

Acute LTE Electromagnetic Field Exposure Modulates the Human Resting-state Functional Connectivity

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1. Introduction

Recently, many studies have applied the neurophysiologic or neuroimage approaches to investigate the possible effects of radiofrequency electromagnetic fields (EMF) on human brain [1]. Besides the findings on regional brain, some studies have demonstrated that acute EMF exposure could modulate the interhemispheric synchronization of temporal and frontal resting EEG rhythms in normal subjects [2] and epileptic patients [3]. Our recent study applied resting state fMRI to investigate the influence of Long Term Evolution (LTE) EMF exposure on brain [4]. We found spontaneous low frequency oscillations were altered by LTE EMF exposure in some brain regions. However, it is still unclear whether acute LTE EMF exposure can modulate the resting state functional connectivity. The present study was aimed to solve this issue with seed-based correlation method.

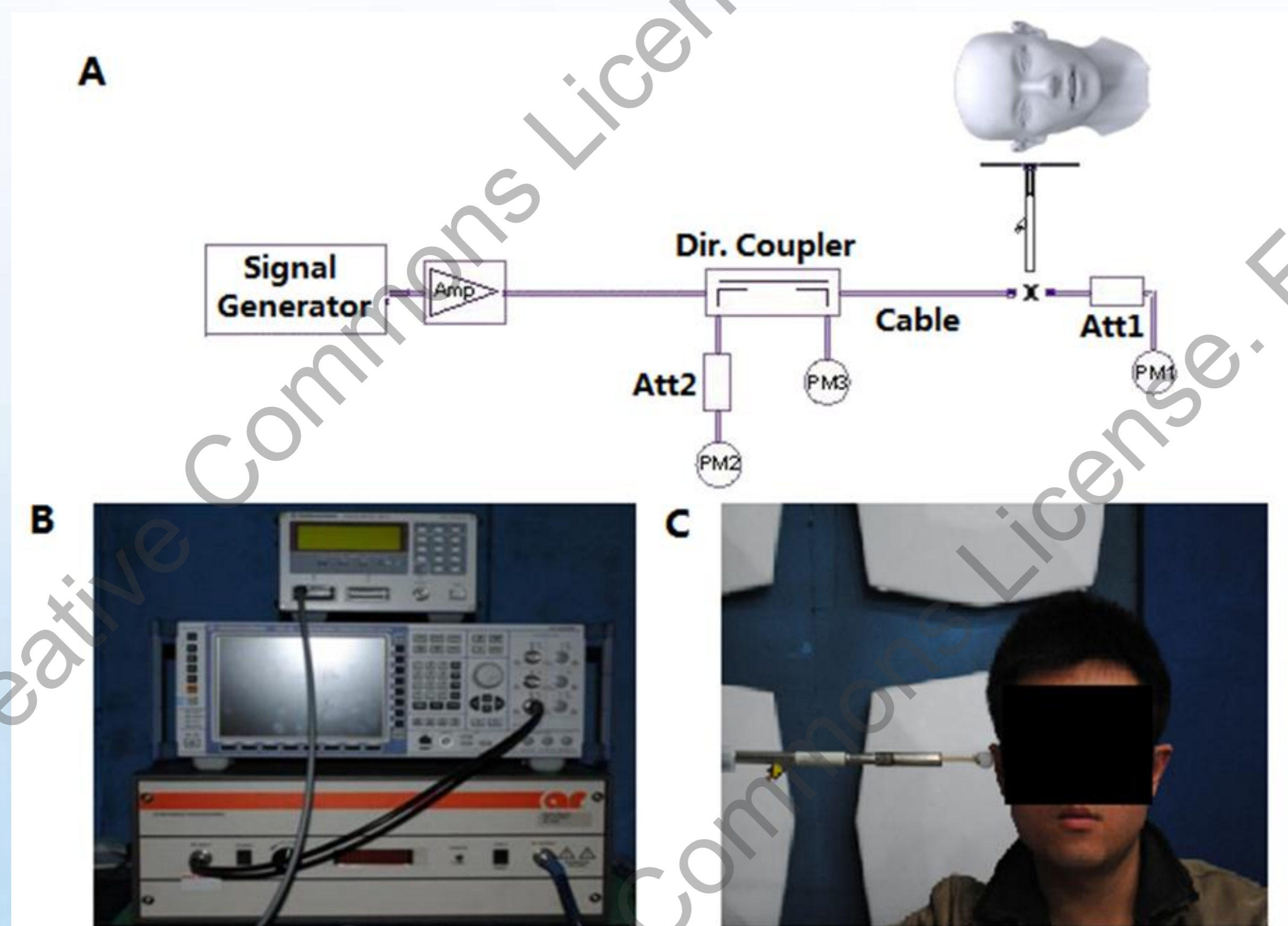


Figure 1. Experimental setup for LTE EMF exposure

2. Method

We designed a controllable near-field LTE EMF exposure environment (figure 1) [4]. 21 right-handed healthy subjects were enrolled to participate in a double, crossover, randomized and counterbalanced experiment including two sessions. In each session, the EMF exposure (real or sham) lasted for 30 min. The structural and resting state fMRI signals were collected before and after the exposure.

The head structural images were segmented interactively to identify 24 tissues with *iSeg*. Then we performed the Finite-Difference Time-Domain (FDTD) simulation [5] to estimate the specific absorption rate (SAR) power distribution induced in different head tissues.

We examined the amplitude of low frequency fluctuation (ALFF) of brain activity [6], and found three brain regions showing significantly decreased ALFF values after real exposure [4]. Then we selected these three regions as seed volumes for the functional connectivity analyses. Data preprocessing included slice time correction, head motion correction, spatially normalization, linear detrend, Gaussian smoothing (FWHM = 4 mm), and temporal band-pass filter with 0.01 ~ 0.1 Hz. Then the mean time series of each seed volume were computed for each condition. Cross-correlation analysis was performed between the mean signals and the time series of all other brain voxels. After performing a Fisher's on correlation coefficients, we used paired t-test to generate the maps of statistical difference between pre- and post-exposure ($P < 0.05$, corrected with Monte Carlo simulation method).

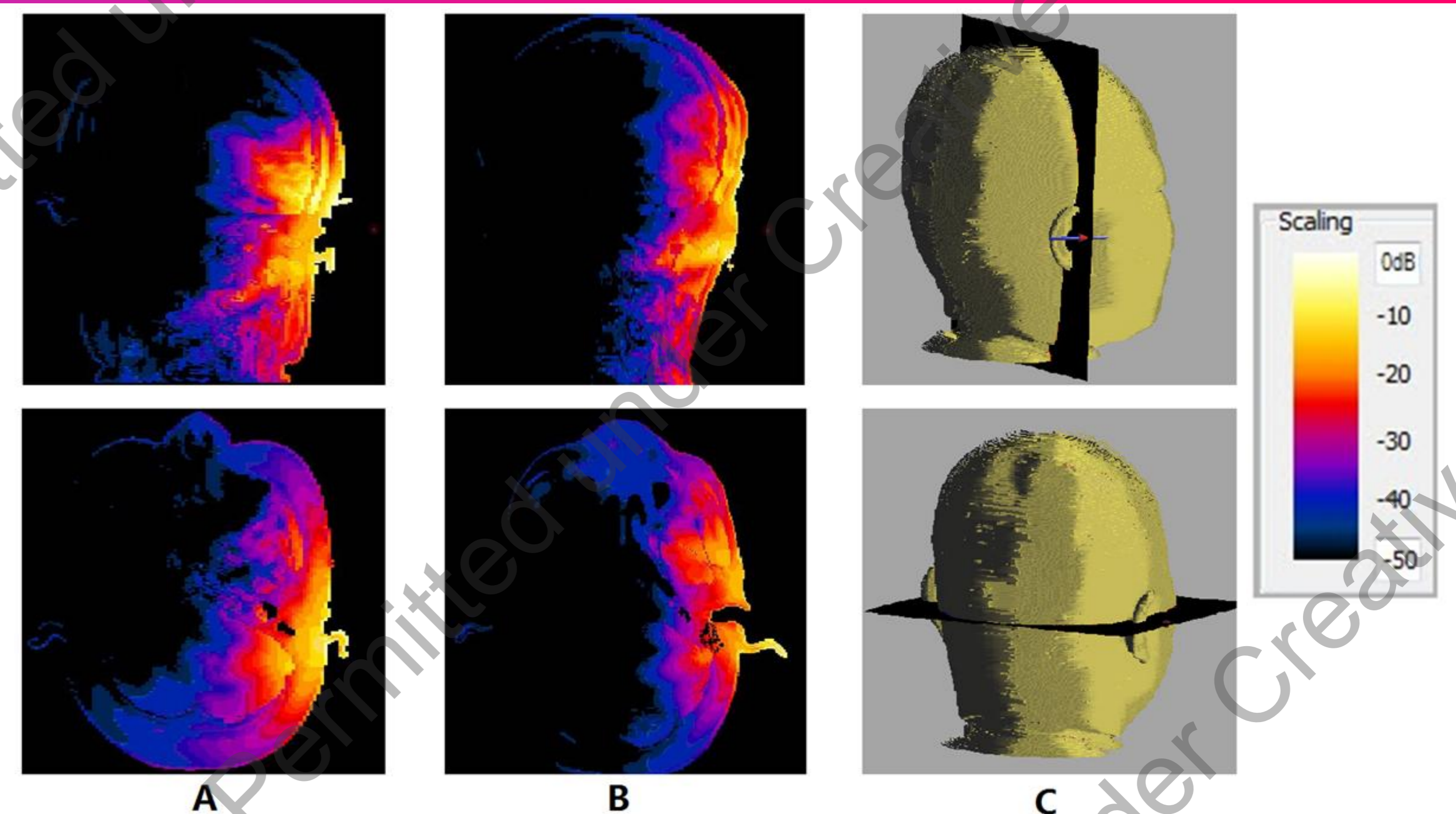


Figure 2. Slice views for estimated SAR power distribution in two subjects. SAR maps are colored according to the bar on the right, and 0 dB equals 2.36 W/kg for a specific voxel.

3. Results and discussions

Figure 2 showed the SAR maps estimated in two subjects. The spatial peak SAR averaging over 10 g tissue was 0.9 and 1.07 W/kg, which were less than the safety limits in ICNIRP guidelines (2 W/kg).

Figure 3A showed the three clusters which have the decreased ALFF values after real exposure. Since the exposure source was on the right side of head, cluster1 was close to this site, while cluster2 was approximate on the homologous areas in the contralateral hemisphere and cluster3 was on the ipsilateral side. Figure 3B-D showed the statistical differences of seed based functional connectivity for each cluster between pre- and post-exposure during real exposure.

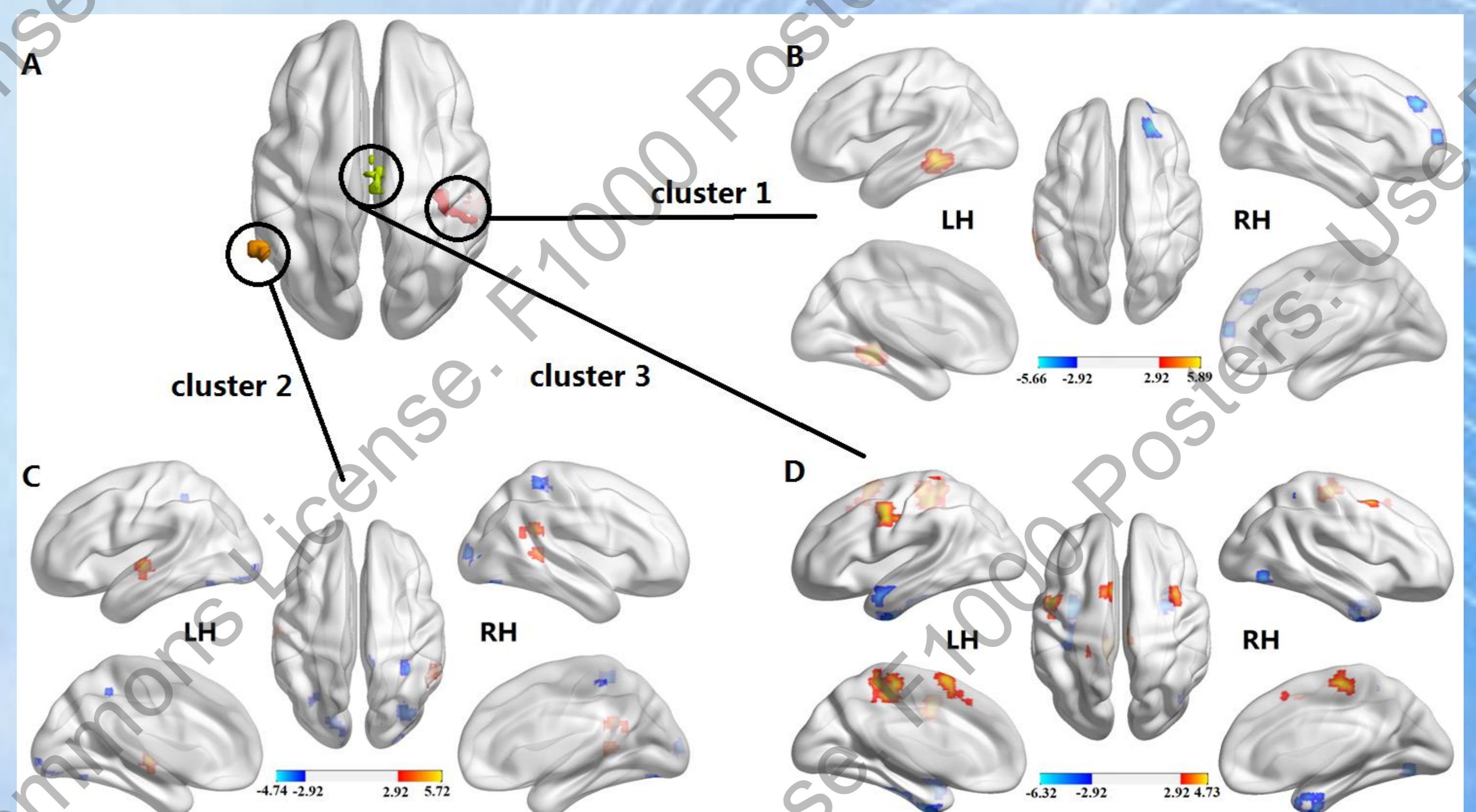


Figure 3. The warm color illustrated the brain regions having lower connectivity after exposure, while the cold color showed the increasing functional connectivity after exposure.

References

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