Alterations of functional connectivity in Parkinson's disease patients

N. Tuovinen¹,², M. Seki¹,⁴, C. Müller¹, E. Reiter¹, M. Nocker¹, M. Schocke⁵, E. Gizewski³,⁴, C. Kremser³,⁴, GK. Wenning¹, W. Poewe¹, C. Scherfler¹,⁴ and K. Seppi¹,⁴

¹Department of Neurology, Medical University of Innsbruck, Innsbruck, Tirol, Austria, ²Department of Neuroscience and Imaging, “G. D'Annunzio” University, Chieti, Abruzzo, Italy, ³Department of Neuroradiology, Medical University of Innsbruck, Innsbruck, Tirol, Austria, ⁴Neuroimaging Research Core Facility, Medical University of Innsbruck, Innsbruck, Tirol, Austria, ⁵Department of Radiology, Medical University of Innsbruck, Innsbruck, Tirol, Austria

Background: Parkinson's disease (PD) patients suffer from several cognitive and motor symptoms suggesting changes in functional brain connectivity. The aim of this work was to identify differences compared to controls and disease progression signs in early-stage PD patients with functional connectivity methods ruling out the change in medication.

Methods: RS-data (TR/TE=2.00s/30ms, 3T Siemens Verio) was acquired for 21 controls and 21 PD patients (disease duration: 2.1±1.2y) with similar medication at follow-up (1.5±0.3y). FSL was used for preprocessing (6mm smoothing, highpass 100s, FLIRT and FNIRT (6/12DOF), regression of WM/CSF/6 motion parameters). AAL-116 parcellated time courses were averaged, Pearson correlated and Fisher-transformed with Matlab. Two-tailed two-group t-tests determined differences (p<0.01). Degree centrality was calculated with Brain Connectivity Toolbox.

Results: Patients presented stronger cerebellar and weaker long-range frontal connectivity at baseline and follow-up compared to controls. At baseline, connectivity between cerebellar and motor regions was stronger. Furthermore, putamen, cingulate and Rolandic connectivity was lower. At follow-up, left caudate had altered connectivity pattern compared to controls with weaker connection with left cerebellum. PD progression showed decreased connectivity of frontal regions especially with left cerebellum. Significantly lower degree centrality at baseline was identified for patients in Rolandic, SMA, postcentral and paracentral regions.

Conclusions: Differences between patients and controls were identified both at baseline and follow-up. Our results confirm previous studies suggesting brain compensation for basal ganglia through cerebellar function and changes in motor region connectivity. In addition, striatal connectivity changes were identified as expected. Cognitive decline of patients might be related to noted connectivity changes in the frontal regions. Furthermore, cingulate connectivity decrease confirms our previous findings suggesting changes in the function of salience network in PD patients. Functional brain connectivity changes in the patients were confirmed by the change in the degree centrality. These observations might account for some clinical symptoms seen in PD patients as identified alterations were located in regions involved in cognitive and motor processes.