

Brain dynamics of mild cognitive impairment during face encoding

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INTRODUCTION

Mild cognitive impairment (MCI), has been proposed as the prodromal stage of cognitive decline preceding AD dementia (Dubois et al., 2010). The most common first clinical symptom of MCI is memory deficit. In addition, brain functional connectivity is affected by means of cognitive aging and accumulation of neuropathology in the brain.

Literature indicates that 1) memory encoding is one of the most affected processes in pathological aging (Clement et al., 2010), 2) MCI shows brain activity changes by means of connectivity increases (hyperactivations) between pairs of sensors (Bajo et al., 2010) and 3) brain activity increases are related with compensatory mechanisms which allow MCIs to achieve the same level of performance as the healthy controls even in the presence of memory difficulties.

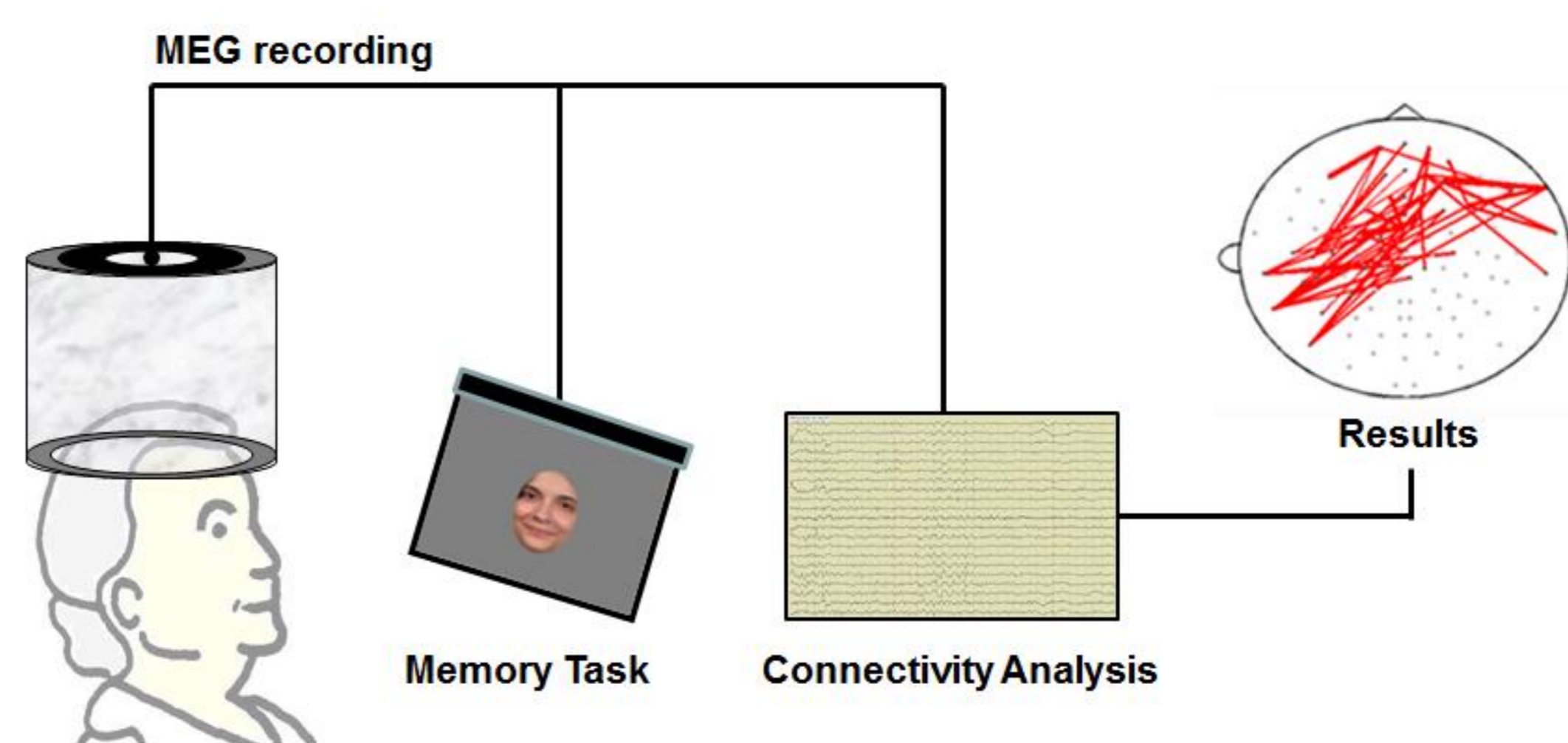
The aims of the present study were to 1) evaluate if memory encoding could differentiate between normal and pathological aging, 2) compare hit rate between healthy adults and MCI, 2) study brain connectivity patterns of MCI during successful encoding of faces and 3) evaluate the possible existence of a compensatory mechanism in MCI.

METHODS

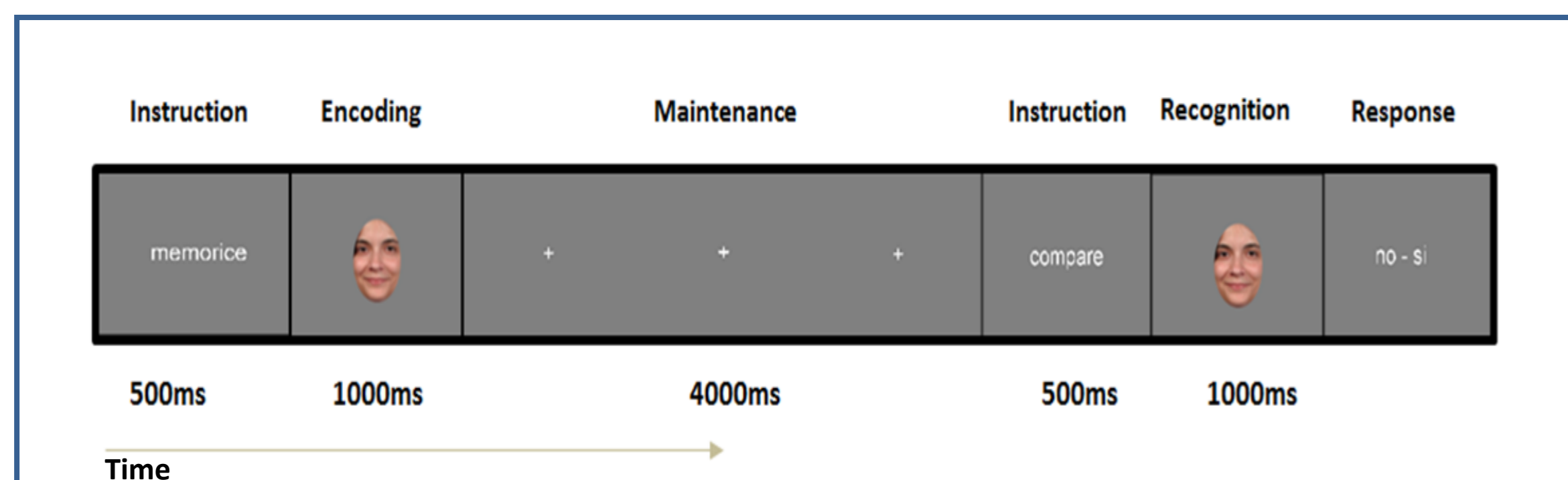
Participants. 16 HC and 16 MCI. Age : 69±4; 72±5; MMSE: 30; 28±2

Task. Face delay match to sample task.

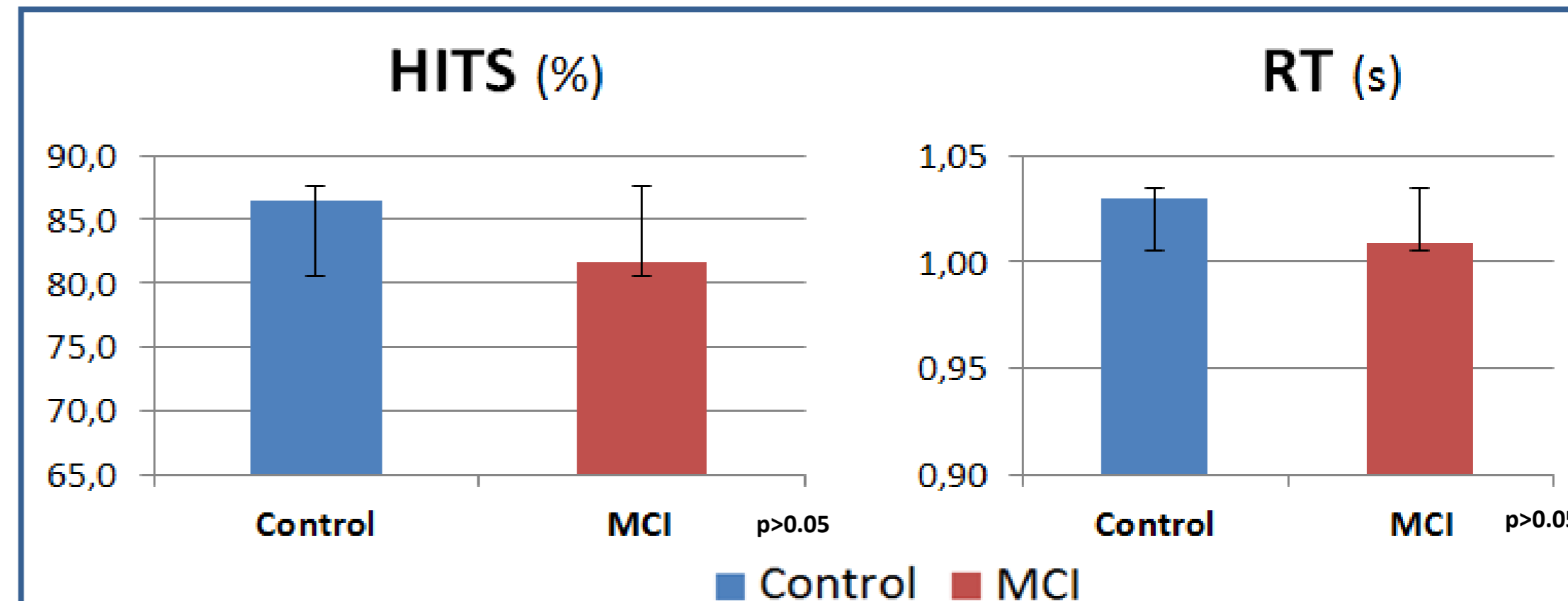
Data acquisition and analysis. Magnetoencephalographic recordings. Connectivity analysis between sensors (Phase Locking Value) in Theta, Alpha, Beta and Gamma. Kruskal - Wallis ($p < 0.01$) and Permutation tests (1000; $p = 0.05$).



COGNITIVE TASK

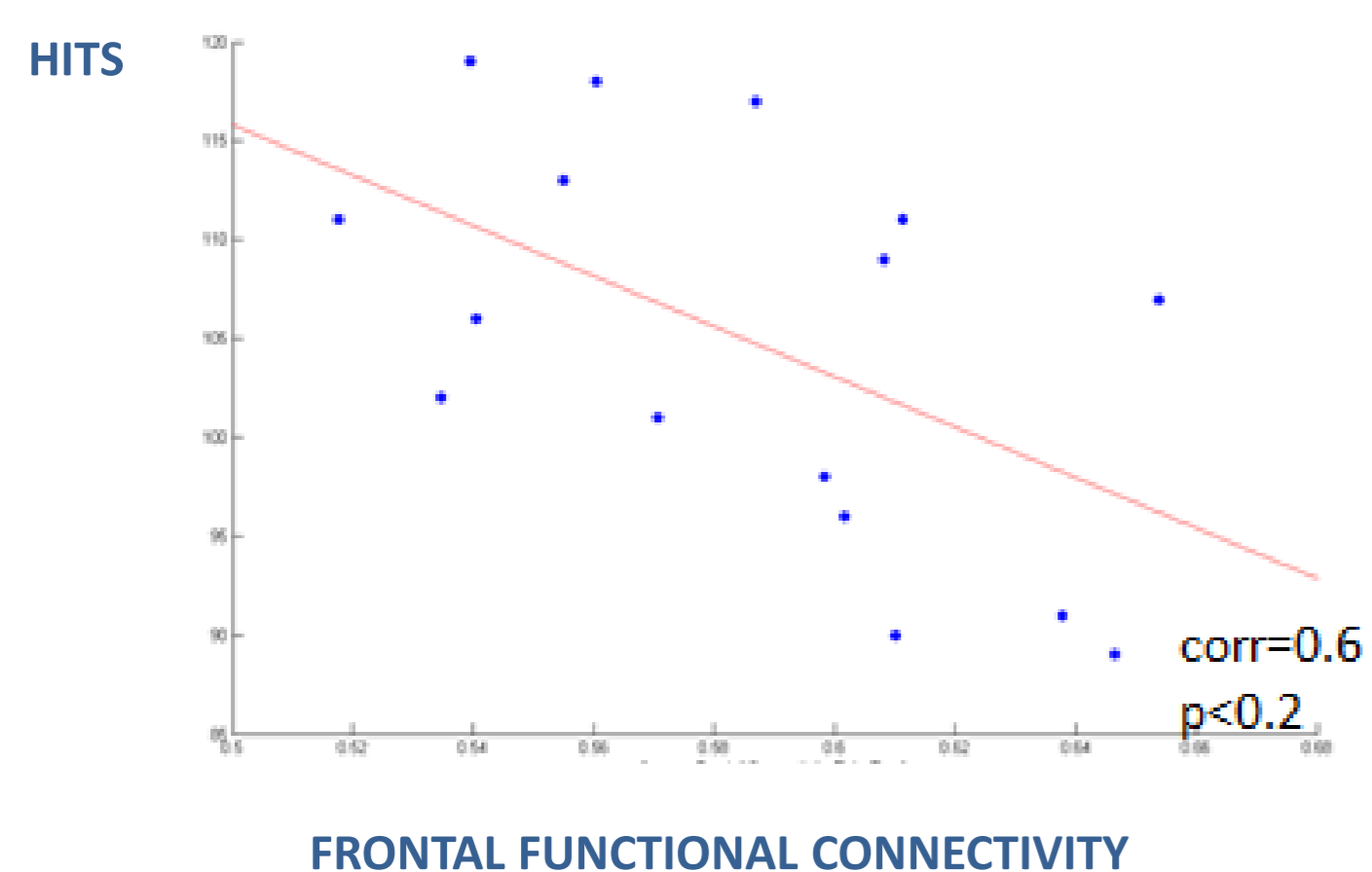


BEHAVIOURAL RESULTS

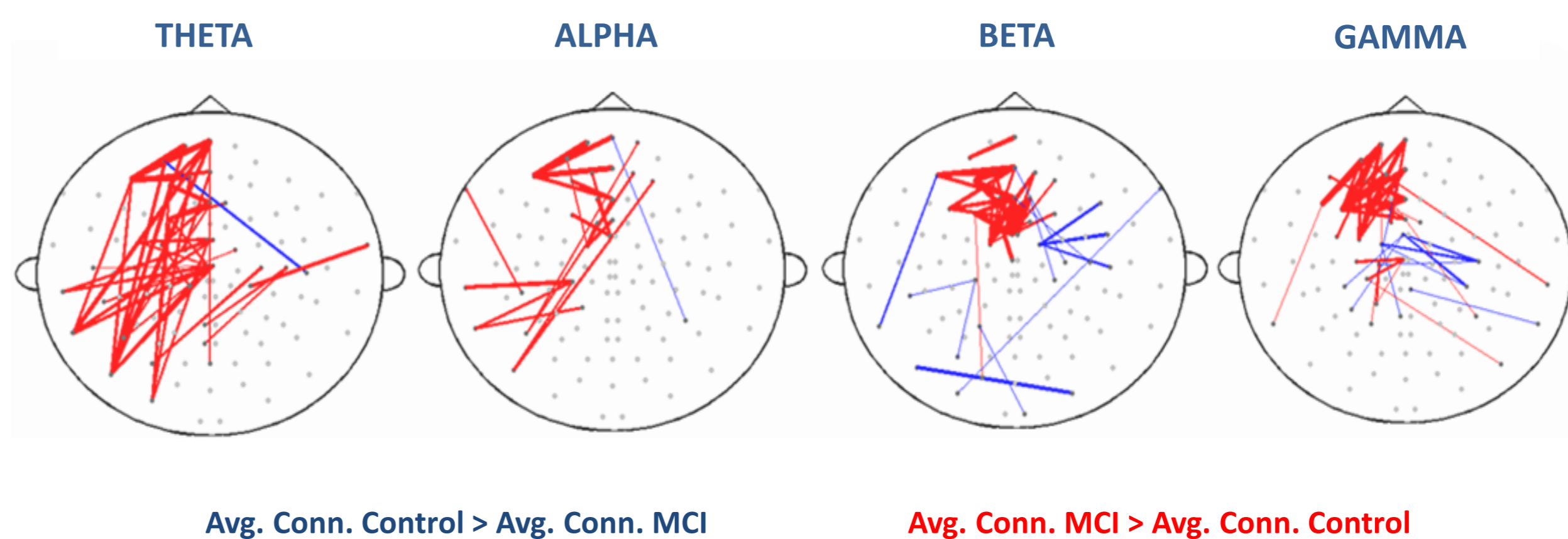


MEG RESULTS

TASK EXECUTION CORRELATES WITH BETA FRONTAL FUNCTIONAL CONNECTIVITY



FUNCTIONAL CONNECTIVITY DIFFERENCES



CONCLUSIONS

MCI, when comparing to healthy controls, is characterized by brain hypersynchronization over frontal and fronto-posterior left sensors during encoding of faces into memory. These patterns are observed in the four frequency bands analysed. The results suggest the possible existence of a compensatory mechanism, which could be necessary for the performance of the task. However, negative correlation between beta synchronization and memory accuracy suggests that those who acquired higher beta values were those who performed the memory task worst.

Therefore, hypersynchronization could be pointing towards a dual compensatory and dysfunctional mechanism which show two neurophysiological states of the MCI brain and which depends on the frequency band analysed.

We conclude that MCI is able to match healthy adult's memory performance by means of brain hypersynchronization. Further studies are needed to study the role which each frequency band have in the memory encoding process.

REFERENCES

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ACKNOWLEDGMENTS

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