International Parkinson and
Movement Disorder Society
Asian & Oceanian Section

6th Asian and Oceanian Parkinson's Disease and Movement Disorders Congress

April 12-14, 2019 • Hangzhou

Late-Breaking Abstracts

LBA 1
This abstract has been withdrawn.

LBA 2
Transcranial direct current stimulation for limb-kinetic apraxia in Parkinson's Disease – a randomized, double-blinded, sham-controlled trial
J.E. Park, H.R. Jang, L.U. Kim, G.J. Park, S.K. Kim, J.E. Bae, J.Y. Hong, M. Hallett (Goyang)
Objective: To examine the effects of transcranial direct current stimulation on limb-kinetic apraxia (LKA) in Parkinson's disease (PD).
Background: Limb-kinetic apraxia is a commonly overlooked phenomenon in PD, characterized as difficulty performing precise and coordinated finger movements. To date, there are no established treatments for this phenomenon, and levodopa medication has been reported to have no effect.
Method: 28 PD patients completed the study. Patients were randomized to two groups; anodal or sham stimulation. For patients assigned to active stimulation, anodal stimulation of the left posterior parietal cortex was performed using 2 mA current for 20 minutes. Patients receiving sham stimulation also underwent the session for 20 minutes. The investigators and patients were both blinded to group allocation. Both groups were instructed to perform repetitive manual tasks including buttoning and unbuttoning, handwriting and coin flipping following the stimulation session. The primary outcome measure was time performing sequential buttoning and unbuttoning, and several secondary outcome measures were obtained (e.g., Unified Parkinson's Disease Rating Scale: UPDRS scores, resting motor threshold: RMT, time writing one's name 10 times, time flipping a coin using each hand, etc.).
Results: Mean age in the active and sham group were 73 and 72 years, respectively. Mean MMSE scores were 25 and 26, respectively. Mixed ANOVA was performed and a significant interaction was found between stimulation type and conditions (medication OFF, ON, tDCS immediate and post-tDCS 24 hours). Patients who received active (anodal) stimulation were found to have a significant decrease in sequential buttoning and unbuttoning times immediately following tDCS and at 24 hours, compared to the medication-OFF state (31% and 29% decrease, respectively). No statistically significant findings were found in the sham group.
Conclusion: Anodal tDCS of the left posterior parietal cortex, relevant in praxis, appears to be effective in ameliorating LKA in PD. Therefore, future long-term projects performing serial stimulation sessions seem worthwhile.

LBA 3
The nigral hyperintensity on 3 Tesla susceptibility-weighted MRI in patients with vascular parkinsonism
K.J. Kim, Y.J. Bae, J.M. Kim (Goyang-Si)
Objective: The loss of nigral hyperintensity on 3 Tesla susceptibility-weighted MR imaging (3T-SWI) in patients with Parkinson's disease (PD) has been reported. In this study, the status of nigral hyperintensity was investigated in patients with VP.
Background: Vascular parkinsonism (VP) and PD may have similar clinical presentations, although the pathological bases of these two conditions are different.
Method: From November 2013 to November 2017, consecutive patients with vascular parkinsonism were included, and age-/sex-matched PD patients and healthy control subjects were compared. Diagnoses were based on the clinical follow-up and 123I-FP-CIT SPECT results. Two blinded readers assessed the nigral hyperintensity on 3T-SWI.
Results: The study included 46 VP patients, 46 PD patients, and 34 control subjects. VP patients showed more frequent lacunes and white matter changes on MRI, compared to PD patients and control subjects. The nigral hyperintensity was more frequently found in VP patients than in PD patients (82.6% vs. 21.7%, OR 19.870, $P<0.001$), and comparable to control subjects. (82.6% vs. 82.4%, OR 1.309, $P=0.683$)
Conclusion: We could find that the nigral hyperintensity on 3T-SWI was more frequently preserved in VP patients than in PD patients. Our findings suggest that the presence or absence of nigral hyperintensity could be helpful in differentiating VP from PD.

LBA 4
Clinical and radiological analysis of Progressive Supranuclear Palsy cases in Kazakhstan
N. Zharkynbekova, R. Kaiyrzhanov, M. Saifullayeva, C. Shashkin, G. Kaishybayeva (Shymkent)
Objective: To make the first clinical and radiological analysis of 12 cases with Progressive supranuclear palsy (PSP) in Kazakhstan.
Background: Progressive supranuclear palsy (PSP) is a debilitating neurological disorder that distinguishes from Parkinson's disease clinically and pathologically. To date, ten clinical variants of PSP have been described in the literature. Recently, quantitative morphometric and volumetric magnetic resonance imaging (MRI) markers of PSP have been introduced to assist clinicians in differentiating PSP from other neurodegenerative causes of parkinsonism. There is lack of reports concerning the clinical and demographic characteristics of Parkinson's disease and PSP from Kazakhstan.
Method: Demographic, clinical and volumetric MRI brain imaging data from 12 PSP patients who had been under the review in the Almaty city movement disorders center were collected and analyzed.
Results: The mean age of the patients at the moment of the last visit was 70.2 years (SD 4.9, range 62-78), the mean age for the disease onset was 64.4 (SD 4.6, range 58-74). The mean duration of the disease was 3.8 (SD 1.9, range 1-8) and M:F ratio was 2:1. Four patients (33%) had mild response to levodopa, two (17%) had moderate response and 6 (50%) had no response to levodopatherapy. Only 50% of patients presented with tremor. Only a half of the patients had symmetric akinetic-rigid syndrome. Mean Movement Disorders Society Unified Parkinson's Disease Rating Scale Part III score was 55.8 (SD 12.11, 33-79). Full upgaze paresis was present in two female patients with no tremor and the disease duration of five years. Reduced range of vertical gaze was noticed in 7 (58%) patients. The main PSP subtype were PSP – parkinsonism (50%) followed by PSP –Steele –Richardsons' type

(17%). Mean Magnetic Resonance Parkinsonism Index (MRPI) comprised 21.8 (SD 2.4, range 17.29-24.37) and mean Midbrain to Pons (M/P) ratio was 0.07 (SD 0.01, range 0.04-0.1). No positive correlation has been found between MRI volumetric measurements and disease duration, as well between MRI measurements and the severity of parkinsonism (Tb – 0.5, p=0.1).
Conclusion: The study results are coherent with the data from literature.

LBA 5
Differential coding and spatial clustering of reward and movement information in the striatal direct and indirect pathways
J.H. Shin, D.H. Kim, M.W. Jung (Seoul)
Objective: To better understand striatal neural processes underlying reward-based learning and movement control, we examined the responses of D1 and D2 medium spiny neurons (MSNs) in the dorsal striatum of mice performing a probabilistic Pavlovian conditioning task.
Background: The direct and indirect pathways of the basal ganglia have long been thought to mediate behavioral promotion and inhibition, respectively. However, this classic dichotomous model has been recently challenged.
Method: We examined the responses of dSPNs(D1) and iSPNs (D2) medium spiny neurons (MSNs) in the dorsomedial striatum of mice performing a probabilistic Pavlovian conditioning task with electrophysiologic recording and in-vivo calcium imaging. We expressed Channelrhodopsin (ChR2) in the dorsomedial striatum in D1-cre and D2-cre transgenic mouse and optogenetically tagged dSPNs and iSPNs in-vivo. We also recorded dSPNs and iSPNs in the dorsal striatum with in-vivo calcium imaging to see whether there is a spatially organized pattern of dSPNs and iSPNs.
Results: dSPNs and iSPNs, which are components of the direct and indirect pathways, respectively, showed diverse arrays of reward and tongue movement-related activity, but with quantitative differences. dSPNs and iSPNs tended to increase and decrease activity as a function of reward value, respectively, suggesting striatal value representation by relative activity levels between dSPNs and iSPNs. D1 MSNs increased activity more strongly than D2 MSNs in association with lick offset, suggesting the involvement of D1 MSNs in suppressing licking behavior. We optogenetically stimulated bilateral dSPNS during lick bouts and revealed transient suppression of lick during light stimulation. In addition, rapid responses to a negative outcome and previous reward signals were stronger among D2 than D1 MSNs, suggesting stronger contributions of D2 MSNs to outcome-dependent behavioral adjustment. Furthermore, with calcium imaging analysis, we revealed that dSPNs and iSPNs form functional local clusters in the dorsal striatum. These local clusters were most prominent for expected value and were not overlapping in different reward-related parameters and motor-related parameters.
Conclusion: These findings confirm that classic dichotomous model is no longer feasible. Reward and motor information are encoded in the dorsal striatum with complex and context-dependent manner. Parallel basal ganglia loop with multiple exclusive local/functional clusters enables complex and similar encoding of the direct/indirect pathway in basal ganglia.

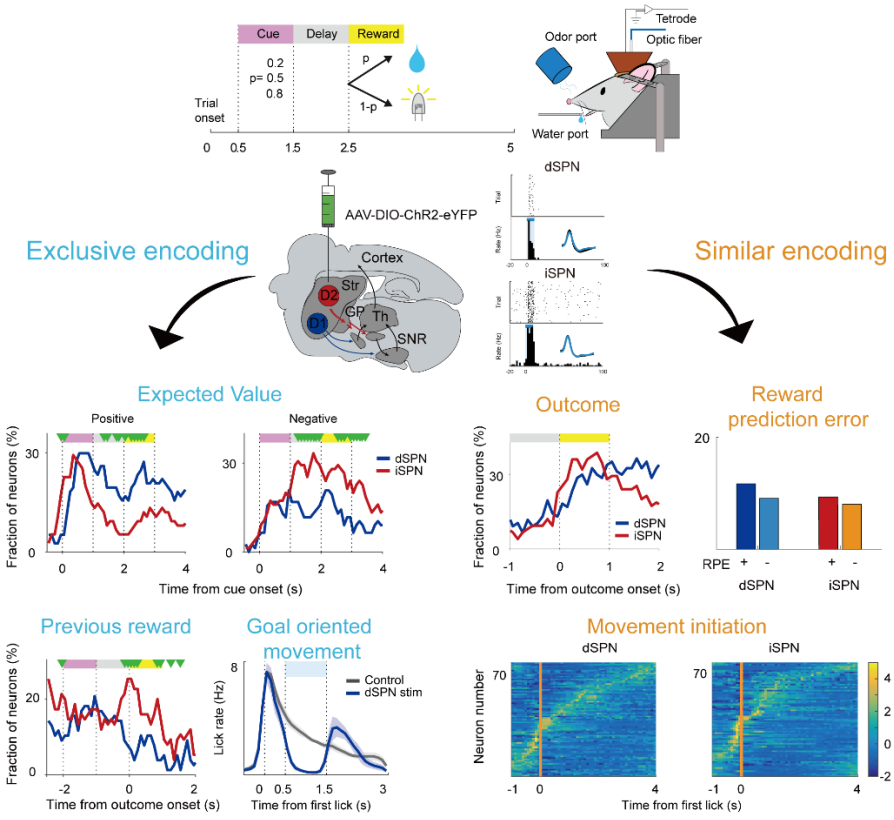

Figure 1

LBA 6

GluRdelta2-regulated climbing fiber pathology generates rhythmic movements in essential tremor

M.K. Pan W.C. Liu, Y.S. Li, C.L. Ni, S.H. Kuo (Taipei)

Objective: To identify the potential causative mechanism of essential tremor (ET).

Background: ET is one of the most prevalent tremor disorders, affecting 4% of individuals aged 40 years or older. The mechanism of ET remains unclear, probably related to the lack of chronic tremor animal model for detailed investigation.

Method: We investigated the cerebellar pathology and related molecular changes in ET patients and aged-matched controls. We investigated the behaviors and cerebellar pathology of a mouse model of corresponding molecular change (GluRdelta2 insufficiency). We also applied intracerebral microinfusion and optogenetic methods to identify the real-time tremor generating circuits in the mouse model.

Results: Here we reported that GluRdelta2-insufficient mice developed tremor in 20 Hz. Similar to ET, the characteristics of mouse tremor was kinetic-predominant, adult onset and worsening with aging. The ET-like tremor in mice responded to ET-medications, including primidone, propranolol and alcohol. Beyond behavioral characteristics, GluRdelta2-insufficient mice recaptured the pathological features in postmortem ET cerebellum: invasion of climbing fibers (CFs) into the parallel fiber territory. Using pharmacological and optogenetic synaptic silencing, we identified that synaptic activities of the hyper-innervated CFs participate in the real-time generation of mouse tremor.

Conclusion: Here we reported that GluRdelta2-insufficient mice developed tremor in 20 Hz. Similar to ET, the characteristics of mouse tremor was kinetic-predominant, adult onset and worsening with aging. The ET-like tremor in mice responded to ET-medications, including primidone, propranolol and alcohol. Beyond behavioral characteristics, GluRdelta2-insufficient mice recaptured the pathological features in postmortem ET cerebellum: invasion of climbing fibers (CFs) into the parallel fiber territory. Using pharmacological and optogenetic synaptic silencing, we identified that synaptic activities of the hyper-innervated CFs participates in the real-time generation of mouse tremor.

LBA 7

Clinical motor outcomes immediately after repetitive transcranial magnetic stimulation (rTMS) for Parkinson Disease in a Filipino cohort

J.A.K. Torres, S.D. Abou Zaki, R. Rosales (Manila)

Objective: The primary objective of this study was to establish the clinical outcomes of patients who underwent rTMS as adjunctive treatment for motor symptoms of PD in a single tertiary care center, in terms of changes in pre- and post-treatment motor component scores (Part 3) of the Movement Disorders Society – Unified Parkinson Disease Rating Scale (MDS-UPDRS).

Background: Repetitive transcranial magnetic stimulation (rTMS) is a non-invasive method of brain stimulation that delivers repetitive trains of stimulation to the cortex (repetitive TMS or rTMS), thereby modulating activity beyond the stimulation period. This modality has been recently available in our institution for the treatment of neuropsychiatric disorders including Parkinson Disease (PD; level of recommendation B).

Method: After ethics approval, chart and medical records review was performed. All eligible patients aged 18 years and above clinically diagnosed with PD by a movement disorder specialist who underwent rTMS at the Neurophysiology Unit in a single tertiary care center from February 2017 to July 2018 were included. Demographic and clinical data as well as MDS-UPDRS motor component scores pre- and post-treatment (immediately after 10 sessions of rTMS) were collected. Repetitive TMS was applied to either or both primary motor cortices (M1).

Results: Subjects were mostly males (67%); Hoehn & Yahr Stage 2.5 (44%); mean age of 64.7 years and mean disease duration of 5.4 years (Table 1). All of the subjects were on levodopa. High-frequency rTMS was delivered to the primary motor cortex in 2,000 pulses per session for 10 sessions. The changes in the motor component scores of the MDS-UPDRS pre- and post-treatment were statistically significant (p-value = 0.00122; Table 3). The mean change in the 2 scores reflected a moderate clinically important difference (Table 3). The primary result of this study among Filipino subjects has substantiated results from clinical trials in Western populations on the contribution of high-frequency repetitive transcranial magnetic stimulation to either or both primary motor cortices to the improvement of motor symptoms in PD. It has been established by functional neuroimaging that excitatory stimulation through high-frequency rTMS of the unilateral or bilateral primary motor cortices facilitates dopamine release in the putamen and caudate thereby addressing the hypodopaminergic state in PD, which thus clinically translates to improvement in motor symptoms such as rigidity and bradykinesia.

The changes in motor component scores of the MDS-UPDRS of the subjects before and immediately after 10 sessions of rTMS were shown to be significant and the mean change of 9.1 points reflected a moderate clinically important difference between the 2 scores. An important factor which may have contributed to these findings is the higher number of pulses delivered during the entire treatment duration. The number of pulses per session and across sessions is a significant predictor of rTMS outcome, with a higher number of pulses per session contributing to larger treatment effects.

Conclusion: High frequency (10 Hz) rTMS to either or both primary motor cortices (M1) at 2,000 pulses per session for a total of 10 sessions provided substantial improvement in the motor symptoms of Parkinson Disease as reflected by the significant changes in the post- and pre-treatment motor component scores of the MDS-UPDRS.

Table 1. Characteristics of subjects with Parkinson Disease (n = 9)

Variable	Value
Age in years, mean ± SD	64.7 ± 8.3
Sex, n (%)	
Male	6 (66.7%)
Female	3 (33.3%)
Disease duration in years, mean ± SD	5.4 ± 2.3
Type of medication, n (%)	
Levodopa	9 (100%)
Dopamine agonist	3 (33.3)
MAO inhibitor	1 (11.1%)
COMT inhibitor	1 (11.1%)
Number of medications, median (IQR)	1 (1 – 3)
On medication at rTMS, n (%)	9 (100%)
Hoehn and Yahr stage, median (IQR)	2.5 (1 – 5)
Stage 1, n (%)	1 (11.1%)
Stage 2, n (%)	1 (11.1%)
Stage 2.5, n (%)	4 (44.4%)
Stage 3, n (%)	2 (22.2%)
Stage 4, n (%)	0
Stage 5, n (%)	1 (11.1%)
With dyskinesia, n (%)	7 (77.8%)
With motor fluctuations, n (%)	6 (66.7%)

Table 3. Motor component score* pre- & post-rTMS treatment for PD (n = 9)

Motor component scores	Hoehn & Yahr Stage	Pre-Treatment score	Post-treatment score$	Change in scores	p-value
Patient 1	2.5	18	13	- 5.0	0.00122
Patient 2	2.5	18	16	- 2.0	
Patient 3	3	21	0	- 21.0	
Patient 4	2.5	9	0	- 9.0	
Patient 5	2.5	7	0	- 7.0	
Patient 6	2	13	7	- 6.0	
Patient 7	3	30	17	- 13.0	
Patient 8	1	7	0	- 7.0	
Patient 9	5	68	56	- 12.0	
			Mean change =	***- 9.1***	

*Part 3 of the Movement Disorder Society - Unified Parkinson Disease Rating Scale (MDS-UPDRS)
$ after 10 sessions

LBA 8

Does gastrectomy delay nigral dopaminergic loss in Parkinson's disease?

P.H. Lee, S.J. Chung, J.S. Baik, J.H. Yoon, J.S. Kim, Y.H. Sohn (Seoul)

Objective: To investigate whether gastrectomy prior to parkinsonian symptom onset could alleviate the neurodegenerative load in Parkinson's disease (PD).

Background: Growing evidence has suggested that Lewy body pathology would arise from the gastrointestinal system and spread to the brain via the vagal nerve, which is modulated by vagotomy.

Method: Fifty-one patients with de novo PD who had undergone gastrectomy prior to motor symptom onset (gastrectomy group) were enrolled from three university hospitals and then, matched with 204 patients with PD without a gastrectomy history (non-gastrectomy group). We performed inter-group comparative analyses of the striatal dopamine transporter availability (28 with a gastrectomy history and matched 112 without) and level of cognitive performance (24 with a gastrectomy history and 107 without). We compared the rates of the longitudinal increases in levodopa-equivalent dose over time between the two groups (follow-up > 2 years) using a linear mixed model.

Results: There was no significant difference in demographic characteristics between the groups. The gastrectomy group had less severely decreased dopamine transporter availability in the posterior putamen compared to the non-gastrectomy group ($p = 0.006$). The gastrectomy group had a slower longitudinal increase in dopaminergic medication doses than the non-gastrectomy group ($p < 0.001$). However, the gastrectomy group showed poorer cognitive performance in attention/working memory ($p = 0.013$), frontal/executive ($p = 0.029$), and memory function domains ($p = 0.023$) relative to the non-gastrectomy group.

Conclusion: Our results suggest that gastrectomy has protective effects on nigrostriatal dopaminergic degeneration and disease progression in subsequent PD, but may be unfavorable for cognitive performance.

TABLE 1. Baseline demographic characteristics, dopamine transporter availability, and longitudinal changes in levodopa-equivalent doses across time in patients with Parkinson's disease

	PD-G+	PD-G-	*p*-value
Demographic characteristics	N = 51	N = 204	
Age (years)	71.22 ± 10.38	70.24 ± 8.98	0.502
Female, % (No.)	31.4% (16)	32.4% (66)	0.893
Age at PD onset (years)	69.33 ± 9.95	68.69 ± 9.10	0.660
PD duration (months)	20.51 ± 26.21	18.55 ± 16.21	0.539
UPDRS-III	23.11 ± 12.34	23.83 ± 9.46	0.715
CCSIT	6.03 ± 2.75	6.12 ± 2.34	0.830
RBD	31.4% (16)	37.3% (76)	0.434
BDI	16.63 ± 9.06	11.89 ± 8.71	0.009

Smoking status			0.480
Ever-smoker	22 (43.1%)	77 (37.7%)	
Never-smoker	29 (56.9%)	127 (62.3%)	
Striatal DAT availability	N = 28	N = 112	
Whole striatum	1.959 (0.094)	1.827 (0.047)	0.210
Anterior caudate	1.995 (0.106)	1.918 (0.053)	0.515
Posterior caudate	1.207 (0.086)	1.289 (0.043)	0.398
Anterior putamen	2.393 (0.120)	2.210 (0.060)	0.176
Posterior putamen	1.741 (0.103)	1.416 (0.052)	0.006
Ventral striatum	2.033 (0.108)	2.012 (0.054)	0.865
Δ LED	N = 29	N = 204	
Estimated beta (SE)	4.9569 (0.4699)	7.0019 (0.2699)	< 0.001

The values are expressed as mean ± standard deviation or percentage (number) for the baseline demographic characteristics, and estimated mean (standard error) for the DAT availability. The estimate (β) is the change in LED per month (Δ LED), i.e., positive value indicates the dose-up of PD medications, and the PD-G+ group exhibited a slower rate of dose-up of PD medications than the PD-G- group (difference: -2.0450 [0.5359], p < 0.001). Abbreviations: PD, Parkinson's disease; PD-G+, PD with a previous history of gastrectomy; PD-G-, PD without a previous history of gastrectomy; UPDRS-III, Unified PD Rating Scale Part III; CCSIT, the cross-cultural smell identification test; RBD, rapid eye movement behavior disorder; BDI, Beck Depression Inventory; DAT, dopamine transporter; LED, levodopa-equivalent dose; SE, standard error.

LBA 9

Type 1 Sialidosis patient with a novel deletion mutation in the NEU1 Gene: case report and literature review

J.H. Ahn, A.R. Kim, C. Lee, N.K.D. Kim, W.Y. Park, M. Kim, J. Youn, J.W. Cho, J.S. Kim (Seoul)

Objective: We aimed to review the previous reports of type 1 sialidosis and compare those with the first case of type 1 sialidosis in Korea.

Background: Recent advances in next-generation sequencing technologies have uncovered the genetic backgrounds of various diseases. Type 1 sialidosis (OMIM#256550) is a rare autosomal recessive lysosomal storage disease caused by a mutation in the NEU1 (OMIM * 608272) gene.

Method: Literature was obtained using the electronic database PubMed (http://www.ncbi.nlm.nih.gov/pubmed/) and Google scholar, and searched keywords included sialidosis, cherry-red spot myoclonus and NEU1. We selected clinical cases or family reports that included genetically confirmed type 1 sialidosis patients and excluded duplicate reports. We summarized selected reports and compared the clinical and genetic characteristics to the patient in our case.

Results: A 36-year-old woman presented with progressive ataxia, myoclonus and seizure since the age of 12. Whole exome sequencing revealed a pathogenic missense variant c.928G>A (p.D310N) and novel c.15_16del (p.P6Qfs*21) of the NEU1 gene as final causal candidate as compound heterozygotes. We reviewed the literature and selected the clinical reports of genetically confirmed type 1 sialidosis patients. A total of 45 patients in 17 reports were identified. Cherry-red spot, myoclonus, ataxia and seizure were reported in 51.2%, 100.0%, 87.8% and 73.7% of patients, respectively. Abnormalities of cognitive function, EEG, and brain MRI and visual symptoms were reported in 22.2%, 40.7%, 66.7% and 69.4% of patients, respectively.Overall, our patient showed similar clinical features to previous type 1 sialidosis patients, but she did not complain of visual symptoms despite having cherry-red spots.

Conclusion: We summarize the clinical features of type 1 sialidosis and report the first case of type 1 sialidosis with novel deletion variant in the NEU1 gene in the Korean population. Our study suggests the importance of ophthalmologic examinations in patients with myoclonus, ataxia and seizure who do not complain of visual symptoms.

Table 1. Characteristics of reported patients with genetically confirmed type 1 Sialidosis

Reference	Numbuer of Case	Ethnicity	Mutation	Type of mutation	Mean AOO	Mean AOE	Sex (M/F)	Cherry red spot	myoclonus	ataxia	Seizure	Abnormal Cognition	Visual Symptoms	Abnormal EEG	Abnormal MRI
Naganawa et al. [4]	2	Japanese	V217M, G243R	Missense	24.5 (17-32)	33.5 (25-42)	1/1	2/2	1/1	1/1	1/1	n.a	1/1	n.a	0/1
Bonten et al. [5]	6	2 African American, 1 Greek, 2 German, 1 Dutch	R294S/L231H R294S/G218A G227R V54M/G378stop G328S/Dpl399HY	Missense Duplication	12.3 (8-17)	n.a	1/5	4/5	5/5	4/4	5/5	1/2	3/3	2/2	n.a
Palmeri et al. [6]	1	Italian	G328S	Missense	17	21	0/1	1/1	1/1	1/1	1/1	1/1	1/1	1/1	1/1
Itoh et al. [7]	1	Japanese	P316S	Missense	14	18	1/0	1/1	1/1	1/1	1/1	n.a	1/1	n.a	n.a
Lai et al. [8]	17	Taiwan	S182G S182G/A319V S182G/Q55*	Missense	19.1 (12-33)	38.1 (25-51)	12/5	3/17	17/17	16/17	13/17	2/17	14/17	3/17	7/17
Ranganath et al. [9]	1	Indian	R294C/N398Tfs*90	Missense Deletion	12	14	0/1	1/1	1/1	1/1	n.a	n.a	n.a	n.a	n.a
Sekijima et al. [10]	1	Japanese	P80L/D135N	Missense	14	17	1/0	1/1	1/1	1/1	n.a	1/1	0/1	n.a	1/1
Sobral et al. [11]	1	Portugal	D234N/R341X	Missense	26	53	1/0	1/1	1/1	1/1	n.a	n.a	1/1	1/1	1/1
Canafoglia et al. [12]	6	Italian	S67I G227R/R305C	Missense	25.3(22-31)	34.3 (27-42)	2/4	0/5	6/6	2/6	1/6	n.a	0/6	n.a	1/6
Schene et al. [13]	2	Dutch	H399_Y400dup/E227R	Missense Duplication	12.5 (12-13)	14.5 (14-15)	0/2	2/2	1/1	1/1	n.a	n.a	2/2	n.a	0/2
Mütze et al. [14]	1	German	S233R/Y268C	Missense	6	8	1/0	1/1	1/1	1/1	0/1	0/1	1/1	1/1	0/1
Gowda et al. [15]	1	Indian	G248C	Missense	9	9	1/0	1/1	1/1	1/1	1/1	0/1	n.a	0/1	0/1
Aravindhan et al. [16]	1	Ecuadorian	P210L	Missense	16	39	1/0	1/1	1/1	1/1	1/1	0/1	1/1	1/1	0/1
Hu et al. [17]	1	Taiwan	S182G/ Q207*	Missense	12	15	1/0	1/1	1/1	1/1	1/1	1/1	n.a	1/1	1/1
Mohammad et al. [18]	1	East-Asian	S182G/G227R	Missense	12	20	0/1	0/1	1/1	1/1	1/1	1/1	0/1	0/1	0/1
Gultekin et al. [19]	1	Turkish	E209Sfs*94 /D310N	Missense Deletion	18	24	1/0	1/1	1/1	1/1	1/1	n.a	n.a	n.a	1/1
Index Patient	1	Korean	R6Qfs*21 / D310N	Missense Deletion	12	36	0/1	1/1	1/1	1/1	1/1	0/1	1/1	1/1	1/1
Mean					17.6 (6-33)	31.8 (8-51)	24/21	22/43 (51.2%)	46/46 (100%)	36/41 (87.8%)	28/38 (73.7%)	6/27 (22.2%)	26/37 (70.2%)	11/27 (40.7%)	24/36 (66.7%)

AOO, Age of onset; AOE, Age of examination; EEG, electroencephalography; MRI, Magnetic Resonance Imaging

Table 2. Comparison between early onset and late onset group

Group	Early onset (n = 31)	Late onset (n = 14)	*p*-value
Mean AOO	13.8 ± 3.2 (n = 31)	26.0 ± 4.2 (n = 14)	0.000
Mean AOE	26.5 ± 11.36 (n = 25)	40.1 ± 7.5 (n = 14)	0.000
Sex (M)	16/31 (51.6%)	8/14 (57.1%)	0.983
Cherry-red spot	20/30 (66.7%)	2/14 (14.3%)	0.004
Myoclonus	28/28 (100%)	14/14 (100%)	-
Ataxia	28/28 (100%)	8/13(61.5%)	0.002
Seizure	23/26 (88.5%)	5/12 (41.7%)	0.005
Abnormal Cognition	8/22 (36.4%)	0/6 (0.0%)	0.141
Visual Symptoms	20/23 (87.0%)	4/13 (30.8%)	0.001
Abnormality on EEG	7/9 (77.8%)	1/1 (100%)	1.000
Abnormality on MRI	5/11 (45.5%)	2/8 (25.0%)	0.633

AOO, Age of onset; AOE, Age of examination; EEG, electroencephalography; MRI, Magnetic Resonance Imaging

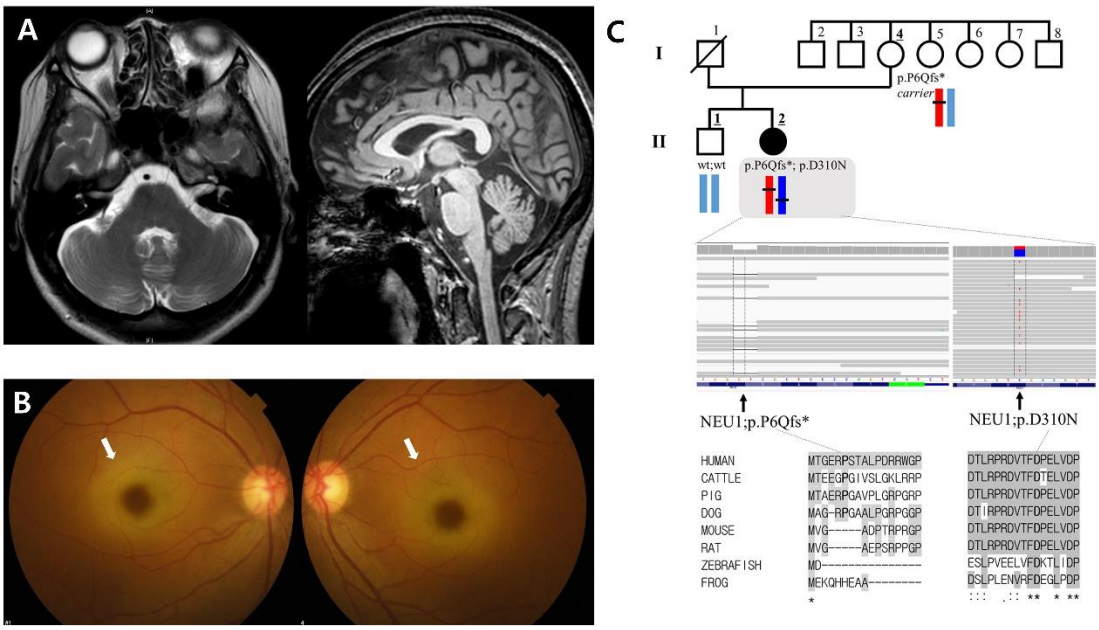

Figure 1

LBA 10

Primary familial brain calcification in China: prevalence, severe phenotypes with biallelic variants

S. Chen, Z.D. Cen, Y. Chen, F. Fu, Q.Q. Pan, X.H. Chen, D.H. Yang, H.T. Wang, H.W. Wu, X.S. Zheng, F. Xie, Z.Y. Ouyang, J.Y. Liu, W. Luo, (Hangzhou)

Objective: Primary familial brain calcification (PFBC) is a rare calcifying disorder of the brain with extensive clinical and genetic heterogeneity. Although being widely reported, genotype-phenotype correlations and disease prevalence are still limited due to clinical selection bias (compared with symptomatic PFBC patients, the asymptomatic have less chance to be genetically identified).

Background: Primary familial brain calcification (PFBC) is a rare neuropsychiatric disorder characterized by bilateral calcification in the basal ganglia. Four causative genes, SLC20A2, PDGFRB, PDGFB, and XPR1(1-2), have been identified for autosomal dominantly inherited PFBC.

Method: A multicenter retrospective cohort study involving PFBC probands enrolled by two different ways: Group I (n=37): probands enrolled by non-systematic collection, which including probands in clinic; Group II (n=236): patients enrolled by a systematic collection by searching in other 50 hospitals' radiology system. Genetic screening of four causative genes for autosomal dominantly inherited PFBC was performed using cDNA. All the variants identified were further confirmed using genomic DNA and classified according to ACMG-AMP recommendations.

Results: Thirty-three variants including 22 novel variants were detected in 37 probands. Among these probands, 83.8% (31/37) are asymptomatic. Two probands with homozygous pathogenic SLC20A2 variants present more severe brain calcification and symptoms. Based on the variant detection rate of probands in Group II, we extrapolate an overall minimal prevalence of PFBC to 6.6 p. 1,000, much higher than previously reported 2.1 p. 1,000.

Conclusion: We identified a higher proportion of genetically confirmed probands with PFBC that were asymptomatic. These asymptomatic patients would be neglected by clinical selection bias and make the disease prevalence underestimated. Considering that PFBC patients with biallelic variants had more severe phenotypes, it would be necessary to offer genetic counseling to prevent birth of individual with biallelic variants.

Reference:

1. Manyam BV, Walters AS, Narla KR. Bilateral striopallidodentate calcinosis: clinical characteristics of patients seen in a registry. Mov Disord. 2001;16 (2):258-264.

2. Lemos RR, Ramos EM, Legati A, et al. Update and Mutational Analysis of SLC20A2: A Major Cause of Primary Familial Brain Calcification. Hum Mutat. 2015;36(5):489-495.

LBA 11

Alteration of brain functional connectivity in Parkinson's disease patients with dysphagia

J.X. Gao, X.J. Guan, Z.D. Cen, Y. Chen, X.P. Ding, Y.T. Lou, S. Wu, B. Wang, Z.Y. Ouyang, M. Xuan, Q.Q. Gu, X.J. Xu, P.Y. Huang, M.M. Zhang (Hangzhou)

Objective: The aim of this study is to investigate the underlying alterations of brain functional connectivity in Parkinson's disease (PD) patients with dysphagia by resting-state functional magnetic resonance imaging.

Background: Dysphagia is a common non-motor symptom in PD patients, whose pathophysiology is poorly understood. There are few studies explaining the relationship between brain functional network and pathophysiological mechanisms in PD patients with dysphagia.

Method: We recruited 13 PD patients with dysphagia and 10 patients without dysphagia, diagnosed by videofloroscopic study of swallowing. Another 13 age-and sex-matched healthy subjects were recruited. Dysphagia was diagnosed by videofloroscopic study of swallowing. Eigenvector centrality mapping was computed to identify functional connectivity alterations among these groups.
Results: PD patients with dysphagia had significantly increased functional connectivity in the cerebellum, left premotor cortex, the supplementary motor area (SMA), the primary motor cortex, right temporal pole of superior temporal gyrus, inferior frontal gyrus, anterior cingulate cortex and insula (ACC), compared with patients without dysphagia (table1). Compared with the NCs, the functional connectivity of the right insula, inferior frontal gyrus, inferior frontal lobe, putamen and globus pallidus decreased in the PD patients without dysphagia(table2). In PD patients with dysphagia, the functional connectivity of left cerebellar tonsil, cerebellum 8 region, cerebellum 9 region and spindle gyrus was increased, compared with NCs (table3).
Conclusion: This research, studying PD with dysphagia by rsfMRI, found that the functional connectivity of the premotor area, SMA, primary motor area, ACC, and insular lobe increased, which may be closely related to the pathophysiological mechanism of dysphagia in PD patients.

Table 1. Difference of functional connectivity between PD patients with dysphagia and PD patients without dysphagia

Brain Regions	L/R	Cluster Size	BA	MNI Coordinate			T Value
				x	y	z	
PD-d > PD-nd							
cerebellar tonsil	L	247	-	-27	-27	-36	3.76
cerebellum 8 region		66					
superior temporal gyrus	R	746	38	39	21	-24	5.10
inferior frontal gyrus		166					
middle frontal gyrus		128					
orbitofrontal gyrus		95					
anterior cingulate		74					
insula		26					
premotor cortex	L	396	6	-42	-3	51	6.83
and supplementary motor area							
precentral gyrus		194					
postcentral gyrus		147					
middle frontal gyrus		67					
primary motor cortex		25					

PD-d, PD with dysphagia; PD-nd, PD without dysphagia; L, left; R, right; BA, Brodman area; MNI, Montreal Neuroscience Institute template.

Table 2. Difference of functional connectivity between PD patients without dysphagia group and NCs

Brain Regions	L/R	Cluster Size	BA	MNI Coordinate			T Value
				x	y	z	
PD-nd < NCs							
insula	R	460	13	33	18	-15	-5.14
inferior frontal gyrus		178					
inferior frontal lobe		50					
putamen		46					
globus pallidus		16					

PD-nd, PD without dysphagia; L, left; R, right; BA, Brodman area; MNI, Montreal Neuroscience Institute template.

Table 3. Difference of functional connectivity between PD patients with dysphagia and NCs

Brain Regions	L/R	Cluster Size	BA	MNI Coordinate			T Value
				x	y	z	
PD-d > NCs							
cerebellar tonsil	L	328	-	-27	-24	-36	3.71
cerebellum 8 region		99					
cerebellum 9 region		42					
spindle gyrus		24					

PD-d, PD with dysphagia; L, left; R, right; BA, Brodman area; MNI, Montreal Neuroscience Institute template.

LBA 12

Evaluation of MYORG mutations as a novel cause of primary familial brain calcification

Y. Chen, F. Fu, S. Chen, Z.D. Cen, F. Xie, X.S. Zheng, D.H. Yang, H.T. Wang, W. Luo (Hangzhou)

Objective: To investigate the clinical, genetic and neuroradiological characteristics of primary familial brain calcification (PFBC) patients with biallelic MYORG mutations in China.

Background: PFBC is typically thought to be inherited as an autosomal-dominant trait and has thus far been associated with SLC20A2 (1), PDGFB (2), PDGFRB (3) or XPR1 (4) mutations. Very recently biallelic mutations in the MYORG gene were identified as a novel genetic cause for autosomal recessive PFBC in twelve Chinese patients from six unrelated families (5).

Methods: We collected clinical and neuroradiological data of 169 Chinese patients with primary familial brain calcification, including 151 sporadic patients and 18 patients from 13 families compatible with an autosomal recessive mode of inheritance. Mutational analysis of MYORG was performed in the cohort.

Results: We identified four, including three novel, MYORG mutations segregating in four families with five patients: one nonsense mutation (c.1431C>A, p.Y477*), one missense mutation (c.687G>T, p.W229C), and two non-frameshift indels (c.348_349insCTGGCCTTCCGC, p.116_117insLAFR; c. 428_442delTGCACTTCTTCATCC, p.143_147delLHFFI). The 12-bp insertion c.348_349insCTGGCCTTCCGC was found in either homozygous or heterozygous state in two probands of our cohort and another Chinese PFBC patient previously reported in the literature. Haplotype analysis of our patients harboring the insertion indicated a founder effect in ethnic Han Chinese population. To date, biallelic MYORG mutations have been reported in 17 patients (including our cohort). Most patients were symptomatic (13/17, 76.5%) and the most recurrent symptoms were movement disorders (10/17, 58.8%), cognitive decline (7/17, 41.2%) and cerebellar symptoms (6/17, 35.3%). All patients had calcifications on comprehensive cranial computed tomography, most frequently located in the basal ganglia (17/17, 100%), cerebellum (17/17, 100%), subcortical white matter (14/17, 82.4%), and thalamus (13/17, 76.5%).

Conclusion: We confirmed MYORG as a novel causative gene for primary familial brain calcification and further expanded the mutational and phenotypic spectrum of MYORG-related primary familial brain calcification.

Reference:

1. Wang C, Li Y, Shi L, et al. Mutations in SLC20A2 link familial idiopathic basal ganglia calcification with phosphate homeostasis. Nat Genet. 2012;44(3):254-6.
2. Keller A, Westenberger A, Sobrido MJ, et al. Mutations in the gene encoding PDGF-B cause brain calcifications in humans and mice. Nat Genet. 2013;45(9):1077-82.
3. Nicolas G, Pottier C, Maltete D, et al. Mutation of the PDGFRB gene as a cause of idiopathic basal ganglia calcification. Neurology. 2013;80(2):181-7.
4. Legati A, Giovannini D, Nicolas G, et al. Mutations in XPR1 cause primary familial brain calcification associated with altered phosphate export. Nat Genet. 2015;47(6):579-81.
5. Yao XP, Cheng X, Wang C, et al. Biallelic Mutations in MYORG Cause Autosomal Recessive Primary Familial Brain Calcification. Neuron. 2018;98(6):1116-23 e5.

LBA 13

Analysis of the characteristic changes of macular thickness in patients with Parkinson′s disease

J.H. Zhao, N. Wu, Y. Wan, L. Song, J. Gan, Z.G. Liu, (Shanghai)

Objective: To analyze the characteristic changes of macular thickness in patients with Parkinson′s disease by spectral-domain optical coherence tomography(SD-OCT). Find out the association between macular thickness and disease progression, visuospatial impairment and asymmetry of motor symptoms.

Background: Impaired macular retina thickness have been demonstrated in Parkinson′s disease patientd using SD-OCT before.

Method: Seventy-one PD patients and sixty-one healthy controls underwent SD-OCT examination, the macular thickness of all retinal quadrant segments, fovea thickness, and macular volume between two groups were comparatively analyzed. Associations between macular measurements and clinical parameters were analyzed using generalized estimated equation fitted with linear regression models.

Results: All quadrants of macular thickness (except fovea and 1mm central zone) in PD group were significantly reduced than those in control group ($P<0.05$). ROC curve analysis revealed that inner superior thickness could predict the presence of PD with area under ROC = 0.727(95%CI 0.662-0.792, $P<0.001$). UPDRS-III scores were negative correlated with inner superior thickness (b=-7.765,

P<0.001), inner nasal thickness (b=-5.112, P=0.008) and inner temporal thickness (b=-5.369, P<0.001). The disease duration had no relationship with macular measurements. We found no difference between the macula parameters of the hemiretinae corresponding to more and less severely affected cerebral hemisphere. MoCA visuospatial subscores were positive correlated with macular volume (b=0.144, P=0.028), outer superior thickness (b=5.623, P=0.037), outer nasal thickness(b=5.680, P=0.010) and outer inferior thickness(b=5.448, P=0.026).
Conclusion: In PD patients, the macular thickness and macular volume are decreased. We did not identify asymmetry between hemiretinae in PD. Some quadrants of macular thickness were associated with disease progression and visuospatial impairment.

LBA 14
The efficacy and safety of STN-DBS under general anesthesia for PD patients
C.W. Chen (Gunagzhou)
Objective: This study was a comparative studies of the efficacy and safety of bilateral subthalamic deep brain stimulation (STN-DBS) for Parkinson's disease (PD) under general anesthesia (GA) in mainland of China with 1-year follow-up.
Background: Deep brain stimulation has been shown to be superior to the best medical therapy in improving motor function and quality of life in advanced Parkinson disease. Today, there are two different anesthesia for PD patients to choose, traditional DBS conducted while the patient is awake with local anesthesia (LA), and new general anesthesia can be performed under asleep DBS for PD patients who cannot tolerate awake DBS.
Method: 19 PD patients who underwent bilateral STN DBS were assigned to the GA group and 23 patients were assigned to the LA group, The PD participants were interviewed and assessed preoperatively and at 1-year postoperatively on medicine (med-on) and off medicine (med-off) using motor and non-motor scales, including Hoehn and Yahr stage, Movement Disorder Society-Unified Parkinson's Disease Rating Scale (MDS-UPDRS), Non-Motor Symptoms Questionnaire for Parkinson's Disease (NMSS), Mini-Mental State Examination (MMSE), MOCA, Unified Dyskinesia Rating Scale (UDysRS) and 39-item Parkinson's Disease Questionnaire (PDQ39), HAMA, HAMD. Their medication dosage was indicated by daily levodopa equivalent doses (LEDD). We compared the above clinical data, stimulating parameters, and adverse effect of patients under general anesthesia (GA) with those who were operated on under local anesthesia (LA). Motor outcome: There was no significant difference on the MDS-UPDRSIII scores in either groups in 1-year fellow-up. The improvement of H-Y stage was significant higher in GA than LA group.
Results: Non-motor outcome: The quality of life (PDQ-39 SI, NMSS and MDS-II scales) did not show difference except that MDS-II scores showed a greater decline in GA group, the scores of PDQ-39 SI and NMSS were improved in LA group in 1-year fellow-up. The sleep evaluation (PSQI, PDSS and ESS) did not show difference in LA group, but the scores of PSQI and PDSS were significantly improvement in GA group (p=0.014, 0.007). The neuropsychological evaluation (HAMA, HAMD and BDI) did not show difference in LA group, but the scales above were all significantly improvement in GA group (p=0.004, 0.021, 0.022). There was no difference in cognitive function (MMSE and MOCA) in both group. The levodopa equivalent daily dose (LEDD) was significantly decreased in both groups postoperatively, and there was no difference in either group. Stimulating parameters: the stimulating parameters gradually increased, the average battery voltage showed significantly higher in GA group compare with LA group (both in onset lateral and onset contralateral, p=0.026, 0.015, respectively), the electric energy of onset contralateral was higher in GA group (P=0.036), the mean frequency and the mean pulse width showed no significant difference in both groups. Adverse effects: The incidence of hiccup and mania were significantly higher in the GA group, the incidence of dyskinesia and deterioration of motor symptom was significant higher in LA group.
Conclusion: Asleep-DBS can be considered as a good alternative method for PD patients undergoing DBS. In GA group, the stimulation parameter was higher, however, the non-motor outcomes including sleep and neuropsychological evaluation were improved better compared with LA group. The incidence of mania and hiccup were higher in GA group, the dyskinesia was higher in LA group.

LBA 15
LRP10 mutations in Chinese patients with familial Parkinson's disease
Y. Chen Z.D. Cen, X.S. Zheng, X.H. Chen, S. Chen, H.T. Wang, D.H. Yang, L.B. Wang (Hangzhou)
Objectives: To investigate the involvement of LRP10 mutations in Chinese patients with familial Parkinson's disease and critically review previous studies assessing LRP10 mutations in patients with Parkinson's disease(1-4).
Backgrounds: Very recently, the LRP10 gene have been identified as a novel genetic cause in individuals affected by Parkinson's disease, Parkinson's disease dementia, or dementia with Lewy bodies(1).
Method: Mutational analysis of the LRP10 gene was performed in the cohort of 205 unrelated Chinese patients with familial Parkinson's disease. Burden analysis was conducted using data from GnomAD database and five genetic studies of LRP10 in patients with Parkinson's disease (including our study).
Results: Three novel potentially pathogenic variants, c.32T>A (p.L11H), c.1184G>A (p.R395H) and c.1333G>A (p.A445T), were detected in three probands of our cohort. Up to date, 13 putative pathogenic variants have been identified in 4,270 (8,540 alleles) fully sequenced patients with Parkinson's disease. A total of 1349 variants in LRP10 fulfilling the same criteria were found in 141,456 (282,912 alleles) individuals from GnomAD database. Burden analysis argued against an over-representation of variant-alleles in patients with Parkinson's disease (p=1.049e-06; odds ratio, 0.3182; 95% confidence interval, 0.1689-0.5463).
Conclusion: The genetic screening of the LRP10 gene in our cohort may provide independent, albeit limited, evidence for the pathogenicity of LRP10 in familial Parkinson's disease. Burden analysis using data from current studies failed to support the association between LRP10 and Parkinson's disease in general. However, it should be noted that it is not easy to detect associations between rare variants and disease in case-control studies, particularly in the context of etiological heterogeneity, incomplete

penetrance, and late-onset diseases like Parkinson's disease5. Besides, the methods of variant detection (Sanger sequencing or next generation sequencing), read depth and ages of onset varied among different studies and public databases. Thus, more robust replication studies are warranted to confirm the involvement of LRP10 in the pathogenesis of Parkinson's disease.

Reference:

1. Quadri M, Mandemakers W, Grochowska MM, Masius R, Geut H, Fabrizio E, et al. LRP10 genetic variants in familial Parkinson's disease and dementia with Lewy bodies: a genome-wide linkage and sequencing study. The Lancet Neurology. 2018;17(7):597-608.
2. Kia DA, Sabir MS, Ahmed S, Trinh J, Bandres-Ciga S, International Parkinson's Disease Genomics C. LRP10 in alpha-synucleinopathies. The Lancet Neurology. 2018;17(12):1032.
3. Tesson C, Brefel-Courbon C, Corvol JC, Lesage S, Brice A, French Parkinson's Disease Genetics Study G. LRP10 in alpha-synucleinopathies. The Lancet Neurology. 2018;17(12):1034.
4. Shi CH, Luo HY, Fan Y, Li YS, Xu YM. LRP10 in alpha-synucleinopathies. The Lancet Neurology. 2018;17(12):1034-5.
5. Visscher PM, Wray NR, Zhang Q, Sklar P, McCarthy MI, Brown MA, et al. 10 Years of GWAS Discovery: Biology, Function, and Translation. American journal of human genetics. 2017;101(1):5-22.

LBA 16

REM sleep behavior disorder in Parkinson's disease patients with and without freezing of gait: Polysomnographic evaluation

Y.H. Sung, J.W. Yang (Incheon)

Objective: We determined whether RBD would be related to the clinical presentation of FOG and might be useful when predicting development of FOG in PD.

Background: Rapid eye movement sleep behavior disorder (RBD) is generally considered as a prodromal marker of Parkinson's disease (PD); in contrast, freezing of gait (FOG) usually develops in advanced PD.

Method: PD patients were divided into 7 PD with FOG (PD+FOG) and 13 PD without FOG (PD-FOG) groups. We selected 22 age- and sex-matched healthy controls. Each participant underwent a detailed neurological examination and sleep assessment using questionnaires and overnight polysomnography.

Results: There was no significant difference in age, sex ratio, disease duration, score of depression scale, and score of sleep scale between PD+FOG and PD-FOG group. Score of UPDRS part II, III, and total which indicate motor severity and disability of activities of daily life was higher in PD+FOG group. RBD frequency and mean percentage of REM sleep without atonia were significantly higher in PD-FOG patients than in PD+FOG patients and healthy controls ($p < .001$ and $p = .03$).

Conclusion: RBD occurred more frequently in PD-FOG patients. In contrast PD+FOG patients had infrequent representation of RBD. This result is contradictory to previous reports and suggests that RBD might be associated with less severe motor symptoms of PD.

LBA 17

Bone mineral density and 25-hydroxyvitamin D levels in patients with Parkinson's disease: a clinical study from plateau area

Y. Cao (Xining)

Objective: To investigate the current status of bone mineral density and 25-hydroxyvitamin D levels in patients with Parkinson's disease in plateau area, and to explore the relationship between bone mineral density and 25-hydroxyvitamin D levels in patients with Parkinson's disease in hypoxic environment.

Background: Parkinson's disease (PD) is a common neurodegenerative disease, which is characterized by insidious onset, slow progression and gradual aggravation. PD patients are prone to falls, and the high risk of fracture is related to myotonia and postural balance disorder, as well as osteoporosis. It was found that bone mineral density (BMD) value and 25-hydroxy vitamin D (25-OH-VitD) concentration levels were closely correlated in PD patients. Hypoxia is an important factor affecting and regulating bone growth and metabolism. Hypoxia causes difficulties in fracture repair and healing, decreases bone density, and increases the rate of chronic bone injury such as osteoporosis. Therefore, there were many patients with osteoporosis in the low-oxygen plateau area, but whether it can affect the changes of bone metabolism indexes in PD patients has not been reported.

Methods: From July 2017 to August 2018, one hundred and two patients with Parkinson's disease including 76 Han, 19 Hui, 7 Tibet nationalities were collected from the outpatient department of neurology and inpatient department of Qinghai Provincial People's Hospital (altitude: 2261m).The PD patients were 44 males and 58 females (mean age 67.1±11.15 years). They were found to be eligible and agreed to participate. All the patients met the latest diagnostic criteria for PD set by the department of neurology of the Chinese medical association. Unified Parkinson's Disease Rating Scale (UPDRS) was used to evaluate the severity of the Disease. All patients were tested using the Lunar iDXA dual energy X-ray bone density measuring instrument manufactured by the USA company to examine the total bone density (BMD), the T value (compared to the young adult 's peak) and the value of Z (compared to the normal peer) of the lumbar spine, the bilateral femoral neck and the bilateral hip joints, as an indicator of osteoporosis. And the serum 25-OH-VitD concentration was measured by chemiluminescence. SPSS20.0 statistical software was used for data processing, group differences were analysis t-test. Pearson's rank correlation was used for correlations. Statistical significance was based on a 5% level.

Results: 1. The incidence of osteoporosis was 24.5% in 102 PD patients in plateau area, and the incidence of bone loss was 45.1%. 2. BMD of lumbar spine, the left hip and the right hip of PD patients in females were lower than those in males , with statistically significant differences (P=0.000, P=0.007, P=0.029). And 25-OH-VitD concentrations in females were significantly lower in males(P=0.013). 3. The concentration of 25-OH-VitD in PD patients of Han nationality was higher than that of hui nationality, and the difference was statistically significant (P=0.006). BMD values of lumbar spine, left femoral neck, right femoral neck and right hip of PD patients of Han nationality were lower compared to Tibetan PD patients, with statistically significant differences (P=0.026, P=0.012, P=0.032, P=0.044). The BMD values of the lumbar vertebra, left femoral neck, left hip and right hip of the hui PD patients were lower than those of the Tibetan patients, with statistically significant differences (P=0.013, P=0.011, P=0.015, P=0.033). The 25-

OH-Vita concentration of the Han, the Hui, and the population of the Tibetan PD , which was no statistically significant difference, (P > 0.05). 4. UPDRS score of PD patients with BMD and T values of left femoral neck (r=-0.356; -0.378), right femoral neck (r=-0.357,-0.308), left hip (r =-0.301, -0.302), right hip (r=-0.289, -0.294) were negatively correlated and statistically significant(P＜0.05). **Conclusion:** It was found that females of the BMD and 25-OH-VitD concentrations levels in PD patients are lower than those males in plateau area. The concentration of 25-OH-VitD in PD patients of Han nationality is higher than that of Hui nationality. The BMD values of PD patients of Han and Hui nationalities are lower than those of Tibetan. The higher the UPDRS score of PD patients is, the more severe the disease is, and the lower the index reflecting osteoporosis is.