Altered causal connectivity of resting state brain networks in amnesic MCI

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Purpose

Most neuroimaging studies of resting state networks in amnesic mild cognitive impairment (aMCI) concentrated on functional connectivity (FC) in a single network^{1,2}. The purpose of the current study was to investigate effective connectivity (EC) among multiple networks in aMCI patients.

Methods

Structural and functional MRI data were collected from 16 aMCI patients and 16 age, gender-matched healthy controls. A method called correlation-purged Granger causality analysis (CPGC)³ was used to examine the causal connectivity of four basic networks in patients and controls – default mode network (DMN), hippocampal cortical memory network (HCMN), dorsal attention network (DAN) and fronto-parietal control network (FPCN).

Results

It was found that the effective connectivity between networks in aMCI patients is significantly altered, as compared with healthy controls, with anterior-posterior FPCN connectivity and hippocampus centered causal links reduced (Left in Figure 1), and within DMN and within right frontal cortex causal pathways enhanced (Right in Figure 1). Some alterations are significantly correlated with the disease severity as measured by mini-mental state examination (MMSE) and California



verbal learning test (CVLT) (Figure 2). When controlling for GM atrophy, the between-group difference is still kept, although the statistical power is decreased. When taking the global signal as covariates, the within-group patterns are changed but the

between-group difference is remained.

Discussion

These results suggest that the causal disconnections may be the underlying substrates of cognitive decline in aMCI patients. The present study extends the previous findings of the coexisting of disconnection and compensation in aMCI to the affective connectivity domain

aMCI to the effective connectivity domain.

Conclusion

The current study firstly applied Granger causality analysis method to resting state of aMCI patients and demonstrated the coexistence of causal disconnection and compensation in aMCI patients, and thus might provide insights into the biological mechanism of the disease.

References

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