**Hippocampal atrophy severely disrupts recruitment of Default Mode Network in TLE**

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**Introduction:** This work consisted on an investigation of the dysfunction of Default Mode Network (DMN) during resting-state functional Magnetic Resonance Imaging in mesial Temporal Lobe Epilepsy (mTLE) patients with Temporal Lobe Epilepsy-Hippocampal Sclerosis (TLE-HS) and without hippocampal atrophy (TLE-NEG). The DMN is a functional connectivity network known by being active during introspective thoughts, such as anticipating and planning the future. Studies have suggested that the DMN is less active or has an uncommon activation in patients with mTLE. However, little is known regarding the impact of hippocampal atrophy in disrupting DMN performance.

**Materials and Methods:** We scanned 95 controls, 21TLE-NEG and 64TLE-HS on a 3T scanner. A seed based analysis was performed from the posterior cingulate cortex (PCC) to identify the DMN (coordinates: 0, -51, 21). Images were pre-processed and analyzed using the SPM12 (http://www.fil.ion.ucl.ac.uk/spm/software/spm12/) with an in-house built routine. After extracting individual activation maps at first-level of SPM, a full-factorial model at second level was applied to compare differences between groups. Significance was determined at p<0.001and clusters> 5 voxels.

**Results:** Patients and controls were balanced for age and gender. We observed that controls presented normal activation of DMN regions, including temporal lobe, parahippocampal gyrus and cerebellum. TLE-NEG displayed a less activated pattern, but with similar spatial distribution. On the other hand, TLE-HS presented more severe disruption of activation with fewer areas recruited and no temporal activation of the DMN.

**Discussion:** The results found are probably due to the important role that hippocampus and associated structures have on the DMN activation since the presence of atrophy is related with worse DMN activation. It is interesting to notice that we did not separate patients considering seizure frequency and thereby both groups mixed subjects with mild and refractory epilepsy. It suggests that the impact of hippocampal atrophy is greater than seizure frequency to generate disruptions on DMN.

**Conclusion:** The present work suggests that the absence of hippocampal atrophy in mTLE allows activations of DMN similar to controls, whereas the presence of hippocampal atrophy in mTLE may impair the proper recruitment of DMN areas such as temporal lobe and hippocampus. In our next step, we plan to investigate associations between cognition, measured by neuropsychological tests and DMN alterations here detected.

**References:** [1] [Maneshi M](http://www.ncbi.nlm.nih.gov/pubmed/?term=Maneshi%20M%5BAuthor%5D&cauthor=true&cauthor_uid=25071712) et al., [Front Neurol.](http://www.ncbi.nlm.nih.gov/pubmed/25071712)14;5:127 [2] Natalie L. Voets et al., [J Neurosci.](http://www.ncbi.nlm.nih.gov/pubmed/?term=Aberrant+Functional+Connectivity+in+Dissociable+Hippocampal+Networks+Is+Associated+with+Deficits+in+Memory) 4281-13.2014.