

## Dysfunction of EEG resting-state networks in *de novo* Parkinson's disease patients

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**Introduction:** Parkinson's disease (PD) is a neurodegenerative disorder characterized by a progressive synucleinopathy that should begin in the brainstem and spread rostrally. However, theories of a primary subcortical pathology are not easy to reconcile with PD heterogeneous clinical manifestations. Dysfunction of selective brain networks could explain this phenomenon. Connectivity analysis may be performed by functional magnetic resonance imaging (fMRI), but also by electroencephalography (EEG) [1], with the advantage of direct measuring of electrical activity with high temporal resolution.

**Objective:** To analyze the differences in EEG resting-state networks (RSNs) between *de novo* PD patients and healthy controls.

**Methods:** 21 PD patients and 20 controls were, so far, enrolled and matched. The analyses used custom-written scripts on the Matlab platform, combined with high-level functions of Fieldtrip toolbox [2]. First, we proceeded to the EEG cortical source localization, through the resolution of forward and inverse problems. Secondly, we calculated the connectivity matrices in the five frequency bands of the EEG ( $\delta$ ,  $\theta$ ,  $\alpha$ ,  $\beta$ ,  $\gamma$ ), based on the imaginary part of coherency [3]. Then, we applied the Newman clustering algorithm to subdivide the connectome into nonoverlapping networks. Finally, we compared RSNs between PD patients and controls through T-test.

**Results:** We identified four main RSNs among those classically described in literature (default-mode, visual, sensorimotor, frontoparietal networks). We found severe dysfunctions in *de novo* PD patients compared to controls in sensorimotor, default-mode and frontoparietal networks, mainly in  $\theta$  and  $\alpha$  frequency bands. No differences were observed in visual network.

**Conclusions:** This study showed that PD is associated with dysfunctions of RSNs since the earliest stages. We indeed demonstrated that not only sensorimotor network, as we expected, but also default-mode and frontoparietal networks are altered. These results contrast with the ascendent theory of the disease and can be useful to better understand the pathophysiology of PD.

### References

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