# P-1262 - AGING AND N-METHYL-D-ASPARTATE RECEPTOR SUBUNITS - NITRIC OXIDE SYNTHASES PATHWAYS IN NOGO-A DEFICIENT RATS

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**Introduction:** It seems that schizophrenia may arise from abnormal neurodevelopment due to aberrant neuron formation and migration and that deletion of Nogo-A (a myelin associated inhibitor of regenerative fiber growth) may lead to schizophrenia-like abnormalities in animal model. Our previous studies reported a high sensitivity of lateral analyses to reveal subtle links and recommended to apply them to validate animal models of diseases accompanied by alterations in brain asymmetry.
**Methods:** Expressions of N-methyl-D-aspartate receptor subunits (NR1, NR2A, NR2B) and activities of subunits-associated synthases of nitric oxide (nNOS, eNOS, iNOS) were evaluated in the right and left cortex of young or old Nogo-A deficient rats.
**Results:** In young controls, no marked laterality was observed in subunits/synthases excepting iNOS but correlation analyses supported links among some subunits, synthases or subunits - synthases. Drops in NR1 (bilaterally) and in NR2B (in the right side) or asymmetrical alterations in NR1 - nNOS pathway were observed in old controls. In young Nogo-A deficient rats, the increase in iNOS (in the left side) and the disruption in left NR1 - right nNOS or in right NR2A - right eNOS pathways were found. And finally in old Nogo-A deficient animals, we observed the increase in NR1 (bilaterally) and in positive correlation between right eNOS - right iNOS.
**Conclusion:** Although some changes in subunits/synthases observed in people with schizophrenia were not found in Nogo-A deficient rats, some alterations in laterality support the validity of this model.
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