ALTERED RESTING STATE NETWORKS IN PATIENTS WITH CLUSTER HEADACHE

M. Filippi1,2, M. A. Rocca1,2, P. Valsasina1, B. Colombo1,2, A. Falini3,4, M. Absinta1, and G. Comi2

1Neuroimaging Research Unit, Scientific Institute Hospital San Raffaele, Milan, Italy, 2Department of Neurology, Scientific Institute Hospital San Raffaele, Milan, Italy, 3CERMAC, Scientific Institute Hospital San Raffaele, Milan, Italy, 4Department of Neuroradiology, Scientific Institute Hospital San Raffaele, Milan, Italy

Introduction
The assessment of low-frequency (<0.1 Hz) fluctuations in functional magnetic resonance imaging (fMRI) data at rest has demonstrated the presence of high temporal coherence between spatially distinct, functionally-related brain regions, which characterizes the RSN of the human brain (1, 2). Aim of this study was to investigate abnormalities of brain resting state networks (RSN) in patients with chronic cluster headache (CH), outside the bout phase, in comparison with healthy individuals.

Methods
RS functional MRI data were acquired from 13 CH patients and 15 matched healthy controls using a 3T MRI scanner. After data pre-processing (realignment, normalization, smoothing and band-pass filtering between 0.01 and 0.08 Hz), Independent component analysis (ICA) was used to decompose RS fMRI data into spatially independent maps and time courses using the GIFT (Group ICA of FMRI Toolbox) software (3). This analysis produced 40 spatially independent maps. Visual inspection of these maps allowed to eliminate components clearly related to artifacts. SPM2 was used to assess the spatial extent of within- and between-groups activations (one-sample t test and ANOVA). Then, for each RS spatially independent map, the average percentage signal change of RS fluctuations inside each significant SPM cluster was compared between controls and patients using a t test for non-paired data.

Results
Our analysis detected 16 RSN with potential functional relevance. Significant between-groups differences in the average percentage signal change of RS fluctuations were found for the sensorimotor network (decreased fluctuation in the primary sensorimotor cortex and supplementary motor area, bilaterally in CH patients) (Figure 1), the primary visual network (decreased fluctuation in V1 in CH patients) (Figure 2), and the secondary visual network (decreased fluctuation in the middle occipital gyrus, bilaterally in CH patients) (Figure 3) (p ranging from 0.03 to 0.007). No differences were found between healthy controls and CH patients in the default mode network and in pain-related networks.

Conclusions
RSN analysis reveals abnormalities inside the visual and motor network in CH patients outside the acute attack. These findings suggest a diffuse dysfunction of functional connectivity which extends beyond the antinoceptive system.

References