

MEETING ABSTRACT

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Expression of NR1 subunit of NMDA receptor and PSD-93/95 in rat hippocampus affected by NR1/NR2 antisense oligodeoxynucleotide

Monika Vrajobá^{1*}, Věra Bubeniková-Valešová¹, Jan Klaschka²

From 1st International Congress on Neurobiology and Clinical Psychopharmacology and European Psychiatric Association Conference on Treatment Guidance Thessaloniki, Greece. 19-22 November 2009

Background

Abnormal protein expression of N-methyl-D-aspartate (NMDA) receptors essential subunits (NR1, NR2) and of associated post-synaptic density proteins (PSD-95, PSD-93) were observed in schizophrenic patients (post-mortem studies) [1,2]. NMDA receptors containing NR2A/B subunit associate in vivo at synapses with PSD-93 and PSD-95 [3]. In the present study, we have silenced the expression of NR1 and/or NR2 proteins in vivo with the goal of assessing the influence of that protein's alteration on the prepulse inhibition of acoustic startle reaction (PPI) and on the expression of related PSD-95/PSD-93 proteins.

Materials and methods

We used antisense oligodeoxynucleotide for NMDA-NR1 individually or in combination with NR2A or NR2B (aNR1, aNR2A, aNR2B) in the rat hippocampus and evaluated the PPI. Western blot was employed to assess the expression of affected proteins (NR1, NR2A and NR2B) and associated PSD-95/93 proteins.

Results

Changes in expression of NR1 were found. We observed a significant decrease in the hippocampi of rats affected by the combination aNR2A/aNR2B when compared with controls; yet we did not detect changes in other applications. In addition, we found significant changes in the expression of PSD-95, namely a decreased level of this protein in groups treated with NR2A or NR2B. There were no significant changes in NR2A/B and PSD-93 expression and in PPI.

Conclusions

Despite the fact, that the short term silencing of NR1/NR2 did not change PPI, there were changes in expression of PSD-95, which were connected with the NR2A/B subunit whose protein expression was not changed. This suggests that the association of PSD-95 with NR2A/B may occur in the early phase of biosynthesis.

Acknowledgements

This work was supported by grants GAČR 309/09/HO72 and MŠMT ČR 1M0517.

Author details

¹Prague Psychiatric Center, Prague, Czech Republic. ²Institute of Computer Science, Academy of Sciences, Prague, Czech Republic.

Published: 22 April 2010

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doi:10.1186/1744-859X-9-S1-S147

Cite this article as: Vrajobá et al.: Expression of NR1 subunit of NMDA receptor and PSD-93/95 in rat hippocampus affected by NR1/NR2 antisense oligodeoxynucleotide. *Annals of General Psychiatry* 2010 **9**(Suppl 1):S147.

¹Prague Psychiatric Center, Prague, Czech Republic