

EFFECTS OF LOW PASS FILTERING AND AUTOCORRELATION ON RESTING STATE FMRI AS INVESTIGATED USING A REGRESSION MODEL IN SPM8

Kayako Matsuo¹, Yung-Chin Hsu², and Wen-Yih I Tseng¹

¹National Taiwan University College of Medicine, Taipei, Taiwan, ²Department of Biomedical Engineering and Environmental Sciences, National Tsing Hua University, Taipei, Taiwan

Introduction:

We investigated a resting state fMRI analysis method on a powerful analysis platform, SPM8 (Wellcome Trust Centre for Neuroimaging, University College London, UK), which provides a convenient computational frame of regression model statistics. We specifically examined an interaction between the low pass filtering (LPF) and the first degree autocorrelation model (AR(1)) [1-2]. As we use a conventional repetition time (TR) such as 2 sec, the LPF for addressing physiological sources such as respiration and pulsation is not efficient because of the Nyquist-Shannon sampling theorem. In contrast, AR is a generally used technique to examine time serial data, as implemented in SPM software [1-4]. We aimed to clarify the effects on signals in the default mode network (DMN), which is typically associated with low frequency fluctuations [5].

Materials and Methods:

Fifteen normal people with a written informed consent were studied using a 3T MR scanner with a 32-channel phased array coil (Trio Tim, Siemens, Erlangen, Germany). A GE-EPI sequence was employed using the following parameters: TR/TE = 2000 ms/24 ms, flip angle = 90 deg, 34 slices, 3 mm thick with no gap interleaved, FOV 256 mm, matrix size 64 x 64, and 180 volumes per run with additional 2 volumes of dummy scans in advance. We discarded the first 3 volumes and used the remaining 177 volumes. We used SPM8 software and in-house MATLAB codes for analyses. We designed a 2-by-2 repeated-measures ANOVA at the group level; one factor was with or without LPF, and the other was with or without AR(1). Individual data underwent the preprocessing of the slice timing, realignment and spatial normalization to the MNI space. After those, we set 2 different procedures for the same data; one applied spatial smoothing with FWHM 8mm, and the other applied an LPF at 0.1Hz by a fourth-order Butterworth filter before the spatial smoothing. We placed 6 seed regions as well as 8 spurious-source regions for time course extractions. The 6 seed regions (12-mm-diameter sphere for each) were the intraparietal sulcus (IPS), the frontal eye field (FEF), and the middle temporal region (MTp) that constituted the task-positive cognitive mode network (CMN), and the medial prefrontal cortex (MPF), the posterior cingulate/precuneus (PCC), and the lateral parietal cortex (LP) that constituted the task-negative DMN [6]. The 6 out of 8 spurious sources were the realign parameters; the remaining 2 were the white matter and cerebrospinal fluid (CSF) derived from individual segmentation results of T1 images. The mean voxel intensity of each extracted region at each volume was used as a time point data in the time course of the 14 regressors. We then computed the time derivatives for the 6 seed-region time courses. We included the total of 20 regressors into the GLM to see the regressions at the first level statistics. For those individual statistics, we specified a high pass filtering at 128 sec (about 0.0078 Hz). We set 2 different GLMs for both of LPF and no LPF processed data: with or without AR(1). Resultant 4 kinds of contrast images were used at the second level random-effects group statistics ($p < 0.001$, uncorrected). The CMN and DMN contrast conditions were separately analyzed using an ANOVA with 2 LPF by 2 AR(1) analysis conditions (all within-subject design).

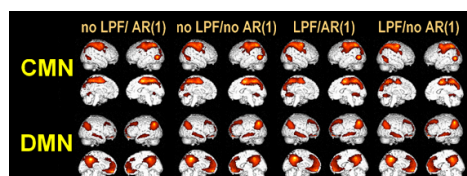


Fig. 1 Rendered activation maps from random-effects group analyses for two contrast conditions and four analysis conditions. N=15.

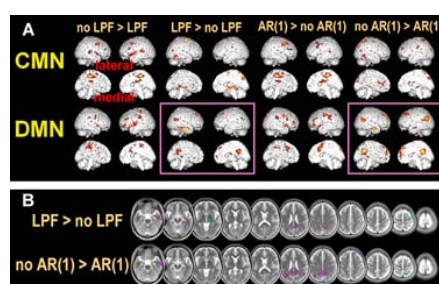


Fig. 2 ANOVA results for the two factors, LPF and AR(1). The purple rectangles in the upper panel A show very similar maps between the comparisons. Sections in the panel B show “correlated” activation with the CMN in purple blobs, whereas that with the DMN in turquoise green. The sections’ right is subjects’ right.

Results and Discussion

The four analysis conditions yielded almost identical maps within each contrast condition (Fig. 1). Comparisons revealed that the LPF and the AR(1) acted in an opposite way with each other; specifically, the LPF enhanced the DMN signals, whereas the AR(1) decreased (Fig. 2). A similar tendency was also shown in the CMN maps; activation maps were quite similar between the “LPF > no LPF” and the “no AR(1) > AR(1)”, as well as, between the “no LPF > LPF” and the “AR(1) > no AR(1)” for both CMN and DMN contrasts. However, section views demonstrated that the reduction by AR(1) was more profound in the DMN than CMN (Fig. 2B). Also, the intensity around the FEF appeared to be increased with the AR(1) application in the CMN. Thus the effects of AR(1) might be different between signal sources; the DMN signal might yield a decrease by the AR(1) application, whereas the CMN signal might not. Another caution might be the small amplitude changes in resting state data, which is in contrast to task fMRI. A previous task fMRI showed that the effect of temporal AR depended on both imaging rate and paradigm frequency [7]. What we want to see is only a small fluctuation in the resting state data; the fluctuation might largely share characteristics with sources that AR(1) addressed, thus tend to be diminished. In contrast to the AR(1), the LPF successfully enhanced the DMN signal amplitude.

Conclusions

The LPF application was effective to detect the DMN activity, whereas the AR(1) was not. We have to be careful in the application of the AR(1) specifically when the DMN fluctuation of the resting state fMRI is of interest.

References

- [1] Friston et al., *Hum Brain Mapp*, 1:153-171, 1994. [2] Friston et al., *NeuroImage*, 12:196-208, 2000. [3] Friston et al., *NeuroImage*, 2:45-53, 1995. [4] Worsley & Friston, *NeuroImage*, 2:173-181, 1995. [5] Auer, *Magn Reson Imaging*, 26:1055-1064, 2008. [6] Fox et al., *Proc Natl Acad Sci USA*, 5:102(27):9673-8, 2005. [7] Purdon & Weisskoff, *Hum Brain Mapp*, 1998;6(4):239-49.