

# DMN is Affected Incongruently by either Internal or External Environments

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## Introduction and Purpose

Task negative functional connectivity (FC) of resting brain has been used to discriminate various cognitive status [1,2,3] and diseases patterns [4,5,6,7,8] in recent years. Among all, default mode networks (DMN) involving posterior cingulate cortex (PCC), precuneus (PCu), medial prefrontal cortex (MPFC), ventral anterior cingulate cortex (vACC), and bilateral inferior parietal cortex (IPC) have been mostly studied [9,10,11]. Although it is pointed out that components of DMN are involved in the processing of various sensory inputs including vision, audition, and somato-sensation [12,13,14], and are also affected by even simple normal physiological activities like eyes-closed (EC) and eyes-open (EO) [15,16,17], the physiological basis and possible baseline of DMN are not clearly understood yet [9,10,11,18]. So, it would be interesting to observe the patterns of DMN under no external sensory input. In this preliminary study, subjects were deprived of external light and visual stimulations, and we aimed to investigate the possible fluctuations of DMN under this condition, and further try to decipher the possible baseline of DMN.

## Methods and Materials

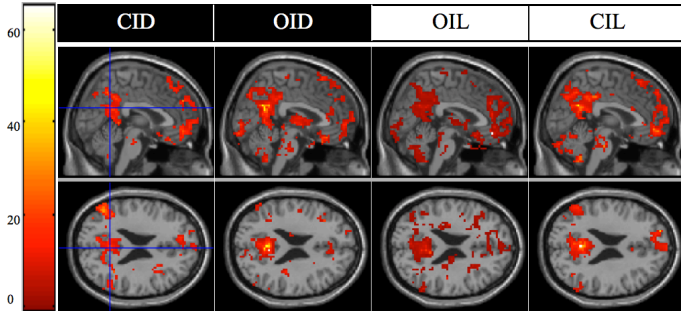
10 healthy right-handed volunteers (F/M=3/7, mean age 31.7±3.3 y/o) first underwent a set of RARE\_T1 anatomical images scan. Then, the following 4 resting-state scan sessions were acquired and counterbalanced across the participants: 1) eyes-closed in dark, CID, 2) eyes-open in dark, OID, 3) eyes-closed in light, CIL, and 4) eyes-open in light, OIL. The deep dark environment was created by covering the MRI scan room with opaque black canvas (0.000 cd/m<sup>2</sup> in the scan room measured by Tektronix J17 photometer when covered, 27.41±2.36 cd/m<sup>2</sup> when all lights on). Each resting-state session lasted for 8 minutes. Resting fMRI data were acquired on a 3.0 Tesla Bruker scanner using EPI sequence (TR/TE 2500/30 ms). Data processing was carried out using SPM5 and REST toolkit (<http://resting-fmri.sourceforge.net>). Preprocessing consisted of: (1) motion correction, (2) linear detrending, (3) normalization, (4) spatial smoothing (FWHM 6 mm), and (5) temporal band-pass filtering (0.01~0.08 Hz). PCC (3,-54,24) [19] was chose as the seed (radius=6 mm) for voxel-wise linear correlation analysis to generate PCC-FC map. Finally, the correlation coefficient maps were converted into z maps by Fisher's r-to-z transform. One-sample t-tests were performed on the individual z maps of the PCC-FC to determine the within-group functional connectivity patterns. The within condition statistical threshold was set at p<0.0005 after correction. Two-sample t-tests were performed to examine between-condition differences of functional connectivity with statistical threshold at p<0.0005.

## Results and Discussion

The one-sample t-test revealed that each component of the DMN do fluctuate across different resting-state conditions (see Fig.1. and table1.2.). Compared CID with OID conditions, when eyes opened in the dark (no interference of the light), the functional connectivity between PCC (BA 23/31) and thalamus increased significantly, which was in concordance with the connectivity between PCC and its neighbouring area like PCu. However, the connectivity between PCC and superior medial frontal cortex decreased a lot. This trend is also observed when comparing CIL with OIL sessions, except some conflicting trend between that of superior medial frontal cortex and medial orbito-frontal cortex. Besides the effect of physiological conditions (EO and EC), the patterns of DMN also showed substantial changes upon the external environmental changes—the light. Under seemly similar EO conditions, PCC had much stronger functional connectivity with both PCu and prefrontal cortex in the light environment, whereas the functional connectivity between PCC and thalamus in stronger in the dark. However, when analysing the possible order effect of DMN [16], we cannot see a synchronous trend between every component in the DMN, which probably due to insufficient cases.

## Conclusions

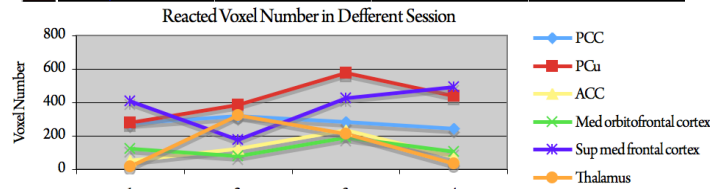
In this study, we focus on the midline DMN structures including PCC, PCu, ACC, and medial prefrontal cortex. Although the cases number is limited, we can still see clearly that DMN do fluctuate across different situations. Both the intrinsic physiological activities and external environments contribute to these changes. Furthermore, each component of DMN does not fluctuate concordantly with each other. More subjects and exquisite experiments are needed to clarify the dynamic change of functional networks of human brains.



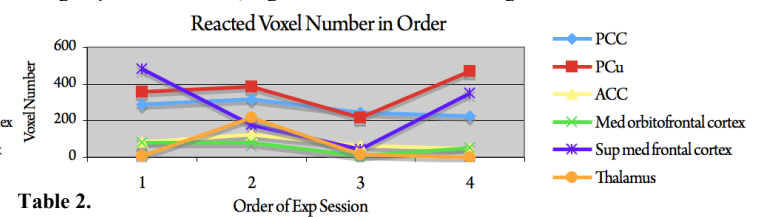
**Fig.1.** PCC-FC under different conditions. The height and extent threshold were set at P<0.0005 (T=8.6103), t score scale is shown on the left. The cross-line indicates PCC (3,-54,24) and the red-yellow color indicates positive t-value. These different conditions were counterbalanced across subjects (So, not all participants received resting-state fMRI sessions in this order). CID, eyes-closed in dark; OID, eyes-open in dark; OIL, eyes-open in light; CIL, eyes-closed in light.

**Table 1.** Reacted voxel number in different conditions (p<0.0005, T=8.6103). FC between PCC and PCu, thalamus, and prefrontal cortex fluctuated significantly but incongruently across different conditions.

**Table 2.** Reacted voxel number in order, ex. all of the first resting-state conditions were grouped as session 1, regardless of the EO/EC or light/dark conditions. The



**Table 1.** Reacted Voxel Number in Different Session



**Table 2.** Reacted Voxel Number in Order

## References:

- [1] J. L. Vincent. Nature 2007. [2] P. Fransson. PNAS 2007. [3] S. G. Horowitz. PNAS 2009. [4] Y. Liu. Brain 2007 [5] J. M. Johnston. 2008. [6] Thilo van Eimeren. Arch Neurol 2009. [7] M. J. Lowe. HBM 2008. [8] S. Laureys. Acta neurol 2002. [9] M. E. Raichle. PNAS 2001. [10] Greicius MD. PNAS 2003. [11] Greicius MD. Cereb Cortex. 2009. [12] Arnott SR. J Neuropsychol 2008. [13] Nobre AC. Brain 1997. [14] Roland PE. Cereb cortex 1995. [15] H. Yang. Neuroimage 2007. [16] Chaogan Yan. PLoS ONE 2009. [17] Q. Zou. HBM 2009. [18] D. A. Gusnard. Nature reviews neuroscience 2001. [19] Changwei Wu. Neuroimage 2008.