# DYSFUNCTION OF THE DEFAULT MODE NETWORK IN EARLY PARKINSON'S DISEASE: A RESTING STATE FMRI STUDY

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**Introduction.** Parkinson's disease (PD) is characterized by dopamine depletion in the nigrostriatal system. According to the basal ganglia-thalamocortical circuit model, this leads to an abnormal modulation of cortico-subcortical networks. Functional MRI (fMRI) revealed less deactivation of Default Mode Network (DMN) during cognitive tasks in not-demented PD patients compared to healthy controls (HC). On the other hand, a recent study showed decreased connectivity within DMN in PD patients with dementia. Resting-state (RS) analysis of DMN in early PD patients might reveal neural correlates of cognitive functions prior to the clinical manifestations of their impairments

**Objective.** Aim of this study was to investigate the integrity of RS DMN connectivity in patients with early PD.

**Methods.** We enrolled 57 early PD patients (Hoehn and Yahr stage I) without cognitive impairment (mild cognitive impairment or dementia) and 24 HC. We analyzed RS fMRI data using a model free (MELODIC) approach in FSL.

**Results.** PD patients showed increased connectivity within the DMN structures including the superior and middle temporal gyri, hippocampi, parahippocampal cortex, precuneus, posterior, middle and anterior cingulate cortices compared to HC bilaterally ($p < 0.05$ FWE corrected) (Figure 1).

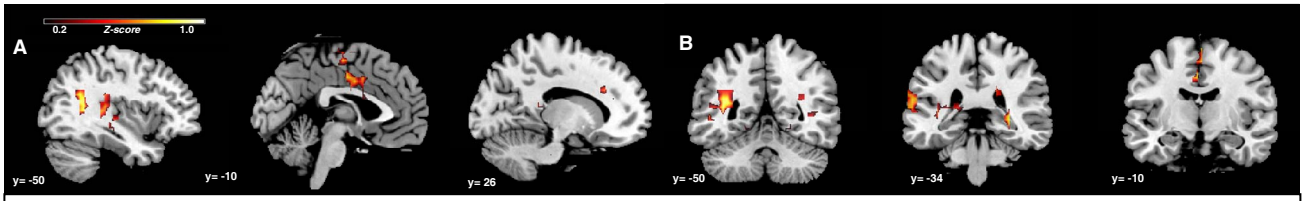


**Figure 1.** Increased resting state (RS) Default Mode Network (DMN) connectivity in patients with early Parkinson's disease (PD) *vs.* healthy controls. Color maps represent significant voxels ($p < 0.05$ FWE corrected). Bar denotes Z scores. Sagittal (A) and coronal (B) sections are shown on the Montreal Neurological Institute standard brain, in neurological convention.

**Conclusion.** We demonstrated a dysfunction of DMN in the very early PD patients without cognitive impairment. There are two possible explanations for our findings. DMN abnormal connectivity in PD could be due to a compensatory mechanism that precedes clinically relevant cognitive impairment. Conversely, it could be secondary to a reduced thalamic outflow to the prefrontal cortex with the impairment of input/output information flows from and to this area and other DMN cortices.

**References.** [1] Van Eimeren et al., Arch Neurol 2009, 66: 877-883; [2] Krajcovicova et al., J Neural Transm 2012, 119:443–454; [3] Ibarretxe-Bilbao et al., Neuroimage 2011, 57:589-597.