practice

Chairs Pramod PAL (India)
Hyeo-il MA (Korea)



Pathophysiology in Essential Tremor : Lessons From Animal Study

Ming-Kai PAN

Department of Medical Research, National Taiwan University Hospital, Taiwan

Essential tremor (ET) is a disease entity with various etiologies. Despite the complexity of pathogenesis, all ET patients share a common symptom: an action tremor, defined by involuntary rhythmic movement. Specifically, tremor can be mathematically described by two features, tremor amplitudes and tremor frequencies. In this talk, we will apply state-of-the-art neural dynamic approaches in awake behaving mice to illustrate how tremor frequencies and amplitudes are ensembled in the brain. We will further describe how different patterns of neuronal synchrony can affect the circuitry dynamics of tremor, therefore leading to differential responses to ET medications. Finally, we will come back to patients and show how these frequency and amplitude coding mechanisms can facilitate novel drug design and therapies in ET.

Personal Information

Full Name Ming-Kai PAN
Current Position Associate Professor
Department Institute of Pharmacology / Department of Medical Research
Organization National Taiwan University College of Medicine/
National Taiwan University Hospital
Country Taiwan



Education / Training

Institution and Location	Degree	Year(s)	Field of Study
National Taiwan University College of Medicine (NTUMC), Taipei, Taiwan	MD	2004	Medicine
Institute of Physiology, NTUMC	PhD	2014	Neuroscience
National Taiwan University Hospital, Taiwan	Residency	2004-2008	Neurology
National Taiwan University Hospital, Taiwan	Fellowship	2008-2010	Clinical electrophysiology
Human motor control section/NINDS/NIH, USA	Fellowship	2008 (intramural)/2009-2010 (extramural)	Clinical electrophysiology

Positions and Honors

Professional Experience

Current

2022~ Associate Professor, Institute of Pharmacology, College of Medicine, National Taiwan University, Taipei, Taiwan
2021~ Attending Physician, Department of Medical Research, National Taiwan University Hospital, Taipei, Taiwan
2024~ Deputy Director, Molecular Imaging Center, National Taiwan University
2024~ Steering committee, Tremor study group, MDS
2024~ Executive Committee, Society for Research on the Cerebellum and Ataxias (SRCA)
2021~ Joint Faculty, Institute of Biomedical Sciences, Academia Sinica, Taipei, Taiwan

Previous

2011~2019 Attending Physician, National Taiwan University Hospital, Taipei, Taiwan Department of Neurology :
2011~2015
Department of Medical Research: 2015~2019,

Honors

2024 Wu Ho-Su TBF Medical Award, Taiwan Bio-developmental Foundation
2024 Outstanding Research Award, National Science and Technology Council
2024 Medical Research Award, The New Century Health Care Promotion Foundation, NCHCPF
2022 Chen-Yuan Lee Medical Memorial Award
2022 International Excellent Young Researcher Project, Ministry of Science and Technology, Taiwan
2020 National Innovation Award
2020 Young Scholar Innovation Award, Foundation of the Advancement of Outstanding Scholarship
2020 Young Scholar Innovation Award, Tien Te Lee Biomedical Foundation
2018 Junior Faculty Award, MDS-AOS SYNERGIES 2018
2018 Awardee, LEAP program, International Parkinson and Movement Disorder Society

Selected Peer Reviewed Publications

1. Chia-Wei Liu, Yi-Mei Wang, Shun-Ying Chen, Liang-Yin Lu, Ting-Yu Liang, Ke-Chu Fang, Peng Chen, I-Chen Lee, Wen-Chuan Liu, Ami Kumar, Sheng-Han Kuo, Jye-Chang Lee, Chung-Chuan Lo, Shun-Chi Wu, **Ming-Kai Pan***. The cerebellum shapes motions by encoding motor frequencies with precision and cross-individual uniformity. *Nature Biomedical Engineering*, 2025 May 27. DOI: 10.1038/s41551-025-01409-5. (IF: 27.7)
2. Chih-Chun Lin, Ke-Chu Fang, Ilaria Balbo, Ting-Yu Liang, Chia-Wei Liu, Wen-Chuan Liu, Yi-Mei Wang, Yen-Ling Hung, Kai-Chien Yang, Scott Kun Geng, Chun-Lun Ni, Christopher P. Driscoll, David S. Ruff, Ami Kumar, Nadia Amokrane, Natasha Desai, Phyllis L. Faust, Elan D. Louis, Sheng-Han Kuo*, **Ming-Kai Pan***. Reduced cerebellar rhythm by climbing fiber denervation is linked to motor rhythm deficits in mice and ataxia severity in patients. *Science Translational Medicine*. 2025 Feb 26;17(787):eadk3922. (Featured article) (IF: 15.8)
3. **Ming-Kai Pan***. Targeting the fundamentals for tremors: the frequency and amplitude coding in essential tremor. *Journal of Biomedical Science*. 2025 Feb 10;32(1):18 (IF: 9.0)
4. Yi-Mei Wang, Chia-Wei Liu, Shun-Ying Chen, Liang-Yin Lu, Wen-Chuan Liu, Jia-Huei Wang, Chun-Lun Ni, Shi-Bing Wong, Ami Kumar, Jye-Chang Lee, Sheng-Han Kuo, Shun-Chi Wu, **Ming-Kai Pan***. Neuronal population activity in the olivocerebellum encodes the frequency of essential tremor in mice and patients. *Science Translational Medicine*. 2024 May 15;16(747):ead11408 (IF: 15.8)
5. Yin-Tzu Hsieh, Kai-Chun Jhan, Jye-Chang Lee, Guan-Jie Huang, Chang-Ling Chung, Wun-Ci Chen, Ting-Chen Chang, Bi-Chang Chen, **Ming-Kai Pan***, Shun-Chi Wu*, Shi-Wei Chu* (Co-corresponding) TAG-SPARK: Empowering High-Speed Volumetric Imaging with Deep Learning and Spatial Redundancy. *Advanced Science* 2024 Nov; 11(41):e2405293. (IF: 14.3)

6. Chun-Lun Ni, Yi-Ting Lin, Liang-Yin Lu, Jia-Huei Wang, Wen-Chuan Liu, Sheng-Han Kuo, Ming-Kai Pan* Tracking motion kinematics and tremor with intrinsic oscillatory property of instrumental mechanics. *Bioengineering & Translational Medicine*. 2022. Oct 22;8(2):e10432, eCollection 2023 Mar
7. Pan M-K*, Li Y-S, Wang S-B, Ni C-L, Wang Y-M, Liu W-C, Lu L-Y, Lee J-C, Cortes EP, Vonsattel J-P, Sun Q, Louis E, Faust P, Kuo S-H*. Cerebellar oscillations driven by synaptic pruning deficits of cerebellar climbing fibers contribute to tremor pathophysiology. *Science Translational Medicine*. 2020 Jan 15;12(526):eeey1769 (Featured article) (IF: 17.956)

Pathophysiology of Dystonic Tremor: Lessons from Imaging and Stimulation Studies

Pattamon PANYAKAEW

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Currently, the MDS Tremor Expert Panels have categorized the movements accompanying dystonia into two types: (1) regular, sinusoidal movements consistent with "tremors in dystonia", and (2) irregular, jerky, non-oscillatory movements, known as "jerky dystonia". Tremor in dystonia is a regular oscillatory movement that occurs in either dystonic or non-dystonic body parts. The term "dystonic tremor", characterized by irregular tremor in the dystonic body regions, and "tremor associated with dystonia", where tremor exhibits in the non-dystonic part, have been discouraged due to a lack of clarity. Jerky dystonia differs from tremor in dystonia, as it is characterized by slow jerks that exhibit variable amplitude and direction, occurring either concurrently or against dystonic muscle co-contraction.

Tremor in dystonia has also been previously described as irregular tremor, characterized by inconsistency in frequency while maintaining the fundamental oscillatory nature. This can be demonstrated by a higher frequency spread throughout the registration, a larger bandwidth at the half-power point of the power spectrum (HBW), and an increased tremor stability index in patients with any tremulous-like movements in dystonia compared to essential tremor. However, it required further neurophysiological studies focusing on the true rhythmic oscillatory movements or tremor in dystonia. Additional neurophysiological characteristics of dystonia, including phasic EMG co-contractions with variable durations, may contribute to the irregular appearance of tremor in dystonia.

The pathophysiology of tremor in dystonia is widespread, involving the cerebello-thalamo-cortical pathway (CTC) and the basal ganglia-thalamo-cortical (BG-TH-C) networks. Impaired cerebellar conditioning and motor adaptation, reduction of cerebellar inhibition to the motor cortex, modulation of tremor amplitude with transcranial alternating current stimulation at the cerebellum, and dysfunction of the functional connectivity of the CTC pathway support the primary involvement of cerebellar networks or cerebellar decoupling hypothesis. The BG-TH-C networks may interact with the CTC pathway to contribute to tremor genesis in dystonia, as demonstrated by the involvement of the dentato-rubro-thalamic and pallidothalamic pathways in tremor-related activities from functional imaging studies. In addition, the optimal DBS target for tremor improvement in dystonia was the Ventralis Oral Posterior Nucleus of the thalamus, which receives the connections from these two pathways, emphasizing the significant network dysfunction in both CTC and BG-TH-C circuits.

Personal Information

Full Name	Pattamon PANYAKAEW
Current Position	Associate Professor of Neurology
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Education / Training

Institution and Location	Degree	Year(s)	Field of Study
Chulalongkorn University, Bangkok, Thailand	Residency in Neurology	2010 - 2012	General Neurology
Chulalongkorn Centre of Excellence for Parkinson's Disease & Related Disorders, Bangkok, Thailand	Fellowship in movement disorders	2012 - 2014	Movement Disorders
Human Motor Control Section NINDS, National Institute of Health, USA	Fellowship in movement disorders	2014 - 2017	Neurophysiology in movement disorders

Positions and Honors

Professional Experience

Current

2023-current Associate Professor of Neurology, Chulalongkorn University, Bangkok, Thailand

Previous

2020-2023 Assistant Professor of Neurology, Chulalongkorn University, Bangkok, Thailand

Honors

2014-2017 King Ananda Mahidol Scholarship Recipient (Medicine)
2018 MDS LEAP graduation (class of 2018)
2018 The first runner up SYNNERGIES research contest (clinical study)
2021-2022 Course Director of MDS-AOS Video Case Discussion Series

2023-2024	Guest Editor (Journal of Dystonia, Special tremor section)
2023-2025	MDS-AOS Educational Committee
2023-2025	Members of MDS Clinical Neurophysiology Study Group
2024-2025	Members of MDS Tremor Study Group
2024-2025	IFCN Educational Committee
2025	Course Director of MDS-AOS School of Clinical Neurophysiology in Movement Disorders

Selected Peer Reviewed Publications

1. Panyakaew P, Carandang RA. Modern Phenomenology of Tremor: How Novel Technology Can Assist in the Diagnosis of Tremor. *Mov Disord Clin Pract*. 2025 Aug 1;doi: 10.1002/mdc3.70241
2. Phokaewvarangkul O, Panyakaew P, Sringean J, Jankates W, Phumphid S, Anan C, et al. Unlocking the daily impact of parkinson's challenges through a comprehensive assessment using the ChulaPD ADL questionnaire. *Sci Rep*. 2025;15(1):26212.
3. Schwingenschuh P, Van der Stouwe M, Pandey S, Hirschbichler S, Panyakaew P, Kojovic M, Mukherjee A, Tijssen MA, Merchant SHI, Vial F; MDS Clinical neurophysiology Study group. Clinical neurophysiology for tremor: Common questions in clinical practice. *Parkinsonism Relat Disord*. 2025 Jan;130:107196.
4. Cho HJ, Shin HW, Panyakaew P, Kassavetis P, Popa T, Wu T, Leodori G, Camacho T, Singh S, Meunier S, Hallett M. Differential induction of Parieto-motor plasticity in writer's cramp and cervical dystonia. *Neurobiol Dis* 2024 Nov;202:106724.
5. Panyakaew P, Wongwan P, Bhidayasiri R. The Goal Attainment Scale Refines Patient-Centered Expectations in Botulinum Toxin Treatment of Cervical Dystonia. *J Mov Disord*. 2024 Oct;17(4):462-465
6. Panyakaew P, Phuenpathom W, Bhidayasiri R, Hallett M. Bedside clinical assessment of patients with common upper limb tremor and algorithmic approach. *Asian Biomed (Res Rev News)*. 2024 Apr 30;18(2):37-52.
7. Panyakaew P, Duangjino K, Kerddonfag A, Ploensin T, Piromsopa K, Kongkamol C, Bhidayasiri R. Exploring the Complex Phenotypes of Impaired Finger Dexterity in Mild-to-moderate Stage Parkinson's Disease: A Time-Series Analysis. *J Parkinsons Dis*. 2023;13(6):975-988.

S10-3

Essential Tremor Plus: Is It a Clinically Relevant Concept?

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The classification of *Essential Tremor Plus* (ET Plus) was introduced by the International Parkinson and Movement Disorder Society in 2018 as part of a revised tremor classification framework. ET Plus is defined as typical essential tremor (ET) with additional "soft neurological signs" that do not fulfill criteria for an alternative diagnosis. This classification aims to better represent the phenotypic heterogeneity observed within ET subjects, challenging the traditional view of ET as a monosymptomatic, benign disorder. This concept, however, has generated significant controversy in clinical and research communities due to its ambiguity, lack of biological validation, and potential for diagnostic instability. Accumulating clinical, neurophysiological, and neuroimaging data have offered divergent perspectives regarding ET Plus. Some studies indicate that ET Plus may constitute a distinct subtype within the ET spectrum, often associated with greater disease severity and accelerated progression. Conversely, other evidence suggests that ET Plus may reflect an early or prodromal phase of overlapping neurodegenerative conditions such as Parkinson's disease or cerebellar ataxias. A critical limitation lies in the subjective nature of the additional neurological signs defining ET Plus, which suffer from variability in clinical assessment and lack standardized evaluation protocols. Moreover, inconsistent biomarker findings further complicate efforts to reliably distinguish ET Plus from classical ET or related disorders.

The incorporation of ET Plus into clinical and research frameworks introduces methodological challenges, including heterogeneity in diagnostic criteria application and the potential inflation of ET prevalence estimates, thereby complicating epidemiological analyses and clinical trial designs. Given these uncertainties, it remains unclear whether the designation of ET Plus contributes to improved patient stratification, prognostication, and therapeutic decision-making, or whether it introduces diagnostic ambiguity without clear clinical benefit.

This lecture will provide a comprehensive and critical appraisal of the current evidence concerning ET Plus, with emphasis on its various pathophysiological basis, clinical characterization, and implications for treatment strategies. By presenting a balanced analysis of the strengths and limitations of the ET Plus concept, this discussion aims to equip movement disorder specialists with an informed perspective to guide both clinical management and future research endeavors. Further, there is a critical need for refinement of the tremor classification system with incorporation of objective biomarkers and clearer clinical thresholds.

Keywords:

Essential Tremor, ET Plus, Tremor Classification, Soft Neurological Signs, Phenotypic Heterogeneity

Personal Information

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Education / Training

Institution and Location	Degree	Year(s)	Field of Study
Korea Univ. College of medicine	PhD.	2009	Neurology

Positions and Honors

Professional Experience

Current

2019- current Professor, Korea University Ansan hospital
2024-current Director, policy and external affairs committee, KMDS
2025-current Director of planning, Korea University Ansan hospital

Previous

2013-2018 Associate Professor, Korea University Ansan hospital
2017-2018 Visiting Scholar, UC San Diego

Selected Peer Reviewed Publications

1. Do-Young Kwon et al. Structured speech tasks reveal temporal facial variability as an objective measure in the de-novo Parkinson's disease patients. *Parkinsonism & Related Disorders* 135 (2025):
2. Do-Young Kwon et al. Effects of freezing of gait on static balance in Parkinson's disease patients with and without a history of falls. *Journal of Mechanics in Medicine and Biology*(2025).
3. Do-Young Kwon et al. "Assessing the Association Between Gadolinium-Based Contrast Agents and Parkinson Disease: Insights From the Korean National Health Insurance Service Database." *Investigative Radiology*(2024): 10-1097.

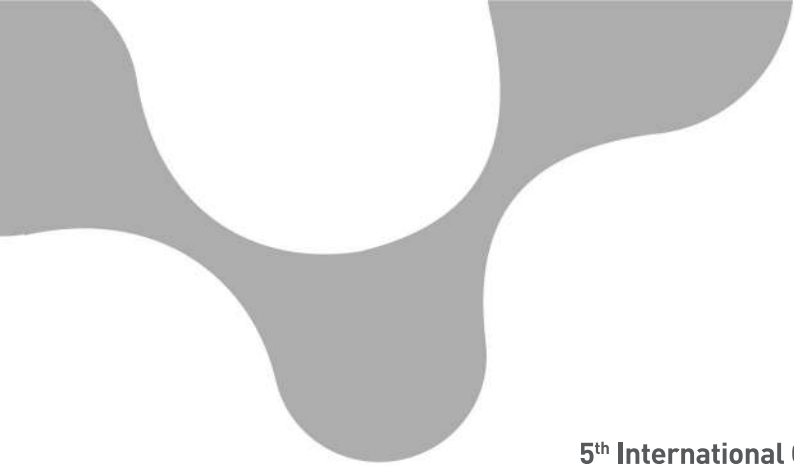
4. Do-Young Kwon et al. Predicting the Future Fall Risk Using Challenging Tasks: Importance of Sensor-Based Quantitative Measurements of Gait in Parkinson's Disease." *Journal of Clinical Neurology* (2024)
5. Do-Young Kwon et al. Comparison of resting tremor at the upper limb joints between patients with Parkinson's disease and scans without evidence of dopaminergic deficit. *Technology and Health Care* 31.1_suppl (2023): 515-523.



ICKMDS 2025

5th International Conference of Korean Movement Disorder Society

**Bridging from the Origin to Clinical Practice:
A Beginning Has an Ending**



ICKMDS 2025

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Bridging from the Origin to Clinical Practice:
A Beginning Has an Ending

Poster Exhibition



Ataxia

P-001

Case Report of Spinocerebellar Ataxia 13 Presenting with Pure Cerebellar Ataxia

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Introduction:

Spinocerebellar ataxia 13 (SCA13) is a rare cause of autosomal-dominant cerebellar ataxia, which is caused by a heterozygous mutation in the KCNC3 gene, which results in voltage-gated potassium channel dysfunction. SCA 13 should be considered in individuals with the following age-related phenotypes. In cases of childhood-onset SCA13, the patient is characterized by progressive ataxia with occasional cognitive decline and/or seizures. We report the SCA13 patient who demonstrated childhood-onset pure cerebellar ataxia.

Case:

A 17-year-old woman visited our clinic with progressive tremor and dysarthria started in her early teens. Developmental delays or seizures were denied. Her mother also exhibited the tremor and gait unsteadiness. Neurological examination revealed cerebellar-type dysarthria, hypermetric saccades, and limb ataxia. The Scale for the Assessment and Rating of Ataxia (SARA) score was 2.5. Other neurological abnormalities, such as pyramidal or extrapyramidal signs or sensory loss, were not observed. The deep tendon reflex was hyporeflexia on the right side. Cognition was grossly intact. She could walk independently and perform most of the activities in daily life.

Discussion:

Brain magnetic resonance imaging (MRI) demonstrated diffuse cerebellar atrophy; the brainstem and middle cerebellar peduncles were relatively preserved. Genetic tests for SCA1, SCA2, SCA3, SCA6, SCA7, SCA8, SCA17, and dentatorubral-pallidoluysian atrophy were normal. Next-generation sequencing using an ataxia panel identified a pathogenic variant, c.1268G>A (p.Arg423His) in the KCNC3 gene. 3-years later, Her symptoms slightly got worse and the SARA score was 5. The brain MRI also showed mildly progressive cerebellar atrophy than the previous study.

P-002

Effects of Home-Based, Remotely Supervised, Cerebellar Transcranial Direct Current Stimulation in Cerebellar Variant of Multiple System Atrophy

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²Neurology, Sungkyunkwan University School of Medicine, Samsung Medical Center, Seoul, Republic of Korea

Background & Objective:

Cerebellar variant of multiple system atrophy (MSA-C) is a leading cause of sporadic degenerative ataxia, for which either disease-modifying therapies or effective symptomatic treatments are currently unavailable for. The aim of this work was to examine whether home-based, remotely supervised transcranial direct current stimulation (tDCS) targeting cerebellum can improve motor and cognitive symptoms in patients with MSA-C.

Methods:

We performed a double-blind, randomized, sham-controlled trial with 84 sessions (for 12 weeks) of daily home-based cerebellar tDCS (2 mA for 20 minutes) in 26 patients with MSA-C. All participants underwent a clinical assessment (International Cooperative Ataxia Rating Scale, Montreal Cognitive Assessment, cerebellar cognitive affective syndrome (CCAS) scale, velocity and cadence of gait, sway area). A follow-up evaluation was conducted at a day, one month, and four months after the stimulation period. Repeated measures analysis of variance was conducted for each outcome with Visit as a within-subjects factor and Group (active vs. sham tDCS group) as a between-subjects factor.

Results:

We observed significant Visit x Group interactions for % changes in ICARS ($F=5.22$, $P=0.003$, $\eta^2=0.085$), CCAS scale ($F=4.11$, $P=0.010$, $\eta^2=0.074$), and gait velocity ($F=9.654$, $P<0.001$, $\eta^2=0.182$).

Conclusions:

A 12-week treatment with home-based, remotely supervised cerebellar tDCS reduces cerebellar motor and cognitive symptoms in individuals with MSA-C, suggesting a promising therapeutic option. This study provides Class II evidence that home-based, remotely supervised cerebellar tDCS is effective and safe in MSA-C.

P-003

Isolated Anti -Yo Antibody Associated Paraneoplastic Cerebellar Degeneration: A Case Report

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Introduction:

We report the first patient in Mongolia with paraneoplastic cerebellar degeneration (PCD) who fulfilled the diagnostic criteria for multiple system atrophy (MSA) and showed a favorable response to immunotherapy.

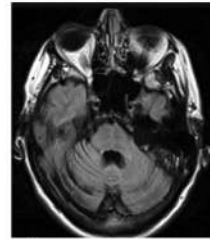
Case:

The patient was a 56-year-old man who was diagnosed with clinically probable MSA, exhibiting cerebellar ataxia, pyramidal signs, and autonomic dysfunction. His clinical course was rapidly progressive, requiring a wheelchair within a year. Cerebellar atrophy with a hot cross bun sign revealed on head magnetic resonance imaging. The patient was referred to a hospital in Beijing where paraneoplastic Anti-Yo antibody (known as Purkinje cytoplasmic antibody 1) was detected. Although whole-body positron emission tomography and tumor markers were negative, a diagnosis of PCD was made. Following the initiation of five administrations of Methylprednisolone pulse therapy, the patient had a substantial stabilization and partial improvement in some of his neurological symptoms.

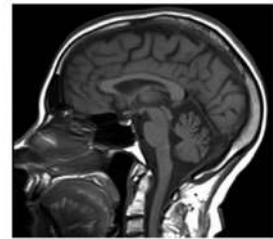
Discussion:

The pathogenesis of PCD is not fully understood, but it is believed to result from autoimmunity against antigens expressed by both the underlying tumor and normal neural tissue, leading to destruction of cerebellar neurons. Anti-Yo antibodies are considered high-risk markers for severe paraneoplastic syndromes, most commonly associated with breast and ovarian carcinomas. The majority of PCD cases with Anti-Yo antibodies are reported in women. Only two cases of Anti-Yo-positive PCD have been reported in men, both associated with lung adenocarcinoma. In conclusion, although onconeural antibody testing is not yet well established in our country, it is crucial to consider that treatable cerebellar ataxia may exist among patients meeting the diagnostic criteria for MSA. Therefore, testing for antineuronal antibodies and cancer screening are essential. The moderate therapeutic response in this case suggests that early immunotherapy can be highly valuable in reversing or stabilizing neurological deficits.

Figure



A hot cross bun sign on axial FLAIR



Cerebellar atrophy on sagittal T1

Keywords

Paraneoplastic cerebellar degeneration, Multiple system atrophy, anti-Yo antibody, Methylprednisolone

P-004

Progressive White Matter Abnormalities on MRI Reveal SYNE1-Related Pure Cerebellar Ataxia: A Diagnostic Mimic of Demyelinating Disease

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Introduction:

SCAR8 is a rare neurogenetic disorder caused by SYNE1 gene mutations, leading to progressive cerebellar ataxia. This case report describes a 38-year-old Filipino female with worsening neurological symptoms over a decade. Extensive investigations ruled out acquired causes, and genetic testing confirmed SCAR8. The patient exhibited classic cerebellar signs without cognitive decline or extracerebellar involvement. Symptomatic and multi-disciplinary management provided partial relief but did not stop disease progression. The case emphasizes the importance of early genetic testing in progressive cerebellar syndromes and highlights the need for supportive care to optimize outcomes.

Case:

A 38-year-old Filipino female experienced a decade-long progression of neurological symptoms, including diplopia, blurred vision, dysarthria, upper limb incoordination, and gait imbalance. Initial symptoms began in 2014, worsening over time to severe motor impairment by 2024. Extensive evaluations ruled out acquired, metabolic, autoimmune, and infectious causes. Serial MRIs showed progressive abnormalities in the brainstem, cerebellum,

and spinal cord, initially suggesting multiple sclerosis or other demyelinating diseases, but these were excluded. Genetic testing identified compound heterozygosity in the SYNE1 gene, confirming SCAR8. The patient exhibited classic cerebellar signs, such as scanning speech, nystagmus, dysmetria, and ataxic gait, without cognitive decline or significant extracerebellar involvement. Management included medications, botulinum toxin injections, and rehabilitative therapies, which provided partial symptomatic relief but did not halt disease progression. This case underscores the diagnostic complexity of SCAR8 and the importance of genetic testing in progressive cerebellar syndromes.

Discussion:

SCAR8 is a rare autosomal recessive spinocerebellar ataxia caused by SYNE1 gene mutations, leading to progressive cerebellar dysfunction. It typically presents in early adulthood with symptoms like gait ataxia, dysarthria, nystagmus, and intention tremor, without cognitive decline or significant extracerebellar involvement. Diagnosis relies on genetic testing, supported by clinical and MRI findings of cerebellar atrophy. Management focuses on symptom control through a multidisciplinary approach, including physical, occupational, and speech therapy, along with medications for spasticity. While life expectancy is not reduced, SCAR8 causes significant disability over time, emphasizing the need for early diagnosis, genetic counseling, and supportive care. Research into gene-targeted therapies is ongoing, but current treatments aim to improve quality of life.

Figure

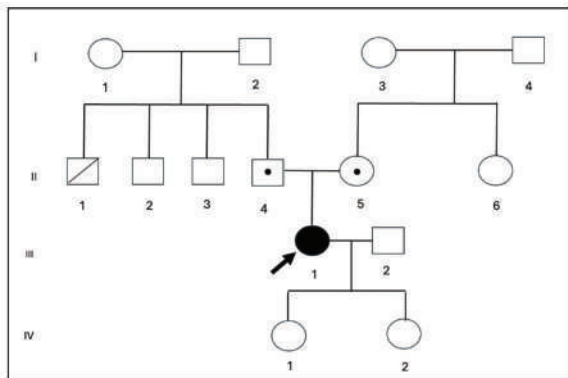


Figure 1. Four-generation Pedigree of the family showing autosomal recessive inheritance of SCAR8 due to biallelic SYNE1 variants. The proband (III-1, indicated by the arrow) is the only affected individual, while both parents (II-4 and II-5, half-shaded) are asymptomatic carriers.

Figure

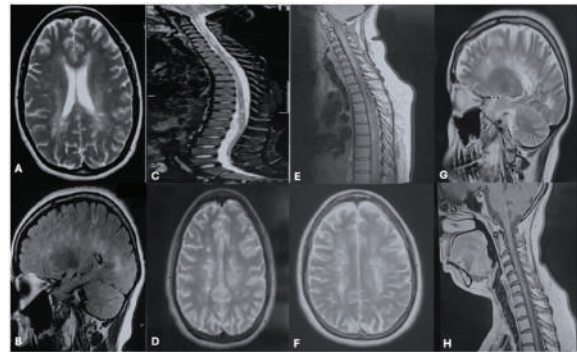


Figure 2. Serial Cranial Magnetic Resonance Imaging (MRI) from 2014 to 2022, demonstrating progressive white matter abnormalities and cerebellar atrophy. In the Year 2014 (A),(B) (C) Magnetic resonance imaging (MRI) scans of the brain and spinal cord demonstrating progressive white matter abnormalities and cerebellar atrophy. The Sagittal MRI of the spinal cord, revealing abnormal signal intensities in the cervical and thoracic regions. These findings illustrate the characteristic neuroimaging features of SCAR8, which can mimic acquired demyelinating diseases but are ultimately due to a genetic ataxia. In the year 2015 serial monitoring of Cranial MRI shows still the progressive white matter abnormalities (D). While the sagittal MRI of spinal cord still revealing abnormal signal intensities in the cervical and thoracic regions (E). In the year 2022 serial monitoring of Cranial MRI shows no significant change in the degree of white matter lesions with reflective of a demyelinating disease (F)(G)(H).

Figure

Category	Test/Panel/Specific Test	Genes/Labors/Assay	Result/Value/Variant Identified	Interpretation/Notes
Genetic	Linkage Panel	SYNE1 (PMID: 314527)	Variant of uncertain significance (VUS)	Not consistent with clinical phenotype
	Whole Exome Panel	SYNE1	Two VUS (missense/frameshift)	Suspicious for SCAR8
	Whole Exome Sequencing	SYNE1, SYNE2	Two SYNE1 VUS (combined in trans); Pathogenic: SC7942 variant	SYNE1 variants consistent with SCAR8; SC7942 unknown
	Recurrent Sequencing Analysis	SYNE1	Pathogenic homozygous for one VUS	Confirms compound heterozygosity in proband
	CR1	-	Bi-allelic (194pG, 99C-4.8-4.17%); Pathogenic: 128-417%.	Normal
	Cholesterol	-	4.7 mg/dL	Normal
	Uric Acid	-	Specific gravity 1.01, pH 6.0, no protein/glucose/ketones	Normal
	ALT	-	28 U/L	Normal
	Bilirubin	-	No 10P smudges, 0.12 smudges, 13 10P smudges, 1030 10 smudges	Normal
	Thyroid Function	TSH, FT4	TSH 0.1 mIU/L, FT4 1.2 ng/dL	Normal
Hematology	Anti-Nuclear Antibody	-	1:10 titer	Normal
	Anti-Smith Antibody	-	4.14 ng/mL	Normal
	Antinuclear Antibody	-	12.4 ng/mL	Normal
	Vitamin B12	-	489 ng/L	Normal
	Vitamin D	-	22 ng/mL	Normal
	Free Long Chain Fatty Acids	-	C20:0 0.19 ng/mL	Normal
	Antinuclear Antibody	-	Negative	Normal
	Anti-peroxidase antibody	-	Negative	Normal
	Glutamate	-	4.4 ng/mL	Normal
	Creatinine	-	26 ng/dL	Normal
Neurophysiology	Clinical Neuro	EMG/EEG, Sensory	Negative	Normal
	Cerebral Blood Flow (CBF), Cerebral Perfusion (CP)	-	CK 100/100, CP 100/100	Normal
	EMG	-	7 mm/s	Normal
	CKP	-	6.4 ng/L	Normal
	ANCA	-	Negative	Normal
	Anti-SSA/SSB	-	Negative	Normal
	DRUGS	-	Negative	Normal
	Anti-cardiolipin antibody	MLI, APL	Negative	Normal
	ANCA	-	Negative	Normal
	Other specific autoantibodies	-	Negative	Normal
Neuroimaging	MR	-	Non-contrast	Normal
	MR	-	Non-contrast	Normal
	MR	-	Non-contrast	Normal
	MR	-	Non-contrast	Normal
	MR	-	Non-contrast	Normal
	MR	-	Non-contrast	Normal
	MR	-	Non-contrast	Normal
	MR	-	Non-contrast	Normal
	MR	-	Non-contrast	Normal
	MR	-	Non-contrast	Normal

Keywords

Cerebellar Ataxia , SCAR 8 , SYNE1, White Matter abnormalities, Leukodystrophy

P-005

Novel *ATAD3A* Intronic Variant: Harel-Yoon Syndrome with Predominant Cerebellar Ataxia and Osteoporosis

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Introduction:

Harel-Yoon syndrome (HAYOS) is a rare autosomal recessive neurodevelopmental disorder caused by biallelic variants in the ATPase family AAA domain-containing protein 3A (*ATAD3A*) gene. Reported phenotypes include global developmental delay, hypotonia, spasticity, hypertrophic cardiomyopathy, optic atrophy, epilepsy, and early mortality.

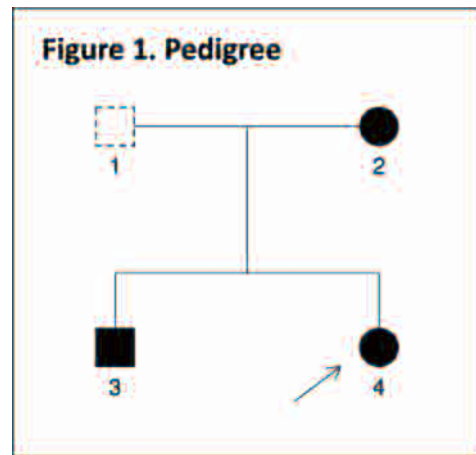
Case:

We report a 33-year-old male who presented with progressive dysarthria, hand tremor, and ataxic gait beginning in adolescence. Brain MRI revealed disproportionate cerebellar atrophy, and bone densitometry demonstrated severe osteoporosis. His past history included recurrent fractures from minor trauma and spontaneous hemopneumothorax. Two of his younger dizygotic twin brothers also exhibited neurological manifestations: one with congenital cortical dysplasia, epilepsy, severe intellectual disability, and short stature; the other with tremor, ataxic gait, osteoporosis, and gaze abnormalities. Whole exome sequencing identified variants of uncertain significance in *ATAD3A* and *MAPK8IP3*. Segregation analysis revealed paternal inheritance of *ATAD3A* variant (NM_001170535.3:c.980G>C) but did not initially support pathogenicity, because the father was an asymptomatic carrier. Subsequent whole genome sequencing revealed an additional deep intronic *ATAD3A* variant (NM_001170535.3:c.751-560C>T), which was maternally inherited, establishing compound heterozygosity and confirming Harel-Yoon syndrome in the proband and affected siblings.

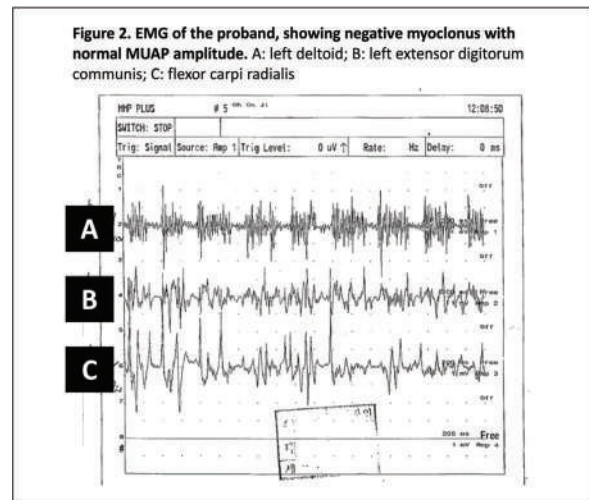
Discussion:

This case expands the clinical spectrum of HAYOS by highlighting predominant cerebellar ataxia and tremor, as well as severe osteoporosis. The diagnostic process emphasizes the importance of considering deep intronic variants in unresolved hereditary ataxia, and of judicious interpretation of variants of uncertain significance.

Figure



Figure



Keywords

Cerebellar ataxia, Genetics, Whole genome sequencing, Hereditary ataxia, Intronic variant

P-006

Multiscale Mathematical Modeling of Ataxia Progression Using Single-Nucleus Transcriptome Trajectories, Patient-Derived Neuronal Epigenomics, and Blood Surrogates

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³Biology, Yogyakarta State University, Sleman, Indonesia

⁴Health Management Laboratory, International University Semen Indonesia, Gresik, Indonesia

Background & Objective:

Ataxia can remain stable for years before abrupt decline, reflecting Purkinje network instability. We developed a multiscale mathematical model that maps single-cell cerebellar changes to vulnerability trajectory, estimates rescue windows before failure, and provides 10-gene blood surrogate for monitoring and stratification.

Methods:

We combined single-nucleus cerebellar trajectories from spinocerebellar ataxia type 1 (GSE246183), patient-derived neurons with DNMT1 mutation and paired methylome/transcriptome (GSE126890), NAD-linked mitophagy perturbation (GSE152289), and an FXTAS blood cohort to train a 10-gene panel (GSE48873; directionality check GSE48902). The model couples epigenetic drift (stochastic), mitochondrial health and mitophagy (three-compartment dynamics), and a Purkinje network stability score driven by cumulative cellular stress. Model states align to the single-cell trajectory via pathway/regulon embeddings with domain-adversarial correction. Validation used bootstrap CIs, leave-dataset-out testing, model ablations, and early-warning analysis.

Results:

The vulnerability trajectory matched pseudotime ($r=0.63$, 95% CI 0.57-0.69; calibration slope 0.87, 95% CI 0.79-0.95). Neurons showed higher vulnerability with a synaptic-program decline -0.37 SD ($p=2.4 \times 10^{-3}$) and an epigenetic change rate 0.028 (95% CI 0.020-0.036). Early-warning signals anticipated loss of stability 7.8 months (IQR 5.6-10.0). Simulated therapy (+25% mitophagy clearance, +10% epigenetic support) restored stability in 47% (95% CI 40-54) with vulnerability improvement 0.13 (95% CI 0.08-0.18) and correlation with nicotinamide adenine dinucleotide signature reversal $r=0.50$ (95% CI 0.38-0.60). 10-gene blood sur-

rogate achieved AUROC 0.79 (95% CI 0.74-0.85) and 0.71 under leave-dataset-out.

Conclusions:

Linking sudden ataxia worsening to loss of Purkinje network stability, the model identifies individualized rescue windows and dosing thresholds, and provides a 10-gene blood surrogate to guide treatment and monitoring.

Keywords

Ataxia, Purkinje, Mathematical modeling, Mitophagy, Biomarker

P-007

Wandering Eyes, Wavering Steps: A Novel Homozygous *SQSTM1* Variant in Filipino Adolescent with Childhood-Onset Ataxia, Dystonia, and Gaze Palsy

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⁵DEPARTMENTS OF ANATOMY & INTERNAL MEDICINE, CEBU INSTITUTE OF MEDICINE, CEBU, Philippines

Introduction:

Objective: To present a genetically confirmed case of childhood-onset neurodegeneration with ataxia, dystonia, and gaze palsy (NADGP) associated with a novel homozygous *SQSTM1* variant in the Philippines.

Background: NADGP is an autosomal recessive disorder typically caused by biallelic loss-of-function *SQSTM1* variants, manifesting with progressive ataxia, vertical gaze palsy, and hyperkinetic movements.

Case:

Methods: A 17-year-old Filipino female presented with progressive gait imbalance, oculomotor apraxia, choreoathetosis, dystonia, and cognitive impairment. The diagnostic workup included evaluation for acquired ataxia, neuroimaging, and whole-exome sequencing.

Discussion:

Results: Genetic analysis revealed a novel homozygous frame-shift *SQSTM1* variant (c.247_248insA; p.Leu83TyrfsTer11), not previously reported in ClinVar. The patient was started on levetiracetam, memantine and coenzyme Q10. Rehabilitation was also initiated for supportive care.

Conclusions:

This report expands the clinical and genetic spectrum of NADGP and highlights the utility of exome sequencing in early-onset movement disorders. The absence of cerebellar atrophy in this case broadens the recognized neuroradiologic profile of NADGP. Identification of this novel variant contributes to global genotype-phenotype correlations.

Keywords

SQSTM1 VARIANT, CHILDHOOD-ONSET NEURODEGENERATION WITH ATAXIA, DYSTONIA, AND GAZE PALSY (NADGP), DYSTONIA, FILIPINO AATAXIA, HYPERKINETIC MOVEMENT DISORDER

Basic Sciences

P-008

Neuroprotective Effects of 2 β -Hydroxybetulinic Acid 3 β -Oleate Isolated from *Euryale ferox* in a Rotenone-Induced Model of Parkinson's Disease

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Background & Objective:

Parkinson's disease (PD) is a progressive neurodegenerative disorder characterized by dopaminergic neuronal loss in the substantia nigra, accompanied by oxidative stress and neuroinflammation. The search for neuroprotective agents with antioxidant and anti-inflammatory properties is of paramount importance. *Euryale ferox* Salisb., known for its ethnomedicinal value, yields 2 β -hydroxybetulinic acid 3 β -oleate (HBAO), a triterpenoid derivative with promising pharmacological potential. This study evaluates the neuroprotective effects of HBAO in an experimental model of Parkinson's disease

Methods:

HBAO was isolated and characterized using standard spectroscopic methods. Parkinsonism was induced in Wistar rats using rotenone administration for 28 days. HBAO was administered orally at graded doses for the duration of the study. Behavioral parameters, including locomotor activity, rotarod performance, and catalepsy tests, were assessed. Biochemical markers of oxidative stress (MDA, SOD, catalase) and pro-inflammatory cytokines (TNF- α , IL-1 β) were measured.

Results:

HBAO treatment significantly improved behavioral performance compared to rotenone-treated controls ($p < 0.05$). Biochemical analyses demonstrated reduced lipid peroxidation and restoration of antioxidant enzyme activities. Furthermore, HBAO attenuated neuroinflammatory cytokine levels and preserved tyrosine hydroxylase-positive neurons in the substantia nigra.

Conclusions:

2 β -hydroxybetulinic acid 3 β -oleate (HBAO) isolated from *Euryale ferox* Salisb. exhibits substantial neuroprotective activity in a rotenone-induced model of Parkinson's disease, likely through its antioxidant and anti-inflammatory mechanisms. These findings support HBAO as a promising candidate for further development in Parkinson's disease therapeutics.

Keywords

2 β -hydroxybetulinic acid 3 β -oleate, Parkinson Disease, *Euryale ferox* Salisb.

P-009

Network--Guided Identification of microRNA--TLR4 Hubs and Phytochemical P2X7 Antagonists for Multi-Target Suppression of Neuroinflammation in Parkinson's disease

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Background & Objective:

Neuroinflammatory signalling is a key but incompletely charted contributor to Parkinson's disease (PD). We aimed to integrate network transcriptomics and structure-based ligand discovery to pinpoint upstream regulators and identify readily translatable small-molecule inhibitors that could jointly restrain microglia-driven neuroinflammation

Methods:

Differential-expression analysis of the GEO dataset GSE26927 was coupled with Gene Ontology enrichment, protein–protein interaction topology and miRNet interrogation to construct a Toll-like-receptor (TLR) signalling interactome. In parallel, a high-fidelity homology model of the human P2X7 purinoceptor was generated. Over 3,500 plant-derived compounds were virtually screened through hierarchical docking, MM-GBSA rescoring, ADMET filtration and 100-ns molecular-dynamics simulation.

Results:

TLR4 ranked as the principal network hub while five microRNAs (miR-21-5p, miR-17-5p, miR-93-5p, miR-7-5p and miR-92b-3p) formed a dense regulatory core predicted to fine-tune TLR4-driven cytokine release. Docking and simulation workflows converged on five stable ligands—silychristin, silybin, rosmarinic acid, nordihydroguaiaretic acid and aurantiamide—that fit the P2X7 binding cavity with ΔG_{bind} values of -57 to -70 kcal mol $^{-1}$, maintain key hydrogen bonds for $>80\%$ of the trajectory and satisfy oral-drug ADMET criteria.

Conclusions:

The miRNA–TLR4 axis and P2X7 pyroptotic node emerge as interconnected checkpoints within the PD inflammatory landscape. The highlighted microRNAs are promising biomarkers or epigenetic levers, whereas the shortlisted phytochemicals provide chemically diverse starting points for P2X7 antagonist development, collectively advancing multi-target strategies to slow PD progression.

P-010

Anti-neuroinflammatory Effect of Cyclobenzaprine by Modulating Necroptosis via TLR4/TRIF/RIPK1/MLKL Pathway in Parkinson's Disease

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Background & Objective:

Parkinson's disease (PD) is a progressive neurodegenerative disorder marked by the selective degeneration of dopaminergic neurons and sustained neuroinflammation. Recent studies implicate necroptosis—a regulated form of necrotic cell death mediated by the TLR4/TRIF/RIPK1/MLKL signaling cascade—as a critical driver of neuroinflammation and neuronal loss in PD. Cyclobenzaprine, a centrally acting muscle relaxant, has shown anti-inflammatory potential in non-neurological models. This study aimed to eval-

uate the neuroprotective effect of cyclobenzaprine by targeting necroptosis in PD.

Methods:

In vivo and in vitro models of PD were employed using 6-OHDA-lesioned rats and MPP⁺-treated SH-SY5Y cells. Cyclobenzaprine was administered at optimized doses, and neuroinflammatory markers (TNF- α , IL-1 β , IL-6), necroptotic pathway components (TLR4, TRIF, RIPK1, MLKL), and dopaminergic neuron survival (TH expression) were assessed using ELISA, Western blotting, qRT-PCR, and immunohistochemistry. Motor function was evaluated through rotarod and open field tests.

Results:

Cyclobenzaprine treatment significantly attenuated neuroinflammation by reducing pro-inflammatory cytokine levels and downregulating TLR4 and TRIF expression. It markedly inhibited the activation of RIPK1 and phosphorylation of MLKL, indicating suppression of necroptosis. Treated PD models exhibited preservation of tyrosine hydroxylase-positive neurons and demonstrated improved motor performance compared to untreated controls

Conclusions:

Cyclobenzaprine confers neuroprotection in PD models by modulating necroptotic signaling via the TLR4/TRIF/RIPK1/MLKL pathway and attenuating neuroinflammation. These findings position cyclobenzaprine as a promising therapeutic candidate for mitigating neurodegeneration in Parkinson's disease

Keywords

Parkinson disease, Necroptosis, Neuroinflammation

P-011

Expression of TDP-43 in Astrocytes Leads to Defective Phenotypes in Neuron and Muscle Cells by Activating the GSK-3 β Signaling Pathway

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Background & Objective:

TDP-43-positive inclusions have also been reported to be associated with the hippocampal CA1 Lewy-body pathology of familial

PD patients bearing a Parkin mutant suggesting that there is likely a link between Parkin and TDP-43. Also, It is known that loss of the nuclear RNA binding protein TAR DNA binding protein-43 (TDP-43) into cytoplasmic inclusions is the strongest correlate to neurodegeneration in frontotemporal degeneration (FTD) and amyotrophic lateral sclerosis (ALS). TDP-43, TAF15, and FUS are DNA/RNA binding proteins, and these lead to neurodegenerative disease when inducing cytoplasmic aggregates. Selective expression of TDP-43 in rat astrocytes also leads to non-cell autonomous neuronal toxicity. TDP-43 is ubiquitously expressed in many tissues and cell types, including glial cells of the central nervous system. Glycogen Synthase Kinase 3 β (GSK3 β) is a Serine/Threonine protein kinase that phosphorylate either threonine or serine and participates in a variety of cellular processes such glycogen metabolism, gene transcription, apoptosis and microtubule stability.

Methods:

To identify the mechanism of neuroinflammatory induction through over-expression of GSK3 β in astrocytes using Shaggy which is the homolog of GSK3 β in drosophila model.

Results:

1. GSK3 β / Shaggy enzyme activity was increased in TDP-43-induced drosophila degenerative model. 2. Inhibition of Shaggy restored the abnormal wing posture in TDP-43-induced drosophila degenerative model. 3. Inhibition of Shaggy restored the locomotor activity of adult flies in TDP-43-induced drosophila degenerative model. 4. Inhibition of GSK3 β restored the neuromuscular junction defects in TDP-43-induced drosophila degenerative model. 5. Cytoplasmic TDP-43 was decreased by the knockdown of GSK3 β 6. Astrocyte-derived TDP43 aggregates are spread and propagation to neurons.

Conclusions:

We could see that to regulate GSK3 β activity might be weaken cystic TDP-43 intermediate neuroinflammatory in degenerative diseases.

P-012

HDAC Inhibition and Acetylation-Driven Disulfide-Linked Aggregation of TDP43: Implications for TDP43 Proteinopathies

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Background & Objective:

TDP43 proteinopathies are characterized by the pathological aggregation and mislocalization of TDP43 protein. These aberrant aggregates disrupt cellular functions and contribute to neurodegeneration. However, the molecular mechanisms driving TDP43 aggregation remain unclear. HDACs regulate protein acetylation, a process implicated in protein aggregation and neurodegenerative diseases. This study aims to elucidate the role of acetylation in TDP43 aggregation, particularly focusing on its mislocalization and oligomerization dynamics.

Methods:

We established a TDP43-BiFC cell model to visualize TDP43 oligomerization in living cells. Using this system, we examined the effects of cellular stress activators and HDAC inhibitors on TDP43 aggregation. The localization and aggregation patterns of TDP43 were analyzed via fluorescence microscopy, and biochemical characterization of aggregates was performed using WB and immunocytochemistry.

Results:

Cellular stress activators induced distinct nuclear and cytoplasmic TDP43 aggregation patterns, indicating the involvement of multiple stress-dependent pathways in TDP43 pathology. Broad HDAC inhibition triggered a time-dependent mislocalization of TDP43 from the nucleus to the cytoplasm, suggesting that HDAC-regulated acetylation is crucial for nuclear TDP43 retention. Furthermore, HDAC inhibition led to increased cellular acetylation, which promoted the formation of stable, SDS-resistant TDP43 oligomers via disulfide-linked aggregation. While phosphorylation was detected within these aggregates, findings suggest that disulfide bonding plays a primary role in driving aggregation rather than phosphorylation.

Conclusions:

Our findings highlight the critical role of acetylation-mediated disulfide-linked aggregation in TDP43 pathology and suggest that HDAC inhibition contributes to TDP43 mislocalization and stable oligomer formation. Targeting this pathway may provide a novel therapeutic strategy for TDP43 proteinopathies.

P-013

DPP-4 Inhibitor Alleviates Gut-Brain Axis Parkinson's Disease Pathology

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Background & Objective:

Dipeptidyl peptidase-4 inhibitors (DPP-4is) have been reported to exhibit therapeutic effects in Parkinson's disease (PD), increasing their potential for drug repurposing. One aspect of PD pathogenesis is thought to be associated with the gut-brain axis, where α -synuclein from the gut is transmitted to the brain via the vagus nerve (VN). We explored whether sitagliptin, a DPP-4i, exhibits a protective effect in a low-dose rotenone-treated gut-brain axis-associated PD model.

Methods:

To explore the effect of sitagliptin, we used the oral rotenone-treated mouse model, which showed spreading of pathological α -synuclein from the intestine in a stereotypic manner via the VN into the midbrain with motor deficits.

Results:

Sitagliptin mitigated rotenone-induced gut inflammation and Toll-like receptor 2 (TLR2) expression, reduced α -synuclein accumulation in the gut, VN and brain, and lessened neuronal loss in the medulla and midbrain with recovery of motor performance. In addition, sitagliptin suppressed inflammation in response to a TLR2 agonist and rotenone in macrophages, enteric glial cells, and enteroendocrine cell lines *in vitro*. In STC-1, an enteroendocrine cell line, sitagliptin also decreased rotenone-induced endogenous α -synuclein levels. The beneficial effects of sitagliptin were maintained even under glucagon-like peptide-1 receptor blockade. Notably, sitagliptin significantly altered the gut microbiome, shifting towards a profile that may counteract PD pathology.

Conclusions:

These findings demonstrated that sitagliptin alleviated α -synuclein deposition in the gut and brain through modulation of TLR2-mediated inflammation and altered the gut microbiome composition towards a more favorable profile, which indicates that DPP-4is can offer a novel therapeutic avenue for managing PD.

P-014

Parkinsonian State-Induced Alteration of Striatal Neuronal Ensemble Dynamics in a Cell Type-Specific Manner

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Background & Objective:

Different subtypes of neurons in the striatum play a significant role in movement and learning by responding to motivational stimuli. In Parkinson's disease (PD), these neurons are linked to immobility and levodopa-induced dyskinesia. However, their activity patterns in the parkinsonian state and their spatiotemporal activity patterns are not well defined. This study therefore set out to delineate the changes in cholinergic interneuron dynamics between healthy and parkinsonian mice to uncover their underlying network mechanisms.

Methods:

We utilized 8 D1-cre mice, 5 A2a-cre mice, and 7 ChAT-cre and 10 PV-cre mice. The mice injected with AAV-flex-Gcamp8f virus underwent an open field test (OFT) combined with in-vivo 1-photon endoscopic calcium imaging. Subsequently, we performed serial tests in a parkinsonian state, which was induced by injection of 6-OHDA into the right medial forebrain bundle.

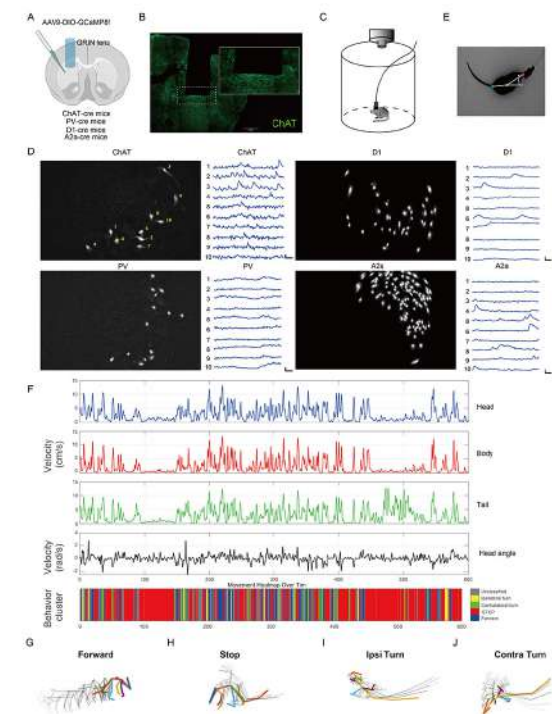
Results:

Compared to the baseline state, D1, A2a, and PV neurons maintained overall significant activation patterns across behavioral phases in the Parkinsonian state, but ChAT neurons lost significant responses during stop, ipsilateral turn, and contralateral turn, indicating a selective impairment in movement-related signaling under dopamine-depleted conditions. Overall pair-wise correlation coefficient was higher in PV than in other cell types followed by cholinergic interneurons and medium spiny neurons. Ensemble decoding performance was markedly impaired in striatal ChAT, D1, and A2a neurons in parkinsonian state. In

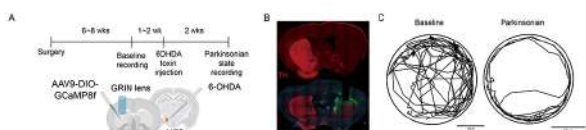
parkinsonian mouse, there was a significant higher mean velocity in stop aligned inhibition (CLOI) compared to random optogenetic inhibition.

Conclusions:
Different striatal neurons significantly encoded distinct behavioral signature; however, we observed a prominent loss of behavioral encoding pattern in cholinergic neurons in the parkinsonian state. The selective inhibition of the cholinergic neurons during the stop phase significantly improved motor dysfunction in the parkinsonian state.

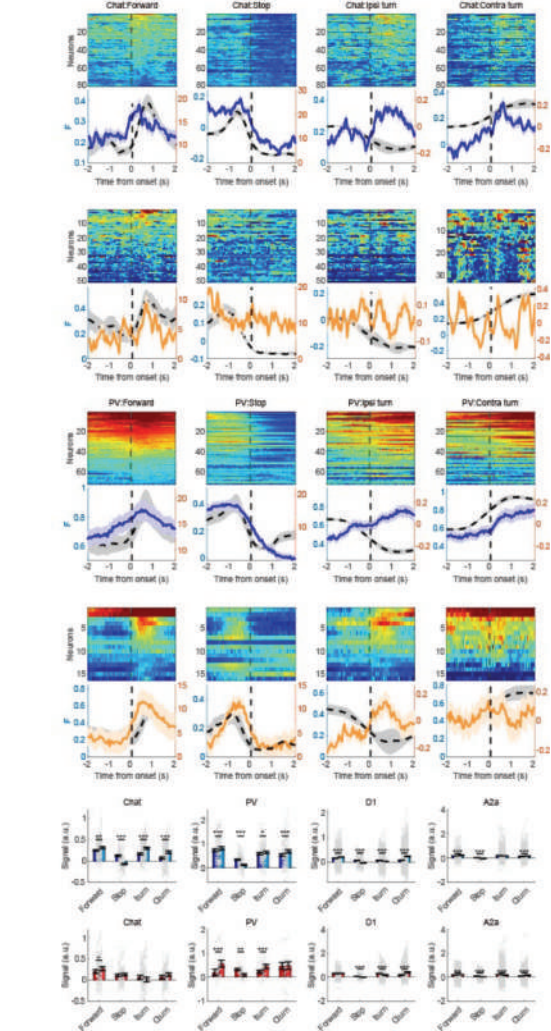
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Figure



Figure



Keywords

Parkinsonism, Striatal cholinergic interneurons, Cholinergic ensembles, Striatal neuronal ensemble dynamic, Spatiotemporal activity patterns

P-016

Therapeutic Intervention of Polyphenols Against α -Synuclein Aggregation Associated With Parkinson's Disease: An AI-Based Drug Discovery Approach

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Background & Objective:

Neurodegenerative diseases are hallmarked by amyloidogenic protein aggregation. Over the recent years, plant-based compounds have been drawing attention in healthcare system due to fewer side effects. Polyphenols are plant-derived compounds that have a significant property to reduce amyloidogenic protein accumulation. Current study was designed to explore the most effective polyphenol in terms of inhibition of α -synuclein (α -syn) fibrillation.

Methods:

A total of 54 polyphenols were selected from different databases for the study. Different analytical web tools, i.e., OSIRIS and Swiss-ADME, were employed for assessing the pharmacokinetic properties of selected polyphenols. DoG SiteScorer and CASTp web servers projected positions were utilized for the investigation of the interaction potential between polyphenols and α -syn. Based on promising interaction of polyphenols with α -syn amyloidogenic amino acids as well as negative binding energy, protocatechuic acid and apigenin were subjected to further in vitro analysis. First, α -syn was incubated under fibrillation conditions with and without polyphenol treatment. In vitro study results, including atomic force microscopy (AFM), thioflavin T (ThT) fluorescence assay, and proteinase K enzymatic digestion assay, revealed the significant inhibition of α -syn fibril formation by protocatechuic acid and apigenin.

Results:

FTIR spectroscopy indicated the inhibitory effect of protocatechuic acid and apigenin through a reduction in β -sheet content. Protocatechuic acid was found to be more effective in α -syn fibril-exposed SH-SY5Y cells viability enhancement via promoting destabilization of toxic fibrils and forming amorphous nontoxic aggregates.

Conclusions:

Protocatechuic acid and apigenin can be utilized for polyphenol-based drug development against synucleinopathies, including Parkinson's disease (PD).

P-017

Common Neuronal DEGs Between Submandibular Gland Tissue and Substantia Nigra in Patients With Parkinson's Disease

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Background & Objective:

Objective; To identify common neuronal molecular markers between submandibular gland (SMG) tissue and substantia nigra (SN) tissue in patients with Parkinson's disease (PD).

Background; Parkinson's disease (PD), the second most prevalent neurodegenerative disorder, is characterized by α -synuclein pathology, a key hallmark of the disease. The molecular mechanisms underlying α -synuclein aggregation, particularly in idiopathic PD (IPD), remain unclear. Post-translational modifications (PTMs), including phosphorylation, ubiquitination, and SUMOylation, have been implicated in α -synuclein aggregation. The SMG, anatomically connected to the brainstem via cranial nerves, exhibits α -synuclein pathology comparable to that of the SN. We hypothesize that RNA signatures in SMG tissue may share molecular biomarkers with SN tissue that contribute to α -synuclein aggregation in patients with IPD.

Methods:

Total RNA sequencing (RNA-seq) was performed on SMG tissue from seven de novo, drug-naïve PD patients and six age- and sex-matched, disease-free controls. Differentially expressed genes (DEGs) from bulk RNA-seq of SMG tissue in PD patients were compared with neuronal DEGs from single-nucleus RNA-seq of SN tissue obtained from postmortem PD brain datasets.

Results:

Among 20 neuronal DEGs identified in the SN of PD patients, 13 were also detected in SMG tissue. These genes are involved in diverse molecular pathways, including ubiquitin activity, chromatin structural regulation, and mitochondrial function.

Conclusions:

The identified DEGs may serve as potential molecular markers associated with PD pathogenesis. Ongoing studies are investigating these mechanisms in PD animal models. This work is supported by the Global Physician-Scientist Training Program from KHIDI.

P-018

Analysis of Differentiation Pathways into Mid-brain Dopaminergic Neurons Using a Double Knock-In Reporter Cell Line

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Background & Objective:

Human embryonic stem cells (hESCs) can be differentiated toward ventral midbrain dopaminergic (vmDA) neurons by neural induction followed by Wnt and SHH signaling, generating progenitors marked by LMX1A, FOXA2, and EN1. However, maturation into functional vmDA neurons remains inefficient, with <50% conversion observed in vitro and in vivo. This study aimed to establish a double knock-in (KI) reporter hESC line to trace the maturation from DA progenitors to vmDA neurons and to analyze underlying molecular pathways, including immune-related expression.

Methods:

A CRISPR/Cas9 or TALEN-based strategy was used to generate a double KI hESC reporter line: eGFP was inserted into the LMX1A locus (progenitor marker) and mCherry into the PITX3 locus (mature vmDA neuron marker, A9/A10 specific). Differentiation toward the dopaminergic lineage was induced. On day 20, eGFP(+) and eGFP(-) progenitor populations were isolated. On day 34, two subsets were sorted: eGFP(+)/mCherry(+), and eGFP(+)/mCherry(-). Bulk RNA sequencing was performed to characterize transcriptional profiles.

Results:

LMX1A-eGFP(+) cells on day 20 displayed robust enrichment of dopaminergic progenitor markers compared to eGFP(-) cells. By day 34, PITX3-mCherry(+) cells showed upregulation of genes associated with neuronal maturation and dopamine synthesis pathways, while PITX3-mCherry(-) subsets retained progenitor-like features. Transcriptomic analyses revealed distinct trajectories of lineage progression and highlighted differential expression of immunologically relevant genes, including HLA isoforms, between progenitor and neuron populations.

Conclusions:

The double KI reporter line enabled precise tracking of mDA neuron differentiation from hESCs. Our findings dissect molecular

pathways governing progenitor-to-neuron transitions and provide insight into immunogenic features of hESC-derived vmDA neurons. These results will inform strategies to improve the efficiency and safety of cell replacement therapies in Parkinson's disease.

Keywords

Human embryonic stem cells, Midbrain dopaminergic neuron, Reporter cell line, RNA sequencing

P-019

Cell-Type Specific Modulation of Subthalamic Nucleus for Rescue of Parkinsonian Symptoms

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Background & Objective:

The subthalamic nucleus (STN) is a primary target for deep brain stimulation (DBS) in Parkinson's disease, yet the specific neuronal subtypes within the STN that mediate symptom relief remain undefined. Clarifying the precise cell types and circuits involved in STN-DBS-induced motor improvement is essential. This study aimed to determine the contribution of STN parvalbumin (PV) neurons to the alleviation of parkinsonian motor deficits through selective neural modulation.

Methods:

Using a PV-Cre transgenic mouse line, we targeted PV-expressing neurons in the STN. Motor performance was assessed in two parkinsonian mouse models (6-OHDA and DAT-PARIS-PV genetic model) with the pole test. Optogenetic stimulation (ChR2, 100 Hz) was applied unilaterally in the 6-OHDA model and bilaterally in the DAT-PARIS model. Cre-dependent (Jx-on) and Cre-independent (Jx-off) viral vectors enabled selective manipulation of PV-STN versus non-PV-STN neurons. Chemogenetic inhibition was achieved using FlexON-hM4Di and FlexOFF-hM4Di constructs. To further evaluate motor asymmetry, unilateral optogenetic stimulation of PV and non-PV STN neurons was tested with the apomorphine rotation assay.

Results:

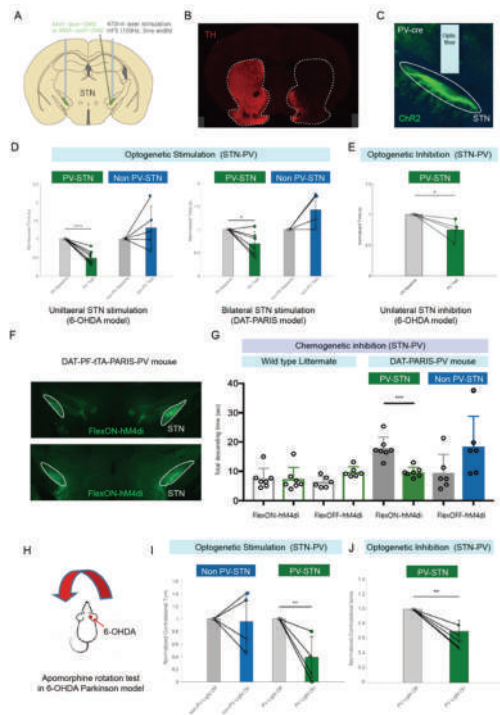
Optogenetic activation of PV-STN neurons significantly improved motor performance in both parkinsonian models, while stimu-

lation of non-PV STN neurons produced no detectable benefit. Similarly, chemogenetic silencing of PV-STN neurons robustly modulated motor symptoms, whereas non-PV-STN inhibition had no effect. In the apomorphine rotation test, unilateral optogenetic activation of PV-STN neurons abolished pathological rotation, an outcome absent with non-PV-STN modulation.

Conclusions:

These findings demonstrate that bidirectional control of PV-expressing STN neurons is sufficient to improve parkinsonian motor symptoms. Targeting this specific STN subpopulation may inform the development of next-generation cell type- and circuit-specific DBS strategies for Parkinson's disease.

Figure



Biomarkers

P-021

Peripheral Blood Autophagy-Lysosomal Pathway Proteins As Early Biomarkers for Parkinson's Disease Detection

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Background & Objective:

Parkinson's disease (PD) is a progressive neurodegenerative disorder affecting 1% of individuals over 60 years. Current diagnostic approaches rely on motor symptoms that manifest after substantial (60-70%) dopaminergic neuronal loss, limiting early intervention opportunities. The autophagy-lysosomal pathway (ALP) degrades misfolded α -synuclein aggregates, and its dysregulation contributes to PD pathogenesis. This study investigated ALP-associated proteins in peripheral blood mononuclear cells (PBMCs) as potential diagnostic biomarkers for early-stage PD detection.

Methods:

We enrolled 37 early-stage PD patients (symptom onset within 3 years) and 40 age-matched essential tremor controls. PBMCs were analyzed for six ALP-associated proteins (ATG5, ATP13A2, BAG3, LAMP1, OPTN, p62) using quantitative immunoblotting. Autophagic flux was assessed using chloroquine treatment and LC3A/B-II accumulation. Lysosomal pH was evaluated using LysoTracker Red DND-99 and flow cytometry. Statistical analyses included Mann-Whitney U tests, Spearman's correlation analysis, and ROC curve analysis.

Results:

OPTN levels were significantly reduced in PBMCs from PD patients compared to controls ($p < 0.05$). Strong positive correlations were observed among ALP-associated proteins, including ATG5 with BAG3 ($r = 0.817$), LAMP1 ($r = 0.774$), OPTN ($r = 0.871$), and p62 ($r = 0.829$), suggesting coordinated pathway dysregulation. ATP13A2 levels positively correlated with UPDRS-III motor scores ($r = 0.356$, $p = 0.042$). Autophagic flux showed positive correlation with age of disease onset ($r = 0.428$, $p = 0.010$). A multivariate bio-

marker model combining LAMP1 and OPTN achieved high diagnostic accuracy with AUC=0.860, sensitivity=81.8%, and specificity=86.1%.

Conclusions:

ALP-associated proteins in PBMCs, particularly OPTN, exhibit dysregulation in early-stage PD patients. The tight correlations among ALP components suggest systematic pathway dysfunction extending beyond the central nervous system. The LAMP1+OPTN biomarker model shows promising diagnostic potential for early PD detection, offering a minimally invasive approach for timely diagnosis before substantial neurodegeneration occurs.

Keywords

autophagy-lysosomal pathway, biomarkers, Parkinson's disease, peripheral blood mononuclear cells

P-022

Identification of Host Genes Modulating Dopaminergic Neurodegeneration Induced by Gut Bacteria-Derived Methylglyoxal

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Background & Objective:

Parkinson's disease (PD) is a progressive neurodegenerative dis-

order marked by dopaminergic neuron loss in the substantia nigra. While its etiology is incompletely understood, accumulating evidence implicates the gut microbiota in PD pathogenesis. To identify microbe-derived factors and host mediators that mechanistically link gut microbial activity to dopaminergic neurodegeneration in PD.

Methods

We combined: (i) microbial genetics using *Escherichia coli* lacking the histone-like nucleoid structuring protein (Δ hns); (ii) metabolite complementation with methylglyoxal (MG); (iii) in vivo neurodegeneration assays in *Caenorhabditis elegans* PD models; (iv) human cohort analyses assessing fecal hns gene abundance in two independent PD cohorts; (v) transcriptomics and RNAi screens to nominate host mediators; and (vi) cross-species validation, profiling ortholog expression in the substantia nigra of aged mice colonized with Δ hns *E. coli* and in MG-treated human neuronal cells, alongside PD patient tissue.

Results

Δ hns *E. coli* conferred neuroprotection in *C. elegans* PD models; this effect was abolished by MG. hns gene abundance was elevated in fecal samples from two independent PD cohorts. Transcriptomic/RNAi analyses identified rig-5 as a key host mediator. Mammalian orthologs—particularly LSAMP—were upregulated in the substantia nigra of aged mice colonized with Δ hns *E. coli*, but downregulated in PD patient SN and in MG-treated human neuronal cells. Together, these data delineate an hns–MG–LSAMP axis connecting microbial activity to host neuronal gene regulation and neurodegeneration.

Conclusion

We uncover a cross-kingdom pathway in which microbial hns and its metabolite MG modulate host LSAMP signaling to influence dopaminergic neurodegeneration. This hns–MG–LSAMP axis provides mechanistic support for the gut–brain link in PD and suggests testable biomarkers and therapeutic targets.

Keywords

Parkinson's disease (PD), gut microbiota, histone-like nucleoid structuring protein (hns), *Escherichia coli* (*E. coli*), methylglyoxal (MG), rig-5, LSAMP

P-023

Balance Biomarker for Early Differentiation of Parkinson's Disease and Multiple System Atrophy With Parkinsonian Type

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Background & Objective:

Parkinson's disease (PD) and multiple system atrophy with parkinsonian type (MSA-P) share early motor symptoms, complicating early differential diagnosis. Despite differences in progression and levodopa response, early differentiation is crucial for treatment planning. Early balance impairment is a potential distinguishing marker. We developed a machine learning model using balance analysis to differentiate early PD and MSA-P.

Methods:

We enrolled 22 healthy controls, 20 PD, and 17 MSA-P patients within three years of onset. Participants stood on two independent force plates (35×35 cm, accuracy 0.05 cm, 100 Hz) for 60 s. Center of pressure (COP) displacement was recorded for each foot and overall balance under eyes open (EO) and eyes closed (EC) conditions. Eight parameters per condition and EO–EC differences were analyzed. Group differences were assessed with ANOVA. Feature selection was performed using LightGBM (LGBM), followed by model training with 4-fold cross-validation.

Results:

Mean ages of healthy controls, PD, and MSA-P patients were 65.4, 64.2, and 68.8 years, respectively. Mean disease durations were 2.4 years for PD and 1.8 years for MSA-P. Mean UPDRS Part III scores were 15.2 for PD and 29.3 for MSA-P, and mean Hoehn and Yahr stages were 2.0 for PD and 2.9 for MSA-P. Except for mean distance in the anteroposterior direction with EO and 95% confidence ellipse area with EC, all parameters showed significant group differences. Post-hoc analysis revealed significant differences between healthy controls and PD/MSA-P in EO, whereas in EC, differences were more pronounced between MSA-P and PD/healthy controls. The LGBM model achieved 86.4% accuracy in classifying healthy controls, PD, and MSA-P, distinguishing PD from MSA-P with 71.2% accuracy and healthy controls from patient groups with 78.2% accuracy.

Conclusions:

Balance analysis with machine learning model could be used as a biomarker for early differentiation of PD and MSA-P.

Keywords

Parkinson's disease, Multiple system atrophy, balance, feature selection, machine learning

P-024

Uncovering Parkinson Disease Progression Related Biomarkers with Plasma Proteomics and Functional Genomics

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Background & Objective:

Parkinson disease (PD) is a progressive neurodegenerative disorder with limited therapeutic options. The lack of effective treatments stems partly from the absence of robust biomarkers. This study's objective was to discover novel plasma protein biomarkers related to progression and treatment response with a longitudinal PD cohort using an integrated approach of advanced proteomics and functional genomics.

Methods:

We combined Orbitrap Astral mass spectrometry with Mag-Net, a novel method for depleting abundant plasma proteins and enriching extracellular vesicles. This platform was applied to a longitudinal cohort of 10 PD patients with paired plasma samples collected at diagnosis and after 2-7 years of disease progression with treatment. We used dimensionality reduction and multivariate analysis to identify protein signatures. (Fig. 1) We also performed a functional genomics screen using a proteome-wide overexpression library to identify genes that modulate alpha-synuclein pre-

formed fibril uptake.

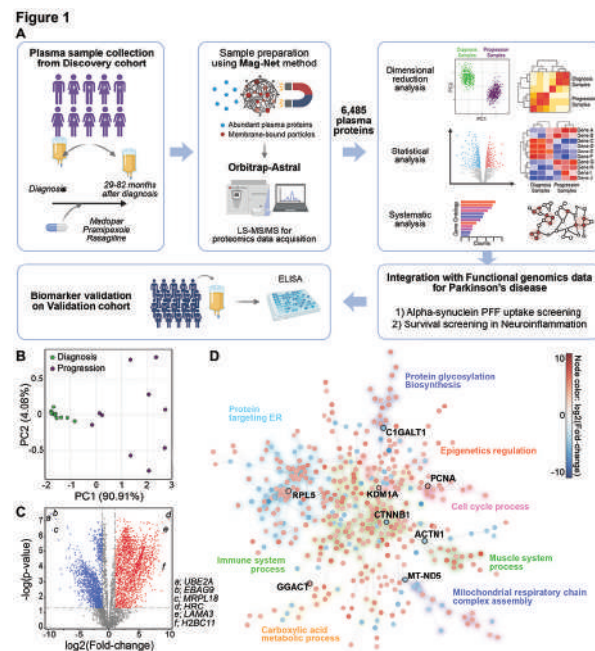
Results:

Our proteomics workflow identified an unprecedented 6,485 plasma proteins. Dimensionality reduction revealed distinct proteomic signatures that separated samples based on disease stage, a separation not possible with conventional proteomic depth. Multivariate analysis identified disease progression and levodopa equivalent daily dose (LEDD) as the primary factors influencing longitudinal proteomic changes. (Fig. 2) Functional genomics screening identified novel regulators of PFF uptake, including DERL1 (decreased PFF uptake) and NUDC (increased PFF uptake). (Fig. 3)

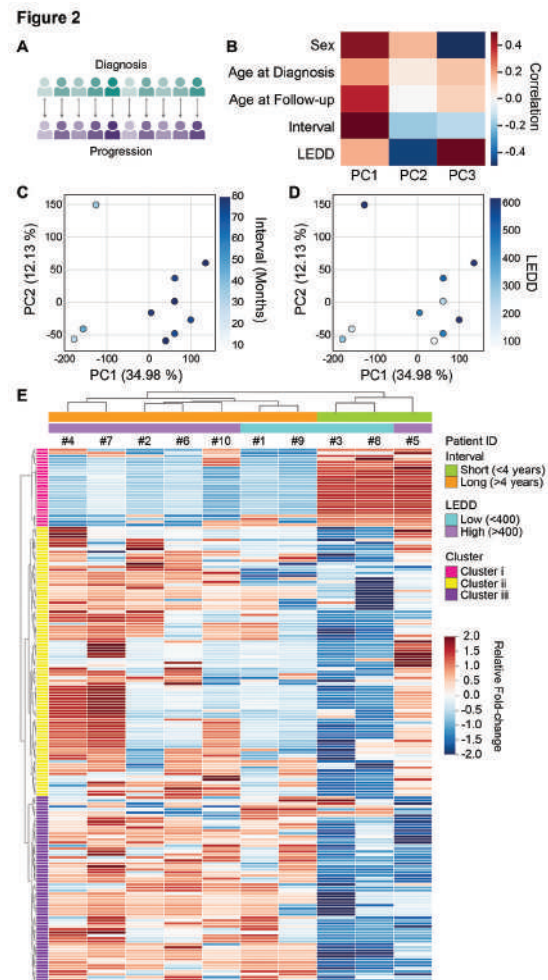
Conclusions:

This study demonstrates an integrated approach to identify new biomarkers and provide insights into PD. The identified biomarkers associated with both disease progression and levodopa treatment have translational potential in both disease monitoring and drug development. These findings lay the groundwork for developing precision diagnostics and personalized therapeutic strategies for Parkinson's disease.

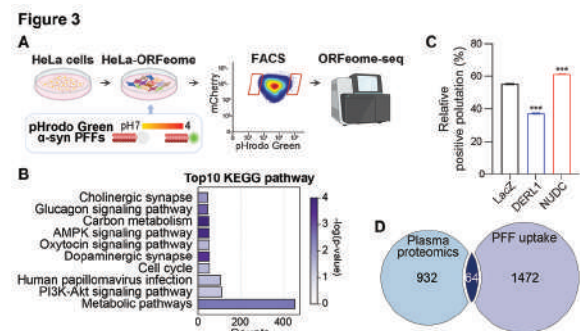
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Figure



Figure



Keywords

Parkinson disease, Biomarker, Proteomics, Mass spectrometry, Functional genomics

P-025

Longitudinal Trajectory of Multimodal Markers for Prodromal Disease Progression in a Patient With Isolated REM Sleep Behavior Disorder

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Introduction:

We report a patient with 20-year history of isolated REM sleep behavior disorder (iRBD) who developed severe neurogenic orthostatic hypotension (OH) highlighted by changes in multimodal imaging markers, thereby providing a novel insight into the progression of prodromal synucleinopathy.

Case:

A 64-year-old man presented to our clinic in 2013 with a preceding 9-year history of dream-enactment behaviors. His symptoms remained stable until 2016, when he began experiencing episodic orthostatic dizziness. These symptoms gradually worsened, clinical tests at that time were unremarkable. In 2021, OH was confirmed by the orthostatic BP tests. The abnormal findings and orthostatic symptoms progressed in 2023. At this time, he appeared to show mild parkinsonian signs and amnesic mild cognitive impairment with multiple domains. Multimodal imaging showed a progressive atrophy of central cholinergic nucleus from 2-year's follow-up and over the subsequent 10 years. A mild presynaptic dopaminergic terminal loss began to be detected at 4-year's follow-up. FDG-PET based Parkinson's disease-related pattern expressions showed an increase above the cut-off at 8-year's follow-up.

Discussion:

Our observation shows longitudinal trajectories in patients with longstanding iRBD, later developed severe neurogenic orthostatic hypotension. Markers showed progression of neurodegeneration in this case compatible with prodromal Lewy body disease rather

than prodromal multiple system atrophy. The trajectory may reflect the heterogeneity in region-specific vulnerability or thresholds against α -synuclein pathology.

Keywords

REM sleep behavior disorder, Neurogenic orthostatic hypotension, Multimodal markers

P-026

Plasma α -Synuclein Aggregates as a Non-Invasive Marker of Parkinson's Disease

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Background & Objective:

Parkinson's disease (PD) is a progressive neurodegenerative disorder characterized by dopaminergic neuronal loss and alpha-synuclein (α Syn) aggregation. Current diagnostic tools, such as clinical assessments and dopamine transporter imaging, are limited by subjectivity, invasiveness, and low specificity, underscoring the need for a reliable blood-based biomarker. This study aimed to evaluate the Constant Shake-Induced Conversion (CSIC) assay, a novel plasma-based method for detecting α Syn aggregates, focusing on its diagnostic performance and clinical applicability.

Methods:

A total of 138 participants were recruited from two centers: 68 idiopathic PD patients and 70 healthy controls. Plasma samples were analyzed using the CSIC assay, with validation by α Syn depletion, ELISA, and Western blotting. Diagnostic accuracy was assessed using receiver operating characteristic (ROC) curve analysis.

Results:

The CSIC assay detected α Syn seeds at femtogram levels within 48 hours. It distinguished PD from controls with an area under the ROC curve (AUC) of 0.8335 ($p < 0.0001$), sensitivity of 70.6%, and specificity of 92.9%. Reproducibility testing demonstrated

intra-assay variability below 10% and inter-assay variability of approximately 20%.

Conclusions:

The CSIC assay provides a sensitive and non-invasive approach for detecting plasma α Syn aggregates with high specificity. Its diagnostic performance supports its potential utility for PD diagnosis and disease monitoring. Further validation across broader synucleinopathies is warranted.

Keywords

Parkinson's disease, α -Synuclein, Plasma biomarker

P-027

The Development of α -Synuclein RT-QulC Assay Using Urine-Derived Neuronal Exosome.

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Background & Objective:

α -synuclein aggregation is a pathological hallmark of Parkinson's disease (PD). The prion-like property of aggregated α -synuclein has been utilized in the development of the diagnostic technique known as real-time quaking-induced conversion (RT-QulC). In addition to CSF based RT-QulC assay, recent studies have demonstrated the presence of α -synuclein aggregates in blood based neuronal extracellular vesicles (NEV) from PD patients. Building upon this observation, the application of RT-QulC to urine-derived NEV will be a fully non-invasive strategy for detecting pathogenic α -synuclein. In this study, we developed urine-derived NEV based RT-QulC assay to differentiating PD from healthy control.

Methods:

In this study, NEVs were isolated from 20 urine samples using L1CAM-conjugated magnetic beads. Four samples were initially used to modify and optimize the isolation method, and these EVs were characterized by evaluating exosomal and neuronal marker expression and morphology using immunoblotting and trans-

mission electron microscopy (TEM). Exosome abundance was quantified prior to applying the RT-QulC assay with an in-house recombinant monomeric α -synuclein. We assessed RT-QulC in 7 PD patients and 9 non-PD patients.

Results:

We successfully isolated NEV from urine, as confirmed by neuronal (CD81), exosomal (L1CAM, TUBB), and α -synuclein markers, as well as TEM, which revealed the characteristic lipid bilayer membrane structure. Our preliminary urine-derived NEV RT-QulC assay distinguished PD from non-PD patients with a sensitivity of 85.7% and a specificity of 77.7%. Six among seven PD samples tested positive, whereas two of the 9 control samples showed positivity.

Conclusions:

This preliminary study demonstrated the potential of urine-derived NEV as a novel and non-invasive source for RT-QulC-based detection of pathogenic α -synuclein in PD.

P-028

Diabetes Mellitus and Parkinson's Disease: Associations with Blood-Based Simoa Biomarkers and Clinical Features

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Background & Objective:

Blood-based biomarkers provide a practical alternative to cerebrospinal fluid (CSF) for monitoring neurodegeneration in Parkinson's disease (PD), but their low levels require ultra-sensitive assays such as single molecule array (Simoa). While prior studies linked Simoa biomarkers to PD features, few examined the role of diabetes mellitus (DM). Because DM may accelerate PD progression through systemic inflammation, we investigated its associations with biomarkers and clinical outcomes.

Methods:

Drug-naïve, early-stage PD patients and healthy controls (HC) were recruited from a biomarker collection database including blood and other specimens. Serum GFAP, NFL, T-tau, UCHL1, and BDNF were measured using Simoa. Demographic and clinical features were summarized, and regression and mediation analyses were conducted adjusting for age, sex, disease duration, and education.

Results:

The study included 92 PD patients and 37 HC. Age (PD: 67.5 [61–74.8] vs. HC: 67 [63–72]) and sex distribution (56.5% vs. 70.3% female) did not differ. Log-transformed GFAP and NfL were elevated in PD compared with HC. In regression models, DM was associated with higher MDS-UPDRS part III scores ($\beta = 7.807$, $p = 0.02$) and lower K-BNT scores ($\beta = -0.586$, $p = 0.039$). DM also predicted higher log-transformed tau ($\beta = 0.523$, $p = 0.017$), which was inversely related to attention ($\beta = -0.252$, $p = 0.025$). Mediation analysis showed DM effects on motor and language were explained only by total/direct effects, whereas attentional decline was mediated indirectly via tau.

Conclusions:

DM contributes to motor and cognitive dysfunction in PD through distinct pathways: direct effects on language and motor outcomes, and tau-mediated effects on attention. Ultra-sensitive blood-based biomarker assays may clarify how systemic comorbidities influence domain-specific neurodegeneration in PD.

Keywords

Biomarker, Simoa, Diabetes

P-029

Parkinson's Disease GWAS-Linked SNCA Carriers Show Slower Non-Motor and Cognitive Progression

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Background & Objective:

A genome-wide association study-linked variant (SNCA rs6826785) was shown to have protective effect of cognitive performance among PD patients in our previous study. However, evidence on the longitudinal outcomes of SNCA rs6826785 carriers remains unknown. To investigate the disease progression between SNCA rs6826785 carriers and noncarriers over 5 years.

Methods:

Early PD patients diagnosed within one year were categorized into SNCA rs6826785 variant carriers and non-carriers. Non-motor symptom progression was evaluated using the Non-Motor Symptom Scale (NMSS), while comprehensive cognitive progression was assessed across five cognitive domains annually. Longitudinal linear mixed models were used to compare changes in

NMSS total and domain scores, as well as cognitive domain performance, between the two groups. To account for multiple comparisons, the false discovery rate (FDR) method was applied, and q-values were calculated, with a significance threshold set at 0.1.

Results:

A total of 194 PD patients, including 164 SNCA rs6826785 carriers and 30 non-carriers were assessed prospectively over a 5-year period. SNCA rs6826785 carriers, compared to noncarriers, had a significantly lower progression rate in NMSS score (carriers vs non-carriers: 2.195 vs 5.951, $p=0.047$, $q=0.047$), NMSS domain 4 (Perceptual problems/hallucinations) score (carriers vs non-carriers: 0.088 vs 0.709 $p=0.0152$, $q=0.045$) and NMSS domain 9 (Miscellaneous) score (carriers vs non-carriers: 0.285 vs 1.469, $p=0.018$, $q=0.028$). SNCA carriers also exhibited significantly slower deterioration in cognitive language domain (carriers vs non-carriers: -0.009 vs 0.096, $p=0.016$, $q=0.082$) after adjustment for confounders and multiple comparisons.

Conclusions:

Carriers of the SNCA rs6826785 variant exhibited a slower decline in non-motor and cognitive functions, particularly in perceptual processing and language performance. These findings suggest that SNCA might serve as a potential therapeutic target.

Table

Table 1. Demographic and Clinical characteristics at baseline

	Non-carriers n=30	SNCA carriers n=164	† p-value
Ethnicity: Chinese	22(73.3%)	143(87.7%)	0.076
Age of onset (year)	62.7 (11.6)	62.7 (8.9)	1.00
Age of diagnosis (year)	63.7 (11.3)	63.4 (8.8)	0.88
Disease duration (year)	1.8 (1.8)	1.5 (0.7)	0.16
Sex: male	23(76.7%)	92(56.4%)	0.061
Education (Year)	11.0 (3.3)	10.6 (4.6)	0.65
Family History: Negative	20 (80.0%)	99 (83.9%)	0.13
LEDD at baseline	166.9 (115.9)	199.4 (141.9)	0.27
MoCA Adjusted Total score	25 (22-28)	26 (23-28)	0.53
MCI (%)	16 (53.3%)	83 (50.6%)	0.84
NMSS Total Score	13 (6-25)	14.5 (9-26.5)	0.54
Total motor score	19 (14-24)	20 (15-26.5)	0.25
Diabetes Mellitus	8 (26.7%)	26 (15.9%)	0.19
Hyperlipidemia	12 (40.0%)	78 (47.6%)	0.55
Hypertension	11 (36.7%)	79 (48.2%)	0.32

Abbreviations: LEDD: Levodopa equivalent daily dose; MoCA score: Montreal Cognitive Assessment score; MCI: mild cognitive impairment; NMSS: non-motor symptom scale;

Categorical variables reported as frequency (%); continuous variables reported as mean ± standard deviation or median and first and third quartile (where appropriate)

† Fisher exact test and two-sample t test or Mann Whitney U test (where appropriate) for categorical and continuous variables, respectively

Table

Table 2: Comparison of NMS and cognitive progression rates between two groups

Longitudinal outcomes	Group	Slope Estimate	95% CI		† p-value	†† q value
			Lower	Upper		
Non-motor						
NMSS total score	0	5.952	2.435	9.468	0.047*	0.047*
	1	2.195	0.992	3.399		
NMSS domain 4 score	0	0.709	0.236	1.183	0.015*	0.045*
	1	0.088	-0.076	0.253		
NMSS domain 9 score	0	1.469	0.5400	2.399	0.018*	0.028*
	1	0.285	-0.037	0.608		
Cognitive						
Memory score	0	0.066	-0.014	0.146	0.3019	0.503
	1	0.112	0.079	0.143		
Visuospatial score	0	-0.109	-0.209	-0.008	0.381	0.476
	1	-0.060	-0.099	-0.0209		
Attention score	0	0.0097	-0.090	0.110	0.659	0.659
	1	-0.014	-0.052	0.024		
Executive score	0	-0.072	-0.1401	-0.005	0.182	0.455
	1	-0.023	-0.049	0.004		
Language score	0	0.096	0.015	0.176	0.016*	0.082*
	1	-0.009	-0.039	0.021		

Abbreviations: NMSS: non-motor symptom scale; NMSS domain 4 (perceptual problems/hallucinations); NMSS domain 9 (miscellaneous symptoms)

† The trend of longitudinal cognitive outcomes from baseline to follow-up visits were examined by means of longitudinal, linear mixed model, adjusting for age, sex, and education year

†† False discovery rate (FDR) method was performed and q values were calculated to control for multiple testing and the threshold of q values was set as 0.1.

Figure

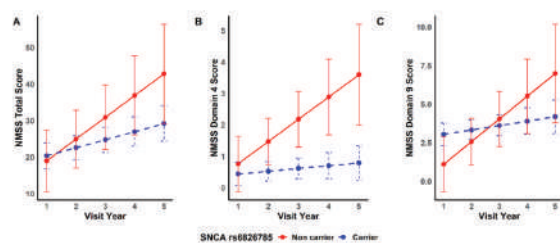


Figure 1: Non-motor progression between SNCA rs6826785 carrier and non-carrier

Dystonia

P-030

X-Linked Dystonia Parkinsonism as a Cause of Isolated Lingual Dystonia

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Introduction:

Dystonia is a movement disorder that causes sustained or intermittent muscle contractions, leading to abnormal posture or repetitive movements. Isolated lingual dystonia (ILD) is a rare subtype of OMD that only affects the tongue and accounts for about 4% of all dystonia cases. This report describes two Filipino males, aged 43 and 48, who developed ILD.

Case:

The first case is a 43-year-old male from Cebu, with no significant family history or roots from Panay Island, was found to carry the X-linked dystonia parkinsonism (XDP)-causing mutation highlighting a novel presentation of ILD linked to XDP. The second case is a 48-year-old male from Roxas City, with a family history suggesting XDP, who presented with ILD initially and later on developed tremor, limb dystonia, and bradykinesia, and then confirmed on genetic testing with XDP.

Discussion:

These cases highlight the importance of considering XDP in the differential diagnosis of ILD among Filipinos, regardless of geographic or familial predispositions. Genetic testing and follow-up examinations are crucial for managing ILD, as it may precede more generalized dystonic and parkinsonian symptoms associated with XDP. This study contributes to the limited literature on ILD and suggests new insights into the clinical variability of XDP.

Figure

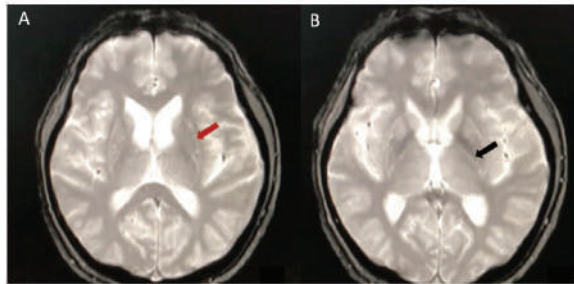
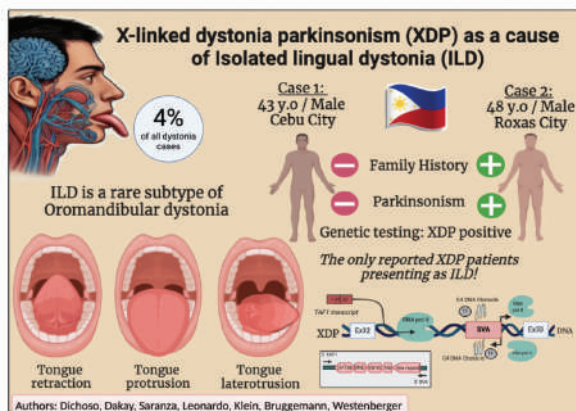


Figure 1. Neuroimaging findings in Case 1. Cranial MRI (Gradient echo sequence) in axial cut showed hyperintensities in the putamen rim (A) and pallidum rim (B).

Figure



Keywords

Isolated lingual dystonia (ILD), X-linked dystonia parkinsonism (XDP)

P-031

Dystonic Tremor in Writer's Cramp Mimicking Primary Handwriting Tremor

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Introduction:

Writer's cramp is a task-specific focal dystonia with peculiarity of occurring only during handwriting. The presence of abnormal posturing and dystonic tremor may exhibit distinctive patterns across

individuals, and may closely resemble primary writing tremor, especially when dystonic postures are mild.

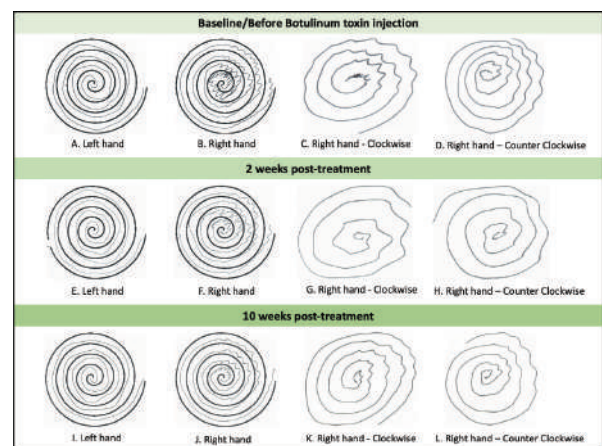
Case:

We present a 48-year-old right-handed Filipino businessman who developed a gradually worsening right-hand tremor confined to handwriting over one year, stabilizing after six months. Neurological examination was otherwise normal, but writing tests revealed dystonic wrist extension and shoulder abduction, along with wrist pronation-supination dystonic tremor during writing. Wavy/oscillating drawing patterns were observed. Diagnosis of writer's cramp was made, which mimicked primary writing tremor due to overlapping features. Multiple oral medications were ineffective, but ultrasound-guided botulinum toxin injections led to clinical improvements, with better handwriting during faster writing and worse at slower speeds.

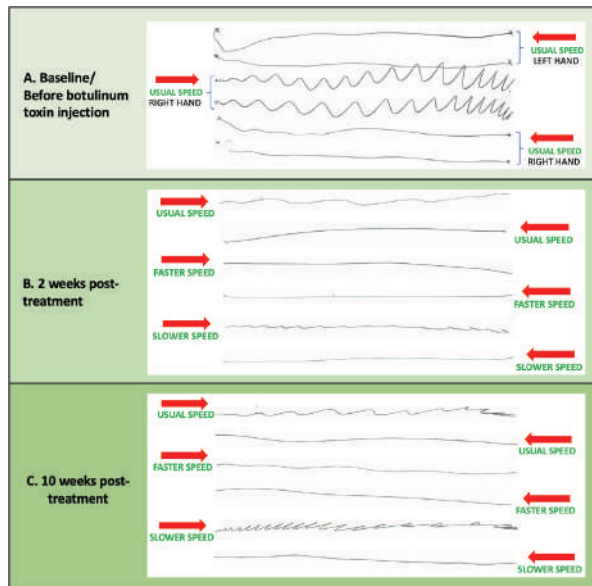
Discussion:

This case underscores the complexity of diagnosing and treating task-specific dystonia with tremor. Incorporating tests like line-drawing at different speeds, including vertical and diagonal lines, can enhance evaluation.

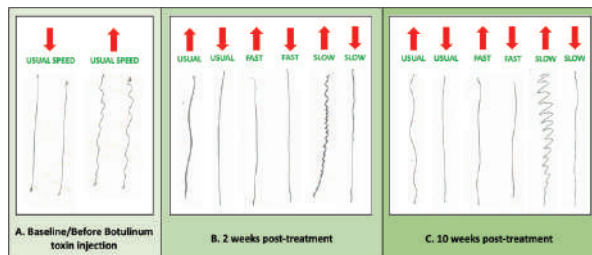
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Figure



Figure



Keywords

writer's cramp, task-specific dystonia, dystonic tremor, primary writing tremor

P-032

Effects of Deep Brain Stimulation on Regional Cerebral Blood Flow in Cervical Dystonia

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Background & Objective:

Cervical dystonia is considered as a network disorder affecting various brain regions in recent days. Presumably, deep brain stimulation (DBS) of the internal segment of globus pallidus (GPi) may exert therapeutic effects for cervical dystonia through modulation of the aberrant brain networks. In the present study, we investigated postoperative regional cerebral blood flow (rCBF) changes after GPi DBS using single-photon emission computed tomography (SPECT) to identify significant activity changes in several relevant brain areas of cervical dystonia patients.

Methods:

A total of 9 patients with idiopathic cervical dystonia were recruited, and SPECT scans were conducted at baseline and 3 months after the bilateral GPi DBS. Voxel-wise changes of rCBF were analyzed using Statistical Parametric Mapping. Symptom severity of dystonia was measured using Toronto Western Spasmodic Torticollis Rating Scale (TWSTRS) at the baseline, and 1 week, and 3 months after GPi DBS.

Results:

At the 3-month follow-up after DBS, rCBF was increased in the left pons and right postcentral gyrus and decreased in the left middle frontal gyrus, left cerebellum, right putamen and pallidum, and left thalamus ($p < 0.001$). Severity of cervical dystonia assessed by TWSTRS was significantly decreased at 1-week and 3-month follow-up ($p = 0.004$).

Conclusions:

Our findings suggest that therapeutic effect of GPi DBS may be associated with changes of activities in the multiple brain areas in the cortico-basal ganglia-cerebellar network. Further longitudinal studies with larger samples and longer follow-up periods are needed to elucidate more detailed neurobiological mechanisms of DBS in cervical dystonia. In addition, associations between rCBF and symptom improvement should be explored to find out which brain regions are more important for the DBS-related functional changes and whether SPECT imaging could be useful to predict the therapeutic effects of DBS in cervical dystonia.

Keywords

Cervical dystonia,, Deep brain stimulation, Regional cerebral blood flow

P-033

Fahr's Syndrome Presenting With Truncal Dystonia in Chronic Idiopathic Hypoparathyroidism

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Introduction:

Fahr's syndrome is a rare, progressive neurological disorder characterized by abnormal, bilateral, and symmetrical basal ganglia calcification. It represents the secondary form of brain calcification, resulting from various metabolic, infectious, or degenerative causes. A broad spectrum of movement disorders has been associated with Fahr's syndrome, including parkinsonism, chorea, tremor, dystonia, athetosis, and orofacial dyskinesia, but truncal dystonia has rarely been reported.

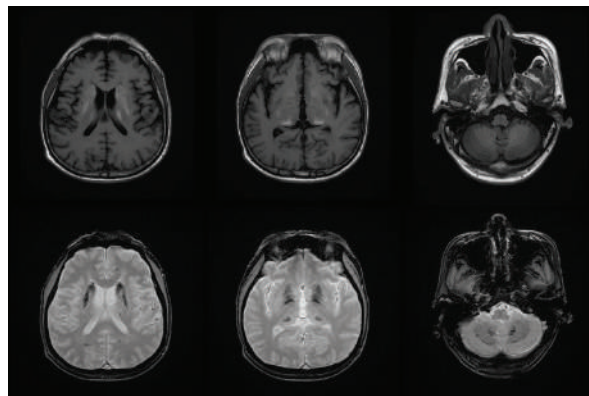
Case:

A 51-year-old Korean male visited the outpatient clinic with a one-month history of abnormal truncal turning. Examination revealed sustained rightward truncal rotation partially relieved by voluntary movement, with asymmetric dystonic posturing of the upper limbs, more prominent on the right. A mild postural tremor was observed without abnormal hand posturing. He had a 10-year history of chronic hypoparathyroidism on long-term calcium therapy without recent changes, and hypertension controlled with medication. Laboratory tests showed hypocalcemia (serum calcium 5.2 mg/dL, reference range: 8.6–10.0; ionized calcium 1.28 mmol/L, reference range: 2.30–2.59), hyperphosphatemia (5.2 mg/dL, reference range: 2.5–4.5), and markedly reduced parathyroid hormone (2.2 pg/mL, reference range: 15–65). Magnetic resonance imaging demonstrated bilateral basal ganglia, thalamus, and dentate calcification with low signal intensities on GRE and high signal intensities on T1-weighted sequences. These findings were consistent with Fahr's syndrome secondary to primary hypoparathyroidism. After intravenous calcium and oral calcitriol, alfacalcidol, and calcium supplementation, calcium levels normalized with partial improvement of dystonia.

Discussion:

We describe a case of Fahr's syndrome with truncal dystonia due to idiopathic hypoparathyroidism. Although uncommon, this case highlights the importance of evaluating metabolic abnormalities in movement disorders and suggests that their correction may aid symptomatic improvement.

Figure



Keywords

Fahr's syndrome, Dystonia, Basal ganglia calcification, Hypoparathyroidism, Calcium

P-034

Uncommon Movement Disorders in Neuropsychiatric Systemic Lupus Erythematosus: A Case of Dystonia and Tremor

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Introduction:

NPSLE is typically presents with headaches, mood disorders, and seizures, the presence of movement disorders is rare (estimated incidence of 0.9%). Identifying movement disorder as a sign of NPSLE is essential for accurate diagnosis and effective treatment, as it can significantly affect a patient's quality of life and complicate the clinical picture of lupus.

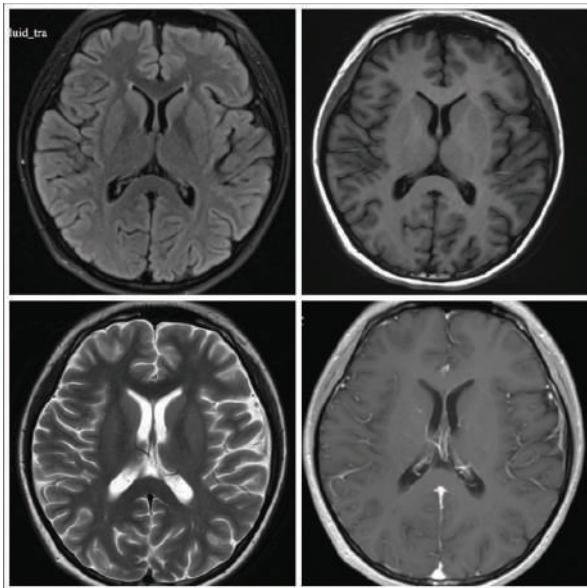
Case:

A 16-year-old girl presented to the hospital with a two-month history of speech difficulties, stuttering, and tongue tremors. She also experienced intermittent hand stiffness and tremor during movement, along with widespread muscle pain and arthralgia, which were exacerbated by anxiety or stress. Routine laboratory tests, including electrolyte levels, were normal. However, serological tests revealed positive antinuclear antibodies (ANA) at a 1:100 titer and RNP/SM antibodies. Head MRI and electrophysiological studies were normal. She was initially treated with Levodopa-benserazide, and trihexyphenidyl. However, her dystonia worsened, leading to a change in treatment to high-dose methylprednisolone, methotrexate, trihexyphenidyl and clonazepam, which resulted in significant improvement.

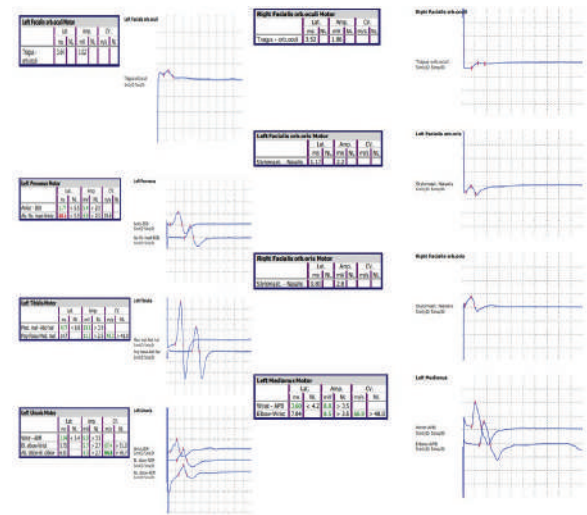
Discussion:

This case highlights dystonia as a rare but important manifestation of NPSLE. The presence of positive ANA and RNP/SM antibodies, along with the absence of other neurological abnormalities, supports the diagnosis of NPSLE-associated dystonia. The patient's initial worsening symptoms with standard treatments underscore the challenges in managing movement disorders in NPSLE. The subsequent improvement with trihexyphenidyl and clonazepam suggests that tailored therapeutic approaches are essential for optimizing outcomes in such cases.

Figure



Figure



Keywords

Dystonia , NPSLE, RNP/SM Antibodies

P-035

SPECT Cerebral Perfusion Patterns in Isolated Cervical Dystonia

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Background & Objective:

Isolated cervical dystonia is characterized by sustained or intermittent neck movements caused by involuntary muscle contractions, resulting in abnormal movements or postures of cervical areas. Despite extensive research, the pathophysiology remains poorly understood. We measured altered regional cerebral blood flow (rCBF) in patients with idiopathic cervical dystonia compared to healthy controls, and explored the correlation between dystonia severity and rCBF patterns.

Methods:

This study was designed as a case-control study. A total of 38 patients with isolated cervical dystonia and 29 healthy controls were recruited. All subjects underwent Toronto Western Spasmodic Torticollis Rating Scale (TWSTRS) and Tc-99m hexamethyl propylene amine oxime perfusion brain SPECT; TWSTRS was used to assess the severity of dystonia. rCBF was compared between groups using voxel-wise statistical analyses. Associations

between rCBF in significant clusters and TWSTRS scores were examined using linear regression.

Results:

Results Compared with controls, patients with cervical dystonia exhibited reduced rCBF in brain regions implicated in sensory processing, motor control, and higher cognitive functions including the left middle temporal gyrus, right postcentral gyrus, right lingual gyrus, left precuneus, right temporal pole, right middle cingulate gyrus, and right anterior cingulate gyrus (Table 1 and Fig 1). Among the seven regions with reduced rCBF, only the right temporal pole showed a negative correlation with TWSTRS scores in dystonia patients. (Fig 2).

Conclusions:

The negative correlation between reduced rCBF in the right temporal pole and increased TWSTRS scores highlights a potential association between altered cerebral perfusion and dystonic symptom severity. Understanding these alterations could offer insights into the neurobiological basis of cervical dystonia, aiding targeted therapeutic interventions. However, further studies are needed for a definitive conclusion.

Table

TABLE 1 | Demographic and clinical characteristics of study participants.

Characteristics	Dystonia (n = 38)	Control (n = 29)	Test
Age (year)	52.9 ± 10.2	55.8 ± 9.2	t = 0.78, p = 0.44
Sex (male/female)	18/20	12/17	p = 0.27
Toronto Western Spasmodic Torticollis Rating Scale	43.9 ± 11.0	—	—

Note: Data are presented as mean ± standard deviation or n (number of patients).

Figure

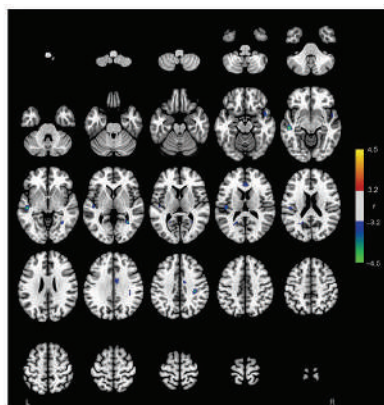
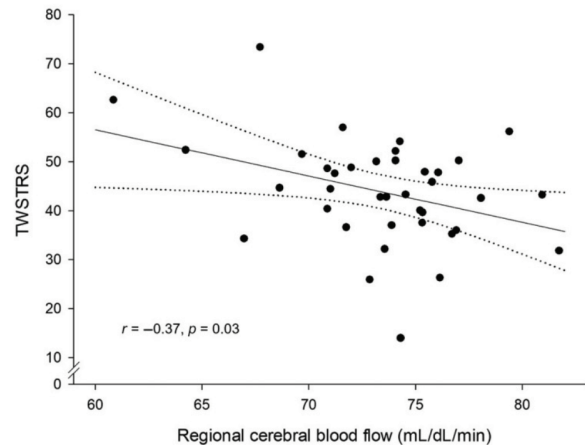


FIGURE 1 | Higher (red-yellow) or lower regional cerebral blood flow (blue-green) in cervical dystonia patients versus healthy controls. Images are shown in neurological convention. The detailed cluster information is shown in Table 2. R, right; L, left.

Figure



Keywords

cervical dystonia, single photon emission computed tomography, cerebral blood flow

P-036

Acute Dystonia Induced by Salbutamol in a Young Adult With a History of Neuroleptic Consumption: A Diagnostic Challenge

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²Neurology, Brigjend H Hasan Basry Regional Hospital, Kandangan, Indonesia

Introduction:

Drug-induced dystonia is often misdiagnosed as a psychiatric condition, with disabling manifestations, impacting patient's quality of life and social functioning.

Case:

An 18-year-old female came with neck rigidity and left-sided head tilt since 1 hour ago after consuming salbutamol for her asthma. She had a history of asthma for 7 years, and consumed salbutamol during asthma attacks 3-4 times monthly. There was no trouble chewing, swallowing, speaking, or motor weakness. No history of tremors, gait disturbances, or fever. No family history of dystonia. She was not on any antipsychotic or other medication recently,

and was not an alcoholic. However, she was diagnosed as having major depressive disorder and pseudoseizure in 2023, and prescribed risperidone, sertraline, and clobazam. She only took the medicine for 3 months and not regularly. On examination, there was a severe head tilt to the left side, likely cervical dystonia. There was also intermittent, non-rhythmic involuntary movement of the left lower part of the face in the perioral region, characterized by facial twitches, with occasional deviation of the mouth's angle to the left side, most likely orofacial dystonia. The other neurological examination was normal. The laboratory result was normal. The symptoms improved after stopping the salbutamol. The patient was given trihexiphenidyl and clonazepam.

Discussion:

There are two hypotheses of acute dystonia, the dopamine hypo-function resulting in a relative overactivity of cholinergic mechanisms. Conversely, neuroleptics induce a paradoxical dopaminergic hyperfunction. The patient's chronic asthma enhanced axonal hyperexcitability as a result of hypoxic insults and hypocapnia from hyperventilation. Salbutamol affects the sodium–potassium pump in skeletal muscles, which might also contribute to axonal hyperexcitability. We also include tardive dyskinesia as a differential diagnosis, because the patient's history of consuming antipsychotics and antidepressants 2 years ago.

Keywords

acute dystonia, salbutamol, B-2 adrenergic

P-037

Effective Treatment of Debilitating Tardive Dyskinesia/Dystonia With Globus Pallidus Deep Brain Stimulation: A Case Report

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Introduction:

Tardive dyskinesia/dystonia (TD) is an abnormal movement disorder occurring after the administration of dopamine receptor blocking agents. TD is characterized by irregular, involuntary movements, primarily involving the tongue, lips, jaw, and face, and occasionally the trunk, limbs, or peri-orbital area. In some patients, TD becomes severely disabling and causes significant distress and stigma. Pharmacological treatment of TD remains difficult and often produces suboptimal results. Surgical treatment may be considered for patients with severe TD who do not

respond to medical therapy. We report a patient with intractable TD, presenting with severe orolingual dyskinesia and cervical dystonia, successfully managed with globus pallidus (GPi) deep brain stimulation (DBS).

Case:

A 67-year-old woman had a long history of psychiatric illness, including depression and delusional disorder, for which she had been taking antipsychotic medications for over two decades. She was referred to the neurology department due to the development of orolingual dyskinesia. Olanzapine, identified as a potential offending agent, was discontinued, but her symptoms did not improve. To manage worsening delusions, aripiprazole was initiated, but dyskinesia progressively worsened, accompanied by cervical dystonia characterized by backward head extension, along with akathisia. Neurologic reevaluation led to the discontinuation of all antipsychotics, and a multidisciplinary approach was adopted in coordination with psychiatry. Genetic testing for Huntington's disease was negative. Despite medication withdrawal, the dyskinesia advanced to involve the entire body, leading to significant functional impairment. The patient had difficulty performing basic daily activities independently, such as tooth brushing and eating while seated. In addition, she was unable to ambulate normally. Tetrabenazine treatment was initiated during psychiatric admission and showed temporary improvement, but symptoms relapsed soon thereafter. Electroconvulsive therapy was then administered over multiple sessions. While mild improvement was noted after the first two treatments, the overall benefit was not sustained. Eventually, the patient was referred back to neurology with severe, medication-refractory TD. The Burke-Fahn-Marsden dystonia rating scale (BFM) motor score was evaluated as 16 points. Given the intractable nature of her symptoms and poor response to conventional therapies, she was evaluated for surgical treatment with GPi DBS. After GPi DBS, there was a significant improvement in the patient's orolingual dyskinesia and cervical dystonia. Her BFM motor score was reduced to 2. The patient became able to perform daily activities and walk independently.

Discussion:

GPi DBS may offer significant symptomatic relief in patients with severe, drug-resistant tardive dyskinesia. Early referral and multidisciplinary evaluation are essential for optimal outcomes in complex cases.

P-038

Unexpected Extrapyramidal Reaction: Acute Tardive Dyskinesia Following Short-Term Metoclopramide Use in a High-Risk Patient

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Introduction:

Tardive dyskinesia (TD) is an involuntary movement disorder linked to dopamine receptor blocking agents (DRBAs), commonly antipsychotics but also gastrointestinal prokinetics such as metoclopramide. It predominantly affects the orofacial region and is associated with risk factors including female sex, advanced age, diabetes, and renal impairment. Although largely preventable, TD is often underrecognized outside psychiatric settings.

Case:

A 49-year-old woman with poorly controlled type 2 diabetes and chronic kidney disease (eGFR 22 mL/min/1.73m²) presented with nausea and vomiting. Laboratory tests showed normocytic anemia and HbA1c 9.2%. She was treated with fluids, proton-pump inhibitor, electrolyte correction, and intravenous metoclopramide at standard dosing. Within 24 hours, she developed repetitive tongue protrusion and orofacial dyskinesia consistent with TD. Neurological examination was otherwise normal, brain CT showed no acute lesion, and electrolytes were within reference. She had no prior history of antipsychotic exposure. Metoclopramide was discontinued, supportive therapy initiated, and ondansetron substituted. Dyskinesia gradually resolved, with full recovery by the second week of hospitalization.

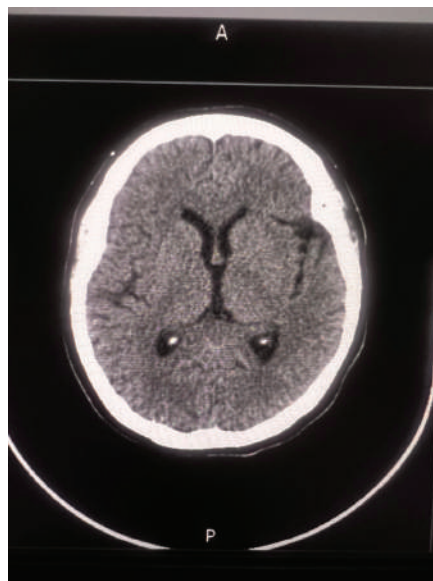
Discussion:

This case underscores that TD may arise after even short courses of metoclopramide, particularly in individuals with risk factors such as diabetes and renal dysfunction. Reduced clearance in kidney disease can enhance dopaminergic blockade and trigger extrapyramidal effects. While TD is classically associated with chronic exposure, acute onset presentations are increasingly reported. Awareness in non-psychiatric practice is essential. In conclusion, Metoclopramide-induced TD can occur after brief exposure in high-risk patients. Vigilance, dose adjustment in renal impairment, and prompt drug discontinuation are key to preventing this disabling complication.

Figure



Figure



Keywords

Tardive Dyskinesia , Metoclopramide , Extrapyramidal, Movement Disorder, High Risk

P-040

Myoclonus-Dystonia Associated with *CAPN3* Variant: Expanding the Phenotypic Spectrum Beyond LGMD

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Introduction:

Mutations in *CAPN3*, encoding calpain-3, cause autosomal dominant or recessive limb-girdle muscular dystrophy type 2A (LGMD2A). Neither dystonia nor myoclonus have been considered part of the *CAPN3*-related phenotype. We present a family with a heterozygous *CAPN3* splice-site variant manifesting as myoclonus-dystonia, broadening the phenotypic spectrum of *CAPN3* mutation.

Case:

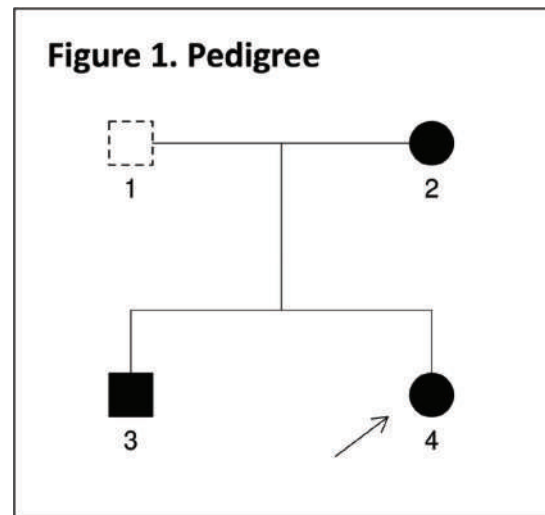
The proband, a 42-year-old woman, was born preterm but had normal early development. In her early 20s, she developed hand clumsiness, involuntary head tilting, and progressive left-predominant generalized dystonia with action-induced myoclonus. Manual muscle testing revealed mild elbow extension weakness, but tendon reflexes were intact. Electromyography showed negative myoclonus, and MRI was unremarkable. Symptoms were partially controlled with valproic acid, clonazepam, and trihexiphenidyl. Her mother developed a similar phenotype in childhood, while her older brother showed a more severe adult-onset course. Given the clear autosomal dominant family history, genetic testing was pursued. Targeted analyses for *TOR1A*, SGCE, and mitochondrial mutations were negative. Whole genome sequencing revealed a heterozygous *CAPN3* splice-site variant (NM_000070.3:c.946-2A>G), which segregated with disease in the proband and her mother.

Discussion:

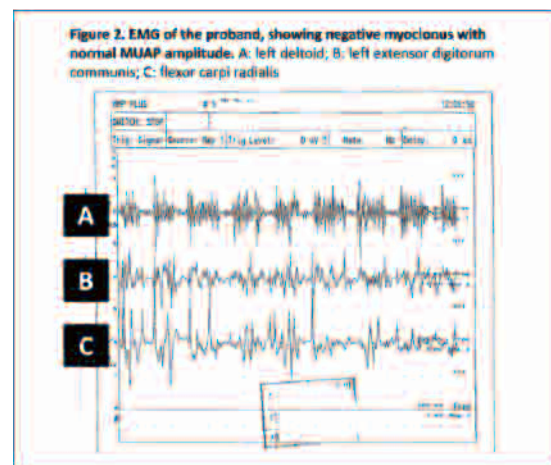
This family demonstrates that CAPN3 variants can present predominantly with myoclonus-dystonia rather than classic muscular dystrophy. In LGMD2A, calpain-3 deficiency destabilizes the dystrophin-gly-

coprotein complex in muscle. Prior studies have detected calpain-3 in astrocytes, supporting a possible neurogenic role. Our findings suggest a novel pathophysiological link between CAPN3 and dystonia, with implications for the interplay between calpain-3 and sarcoglycan complexes in both muscle and brain.

Figure



Figure



Keywords

Myoclonus Dystonia, Genetic dystonia, CAPN3, Limb-girdle muscular dystrophy

P-041

Adult-Onset DYT-KMT2B in a Filipino Male: Pathogenicity of a Novel De Novo p.Thr1650Met Variant

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Introduction:

DYT-KMT2B is predominantly an early-onset form of dystonia caused by truncating variants in the *KMT2B* gene. Patients with adult onset are rare and are often associated with missense variants, for which pathogenicity is challenging to ascertain (variants of uncertain significance, VUS).

Case:

A 47-year-old Filipino male developed progressive focal and generalized dystonia beginning at the age of 33 years. Comprehensive neurologic evaluation, targeted genetic sequencing, and DNA methylation profiling were performed. Family members underwent segregation analysis to evaluate inheritance and variant origin. Whole exome sequencing revealed a heterozygous c.4949C>T (p.Thr1650Met) missense variant in *KMT2B*, initially interpreted as VUS. DNA methylation epigenotype analysis demonstrated a characteristic hypermethylated pattern in the patient supporting a disease-causing relevance. Further, by haplotype analysis of three unaffected, mutation-negative family members (mother and two brothers, father was not available) using eight microsatellite markers, we demonstrated that the variant arose de novo. This enabled reclassification of the variant as pathogenic (Class 5). Clinical management with standard oral medications and chemodestruction resulted in partial improvement; advanced interventions were restricted due to socioeconomic considerations.

Discussion:

This case spotlights a novel pathogenic, de novo missense *KMT2B* variant as the cause of adult-onset dystonia, expanding the known genetic spectrum of DYT-KMT2B. It highlights

the clinical utility of methylation profiling for clarifying ambiguous genetic findings and underscores the importance of family studies in confirming variant origin. Early recognition of de novo variants through advanced genetic and epigenetic testing is essential for diagnosis, counseling, and personalized management in rare movement disorders.

P-042

Dysphagia And Hyphonia as Initial Presentation of XDP: A Case Report

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Introduction:

X-linked Dystonia Parkinsonism is an endemic disease in the Philippine island of Panay with variable clinical features. Typically, dystonia occurs focally, affecting the limbs, jaw, neck, trunk, and eyes. This paper aims to emphasize unique symptoms that patients can initially manifest with.

Case:

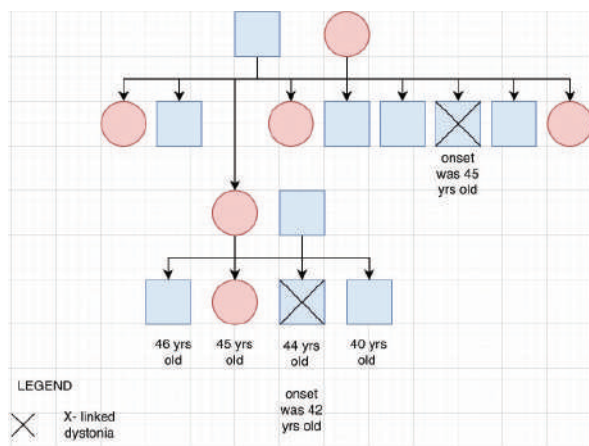
We report a 44-year-old Filipino male who initially presented with hyphonia and dysphagia. Initially thought to be Amyotrophic Lateral Sclerosis, EMG-NCV was done but revealed normal results. In a span of 2 years, dystonic posturing was observed affecting the left leg more than the right, and speech became incomprehensible. He had jaw opening dystonia and right lateral torticollis. The left sternocleidomastoid appeared hypertrophied while the legs were atrophied. Parkinsonism was noted on bradykinetic finger tapping and alternating hand movements. Family history revealed an uncle to have X-linked Dystonia Parkinsonism. Their geographical ancestry was traced to Roxas, Panay. The patient underwent genetic testing revealing SVA retrotransposon insertion in TAF1/DYT3 confirming the diagnosis of X-linked dystonia parkinsonism. Currently maintained on Levodopa + Carbidopa 300mg/75mg/day and Biperiden 6mg/day.

Discussion:

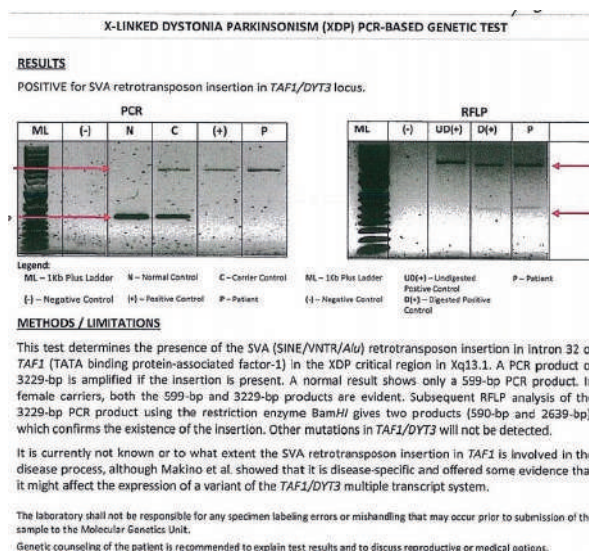
X-linked dystonia-parkinsonism (XDP) afflicts primarily adult Filipino men. The initial clinical course is variable with the majority of dystonic postures occurring on the lower extremities. In our case, presented with bulbar impairment only comprises 28% of the total cases recorded. Laryngeal dystonia is due to poor palatal movement and hypernasality. The hypophonia is due

to affectation of adductor muscles. Contractions of the tongue lead to dysphagia. Previous linkage and association studies have demonstrated that the XDP locus is within a 427 kb genetic interval on chromosome Xq13.1 but as to what muscle group to be affected first, data still lacking. The cornerstone treatment is still mainly symptomatic and supportive depending which symptom is predominant.

Figure



Figure



Keywords

XDP, Dystonia, Dysphagia, Hyphonia, DYT3

P-043

Clinical Characteristics of Patients With Oromandibular Dystonia at Ngoerah General Hospital Denpasar

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Background & Objective:

Dystonia is a movement disorder characterized by persistent involuntary muscle contractions and often causes an abnormal body posture. Oromandibular dystonia is a focal dystonia that involves the tongue, jaw, and mouth area. It is rarely reported, comprises around 3-5% of all types of dystonia. Delayed in making a diagnosis are quite common. This study aims to determine the demographic and clinical characteristics of patients with oromandibular dystonia.

Methods:

This research is a descriptive study with a cross-sectional method to subjects diagnosed with oromandibular dystonia at Ngoerah General Hospital Denpasar from August 2022 to March 2024. Data was taken from medical records, including age, gender, etiology, distribution area, and given treatment.

Results:

Seventeen patients (7 men, 10 women) were identified in our study. The mean age of onset of symptoms was 57.76 ± 3.81 years. All patients had no previous family history of movement disorders. Most patients (41.2%) had unknown etiology (idiopathic). Cases of oromandibular dystonia were more frequently found in women (58.8%), in accordance with several previous literature. Oromandibular dystonia is generally associated with other movement disorders. A multidisciplinary approach with a combination of medication, speech therapy, and nutritional therapy can be tailored to the individual needs of each patient.

Conclusions:

Oromandibular dystonia is a focal dystonia that has a significant impact in decreasing patient's quality of life. This study can be used as a basis for further research in determining the clinical pictures of oromandibular dystonia.

Keywords

oromandibular dystonia, dystonia, clinical characteristics

P-044

Sequential Selective Peripheral Denervation Targeting Sternocleidomastoid and Splenius Capitis in Refractory Torticollis: A Case Report

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Introduction:

Torticollis, from Latin tortum collum and Italian torti colli ('twisted neck'), is a form of cervical dystonia marked by involuntary neck muscle contractions causing abnormal head rotation toward the affected side. Treatment options include physiotherapy, pharmacotherapy, botulinum toxin injections, and surgery. Surgery is typically reserved for refractory cases unresponsive to conservative treatments.

Case:

A 44-year-old woman has experienced frequent involuntary head turning to the left and difficulty turning to the right for the past two years, with symptoms progressively worsening. Initial treatment with oral medication, physiotherapy, and botulinum toxin showed no improvement. Selective peripheral denervation (SPD) of the right sternocleidomastoid was performed 1.5 years after symptom onset, guided by EEG mapping. Postoperatively, the Toronto Western Spasmodic Torticollis Rating Scale-2 (TWSTRS-2) score showed significant improvement, decreasing from 42/94 to 13/24. The improvement lasted only two months before symptoms recurred. A subsequent injection provided relief for only three weeks. Six months after the initial procedure, the patient underwent a second surgical intervention targeting left splenius capitis muscle. Three months postoperatively, TWSTRS-2 scores showed further improvement, with severity: 8/24, disability: 1/30, and pain: 5/40, indicating ongoing clinical benefit following SPD.

Discussion:

Recurrence following selective peripheral denervation (SPD) has been reported in approximately 10–15% of patients. In this case, initial SPD targeting the right sternocleidomastoid muscle yielded short-term improvement; however, symptom recurrence necessitated further intervention. Although current clinical guidelines do not endorse immediate bilateral or contralateral denervation, existing evidence supports a selective and staged

SPD approach, particularly targeting muscles such as the splenius capitis, when conservative treatments have proven ineffective.

Genetics and Epidemiology in Movement Disorders

P-045

Movement disorders in Adults with Metabolic Encephalopathy

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Background & Objective:

Movement disorders may be the harbinger of an underlying systemic disease or can be the presenting feature of or may emerge during the course of metabolic encephalopathy. Studying the co-existence of these two neurologic entities can provide new insights because each systemic disease can affect the brain in different ways.

Objectives:

To determine the prevalence and characteristics of movement disorders in adult patients diagnosed with various types of metabolic encephalopathies in the Philippines.

Methods:

This is a descriptive cross-sectional study based on a 7-year chart review of admitted adult patients diagnosed with any of the fourteen different types of metabolic encephalopathy who experienced movement disorders during their hospital stay. Movement disorders were characterized based on phenomenology, onset, and distribution.

Results:

Out of 9,773 neurologic referrals, 2,055 patients (17%) were diagnosed with metabolic encephalopathy, mainly the hypoxic-ischemic type. Only 4% (76) exhibited involuntary movements, primarily linked to hypoxic-ischemic encephalopathy. Three movement disorders were reported: myoclonus (64), tremor (10), and parkinsonism (2). The 6th decade was the most common age for these disorders, predominantly in males. Most cases ap-

peared acutely within 7 days after onset of encephalopathy and were mostly diffuse and multifocal.

Conclusions:

The presence of movement disorders may serve as additional clues to the underlying systemic dysfunction responsible for the encephalopathic state. The acute onset of involuntary movements underscores the rapid neurological impact of these conditions.

Table

Clinical and demographic profile		Movement disorders (all cases) (n=70)		Myoclonus (n=64)		Tremor (n=10)		Parkinsonism (n=2)	
		Number	Percentage	Number	Percentage	Number	Percentage	Number	Percentage
Age (in years)	18-29	2	2.86%	1	1.56%	1	10%	0	0%
	30-39	6	8.57%	5	7.81%	0	0%	0	0%
	40-49	2	2.86%	1	1.56%	0	0%	0	0%
	50-59	8	11.43%	6	9.37%	2	20%	0	0%
	60-69	23	32.86%	20	31.25%	2	20%	1	50%
	70-79	11	15.71%	10	15.62%	1	10%	0	0%
	80-89	13	18.57%	13	20.31%	4	40%	0	0%
Sex	< 50	7	10.0%	8	12.5%	0	0%	1	50%
	Male	41	58.57%	37	57.81%	4	40%	0	0%
Types of metabolic encephalopathy	Female	33	46.93%	27	42.19%	6	60%	1	100%
	Hypoxic-ischemic	48	68.57%	38	59.38%	2	20%	0	0%
	Hypoglycemic	4	5.71%	4	6.25%	0	0%	0	0%
	Uremic	16	22.86%	14	21.88%	2	20%	0	0%
	Hepatic	1	1.43%	1	1.56%	0	0%	0	0%
	Hypocalcemic	0	0%	0	0%	0	0%	0	0%
	Hyperglycemic	1	1.43%	1	1.56%	0	0%	0	0%
	Hyperkalemic	0	0%	0	0%	0	0%	0	0%
	Hypernatremic	0	0%	0	0%	0	0%	0	0%
	Sepsis	7	10.0%	2	3.12%	2	20%	2	100%
	Hypoglycemic	4	5.71%	2	3.12%	2	20%	0	0%
	Hypomagnesemic	1	1.43%	1	1.56%	0	0%	0	0%
	Hypocalcemic	0	0%	0	0%	0	0%	0	0%
	Hyperkalemic	1	1.43%	0	0%	1	10%	0	0%
	Hypernatremic	0	0%	0	0%	0	0%	0	0%
Onset	< 7 days	71	95.43%	60	93.75%	10	100%	1	50%
	7-14 days	4	5.71%	4	6.25%	0	0%	0	0%
	> 14 days	1	1.43%	0	0%	0	0%	0	0%
Distribution	Focal	13	18.57%	13	20.31%	0	0%	0	0%
	Multifocal	28	39.43%	21	32.81%	8	80%	2	100%
	Diffuse	39	55.43%	26	40.62%	2	20%	0	0%
	Missing data	0	0%	0	0%	0	0%	0	0%

Figure

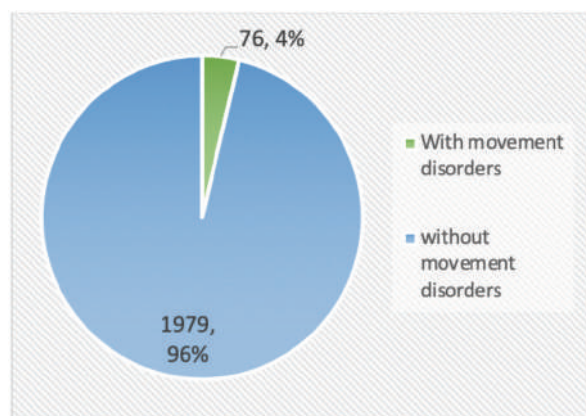


Figure 1. Frequency of movement disorders among patients with metabolic encephalopathy (7-year period)

Figure

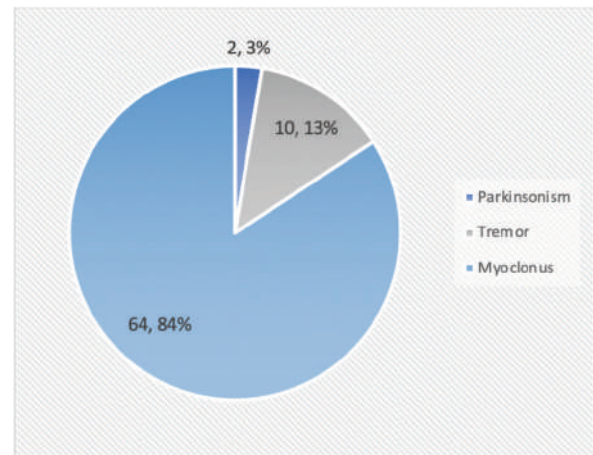


Figure 2. Frequency of movement disorders in patients with all-cause metabolic encephalopathies

Keywords

movement disorders, metabolic encephalopathy

P-047

Ethnic Implication of BDNF rs6265 Polymorphism in Korean Longitudinal Cohorts With Parkinson Disease

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Background & Objective:

Brain-derived neurotrophic factor supports dopaminergic neuron's endurance and dopamine release; its Val66Met polymorphism may modify Parkinson's disease risk, although evidence in Asians is limited. This study aimed to explore how the BDNF rs6265 genotypes could describe the characteristics and longitudinal progression patterns of PD patients in the Korean population.

Methods:

A total of 247 patients were enrolled and followed for an average of 50.9 ± 23.9 months. Baseline and/or periodic assessments captured motor severity, non-motor burden, cardiac denervation. The repeated measures were manipulated to infer any genotypic differences in the trajectories of each domain.

Results:

Genotype frequencies were Val/Val 31.2%, Val/Met 49.0%, and Met/Met 19.8% (Hardy-Weinberg $P = 0.847$); Met/Met prevalence was remarkably higher than in Caucasians. The baseline clinical characteristics and dopamine availability were comparable across the genotypes, but Met-allele carriers showed more denervated myocardium with a trend for allele dose effect. Longitudinal modelling revealed that the Val/Val genotype significantly worsened in a non-linear manner, compared to the nearly flattened progression of the Met/Met. The study did not prove significant genotype-specific trajectories in the domains of non-motor features, quality of life, and cardiac denervation in the longitudinal analyses.

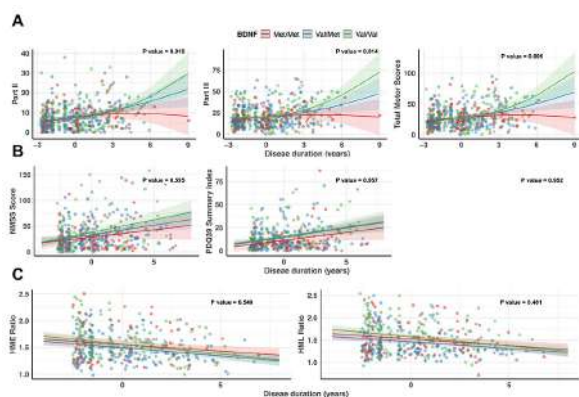
Conclusions:

The differences in cardiac denervation at diagnosis, non-motor burden, and motor progression might suggest an ethnic role of Val66Met polymorphism in the evolution and progression of Parkinson's disease.

Table

WFO Population	Overall	Male	Female	Age Group	Median	Post-Test Result
12/21	361	273 (76%)	127 (35%)	20-29	28	
Age of participants prior to COVID	47 (9.1%)	46 (9.0%)	47 (9.1%)	30-39	37	0.384
Sex	217 (60.1%)	217 (60.1%)	217 (60.1%)	40-49	45	0.867
Duration of onset of symptoms, mean (median) (IQR)	1,803 (36-100)	1,803 (36-100)	1,803 (36-100)	50-59	50	0.504
Total time from onset to PCR	36 (10.0%)	36 (10.0%)	36 (10.0%)	60-69	60	0.000
Time from onset to PCR	49 (13.6%)	49 (13.6%)	49 (13.6%)	70-79	71	0.124
Time from onset to PCR	20 (5.6%)	20 (5.6%)	20 (5.6%)	80-89	80	0.000
Time from onset to PCR	11 (3.1%)	11 (3.1%)	11 (3.1%)	90-99	90	0.000
Time from onset to PCR	3 (0.8%)	3 (0.8%)	3 (0.8%)	100-109	100	0.000
Time from onset to PCR	1 (0.3%)	1 (0.3%)	1 (0.3%)	110-119	110	0.000
Time from onset to PCR	1 (0.3%)	1 (0.3%)	1 (0.3%)	120-129	120	0.000
Time from onset to PCR	1 (0.3%)	1 (0.3%)	1 (0.3%)	130-139	130	0.000
Time from onset to PCR	1 (0.3%)	1 (0.3%)	1 (0.3%)	140-149	140	0.000
Time from onset to PCR	1 (0.3%)	1 (0.3%)	1 (0.3%)	150-159	150	0.000
Time from onset to PCR	1 (0.3%)	1 (0.3%)	1 (0.3%)	160-169	160	0.000
Time from onset to PCR	1 (0.3%)	1 (0.3%)	1 (0.3%)	170-179	170	0.000
Time from onset to PCR	1 (0.3%)	1 (0.3%)	1 (0.3%)	180-189	180	0.000
Time from onset to PCR	1 (0.3%)	1 (0.3%)	1 (0.3%)	190-199	190	0.000
Time from onset to PCR	1 (0.3%)	1 (0.3%)	1 (0.3%)	200-209	200	0.000
Time from onset to PCR	1 (0.3%)	1 (0.3%)	1 (0.3%)	210-219	210	0.000
Time from onset to PCR	1 (0.3%)	1 (0.3%)	1 (0.3%)	220-229	220	0.000
Time from onset to PCR	1 (0.3%)	1 (0.3%)	1 (0.3%)	230-239	230	0.000
Time from onset to PCR	1 (0.3%)	1 (0.3%)	1 (0.3%)	240-249	240	0.000
Time from onset to PCR	1 (0.3%)	1 (0.3%)	1 (0.3%)	250-259	250	0.000
Time from onset to PCR	1 (0.3%)	1 (0.3%)	1 (0.3%)	260-269	260	0.000
Time from onset to PCR	1 (0.3%)	1 (0.3%)	1 (0.3%)	270-279	270	0.000
Time from onset to PCR	1 (0.3%)	1 (0.3%)	1 (0.3%)	280-289	280	0.000
Time from onset to PCR	1 (0.3%)	1 (0.3%)	1 (0.3%)	290-299	290	0.000
Time from onset to PCR	1 (0.3%)	1 (0.3%)	1 (0.3%)	300-309	300	0.000
Time from onset to PCR	1 (0.3%)	1 (0.3%)	1 (0.3%)	310-319	310	0.000
Time from onset to PCR	1 (0.3%)	1 (0.3%)	1 (0.3%)	320-329	320	0.000
Time from onset to PCR	1 (0.3%)	1 (0.3%)	1 (0.3%)	330-339	330	0.000
Time from onset to PCR	1 (0.3%)	1 (0.3%)	1 (0.3%)	340-349	340	0.000
Time from onset to PCR	1 (0.3%)	1 (0.3%)	1 (0.3%)	350-359	350	0.000
Time from onset to PCR	1 (0.3%)	1 (0.3%)	1 (0.3%)	360-369	360	0.000
Time from onset to PCR	1 (0.3%)	1 (0.3%)	1 (0.3%)	370-379	370	0.000
Time from onset to PCR	1 (0.3%)	1 (0.3%)	1 (0.3%)	380-389	380	0.000
Time from onset to PCR	1 (0.3%)	1 (0.3%)	1 (0.3%)	390-399	390	0.000
Time from onset to PCR	1 (0.3%)	1 (0.3%)	1 (0.3%)	400-409	400	0.000
Time from onset to PCR	1 (0.3%)	1 (0.3%)	1 (0.3%)	410-419	410	0.000
Time from onset to PCR	1 (0.3%)	1 (0.3%)	1 (0.3%)	420-429	420	0.000
Time from onset to PCR	1 (0.3%)	1 (0.3%)	1 (0.3%)	430-439	430	0.000
Time from onset to PCR	1 (0.3%)	1 (0.3%)	1 (0.3%)	440-449	440	0.000
Time from onset to PCR	1 (0.3%)	1 (0.3%)	1 (0.3%)	450-459	450	0.000
Time from onset to PCR	1 (0.3%)	1 (0.3%)	1 (0.3%)	460-469	460	0.000
Time from onset to PCR	1 (0.3%)	1 (0.3%)	1 (0.3%)	470-479	470	0.000
Time from onset to PCR	1 (0.3%)	1 (0.3%)	1 (0.3%)	480-489	480	0.000
Time from onset to PCR	1 (0.3%)	1 (0.3%)	1 (0.3%)	490-499	490	0.000
Time from onset to PCR	1 (0.3%)	1 (0.3%)	1 (0.3%)	500-509	500	0.000
Time from onset to PCR	1 (0.3%)	1 (0.3%)	1 (0.3%)	510-519	510	0.000
Time from onset to PCR	1 (0.3%)	1 (0.3%)	1 (0.3%)	520-529	520	0.000
Time from onset to PCR	1 (0.3%)	1 (0.3%)	1 (0.3%)	530-539	530	0.000
Time from onset to PCR	1 (0.3%)	1 (0.3%)	1 (0.3%)	540-549	540	0.000
Time from onset to PCR	1 (0.3%)	1 (0.3%)	1 (0.3%)	550-559	550	0.000
Time from onset to PCR	1 (0.3%)	1 (0.3%)	1 (0.3%)	560-569	560	0.000
Time from onset to PCR	1 (0.3%)	1 (0.3%)	1 (0.3%)	570-579	570	0.000
Time from onset to PCR	1 (0.3%)	1 (0.3%)	1 (0.3%)	580-589	580	0.000
Time from onset to PCR	1 (0.3%)	1 (0.3%)	1 (0.3%)	590-599	590	0.000
Time from onset to PCR	1 (0.3%)	1 (0.3%)	1 (0.3%)	600-609	600	0.000
Time from onset to PCR	1 (0.3%)	1 (0.3%)	1 (0.3%)	610-619	610	0.000
Time from onset to PCR	1 (0.3%)	1 (0.3%)	1 (0.3%)	620-629	620	0.000
Time from onset to PCR	1 (0.3%)	1 (0.3%)	1 (0.3%)	630-639	630	0.000
Time from onset to PCR	1 (0.3%)	1 (0.3%)	1 (0.3%)	640-649	640	0.000
Time from onset to PCR	1 (0.3%)	1 (0.3%)	1 (0.3%)	650-659	650	0.000
Time from onset to PCR	1 (0.3%)	1 (0.3%)	1 (0.3%)	660-669	660	0.000
Time from onset to PCR	1 (0.3%)	1 (0.3%)	1 (0.3%)	670-679	670	0.000
Time from onset to PCR	1 (0.3%)	1 (0.3%)	1 (0.3%)	680-689	680	0.000
Time from onset to PCR	1 (0.3%)	1 (0.3%)	1 (0.3%)	690-699	690	0.000
Time from onset to PCR	1 (0.3%)	1 (0.3%)	1 (0.3%)	700-709	700	0.000
Time from onset to PCR	1 (0.3%)	1 (0.3%)	1 (0.3%)	710-719	710	0.000
Time from onset to PCR	1 (0.3%)	1 (0.3%)	1 (0.3%)	720-729	720	0.000
Time from onset to PCR	1 (0.3%)	1 (0.3%)	1 (0.3%)	730-739	730	0.000
Time from onset to PCR	1 (0.3%)	1 (0.3%)	1 (0.3%)	740-749	740	0.000
Time from onset to PCR	1 (0.3%)	1 (0.3%)	1 (0.3%)	750-759	750	0.000
Time from onset to PCR	1 (0.3%)	1 (0.3%)	1 (0.3%)	760-769	760	0.000
Time from onset to PCR	1 (0.3%)	1 (0.3%)	1 (0.3%)	770-779	770	0.000
Time from onset to PCR	1 (0.3%)	1 (0.3%)	1 (0.3%)	780-789	780	0.000
Time from onset to PCR	1 (0.3%)	1 (0.3%)	1 (0.3%)	790-799	790	0.000
Time from onset to PCR	1 (0.3%)	1 (0.3%)	1 (0.3%)	800-809	800	0.000
Time from onset to PCR	1 (0.3%)	1 (0.3%)	1 (0.3%)	810-819	810	0.000
Time from onset to PCR	1 (0.3%)	1 (0.3%)	1 (0.3%)	820-829	820	0.000
Time from onset to PCR	1 (0.3%)	1 (0.3%)	1 (0.3%)	830-839	830	0.000
Time from onset to PCR	1 (0.3%)	1 (0.3%)	1 (0.3%)	840-849	840	0.000
Time from onset to PCR	1 (0.3%)	1 (0.3%)	1 (0.3%)	850-859	850	0.000
Time from onset to PCR	1 (0.3%)	1 (0.3%)	1 (0.3%)	860-869	860	0.000
Time from onset to PCR	1 (0.3%)	1 (0.3%)	1 (0.3%)	870-879	870	0.000
Time from onset to PCR	1 (0.3%)	1 (0.3%)	1 (0.3%)	880-889	880	0.000
Time from onset to PCR	1 (0.3%)	1 (0.3%)	1 (0.3%)	890-899	890	0.000
Time from onset to PCR	1 (0.3%)	1 (0.3%)	1 (0.3%)	900-909	900	0.000
Time from onset to PCR	1 (0.3%)	1 (0.3%)	1 (0.3%)	910-919	910	0.000
Time from onset to PCR	1 (0.3%)	1 (0.3%)	1 (0.3%)	920-929	920	0.000
Time from onset to PCR	1 (0.3%)	1 (0.3%)	1 (0.3%)	930-939	930	0.000
Time from onset to PCR	1 (0.3%)	1 (0.3%)	1 (0.3%)	940-949	940	0.000
Time from onset to PCR	1 (0.3%)	1 (0.3%)	1 (0.3%)	950-959	950	0.000
Time from onset to PCR	1 (0.3%)	1 (0.3%)	1 (0.3%)	960-969	960	0.000
Time from onset to PCR	1 (0.3%)	1 (0.3%)	1 (0.3%)	970-979	970	0.000
Time from onset to PCR	1 (0.3%)	1 (0.3%)	1 (0.3%)	980-989	980	0.000
Time from onset to PCR	1 (0.3%)	1 (0.3%)	1 (0.3%)	990-999	990	0.000
Time from onset to PCR	1 (0.3%)	1 (0.3%)	1 (0.3%)	1000-1009	1000	0.000
Time from onset to PCR	1 (0.3%)	1 (0.3%)	1 (0.3%)	1010-1019	1010	0.000
Time from onset to PCR	1 (0.3%)	1 (0.3%)	1 (0.3%)	1020-1029	1020	0.000
Time from onset to PCR	1 (0.3%)	1 (0.3%)	1 (0.3%)	1030-1039	1030	0.000
Time from onset to PCR	1 (0.3%)	1 (0.3%)	1 (0.3%)	1040-1049	1040	0.000
Time from onset to PCR	1 (0.3%)	1 (0.3%)	1 (0.3%)	1050-1059	1050	0.000
Time from onset to PCR	1 (0.3%)	1 (0.3%)	1 (0.3%)	1060-1069	1060	0.000
Time from onset to PCR	1 (0.3%)	1 (0.3%)	1 (0.3%)	1070-1079	1070	0.000
Time from onset to PCR	1 (0.3%)	1 (0.3%)	1 (0.3%)	1080-1089	1080	0.000
Time from onset to PCR	1 (0.3%)	1 (0.3%)	1 (0.3%)	1090-1099	1090	0.000
Time from onset to PCR	1 (0.3%)	1 (0.3%)	1 (0.3%)	1100-1109	1100	0.000
Time from onset to PCR	1 (0.3%)	1 (0.3%)	1 (0.3%)	1110-1119	1110	0.000
Time from onset to PCR	1 (0.3%)	1 (0.3%)	1 (0.3%)	1120-1129	1120	0.000
Time from onset to PCR	1 (0.3%)	1 (0.3%)	1 (0.3%)	1130-1139	1130	0.000
Time from onset to PCR	1 (0.3%)	1 (0.3%)	1 (0.3%)	1140-1149	1140	0.000
Time from onset to PCR	1 (0.3%)	1 (0.3%)	1 (0.3%)	1150-1159	1150	0.000
Time from onset to PCR	1 (0.3%)	1 (0.3%)	1 (0.3%)	1160-1169	1160	0.000
Time from onset to PCR	1 (0.3%)	1 (0.3%)	1 (0.3%)	1170-1179	1170	0.000
Time from onset to PCR	1 (0.3%)	1 (0.3%)	1 (0.3%)	1180-1189	1180	0.000
Time from onset to PCR	1 (0.3%)	1 (0.3%)	1 (0.3%)	1190-1199	1190	0.000
Time from onset to PCR	1 (0.3%)	1 (0.3%)	1 (0.3%)	1200-1209	1200	0.000
Time from onset to PCR	1 (0.3%)	1 (0.3%)	1 (0.3%)	1210-1219	1210	0.000
Time from onset to PCR	1 (0.3%)	1 (0.3%)	1 (0.3%)	1220-1229	1220	0.000
Time from onset to PCR	1 (0.3%)	1 (0.3%)	1 (0.3%)	1230-1239	1230	0.000
Time from onset to PCR	1 (0.3%)	1 (0.3%)	1 (0.3%)	1240-1249	1240	0.000
Time from onset to PCR	1 (0.3%)	1 (0.3%)	1 (0.3%)	1250-1259	1250	0.000
Time from onset to PCR	1 (0.3%)	1 (0.3%)	1 (0.3%)	1260-1269	1260	0.000
Time from onset to PCR	1 (0.3%)	1 (0.3%)	1 (0.3%)	1270-1279	1270	0.000
Time from onset to PCR	1 (0.3%)	1 (0.3%)	1 (0.3%)	1280-1289	1280	0.000
Time from onset to PCR	1 (0.3%)	1 (0.3%)	1 (0.3%)	1290-1299	1290	0.000
Time from onset to PCR	1 (0.3%)	1 (0.3%)	1 (0.3%)	1300-1309	1300	0.000
Time from onset to PCR	1 (0.3%)	1 (0.3%)	1 (0.3%)	1310-1319	1310	0.000
Time from onset to PCR	1 (0.3%)	1 (0.3%)	1 (0.3%)	1320-1329	1320	0.000
Time from onset to PCR	1 (0.3%)	1 (0.3%)	1 (0.3%)	1330-1339	1330	0.000
Time from onset to PCR	1 (0.3%)	1 (0.3%)	1 (0.3%)	1340-1349	1340	0.000
Time from onset to PCR	1 (0.3%)	1 (0.3%)	1 (0.3%)	1350-1359	1350	0.000
Time from onset to PCR	1 (0.3%)	1 (0.3%)	1 (0.3%)	1360-1369	1360	0.000
Time from onset to PCR	1 (0.3%)	1 (0.3%)	1 (0.3%)	1370-1379	1370	0.000
Time from onset to PCR	1 (0.3%)	1 (0.3%)	1 (0.3%)	1380-1389	1380	0.000
Time from onset to PCR	1 (0.3%)	1 (0.3%)	1 (0.3%)	1390-1399	1390	0.000
Time from onset to PCR	1 (0.3%)	1 (0.3%)	1 (0.3%)	1400-1409	1400	0.000
Time from onset to PCR	1 (0.3%)	1 (0.3%)	1 (0.3%)	1410-1419	1410	0.000
Time from onset to PCR	1 (0.3%)	1 (0.3%)	1 (0.3%)	1420-1429	1420	0.000
Time from onset to PCR	1 (0.3%)	1 (0.3%)	1 (0.3%)	1430-1439	1430	0.000
Time from onset to PCR	1 (0.3%)	1 (0.3%)	1 (0.3%)	1440-1449	1440	0.000
Time from onset to PCR	1 (0.3%)	1 (0.3%)	1 (0.3%)	1450-1459	1450	0.000
Time from onset to PCR	1 (0.3%)	1 (0.3%)	1 (0.3%)	1460-1469	1460	0.000
Time from onset to PCR	1 (0.3%)	1 (0.3%)	1 (0.3%)	1470-1479	1470	0.000
Time from onset to PCR	1 (0.3%)	1 (0.3%)	1 (0.3%)	1480-1489	1480	0.000
Time from onset to PCR	1 (0.3%)	1 (0.3%)	1 (0.3%)	1490-1499	1490	0.000
Time from onset to PCR	1 (0.3%)	1 (0.3%)	1 (0.3%)	1500-1509	1500	0.000
Time from onset to PCR	1 (0.3%)	1 (0.3%)	1 (0.3%)	1510-1519	1510	0.000
Time from onset to PCR	1 (0.3%)	1 (0.3%)	1 (0.3%)	1520-1529	1520	0.000
Time from onset to PCR	1 (0.3%)	1 (0.3%)	1 (0.3%)	1530-1539	1530	0.000
Time from onset to PCR	1 (0.3%)	1 (0.3%)	1 (0.3%)	1540-1549	1540	0.000
Time from onset to PCR	1 (0.3%)	1 (0.3%)	1 (0.3%)	1550-1559	1550	0.000
Time from onset to PCR	1 (0.3%)	1 (0.3%)	1 (0.3%)	1560-1569	1560	0.000
Time from onset to PCR	1 (0.3%)	1 (0.3%)	1 (0.3%)	1570-1579	1570	0.000
Time from onset to PCR	1 (0.3%)	1 (0.3%)	1 (0.3%)	1580-1589	1580	0.000
Time from onset to PCR	1 (0.3%)	1 (0.3%)	1 (0.3%)			

Figure



P-048

Lack of Diagnostic Method for Huntington's Disease in Mongolia

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Introduction:

Huntington disease (HD) is an inherited progressive neurodegenerative disorder characterized by choreiform movements, psychiatric problems, and dementia. Clinical characteristics of cortical and subcortical dementia show a subtle overlap, which complicates a distinct diagnosis. The definite diagnosis is exclusively provided in the case of patients who present with clinical syndrome and have pathogenetic mutations or histopathological evidence.

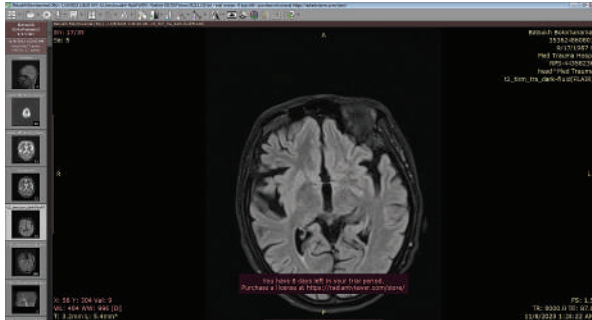
Case:

38 years old female was admitted to our Alt-Bold neurology clinic because of the irregular movement, ataxia, memory impairment, dysarthria, dysphagia, dysphonia, loss of bladder control, constipation, hyposomnia, and cognitive impairment. She has experienced first symptom since 2015 with irregular, jerky movement in her hands and worsening the other symptoms gradually such as insomnia, ataxia, and cognitive impairment. Deep tendon reflexes were high in all extremities, Babinski reflexes and planter clonus also occurred bilaterally. Romberg's sign was positive and fell in various directions. In November 2023, cranial magnetic resonance imaging (MRI), diffusion-weighted imaging (DWI) and magnetic resonance angiography (MRA) suggested the following: (i) moderate brain atrophy with shrinkened putamen; (ii) neurodegeneration such as fronto-temporal lobe atrophy.

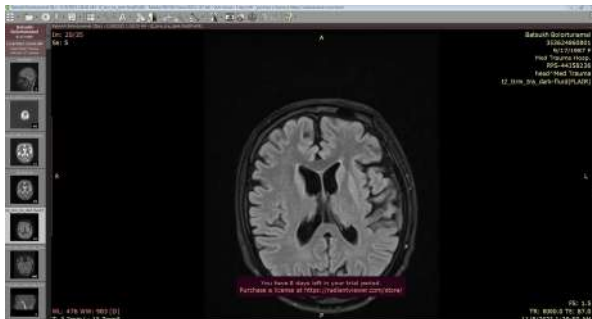
Discussion:

The case was classified as probable cognitive decline that occurs as a symptom of Huntington's Disease according to the symptoms, nervous examination and MRI findings, however there is a lack of diagnostic confirmation to differentiate it from other types of neurodegenerative diseases and confusion about other type of dementias. There was not enough diagnostic method such as genetic testing and other advanced diagnostic device required for many patients in Mongolia. Problems with diagnosis also make it difficult to choose the appropriate treatment for the patients. It is necessary to diagnose movement disorders at an early stage and maintain their quality of life.

Figure



Figure



Figure



Keywords

Huntington, chorea, differential, genetic, epidemiology

P-049

Effects of Parkinson's Disease-Related Genes Parkin, PINK1 and CHCHD2 in Mitochondria

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Background & Objective:

The purpose of this study is to understand the change of mitochondria morphology for some genes to cause young onset familial Parkinson's diseases using drosophila. We would study PINK1 knock-out (PINK1^{-/-}), Parkin knock-out (Parkin^{-/-}) and dCHCHD2 knock-out (dCHCHD2^{-/-}) in normoxia or hypoxia condition. In cellular level, Parkinson's disease (PD) has prominent mitochondrial dysfunction. PINK1 and Parkin, which encode mitochondrial protein kinase and cytosolic ubiquitin (Ub) ligase, relatively, were identified as the genes responsible for the autosomal recessive form of juvenile Parkinson's disease. Parkin gene encodes an ubiquitin ligase (E3), as we know, induces mitophagy to control mitochondria quality. And loss of Parkin gene results in degradation of mitochondria. Parkin, PINK1 and DJ-1 are associated with mitophagy and mitochondria homeostasis. The newly identified PD-related gene CHCHD2, which encodes a mitochondrial protein, was one of etiologies of PD. The loss of CHCHD2 in Drosophila causes abnormal matrix structures and impaired oxygen respiration in mitochondria, leading to oxidative stress.

Methods:

A. Drosophila stocks. All Drosophila stocks were kept under standard food conditions, normal temperature conditions (25°C), and normal humidity conditions (60%). Those flies were obtained from the Bloomington Drosophila Stock Center, Baylor College of Medicine and University of Pennsylvania. B. Hypoxia/Normoxia condition. Pink1 (B9), Parkin, CHCHD2 and normal controlled flies to be raised from eggs under average 6% O₂/94% N₂ condition comparing flies under normoxia (average 21% O₂). C. Whole-mount immunostaining and fluoromicroscope check. The mitochondrial morphology of the indirect flight muscles and the number of TH-positive neurons were analyzed by whole-mount immunostaining as previously described. D. Survival assay and climbing assay. For the lifespan studies, ~75 adult flies for each group per vial (25mm diameter X 95mm height) were maintained at 25°C, transferred to fresh fly food, and scored for survival ev-

ery 4 days. For a climbing assay, vials (25mm diameter x180mm height) containing 20–25 flies were tapped gently on the table and left standing for 18seconds. The number of flies that climbed at least 60mm was recorded. E. Immunohistochemistry. Adult fly thoraces dissected from 7-day-old male flies were fixed with 4% formaldehyde in fixation buffer (100 mM PIPES, 1 mM EGTA, 1% Triton X-100, and 2 mM MgSO₄, pH 6.9) and blocked with wash buffer (50 mM Tris, 150 mM NaCl, 0.1% Triton X-100, 0.5 mg/mL BSA, pH 6.8) with 10 mg/mL BSA. All images were obtained under an LSM710 Carl Zeiss confocal microscope.

Results:

1.UAS-Mito GFP; MHC-GAL4 for Normoxia (a) and Hypoxia (b) state Normal control (UAS-Mito GFP/+ ; MHC-GAL4/+) (Normoxia) : No abnormal deformity of mitochondria and aligned between the myofibrils. Normal control (Hypoxia) : Mild deformity of mitochondria with mildly decreased GFP signal within normal myofibrils. 2.PINK1 -/- (B9) for Normoxia (a) and Hypoxia (b) state PINK1 -/- (B9/y ; MHC- mito GFP) (Normoxia) : Moderate to severe deformity of mitochondria. Many mitochondrial clumps with overlapped mitochondria (Fusion) GFP signal is reduced. PINK1 -/- (Hypoxia) : Severe deformity of mitochondria & various mitochondrial clumps. (fusion and fission) And GFP signal is much more reduced. Irregular shaped and large mitochondrial clumps. 3.Parkin -/- (dPark[25]/dPark [1]) for Normoxia (a) and Hypoxia (b) state Parkin -/- (MHC-Gal4, UAS-mito GFP/+ ; dPark[25]/dPark[1]) (Normoxia) : Severe deformity of mitochondria. Irregular GFP signal and mitochondrial clumps Parkin -/- (Hypoxia) : Severe deformity of mitochondria. Big mitochondrial clumps & GFP signal is so much reduced. 4.Knock-out CHCHD2 for Normoxia (a) and Hypoxia (b) state dCHCHD2 -/- (Knock-out CHCHD2/y ; MHC- mito GFP) (Normoxia) : Mild abnormal deformity of mitochondria such like fission following aligned between the myofibrils. dCHCHD2 -/- (Hypoxia) : Near normal to mild deformity of mitochondria with some fission or defragmentation but rare mitochondrial clumps* Green colored GFP, Red colored Actin, Blue colored Nucleus in fluoromicroscope. 5. This table shows the observations of fluorescence microscopy for the change of the mitochondrial shape under genetic mutation for Parkinson's disease. 6. Western Blot outcome for Normoxia vs. Hypoxia Hypoxia-inducible factor 1 (HIF1-beta) is overall increased in the hypoxia state. Ref2P is a component of protein aggregates in defective flies in autophagy or neurodegenerative diseases and thus, the level of that for d-Park, B9-, Knock-out CHCHD2 in normoxia is elevated. 4E-BP represents reactive oxygen specificities in mitochondria, and in the case of normoxia, B9- and Knock-out CHCHD2 drosophila the values of 4E-BP are elevated. Tubulin, which means microtubule constitution, was used as a control, and the level is constant regardless of normoxia or hypoxia.

Conclusions:

From this study, we found that hypoxia caused moderate to severe damage on mitochondria except dCHCHD2 knock out flies. In details, under average 6% O₂ concentration, normal control (UAS-Mito GFP/+; MHC-GAL4/+) showed mild deformity of mitochondria with reduced GFP (green fluorescent protein) signal but expressing normal myofibrils. And Parkin knock out (MHC-Gal4, UAS-mito GFP/+; dPark [25]/dPark [1]) made big mitochondrial clumps and depicted GFP signal might be reduced. Also, PINK1 knock out (B9/y;; MHC- mito GFP) demonstrated severe deformity of mitochondria and various mitochondrial clumps such like fusion and fission. Furthermore, GFP signal is reduced. However, dCHCHD2 knock out made an expression of moderate deformity of mitochondria such as fission or defragmentation under normoxia pressure and in hypoxia condition, we could observe near normal to mild deformity of mitochondria with some fission or defragmentation but rare mitochondrial clumps. Thus, we a little understood the physiological and pathological roles of PINK1, Parkin and CHCHD2. Also, CHCHD2-deficiency flies may show be rescued under hypoxia because of decreased oxidative stress. These results suggest PINK1 and Parkin gene as well as dCHCHD2 knock out are major causes of mitochondrial dysfunction to lead young onset Parkinson's disease.

P-051

Association Between Instrumental Activities of Daily Living and Incidence of Parkinson's Disease: A Nationwide Population-Based Cohort Study

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Background & Objective:

Several studies have investigated the risk or prodromal factors for early diagnosis of Parkinson's disease (PD). Instrumental activities of daily living (IADL) involve both motor and non-motor functions, and have been used as a screening tool for dementia. This study aimed to examine the association between IADL de-

pendency and PD risk and identify specific IADL items linked to an increased likelihood of developing PD.

Methods:

This population-based cohort study used data from the Korean National Health Insurance Service database, that contains information on long-term care services. Individuals who underwent at least one health screening and completed the geriatric assessment sheet between 2009 and 2021 were included in this study. Information on IADL was extracted from the geriatric assessment sheet of long-term care services database. Participants were categorized into quartiles according to their IADL dependency. PD was defined based on the ICD-10 code (G20) and rare intractable disease registration code (V124). The Fine-Gray subdistribution hazard model was used to assess subsequent PD risk according to IADL dependency.

Results:

During a mean follow-up period of 3.78 ± 3.34 years, 308 of the 21,662 participants developed PD. The highest IADL dependency was significantly associated with an increased risk of PD (adjusted hazard ratio [aHR] = 1.458, 95% confidence interval [CI] 1.037–2.050), particularly among women or those aged 75 years and older. Among the 10 IADL items, dependency in financial management (aHR 1.420, 95% CI 1.057–1.909, $p = 0.0201$) or telephone use was significantly associated with an increased risk of PD (aHR 1.536, 95% CI 1.204–1.961, $p = 0.0006$).

Conclusions:

Our results suggested that individuals with IADL dependency were at an increased risk of PD. Further research on other ethnicities that considers sociocultural differences is required to confirm this association.

such as isolated hypomyelination or hereditary dystonia type 4. Nevertheless, familial adult-onset presentations have been rarely reported.

Case:

We report a Korean family with *TUBB4A*-related leukodystrophy carrying a heterozygous c.286G>A (p.Gly96Arg) variant. The proband, a male in his early thirties, initially developed hand tremor in his early twenties, which gradually worsened and was followed by gait disturbance, dysarthria, and mild cognitive impairment in his late twenties. Neurological examination revealed spasticity, cerebellar ataxia, hyperreflexia, positive Babinski signs, and postural tremor, without evidence of dystonia or parkinsonism. Brain magnetic resonance imaging demonstrated diffuse periventricular and subcortical T2 hyperintensities with mild cerebellar atrophy, while the basal ganglia were preserved. A targeted next-generation sequencing ataxia panel identified the pathogenic *TUBB4A* c.286G>A variant in the proband. Subsequent familial mutation testing confirmed the same variant in his mother and two siblings, all of whom presented with varying degrees of spastic-ataxic gait disturbance, tremor, and dysarthria. The mother developed head tremor in her forties, followed by rapid progression to complete loss of ambulation within 5 years and subsequent cognitive decline, leading to institutional care. In contrast, the father and another sibling tested negative for the variant and remained asymptomatic.

Discussion:

This report describes an adult-onset *TUBB4A*-related leukodystrophy presenting as a spastic-ataxic syndrome with tremor, thereby expanding the clinical spectrum of *TUBB4A*-associated disorders and highlighting the importance of genetic testing in suspected hereditary spastic-ataxic disorders.

P-052

Adult-Onset Familial *TUBB4A*-Related Leukodystrophy in a Korean Family: A Case Report

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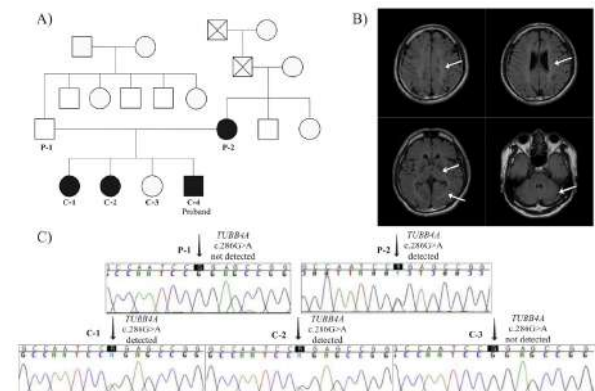
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Introduction:

Tubulin beta-4A class IVa (*TUBB4A*)-related leukodystrophy is an uncommon autosomal dominant disorder with a broad phenotypic spectrum, ranging from infantile-onset hypomyelination with atrophy of the basal ganglia and cerebellum to milder forms

Figure



P-053

Adolescent-Onset Spastic Ataxia Without Myopathy in a Patient With Heterozygous *MSTO1* Mutation

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Introduction:

MSTO1 encodes a cytoplasmic protein essential for mitochondrial fusion. Recessive *MSTO1* mutations usually cause early-onset myopathy, motor delay, corticospinal dysfunction, cerebellar atrophy, and elevated creatine kinase (CK). Dominant *MSTO1* mutations are extremely rare, with one family reported showing late-onset, mild motor impairment, absent cerebellar signs, and low vitamin D.

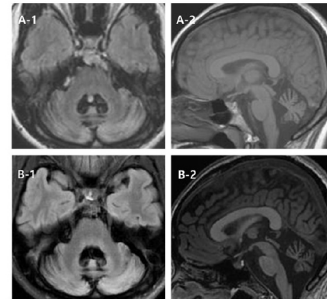
Case:

A 20-year-old Korean woman developed tremor at age 10 and gait difficulty at 13. Development was normal, no neuromuscular disease except two episodes of presumed aseptic meningitis. Exam showed saccadic hypermetria, gaze-evoked nystagmus, limb dysmetria (L>R), spasticity, hyperreflexia, cerebellar dysarthria, and wide-based ataxic gait; she remained independently ambulatory. Labs including CK and lactic acid were normal; vitamin D was 8.7 ng/mL. Brain MRI showed severe pontocerebellar atrophy, largely stable over 14 years. EMG/NCS were normal. Genetic tests for common ataxias and 27-gene panel were negative. Whole-exome sequencing revealed heterozygous likely pathogenic *MSTO1* c.22G>A, p.Val8Met, previously reported in a Hungarian family. Gait progressed slowly, she required minimal assistance, cognition intact.

Discussion:

This is the second reported case of dominant *MSTO1* mutation. Unlike recessive cases, she had late-onset, slowly progressive spastic ataxia with normal CK and preserved muscle function. Compared to the Hungarian family with the same variant, she showed cerebellar involvement without psychiatric or neurodevelopmental features, indicating variable expressivity. Vitamin D deficiency was shared. MRI confirmed cerebellar pathology despite mild functional impairment, supporting relevance in adolescent-onset cases. In summary, this case highlights a distinct phenotype of dominant *MSTO1* mutation, differing from both recessive cases and the previously reported family.

Figure



Brain MRI images taken at age 20 showed diffuse cerebellar atrophy in axial FLAIR (A-1) and sagittal FLAIR (A-2). In the image taken at the age of 34, mild progression of cerebellar hemisphere atrophy was observed in axial FLAIR (B-1) and sagittal FLAIR (B-2).

Keywords

Cerebellar Ataxia, *MSTO1*, Mutation, Whole Exome Sequencing

P-055

Can Occupational Pesticide Exposure Increase the Chance of Parkinson's Disease Risk?

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Background & Objective:

Impact of molecular mechanism of long-term pesticide exposure on the pathophysiology of Parkinson's disease (PD) are still unclear. Additionally, PD is linked to changes in the expression levels of transcription factors (TFs) such as FOXA1 and NURR1. Therefore, any dysregulation in these TFs may interfere with neuronal maintenance processes, thereby making PD more likely. The purpose of this study was to identify environmental stressors that interact with the genes that encode the transcription factors FOXA1 and NURR1.

Methods:

A total of 44 Peripheral blood lymphocytes (PBL) samples were collected from 17 subjects from pesticide exposed group and 27 healthy age and gender matched controls (HC). In the current investigation, PD biomarkers FOXA1 and NURR1 have been used. In order to identify any abnormalities in the expression levels of NURR1 and FOXA1, the transcriptomes of peripheral blood lymphocytes obtained from blood samples of rural agricultural

laborers who had been exposed to pesticides for an extended period of time within the Indian subpopulation were profiled.

Results:

Based on the Nurr1 expression profile, the odds ratios (ORs) for developing Parkinson's disease (PD) were 0.52 ($p < 0.05$) for males and 0.172 ($p > 0.05$) for females. However, the OR for Foxa1 expression was 1.17 ($p > 0.05$) in females and 0.74 ($p < 0.05$) in males. The pesticide exposure group's expression of Foxa1 and Nurr1 mRNA was significantly lower than that of the healthy controls (Table). Considering that the UPDRS scale has trouble measuring the motor symptoms of Parkinson's disease in its early phases, this result suggests that pesticide exposures may be in the early stages of dopamine neuron degeneration. Nevertheless, the risk factor for male and female groups was not identified in the current investigation.

Conclusions:

This study indicates that pesticide exposure may elevate PD risk, utilizing transcription factors as markers.

Table

Table: Gene expression profiling of Foxa1 and Nurr1 gene from peripheral blood of agricultural workers.

Gender	TF	R2	CI-95%	p-Value
Male	Nurr1	0.520	0.15-0.54	0.002
	Foxa1	0.740	0.44-0.90	0.001
Female	Nurr1	0.170	0.04-0.62	0.08
	Foxa1	0.170	0.04-0.56	<0.06

Keywords

Agriculture Pesticide Exposure, Organophosphorus, Parkinsonism, Transcription Factors

P-056

A Novel SPG11 Splice-Site Mutation in Hereditary Spastic Paraplegia: Clinical, Radiologic, and Genetic Insights from the First Philippine Case

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Background & Objective:

Hereditary spastic paraplegias are clinically and genetically heterogeneous disorders marked by progressive spasticity and weakness, often complicated by cognitive impairment and distinctive neuroimaging features. SPG11 is the most common autosomal recessive HSP globally but remains underdiagnosed in resource-limited populations. This paper aims to report the first genetically confirmed case of SPG11-associated hereditary spastic paraplegia (HSP) in the Philippines, featuring a previously undocumented splice-site mutation and comprehensive phenotypic characterization.

Methods:

A 26-year-old female from a non-consanguineous family, with a decade-long history of progressive lower limb spasticity and cognitive decline, underwent thorough clinical, neurologic, and imaging assessments. The whole-exome sequencing identified two pathogenic SPG11 variants, including the novel c.3039-1G>A canonical splice-site mutation.

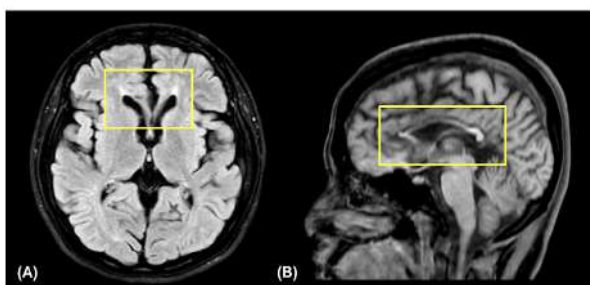
Results:

The patient exhibited classic SPG11-HSP features: progressive spastic paraplegia, cognitive impairment, and radiologic signs, including the thinning of the corpus callosum and "ears-of-the-lynx." Genetic analysis confirmed compound heterozygosity for two loss-of-function SPG11 variants, one of which is previously unreported. Symptomatic management with oral baclofen improved spasticity moderately. Genetic counseling and multidisciplinary care were provided.

Conclusions:

This case highlights the expanding mutational spectrum of SPG11 and reinforces the diagnostic value of genomic testing, especially in atypical spastic paraplegias. Early recognition, neuroimaging evaluation, and multidisciplinary management including rehabilitation and counseling are essential to optimize outcomes for SPG11-HSP. Further research is needed to address genotype-phenotype correlations and develop targeted therapies for complex forms of HSP.

Figure



Table

Table 1. Summary of pathogenic variants identified in the patient with SPG11-related HSP

Gene	Position	Transcript	Nucleotide	Zygosity	MAF/TW	Inheritance	Class
SPG11	15:44613537	NM_025137.4	c.3039-IG>A	Het	NR/NR	AR	Pathogenic
SPG11	15:44633499	NM_025137.4	c.1735+31735+6del	Het	0.000001842/NR	AR	Pathogenic

AR- autosomal recessive; c- coding DNA sequence; Het- heterozygous; MAF- maximal minor variant allele frequency; NM- curated mRNA transcript; NR- not reported; SPG- spastic paraplegia gene; TW- Taiwan biobank

Keywords

Hereditary Spastic Paraplegia, Spastic paraplegia, HSP, SPG11, Ears-of-the-lynx

P-057

From Past Trends to Future Challenges: A Time-Series Analysis and Projection of Parkinson's Disease Prevalence in Indonesian Older Adults with Estimated Burden for 2030 and 2040

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Background & Objective:

Parkinson's disease (PD) is a growing public health concern worldwide, particularly in aging populations. In Indonesia, the burden of PD is expected to rise in the coming decades, yet limited evidence exists on future projections of prevalence in specific age groups. This study aimed to analyze trends and project the future prevalence of Parkinson's disease among Indonesians aged 50–54 years, using historical prevalence data to estimate the burden up to 2030 and 2040.

Methods:

Prevalence data for PD in Indonesia were obtained from the Global Burden of Disease (GBD) Study 2017–2021. A time-series approach was applied using R statistical software to calculate the average annual growth rate and to project prevalence estimates for 2030 and 2040. Comparative analyses were conducted to assess temporal changes and the magnitude of the projected increase.

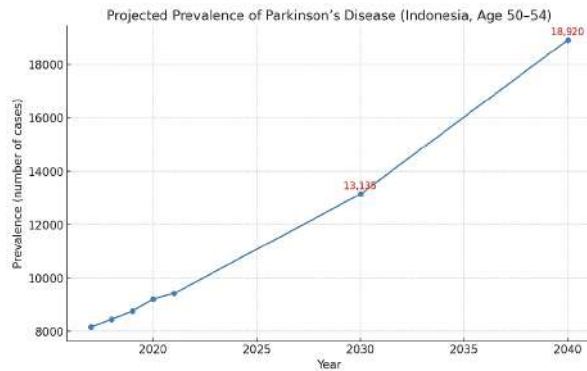
Results:

Analysis of Parkinson's disease (PD) prevalence in the 50–54-year-old group in Indonesia showed a rise from 8,153 cases in 2017, 8,442 in 2018, 8,741 in 2019, 9,192 in 2020, to 9,409 in 2021, reflecting an average annual growth of 3.65%. Projections estimate an increase to 13,135 cases by 2030 and 18,920 cases by 2040, nearly doubling over two decades. These findings confirm a sustained upward trend and underscore the growing public health challenge of PD in Indonesia's older productive-age population.

Conclusions:

The prevalence of Parkinson's disease among Indonesians aged 50–54 years rose from 8,153 in 2017 to 9,409 in 2021 and is projected to reach 13,135 in 2030 and 18,920 in 2040, showing a consistent upward trend and underscoring the need for targeted public health strategies.

Figure



Keywords

Parkinson's Disease, Prevalence , Projections, Trend Burden, Epidemiology

P-058

Dual Genetic Etiology in Adult-Onset Ataxia: Spinocerebellar Ataxia Type 3 with a Pathogenic *NOTCH3* Variant

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Introduction:

Spinocerebellar ataxia type 3 (SCA3) is the most common autosomal dominant cerebellar ataxia, caused by *ATXN3* CAG expansion, and typically presents with progressive gait disturbance, dysarthria, and oculomotor dysfunction. Cerebral autosomal dominant arteriopathy with subcortical infarcts and leukoencephalopathy (CADASIL), due to *NOTCH3* mutations, usually manifests with migraine, ischemic strokes, and confluent white matter hyperintensities (WMH). Their coexistence in a single patient is exceptionally rare.

A diagnostic challenge arises when the clinical phenotype and neuroimaging findings are discrepant, risking misdiagnosis.

Case:

A 51-year-old man had a 2–3 year history of progressive gait unsteadiness, dysarthria, and ocular fixation difficulty. Family history included multiple relatives with ataxia, but genetic testing

was declined. Examination revealed broken saccades, overshoot nystagmus, staccato speech, wide-based gait, and mild dysmetria. Brain MRI showed mild cerebellar atrophy with confluent WMH, lacunes, and microbleeds in the basal ganglia and thalamus. Genetic testing confirmed SCA3 with *ATXN3* expansion (~70 repeats) and identified a heterozygous *NOTCH3* variant (c.160C>T, p.R54C).

Discussion:

This case highlights the clinical implications of dual-genetic etiologies in adult-onset ataxia. The *ATXN3* expansion explained the progressive cerebellar syndrome, while MRI findings strongly suggested CADASIL. Although migraine and cognitive decline were absent, the *NOTCH3* variant supported combined pathology. Similar reports show that CADASIL may rarely present with ataxia and that *NOTCH3* mutations can modify SCA phenotypes. The clinicoradiologic mismatch in this patient illustrates the risk of attributing all findings to a single disorder. Clinicians should remain alert when clinical and imaging findings are discordant. Careful integration of clinical, radiologic, and genetic findings is essential to avoid diagnostic deviation, guide prognosis, and inform counseling.

Keywords

Spinocerebellar Ataxias, NOTCH3 Gene, CADASIL, Adult-Onset Ataxia, Genetic Predisposition to Disease

P-059

siRNA-Mediated Gene Silencing of *CTSB* and *USP30* Targeting Lysosomal and Mitochondrial Pathways to Enhance Alpha-Synuclein Clearance in Parkinson's Disease

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Background & Objective:

Parkinson's disease (PD) remains without disease-modifying therapies, as current treatments primarily provide symptomatic relief. A key clinical challenge is the progressive accumulation of misfolded α -synuclein, driving neurodegeneration. Gene silencing strategies targeting regulatory pathways involved in α -synuclein clearance offer promising therapeutic potential. Cathepsin B (CTSB) and ubiquitin-specific protease 30 (USP30) regulate lysosomal and mitochondrial pathways, respectively, which are

central to α -synuclein homeostasis. Targeting these genes with RNA interference may enhance proteostatic mechanisms and promote α -synuclein degradation.

Methods:

We employed a computational pipeline using DSIR software for siRNA design, MaxExpect for secondary structure prediction, and DuplexFold for duplex stability assessment. Candidate siRNAs against CTSB and USP30 were ranked by hybridization energy, structural accessibility, and corrected efficacy scores.

Results:

A total of 20 siRNA candidates were generated, with 10 sequences targeting CTSC and 10 targeting USP30 (Table 1). For CTSC, the top candidate (position 200) achieved a DSIR score of 106.4, while other high-ranking sequences showed scores ranging from 71.1 to 96.2. For USP30, the highest score was 93.8 (position 677), with several candidates scoring above 85, indicating strong silencing potential. Both target sets demonstrated favorable secondary structure accessibility and stable duplex formation, supporting their predicted functional efficiency.

Conclusions:

This study provides *in silico* evidence that silencing CTSB and USP30 with siRNA may enhance α -synuclein clearance via lysosomal and mitochondrial pathways. Clinically, this strategy could represent a disease-modifying approach beyond symptomatic therapy. Validation in experimental models, optimization of CNS delivery, and off-target assessment remain essential next steps, underscoring the translational promise of gene silencing in movement disorders.

Table

Table 1. Sequence siRNAs predicted for the gene of CTSC and USP30, and their parameters

[illegible]

Keywords

Parkinson Disease, Alpha-synuclein, Gene silencing, CTSB, USP30

P-060

Generalized Chorea and Ballismus as a rare presenting symptom of JAK2-Positive Polycythemia Vera Rubra

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Background & Objective:

Objective: To report a rare presentation of generalized chorea and ballismus in a patient with polycythemia vera rubra (PCVR) positive for the JAK2 V617F mutation. **Background:** PCVR is an acquired myeloproliferative neoplasm primarily presenting with erythrocytosis and thromboembolic complications. Neurological symptoms are usually vascular; movement disorders such as chorea and ballismus are rarely described, particularly in Asian populations.

Methods:

A 52-year-old Filipino female with no prior neurologic disease developed recurrent, brief episodes of involuntary movements affecting both lower extremities, consistent with chorea and ballismus, in addition to dysarthria and teeth grinding. Laboratory workup showed erythrocytosis, leukocytosis, and positive JAK2 mutation. Brain MRI demonstrated non-specific bilateral basal ganglia calcification. Other etiologies, including infectious, metabolic, autoimmune, and hereditary causes, were excluded.

Results:

The patient was diagnosed with PCVR with secondary generalized chorea and ballismus. Symptomatic control was achieved with risperidone and clonazepam. Therapeutic phlebotomy and antiplatelet therapy led to complete and sustained resolution of symptoms on follow-up.

Conclusions:

This case highlights generalized chorea and ballismus as unusual but important manifestations of JAK2-positive PCVR. Early recognition and hematologic treatment can lead to full resolu-

tion of movement symptoms. Hematologic etiologies should be considered in the differential diagnosis of unexplained movement disorders.

P-061

Genetic and Phenotypic Insights into MERRF: The MT-TK m.8344A>G Variant in a Filipino Family with Progressive Myoclonus Epilepsy

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Background & Objective:

To characterize the clinical spectrum and genetic basis of MERRF syndrome in a Filipino family, focusing on the pathogenic m.8344A>G MT-TK variant and its heteroplasmy. Background: MERRF (Myoclonic Epilepsy with Ragged-Red Fibers) is a rare maternally inherited mitochondrial disorder marked by progressive myoclonus, epilepsy, and myopathy. Due to heteroplasmy, clinical presentations and disease severity can vary widely, warranting detailed family-based study.

Methods:

A Filipino family presented with variable neurologic manifestations suggestive of MERRF. Clinical assessment, neurologic exams, and next-generation sequencing targeting mitochondrial DNA were performed. Heteroplasmy levels of the MT-TK m.8344A>G variant were quantified, and clinical-genetic correlation was assessed.

Results:

Three affected individuals carried the pathogenic m.8344A>G variant with heteroplasmy levels ranging from 9.5% to 77.3%. The highest heteroplasmy corresponded to earlier onset and more severe disease. Clinical features included myoclonic epilepsy, myopathy, edema, and cognitive symptoms. Management was primarily symptomatic, using levetiracetam, coenzyme Q10, B vitamins, and L-carnitine. Genetic counseling was provided.

Conclusions:

This report underscores the clinical impact of variable heteroplasmy of the MT-TK m.8344A>G mutation in MERRF and highlights the importance of family-based genetic analysis. Individualized management and multidisciplinary care are essential for patients with mitochondrial disorders. Regular follow-up and genetic counseling are critical in supporting affected families

and guiding medical decisions.

Keywords

MERRF syndrome, m.8344A>G, heteroplasmy, pathogenicity

P-062

Geospatial and Statistical Analysis of Health Workers, Therapy Workers, and Psychology Workers and Happiness for Parkinson's Disease in Indonesia: Using Open Government Data

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Background & Objective:

Indonesia is the fourth most populous country in the world and also has a large elderly population, which puts it at high risk for Parkinson's disease. This study identifies the relationship between health workers, therapy workers, and psychology workers and happiness in Parkinson's disease in Indonesia using statistical tests and geospatial mapping.

Methods:

The method used was statistical analysis with OLS and geospatial mapping analysis using QGIS. The variables identified were health workers, therapy workers, psychology workers, and the happiness index for Parkinson's disease in Indonesia. The 2021 data is the latest available from the government at the IHME and BPS.

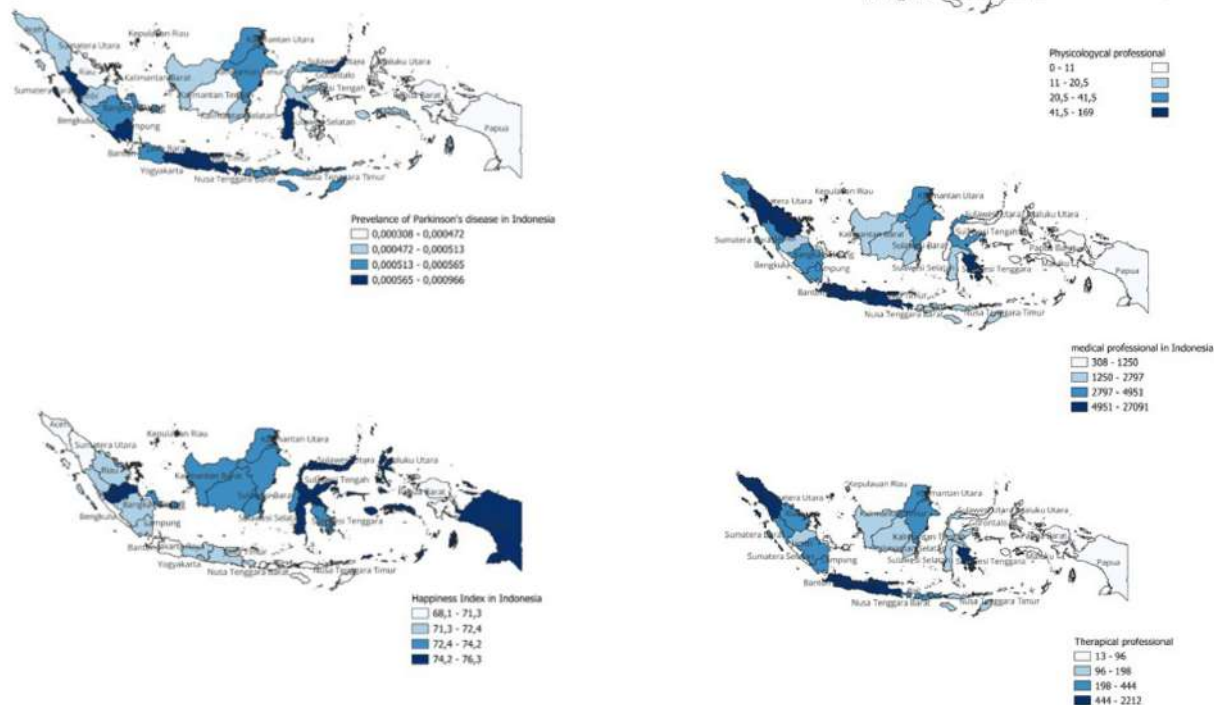
Results:

The statistical results show no significant relationship between health workers, therapy workers, and psychology workers and happiness in Parkinson's disease in Indonesia, but a negative relationship is present. Mapping across 34 provinces also shows inconsistent associations in influencing Parkinson's disease prevalence. However, some provinces show a negative correlation, where higher happiness leads to a lower incidence of Parkinson's disease. This is particularly true in eastern Papua. The number of psychologists and therapists tends to align with Parkinson's prevalence, likely due to factors such as urbanization, population density, and detection capabilities. In general, Parkinson's prevalence is higher in Java, Bali, and Sulawesi (urban and accessible areas), while it is lower in Papua and Maluku (where there is a shortage of healthcare workers and prevalence may be underreported).

Conclusions:

Therefore, the government needs to increase happiness and medical personnel to reduce the incidence of Parkinson's in Indonesia.

Figure



Figure

Keywords

Geospatial, Statistical, Parkinson's Disease, Indonesia, GIS

P-063

Genome-Wide and Polygenic Analyses of Idiopathic Parkinson's Disease in Koreans

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Background & Objective:

Parkinson's disease (PD) is the second most common neurodegenerative disorder in the elderly. Idiopathic PD (IPD) is a complex condition, thought to result from a combination of genetic and environmental factors. Genetically, IPD is considered a polygenic disorder, with multiple risk alleles of mostly weak to moderate effect contributing to disease susceptibility. To date, more than 90 single-nucleotide polymorphism (SNP) associations with IPD have been identified through genome-wide association studies (GWAS) in European populations.

Methods:

To investigate the genetic architecture of IPD in the Korean population, we conducted a GWAS of 10,139 individuals, including 2,793 patients with IPD and 7,446 controls, using the Korean Biobank Array.

Results:

We identified genome-wide significant associations ($P < 5 \times 10^{-8}$) at the SNCA, SLC45A3-NUCKS1, SLC2A13, and LRRK2 loci. In addition, several exonic variants in SLC41A1, MCCC1, PGBD1, ZSCAN31, MAS1L, and LRRK2 showed suggestive associations, supporting roles for these genes in IPD not only in Europeans but also in East Asians. We also confirmed that 62 of 90 previously reported Mendelian genes for hereditary parkinsonian disorders were nominally associated with the disease ($P < 0.05$) in our dataset. Building on these findings, we will compute a polygenic risk score (PRS) to assess its contribution to disease risk in Koreans, with particular focus on its influence on the age at onset of IPD. Furthermore, we will evaluate the impact of PRS on phenotypes and disease penetrance in carriers of specific mutations, such as LRRK2 G2385R.

Conclusions:

Together, our GWAS and PRS analyses will advance understanding of the genetic architecture, penetrance, and phenotypic variability of IPD in the Korean population.

Keywords

Parkinson's disease, genome-wide association study, genetics, polygenic risk score, LRRK2

Infection- and Immune-mediated Movement Disorders

P-064

Unveiling Action Tremor in Multiple Sclerosis : Serial Cases

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Introduction:

Multiple sclerosis (MS) is a chronic central nervous system disorder. Tremor, affecting up to one-third of patients with MS, is rarely an initial symptom but can cause severe disability from cerebellar pathway involvement.

Case:

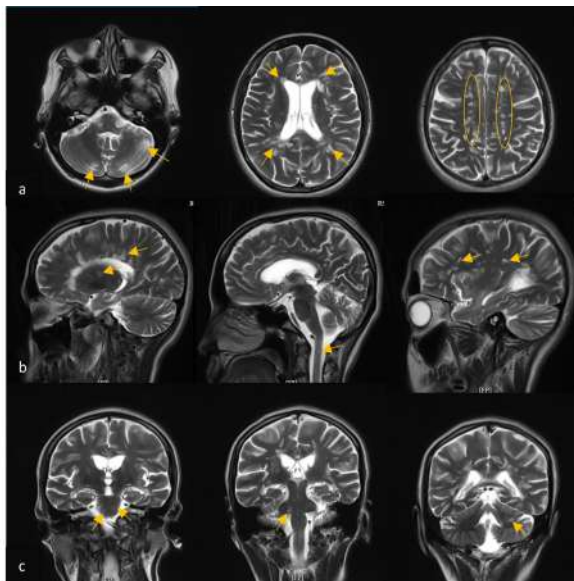
A 28-year-old woman presented with progressive right leg weakness, followed by left-hand and head tremor with right-eye blurred vision. Examination showed monoparesis, cerebellar tremor, cranial nerves palsy, and positive Lhermitte's sign. Brain MRI revealed multiple ovoid demyelinating lesions in periventricular, brainstem, and cerebellar regions. Patient was diagnosed with probable MS with cerebellar ataxia and intentional tremor, treated with pulse dose methylprednisolone, topiramate, and clobazam. Case 2: A 28-year-old woman with Relapsing-remitting MS developed progressive right-hand tremor since 2018 with visual blurring, weakness, spastic hemiparesis, and imbalance. MRI showed demyelinating lesions in the centrum semiovale, corona radiata, frontal lobes. Patient also had severe depression with psychotic features. A diagnosis of kinetic tremor secondary to MS was made, and patient was treated with methylprednisolone, azathioprine, gabapentin, and psychiatric therapy.

Discussion:

Tremor is a disabling manifestation of multiple sclerosis (MS), affecting 25–58% of patients, with meta-analysis reporting 33.3%. It seldom appears at onset, usually emerging years later. Both cases involved young women, consistent with the female predominance (2–3:1). The tremors were action type—intention and kinetic—linked to cerebello-thalamo-cortical lesions, supported by MRI brain and spinal findings. MS tremor is heterogeneous, commonly affecting the upper limbs, though head tremor occurred in Case 1. Management is difficult, as symptomatic

drugs such as topiramate, levetiracetam, clobazam, and clonazepam provide only partial benefit, as seen in our patients who improved modestly yet retained coordination deficits.

Figure



Figure



Keywords

Action Tremor, Multiple Sclerosis, Demyelinating Lesion

P-065

Epstein-Barr Virus Meningitis Followed by Autoimmune GFAP Astrocytopathy with Opsoclonus-Myoclonus Syndrome in an Adolescent: A Case Report

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Introduction:

Autoimmune glial fibrillary acidic protein (GFAP) astrocytopathy is a rare inflammatory central nervous system disorder that can be triggered by preceding viral infections. Epstein-Barr virus (EBV) is a neurotropic virus, but the occurrence of GFAP astrocytopathy following EBV meningitis has rarely been documented.

Case:

A previously healthy 16-year-old female presented with a one-week history of severe headache, dizziness, and nausea. Initial cerebrospinal fluid (CSF) analysis revealed lymphocytic pleocytosis with an opening pressure of 210 mmH₂O, and multiplex PCR from CSF was positive for Epstein-Barr virus. Brain MRI showed decreased ADC at the splenium of the corpus callosum and diffuse leptomeningeal enhancement. The patient was treated with intravenous acyclovir (30 mg/kg/day) and corticosteroid therapy. On hospital day 6, she developed new neurological symptoms including drowsiness, confusion, opsoclonus, and myoclonus. A follow-up CSF analysis again demonstrated lymphocytic pleocytosis, and autoimmune encephalitis panel was additionally requested. This subsequently confirmed anti-GFAP antibody positivity, establishing the diagnosis of autoimmune GFAP astrocytopathy. Upon confirmation, intravenous immunoglobulin (2 g/kg over 5 days) were initiated in addition to ongoing antiviral treatment. The patient's neurological manifestations, including opsoclonus-myoclonus and cognitive impairment, gradually improved with immunotherapy and rehabilitation. She was discharged with a tapering regimen of oral corticosteroids.

Discussion:

This case highlights a rare but important sequence of EBV meningitis followed by autoimmune GFAP astrocytopathy presenting with opsoclonus-myoclonus syndrome. Viral meningoencephalitis, particularly EBV infection, may act as a trigger for secondary autoimmune astrocytopathy in adolescents. Early recognition and timely immunotherapy are essential for favorable outcomes.

P-066

Revealing Alterations In Proteasome And Immune Response In The Substantia Nigra Of Parkinson's Disease: Insights From Geo Database And Experimental Models

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Background & Objective:

Objectives:

To investigate alterations in proteasome function and immune responses in Parkinson's disease (PD) by integrating transcriptomic data from GEO. Additionally, to elucidate whether these changes are linked to the response mechanisms of neurons and PD neurons to influenza virus infection.

Methods:

Gene expression data from the substantia nigra of PD patients and controls were analyzed using the GEO datasets GSE8397 and GSE20292. Batch effects were corrected using the "Combat" algorithm, and differentially expressed genes (DEGs) were identified with the "limma" package in R. GO/KEGG pathway enrichment and immune infiltration analysis of 29 immune cell types was performed. A LUHMES PD cell model was co-cultured with HMC3 microglia and infected with the A/WS/33 H1N1 influenza strain. Flow cytometry was used to analyze immune cell populations, HLA class I/II expression, and viral infection. Cytokine profiles were assessed via ELISA, while Western blotting measured alpha-synuclein phosphorylation, dopaminergic neuron death, and proteasomal/lysosomal protein degradation.

Results:

DEG analysis revealed significant downregulation of CCL5, UBA7, and PSMB5, related to immune and proteasome system. GO/KEGG analysis highlighted alterations in the proteasome pathway, and immune infiltration analysis showed increased HLA-I expression in PD patients. In the PD model, flow cytometry showed reduced influenza nucleoprotein levels compared to controls (healthy neurons), while virus infection led to increased HLA-I expression in the PD group, which was in line with GEO data. ELISA data revealed elevated IL-6 and CCL2 levels in both control and PD groups following infection, while TNF- α , IL-10, IL-1 β , and IL-12p70 levels remained unchanged.

Increased HLA-I expression in PD suggests an enhanced immune response and altered viral susceptibility. HMC3 microglia potentially protect neurons and PD cells from viral infection by regulating IL-6 and CCL2.

Keywords

Parkinson's disease , Influenza virus

P-067

Epigallocatechin-3-Gallate Suppresses Helicobacter pylori Virulence with Implications for Parkinson's Disease Management

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Background & Objective:

Emerging evidence suggests that *Helicobacter pylori* infection is strongly associated with Parkinson's disease (PD), where it exacerbates motor symptoms, accelerates disease progression, and interferes with levodopa absorption. Eradication of *H. pylori* has been shown to improve motor performance and therapeutic responsiveness, highlighting this pathogen as a modifiable risk factor in PD. However, reliance on conventional antibiotics raises concerns regarding resistance and treatment tolerance. This has stimulated growing interest in alternative strategies, particularly natural dietary compounds with antibacterial potential. Green tea, one of the most widely consumed beverages worldwide, contains epigallocatechin-3-gallate (EGCG), a polyphenol with documented inhibitory activity against *H. pylori*. The objective of this study was to investigate the antipathogenic mechanisms of EGCG against *H. pylori* using bioinformatics tools, with the aim of exploring its potential as a dietary intervention to mitigate PD-related risks.

Methods:

In this study, bioinformatics platforms including STITCH v.5.0, VICMPred, and VirulentPred were applied to characterize protein targets, functional roles, and virulence profiles of *H. pylori*. Additionally, BepiPred v.2 and PSORTb v.3 were utilized to predict B-cell epitopes and subcellular localization of bacterial virulence factors.

Results:

Our results revealed that EGCG interacts with critical *H. pylori* virulence determinants, including enoyl-ACP reductase (*fabI*), type II DNA modification enzyme (*jhp_0044*), 7- α -hydroxysteroid dehydrogenase (*jhp_0409*), and type II DNA methyltransferase (*jhp_0435*). These proteins play essential roles in bacterial metabolism, survival, and pathogenicity.

Conclusions:

In conclusion, EGCG from green tea shows promise as a natural dietary agent to suppress *H. pylori* infection, which may in turn reduce its negative impact on PD progression and treatment outcomes. Further laboratory and clinical validation are warranted to confirm these computational findings and assess the broader neuroprotective role of green tea in Parkinson's disease.

Keywords

Helicobacter pylori, Epigallocatechin-3-gallate, Parkinson's disease, Gut-brain axis

P-068

Machine Learning-Derived Immune-Striatal Interaction Score from CSF and PBMC Single-Cell Profiles to Predict Post-Infectious Movement Disorders

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Background & Objective:

Autoimmune responses after infection or vaccination can disrupt basal ganglia circuits and cause movement disorders. Existing diagnostics are non-specific and delay treatment. We aimed to develop a computational immune-striatal interaction score that integrates PBMC, CSF, and striatal receptor data to predict risk and identify therapeutic targets in immune-mediated movement disorders.

Methods:

Single-cell RNA-seq data from PBMC in vaccination-associated encephalitis (GSE205606/7), CSF immune repertoires in narcolepsy (GSE247134), and striatal receptor modules from a

Huntington disease atlas (GSE242195) were harmonized with SCTransform and batch-balanced kNN. Ligand-receptor interactions were mapped using NicheNet and CellPhoneDB to generate immune to striatal HitMaps. The score was derived from ligand load, receptor density, ligand-receptor weights, and graph centrality, trained with XGBoost under leave-dataset-out cross-validation. SHAP defined a 12-analyte biomarker panel, and in-silico simulations modeled score reduction after cytokine blockade.

Results:

The score distinguished cases from controls with AUROC 0.833 (95% CI 0.79-0.87). In encephalitis, scores were higher (median 0.71 vs 0.28, $p < 0.001$) and correlated with CSF pleocytosis ($r = 0.62$) and CRP ($r = 0.53$). Key edges included CXCL13 to CXCR5, CXCL10 to CXCR3, TNFSF13B to TACI, and C3a to C3aR. DRD2 neurons showed stronger TNF and interferon inputs predicting parkinsonism, while DRD1 neurons favored complement and glutamate edges linked to chorea. Simulated blockade reduced scores by -31% with JAK inhibition, -27% with anti-TNF, and -19% with BAFF blockade. The 12-marker panel preserved performance (AUROC 0.812).

Conclusions:

This study presents a computational immune-striatal interaction score linking PBMC and CSF immune states to striatal vulnerability. It predicts risk of immune-mediated movement disorders and enables therapeutic simulations, with a reduced biomarker panel supporting clinical translation.

Keywords

Immune-Striatal, Single-Cell, Post-Infectious, Ligand-Receptor, Biomarkers

Neuroimaging in Movement Disorders

P-069

Association of Olfactory Loss with Cognition is Mediated by Striatal Dopamine Loss and Cerebral Perfusion in Parkinson's Disease

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Background & Objective:

Hyposmia is a common non-motor symptom in Parkinson's disease (PD). Association of hyposmia with cognition has been largely studied, however, the role of subregional striatal dopaminergic degeneration and cerebral perfusion on this association has not been investigated yet. This study investigated whether olfactory loss in PD was associated with subregional striatal dopamine loss and cerebral perfusion and their effect on cognition.

Methods:

This retrospective study reviewed the anonymized data collected during routine clinical care of drug-naïve newly diagnosed patients with PD who underwent the Cross-Cultural Smell Identification Test (CCSIT) and dual-phase N-(3-[¹⁸F]fluoropropyl)-2β-carbomethoxy-3β-(4-iodophenyl) nortropine (18F-FP-CIT PET) scans. General linear models were used to determine the relationship of olfactory loss with subregional dopamine transporter availability (DAT) and cerebral perfusion. A Cox regression model was used to compare the risk of developing PD dementia (PDD) between the groups. A mediation analysis was used to explore whether the imaging features that correlates with olfactory loss mediate the relationship between hyposmia and cognitive outcome.

Results:

343 patients with PD (mean age, 69.8 years; 179 women) were enrolled. DAT availability in all striatal subregions and regional perfusion in the bilateral parieto-occipital area was positively correlated with CCSIT scores. A survival analysis showed that the rate of PDD conversion was significantly higher when CCSIT scores were lower (hazard ratio = 0.86, 95% confidence interval = 0.77–0.95, $P = 0.004$), which was largely mediated by DAT availability in the posterior putamen or parieto-occipital cerebral perfusion in mediation analyses.

Conclusions:

This study demonstrated that the relationship between olfactory loss and dementia conversion was largely mediated by striatal subregional dopaminergic degeneration and regional cerebral perfusion.

P-070

Clinical Efficacy of Repetitive Deep TMS in Patient with Parkinson's disease

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Background & Objective:

Pharmaceutical therapy can provide effective symptomatic relief for Parkinson's disease initially, but symptoms of PD become more complex and refractory to pharmaceutical therapy as the disease progresses, necessitating alternative therapeutic approaches. Repetitive transcranial magnetic stimulation (rTMS) is a noninvasive technique for brain stimulation, which is recommended as a potential therapeutic tool for various neurological and psychiatric disorders. This study aimed to investigate the effects of deep rTMS using an H-coil on cerebral perfusion and motor function in PD.

Methods:

Five PD patients underwent high-frequency dTMS over the supplementary motor area (SMA) for two weeks. Clinical assessments, including the Unified Parkinson's Disease Rating Scale (UPDRS), Hoehn-Yahr scale, and Timed-Up-Go (TUG) test, were conducted before and three months after dTMS. Brain perfusion was evaluated using single-photon emission computed tomography (SPECT) and analyzed with statistical parametric mapping

Results:

There were no statistically significant changes in Hoehn-Yahr scale, TUG, or UPDRS motor scores following dTMS intervention. However, SPECT analysis revealed a significant increase in regional cerebral blood flow (rCBF) in the right cerebellum after dTMS treatment.

Conclusions:

This study showed that the application of high frequency rTMS to the SMA may enhance cerebellar CBF. Although the improvement in motor function after rTMS was not statistically significant, there was a trend toward improvement in motor function tests after rTMS application. Therefore, we cautiously suggest that rTMS procedure may be a new therapeutic option for PD. Further studies are necessary to investigate associations between changes in rCBF and those in motor function tests after rTMS application in PD.

Keywords

Parkinson's disease, deep transcranial magnetic stimulation, regional cerebral blood flow

P-071

Difference Between ¹⁸F-FP-CIT PET and Susceptibility Map-Weighted Imaging in Evaluation of Nigrostriatal Dopaminergic Degeneration

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Background & Objective:

Dopamine transporter imaging such as ¹⁸F-FP-CIT PET is used as the diagnostic criteria by the IPMDS. Currently, susceptibility map-weighted imaging (SMWI) can be used as an emerging imaging biomarker for IPD. Therefore, this study aimed to evaluate association between two imaging biomarkers for evaluation for the diagnosis of primary parkinsonism.

Methods:

Total of 51 patients (29 IPD, 22 non-IPD) who underwent both SMWI and PET within 1 year were collected. For quantitative image analysis, the volume of hyperintensity in the SNpc was measured (mPDIa and N1q, Heuron®), and for DAT binding on PET, the striatal binding ratio (SBR) was measured in each subregion of the brain (BTXBrain-DAT, Brightonix Imaging®). Correlation analysis was used to evaluate the association between the hyperintensity loss in the SNpc and the dopaminergic denervation in the striatum.

Results:

The group with IPD showed a moderate positive correlation between the volume of hyperintensity in the right SNpc and the SBR in the putamen ($p:0.406$, $p=0.029$). However, in the group without IPD, moderate to strong positive correlations were observed as follows: between the volume of hyperintensity in the right SNpc and the SBR in the pallidum ($p:0.632$, $p=0.002$), putamen ($p:0.552$, $p=0.008$), caudate ($p:0.434$, $p=0.044$), and ventral striatum ($p:0.426$, $p=0.048$), and between the volume of hyperintensity in the left SNpc and the SBR in the pallidum

($p:0.632$, $p=0.002$).

Conclusions:

The quantitative image analysis demonstrated that there was lack of significant correlation between striatal dopaminergic denervation by PET and nigral hyperintensity loss by MRI in the group with IPD, but there were several correlations in the group without IPD. These results suggest that PET shows nigrostriatal presynaptic terminal integrity, while SMWI shows microstructural integrity of SNpc, so their associations may be a marker for nigrostriatal tract integrity.

P-072

Automated Deep Learning Model for Differentiating Parkinson's Disease Using Dopamine Transporter Uptake Imaging

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Background & Objective:

Dopamine transporter (DAT) imaging is essential for diagnosing Parkinson's disease (PD) by visualizing presynaptic dopaminergic activity. However, interpretation relies on nuclear medicine specialists, introducing subjectivity and limiting scalability. We developed and validated an automated deep learning model to distinguish PD from normal controls using DAT imaging with clinical variables.

Methods:

A retrospective dataset of 680 patients (41,573 slices) from Haeundae Paik Hospital was used to train a two-dimensional convolutional neural network. Input data included maximum intensity projection (MIP) images and standardized clinical variables (age, sex, UPDRS, Hoehn & Yahr stage). External validation was conducted on 187 patients (5,720 slices) from Dong-A University. Grad-CAM was applied to visualize image regions contributing to predictions.

Results:

On external validation, the model achieved AUROC 0.94, sensitivity 92%, specificity 90%, F1 score 0.91, and accuracy 91%. Grad-CAM confirmed the model focused on clinically relevant striatal regions.

Conclusions:

This CNN-based model demonstrates robust and generalizable performance for differentiating PD from normal controls using DAT imaging. It offers an objective, scalable, and interpretable tool that may support clinical decision-making.

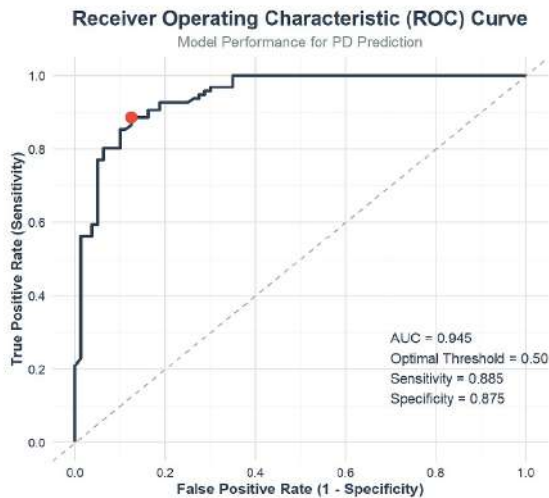
Table

Table 1 Feature Distribution and AI Prediction Accuracy in PD Cases (Total PD = 96)

Features	Correct AI prediction	p-value*
Age		
< 60 (n=76)	20 (100%)	0.113
≥ 60 (n=20)	65 (85.5%)	
Sex		
Male (n=47)	42 (89.4%)	1.000
Female (n=49)	43 (87.8%)	
Area of decreased uptake in Striatum		
Both (n=89)	80 (89.9%)	0.632
Right dominant (n=14)	11 (78.6%)	
Left dominant (n=16)	13 (81.2%)	
Posterior (n=5)	5 (100.0 %)	
Dorsal (n=17)	16 (94.1%)	
Caudal (n=19)	18 (94.7%)	
Ventral (n=5)	5 (100.0%)	

* Statistical differences between groups were analyzed using Fisher's exact test.

Figure



Keywords

Parkinson's disease, Deep learning, Radionuclide imaging

P-073

Clinical Significance of Automatically Assessed Perivascular Space Volume in Subcortical White Matter of Parkinson's Disease

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Background & Objective:

This study aimed to examine the relationship between automatically quantified perivascular space (PVS) volume located in the subcortical white matter (WM), baseline motor deficits and cognitive function, as well as longitudinal motor and cognitive outcomes in patients with Parkinson's disease (PD).

Methods:

This retrospective cohort study included 270 consecutive patients with newly diagnosed PD who underwent brain MRI at initial assessment. Subcortical WM PVS volume was automatically measured using high-resolution T2-weighted images. Multivariate linear regression analyses evaluated the association between subcortical WM PVS volume and baseline Unified PD Rating Scale Part III, Mini-Mental State Examination, and Montreal Cognitive Assessment scores. Cox regression models assessed the prognostic value of baseline subcortical WM PVS volume for the risk of developing freezing of gait (FOG) and dementia.

Results:

Greater subcortical WM volume, particularly in the frontal and parietal regions, correlated with milder parkinsonian motor deficits and better global cognitive function at baseline. Higher subcortical WM PVS volume, especially in the frontal region, was linked to a lower risk of FOG (hazard ratio: 0.450, 95% confidence interval (CI): 0.221–0.916, $p = 0.028$). Additionally, greater subcortical WM PVS volume, especially in the occipital region, was associated with a reduced risk of dementia conversion (hazard ratio: 0.358, 95% CI: 0.134–0.958, $p = 0.041$) in Cox regression models.

Conclusions:

These findings indicate that subcortical WM PVS can be an indicator of unfavorable clinical outcomes in PD.

Keywords

dementia, freezing of gait, Parkinson's disease, perivascular space, subcortical white matter

P-074

Association of Glymphatic Flow at Diffusion Tensor Image With Motor Symptoms and Striatal Dopamine Degeneration in Parkinson's Disease

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Background & Objective:

This study aimed to investigate the association between the diffusion tensor image (DTI) analysis along the perivascular space (ALPS) index, dopamine transporter (DAT) availability in the posterior putamen, and baseline parkinsonian motor deficits in patients with Parkinson's disease (PD).

Methods:

This retrospective study included 207 patients with newly diagnosed PD who underwent brain MRI and 18F-FP-CIT PET between January 2015 and December 2020. The DTI-ALPS index was used to assess glymphatic system function. Motor function was assessed using the Unified Parkinson's Disease Rating Scale Part III (UPDRS-III). Multiple linear regression and mediation analyses were performed to evaluate their associations.

Results:

Multiple linear regression analysis showed that both DAT availability in the posterior putamen and ALPS index were negatively associated with UPDRS-III scores after adjusting for age at symptom onset and sex. Mediation analyses showed that the ALPS index affected UPDRS-III scores both directly ($\beta = -10.116$) and indirectly via DAT availability in the posterior putamen ($\beta = 2.509$). When the ALPS index was located as a mediator, posterior putamen DAT availability affected UPDRS-III scores both directly ($\beta = -5.494$) and indirectly via ALPS index ($\beta = 0.377$).

Conclusions:

These findings suggest that glymphatic dysfunction is associated with severe motor deficits in PD, together with nigrostriatal dopaminergic degeneration.

P-075

Association of Glymphatic Flow at Diffusion Tensor Image With Cognition and Striatal Dopamine Degeneration in Parkinson's Disease

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Background & Objective:

This study aimed to investigate the association between the diffusion tensor image (DTI) analysis along the perivascular space (ALPS) index, dopamine transporter (DAT) availability in the associative striatum, and baseline cognitive function in patients with Parkinson's disease (PD).

Methods:

This retrospective study enrolled 207 patients with newly diagnosed PD who underwent brain MRI and 18F-FP-CIT PET from Yongin Severance Hospital between January 2015 and December 2020. The DTI-ALPS index was used to assess glymphatic system function. Cognitive function was measured using the Mini-Mental State Examination (MMSE). Multiple linear regression and mediation analyses were performed to evaluate their associations.

Results:

Two multiple linear regression analyses were performed to investigate the association with the MMSE scores, DTI-ALPS index, and DAT availability in the caudate (model 1) or anterior putamen (model 2). In model 1, both caudate DAT availability and ALPS index were positively associated with MMSE scores. Mediation analyses showed that the DTI-ALPS index directly affected MMSE scores ($\beta = 1.176$) with no significant indirect effect via caudate DAT availability. When the DTI-ALPS index was located as a mediator, caudate DAT availability directly affected MMSE scores ($\beta = 0.441$) without significant indirect effect via ALPS index ($\beta = -0.006$). In model 2, both anterior putamen DAT availability and ALPS index were positively associated with MMSE scores. Mediation analyses showed that the ALPS index had both direct ($\beta = 1.317$) and indirect effects via anterior putamen DAT availability ($\beta = -0.169$) on MMSE scores. When the ALPS index was located as a mediator, anterior putamen DAT availability affected MMSE scores directly ($\beta = 0.278$) and indirectly via ALPS index ($\beta = -0.441$).

Conclusions:

These findings suggest that glymphatic dysfunction is associated with worse cognitive function in PD, together with nigrostriatal dopaminergic degeneration.

P-076

Striatal Dopamine Dysfunction and Cerebral Perfusion Alterations in Drug-Induced Parkinsonism

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Background & Objective:

This study aimed to investigate whether subtle striatal dopamine deficits and cerebral perfusion alterations are observed in patients with drug-induced parkinsonism (DIP).

Methods:

We consecutively enrolled 82 patients with DIP who showed visually preserved dopamine transporter (DAT) availability in late-phase ¹⁸F-FP-CIT PET scans. We performed comparative analyses of striatal DAT availability on late-phase ¹⁸F-FP-CIT PET images and regional uptake on early-phase ¹⁸F-FP-CIT PET images (i.e., regional cerebral perfusion) between patients with DIP, those with Parkinson's disease (PD; propensity-score matched; n = 164), and healthy controls (n = 50).

Results:

Compared with healthy controls, patients with DIP showed decreased DAT availability in the caudate and reduced cerebral perfusion in the frontal, occipital, and limbic cortices. Patients with DIP also exhibited more severely reduced cerebral perfusion in the frontal and limbic cortices than patients with PD, whereas regional uptake in the occipital lobe was comparable between the DIP and PD groups.

Conclusions:

The results of this study demonstrated that patients with DIP exhibit subtle nigrostriatal dopaminergic dysfunction and cerebral hypoperfusion in the frontal, limbic, and occipital regions.

Keywords

cerebral perfusion, dopamine transporter, drug-induced parkinsonism, Parkinson's disease

P-077

Spatiotemporal Evolution Patterns of Nigrostriatal Dopamine Depletion in Parkinson's Disease

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Background & Objective:

Dopamine neurons in the ventrolateral tier of the substantia nigra pars compacta, which project to the posterior putamen, are known to be the most vulnerable in Parkinson's disease (PD), while there is an individual variability in the involvement of dopamine neurons projecting to the other striatal regions. This study aims to elucidate the spatiotemporal evolutionary patterns of nigrostriatal dopamine depletion in PD.

Methods:

A total of 698 patients with PD and 40 healthy controls who underwent ¹⁸F-FP-CIT PET scans were retrospectively enrolled in this study. After quantifying the dopamine transporter availability in each striatal subregion, we applied Subtype and Stage Inference (SuStaln), a probabilistic machine learning method, to characterize the spatiotemporal progression patterns of striatal dopamine depletion in PD.

Results:

SuStaln analysis identified two subtypes with distinct spatiotemporal evolutionary patterns of nigrostriatal dopamine depletion in PD: Subtype 1 whose striatal dopamine depletion first appeared in the posterior putamen and Subtype 2 with diffuse striatal dopamine depletion. The majority of patients with PD were classified into the Subtype 1, and showed the pattern of progression of striatal dopamine depletion in the following order: more-affected posterior putamen, less-affected posterior putamen, more-affected anterior putamen, more-affected ventral putamen, less-affected ventral putamen, less-affected anterior putamen, more-affected caudate, less-affected caudate,

more-affected ventral striatum, and less-affected ventral striatum.

Conclusions:

Our SuStaln model uncovered the spatiotemporal evolutionary patterns of nigrostriatal dopamine depletion in PD.

Keywords

dopamine transporter, 18F-FP-CIT PET, Parkinson's disease, Subtype and Stage Inference

P-078

Volume and Diffusion Tensor Imaging of the Cholinergic System in Synucleinopathies and Tauopathies

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Background & Objective:

Degeneration of the cholinergic neurons and their projecting fibers is a common feature across various neurodegenerative disorders. Here, we aimed to investigate gray and white matter involvement of the central cholinergic system in patients with synucleinopathies and tauopathies.

Methods:

We recruited patients with synucleinopathies (47 idiopathic REM sleep behavior disorder [IRBD], 181 Parkinson's disease [PD], 38 dementia with Lewy bodies [DLB], and 37 multiple system atrophy [MSA]), tauopathies (16 corticobasal syndrome [CBS], 31 progressive supranuclear palsy [PSP], and 112 Alzheimer's disease [AD]), and 67 healthy controls (HC). Diffusion tensor imaging was used to calculate free water-extracted mean diffusivity (FWeMD) in the pedunclopontine nucleus (PPN)-thalamus (TH) pathway (PPN-TH) and in the medial and lateral nucleus basalis of Meynert (NBM)-cortical pathways (NBM-MP and NBM-LP, respectively). 3D T1-weighted images were used to measure NBM volume.

Results:

Compared to HCs, NBM volume was reduced in all neurodegenerative groups except MSA. Patients with DLB and AD had smaller BF volume than those with IRBD and PD. FWeMD in the PPN-TH pathway was higher in PD, DLB, MSA, CBS, and PSP than in HC, IRBD, and AD. FWeMD in the NBM-MP pathway was higher in AD than in HC, PD, MSA, and PSP. Within the PD spectrum, FWeMD in the PPN-TH pathway was higher in PD than in IRBD, while NBM volume was lower in PDD than in PD. Within the AD spectrum, FWeMD in the NBM-MP pathway was higher in AD dementia than in prodromal AD, whereas NBM volume in prodromal AD was lower than in HC but higher than in AD dementia. Cholinergic imaging markers distinguished each neurodegenerative disorder and correlated with global cognition.

Conclusions:

PD was characterized by early damage to the PPN-TH pathway followed by later BF degeneration, whereas AD showed early BF degeneration and subsequent involvement of the NBM-MP pathway, without PPN-TH involvement. Both PPN-TH and NBM were prominently affected in DLB.

Keywords

Cholinergic, Diffusion tensor imaging, Parkinson's disease, Alzheimer's disease, Dementia with Lewy bodies

P-079

Hippocampal Subfields Shape in Early Parkinson's Disease: A Longitudinal MRI Study

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Background & Objective:

Hippocampal subfields are important in cognitive function in Parkinson's Disease (PD). This prospective cohort study longitudinally investigated hippocampal subfield shape deformation and atrophy in early-stage PD, using spherical harmonic-based shape analysis.

Methods:

A cohort of early PD (n=146; disease duration=14.9 ± 9.5 months; 44% female) and matched healthy controls (HC) (n=59; 56% female) underwent MRI and cognitive assessments with follow-up after two years. Hippocampal subfields were segmented using FreeSurfer, and shape deformations were analysed via SPHARM-PDM (Fig 1).

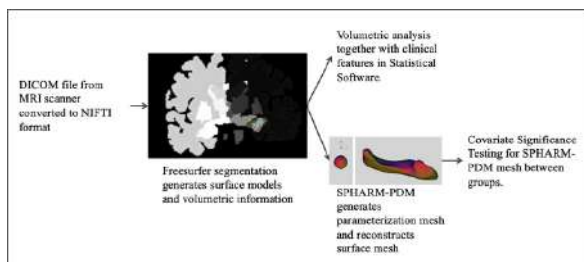
Results:

Shape analysis identified selective morphological changes in hippocampal subfields (CA1, CA3, subiculum, and fimbria) at baseline in early PD patients compared to HCs, despite no volumetric differences. Longitudinally, atrophy in the right hippocampus, and selective hippocampal subfields (right CA1, and left fimbria) were present. In the PD-Mild Cognitive Impairment (PD-MCI) subgroup (n=27), localised shape deformations in CA1, CA3, GC-ML-DG, and fimbria were observed at baseline when compared to PD-Normal Cognition (PD-NC) (n=84; Fig 2). Longitudinally, left fimbria atrophy emerged as a sensitive biomarker predicting cognitive deterioration, especially in visuospatial and memory domains.

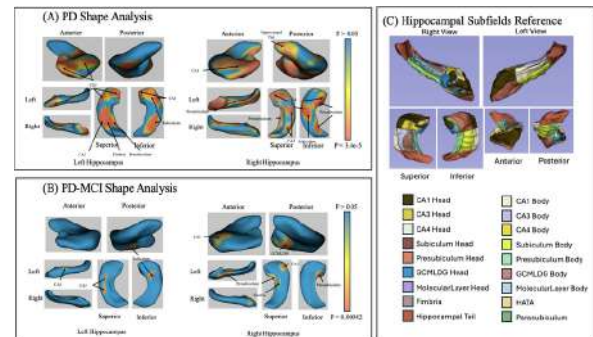
Conclusions:

Shape analysis provides superior sensitivity to volumetric analysis for detecting neurodegeneration that precedes gross volumetric atrophy in de-novo PD. This study highlights the potential of hippocampal subfield shape analysis for early detection of cognitive decline in PD. One of the subfields, left fimbria, arises as a promising biomarker for predicting cognitive decline in PD and guiding early targeted interventions. This is the first study of longitudinal hippocampus shape analysis in early PD patients, and used spherical harmonics based shape analysis, which is different from traditional FSL-FIRST protocol, and provides higher sensitivity for surface morphometry.

Figure



Figure



Keywords

magnetic resonance imaging, shape analysis, morphometry, parkinson's disease, hippocampus

P-080

An Elderly Man With a 4-Year History of Gait Unsteadiness and Intermittent Falls: Clinical Usefulness of Dopaminergic Imaging for Atypical Parkinsonism

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Introduction:

Accurate differential diagnosis of atypical parkinsonism remains challenging because clinical features are often nonclassical.

Case:

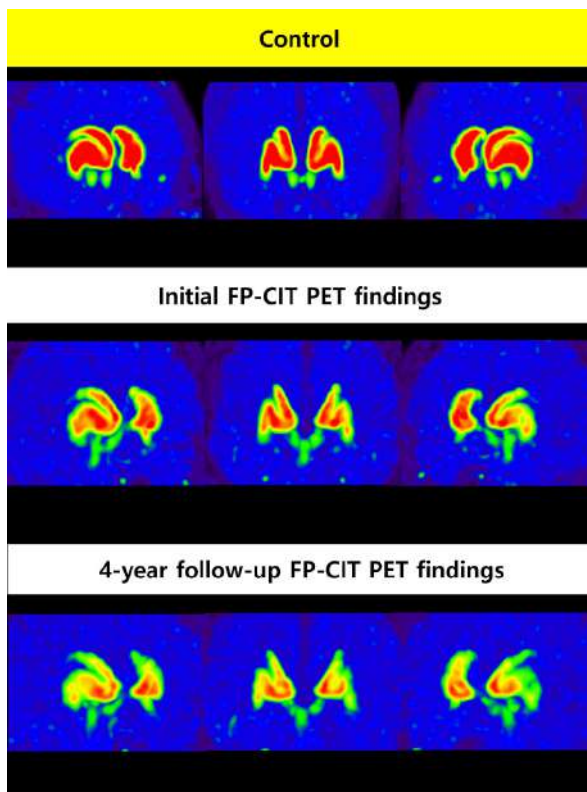
A 76-year-old man presented with gait unsteadiness and intermittent falls over six months. Neurological examination showed mild slowness but no tremor, rigidity, or ataxia. Brain magnetic resonance imaging (MRI) was unremarkable. Initial ¹⁸F-FP-CIT PET (Fig 1. Middle part) demonstrated mild dopamine depletion in the left posterior putamen and caudate nucleus. Over the following years, he experienced progressive slowness and recurrent falls. By year four, falls occurred several times per month, and examination revealed impaired vertical saccades ("round-the-houses" sign), mild bradykinesia, and bilateral postural instability during tandem gait. Repeat ¹⁸F-FP-CIT PET (Fig 1. Lower part)

revealed progressive dopamine loss in both posterior putamen and caudate nuclei, a pattern atypical for PD. During subsequent follow-up, extraocular movement slowed further with partial limitation of downgaze. The patient developed moderate bradykinesia, severe postural instability, frequent falls, and required assistance to walk. Despite treatment with levodopa (600 mg daily) and safinamide (50 mg daily), motor symptoms persisted. Ultimately, he met Movement Disorders Society criteria for clinically probable progressive supranuclear palsy (PSP).

Discussion:

This case highlights the diagnostic value of dopaminergic imaging in atypical parkinsonism. In Parkinson's disease, dopamine loss predominates in the posterior putamen relative to the caudate, whereas PSP typically shows comparable loss across both regions. In conclusion, careful integration of clinical findings and detailed dopaminergic imaging can improve diagnostic precision and management of parkinsonian syndromes.

Figure



P-081

Gait Impairment Associated With Neuroimaging Biomarkers in De Novo Parkinson's Disease

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Background & Objective:

Gait impairment is a cardinal feature of Parkinson's disease (PD), but the neural correlates underlying quantitative gait dysfunction in medication-naïve patients remain poorly understood. This study investigated the relationships among quantitative gait parameters, clinical symptoms, cognitive function, and neuroimaging biomarkers in de novo PD patients.

Methods:

We enrolled 130 patients with de novo PD and 184 healthy controls. Quantitative gait analysis was performed using the GAITRite system. All participants underwent ¹⁸F-FP-CIT PET imaging and brain MRI with FreeSurfer volumetric analysis. Path analysis examined direct and indirect relationships among dopaminergic dysfunction, brain volumes, motor symptoms, cognition, and gait parameters.

Results:

De novo PD patients exhibited significantly slower gait velocity, shortened step and stride lengths, increased double support time, and greater gait variability compared to controls (all $p < 0.0001$). Gait parameters correlated significantly with dopamine transporter binding in the putamen, with direct effects comprising 87% of the total effect on step length. Limbic system volumes (anterior cingulate, thalamus, hippocampus, amygdala) showed both direct (64-75%) and cognition-mediated indirect effects on gait parameters.

Conclusions:

These findings demonstrate that gait dysfunction in de novo PD results from both direct dopaminergic deficits and limbic system involvement, with cognitive-motor interactions contributing to gait variability. The identification of specific neural correlates supports the utility of quantitative gait analysis as an early diagnostic biomarker and suggests targets for personalized therapeutic interventions.

Other Movement Disorders

P-082

Subacute Sclerosing Panencephalitis in a 27-Year-Old Male: A Forgotten Threat in the Post-Measles Era

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Introduction:

Subacute sclerosing panencephalitis (SSPE) is a rare, progressive, and often fatal neurological disorder resulting from persistent infection with the measles virus. SSPE usually manifests 7–10 years after measles infection, though the latency varies from 1 month to 27 years. Despite widespread immunization programs significantly reduced disease burden, measles remains common in developing countries.

Case:

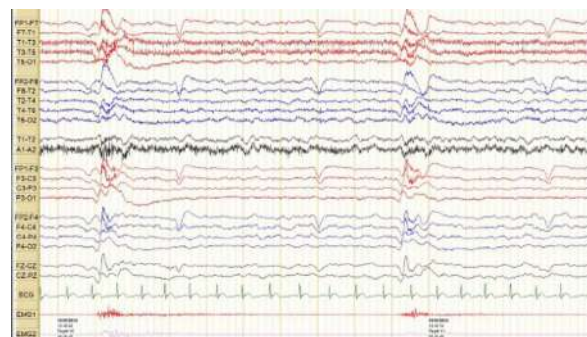
A 27-year-old male presented with a one-year history of progressive myoclonic jerks, initially involving sudden axial muscle contractions described as being "thrown backward," later progressing to bilateral, rhythmic upper limb jerks occurring every 7–10 seconds. Approximately 10 months prior to presentation, he developed cognitive decline and regression of speech, which gradually worsened. Neurological examination revealed normal visual acuity and extraocular movements, with no cranial nerve deficits. The patient's family was unable to provide a reliable vaccination history and denied any known history of measles infection. Diagnosis of SSPE was confirmed by elevated anti-measles IgG titers in cerebrospinal fluid (7.77 IU/mL) and serum (7.33 IU/mL).

Discussion:

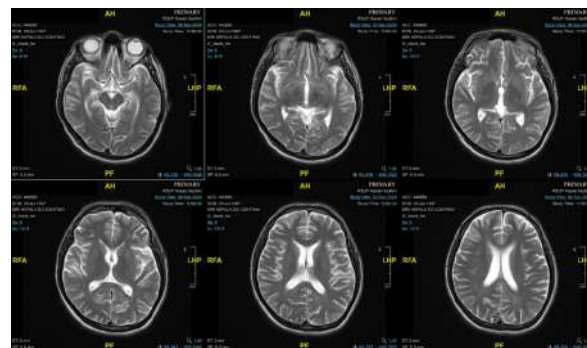
While most measles infections resolve without sequelae, the virus may remain dormant for some time before triggering an inflammatory response which presents as SSPE. Diagnosis was established using Dyken's criteria, with the presence of two major criteria; elevated anti-measles antibody titers in serum and CSF, and a characteristic progressive clinical course (although the age of onset was atypical) — and one minor criterion, the presence of periodic, stereotyped electroencephalographic discharges (Radermecker complexes). Therapeutic interventions for SSPE remain limited, costly, and non-curative, with treatment available only in select specialized centers worldwide. Thus,

effective measles vaccination remains the most reliable and cost-effective strategy for preventing this devastating neurological disorder.

Figure



Figure



Keywords

Subacute Sclerosing Panencephalitis, Measles, Myoclonic

P-083

Adult-Onset Huntington's Disease With Unusual Presentation of Dopa-Responsive Lower Limb Dystonia

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Introduction:

Huntington's disease (HD) is an inherited, progressive neurodegenerative disease. The predominant motor symptoms tend to

vary by the onset age. Presentation beginning with lower limb dystonia is rare, particularly with documented dopa-responsiveness. Herein, we report a case of an adult-onset HD initially presenting as dopa-responsive lower limb dystonia.

Case:

A 27-year-old female patient presented with a six-month history of stiffness in her right leg while walking. She felt the stiff rotation of her pelvis and intorsion of the right foot during ambulation. She swirled her pelvis to circumscribe the right leg and swung it faster than the left to reduce the swing phase. Her right foot often exhibited inversion. Blood tests were non-contributory to her symptoms. An ophthalmologic examination revealed no Kayser-Fleischer rings. CSF analysis excluded hypoglycorrachia. Panel genetic tests yielded negative results. Brain MRI and dopamine imaging showed no abnormalities. A presumptive diagnosis of dopa-responsive dystonia was made by partial response to levodopa. After two years, she developed involuntary twisting movements of her lips and tongue, followed by irregular, involuntary movements in her right hand and foot. CAG expansions (17/44 alleles) in the *HTT* gene were confirmed. She was ultimately re-diagnosed with atypical adult-onset HD, initially presenting as dopa-responsive lower limb dystonia.

Discussion:

Age at onset has been proposed as a factor influencing distinct endophenotypes. HD has been shown to exhibit age-related pathobiological variability. The patient's age at onset falls between the known juvenile and late-onset forms of HD, where shared characteristics may reside and act differently. This may explain the transformation of the motor phenotype from dystonia to hyperkinesia. This case highlights the unusual initial presentation of adult-onset HD and the importance of surveilling phenotypic evolution.

P-084

Normal Pressure Hydrocephalus, the Underappreciated Entity in the Diagnosis of Cognitive Decline With Gait Difficulty

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Background & Objective:

Hydrocephalus, normally in the case of normal pressure hydrocephalus, manifests deterioration in either cognition or walking, which is often misdiagnosed as another entity of dementia or Parkinsonism. Previous literature reported that approximately 6% of the whole dementia population is accounted for, although this percentage might vary and be underappreciated. It is estimated that less than 20% of patients with NPH are correctly diagnosed. We sought to explore the actual and practical prevalence of dementia by hydrocephalus in the clinical practice of the single institute cohort.

Methods:

The current registry-based retrospective cohort study investigated 137 patients who were diagnosed with dementia in a single institution.

Results:

Of 137 total study population, 63(46.0%) were diagnosed Alzheimer dementia (AD) whom are followed by hydrocephalus (n=34, 24.8%), vascular dementia (n=25, 18.2%), frontotemporal dementia (n=5, 3.6%), dementia with Lewy body (n=2, 1.5%), and undetermined (n=8, 5.8%). Patients whose MMSE score is >20 but early behavioral issues were higher in the hydrocephalus (n= 18/31, 58.1%), which was greater than AD (n= 5/36, 13.9%; $p < 0.001$). In neuropsychiatric inventory test in patients whose MMSE > 20, the hydrocephalus group demonstrated higher prevalence in the domains of delusions (hydrocephalus vs AD; 15/31 (48.4%) vs 4/36 (11.1%); $p < 0.001$) and disinhibition (12/31 (38.7%) vs 2/36 (5.6%); $p < 0.001$). In 14 out of 137 total study population who were previously diagnosed with AD at a previous institution or private clinic elsewhere, twelve (12/14, 85.7%) were re-evaluated to be diagnosed as dementia by hydrocephalus. All of them presented either mild frontal gait or signs of disproportionately enlarged subarachnoid space hydrocephalus and callosal bowing compared to those of AD.

Conclusions:

Our data demonstrated that the prevalence of dementia by hydrocephalus may be higher than being understood to date. Dementia of hydrocephalus might be misdiagnosed as AD.

P-085

Stereotypy and Other Movement Disorders following Osmotic Demyelinating Syndrome: A Case Report

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Introduction:

Background: Osmotic demyelinating syndrome (ODS) is an uncommon and serious neurological condition characterized by demyelination from oligodendrocyte loss, primarily caused by rapid serum sodium correction. Striatal involvement is associated with an array of hypokinetic and/or hyperkinetic movement disorders; however, stereotypy has never been recorded in ODS.

Objective: This paper aims to present a case of stereotypy as a unique manifestation of ODS in a Filipino adult female, along with the co-occurrence of other abnormal involuntary movements.

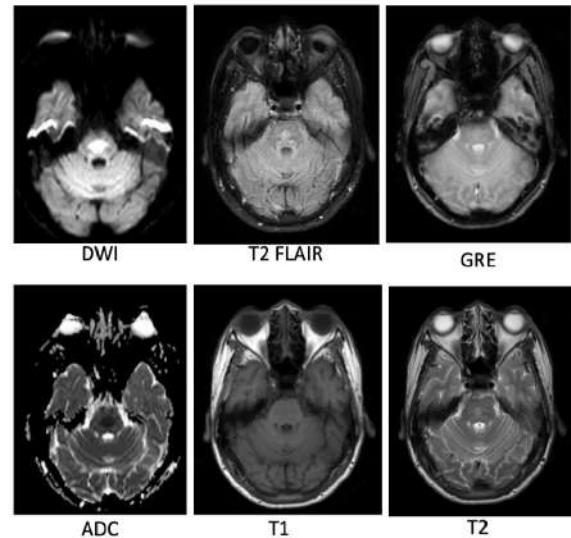
Case:

A 63-year-old Filipino woman had severe hyponatremia (99.7 mmol/L) due to inadequate oral intake during a COVID-19 infection. Unfortunately, this was rapidly corrected at a net increase of 16.3 mmol/L in 20 hours. Subsequently, dysarthria and dysphagia developed. ODS was confirmed by cranial MRI findings of both central pontine myelinolysis (CPM) and extrapontine myelinolysis (EPM) of the bilateral corpus striatum. Initially, symptoms improved; however, parkinsonian signs emerged six months later. Stereotypy followed after three months, exhibited as involuntary, repetitive, and purposeless kicking and stomping movements while lying supine. Other movement disorders followed shortly, including dystonia, chorea, mild myoclonus, and possible catalepsy.

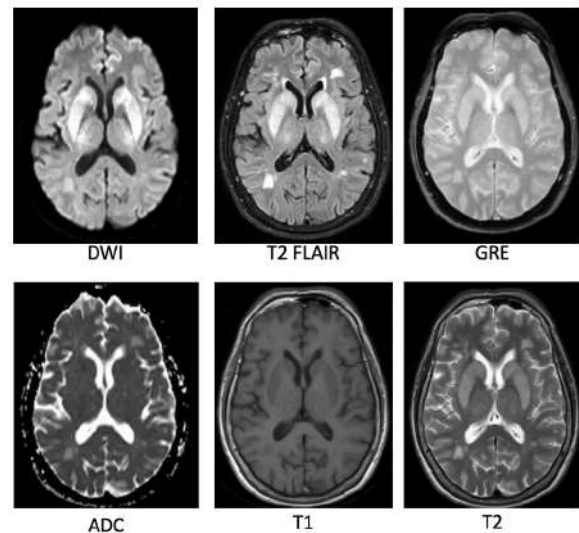
Discussion:

Dysarthria and dysphagia may be due to CPM of the corticobulbar tract in the basis pontis or secondary to bradykinesia of orolingual movements. Meanwhile, EPM could lead to various movement disorders due to its effect on the basal ganglia; however, stereotypical movements have never been recorded in ODS. These involuntary movements can be diverse, appear late, evolve over time, and persist despite treatment. Adherence to safe correction rates of sodium (<8–10 mmol/L/day) is crucial to prevent iatrogenic complications.

Figure



Figure



Keywords

Osmotic demyelination syndrome, stereotypy, movement disorders

P-086

Hemichorea As the Presenting Feature of Polycythemia Vera: A Case Report

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Introduction:

Chorea is characterized by involuntary, irregular movements that are typically random and flowing. It is linked to various causes, including genetic, immunologic, vascular, infectious, metabolic, or drug-induced origins. In some cases, chorea is confined to one side, a condition referred to as hemichorea. Polycythemia vera (PV) is a myeloproliferative neoplasm marked by erythrocytosis, with possible leukocytosis, and thrombocytosis. Neurologic symptoms occur in 50%–80% of PV cases, but chorea is rare, reported in only 0.5% to 5% of cases. These cases are usually generalized with orofacial and brachial involvement. This report describes a patient in whom hemichorea was the only manifestation of PV.

Case:

We report a 55-year-old Filipino woman presenting with sudden-onset right-sided involuntary movements and subtle orofacial dyskinesia. She had no history of stroke, no autoimmune, metabolic, or infectious diseases, nor any medication use. Neurologic examination showed right-sided choreiform movements involving both proximal and distal limbs, with minimal facial involvement. Lab results revealed elevated hemoglobin (17.5 g/dL) and hematocrit (54.8%), leukocytosis, thrombocytosis, decreased erythropoietin, and positive JAK2 V617F mutation. Brain MRI showed no acute lesions. A provisional diagnosis of PV was made. She was treated with hydroxyurea, aspirin, and valproic acid. Improvement was noted after phlebotomy. One month later, her hemoglobin and hematocrit normalized, and choreiform movements had mostly resolved, allowing her to resume normal function.

Discussion:

While the exact mechanism by which chorea develops from PV is still unknown, several hypotheses are being investigated. This case highlights hemichorea as a rare but possible sole presenting feature of PV. It should be considered in the differential diagnosis of new-onset chorea in middle-aged and older patients. Early diagnosis and appropriate hematologic treatment can lead to complete symptom resolution.

Table

Table 1. Results of serial complete blood count of the patient on first presentation, post-phlebotomy, and on follow up.

	Day 1 First presentation	Day 3 Post- phlebotomy	Day 31 Follow-up
Red blood cell count (per $10^{12}/L$)	6.16	5.08	4.75
Hemoglobin (g/dL)	17.5	14.4	13.4
Hematocrit (%)	54.8	44.9	42.5
Mean corpuscular volume (fL)	89	88	90
Mean corpuscular hemoglobin concentration	0.319	0.321	0.315
Mean corpuscular hemoglobin (pg)	28.4	28	28
White blood cell count (per $10^9/L$)	23.0	20.64	13.01
Platelet count (per $10^9/L$)	619	555	366

Keywords

Hemichorea, Chorea, Polycythemia vera

P-087

Hemichorea-Hemiballismus as Initial Presentation of Cerebral Toxoplasmosis With Unusual Serology In HIV Positive: A Rare Case Report

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Introduction:

Movement disorders related to opportunistic infections in people with Human Immunodeficiency Virus (HIV) occur in about 2–3%, most often choreiform or parkinsonism. Hemichorea–hemiballismus as the initial manifestation of cerebral toxoplasmosis is rare. In most patients with cerebral toxoplasmosis, anti-Toxoplasma IgG is positive, but our case was unusual with negative IgG and positive IgM serology.

Case:

A 44-year-old male presented with 2 days of involuntary movements of the right limb and slurred speech, preceded by progressive headache and 17-kg weight loss over 1 month. Neurological exam was otherwise normal, except for irregular, non-rhythmic, large-amplitude choreiform and ballistic movements of the right limb. Brain MRI with contrast revealed multiple ring-enhancing lesions in the left thalamus, bilateral basal ganglia, frontal to occipital lobes, and cerebellum, suggestive of cerebral toxoplasmosis. HIV was confirmed with CD4 count 16 cells/ μ l. Serology showed anti-Toxoplasma IgM positive but IgG negative. Differential diagnoses ruled out included cryptococcosis (CrAg negative), tuberculosis (negative), syphilis (TPHA negative), primary CNS lymphoma (neuroimaging not compatible), and drug-induced causes (none). Therapy with dexamethasone, trimethoprim-sulfamethoxazole, pyrimethamine, and clindamycin was started, with haloperidol and clonazepam for abnormal movements. The patient improved and was discharged with ART planned two weeks later.

Discussion:

The movement disorder likely resulted from subcortical involvement, especially basal ganglia and subthalamic nuclei, common targets of HIV and *T. gondii* and diffuse cerebral involvement affecting the central dopaminergic pathway. The rarity lies in the IgM+/IgG– serology, as IgG positivity is usually universal in cerebral toxoplasmosis. Effective management requires both causative treatment against *T. gondii* and symptomatic neuroleptics or GABAergic drugs. Early recognition is essential for favorable outcome.

Keywords

hemichorea-hemiballismus, cerebral toxoplasmosis, HIV

P-088

Patient-Perceived Response to Medical Treatment in Hemifacial Spasm

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Background & Objective:

Compared to botulinum toxin injection and surgery, the therapeutic response to oral medications for hemifacial spasm (HFS) remains underexplored. This study aimed to compare patients' perceptions of the therapeutic response to medication for HFS.

Methods:

We reviewed records of 117 patients who received pharmacological treatment upon diagnosis of HFS. Four medications—clonazepam, trihexyphenidyl, carbamazepine, and baclofen—were prescribed sequentially for seven days each. Patients recorded whether each medication was beneficial, and therapeutic responses were compared using Cochran's Q test.

Results:

Among 63 patients in the final analysis, 37 (59%) reported a favorable response to at least one medication. However, each medication showed a therapeutic response of approximately 20–30%, with no significant differences overall ($p=0.400$) or within the subgroup of 27 patients with neurovascular contact on magnetic resonance imaging ($p=0.972$).

Conclusions:

Oral medication may serve as a useful initial treatment for HFS, with careful consideration of potential adverse effects.

Table

Table 1. Demographic and Baseline Characteristics of Patients

Total	63 (100%)
Male	19 (30%)
Female	44 (70%)
Age of Onset, years	55.2 (12.2)
Age of Visit, years	58.1 (11.0)
Duration of Disease, months	33.6 (57.6)
> 1 year	29 (48%)
≤ 1 year	31 (52%)
History of Facial Palsy (n=44) *	
Yes	12 (27%)
No	32 (73%)
IAC MRI (n=40) *	
Neurovascular contact	27 (68%)
Absent neurovascular contact	13 (32%)

Note: Data are presented as mean (SD) or N (%)

* Percentages for history of facial palsy and IAC MRI findings are based on available data.

Abbreviations: IAC MRI = internal auditory canal magnetic resonance imaging

Figure

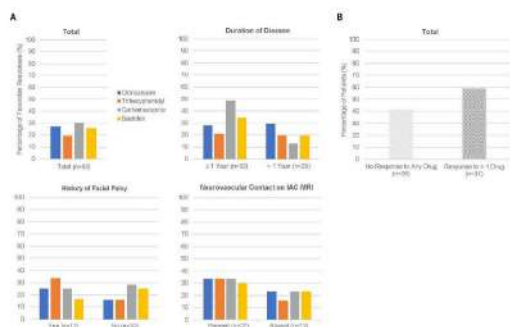


Figure 1. (A) Percentage of favorable responses to each medication in the overall population and across subgroups defined by disease duration, history of facial palsy, and IAC MRI findings. (B) Proportion of patients who responded to at least one medication versus none.

Table

Table 2. Patient-Reported Favorable Responses to Medications in Hemifacial Spasm

	Clonazepam n/Total no. (%)	Trihexyphenidyl	Carbamazepine	Baclofen	p-value*
Total (n=63)	17/63 (27)	12/63 (19)	19/63 (30)	16/63 (25)	0.400
Duration of HFS					
> 1 year (n=29)	8/29 (28)	6/29 (21)	14/29 (48)	10/29 (34)	0.065
≤ 1 year (n=31)	9/31 (29)	6/31 (19)	4/31 (13)	6/31 (19)	0.334
History of facial palsy					
Yes (n=12)	3/12 (25)	4/12 (33)	3/12 (25)	2/12 (17)	0.753
No (n=32)	5/32 (16)	5/32 (16)	9/32 (28)	8/32 (25)	0.313
Neurovascular contact on IAC MRI					
Present (n=27)	9/27 (33)	9/27 (33)	9/27 (33)	8/27 (30)	0.972
Absent (n=13)	3/13 (23)	2/13 (15)	3/13 (23)	3/13 (23)	0.925

*p-values were calculated using Cochran's Q test to compare therapeutic responses across the four medications within each subgroup.

Abbreviations: HFS = hemifacial spasm; IAC MRI = internal auditory canal magnetic resonance imaging

Keywords

Hemifacial Spasm, Medications, Treatment, Patient-centered

P-089

Early Phenotypic Features of Beta-Propeller Protein-Associated Neurodegeneration: Insights from a Korean Series

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Background & Objective:

Beta-propeller protein-associated neurodegeneration (BPAN) is a rare X-linked disorder caused by pathogenic variants in WDR45 gene. Early diagnosis remains challenging due to nonspecific presentations in childhood.

Methods:

We report six pediatric patients with BPAN, identified through genetic testing performed during the evaluation of neurodevelopmental disorders.

Results:

All were female and exhibited early developmental delay, severe language impairment, and varying degrees of motor dysfunction. Seizures occurred in four patients with varying severity. Two patients showed signs of central precocious puberty. Serum neuron-specific enolase was elevated in all tested patients. Brain MRI revealed corpus callosum thinning in all cases. Iron accumulation in the substantia nigra and globus pallidus was observed in only two older patients. WDR45 variants included two nonsense, two splice-site, one in-frame deletion, and one novel frameshift deletion.

Conclusions:

Our findings highlight early clinical features that may aid in recognizing BPAN prior to the emergence of distinctive MRI abnormalities or degenerative-phase manifestations.

Keywords

Beta-propeller protein-associated neurodegeneration, Neurodevelopmental disorder, Enolase, Magnetic Resonance Imaging

P-090

Two Cases of Middle Cerebral Artery Occlusion Mimicking Hemifacial Spasm

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Introduction:

Hemichorea refers to continuous, irregular, involuntary movements involving one side of the body. Middle cerebral artery (MCA) occlusion rarely presents as predominant hemifacial dyskinesia, leading to diagnostic delays. We report two patients with abrupt-onset hemifacial spasm caused by MCA occlusion secondary to Moyamoya disease and cardioembolic stroke

Case:

Case 1: A 62-year-old woman presented with continuous, involuntary movements of the left face, initially suspected to be hemifacial spasm. Neurological examination showed choreiform movements of the left face, with subtle involvement fingers and foot. Brain MRI showed no acute lesions, while cerebral angiography demonstrated right distal internal carotid narrowing with MCA and ACA obliteration, consistent with Moyamoya disease. Brain SPECT revealed cortical hypoperfusion in the right frontoparietal area. Her symptoms improved spontaneously within one month, correlating with development of collateral circulation. Case 2: A 55-year-old woman with atrial fibrillation and mitral stenosis presented with sudden-onset left-sided facial movement. On neurological examination, choreic movements were prominent in the left face with slight fingers and foot involvement. CT angiography revealed acute right MCA occlusion. Spontaneous recanalization occurred within 24 hours, resulting in complete symptom resolution.

Discussion:

These cases indicate that hemifacial dyskinesia may be a presenting neurological symptom of MCA occlusion in the absence of its other classical clinical features. Clinicians should consider MCA occlusion as differential diagnosis of abrupt onset hemifacial dyskinesia. Early recognition and vascular evaluation are essential for distinguishing MCA occlusion from primary hemifacial spasm and for preventing secondary ischemic events

P-091

Challenges in the Diagnosis and Management of Functional Movement Disorders in a Developing Nation: Insights from the Philippines

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Background & Objective:

This study examines how age, gender, symptom duration, and clinical phenotypes relate to Functional Movement Disorders (FMDs) in the Philippines. It discusses diagnostic and management challenges, such as care fragmentation and economic constraints, and analyzes how education influences adherence to psychiatric referrals and treatment plans.

Methods:

This cross-sectional study integrated with a review of individual patient data aimed to investigate the demographic, clinical, and behavioral characteristics of patients diagnosed with FMD in the Philippines.

Results:

The study included 33 patients, with a female predominance (69.7%) and a mean age of symptom onset in the late 20s. Tremor was the most frequently observed phenotype (42%), followed by dystonia (30%), facial spasms (24%), jerks/myoclonus (24%), and weakness (6%). Psychiatric comorbidities were common, with anxiety disorders (36.6%) and mood disorders (33.33%) being the most prevalent. A significant delay of approximately 1.87 years from symptom onset to initial medical consultation was noted, with a strong correlation between duration of symptoms and time to first consultation ($r = 0.804$, $p < 0.001$). Only 54.5% of patients referred for psychiatric care returned for follow-up, highlighting gaps in continuity of care. Educational attainment appeared to moderate health-seeking behavior, with college-educated patients more likely to pursue follow-ups than those with only a high school education.

Conclusions:

The findings highlight the complex biopsychosocial nature of FMD and the need for a holistic, interdisciplinary approach. Addressing FMD in the Philippines requires policy reform, improved primary care training, public education, standardized diagnostics, multidisciplinary neuropsychiatric care, longitudinal studies, and digital health technologies for diagnosis, monitoring, and therapy.

P-092

Quality of Lives of Caregivers in Huntington's Disease Families in Korea: K-HDQoL-C Analysis From the KHDC Database

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Background & Objective:

Huntington's disease (HD) is a progressive neurodegenerative disorder that burdens caregivers, causing distress and reduced quality of life (QoL). Although the Korean version of the HD QoL Battery for Carers (K-HDQoL-C) has been validated, studies on caregiver QoL in HD remain scarce, particularly regarding patient clinical features. We aimed to identify clinical and neuropsychiatric factors associated with caregiver QoL.

Methods:

This cross-sectional study included 82 caregivers of genetically confirmed HD patients from a multicenter Korean cohort (KHDC). Caregiver measures were demographics, Zarit Burden Interview-12 (ZBI-12), and Neuropsychiatric Inventory (NPI). Patient variables were motor/functional status (Unified Huntington's Disease Rating Scale, UHDRS), cognition (MMSE, MoCA), and neuropsychiatric symptoms. Caregiver QoL was assessed using K-HDQoL-C (Part 2: aspect of caring; Part 3: satisfaction with life; Part 4: feelings about life). Spearman correlation was applied.

Results:

Mean caregiver age was 54.2 years; 53.7% female. Lower UHDRS functional assessment ($r=-0.252$), independence ($r=-0.248$), and functional capacity ($r=-0.342$) correlated inversely with Part 2. Lower MMSE ($r=-0.230$) also showed inverse correlation. Greater neuropsychiatric symptoms (NPI frequency*severity $r=0.249$; distress $r=0.371$) correlated positively with Part 2. Higher UHDRS independence ($r=0.253$) and functional capacity ($r=0.301$) correlated positively with Part 3. ZBI-12 correlated positively with Part 2 ($r=0.282$) and Part 4 ($r=0.325$), and inversely with Part 3 ($r=-0.441$). All $p<0.05$.

Conclusions:

Caregiver burden was greater with poorer patient functional and cognitive status, while higher distress was linked to negative caregiving perceptions and lower life satisfaction. Greater independence and functional capacity were associated with higher life satisfaction. These findings support that multidisciplinary care for patient's functional, cognitive and emotional status reduce the caregiver burden and their QoL.

Keywords

Huntington's disease, caregiver, Quality of life, cohort, neurodegenerative

P-093

Evaluating the Feasibility of Hybrid Therapeutic Education for Neurodegenerative Disorders: A Systematic Synthesis and Meta-Analysis

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Background & Objective:

Mixed therapeutic education and telerehabilitation are increasingly used in patients with neurodegenerative disorders such as Multiple System Atrophy (MSA) and Parkinson's Disease (PD). However, evidence regarding the feasibility of these programs remains limited. This study aimed to assess the feasibility (retention/program completion) of primary studies evaluating virtual, in-presence, or hybrid interventions in patients with MSA/PD.

Methods:

The PRISMA method used in the literature search identified eight relevant studies; three primary studies with numerical retention data were analyzed: Fayet et al. (2025, MSA pilot, n=15), Dorsey et al. (2012, RCT of virtual housecalls for PD, n=20), and Johnson et al. (2024, pilot RCT of telerehabilitation for PD, n=20). Data on retention proportions (events/n) were pooled using a DerSimonian–Laird random-effects meta-analysis model with logit transformation. Analysis was performed on the primary outcome of participant retention.

Results:

A total of 55 participants (MSA and PD) from three studies were included; 52 completed the program (individual retention: 86%, 100%, and 95%). Meta-analysis yielded a pooled retention of 92.2% (95% CI: 79.8–97.3%), with low heterogeneity ($Q=1.59$; $\tau^2=0$; $I^2=0\%$). Studies reported high satisfaction, good acceptability, and no serious adverse events. Clinical outcomes such as quality of life (PDQ-39, EQ-5D), motor function (Mini-BESTest, TUG), and disease scores showed improvements in several domains, but results were heterogeneous across studies.

Conclusions:

Thus, this blended education and telerehabilitation program for MSA/PD patients demonstrated high feasibility, with average retention >90% and acceptance by both patients and caregivers. Preliminary clinical evidence suggests potential benefits on quality of life and motor function, although numerical data are limited. Larger-scale studies with uniform outcome reporting are needed to evaluate clinical effectiveness and long-term impact

Keywords

Hybrid Therapeutic Education, Neurodegenerative Disorder, PRISMA

Parkinson's Disease: Medical Treatment

P-094

Herbal Remedy Treatments for Parkinson's Disease Patients in the Philippines

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Background & Objective:

Parkinson's disease (PD) is a progressive neurodegenerative disorder characterized by motor and non-motor symptoms. In the Philippines, conventional pharmacotherapy is widely used, but interest in complementary herbal remedies is growing.

Methods:

A structured literature search was conducted across PubMed, Scopus, and Google Scholar for English-language articles (2018–2025) using keywords "Parkinson's disease," "herbal," and "Philippines." Inclusion criteria encompassed clinical trials, observational studies, ethnobotanical surveys, and preclinical research on herbal interventions relevant to PD. Data were extracted on study design, population, intervention, outcomes, and safety.

Results:

Philippine PD patients commonly self-medicate with readily available herbs, motivated by cost, cultural acceptance, and perceived safety. *Mucuna pruriens* shows the most promise—mirroring global findings of levodopa content and clinical efficacy—yet formal RCTs in the Philippine context are lacking. Curcumin and ginger benefit from robust preclinical data but need human trials. Ethnobotanical surveys highlight sambong and tawa-tawa, but mechanistic and clinical validation is pending. Key challenges include variability in herbal preparations, unstandardized dosing, potential herb–drug interactions, and the absence of regulatory oversight.

Conclusions:

Herbal remedies hold potential as adjunctive therapies for PD in the Philippines. *Mucuna pruriens* leads in clinical promise, while curcumin and ginger warrant translational research. A concerted effort to standardize preparations, rigorously test efficacy, and monitor safety will be critical to integrating these botanicals into holistic, culturally sensitive PD management.

Table

Herb (Local Name)	Active Component(s)	Mechanism	Philippine Evidence	Global Evidence
<i>Mucuna pruriens</i> (barag)	Levodopa	Dopamine precursor	Manila pilot: +20 % UPDRS-III, mild GI upset	RCTs: similar efficacy to L-DOPA
<i>Curcuma longa</i> (luyang dilaw)	Curcumin	Antioxidant, anti-inflammatory	Ethnobotanical use for stiffness	Rodent PD: motor deficits
<i>Zingiber officinale</i> (luya)	Gingerols	Anti-inflammatory, antioxidant	Patient-reported muscle relief	Preclinical: neuroprotection (rats)
<i>Blumea balsamifera</i> (sambong)	Flavonoids, tannins	Anti-inflammatory, diuretic	Folk use to reduce rigidity and edema	Limited PD-specific data
<i>Euphorbia hirta</i> (tawa-tawa)	Polyphenols, flavonoids	Antioxidant, anti-inflammatory	In vitro neuroprotective effects (UP Diliman)	Emerging preclinical evidence

Keywords

herbal remedy for PD, systematic review, *Mucuna pruriens*

P-095

Correlation Between Vitamin D and Hoehn & Yahr Stage in Parkinson's Disease: A Structured Review of Current Evidence

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Background & Objective:

Parkinson's disease (PD) is a progressive neurodegenerative condition primarily manifested by motor impairment. The Hoehn and Yahr (H&Y) scale remains the most commonly applied tool for evaluating disease severity. Recent findings suggest a potential association between vitamin D status and PD progression. This study aims to systematically investigate the relationship between serum 25-hydroxyvitamin D (25[OH]D) concentrations and H&Y staging in PD, with a particular emphasis on young-onset Parkinson's disease (YOPD).

Methods:

A PRISMA-compliant meta-analysis was performed using data from 30 eligible studies (25 observational, 3 randomized controlled trials, 2 meta-analyses) involving over 6,400 patients across various regions. Data extracted included vitamin D concentration, H&Y stage, correlation coefficients, and subgroup analyses in YOPD patients. Study quality was assessed using Newcastle-Ottawa and Jadad scales.

Results:

Most investigations have demonstrated a significant inverse association between vitamin D concentration and Hoehn and Yahr (H&Y) stage, with correlation coefficients ranging from $r = -0.1$ to -0.7 . A total of fifteen representative studies comprising more than 3,000 participants reported consistent findings, with weighted mean differences (WMD) between -5.27 and -11.6 ng/mL. Interventional trials indicated that vitamin D3 supplementation was associated with a slower progression of motor symptoms. Subgroup analyses focusing on young-onset Parkinson's disease (YOPD) showed comparable patterns, although interpretation was constrained by the limited number of studies and small sample sizes. From a mechanistic perspective, vitamin D is thought to influence disease progression by supporting dopaminergic neuron survival and attenuating neuroinflammatory processes.

Conclusions:

Vitamin D deficiency is consistently associated with increased severity of Parkinson's disease (PD), including in young-onset subgroups. The role of vitamin D as both a biomarker and a potential therapeutic target is increasingly supported by current evidence. Nevertheless, the existing literature highlights the need for further age-stratified and longitudinal studies, particularly in young-onset PD populations, to clarify causality and long-term effects. Considering its safety profile and biological plausibility, routine oral vitamin D supplementation may be recommended as an adjunctive strategy to attenuate disease progression in PD patients.

Table

Table 1. Extracted Data Matrix

Study	Sample Size (N)	Mean Vit D (ng/mL)	H&Y Range	Correlation (r)
Suzuki et al. 2023	114	25.1	1-4	-0.46
Sato et al. 2020	300	22.5	1-5	-0.51
Senel et al. 2021	152	21.7	1-3	-0.33
Beyazal & Yildiz 2017	78	19.6	1-4	-0.41
Peterson et al. 2013	286	24.8	1-5	-0.191
El Sayed et al. 2022	78	14.2	1-4	-0.45
Habibi et al. 2019	56	17.9	1-3	-0.52
Martinez-Horta et al. 2024	102	27.4	1-3	-0.38
Akinyemi et al. 2023	94	18.3	1-5	-0.29
Costa et al. 2023	88	16.7	2-5	-0.42
Chen et al. 2022	204	21.3	1-4	-0.35
Mahmoudi et al. 2021	83	20.5	1-3	-0.27
Kaya et al. 2020	76	19.1	1-4	-0.36

Figure

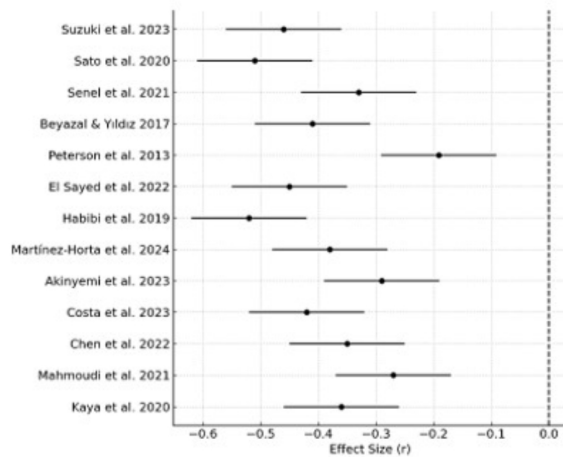
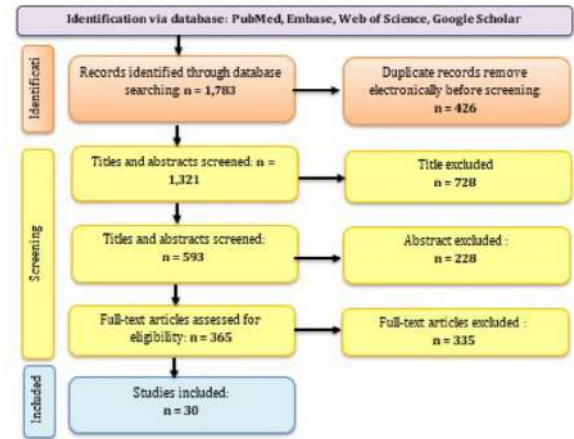


Figure 1. Forest Plot.

Correlation between serum vitamin D levels and Hoehn & Yahr stage in Parkinson's disease

Figure



Keywords

Vitamin D, Parkinson, YOPD, Hoehn Yahr

P-096

Burning Mouth Syndrome Linked With Parkinson's Disease

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Background & Objective:

Burning mouth syndrome (BUS) is a painful intraoral sensation that is not associated with any physiological and laboratory results signifying a pathology. Post-menopausal women more affected with BUS and it is mostly an old age disease. A reduction in the endogenous dopamine and dopamine dysregulation are the possible aetiology. Burning mouth syndrome can occur as an aftermath of Parkinson's disease. We aimed to study the short- (≤ 3 months) and long-term (> 3 months) effectiveness and sustainable benefit of the treatment strategies and the associated side effects.

Methods:

Retrospective analysis was performed on data collected from 10 patients undergoing treatment with carbidopa/levodopa 25/100mg titrated to 1.5 tablets three times daily. There was reduction in symptoms of Parkinsonism but they developed BUS. Antifungal therapy was given with Candid V6 vaginal tablets without any resolution. The Parkinson's drug was suspended

temporarily and patient was put on Pramipexole. There was reduction in the symptoms in all of them.

Results:

Nine of 10 patients (90.0%) reported improvement in pain severity and in global mood improvement. Treatment was successful due the withdrawal of dopamine and replacing it with a D2 and D3 dopamine receptor agonist. The excessive presence of dopamine D1 activity compared to D 2 activity was the probable reason for BUS in this patient.

Conclusions:

Based on these results, Pramipexole appears to be an efficacious treatment option. The treatment was well tolerated with less adverse events.

P-097

Botulinum Toxin the Bullet To Take Charge of Sialorrhea

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Introduction:

Sialorrhea can be troublesome symptom in patients suffering from Parkinson's disease. Various modalities are available such as anticholinergic drugs, radiation therapy, surgery, and the behavioural therapy and the potent being botulinum neurotoxin injection. In this case series we would like to highlight the positive outcome and adverse events if any in the administration into the major salivary glands.

Case:

Patients with Parkinson's disease presenting with sialorrhea were administered with 2500 U botulinum neurotoxin type B. Ultrasound guided injections were given bilaterally into the submandibular and parotid salivary gland. The patients were evaluated at 1 week then at and 3 months after the first injection. Results There was both subjective and objective improvement in the patients. Most of the symptoms were normal within a week after the administration. There was 100 % technical success rate and overall effectiveness of the procedure was 72 %. Repeated injection was not an enhancer of clinical success rate. The toxin related side effect was an increase in salivary thickness. There were no major side effects in the procedure.

Discussion:

The injection of botulinum toxin had improved the oral quality of life and further it is effective and safe as a drug.

P-098

Development of Natural Exosome-Based Nanocarriers for Sustained Intranasal Delivery of Rotigotine in Parkinson's Disease

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Background & Objective:

Parkinson's disease (PD) is a progressive neurodegenerative disorder characterized by dopaminergic neuronal loss and impaired neurotransmission. Conventional drug therapies, including dopaminergic agonists like rotigotine, face limitations such as poor bioavailability, rapid metabolism, and limited blood–brain barrier (BBB) penetration. This study aims to develop a natural exosome nanovesicle (EN) based delivery system derived from garlic (G), colocasia (C), maize (M), and potato (P), for efficient intranasal delivery of rotigotine. The objective was to achieve high drug encapsulation efficiency, sustained drug release, enhanced nasal mucosal permeability, and potential BBB penetration to improve therapeutic efficacy in PD.

Methods:

ENs were isolated from natural sources and characterized for particle size, polydispersity index (PDI), and zeta potential using a Malvern Zetasizer. Morphological integrity was confirmed through fluorescence microscopy and transmission electron microscopy (TEM). Rotigotine was loaded into ENs by co-incubation, and encapsulation efficiency was quantified using a validated HPLC method (C8 column, 60:40 acetonitrile:buffer, $\lambda = 224$ nm). In vitro drug release was assessed using a Franz diffusion cell with dialysis membrane, while ex vivo permeation was studied across goat nasal mucosa. Drug release and permeation profiles of exosomal formulations were compared with free drug formulations.

Results:

ENs exhibited nanoscale particle sizes (119.1–145.5 nm) with spherical morphology and maintained structural stability before and after drug loading. Zeta potential values (-1.20 to -9.49 mV) confirmed colloidal stability. High drug encapsulation efficiency was achieved (91.51–99.32%). In vitro release studies demon-

strated sustained and nearly complete drug release (~99–100%) within 24 hours for exosomal formulations, compared with only 68.65% for the free drug at 30 hours. Ex vivo studies revealed significantly improved nasal mucosal permeation, with maize-derived ENs achieving ~97.49% penetration within 6 hours, while free drug reached only 54.99%. Overall, exosome formulations provided superior retention, sustained release, and enhanced mucosal transport compared to free rotigotine.

Conclusions:

The study demonstrates that natural exosome nanovesicles serve as a stable, biocompatible, and highly efficient drug delivery system for rotigotine. EN-based formulations achieved superior encapsulation, sustained release, and enhanced mucosal permeation, underscoring their potential for non-invasive, intranasal, nose-to-brain delivery in Parkinson's therapy. Future work will focus on in vivo brain distribution, toxicity profiling, and therapeutic efficacy evaluation to enable clinical translation of this exosome-based neurotherapeutic platform

Keywords

Exosome nanovesicles, Rotigotine, Intranasal drug delivery, Parkinson's disease, Sustained release

P-099

DSP-4-Induced Noradrenergic Lesion as a Model of Levodopa-Resistant Dyskinesia: Role of the Norepinephrine Pathway

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Background & Objective:

While levodopa (LD) remains the gold-standard therapy for Parkinson's disease (PD), a subset of patients shows limited benefit due to neurochemical changes beyond the dopaminergic system. Degeneration of the locus coeruleus (LC) and norepinephrine (NE) depletion may influence LD responsiveness. We aimed to develop a dual-lesion PD model by combining rotenone-induced nigrostriatal damage with DSP-4-mediated LC ablation to replicate LD resistance and enable preclinical therapeutic screening.

Methods:

Male C57BL/6 mice (8–10 weeks) were allocated to control, rotenone, rotenone+LD, or rotenone+DSP-4+LD groups. Rotenone

(2.5 mg/kg/day, i.p., 21 days) induced nigrostriatal degeneration. DSP-4 (50 mg/kg, i.p., day 14) selectively ablated LC neurons. LD (20 mg/kg, i.p.) was given acutely before behavioral testing (open field, rotarod, bar test) on day 22. Striatal DA and NE levels were quantified by HPLC-ECD. Tyrosine hydroxylase (TH) immunohistochemistry assessed neuronal integrity in substantia nigra pars compacta (SNpc) and LC.

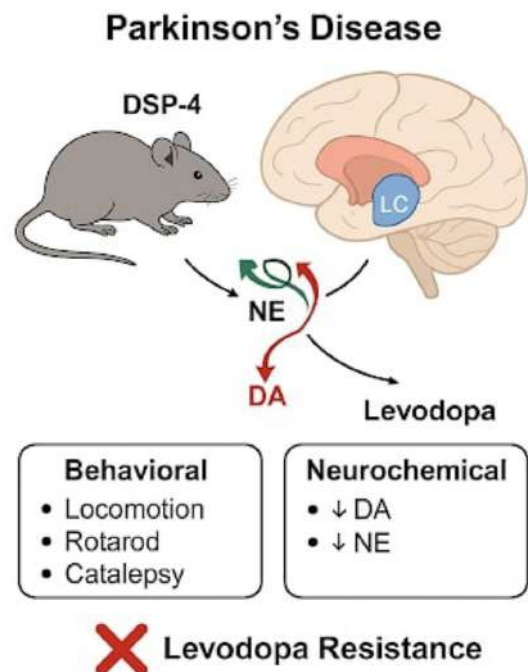
Results:

Rotenone reduced motor activity and striatal DA (~65%, $p < 0.001$), all improved by acute LD ($p < 0.01$). DSP-4 co-lesion further depleted DA (~78%) and NE (~85%, $p < 0.001$) and abolished LD-induced motor recovery ($p > 0.05$ vs. rotenone baseline). TH staining confirmed marked SNpc and LC neuronal loss.

Conclusions:

The combined rotenone–DSP-4 paradigm yields a reproducible PD model with clinically relevant LD resistance, highlighting the LC–NE system's role in maintaining dopaminergic therapy efficacy. This dual-lesion approach offers a translational platform for testing interventions targeting NE–DA interplay.

Figure



Keywords

DSP-4, Levodopa resistance, Parkinson's disease, locus coeruleus, norepinephrine

P-100

Opicapone for Nocturnal Hypokinesia in Patients With Parkinson's Disease: An Open-Label Pilot Clinical Trial

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Background & Objective:

Nocturnal hypokinesia (NH) is common in patients with moderate-stage Parkinson's disease (PD). To enable the accurate assessment of NH, the Nocturnal Hypokinesia Questionnaire (NHQ) was developed. This study aimed to assess the effect of bedtime opicapone administration on NH and to evaluate its clinical utility.

Methods:

This study was designed as an open-label, single-arm pilot clinical trial involving patients with PD and NH. The primary endpoint was the change in NHQ score from baseline to week 6. Secondary endpoints included changes over six weeks on various motor and non-motor scales. The safety of opicapone was evaluated based on the incidence of adverse events.

Results:

Fifteen patients were included in the study. The mean change of NHQ score after the 6-week treatment was -0.9 points (95% confidence interval [CI]: -2.4, 0.5; $p=0.187$), which was not statistically significant. However, NHQ Domain 2 (getting out of bed) demonstrated a significant improvement, with a mean change of -0.3 points (95% CI: -0.6, -0.1; $p=0.025$). No secondary endpoints showed significant differences after opicapone treatment. The baseline NHQ score exhibited significant correlations with the Movement Disorder Society-Unified Parkinson's Disease Rating Scale Part II score ($p=0.543$; $p=0.036$), Parkinson's Disease Questionnaire-39 total score ($p=0.548$; $p=0.035$), and New Freezing of Gait Questionnaire score ($p=0.566$; $p=0.028$). Opicapone treatment was well tolerated.

Conclusions:

Nighttime administration of 50 mg opicapone in patients with PD and NH improved nighttime mobility. The NHQ allows for a more detailed and specific assessment of NH compared to other sleep questionnaires and may serve as a useful tool for evaluating NH in future clinical studies.

P-101

Vutiglabin, a PON2 Activator, in Phase 2a Clinical Trial as a Novel Treatment for Early Parkinson's Disease

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Background & Objective:

Parkinson's disease (PD) is a progressive neurodegenerative disorder characterized by dopaminergic neuron loss, mitochondrial dysfunction, and oxidative stress. Recent therapeutic strategies have shifted from symptomatic relief to disease modifying therapies that can slow disease progression. PON2 is an enzyme that plays a crucial role in antioxidant defense and mitochondrial function. Therefore, we developed Vutiglabin, a novel orally active small molecule designed to augment PON2 activity as a potential therapeutic agent for PD.

Methods:

In preclinical studies, Vutiglabin effectively crossed the blood-brain barrier and bound to PON2, leading to neuroprotective effects. It significantly reduced mitochondrial dysfunction, oxida-

tive stress, and neuroinflammation in mouse models of PD. This protected dopaminergic neurons, improved motor function, and was shown to be entirely dependent on PON2 activity.

Results:

Building on these promising preclinical findings and favorable safety data from a Phase 1 clinical trial, we are currently conducting a randomized, double-blind, placebo-controlled Phase 2a clinical trial (NCT06329141) in patients with early PD. The primary objectives are to evaluate the safety and efficacy of Vutiglabin. The primary endpoint is the changes in motor function measured by the MDS-UPDRS Part III score. Secondary and exploratory endpoints include assessments of non-motor symptoms, cognitive function, and paraoxonase activity in the blood.

Conclusions:

The results from this trial will bridge robust preclinical data with clinical evidence, supporting Vutiglabin as a novel disease-modifying therapy for PD.

Keywords

paraoxonase, mitochondria, Vutiglabin, parkinson's disease, Phase 2a

P-102

AUTAC as a Novel Strategy for the Degradation of α -Synuclein Aggregates in Parkinson's Disease

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Introduction:

Parkinson's disease (PD) and related synucleinopathies are characterized by pathological aggregation of misfolded α -synuclein (α -syn), leading to neuronal dysfunction and progressive neurodegeneration. Effective clearance of α -syn aggregates remains a major therapeutic challenge, as conventional PROTAC-based targeted protein degradation (TPD) strategies are limited to soluble proteins and show little efficacy against large aggregates.

Case:

In this study, we developed a series of 34 autophagy-targeting chimeras (AUTACs) designed to selectively degrade α -syn aggregates via the autophagy-lysosome pathway. Each AUTAC consists of an α -syn-binding warhead (Anle138B) linked to a

guanine-derived degradation tag that recruits the autophagy receptor SQSTM1/p62, facilitating selective sequestration of α -syn aggregates into autophagosomes. Treatment of A53T α -syn-expressing cell lines with AUTAC resulted in a marked reduction of α -syn aggregates.

Discussion:

These findings suggest that α -syn-targeting AUTACs represent a promising therapeutic strategy for the targeted reduction of pathological α -syn aggregation in PD and related disorders.

P-103

Investigating Supervisor-Supported Medication Adherence in Parkinson's Patients with Cognitive Impairment

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Background & Objective:

Levodopa remains the first line treatment for PD and some parkinsonism; however, it has strict instructions. Adherence to medication improves clinical outcome. Many factors affect treatment success, including cognitive status and medical supervisors. This study aims to describe the impact of MS on treatment adherence in Parkinsonism, especially those who have cognitive impairment.

Methods:

Study population were outclinic patients with parkinsonism receiving Levodopa treatment who came in July 2025. Subjects were divided into two groups: with and without supervised medication. Adherence is defined as correct dose, time punctuality and correct instructions. MoCA-INA was used to assess cognitive status.

Results:

A total 23 patients were included in the study and divided into two groups: good adherence (n=19) and bad adherence (n=4). Most are male (n=15) and the average age were 61.2 (41-79) years. Parkinson's disease made up the largest group (n=13), followed with vascular parkinsonism (n=5) and atypical parkinsonism (n=5). Cognitive impairment was discovered in 15 (65.2%) subjects, with the average MoCA-INA score of 21 (5-29). Most

have moderate impairment (n=8). Approximately 43% of patients have MS, who was either their spouse (60%) or their children. Subjects with cognitive impairment tend to have medication non-adherence compared with those without cognitive impairment (3/12 (25.0%) vs 1/7 (14.3%), $p=1.0$). The presence of MS was associated with a trend toward good medication adherence, albeit statistically significant (9/10 (90.0%) with MS vs 10/13 (76.9%) without MS, $p=0.6$).

Conclusions:

Cognitive impairment may negatively affect medication adherence. The presence of medication supervisors may play an important role in addressing this issue. A larger sample size is needed to overcome the current statistical limitations.

Table

Tabel 1. Characteristics of patients

	Good adherence	Bad adherence
Sex		
Male	14	1
Female	6	3
Age		
<50	5	1
51-60	2	1
61-70	8	
>70	4	2
Type of parkinsonism		
Parkinson's disease (PD)	12	1
Non-PD		
Vascular	4	1
Atypical	3	2
Medication supervisor		
Yes	9	1
No	10	3
Type of medication supervisors		
Spouses	5	1
Children	4	-
Medications		
Levodopa only	10	3
Levodopa and Pramipexol	1	1
Levodopa and trihexyphenidyl	3	-
Levodopa+pramipexole+THP	5	-
Cognitive impairment (MoCA-Ina score)		
Normal	7	1
Mild cognitive impairment	5	1
Moderate cognitive impairment	7	1
Severe cognitive impairment	0	1

Table

Tabel 2.

	Adherence		P
	Good	Bad	
Cognitive impairment			
Yes	12	3	1.0
No	7	1	
Supervised medication			
Yes	9	1	0.6036
No	10	3	

Keywords

cognitive impairment, levodopa, medication supervisors, parkinson's disease

P-104

Harnessing the Local Wisdom of Central Kalimantan's *Eleutherine palmifolia* (Dayak Onion) in Parkinson's Disease Therapy: A Molecular Docking and Ethnopharmacological Study"

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Background & Objective:

Dayak Onion is a traditional plant used by the Dayak people (Kalimantan, Indonesia) to empirically treat various ailments. Various studies have shown this plant to be rich in compounds with potential as therapeutic candidates. This is relevant given that current therapy for Parkinson's disease (PD) is limited to alleviating clinical symptoms. It is hoped that this research will reveal new hope for PD therapy. The purpose of this in silico study was to examine the potential protective effects of chemicals found in dayak onion against PD

Methods:

In this work, we used a variety of bioinformatics approaches, including network pharmacology, molecular docking, and molecular dynamics, to investigate the possible therapeutic advantages and illustrate the fundamental mechanisms of dayak onion compounds against PD.

Results:

The 10 target proteins can interact with all chemicals, according to molecular docking studies. Possible chemicals from dayak onion can be used to assess the ESR1, HSP90AA1, and AKT1 proteins in PD in silico. The chemicals methylantraquinone, eutherine, and anthraquinone have the lowest docking scores against a number of target proteins when compared to levodopa, which is their control. Among the target proteins, Methylantraquinone compound has the lowest docking scores (kcal/mol): HSP90AA1 (-11.18), AKT1 (-10.34), and ESR1 (-8.53). At -10.08 for HSP90AA1, -10.07 for AKT1, and -8.53 for ESR1, Eutherine has the lowest docking scores. Meanwhile, the Anthraquinone compound is known to have the lowest docking score against the HSP90AA1 protein (-10.58), AKT1 (-9.73) and ESR1 (-8.25).

Conclusions:

Dayak onion contain methylantraquinone, eutherine, and anthraquinone molecules that have a good pharmacokinetic profile and can bind to protein targets implicated in PD. They can be submitted for additional in vitro and in vivo studies.

Keywords

Parkinson disease, Bawang dayak, Methylantraquinone, Eleutherine, Anthraquinone

P-106

Disease-Modifying Therapeutic Strategies Targeting Alpha-Synuclein in Parkinson's Disease: A Systematic Review

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Background & Objective:

Parkinson's disease (PD) is a progressive neurodegenerative disorder characterized by the accumulation of misfolded alpha-synuclein (a-syn) aggregates, which play a central role in its pathogenesis. Given the limitations of current symptomatic therapies, a-syn has emerged as a promising therapeutic target for disease modification.

Methods:

A systematic literature search was conducted in PubMed, Scopus, Web of Science, and Cochrane Library from January 2010 to July 2025. Eligible studies included randomized controlled trials, preclinical investigations, and clinical trials evaluating a-syn-targeted interventions. Data were extracted on therapeutic strategies, efficacy, safety, and clinical outcomes.

Results:

Eighteen studies met the inclusion criteria, comprising 10 preclinical and 8 clinical trials. Therapeutic strategies were grouped into three main categories: (1) immunotherapy (monoclonal antibodies and vaccines), which demonstrated significant reduction in a-syn aggregation and favorable safety in early-phase trials; (2) small molecules and aggregation inhibitors, which showed promise in preventing a-syn misfolding and promoting clearance in preclinical models, though clinical translation remains limited; and (3) gene-silencing and RNA-based therapies, which effec-

tively reduced a-syn expression in animal models, with ongoing clinical evaluation. While most interventions were well tolerated, consistent disease-modifying effects in large-scale clinical trials are yet to be established.

Conclusions:

Targeting a-syn represents a promising avenue for disease modification in PD. Immunotherapy and gene-silencing approaches are the most advanced, while small molecules hold potential for future translation. However, current evidence remains preliminary, highlighting the need for large, long-term clinical trials to confirm efficacy and establish a-syn-directed therapies as a cornerstone in P. V management.

Keywords

Disease-Modifying Therapeutic, Targeting Alpha-Synuclein, Parkinson's Disease

Parkinson's Disease: Motor Features

P-107

Elucidating the Genetic Alterations in *MT-ND1* Gene, Mitochondrial Membrane Potential (MMP) and Clinical Phenotypes in Parkinson's Disease (PD) Patients in Indian Population

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Background & Objective:

Parkinson's disease (PD) is a neurodegenerative disorder accompanied by depletion of dopamine and loss of dopaminergic neurons (DA) in the brain that is believed to be responsible for the motor and non-motor symptoms of PD. However, the association of possible factors with PD is need to be investigated in Indian population. The aim of the present study is to determine the association of lifestyle, environmental factors, biochemical parameters, mitochondrial membrane potential (MMP) and genetic insights of the complex I which are the most vulnerable part of mtDNA such as *MT-ND1* gene in PD patients.

Methods:

Using a standardised questionnaire and UPDRS scaling, PD patients and control group of about 182 study participants were interviewed on demographic, lifestyle and environmental factors. Mitochondrial DNA was isolated from blood samples followed by PCR and sequencing data to unravel the mutations in *MT-ND1* gene and understand the mitochondrial copy number variations among the study participants. The Mitochondrial membrane potential (MMP) was analysed in the EOPD and LOPD subjects lymphocytes using Mitoprobe JC-1 Assay kit (M34152) in the Attune next flow cytometry.

Results:

In our study, the *MT-ND1* alteration with A3843G variant was significant at $p < 0.05$ for AG allele in all the three PD groups but the highest prevalence was observed in late-onset group. Also, we found that the occupational exposure to agriculture and industry showed an increased risk among the late-onset group. The biomarkers uric acid (UA) and dopamine (DA) were significant at $p < 0.05$ in all the three PD age groups. The mitochondrial membrane potential (MMP) analysis, LOPD showed slight variations in the MMP compared to EOPD subjects.

Conclusions:

Thus, the mutational screening initiated the research in mtDNA; moreover, this study aid to focus future research in identifying mtDNA copy number and sequencing analysis in PD. In conclusion, we suggest that further research is mandatory to detect other possible associations among PD.

P-108

Mutational Analysis of *TFAM* Gene and Analysis of Oxidative Stress Among Parkinson's Disease (PD) Patients From Punjab Population

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Background & Objective:

Parkinson's disease (PD) is a progressive motor system disorder which distress several parts of the brain, in particular substantia nigral area that is characterised by the loss of dopaminergic neurons. Mutations/polymorphisms in mitochondrial DNA (mtDNA) have been implicated in PD. The mitochondrial transcription factor A (TFAM) controls the transcription of mtDNA and regulates the mtDNA-copy number, thus being important for maintaining ATP production and maintain the oxidative stress in the cell. The goal of this case-control study was to investigate the genetic alterations in the *TFAM* gene and its related oxidative stress among the PD patients from Punjab population, India.

Methods:

52 PD patients and 52 sex- and age-matched controls were included and we found that we recruited $n=37$ male and $n=15$ female study subjects among the PD and control subjects. The PD patients were categorised as late onset PD (LOPD) and early onset PD (EOPD) where we had recruited $n=46$ LOPD subjects while $n=6$ EOPD subjects. Unified Parkinson's disease Rating Scale (UPDRS) and informed consent was obtained. Genomic DNA was isolated and mutational analysis was performed. The levels of SOD and CAT for oxidative stress among PD subjects were analysed by HPLC.

Results:

We found that PD patient's plasma had lower superoxide dismutase (SOD) and higher catalase (CAT) activity, resulting in lower SOD/CAT ratio. It was observed that abnormalities in mitochondrial complexes (I, III, V) are more common in PD, and it may have resulted for oxidative stress. Also, we found that the PD patients had variation in the *TFAM* gene at exon 1 in region S12T more significantly among the LOPD subjects than the controls.

Conclusions:

In conclusion, our findings authenticate that mutations or alterations in the *TFAM* gene could enhance the chance of oxidative stress which has a potential role behind PD and we recommend to conduct more such concurrent research to understand their unique mechanism for better diagnosis and therapeutic for PD.

P-109

Bacopaside-I Attenuates Neurodegeneration and Motor Deficits in a Rat Model of Parkinson's Disease via Modulation of Oxidative Stress and Neuroinflammation

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Background & Objective:

Chronic administration of *Bacopa monnieri* extract has shown neuroprotective potential in various models of neurodegenerative diseases including Parkinson's disease (PD), Alzheimer's disease, depression, and cognitive impairments. However, the precise molecular mechanisms remain poorly understood. To investigate the neuroprotective effects of Bacopaside I (BS-I), a major triterpenoid saponin from *Bacopa monnieri*, in a rotenone-induced rat model of PD, and to elucidate the underlying molecular mechanisms.

Methods:

Male rats were administered rotenone (2 mg/kg body weight, subcutaneously) daily for four consecutive weeks to induce PD-like pathology. BS-I was administered orally at doses of 5, 15, and 45 mg/kg during the same period. Behavioral tests including the rotarod, footprint, and grip strength assessments were conducted. Dopamine levels, oxidative stress markers, and gene/protein expression of DAT, VMAT2, and Tyrosine Hydroxylase (TH) were analyzed using standard biochemical and immunohistochemical techniques.

Results:

BS-I significantly improved motor performance across behavioral tests compared to the rotenone-only group. It attenuated rotenone-induced dopaminergic depletion and oxidative stress. Moreover, BS-I upregulated the expression of DAT and VMAT2 genes and increased the number of TH-positive neurons in the substantia nigra, indicating dopaminergic neuron preservation.

Conclusions:

Bacopaside I exerts neuroprotective effects in a rotenone-induced rat model of PD by enhancing dopaminergic signaling and mitigating oxidative stress. These findings suggest that natural saponins like BS-I could serve as promising therapeutic agents targeting molecular pathways involved in PD pathogenesis.

Keywords

Bacopaside I,, rotenone model, neuroprotection, Tyrosine Hydroxylase, dopamine

P-110

Impact of Amyloid Copathology on Effects of Regular Physical Activity in Parkinson's Disease

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Background & Objective:

Amyloid copathology contributes to clinical heterogeneity in Parkinson's disease (PD). We aimed to examine whether regular physical activity influences disease progression in the presence of amyloid copathology.

Methods:

We included participants with PD from the Parkinson's Progression Markers Initiative (PPMI) who completed the Physical Activity Scale for the Elderly (PASE) at least three times over more than four years. Participants were categorized as amyloid-negative or -positive based on cerebrospinal fluid A β_{1-42} levels. Regular physical activity was quantified using both average PASE scores and adjusted PASE scores, with the adjusted scores accounting for age at onset and baseline motor severity. Associations between physical activity and longitudinal changes in motor, cognitive, and biofluid markers were assessed using linear mixed-effects models.

Results:

In the amyloid-negative group (n = 134), higher average and adjusted PASE scores were associated with slower progression of square-root transformed MDS-UPDRS part III scores (interaction with disease duration; average scores, estimate = -5.280×10^{-5} , $p < 0.001$; adjusted scores, estimate = -0.455×10^{-2} , $p < 0.001$), and serum neurofilament light chain levels (average scores, estimate = -2.350×10^{-5} , $p = 0.015$; adjusted scores, estimate = -1.537×10^{-3} , $p = 0.049$), as well as a lower risk of conversion to Hoehn and Yahr stage 3 (average scores, hazard ratio [HR] 0.991, 95% CI 0.984~0.997, $p = 0.006$; adjusted scores, HR 0.530, 95% CI 0.336 ~ 0.837, $p = 0.006$). In contrast, among amyloid-positive patients (n = 54), physical activity was associated only with reduced progression of axial motor deficits, while other outcomes showed no significant associations.

Conclusions:

Our findings indicate that amyloid copathology may attenuate the protective effects of regular physical activity on motor, cognitive, and biomarker progression in PD, highlighting the importance of personalized therapeutic strategies based on underlying pathology.

P-111

Sensor-Based Assessment of Asymmetry in Upper Limb Bradykinesia Among Patients With Parkinson's Disease and Scans Without Evidence of Dopaminergic Deficits (SWEDDs)

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Background & Objective:

Asymmetry of motor manifestation is a hallmark feature of Parkinson's disease(PD). However, few studies have quantitatively examined this feature in patients with scans without evidence of dopaminergic deficits (SWEDDs). This study aimed to investigate the difference of upper limb asymmetry of bradykinesia between PD and SWEDDs using gyro sensors, mainly focusing on finger tapping and forearm rotation task.

Methods:

We recruited 23 early-stage, drug-naïve PD patients and 23 SWEDDs patients. We recorded gyro sensor signals during 15-second finger tapping and forearm rotation tasks. Bilateral asymmetry indices were calculated and compared between groups. Additionally, repeated measure analysis of variance (ANOVA) was used to examine interaction of task and group, and Spearman correlation analysis was conducted with clinical motor scores.

Results:

No significant group differences were found during finger tapping. However, during forearm rotation, PD patients exhibited significantly greater asymmetry than SWEDDs patients in several indices of quantitative analysis, including RMS angular velocity, peak angular displacement, peak power and total power. Task × group interaction effects were significant only in forearm

rotation, and asymmetry indices from forearm rotation showed significant correlations with clinical motor scores.

Conclusions:

Quantitative measurements of forearm rotation could be sensitive task for detecting motor asymmetry in PD and for differentiating PD from SWEDDs. These results suggest that quantitative motor asymmetry indices using wearable sensors could aid clinicians in the identification of potential SWEDD.

P-112

Relationship Between Clinical Symptoms and Ageing, Parkinson Disease

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Background & Objective:

To estimate the relationship between age groups and disease progression in Parkinson's disease (PD).

Methods:

Data were obtained from the Parkinson's disease database of neurology clinics at tertiary care hospitals between January 1, 2019, and March 31, 2022. A total of 72 patients aged between 51 and 81 years were included in this retrospective study. We analyzed the time taken to progress through various Hoehn and Yahr (H&Y) stages using SPSS version 23.

Results:

Of the 72 patients (84.7 % female), the mean age was 64.3 ± 8.9 years. Among them, 19.2 % were in the middle-aged group and 80.8 % were in the older-aged group; there were no young-onset cases in this sample. The mean disease durations by H&Y stage were as follows: Stage II – 1.6 ± 0.8 years; Stage III – 5.0 ± 2.2 years; Stage IV – 8.8 ± 1.6 years; Stage V – 10.8 ± 3.6 years ($p < 0.01$). Young-onset patients (reported in previous studies) showed significantly longer time to reach Stages IV and V but shorter time to develop wearing-off and dyskinesia. In our study, the mean age at diagnosis was 60.6 ± 8.3 years, which is consistent with previous findings that the prodromal stage of PD typically occurs between the ages of 40 and 60. Conversely, younger age at diagnosis was significantly associated with higher H&Y stage ($p = 0.01$) (see Table 1). Gender and ethnicity were not associated with H&Y stage.

Conclusions:

The Hoehn and Yahr transition time is a valuable indicator of disease progression in Parkinson's disease and may serve as a useful outcome measure in clinical studies evaluating therapeutic interventions and prognostic factors.

Table

Firstly diagnosed age	Hoehn and Yahr stage				Total
	II stage	III stage	IV stage	V stage	
Middle age between 40-59	16.7%	50.0%	40.0%	100.0%	42.3%
Old age more than 60	83.3%	50.0%	60.0%	0.0%	57.7%
Total	100.0%	100.0%	100.0%	100.0%	100.0%

Keywords

Parkinson disease, Hoehn and Yahr stage

P-114

Lifestyle Determinants and Tremor as Predictors of Parkinson's Disease: Evidence from Machine Learning Models

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Background & Objective:

Parkinson's disease is a progressive neurodegenerative disorder characterized by motor symptoms, particularly tremors. In addition to genetic factors, scientific evidence suggests that lifestyle factors such as smoking, alcohol consumption, physical activity, sleep patterns, and diet can influence the risk of Parkinson's. Early detection is crucial for patients to receive optimal treatment. With the support of machine learning, lifestyle data and clinical symptoms such as tremors can be comprehensively analyzed to predict disease risk. This approach offers potential as a non-invasive method to aid in the early diagnosis of Parkinson's.

Methods:

This study used Logistic Regression and Random Forest algorithms to predict Parkinson's risk based on lifestyle factors (smoking, alcohol consumption, physical activity, sleep quality, dietary patterns) and tremor symptoms. The data were pro-

cessed as input features, and model performance was evaluated using AUC. This machine learning approach is a non-invasive method to support early Parkinson's detection.

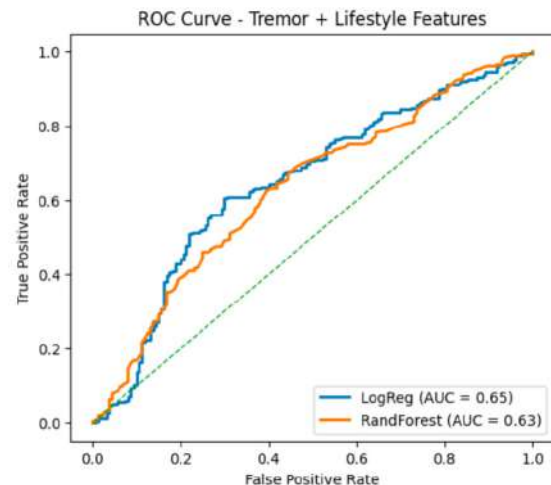
Results:

Analysis using two machine learning models, Logistic Regression and Random Forest, showed that the combination of tremor and lifestyle factors contributed to Parkinson's disease prediction, although their performance was moderate. Logistic Regression produced an AUC of 0.65, while Random Forest achieved 0.63. Feature importance analysis in Random Forest indicated that lifestyle factors such as diet quality, sleep quality, physical activity, and alcohol consumption played a more dominant role than tremor. Meanwhile, smoking showed the lowest contribution to the prediction.

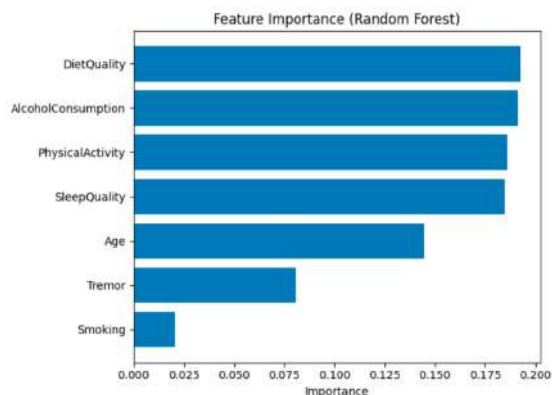
Conclusions:

This study shows that tremor and lifestyle factors predict Parkinson's disease, although model accuracy is still limited. Feature importance analysis indicated that diet quality, alcohol consumption, sleep quality, and physical activity had a more dominant contribution than tremor or smoking habits.

Figure



Figure



P-115

Dysphonia in Parkinson's Disease: Prevalence and Its Impact on Quality of Life

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Background & Objective:

Voice disorders affecting up to 90% Parkinson's disease (PD) patients, and often appear early. As the disease progresses, many develop hypokinetic dysarthria, impairing speech intelligibility and communication. Recognizing these changes is important for management and improve quality of life. This study aimed to describe voice disorders in PD patients and their relationship to the quality of life.

Methods:

This cross-sectional observational study was conducted at the Neurology and Otolaryngology clinics of Indonesia's national referral hospital, Cipto Mangunkusumo Hospital. Patients with confirmed PD were enrolled. Demographic data, disease severity (Unified Parkinson's Disease Rating Score/UPDRS), and quality of life (Parkinson's Disease Questionnaire, PDQ-39) were recorded. All subjects underwent voice assessment using the GRBAS-I

scale. Correlations between dysphonia severity and clinical parameters were analysed.

Results:

A total of 25 PD patients were included. The mean age was 55.1 ± 11.8 years, with 68% male. The mean disease duration was 5.32 ± 3.65 years, mean total UPDRS score of 48.84 ± 20.89 and a mean PDQ-39 score was 46.16 ± 22.7 . All subjects exhibited dysphonia, with a median total GRBAS-I score of 4 (range 2–15). A correlation was observed between the UPDRS motor domain and the GRBAS-I scale, showing a trend toward a moderate positive relationship ($\rho = 0.389$; $p = 0.055$). Both speech ability ($\rho = 0.720$; $p < 0.01$) and facial expression ($\rho = 0.662$; $p < 0.01$) showed strong, statistically significant positive correlation with GRBAS-I. Regarding quality of life, the strongest correlation with voice impairment was found in the PDQ-39 communication domain ($\rho = 0.228$).

Conclusions:

Dysphonia is highly prevalent among PD patients. Voice impairment showed a strong association with speech ability and facial expression. The observed trends suggest that greater dysphonia severity is linked to poorer communication-related quality of life.

Table

Age (years)		55.1 ± 11.8
Gender	F	8 (32%)
	M	17 (68%)
Duration of illness (years)		5.32 ± 3.65
UPDRS		
Total		48.84 ± 20.89
Part	I	6.16 ± 3.36
	II	12.32 ± 8.16
	III	29.12 ± 17.68
	IV	2.64 ± 2.69
PDQ 39		
Total		46.16 ± 22.7
Domain	I	16.52 ± 9.3
	II	8.32 ± 6.58
	III	7.4 ± 6.4
	IV	2.7 ± 3.2
	V	1.1 ± 1.4
	VI	4.4 ± 3.2
	VII	2.32 ± 2.2
	VIII	3.3 ± 2.9

Figure 1. Data distribution

Keywords

Parkinson Disease, Dysphonia, Motor Features, Quality of Life, Cross-Sectional Studies

P-116

A Case Report: Diphasic Dyskinesia of Lower Limbs and Hemitremors Associated With Levodopa Fluctuations in Parkinson's Disease Post Magnetic Resonance-Guided Focused Ultrasound (MRgFUS) Thalamotomy

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Introduction:

Dyskinesias are common complications of long-term levodopa therapy in patients with Parkinson's Disease (PD). While most cases documented are peak-dose in nature, diphasic dyskinesia occurs less frequently and is often mistaken as wearing off. They emerge during the rising and falling phases of plasma levodopa levels and may complicate the management. The use of MR-guided focused ultrasound (MRgFUS) thalamotomy for tremor control on levodopa-induced motor complications remains undiscovered to this day.

Case:

We present a 54-year-old Filipino female with a 9-year history of tremor-predominant PD. The patient was managed with multiple therapeutic regimen including levodopa-carbidopa, rasagiline, levodopa-carbidopa-entacapone, amantadine, entacapone, and clonazepam with variable control of tremor, dystonia, and dyskinesia. She underwent left-sided MRgFUS thalamotomy in October 2023 for disabling right hemibody tremor, achieving sustained tremor reduction. One year post-operatively, she developed new involuntary movements characterized as rhythmic flexion extension of the left lower limb with ipsilateral hemitremor. These appeared predictably around 15 to 20 minutes after levodopa intake ("on" onset) and approximately 30 to 40 minutes before the next dose ("off" onset), consistent with diphasic dyskinesia. Adjustment of levodopa-carbidopa treatment provided symptomatic relief.

Discussion:

This case illustrates a rare manifestation of lower limb dyskinesia with ipsilateral hemitremor associated with levodopa fluctuations post-MRgFUS thalamotomy. The patient's long complex therapeutic history emphasizes the need to recognize diphasic dyskinesia as a distinct and different entity from peak-dose dyskinesia or wearing-off phenomenon, particularly in the post-ablative setting, to guide rational adjustment of dopaminergic

therapy. Further studies are warranted to shed light on the relationship between MRgFUS thalamotomy and levodopa-induced motor fluctuations.

Keywords

Parkinson's disease, diphasic dyskinesia, levodopa, MRgFUS, thalamotomy

P-117

Associations Between Gait Dual-Task Cost and Cognitive Function in Parkinson's Disease and Healthy Older Adults

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Background & Objective:

Gait-cognition relationships in Parkinson's disease (PD) are well documented. However, evidence linking cognition to dual-task cost (DTC), an index of gait decline under cognitive or motor load, is limited. This study compared basic gait parameters and DTC between PD patients and healthy older adults (HO) and examined their associations with cognition.

Methods:

Seventy-one PD patients and 59 HOs underwent gait assessment with the GAITRite system under four conditions: preferred speed, cognitive dual task (serial 7 subtraction), cognitive-motor dual task (cellphone use), and challenging gait condition (backward walking). Gait parameters were grouped into five domains: pace, variability, rhythm, asymmetry, and postural control. Cognition was assessed with the K-MMSE and K-MoCA. Group differences were tested for basic parameters and DTC. Linear regression, adjusted for age, sex, education, and leg length, identified gait predictors of cognitive performance.

Results:

Compared with HOs, the PD group was younger but had fewer years of education and lower K-MMSE and K-MoCA scores. Group differences in gait were greater under dual-task conditions, most prominently during the cellphone dual task. Across domains, PD patients showed poorer performance in pace, rhythm, and asymmetry. For DTC, significant group differences were observed only in swing time asymmetry during the cellphone dual task, with higher costs in PD. In predicting K-MoCA, step time DTC during the serial subtraction task was significant

in HOs, whereas stance time asymmetry DTC during backward walking was significant in PD.

Conclusions:

Compared with healthy older adults, PD patients exhibited deterioration in most gait parameters under dual-task conditions, with the most pronounced differences during the cellphone dual task and backward walking. For cognition, associations were stronger with K-MoCA than K-MMSE, involving rhythm-related parameters in healthy older adults and asymmetry-related parameters in PD.

P-118

Exploring Motor Trajectories of Parkinson's Disease Patients Receiving Deep Brain Stimulation Versus Levodopa

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Background & Objective:

Parkinson's disease (PD) is a progressive neurodegenerative disorder with motor and non-motor symptoms imposing a heavy burden on patients. Levodopa has been the gold-standard treatment due to its rapid motor improvement and quality-of-life benefits. However, long-term use leads to motor fluctuations and reduced efficacy after 5–10 years, limiting functional independence. Deep brain stimulation (DBS) has emerged as an effective surgical option for PD, targeting the subthalamic nucleus or globus pallidus internus. By modulating pathological basal ganglia oscillations, it offers durable motor improvements, reduces medication needs, and enhances quality of life. Still, DBS carries surgical risks and questions about optimal timing, creating a clinical dilemma between pharmacological treatment and surgery. Recent studies provide new insights, compares DBS and levodopa regarding motor progression and functional outcomes by mapping scientific literature to clarify their roles in PD management.

Methods:

A bibliometric analysis of 306 articles (2020–2025) used the query Parkinson; deep brain stimulation; levodopa; motor progression. Analyses examined publication trends, citations, collaborations, and thematic mapping with VOSviewer.

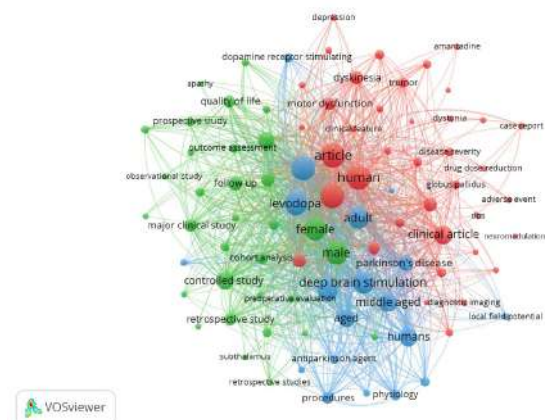
Results:

Keyword co-occurrence identified three clusters: clinical features and complications linked to levodopa; outcome and methodology terms; surgical and physiological concepts emphasizing neuro-modulation. Temporal visualization showed a shift from levodopa-focused studies (2020–2021) to DBS procedural innovations and long-term outcomes (2022–2025). The US led publications (n=90) and citations (n=1331), while Germany had the strongest collaborations. Emerging research was noted in other countries, reflecting global expansion.

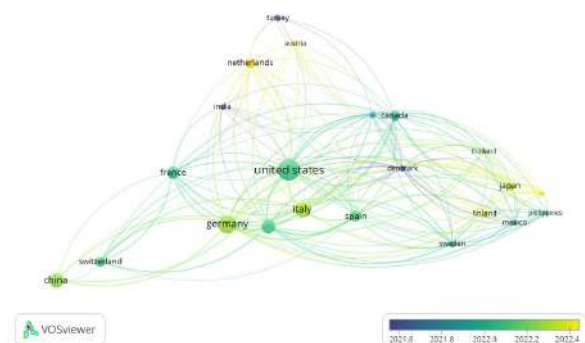
Conclusions:

This review highlights a growing interdisciplinary and focus on optimizing DBS and levodopa in PD motor management, supporting complementary use and the need for further comparative studies to guide treatment timing and integration.

Figure



Figure



Table

No	Country	Documents	Citations	Total link strength
1	Germany	67	1348	118
2	United Kingdom	37	854	113
3	Italy	42	824	94
4	United States	90	1331	87
5	Spain	25	648	74
6	Canada	20	305	60
7	Denmark	8	244	47
8	Australia	9	219	46
9	Sweden	9	160	45
10	France	30	547	44

Keywords

Deep brain stimulation, Levodopa therapy, Motor symptoms, Parkinson's disease

Parkinson's Disease: Non-medical, Surgical Treatment

P-119

Investigating the Role of Yoga practice on Stress Biomarker and Autonomic Dysfunction in Parkinson Disorder

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Background & Objective:

The present study's aim to explore the role of yoga practices on serum cortisol and heart rate variability (HRV) among patients with symptom of Parkinson disorder.

Methods:

Total 80 newly diagnosed patients from 18-40 years both male and female genders were included and diagnosed by previous record and clinical examinations. Patients were randomly assigned to one of two groups: conventional treatment with regular yogic practices (group A) and conventional treatment without yoga (group B). Yoga sessions were conducted for the group A, for three months and group B continued to take their prescribed medication only. HRV test was done to assess the severity of mental disorder also we tested serum cortisol level in fasting state of patients; suggest deviation of thoughts. Social, occupational functioning assessment scale (SOFAS) was used to evaluate overall level of performance.

Results:

The patients were 88% male and 12% female. Heart rate variability showed a significant improvement in yoga intervention group ($p=0.034$) after three month follow up. There was a reduction in serum cortisol level in yoga intervention group (9.81 ± 3.75 to 8.70 ± 3.41) not up to the significant level, but in the control group, there was slight increase (8.42 ± 3.91 to 9.0 ± 4.43) in level of serum cortisol after 3 month follow up. Moreover, the yoga group also saw reductions in drug addiction.

Conclusions:

As per our conclusion, 3 month yoga intervention is a beneficial and non-invasive treatment for Parkinson related symptoms. Our investigation also points to the need of more yoga sessions that directly help all patients.

Keywords

Yoga Intervention, Serum Cortisol

P-120

A Novel Approach to Management of Levodopa-Induced Dyskinesia Using Deep Transcranial Magnetic Stimulation

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Introduction:

Levodopa-induced dyskinesia (LID) is one of the most disabling motor complications in patients with Parkinson's disease (PD) undergoing long-term levodopa therapy. Despite its significant impact on quality of life, treatment options are limited and often invasive or associated with side effects. Recent studies have suggested that the supplementary motor area (SMA) plays a key role in the pathophysiology of LID, with hyperactivity in this region contributing to dyskinetic symptoms. Low-frequency (1 Hz) repetitive transcranial magnetic stimulation (rTMS) has been shown to suppress cortical excitability and may help rebalance dysfunctional motor circuits. However, the potential of deep rTMS, which penetrates broader and deeper cortical areas, has not been fully explored in this context.

Case:

we present two patients with advanced PD and severe LID who underwent low-frequency (1 Hz) deep repetitive transcranial magnetic stimulation (rTMS) targeting the supplementary motor area (SMA) using an H-coil. Both patients showed clinically meaningful improvements in dyskinesia severity, with reductions in Unified Dyskinesia Rating Scale (UdysRS) and Unified Parkinson's Disease Rating Scale (UPDRS) scores, without any adverse effects.

Discussion:

These findings suggest that low-frequency deep rTMS targeting the SMA could represent a promising therapeutic alternative for PD patients with LID, especially in cases where medication adjustments are ineffective or not well tolerated, and where invasive procedures like deep brain stimulation are not preferred. However, given the small sample size, further randomized controlled studies are needed to validate these findings, determine optimal stimulation parameters, and clarify the underlying neurophysiological mechanisms.

Keywords

Levodopa-induced dyskinesia, repetitive transcranial magnetic stimulation, supplementary motor area

P-121

Cycling Exercise for Autonomic Symptoms in Early Parkinson's Disease

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Background & Objective:

The effects of exercise on autonomic symptoms in Parkinson's disease (PD) remain unclear. This study investigated domain-specific effects of cycling exercise in early PD.

Methods:

This exploratory analysis of a randomized controlled trial included 30 patients with early PD, assigned to a cycling exercise group ($n=19$) and a control group ($n=11$). The intervention consisted of supervised cycling sessions (40–60 minutes, three times per week) for 24 weeks. Autonomic symptoms were assessed using the Scale for Outcomes in Parkinson's Disease–Autonomic (SCOPA-AUT), which includes gastrointestinal, urinary, cardiovascular, thermoregulatory, pupillomotor, and sexual domains.

Results:

After 24 weeks, there was a trend toward a smaller increase in the SCOPA-AUT total score in the cycling exercise group compared to the control group, although the difference was not statistically significant ($p=0.082$). Among the SCOPA-AUT domains, the urinary subscore increased significantly less in the cycling exercise group (median difference, -2.0 points; 95% confidence interval, -4.0 to -1.0 ; $p=0.017$), whereas no significant differences were observed in the other autonomic domains. Of the urinary subitems, only urinary frequency improved significantly in the cycling exercise group compared to the control group ($p=0.032$).

Conclusions:

Cycling exercise may attenuate urinary symptoms, particularly urinary frequency, in patients with early PD. These findings warrant further evaluation in large-scale trials.

Keywords

Parkinson's disease, cycling, exercise, autonomic, urinary

P-122

Targeted Stereotactic Lesioning in Parkinson's Disease: Case Series of VIM Thalamotomy and Pallidothalamic Tractotomy

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Introduction:

In Parkinson's disease (PD), medications are effective in the early stages, but with disease progression and escalating doses, motor complications and refractory symptoms may develop. In such cases, surgical options may be considered. We report a case series of PD patients treated with stereotactic lesioning.

Case:

CASE 1

A 75-year-old male with a 2-year history of Parkinson's disease (Hoehn & Yahr stage 2) presented with left-predominant resting tremor and rigidity. Despite treated with levodopa and carbidopa 100/25 mg every 8 hours, pramipexol 0.375 mg every 24 hours, and trihexyphenidyl 2 mg every 12 hours for two years, symptoms persisted and tremor became increasingly disabling. He underwent ventral intermediate thalamus (VIM) lesioning which resulted in complete tremor resolution, allowing discontinuation of pramipexole and trihexyphenidyl. His UPDRS III score improved to 15, representing a 53.1% reduction from baseline.

CASE 2

A 57-year-old male with a 6-year history of Parkinson's disease (Hoehn and Yahr stage 4) presented with freezing of gait during gait initiation and turning. He had been on levodopa/carbidopa/entacapone six times daily and pramipexole 0.375 mg once daily for 3 years and the effect of levodopa had shortened to about 2 hours. The preoperative UPDRS III score was 53. He underwent stereotactic pallidothalamic tractotomy, resulting in marked improvement with levodopa reduced by 200 mg/day. However, intermittent freezing of gait recurred after two months.

Discussion:

Target selection of stereotactic lesioning in PD depends on the predominant symptom. VIM thalamotomy is effective for tremor, consistent with its role as a relay of the cerebello-thalamic-cortical pathway but offers limited benefit for rigidity and bradykinesia. While pallidothalamic tractotomy interrupts the GPi-thalamus pathway, thus offers broader control of tremor, rigidity, and dyskinesia

Figure



Figure



Keywords

Stereotactic Lesioning, Thalamotomy, Pallidothalamic Tractotomy, Parkinson Disease

P-124

Transforming Elderly Parkinson's Disease Care in Indonesia: Interactions of Digital Aging, Depression, and Quality of Life

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Background & Objective:

Digital health technologies offer transformative potential for managing chronic conditions, but "digital aging"—disparities in technology access and use among the elderly—may exacerbate existing inequalities. The interplay between digital access, the high prevalence of depression, and quality of life (QoL) in Indonesian PD patients remains unquantified, representing a significant knowledge gap in resource-limited settings.

Methods:

Using Indonesia Family Life Survey (IFLS), the sample included 387 adults aged ≥ 60 with a self-reported PD diagnosis. The primary outcome was QoL (EQ-5D Visual Analogue Scale (EQ-VAS)) with the exposure was a Digital Access Index (DAI; range 0-3) derived from mobile phone ownership, internet use, and computer use. Depression was measured by the CES-D-10 score. We used multivariable linear regression adjusted for age, sex, residence, wealth, education, and comorbidities to assess associations. Structural equation modeling with bootstrapping (5,000 reps) quantified depression as a mediator.

Results:

Each unit increase in the DAI was associated with a 3.15-point higher EQ-VAS score (95% CI: 1.02 to 5.28, $p < 0.05$) after full adjustment. Depression was a strong, independent predictor ($\beta = -2.05$ per CES-D point, $p < 0.01$). Mediation analysis confirmed a significant indirect effect: 43% of DAI's total effect on QoL was mediated through lower depression scores ($\beta = 0.134$, 95% Bootstrapped CI: 0.082 to 0.196). Significant disparities in digital access were observed, correlating with older age, female gender, rural residence, and lower socioeconomic status.

Conclusions:

The study highlights digital inclusion as a potent, underutilized lever for improving neurological care. However, existing socio-demographic disparities in digital access risk widening health inequities. National PD strategies must integrate digital health tools while implementing targeted policies to overcome the digital divide for the most vulnerable elderly populations.

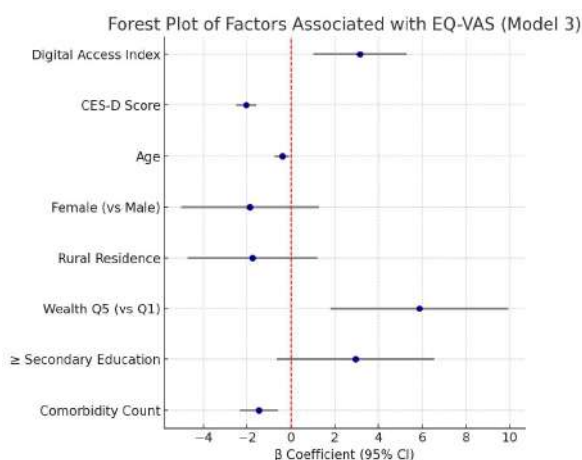
Table

Characteristic	Overall	Low DAI (0-1)	High DAI (2-3)	p-value
Age, years (mean, SD)	68.7 (6.9)	71.2 (7.1)	65.1 (5.2)	<0.001
Female, n (%)	212 (54.8%)	148 (62.7%)	64 (42.4%)	<0.001
Rural Residence, n (%)	183 (45.8%)	132 (55.9%)	51 (32.1%)	<0.001
Wealth Quintile 4 or 5, n (%)	121 (31.3%)	45 (18.1%)	76 (48.3%)	<0.001
≥ Secondary Education, n (%)	87 (22.5%)	25 (10.6%)	62 (41.1%)	<0.001
Comorbidity Count (mean, IQR)	2 (1-3)	2 (1-3)	2 (1-3)	0.45
CES-D Score (mean, SD)	14.2 (5.1)	15.8 (4.9)	11.9 (4.6)	<0.001
EQ-VAS Score (mean, SD)	62.5 (18.3)	57.1 (17.5)	70.8 (16.1)	<0.001
Digital Access Index (DAI), n (%)				
-0 (Lowest)	118 (30.5%)	—	—	—
-1	118 (30.5%)	—	—	—
-2	97 (25.1%)	—	—	—
-3 (Highest)	54 (14.0%)	—	—	—

Table

Variable	Model 1: β (95% CI)	Model 2: β (95% CI)	Model 3: β (95% CI)
Digital Access Index (per 1-point increase)	6.82 (4.11, 9.53)**	5.01 (2.65, 7.37)**	3.15 (1.02, 5.28)*
CES-D Score (per 1-point increase)	—	-2.18 (-2.65, -1.71)**	-2.05 (-2.51, -1.59)**
Age (per 1-year increase)	-0.55 (-0.95, -0.15)*	-0.48 (-0.84, -0.12)*	-0.41 (-0.76, -0.06)*
Female (Ref: Male)	-3.10 (-6.88, 0.68)	-2.15 (-5.45, 1.15)	-1.88 (-5.05, 1.29)
Rural Residence (Ref: Urban)	-2.95 (-6.45, 0.55)	-2.11 (-5.21, 0.99)	-1.75 (-4.72, 1.22)
Wealth Q5 (Ref: Q1)	8.11 (3.22, 13.00)**	6.55 (2.25, 10.85)**	5.88 (1.82, 9.94)**
≥ Secondary Education	5.88 (1.05, 10.71)*	3.99 (0.15, 7.83)*	2.95 (-0.65, 6.55)
Comorbidity Count	-2.05 (-3.11, -0.99)**	-1.55 (-2.45, -0.65)**	-1.48 (-2.36, -0.60)**
R ² / Adjusted R ²	0.28 / 0.26	0.47 / 0.45	0.49 / 0.47

Figure



Keywords

Parkinson's Disease, Digital Aging, Depression, Health Disparities

Parkinson's Disease: Nonmotor Features

P-125

Triple Modulation of GSK-3 β , NLRP3, and STING by Ternary Thiazole–Coumarin–Azomethine Derivatives: A Pharmacoinformatic Strategy Against Neuroinflammation and Tau Pathology in Parkinson's Disease

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Background & Objective:

Parkinson's disease (PD) encompasses both motor impairments and nonmotor symptoms, including depression and cognitive decline, which are increasingly attributed to tau hyperphosphorylation and persistent neuroinflammation. Glycogen synthase kinase-3 β (GSK-3 β) drives pathological tau aggregation, while the stimulator of interferon genes (STING) and NLRP3 inflammasome pathways mediate chronic microglial activation and pro-inflammatory signaling. Given the interconnected roles of these targets in PD pathogenesis, this study explores ternary thiazole–coumarin–azomethine derivatives as multitarget ligands designed to inhibit GSK-3 β , modulate STING, and suppress NLRP3, aiming to alleviate neuroinflammation and tauopathy in Parkinson's disease.

Methods:

Five ternary thiazole–coumarin–azomethine derivatives were docked against GSK-3 β (3GB2), STING (6DXL), and NLRP3 (7ALV) using AutoDock Vina, with Tideglusib, amidebenzimidazole, and RM5 as references. ADMET properties were evaluated via pkCSM.

Results:

Derivatives 6i, 6j, and 6k exhibited strong multitarget binding to GSK-3 β (–10.0 kcal/mol), STING (–9.4 to –9.8 kcal/mol), and NLRP3 (–10.7 for 6i and –11.1 for 6k), suggesting potent inflammasome inhibition. Pharmacokinetic predictions revealed 100% human intestinal absorption for all three, with non-inhibition of CYP1A2, and modest BBB permeability (log BB values from –1.07 to –1.25). Despite high lipophilicity (log P > 6), the compounds maintained moderate solubility (log S around –3.3 to –3.5) and

acceptable total clearance rates, with 6i being the slowest cleared (0.03 log mL/min/kg). All candidates showed one Lipinski violation (due to high log P) and low predicted toxicity ($LD_{50} > 3$ mol/kg). Compound 6i–6k emerge as multitarget neuroprotective leads.

Conclusions:

Ternary thiazole–coumarin–azomethine derivatives depicted multitarget agents for Parkinson's disease. Their ability to modulate GSK-3 β , STING, and NLRP3 warrants further validation through rigorous experimental/clinical research.

Table

GENERAL PROFILES			
Molecules/Pubchem ID	6i	6j	6k
MW	521.554	551.58	600.45
Binding Affinity			
GSK-3 β (kcal/mol)	-10.0	-10.0	-10.0
NLRP3 (kcal/mol)	-10.7	-9.4	-11.1
STING (kcal/mol)	-9.4	-9.5	-9.8
LIPOPHILICITY			
Consensus Log P	6.0479	6.0565	6.8104
WATER SOLUBILITY			
Log solubility (log mol/L)	-3.336	-3.442	-3.466
PHARMACOKINETICS			
BBB Permeability (log BB)	-1.078	-1.256	-1.225
CYP1A2 inhibitor	No	No	No
Intestinal Absorption (Human) (% Absorbed)	100	100	100
Total Clearance (log ml/min/kg)	0.03	0.066	-0.122
DRUGLIKENESS			
Lipinski #violations	1	1	1
Bioavailability Score	0.55	0.55	0.55
MEDICINAL CHEMISTRY			
Leadlikeness #violations	2	2	2
Synthetic Accessibility	4.2	4.37	4.19
TOXICITY			
Oral Rat Acute Toxicity (LD_{50}) (mol/kg)	3.205	3.376	3.214

Table 1. Pharmacoinformatic Profile

Keywords

Parkinson's disease, Neuroinflammation, GSK-3 β / NLRP3 / STING, Nonmotor, Pharmacoinformatics

P-126

Sleepless Pressure: Unmasking Nocturnal Hypertension in Parkinson's Disease, An Indian tertiary-care Perspective

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Background & Objective:

PD is a progressive neurodegenerative disorder with motor and non-motor manifestations. NAH, an underrecognized cardiovascular complication linked to autonomic dysfunction, is increasingly relevant in India, where hypertension prevalence and metabolic comorbidities are high. The objective of this study is to determine prevalence and determinants of nocturnal arterial hypertension (NAH) in Parkinson's disease (PD) in India

Methods:

A 9-month cross-sectional study at a tertiary neurology center recruited PD patients meeting MDS criteria with age- and gender-matched controls (1:2). Severity was assessed by MDS-UPDRS and Hoehn & Yahr (H&Y) scales. NAH and BP profiles were measured by 24-hour ABPM. Compliance was assessed via MARS-5. Predictors were identified using logistic regression.

Results:

We enrolled 102 participants, 34 patients and 68 controls. Mean PD age was 66.2 ± 7.6 years, age at diagnosis 61.1 ± 6.8 years; 67.6% were male. Median disease duration was 8 years, 55.9% in advanced H&Y stage (median stage 3). 88.2% were on levodopa (mean dose 410.5 ± 154.3 mg); 44.1% had moderate adherence. NAH prevalence was 52.9%, and 41.1% were "non-dippers." Predictors included advanced H&Y stage (OR 3.2, $p=0.01$), MDS-UPDRS Part I ≥ 12 (OR 2.7, $p=0.03$), age at PD diagnosis > 60 (OR 3.8, $p=0.004$), and type 2 diabetes (OR 1.9, $p=0.04$).

Conclusions:

Over half of PD patients had NAH. Advanced stage, higher non-motor burden, later diagnosis, and diabetes predicted NAH. Routine ABPM screening is recommended, especially for patients >60 or with metabolic comorbidities.

Keywords

Parkinson's disease, Nocturnal Hypertension, Autonomic Dysfunction, Non Motor Symptoms

P-127

Nocturnal and Early Morning Hypoglycemia in Parkinson's Disease: An Indian Perspective

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Background & Objective:

Emerging evidence links PD with disturbances in glucose metabolism. Shared mechanisms such as mitochondrial dysfunction, oxidative stress, and neuroinflammation may predispose to insulin resistance and hypoglycemia. In India, where diabetes prevalence is high, understanding this link has direct clinical relevance for both neuroprotection and metabolic control and the objective of the study was to determine the prevalence and risk factors of hypoglycemic episodes in Indian patients with Parkinson's disease (PD) using two-week continuous glucose monitoring (CGM).

Methods:

Forty PD patients and twenty age- and gender-matched controls were recruited from a tertiary-care neurology clinic. Demographic and clinical data were collected, and autonomic symptoms were evaluated using the SCOPA-AUT scale. Participants underwent 14-day CGM using the Freestyle Libre Pro system. Hypoglycemia was defined as interstitial glucose <70 mg/dL for ≥5 minutes. Predictors were analyzed using multivariate logistic regression.

Results:

Hypoglycemic episodes were detected in 22 PD patients (55%), totaling 146 episodes with a mean duration of 11.2 ± 4.1 min. 68% of events occurred between midnight and 6:00 am. Prevalence of hypoglycemia was higher in PD than controls (55% vs 35%; p=0.12), but episode frequency was significantly greater (4.1 vs 1.6 episodes/person; p=0.0004). Independent predictors in PD included lower 2-week mean glucose (OR -0.27, 95% CI -0.49 to -0.06; p=0.014), higher glycemic variability index (OR 0.48, 95% CI 0.09 to 0.87; p=0.019), and higher SCOPA-AUT score (OR 0.25, 95% CI 0.08 to 0.42; p=0.037).

Conclusions:

Indian PD patients demonstrate frequent nocturnal and early morning hypoglycemia, particularly with greater autonomic dysfunction and glycemic variability. CGM should be considered in PD management, especially for those with diabetes or advanced non-motor symptom burden, to enable timely interventions.

Keywords

Autonomic dysfunction, Parkinson's disease, Hypoglycemia,

Continuous glucose monitoring, India

P-128

The Nature and Severity of Non-Motor Symptoms in Drug-Naïve Early Parkinson's Disease Patients: Korean Cohort Data

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Background & Objective:

NMS significantly impact the QoL of PD patients and may appear before motor symptoms. However, distinguishing them from dopaminergic treatment side effects is challenging. Drug-naïve PD patients provide a unique opportunity to study the natural presentation of NMS without pharmacological confounders. This study aimed to assess the distribution and severity of non-motor symptoms (NMS) in drug-naïve early PD patients to inform future NMS management strategies.

Methods:

We retrospectively analyzed drug-naïve PD patients initially diagnosed from January 2023 to December 2024. All patients underwent MDS-UPDRS Part I and SCOPA-AUT assessments before dopaminergic treatment initiation.

Results:

A total of 40 drug-naïve PD patients were included. Among NMS, urinary dysfunction (92.5%), gastrointestinal dysfunction (85.0%), and sleep disturbances (57.5%) were the most prevalent. Depression (47.5%) and anxiety (45.0%) were also frequent. Autonomic symptoms were common, including fatigue (52.5%) and lightheadedness on standing (40.0%). Pupillomotor dysfunction (10.0%) and hallucinations (2.5%) were rare but severe when present. Impulse control disorder-related symptoms (5.0%) and hypersalivation were observed at lower rates. The burden of NMS varied widely, with urinary and cardiovascular dysfunctions showing the highest burden, while neuropsychiatric symptoms, though less frequent, were severe in some cases.

Conclusions:

NMS were highly prevalent in drug-naïve early PD patients, with autonomic dysfunctions, particularly urinary and gastrointestinal symptoms, being the most frequently reported. Mood-related symptoms such as depression and anxiety were also common, while certain symptoms like hallucinations were rare but notably

severe. These findings highlight the need for systematic NMS evaluation and early intervention in PD management. Given the variability in symptom prevalence and severity, a personalized approach to NMS treatment is warranted.

Table

Table 1. The demographic and clinical characteristics of Drug-Naïve patients with Parkinson's disease.

	Total (n=40)
Female sex, n (%)	20 (50.0 %)
Age, y	72.15±7.75
Duration of PD, y	1.30±0.99
Education, y	10.05±6.03
MoCA-K	22.03±5.09
Smoking, n (%)	
Never	25 (62.5)
Quit	10 (25)
Currently	5 (12.5)
Alcohol, n (%)	
Never	22 (55)
Quit	10 (25)
Currently	8 (20)
BMI	23.92±3.17
Hoehn and Yahr Scale	2.00±0.44
MDS UPDRS Part 1	7.80±5.91
MDS UPDRS Part 2	7.58±5.97
MDS UPDRS Part 3	31.45±9.67
PIGD score	2.55±2.25
Tremor score	5.53±4.22
SCOPA-AUT	
K-MMSE*	26.33±3.08
K-GDS*	15.20±7.18
FAZEKAS Scale**	
0	8(22.2)
1	21(58.3)
2	6(16.7)
3	1(2.8)

*10 missing data

**4 missing data

Table

Table 2. Distribution of Non-motor Sx and Autonomic Sx of Drug Naïve PD patients

	Mean score of all 49 patients	Number of symptomatic patients, n (%)	Mean score of symptomatic patients
MDS UPDRS Part 1			
-Cognitive impairment	0.55±0.64	19 (47.5)	1.16±0.38
-Hallucinations and psychosis	0.05±0.32	1 (2.5)	2.00±0.00
-Depressed mood	0.60±0.71	19 (47.5)	1.26±0.45
-Anxious mood	0.56±0.75	18 (45.0)	1.28±0.58
-Apathy	0.40±0.59	14 (35.0)	1.14±0.36
-Feature of DDS	0.05±0.22	2 (5.0)	1.00±0.00
-Sleep problems	1.06±1.16	23 (57.5)	1.87±0.92
-Daytime sleepiness	0.70±0.91	16 (40.0)	1.75±0.45
-Pain and other sensations	0.53±0.82	15 (37.5)	1.40±0.74
-Urinary problems	1.06±1.05	25 (62.5)	1.72±0.79
-Constipation problems	0.80±0.97	22 (55.0)	1.45±0.86
-Light headedness on standing	0.66±0.97	16 (40.0)	1.69±0.78
-Fatigue	0.73±0.85	21 (52.5)	1.38±0.67
SCOPA-AUT			
-Gastrointestinal dysfunction	3.03±2.88	34 (85.0)	3.56±2.89
-Urinary dysfunction	5.60±4.21	37 (92.5)	6.05±4.04
-Cardiovascular dysfunction	1.20±1.80	21 (52.5)	2.29±1.93
-Thermoregulatory dysfunction	0.06±1.87	15 (37.5)	2.03±2.33
-Pupillomotor dysfunction	0.18±0.59	4 (10.0)	1.75±0.96

P-129

Prevalence of Muscle Mass Loss and Its Association With Orthostatic Hypotension and Related Symptoms in Parkinson's Disease

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Background & Objective:

To investigate the prevalence of muscle mass loss in Parkinson's disease (PD) patients compared with that in the general population and to explore its relationship with orthostatic hypotension (OH) and associated symptoms.

Methods:

We prospectively enrolled PD patients (Hoehn and Yahr of 3 or lower) and matched them 1:5 ratio by age, sex, and height with general population from the Korea National Health and Nutrition Examination Survey data. Appendicular muscle mass was assessed by dual-energy X-ray absorptiometry and cutoff values for muscle mass loss defined by the Asian Working Group for Sarcopenia 2019. In PD patients, blood pressure was recorded in the supine position and at 30 seconds, 1, 3, and 5 minutes after standing. OH was defined according to the international consensus, and symptoms were evaluated using items 14 to 16 of the Scales for Outcomes in Parkinson's Disease—Autonomic Dysfunction questionnaire.

Results:

We analyzed 409 PD patients (mean age, 71.1±9.5 years; 52.3% men) and 2045 matched controls. Muscle mass loss was more prevalent in the PD patients (35.0%) than controls (24.8%), especially in men and individuals aged ≥70 years. Among PD patients, prevalence of OH did not differ between the low muscle mass (LMM) and normal muscle mass (NMM) groups (46.8% vs.

38.6%). The LMM group showed significantly greater drops in systolic BP ($P=0.036$) and diastolic BP ($P=0.009$) during 5 minutes after standing. A significant difference was observed only at 30 seconds after standing in both systolic BP (-18.7 ± 1.7 mmHg vs. -13.0 ± 1.1 mmHg, $P=0.020$) and diastolic BP (-7.8 ± 1.1 mmHg vs. -4.2 ± 0.6 mmHg, $P=0.012$). No differences were observed in the frequency or severity of its symptoms.

Conclusions:

Patients with mild to moderate PD showed higher risk of muscle mass loss. It associated with transient orthostatic BP instability in the early period after standing, it was insufficient to cause OH or associated symptoms.

Keywords

Parkinson's disease, Muscle mass, Sarcopenia, Orthostatic hypotension, Autonomic dysfunction

P-130

Association Between Cognitive Adverse Effects of Anticholinergic Medication and Development of Dementia in Parkinson's Disease

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Background & Objective:

Approximately one-third of patients with Parkinson's disease develop dementia, and anticholinergic use has been associated with increased risk, particularly with prolonged exposure. This study investigated whether short-term anticholinergic use was associated with differing dementia incidence between cognitive and non-cognitive adverse effects groups.

Methods:

A retrospective chart review was conducted on 209 patients with Parkinson's disease initially prescribed anticholinergics. Patients were grouped by continued use without adverse effects or discontinuation due to cognitive or non-cognitive adverse effects. Multivariable logistic regression was used to evaluate the association between cognitive adverse effects and dementia incidence. Kaplan-Meier survival curves were used to compare

dementia incidence among groups.

Results:

Of the 155 patients analyzed, 62 continued long-term use without adverse effects, while 12 and 81 discontinued due to cognitive and non-cognitive adverse effects, respectively. Baseline characteristics and anticholinergic dosages did not significantly differ between the cognitive and non-cognitive adverse effects groups. After adjusting for age, sex, and disease duration, no significant differences in dementia incidence were observed between the cognitive and non-cognitive adverse effects groups (adjusted OR = 1.17; 95% CI: 0.13–10.22, $p = 0.890$) or between the cognitive adverse effects and long-term use groups (adjusted OR = 1.57; 95% CI: 0.23–10.89, $p = 0.646$).

Conclusions:

The association between cognitive adverse effects of anticholinergics and the development of dementia has not been established. Prospective studies that include comprehensive test of individual variability in anticholinergic sensitivity among patients with PD and its impact on cognition are needed.

Figure

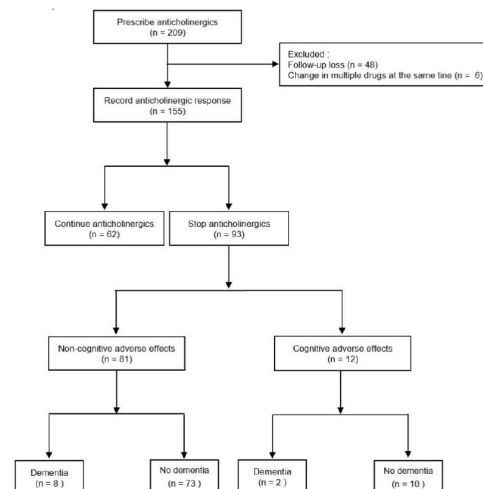


Fig. 1. Flow diagram of patient selection and classification based on anticholinergic adverse effects and dementia outcomes.

Table

Table 1. Baseline demographic and clinical characteristics of cognitive and non-cognitive adverse effects groups.

	cognitive adverse effect (n=12)	non-cognitive adverse effect (n=81)	p-value
Age, years (SD)	69.3 (6.38)	66.8 (7.43)	0.232
Female, n (%)	6 (50)	45 (56)	0.725
Duration of disease, years (SD)	1.82 (1.72)	2.12 (2.35)	0.747
Drug dose, mg/d (SD)			
Trihexyphenidyl	5.00 (1.29)	5.45 (1.18)	0.277
Benzotropine	0	5.63 (1.06)	1.000
Amantadine	166.67 (83.49)	217.50 (81.95)	0.103
LEDD at prescription, mg/d (SD)	126.26 (186.83)	193.70 (352.29)	0.903
Cognitive adverse effect			
Memory loss, n (%)	6 (50)		
Hallucination, n (%)	6 (50)		
Non-cognitive adverse effect			
Dry mouth, n (%)		20 (25)	
Voiding difficulty, n (%)		1 (1)	
Dizziness, n (%)		7 (9)	
Other symptoms, n (%) *		53 (65)	
Incidence of dementia, n (%)	2 (17)	8 (10)	
LEDD at diagnosis of dementia, mg/d (SD)	750.00 (0.00)	627.19 (227.60)	0.171

Abbreviation: LEDD, levodopa equivalent daily dose

t-tests were used for age and LEDD at diagnosis of dementia; Mann-Whitney U tests were used for sex, duration of disease, trihexyphenidyl, amantadine, and LEDD at prescription; Chi-square test was used to analyze the incidence of dementia between the groups.

*Other symptoms included dry eye, insomnia, anxiety, nausea, vomiting, tachycardia, hypotension, leg edema, vision loss, livedo reticularis, and itching.

Figure

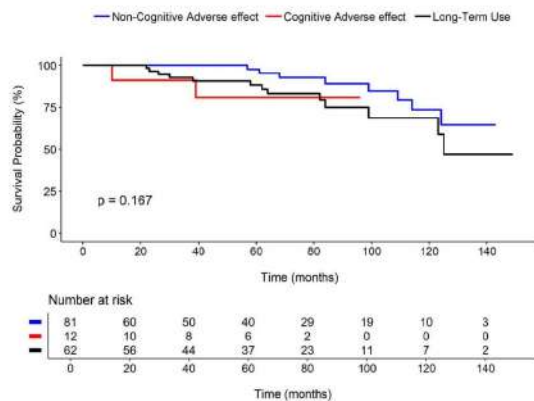


Fig 2. Kaplan-Meier survival curves comparing three groups: Non-Cognitive Adverse Effects (blue), Cognitive Adverse Effects (red), and Long-Term Use (black). No statistically significant difference was observed among the groups (log-rank test, $p = 0.171$). Censoring marks are omitted. The number at risk is shown below the x-axis.

Keywords

Parkinson's disease, Anticholinergics, Cognition, Dementia

P-131

Subjective Cognitive Complaints in Parkinson's Disease Patients With Normal Cognition Predict Development of Dementia: A 5-Year Longitudinal Observation

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Background & Objective:

This study aimed to examine whether subjective cognitive complaints (SCCs) measured by the SCCQ-PD questionnaire can predict the development of dementia in initially cognitively normal patients with Parkinson's disease (PD).

Methods:

We retrospectively analyzed 53 PD patients with normal cognition at baseline who were followed for an average of 5 years. SCCs were assessed using the SCCQ-PD questionnaire, and subjects were grouped by SCCQ-PD scores (low, intermediate, high). Cox proportional hazard models were used to identify predictors of dementia.

Results:

Baseline demographic characteristics and cognitive performance did not differ significantly among groups, while patients with high SCCQ-PD scores showed poorer instrumental activities of daily living than those with low SCCQ-PD scores. During follow-up, 32.1% of patients developed dementia. High SCCQ-PD scores were significantly associated with an increased risk of future dementia (adjusted hazard ratio = 5.37, $p = 0.018$) after controlling for age and disease duration.

Conclusions:

SCCs assessed by the SCCQ-PD questionnaire are predictive of future dementia in cognitively normal patients with PD. These findings suggest that the SCCQ-PD may serve as a valuable predictive marker of cognitive decline.

P-132

Corynoxine-Mediated Suppression of NLRP3 Inflammasome as a Neuroprotective Agent in Parkinson's Disease

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Background & Objective:

Parkinson's disease (PD) is a progressive neurodegenerative disorder characterized not only by motor dysfunction but also by a wide spectrum of non-motor symptoms, including cognitive impairment, mood disturbances, and autonomic dysfunction. Increasing evidence suggests that chronic neuroinflammation plays a key role in the pathogenesis of these non-motor features, particularly through the activation of the NLRP3 inflammasome complex. Persistent activation of NLRP3 drives the release of pro-inflammatory cytokines, exacerbating neuronal damage beyond dopaminergic systems. This study aims to explore the therapeutic potential of corynoxine as a ligand targeting NLRP3 to exert anti-inflammasome and neuroprotective effects in PD.

Methods:

AutoDock Vina was used to dock three corynoxine derivatives against NLRP3 (7ALV), using RM5701 as a reference. ADMET properties and pharmacokinetic studies were evaluated via pkCSM and SwissADME.

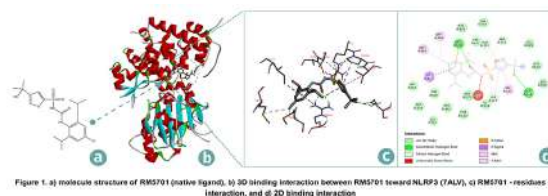
Results:

All ligand derivatives (CID-10475115, CID-10091424, and CID-21148450) demonstrated strong binding affinity to NLRP3 (−7.5 to −8.8 kcal/mol) and high intestinal absorption (>95%), indicating efficient oral uptake. Moderate BBB permeability values (log BB −0.412 to −0.578) suggested the ability to reach the central nervous system, while no CYP1A2 inhibition was observed, reducing the likelihood of metabolic interactions. All compounds complied with Lipinski's rules and exhibited acceptable predicted toxicity (LD50 ≈ 2.5 mol/kg), with one derivative showing lower lipophilicity (Log P 3.122) compared to the others.

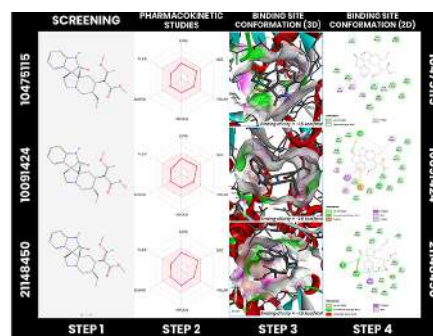
Conclusions:

Among the tested derivatives, ligand CID-10091424 exhibited the most favorable balance of properties. These features highlight its suitability for further exploration as an NLRP3-targeting agent in alleviating non-motor symptoms of Parkinson's disease. In addition, clinic trials and research are needed.

Figure



Figure



Table

Table 1. ADMET Profile of ligand derivatives

GENERAL PROFILES			
Molecules/PubChem CID	10475115	10091424	21148450
MW (g/mol)	384.476	384.476	420.937
BINDING AFFINITY			
NLRP3 (7ALV) (kcal/mol)	-7.5	-8.8	-7.6
LIPOPHILICITY			
Consensus Log P	27.002	27.002	3.122
WATER SOLUBILITY			
Log Solubility (log mol/L)	-3.996	-3.996	-4.605
PHARMACOKINETICS			
BBB Permeability (log BB)	-0.412	-0.412	-0.578
CYP1A2 inhibitor	No	No	No
Intestinal Absorption (Human) (%Absorbed)	96.234	96.234	95.196
Total Clearance (log ml/min/kg)	0.809	0.809	1.125
DRUGLIKENESS			
Lipinski (Violation)	0	0	0
Bioavailability Score	0.55	0.55	0.55
MEDICINAL CHEMISTRY			
Leadlikeness (Violation)	1	1	1
Synthetic Accessibility	4.78	4.78	4.86
TOXICITY			
Oral Rat Acute Toxicity (LD50) (mol/kg)	2.482	2.482	2.641

Keywords

Parkinson's disease, Corynoxine, NLRP3 Inflammasome, Neuro-protective, Neuroinflammation

P-133

Association Between Esophageal Motility and Gait in Early stage of Parkinson's Disease

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Background & Objective:

Parkinson's disease (PD) patients frequently develop non-motor manifestations, particularly gastrointestinal symptoms. This study aimed to investigate the association between esophageal motility and PD symptoms, focusing on autonomic dysfunction and gait.

Methods:

From September 2023 to September 2024, patients with PD underwent assessments including clinical rating scales, autonomic tests, gait analysis, and HRM. In the control group, HRM was performed between April 2021 and August 2024. Manometric parameters were compared between groups, and correlations with clinical measures were analyzed within the patient group.

Results:

Compared with controls, patients with PD demonstrated significant differences in striate esophageal contractile integral (SECI), distal contractile integral (DCI), and distal latency, but these differences did not remain significant after adjusting for age and hypertension. Within-group analysis revealed that UPDRS part III scores were negatively correlated with SECI ($\rho = -0.279$, $p = 0.022$), while lower esophageal sphincter (LES) residual pressure integrated relaxation pressure (RP IRP) was positively correlated ($\rho = 0.317$, $p = 0.009$). No associations were observed with autonomic tests. For gait parameters, upper esophageal sphincter basal pressure (UES BP) correlated positively with velocity, cadence, step length, swing phase, and single support time. In contrast, LES basal pressure and LES RP IRP were negatively correlated with %GC of swing phase and single support time.

Conclusions:

Although this study was conducted in early-stage patients and no significant differences in HRM findings were observed compared with the control group, correlation analyses within the patient demonstrated an association between esophageal motility and gait. Certain parameters (UES BP) showed positive correlations with gait improvement, yet these values are known to decrease in advanced PD. In this context, HRM may serve as a useful biomarker reflecting symptom progression in PD.

P-134

Cognitive Domain Differences and the Role of Depression and Anxiety in Subjective and Mild Cognitive Impairment in Parkinson's Disease

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Background & Objective:

Subjective cognitive decline in Parkinson's disease (PD-SCD) is regarded as a prodromal stage with elevated risk of mild cognitive impairment (PD-MCI) and dementia. Distinguishing cognitive and affective features may aid early identification. This study compared cognitive profiles of PD-SCD and PD-MCI and examined the impact of depressive, anxiety, and other behavioral symptoms.

Methods:

Fifty-two patients (PD-SCD = 21, PD-MCI = 31) completed the Seoul Neuropsychological Screening Battery-II, Mini-Mental State Examination, Montreal Cognitive Assessment, Short Geriatric Depression Scale (SGDS), and Neuropsychiatric Inventory (NPI). Scores were standardized to z-scores. Group comparisons, correlations, and logistic regression were conducted. Longitudinal follow-up was available after 2–3 years.

Results:

PD-MCI patients performed worse across domains, most prominently in memory (-1.37 z, $p < 0.001$). Within this group, higher SGDS and NPI-depression were associated with poorer executive performance (Trail Making Test) and showed trends with processing speed and visuospatial memory. Anxiety related modestly to executive and attentional tasks, while other NPI domains were negligible. These links were not evident in PD-SCD. Logistic regression identified RCFT recall, SVLT, MMSE, CWST, and COWAT as predictors (AUC = 0.88; accuracy 79%), with RCFT recall strongest. SVLT best reflected progression.

Conclusions:

PD-MCI showed broader and more severe deficits than PD-SCD, especially in memory and executive/processing speed. Depressive and anxiety symptoms were tied to executive decline. RCFT recall and SVLT were the most reliable predictors of progression. Although the modest sample size warrants caution, the results suggest that combining memory assessments with affective measures may strengthen efforts toward early detection of cognitive decline in Parkinson's disease.

Table

<Table 1>. Group comparison of cognitive domains (PD-SCD vs PD-MCI)

Domain	PD-SCD (mean z)	PD-MCI (mean z)	Diff	p-value
Memory (SVLT, RCFT recall)	+0.65	-0.72	-1.37	<0.001
Executive/Speed (TMT, CWST, DSC, COWAT)	+0.39	-0.49	-0.89	<0.001
Visuospatial (RCFT copy)	+0.28	-0.59	-0.87	0.001
Language (BNT)	+0.21	-0.53	-0.74	0.010
Global (MMSE, MoCA)	+0.42	-0.54	-0.96	<0.001

Table

<Table 2>. Correlations between affective/behavioral symptoms and cognitive performance (PD-MCI group)

Predictor	Cognitive Test	r	p-value
SGDS	TMT	-0.48	0.020
SGDS	DSC	-0.36	0.060
SGDS	RCFT recall	-0.42	0.070
NPI-Depression	TMT	-0.39	0.040
NPI-Anxiety	TMT	-0.29	0.110
NPI-Anxiety	CWST	-0.27	0.140

Table

<Table 3> Logistic regression predictors of PD-MCI and longitudinal change

Predictor	Odds Ratio	95% CI	p-value
RCFT recall	0.017	0.00-1.06	0.054
SVLT	0.21	0.04-1.21	0.084
MMSE	0.34	0.08-1.37	0.120
CWST	0.45	0.09-2.05	0.290
COWAT	0.53	0.12-2.39	0.350

Keywords

Parkinson's disease, PD-SCD(Subjective cognitive decline), PD-MCI(Mild cognitive impairment), Cognitive domains, Affective symptoms

P-135

Association of Olfactory Dysfunction With Brain Network, Atrophy, and Cognitive Functions in Parkinson's Disease

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Background & Objective:

Olfactory dysfunction is a highly prevalent and earliest non-motor symptom in patients with Parkinson's disease (PD). Olfactory dysfunction is associated spreading patterns of synucleinopathy, but its pathophysiology remains unclear. Thus, this study investigated association between olfactory functions and brain network, and atrophy pattern in PD.

Methods:

This retrospective observation study included 174 drug-naïve patients with PD who underwent Cross-Cultural Smell Identification Test (CC-SIT), neuropsychological assessments, 18F-FP-CIT PET scans and brain MRI at initial assessment. The patients were classified into three subgroups according to CC-SIT score, normosmia (8-12), hyposmia (5-8), and anosmia (0-4). Among them, 66 patients (37.93 %) additionally completed assessments brain diffusion tensor imaging. Associations between olfaction and clinical/imaging variables were assessed using correlation analysis. The severity of hyposmia was negatively correlated with network degree measures, indicating that reduced olfactory function is associated with more impaired connectivity across identified subnetworks.

Results:

A total of 174 patients of PD was included, 31 with normal olfactory function, 96 with hyposmia, and 47 with anosmia. The severity of olfactory dysfunction was associated with cognition.

Patients with anosmia showed lower scores in memory, visuospatial, and executive domains compared to those with hyposmia or normosmia ($p < 0.05$). Imaging analyses revealed that atrophy in the limbic structures were neural correlates of olfactory dysfunction. After adjusting age, sex, and education, individuals with olfactory dysfunction exhibited significantly lower subnetwork connectivity compared to those with normal olfaction (Normosmia vs Anosmia $p=0.008$; Hyposmia vs Anosmia, $p=0.009$).

Conclusions:

This indicates that reduced olfactory function is associated with more impaired connectivity across identified subnetworks and affects cognition in Parkinson's disease.

P-136

Amyloid Copathology Affects the Atrophy Rate of Hippocampal Subfields in Parkinson's Disease

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Background & Objective:

Mounting evidence suggests that coexistent Alzheimer's disease (AD) pathology may influence the clinical manifestations and prognosis of patients with Parkinson's disease (PD). While hippocampal atrophy is not an uncommon finding in PD, the underlying pathomechanism remains unclear, particularly with respect to the contribution of AD copathology. In this study, we investigated the potential association between amyloid- β (A β) burden and the rate of hippocampal subregional atrophy.

Methods:

This retrospective longitudinal study included 138 patients with PD who underwent standardized neuropsychological assessment, 18F-Florbetaben (FBB) positron emission tomography

(PET) imaging, and at least two separate 3-dimensional magnetic resonance imaging (MRI) scans. A linear mixed model was constructed to compare the rate of hippocampal subfield atrophy and longitudinal changes in neuropsychological test scores.

Results:

Forty-eight patients (34.8%) revealed positive amyloid PET scans. Compared to amyloid-negative group, amyloid-positive group showed faster rate of hippocampal atrophy ($P=0.041$) and cognitive decline in terms of global cognitive score ($P=0.004$). Among the hippocampal subfields, atrophy rate of the subiculum revealed the most prominent difference ($P=0.006$) followed by CA2/3 ($P=0.038$). The effect of amyloid burden on global cognitive score was mediated by the rate of subiculum atrophy.

Conclusions:

Amyloid copathology appears to affect the rate of hippocampal atrophy, especially in the subiculum, and cognitive decline in PD.

P-137

TRPV1 as a Potential Therapeutic Target for Pain in Parkinson's Disease

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Background & Objective:

Pain is a debilitating feature of Parkinson's disease (PD) that significantly impairs quality of life. Although Transient Receptor Potential Vanilloid 1 (TRPV1) is associated with inflammation—a known pathomechanism of PD—and pain in general, its specific role in PD-related pain remains unclear.

Methods:

We measured mRNA expression of TRP channels in Peripheral Blood Mononuclear Cells (PBMCs) from PD patients (with and without pain) and neurological controls, correlating with clinical severity. Pain scores, TRPV1 expression, and inflammatory pathway genes (hsa04750) were analyzed using the PPMI database and microarray dataset GSE8397 of postmortem PD and control brain tissue. A striatal Preformed Fibril (PFF) injection PD mouse model ($\pm$ Lipopolysaccharide (LPS)) was used for behavioral tests to evaluate analgesic effects of a TRPV1 antagonist, with TRPV1

protein measured in brain tissue.

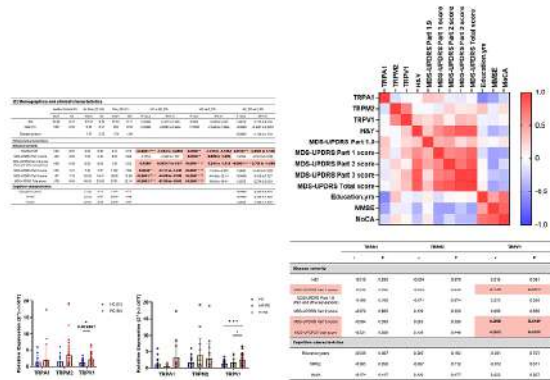
Results:

TRPV1 mRNA expression was significantly higher in PBMCs from PD patients, especially those with pain, while TRPA1 and TRPM2 showed no group differences. Higher TRPV1 levels correlated with more severe motor and non-motor symptoms. In the PPMI cohort, pain scores and TRPV1 remained persistently elevated in PD patients. Transcriptomic analysis revealed upregulation of inflammatory pathway genes—including TRPV1, NGF, PLCB4, and MAPKs—in high-pain PD. In GSE8397, TRPV1 and neuroinflammatory genes were increased in the lateral substantia nigra of PD brains, with gene set enrichment confirming activation of pain and inflammation-related pathways. In the PD mouse model, both PFF and PFF+LPS groups showed motor deficits and heightened pain sensitivity. TRPV1 antagonist treatment reduced thermal pain sensitivity, and TRPV1 protein levels at the injection site decreased post-treatment.

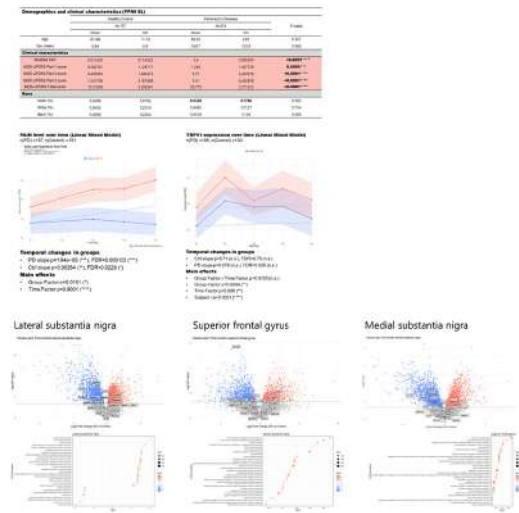
Conclusions:

TRPV1 upregulation in blood and brain is linked to increased pain and symptom severity in PD. TRPV1 antagonism selectively reduced pain in animal models, supporting its potential as a therapeutic target for PD-related pain..

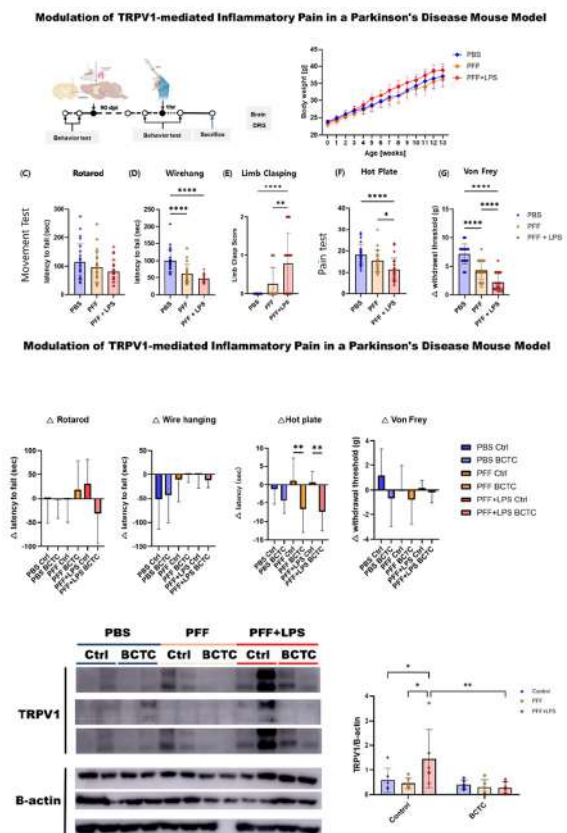
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Figure



Figure



Keywords

Parkinson's disease, inflammation, pain, TRPV1

P-138

Neuropsychiatric Manifestations in Parkinson's Disease and Their Link to Caregiver Stress

Tungalag ENKHBAT, Bolortsetseg KHATANBAATAR

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Background & Objective:

Parkinson's disease (PD) is a progressive neurodegenerative disorder presenting with both motor and non-motor symptoms. In Mongolia, limited data exist regarding the clinical profile of PD patients, particularly the burden of neuropsychiatric symptoms (NPS). Objective: To describe the frequency of motor and non-motor symptoms, with emphasis on neuropsychiatric manifestations, among Mongolian patients with PD and to assess their impact on daily functioning and caregiver burden.

Methods:

A cross-sectional observational study was conducted among 50 patients diagnosed with idiopathic PD attending a neurology clinic in Ulaanbaatar and Umnugovi province. All participants fulfilled the UK Parkinson's Disease Society Brain Bank diagnostic criteria. Motor severity was assessed using the Unified Parkinson's Disease Rating Scale (UPDRS) and Hoehn & Yahr staging. Non-motor and neuropsychiatric symptoms were evaluated with the Neuropsychiatric Inventory (NPI), and caregiver burden was measured using the Zarit Burden Interview (ZBI).

Results:

The mean age of patients was 61.2 ± 8.9 years, with a male pre-dominance (58%). Median disease duration was 4.2 years (IQR 2–6). Tremor and bradykinesia were the most common motor symptoms. At least one neuropsychiatric symptom was observed in 64% of patients, with depression (52%), anxiety (28%), apathy (22%), and irritability (20%) being the most frequent. Cognitive impairment was identified in 18% of cases. Higher NPI scores correlated significantly with functional disability and caregiver burden ($p < 0.01$). Caregivers reported mild to moderate burden in 34% of cases and severe burden in 8%.

Conclusions:

This study demonstrates that neuropsychiatric symptoms are common among Mongolian PD patients and represent a major determinant of caregiver burden. Early recognition and multidisciplinary management of non-motor symptoms, alongside motor control, are essential to improve quality of life for both patients and caregivers.

Keywords

Parkinson's disease, Mongolia, neuropsychiatric symptoms, caregiver burden, motor and non-motor symptoms

P-139

Classification of Parkinson's Disease through Acoustic Feature Analysis Using Machine Learning Algorithms in Python

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Background & Objective:

Parkinson's disease is a neurodegenerative disorder that affects motor function and voice quality. Early detection is crucial, but conventional methods are often time-consuming and expensive. Acoustic changes in a patient's voice can be a non-invasive indicator. With the support of machine learning, voice data can be analyzed to differentiate Parkinson's patients from healthy individuals. Python provides a computational library to support this process, so this research focuses on Parkinson's classification through acoustic features using Python-based machine learning.

Methods:

This study used an acoustic dataset of Parkinson's patients with an 80:20 split between training and testing data. The Random Forest Classifier algorithm was applied for classification due to its ability to handle multiple features and provide feature importance analysis. Evaluation was performed using accuracy, precision, recall, F1-score, and AUC, with visualization using a confusion matrix, ROC curve, and key feature analysis.

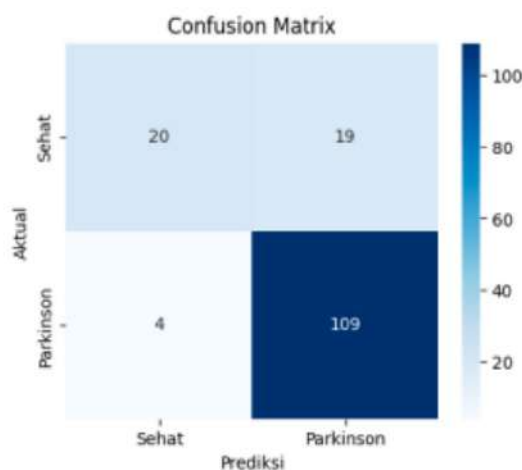
Results:

The Random Forest model could classify Parkinson's disease with high accuracy. The confusion matrix showed that 109 Parkinson's patients were correctly detected, although 19 healthy cases were misidentified. The ROC curve produced an AUC of 0.92, indicating the model's ability to distinguish between the two classes. Feature analysis showed that the logarithmic energy variables (delta and delta-delta) and the MFCC coefficient contributed the most to the prediction, consistent with medical findings that acoustic changes in voice are closely related to Parkinson's symptoms.

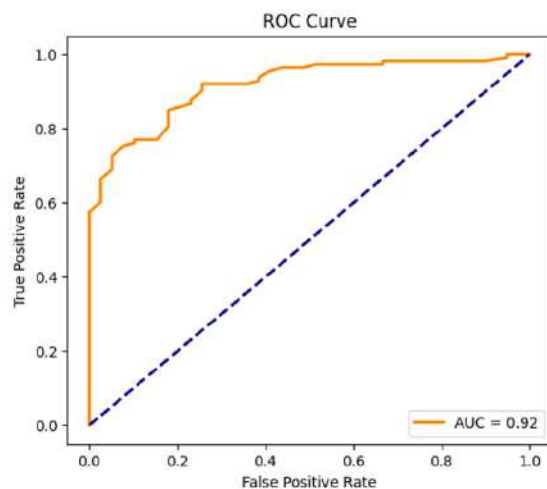
Conclusions:

This study demonstrates that the Random Forest algorithm can classify Parkinson's disease from acoustic features with high accuracy. Feature analysis confirms that logarithmic energy variables and MFCC play a significant role in prediction. These results underscore the potential of speech-based ML as a non-invasive diagnostic tool for Parkinson's disease.

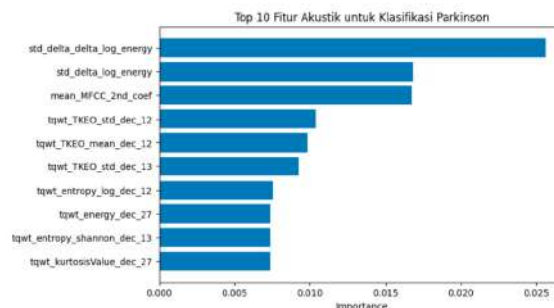
Figure



Figure



Table



Keywords

Parkinson's Disease, Parkinson's Disease: Nonmotor Features, Machine Learning, Acoustic Feature Analysis, MFCC

P-140

Obstructive Sleep Apnea in Parkinson's Disease: The Role of Positive Airway Pressure Therapy

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Background & Objective:

Parkinson's disease (PD) is a chronic neurodegenerative disorder with both motor and non-motor features. Obstructive sleep apnea (OSA) is a frequent comorbidity in PD patients. The high prevalence of both conditions contributes to a reduced quality of life and an increased risk of cognitive decline. This study aimed to examine the available literature exploring the effect of positive airway pressure therapy on improving OSA and cognition of PD patients with OSA.

Methods:

A systematic literature review was performed using several core databases, such as PubMed and Cochrane according to PRISMA statement. Parkinson, obstructive sleep apnea, and positive airway pressure were used as the search term. Original researches, specifically randomized controlled trials (RCTs) were incorporated into this study.

Results:

Three randomized controlled trials with a total of 266 participants with PD (170 participants with PD and OSA) were eligible

for this study. The results showed that PD patients with OSA exhibit greater cognitive impairment. While short-term Continuous PAP (CPAP) therapy (3-6 weeks) may not lead to significant cognitive improvements, a longer-term intervention of six months has been shown to improve Montreal Cognitive Assessment (MoCA) scores by 1.44 (95% CI [0.09, 2.79]) compared to a control group. In one study, CPAP therapy was shown to be effective in treating OSA by significantly reducing the Apnea-Hypopnea Index (AHI) and the percentage of time spent with oxygen saturation below 90%. Additionally, subjects on CPAP showed an increase in N3 sleep, indicating deeper, more restorative sleep. Three weeks of treatment also resulted in significant decreases in arousal index ($t=3.4$, $p=0.002$) and daytime sleepiness ($p=0.011$).

Conclusions:

According to the study's findings, PAP improves OSA and cognition in PD patients with OSA. However, research on the function of PAP in PD patients with OSA remains limited. More well-designed randomized controlled trials are needed.

Keywords

parkinson, obstructive sleep apnea, positive airway pressure

P-141

Gastric Emptying and Colonic Transit Time in Relation to Cardiac MIBG and RBD in Parkinson's Disease: Insights into Parasympathetic and Sympathetic α -Synuclein Propagation

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Background & Objective:

The 'body-first' and 'brain-first' hypothesis classifies Parkinson's disease (PD) based on the pattern of α -synuclein propagation. However, recent studies have raised doubts about this dichotomy, and a further subdivision of the body-first into parasympathetic- and sympathetic-subtype has been proposed. Cardiac MIBG scintigraphy and gastric dysmotility may reflect sympathetic and parasympathetic denervation, respectively. In this study, we investigated the associations among gastric emptying time (GET), colonic transit time (CTT), cardiac MIBG and REM sleep behavior disorder (RBD) in PD, and explored potential pattern of α -synuclein propagation.

Methods:

We prospectively enrolled 21 PD patients (mean age 66.9 ± 9.2) who underwent cardiac MIBG scintigraphy. GET was measured by gastric scintigraphy after solid meal (bread and fried egg). CTT was assessed using radio-opaque markers (Kolomark™) with the 4-day method. Acquired GET and CTT were compared between PD with and without RBD (PD+RBD, $n=10$; PD-RBD, $n=11$), and according to the presence of cardiac sympathetic denervation (CSD).

Results:

PR+RBD group showed significantly longer GET compared to PD-RBD ($T1/2=68.8 \pm 20.0$ vs. 51.5 ± 16.2 minutes, $p=0.041$). No significant difference was observed between PD+CSD and PD-CSD. Multiple linear regression demonstrated an independent association between RBD and GET ($\beta=0.578$, $p=0.040$), whereas not with H/M ratio ($p=0.753$). Delayed CTT was common in both PD+RBD and PD-RBD groups (77.8% vs. 80.0%). Although total CTT did not differ between groups, the number of markers retained in the right colon was significantly higher in PD+RBD.

Conclusions:

Gastric emptying time was associated with RBD but not with cardiac MIBG scintigraphy. These findings support the concept of distinct α -synuclein propagation pathways via parasympathetic (vagal) and sympathetic nerves. In CTT, only the proximal colon showed delayed transit in PD+RBD, likely reflecting the predominant vagal innervation of the proximal colon.

Keywords

Gastric emptying time, cardiac MIBG, colonic transit time, RBD, Parkinson's disease

P-142

Exploring Voluntary Saccade Hypometria in the Differentiation of MSA and PD

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Background & Objective:

Saccadic eye movements are essential for vision, and their impairment is a potential biomarker in parkinsonism. Reflexive sac-

acades are often preserved in Parkinson's disease (PD), whereas voluntary saccades are impaired. Data on multiple system atrophy (MSA) remain limited. We compared reflexive and voluntary saccades in PD, MSA, and controls.

Methods:

We studied 22 PD, 13 MSA, and 18 controls. Reflexive saccades were elicited with 15° horizontal jumps; voluntary saccades used a memory-guided paradigm. Latency, gain, peak velocity, and intersaccadic pause were measured. Group comparisons used Mann–Whitney U and Wilcoxon tests.

Results:

Reflexive gain was preserved in PD and MSA, but latency was prolonged versus controls ($p=0.005$). Voluntary saccades differed: overshoot in controls (gain 1.18), mild hypometria in PD (0.96), marked hypometria in MSA (0.85; PD vs MSA $p=0.029$). Peak velocity was highest in controls (297°/s), reduced in PD (187°/s) and MSA (206°/s) with no difference ($p=0.271$). Intersaccadic pauses were longer in PD (1415 ± 354 ms) and MSA (1530 ms) than controls (1130; $p<0.01$), but not between patients. Within groups, voluntary saccades were slower than reflexive (PD $p<0.001$; MSA $p=0.02$). PD showed a paradoxical gain increase ($p=0.001$), absent in MSA ($p=0.937$). Controls overshoot voluntary vs reflexive ($p=0.001$).

Conclusions:

PD and MSA share delayed reflexive latency and impaired voluntary speed and pauses. Yet PD shows paradoxical gain increase, while MSA shows greater hypometria without compensation. Voluntary saccadic gain—its rise in PD and marked drop in MSA—may serve as a simple biomarker to distinguish PD from MSA and support early recognition of atypical parkinsonism.

Keywords

Parkinson disease, Multiple system atrophy, Saccades, Eye Movements, Biomarkers

P-143

Social Cognition in Patients with Parkinson's Disease: a Pilot Study with Facial Affect Tasks

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Background & Objective:

Patients with Parkinson's disease (PD) often have masked faces that impair social communication. They also frequently experience emotional-processing impairments. Some research suggests that pain empathy deficits in PD. However, the preservation of social cognition in PD patients and its relationship with other cognitive domains remain unclear. We aimed to investigate these aspects using facial affect tasks of the Florida Affect Battery (FAB).

Methods:

Patients with PD who underwent the Seoul Neuropsychological Screening Battery II (SNSB-II) within the past year were enrolled. They were assessed with the Korean version of the FAB, a tool designed to evaluate the ability to recognize and interpret emotions in others, especially through nonverbal signals. The study focused on facial affect tasks, which include five subtests: facial identity discrimination, facial affect discrimination, facial affect naming, facial affect selection, and facial affect matching.

Results:

A total of sixteen patients were enrolled: cognitively preserved (N=4), with mild cognitive impairment (MCI) (N=10), and with dementia (N=2). The mean (SD) age was 69.06 (7.13) years, the disease duration was 3.79 (2.27) years, and the education period was 9.63 (5.25) years. Patients with PD generally showed impairments in facial affect tasks, with facial affect matching being the most affected. Those with PD dementia exhibited particularly severe deficits in facial affect naming, selection, and matching. Errors were more frequent in negative emotions, especially sadness (n=19) and anger (n=11), in facial affect naming. Similarly, in facial affect matching, sadness (n=24) and anger (n=20) were also more impaired.

Conclusions:

Patients with PD show impairments in facial affect recognition even in normal or MCI stages. Although this study involved a small sample, it highlights the presence of social cognition deficits in PD and underscores the need for further research in this area.

Table

	Cognitively preserved	Mild cognitive impairment	Dementia
N	4	10	2
Sex, M/F	2/2	6/4	1/1
Age (yr)	69.0±5.8	69.2±7.8	69.0±3.5
Education (yr)	8.8±3.3	10.5±5.4	7.0±7.1
PD duration	2.8±0.8	4.2±2.5	6.0±2.8
H&Y stage	2.3±0.4	2.5±0.2	2.8±0.4
Fazekas	1.3±0.5	1.1±0.7	1.5±0.7
K-MMSE	28.3±1.1	25.5±2.5	22.0±1.4
CDR	0.4±0.2	0.5	0.8±0.4
CDR-SOB	0.6±0.4	1.6±0.7	4.3±0.4
GDS	2.0	3.0	4.0

Table

Table 2. Total subtest scores in each cognitive subgroup

	Cognitively preserved	Mild cognitive impairment	Dementia
Facial identity discrimination	18.5±1.1	17.8±1.8	18.0±1.4
Facial affect discrimination	18.3±1.3	18.0±1.0	18.5±1.5
Facial affect naming	17.8±1.6	17.7±1.4	16.5±3.5
Facial affect selection	18.8±1.6	18.3±2.0	17.5±0.7
Facial affect matching	16.5±2.6	15.9±2.9	14.5±0.5

Keywords

Parkinson's disease, Social cognition

P-144

A Singing Therapy Model Based on Motor–Emotion Interconnection for Patients With Parkinson's Disease

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Background & Objective:

This study aimed to develop a singing therapy model based on the interconnection of motor and emotional functions for patients with Parkinson's disease, addressing motor impairments, voice dysfunction, and reduced emotional expression through structured music-based therapy.

Methods:

A theoretical review considered the vocal and emotional features of Parkinson's patients, principles of therapeutic singing, motor–emotional neural processes, and music-induced neuroplasticity. A systematic review of 20 international studies (1990–2020) on singing interventions for Parkinson's disease was conducted. The findings were synthesized into a framework linking musical elements—rhythm, tonality, and structural factors—with motor and emotional processes.

Results:

The model demonstrated that vocalization and singing can regulate breathing, enhance phonation, improve articulation, and promote emotional communication. Key mechanisms included stabilized breathing, improved vocal quality via rhythm and pitch, volume control across phrases, and articulation through lyric repetition. Lyrics also supported memory recall, emotional re-experience, and empathic engagement, thereby reinforcing motor–emotional interconnections.

Conclusions:

The proposed model integrates structured singing with the specific motor and emotional challenges of Parkinson's disease. It highlights the therapeutic potential of singing as a sensorimotor and affective process that fosters neuroplasticity and sustained dopaminergic stimulation. Further research should refine protocols and validate effectiveness in clinical practice.

Keywords

Parkinson's Disease, Music Therapy, Singing, Therapeutic Model

P-145

Cognitive Continuum in Parkinson's Disease: Insights from 44 Patients in a Tertiary Memory Clinic

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Background & Objective:

Background Cognitive impairment in Parkinson's disease (PD) spans from mild cognitive impairment (MCI) to dementia (PDD). Data from Southeast Asia remain limited, particularly on domain-specific profiles. This study aimed to characterize cognitive patterns across the continuum of MCI-PD and PDD in an Indonesian cohort.

Methods:

MethodsWe retrospectively reviewed 44 PD patients who underwent comprehensive neuropsychological testing between 2019–2025 in a tertiary memory clinic. Cognitive domains assessed included memory, executive function, attention, visuospatial, and language. Functional status was determined by instrumental activities of daily living (IADL). Patients were classified as MCI-PD or PDD using MDS criteria.

Results:

Results Of 44 patients, 4 met criteria for MCI-PD and 40 for PDD. MCI-PD patients (mean MMSE 27.3; MoCA 25.3) demonstrated preserved IADL and predominantly executive dysfunction with mild memory deficits. In contrast, PDD patients showed multi-domain impairment, with memory and visuospatial deficits present in over 70%, alongside near-universal executive dysfunction. IADL was impaired in nearly all PDD cases. The small proportion of MCI-PD likely reflects referral bias to a memory clinic, where patients present at more advanced stages.

Conclusions:

Conclusion Our findings reinforce the concept of a cognitive continuum in PD, beginning with executive dysfunction in MCI-PD and progressing to widespread deficits in dementia. Memory and visuospatial impairment may serve as clinical markers of transition to PDD. These results emphasize the need for earlier cognitive screening in PD and provide baseline data from an Indonesian tertiary center.

Keywords

Parkinson's disease, mild cognitive impairment, Parkinson's disease dementia, Neuropsychology test, Indonesia

P-146

Volitional Saccadic Dysfunction and Visual Hallucination in Parkinson's Disease

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Background & Objective:

Visual hallucinations are a common symptom of Parkinson's disease (PD) psychosis. The underlying mechanisms are multifactorial, involving dopaminergic and cholinergic dysfunction, abnormalities in the visual pathways, impaired attentional control, and ocular dysfunction including retinopathy. Volitional saccade dysfunction, such as saccadic hypometria, is frequently observed in PD. We hypothesized that impaired saccadic control may contribute to hallucinations.

Methods:

A total of 72 drug-naïve patients with PD were enrolled. Saccadic performance was assessed using video-nystagmography (SLVNG, SLMED, Seoul, South Korea). Horizontal and vertical saccades were measured, and mean latency, amplitude and

velocity were calculated. The Scales for Outcomes in PD-Psychiatric Complications (SCOPA-PC) questionnaire was administered, and item 1 and 2 were used to evaluate the severity of hallucinations and illusions. Partial correlation analyses were performed with age, sex and educational level as covariates.

Results:

The mean age of the patients was 65.6 ± 9.9 , and 40.3% were female. Mean horizontal saccadic latency was 0.26 ± 0.09 s, amplitude was $22.40 \pm 4.47^\circ$, and velocity was $400.28 \pm 78.61^\circ/\text{s}$. Mean vertical saccadic latency was 0.30 ± 0.11 s, amplitude was $19.94 \pm 5.08^\circ$, and velocity was $331.06 \pm 83.66^\circ/\text{s}$. Correlation analysis showed that the severity of visual hallucinations was negatively correlated with horizontal saccade amplitude ($\rho = -0.308$, $p = 0.013$). No significant associations were found hallucination severity and other saccade parameters, nor with illusions.

Conclusions:

Volitional saccadic hypometria is significantly associated with hallucinations in PD. Reduced horizontal saccade amplitude may reflect dysfunction in the fronto-striato-collicular-cerebellar circuit that underlies volitional saccade generation and overlaps with attentional and visuospatial processing. Disruption of this network may predispose patients to hallucinations.

Keywords

Parkinson's disease, Visual Hallucination, Saccade

P-147

Sleep–Autonomic Interactions in Parkinson's Disease and Multiple System Atrophy: A Comparative Clinical Study with Structural Equation Modeling

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Background & Objective:

Parkinson's disease (PD) and multiple system atrophy (MSA) are α -synucleinopathies that share autonomic dysfunction and sleep disturbances, but differ in trajectories and pathophysiological mechanisms. This study compared sleep and autonomic profiles in drug-naïve PD and MSA and examined disease-specific associations between these domains.

Methods:

A retrospective cohort of 136 drug-naïve PD and 67 MSA underwent overnight polysomnography (PSG) and autonomic evaluation, including autonomic function testing (AFT) and cardiac [I-123] MIBG scintigraphy. Group comparisons were performed for sleep and autonomic parameters. Variables showing significant associations and intergroup differences in Spearman correlation analyses were subsequently entered into structural equation models (SEM) to explore latent relationships.

Results:

The PD group was older than the MSA group, with no differences in sex, disease duration, or autonomic-related comorbidities. In MSA patients, PSG revealed reduced stage N3 sleep and higher respiratory disturbance index (RDI), periodic limb movement index (PLMI), and arousal index. Cardiac MIBG uptake was more reduced in PD. Autonomic testing in MSA showed greater blood pressure drop during head-up tilt and higher Composite Autonomic Severity Score (CASS), except for the cardiovagal subscore. SEM in MSA demonstrated a significant negative association from autonomic dysfunction to sleep parameters, indicating that greater autonomic impairment was associated with poorer sleep quality. A marginal covariance between autonomic and sleep constructs suggested a potential interrelationship rather than a strictly unidirectional effect.

Conclusions:

In MSA, both autonomic and sleep parameters were significantly more impaired than in PD. SEM further indicated that these domains are closely linked, whereby greater autonomic dysfunction is associated with poorer sleep quality. This interrelationship highlights the need for integrated management strategies addressing both domains.

P-148

Peripheral and Central Inflammatory Profiles Associate With Autonomic Dysfunction in Parkinson's Disease

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Background & Objective:

Inflammation is a key mechanism in Parkinson's disease (PD), and autonomic dysfunction is a major determinant of quality of life. However, the link between autonomic dysfunction and inflammation remains poorly understood. We investigated whether peripheral and central inflammatory marker relate to autonomic function tests in PD.

Methods:

We analyzed blood from 167 drug naïve PD patients. Several peripheral inflammatory indices including PHR(platelet-to-HDL ratio), MHR(monocyte-to-HDL ratio), and NHR(neutrophil-to-HDL ratio) were evaluated. In a sub population (n=85) among them, markers reflecting central nervous system including GFAP and NfL were measured by Simoa analysis. expiratory-inspiratory ratio, HRV(heart rate variability), Valsalva ratio (VR), head up tilt test, sympathetic skin response, cardiac MIBG uptake, and K COMPASS 31 were evaluated. Associations used linear, logistic, and proportional odds models adjusted for age, sex, and covariates.

Results:

HRV was significantly correlated with PHR ($p=0.024$), and VR with MHR ($p=0.033$). In ordinal models, VR demonstrated significant associations with both MHR and NHR ($p=0.005$; $p=0.006$). GFAP and NfL correlated with motor severity ($p<0.05$); GFAP inversely correlated with HRV and VR ($r=-0.305$, $p=0.047$; $r=-0.317$, $p=0.006$) and positively correlated with K COMPASS 31 ($p=0.044$, $p=0.015$). NfL inversely correlated with VR ($r=-0.498$, $p<0.001$) and positively correlated with MIBG uptake, and K COMPASS score (all $p<0.05$).

Conclusions:

Peripheral and central inflammation—particularly measured by MHR and GFAP—align with autonomic impairment in drug naïve PD, supporting the concept of a peripheral–central inflammatory axis that may underlie or reflect the severity of autonomic dysfunction.

Keywords

Parkinson's disease, Autonomic dysfunction, Peripheral inflammation, Single molecule array

P-149

Underreported Non-Motor Manifestations and Their Impact on Parkinson's Disease Outcomes: An Integrative Bibliometric Analysis

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Background & Objective:

Parkinson's disease affects over 10 million people worldwide and is the fastest-growing neurodegenerative disorder in prevalence and disability. Beyond tremor and rigidity, non-motor symptoms (NMS) such as depression, sleep disturbance, autonomic dysfunction, and cognitive decline affect up to 90% of patients and often appear years before motor signs. These symptoms are closely linked to reduced quality of life, caregiver burden, and higher healthcare costs, yet remain under-recognized and unevenly addressed. Bibliometric analysis offers a systematic way to map scientific output, identify gaps, and guide research toward a more holistic approach to care.

Methods:

Data from PubMed, Scopus, and Web of Science (2020–2025) were analyzed using the query ("Parkinson*" AND ("non motor syndrome" OR "non-motor symptom*" OR "NMS") AND ("hidden burden" OR "underreported" OR "quality of life"). Co-occurrence, co-authorship, and visualization analyses were conducted with VOSviewer version 1.6.20.

Results:

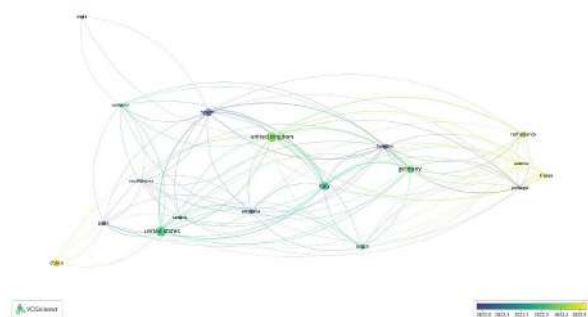
A total of 383 articles on Parkinson's disease were published between 2020 and 2025, with many addressing non-motor symptoms (NMS) such as depression, anxiety, fatigue, insomnia, and cognitive impairment. Co-occurrence analysis revealed steady growth of NMS research alongside motor studies, underscoring their impact on quality of life and daily function. Publication output increased consistently, led by the United States, Germany, and the United Kingdom, while China, Japan, and South Korea showed rapid growth, reflecting strong international collaboration and rising global interest in NMS.

Conclusions:

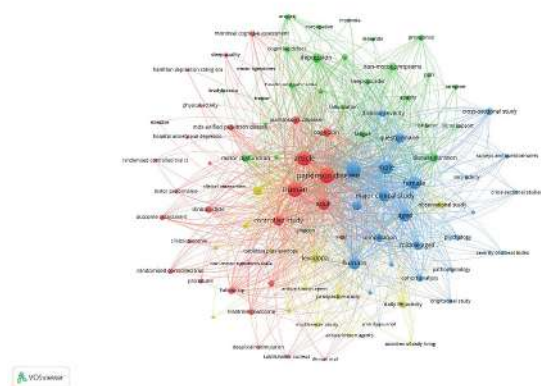
This bibliometric analysis highlights the rising focus on NMS in Parkinson's disease and their major impact on quality of life. While Western countries dominate output, Asian contributions are expanding. Future studies should clarify progression, com-

pare management across regions, and develop targeted interventions through global collaboration.

Figure



Figure



Keywords

Bibliometric analysis, Parkinson's disease, Non-Motor Symptoms

P-150

Gut Microbiota And Neurodegeneration: A Bibliometric Study Of The Gut–Brain Axis In Parkinson’s Disease

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Background & Objective:

Parkinson’s disease (PD) is increasingly recognized as a multisystem disorder. Gastrointestinal dysfunction and α -synuclein in the gut often precede motor symptoms, supporting a gut–brain axis role. This study maps publication trends, research domains, and collaborations on the gut–brain axis in PD, and integrates bibliometric findings with mechanistic and translational evidence.

Methods:

Data were retrieved from Scopus (2020–2025). Only English peer-reviewed and full articles were included. Bibliometric indicators (keyword co-occurrence and co-authorship) were analyzed and visualized with VOSviewer (v1.6.20).

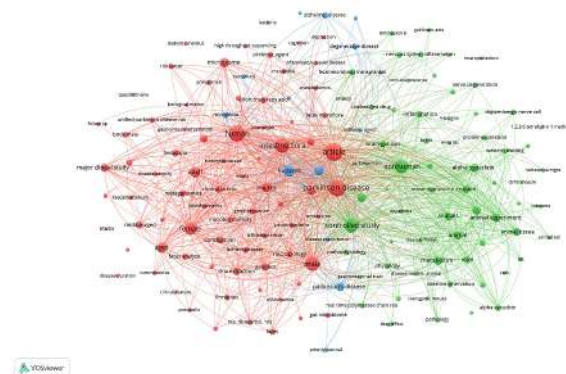
Results:

A total of 306 articles were identified, with outputs nearly tripling between 2020 and 2025. Keyword mapping showed three clusters: (1) clinical studies on gastrointestinal symptoms and microbiome composition, (2) mechanistic work on α -synuclein transmission, barrier disruption, and inflammation in animal models, and (3) methodological advances in sequencing and bioinformatics. Overlay mapping indicated a shift from descriptive microbiome surveys dominant in 2020–2021 to mechanistic and therapeutic approaches, including probiotics and fecal microbiota transplantation, which became more prominent after 2022. Collaboration networks revealed the United States as an early leader, Europe as a dense hub, and China with other Asian countries as emerging contributors.

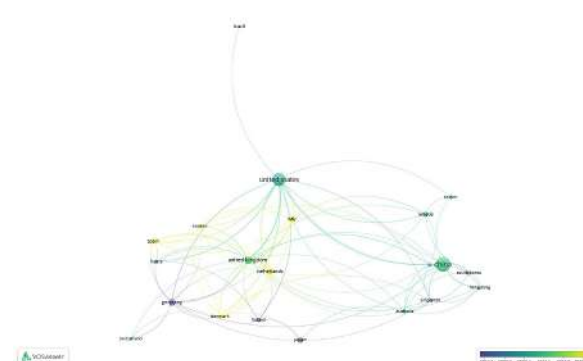
Conclusions:

Silent intestinal inflammation and dysbiosis are central to PD pathogenesis, providing opportunities for biomarkers and microbiome-based therapies. Bibliometric mapping shows rapid growth but methodological heterogeneity. As analyses rely on metadata, findings should be interpreted cautiously. Standardized protocols, longitudinal cohorts, and multi-omic approaches are needed to clarify causality and enable gut-targeted strategies for early diagnosis and intervention.

Figure



Figure



Keywords

α -Synuclein, Bibliometric Analysis, Dysbiosis, Gut–Brain Axis, Parkinson’s Disease

P-151

Association Between Constipation and Parkinson's Disease: A Propensity Score Matching Analysis Using the Korean National Health Insurance Service National Sample Cohort

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Background & Objective:

Constipation a well-recognized prodromal symptom of Parkinson's disease (PD) often precedes motor symptom onset by several years. While the brain-gut axis has been implicated in PD pathogenesis, longitudinal evidence linking constipation, laxative use, and PD risk remains limited in Asian populations. This study investigated these associations in a Korean nationwide cohort.

Methods:

We conducted a retrospective cohort study using the Korean National Health Insurance Service's National Sample Cohort. Individuals aged ≥ 50 years without a history of PD or constipation were identified and categorized into constipation and non-constipation groups, defined by ICD-10 code K590 or ≥ 60 consecutive days of laxative prescriptions between 2008 and 2009. Propensity score matching (PSM) (1:3) was applied to balance demographics and comorbidities. Participants were followed for PD incidence (G20) from 2010 to 2019. Cox proportional hazards models were used to estimate hazard ratios (HRs) with 95% confidence intervals (CIs).

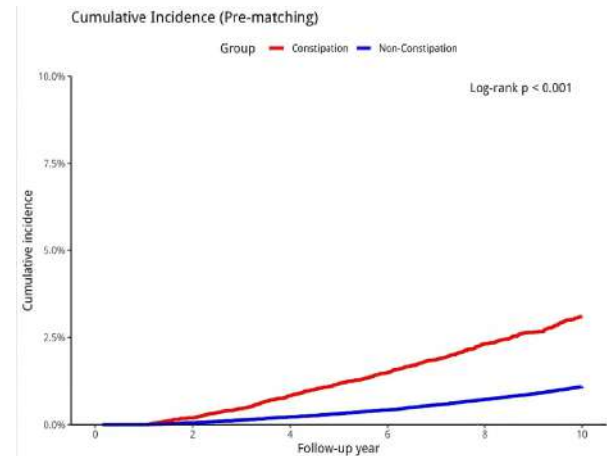
Results:

In PSM cohort ($n=47,580$), 945 PD cases were identified over 11 years. Incidence rates per 100,000 person-years were 162 and 314 in the non-constipation and constipation groups, respectively. 5-year cumulative incidence rates were 0.49% and 1.17% (relative risk [RR]=2.04), and 10-year rates were 1.61% and 3.10% (RR=1.92), indicating persistent significant differences. Adjusted Cox modeling indicated constipation was associated with nearly double the PD risk (hazard ratio [HR], 1.95; 95% confidence interval [CI], 1.71–2.23; $p<0.001$). Constipation showed consistent, significant positive association with PD onset across including absolute risk, incidence rates, KM analysis, and Cox regression.

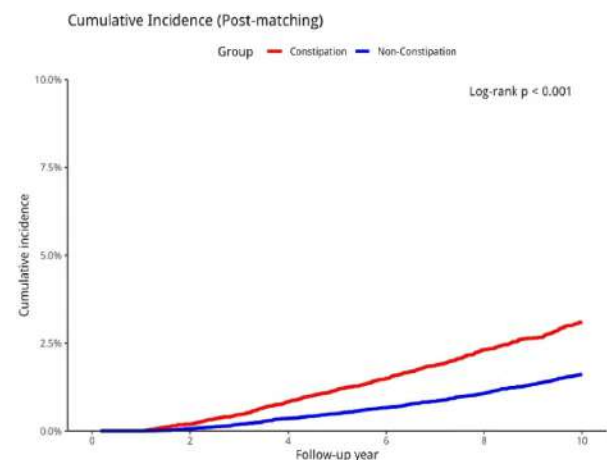
Conclusions:

Constipation was significantly associated with increased PD risk in this population-based cohort. These findings support constipation as an early clinical indicator of PD and highlight the potential value of monitoring gastrointestinal symptoms for timely detection and intervention.

Figure



Figure



Keywords

Parkinson disease, Constipation, Prodromal symptoms, Gastrointestinal Motility Disorders, Cohort studies

P-152

Therapeutic Potential of Probiotics Prebiotics and Synbiotics for Constipation in Parkinson's Disease

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Background & Objective:

Constipation is a common nonmotor symptom of Parkinson's disease (PD) that significantly reduces quality of life. Recent evidence suggests that gut microbiota-targeted therapies such as probiotics, prebiotics, and synbiotics may offer clinical benefits. This study aims to evaluate their therapeutic potential and underlying mechanisms in managing PD-related constipation.

Methods:

A systematic literature search was performed using PubMed, Cochrane Library, ScienceDirect, ClinicalTrials.gov, and other relevant databases to identify preclinical and clinical studies evaluating the effects of probiotics, prebiotics, and synbiotics on constipation in PD.

Results:

Probiotic, prebiotic, and synbiotic supplementation demonstrated beneficial effects on gastrointestinal function, including improvements in colonic transit time, stool consistency, and defecation frequency. In human studies, inulin (a prebiotic) was associated with enhanced bowel movement frequency, while probiotic strains such as *Bifidobacterium lactis* and *Lactobacillus casei* improved stool frequency and consistency. Synbiotics produced synergistic outcomes by combining microbiota modulation with substrate provision, leading to increased production of short-chain fatty acids, improved mucin secretion, and enhanced gut motility and barrier integrity.

Conclusions:

Microbiota-targeted interventions using probiotics, prebiotics, and synbiotics show promising potential in managing constipation in PD. Nonetheless, further robust randomized controlled trials are necessary to determine the most effective strains, combinations, dosages, and treatment durations, as well as to elucidate the precise underlying mechanisms.

Keywords

Parkinson's disease, constipation, gut microbiota, probiotics, prebiotics, synbiotics, gastrointestinal dysfunction, neurogastroenterology, gut-brain axis, short-chain fatty acids

Parkinson's Disease: Others

P-153

Insulin Resistance Can Predict Mortality in Parkinson's Disease Patients

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Background & Objective:

Parkinson's disease (PD), a progressive neurodegenerative disorder, is increasingly linked to insulin resistance—even in non-diabetic patients, where it affects about two-thirds. Tools like HOMA-IR and the TyG index help detect this hidden metabolic dysfunction. Elevated alpha-synuclein may impair insulin secretion, and brain-specific insulin resistance has been observed in PD, independent of diabetes. This study explores the link between insulin resistance and PD mortality, aiming to inform new management strategies.

Methods:

We analyzed data from 3,193 Parkinson's disease patients in the UK Biobank, a large-scale biomedical database. Of these, 1,387 had confirmed mortality records.

Results:

Among Parkinson's disease mortality cases, a Pearson's correlation of -0.15 ($p < 0.001$) was observed between days lived and the TyG index, indicating a significant inverse relationship. Linear regression further supported the association between higher TyG index values and reduced survival, suggesting its potential as a predictor of survival outcomes in PD patients.

Conclusions:

These findings highlight the potential of insulin resistance markers, such as the TyG index, as valuable prognostic tools in Parkinson's disease. They may aid in patient assessment and management and warrant further research to deepen our understanding of this relationship.

Figure

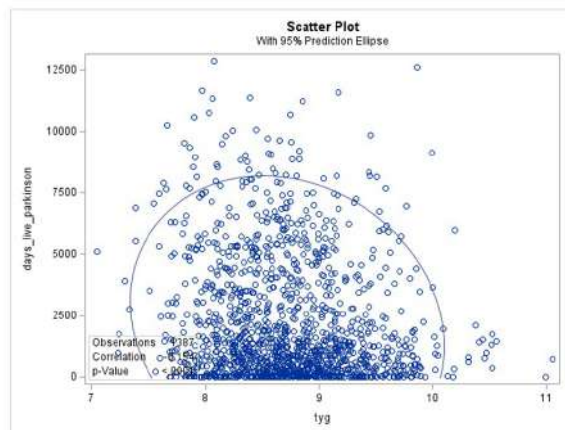


Figure 1- Correlation between survival in Parkinson's disease patients and TyG index

Table

Table 1- Linear regression analysis results of the association between insulin resistance index and survival in Parkinson's disease patients

Parameter Estimates							
Variable	DF	Parameter Estimate	Standard Error	t Value	Pr > t		
Intercept	1	7985.57102	1009.45766	7.91	<.0001		
tyg	1	-667.73101	115.50162	-5.78	<.0001		
Variable		N	Mean	Std Dev	Sum	Minimum	Maximum
tyg		1387	8.72170	0.56185	12097	7.04695	11.05094
days_live_parkinson		1387	2162	2444	2998442	0	12849

Keywords

Parkinson, Biomarker, Insulin resistance, Survival

P-155

Dipeptidyl Peptidase-4 Inhibitors and Risk of Parkinson's Disease: Nationwide, Nested Case-Control Study Including Non-Diabetic Comparators

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Background & Objective:

Parkinson's disease (PD) is a neurodegenerative disorder, and there is currently no disease-modifying treatment available. Previous studies have suggested that dipeptidyl peptidase-4 inhibitors (DPP-4is) may have disease-modifying effects in PD. However, there have been very few studies including a group without diabetes. This study aims to examine whether DPP-4is can reduce the incidence of PD, even compared to non-diabetic individuals.

Methods:

We conducted a nationwide, nested case-control study using data from the Korean National Health Insurance Service between 2010 and 2022. We identified 44,613 incident PD cases aged 40–80 years and selected 178,452 matched controls without PD based on age, sex, and follow-up duration using incidence density sampling. Adjusted odds ratios (aORs) for PD were calculated according to DPP-4i use and cumulative duration, with stratification by age (<70 vs ≥70 years) and CVD status.

Results:

Among individuals aged <70 years, DPP-4i use was associated with a significantly reduced PD risk compared to the non-diabetic population (aOR 0.88, 95% CI 0.81–0.95), with a dose-dependent trend observed for longer duration of use (aOR 0.84 [0.73–0.96] for ≥3 years; P for trend = 0.003). The strongest protective association was found in younger individuals with CVD, where DPP-4i use was associated with lower PD risk regardless of duration (aOR 0.74, 95% CI 0.62–0.87).

Conclusions:

DPP-4i use may be associated with a reduced risk of developing PD, especially among younger individuals and those with coexisting CVD, even relative to individuals without diabetes.

P-156

The Clinical Usefulness of Thyroid Uptake in ¹²³I-MIBG Myocardial Scintigraphy: Model-implied Predictions

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Background & Objective:

¹²³I-metaiodobenzylguanidine (¹²³I-MIBG) myocardial scintigraphy has been used to assess the severity of myocardial denervation in Parkinson's disease (PD). The thyroid gland receives sympathetic innervation, and its uptake can be simultaneously visualized alongside the heart. However, its clinical utility has seldom been investigated. The purpose of this study was to ascertain whether thyroid-to-mediastinum uptake ratios (TMR) could reflect the clinical aspects of PD.

Methods:

A total of 233 patients were studied in this cohort. Myocardial scintigraphy was reassessed two or more times. Early and delayed heart-to-mediastinum uptake ratios (HMR) and early TMR were calculated. The *initial* early TMR was extrapolated from a random intercept and fixed slope mixed model. Baseline assessments captured orthostatic stress, circadian blood pressure variability, motor severity, non-motor burden, and quality of life. These clinical measurements were associated with observed and model-inferred estimates of their respective HMR and TMR.

Results:

Early PD patients were enrolled and followed for an average of 57.3 ± 22.1 months. The average age at diagnosis was 68.7 ± 8.7 years, with 51.9% of patients being male. Disease duration at diagnosis was 1.0 (interquartile range, IQR, 1.0) years, and the median Hoehn and Yahr stage was 2.0 (IQR, 1.0). The trajectories of observed HMR and TMR were comparable across the time points; thus, the initial TMR values were estimated by the mixed model. The significant negative correlations were observed between HMR and orthostatic BP changes. The HMR values were inversely associated with non-motor burden, quality of life, and motor aspects of Parkinson's disease. These patterns were not observed with TMR.

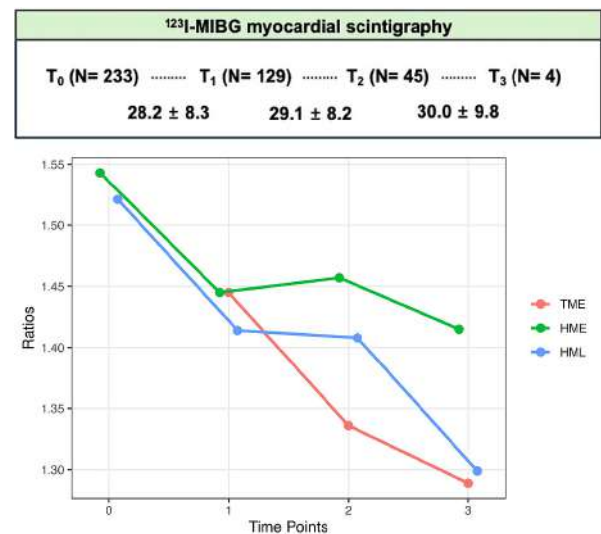
Conclusions:

The contemporary thyroid uptakes in ¹²³I-metaiodobenzylguanidine myocardial scintigraphy appear to have little clinical significance compared to myocardial uptakes.

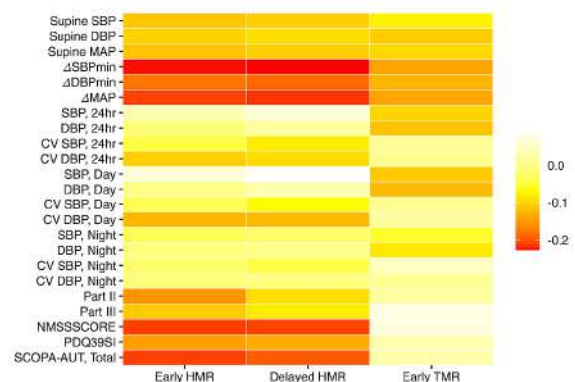
Table

	Overall 233
N	
Age at diagnosis, years (mean (SD))	68.73 (8.68)
Male (%)	121 (51.9)
Disease duration at diagnosis, years (median [IQR])	1.00 [0.50, 1.50]
Diabetes mellitus (%)	44 (18.9)
Dyslipidemia (%)	70 (30.0)
Hypertension (%)	114 (48.9)
Nonsmoker (%)	229 (98.3)
Follow-up duration, months (mean (SD))	57.25 (22.09)
HY stage (median [IQR])	2.00 [1.00, 2.00]
Converted MDS-UPDRS Part II (median [IQR])	5.00 [3.00, 9.00]
Converted MDS-UPDRS Part III (mean (SD))	18.82 (10.64)
Total Motor Scores (Part II + Part III) (median [IQR])	22.60 [15.60]
Early H/M ratio (mean (SD))	1.54 (0.30)
Delay H/M ratio (mean (SD))	1.52 (0.36)
Early T/M ratio (mean (SD))	1.50 (0.15)
Delay T/M ratio (mean (SD))	1.67 (0.64)

Figure



Figure



P-157

Cortical Substrate of Impulse Control Disorder and Levodopa-Induced Dyskinesia in Parkinson's Disease

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Background & Objective:

Impulse control disorder (ICD) and levodopa-induced dyskinesia (LID) in Parkinson's disease are both associated with dopamine therapy and exhibit overlapping frontostriatal dysfunction, suggesting a shared neural mechanism. The underlying cortical neural substrate remains to be elucidated. In this study, we aimed to investigate the longitudinal association between baseline ICD and the risk of developing LID.

Methods:

We analysed data of participants of the Parkinson's Progression Markers Initiative study who were followed up for more than 4 years ($N=464$). We employed Cox proportional hazard regression models to assess the association between baseline ICD and future risk of LID. Using morphometric similarity network analysis, we identified a subnetwork showing significant differences in connectivity between the groups of Parkinson's disease patients with and without ICD and determined its hub regions. Network topological measures of these hub regions were examined for associations with both ICD status and subsequent LID risk. Causal mediation analysis was performed to test whether the relationship between ICD and LID was mediated by the topological features of the identified hub regions.

Results:

We found that the presence of ICD was associated with an increased risk of LID independent of putaminal dopaminergic depletion. The group of Parkinson's disease patients with ICD showed lower morphometric similarity network connectivity centred on bilateral paracentral lobule and left anterior cingulate cortex. The influence of ICD-related morphometric similarity network disruption on LID development was mediated by the within-module z-score of the left paracentral lobule, which on average belongs to a module comprising sensorimotor network regions.

Conclusions:

This suggests that a common pathophysiological mechanism of motor inhibition failure may underlie impulse control disorder and levodopa-induced dyskinesia.

P-158

Impact of Amyloid-Beta Pathology on Longitudinal Motor and Non-Motor Symptoms in Parkinson's Disease

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Background & Objective:

Alzheimer's disease pathology, which is characterized with amyloid-beta and tau aggregation, is commonly observed in patients with Parkinson's disease (PD). Presence of amyloid-beta is associated with worse cognition and higher risk of dementia, but its effect on longitudinal symptomatic trajectory is not elucidated. We aimed to investigate the impact of amyloid-beta pathology on longitudinal trajectories of motor and non-motor symptoms in PD.

Methods:

388 early PD patients (disease duration less than 3 years) who had followed up more than 3 years and who had available baseline cerebrospinal fluid study were included for the analysis from PPMI database. Amyloid-beta pathology was defined to be elevated CSF ptau/Abeta42 ratio > 0.02 . Baseline characteristics were compared between amyloid-beta positive and negative group using chi-square test and independent t-test. The impact of amyloid-beta pathology on motor symptom, non-motor symptom, and dopamine transporter availability were investigated using linear mixed effects model.

Results:

Sixty-nine patients had positive amyloid-beta pathology at baseline. Amyloid-beta positive patients were older and had APOE4 allele more frequently. Other clinical features and dopamine transporter availability were comparable between the two groups. The presence of amyloid-beta pathology was associated with faster deterioration of MoCA score and increase of MDS-UPDRS part I score. The presence of amyloid-beta pathology was associated with the faster increase in MDS-UPDRS part II and III scores, and faster increase in postural instability-gait disturbance and akinetic-rigid symptoms. The presence of amyloid pathology was not associated with the longitudinal dopamine transporter availability or motor complications including motor fluctuation, levodopa-induced dyskinesia, and freezing of gait.

Conclusions:

Amyloid-beta pathology is associated with faster deterioration of motor and non-motor symptoms of PD, independent of ni-

grostriatal dopaminergic degeneration.

P-159

Clinical Characteristics of Geriatric Patients with de novo Parkinson's Disease in Comparison to the Non-Geriatric Population: Adapting to Changes in the Era of Aging

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Background & Objective:

Parkinson's disease (PD) is increasingly diagnosed in older adults. Despite this trend, the clinical features of geriatric patients with PD are not thoroughly defined. This study aimed to compare the clinical characteristics of geriatric patients (aged ≥ 75 years) with de novo PD against those of non-geriatric patients (aged < 75 years) newly diagnosed with PD.

Methods:

This retrospective analysis enrolled 110 patients aged 50 years or older with de novo PD from our hospital's Parkinsonism registry between 2017 and 2023. Clinical evaluations included motor assessment via the Unified Parkinson's Disease Rating Scale Part III, and global cognitive function was measured using the Montreal Cognitive Assessment (MoCA). Nonmotor symptoms, including depression, anxiety, and fatigue, were assessed with other scales, and autonomic dysfunction—was assessed with the Scale for Outcomes in Parkinson's Disease—Autonomic (SCOPA-AUT).

Results:

Geriatric patients with PD (n=37) exhibited significantly lower cognitive performance (lower MoCA scores, $p < 0.001$) and more pronounced autonomic dysfunction (higher SCOPA-AUT scores, $p = 0.039$) in comparison to non-geriatric PD patients (n=73) (Table 1 and Figure 1). In multivariable logistic regression analysis (Table 2), lower MoCA scores (odds ratio [OR]: 0.764, 95% confidence interval [CI]: 0.671–0.870, $p < 0.001$) and elevated SCOPA-AUT scores (OR: 1.064, 95% CI: 1.003–1.129, $p = 0.039$) emerged as significant independent predictors of geriatric PD.

Conclusions:

These findings reveal a distinct clinical phenotype among geriatric patients with de novo PD, underscoring the value of early detection and proactive management of cognitive and autonomic impairments in this group. The results further emphasize the need for individualized assessment and therapeutic interventions tailored to the specific requirements of geriatric patients with PD.

Table

Table 1. Comparison of clinical features between non-geriatric and geriatric patients with de novo Parkinson's disease

Variable	Total (n=110)	Non-geriatric PD (n=73)	Geriatric PD (n=37)	P value
Age, yr	70.65 $\pm$ 8.17	66.18 $\pm$ 5.98	79.49 $\pm$ 3.13	<0.001
Gender-female	57 (51.82%)	36 (49.32%)	21 (56.76%)	0.5919
Body weight, kg	60.47 $\pm$ 10.16	61.57 $\pm$ 9.86	58.3 $\pm$ 10.54	0.1206
Height, m	1.60 $\pm$ 0.09	1.62 $\pm$ 0.08	1.58 $\pm$ 0.09	0.0265
Body mass index	23.37 $\pm$ 2.69	23.41 $\pm$ 2.71	23.28 $\pm$ 2.69	0.8125
Disease duration, yr	1.29 $\pm$ 0.93	1.35 $\pm$ 0.96	1.17 $\pm$ 0.86	0.3093
Level of education, yr	10.14 $\pm$ 5.01	11.29 $\pm$ 4.7	7.88 $\pm$ 4.88	<0.001
Diabetes mellitus, n(%)	29 (26.36%)	19 (26.03%)	10 (27.03%)	>0.99
Hypertension, n(%)	55 (50%)	35 (47.95%)	20 (54.05%)	0.6865
Previous faller, n(%)	44 (40%)	27 (36.99%)	17 (45.95%)	0.4837
UPDRS-III (motor)	22.29 $\pm$ 11	21.44 $\pm$ 10.75	23.97 $\pm$ 11.45	0.2668
H&Y stage	2 (2, 2)	2 (2, 2)	2 (2, 2)	0.7705
MoCA-K (cognition)	23.85 $\pm$ 4.35	25.32 $\pm$ 2.95	20.95 $\pm$ 5.17	<0.001
BDI(depression)	8.16 $\pm$ 6.97	7.49 $\pm$ 7.03	9.49 $\pm$ 6.74	0.1529
BAI(anxiety)	5.41 $\pm$ 6.01	5.11 $\pm$ 5.92	6 $\pm$ 6.22	0.4734
PFS (fatigue)	39.47 $\pm$ 16.81	37.81 $\pm$ 17.59	42.76 $\pm$ 14.83	0.1246
SCOPA-AUT (dysautonomia)*	11.05 $\pm$ 7.82	9.62 $\pm$ 7.21	13.86 $\pm$ 8.32	0.0103

* This table was summarized as appropriate according to normality test with Kolmogorov-Smirnov test and the presence of warning from chi-squared test: numerical data are presented as mean S.D or interquartile range and Non-numerical data are shown as number (%).

*Total score of SCOPA-AUT was calculated except for sexual domain, since many patients did not answer the question for the sexual dysfunction.

UPDRS, Unified Parkinson Disease Rating Scale (UPDRS); H&Y, Hoehn and Yahr; MoCA-K, Korean version of Montreal Cognitive Assessment; BDI, Beck Depression Inventory; BAI, Beck Anxiety Inventory; PFS, Parkinson's disease Fatigue Scale; SCOPA-AUT, Scales for Outcomes in Parkinson's disease – Autonomic

Table

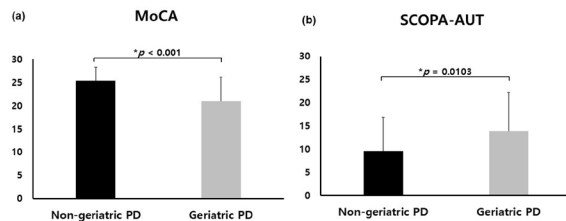
Table 2. The logistic regression analyses of clinical characteristics for geriatric PD in patients with de novo PD

Variable	Univariable			Multivariable		
	Odds ratio	95% CI	p-value	Odds ratio	95% CI	p-value
Gender-female	1.3490	0.6086 – 2.9900	0.4611			
Body weight, kg	0.9678	0.9294 – 1.0077	0.1127			
Height, m	0.0040	0.0001 – 0.4836	0.0242			
Body mass index (BMI)	0.9820	0.9468 – 1.1389	0.8105			
Disease duration, yr	0.7972	0.5085 – 1.2580	0.3234			
Level of education, yr	0.8622	0.7882 – 0.9433	0.0012			
Diabetes mellitus, n(%)	1.0536	0.4304 – 2.5743	0.9105			
Hypertension, n(%)	1.2773	0.578 – 2.8229	0.5452			
Previous faller, n(%)	1.4481	0.6491 – 3.2306	0.3657			
UPDRS-III (motor)	1.0212	0.985 – 1.0587	0.2541			
H&Y stage	1.0432	0.405 – 2.6870	0.9302			
MoCA-K (cognition)	0.7547	0.6621 – 0.8603	<0.001	0.7642	0.6712 – 0.8701	<0.001
BDI(depression)	1.0410	0.984 – 1.1013	0.162			
BAI(anxiety)	1.0244	0.9604 – 1.0927	0.4633			
PFS (fatigue)	1.0180	0.9938 – 1.0427	0.1458			
SCOPA-AUT (dysautonomia)*	1.0726	1.0176 – 1.1306	0.0091	1.0640	1.0031 – 1.1286	0.0391

*Multivariable logistic regression analysis with step-wise selection was performed.

PD, Parkinson's disease; CI, confidence interval; UPDRS-III, the Unified Parkinson's disease rating scale-part 3; MoCA-K, Korean version of Montreal Cognitive Assessment; BDI, Beck Depression Inventory; BAI, Beck Anxiety Inventory; PFS, Parkinson's disease Fatigue Scale; SCOPA-AUT, Scales for Outcomes in Parkinson's disease – Autonomic

Figure



Keywords

Aged, Cognition, Geriatrics, Parkinson Disease

P-160

Trends in Parkinson's Disease Hospitalizations in Germany, 2000-2023

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Background & Objective:

Parkinson's disease is a progressive neurodegenerative disorder that significantly impacts quality of life. Management often requires long-term medical and inpatient care, particularly as the disease advances. Understanding hospitalization trends can provide insights into disease burden, healthcare utilization, and regional disparities in access to specialized care. This study examines hospitalization trends for Parkinson's disease in Germany, focusing on regional differences at the federal state level.

Methods:

Using data from the German Federal Statistical Office, annual hospitalizations for Parkinson's disease (ICD-10 G20) from 2000 to 2023 are examined. Age-standardized hospitalization rates (ASHRs) per 100,000 inhabitants, based on the 2013 European standard population, were calculated. Joinpoint regression analysis was employed to identify temporal trends, and Average Annual Percent Changes (AAPCs) with 95% confidence intervals were computed.

Results:

Nationally, the ASHR for Parkinson's disease increased from 37.1 per 100,000 inhabitants in 2000 to 46.0 in 2023 (Fig. 1). Substantial regional differences were observed across Germany's federal states. In 2023, the highest rates were recorded in Saarland

(63.2) and Thuringia (61.4), while the lowest rates were seen in Baden-Württemberg (35.0) and Bavaria (41.2) (Fig. 2). The relative increases in ASHRs over the study period varied widely, with the largest increases in Thuringia (+104.7%) and Saxony (+60.0%) and the smallest in Baden-Württemberg (+0.6%) and North Rhine-Westphalia (+11.2%).

Conclusions:

The findings reveal significant regional heterogeneity in Parkinson's disease hospitalization trends, suggesting variations in disease prevalence, healthcare infrastructure, or diagnostic practices across federal states. The limited granularity of the data underscores the need for further research to explore the underlying causes of these disparities, including demographic, environmental, and healthcare system factors.

Figure

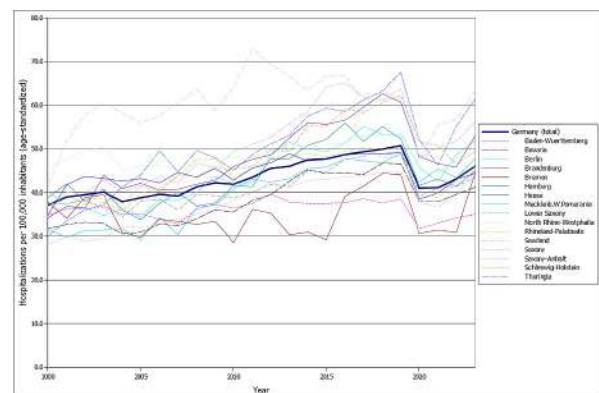


Fig. 1: Parkinson's disease hospitalizations per 100,000 inhabitants (age-standardized) in Germany by federal state, 2000-2023

Figure

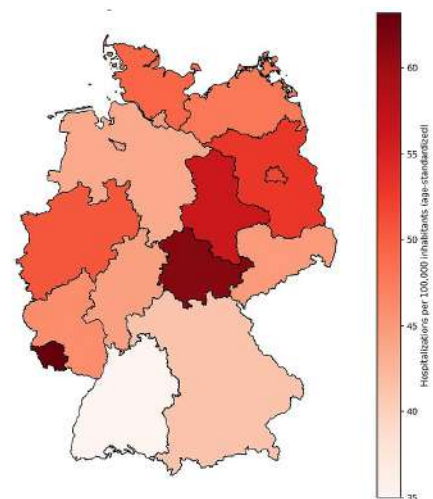


Fig. 2: Geographical distribution of Parkinson's disease hospitalizations in Germany by federal state (per 100,000 inhabitants, age-standardized, 2023)

Keywords

Parkinson's, Trend, Hospitalization, Germany, Joinpoint

P-162

Antioxidative and Anti-inflammatory Activity After Probiotic Intervention in Parkinson's Patients: A Systematic Review and Meta-Analysis Randomized Controlled Trials

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Background & Objective:

There is increasing evidence highlighting the potential role of the gut-brain axis in the pathogenesis of parkinson's disease patients. This meta-analysis aims to determine the antioxidative and anti-inflammatory activity after probiotic intervention in parkinson's disease patients.

Methods:

The systematic review and meta-analysis are reported based on PRISMA guidelines. The Embase, Cochrane Library, PubMed, and Web of Science databases were comprehensively searched for randomized controlled trials (RCTs) studies reporting the intervention of probiotics on inflammation markers and oxidative stress in Parkinson's patients during the past 10 years. Outcomes included interleukin-10 (IL-10), tumor necrosis factor-alpha (TNF- α) (anti-inflammatory parameters), and malondialdehyde level (MDA), and glutathione level (GSH) (antioxidative parameters) after the probiotic intervention. The results were reported as the Mean Difference (MD) with 95% confidence intervals (CI) and a random-effects model.

Results:

The meta-analysis included 7 RCTs that fulfilled the inclusion criteria with a total of 410 participants. Probiotic intervention significantly reduced the TNF-alpha compared with the control group (MD -8.60; 95%CI [-11.80 to -5.40]; I²=82%; P<0.001). Probiotic intervention significantly increased the IL-10 compared with the control group (MD 2.71; 95%CI [0.29 to 5.14]; I²=84%;

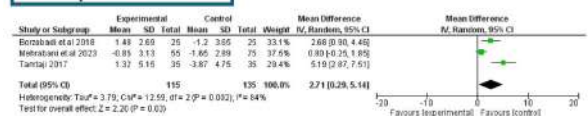
P=0.03). There is shown a significantly reduced malondialdehyde level ($\mu\text{mol/L}$) after the probiotic intervention compared with the control group (MD -0.51; 95%CI [-0.88 to -0.15]; I²=40%; P=0.006). Probiotic intervention significantly increased the glutathione level ($\mu\text{mol/L}$) compared with the control group (MD 40.29; 95%CI [30.57 to 50.02]; I²=0%; P<0.001).

Conclusions:

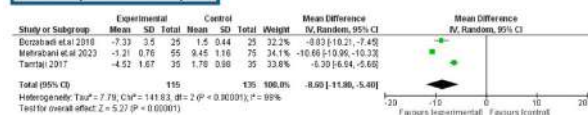
Probiotic intervention may be suitable for improving the oxidative and anti-inflammatory activity in parkinson's disease patients. Further research is warranted to determine the optimal treatment protocol for Parkinson's disease patients.

Figure

Forest plot IL-10 Level

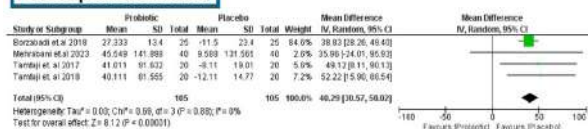


Forest plot TNF-Alpha

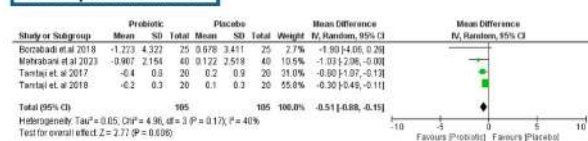


Figure

Forest plot GSH Level



Forest plot MDA Level



Keywords

Antioxidative, Anti-inflammatory, Probiotic, Parkinson's

P-163

Autopsy-Confirmed Parkinson's Disease With a Novel GBA p.Asn227Ser Variant and Brainstem-Predominant Lewy Pathology: A 37-Year Young-Onset Case

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Introduction:

Variants in GBA, encoding lysosomal glucocerebrosidase, are the strongest common genetic risk for Parkinson's disease (PD), yet clinicopathologic data from Korea are scarce. We report an autopsy-confirmed PD case harboring a heterozygous GBA c.680A>G (p.Asn227Ser) variant not previously described in Korean PD cohorts.

Case:

A woman developed young-onset PD at age 34 with bradykinesia and rest tremor. Severe wearing-off and levodopa-induced dyskinesia led to bilateral STN-DBS at 56 (mUPDRS off 31/HY 4 to on 17/HY 2.5). Cognition remained intact (MMSE 30/30 before and 3 months after DBS), and there was no family history. Despite optimized therapy, she had frequent falls late in the course and died at 69 from a large right basal ganglia/frontal intracerebral hemorrhage. Consent for brain donation was obtained.

Discussion:

This profile (young onset, high motor burden, DBS for dyskinesia) alongside 37-year survival without dementia contrasts with typical PD-GBA trajectories. Autopsy showed brain weight 1,160 g with mild substantia nigra pallor and mild neuronal loss in the locus coeruleus. Alpha-synuclein immunohistochemistry revealed

mild Lewy bodies confined to the ventral striatum and medulla, without neocortical involvement; beta-amyloid showed only mild angiopathy without plaques; phosphorylated-tau showed mild neurofibrillary tangles (moderate in the amygdala); TDP-43 was negative. Vascular co-pathology included arteriolosclerosis and multiple infarcts. Whole-genome sequencing of brain tissue identified heterozygous GBA c.680A>G (p.Asn227Ser). To our knowledge, this is the first autopsy-confirmed PD with GBA in Korea. Brainstem-predominant Lewy pathology provides a plausible substrate for preserved cognition and expands the clinicopathologic spectrum of PD-GBA in East Asians.

Keywords

GBA, Parkinson's disease, young-onset, Lewy body disease, autopsy

P-164

Socioeconomic and Caregiver Burden of Parkinson's Disease in Southeast Asia: A Systematic Review

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Background & Objective:

Parkinson's disease (PD) is a progressive neurodegenerative disorder whose prevalence is increasing globally as populations age. In Southeast Asia (SEA), demographic transitions signal an impending rise in PD burden, yet investigations into its socioeconomic and caregiver impacts remain limited. This systematic review aims to synthesize available evidence on the economic burden, caregiver strain, and health system readiness for PD in SEA.

Methods:

We reviewed publications from 2000–2024 in PubMed and Scopus, as well as GBD reports, focusing on PD's non-clinical impacts in SEA. Key domains included direct/indirect costs, caregiver workload, and service availability across SEA countries.

Results:

The GBD 2016 report estimated 409,655 prevalent PD cases in SEA in 2016, with SEA showing an approximate 24.7% increase

in age-standardized rates between 1990 and 2016, the highest among Southeast Asian nations such as Thailand (31.7%) and Indonesia. Economic burden data are scarce; however, globally, caregivers provide up to 22 hours per week of informal care, contributing significant indirect costs. Telemedicine, as evaluated in broader Asian contexts, reduced transportation costs by around 30–35%, suggesting potential applicability in SEA.

Conclusions:

PD in Southeast Asia presents a growing public health challenge with substantial societal and caregiver implications, yet data on economic and systemic impacts remain sparse. There is an urgent need for SEA-specific research on cost-of-illness, caregiver burden, and health service availability to inform policy and health system strengthening.

Keywords

Parkinson's disease, Southeast Asia, Caregiver strain, Health system readiness, Systematic review

P-165

Influence of Amyloid Copathology on White Matter Network Alterations in Parkinson's Disease

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Background & Objective:

While Alzheimer copathology is known as one of the major factors that aggravates cognitive and neuropsychiatric symptoms (NPS) in Parkinson's disease (PD), their mechanistic link remains elusive. We investigated whether amyloid- β (A β) elicit white matter (WM) network disruption and further explored the poten-

tial clinical consequences.

Methods:

We included patients with PD who underwent 18F-florbetaben PET, diffusion-tensor imaging (DTI), and neuropsychological assessments. After extracting A β -susceptible WM networks by comparing DTI-metrics (Fractional anisotropy [FA] and mean diffusivity [MD]) according to A β status, linear regression analyses were performed to explore the associations between A β burden, integrity of identified WM networks, and clinical measures (cognition and NPS). Path analyses were performed to determine whether the network integrity mediated the effect of A β on clinical measures.

Results:

Among 165 patients enrolled, 48 showed amyloid-positive (A β +) scan (29.1%). A β +PD revealed compromised FA-weighted (FA-WMN) and MD-weighted WM networks (MD-WMN) in cortico-cortical and cortico-striatal-limbic WM networks. Global A β burden was associated with cognitive dysfunction (attention: β =−0.274, P <0.001; frontal/executive: β =−0.234, P =0.002) and greater NPS burden (motivation: β =0.496, P <0.001; affective dysregulation: β =0.409, P <0.001). Path analyses confirmed that the FA-WMN integrity completely mediate the A β -cognition relationship and partially mediate the A β -NPS relationship. In subgroup analyses within A β +PD, striatal A β burden was associated with memory impairment (β =−0.391, P =0.009) and apathetic symptoms (β =0.418, P =0.004), with both relationships mediated through the compromised MD-WMN integrity.

Conclusions:

Our findings suggest that coexistent A β may elicit additive vulnerability in WM microstructures in PD, with WM network degeneration potentially mediating the contribution of A β to cognitive and neuropsychiatric symptoms.

P-166

Skeletal Muscle Index as Motor Reserve Proxy in Parkinson's Disease

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Background & Objective:

Motor reserve (MR) explains the clinical heterogeneity of Parkinson's disease (PD), even with comparable dopaminergic loss. However, physically measurable proxies of peripheral contributors and corresponding neural implementations remain poorly characterized. This study investigated whether body composition may serve as an MR proxy and identified its cortical neural substrate and implementation using morphological similarity networks.

Methods:

This prospective case-control study analyzed data from 98 patients with early- and late-stage de novo PD and 63 healthy controls. Bioelectrical impedance variables that moderated the association between motor symptom severity and striatal dopamine transporter (DAT) availability, along with associated cortical subnetworks, were identified using threshold-free cluster-enhancement analysis. The subnetwork's moderating effects and relationship with body composition were assessed across three diagnostic groups. We analyzed independent data from the Parkinson's Progression Markers Initiative cohort, of whom 190 remained treatment-naïve over one year, comparing longitudinal motor symptom changes between high- and low-MR groups stratified by the identified cortical subnetwork.

Results:

Only skeletal muscle index (SMI) significantly moderated the association between anterior putaminal DAT availability and motor symptom severity in early-stage PD. We identified an SMI-associated subnetwork, centered on the left caudal middle frontal gyrus, showing stronger connectivity in high-SMI individuals. This connectivity was higher in the high-SMI group than in HCs, a pattern absent in the low-SMI group. The longitudinal analysis revealed a steeper motor decline in the high-MR group.

Conclusions:

Skeletal muscle was identified as a modifiable reserve in early-stage PD, supported by a cortical neural substrate providing neural compensation via this network.

Keywords

motor reserve, skeletal muscle index, morphological similarity network, neural compensation, longitudinal analysis

P-167

Perceptions of Neuropalliative Care in Parkinsonism: Implications from a Multicenter Survey in Korea

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Background & Objective:

Parkinsonism, particularly Parkinson's disease (PD), is a progressive neurodegenerative disorder with motor and non-motor symptoms that negatively affect quality of life and increase caregiver burden. Neuropalliative care has recently emerged for chronic neurological disorders, including PD. This study evaluated awareness and perceptions of palliative care among patients with parkinsonism, their caregivers, and physicians in Korea, and identified factors associated with caregiver burden.

Methods:

A multicenter, cross-sectional study was conducted with 112 patients, 110 caregivers, and 61 physicians. Structured questionnaires assessed awareness and attitudes toward neuropalliative care. Caregiver burden was measured using validated

Korean version of the Parkinson's Disease Questionnaire-Carer (PDQ-Carer).

Results:

Awareness of palliative care differed markedly: 31.3% of patients and 27.5% of caregivers versus 90.2% of physicians. Caregivers were stratified by PDQ-Carer score into low- (≤ 34 , $n=63$) and high-burden (>34 , $n=47$) groups. High-burden caregivers supported patients with longer disease duration, poorer cognition, higher Hoehn and Yahr stage, and greater levodopa equivalent daily doses (LEDD). Regression analysis showed that caregiver demographics were not associated with burden, whereas caregiving duration, patient cognitive decline, Hoehn and Yahr stage, LEDD, and disease duration were correlated. In the multivariable model, only patient disease duration remained significant ($B=2.048$, 95%CI 0.083-4.012, $p=0.041$).

Conclusions:

There is a significant awareness gap in palliative care between physicians and patients/caregivers. The caregiver burden was mainly linked to disease duration as the strongest predictor. Education and structured neuropalliative strategies are needed to support patients with PD and their families

Keywords

Parkinsonism, Neuropalliative care, caregiver burden, PDQ-Carer

P-168

Exploring the Relationship Between Spiritual Well-Being and Clinical Variables in Parkinson's Disease: Insights from a Cross-Sectional Study

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Background & Objective:

Spiritual well-being (SWB) has emerged as a significant psychosocial dimension in chronic neurodegenerative disorders such as Parkinson's disease (PD). However, few studies have systematically examined how SWB—particularly its subcomponents—relates to non-motor symptoms and other clinical characteristics of PD. To investigate the relationship between spiritual well-being

and clinical variables in patients with PD, using validated scales for spiritual health and PD symptoms

Methods:

In this cross-sectional study, 40 patients with idiopathic PD were evaluated using the Functional Assessment of Chronic Illness Therapy–Spiritual Well-Being scale (FACIT-SP12) and the Spiritual Well-Being Scale (SWBS), including their subscales (Meaning, Peace, Faith, Religious Well-Being [RWB], and Existential Well-Being [EWB]). Clinical assessments included the Non-Motor Symptom Scale (NMSS), Beck Depression Inventory (BDI), Beck Anxiety Inventory (BAI), and PDQ-39. Spearman correlations and multiple linear regression analyses were performed. Covariates such as age, sex, coping style, and perceived social support were tested for adjustment

Results:

FACIT-SP12 scores showed a negative correlation with BDI ($\rho = -0.80$, $p < 0.05$) and PDQ-39 ($\rho = -0.44$), and a positive correlation with NMSS ($\rho = +0.57$). The Peace subscale was notably associated with both NMSS and PDQ-39. In contrast, Faith and RWB scores were primarily influenced by religious affiliation, not clinical symptoms. Interestingly, EWB showed a positive association with depressive symptoms (BDI; $\beta = +0.56$, $p = 0.07$).

Conclusions:

Spiritual well-being, especially its "Peace" and "Existential" dimensions, is meaningfully associated with PD patients' emotional and symptomatic burden. This highlights the need to consider spiritual health in holistic management of PD, particularly in addressing non-motor symptoms such as depression and reduced quality of life

Keywords

Parkinson's disease, Spiritual well being, non motor symptoms

P-169

Impact of Socioeconomic Status on the Outcomes of Parkinson's Disease: A Nationwide Population-Based Matched Cohort Study in Korea

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Background & Objective:

The impact of socioeconomic status (SES) on medical outcomes in Parkinson's disease (PD) remains unclear. Using a nationwide cohort in Korea, we investigated the association between individual income level and the risk of disability and cognitive dysfunction.

Methods:

We included patients diagnosed with PD (ICD-10 code G20) and registered with the rare intractable diseases program (V124) between 2005 and 2019 (n = 95,093). Patients with atypical or secondary parkinsonism were excluded. Age-, sex-, and comorbidity-matched controls without PD were enrolled. Subjects were classified into five categories based on the National Health Insurance (NHI) premium, a proxy for income level.

Results:

The medical aid group had a significantly lower risk of disability compared with non-medical aid groups, but showed a higher risk of cognitive impairment. Among participants aged <64 years, higher income levels were associated with a reduced odds ratio (OR) for disability registration. In contrast, among those aged ≥65 years, higher income levels were associated with increased disability registration. PD patients demonstrated a decreased risk of cognitive dysfunction with increasing income levels.

Conclusions:

In Korea, SES indicators such as income level and insurance type were significantly associated with risks of disability and cognitive dysfunction in PD.

P-170

Enhancing Parkinson's Disease Diagnosis in Mongolia: Comparison of UK Brain Bank and MDS Clinical Criteria at the Second State Central Hospital (2020–2025)

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Background & Objective:

Background: Accurate diagnosis of Parkinson's disease (PD) remains challenging, particularly in settings with limited diag-

nostic resources. This study aimed to evaluate and compare the diagnostic outcomes of two widely used clinical criteria: the UK Parkinson's Disease Society Brain Bank (UKBB) and the Movement Disorder Society (MDS, 2015) criteria in a Mongolian clinical cohort.

Methods:

Methods: We retrospectively reviewed 30 patients diagnosed with PD between 2020 and 2025 at the Neurology Department of the Second State Central Hospital. Inclusion was based on cardinal clinical features, including bradykinesia, tremor, postural instability, and rigidity. Patients' diagnoses were assessed according to the UKBB and MDS criteria.

Results:

Results: The mean age of the cohort was 73.57 ± 12.28 years (range 40–95). Using UKBB criteria, 16 patients were confirmed PD, while 14 did not meet the criteria. Using MDS criteria, 16 patients were confirmed, 3 were classified as probable, and 11 were excluded. Notably, the 16 patients with confirmed diagnoses fully overlapped between the two criteria. However, some patients classified as excluded by UKBB were identified as probable PD under MDS criteria, highlighting discrepancies between the two diagnostic frameworks.

Conclusions:

Conclusion: Our findings demonstrate that while both UKBB and MDS criteria identified a core group of PD patients consistently, MDS criteria may capture additional probable cases not recognized by UKBB. This underscores the importance of applying multiple diagnostic frameworks in clinical practice, particularly in resource-limited settings. Moreover, these results highlight the need for ongoing education and skill development for junior physicians and enhanced training programs within our hospital to improve diagnostic accuracy and patient care in Parkinson's disease.

P-171

Evaluating Oxidative Stress, Bone Density, Cognitive Function, and Serum 25-Hydroxyvitamin D Levels in Post-Menopausal Women with Parkinson's Disease

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Background & Objective:

Vitamin D plays a significant role in bone health, and its deficiency in Parkinson's disease (PD) may exacerbate bone-related complications, such as osteoporosis and fractures. The objective of this study is to assess the levels of 25-Hydroxyvitamin D(25(OH)D), bone mineral density (BMD), cognitive difficulties, non-motor symptoms, and oxidative stress in serum of post-menopausal women with PD.

Methods:

This cross-sectional study included 482 post-menopausal women with PD, excluding those with an illness duration of more than 10 days or those who had previously received steroid therapy or vitamin D/calcium supplements. Serum levels of vitamin E, vitamin C, malondialdehyde (MDA), protein carbonyl, and neuro-lipofuscin were measured using biochemical and electrochemiluminescence immunoassay methods. Serum 25-Hydroxyvitamin D (25(OH)D) levels were measured in routinely collected blood samples from PD patients using immunoassay.

Results:

PD patients exhibited significantly lower serum 25(OH)D levels compared to healthy controls ($P < 0.001$). A positive correlation was found between serum 25(OH)D levels and Functional Ambulation Classification (FAC) scores ($p = 0.001$). A negative correlation was observed between malondialdehyde (MDA) and antioxidants (vitamin C and vitamin E), while MDA showed a positive correlation with other oxidative stress markers. Additionally, PD patients with lower 25(OH)D levels had a higher frequency of falls ($P = 0.05$) and insomnia ($P = 0.01$). They also scored higher on the Pittsburgh Sleep Quality Index (PSQI; $P = 0.01$), depression ($P = 0.05$), and anxiety ($P = 0.001$). Moreover, these patients had significantly lower bone mineral density (BMD) in the lumbar spine ($P = 0.01$) and femoral neck ($P < 0.001$).

Conclusions:

PD is linked to lower serum 25(OH)D levels, reduced bone mineral density, and antioxidant deficiencies. These deficiencies are associated with increased falls, sleep issues, and mood disorders in post-menopausal women with PD.

Keywords

Parkinson's disease, vitamin D, post-menopausal women, non-motor symptoms

P-172

Therapeutic Benefits of Laughter Yoga, Deep Breathing Exercises, and Withania Somniferaroot Extract in Parkinson's Disease Patients

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Background & Objective:

This study aims to evaluate whether laughter yoga, deep breathing exercises, and Withania somnifera root extract (WSE) supplementation can improve motor and non-motor symptoms, stress levels, and glycemic control in Parkinson's disease (PD) patients with dementia.

Methods:

A cross-sectional study was conducted involving 110 PD patients (ages 50–75) diagnosed with dementia. Baseline assessments included age, family history of PD, physical activity, waist circumference, fasting glucose, insulin levels, glucose tolerance test (GTT), glycosylated hemoglobin (HbA1c), MRI, and CT scans. Patients participated in laughter yoga sessions consisting of 30-minute educational lectures followed by 30-minute intensive laughter and clapping exercises, five times per week for three months. Additionally, they received a daily 50g dose of WSE for one month. The Unified Parkinson's Disease Rating Scale (UPDRS III) was used to compare motor function changes between the treatment and control groups over three months. Non-motor symptoms, quality of life (PDQ-8), and stress levels were also assessed using a standardized Perceived Stress Scale.

Results:

Participants practicing laughter yoga and deep breathing, along with WSE supplementation, showed significant improvements in UPDRS III scores ($p < 0.001$), better glycemic control with reduced HbA1c levels, and enhanced MRI findings indicating increased mobility. Quality of life improved significantly in the treatment group compared to the control group ($p < 0.005$). Higher stress levels were linked to hyperglycemic complications in PD patients.

Conclusions:

This study highlights the therapeutic value of laughter yoga, deep breathing, and WSE supplementation in managing PD symptoms. These non-pharmacological interventions may delay disease progression, improve quality of life.

Keywords

Parkinson's disease, laughter yoga, Withania somnifera root

P-173

MicroRNA Profiles in Cerebrospinal Fluid Might Be Differential Markers in Parkinson's Disease

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Background & Objective:

Parkinson's disease (PD) remains difficult to diagnose early and to distinguish from atypical parkinsonian syndromes (APS). Current cerebrospinal fluid (CSF) protein biomarkers show limited specificity and sensitivity. MicroRNAs (miRNAs) are stable, regulatory molecules that may provide novel diagnostic insights.

Methods:

We profiled CSF miRNA expression in PD patients, healthy controls, and APS cohorts (multiple system atrophy, progressive supranuclear palsy, corticobasal syndrome) using microarray analysis. Subgroup comparisons were conducted for tremor-dominant (TD) and postural instability/gait disturbance (PIGD) PD phenotypes. Differential expression and pathway enrichment analyses were performed.

Results:

Compared with controls, PD showed significant upregulation of **hsa-miR-5787**, **hsa-miR-6750-5p**, and **hsa-miR-6125**, and downregulation of **hsa-miR-3613-3p** and **hsa-miR-877-5p**. TD patients exhibited unique increases in **miR-5000-5p** and **miR-455-3p**, while PIGD patients showed reductions in **miR-3665**, **miR-6087**, and **miR-6090**. Importantly, **hsa-miR-3921** and **hsa-miR-3613-3p** emerged as PD-specific markers not observed in APS groups. Functional analyses linked dysregulated miRNAs to autophagy, oxidative stress, and cytoskeletal regulation, processes central to PD pathogenesis.

Conclusions:

CSF-derived miRNA signatures distinguish PD from controls, reveal subtype-specific profiles, and differentiate PD from APS. These findings highlight **hsa-miR-3613-3p**, **hsa-miR-3921**, and **hsa-miR-6125** as promising biomarker candidates. Larger longitudinal studies are required to validate their clinical utility and clarify mechanistic roles in PD.

P-174

Effect of a Live-Streaming Tele-Exercise in Parkinson's Disease: A Multi-Center, Controlled Trial in South Korea

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Background & Objective:

Despite the benefits from exercise in Parkinson's disease (PD), there are limitations in accessibility to the exercise due to parkinsonian symptoms, and live-streaming, real-time tele-exercise may offer a practical alternative. This study evaluated the effects and feasibility of a regularly implemented live-streaming tele-exercise program in patients with PD.

Methods:

We recruited PD patients from 6 university-based hospitals. Participants were allocated to either a real-time tele-exercise intervention group for 12 weeks or an active control group who received a standardized exercise template for self-practice. We evaluated parkinsonian motor and non-motor symptoms, and quality of life at the baseline and after the intervention.

Results:

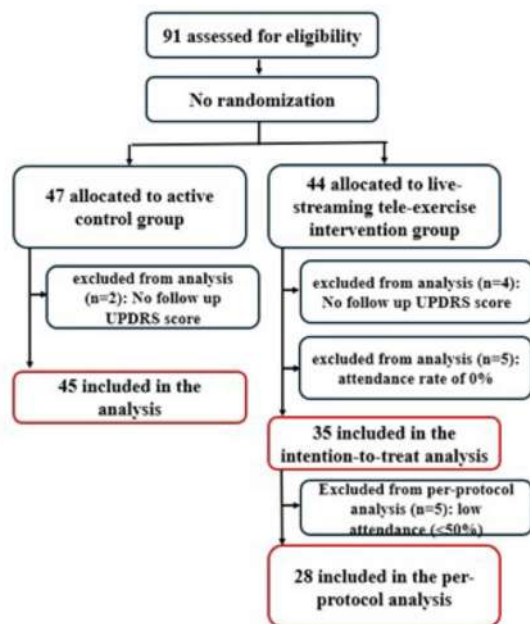
Of the 91 enrolled participants (intervention: n = 44; control: n = 47), 18 were excluded due to loss to follow-up or low attendance. In both groups, there were significant improvements in Parkinson's Disease Questionnaire-39 (PDQ-39), Non-Motor Symptoms Scale (NMSS), and International Physical Activity Questionnaire (IPAQ), while the improvements in Montreal

Cognitive Assessment (MoCA) and MDS-unified PD rating scale (MDS-UPDRS) Part III scores were detected in the intervention group. When we compared two groups, there were significantly greater improvements in MDS-UPDRS Part III (−3.7 points) and IPAQ sum (+763 points) in the intervention group ($p = 0.039$, $p = 0.046$, respectively) after adjusting age and sex.

Conclusions:

Live-stream tele-exercise enables home-based training for patients with PD, offering greater motivation than pre-recorded videos. Clinicians should provide appropriate exercise modalities and motivation, as these may directly enhance quality of life.

Figure



Table

Table 1. Baseline characteristics

	Active control control (N=45)	Intervention group (N=28)
Age, mean (SD)	65.5 (7.6)	68.6 (5.8)
Years of education, mean (SD)	12.9 (2.7)	12.1 (3.2)
Male sex, n (%)	23 (51)	12 (43)
Smoker, n (%)	3 (6.7)	0 (0)
Hypertension, n (%)	8 (17.8)	2 (7.1)
Diabetes mellitus, n (%)	4 (8.9)	1 (3.6)
Cardiovascular event, n (%)	3 (6.7)	0 (0)
Stroke, n (%)	1 (2.2)	0 (0)
Baseline score, mean (SD)		
H&Y stage		
1	15 (33.3)	5 (17.9)
2	27 (60.0)	22 (78.6)
3	3 (6.7)	1 (3.6)
PDQ-39	16.8 (3.0)	17.9 (2.2)
MoCA	26.3 (0.4)	26 (0.4)
NMSS	23.4 (21.1)	27.3 (18.4)
MDS-UPDRS 3 score	17.2 (8.6)	18.9 (8.6)
IPAQ		
IPAQ sum	1668.0 (1470.1)	1072.2 (968.8)
IPAQ category, n (%)		
Low	6 (13.3)	8 (28.6)
Moderate	31 (68.9)	17 (60.7)
High	8 (17.8)	3 (10.7)

Abbreviations: SD, standard deviation; MDS-UPDRS, Movement disorder society unified Parkinson's disease rating scale; H&Y, Hoehn and Yahr stage; PDQ-39, Parkinson Disease Questionnaire 39 Summary Index score; MoCA, Montreal Cognitive Assessment; NMSS, Non-motor symptoms scale; IPAQ, International Physical Activity Questionnaire

Table

Table 2. Between-group changes from baseline to 12 weeks in the intervention and control groups

Scores	Δ ACG (Change from baseline)	Δ IG (Change from baseline)	Δ IG - Δ ACG, mean (SE)	Δ ACG - Δ IG, 95% CI	P-value
H&Y stage	0.0	0.0	-0.06 (0.07)	-0.19 to 0.07	1.000
PDQ-39	-2.8	-3.6	-0.79 (1.65)	-4.02 to 2.45	0.278
MoCA	0.0	0.5	0.48 (0.32)	-0.14 to 1.10	1.000
NMSS	-3.8	-8.9	-5.12 (2.66)	-10.32 to 0.09	0.209
MDS-UPDRS 3 score	1.3	-2.4	-3.70 (1.23)	-6.11 to -1.30	0.039
IPAQ total score	468.5	1231.5	763.03 (376.60)	24.90 to 1501.15	0.046

Between-group comparisons of changes from baseline were analyzed using rank ANCOVA, with baseline score, age, and sex included as covariates. Values are presented as mean change from baseline (Δ), with standard error (SE) for the between-group difference. P-values represent the statistical significance of the group difference after adjustment. Statistical significance was set at $p < 0.05$. Abbreviations: IG, intervention group; ACG, active control group; SE, Standard error; CI, confidence intervals

Keywords

Parkinson's disease, Live-stream, Tele-exercise, MDS-UPDRS

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Epidemiological and Prognostic Characteristics of Parkinson's Disease by Age of Onset: Insights From the Korean National Sample Cohort

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Background & Objective:

The prognosis of Parkinson's disease (PD) is known to vary based on the age of onset. This study aimed to compare the epidemiological characteristics and long-term prognostic differences between Early-Onset PD (EOPD) and Late-Onset PD (LOPD) using data from the Korean National Sample Cohort.

Methods:

We analyzed data from the Korean National Sample Cohort from 2002 to 2019. The study population included patients with PD aged 20 years or older, who were categorized into EOPD and LOPD groups, alongside a 1:5 randomly matched control group of individuals without PD. We analyzed epidemiological trends, such as incidence and prevalence, and compared prognostic factors including Body Mass Index (BMI), the Charlson Comorbidity Index (CCI), smoking status, and household income.

Results:

The results revealed significant epidemiological changes between 2003 and 2018. Per 100,000 people, the incidence of PD increased from 11.36 to 26.10, while the prevalence rose sharply from 29.15 to 174.52. During this period, the proportion of EOPD among all PD patients decreased from 8.81% to 4.42%. In the 3-year follow-up, the LOPD group had a higher CCI at baseline than the EOPD group. Furthermore, both the BMI and smoking rate significantly decreased in the LOPD group and their controls, but not in the EOPD group. Notably, patients with EOPD experienced a significant decrease in household income three years after their diagnosis.

Conclusions:

In conclusion, the incidence and prevalence of PD in South Korea are increasing, while the proportion of EOPD is decreasing. Our findings indicate that LOPD patients present with a higher comorbidity burden and undergo more significant changes in health-related behaviors post-diagnosis, whereas EOPD patients are more vulnerable to adverse socioeconomic outcomes. These distinct prognostic pathways underscore the need for differenti-

ated management strategies tailored to the age of onset for PD patients.

Keywords

Parkinsonism, Epidemiology, Korea, Socioeconomic

P-176

The Amplification of Genetic Risk: How Modern Exposures Are Driving Parkinson's Disease Incidence

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Background & Objective:

The escalating burden of Parkinson's disease (PD) in developed countries is poorly understood. While polygenic risk scores (PRS) quantify genetic susceptibility, their interaction with evolving environmental factors, captured by birth cohort, and their effect across age groups remains unquantified at a population level.

Methods:

We conducted a systematic review and individual participant data meta-analysis following PRISMA-IPD guidelines. We pooled data from 24 studies (62,811 cases, 224,822 controls) to analyze the association of a standardized PD-PRS with PD risk. Stratified analyses were performed by age-of-onset groups (<50, 50-64, 65-79, ≥80 years) and birth cohort. Temporal trends were assessed via meta-regression of incidence rates from 55 population-based studies.

Results:

A one-standard-deviation increase in PRS was associated with an odds ratio (OR) of 2.41 (95% CI: 2.32-2.51, $p < 1 \times 10^{-100}$) for PD. This effect was non-linear, strongest in the top decile (OR=11.55, CI:10.80-12.35 vs. lowest decile), and significantly modified by age (p -interaction<0.001), with the strongest effect in the <50 years group (OR=2.92, CI:2.71-3.15) that attenuated with older age. Critically, the effect of PRS strengthened across successive birth cohorts (p -interaction=0.007), from an OR of 2.28 (CI:2.15-2.42) for the <1930 cohort to 2.72 (CI:2.54-2.91) for the ≥1960

cohort. Concurrently, incidence rates accelerated significantly after 2004 (AAPC increased from +0.8% to +2.1%).

Conclusions:

The genetic risk for PD is non-linear and most potent for early-onset cases. The strengthening association of PRS with PD risk in later birth cohorts provides compelling evidence for gene-environment interaction, suggesting modern exposures in developed nations are potentiating the biological pathways of genetic susceptibility. This underscores an urgent need to identify modifiable risk factors and justifies the use of PRS to stratify risk in future preventive trials.

Table

Characteristic	Overall (n=287,633)	Cases (n=62,811)	Controls (n=224,822)	Standardized Difference
Age, years (mean ± SD)	65.8 ± 12.1	69.3 ± 10.8	62.9 ± 12.3	0.38
Sex, Female (%)	141,211 (49.1%)	25,245 (40.2%)	115,966 (51.6%)	0.23
Genetic Ancestry, n (%)				
- European	248,205 (86.3%)	52,901 (84.2%)	195,304 (86.9%)	0.08
- East Asian	39,428 (13.7%)	9,910 (15.8%)	29,518 (13.1%)	0.08
Polygenic Risk Score (PRS), mean ± SD	0.00 ± 1.00	0.71 ± 0.93	-0.20 ± 0.97	0.97
PRS Decile, n (%)				
- Decile 1 (Lowest risk)	28,763 (10.0%)	1,885 (3.0%)	26,878 (12.0%)	
- Deciles 2-4	86,290 (30.0%)	8,537 (13.6%)	77,753 (34.6%)	
- Deciles 5-7	86,290 (30.0%)	15,174 (24.2%)	71,116 (31.6%)	
- Deciles 8-9	57,527 (20.0%)	17,258 (27.5%)	40,269 (17.9%)	
- Decile 10 (Highest risk)	28,763 (10.0%)	19,957 (31.8%)	8,806 (3.9%)	
Age at Diagnosis/Assessment, n (%)				
- <50 years (Early-Onset)	34,516 (12.0%)	4,517 (7.2%)	29,999 (13.3%)	
- 50-64 years (Mid-Onset)	89,166 (31.0%)	18,843 (30.0%)	70,323 (31.3%)	
- 65-79 years (Late-Onset)	126,568 (44.0%)	30,949 (49.3%)	95,619 (42.5%)	
- ≥80 years (Very-Late-Onset)	37,393 (13.0%)	8,502 (13.5%)	28,891 (12.9%)	

Table

PRS Decile	Odds Ratio (95% CI)	P-value	Absolute Risk (%)
1 (Lowest)	1.00 (Reference)		0.7%
2	1.52 (1.42–1.63)	<0.001	1.1%
3	1.98 (1.85–2.12)	<0.001	1.4%
4	2.45 (2.29–2.62)	<0.001	1.7%
5	3.05 (2.86–3.26)	<0.001	2.1%
6	3.88 (3.64–4.14)	<0.001	2.7%
7	4.85 (4.55–5.18)	<0.001	3.3%
8	6.25 (5.86–6.67)	<0.001	4.3%
9	8.10 (7.59–8.65)	<0.001	5.5%
10 (Highest)	11.55 (10.89–12.33)	<0.001	7.7%

Table

Birth Cohort	N (Cases / Controls)	OR per 1-SD PRS (95% CI)	P-value
<1930 (Reference)	12,501 / 58,244	2.28 (2.15–2.42)	<1×10 ⁻¹⁰⁰
1930–1944	18,845 / 72,110	2.39 (2.27–2.51)	<1×10 ⁻¹⁰⁰
1945–1959	21,450 / 65,331	2.51 (2.40–2.63)	<1×10 ⁻¹⁰⁰
≥1960	10,015 / 29,137	2.72 (2.54–2.91)	<1×10 ⁻¹⁰⁰

Keywords

Parkinson's Disease, Polygenic Risk Score, Genetic Epidemiology, Gene-Environment Interaction

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Caregiver Perception, Knowledge, and Attitude Toward Elderly with Parkinson Disease: Insights into Preventing Sarcopenia and Frailty

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Background & Objective:

The risk of sarcopenia in elderly with Parkinson's Disease (PD) is heavily influenced by caregivers. In Indonesia, care is provided by both informal family and formal paid caregivers. This study investigates how the effects of caregiver knowledge, attitude, and social capital on sarcopenia risk are moderated by caregiver type.

Methods:

A cross-sectional analysis was conducted using data from the Indonesia Family Life Survey (IFLS-5). The sample included elderly (≥60 years) with a PD diagnosis and an identified primary caregiver (n=312). Sarcopenia risk was defined as calf circumference <31 cm. Caregivers were classified as Informal Family or Formal. Multivariable logistic regression models were stratified by caregiver type to examine associations.

Results:

The drivers of sarcopenia risk were fundamentally different between groups. For ****Informal Family caregivers**** (n=194), a positive attitude (Adjusted Odds Ratio [AOR]=0.61; 95% CI: 0.42-0.89) and high social capital (AOR=0.65; 95% CI: 0.50-0.85) were significantly protective; knowledge was not. Conversely, for ****Formal caregivers**** (n=118), high knowledge was the primary protective factor (AOR=0.51; 95% CI: 0.31-0.84). Crucially, a significant interaction revealed this knowledge effect was dramatically enhanced by high social capital (AOR=0.58; 95% CI: 0.36-0.93), indicating formal caregivers require social integration to effectively apply their expertise.

Conclusions:

The mechanisms preventing sarcopenia are context-specific. Interventions must be tailored: supporting family caregivers through psychosocial and community-based strategies, and strengthening formal caregivers through clinical training coupled with efforts to integrate them into the household's social fabric. A universal approach will be less effective than one targeted to caregiver type.

Table

Characteristic	Overall (n=312)	Informal Family (n=194)	Formal (n=118)	p-value
Sarcopenia Risk (CC<31cm), %	46.8	51.5	39.0	0.028
Age, mean (SD)	73.1 (7.2)	73.8 (7.5)	72.0 (6.5)	0.032
Urban Residence, %	58.3	48.5	74.6	<0.001
Top HH Expenditure Quintile, %	38.5	28.9	54.2	<0.001
Caregiver Age, mean (SD)	47.3 (14.1)	54.8 (12.3)	35.1 (8.4)	<0.001
High Knowledge (p2), %	36.5	30.9	45.8	0.009
Positive Attitude (p4), %	65.4	72.2	54.2	0.002
Perception of Disease Severity (mean ADLs/IADLs)	3.7 (1.4)	3.9 (1.3)	3.3 (1.5)	0.014
Social Capital Index, mean (SD)	0.01 (1.0)	0.35 (0.9)	-0.52 (0.8)	<0.001

Table

Variable	Informal Family AOR (95% CI)	Formal AOR (95% CI)	p-diff
Caregiver Knowledge (High)	0.82 (0.60–1.12)	0.51 (0.31–0.84)*	0.048
Caregiver Attitude (Positive)	0.61 (0.42–0.89)*	0.92 (0.65–1.30)	0.078
Caregiver Perception (High Severity)	0.71 (0.54–0.93)*	0.65 (0.44–0.97)*	0.065
Knowledge × Social Capital	1.10 (0.80–1.52)	0.59 (0.36–0.93)*	0.010
Attitude × Social Capital	0.96 (0.74–1.25)	0.89 (0.63–1.26)	NS
Perception × Social Capital	0.92 (0.70–1.21)	0.84 (0.58–1.21)	NS
Elderly Age	1.08 (1.02–1.15)*	1.11 (1.04–1.19)*	NS
Elderly Female	2.95 (1.50–5.80)*	3.21 (1.40–7.38)*	NS
High HH Expenditure	**0.52 (0.30–0.90)**	1.15 (0.60–2.20)	0.018

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Determinants of Adherence and Validity in Parkinson's Disease Home Diaries: Insights from an Indonesian Pilot Study

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Background & Objective:

Motor complications occur in up to 50-70% of Parkinson's disease patients, impairing daily function and quality of life. Validated and reliable home diaries are needed, as hospital-based assessment is unable to monitor daily symptoms. This study aims to evaluate factors influencing adherence and validity of Parkinson's diary completion.

Methods:

This observational study at the Neurology Outpatient Clinic, Cipto Mangunkusumo Hospital, collected data on demographics, cognition (education level and MoCA-INA scores), and motor fluctuations (MDS-UPDRS). Participants completed a home-based Parkinson's Diary, which was compared with clinical assessments during hospital visits.

Results:

Fourteen patients with Parkinson's disease were enrolled. Female patients and those < 60 years were more likely to experience dyskinesia and OFF periods, with dyskinesia and OFF more

frequent in disease onset > 40 years. No significant differences in motor fluctuations were found across Parkinson's disease stages. Higher UPDRS scores were observed in patients with dyskinesia (M = 43.60 ± 23.28) and OFF periods (M = 50.86 ± 24.01) compared to those without. Incomplete Parkinson's Diaries were common in patients > 60 years and all patients with cognitive impairment. Lower education level (< 12 years) was significantly associated with incomplete diaries. Patients < 60 years, higher education, and normal MoCA-INA results tended to provide complete and UPDRS-consistent records.

Conclusions:

Diary adherence was better among patients with younger age, higher education, and preserved cognition, suggesting that both age and cognitive status may influence adherence and the validity of self-reported Parkinson's disease diaries. A larger sample size is warranted to enhance clinical significance and minimize potential bias.

Figure

Keywords

Determinants, Adherence, Validity, Parkinson's Disease, Diary

P-179

A Project and Practice Based Curriculum for Short-Term Training of Nurses in Parkinson's Care in Indonesia

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Background & Objective:

Parkinson's disease is a progressive neurodegenerative disorder with an increasing prevalence in Indonesia. According to WHO, in 2020 there were 8,492 deaths attributed to Parkinson's disease in Indonesia, with an age-adjusted mortality rate of 4.8 per 100,000 population. Another study estimated 146,236 Parkinson's cases in Indonesia in 2016, the highest prevalence in Southeast Asia. Nurses play a crucial role in timely medication administration, complication prevention, and patient-family education. However, local evidence shows that 62.4% of nurses have never received education on Parkinson's disease, and the majority only possess a "moderate" level of knowledge (Sari et al., 2022). This highlights the urgent need for a structured and practice-oriented training program.

Methods:

This study employs a Research and Development (R&D) approach, focusing on two main stages: needs analysis and curriculum design. The needs analysis was conducted through literature review and examination of existing studies on nurses' knowledge of Parkinson's disease in Indonesia. Based on these findings, a short-term project- and practice-based curriculum was designed to provide intensive yet applicable learning experiences for nurses.

Results:

The curriculum was structured for two weeks (60 hours), comprising 30% theoretical instruction and 70% practice. Core topics include: (1) assessment of motor and non-motor symptoms, (2) dopaminergic medication management with emphasis on timing, (3) therapeutic communication and family education, (4) fall prevention and complication management, (5) basic rehabilitation strategies, and (6) multidisciplinary case simulation. Practical components are complemented with group projects such as drafting a fall-prevention SOP, developing patient/caregiver educational media, and creating simple assistive device prototypes. Evaluation methods include pre-post tests, skills observation during simulation, and project output assessment.

Conclusions:

This project- and practice-based curriculum is expected to enhance nurses' knowledge, practical skills, and professional attitudes in caring for Parkinson's patients. Direct practice and project activities provide opportunities for nurses to generate applicable innovations for clinical settings.

Table

Project- and Practice-Based Curriculum for Short Training of Nurses in Parkinson's Care in Indonesia

This curriculum is designed as a short, intensive training program for nurses in Indonesia, focusing on project- and practice-based learning to improve knowledge, skills, and holistic care in Parkinson's disease management.

Week / Module	Topics & Activities	Learning Method	Project / Output	Duration
Week 1 - Clinical Foundations Module 1: Understanding Parkinson's	- Epidemiology & pathophysiology - Clinical motor & non-motor symptoms	Mini-lecture + symptom observation workshop	Checklist for Parkinson's symptom assessment	4 hrs
Week 1 - Clinical Foundations Module 2: Medication Management	- Levodopa and dopaminergic drugs - Importance of timing - Side effects & off-period handling	Simulation with medication timing Role-play for missed dose scenarios	Simple Medication Reminder System design (poster/app prototype)	6 hrs
Week 1 - Clinical Foundations Module 3: Communication & Family Education	- Therapeutic communication - Patient with dysarthria - Caregiver guidance	Role-play + practice teaching session	Patient/caregiver leaflet or video education tool	4 hrs
Week 2 - Applied Nursing Practice Module 4: Rehabilitation & Fall Prevention	- Basic physiotherapy and mobility training - Nutrition & aspiration prevention	Bedside practice + simulation	Draft SOP 'Fall Prevention in Parkinson's Patients'	6 hrs
Week 2 - Applied Nursing Practice Module 5: Multidisciplinary Case Simulation	- Collaboration with physician, physiotherapist, nutritionist	Group case study discussion + simulation	Comprehensive Holistic Care Plan for a simulated patient	6 hrs
Week 2 - Applied Nursing Practice Module 6: Final Project - Nursing Innovation	- Group project on innovation for Parkinson's care	Project-based learning + mentoring	Presentation of project (assistive tool prototype, educational media, SOP-model)	12 hrs

Evaluation Sheet

This sheet is designed to evaluate participants' performance throughout the training. Trainers can fill in based on observation, test results, and project outcomes.

Evaluation Component	Indicators	Score (1-5)	Comments
Knowledge	Pre-test & Post-test results; understanding of Parkinson's disease		
Skills	Clinical skills during OSCE and simulation (medication timing, mobility, communication)		
Project Output	Quality and practicality of project (SOP, tool, leaflet, care plan)		
Professional Attitude	Participation, teamwork, reflection journal submission		

Keywords

Parkinson's disease, nursing, curriculum

P-180

Precision RNAi Approaches to Silence SNCA and Inflammatory Mediators in Parkinson's Disease: A Bioinformatics Approach

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Background & Objective:

Parkinson's disease (PD) is a debilitating neurodegenerative disorder marked by progressive dopaminergic neuron loss, motor dysfunction, and non-motor symptoms. Accumulation of α -synuclein (SNCA) and neuroinflammation mediated by cytokines such as TNF- α and IL-1 β are key contributors to disease progression. RNA interference (RNAi) using small interfering RNAs (siRNAs) offers a promising approach to selectively silence pathogenic genes. This study aimed to design and evaluate siRNA candidates targeting SNCA, TNF- α , and IL-1 β using comprehensive bioinformatics analyses, providing a foundation for RNAi-based therapeutic strategies in PD.

Methods:

mRNA sequences of SNCA (NM_000345.4), TNF- α (NM_000594.4), and IL-1 β (NM_000576.3) were retrieved from GenBank. siRNA candidates were designed using DSIR with a score threshold >90 to ensure high predicted efficacy. The siRNA Scales tool was used to filter potential candidates. Secondary structures of the guide strands were analyzed using MaxExpect to assess folding free energy, while DuplexFold evaluated binding free energy between the guide strand and the target mRNA to predict binding stability and silencing potential.

Results:

Multiple high-potential siRNA candidates were identified, with DSIR scores of 103.9 for SNCA, 101.5 for TNF- α , and 101.5 for IL-1 β , exceeding the efficacy threshold. MaxExpect analysis indicated minimal self-structure formation in guide strands. DuplexFold analysis revealed strong binding free energies between guide strands and target mRNAs. Candidates also demonstrated favorable GC content, reducing off-target risks and enhancing thermodynamic stability.

Conclusions:

This bioinformatics study successfully designed potent siRNA candidates targeting SNCA, TNF- α , and IL-1 β , demonstrating favorable thermodynamic properties for potential gene silencing in Parkinson's disease. These candidates provide a basis for future in vitro and in vivo validation, paving the way for RNAi-based therapeutic strategies in neurodegenerative disorders.

Table

Table 1. Predicted siRNAs against SNCA gene

No.	Pos.	SS Sequence	AS Sequence	Score	Corrected OT Score	OT Pos
1	190	CGACGACAGUGUGUGAAAG	UUACACCCACACUGUGUGGAA	103.9	102.9	1
2	2692	GGAUUUAUUGUGGUAACAUAU	UUUGUAUCCACAAUAAUCCUU	103.5	77.5	4
3	2668	GGAAAGUUGGACCAUGAAU	UUCUAUGGUCACAAUUCUCA	102.9	76.9	4
4	2263	GAUAUUCUAGACUUGUAGC	UAGCAUUCUAGAAUUAUCUG	102.4	80.4	4
5	883	GCUCUAAUUCUACGAUGUAAA	UAAACUUCUGAGAAUUGAAGCC	102.2	94.2	4
6	3080	GCACUAGUGUGAGUAGCAAC	UUGCAUCUACUACACUAGUGC	101.7	71.7	4
7	686	GAUGUCCAUCCUGUACAAGU	UUGUACAGGAUUGAAACUUCU	101.6	95.6	4
8	1161	CACUUGUGUUUAUUAUAAU	UAUAUAUAAACAAGAGUGAA	101.5	90.5	4
9	1750	GGAAACUAGCAGUAGAAGA	UUUACACUGCUUAGUCCCG	100.7	83.7	4
10	2622	GGACAGAUACAGACUGAUGC	UAUACUCUGUAGUUCUGUCUC	100.6	74.6	4

Table 2. Predicted siRNAs against TNF gene

No.	Pos.	SS Sequence	AS Sequence	Score	Corrected OT Score	OT Pos
1	1543	GAUUAAGUGUUAACAAACUG	UUGUUUGUACACUUUAUUCAG	101.5	86.5	1
2	795	GACUCAGCGGUGAGAUCAUC	UUGAUUCUACGCGUGAGUUG	99.4	92.4	1
3	1204	GAGAAGACUACCAUGAAAU	UUCUAUGGUGAGGUCUUCUCA	97.7	85.7	1
4	404	GUCAGAUCAUUCUCCGAAC	UUCGGAGAAGAUUGUCUACUG	96.7	92.7	1
5	522	GGUUGAGGUGAGAGUAGAAC	UUAUCUCUACGUCACGCGA	95.1	90.1	1
6	436	GCCUUGAGCCCAUGUUGAGC	UAGACAUUUGGCAUACAGGCU	95.0	91.0	1
7	660	CCUACAGACCAAGGUCAACC	UUGACCUUUGUCUGUAGGAG	94.8	88.8	1
8	1003	AGCUUAGAGACUUAAGAACCA	UUGUUGCUUAAAGUCUCUAA	94.7	84.7	1
9	1082	GCAACCAUAGAGAUUACAAC	UUGAAUUCUAGUGGUGGCGA	94.3	84.3	1
10	990	GGGUGGAACCAAGCUGAUA	UAGCUUGGGUUGCAGCCUA	93.8	84.8	1

Table 3. Predicted siRNAs against IL1b gene

No.	Pos.	SS Sequence	AS Sequence	Score	Corrected OT Score	OT Pos
1	645	GGUUGUAGAAAGAUAAAGC	UUUUAUUCUACACGCGAG	101.5	95.5	1
2	741	AGAUAGAAUCAAUAAACAGC	UUGUUAUUGAUUUAUUCUUG	101.2	94.2	1
3	723	AGGCAUUGUUCUACAAGA	UUCUUGUAGAAACAAGCCU	100.5	93.5	1
4	1452	CAAUUUAUUAUUGUUGAUG	UUGACAGAUAGAUUAUUG	100.4	85.5	1
5	738	ACAAGAUAGAAUCAAACAA	UUGUUGUUAUUAUUCUUGU	99.2	92.2	1
6	430	CGAUGACCCUGUACGACACU	UGAGUGGACAGGUGCGAUGU	98.7	94.7	1
7	1465	GUUCAUGGUGUUAAGAAUA	UUUAUUAACAGAACAUUGAAC	97.3	83.3	1
8	725	CGAUUUGUUCUACAAGAUUA	UAUUAUAGAAACAAGUCCU	97.0	90.0	1
9	735	UCAACAAGAUAGAAUUAUA	UAUUAUUAUUAUUGUAGAA	96.7	89.7	1
10	878	CAAUUUGUUCUUCUAAAGA	UUUUAAGGAAGACACAAUUGCA	96.3	88.3	1

Keywords

siRNA, Parkinson's disease, SNCA, TNF- α , IL-1 β

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An Analysis of the Role of Domestic General Government Health Expenditure, External Health Expenditure, and Out-of-Pocket on the Death of Parkinson's Disease in ASEAN-5.

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Background & Objective:

Parkinson's disease shows a significant trend of increasing prevalence in Asia, including Southeast Asia, due to aging populations and increasing awareness. This study aims to identify the interrelationships between government, the private sector, and individual awareness of the death of Parkinson's Disease in ASEAN-5.

Methods:

The methods used in this study are fixed effects and random effects models. The data used are panel data, combining data series from 2000 to 2021 with cross-sectional data from ASEAN-5 (Indonesia, Malaysia, Thailand, Singapore, and the Philippines). The data are sourced from the World Health Organization and IHME. The independent variables used were Domestic General Government Health Expenditure (DGGH-E), External Health Expenditure (EHE), and Out-of-Pocket Expenditure, with the dependent variable being the Death of Parkinson's Disease.

Results:

The results showed that the best model from the Hausman test was the random effect model, as the sig value was more than 0.05. The random effect model or fixed effect model showed that the variables EHE and Out-of-Pocket significantly and negatively reduced the risk of the Death of Parkinson's Disease in ASEAN, as indicated by p value less than 0.05 or t table (2.16) less than t statistics (t statistics = -2.21 & -2.19). But, DGGH-E, is significant and positive to the Death of Parkinson's Disease. The result that significantly reduced the death of Parkinson's Disease was

external expenditure, marked by the largest coefficient value among the other independent variables at -0.000192%, followed at -0.278%, out-of-pocket expenditure as a percentage of current health expenditure at -0.000279%.

Conclusions:

Therefore, government efforts are needed to collaborate with external parties to provide health insurance to support public health, increase government but must be on target, and increase community income to increase out-of-pocket expenditure and reduce the death of Parkinson's disease in ASEAN.

Figure

	(fixed) Death parkinson	(random) Death parkinson
outofpocket	-0.0000346** (-2.68)	-0.0000279* (-2.21)
externalexpen diture	-0.000148 (-1.74)	-0.000192* (-2.19)
governmentex penditure	0.000296*** (6.45)	0.000338*** (7.20)
_cons	0.00455*** (5.12)	0.00393*** (4.45)
R-squared	0.760	
<i>t</i> statistics in parentheses		
* $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$		

Keywords

Government Expenditure, External Expenditure, Out of Pocket, Parkinsons, ASEAN

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Network Pharmacology-Guided Docking and MD Simulation of LRRK2 for Novel Therapeutic Discovery in Parkinson's Disease

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Background & Objective:

Parkinson's disease (PD) is a progressive neurodegenerative disorder characterized by protein misfolding, mitochondrial

dysfunction, and the destruction of dopaminergic neurons. Leucine-rich repeat kinase 2 (LRRK2) is an appealing target for pharmacological intervention because of its pivotal involvement in neuronal function and α -synuclein disease.

Methods:

The current study employs network pharmacology, molecular docking, and molecular dynamics (MD) modeling to thoroughly evaluate and confirm LRRK2 modulators. We got PD-related genes from OMIM, DisGeNET, and GeneCards, and we got candidate bioactive compounds from SwissTargetPrediction, TCM-SP, and DrugBank. STRING was used to create protein-protein interaction networks, and pathway enrichment analysis (KEGG, GO) confirmed that LRRK2 is a central target for synaptic signaling and autophagy control. The crystal structure of LRRK2 (PDB ID: 6VP6) was determined for docking analysis. Using AutoDock Vina, we virtually screened the natural and synthetic compounds that made the short list. We then chose the best-ranked ligands with a high binding affinity for the kinase domain. We used 100 ns MD simulation (GROMACS) to look at the conformational stability, intermolecular hydrogen bonding, and binding free energy (MM-PBSA) of the lead complexes.

Results:

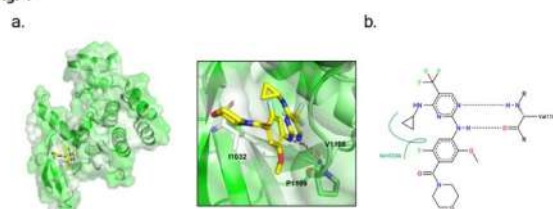
The findings indicate that numerous flavonoid derivatives demonstrate significant and stable interactions with the ATP-binding region of LRRK2, suggesting potential for the control of kinase activity associated with PD development.

Conclusions:

This comprehensive bioinformatics approach facilitates the utilization of network pharmacology, molecular docking, and molecular dynamics modeling to identify disease-modifying lead candidates for Parkinson's disease. The publication identifies LRRK2 as a verified computational target and offers a cost-effective approach to rational drug discovery in neurodegeneration.

Figure

Fig. 4



3D (a) and 2D (b) representation of the interaction between LRRK2 and its co-crystallized inhibitor. LRRK2 is represented from White (hydrophobic) to green (hydrophilic) using the Eisenberg hydrophobicity scale.

Keywords

Parkinson's disease, Leucine-rich repeat kinase 2, Molecular docking, Molecular dynamics (MD) simulation, Network pharmacology

P-183

Fabrication, Characterization of Ganodric Acid-Dextran Nanoformulation Against Parkinson's Disease-Induced Functional Damage in Rats via Inhibition of Caspase-3/Interleukins-6 and 1 β

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Background & Objective:

Parkinson's disease (PD) is indeed the second most commonly diagnosed neurodegenerative disorder, following Alzheimer's disease. Parkinson's disease is characterized by a progressive loss of dopamine-producing neurons in the brain, leading to motor symptoms such as tremors, bradykinesia (slowness of movement), rigidity, and postural instability. The current study was to fabricate the Ganodric acid-Dextran nano-formulation (GANF) and scrutinized the neuroprotective effect against the PD in a rat model and scrutinized the possible mechanism.

Methods:

6-hydroxydopamine (5 μ L) was administered for induction the PD in the rats and rats were received the oral administration of ganodric acid and GANF. Estimated the neurotransmitters, inflammatory cytokines like TNF- α , IL-1 β , IL-6, TGF- β ; inflammatory parameters such as COX-2, PGE2, iNOS, NF- κ B; antioxidant parameters viz., SOD, CAT, MDA, GSH; apoptosis parameters like Bax, Bcl-2, caspase-3, respectively. We also estimated the mRNA expression.

Results:

Both ganodric acid and GANF significantly ($P < 0.001$) suppressed the PD induced high turning values and also decreased the level of acetyl choline (ACh) and acetylcholinesterase (AChE). GANF altered the level of neurotransmitters (HVA, dopamine, 5-HT, 5-HIAA and DOPAC); inflammatory cytokines such as TNF- α , IL-1 β , IL-6, TGF- β ; antioxidant parameters such as MDA, GSH, CAT, GPx, CAT; inflammatory parameters like COX-2, PGE2, iNOS, NF- κ B; apoptosis parameters viz., Bax, Bcl-2 and caspase-3, respectively. GANF rats effectively altered the expression of TNF- α , IL-1 β , NF- κ B, COX-2, p53, Bax, caspase-3 and caspase-9.

Conclusions:

We can conclude that GANF exhibited the protective effect against the Parkinson disease via Inhibition of Caspase-3/Interleukins-6 and 1 β .

P-184

Neuro-Modulatory Activity of Naringenin in Rotenone-Induced Neurotoxicity in Mice Model of Parkinson's Disease via Suppressing NF- κ B Pathway

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Background & Objective:

Parkinson diseases is age related neurodegenerative diseases in which dopaminergic neurons get destructs and accumulation of protein (α -synuclein) in the substantia nigra pars compacta region of brain. It is well accounted in the literature that due to production of pro-inflammatory cytokines, deterioration of DA nigrostriatal pathway occurred and subjected to inflammation-induced oxidative stress and neurotoxicity. The current study was design to explore the action of polyphenolic compound Naringenin in rotenone-induced neurotoxicity of Parkinson's disease in animals.

Methods:

All the mice were randomly divided into five groups and treated with Naringenin at the dose level of 25, 50 and 100 mg/kg b.w after the intoxication of rotenone. All the experimental mice after administration of test drugs were subjected to behavioural tasks to evaluate the motor deficit. Mice were euthanized and dissect out the brain for further study (biochemical parameter, immunochemical staining to determine the expression of inflammatory markers).

Results:

Motor impairment were improved after the treatment of Naringenin through behavioural analysis. Reduced activation of NF- κ B pathway which suggested downregulation in pro-inflammatory cytokines [{TNF- α (56.5%)}, {IL-1 β (54.4%)}, {IL-6 (64.5%)}] production and upregulation in anti-inflammatory cytokines in Nar-

ingenin supplemented groups. Administration of Naringenin also dose dependently reversed the levels of Biochemical parameter such as antioxidant enzymes cytokines [{SOD (58.6%)}, {CAT (45.5%)}, {MDA (69.4%)}] in drugs treated groups. Naringenin supplementation also secured the degeneration of DA neurons in substantia nigra pars compacta.

Conclusions:

This study speculates naringenin accredited to antioxidant and anti-neuroinflammatory properties against rotenone-induced neurotoxicity via reduced activation of NF-κB pathway and a prominent drug in the protection of PD.

Keywords

Parkinson's disease, Naringenin, Inflammation, Antioxidant, motor deficit

P-185

Anti-H. pylori IgG Seropositivity and Its Clinical Relevance in Parkinson's Disease

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Background & Objective:

Helicobacter pylori (H. pylori) infection has been implicated in Parkinson's disease (PD) and levodopa absorption, but studies on H. pylori infection history (anti-H. pylori IgG seropositivity) have shown conflicting results across populations. We investigated (1) whether anti-H. pylori IgG seropositivity differs between PD and controls, and (2) whether it is associated with clinical features including levodopa equivalent daily dose (LEDD).

Methods:

Participants were recruited prospectively from Hallym University Sacred Heart Hospital and Hallym University Chuncheon Sacred Heart Hospital between June 2024 and June 2025. A total of 123 PD patients and 671 controls were included, and age- and

sex-matching was performed (Table 1). Anti-H. pylori IgG was measured, and clinical features of PD were compared between seropositive and seronegative patients (Tables 2–3).

Results:

No significant difference in anti-H. pylori IgG seropositivity was observed between PD patients and matched controls (45.5% vs 51.2%, $p=0.925$). Within PD, age, disease duration, H&Y stage, and MDS-UPDRS part III did not differ by serostatus (Table 2). In treated PD patients, the LEDD was numerically higher in seropositive patients than seronegative patients (777.98 mg vs 651.59 mg), though this difference was not statistically significant ($p=0.168$, Table 3).

Conclusions:

This is the first Korean population-based study to compare anti-H. pylori IgG seropositivity between PD patients and controls. Seropositivity was not different between PD and controls and showed no clinical impact, including LEDD. While prior studies suggested possible effects of H. pylori on levodopa absorption, results remain controversial and may reflect regional or ethnic variability. As anti-H. pylori IgG does not perfectly represent active infection, further studies using urea breath test or rapid urease test are warranted.

Table

Table 1. Comparison of Anti-H. pylori IgG Seropositivity Between Age and Sex Matched Controls and PD Patients

	Control (n=123)	PD (n=123)	p value
Age (years)	69.3 ± 10.0	69.4 ± 10.1	0.939
Sex (female)	67 (54.5%)	67 (54.5%)	1.000
Anti-H.pylori IgG			
positive	63 (51.2%)	56 (45.5%)	0.925
negative	60 (48.8%)	67 (54.5%)	

Table

Table 2. Clinical Characteristics of PD Patients According to Anti-H. pylori IgG Seropositivity

	IgG (-) PD (n=56)	IgG (+) PD (n=66)	p value
Age (years)	69.98 ± 11.61	68.84 ± 8.65	0.486
Sex (female)	32 (57.1%)	34 (51.5%)	0.101
Disease duration (years)	3.04 ± 2.71	3.86 ± 4.73	0.308
H&Y stage	1.93 ± 0.82	2.15 ± 0.91	0.205
MDS-UPDRS part III	24.27 ± 9.48	24.71 ± 4.95	0.880

Table

Table 3. Comparison of Clinical Features and LEDD in Treated PD Patients According to Anti-H. pylori IgG Seropositivity

	IgG (-) treated PD (n=33)	IgG (+) treated PD (n=40)	p value
Age (years)	71.03 ± 8.90	68.30 ± 8.63	0.191
Sex (female)	21 (63.6%)	19 (47.5%)	0.168
Disease duration (years)	4.38 ± 2.49	5.62 ± 5.33	0.301
H&Y stage	1.93 ± 0.80	2.20 ± 0.96	0.223
MDS-UPDRS part III	20.61 ± 8.56	22.30 ± 8.93	0.584
LEDD (mg)	651.59 ± 264.21	777.98 ± 433.57	0.168

Keywords

Parkinson's disease, Helicobacter pylori, Korean population, Seropositivity

P-186

Mapping Two Decades of Global Research on Mesenchymal Stem Cell Therapeutic Strategy for Parkinson's Disease: A Bibliometric and Network Analysis (2000–2025)

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Background & Objective:

Parkinson's disease (PD) is recognized as one of the most common neurodegenerative disorders worldwide. Primary pathological mechanisms of PD involve excessive generation of reactive oxygen species leading to apoptosis and neuroinflammation. In recent years, mesenchymal stem cells (MSCs) have been proposed as a prospective therapeutic alternative with notable competence in proliferative systems and minimal adverse effects. The aim of this study is to advance the global intellectual trajectory of MSC research for PD over the past two decades, focusing on shifting themes, countries, citations, and years. This approach highlights emerging domains and underexplored areas that signify critical research gaps.

Methods:

This study incorporated an array of methods using 1590 articles from 2000-2025 extracted from Scopus database, analysed using

the 'Bibliometrix' package in R for co-occurrence and thematic map, in addition country level density visualization generated using VOSviewer software version 1.6.19.

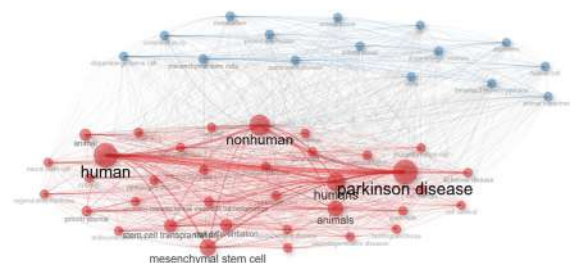
Results:

The co-occurrence network between 2000 and 2025 indicated the first cluster appeared to center around Parkinson's disease and its relevance to both human and nonhuman studies, while the second cluster was oriented towards "animal models," and "metabolism" related research. Publications on related keywords peaking in 2024 with 128 papers. The "Parkinson disease" cluster's position as a basic theme suggested that research in this domain was highly specialized and developed. The United States, followed by China, led global contributions on this topic. The topic trend developed from adoptive immunotherapy in the early 2000s, and by 2024, research directions had become more specific towards "adipose-derived stem cells" and "pharmaceutics."

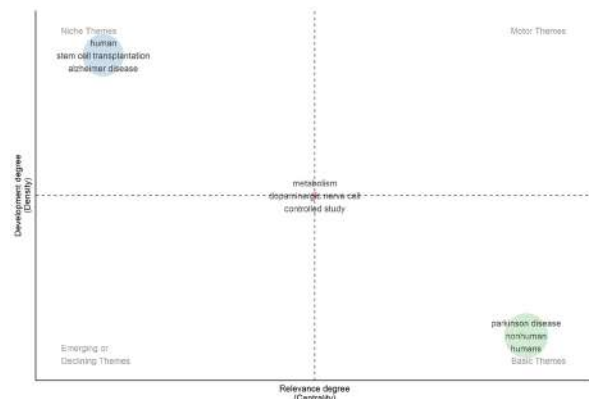
Conclusions:

This bibliometric study network provides a valuable overview of the research landscape related to Parkinson's disease and stem cell therapies. It highlights the key themes, relationships, and potential areas for further investigation.

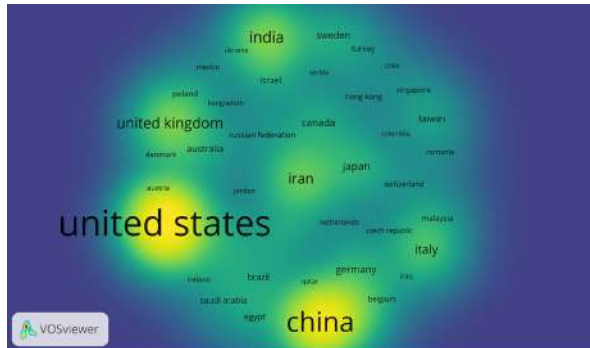
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Figure



Figure



Keywords

Parkinson's disease, Mesenchymal stem cells, Movement disorder, Stem cell-based

P-187

Effects of Music and Rhythmic Auditory Stimulation Interventions on Motor Function in Parkinson's Disease: A Meta-analysis of Randomized Controlled Trials

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Background & Objective:

Non-pharmacological music-based interventions include rhythmic auditory stimulation (RAS) and music-based movement therapy (MMT). Evidence regarding aromatherapy on PD motor outcomes is limited. This study conducted a meta-analysis of RCTs reporting motor outcomes (UPDRS-III) and gait velocity in PD patients.

Methods:

The literature search focused on open-access RCTs of music/RAS in PD (PubMed/PMC/Frontiers/JNER/PLoS). Numerical data (mean $\pm$ SD post-intervention and sample size per group) were extracted from available full-text articles. For gait velocity, the pooled mean difference (MD, m/s) was used with a fixed-effects model when the study size was small; for the UPDRS-III, the pooled MD was used using a random-effects model (DerSimonian-Laird). Heterogeneity was assessed using Q and I^2 .

Results:

The result show that there are three studies reported post-intervention UPDRS-III scores ($k = 3$) and two studies reported post-intervention gait velocity ($k = 2$). Gait velocity (m/s): pooled MD = +0.11 m/s (95% CI 0.062 to 0.157, $k = 2$, $I^2 = 0\%$), indicating an increase in gait speed in the music/RAS group compared to the control. A change of ~ 0.10 m/s is generally considered clinically significant in the neurological/elderly population. UPDRS-III (motor): pooled MD = -3.04 points (95% CI -5.87 to -0.22, $k = 3$, $I^2 = 0\%$), indicating a decrease in motor scores (improvement) in the intervention group. A reduction of approximately 3 points could potentially approach or meet the reported MCID threshold (~ 2.5 –5 points), but interpretation should be cautious due to the relatively small number of studies.

Conclusions:

Preliminary evidence from open-access RCTs suggests that music/RAS-based interventions are associated with improvements in motor function in PD, including increased gait speed (~ 0.11 m/s) and decreased UPDRS-III scores (~ 3 points). However, these results need to be confirmed by a more comprehensive meta-analysis to strengthen the effect estimates.

Keywords

Parkinson disease, music therapy, rhythmic auditory stimulation, gait speed, UPDRS

P-188

Moyamoya Disease and the Risk of Parkinson's Disease: A Mendelian Randomization Analysis

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Background & Objective:

Research on Moyamoya disease (MMD) and the risk of Parkinson's disease (PD) using the Korean National Health Insurance Service database suggests a potential link through interactions between α -synuclein and RNF213, as evidenced by in vitro and postmortem studies. This study investigates the causal relationship between MMD and PD using Mendelian randomization (MR) analysis.

Methods:

We selected single-nucleotide polymorphisms (SNPs) as exposure from the Korean genome-wide association study (GWAS) involving 216 MMD patients and 296 controls, and SNPs as outcome from the Korean GWAS for PD (1,036 cases and 1,208 controls). Two-sample MR analyses were conducted to estimate the associations between MMD and PD.

Results:

The inverse variance weighted method indicated no significant association between MMD and PD (odds ratio = 1.0056, 95% confidence interval = 0.9847-1.0269; $p = 0.603$), consistent with the results from MR Egger, weighted median, and weighted mode analyses.

Conclusions:

These findings suggest no evidence of a causal relationship between MMD and PD based on the Korean GWAS data. Further larger cohort studies and MR analyses are needed to explore potential causal relationships.

Keywords

Moyamoya Disease, Parkinson's Disease, Mendelian Randomization, Genome-Wide Association Study, Causal Relationship

P-189

Gut Microbiota Alterations in Parkinson's Disease and REM Sleep Behavior Disorder

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Background & Objective:

Gut microbiota is increasingly implicated in Parkinson's disease (PD) and prodromal conditions such as REM sleep behavior disorder (RBD). We aimed to characterize microbial diversity and taxonomic differences among PD, RBD, and healthy controls (HC).

Methods:

Fecal microbiota from PD ($n=54$), RBD ($n=10$), and HC ($n=31$) were analyzed. Alpha diversity and beta diversity (Unweighted UniFrac) were assessed. Differential abundance testing was performed at genus and phylum levels.

Results:

Alpha diversity showed higher Richness in PD than HC ($p=0.008$). Beta diversity revealed different trends between PD and HC (adjusted $p=0.076$), suggesting community structure shifts. Genus-level analysis showed *Methanobrevibacter*, *Enterobacter*, *Lentihominibacter*, *Coprobacillus*, and *Mogibacterium* were higher in PD/RBD compared with HC. At the phylum level, *Methanobacteriota* were increased in PD/RBD, while *Ascomycota* (fungi) decreased in PD/RBD compared to HC. HC samples tended to harbor more eukaryotic fungi, whereas PD showed higher archaeal abundance.

Conclusions:

PD and RBD patients exhibit altered gut microbial communities with increased methanogenic archaea and reduced fungi, alongside enrichment of specific genera. These findings support gut dysbiosis as a feature of PD pathogenesis and suggest microbial signatures that may distinguish prodromal and clinical PD.

Keywords

Parkinson's disease, gut microbiome

Parkinsonism (Secondary and Parkinsonism-plus)

P-190

Association of Choroid Plexus Volume With Brain Atrophy and Glucose Metabolism in Multiple System Atrophy

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Background & Objective:

The choroid plexus (CP) is a highly vascularized structure critical for cerebrospinal fluid (CSF) production and brain waste clearance via the glymphatic system. While previous studies have reported CP volume (CPV) enlargement in neurodegenerative diseases such as Alzheimer's disease (AD) and Parkinson's disease (PD), structural changes in the CP in multiple system atrophy (MSA) remain poorly understood. We aimed to investigate CPV in MSA and explore its associations with disease severity, brain atrophy, and glucose metabolism.

Methods:

We retrospectively analyzed 87 patients with clinically established or probable MSA and 84 age-matched healthy controls who underwent 3T brain MRI and 18F FDG-PET. Volumetric analyses were conducted using FastSurfer for automated segmentation of CPV. Multivariate linear regression analyses were performed to assess the relationship between CPV and clinical severity (Unified MSA Rating Scale; UMSARS), regional brain atrophy, and FDG uptake, adjusting for confounding factors.

Results:

CPV was significantly smaller in MSA patients compared to controls (1.00 ± 0.27 vs. 1.30 ± 0.26 , $P < 0.001$). In contrast to findings in AD and PD, this CP atrophy was consistently observed in both MSA-C and MSA-P subtypes. CPV showed no significant association with UMSARS scores. However, smaller CPV was significantly correlated with more severe regional brain atrophy, particularly in the cerebellar hemispheres and midline vermis, and with reduced glucose metabolism in the brainstem, cerebellum, thalamus, and ventral diencephalon. These associations were more robust in patients with the cerebellar subtype of MSA.

Conclusions:

This study demonstrates that CP atrophy is a characteristic neuroimaging feature of MSA and contrasts with the CP enlargement observed in other neurodegenerative conditions, highlighting CP as a novel site of structural involvement in MSA and underscore its relevance in disease pathophysiology.

Keywords

choroid plexus, multiple system atrophy

P-191

Plasma Inflammatory Proteins Linked to Disease Severity and Longitudinal Progression in Progressive Supranuclear Palsy: A Prospective Cohort Study

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Background & Objective:

Although neuroinflammation is increasingly recognized as a key driver of progressive supranuclear palsy (PSP), the role of blood inflammatory markers remains unrevealed. In this prospective cohort study, we aimed to characterize plasma inflammatory protein profiles in PSP patients and to assess their associations with clinical severity and one-year longitudinal progression, neurodegeneration biomarkers, and brain morphometric changes.

Methods:

We included 71 probable PSP patients and 30 age-matched healthy controls. Principal component (PC) analysis of plasma 39 inflammatory proteins were applied to derive composite inflammatory profiles. All patients underwent baseline assessments, including PSP-Rating Scale (PSPRS), global cognitive function tests, and volumetric MRI. Blood biomarkers, including neurofilament light chain (NfL), phosphorylated tau 231, sTREM2, and glial fibrillary acidic protein, were also measured. Follow-up PSPRS assessments were available for 56 patients at 6 months and 45 at 12 months.

Results:

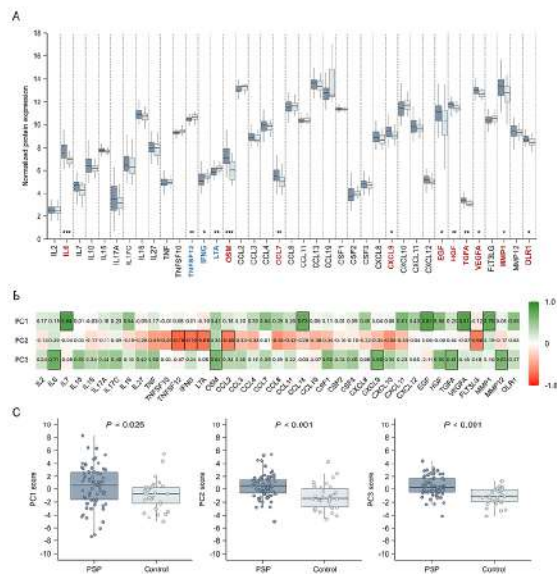
PC analysis identified a three-component solution explaining 55.6% of the variance, and scores of each component were elevated in the PSP group compared to controls. PC1, mainly driven by interleukin (IL)-7, vascular endothelial growth factor A, and pro-epidermal growth factor, was associated with greater clinical severity, higher soluble TREM2 levels, and more pronounced atrophy in the cerebellum, middle temporal gyrus, and limbic areas. PC3, mainly driven by IL-6, C-X-C motif chemokine 9, and

oncostatin-M, was associated with greater clinical severity and faster disease progression, higher NfL levels, and more extensive atrophy in the precuneus, inferior frontal gyrus, caudate, and, to a lesser extent, brainstem regions. PC2 showed no associations with baseline and longitudinal outcomes.

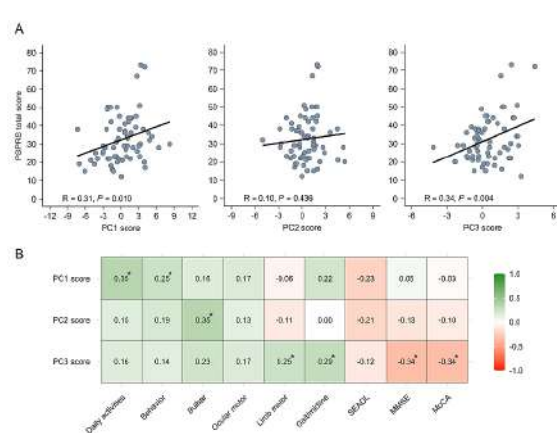
Conclusions:

These observations provide mechanistic and clinical insights into the potential contribution of peripheral inflammation to the neurodegenerative processes underlying PSP.

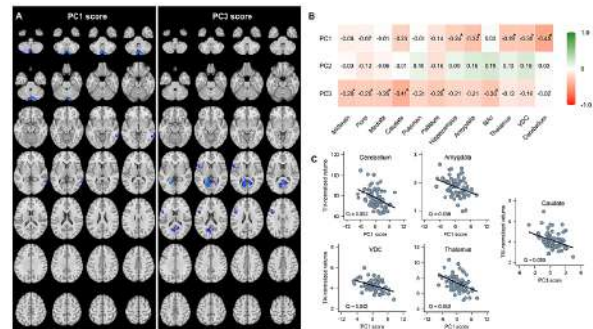
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P-192

Estimating the Progression of Multiple System Atrophy in a Single-Center Cohort

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Background & Objective:

Previous studies have proposed progression rate and model in multiple system atrophy (MSA). Our objective was to evaluate progression of MSA and to establish a simple predictive model.

Methods:

Patients were retrospectively identified from the Seoul National University Hospital Movement Disorder Center MSA cohort. Progression was assessed with the Unified Multiple System Atrophy Rating Scale (UMSARS). At the time of the first UMSARS evaluation, clinical variables included REM sleep behavior disorder (confirmed by polysomnography or determined clinically with the RBDSQ), presence of the hot cross-bun sign, urinary retention, urinary urge incontinence, and orthostatic hypotension. A logarithmic model was constructed in patients with three serial UMSARS assessments and tested for performance in those with only two

Results:

Among patients with UMSARS assessments within three years, 77 had 1-year follow-up data and 39 had 2-year follow-up data. The mean change in UMSARS scores was 11.87 ± 11.75 from baseline to 1 year and 8.22 ± 10.64 from 1 to 2 years, indicating substantial inter-individual variability. The logarithmic model predicted UMSARS Part I + Part II total scores with an R^2

of 0.679 and a mean absolute error of 4.86. Gaussian mixture modeling applied to the 39 patients based on annual UMSARS score changes identified three subgroups: (1) similar progression between years 1–2, (2) greater increase during year 1, and (3) greater increase during year 2. However, clinical characteristics did not differ significantly across these subgroups.

Conclusions:

Our study demonstrated that the progression rate in our cohort was comparable to those reported in other cohorts. The logarithmic model achieved moderate predictive performance at the individual level. Also, no significant clinical differences were observed among the identified subgroups, suggesting that future research should explore additional biomarkers to better predict disease progression in MSA.

Keywords

MSA, UMSARS

P-193

Levodopa Responsiveness in Vascular Parkinsonism: A Within-Subject Pre–Post Analysis of UPDRS Part III and Timed Up-and-Go

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Background & Objective:

Background Vascular parkinsonism is rare disorder characterized by symmetric bradykinesia, and gait disturbance. It has generally been considered to respond poorly or not at all to levodopa treatment. Although several studies have suggested adjunctive therapeutic options for vascular parkinsonism, the efficacy of levodopa has not been objectively demonstrated. In this study, we aimed to evaluated levodopa responsiveness in patients with vascular parkinsonism

Methods:

In this study, we conducted a single-center, within-subject pre–post analysis in patients with vascular parkinsonism at Chung-Ang university hospital. We analyzed baseline demographics and evaluated the effect of levodopa by measuring Unified Parkinson's Disease Rating Scales (UPDRS) part III and Timed Up-and-Go (TUG) scores before and after administration. Statistical testing used one-sided Wilcoxon signed-rank tests (alternative: post < pre) with Holm correction fortwo results.

Results:

We enrolled 22 patients with vascular parkinsonism, including 12 female (54.5%), with mean age of 77.8 ± 7.6 years and mean Levodopa equivalent daily dose (LEDD) of 363.6 ± 169.7 mg/day. The Wilcoxon signed-rank test revealed that there was significant improvement in UPDRS part III after levodopa administration ($p < 0.001$). The median change -8.0 points (bootstrap median 95% CI -12.5 to -3.0), and the mean change -9.73 (95% CI -13.68 to -6.23). The TUG ($n=10$) also improved significantly after levodopa, with a median change of -4.5 s (bootstrap 95% CI -6.5 to -2.0) and a mean change of -4.50 s (95% CI -6.00 to -3.20) ($p = 0.003$). Both results remained significant after Holm correction.

Conclusions:

In this study, we found that levodopa was associated with improvements in motor severity and mobility among patients with VP. Although VP is often views as poorly responsive to levodopa, our results suggest measurable benefit in patients with VP. Further research is required to apply this to the clinical practice.

Table

Table 1. Clinical characteristics of patients with vascular parkinsonism.

	Mean(SD)
Age (year)	77.8 ± 7.6
Sex (female : male)(%)	54.5 : 45.5
LEDD (mg/day)	363.6 ± 169.7
Pre UPDRS partIII	31.0 ± 11.1
Post UPDRS partIII	21.3 ± 10.1
Pre TUG (sec)	20.3 ± 5.2
Post TUG (sec)	15.8 ± 3.5

SD; Standard deviation, LEDD; levodopa equivalent daily dose, UPDRS; Unified Parkinson's Disease Rating Scales, TUG; Timed Up-and-Go

Table 2. Within-subject levodopa response ($\Delta = \text{Post} - \text{Pre}$)

	Δ Median	95% CI	Δ Mean	95% CI	p value	p value (Holm-adjusted)
UPDRS III	-8.0	-12.5 to -3.0	-9.7	-13.7 to -6.3	< 0.001	< 0.001
TUG	-4.5	-6.5 to -2.0	-4.5	-6.0 to -3.1	0.003	< 0.001

Keywords

vascular parkinsonism, levodopa, UPDRS, TUG

P-194

Case Series of Parkinson Plus Syndromes

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Introduction:

Parkinson plus syndrome may pose a diagnostic challenge especially in centres without movement disorder specialist, however keen observation and astute clinical examination provide clinical diagnosis.

Case:

78yr old male presented with recurrent falls and difficulty ambulating for 6months. He also is having urinary incontinence for 2weeks. Physical examination showed bradykinesia of both upper limbs with magnetic gait. His MMSE (mini-mental status examination score) was 24. He was promptly diagnosed as probable normal pressure hydrocephalus with a tap test performed. CT brain showed communicating hydrocephalus with Evan's ratio of 0.4. Pre tap test-time to go 6metres :4mins 52 seconds with a remarkable improvement in post tap time to go 6metres :43seconds. 72years old male presenting with slowness of movements for 3years with rapid progression of symptoms and partial response to levodopa. Physical examination showed exaggerated corticospinal response with bilateral past-pointing. There are also supportive non-motor symptoms such as stridor, inspiratory sigh and pathological laughter. A clinical diagnosis of multisystem atrophy was made. 75 yrs old male presented with recurrent falls for 2years. He also noticed tremor and slowness of movements for 3years. He showed poor response to Levodopa (250mg TDS madopar=600mg L dopa). Physical examination showed upwards gaze restriction with procerus sign, axial rigidity, postural tremor with bilateral bradykinesia more marked on left side. There was significant postural hypotension (standing 100/70mmHg, sitting 140/88) He was diagnosed as progressive supranuclear palsy.

Discussion:

In conclusion, case series demonstrate the importance of identifying clinical cues to diagnose Parkinson plus syndromes.

Keywords

Parkinson plus syndrome, normal pressure hydrocephalus, multisystem atrophy, Progressive supranuclear palsy

P-195

A Case of Acute Dysphagia in Proximal Myotonic Myopathy With Benign Tremulous Parkinsonism: Double Jeopardy of Swallowing

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Introduction:

Proximal Myotonic Myopathy (PROMM) is a multisystem myopathy characterized by proximal weakness and electrical myotonia, whereas benign tremulous parkinsonism (BTP) is a tremor-domi-

nant syndrome with a relatively slow rate of deterioration. Here, we report a case of acute dysphagia developed in a patient with coexistence of PROMM and BTP.

Case:

A 66-year-old man initially presented resting tremor in his left hand for 6 months in 2018. He had a previous history of bilateral cataract surgery at age of 65. Neurologic examination showed mild left-sided proximal weakness, but there were no significant bradykinesia. Other clinical syndrome included angina, atrial fibrillation and baldness. Electromyography demonstrated a waxing and waning pattern of repetitive discharge suggesting electrical myotonia, supporting a diagnosis of myotonic dystrophy. The DMPK gene study showed a normal CTG repeat length. 3 years later, he developed mildly asymmetric parkinsonism with left predominance, including bradykinesia with resting tremor and rigidity, compatible with Hoehn and Yahr scale stage 2.5. FT-CIT PET study revealed a marked bilateral presynaptic dopaminergic deficit (more severe on the right side of basal ganglia), and thus rasagiline treatment was initiated. For more than 3 years, he had been keeping an independent daily activity with regular exercise (bicycling and weight training). In July 2025, he was admitted our hospital for 2-month onset swallowing difficulty, and a levodopa trial showed no benefit. The VFSS study recommended L-tube insertion based on ASHA NOMS swallowing level scale 1 (level 7 = normal), describing decreased swallowing reflex and decreased upper esophageal sphincter function.

Discussion:

We report a rare case of acute dysphagia in a patient with PROMM coexisting with BTP. This case emphasizes that rapid progressive symptoms like severe swallowing dysfunction could develop in a patient with complex neuromuscular and movement disorders.

Keywords

dysphagia, proximal myotonic myopathy, benign tremulous parkinsonism

P-196

Atypical Vascular Parkinsonism From a Midbrain Lesion With Excellent Dopaminergic Response

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Introduction:

Vascular parkinsonism is typically caused by ischemic lesions in the striatum or periventricular white matter and usually shows poor response to dopaminergic agents. However, parkinsonism due to ischemic lesions in the midbrain is rare. We present a case of parkinsonism resulting from an ischemic lesion in the midbrain that exhibited a favorable response to dopaminergic therapy, with anatomical considerations discussed.

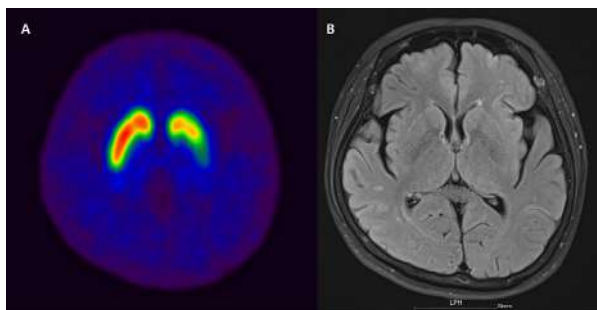
Case:

A 70-year-old woman presented with a one-year history of a right-hand tremor, which worsened with anxiety. She also developed right leg tremor and mild slowness in right limb movements, accompanied by mild rigidity and slight bradykinesia. Non-motor features such as REM sleep behavior disorder, hyposmia, and depression were absent, except for mild constipation. FP-CIT PET revealed decreased DAT binding in the left striatum but not the right.(Figure 1) Brain MRI showed an old ischemic lesion in the left midbrain without basal ganglia involvement.(Figure 1 and Figure 2) Treatment with rasagiline and amantadine improved her mild slowness. Over 18 months of follow-up, no symptom progression was observed.

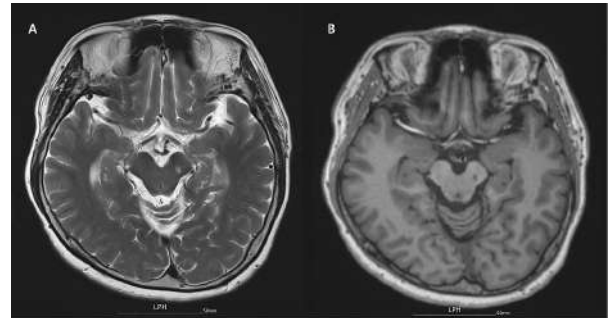
Discussion:

Parkinson's disease is characterized by degeneration of dopaminergic neurons in the substantia nigra with preserved postsynaptic systems, leading to good response to dopaminergic agents. In contrast, most cases of vascular parkinsonism are due to striatal or white matter involvement, resulting in poor response. This case uniquely demonstrates that a midbrain ischemic lesion affecting presynaptic dopaminergic neurons can cause parkinsonism that is responsive to dopaminergic therapy, consistent with the anatomical basis of presynaptic dopamine depletion.

Figure



Figure



P-197

Vascular Parkinsonism Followed by Post-Stroke Hemiballism/Hemichorea

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Introduction:

Movement disorders can be broadly divided into hypokinetic (e.g., parkinsonism) and hyperkinetic syndromes (e.g., dystonia, chorea, tremor). Cerebrovascular disease is a major cause of secondary movement disorders, often involving basal ganglia lesions. We report a patient who developed both vascular parkinsonism and post-stroke hemiballism/hemichorea.

Case:

A 75-year-old man presented with gradually progressive bradykinesia and gait disturbance. Examination revealed decrement with repetitive hand and foot movements, reduced step length, and decreased arm swing. Cognitive impairment was suspected (K-MMSE 20/30). ^{18}F -FP-CIT PET showed reduced dopamine transporter uptake, more marked in the left caudate and anterior putamen. Brain MRI demonstrated diffuse white matter disease and a right thalamic lacune. Levodopa (200 mg/day) produced no benefit. Eight months later, he developed acute, involuntary dance-like movements of the left upper limb and face. Brain MRI revealed an acute infarct in the right subthalamic nucleus, consistent with hemiballism/hemichorea. He was diagnosed with lacunar stroke and treated with dual antiplatelet therapy and statin. Symptomatic treatment with risperidone was declined by the patient.

Discussion:

This case illustrates the coexistence of two contrasting movement disorders—progressive vascular parkinsonism and acute post-stroke hyperkinetic movements—in the same patient. Progressive vascular parkinsonism typically shows diffuse white matter changes, symmetric features, cognitive decline, urinary symptoms, and poor levodopa response, as in our patient. Subthalamic nucleus infarction disrupts inhibitory control of the thalamocortical pathway, resulting in hyperkinetic movements such as hemiballism and hemichorea. Cerebrovascular disease can lead to diverse movement disorders depending on lesion location. Secondary prevention with antiplatelet therapy and risk-factor management remains crucial.

Keywords

Vascular parkinsonism, Cerebrovascular disease, Subthalamic nucleus, Hemichorea

P-198

Fahr's Syndrome Following Total Thyroidectomy: Commonly Overlooked Neurological Warning Signs: Rare Case Series

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Introduction:

Post-total thyroidectomy Fahr syndrome is a rare secondary clinical manifestation characterised by symmetrical calcification in the basal ganglia, dentate nucleus, and white matter of the cerebral cortex following iatrogenic hypoparathyroidism.

Case:

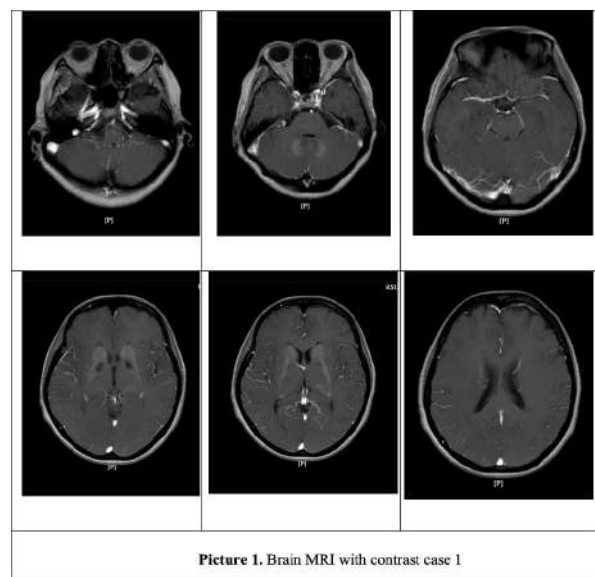
We report two cases of secondary Fahr syndrome due to hypoparathyroidism following total thyroidectomy in a year. The first patient, a 55-year-old woman, presented with tonic-clonic generalised seizures, parkinsonism, and cognitive decline. Laboratory showed hypocalcaemia (5.6 mg/dL), hyperphosphataemia (8.8 mg/dL), low PTH levels (5.5 pg/mL). MRI showed multiple bilateral and symmetrical lesions in the caudate nucleus, lentiform nucleus of the thalamus and right and left. The second patient, a 58-year-old woman, presented with vertigo, coordination disorders, intention tremor, and cognitive decline. Laboratory showed hypocalcaemia (7.5 mg/dL), hyperphosphataemia (8.0 mg/dL),

low PTH levels (6.3 pg/mL). CT-scan showed symmetrical calcifications in the basal ganglia and cerebellum. These findings supported the diagnosis of Fahr's syndrome. Both patients were given calcium replacement therapy, levothyroxine dose adjustment and monitoring for neurological disorders showed improvement. However, cognitive impairment persisted.

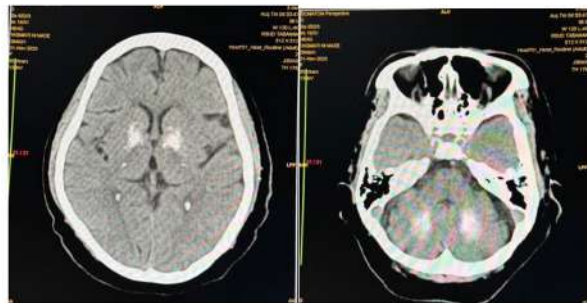
Discussion:

Secondary Fahr's syndrome is a complication that arises through chronic mechanisms due to calcium-phosphate metabolism dysregulation can lead to the formation of irreversible cerebral calcifications. Although metabolic correction may improve some symptoms, established cerebral calcifications rarely regress. Long-term complications of secondary Fahr's syndrome are significantly reduce patients' quality of life. It's important to understand Fahr's syndrome is not merely a radiological diagnosis, but a progressive neurodegenerative syndrome that can cause long-term disability. Early diagnosis and interdisciplinary management are necessary to prevent symptom progression and improve patients' quality of life.

Figure



Figure



Picture 2. Brain CT-Scan with contrast case 2

Table

	Case 1	Case 2
Etiology	Total Thyroidectomy	Total Thyroidectomy
Gender	Female	Female
Age	55	58
Family History	None	None
Neurological deficits	General motor tonic-clonic seizure, parkinsonism, cognitive impairment	Vertigo, coordination disorder, intention tremor, cognitive impairment.
Radiology examination	Brain MRI with contrast; multiple lesion bilateral symmetrical at nucleus caudatus, nucleus lentiformis, thalamus and nucleus dentatus dextra-sinistra.	Brain CT-scan with contrast; symmetrical <u>calcification</u> at basal ganglia and cerebellum.
Phosphate (mg/dL)	8.8	8
Calcium (mg/dL)	5.6	7.5
PTH (pg/mL)	5.5	6.3
Vitamin D (ng/mL)	25	30

Table 1. Clinical and biological characteristics of patients

Keywords

Fahr's syndrome, hypoparathyroidism, total thyroidectomy

P-199

Comparison of Early- and Late-Onset Progressive Supranuclear Palsy

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Introduction:

Progressive Supranuclear Palsy (PSP) is a neurodegenerative tauopathy characterized by vertical gaze palsy, postural instability, rigidity, and cognitive decline. We present two contrasting PSP cases

Case:

Case 1:

A 63-year-old female began experiencing balance issues at age 55. Over eight years, she developed progressive axial rigidity, dysphagia, vertical gaze palsy, facial hypomimia, and frequent choking. Neurological exam revealed supranuclear gaze palsy, increased tone, and a positive Romberg sign. MRI showed the classic "Hummingbird sign." She was diagnosed with PSP-Richardson's syndrome (PSP-RS).

Case 2:

A 34-year-old male initially presented with right arm numbness and tremor at age 30. Symptoms progressed to bradykinesia, gait instability, diplopia (on right gaze), memory impairment, and frequent nighttime urination, suggesting early autonomic involvement. Neurological exam revealed horizontal nystagmus, limb rigidity, poor coordination, and brisk reflexes. These findings were consistent with PSP with Parkinsonism predominance (PSP-P).

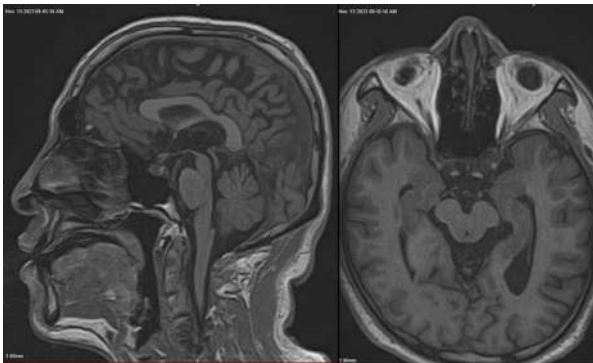
Discussion:

This comparison highlights clinical variability in PSP. Late-onset PSP-RS typically shows early postural instability and gaze palsy, while early-onset PSP may mimic Parkinson's disease, with subtle cognitive and autonomic features. Early recognition is crucial for accurate diagnosis and management

Figure



Figure



Keywords

progressive supranuclear palsy, Richardson's syndrome, Hummingbird sign

P-200

Modulation of Amyloidogenic Enzymes by "Dayak-Onion" Compounds as Biomarker and Therapeutic Potential in Movement Disorders

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Background & Objective:

Eleutherine bulbosa (Dayak Onion), an indigenous spice from Borneo Island, contains diverse bioactive compounds with reported pharmacological activities. Accumulation of amyloid- β (A β) plaques and tau aggregates, well established in Alzheimer's disease, also contributes to cognitive decline and motor dysfunction in parkinsonism-plus syndromes, including dementia with Lewy bodies, Parkinson's disease dementia, progressive supranuclear palsy, and corticobasal syndrome. Identifying natural modulators of amyloidogenic pathways may therefore yield biomarker candidates and therapeutic leads for these disorders.

Methods:

This study investigated the anti-amyloidogenic potential of *E. bulbosa* compounds using in silico approaches. Drug-likeness, pharmacokinetic properties, and predicted toxicity were assessed via SwissADME and PkCSM, resulting in the selection of nine compounds with favorable profiles. Molecular docking (Autodock Tools 1.5.6 and Biovia Discovery Studio) was performed against key amyloidogenic targets: A β (helical and β -sheet conformations), tau protein, β -secretase (BACE-1), and γ -secretase.

Results:

The nine selected compounds demonstrated strong inhibitory potential against BACE-1 and γ -secretase, enzymes central to A β production (Figure 1) two key enzymes involved in A β plaque formation. Among these, eleutherin showed the highest binding affinity for both targets (Figure 1, binding mode). Conversely, low binding affinity was observed toward A β aggregates themselves, suggesting that these compounds act primarily by modulating amyloid precursor protein cleavage rather than directly inhibiting fibril formation.

Conclusions:

In conclusion, *E. bulbosa* compounds—particularly eleutherin—emerge as promising modulators of amyloidogenic enzymes. These findings highlight their potential role as molecular biomarkers and therapeutic leads in secondary parkinsonism and parkinsonism-plus syndromes where amyloid and tau pathologies contribute to disease progression. Further experimental validation is required to establish translational relevance.

Figure

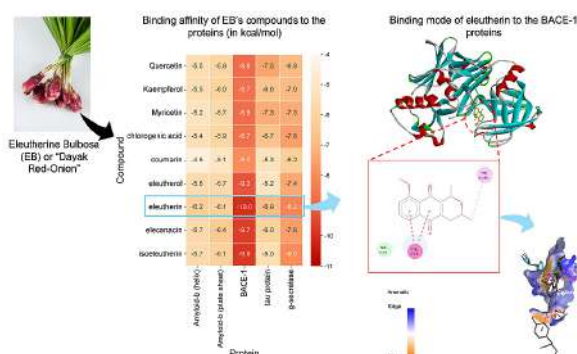


Figure 1. Research results

Keywords

amyloid-beta, Parkinsonism, amyloidogenic modulator, anti-amyloidogenic

Scales

P-201

The Relationship Between Korean Form of Mini-Mental State Examination and Montreal Cognitive Assessment and Conversion of K-MOCA to K-MMSE in Patients With Parkinson's Disease

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Background & Objective:

We aimed to investigate the correlation between K-MMSE and K-MoCA scores in a diverse cohort of patients with neurological

disorders with Parkinson's disease (PD). The Mini-Mental State Examination (MMSE) and the Montreal Cognitive Assessment (MoCA) are commonly used tools to assess cognitive function in patients with Parkinson's disease (PD). However, the MMSE has limitations, including copyright restrictions and a ceiling effect, which makes it less sensitive for detecting mild cognitive impairment, particularly in highly educated individuals. This study aimed to evaluate the validity of the MMSE-MoCA conversion across different educational levels in patients with PD.

Methods:

In this retrospective study, we investigated PD patients who completed both K-MMSE and K-MoCA on the same day at Chung-Ang university hospital from April 2010 and December 2020. We calculated using equipercntile method to equate MMSE scores based on MoCA scores. The MoCA-MMSE conversion scales was validated according to educational level (low educated: ≤ 6 years; intermediate educated 7-12 years, high educated: ≥ 13 years). The mean, median, root mean squared error (RMSE) of the difference between the actual and equated MMSE scores were calculated. Intraclass correlation coefficients (ICCs) and R-squared (R^2) were also calculated.

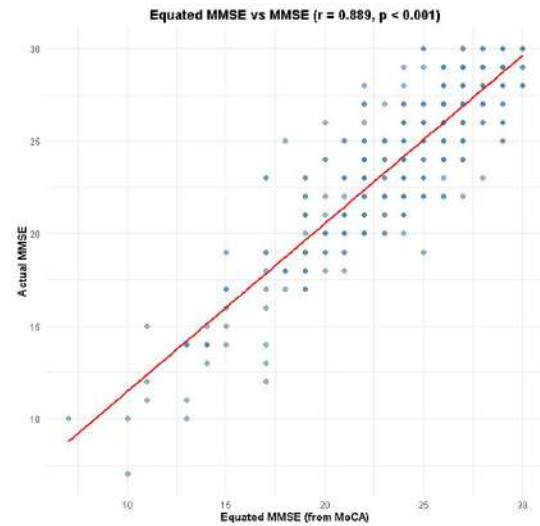
Results:

The difference between actual and equated MMSE scores had median value of 0, with small mean and RMSE of difference. ICCs and R^2 between actual and equated MMSE scores demonstrated a high correlation (ICCs 0.890, R^2 0.783). In the classification according to educational level, all groups had similar values of the median, mean, RMSE, ICCs and R^2 between actual and equated MMSE scores.

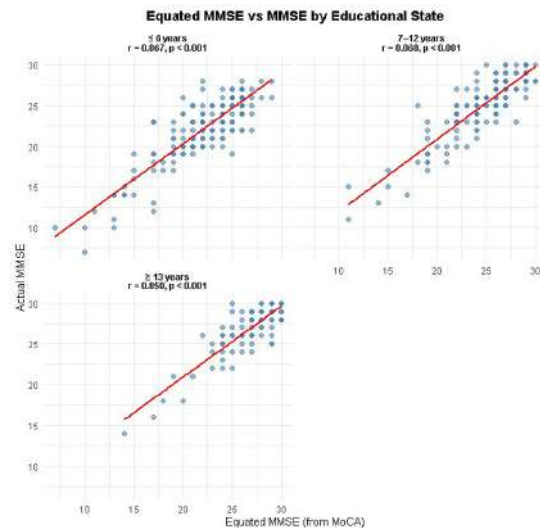
Conclusions:

Our findings suggest that validity of conversion MMSE tables derived from K-MoCA patients with PD, regardless of educational level and also suggest that K-MoCA may serve as a viable alternative to the MMSE for cognitive screening.

Figure



Figure



Keywords

K-MoCA, K-MMSE, Parkinson's disease

Technologies and AI for Movement Disorder

P-202

An Interpretable Deep Learning Framework for PSG-Based Classification of REM Sleep Behavior Disorder and Parkinson's Disease

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Background & Objective:

Early identification of Parkinson's disease (PD) remains a critical frontier. While REM sleep behavior disorder (RBD) is a well-established prodromal marker, conventional polysomnography (PSG)-based diagnostic approaches are limited in generalizability and interpretability. We aimed to develop an interpretable deep learning framework to classify RBD and PD across multi-center PSG datasets.

Methods:

PSG data from 233 individuals (PD with/without RBD, isolated RBD, controls) from four centers were analyzed. Two parallel AI pipelines were designed: an RBD classifier using chin EMG during REM, and a PD classifier leveraging multi-channel EEG/EOG/EMG (Fig.1). Subject-specific EMG normalization based on NREM segments enhanced inter-center robustness. Signals were segmented into 3-sec mini-epochs, and attention-weighted diagnostic scores were computed. Sleep stage information and latent space clustering were further incorporated for PD classification.

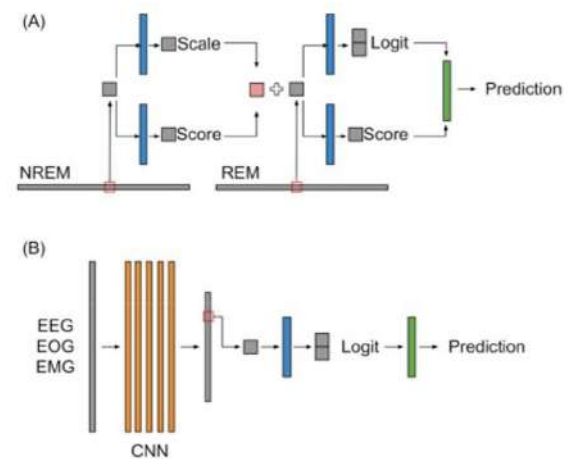
Results:

Our framework achieved strong performance, with AUC scores of 0.817 for RBD and 0.792 for PD. Two key findings emerged: 1) Model accuracy dropped minimally with only NREM (N2: -1.5%, N3: -2%), but markedly with REM alone (-10%) (Fig.2). 2) PD patients exhibited reduced sleep pattern diversity during N2, occupying only 31% of pattern diversity in controls (Δ entropy $\approx$ -1.7 bits) (Fig.3), indicating a collapse in neurophysiological flexibility. The RBD classifier also benefited from adaptive amplitude normalization, outperforming traditional methods.

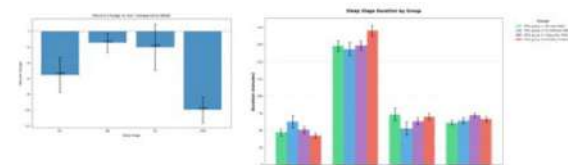
Conclusions:

This interpretable deep-learning framework achieves robust cross-center classification of RBD and PD with standard PSG and provides novel insights into neurodegeneration. Findings challenge REM-centric paradigms and highlight reduced neural flexibility in early PD. By uniting strong diagnostic performance with interpretability, the model supports a clinically scalable AI-assisted diagnosis.

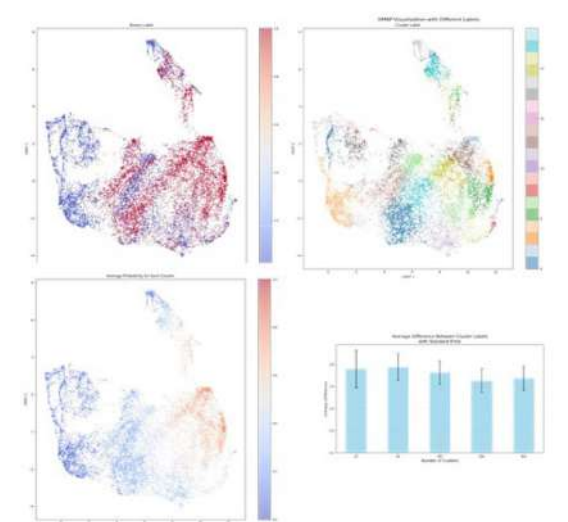
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Figure



Figure



Keywords

Interpretable deep learning, REM sleep behavior disorder, Parkinson's disease, Polysomnography, NREM sleep

P-203

Machine Learning-Enhanced Statistical Analysis of Voice Acoustic Biomarkers for Early-Stage Parkinson's Disease Detection: A Multi-Algorithm Comparative Study

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Background & Objective:

Parkinson's disease (PD) is a progressive neurodegenerative disorder that affects motor control, including voice production systems, often manifesting in early disease stages. Traditional diagnostic approaches rely on subjective clinical assessments with limited sensitivity for early-stage detection. This study presents a comprehensive multi-algorithm comparative analysis to identify optimal machine learning approaches for early-stage PD detection using statistical analysis of voice acoustic biomarkers, providing an objective, non-invasive screening methodology.

Methods:

We analyzed 195 voice recordings (147 PD patients, 48 controls) with 22 acoustic biomarkers including MDVP parameters, jitter/shimmer measurements, and spectral features. Six algorithms were compared: GBM, Random Forest, SVM, Neural Network, Elastic Net, and Logistic Regression using 10-fold cross-validation with ROC-AUC analysis.

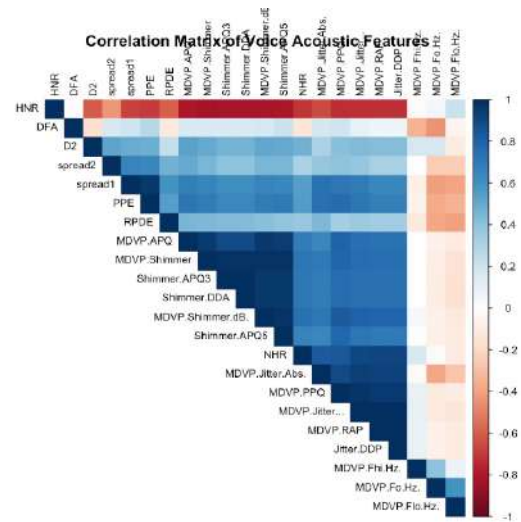
Results:

Multi-algorithm comparison revealed GBM as the optimal classifier, achieving superior performance metrics: 92.11% accuracy, 93.10% sensitivity, 88.89% specificity, and 97.70% AUC. Statistical cross-validation confirmed robust model stability across all validation folds. Feature importance analysis identified five critical acoustic biomarkers: PPE (Pitch Period Entropy), spread1, MDVP.Fo.Hz, spread2, and MDVP.Flo.Hz. Correlation matrix analysis revealed significant inter-parameter relationships among MDVP features and shimmer measurements, while HNR demonstrated inverse correlations with perturbation measures. Statistical comparison between groups showed significantly reduced fundamental frequency parameters in early-stage PD patients compared to controls.

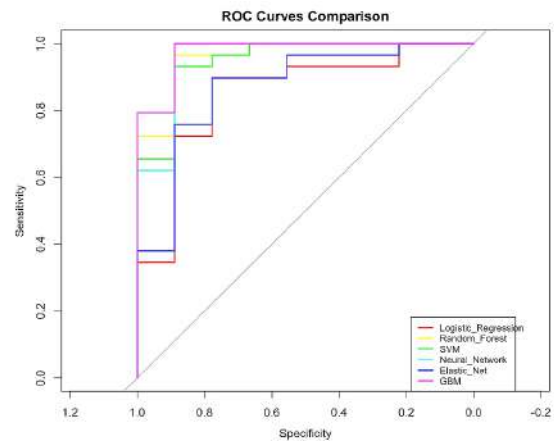
Conclusions:

Machine learning-enhanced statistical analysis of voice biomarkers provides exceptional early-stage PD detection (AUC=0.977). Key biomarkers reflect early motor control deterioration, offering clinical potential for non-invasive screening and monitoring, enabling timely intervention and improved outcomes.

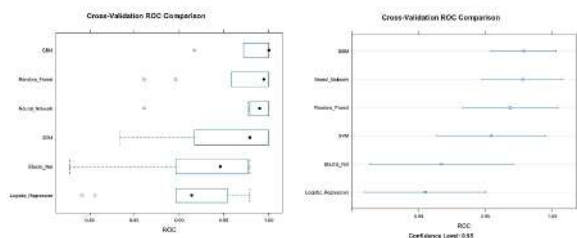
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Keywords

Machine Learning, Parkinson's Disease, Voice Acoustic Biomarkers, Gradient Boosting Machine

P-204

A Smartphone-Based Screening for Parkinson's Disease Integrating Digital Biomarkers and Clinical Risk Factors

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Background & Objective:

PD diagnosis relies mainly on clinical assessment, yet delays are common due to low awareness, limited specialist access, and socioeconomic barriers. We aimed to develop and validate a smartphone-based screening method using multimodal digital biomarkers combined with clinical risk factors.

Methods:

This study was conducted at Seoul National University Hospital from Dec 2023–Oct 2024. Patients with PD and age-matched controls were recruited; those with levodopa-induced dyskinesia were excluded. A self-administered application was developed to collect (1) motor and speech/cognitive digital biomarkers, and (2) a structured questionnaire covering medical history and non-motor symptoms. Iterative AI/ML-based feature refinement resulted in a final version comprising five motor function tests, a speech test, a color test, and the questionnaire.

Results:

A total of 241 patients with PD and 140 control subjects were enrolled for the initial version. Among nine motor tests in the initial version, six showed acceptable accuracy and included in the final version of application. Speech test, Color test and the Survey Questionnaire also showed acceptable accuracy and included in the final version of application. A new set of 204 patients with PD and 92 control participants was enrolled for the final version. The classifier achieved an AUROC of 86.20% when all tests were included. Among the individual tests, the Survey Questionnaire demonstrated the highest (AUROC 83.97%). The optimal performance was observed when Spiral test, Touch test, and the Survey Questionnaire were combined, yielding an AUROC of 90.53%.

Conclusions:

A smartphone-based platform integrating multimodal digital biomarkers with structured questionnaires demonstrated high accuracy (AUROC >90%) for PD screening. The tool is portable, user-friendly for older adults, and scalable without additional hardware.

Keywords

Parkinson's disease screening, Digital biomarkers, Smartphone-based application

P-205

Development of Immersive Gamification Technology Systems (ImGTS) for Improved Gait and Posture in Parkinson Disease (PD)

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Background & Objective:

Patients with PD commonly have impaired posture and gait, limiting their daily function and increasing fall risk. Standard-of-care dopaminergic medications do not fully address postural instability, gait automaticity, and fall risk in PD. Yet rehabilitation effectively reduces the risk of falls, making access to more options for rehabilitation crucial, especially in resource-limited countries. This study aimed to develop ImGTS using virtual reality (VR) for PD rehabilitation that are adapted for the Philippine context.

Methods:

ImGTS prototypes were developed through an iterative process involving alpha and beta testing. Eleven participants, namely game developers, rehabilitation medicine specialists, occupational and physical therapists, movement disorder specialists; and patient caregivers, joined focus group discussions (FGD) on feasibility, game elements, and potential challenges in using ImGTS in PD rehabilitation. Feedback from the FGD guided subsequent design revisions. A second cycle of prototype testing-feedback-design revision will follow.

Results:

Preliminary FGD results indicated that ImGTS was perceived as feasible as an engaging therapy option if it contains the following features: familiar and traditional Filipino elements and values, user training, and implementation in centers rather than homes. Potential challenges in using ImGTS in PD that emerged in the FGD included cost, need for technical support, complexity of setup, connectivity issues, and health and safety concerns that can be minimized by customizing games to patient profiles.

Conclusions:

These findings inform the iterative design process for the ImGTS prototype as a culturally contextualized therapy option to improve posture and gait in PD, and provide basis for pre-clinical testing in healthy adults.

Keywords

Parkinson Disease, Gait, Posture, Virtual Reality, Physical Rehabilitation

P-207

Surface Electromyography in Movement Disorder Management: Applications and Diagnostic Approaches in Developing and Developed Countries

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Background & Objective:

Over the last decades, the prevalence of movement disorders rose fourfold and misdiagnosis remains common. Surface electromyography (sEMG) provides objective, low-cost diagnostic data and has started to be used in developed countries. However its feasibility and performance in low- and middle-income countries (LMICs) are poorly studied. This study reviews current evidence on sEMG applications, highlighting its possible implementation in LMICs.

Methods:

This review followed PRISMA guidelines. Studies in the last decades evaluating sEMG use on movement disorder were retrieved from Cochrane, ScienceDirect, Scopus, PubMed, Taylor and Francis, Wiley, EBSCO, and Sage Journals using combination of Boolean operators, MeSH terms, and analyzed qualitatively.

Results:

Four studies with a total of 830 participants were reviewed, covering Parkinson's disease, tremor, and myoclonus. Surface electromyography (sEMG) was used either alone or combined with high-density sEMG (HD-sEMG), accelerometry, electroencephalography (EEG), and machine or deep learning methods. HD-sEMG shows positive results but lacks feasibility for implementation due to advanced hardware needs. sEMG with EEG and accelerometry are feasible in referral hospitals. And sEMG alone or sEMG combined with machine or deep learning offers a scalable approach for diagnosing movement disorders in low- and middle-income countries (LMICs). Overall these approaches showed strong performance in classifying Parkinson's disease, the assessment of swallowing muscle function, and the diagnosis of challenging tremor cases.

Conclusions:

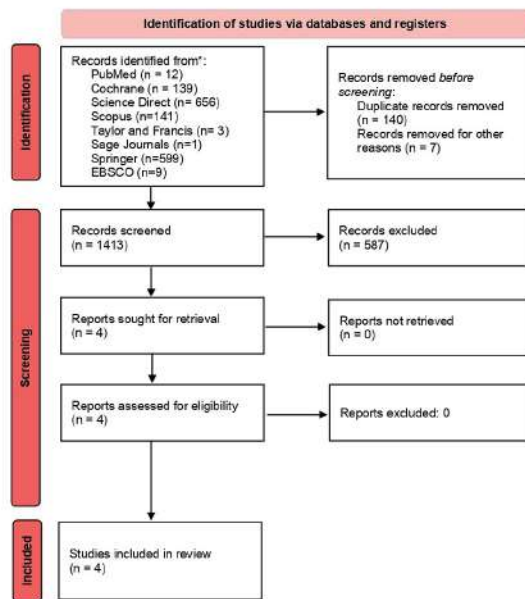
sEMG demonstrates strong potential for improving diagnosis and therefore management of movement disorders in low- and

middle-income countries (LMICs) where access to specialized personnel, diagnostic tools, and advanced facilities are limited. However, its application requires trained personnel for operation and accurate data interpretation.

Table

Author (Year)	Measurement Methods	Strengths	Limitations	Application	Suitability in LMICs
Everio CSJ et al., 2022	Polymyography (sEMG + accelerometry) combined with EEG	Re-evaluate polymyography data with a more systematic and consistent approach, focus on cases that are difficult to differentiate clinically, large and representative sample size	Data need to be interpreted by an experienced neurophysiologist, retrospective study and the potential of circular reasoning	A stepwise approach that can serve as a diagnostic framework: contraction pattern (alternating vs. synchronous), rhythm (regular vs. irregular), EMG burst duration, evidence of cortical drive from EEG	Medium-High: portable and adaptable, interpretation of polymyography results requires a trained neurophysiologist
Razavi Khosro et al., 2023	Pre-trained Deep Tensor Learning (DTL) structure and conventional machine learning (ML) models from sEMG signals	The combination of DTL (for feature extraction) and ML (SVM for classification) provides superior accuracy and efficiency in the sEMG signal-based Parkinson's disease classification process.	Requires large database and expert in multiple fields	A non-invasive tool for diagnosing and monitoring Parkinson's disease, which can support clinical decisions, assess disease progression, and has the potential to be integrated with various assistive devices and rehabilitation systems such as rehabilitation robots and prostheses	High: two-channel sEMG and transfer learning can be cost-contained, barriers include hardware costs and software or third-party licenses
Nakamori et al., 2024	High-density surface electromyography (HD-sEMG) of the suprapyloric muscles during banger elevation, before and after cervical paravertebral interlaminar current stimulation	Non-invasive, detailed spatial and temporal analysis of motor unit (MU) recruitment, capable of detecting subtle physiological changes due to intervention, validated method for swallowing-related muscles	Single-site, single-group design, small sample size, lacks direct correlation with swallowing function, no placebo group, needs advanced equipment and trained operator	Assessment of MU recruitment and response to neurostimulation in PD patients; potential tool for evaluating dysphagia and effect of intervention	Low: Requires HD-sEMG equipment, computational tools, trained personnel, and intervention device, difficult to implement in resource-limited settings
Kozulin et al., 2024	Surface EMG (sEMG) during MDS-UPDRS II tasks ("finger tapping" and "hand movements") pre- and post-levodopa intake, with analysis of number of peaks and activation intervals	Objective quantification of bradykinesia feature using sEMG, detects levodopa-induced changes, can enhance pre- and post-levodopa intake, with analysis of number of peaks and activation intervals	Small sample (8 PD, 7 controls), only two tests analyzed, limited generalizability, absence of direct pathological validation, mostly descriptive	Enhances objectivity of MDS-UPDRS II motor scoring using number of muscle activation peaks and timing, potentially useful for real-time bradykinesia monitoring	Medium-high: sEMG systems are wireless and relatively portable, low infrastructure requirement, suitable for adapted use in LMICs with minimal cost compared to imaging

Figure



Keywords

surface electromyography, movement disorders, diagnostic approaches, low- and middle-income countries

P-208

Machine Learning Approaches using Surface Electromyography Features to Classify Parkinson's Disease during Sit-to-Walk

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Background & Objective:

Among daily functional activities, the sit-to-walk (STW) task represents a complex motor sequence requiring precise neuromuscular timing and coordination [1]. This study investigated neuromuscular abnormalities in Parkinson's disease (PD) during the STW task using surface electromyography (sEMG) and evaluated the potential of explainable machine learning (ML) frameworks for digital biomarker discovery.

Methods:

Data were collected from 102 individuals with PD and 50 age-matched healthy controls (HCs) during a standardized STW task and segmented into three phases: (1) total STW sequence, (2) sit-to-stand, and (3) gait initiation. Raw sEMG signals from eight bilateral lower-limb muscles were preprocessed, filtered, normalized, aligned to the right leg as the leading step, and time- and frequency-domain features and co-contraction indices were extracted. Feature importance was first estimated using a baseline Random Forest (RF) model, and the top 20 ranked features were selected for downstream ML modelling (Figure 1). Model interpretability was using Shapley Additive Explanations (SHAP) values to identify phase- and muscle-specific discriminative features.

Results:

Feature importance analysis revealed that the laterality variable emerged as the most discriminative predictor. Among the sEMG-derived variables, the mean frequency during STW Phase 1 (sit-to-stand) was identified as the strongest feature for distinguishing PDs from HCs. The RF classifier yielded the best results (accuracy = 92.6% and AUC = 0.97). SHAP analysis further highlighted frequency-domain features and co-contraction indices of the rectus femoris and biceps femoris short head during STW Phase 1 (Figure 2).

Conclusions:

These findings suggest maladaptive muscle coordination strategies in PD and underscore the sensitivity of STW neuromuscular features for classification [2]. Feature-based ML provided superior classification performance, while SHAP analysis enhanced interpretability by identifying meaningful digital biomarkers.

Figure

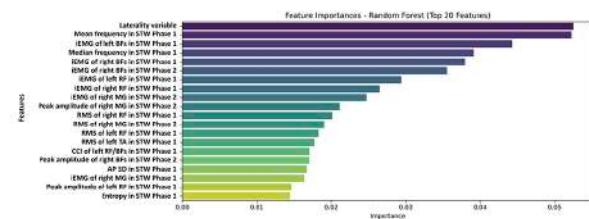


Figure 1. Top 20 ranked sEMG-derived features for classifying people with Parkinson's disease and healthy controls. STW, Sit-to-walk; IEMG, Integrated EMG; BF, Biceps Femoris short head; RF, Rectus Femoris; MG, Medial Gastrocnemius; RMS, Root mean square; TA, Tibialis Anterior; CCI, Co-contraction Index; AP SD, standard deviations along the anterior-posterior.

Figure

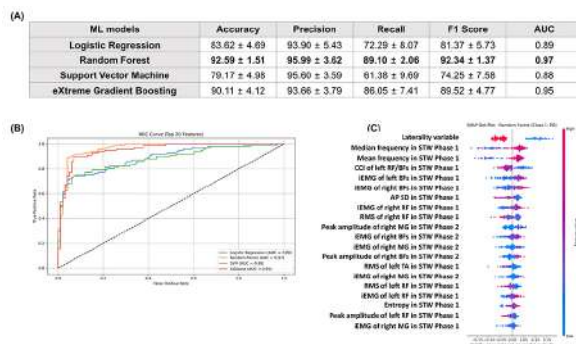


Figure 2. (A) Performance comparison of machine learning models for people with Parkinson's disease and healthy controls classification using sEMG-derived features, (B) Receiver operating characteristic (ROC) curves of machine learning models, and (C) SHAP feature importance summary plot, highlighting the top 20 predictive sEMG-derived features.

Keywords

Parkinson's Disease, Surface Electromyography, Sit-to-Walk, Machine Learning, Digital biomarker

P-211

Data Driven Subtyping Patterns of Deep Gray Matter Atrophy, Dopamine Availability, and Cognition in Early Parkinson's Disease

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Background & Objective:

Cognitive impairment is a prevalent non-motor symptom in Parkinson's disease (PD). PD is characterized by striatal dopamine depletion and deep gray matter atrophy, with early cognitive decline often attributed to dopaminergic system dysfunction. We aimed to explore disparities in cognition based on based on striatal dopamine availability and regional brain volume among PD patients.

Methods:

This study included 287 patients with newly diagnosed PD. They all underwent brain magnetic resonance imaging, positron emission tomography with ¹⁸F-N-(3-fluoropropyl)-2beta-carbon ethoxy-3beta-(4-iodophenyl) nortropane, and ¹²³I-MIBG scintigraphy. Using the Subtype and Stage Inference (SuStain) machine-learning technique, we categorized PD patients into subtypes by analyzing their progression patterns in deep gray matter atrophy and dopamine availability, and evaluated differences in cognition among these subtypes.

Results:

SuStain algorithm categorized them into four distinctive subtypes. Motor symptoms in subtype 2 was milder than other subtypes. Subtype 2 showed higher cognitive function and subtype 3 and 4 displayed larger proportion of dementia. Subtype 1 and 2 generally showed less decrease of brain atrophy and dopamine availability than subtype 3 and 4. Subtype 1 showed early dopamine availability decrease and late brain volume reduction, while in subtype 2, brain atrophy was decreased initially, followed by dopamine availability. Subtype 3 and 4 were mixed type, in which initial change was globus pallidus atrophy and dopamine availability and brain atrophy were reduced similarly.

Conclusions:

Our finding underscores the association between decreased dopamine availability and volume with cognitive dysfunction, highlighting the spatiotemporal association between dopaminergic and structural pathology in cognitive impairment in PD.

Keywords

Parkinson's disease, cognition, deep gray matter atrophy, dopamine availability, SuStain

P-212

Brain Atlas-Based Machine Learning for Parkinsonian Disorders Classification and Evaluation of Classification Performance Across Disease Duration

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Background & Objective:

Despite the different prognosis and response to medication, Parkinson's disease (PD), multiple system atrophy (MSA) and progressive supranuclear palsy (PSP) can share common clinical manifestations, thus the differential diagnosis can be challenge in clinical practice. Therefore, we applied various machine learning models for the differential diagnosis of degenerative parkinsonian disorders in this study.

Methods:

We retrospectively collected brain MRI of 913 subjects with parkinsonism (494 with PD, 242 with PSP, and 197 with MSA). In all subjects, the volumes of brain structures based on 12 brain atlases were calculated and used to develop differential diagnostic tool using machine learning. We compared the diagnostic performance among the tools with various machine learning techniques, and the performance of the best model based on the disease duration.

Results:

Among the various machine learning techniques, the model with combination of Recursive Feature Elimination with Cross-Validation and Random Forest model (RFECV-RF) demonstrated the

highest area under curve (AUC) for the differential diagnosis. The AUC was higher than 0.9 for the prediction of PD vs. others, PSP vs. others, and MSA vs. others. The diagnostic performance was better with longer disease duration from 1 year to 4 years, and the best at 4 years of disease duration.

Conclusions:

Our study revealed the changes in the brain structures are different among the degenerative parkinsonian disorders, and these changes can be used for the differential diagnosis. Based on our results, we suggested differential diagnostic tool in parkinsonian disorders using machine learning.

P-213

AI-Driven Integration of Single-Cell Purkinje Trajectories and DNMT1 Epigenetic Dysregulation to Derive Blood-Based Biomarkers for Cerebellar Ataxia

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⁴Computational Science Research Laboratory, BLK Muhammadiyah University, Bulukumba, Indonesia

Background & Objective:

Cerebellar ataxias have varied genetic causes but converge on Purkinje cell loss. Single-cell studies capture this decline but are not feasible clinically. We mapped Purkinje trajectories in SCA1, integrated DNMT1-related epigenetic changes, and tested whether FXTAS blood profiles mirror these states to enable a digital biopsy.

Methods:

Datasets were obtained from the Gene Expression Omnibus (GEO). Single-nucleus RNA-seq of SCA1 cerebellum (GSE246183, 54 samples) defined Purkinje trajectories with Harmony and Monocle3. DNMT1-mutant induced neurons (GSE126890, 56 samples) with RNA-seq and methylomes identified DMR-gene couplings and synaptic modules, aligned by dynamic time warping. FXTAS blood transcriptomes (GSE48873, GSE48902, 39 samples) were ComBat-normalized and mapped to trajectories using a domain-adversarial network and kernel ridge regression.

A 10-gene panel was derived with nested LASSO. Performance was assessed by 100 Monte-Carlo cross-validations, bootstrap confidence intervals, calibration, and decision-curve analysis.

Results:

Purkinje trajectories separated SCA1 from controls with AUROC 0.81 (95% CI 0.72-0.89) and accuracy 78% (95% CI 68-87). A DNMT1-driven synaptic module enriched for ion-channel genes correlated with progression (p 0.52, 95% CI 0.42-0.61, FDR $<1 \times 10^{-4}$). DMR-DEG coupling explained 33% of variance (adj. R^2 0.33, $P < 1 \times 10^{-4}$). Blood-to-brain prediction gave R^2 0.36 (95% CI 0.12-0.58), MAE 0.15, and advanced classification AUROC 0.76 (95% CI 0.63-0.88) with Brier 0.19. The 10-gene panel (EAP1, RNF10, HDAC5, APP, SOD1, ATXN7, ATXN3, COX6C, COX7B, DFFA) was stable in 79% of resamples and overlapped FX premutation signals. Decision-curve analysis showed benefit across thresholds 0.25-0.65.

Conclusions:

This computational framework links DNMT1-driven epigenetic changes to Purkinje cell trajectories and translates them into a 10-gene blood panel, offering a digital biopsy for ataxia staging and patient stratification.

Keywords

Purkinje trajectories, DNMT1 epigenetic remodeling, 10-gene blood panel, Digital biopsy, Domain-adversarial network

P-214

Social-Jetlag-Aware Machine Learning Twin Integrates Neurodegeneration, Dopaminergic Dysfunction, and Daily Routines to Predict Quality-of-Life Decline in Parkinson's Disease

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Background & Objective:

Medication timing is critical in Parkinson's disease (PD), although many patients experience "social jetlag," where social schedules misalign with biological rhythms. We aimed to build a machine learning (ML) framework linking social jetlag, biologic

vulnerability, and digital behavior to predict quality-of-life (QoL) decline and guide personalized chronotherapy.

Methods:

Three open cohorts were harmonized. mPower (n=1,612) provided smartphone-based tapping, gait, and voice tasks time-stamped medication use and PDQ-8. Fox Insight (>54,000) supplied sleep-wake patterns, employment, caregiving, lifestyle to derive social-jetlag metrics (midpoint sleep gap, irregularity). PPMI (n=1,084) contributed EQ-5D-5L, PDQ-39, MDS-UPDRS, DaTscan binding ratios (SBR), UPSIT, RBDSQ, serum neurofilament light chain (NfL). Symptom-phase trajectories modeled generalized additive models. Gradient-boosted ML with double machine learning estimated average, individualized treatment effects (CATEs) of delayed medication, controlling for age, sex, UPDRS, SBR, UPSIT, RBDSQ, NfL. Domain adaptation transported effects across cohorts, and policy learning simulated dosing retiming.

Results:

In PPMI, medication delays >60 minutes reduced EQ-5D by -0.042 (95% CI -0.055 to -0.028). Larger effects were seen in patients with low SBR (CATE -0.067, $p < 0.01$) and high NfL (CATE -0.053, $p = 0.004$). In Fox Insight, each 1-hour social-jetlag increase reduced PDQ-39 by 2.1 points (95% CI -2.9 to -1.3). In mPower, diurnal sensor features predicted PDQ-8 with $R^2 = 0.43$, and performance improved to $R^2 = 0.51$ when social-jetlag was included. The mapped EQ-5D showed a mean absolute error of 0.076. Targeted retiming reduced low-QoL prevalence by 5.8% (95% CI -7.6 to -4.1).

Conclusions:

This ML framework shows that social jetlag and neurodegeneration jointly accelerate QoL decline in Parkinson's disease. It delivers precision dose-clock recommendations with direct clinical utility for managing daily motor fluctuations.

Keywords

Parkinson's disease, Social jetlag, Chronotherapy, Machine learning, Quality of life

P-216

A Multimodal AI Framework for Predicting Parkinson's Disease Progression: Augmented by Wearable Sensor and Patient-Reported Outcome Data

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Background & Objective:

The heterogeneous progression of Parkinson's disease (PD) necessitates a paradigm shift from episodic clinic assessments to continuous, objective monitoring. While artificial intelligence (AI) models leveraging wearable sensors and patient-reported outcomes (PROs) show promise, the comparative efficacy of data modalities remains poorly synthesized, and multimodal AI approaches are underexplored.

Methods:

This study comprised a systematic review/meta-analysis of existing AI models (k=48 studies) and a novel quantitative analysis developing a multimodal deep neural network (DNN). The DNN fused wearable sensor-derived features (e.g., gait velocity, tremor power) and PROs (MDS-UPDRS Part II) from the open-source LPP dataset (N=75 patients) to predict the change in motor severity (Δ UPDRS-III). Model performance was evaluated using nested cross-validation.

Results:

The meta-analysis established that multimodal models significantly outperformed unimodal approaches (Pooled MAE: 3.12 vs. >4.85). Our novel DNN validated this finding, achieving state-of-the-art performance (MAE=3.28, R^2 =0.75) and a 25% error reduction over a comparable multimodal Random Forest (MAE=4.05). Ablation studies confirmed the synergistic effect of multimodal data, with sensor-only (MAE=5.12) and PRO-only (MAE=6.83) models performing substantially worse. Interpretability analysis (SHAP values) identified gait velocity, disease duration, and the patient's self-reported motor function as the most salient predictive features, aligning with clinical knowledge.

Conclusions:

This work provides the first quantitative meta-synthesis demonstrating the superiority of multimodal AI for predicting PD progression. By developing and validating a highly accurate and interpretable DNN, we bridge a critical gap between proof-of-concept and clinically actionable tools. Our results robustly argue for the integration of multimodal digital biomarkers into future clinical trials and personalized disease management strategies.

Table

Characteristic	Category	Number of Studies (k=48)	%
Data Modality	Sensor-only	28	58.3%
	PRO-only	8	16.7%
	Multimodal	12	25.0%
ML Model Type	Traditional (RF, SVM)	35	72.9%
	Deep Learning	13	27.1%
Prediction Task	Regression (e.g., UPDRS score)	31	64.6%
	Classification (e.g., Hoehn & Yahr stage)	17	35.4%

Table

Variable	Mean (SD) or Count (%)	Measurement
Age (years)	64.7 (8.2)	Continuous
Sex (Male)	48 (64%)	Categorical
Disease Duration (years)	7.1 (4.5)	Continuous
Levodopa Equivalent Dose (mg/day)	725.3 (325.6)	Continuous
ΔUPDRS-III (OFF-ON)	21.5 (8.7)	Continuous (Target)
Gait_Velocity (m/s)	0.98 (0.21)	Continuous (Sensor Proxy)
PRO_Motor_Total (0-52)	18.4 (9.1)	Ordinal (PRO Proxy)

Table

Model	Data Modality	Mean Absolute Error (MAE)	Root Mean Squared Error (RMSE)	R^2
Linear Regression	Multimodal	4.91	6.12	0.51
Random Forest	Multimodal	4.05	5.28	0.63
Proposed DNN	Multimodal	3.28	4.35	0.75
Random Forest	Sensor-only	5.12	6.45	0.41
Random Forest	PRO-only	6.83	8.12	0.18

Keywords

Parkinson's Disease, Digital Biomarker, Artificial Intelligence, Multimodal Learning, Wearable Sensors

P-217

Palatal Tremor Versus Palatal Myoclonus: Revisiting the Term Based on Video-Based Analysis

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Background & Objective:

Rhythmic jerky movement of the soft palate and uvula is a rare movement disorder that is caused by idiopathic etiology or structural lesions affecting the Guillain Mollaret triangle (GMT). However, there are debates regarding the description of this as a tremor or myoclonus. Thus, objective tracking of the soft palate/uvula is required to analyze the rhythmicity or shape of the palatal movement. With recent development in computer vision technology, we can reliably track soft palate or uvula within the 2D RGB video clip.

Methods:

We collected videos of palatal tremor/myoclonus from published articles. Specifically, we searched PubMed using the terms palatal tremor OR myoclonus and retrieved supplementary video files when available. Videos were included if they demonstrated more than 10 visible cycles of tremor/myoclonus with clear visualization of the soft palate. For comparison, we used archived videos of patients with essential tremor (ET) presenting with head tremor. Video analysis was performed using a previously validated algorithm (Shin et al., 2025), which tracks the displacement of the soft palate/uvula across frames. The extracted motion traces were then subjected to power spectrum analysis to quantify the rhythmicity of palatal movements.

Results:

We analyzed 12 videos of palatal tremor/myoclonus and 30 videos of essential tremor (ET). In the palatal tremor/myoclonus group, the distribution of rhythmicity and sinusoidality varied across participants. A subset of patients demonstrated a high sinusoidality index, suggestive of jerkiness consistent with myoclonus. However, no significant group-level differences in rhythmicity or directionality were observed between the essential tremor and palatal tremor/myoclonus groups.

Conclusions:

This study may contribute to revisiting the appropriate term for the current concept of palatal tremor or palatal myoclonus. Based on the waveform analysis, the term may be attributed based on individual cases based on waveform of sinusoidality.



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1. Hauser RA, et al. 2021;133(7):721-727. 2. Ferreira JJ, Lee JY, et al. J Neurol. 2024 Oct;271(10):6729-6738. 3. ONgentys (opicapone) Summary of Product Characteristics. November 2024. 4. Ferreira JJ, et al. Mov Disord. 2022 Nov;37(11):2272-2283. 5. Ferreira JJ, et al. Mov Disord. 2020;35(Suppl S1):S447. Abstract 998. 6. Lees A, et al. Safety profile of Opicapone in the management of Parkinson's Disease. J Parkinsons Dis. 2019;9(4):733-740. 7. Reichmann H, et al. Transl Neurodegener. 2020;9(1):9. 8. Santos Garcia D, et al. Brain Sci. 2022;12(3):383. 9. Metta V, et al. J Neural Transm (Vienna). 2024 Jan;131(1):25-30.

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ADOPTION STUDY²

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목적: 파킨슨병 환자의 약효소진(wearing off) 치료에 First-line approach로서 Opicapone 50mg 또는 레보도파 100mg 추가 효과를 평가하기 위한

다음과 같은 PD 환자:

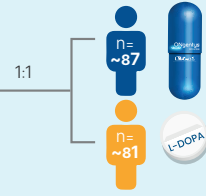
- 초기 운동장애
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- 레보도파
≈ 400MG
- 1일 3회 복용
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평가변수

Baseline 대비 OFF-TIME 변화

2차
평가변수

내약성, 기능적 운동 및 비운동 요소 평가
(MDS-UPDRS, PDQ-8 & CGI-I 및 CGI-C 척도)

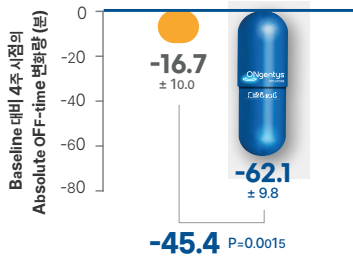


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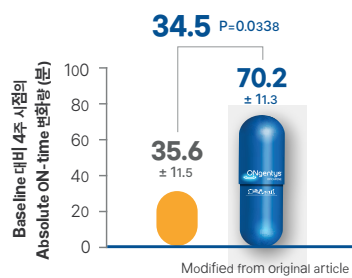
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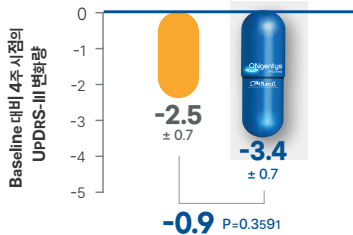
ON-TIME



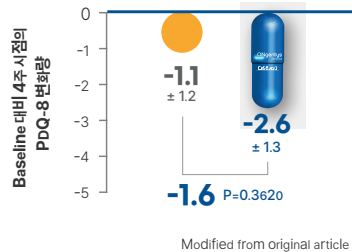
Point 01

1일 1회 복용하는 온젠티스[®]는 초기 운동장애 환자에서 레보도파 100 mg 추가에 비해 OFF-time 및 ON-time을 유의하게 더 많이 개선 하였습니다.²

운동성 증상 (MDS-UPDRS III)



삶의 질 (PDQ-8)



Point 02

레보도파 100 mg 대비 운동성 증상 및 삶의 질을 개선하는 경향성을 보였습니다.²
(Numerical Improvement)

전반적 개선지수 평가 (CGI-I, CGI-C)

개선된 대상자 비율 ('매우 개선됨', '개선됨', '약간 개선됨')으로 평가된 대상자 비율



임상의



환자



Modified from original article

Point 03

임상의 및 환자에 의해 평가된 전반적 개선 비율이 온젠티스[®]에서 더 높게 나타났습니다.²

MDS-UPDRS : Movement Disorder Society-Unified Parkinson's Disease Rating Scale,

PDQ-8 : Parkinson's Disease Questionnaire-8,

CGI-I : Clinical Global Impression of Improvement,

CGI-C : Patient Global Impression of Change

References 1. Ferreira JJ, et al. Effect of Opicapone on Levodopa Pharmacokinetics in Patients with Fluctuating Parkinson's Disease. Mov Disord. 2022 Nov;37(11):2272-2283
wearing-off in parkinson's disease patients: The korean ADOPTION trial. Movement Disorders Clinical Practice, 2024.

2. LEE JY, et al. Opicapone to treat early
3. 온젠티스캡슐 허가정보, 의약품안전나라 [Cited 2024.05.30]. Available from: <https://nedrug.mfds.go.kr/>

온젠티스[®] 캡슐 제품요약정보

【제품명】 온젠티스캡슐 50밀리그램(오피카폰) **【원료약품 및 그 분량】** 이 약 1캡슐 중 유효성분: 오피카폰(별표) 50mg

【용법·용량】 1. 이 약의 권장용량은 1일 1회 50mg이다. 이 약은 취침 전에 레보도파/도파 탈탄산염 복합제(DDCI)의 보조치료제

carbidopa)와 최소 1시간 이상 간격을 두고 경구 투여한다. 이 약을 투여하기 1시간 전부터 1시간 후까지 공복상태를 유지한다. 2. 이 약은 레보도파 표준요법의 부가요법으로 투여되고, 레보도파의 효능을 증강시키므로 레보도

파 관련 도파민성 부작용을 즉 운동이상증, 환각, 구역, 구토 및 기립성저혈압을 감소시키기 위해서는 이 약 투여를 시작한 이후 환자의 임상적 상태에 따라, 복용량 당 레보도파의 양을 감소 시키거나 복용 간격을 증가시키므로써 레

보도파의 용량을 조절할 필요가 있다(2. 신중투여할 참고). 3. 이 약의 복용을 잊었을 경우: 이 약의 복용을 잊었을 경우, 다음 복용을 예정대로 복용한다. 놓친 용량을 보충하기 위해 권장량을 초과하여 투여해서는 안된다. 4. 고령

자: 고령자에 대한 이 약의 용량조절은 요구되지 않는다(8. 전문가를 위한 정보참고). 85세 이상의 고령자에 대해서는 임상적 경험에 제한적이며 주의해서 투여해야 한다. 5. 소아: 소아에 대한 이 약의 사용은 권장되지 않

는다. 6. 신장에 환자: 이 약은 신장으로 배설되는 양이 낮아(8. 전문가를 위한 정보참고) 신장에 환자에 대한 이 약의 용량조절은 요구되지 않는다. 7. 간장에 환자: 경증의 간손상 환자(Child-Pugh A)에 대한 이 약의 용량조

절은 요구되지 않으며, 중등도 간손상 환자(Child-Pugh B)에 대한 이 약의 임상적 경험은 제한적이며 투여 시 용량조절이 필요할 수 있다. 중증의 간손상 환자(Child-Pugh C)에 대한 임상적 경험은 없으므로 이 약의 사용은 권

장 되지 않는다(8. 전문가를 위한 정보참고). **【사용상의 주의사항】** 1. 다음 환자에는 투여하지 말 것: 1) 주성분 또는 부형제, 캡슐 기재 등에 과민성인 환자 2) 크롬천화성세포증, 부신경절종 또는 다른 카테콜아민분비 종양

환자 3) 신기능저하 약성증후군 및/또는 비외상성 혈로근용해증 환자 4) 파킨슨병 치료제 이외의 모노아민 산화효소(MAO-A 및 MAO-B) 억제제 (예: 페닐진, 트라닐시프롤린, 모클로베מיד)를 투여 중인 환자(상호작용 참고) 5)

인부 6) 수유부 7) 이 약을 함양하고 있으므로, 갈락토스 불내성(galactose intolerance), 유당불내성 결핍증(total lactase deficiency) 또는 포도당갈락토스 흡수장애(glucose-galactose malabsorption) 등

의 유전적인 문제가 있는 환자에는 투여하면 안 된다. (후략) **【제조사】** Bial - Portela & C^o, S.A. Àv. da Siderurgia Nacional 4745-457 S. Mamede do Coronado Portugal

정주시 흥덕구 산단로 149 **【판매처】** 에스케이케미칼(주) 경기도 성남시 분당구 판교로 310

* 처방하기 전 제품설명서 전문을 참고하십시오. 최신 허가사항에 대한 정보는 '의약품안전나라' (<https://nedrug.mfds.go.kr/index/>)에서 확인할 수 있습니다.

【효능·효과】 레보도파/도파 탈탄산염 복합제(DDCI) 표준요법으로 증상이 개선되지 않는 운동장애 증상

【용법·용량】 1. 이 약의 권장용량은 1일 1회 50mg이다. 이 약은 취침 전에 레보도파/도파탈탄산 염소염 복합제(benserazide 또는

carbidopa)와 최소 1시간 이상 간격을 두고 경구 투여한다. 이 약을 투여하기 1시간 전부터 1시간 후까지 공복상태를 유지한다. 2. 이 약은 레보도파 표준요법의 부가요법으로 투여되고, 레보도파의 효능을 증강시키므로 레보도

파 관련 도파민성 부작용을 즉 운동이상증, 환각, 구역, 구토 및 기립성저혈압을 감소시키기 위해서는 이 약 투여를 시작한 이후 환자의 임상적 상태에 따라, 복용량 당 레보도파의 양을 감소 시키거나 복용 간격을 증가시키므로써 레

보도파의 용량을 조절할 필요가 있다(2. 신중투여할 참고). 3. 이 약의 복용을 잊었을 경우: 이 약의 복용을 잊었을 경우, 다음 복용을 예정대로 복용한다. 놓친 용량을 보충하기 위해 권장량을 초과하여 투여해서는 안된다. 4. 고령

자: 고령자에 대한 이 약의 용량조절은 요구되지 않는다(8. 전문가를 위한 정보참고). 85세 이상의 고령자에 대해서는 임상적 경험에 제한적이며 주의해서 투여해야 한다. 5. 소아: 소아에 대한 이 약의 사용은 권장되지 않

는다. 6. 신장에 환자: 이 약은 신장으로 배설되는 양이 낮아(8. 전문가를 위한 정보참고) 신장에 환자에 대한 이 약의 용량조절은 요구되지 않는다. 7. 간장에 환자: 경증의 간손상 환자(Child-Pugh A)에 대한 이 약의 용량조

절은 요구되지 않으며, 중등도 간손상 환자(Child-Pugh B)에 대한 이 약의 임상적 경험은 제한적이며 투여 시 용량조절이 필요할 수 있다. 중증의 간손상 환자(Child-Pugh C)에 대한 임상적 경험은 없으므로 이 약의 사용은 권

장 되지 않는다(8. 전문가를 위한 정보참고). **【사용상의 주의사항】** 1. 다음 환자에는 투여하지 말 것: 1) 주성분 또는 부형제, 캡슐 기재 등에 과민성인 환자 2) 크롬천화성세포증, 부신경절종 또는 다른 카테콜아민분비 종양

환자 3) 신기능저하 약성증후군 및/또는 비외상성 혈로근용해증 환자 4) 파킨슨병 치료제 이외의 모노아민 산화효소(MAO-A 및 MAO-B) 억제제 (예: 페닐진, 트라닐시프롤린, 모클로베מיד)를 투여 중인 환자(상호작용 참고) 5)

인부 6) 수유부 7) 이 약을 함양하고 있으므로, 갈락토스 불내성(galactose intolerance), 유당불내성 결핍증(total lactase deficiency) 또는 포도당갈락토스 흡수장애(glucose-galactose malabsorption) 등

의 유전적인 문제가 있는 환자에는 투여하면 안 된다. (후략) **【제조사】** Bial - Portela & C^o, S.A. Àv. da Siderurgia Nacional 4745-457 S. Mamede do Coronado Portugal

정주시 흥덕구 산단로 149 **【판매처】** 에스케이케미칼(주) 경기도 성남시 분당구 판교로 310

* 처방하기 전 제품설명서 전문을 참고하십시오. 최신 허가사항에 대한 정보는 '의약품안전나라' (<https://nedrug.mfds.go.kr/index/>)에서 확인할 수 있습니다.

ADD ON TO LIFE

"I'M SO HAPPY! DAD'S RESPONDING
BETTER TO MEDICATION AGAIN!"

Is Ongentys[®] just another COMT* inhibitor?

- Ongentys[®] 50 mg is a **once daily-dosing** COMT inhibitor¹
- Ongentys[®] delivered **smooth and sustained** COMT inhibition vs entacapone^{2**}
- Ongentys[®] induced greater levodopa bioavailability vs entacapone^{2***}
- Ongentys[®] met predefined criteria for non inferiority vs entacapone in OFF-time reductions³
- Patient who **switched from entacapone** and ended the OLE study on Ongentys 50mg **achieved a significant additional 68.2 minutes OFF-time reduction per day** (n=40, LS mean change, -68.2 min; p=0.0025)⁴
- Contrary to tolcapone, **no liver monitoring** is required with Ongentys[®]1,5

Ongentys is indicated as adjunctive therapy to preparations of levodopa/DOPA decarboxylase inhibitors (DDCI) in adult patients with Parkinson's disease and end-of-dose motor fluctuations who cannot be stabilised on those combinations.

1. Ongentys[®] Summary of Product Characteristics. 2. Rocha J et al. *Eur J Clin Pharmacol* 2014;70:1059-71. 3. Ferreira J et al. *Lancet Neurol* 2016;15:154-65. 4. Ferreira et al, 2019. Switching from double-blind entacapone or placebo to open-label opicapone: change in OFF-/ON-time from patients who ended the 1-year BIPARK-I extension on opicapone 50 mg. Poster presented at Presented at the XXIV World Congress of Neurology (WCN 2019), Dubai, United Arab Emirates. 27-31 October 2019. 5. Tasmar[®] Summary of Product Characteristics.

* Catechol-O-Methyltransferase

** Randomized, double-blind, gender-balanced, parallel-group study performed in 4 groups of 20 healthy subjects each. Eligible subjects were enrolled into one of the dosing groups. From the 20 healthy subjects per group, 4 received placebo (PLC) during the entire

study. 16 subjects in one of the groups received PLC once daily for 11 days, and on day 12, 200 mg entacapone (ENT) concomitantly with each levodopa/carbidopa (LC) dose (3 times separated by a 5-h interval). 16 subjects in each of the remaining three groups received respectively 25, 50, and 75 mg opicapone (OPC) once daily for 11 days, and on day 12, PLC concomitantly with each LC dose.

*** Pharmacokinetic studies carried out in healthy patients.

AUC Opicapone 50 mg vs levodopa/carbidopa 100/25 mg + entacapone 200 mg: 148 (CI 106; 208)

AUC Opicapone 50 mg vs levodopa/carbidopa 100/25 mg: 165 (CI 139; 195)

AUC Opicapone 50 mg vs levodopa/benserazide 100/25 mg: 165 (CI 136; 201)

[Ongentys capsule 50 mg] Product information

Product name Ongentys capsule 50 mg (Opicapone) **Ingredient and Contents** 1 capsule (321 mg) of Ongentys contains active ingredient : Opicapone 50 mg **Indication** adjunctive therapy to preparations of levodopa/ DOPA decarboxylase inhibitors (DDCI) in adult patients with Parkinson's disease and end-of-dose motor fluctuations who cannot be stabilized on those combinations **Administration** 1) The recommended dose of opicapone is 50 mg. Ongentys should be taken once-daily at bedtime at least one hour before or after levodopa combinations. 2) Dose adjustments of antiparkinsonian therapy: Opicapone enhances the effects of levodopa. Hence, it is often necessary to adjust levodopa dosage within the first days to first weeks after initiating the treatment with opicapone. 3) Missed dose: If one dose is missed, the next dose should be taken as scheduled. The patient should not take an extra dose to make up for the missed dose. 4) Elderly: No dose adjustment is needed for elderly patients. Caution must be exercised in patients ≥ 85 years of age as there is limited experience in this age group. 5) Paediatric population: Ongentys is not recommended in Paediatric patients 6) Renal impairment: No dose adjustment is necessary in patients with renal impairment, as opicapone is not excreted by the kidney. 7) Hepatic impairment: No dose adjustment is necessary in patients with mild hepatic impairment (Child-Pugh Class A). There is limited clinical experience in patients with moderate hepatic impairment (Child-Pugh Class B). Caution must be exercised in these patients and dose adjustment may be necessary. There is no clinical experience in patients with severe hepatic impairment (Child-Pugh Class C), therefore, Ongentys is not recommended in these patients. **Contraindication** 1) Hypersensitivity to the active substance or to any of the excipients. 2) Pheochromocytoma, paraganglioma, or other catecholamine secreting neoplasms. 3) History of neuroleptic malignant syndrome and/or non-traumatic rhabdomyolysis. 4) Concomitant use with monoamine oxidase (MAO-A and MAO-B) inhibitors (e.g. phenelzine, tranylcypromine and moclobemide) other than those for the treatment of Parkinson's disease. 5) Pregnant, possibly pregnant or lactating women 6) Patients with Genetic problem such as galactose intolerance, Lapp lactase deficiency, glucose-galactose malabsorption **Manufacturer** Bial - Portela & C^ª, S.A., Áv. da Siderurgia Nacional 4745-457 S. Mamede do Coronado Portugal **Seller** SK Chemicals Co., Ltd. 310 Pangyo-ro, Bundang-gu, Seongnam-si, Gyeonggi-do

※ Please refer to the full product manual before prescribing. Information on the latest approvals can be found at the 'Integrated Drug Information System (<https://nedrug.mfds.go.kr>)'.

Stalevo®

Stabilized *levodopa* response during long-term treatment¹⁻³

For Parkinson's disease patients experiencing end-of-dose **wearing-off** on levodopa/DDCI treatment^{1,2,4}

With 6 dosage strengths of levodopa, Stalevo® provides dosing flexibility.⁴

50 mg

75 mg

100 mg

125 mg

150 mg

200 mg

Illustration is an artistic rendition. Not necessarily representative of clinical effects.

DDCI, dopa decarboxylase inhibitor.

References 1. Larsen JP, et al. The tolerability and efficacy of entacapone over 3 years in patients with Parkinson's disease. Eur J Neurol. 2003;10(2):137-146. 2. Rinne UK, et al. Entacapone enhances the response to levodopa in parkinsonian patients with motor fluctuations. Ncomet Study Group. Neurology. 1998;51(5):1309-1314. 3. Fedotova Elu, et al. Zh Nevrol Psikiatr Im S S Korsakova. 2012;112(5):50-5. 4. 스타레보 필름코팅정100/25/200밀리그램 제품정보. Available at: <https://medrug.mfds.go.kr/bpp/CCBB01/getItemDetailCache?cacheSeq=200410072au> pdateTs2023-12-31%2011:34:41.0b (accessed on 7 Feb 2024)

스타레보 필름코팅정 50/12.5/200 mg, 75/18.75/200 mg, 100/25/200 mg, 125/31.25/200 mg, 150/37.5/200 mg, 200/50/200 mg [주성분] 레보도파/도파탈탄산효소(Dopadecarboxylase; DDC)억제제 표준치료제로 개선되지 않는 파킨슨병 환자의 운동 증상(end-of-dose motor fluctuation)의 치료 [용법·용량] 이 약은 1회 1정, 하루 3~4회, 식사 후 복용한다. 일반적으로 레보도파/도파탈탄산효소억제제 표준치료제로 치료 받고 있는 환자에 사용된다. 시간 용량 중 하나를 선택하여 최대 1일 복용량을 투여한다. 스타레보 50/12.5/200 mg, 75/18.75/200 mg, 100/25/200 mg, 125/31.25/200 mg의 일일 최대 권장용량은 10정이며, 스타레보 200/50/200 mg의 일일 최대 권장용량은 7정이다. 기존에 엔타카폰을 복용하고 있지 않던 환자가 스타레보의 복용을 시작할 경우 다른 항파킨슨병 약물의 용량 조절이 필요할 수도 있다. 18세 이하 환자에 대해 스타레보의 투여는 권장되지 않는다. [금기] 이 약의 성분에 과민성인 환자, 일부 및 수유부, 중증의 간장애 환자, 혈우각성 뇌내장, 미진단성 피부질환이 의심되거나 흑색증의 병력이 있는 환자, 크롬친화성 세포종 환자, 비선택적 모노아민산화효소(MAO-A 및 MAO-B) 억제제 및 선택적인 MAO-A 억제제 또는 선택적인 MAO-B 억제제와의 병용 투여, 신경이완제(성중추)인 neuroleptic malignant syndrome(NMS) 및/또는 비의상성 혈문관용해증 병력이 있는 환자, 과당 불내성, 포도당-갈락토스 흡수장애 또는 수크랄제-이소말타이제 기능부전의 희귀한 유전적 장애가 있는 환자, [경고 및 주의] 약물에 의한 부작용으로 증상의 치료에는 권장되지 않는다. 위궤양 혹은 심부전장애, 경련, 심근경색 및 심실부정맥의 병력이 있는 환자, 허혈성 심질환, 중증 심혈관계 또는 폐질환, 기관지천식, 간장애 또는 신장애, 내분비계 질환, 만성 광우각성 뇌내장이 있는 환자, 정신질환을 앓고 있거나 그 병력이 있는 환자, 전신 마취를 요하는 경우, 장기 치료 시 혹은 중대한 중단하는 경우에는 주의해야 한다. 정신적 변화, 자살성향을 동반한 우울증, 기타 반사회적인 행동들이 나타나는지 주의 깊게 모니터링 해야 한다. 살사를 보인 환자는 제증에 대한 관리가 권장된다. 약 투여와 연관성이 의심되는 지속적인 살사가 나타날 경우, 투여를 중단해야 한다. 상대적으로 짧은 기간에 진행성 식욕부진, 무력증 및 체중 감소를 경험한 환자들에는 간기능 검사(포함하여 전반적인 건강 검진)가 고려되어야 한다. 중독 조절 장애(예: 병적인 도박, 성욕 증가, 성행동장애)의 발생을 주기적으로 모니터링 해야 한다. 운전 및 기계 조작 시 주의한다. 졸음, 갑작스런 수면증상 등이 나타나는 환자는 이러한 증상이 없어질 때까지 자동차 운전 및 위험이 수반되는 기계조작을 하지 않도록 주의한다. [상호작용] 레보도파를 고혈압 약제와 병용할 경우 중추성 기압성 저혈압이 발생할 수 있다. MAO-A 억제제, 심혈계 항우울제, 노르아드레날린 제제와 병용 시 주의해야 한다. 이 약은 설파닐아마이드(MAO-B 억제제)와 병용 투여 시 1일 용량 10 mg를 초과해서는 안 된다. 약물 도파민 수용체 차단제(항진단약) 및 항구토제, 비마도인, 파파베린과 병용 투여 시 약효의 감소에 대해 주의 깊게 모니터링 하도록 한다. 와파린을 투여받고 있는 환자가 스타레보 투약을 시작할 경우 INR 수치의 조절이 권장된다. 이 약과 철제제는 적어도 2-3 시간의 간격을 두고 투여해야 한다. 고단백 식단에 의해 이 약이 흡수가 저해될 수 있다. [이상반응] 빈혈, 혈소판감소증, 신경이완제(성중추)인 neuroleptic malignant syndrome(NMS), 혈문관용해증, 심근경색증, 불규칙한 심장 리듬, 기압성 저혈압, 고혈압, 갑작스런 졸음, 정신 장애(예: 기억력 장애, 치매), 정신병, 불안, 자살 성향을 포함하거나 포함하지 않는 우울증, 운동이상증, 파킨슨병 악화, 떨림, on and off 현상, 경련, 졸음, 현기증, 근긴장이상, 불면증, 환각, 혼돈, 초조, 비정상적인 꿈, 호흡곤란, 위장관계출혈, 위장관계 증상(구역, 구토, 소화불량, 복통, 변비, 설사, 구강건조), 연하곤란, 체중 및 식욕 감퇴, 두통, 노 이외의 변색(피부, 손톱, 머리칼, 땀), 시야흐림, 피로, 무력증, 다한증, 쓰러짐, 대장염, 병적다발, 성욕증가 및 성욕과다증, 발진, 두드러기, 근육, 근골격계 및 결합조직 통증, 근육 연축, 관절염, 요로감염, 요저류, 가슴통증, 말초부종, 보행장애, 권태감. ● 보다 자세한 사항은 제품설명서 전문을 참고하시기 바랍니다.



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with Once-Daily Regimen⁴⁻⁶**

| 50 mg | 150 mg | 200 mg | 300 mg | 400 mg |

Illustration is an artistic rendition. Not necessarily representative of clinical effects.

XR, extended release.

References

1. Cutler AJ, et al. Clin Ther. 2011;33(11):1643-1658. 2. Young AH, et al. J Clin Psychiatry. 2010;71(2):150-162. 3. Lindstrom L, et al. J Affect Disord. 2017;213:138-150.
4. Figueroa C, et al. Prog Neuropsychopharmacol Biol Psychiatry. 2009;33(2):199-204. 5. Peuskens J. Neuropsychiatr Dis Treat. 2011;7:549-564.
6. 세로켈서방정: 50 밀리그램, 150 밀리그램, 200 밀리그램, 300 밀리그램, 400 밀리그램 (쿠에티아핀푸마르산염) 제품정보. Available at: <https://medrug.mfds.go.kr> (accessed on 7 Oct 2024)

세로켈®정
(쿠에티아핀푸마르산염)



세로켈서방정®
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엔틱사정은 중등도-중증 알츠하이머병 환자에서 사용 가능한 메만틴 성분의 치료제입니다.¹

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QD, once daily; BID, twice daily.

References

1. 엔틱사정20밀리그램(메만틴염산염) Available at: <https://medrug.mfds.go.kr> (accessed on 18 Apr 2025)
2. Gomolin IH, et al. J Am Geriatr Soc. 2010;58(9):1812-1813.
3. Jones RW, et al. Int J Geriatr Psychiatry. 2007;22(3):258-262.
4. Bassil N, et al. Expert Opin Pharmacother. 2010;11(10):1765-1771.
5. Srivastava K, et al. Patient Prefer Adherence. 2013;7:419-434.

Product Information

엔틱사정20밀리그램
(메만틴염산염)



※ 처방하시기 전 제품에 대한 자세한 내용은 제품설명서 전문을 참조하시기 바랍니다.

Requip PD 는 Motor & Non-motor symptom 개선을 통해 환자의 QOL을 향상시킵니다. 1-6



한인제약

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혼자서는 화장실조차
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행복찾기!

할아버지가 언제나 행복하게
아침을 맞이할 수 있도록
선생님의 도움이 필요합니다.

REQUIP PD 24 HOUR
ropinirole

리quipPD정 투여 금기⁷

1. 로피니롤 또는 이 약의 구성성분에 과민한 자 2. 중증 신기능 저하 환자(크레아티닌 청소율 <30ml/분) 3. 간기능 저하환자 4. 임부, 수유부 및 임신 가능성이 있는 여성 5. 이 약은 유당을 함유하고 있으므로, 갈락토오스 불내성(galactose intolerance) Lapp 유당분해효소 결핍증(Lapp lactase deficiency) 또는 포도당-갈락토오스 흡수장애(glucose-galactose malabsorption) 등의 유전적인 문제가 있는 환자에게는 투여하면 안 된다.

* 여기서 언급된 환자 및 증상은 가상의 인물입니다.

References. 1. Ray Chaudhuri K, Improvements in nocturnal symptoms with ropinirole prolonged release in patients with advanced Parkinson's disease, Eur J Neurol. 2012 ;19(1):105-113
2. Pahwa R et al, Ropinirole 24 hour prolonged release: randomized, controlled study in advanced Parkinson disease, Neurology 2007;68(14):1108-15. 3. Gallagher DA; CNS Drugs; 2008;22:563-586
4. Rektorova I et al. Clinical Neuropharmacol 2008;31(5):261-6 5. Pablo Martinez-Martin, et al. Movement Disorders 2011;26(3): 399-406 6. Chaudhuri KR et al. European Journal of Neurology 2012;19:105-113
7. Requip PD 2mg, 4mg, 8mg Korea Product Information

Safety Information⁷

Reference. 리quip PD정 2mg, 4mg, 8mg 국내 허가사항 중 사용상의 주의사항 1. 경고1) 로피니롤의 말초 도파민 효능작용으로 인하여 중증의 심혈관계 질환 환자는 주의하여 처치한다. 2) 항고혈압약 및 항부정맥약과의 병용투여는 연구되지 않았다. 다른 도파민 효능약과 마찬가지로 병용투여 시에 저혈압, 서맥 또는 기타 부정맥을 유발할 가능성이 있으므로 주의한다. 3) 중요한 정신질환환자는 치료상의 유익성이 위험성을 상회한다고 판단되는 경우에만 도파민 효능약으로 처치한다. 4) 갑작스런 수면 발생 2. 투여금기 1) 로피니롤 또는 이 약의 구성성분에 과민한 자 2) 중증 신기능 저하 환자(크레아티닌 청소율 <30ml/분) 3) 간기능 저하 환자 4) 임부, 수유부 및 임신가능성이 있는 여성 5) 유당 관련 질병 및 장애 등의 유전적인 문제가 있는 환자 3. 신중투여 1) 중증 심혈관계 질환(특히 심장동맥질환) 환자에서 특히 치료 초기에서의 저혈압 위험 때문에 혈압 모니터링이 권장된다. 2) 이 약(서방정) 4mg은 황색5호(설텔로우 FCF, Sunset Yellow FCF)를 함유하고 있으므로 이 성분에 과민하거나 알레르기 병력이 있는 환자에는 신중히 투여한다. 4. 이상반응 매우 흔하게(>1/10), 흔하게(>1/100, <1/10), 때때로(>1/1,000, <1/100), 드물게 (>1/10,000, <1/1,000), 매우 드물게(<1/10,000), 기타로 구분하였다. 1) 단독요법 : 매우 흔하게 - 졸음, 오심, 흔하게 - 환각, 어지러움, 갑작스런 수면, 변비, 말초 부종, 때때로 - 기립성 저혈압, 저혈압 2) 병용요법 : 매우 흔하게 - 운동이상증, 흔하게 - 환각, 졸음, 어지러움, 갑작스런 수면, 기립성 저혈압, 저혈압, 오심, 변비, 말초 부종

수입의약품

전문의약품

Product Information

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(로피니롤염산염)



리quipPD정4일리그래
(로피니롤염산염)



리quipPD정8일리그래
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- 3/ 국내 최초 12.5mg 출시 및 6가지 전 함량 보유

References 1. Kishi T, et al. *Mol Psychiatry*, 2022 Feb;27(2):1136-1144. 2. D Popovic, et al. *Acta Psychiatr Scand* 2014; 129: 368-374. 3. 쿠에타핀[®]정 제품설명서. 4. IQVIA 2021-2024년, Sales 기준, Quetiapine IR 부문

쿠에타핀정 (쿠에타이핀푸마르산염) 12.5mg / 25mg / 50mg / 100mg / 200mg / 300mg

원료약품 및 분량: 쿠에타이핀 12.5mg, 25mg, 50mg, 100mg, 200mg, 300mg 성상: 흰색의 원형 필름코팅정제, 주황색의 원형 필름코팅정제, 흰색의 원형 필름코팅정, 노란색의 원형 필름코팅정제, 흰색의 장방형의 필름코팅정제, 흰색의 장방형의 필름코팅정제 **효능 - 효과:** 정신분열병, 양극성장애 (양극성장애 1형과 관련된 조증삽화의 급성 치료, 양극성장애의 우울삽화의 급성 치료, 쿠에타이핀 투여로 조증, 혼재 또는 우울삽화에 반응을 보인 환자들에 있어서, 양극성장애의 재발방지) **경고:** 1. 자살상향 및 항우울제 2. 고혈당 및 당뇨병 3. 신경이완제 악성 증후군(Malignant Neuroleptic Syndrome) 4. 후체외로 증상 및 지연성운동이상증(Tardive Dyskinesia) 5. 치매 노인 환자 6. 중증 호흡기감염증 및 무과립구증 7. 뇌혈관 질환 이상반응 위험 증가 8. 장액혈전증 위험 9. 동반 질환: 이 약은 심혈관계 질환, 뇌혈관 질환, 다른 저혈압 요인이 있는 환자에서는 주의하여 사용해야 한다. 쿠에타이핀은 특히 초기 중량 기간 동안 기립성 저혈압을 야기할 수 있다 10. 중증피부유해반응(Severe Cutaneous Adverse reaction, SCAR) **금기:** 1. 이 약 또는 이 약의 구성성분에 과민반응 환자 2. 혼수상태의 환자가 이 약이 혼수를 악화시킬 수 있다 3. 바르비투르산염 등의 중추신경억제제의 강한 영향 아래 있는 환자(중추신경억제작용이 증강될 수 있다) 4. 에피네프린을 투여 중인 환자 5. 당뇨병 환자 또는 당뇨병 병력이 있는 환자 6. 이 약은 유당을 함유하고 있으므로, 갈락토오스 불내성(galactose intolerance), Lapp 유당 분해 효소 결핍증(Lapp lactase deficiency), 포도당-갈락토오스 흡수장애(glucose-galactose malabsorption) 등의 유전적인 문제가 있는 환자에게는 투여하면 안 된다. **신중투여:** 1. 간장애환자 (이 약은 간에서 주로 대사되므로 소실을 감소에 따라 혈중 농도가 증가할 수 있다. 저장량으로 시작하여야 하며, 환자의 상태를 모니터링하면서 점차적으로 증량한다) 2. 심혈관계 질환, 뇌혈관계 질환 혹은 저혈압이거나 저혈압의 위험이 있다고 알려진 환자(최초 투여동안 일과성 저혈압이 나타날 수 있다) 3. 간질이나 유사 질환 병력을 포함하는 경련성 질환 환자 4. 고령자 5. 자살 시도 또는 자살 관념화 환자 6. 당뇨병 기록, 고혈당 혹은 비만 등의 당뇨병의 위험인이자 있는 환자 7. 일부 또는 임신하고 있을 가능성이 있는 여성 및 수유부 **저장방법:** 기밀용기, 실온 (1~30°C) 보관

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REFERENCES

1. Chenu CV et al. *Euro Neurol Rev* 2016;11(Suppl. 2):2-6. 2. Calabresi P et al. *Euro Neurol Rev* 2017;12(Suppl. 5):2-6. 3. Perez-Llovet S et al. *Expert Rev Neurother* 2016;16(2):245-258. 4. Borghani R et al. *Mov Disord* 2014;29(7):229-237. 5. Schapira AHV et al. *JAMA Neurol* 2017;74(2):216-224. 6. Cattaneo C et al. *J Parkinsons Dis* 2016;6(1):165-173. 7. Cattaneo C et al. *J Parkinsons Dis* 2017;7(1):95-101. 8. Cattaneo C et al. *J Parkinsons Dis* 2017;7(4):629-634. 9. Ruppam Borghani et al. *Mov Disord* 2014;29(10):1273-1280. 10. Cattaneo C et al. *J Parkinsons Dis* 2020;10(1):89-97.

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REFERENCE 1. Seltzer B, et al. Efficacy of donepezil in early-stage Alzheimer disease: a randomized placebo-controlled trial. *Arch Neurol.* 2004; 61(12):1852-6.

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References. 1. van Dyck CH, Swanson CJ, Aisen P, et al. Lecanemab in early Alzheimer's disease. N Engl J Med. 2023;388(1):9-21. 2. Leqembi Korea Product Information. 24.11.15.



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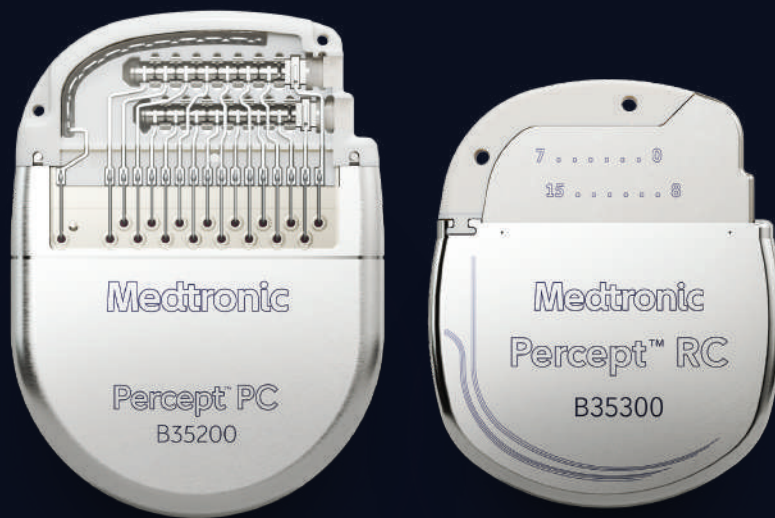
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Reference

1. Feldman H, et al. J Am Geriatr Soc 2003;51:737-44. 2. Mehta S, et al. Indian J Psychiatry 2012;54:337-43. 3. Feldman H, et al. Int J Geriatr Psychiatry 2005;20:559-69. 4. Gauthier S, et al. Int Psychogeriatr 2002;14:389-404. 5. Holmes C, et al. Neurology 2004;63:214-19. 6. Martin M, et al. Alzheimer Disease & Associated Disorders 2011;25:333-40. 7. Whitehead A, et al. Int J Geriatr Psychiatry 2004;19:624-33. 8. Farlow MR. Psychiatry Clin Ther 2010;32:1234-51. 9. DeKosky S. Alzheimer Dis Assoc Disord 2003;17:S99-S104. 10. Winblad B, et al. Neurology 2001;57:489-495.



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REFERENCES 1) Kwon and Son, Biomol Ther21(3), 181-189(2013), in vivo : rat, dog

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