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RESTING-STATE BRAIN NETWORKS IN PATIENTS WITH PARKINSON'S DISEASE AND IMPULSE CONTROL DISORDERS

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BACKGROUND AND AIM

Evidence from previous brain metabolism, functional and morphometric imaging studies have consistently demonstrated a dysfunction within the meso-cortico-limbic-striatal circuit in Parkinson's disease (PD) patients with Impulse control disorders (ICD). We aimed to investigate resting-state neural networks connectivity changes in PD patients with and without ICD.

METHODS

Fifteen patients with PD with ICD (ICD+), 15 patients with PD without ICD (ICD-) and 24 age and sex-matched healthy controls (HC) were enrolled in the study. To identify patients with and without ICD and/or punding, we used the Minnesota Impulsive Disorders Interview (MIDI) and a clinical interview based on diagnostic criteria for each symptom. All patients underwent a detailed neuropsychological evaluation. Whole brain structural and functional imaging was performed on a 3T GE MR scanner. Statistical analysis of functional data was completed using BrainVoyager QX software.Voxel-based morphometry (VBM) was used to test whether between-group differences in connectivity were related to structural abnormalities.

RESULTS

Demographic, clinical, behavioural and neuropsychological features of ICD+ and ICD- PD patients and HC are shown in tab. 1 and tab. 2. PD patients with ICD showed an increased connectivity within the salience and default-mode networks, as well as a decreased connectivity within the central executive network ($p<0.05$ corrected) (Fig.1). ICD severity was correlated with both salience and default mode networks connectivity changes only in the ICD+ group (Fig.2). VBM analysis did not reveal any statistically significant differences in local GM between ICD+ and ICD- patients and between all patients and HC ($p<0.05$. FWE).

Parameter	HC (n=24) mean±SD	ICDs+ (n=15) mean±SD	ICDs- (n=15) mean±SD	p -value
Age	63.54±6.7	62.87±8.6	63.14±8	0.789
Education	10.3±3.7	9.8±5	12.9±8	0.106
Gender (M/F)	17/7	11/3	11/2	0.163
Disease Duration	n.a	5.3±2.9	6.6±3.9	0.266
H&Y stage	n.a	1.3±0.5	1.4±0.6	0.591
UPDRS III	n.a	10.9±4.5	12.1±4.4	0.560
HAMD	7.2±2.7	8.6±4.7	7.4 ±3.9	0.575
HADS	9.6±3.7	11.3±8.2	8.1±7.5	0.223
MIDI score	n.a.	6.87±3.27	0.4±0.5	**<0.001**
Total LEDD (mg daily)	n.a	477.3 ±222.9	532.1±207.2	0.481
LEDD DA (mg daily)	n.a.	243.3±82.1	243.3±90.2	0.921

Tab. 1. H&Y stage: Hoehn & Yahr stage; UPDRS: Unified Parkinson's Disease Rating Scale; HAM-D: Hamilton Depression Rating Scale; HADS: Hospital Anxiety and Depression Scale; MIDI: Minnesota Impulsive Disorders Interview; LEDD: Levodopa Equivalent Daily Dose; DA: dopamine agonist.

Cognitive Tasks	ICDs+ (mean±SD)	ICDs- (mean±SD)	U	p-value
MMSE	26.5±2.2	27.1±1.8	75.5	0.452
Immediate recall (Rey)	33.6±7.2	43.8±11.4	45.5	**0.027**
Delayed recall (Rey)	6.3±2.6	9±3	43.5	**0.034**
WCST-global score	101.4±34.3	82.1±30.3	50.0	**0.045**
ROCF- copy task	24.6±8.9	29.2±4.8	62.5	0.165
RCPM	22.2±7.9	28.7±5.5	43.5	**0.021**
Verbal Fluency	24.5 ± 13.4	37.5 ± 10.4	38.5	**0.009**
Semantic Fluency	15.1 ± 5.7	20 ± 4.3	36.0	**0.007**
TMT:B	181.1 ± 98.6	119.2 ± 68.5	47.0	**0.033**
IST	10.5 ± 7.8	13.9 ± 7.5	64.5	0.197
Attentional matrices	45.8 ± 11.3	51.5 ± 6.4	61.5	0.151

Tab. 2. MMSE: Mini Mental State Examination; WCST: Wisconsin Card Sorting Test; ROCF: Rey-Osterrieth Complex Figure Test; RCPM: Raven's 47 Coloured Progressive Matrices. IST: Interference of Stroop Test. Significant results are reported in bold.

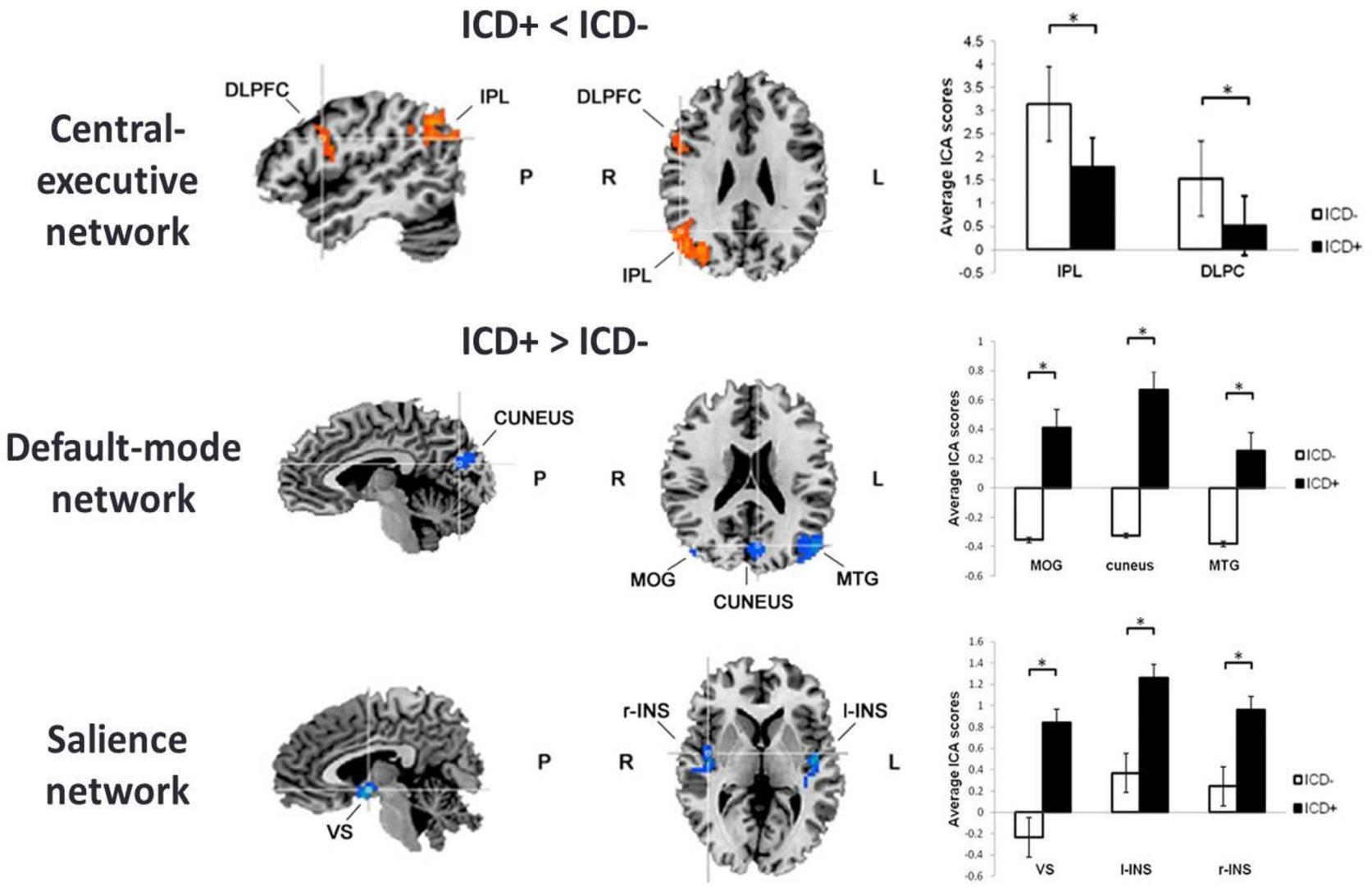

Fig. 1. IPL: inferior parietal; DLPFC: dorsolateral prefrontal cortex; MTG: middle temporal gyrus; VS: ventral striatum; l-INS: left insula; r-INS: right insula.

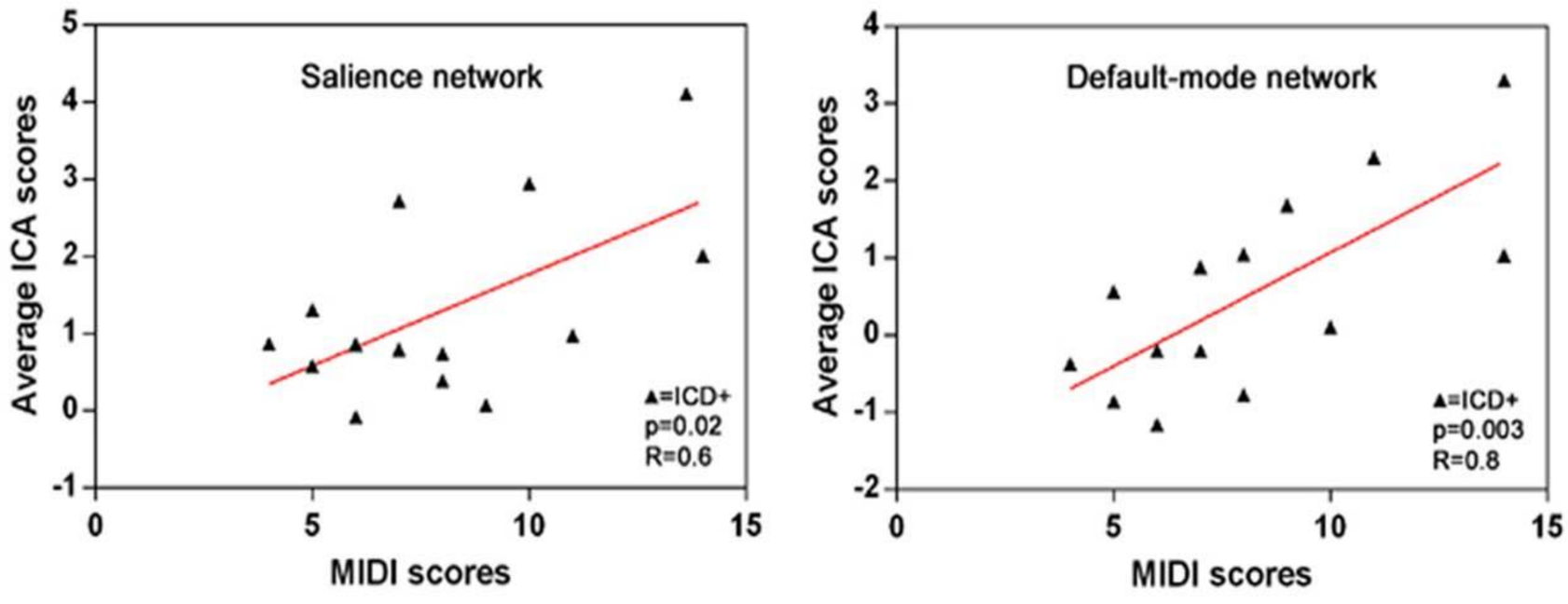

Fig. 2. Correlation analyses between MIDI scores and SN and DMN average ICA z-scores in ICD+ patients.

DISCUSSION AND CONCLUSIONS

The presence of a disrupted connectivity within the three core neurocognitive networks may be considered as a potential neural correlate of ICD presence in patients with PD. Our findings provide additional insights into the mechanisms underlying ICD in PD, confirming the crucial role of an abnormal prefrontal-limbic-striatal homeostasis in their development.