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The effect of 5-HTTLPR polymorphism on EEG current source density

E. Proshina[1]*, A. Savostyanov[1, 2, 3], A. Bocharov[1, 3], G. Knyazev[1]

[1] *Scientific Research Institute of Physiology and Basic Medicine Novosibirsk, Russia*
[2] *Institute of Cytology and Genetics, Novosibirsk, Russia*
[3] *Novosibirsk State University, Novosibirsk, Russia*
** e-mail: proshinaea@physiol.ru*

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Motivation and Aim: The S allele of the promoter region of the serotonin transporter gene (5-HTTLPR) is associated with increased risk of depression and other mental disorders. Evidence linking this polymorphism with individual variation in electrophysiological properties of resting state brain networks is still very limited. We aimed to explore the effect of 5-HTTLPR polymorphism on source-level EEG activity in eyes-closed and eyes-open resting condition.

Methods and Algorithms: EEG recordings were performed using 100 electrodes positioned in an elastic cap according to the International 10-10 system with a Cz as the reference. The fronto-central electrode was used as the ground. The signals were amplified using 'Neuroscan (USA)' amplifiers, with a 0.1–100 Hz analog bandpass filter and continuously digitized at 1000 Hz. Electrode impedances were kept at or below 5 kilo-ohms. Artifacts were corrected using independent component analysis in the EEGlab toolbox (http://www.sccn.ucsd.edu/eeglab/) and EEG data were recomputed to the average reference and down-sampled to 250 Hz. The standardized Low Resolution Brain Electromagnetic Tomography method [1] (sLORETA) was used to localize the sources of scalp-recorded EEG data. Current source density data were analyzed using second-level full factorial design in the SPM 12 toolbox (http://www.fil.ion.ucl.ac.uk/spm). There were three factors – the between-subject group factor (L/L vs. other two genotypes) and two within-subject factors, i. e., condition (eyes closed vs. eyes open) and frequency band (seven levels). Of primary interest was the main effect of the group factor and its interactions with the two other factors.

Results: As compared to L homozygotes, S-allele carriers showed lower current source density, with this effect being most pronounced in alpha2 and beta bands in areas overlapping with the default mode regions, the orbitofrontal, temporal cortices, and in the insula.

Conclusion: The effect of genotype was significant in brain regions that are involved in self-referential and emotional processing. We can assume that this effect may reflect the predisposition to emotional disorders inherent to S allele carriers.

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References

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