COMPARISON OF NAA DYNAMICS IN TASK-ACTIVATED MOTOR CORTEX IN THE NORM AND SCHIZOPHRENIA

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Target audience. This study could be interesting for the basic scientists working with fundamental brain metabolism research, and to medical doctors for the development of new approaches of brain disorders treatment. New MRS technique for "task loaded" brain metabolism study is proposed.

Purpose. Metabolic features of motor cortex activation are poorly studied in schizophrenia[1] despite significant motor disorders were reported[2]. The aim of this study was the analysis of motor cortex metabolism dynamics during period of BOLD response to fMRI motion task.

Patients and methods. The patient group consisted from 8 males of 16–28 years old in initial stage of schizophrenia. All patients were treated with antipsychotic drugs. The group of 9 age matched healthy males was used as a control group.

Philips Achieva 3.0T scanner was used for this study. Volume of interest in motor cortex was defined using fMRI data. Area of activation was obtained for "press the button" paradigm – patient pressed the bottom in response to the single auditory stimulus transmitted repeatedly with the interval of 18 s (EPI FFE BOLD sequence, TR = 3000, TE = 30). The BOLD response signal was measured with time resolution of 3 seconds. Dynamic 1H MRS (PRESS, TE = 30 ms TR = 3000) was performed; spectroscopic data were collected each 3 seconds after "press the button" task execution. Auditory stimulus was repeated 98 times and 98 × 7 FID signals were collected. The same method was applied for spectra acquisition in "rest" condition. All 98 signals obtained for time points t = 0, 3, 6, 9, 12, 15, 18 seconds after stimulus were summarized to improve resulting SNR. Spectra processing was done using custom made software. Apodization filtering (LB = 20, GB = -5) and manual phase correction was applied to obtain NAA, Cho and Cr signal intensities.

Results and conclusions. BOLD signal in both groups demonstrated maximum at the 6^{th} second after motor task execution, with significantly lower signal intensity for patients in comparison with the control group. For healthy volunteers, NAA decreased ($p>0.95$) at the 12^{ve} second after stimulus presentation and returned to initial value at the 15^{th} second, Cho and Cr intensities remained unchanged. For schizophrenia patients, no significant intensity changes were found for all NAA, Cr and Cho. Meanwhile, stable intensities of NAA, Cr and Cho were observed in dynamic for absence of motor activation. Thus, only NAA intensity changed significantly as a response for motor cortex activation, and NAA intensity change is shifted from BOLD response by 6 seconds. Also, patients and control group demonstrated different NAA kinetics in motor cortex.

Different behavior of NAA for schizophrenia patients and control group might be related with difference in location (or activity) of ASPA[3]. Substantial amount of axons are involved into ASPA expression [4]. This enzyme hydrolyzes NAA into acetate and aspartate. Significant NAA decrease at 12^{ve} second after motor task execution might be associated with high neuronal activity. In proposed model, NAA is transferred to oligodendrocytes to transport acetyl CoA and aspartate in respond to increased metabolic demands.

References.

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